

GENERAL QUESTIONS OF ANESTHESIOLOGY

Fundamentals of anesthesia

1.1. Historical information

People always wanted to overcome the sufferings caused by pain. The history of civilization left numerous documented evidences of permanent search for ways and methods of anaesthesia. The first written mention of the pain relieving medicines was found in Egypt (described in Ebers papyrus 4 - 5 thousand years ago). Much attention was paid to this problem by the doctors of ancient Greece and Rome. They used wine, mandrake root, opium, Indian hemp, henbane and thorn-apple. In the East, in the mountains of Tibet, acupuncture and massage were widely used with anaesthetic aim.

However, until the eighteenth century there was no radical ways of pain relieving. At this time, because of the fundamental discoveries in natural sciences the preconditions of new medical possibilities appeared. In 1776 chemist Priestley synthesized nitrous oxide - an anaesthetic, which is still widely used in anaesthesiology. Another chemist, Davy, on April 9, 1779 for the first time tested the effect of nitrous oxide on himself. Later he wrote: "Nitrous oxide, along with other properties, has the ability to relieve pain and thus can be successfully used in surgical operations". And only 25 years later, an English surgeon Hickman began to use the "laughing gas" in medical practice. However, this method of anaesthesia had not received adequate acceptance in Europe. At the same time in America dentist Wells began to apply nitrous oxide anaesthesia. In April 1842 his compatriot surgeon Long first used ether anaesthesia in practice. (It should be noted, that diethyl ether was synthesized by well-known chemist Paracelsus a few centuries before). With ether anaesthesia Long performed eight operations, and his observations were never published.

However, priority in the use of ether narcosis belongs to another American researcher - Morton. On October 16, 1846 at Boston University clinic he successfully administered ether narcosis during surgical removing of hemangioma in public. The operation was performed by surgeon Warren. A special Morton's contribution was a preliminary study of ether narcosis effect on animals, which was the beginning of the experimental study of general anaesthesia techniques.

Therefore, the 16th of October, 1846 is considered to be the birthday of anaesthesiology.



In few months enthusiastic followers of ether narcosis appeared in all civilized countries. At the beginning of February 1847 this type of narcosis was performed by professor F.I. Inozemtsev in Moscow clinic, two weeks later it was performed in St. Petersburg by our compatriot, surgeon Mykola Ivanovych Pirogov. This outstanding surgeon and the first anaesthesiologist played prominent role in the history of ether narcosis. He was the first to argument theoretical basis for the action mechanism of ether on the central nervous system; he proposed alternative ways of administration of ether (into the trachea, in the blood, into the gastrointestinal tract). Invaluable experience of ether narcosis M. I. Pirogov described in his monograph “On the application of sulphuric ether vapours in operational medicine” published in 1847.

In the Crimean-Turkish war (1853 – 1856) our compatriot performed hundreds of successful ether anaesthesias during surgeries on gunshot injuries.

In 1937 Guedel determined the clinical stages of ether narcosis, which are still considered to be classic.

In 1847 a prominent scientist Simpson introduced into clinical practice another preparation for narcosis - chloroform.

Since that time anaesthesiology has begun its scientific development.

For 150 years of anaesthesiology history scientists proposed and implemented in clinical practice dozens of anaesthetic preparations, both inhalation and non-inhalation, as long as various types and methods of pain relief. This stimulated development of operative surgery and allowed various range of surgical interventions in all organs and systems of the body.

Anaesthesiology is a science that studies how to protect the organism from operating injuries. It improves the well-known and develops new methods of preparing patients for surgeries, providing anaesthesia, controlling the body functions during the operation and in postoperative period.

1.2. Preparing of the patients for the surgery

During the preoperative period an anaesthesiologist should evaluate the patient's condition (main disease and comorbidities, degrees of their compensation), provide correction of health disorders, prevent complications that may occur during the operation, etc. In another words anaesthesiologist should create perfect conditions to perform anaesthesia with the largest adequacy and the least harm to the patient.

The tasks of an anaesthesiologist are:

1. Evaluation of somatic and mental condition of the patient:
 - determine the severity of main disease, concerning operation, which is planned;
 - identify comorbidities (of the cardiovascular, respiratory, digestive, nervous and endocrine systems, etc.);
 - define the psychological and emotional state of the patient (his/her attitude to future operations, anaesthesia, etc.).
2. Preoperative preparation of the patients.
3. Determination of the anaesthetic risk level.
4. Choice of the best anaesthesia method.
5. Postoperative treatment.

In the preoperative period it is essential to clarify the nature of operation: planned, urgent or emergency:

- a) in case of planned surgeries with decompensated comorbidities there should be an adequate therapy performed at first; If necessary ask other specialists to take part in the treatment;
- b) if an operation is urgent the main vital parameters should be stabilized in short term, further correction should be made during the operation;
- c) in case of emergency the patient is transferred to the operating room as soon as possible, as anaesthesia and surgery are critical factors in saving his/her life. Stabilization of vital functions is achieved on the operating table.

During the examination of the patient special attention should be paid to the condition of

the central and peripheral nervous system (mental and emotional lability, sleep disorders, the presence of anxiety and fear, paresis and paralysis). Constitution of the body, anthropometric data (weight and height) should be determined. The attention is paid to the subcutaneous fat and its distribution, peripheral veins.

While examination should be assessed: the skin and its colour, temperature, humidity and turgor of tissues (particular attention is paid to the capillary refill - press the nail of the patient for 5 seconds and if the white spot after pressing will not disappear in 2 seconds microcirculation is violated); anatomical features of the upper respiratory tract: the width of mouth opening, the size of oral cavity, presence of dentures, caries teeth, type of bite, the size of the tongue, size of tonsils, size of the nasal passages; shape of the neck and its size, the size of the thyroid gland.

Reserves of respiratory system are determined by spirometry tests (breath-holding after maximal inspiration and expiration – tests of Stange and Hensch). Normally they are 50 - 60 sec. and 35 - 45 sec., respectively. If necessary, more detailed study called spirometry is made.

The breathing rate should be determined and the chest should be examined (palpation, percussion and auscultation).

While assessing the state of the cardiovascular system, it is necessary to auscultate the heart, measure blood pressure and evaluate the pulse. In case of cardiac rhythm disorders, it is necessary to measure pulse deficit (the difference between the heart rate and the pulse rate on the radial artery per minute). An electrocardiogram should be made in order to make a detailed study of the disorder.

Examination of the digestive system should start from the tongue, which gives information about the degree of organism dehydration (dry tongue, marked buds, available longitudinal grooves), the severity of stomach inflammation (the tongue is coated), convulsions in anamnesis (scars on the tongue from biting), manifestations of avitaminosis and fungal diseases ("geographic", raspberry tongue). Also the inspection, palpation, percussion and auscultation of the abdomen should be made. Always check the symptoms of peritoneal irritation and muscular defence of the anterior abdominal wall. Ask the patient about the distension of the abdomen, nature, and frequency of defecations.

The urinary system should be assessed by palpation of the kidneys and lower abdomen (projection areas of the bladder), check of Pasternacki symptom. The frequency, volume and nature of urination, colour and odour of the urine should be determined. In urgent and emergency surgery with suspicion of renal insufficiency the bladder should be always catheterized in order to control the urination during surgery.

Don't forget to assess the probability of thrombosis: identify peripheral veins of lower extremities, swelling and pain of the ankles. Laboratory tests and instrumental examinations are also very important. The required minimum of these examinations depends on the urgency of an operation.

In emergency surgery as soon as possible (often right during the operation) the following

analyses are made:

1. Clinical blood test.
2. Clinical urine analysis.
3. Blood group and Rh identification
4. ECG .
5. Glucose control.

Urgent operational interventions allow to perform the listed above diagnostic minimum is necessary in the preoperative period and also require:

6. X-ray examination of the chest.
7. Examination of the patient by a therapist (paediatrician) or a required specialist.

In preparing patients for planned operational interventions the functions of other organs and systems are examined in a more detailed way:

8. Biochemical analysis of the blood (bilirubin, urea, creatinine, protein and its fractions, electrolytes, coagulogram, transaminase, cholinesterase, etc.).

9. Functional tests to determine the degree of compensation of the respiratory, cardiovascular, central nervous systems, organs of detoxification and secretion (according to the indications).

10. Special instrumental investigations (endoscopy, angiography, ultrasound examination, scanning, etc.).

After you found a disease, which requires preoperative correction, treat it together with other leading experts and a responsible doctor. Duration of the therapy is limited on the one hand, by the nature and degree of compensation of this disease, on the other hand – by emergency of planned surgery.

2. Preoperative preparation of the patients (premedication) is divided into previous and immediate.

Previous premedication is a treatment given to the patients hours before the surgery (usually in the evening before the invasive procedure). It includes therapy of different dysfunctions as well as sedative treatment.



One of the obligations of the anaesthesiologist is to provide psychological and emotional comfort to the patient before the surgery. To achieve this aim use the following:

- psychological effect of medical personnel - soothing, honest conversation of an anaesthesiologist with the patient, including explanation of the basic stages of future operation and anaesthesia, provides the sense of a confidence and develops belief in favourable outcome of the treatment;

- sedative and analgesic therapy (therapeutic doses of benzodiazepines and sleeping pills for example).

A full, deep sleep and psycho-emotional balance of the patients before the surgery are very important for prevention of undesirable reactions of the vegetative and endocrine systems. They allow to create the best background for the uncomplicated introduction and providing of anaesthesia with quick end of narcosis.

Preparation of the gastrointestinal tract includes one obligatory condition - "empty stomach" (in order to prevent aspiration of gastric contents into the lungs). The patients should:

- follow the light diet during 2 – 3 days before the operation;
- not to eat at least 6-8 hours before the surgery;
- take H₂blockers (ranitidine, cimetidin, etc.) before an operation to reduce gastric juice production and decrease its acidity;
- undergo cleansing enemas (in the evening and in the morning before an operation, however not before all surgeries; this method plays an important role in detoxification of the organism and soon restoration of the intestinal peristalsis in the early postoperative period;

In cases of urgent surgical interventions stomach should be clean with inserted gastric probe 1 hour before the operation; give 200 mg of cimetidine solution intravenously and 15 - 20 min. before the anaesthetic introduction ask the patient to drink 15 ml of 0.3 M sodium citrate solution.

Those steps will minimize the risk of vomiting, regurgitation and aspiration of the gastric

contents into the airways (one of the most common and severe complications of anaesthesia).

Immediate premedication in planned surgery is performed 30-40 minutes before the intervention. The classical scheme includes such medicines (given i/m):

- a) peripheral M-cholinolytics (0.1% atropine sulphate solution or 0.1%, metacin solution, dose: 0.01 mg / kg);
- b) antihistaminic drug (1% dimedrol solution or 2.5%, pipolfen solution, 1-2 ml of 2% suprastin solution);
- c) narcotic analgesics (1ml of 2% promedol solution or morphine hydrochloride, dose: 0.4 mg / kg).

In urgent operations premedication medicines are often used intravenously (right before induction). If there are special indication you can include into the typical premedication other medicines, such as: tranquilizers (2-4 ml of 0.5% Diazepam solution, 0.25% droperidol solution, etc.), non-narcotic analgesics (2 ml of 50% analgin solution, 5 ml of baralginum solution, etc.), steroids (60-90 mg of prednisolone solution), etc.

You should avoid rapid intravenous administration of antihistamine preparations, tranquilizers and narcotic analgesics in patients with deficiency of circulating blood volume due to the high probability of hypotension!

With properly chosen and performed premedication the patient is calm and a bit sleepy before the operation and to the operating-room he/she is transported on a trolley.

3. Evaluate the anaesthetic risk before each surgery. In Ukraine we traditionally used the classification of risk degrees proposed by V. Holohorskyi in 1982. It includes:

I. Somatic condition of the patients:

1. The patients without organic pathology or with local disease without systemic disorders.
2. The patients with light or moderate systemic disorders associated or not associated with surgical pathology, which moderately violate general condition.
3. The patients with severe systemic disorders, associated or not associated with surgical pathology that significantly violate vital functions.
4. The patients with the most severe systemic disorders associated or not associated with surgical pathology, which are life-threatening.
5. The patients in critical condition who can die within 24 hours with or without the surgery.

II. The severity of operative intervention.

- A. Small operations on the body surface and hollow organs (uncomplicated appendectomy

and hernial plastic, haemorrhoidal surgeries, amputation of fingers, etc.).

B. Operations of average size (amputation of the limb segments, complicated appendectomy and hernial plastic, peripheral vascular surgery).

C. Large operations (radical operations on the organs of the chest and abdomen, enlarged limb amputations).

D. Operations on the heart and main vessels.

E. Emergency surgery.

For example: a young patient without comorbidities is prepared for planned biliary tract surgery (calculous cholecystitis). The degree of operational risk is 1C. However, biochemical examination has revealed high level of glucose, and diabetes mellitus was diagnosed. Thus the degree of operational risk is 2C. In case of emergency operations of the same patient, the risk degree will be 2CE.

Now ASA (American Society of Anaesthesiologists) classification is widely used.

The tasks of medical staff in preparing of the patient to the surgery are:

- to create best conditions for psychological comfort of the patient. The appearance of the medical personnel should reassure the patient. Neat uniforms, accurate fulfilment of various manipulations, calm voice, encouraging smile and soothing conversation of a nurse or doctor are very important for the patient and develop confidence in a successful outcome of the treatment;

- to monitor changes of the patient's state (colour of the skin, consciousness, body temperature, character of the respiration, blood pressure and pulse, urination volume, etc.);

- to perform clearly, professionally and skilfully the necessary manipulations (getting of i/v access; taking of blood samples for laboratory tests; intravenous, intramuscular and subcutaneous injections; insertion of gastric and urinary probes, etc.);

- to follow the rules of asepsis and antisepsis in the department;

- to perform premedication in a ward or in the operating room.

Remember, that some medicines must not be mixed in one syringe, like narcotics and atropine. After effective premedication patients can feel sleepy, so as it was mentioned above they are transported on a trolley to the operating room (don't forget to control their condition during the transportation).

1.3 Anaesthesia and its preparations.

Anaesthesia is a set of methods applied to patients during operations and painful manipulations in order to protect their life and health.

Anaesthesia includes the following components:

- drug-induced sleep;

- pain relieve;
- neuro-vegetative protection;
- muscle relaxation;
- adequate ventilation;
- optimal level of blood circulation;
- normalization of metabolic processes in tissues.

Narcosis (from 'narkosis' - get torpid) is the process of temporary reversible depression of the central nervous system caused by pharmacological agents.

Analgesia (from Greek 'analgesio' - no pain) – absence of pain sensitivity.

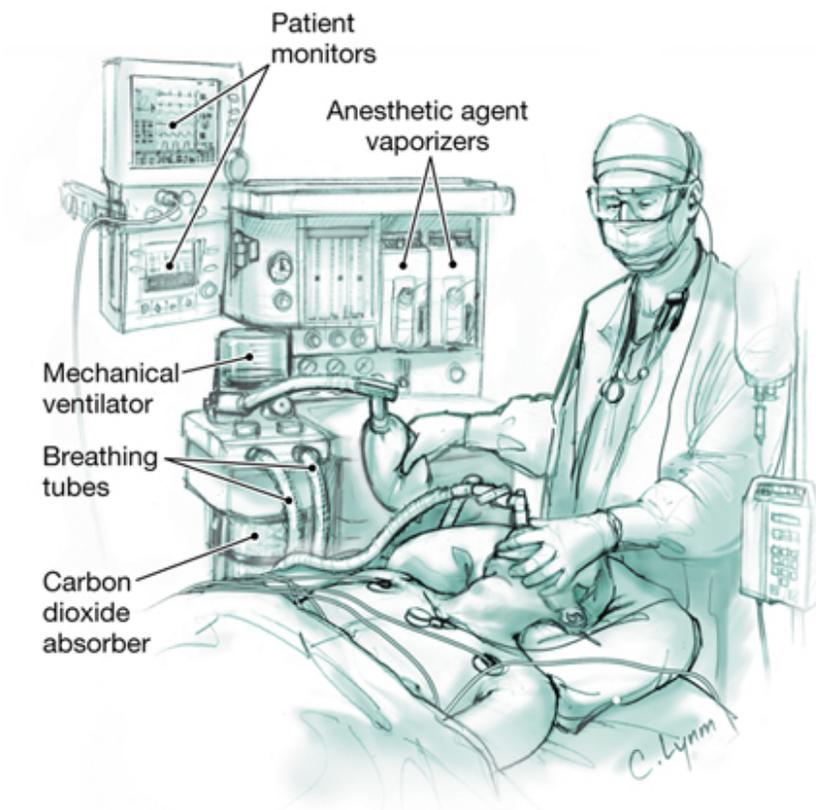
Anaesthesia (from Greek 'anaestasio' – loss of sensation) - a set of methods used to remove all kinds of sensitivity.

Anaesthetics - pharmacological agents, which eliminate sensitivity.

Anaesthetics can have general (agents for narcosis) or local action (medicines for regional anaesthesia).

Depending on the way of admission anaesthetics are divided into inhalational and non-inhalational (mostly intravenous).

During inhalation anaesthesia gases or vapour of anaesthetics enter the patient's through the airways and lungs (anaesthesia machines are connected with the mask, endotracheal tube, tracheostomy tube, etc.) This type of anaesthesia is characterized by the simplicity of the method and high degree of control. The most common anaesthetics are: nitrous oxide, sevoflurane, isoflurane, xenon. Ether and phtorotan have now historical meaning.



Non-inhalational anaesthesia can be delivered through intramuscular injection, orally or per rectum, but the most common way is intravenous administration.

Those first ways are less popular in adult anaesthesiology, however still popular in paediatric anaesthesiology.

Characteristic feature of intravenous total anaesthesia is its difficult control: the anaesthesia becomes dependable on the biological transformation (neutralization and excretion) of anaesthetic in the body.

Intravenous anaesthetics include barbiturates (hexenal, thiopental sodium) sodium oxybate, propofol, ketamine, etc. In the past viadril, propanidid, altezin were also used. There are combined methods of general anaesthesia, like: neuroleptanalgesia, ataralgesia, central analgesia and others.

Unlike general anaesthesia (narcosis), during regional anaesthesia patients are conscious.

Depending on the site of local anaesthetic administration (dicaine, novocain, lidocaine, bupivacaine, etc.), regional anaesthesia is divided into:

topical (surface); infiltrative; plexus and single nerves blockades; neuroaxial blockades - epidural, spinal anaesthesia.

Inhalation anaesthesia implies respiratory introduction of anaesthetics into the body. It can be performed through open, semi-open, semi-closed and closed circuits.

In the open circuit the patients inhale and exhale into the atmosphere; in semi-open they inhale oxygen or oxygen-air mixture, and exhale into the atmosphere. In closed and semi-closed circuits the patients inhale oxygen only from gas cylinders, and exhale completely (in closed circuits) or partially (in semi-closed circuits) into the chemical absorber (absorbs carbon dioxide). Respiratory mixture with eliminated carbon dioxide can be used for breathing again.

1. Nitrous oxide (N₂O) is a gas stored in liquefied state under the pressure of 50 atmospheres in the cylinders of grey colour (in French-speaking countries, these tanks are painted blue!). From 1 kg of liquid N₂O about 500 litres of colourless inflammable stable gas are formed. To prevent gas freezing at the outlet of the container it is necessary to use a special type of reductors (ribbed). Gas supply of the anaesthetic machine is performed through a high-pressure hose. Dosage control is made with special flow measurement devices (rotameters).

Nitrous oxide should be always combined with oxygen in the anaesthetic mix, otherwise patient can die; usually a ratio of 1:1, 2:1, 3:1 are used.

Note that false connection of nitrous oxide supply hose to the oxygen supply inlet is dangerous for the patient!

This gas does not cause unconsciousness, has a weak anaesthetic and mild analgesic (pain-relieving) properties. It is not toxic for the patient and it can be combined with other inhalation and intravenous anaesthetics.

Harmful effects of nitrous oxide are noticeable when its concentrations in the respiratory mixture is over 75 - 80% (in these cases, the oxygen content in the mixture is lower than 20%, which is life-threatening).

Nitrous oxide is used in combination with other anaesthetics to enhance their effects during various anaesthesias, in the form of oxide-oxygen mixture - for pain relief during attacks of angina pectoris, myocardial infarction, complicated and uncomplicated childbirth.

2. Anaesthetic ether was widely used in anaesthesiology for about 150 years. Although currently ether is not used it is a classic of anaesthesiology.

Ether is a colourless volatile liquid with a specific odour. It is produced in orange bottles of 100 and 150 ml. Boiling point of ether is 37°. It is explosive!

Ether is a potent anaesthetic, which causes unconsciousness, analgesia and muscle relaxation. It has sympathomimetic effects (causes tachycardia, increases blood pressure, stimulates the activity of the salivary and bronchial glands, increases level of sugar in the blood, stimulates the respiratory centre).

In toxic doses, ether depresses the activity of the heart, liver and kidneys. It irritates the respiratory tract. Therefore, rapid increase of its concentration in the breathing mix can cause

laryngeal or bronchial spasm.

Ether is an universal anaesthetic. It can be used in open, semi-open, semi-closed and closed circuits, in combination with other anaesthetics, during mask and intubation anaesthesia. Ether became popular as an anaesthetic because of its large therapeutic range and safety.

Ether as an anaesthetic is contra-indicated for patients with hypertension, diabetes mellitus, acute and chronic inflammation of the respiratory system. During ether narcosis, it is strongly contraindicated to use an open flame and it is dangerous to use diathermy!

During the surgery we usually evaluate the course of general anaesthesia according to the classic scheme of 4 ether narcosis stages.

Clinical course of ether narcosis (by Guedel).

I. Analgesia stage lasts from the start of vapour inhalation up to the loss of consciousness. It is characterized by the gradual loss of pain sensitivity (temperature and tactile are preserved) and depression of consciousness. During this stage you can do short-time surgery and painful manipulations.

II. Excitement stage. The patient loses consciousness. Motor and verbal excitement appear. Skin is hyperaemic, breathing is deep and noisy, the teeth are clenched, the eyeballs make floating movements, the pupils are dilated, all kinds of reflexes are increased. Blood pressure is increased, as well as heart rate; even ventricular fibrillation can occur.

During this stage various surgical interventions and manipulations are contraindicated! Just control the vital parameters and wait.

With the deepening of narcosis exciting symptoms fade, cross-striated muscles relax and breathing normalizes.

III. Surgical stage (stage "of narcotic sleep") is divided into three levels:

III.1. It is characterized by quiet sleep, deep and rhythmic breathing. The pupils are narrow, their reaction to light is preserved, the eyeballs are "floating." Ciliary, swallowing reflexes and reaction to weak pain stimuli disappear.

III.2. Corneal reflex is absent, the eyeballs are fixed in the central position, the pupils are narrow, and do not react to the light, breathing is rhythmic, blood pressure and heart rate are stable. The majority of surgical interventions are performed during this stage.

III.3. It is characterized by progressive depression of reflexes and vital functions. There may be signs of toxic ether effects: the dilated pupils, lack photoreaction, depression of respiration (with predominant diaphragmatic type of breathing) and cardiovascular activity, total lack of reaction to painful stimulation.

IV. Agonal stage is extremely dangerous, so you never intentionally deepen anaesthesia this much! In case of ether overdose there is a progressive depression of respiration and cardiac activity followed by cardiac arrest.

Pre-agonal signs of ether overdose: clammy cold sweat, grey skin colour, the sphincter relaxation and spontaneous urination and defecation.

During the recovering of the patient the same stages are observed, however in reverse order.

3. Fluothane (halothane) was widely used at the end of the 20th century, however now it is not popular. It was discovered by Raventos in 1956. It is a clear liquid with a sweet odour, not explosive. It is produced in bottles of dark glass (capacity 50, 150, 250 ml).

Fluothane is characterized by severe anaesthetic and weak analgesic effect. It depresses the secretion of the salivary and the bronchial glands, extends the bronchi. The drug depresses the larynx and throat reflexes, the respiratory centre and relaxes the cross-striated muscles. It also causes myocardial depression.

Fluothane has a short therapeutic range, its overdose is characterised by hypotension and bradycardia.

During fluothane narcosis it is contraindicated to use sympathomimetics! Fluothane increases catecholamine sensitivity of the myocardium. Adrenaline, noradrenaline and other drugs of this group during fluothane anaesthesia can cause myocardial fibrillation, followed by cardiac arrest.

For the narcosis fluothane should be filled in special evaporators of anaesthetic machines, which are situated beyond the circulation circuits. This is the way of overdose prevention.

Mask fluothane-oxygen narcosis was widely used in paediatric anaesthesiology, during short-term and small traumatic surgery. As medical narcosis - (manipulation of despair) - it is used for the immediate spasm relief when there is no venous access (epileptic state, seizures in case of eclampsia, meningitis, tetanus) for the treatment of bronchial spasm in patients with asthmatic conditions.

Fluothane anaesthesia is contraindicated for the patients with low blood pressure, deficiency of circulating blood volume (massive haemorrhage), heart failure, insufficiencies of parenchymal organs (liver-kidney failure).

4. Sevoflurane (Sevoran) was first synthesized in 1969 in the USA, now it is leading among the inhalational anaesthetics. It is a clear, colourless, mobile liquid with floral odour. It is produced in bottles of 100 and 200 ml.

Sevoran can be applied for initial anaesthesia, and for its maintaining. Sevoran is easily controlled: rapid induction of patients and quick recovery after termination of anaesthetic supply are its known benefits.

Initial narcosis is characterized by minimal excitement and absence of upper respiratory tract irritation. Sevoran causes dose-dependent inhibition of respiratory function and decrease of blood pressure. Therefore, its dose should be selected individually to achieve the desired effect, taking into account age and state of the patient.



Sevoflurane is administered by inhalation in a mixture of oxygen and nitrous oxide at a concentration of 0.5-3%, using the minimum-flow anaesthesia (0,5-2 litres of oxygen per minute). It also works with barbiturates, benzodiazepines and opioid analgesics.

Easiness of control, as well as minor side effects of sevoflurane (sleepiness after a general anaesthesia, dizziness, depression of respiratory and cardiac activity, rarely - nausea, vomiting) contributed to its wide introduction in anaesthesiology.

5. Isoflurane (forane) is a colourless inflammable liquid. It has high resistance to different agents, so it can be saved without special safety measures.

The main features of isoflurane anaesthesia are rapid falling asleep of the patient without excitement phenomena and the presence of analgesic effect after waking from anaesthesia. Isoflurane does not have toxic effects on respiration and cardiovascular system, however it has quite noticeable muscle relaxant effect. During forane anaesthesia tachycardia and increased tissue bleeding can occur.

6. Enflurane (Ethrane) was first described in 1968. It is a clear inflammable liquid with a sweetish odour. It does not cause excitement, has potent anaesthetic and weak analgesic effect (two times weaker than fluothane). During anaesthesia heart rate and respiratory rate increase, blood pressure decreases. It slightly depresses reflexes of the larynx and throat. According to the safety of its use it is placed between anaesthetic ether and fluothane. It can be combined with intravenous anaesthetics.

7. Xenon.

Xenon has been allowed for medical use as a preparation for inhalation general anaesthesia since 1999. This drug belongs to inert gases, that is why it does not enter into any chemical reaction in the body and it is not subjected to biological transformation. However, it has significant analgesic and anaesthetic effects (1.5-2 times more potent than nitrous oxide).

Clinical course of Xenon narcosis: after 5-6 inhalations of narcotic concentration of xenon the first stage occurs – peripheral paraesthesia and hypalgesia. Numbness and heaviness in the legs gradually rises upwards, spreading onto the skin of the abdomen, chest, neck and the head. In 2-3 minutes appears the second stage of euphoria and psychomotor activity, that is rapidly changing into the third stage of complete analgesia and partial amnesia. With the loss of consciousness, there is the fourth stage of complete analgesia and anaesthesia (corresponding to the first surgical stage of ether narcosis). At this stage, in conditions of mono-narcosis and spontaneous respiration, it is possible to perform surgery without the narcotic analgesics. Cardiovascular and respiratory systems (gas exchange) are stable.

Analgesia occurs in case of inhalation of 30-40% mixture with oxygen, the loss of consciousness occurs during inhalation of 65-70% of mixture. Muscle relaxation is quite significant. Recovery fast: in 2-3 minutes after gas supply is turned off, the patient is entirely conscious with pleasant subjective sensations.

Xenon can be used as an anaesthetic for various surgical operations, childbirth, painful manipulations; for relieving of pain attacks and treatment of pain syndromes and depression states in all fields of medicine, especially in patients of high risk groups. It is not toxic, that is why xenon should become an anaesthetic of choice, "gold reserve" during operations of patients with high anaesthesiology risk.

Xenon is used as a single component of anaesthesia (mask and endotracheal option) or in combination with various intravenous preparations: sedatives, narcotic and non-narcotic analgesics, neuroplegics, tranquilizers, gangliolytics etc.

Xenon can also be used for treatment at pre-hospital stage (emergency and urgent medical care to relieve pain in case of myocardial infarction, angina pectoris, burn or skeletal injuries, asthma attacks with the usage a special devices (mixture of Xe: O₂ - 50:50). It can be used as an antidepressant in neurotic and stress-anxiety disorders and other neurological conditions, to relieve abstinent syndrome, in treatment of motor aphasia, dysarthria.

Non-inhalational anaesthesia occurs in case of the parenteral injection of anaesthetics. The most common way of administration is intravenous route.

1. Derivatives of barbituric acid.

	R_1	R_2	
4-218 Thiopental	$-C_2H_5$	$-\underset{\text{CH}_3}{\text{CH}}-(CH_2)_2CH_3$	(2-S)
4-219 Hexobarbital	$-CH_3$		(1-CH ₃)
4-220 Pentobarbital	$-C_2H_5$	$-\underset{\text{CH}_3}{\text{CH}}-(CH_2)_2CH_3$	
4-221 Amobarbital	$-C_2H_5$	$-CH_2CH_2CH(CH_3)$	
4-222 Phenobarbital	$-C_2H_5$		
4-223 Barbitol	$-C_2H_5$	$-C_2H_5$	

Sodium thiopental (hexenal) was first described by Lundy in 1934. It is a powder of green (sodium thiopental) or white (hexenal) colour. It is produced in bottles of 0.5 and 1 gram. Immediately prior to anesthesia, this powder is dissolved with distilled water (isotonic solution of sodium chloride) up to 1% concentration.

Barbiturates have narcotic effect (cause unconsciousness) with minor pain relief effect. Therefore, they are used for initial narcosis, during the painless manipulations. For anaesthesia during barbituric narcosis it is necessary to introduce additional narcotic analgesics (morphine hydrochloride, phentanyl).

Easy, pleasant for the patient falling asleep and lack of excitement at the same time led to widespread use of sodium thiopental and hexenal in the practice of an anaesthesiologist.

In addition, due to strong anticonvulsant effect barbiturates are used for removal of cramps in patients with epilepsy, tetanus, meningitis, eclampsia etc.

Derivatives of barbituric acid have parasympathomimetic properties. Therefore, at the use of them the following complications are possible: cough, bronchial spasm, laryngeal spasm. They inhibit the activity of the respiratory centre, with fast intravenous administration of large doses of sodium thiopental or hexenal respiratory arrest is possible.

Barbiturates may be used only when the apparatus for artificial ventilation of the lungs is available!

A nurse – anaesthetist should prepare anaesthetic for anaesthesia and give it to the patient by the anaesthesiologist's instruction. She should dissolve in aseptic conditions 1 gram of sodium thiopental in 100 ml of 0.9% solution of sodium chloride (up to 1% concentration). After venous puncture and managing system for infusion therapy (only by the anaesthesiologist's instruction)

she should introduce 1-2 ml of anaesthetic intravenously. Waiting for 1 - 2 minutes, she should determine if the patient has sensitivity to thiopental sodium, if the solution is not under the skin. Then she should introduce the rest of the drug. The dose of anaesthetic is especially individual (from 4 to 8 mg / kg of body weight). Barbituric narcosis lasts 15 - 30 minutes. To continue narcotic sleeping the patient should be administered 10 - 20 ml of 1% solution or use other anaesthetics.

2. Sodium oxybutyrate (GOBA).

Sodium salt of gamma-oxybutyric acid (GOBA) was first described by H. Laborit in 1960. It is produced in ampoules of 10 ml of 20% solution. It has sedative and narcotic effect. Anaesthetic effect is not significant.

Sodium oxybutyrate - derivative of gamma-aminobutyric acid - a natural metabolite of the organism. Therefore, its use does not cause toxic effects in patients. Involving in metabolic processes, GOBA normalizes cellular respiration, transmembrane potential, moving potassium from blood plasma into the cells, due to that excitation processes of cells of the heart, brain, etc. decrease.

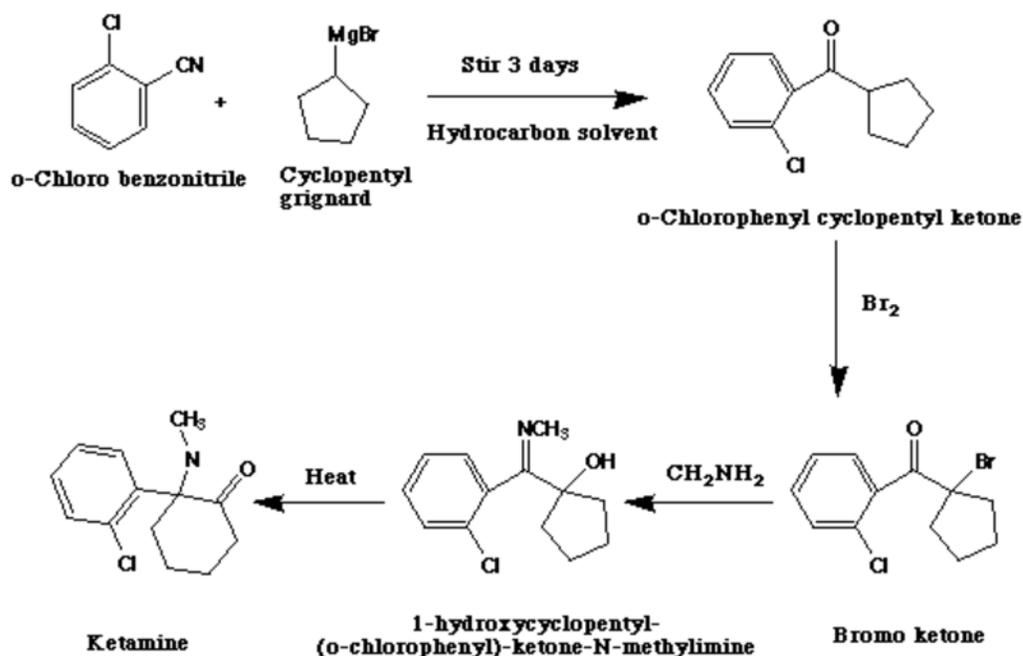
Sodium oxybutyrate also belongs to the group of anti-hypoxants, so it is widely used in intensive care of patients with disabilities of the cardiovascular system (in shock), with severe hypoxic conditions (after having survived clinical death, brain damage, in obstetrics). Anti-hypoxic dose (20-40mg/kg body weight) effectively affects tissue respiration, optimizing it. GOBA at this dose does not influence on consciousness of the patient.

As a means of anaesthesia, it is a method of choice in accompanying diseases of the cardiovascular system, the liver, the kidneys, diseases of the endocrine organs, in neurosurgical operations and in the pathology of pregnancy. In the intravenous method, it can be used intramuscularly, orally, rectally.

For anaesthesia GOBA is used at a dose of 70 - 120 mg / kg of body weight. A nurse - anaesthetist introduces it, following doctor's instructions, intravenously slowly by 10 ml. In order to prevent spasms, GOBA should be introduced together with barbiturates. For this 20-milliliter syringe should be filled with sodium oxybutyrate (10 ml) and sodium thiopental (10 ml). This mixture can be administered quickly. After repeated (3-5 times) injection of the mixture in 5 - 10 minutes the patient falls asleep, which resembles the physiological sleep. A rare, deep breathing, muscle relaxation, inhibition of reflexes are observed. Haemodynamics is not disturbed. Narcotic sleep lasts for 1 - 1,5 hours, its after-effect - up to 5 hours. At this time the patient needs to be looked after because retraction of the tongue, disorder of the lung ventilation may occur.

3. Ketamine.

Synthesis of Ketamine



Ketamine (ketanest, kalipsol, ketalar, velonarkon, petar) was described by Karsen and Domino in 1965. This group of drugs, has the ability to depress the function of some parts of the central nervous system and increase the activity of others, causing a so-called dissociative narcosis. Ketamine is a clear liquid, unstable to light, produced as 1 or 5% solution in bottles of darkened glass (10 ml), or in ampoules (2 ml). Ketamine is a strong anaesthetic, causing a deep sleep with moderate anesthesia. Due to selective stimulation of certain parts of the brain (limbic structures), during ketamine mono-narcosis patients may have visual hallucinations. In addition, ketamine causes a slight neuro-vegetative inhibition, relaxation of the cross - striated muscles with preserved tendon reflexes. It increases blood pressure, heart rate, slightly depresses respiration and stimulates the production of cerebrospinal fluid (liquor).

According to its characteristics ketamine is a tool of choice in patients with deficiency of circulating blood volume (hypohydration, hypotension, and various types of shock, collapse), in the need of anaesthesia with preserved spontaneous breathing. Due to its universal administration (intravenous, intramuscular and rectal) this anaesthetic is widely used in paediatric anaesthesiology.

Ketamine can be administered intravenously at doses of 2 - 3 mg / kg or intramuscularly at doses of 7-15 mg / kg. To avoid unwanted hallucinatory effect it is used with tranquilizer solution (1 - 2 ml of 0.5% sibazon solution).

Ketamine can be combined with various inhalation and non-inhalation preparations in the combined narcosis.

Ketamine anaesthesia is contraindicated for patients with hypertension, liquor hypertension (traumatic brain injury, epilepsy), with mental disorders.

4 Propofol (diprofil, diprivan, isoprivan).



The anaesthetic is produced in the form of white fat emulsion in 20 ml ampoules or bottles of 50 and 100 ml. 1 ml of preparation contains 10 mg of active substance. It can be administered intravenously with different infusion means (optimally - 5% glucose solution), except for blood and plasma (danger of deemulgation).

Propofol has a strong hypnotic and sedative effect, slight anaesthetic effect. Therefore, it should be used in combination with analgesics.

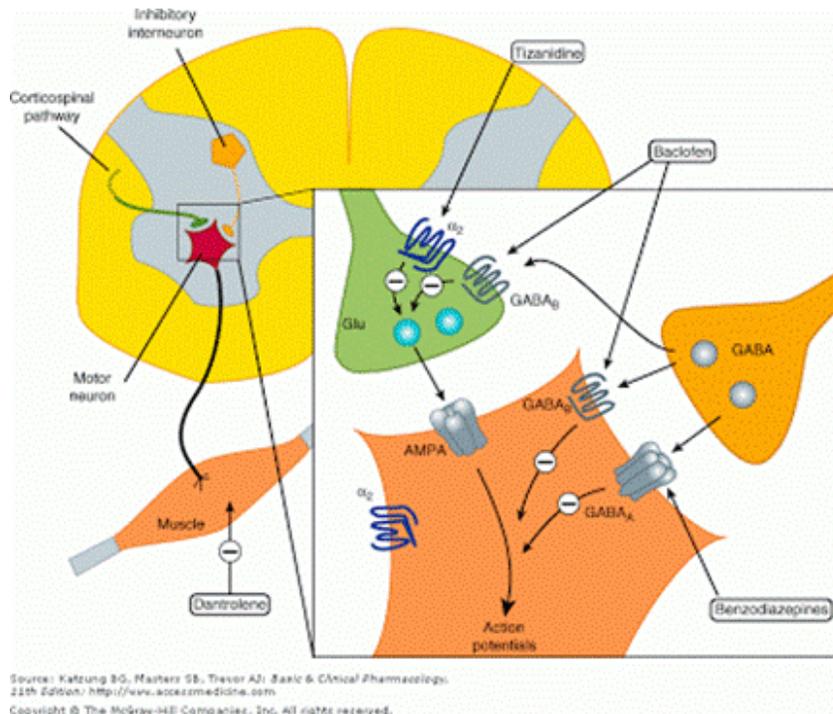
Intravenous injection at a dose of 2-2.5 mg / kg (15-20 ml) causes sleeping in 40 seconds (disappearance of the ciliary reflex). The duration of narcotic sleep is 20 - 30 minutes. Diprivan does not cause excitement, somewhat weakens the breathing, has a weak hypotensive effect without tachycardia. Inhibition of laryngeal reflexes and throat allows to apply laryngeal mask (a means of maintaining airway of the respiratory passages in self breathing and performing artificial ventilation of the lungs).

Diprivan is used for initial narcosis and as a component of balanced anaesthesia during long-term operations. This anaesthetic is injected again in 20 - 30 minutes at a dose of 100 mkg / (kg. min) - (fractional, by 2 ml, followed by a decrease at dose of $\frac{1}{4}$ each time of next administration or continuously, using a dispenser).

After diprivan narcosis (even long-term) the patients wake up fast during 10 minutes. This feature, as well as normalization of haemodynamics during laparoscopic surgery, the minimum number of complications and contraindications make propofol a drug of choice in modern anaesthesiology.

Combined general anaesthesia with muscle relaxants.

To achieve general anaesthesia most effectively use a combination of several anaesthetics should be. In these cases, the dose of each anaesthetic is reduced (and, respectively, their toxicity), therapeutic effect is increased. The use of muscle relaxants and muscle relaxation by themselves still reduce the need for anaesthetic, facilitating optimal performance of surgeons in the depth of the wound.



Muscle relaxants are divided into depolarizing and anti-depolarizing. The first are short-term, causing febrile twitching of the facial muscles, muscles of the trunk, the limbs, the diaphragm, followed by relaxation of the muscles and vocal cords up to 4-6 minutes. Ditylin (succinylcholine) is produced in ampoules of 5 ml of 2% solution, usually used for tracheal intubation at a dose of 2 mg / kg (7 - 10 ml) intravenously.

Anti-depolarizing muscle relaxants cause long-term (from 25 min. to 1,5 hours) muscle relaxation in patients without prior fibrillation, so they are used during prolonged operation on the organs of the abdominal cavity, the chest, etc. They include tubocurarine chloride, pavulon, arduan, tracrium. Arduan is produced in bottles, 4 mg of dry substance. Before the administration muscle relaxant should be dissolved in 2 ml of isotonic solution of sodium chloride, applied at a dose of 0.04 mg / kg (1.5-2 ml) intravenously, every 40 - 45 minutes the drug is re-administered in the half dose.

As a multi component intravenous narcosis with muscle relaxation and artificial

ventilation of the lungs neuroleptanalgesia, ataralgesia, central analgesia are often used.

Neuroleptanalgesia (NLA). For NLA neuroleptic droperidolum and narcotic analgesics phentanyl are used either alone or in combination with other inhalation or non-inhalation anaesthetics.

Droperidolum is produced in bottles: of 10 ml transparent colourless liquid, which contains 25 mg of preparation (0.25% solution). The drug has a marked antishock effect: it blocks extra excitement of the sympathetic-adrenal system, normalizes the activity of the reticular formation, relieves spasm of arterioles (alpha adrenergic influence on the system of microcirculation), does not cause unconsciousness. Duration of the drug action is up to 3-4 hours.

Phentanyl is produced in ampoules of 2 ml of 0.005% solution. It has short-term analgesic effect (up to 30 minutes), which allows to manage anaesthesia by minimizing the risk of drug inhibition of the respiratory centre in patients in the postoperative period.

When using the classic technique of NLA is administered intravenously 0,25-0,5 mg/kg of droperidolum and 0.005 mg/kg of phentanyl (6-8 ml of droperidolum solution and 6-8 ml of phentanyl solution for 1 hour anaesthesia). To cause unconsciousness the respiratory mixture of dinitrogen oxide and oxygen in the ratio 70%: 30% is used. After the introduction of muscle relaxants the patient is intubated, artificial ventilation of the lungs is performed. To maintain narcosis 1-2 ml of droperidolum and phentanyl should be administered every 20-30 minutes. Tachycardia is indication for re-administration of phentanyl, hypertension for re-administration of droperidolum.

Neuroleptanalgesia is used for very weak patients with high operational risk, in disorder of haemodynamics and the need of artificial ventilation of the lungs in the postoperative period.

Ataralgesia is the way of general anaesthesia with sedation preparations (ataractics) and narcotic analgesics.

Ataractic seduxen (diazepam, relanium, sibazon) is produced in ampoules of 2 ml of 0.5% solution. It cannot be dissolved or mixed with other drugs (white precipitate is formed!).

For narcosis seduxen is administered at a dose of 0.3-0.5 mg/kg and intravenously mixture of dinitrogen oxide with oxygen (2:1) is used. Anaesthetic effect is obtained by introducing phentanyl fractionally or other narcotic analgesic (morphine hydrochloride, dipidolor, pentazocine) at doses as for NLA. To achieve muscle relaxation muscle relaxants are used, and to ensure adequate breathing - artificial ventilation of the lungs is used. During the surgery by indications (tachycardia, increased blood pressure) seduxen is re-administered (usually in 1,5 hour) and phentanyl (in 20-30 min.)

The advantage of this narcosis is its minimal adverse effects on various organs and systems so that ATA is used in patients with accompanying pathology of the heart, the liver, the kidneys etc.

Central analgesia is the way of narcosis in which anaesthesia, hyporeflexia and neuro-

vegetative protection in patients is reached by administration of large doses of narcotic analgesics (1% morphine hydrochloride solution up to 20-30 ml, 0.005% of phentanyl solution - up to 100 ml). Other components (relaxation, unconsciousness, support of gas exchange and blood circulation) are used by general rules.

Central analgesia is indicated for patients with severe heart failure, combined heart defects, in case of prolonged artificial ventilation of the lungs in the postoperative period.

1.4 The work of anaesthetic team in the operating room

The success of anaesthesia and of surgery largely depends on accurate and coordinated actions of anaesthesiological team (a doctor-anaesthesiologist and nurses anaesthetists), their experience, concentration, knowledge, skills and available medical equipment.

Necessary medical equipment and instruments:

- system of centralized oxygen and nitrous oxide supply;
- anesthetic machine or artificial ventilation apparatus ("Phase", "Leon", etc.);
- equipment to monitor the functional state of the organism (cardiomonitor - for continuous ECG registration, pulse oximeter - for continuous measurement of oxygen saturation of the blood, volumeter - to monitor the volumes of inspiration and exhalation of the patient, tonometer, phonendoscope, stopwatch, etc.).
- electric pumps, laryngoscopes with a set of blades, set of intubation tubes;
- tools and details of devices: high pressure hose (to supply oxygen and dinitrogen oxide), masks for breathing apparatus, hose pipes, connectors, adapters, system of removal of exhaled gases.
- additional instruments: anaesthesiologist's forceps, clamps, mouth dilator, syringe for inflating cuff endotracheal tubes, flexible conductors for tubes, a tray for bottles, a set of probes and catheters;
- an anaesthetist's table, completed with preparations for narcosis, infusion therapy and others; system for infusion therapy, syringes, sterile rubber gloves, tourniquet, bandages.

An anaesthesiologist personally is responsible for the availability and serviceability of narcosis and respiratory apparatus and accessories, an anaesthetist –c for its asepsis and antisepsis, a table equipment and its management.

Before anaesthesia an anaesthesiologist must check:

- availability of oxygen in the system;
- working condition of narcosis and respiratory apparatus. He personally connects hoses of oxygen and dinitrogen oxide supply to the corresponding connecting pipes of dosimeters, turns on breathing apparatus, and having connected breathing circuit, checks it for hermetic. For this

purpose, he closes by his finger adapter of breathing circuit of working apparatus, following the manometer. In its indications of 300 mm. of water column dehermetisation valve must operate;

-efficiency work of electric suction machine (having closed a rubber hose by finger, follows the vacuum manometer deflection);

-working condition of laryngoscopes. After its turning on the lamp must light;

-state of cardiomonitor, pulse oximeter.

Preparing an anaesthetist's working place

While preparing the anaesthetist's table to work one should follow the order of setting instruments and medical preparation for narcosis and intensive care.

First, a table is covered with a sterile cloth. Then successively disposable syringes are put: in the lower left corner - two of 20-milliliter for 1% thiopental sodium solution (hexenal, propofol) in the lower right corner - syringes for muscle relaxants: 10 ml of capacity for 2% Dithylin solution (listenon, myorelaxant) and 2 ml for 0.2% for arduan solution. Behind them 5-milliliter syringe is located for narcotic analgesics (0.005% phentanyl solution). In the upper right corner there should be 5 ml syringe for solutions of sibazon, droperidolum, ketamine. In the upper left corner - syringes of 5 and 10 ml capacity for other drugs (ganglioblockers, clopheline, dalargin, etc.). Among them there are placed two glass vessels (of 100 ml of capacity) filled with 0.9% sodium chloride solution. In the left bowl the powder of sodium thiopental is dissolved up to 1% concentration; 0.9% sodium chloride solution in the right bowl is used for dissolving other drugs just before their intravenous introduction.

In a separate tray must be sterile gauze wipes and forceps, next to a bottle with antiseptic solution (70% of ethyl alcohol).

The desk drawer is designed to store pharmacological preparations needed during surgery, intravenous catheters, disposable system for transfusions, adhesive plaster, etc.

On the shelf under the box an anaesthetist places the bottles of infusion-transfusion preparations.

In a separate tray a set for tracheal intubation is formed: laryngoscopes with a set of blades (large, medium, small), intubation tubes of various sizes and throat mask, air-way, mouth dilator, a connector of the intubation tube, syringe for cuff blow of the intubation tube, Kocher clamp, napkins.

1.5. Complications of general anaesthesia, their prevention and treatment

Complications may occur at different stages of anaesthesia: during premedication, initial narcosis, during maintenance of narcosis and after narcosis period.

The most responsible for an anaesthesiologist stages are: giving anaesthesia, awakening

and early after narcosis period.

The complications may be associated with:

- a) the specific influence of anaesthetic;
- b) the type and method of anaesthesia;
- c) main or accompanying disease;
- d) the nature of surgery.

The basis of prevention of complications is adequate preparation of the patient for surgery and anaesthesia, and careful monitoring of patients at all stages of anaesthesia.

Complications concerning the respiratory system are with the following disorders:

- a) of airway of the air passages (accumulation of phlegm, tongue retraction, aspiration of vomit, laryngospasm, bronchospasm, etc.)
- b) of the regulation of respiration (respiratory centre depression drugs, hypoxia, etc.)
- c) of neuromuscular conduction (influence of muscle relaxants, antibiotics, disorder of electrolyte metabolism),
- g) of the lungs (pneumonia, atelectasis, pulmonary oedema, etc.).

Any disorder of respiration leads to oxygen starvation (hypoxia) and accumulation of carbon dioxide (hypercapnia).

Hypoxia. Respiratory, or hypoxic hypoxia occurs during hypoventilation, respiratory passage disorders, reducing the oxygen volume in the inhaled mixture, pulmonary pathology. Characteristic feature is decreasing PaO₂ <80 mmHg

Clinical characteristics of hypoxia. I stage of compensation (anaesthetic); II stage decrease of compensatory reactions (narcotic), III stage of decompensation (toxic).

At the first signs of hypoxia one should primarily:

- a) to listen to the respiration of the patient of both lungs,
- b) to ensure airway of the respiratory passages,
- c) to check the narcosis apparatus and oxygen supply.

Hypercapnia occurs in hypoventilation of the lungs due to respiratory depression by anaesthetics or drugs, in case of non-physiologic operating position of the patient, absence or ineffectiveness of adsorbent, large amounts of "dead space"; in the postoperative period - with inadequate anaesthesia.

Hypercapnia stimulates the sympathetic-adrenal system. The increase of blood pressure, increased salivation, bright red colour of the skin and mucous membranes, extra-systoles are observed. Dilatation of the capillary vessels leads to increased capillary bleeding, which is one of the reasons of delayed waking from narcosis and can lead to cerebral oedema.

In case of hypercapnia it is necessary to normalize pulmonary ventilation (auxiliary artificial ventilation or mechanical ventilation), to replace chemical absorber in narcosis apparatus.

Complications concerning the cardiovascular system may be caused by inadequate gas exchange, changes in circulation blood volume, weakening of cardiac activity, disturbance of peripheral circulation, changes in rheological properties, clotting and fibrinolytic systems of the blood. Changing blood pressure, cardiac arrhythmia, cardiac arrest, embolism and thrombosis are observed:

Disorder of normal rhythm and beat of heart activity. Tachycardia occurs in abnormal gas exchange and transport of blood gas, blood loss, inadequate anaesthesia, reflex stimulation of the heart, administration of atropine sulphate, ether. Manifested tachycardia may be the precursor of fibrillation of the heart ventricles. Bradycardia. Its cause is severe hypoxia, stimulation of the vagus nerve, overdose of fluothane or methoxyflurane, administration of dithylin. It may precede cardiac arrest. Arrhythmia is the result of hypercapnia, stimulation of the vegetative nervous system receptors.

Treatment – adequate ventilation, neuro-vegetative blockade, in brady-arrhythmia – the introduction of atropine.

Arterial hypertension may occur due to hypercapnia, hypoxia, painful stress, effects of anaesthetics (ketamine) and adrenomymetics.

Arterial hypotension: a) with tachycardia is developed in hypovolemia of any origin. Treatment is to restore blood circulation volume. In cases of suspected acute adrenal insufficiency hormones are prescribed. In acute heart failure that is due to myocardial infarction or lack of blood and fluids transfusion cardio-tonics are administered;

b) with normocardia - occurs in decrease of vascular tone on a background of moderate vagotonia (sodium-thiopental narcosis). Vasopressors are prescribed;

c) with bradycardia – occurs in severe hypoxia, fluothane and narcotic analgesics overdose, vagotonia.

Having determined decrease of BP, an anaesthesiologist should stop giving anaesthetic and define the cause of hypotension!

Complications concerning the digestive system.

Vomiting is caused by hypoxia, hydration, the influence of anaesthetics (on the vomiting centre and the mucous membrane of the stomach), stimulation of reflexogenic zones (root of the tongue, pharynx).

Regurgitation occurs during mechanical ventilation of the lungs in patients with "full" stomach, lowering the head end during operation at the time of fibrillation of the muscles while administering dithylin for intubation. Lack of symptoms is dangerous prior to aspiration

Complications concerning the nervous system.

Slow awakening from general anaesthesia is caused by prolonged hypoxemia and hypercapnia, anaesthetics overdose, in children – on the basis of hypothermia.

Hypoxia of the brain can be complicated by brain oedema, focal lesions, decerebration. Spasms, mental disorders are possible. Normalization of gas exchange and adequate anaesthesia provide prevention of the complication of the CNS.

As the result of compression or dilatation of the peripheral nerves (brachial plexus, fibular nerve etc.) neuritis, paresis, paralysis can be observed.

To prevent the occurrence of this pathology you should put the limbs properly and control their position during surgery and in postoperative period.

Disorders of thermoregulation. Hypothermia (spontaneous) occurs on the basis of increased heat loss and blockade of thermoregulation centres. To prevent the development of this disease, you should maintain normal body temperature.

Malignant hyperthermia occurs most often in young people and in children in postoperative period. During the operation use of muscle relaxants, fluothane and other drugs should be made with particular caution.

Anaesthesia is meant to protect the body from operating injuries, but it can also be a cause of serious reactions and complications by itself. Therefore, an anaesthesiologist should inform the patient before surgery about possible complications, side effects and consequences of anaesthesia.

Complications of anaesthesia and the effects of narcosis are divided into:

1. Common: nausea, sore throat, tremor, dizziness and disturbance of consciousness, headache, itching, pain in the back and lumbar, muscle pain, confused consciousness.
2. Uncommon: postoperative pulmonary infection, injury of the teeth, the lips, the tongue, awakening during general anaesthesia.
3. Rare and very rare: nerve damage associated with general anaesthesia, nerve injury associated with regional anaesthesia, allergic reaction (anaphylaxis), injury of the eyes during general anaesthesia, death or brain injury.

Common side effects and complications of anaesthesia.

Nausea is a frequent result of anaesthesia, which occurs in about 30% of cases in the postoperative period. Nausea is often typical to the general, than regional anaesthesia. For its prevention it is necessary to provide a patient with adequate pain relief, he/she should not be active during the first hours after surgery – to sit and get up, to drink water and to eat. Deep breath with slow inhalation of the air can decrease nausea.

Sore throat. It can vary from discomfort to severe constant pain during talking or swallowing. The mouth can be dry. These symptoms may be a few hours after surgery, and may remain for two more days.

Tremor, as the result of anaesthesia is a frequent problem because it causes great discomfort, although it is not a danger to the organism and lasts about 20-30 minutes. Tremor may occur after general anaesthesia and be a complication of epidural and spinal anaesthesia. The thermal comfort of the patient before and during surgery decreases the probability of tremor occurrence.

Dizziness and loss of consciousness. The residual effect of anaesthetics may cause decrease of blood pressure, in addition dehydration can cause the same effect, which is not so rare after surgery. Decreased blood pressure may cause dizziness, weakness, loss of consciousness.

Headache. It can be caused by a drug used for anaesthesia, the operation itself, dehydration and the patient's anxiety. Often headache disappears in a few hours after anaesthesia and is treated with analgesics easily. A severe headache may be a complication of spinal or epidural anaesthesia.

Itching. Usually it is adverse reaction to anaesthesia medication (e.g. morphine), but itching may be a manifestation of an allergic reaction.

Backache and pain in the lumbar. During the operation the patient is in a constant position on a firm operating table for a long time, which may cause "tired" back and lead to backache after surgery.

Pain in the muscles. Most often this pain occurs in young males, it is often associated with the use of muscle relaxants during anaesthesia, particularly dithylin. Pain in the muscles is the result of narcosis (general anaesthesia), it is symmetrical, often localized in the neck, the shoulders, the upper abdomen and lasts until 2-3 days after surgery.

Confused consciousness. Some patients, usually elderly, suffer from confused consciousness after surgery and anaesthesia. Their memory may deteriorate, and their behaviour may differ from typical for them usual state.

Uncommon side effects and complications of anaesthesia.

Postoperative pulmonary infection. This problem is the most common consequence of narcosis (general anaesthesia). A few simple measures will significantly reduce the risk of this complication:

- Smokers should quit smoking for about 6 weeks before surgery;
- The patients with chronic lung disease should be operated in a state of remission;
- Adequate pain relief after surgery is the key to effective breathing and ability to cough, and, consequently, to reduce the risk of pulmonary infection.

Injuries of the teeth, the lips, the tongue. General anaesthesia is a risk for teeth injury, which occurs in about 1 case in 45,000 anaesthesias. Minor injuries of the lips or the tongue are common in about 5% of cases of general anaesthesia.

Awakening during anaesthesia. Some of the patients during surgery may come to

consciousness, they can remember some episodes of the operation. It is very unpleasant complications of anaesthesia, however, it is quite a rare event, since the modern system of monitoring can prevent the development of such complication.

Rare and very rare side effects and complications of anaesthesia.

Nerve injury, as a complication of anaesthesia. This type of complication is characterized by a sense of numbness, tingling or pain. There may be a disorder of heat or cold sensitivity. Additionally, there may be a feeling of weakness or paralysis. Typically, all complaints disappear in a few days or months. Complete recovery can sometimes be delayed up to a year. The most common is ulnar nerve injury in the ulna nerve area and fibular nerve in the knee.

Nerves may be injured during epidural and spinal anaesthesia. This complication is rare and usually disappears within a few weeks or months. Cases of temporary immobilization (paralysis) of one or two extremities are very rare (1:50 000).

Reasons that may cause nerve injury:

- Nerve may be injured by a surgeon during some operations (it is sometimes difficult and unavoidable);
- The position in which the patient lies on the operating table can lead to nerve compression or tension, damaging it;
- The use of tourniquets by a surgeon to reduce the amount of blood loss during surgery puts pressure on the nerve, also contributing to its injury;
- The cause of nerve compression may be post-operative swelling of the tissues.

Allergic reaction (anaphylaxis). During anaesthesia, as well as, during the patient's stay in hospital medications may cause severe allergic reaction - anaphylaxis. The frequency of development is 1:15000 of anaesthesia. Usually, an anaesthesiologist successfully diagnoses and treats this terrible complication, however, statistically one of twenty of such severe reactions can lead to death.

Injury of the eyes during general anaesthesia. This is a rare complication of narcosis. The most frequent type of eye injury during and after general anaesthesia is damage of the cornea (1:2000 anaesthesias). This condition does not affect visual acuity, but may cause a dark point in the damaged eye. Most corneal injury is due to the fact, that during anaesthesia the patient's eyelids are not always completely closed. Consequently, the cornea becomes dry and the eyelid "sticks" to it from inside. Then, when the patient opens the eyes and corneal injury occurs.

Death or brain injury.

1.6. Postoperative care of surgical patients

After complex operations the patients are transferred to the department of intensive therapy. Medical personnel of the department should continuously look after the patients

(monitor vital functions) and provide intensive care.

During the postoperative period of patients after long hours of surgery under general anaesthesia combined with the use of muscle relaxants, a special attention should be paid to the respiratory function and the cardiovascular system activity.

The patients, who underwent complicated surgery, particularly on the digestive organs of the gastrointestinal tract, should be provided with a balanced intensive care for a long period of time until the restoration of the physiological processes of digestion.

Planned postoperative prescriptions (general regulations):

1) administration of adequate analgesia (with the use of narcotic and non-narcotic analgesics);

2) prescribing infusion therapy (at a rate of 40 ml per 1 kg of body weight per 24 hours - the regime of moderate haemodilution)

3) providing the organism with the most important electrolytes (daily need: Na⁺ – 2 mmol / kg, Cl⁻ – 2 mmol / kg, K⁺ – 1 mmol / kg, Mg⁺⁺ – 0.2 mmol / kg, Ca⁺⁺ – 0,3 mmol / kg)

4) prescribing parenteral nutrition to the patient, based on

- The necessary energy providing - 25 kcal / kg per 24 hours;

- The daily need of proteins should be 1.4 g/kg;

- The ratio of ingredients for parenteral nutrition:

proteins: carbohydrates: fat = 2: 7: 1,

- It is necessary to prescribe vitamins (at the therapeutic dose);

5) by indications, with a prophylactic or therapeutic purpose, the administration of antibiotics;

6) maintaining therapy, aimed at preventing decompensation of the main and probable accompanying pathology;

7) symptomatic therapy;

8) physiotherapy treatment and exercise therapy;

9) providing different laboratory and instrumental investigations.

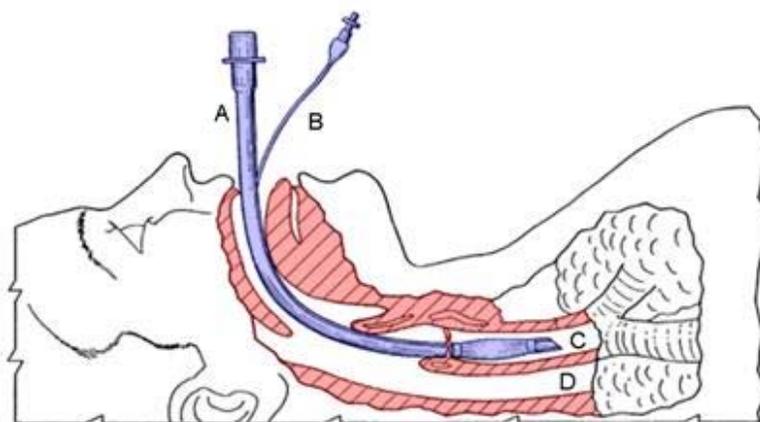
Example. A 47-year-old patient K., 72 kg, survived operation on stomach cancer. Surgery - gastrectomy. Accompanying pathology: chronic bronchitis of a smoker.

The patient was prescribed:

1) for pain - 1% promedol solution, 1 ml intramuscular;

- 2) 0.9% sodium chloride solution - 800 ml, drip intravenously;
- 3) 10% solution of glucose - 400 ml
 - 7.5% potassium chloride solution - 20 ml
 - 25% magnesium sulphate solution - 3 ml
 - insulin solution - 12 units;to administer intravenously, drip, 3 bottles (1270 ml) during 4 hours;
- 4) 8% polyamines solution - 400 ml;
- 5) 20% intralipid solution - 500 ml, drip intravenously during 3 hours;
- 6) 20% glucose solution - 400 ml
 - insulin solution 24 units;to administer intravenously, drip slowly during 2 hours;
- 7) Vitamin C, 5% solution, 5 ml intravenously, 3 times (at 8.00, 16.00, 24.00)
 - vitamins B1, B6 - 1 ml intramuscular;
- 8) ceftriaxone 1g intravenously twice a day (at 10.00 and at 22.00);
- 9) Steam inhalation with sodium hydrocarbonate solution, coltsfoot infusion;
- 10) exercise therapy, breathing exercises with the creation of positive pressure on exhalation;
- 11) Laboratory tests: blood analysis, hematocrit, biochemical blood analysis (glucose, proteinogram, coagulogram, amylase, bilirubin, Na, K, Cl, options of acid-base state, the toxicity of blood serum), urinalysis.

1.7. Medical manipulations and operations



Tracheal intubation

Indications: the necessity of artificial ventilation of the lungs in patients with combined anaesthesia using muscle relaxants, providing respiratory care to patients with acute and chronic respiratory failure, making manipulation (bronchoscopy) in tracheal-bronchial tree, performing cardiopulmonary-cerebral resuscitation.

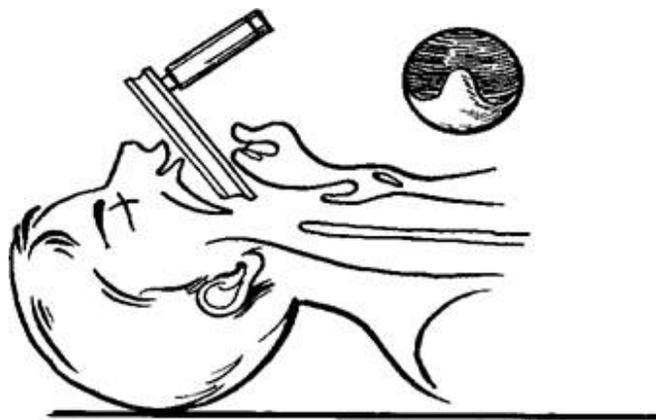
Necessary equipment: A laryngoscope with a set of blades, intubation tubes of different sizes, electrical or mechanical suction machine, apparatus for artificial ventilation of the lungs, atropine sulphate solution, preparations for anaesthesia and muscle relaxants, gauze napkins, phonendoscope.

Methods of intubation. The trachea is often intubated by mouth with laryngoscope in direct laryngoscopy.

For prevention of pathological reflexes the patient is previously injected intravenous 0.1% atropine sulphate solution (0.4 - 0.5 ml) and anaesthetic (e.g. sodium thiopental) for narcosis.

Mask way is used for additional artificial ventilation of the lungs with oxygen, along with administration of muscle relaxation solution.

After full muscle relaxation an anaesthesiologist opens the patient's mouth by his right hand fingers (grasps the incisors of the upper jaw with the second finger tip and moves the chin down with the third finger), he takes the working laryngoscope by his left hand and gently introduces its blade into the mouth cavity.

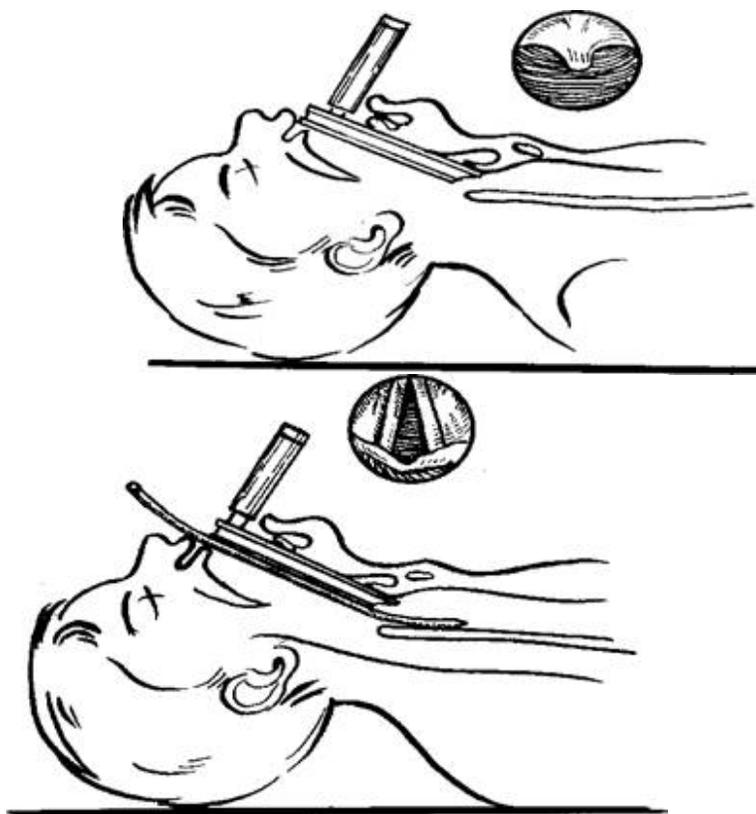


Pic. 1.1 A laryngoscope is putted into the oral cavity.

The root of the tongue is pulled up, the soft palate and the tonsils are visible

Without efforts, carefully in order not to injure soft tissues, he moves the blade deep and medially into, pushing the tongue to the left. The entrance in the throat in and back wall of the throat are visible. With further introduction of the blade the larynx-throat opens with overhanging layer of its epiglottis. An anaesthesiologist displaces it upwards (when using direct blade takes the epiglottis free end and raises it up; while using indirect laryngoscope puts it under the basis of the epiglottis and pressing shifts it upwards). The entrance to the trachea opens, formed by the glottis of inverted trapezoid form. Its side walls are the vocal

cords.



Pic. 1.2-3 The epiglottis is raised by the laryngoscope's blade.

The glottis is visible

By his right hand an anaesthesiologist gently introduces the intubation tube of appropriate size into the glottis, so that its blowing cuff was hidden immediately behind the vocal chords.

The intubation tube is connected to the triple connector of respiration circuit of the apparatus of artificial ventilation of the lungs. Tube cuff of the tube is blown with air, due to that it obturates free lumen of the trachea and seals system "the lungs - apparatus of artificial ventilation of the lungs".

He checks accuracy of the intubation tube position in the trachea, listening to the breathing with phonendoscope on both sides of the chest. In all regions of the lungs vesicular breathing the same sonority should be available (without abnormal noise).

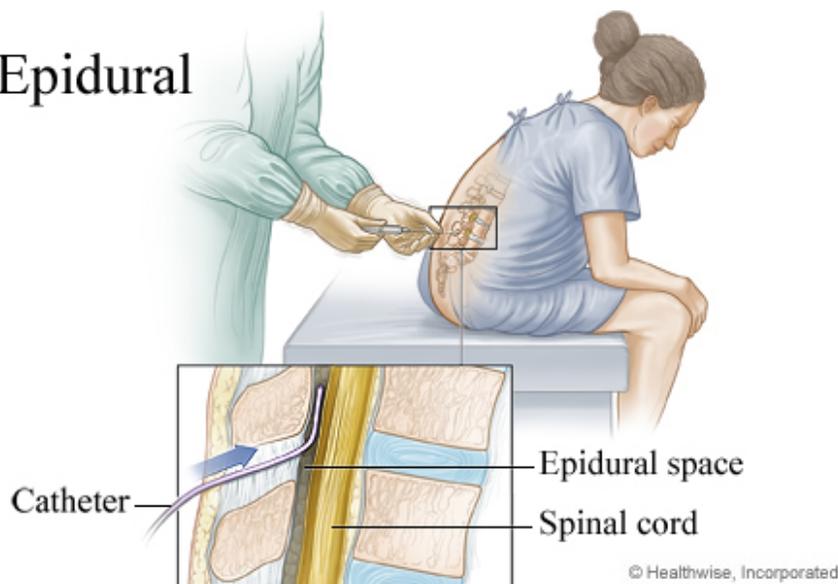
The intubation tube is fixed by adhesive plaster or a bandage, around the patient's head.

In the case of clinical death for effective ventilation of the lungs the trachea should be immediately intubated, without prior administration of anaesthetics. At presence of the mucus, the blood, the gastric contents or foreign bodies in the mouth and the throat an anaesthesiologist performs sanitation of the respiratory passages before the intubation, using

electrical or mechanical suction machine or a gauze tampon at the clips.

Epidural anaesthesia.

Epidural



Indications: anaesthesia in surgery on the abdomen, the pelvis and the lower extremities; intensive therapy of pathological conditions in which a temporary medication denervation of appropriate segments of the body is necessary (asthmatic status, myocardial infarction, pulmonary oedema, acute pancreatitis, paralytic intestinal obstruction, frostbite of the lower limbs etc.).

Picture 1.4 Choice of the place for puncture in epidural anaesthesia, depending on the required level of anaesthesia

Necessary equipment: a needle with a stylet for puncture of epidural space (Tyoxi), Dyufo needle, a needle for intramuscular injections, syringes of 2 ml of capacity with a light piston moving and of 10 ml with the adapter to the catheter, epidural catheter, Kocher clamp, sterile gauze tampons, a solution of ethanol, adhesive plaster, the solution of local anaesthetic.

Methods of administration

The equipment is placed on the sterile surface (a nappy) of the manipulation table.

The patient is placed on the operating table lying on his side or sitting, bending his body maximum (moving the head to the knees).

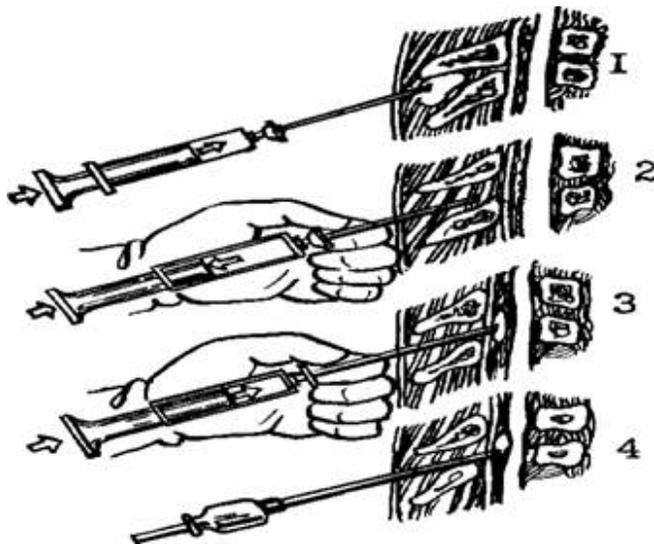
A place for puncture is chosen, according to the indications (the required level of anaesthesia).

The operating field and the anaesthesiologist's hands are washed according to the rules of asepsis and antisepsis.

Infiltration anaesthesia of soft tissues from the puncture along the needle (introducing it in the sagittal direction, in the middle between the osseous processes of the spine) is administered.

Piercing the skin and subcutaneous fat tissue with Dyuflo needle, creating a channel of 2 - 2.5 cm in depth.

Along the channel in the sagittal direction Tyoxi needle with a stylet is introduced in a depth of 3 - 4 cm, piercing upper osseous and intra-osseous ligaments.



Picture. 1.5 Sequence in which needle penetrates the tissues

moving it into the epidural space.

1 - the introduction of anaesthetic into the subcutaneous tissue;

2 – the needle - between osseous ligaments;

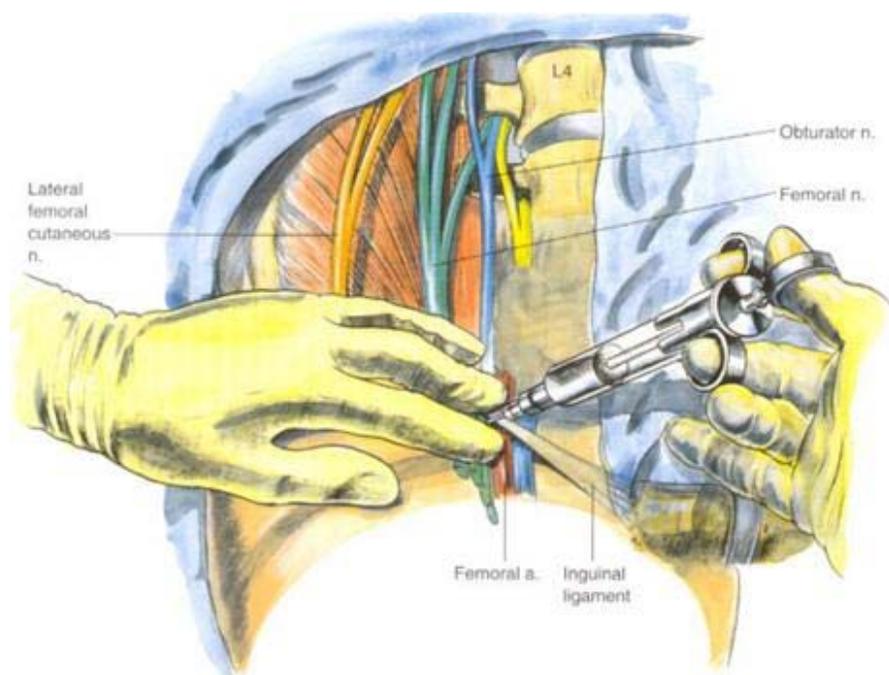
3.4 - the tip of the needle is in the epidural canal, the anaesthetic is injected easily (3), and it does not flow (4).

A 2 ml syringe is filled with isotonic sodium chloride solution, so that it contained a small air bubble. A stylet is removed from the Tyoxi needle and the syringe should be attached to its cannula. Continue to push gently the needle, while easily pressing the syringe piston. Monitor the air bubble. After passing the yellow ligament with the needle "a downfall" is felt: the syringe piston moves freely forward with the slightest effort of the anaesthesiologist, an air bubble is not deformed. The syringe should be carefully disconnected, leaving in the cannula a hanging drop of isotonic solution. The patient is asked to take a deep breath. Drawing of the drops into the cannula confirms that the tip of the needle is in the epidural space.

If one-stage anaesthesia is expected, the patient is injected into the epidural space a test-dose of anaesthetic first (3 - 5 ml 2% lidocaine solution), and in the absence of symptoms of spinal anaesthesia – in 5 minutes - a full dose of anaesthetic.

In case of a long-term anaesthesia the epidural space is catherized, moving the catheter through the Tyoxi needle to a depth of 7 - 8 cm. The needle is carefully removed, an aseptic bandage is put on the place of the puncture, and the catheter is fixed to the skin by adhesive plaster. A syringe with adapter is attached to the outer end of the catheter. First a test dose is administered and then, in the absence of symptoms of spinal anaesthesia - the rest of the required dose of anaesthetic.

Conductive anaesthesia.



In large traumatic operations (femur amputation, under-knee leg amputation) the most reasonable is the use of methods of conductive anaesthesia by blocking the sciatic and femoral nerve in the proximal regions.

The rear (proximal) blockade of the sciatic nerve (according to Rai).

Indications: In case of large operations (usually amputation of the lower third of the thigh), especially when the patient in some reason cannot be placed on the side or on the abdomen.

Necessary equipment: a 10-centimeter needle type UP 20G cut on 15-30 degrees; an anaesthetic - 20-30 ml of 1% xylonest solution (or mepivacaine, or lidocaine).

Anatomical orientation: a large trochanter of the femur, a sciatic tuber.

Methods of administration. The patient's position: on the back, the assistant lifts up the patient's leg, bending it at 90 degrees in the hip and knee joints. An anaesthesiologist marks the line between the large trochanter and the sciatic tuber, strictly in the middle of which, perpendicular to the skin introduces the needle, pushing it into the cranial direction.

At a depth of 5-10 cm during electrical stimulation (0.3 mA / 0.1 ms) foot bending appears (fibular nerve stimulation) or its extension (tibial nerve stimulation);

Blockade of the femoral nerve in the inguinal region (by Vinnie Rosenblatt).

Indications: In case of large operations (amputation of the lower limb).

Necessary equipment: a 5-centimeter needle type UP 18G; an anaesthetic - 20-30 ml of 1% xyloest solution (or mepivacaine, or lidocaine).

Anatomical orientation: the inguinal fold, the femoral artery.

Methods of administration. The patient's position on his/her back, the lower limb is slightly drawn aside and rotated outward. The place of injection: 2 cm below the inguinal fold and 1.5 cm outside of the artery. A needle with electric stimulator is injected at an angle of 30 degrees into the skin in cranial direction to the sense of a double downfall on the passage of two fascias. Motor response is proved by contraction of the quadratus femoris muscle and the patella tingling during electrical stimulation 0.3 mA / 0.1 ms

The distal sciatic nerve blockade (by Meyer).

Indications:

- Anaesthesia during operations on the foot and the ankle joint;
- Treatment of chronic pain, postoperative analgesia of the tissues located distal to the knee joint;
- Sympathicolise in diabetic gangrene, disorder of the peripheral circulation and in treatment of wounds of the legs and the feet, which do not heal for along time.

Necessary equipment: a 5-10 centimeter needle type UP 22G; an anaesthetic - 20-30 ml of 1% xyloest solution (or mepivacaine, or lidocaine).

Anatomical orientation: the popliteal fold, fossa poplitea, arteria poplitea.

Methods of administration. The patient should be placed on the side of a healthy limb, which is bent in the knee and thigh joints. The injured limb should be straightened out; between the legs a small pillow is placed. Side protuberances of the knee are fixed by a thumb and middle finger (two bottom corners of the triangle), and by the index finger - top of an equilateral triangle, which limits the upper half of the popliteal fossa. The point of injection should be determined, which is 1-2 cm outside of the top of the triangle, near the medial edge of the tendon of the thigh biceps muscle. A needle is introduced at an angle of 30-45 degrees into the skin in the cranial and slightly medial direction to a depth of 4-6 cm. In response to electrical stimulation dorsal or plantar flexion of the foot occurs, indicating close to the sciatic nerve location of the needle.

Remarks.

The sciatic nerve is always outside of the popliteal artery.

The distal sciatic nerve blockade is particularly effective for prolonged anaesthesia. For

this purpose the needle 19,5 G and catheter 20G should be used, which is injected to a depth of 4-5 cm cranially from needle cut. The anaesthetic is administered at a speed of 6 ml/hour, with optimum use of continuous administration of anesthetic by infusion pump.

1.8. Control tests and tasks

1. Preparations of sodium thiopental are produced

A - in ampoules (10 ml of 20% solution);

B - in bottles (1 g, 0.5 g);

C - in ampoules (10 ml of 1% solution);

D - in ampoules (10ml of 20% solution);

E - in bottles of 4 mg.

2. Arduan - is

A – a non-inhalation anaesthetic;

B – a depolarizing muscle relaxant;

C – anti-depolarizing muscle relaxant;

D -a narcotic analgesic;

E - an inhalation anaesthetic.

3. In administering neuroleptanalgesia are used:

A- droperidolum and seduxen;

B - phentanyl and seduxen;

C - phentanyl and droperidolum;

D - morphine and sodium thiopental;

E - phentanyl and ketamine.

4. In administering ataralgesia are used:

A - droperidolum and seduxen;

B - phentanyl and seduxen;

C - phentanyl and droperidolum;

D - morphine and sodium thiopental;

E - phentanyl and ketamine.

5. Diprivan is produced

A - in ampoules (10 ml of 20% solution);

B - in bottles (1 g, 0.5 g);

C - in ampoules (10 ml of 1% solution);

D - in ampoules (10ml of 20% solution);

E - in bottles of 4 mg.

6. Signs of fluothane overdose:

A - tachycardia;

B - hypertension;

C - hypotension and bradycardia;

D – comma, asystolia;

E - maximum constriction of the pupils.

7. At what stage of ether narcosis the pupils' reaction to the light disappears:

A - I

B - II

C - III1

D - III2

E - IV

8. Ether is expedient to be administered to patients with:

A - hypotension due to blood loss;

B - acute liver failure;

C - bronchitis;

D - acute renal failure;

E – diabetes mellitus.

Task 1

A 45-year-old patient G., is prepared for planned surgery on the stomach ulcer. Case history: diabetes mellitus, daily receives 28 units of insulin. At the time of examination glycaemia has been compensated. Objectively: the respiratory and the cardiovascular systems are in the normal state. Increased indices of urea (9,6 mmol / l) and creatinine (0,12 mmol / l).

Determine the degree of operating and anaesthesiological risk.

Task 2

During administering mask narcosis an anaesthesiologist noted the patient's sudden inspiration dyspnoea with inability to make an active inspiration. The patient's face became

cyanotic, the neck veins became swollen, the pupils became dilated.

Specify the complication, its probable causes and sequence of nurse (anaesthetist) actions.

Task 3

What preoperative preparation does a 73-year-old patient S. need? He has been admitted to the surgical department with clinical signs of adhesive intestinal obstruction. On examination of the patient hypotension (BP -90 and 60 mm Hg.), tachycardia (heart rate - 112 per minute) have been revealed

Task 4

During intravenous anaesthesia the patient's breathing became discontinuous, noisy, "gurgling": from the mouth gastric content appeared. What has happened? Determine the sequence of emergency aid.

Task 5

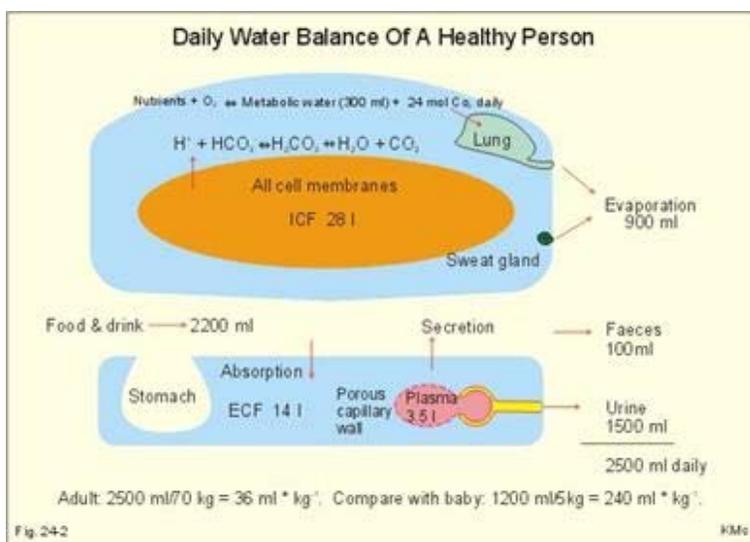
While performing tracheal intubation, an anaesthesiologist observed stomach contents in the mouth of the patient, which slowly moved between the vocal chords into the trachea.

Determine the type of complication, its cause and probable course of the disease, and emergency aid that is necessary for the patient.

Task 6

An anaesthesiologist performed intubation to the patient and connected the intubation tube to the breathing apparatus. With air supply there appeared typical "gurgling" sound. Simultaneously, in the patient began to project epigastric area of the abdomen, breathing in the chest was not available.

Determine the type of complication and urgent aid.



Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobin concentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW * (Na_p - 140) / 140 ,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, $0,6 * BW$ volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

9.4 Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 BW$$

K- potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

$0,6 \cdot BW$ – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) \cdot 0,2 \cdot BW,$$

Na_d - sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

$0,2 \cdot BW$ – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonic overhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 \text{ BW},$$

Cl_d - chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chlorine solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6 \text{ BW},$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

$0,6 * BW$ – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d – calcium deficiency, mmol

Cl_p – plasma calcium concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorbition.

9.5 Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO_2 . Normally pCO_2 of arterial blood is 36-44 mm Hg. Hypercapnia (pCO_2 increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO_2 less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H^+ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is $0 \pm 1,5$. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H⁺ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H⁺ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patient, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patient, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patient, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patient, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patient, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration

88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

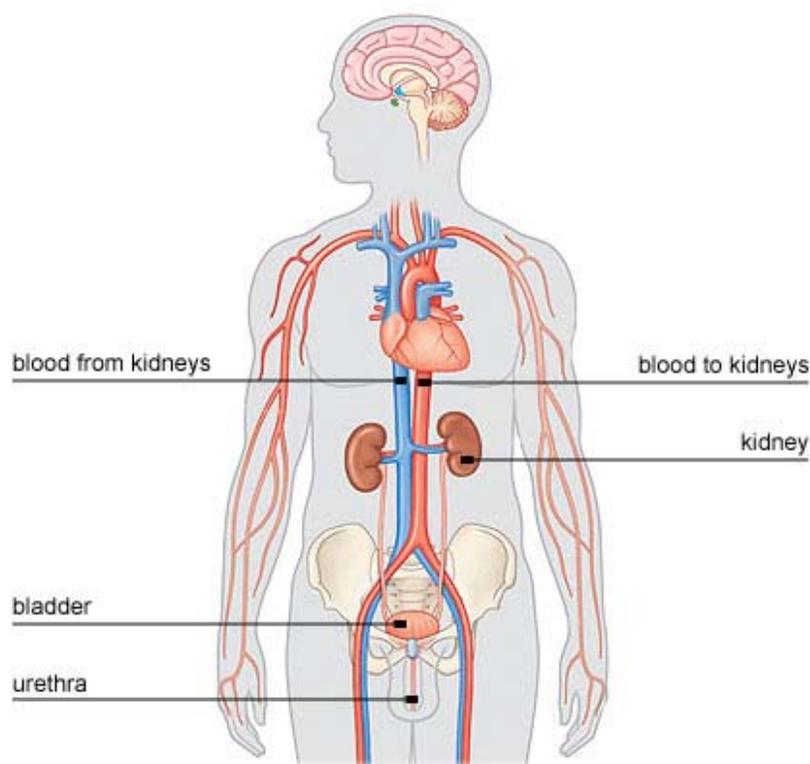
Task 7.

Patience, the patient of 50 with body weight 75 kg, was transported to the admission unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow breathing. Blood tests show: pH 7,18, pCO_{2a} 78 mm Hg, pO_{2A} – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patience, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, pCO_{2a} 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

Violations of homeostasis and their correction.



9.1 The importance of the water to the organism.

Life on earth was born in the water environment. Water is a universal solvent for all the biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the atrial walls, baroreceptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals reveal into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution). This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.

9.2 Importance of osmolarity for homeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of

another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hypoosmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.

9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;

- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobin concentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

Ht_p – patient's hematocrit, l/l,

BW – body weight, 0,2*BW – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

V_{H₂O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, 0,6*BW volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

9.4 Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K- potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

0,6*BW – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 \text{ BW},$$

Na- sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

0,2*BW – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonic overhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 \text{ BW},$$

Cl_d- chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat hyponatremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chloride solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hypernatremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hypernatremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

$0,6 * BW$ – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d – calcium deficiency, mmol

Ca_p – plasma calcium concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorbition.

9.5 Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompansated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO₂. Normally pCO₂ of arterial blood is 36-44 mm Hg. Hypercapnia (pCO₂ increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO₂ less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H⁺ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is 0±1,5. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung

ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H⁺ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H⁺ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

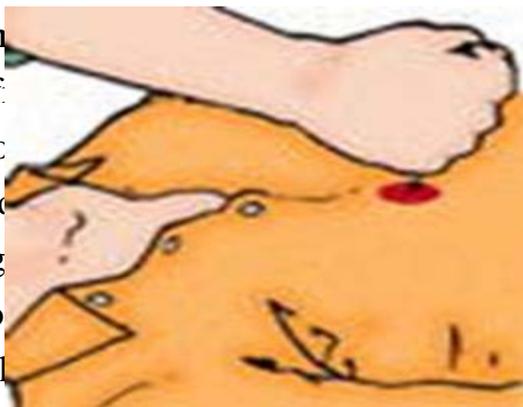
BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Cardiopulmonary

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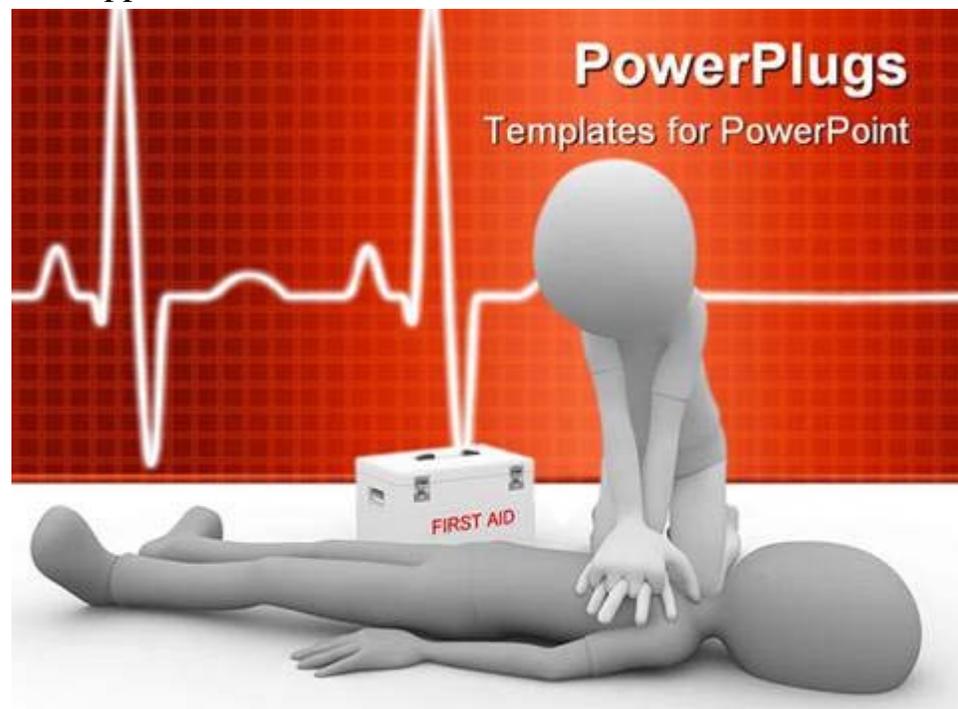


Fig. Importance of CPR

Intensive care and resuscitation in many cases may prevent and eliminate energy and substrate deficiency, which appear during the terminal state and consequently save the organism from death.

Intensive care is a complex of methods, which allows a temporary replacement of vital functions. These methods are used to prevent the adaptation mechanisms exhaustion and to avoid the terminal state appearance.



Fig. Checking of patients condition

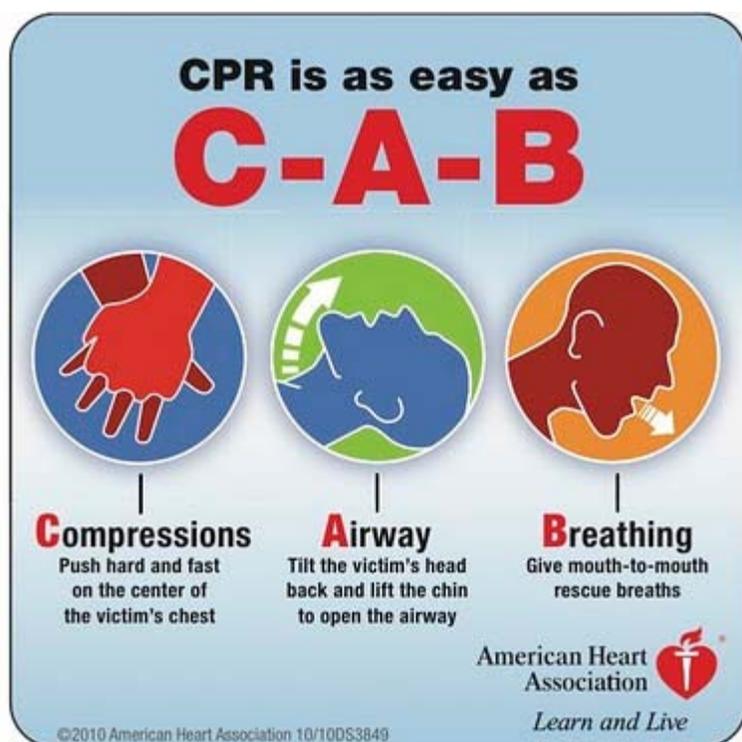


Fig Method of CPR

Reanimatology is a science about vitalization of the organism, prevention and treatment of the terminal states (according to V. Negovski).

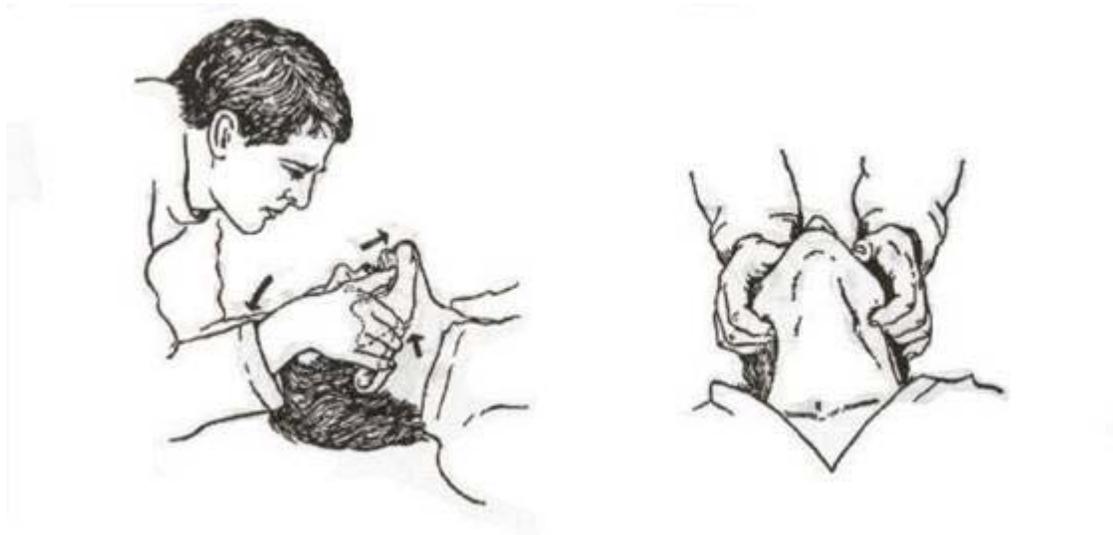


Fig. Artificial lung ventilation

Human being for existence needs continuous entry and consumption of oxygen and elimination of carbonic acid. Those processes are provided with the coordinated functioning of respiration and circulation under the control of the central nervous system. These 3 systems are so called “triple gates of death” (lungs, heart and brain). Arrest of vital functions (death) might be sudden (accidents) or quite predictable consequence of aging or an incurable disease.



Fig. Cleaning of oral cavity

The whole process of dying might be divided into next stages: Preagony. Physiologic mechanisms of vital activity are deeply exhausted: central nervous system is depressed (coma is possible);

heart sounds are weak, pulse is thready, systolic blood pressure is lower than the critical level of 70 mm Hg; external respiration is weak and not effective, tidal volume and respiratory frequency are inadequate; functions of parenchymal organs are violated. Preagony can last for minutes, hours or even days. During this time condition of patient becomes worse and finally everything ends with a terminal pause. Patient faints, blood pressure and pulse become hard to measure, respiratory arrest appears and reflexes are lost.



Fig. Artificial lung ventilation

Terminal pause ends within a minute and final stage – agony begins. According to the complete exhaustion of superior control centers of the CNS lower centers (bulbar respiratory and vasomotor centers, reticular formation) are getting more active. Muscular tone, reflexes and external respiration (chaotic, with auxiliary respiratory muscles participation) are restored.



Fig. Position of hands

The pulse is palpated over the main arteries; systolic blood pressure may rise up to 50_70 mm Hg (due to temporary renewed vascular tone). At the same time irreversible cell metabolic changes take place: reserves of high_energy substances are burnt out and in 20_40 seconds clinical death appear.



Fig. Artificial lung ventilation

In quite a long list of pathological cases (drowning, electrical or lightning injuries, strangulated asphyxia, communication accidents, myocardial infarction, etc.) clinical death appears suddenly, without any previous sings of dying.

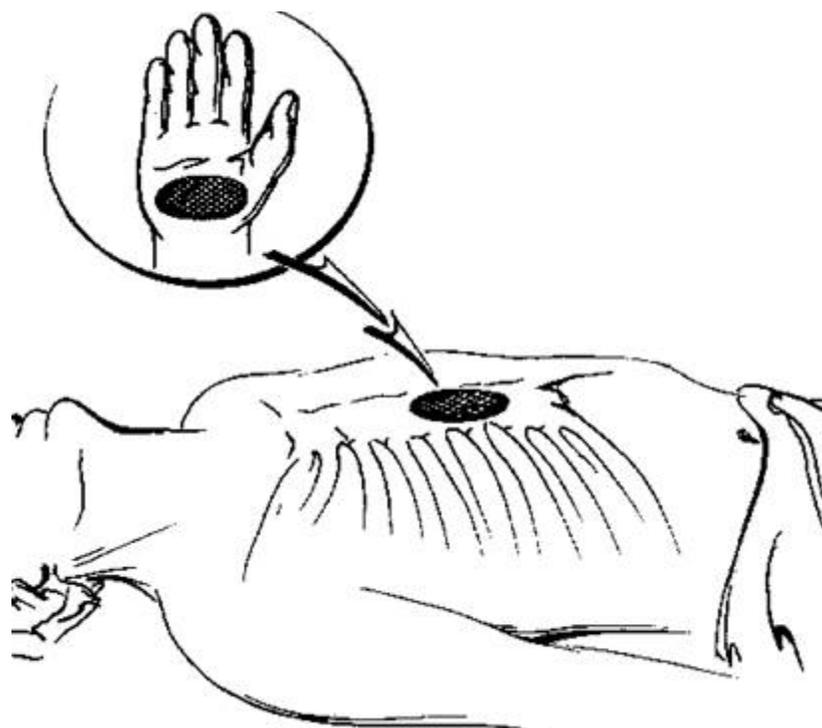


Fig. Position of precordial beat

Main sings of clinical death are:

1. Lack of pulse over the main arteries (carotid, femoral arteries)
2. Persistent pupil's dilatation with a lack of photoreaction.
3. Lack of unassisted ventilation.

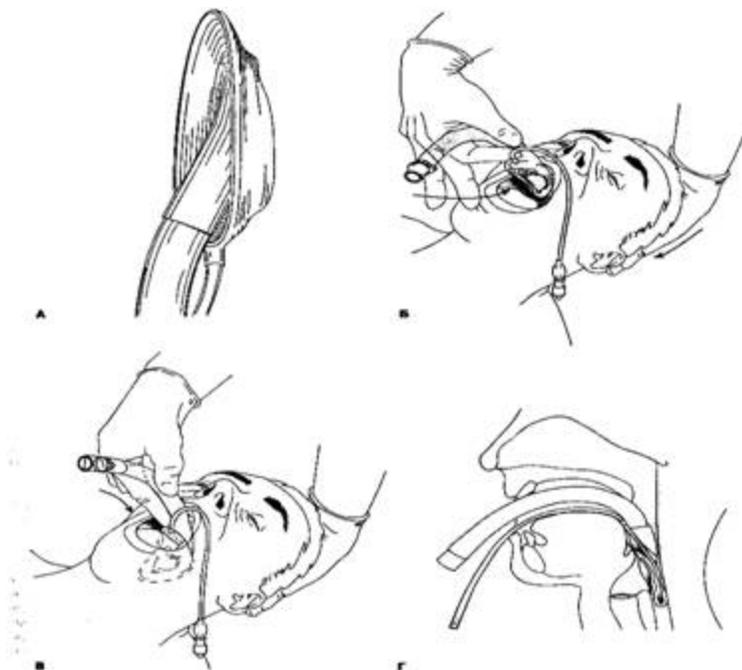


Fig. Laryngeal mask like alternative method of intubation

Additional sings of clinical death:

1. Changes of the skin color (gray or cyanotic)
2. Unconsciousness

3. Lack of reflexes and muscular atony.

The effectiveness of resuscitation is greatly affected by the temperature of the environment and the duration of dying.



Fig. Position of hands during CPR

Normally clinical death caused by a sudden cardiac arrest in case of normal environment temperature lasts nearly 5 minutes. In case of hypothermia – 10 minutes and more.



Fig. Heart massage

The longer process of dying lasts, the shorter clinical death is and thus lower are the chances of positive resuscitation outcome.



Fig. Heart massage

Biological death appears as a result of irreversible changes of the whole organism, especially of the CNS changes.

Stages and phases of resuscitation.

Resuscitation is a complex of actions, which prevent irreversible changes and restore vital functions of an organism in a state of clinical death. A person, who conducts these actions is called a rescuer.

The final goal of resuscitation is to bring back life of full value to a patient after clinical death. This task might be realized only with immediate, professional and sequential measures.



Fig. steps of CPR

Nevertheless always care about your own safety, as your duty is to help, not to increase the number of victims. Pay attention to the conditions in which clinical death appeared, make sure you are not in danger, use gloves and eyewear if they are available. Of course final decision about priorities belongs to you, but every biological liquid contacting your skin and mucosa is a potential infection source. If you feel unwilling to perform rescue breathing mouth_to_mouth or physically it is impossible to ventilate the patient for some reason at least do chest compressions.



Fig. New recommendation for performing CPR

According to the modern level of resuscitation

knowledge blood flow is the most important target of CPR.

The first stage of resuscitation is basic life support. It is

conducted by a rescuer who is not obligatory a health care professional, but a witness who acquired basic life support skills.

Fig. Precardial beat

After clinical death is stated (try not to evaluate respiration (B)

and heart action (C) more than 10 seconds) a rescuer should

immediately start basic life support (optimal position of patient who is on the flat surface lying supinely).

Successful cardiopulmonary resuscitation is based on three pillars:

I. Airways (A). To make

ABC check upper airways free use triple

method of Peter Safar: 1. Open the mouth of the patient and empty the oral cavity, if necessary, from foreign bodies and liquids such as vomit, sputum, false jaws, blood cloths, etc (using your finger or forceps with surgical drape).

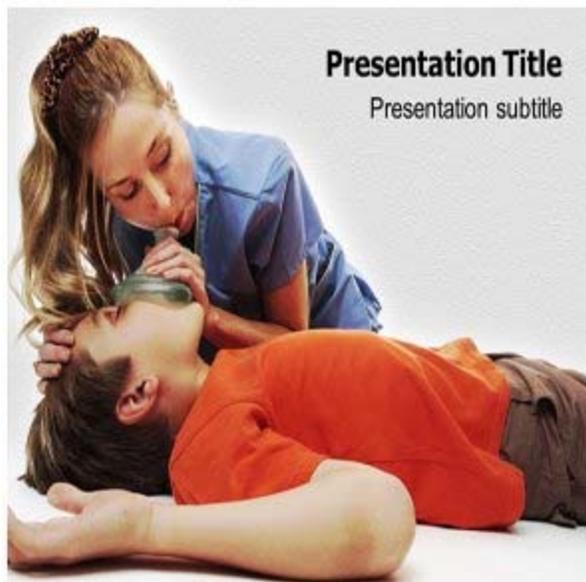


Fig. Ventilation through mask

2. Tilt the head backwards (remember, that in case of each trauma patient we always suspect neck injury, so tilting and sharp neck moves should be rather avoided). After this in most cases upper airways become conductive (soft palate and tongue are not blocking air passage any more).

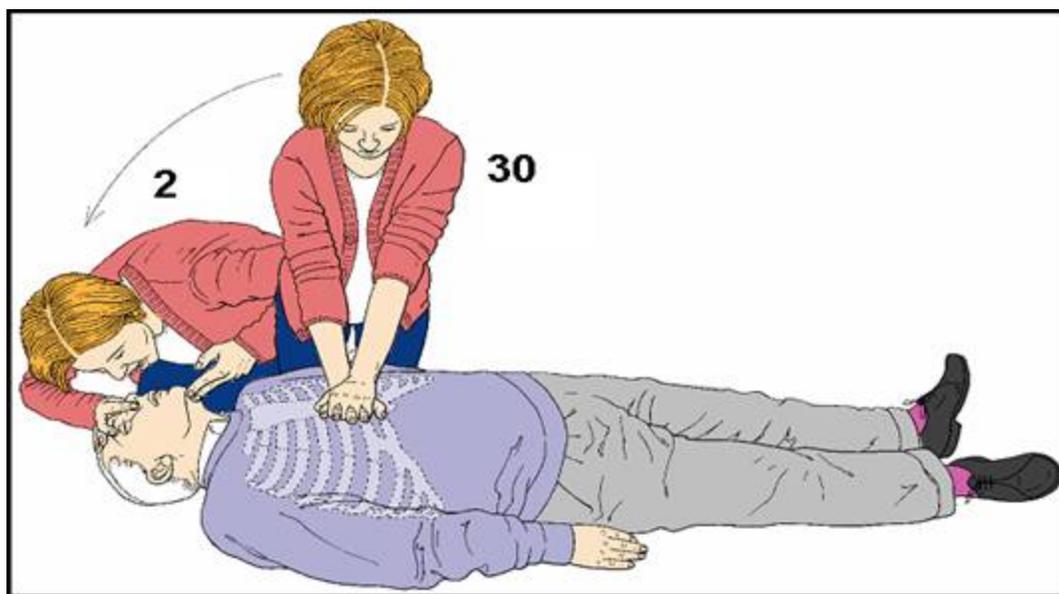


Fig. Artificial lung ventilation and heart massage



Fig. Artificial lung ventilation

3 . Thrust the jaw forward. In all cases this part of Safar method provides final air passage.



Fig. Defibrillation

You can also use simple airway adjuncts such as oropharyngeal and nasopharyngeal airways.

II. Breathing (B). Respiratory support in conditions of BLS is usually mouth_to_mouth ventilation. If only there is a chance use devices for pre_hospital ventilation: pocket resuscitation masks of different types or at least handkerchief. Place closely your mouth

over that of the patient and make a normal exhale (volume 600_800 ml). Remember to keep the airways conductive using the methods

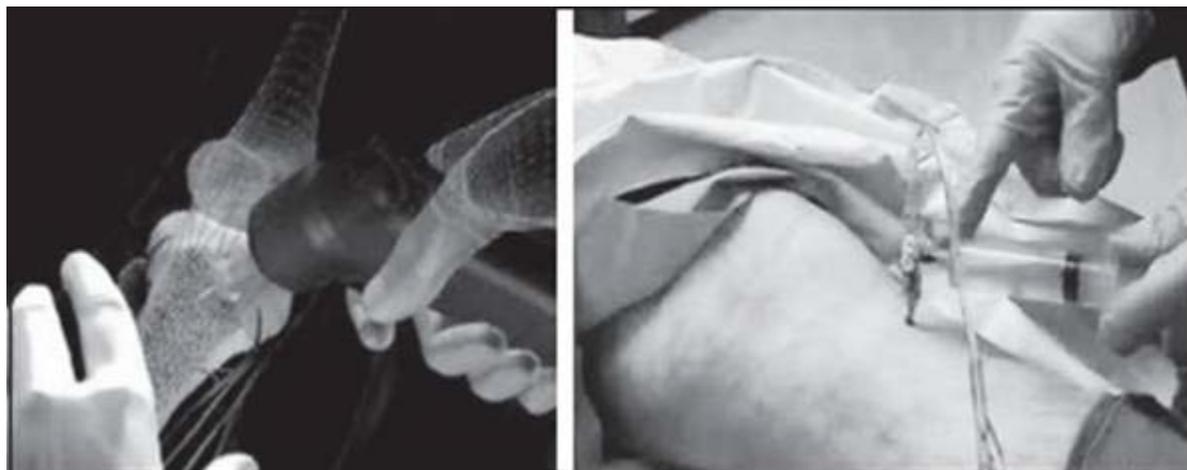


Fig. Using of intraosseal injection during CPR

described above; use fingers of your free hand to close the nostrils of the patient. In case of correct ventilation chest rises and falls silently. Repeat this action one more time.

III. External heart massage (C– circulation). Standing aside the patient (on your knees), place your hands in the center of the chest: heel of the hand in the middle of the lower part of sternum or between lower and middle thirds of sternum. Don't lose precious time looking for anatomic landmarks, as soon as possible start heart massage. Pay attention to your fingers – they should not lean on the chest, otherwise you will break the ribs during compressions. The frequency of compressions should be 100 per minute. It means that until you haven't provided airways with endotracheal tube you make 30 compressions per every 2 breathes. Heart massage is extremely important: do it correctly as its efficiency (and thus cerebral blood circulation) depends on your technique. Use the most developed muscles of your body – back muscles, keep your elbows straight and compress the chest with the power of your trunk, not upper limbs. Every compression should be 5_6 cm deep. After each compression don't forget to let the chest recoil completely. Signs of effective resuscitation actions are pupils contraction,

normalization of the skin color, appearance of peripheral pulse synchronized with the massage, sometimes even possibility of blood pressure measurement. Sometimes heart action restores even during BLS.



Fig. Defibrillation

Fig. Defibrillation

Second stage is advanced life support, which is provided by health_ care professionals in hospitals with the usage of medicines, diagnostic and therapy equipment. The main ideas of ALS are: determination of cardiac arrest type (shockable/nonshockable rhythm), pharmaceutical and electric treatment, usage of advanced artificial ventilation (if available also devices for heart massage) and therapy of reversible clinical death reasons.

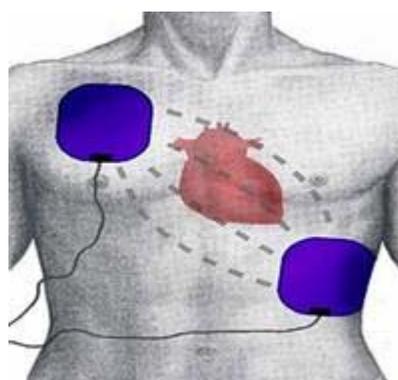


Fig. Defibrillation

When resuscitation team works together functions of rescuers must be divided in order to gain maximal efficiency. After CPR started the main purpose is to decide whether the

defibrillation is necessary or not, other words: to monitor the type of cardiac arrest. Shockable rhythms are ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT without pulse); nonshockable rhythms are asystole (A) and pulseless electrical activity (PEA) of the heart.

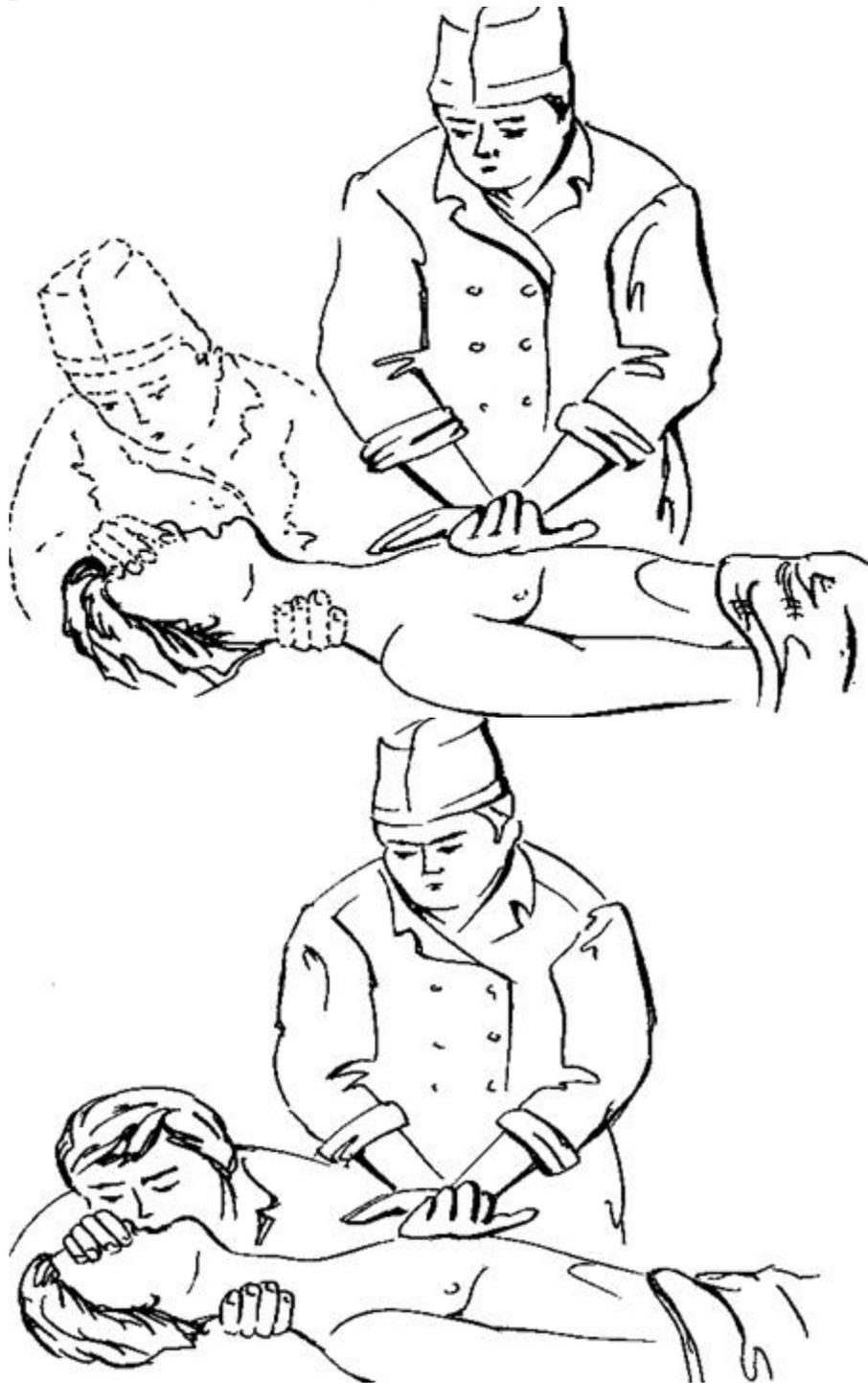


Fig. Performing CPR by team

Without energetic resources VF and pulseless VT quickly change into PEA and asystole, so to shorten the time between arrest and defibrillation paddles

visualization (apex_sternum position) should be used even before the electrodes will be placed on the chest. For the first defibrillation use the dose of 360 Joules for monophasic defibrillators (old models) and 150_200 Joules for biphasic defibrillators (modern devices).

Subsequent shocks might be of the same (200 J) or escalated energy (150_360 J) – the efficiency of energy increase is not proved, so it depends on you and local standards.

Cardiac arrest types:

1. Asystole – flat line on ECG
2. Ventricular fibrillation – chaotic contractions of myocardial fibers visualized on ECG as waves of different shape and amplitude (high, medium and low).
3. PEA – different ECG rhythms, including normal, but combined with the lack of effective systole (pulse).

Simultaneously a venous access attempt should be done, as after third defibrillation administration of adrenaline (1 ml of 0,1% solution = 1 mg followed by 20 ml of 0,9% NaCl solution) and amiodarone (6 ml of 5% solution = 300 mg with 5% Glucose solution, total volume – 20 ml) are required. Adrenaline administration should be repeated every 3_5 minutes in the same dose. In case of PEA and asystole adrenaline should be given from the moment of intravascular access achievement. Atropine is not any more included into the official algorithms of ALS if the cardiac arrest is not caused by vagal effect.

However according to actual Ukrainian standards single administration of Atropine is still recommended (3 ml of 0,1% solution=3 mg, followed by 20 ml of 0,9% NaCl solution).

If, however, venous access attempts are unsuccessful within 2 minutes you should think about alternatives, such as intraosseous access. As for the medicine delivery via the tracheal tube – it is no longer recommended. Central venous line insertion is a prerogative of the most skilled and competent members of the team. Previously it was thought, that triple doses of resuscitation drugs given through endotracheal tube or through the needle in the crico_thyroid membrane will be effective, but according to the actual

recommendations such way of admission is unpredictable and thus can not be an alternative. Intracardiac delivery of drugs nowadays has rather historical value: in most developed countries it is not practiced any more.

Mechanical ventilation is much more effective than the mouth_to_mouth one. There are different types of devices for respiration (carrying and stationary) and numerous devices which play the role of connector between patient's airways and apparatus for artificial pulmonary ventilation (ventilation masks, laryngeal masks and tubes, combitubes, endotracheal tubes, etc). In case of CPR the endotracheal tube with cuff is an absolute golden standard, as it allows asynchronous ventilation/massage and protects patient from aspiration (cardiac arrests are mostly sudden, so there is always a risk of regurgitation, aspiration and thus aspiration syndrome development). Under control of direct laryngoscopy it's possible to clean upper airways with electric or pneumatic suction device and what is even more important – to intubate the trachea.

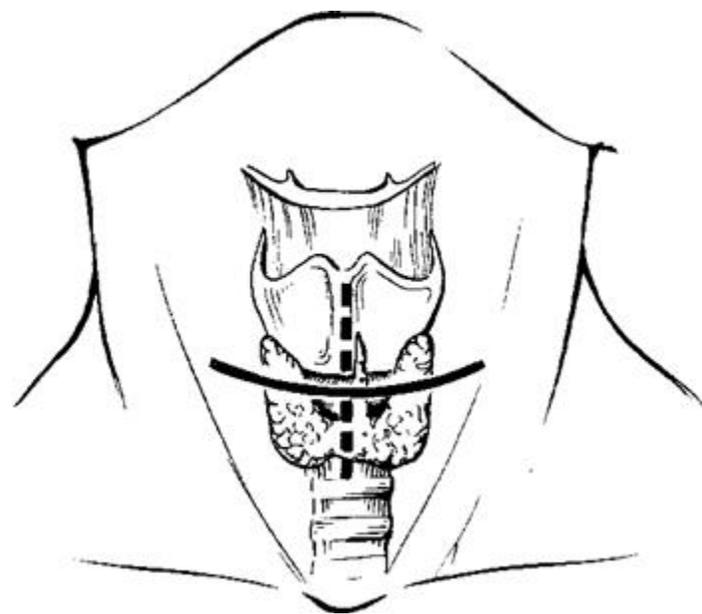


Fig. Conicotomy

During the CPR think about reversible cardiac arrest reasons and try to treat them: there are easy mnemonic schemes of 4 H and 4 T for this purpose. So, the reversible causes of the clinical death are: hypoxia tension pneumothorax hypovolemia tamponade (cardiac)

hypothermia toxins hypo/hyper electrolytic and metabolic disorders thrombosis
 Treat them with: oxygen and artificial ventilation in case
 of hypoxia; crystalloids and colloids in case of hypovolemia; warming
 (including warm infusions) in case of hypothermia and proper
 electrolyte infusions in case of electrolytes and metabolic disorders
 (for example use 5_10 ml of 10% calcium chloride solution if you
 suspect hyperkalemia or hypocalcemia caused by dialysis, hemolysis,
 massive tissue damage, etc.).



Fig. Scheme of CPR

Use needle thoracocentesis for tension pneumothorax, needle pericardiocentesis for cardiac tamponade, antidotes and detoxification methods for toxic agents and thrombolytic therapy for thrombosis (if required).

The third stage of resuscitation is post_resuscitation care provided also in intensive treatment unit.



Fig. Scheme of CPR

On the first stage of this care check again the patient's condition: monitor constantly condition of cardiovascular and respiratory systems, measure blood pressure and central venous pressure, evaluate CNS state (reflexes, neurological deficiency), perform laboratory tests (take blood and urine samples, liquor if necessary), etc. Well_planned, comprehensive examination allows us to identify homoeostasis disorder and choose optimal treatment. After main parameters are stabilized central nervous system becomes your main concern: protect the brain from hypoxia by all available means, because hypoxic damage of neurons is usually irreversible. To achieve this purpose you should:

– provide adequate oxygenation (however excess of oxygen is no longer recommended, so keep blood saturation at the level 94_98%); hyperventilation might be used in case of brain oedema (first 12_24 hours of artificial ventilation);

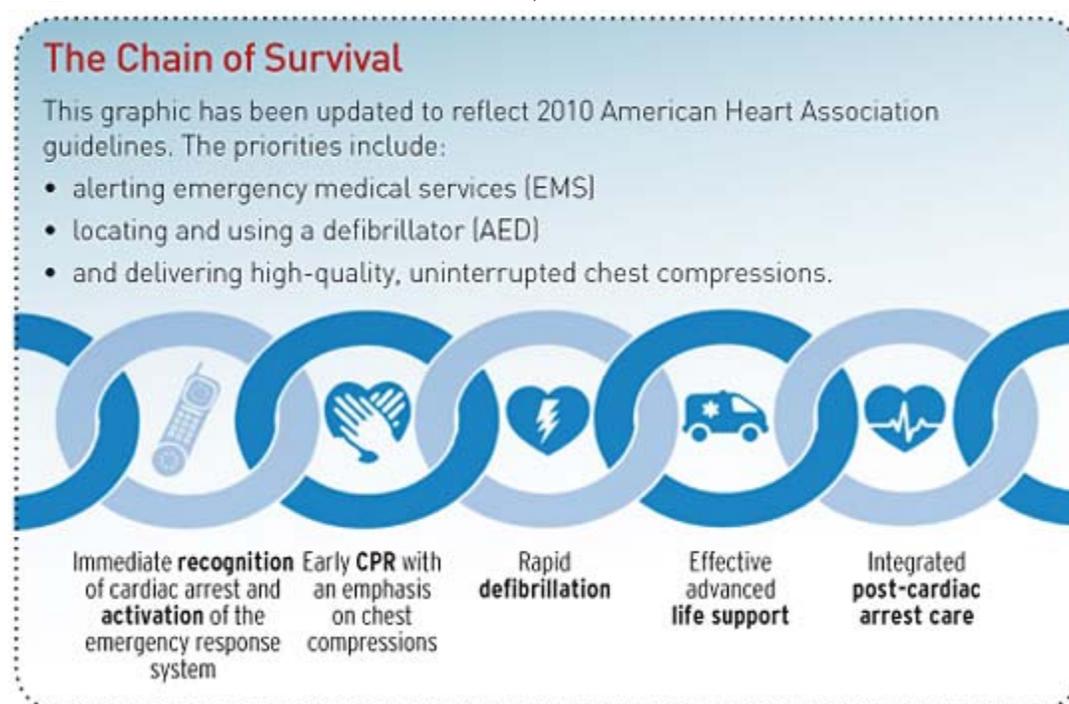


Fig. The chain of survival

– decrease metabolic needs of the CNS by craniocerebral hypothermia (give saline solution with the temperature 2 °C in order to lower body temperature to 32_34 °C for 12_24 hours) or by continuous narcosis (sodium thiopental 3_5 mg/kg, diazepam 0,2 mg/kg, neuroleptics, etc.);

- control blood glucose level (avoid hyperglycaemia over 10 mmol/l; hypoglycaemia can not be accepted at all);
- additionally prescribe antihypoxants like sodium oxybate (20_40 mg/kg every 4 hours), cytochrome C (0,5 mg/kg i/v); antioxidants like tocopheryl acetate (500 mg i/v), B_vitamins (2_3 ml), ascorbic acid (5 ml of 5% solution 3 times a day); calcium antagonists like verapamil (2 ml 3 times a day), magnesium sulphate (5_10 ml of 25% solution i/v every 4 hours with blood pressure control);
- improve cerebral perfusion with haemodilution (give crystalloids to get hematocrit 0,3_0,35 l/l), relative hypertension (20_30% over the normal level), solutions influencing rheological properties of the blood and microcirculation (rheopolyglucin, 2 ml of 0,5% curantil solution, heparin 5000 units every 4 hours, etc);
- treat cerebral oedema with mannitol (1g/kg), furosemide solution 10 mg i/v 3 times a day), dexametazon solution (8 mg every 4 hours);
- use hyperbaric oxygenation from the 5_th day of post_resuscitation care (totally 10 procedures);
- give nootropic drugs and neuroprotectors (piracetam, cerebrolysin, aminalon, etc.)



Fig. Trainings

Cardiac arrest in special circumstances

Regardless to the cardiac arrest reasons the main factors of thanatogenesis [*Thanatos – god of death in Ancient Greece] are hypoxia, hypercapnia, electrolytic disorders and pathological reflexes. In 90% of cases heart stops in the moment of diastole, in 10% – systole. Heart, lungs and brain are the death entrance gate.

In_hospital cardiac arrest.

The most common reasons of primary cardiac arrest:

- a. acute cardiac failure (coronary disease, myocardial infarction, rhythm disorders, sudden coronary death)
- b. acute obstruction of main vessels (pulmonary thromboembolism)
- c. acute and severe deficiency of blood volume (significant blood loss, dehydration)
- d. acute decline of peripheral vessels resistance (acute suprarenal failure, anaphylactic shock, somatogenic collapse in case of acute intoxication, orthostatic medication collapse)

The most common reasons of acute respiratory failure:

- a. airways obstruction (tongue, vomit, foreign bodies)
- b. inhibition of respiratory center (opiates, anesthetics)
- c. disorders of breathing biomechanics (convulsions, myasthenia, tension pneumothorax or hemothorax)
- d. restrictive disorders (massive pneumonias, shock lung syndrome, pneumothorax or hemothorax).

The most common reasons of primary brain death: acute vascular disorders (subarachnoid hemorrhage, hemorrhagic and ischemic strokes, brain dislocation).

Patients with a predictably high risk of sudden death should be constantly under complex vital monitoring. In case of compensation failures medical personnel of the department should intrude with a treatment directed at correction of disorders and intensive care. That is why an in_hospital sudden death should be an exception. Never the less hospital staff should be prepared to provide immediate life support.

Here is the list of equipment for in_hospital resuscitation:

1. Portable manual respirator.
2. Oxygen supply (cylinder).
3. Electric suction device with suction catheters.
4. Electrocardiograph, defibrillator, tonometer.
5. Mouth_gag, tongue forceps, clips.
6. Set of face masks and airways.
7. Laryngoscope with a set of tubes.
8. Set for conicotomy and for pericardiocentesis.
9. Solutions of adrenaline, atropine, sodium bicarbonate, lidocaine, steroids, colloids and crystalloids.
10. Infusion sets, i/v catheters, syringes of different sizes.
11. Bandages, medical napkins, antiseptic solutions.

Peculiarities of in_hospital resuscitation.

Patient is usually lying in bed.

1. To get a firm surface [efficiency of chest compressions depends on this] lay under the patient spinal board or move him/her on the floor.
2. Duration of the first stage should be minimal (5 to 7 minutes) as the beginning of advanced life support in intensive care unit is more important.
3. In witnessed primary cardiac arrest caused by ventricular fibrillation during first 20_30 seconds precordial thump might be effective (so called “mechanical defibrillation”).
4. Sometimes tracheostomy or conicotomy might be necessary in case of upper airways obstruction (laryngospasm, stenosis of larynx, foreign body in the glottis).

The most favorable resuscitation prognosis is connected with primary respiratory arrest and the most unfavorable – with the primary cerebral death.

Drowning: types and resuscitation.

There are different types of drowning thanatogenesis.

True drowning. Most victims under the water according to a reflex stop breathing. But after some time due to hypercapnic

stimulation of the respiratory center they unwillingly begin to make respiratory movements. Liquid gets to the lungs: fresh water, which is hypoosmolar to plasma, diffuses easily through blood-air barrier into the blood, thus increasing its volume. Extra 1500-2000 ml of water in addition to hypoxia lead to cardiac arrest. At the same time osmotic hemolysis (caused by rapid lowering of plasma osmolarity) and hyperpotassemia are also cardiac arrest factors.

In case of salt water true drowning fluid part of blood according to the osmotic gradient moves from bloodstream into the bronchi and trachea. This way surfactant is being destroyed and pulmonary edema begins.

Dry drowning. 8-10% of victims in the moment of water aspiration have reflexive vocal cords closure. This prevents further water entering into the lower airways. The cardiac arrest is caused by hypoxia.

Syncopal drowning happens in 5 % of cases. Due to fear, immersion in cold water, injury of reflexogenic zones caused by falling primary cardiac arrest appears. It is called "syncope". Those victims have gray color of skin and there is no water in their airways.

Peculiarities of resuscitation.

1. In case of true drowning early respiratory support is the most important. Right after airways management (head titling, oral cavity cleaning) rescue breathing should be provided. Don't waste victim's precious time on shaking out the water by pressing the abdomen or lowering the head: the amount of water inside is not that dangerous and your efforts are hopeless and unnecessary. The only thing you really can achieve by these actions is vomiting and aspiration of gastric contents, which are much more dangerous, than aspiration of water.
2. During resuscitation of the patient, who drowned in sweet water, 10 % solution of calcium chloride is used (5 or 10 ml).
3. All patients who were drowning should be transported to the ICU and observed most carefully (few days).
4. To avoid secondary drowning (fulminant pulmonary edema causing death) patients with true drowning should receive respiratory support with positive end expiratory pressure.

5. On the third stage of resuscitation patients with true drowning in sweet water should receive solution of sodium bicarbonate in order to prevent renal failure (renal tubules are being blocked by hemoglobin, which accumulates due to hemolysis) and diuretics.

6. On the third stage of resuscitation patients with true drowning in salt water should receive hypotonic infusions (in order to correct hyperosmotic hypohydration).

In case of dry drowning and syncopal drowning prognosis is favorable even after prolonged clinical death, unlike drowning in sweet water. In case of drowning in cold water (0°C) biological death is not stated until the body becomes warm, resuscitation lasts much longer, than in conditions with normal temperature.

Resuscitation in case of mechanical injuries (fall from a height, car accidents).

1. Clinical death might be caused by severe injuries incompatible with life or reflex cardiac arrest. It is obvious, that resuscitation will be successful in second case, but not first. If it is a witnessed cardiac arrest precordial thump might be effective.

2. In trauma cases you should be careful with the neck of the patient: always suspect backbone injury until it is not excluded with additional methods of diagnostics (computer tomography diagnostics, X_ray diagnostics). Don't tilt the head backwards: in such cases it's enough to thrust the jaw forward.

3. If the patient has fractures of facial skeleton or injuries of face soft tissues mouth_to mouth respiration might be ineffective or even impossible, however mouth_to_nose respiration might be useful.

Transportation, if it's possible, should be in a save position with head turned aside.

4. Constantly examine patient's condition: injuries, which seem to be unimportant at the beginning sometimes change into life threatening; this is why examinations should be repeated and accurate.

Transportation of severe patients sometimes ends with decompensation of main vital systems.

Electric trauma and lightning stroke.

1. After electric injury or lightning stroke clinical death might happen due to primary respiratory arrest (spasms of respiratory muscles, respiratory center damage), ventricular fibrillation or cerebral affection (in last case vital signs are minimal, so there were cases, when patients were buried “alive” and “raised”; this could be the reason of a superstitious belief that for bringing back life after lightning stroke victim should be buried).
2. As soon as possible break the contact between victim and electricity source. Still, remember that your own safety is of not less importance.
3. In most cases electric defibrillation will be necessary (so called shockable rhythms).
4. Even patients who seem to be fine according to their vital parameters should be admitted to the ITU and observed most carefully for few days. There is always a risk of sudden cardiac arrest due to violation of myocardial excitability and conductivity during first 24 hours after electric trauma or lightning stroke.

Mechanical asphyxia.

1. In case of mechanical asphyxia first of all provide the patency of airways (clear the oral cavity, throat, larynx with your finger or any available equipment (clamp, forceps, aspirator) with laryngoscopy or without it and then decide what type of respiratory support patient needs.
2. If there is no chance to treat the obstruction of upper airways using usual methods or tries were unsuccessful urgent cricoidotomy (3.3) or tracheostomy (3.4, in hospital conditions) are the only ways of rescuing patient’s life.
3. Never ever try to push “deeper” the foreign body you can’t take out!

Medical operations and manipulations

Mouth_to_mouth ventilation.

Indications: respiratory arrest or ineffective patient’s breathing, when there is no respiratory apparatus.

Necessary equipment: napkin or handkerchief; artificial airway,

gloves– if available.

Procedure: First of all free upper airways – open the mouth of the patient and clear oral cavity as mentioned above: turn the head aside, open the mouth and remove vomit, blood clots, foreign bodies with a finger. Then tilt head backwards and thrust the jaw forward.

To make mouth-to-mouth ventilation less unpleasant you can put a napkin or a piece of bandage on the mouth of the victim. Close patient's nostrils with your fingers, press your mouth against the mouth of the patient and make a forced expiration. Inhaling the air observe the chest: if it moves according to your respiratory efforts breathing is effective. However if chest is not rising check again airways patency: thrust the jaw placing your fingers over its angle and moving them forward (lower teeth should overlap upper teeth).

Your aim is to exhale nearly 600_800 ml of air with the frequency of 10 times per minute (2 breathes to 30 compressions) during CPR. If it is respiratory arrest alone you can make 15_20 breathes per minute.

Chest compressions (External heart massage).

Indications: cardiac arrest (clinical death).

Necessary equipment: doesn't need any; gloves if they are available.

Procedure: place the patient on the firm flat surface in supine position; if you have an assistant one of you should provide airways patency and breathing, another – chest compressions. Staying on your knees beside the patient place one hand in the middle of the chest and cover it with another. Your fingers should not touch the chest, otherwise while compressions you will break the ribs. Frequency of compressions should be 100 per minute*, depth – 5_6 cm. Keep your elbows straight and use mainly muscles of your back (weight of the body): thus you will exhaust slower. In case of effectively provided CPR you might observe constriction of pupils, normalization of skin color, pulse on peripheral vessels, sometimes it's even possible to measure blood pressure.

Heart puncture.

There are two types of heart puncture: puncture of heart cavity

(previously used for adrenaline injection or in case of air embolism) and pericardial puncture performed for extraction of blood in case of haemopericardium.

Indications: air embolism, haemopericardium.

Necessary equipment: 7_10 cm needle, 10_20 ml syringe.

Procedure: find the fourth intercostal space and puncture the skin with a saline filled syringe 1_1,5 cm to the left from the sternum border.

Needle should be directed over the fifth rib sagittally and a bit to the middle, all the time control the needle position pulling the plunger back. At a depth of 4_5 cm you will feel a kind of resistance – wall of the right ventricle, after that expect appearance of blood in the syringe – sign of the needle located in the left ventricle. Control the hub with your left hand and push the plunger with the right hand to infuse the medicine from the syringe (or aspirate the air in case of air embolism).

Electric heart defibrillation

Indications: ventricular fibrillation, ventricular tachycardia without pulse.

Necessary equipment: defibrillator, electrode paste (electricity conductive gel).

Procedure: evaluate the rhythm during CPR as soon as possible.

After stating “shockable rhythm” defibrillate immediately. According to the actual CPR recommendations heart massage should be interrupted only for a moment of defibrillation itself and continued during paddles placement and charging. So while 2 rescuers are continuing CPR the third should place the paddles in sternum_apex position (previously putting on them layer of electrode gel), choose the correct mode on the defibrillator and press charge button. Begin with 150_200 J in case of biphasic defibrillator and 360 J in case of monophasic. Make sure neither you nor your colleagues are contacting the patient or equipment, otherwise you might get injured. At the moment of defibrillation remove the source of the oxygen from the patient and stop the infusion. After defibrillator is loaded press “defibrillate” button on the paddle and at the moment

defibrillation is over restart the massage. Put paddles on their place without crossing or contacting them in the air. In two minutes check the rhythm and vital signs again and if necessary – repeat the defibrillation with a greater voltage (300_360J).

Paddles placement: a) one electrode is placed in front of the heart (anterior position) and another one on the back, behind the heart, between the scapula (posterior position); b) apex_ sternum position: one electrode is placed over the heart and another one on the right side of the sternum.

2.5. Control tests

1. Name the stages of dying:

A.Preagony, terminal pause, agony, clinical death;

B.Agoty, clinical and biological death;

C.Coma, agony, clinical death;

D.Preagony, agony, clinical death, social death;

E.Hypotension (blood pressure lower than 80 mm Hg)

2. What sign is not one of the main clinical death signs?

A.lack of blood pressure

B.lack of pulse on femoral arteries

C.lack of breathing

D.mydriasis and lack of photoreaction

E.lack of pulse on carotid arteries

3. What should be expiratory volume during “mouth_to_mouth” ventilation?

A.1700_2000 ml

B.500_700 ml

C. 1200_1600 ml

D. 800_1100 ml

E. maximal exhalation

4. The duration of clinical death in conditions of normal temperature is:

A. 7_12 minutes

B.1_3 minutes

C. 4_5 minutes

- D. 5_7 minutes
- E. 10 minutes
5. Name the exact hand placement during heart massage:
- A. on the chest, fingers to chin
- B. lower third of the sternum, fingers along ribs
- C. middle of the chest
- D. 4_th intercostal space, left part of the chest
- E. on the chest in the heart area
6. What are the signs of effective artificial ventilation?
- A. narrowing of the pupils
- B. noises during ventilation
- C. silent chest movements
- D. bulged out epigastrium
- E. dizziness of the rescuer
7. Choose an absolute sign of cardiac arrest:
- A. flat line on ECG
- B. unconsciousness
- C. cyanotic color of skin
- D. lack of pulse over carotid arteries
- E. lack of blood pressure
8. Choose the place of heart puncture:
- A. 5_th intercostal space, in the place of apex projection
- B. 3_d intercostal space, on the left from sternum
- C. lower edge of the 4_th rib
- D. 4_th intercostal space, 1,5 cm on the left from the sternum
- E. in the place where heart action is noticeable
9. What medicines are usually used during CPR?
- A. adrenaline, atropine, cordaron
- B. adrenalin, dopamine
- C. atropine, dopamine
- D. atropine, steroids, dopamine, magnesium
- E. adrenaline, magnesium
10. What is the reason of Sodium bicarbonate usage during CPR?
- A. correction of metabolic acidosis

- B. prevention of respiratory acidosis
 - C. liquidation of hypoxia
 - D. treatment of metabolic alkalosis
 - E. protection of central nervous system
11. Choose the indication for defibrillation during CPR:
- A. low blood pressure
 - B. asystole
 - C. PEA
 - D. VF
 - E. lack of pulse over carotid arteries

Task 1.

Andrew, the student of medicine, is a witness of an accident: wireman, who was working with a transformer, suddenly was kicked few meters from it. During examination it turned out, that victim is unconscious, his pupils are dilated and do not react on light, his skin is pale; there is no pulse over carotid arteries; fingers of the right hand are burnt, in the middle of the palm there is a lacerated wound.

What diagnosis should Andrew think about? What should be done?

What is the order of these actions?

Task 2.

Annie, the nurse, is a witness of such situation: a man of 40, lying at the city bus station, is being ventilated by a passing pedestrian through chest pressing and arms abducting. The victim is unconscious, his pupils are dilated, skin is pale and there are no pulse, no breathing and no reflexes.

What diagnoses should Annie think about? What way should Annie provide the CPR?

Task 3.

Walking near the lake Andrew, the student of medicine, suddenly notices a 4-year old girl who fell to the water and disappeared under the surface. What should Andrew do?

Task 4.

„The laymen”, local group of amateur rescuers, dragged from the water a young girl. They turned her down at the same time pressing with the knee her abdomen in order to force the water out from the airways. After

that they putted her on her back, attached with a pin her tongue to the chin (in order to provide airways potency). Next they started CPR: forced maximal breathes (14 per minute) together with chest compressions (4 cm deep, 60 per minute). Name 5 mistakes of „The layman”.

Task 5.

„The Dream team”, group of professional rescuers, started CPR of a patient in a state of clinical death. Then they cleared the upper airways with an electric suction machine, connected respiratory machine. Next they gave adrenaline (0,5 ml of 0,1% solution) sodium bicarbonate (4% solution, 10 ml), calcium chloride (10%, 5 ml) and atropine (0,5 ml of 0,1% solution) at the same time and connected patient to a monitor (flat line on the ECG). After that rescuers did a defibrillation of 300 J, which resulted in a flat line on the ECG. They repeated everything again, this time with defibrillation of 400J. Unfortunately there was no effect. Patient died.

Name 5 mistakes of „The Dream team

The importance of the water to the organism.

Life on earth was born in the water environment. Water is a universal solvent for all the biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homoeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the atrial walls, baroreceptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals reveal into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution). This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.

Importance of osmolarity for homoeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hyposmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.

9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobin concentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, 0,6* BW volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K - potassium deficiency, mmol;

K_p - potassium level of the patient mmol/l;

$0,6 * \text{BW}$ - total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 \text{ BW},$$

Na- sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

0,2*BW – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonicoverhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 \text{ BW},$$

Cl_d- chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

0,2*BW – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chlorine solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p - plasma magnesium concentration of the patient, mmol/l

0,6*BW - extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d - calcium deficiency, mmol

Ca_p - plasma calcium concentration of the patient, mmol/l

0,2*BW - extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorbition.

Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO₂. Normally pCO₂ of arterial blood is 36-44 mm Hg. Hypercapnia (pCO₂ increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO₂ less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H⁺ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is 0±1,5. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat

acute and chronic respiratory violations. When $p\text{CO}_2$ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

$$\text{pH} > 7,45,$$

$$p\text{CO}_{2a} < 33 \text{ mm Hg}$$

$$\text{BE} < +1,5 \text{ mmol/l.}$$

However prolong alkalosis brings decrease of BE due to compensatory retain of H^+ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H^+ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

$$\text{pH} < 7,35,$$

$$p\text{CO}_{2a} < 35 \text{ mm Hg}$$

$$\text{BE} (-3) \text{ mmol/l.}$$

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * \text{BE} * \text{BW}$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H^+ ions concentration. Blood tests show:

$$\text{pH} > 7,45,$$

$$p\text{CO}_{2a} \text{ normal or insignificantly increased (compensatory reaction)}$$

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patient, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patient, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patient, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patient, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patient, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration 88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

Task 7.

Patient, the patient of 50 with body weight 75 kg, was transported to the admission

unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow breathing. Blood tests show: pH 7,18, $p\text{CO}_2\text{a}$ 78 mm Hg, $p\text{O}_2\text{A}$ – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patience, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, $p\text{CO}_2\text{a}$ 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

ACUTE VIOLATION OF CIRCULATORY ACTIVITY

Diagnosis and treatment

Introduction

Acute circulatory insufficiency

Anatomy and physiology

Life is provided through a variety of mechanisms, however all of them depend on proper circulation. Circulation itself consists of 2 parts: work of heart (pump of the body) and vessels, through which blood is pumped to the most remote organs and tissues. During every systolic contraction heart pumps 70_80 ml of blood (so called stroke volume). So if the heart rate is 70 beats per minute, heart pumps nearly 5 liters of blood, what makes more than 7 tones per day.

From the left ventricle blood gets to the arterial system of the systemic circuit. Arteries contain 15% of the whole circulating blood volume; they carry blood from the heart to their distal departments – arterioles (vessels of resistance). Arterioles themselves are defining blood distribution: in condition of constriction (spasm) they make blood supply of the capillaries impossible (ischemia appears). On the contrary, in condition of dilatation they provide maximal oxygenation. When arterioles are blocked due to the spasm blood is flowing through the arterio_venous anastomoses directly to the venous system.

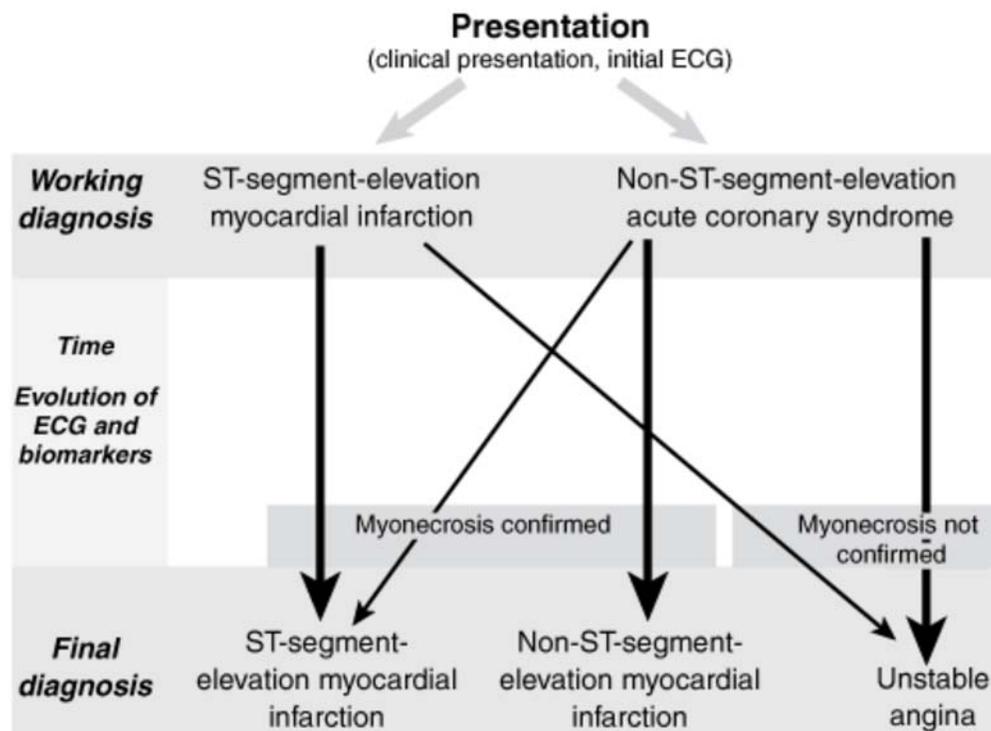
Distribution of blood in the vascular bed (% of CBV).

- a. heart cavity 3%
- b. arteries 15%
- c. capillaries 12%
- d. venous system 70%

Among the natural vasoconstrictors (agents, which cause constriction of the blood vessel) are epinephrine, norepinephrine,

serotonin, angiotensin II. Stress enhances the secretion of catecholamines, their blood concentration increases and arterioles constrict. Spasm of the arterioles is the basis of blood flow centralization: peripheral flow is disregarded in order to provide brain with the oxygenated blood as long as possible. To the group of vasodilators (agents, which provide dilatation of the vessels) belong “acid” metabolites (lactate, pyruvate, adenylic acid, inosinic acid),

—
bradykinin, acetylcholine, different medicines (neuroleptics, β -adrenergic antagonists, peripheral vasodilators, ganglionic blocking agents, etc.), some exogenous poisons. All of them cause blood flow decentralization: opening of arterioles and distribution of the blood from central vessels to the capillary bed.



Capillaries are the interweaving network of the smallest body vessels with the general length of 90_100 thousands kilometers. However simultaneously work only 20_25% of them. They provide metabolic exchange bringing oxygen and nutrients to the tissues and take back wastes of metabolism. Periodically, every 30_40 seconds one of them get closed and others open (vasomotion effect). Capillaries contain 12% of the whole circulating blood volume, but

different pathological conditions can increase this amount even 3 and more times.

“Used” blood from the capillaries flows to the venous system.

Veins are the blood reservoir, which contains 70% of the total circulating blood volume. Unlike arteries they are capable of volume control and thus they influence the amount of blood, which returns to the heart.

The most important haemodynamic index of venous system is central venous pressure. CVP represents the pressure which blood causes to the walls of cava veins and right atrium. This parameter is an integral index of circulating blood volume, systemic vascular resistance and pump function of the heart. It can be measured with a special device called “phlebotonometer” (pic. 4.9) or with a usual infusion set and a ruler. Normally CVP measured from the sternum point is 0_14 cm H₂O and from midaxillary line _ 8_15 cm H₂O.

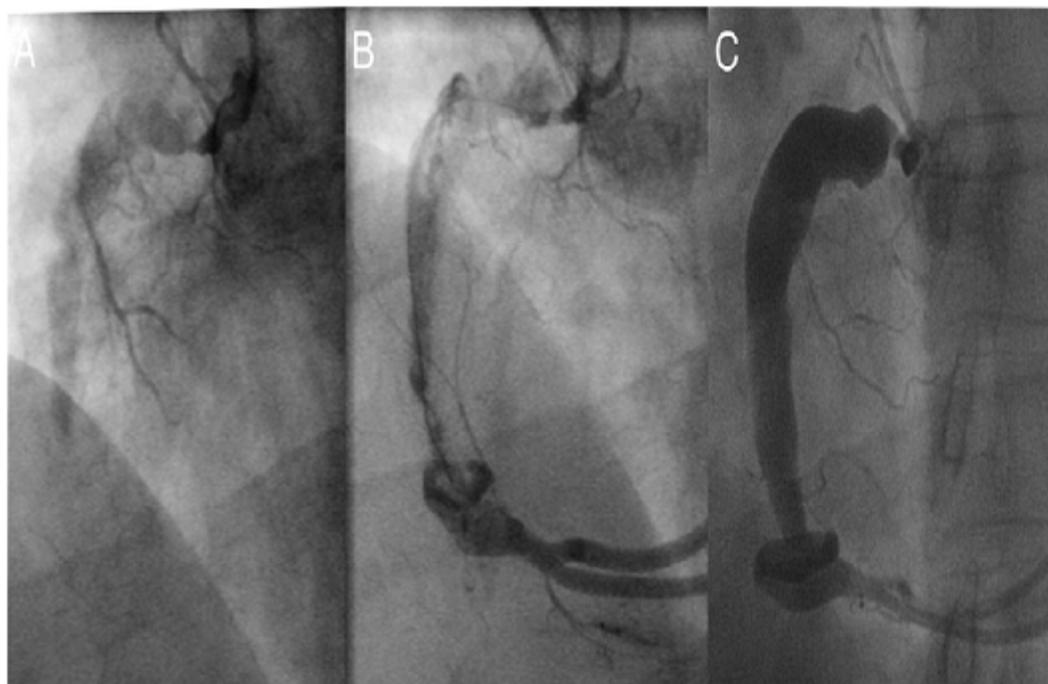


Fig. Angiografia of patient with MI

Central venous pressure decreases (sometimes even to negative)

in case of:

- blood loss
- excessive water loss (dehydration)

– distributive shock (decrease of peripheral resistance due to venous and arterial dilatation)

In those conditions decreases volume of blood returning to the heart and thus suffers cardiac output. In case of negative CVP cardiac arrest is highly probable.

–
Central venous pressure increases in case of:

- heart failure (insufficiency of left or right ventricle)
- hypervolemia (excessive blood infusion, improper infusion therapy)
- obstructions to blood flow (pulmonary embolism, cardiac tamponade, etc.)

When CVP over 15_16 cm H₂O is combined with left ventricle insufficiency the risk of pulmonary edema is very high.

Blood pressure is an integral index of arterial part of systemic haemodynamics. Talking about blood pressure we may refer to systolic, diastolic, pulse and mean arterial pressure. Systolic (P_{syst}) and diastolic (P_{diast}) pressures are measured with the manometer (method with the usage of phonendoscope was invented by M. Korotkoff). Pulse pressure (PP) is a difference between systolic and diastolic blood pressure.

Mean arterial pressure (MAP) is calculated according to the formula:

$$\text{MAP} = P_{\text{dias}} + \frac{1}{3} \text{PP} \text{ mm Hg}$$

MAP defines the level of pressure necessary for the metabolic exchange in the tissues. Its measurement allows the evaluation of tissue perfusion level.

Blood pressure depends on different factors, but the most important are cardiac output and vascular resistance (mostly arterioles). This dependence is direct, thus you can increase blood pressure using:

- infusion of vasoconstrictors – solutions of epinephrine, phenylephrine (mesaton), etc. (they will increase the vascular resistance);

- infusion of hydroxyethyl starch solutions or saline (they will increase circulating blood volume)
- infusion of cardiac glycosides or other medicine which stimulate Myocardium

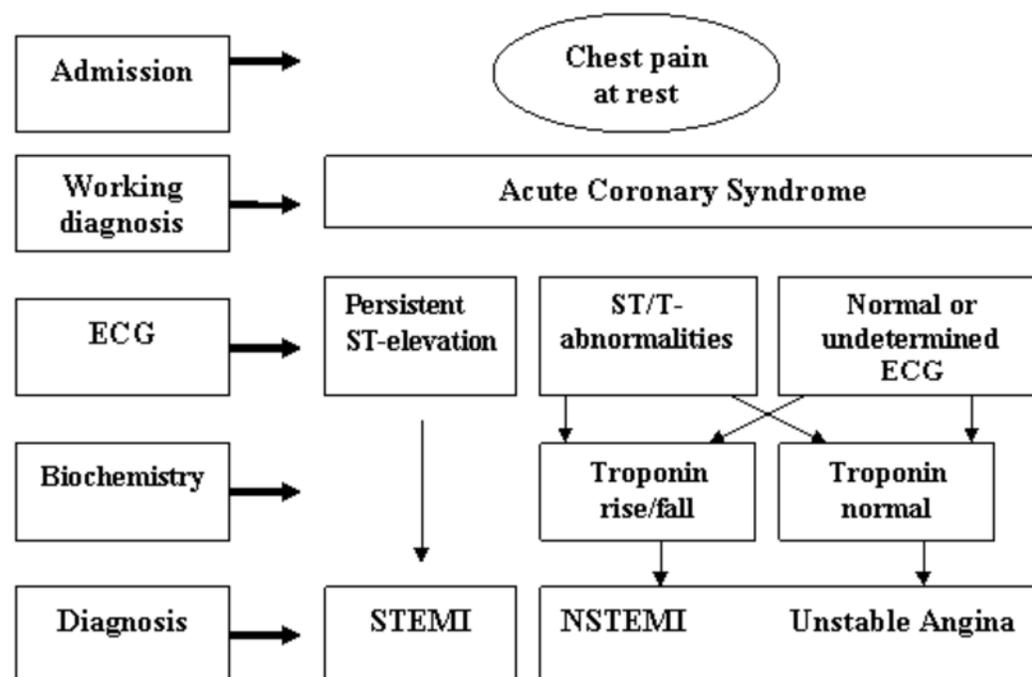


Fig. Chest pain

General volume of blood in the body of a healthy adult is nearly 7% from the body weight: 70 ml per kilogram for male and 65 ml per kilogram of body weight for female. Of course circulating blood volume is lower, because part of blood is out of metabolic processes

– as a reserve. CBV can be measured with the infusion of coloring substance to the blood flow (Evans blue, polyglucin) and later evaluation of its dissolution degree.

Therefore measurement of CVP, BP, cardiac output and circulating blood volume allow to evaluate condition of circulation system of the patients and to provide adequate correction.

4.2. Acute heart failure; shock and collapse

Acute cardiovascular failure is a state of cardiac and vascular inability to provide adequate supply of tissue metabolic needs with oxygenated blood and nutrients. This, earlier or later, causes cellular death.

The reasons of the failure vary greatly: mechanic injuries, blood loss, burns, dehydration, exogenous and endogenous intoxications, immediate hypersensitivity reaction, ischemic heart disease, neural and humoral regulation disorders of vascular tone.

Acute cardiac failure is a disorder of heart pumping action. It develops due to primary heart problems or secondary, under the influence of extracardiac factors such as infection or intoxication.

There are two types of heart failure: left_sided and right_sided.

Left_sided heart failure is an inability of left ventricle to pump blood from the pulmonary circuit to the systemic circuit. The most common reasons of it are myocardial infarction, mitral insufficiency, left AV valve stenosis, aortic valve stenosis, aortal insufficiency, hypertonic disease, coronary sclerosis, acute pneumonia.

Coronary circulation is possible only during the diastole and in those conditions every violation of coronary passability decreases cardiac output. This way during the systole part of the blood is not injected into aorta, but stays in the left ventricle. Diastolic pressure in the left ventricle increases and blood is literally forced to stagnate in the left atrium. At the same time right ventricle functions normally and continues to pump usual amounts of blood to the pulmonary circuit.

Thus hydrostatic pressure in the vessels of pulmonary circulation increases, fluid part of the blood moves first to the lung tissue and then, through alveolar_capillary membrane, to the alveolar lumen.

Clinically pulmonary edema begins with dyspnea (during physical activity or rest). Later attacks of dyspnea are connected with persistent cough with white or pink blood_tinged phlegm. During the attack patient tries to sit as in this position breathing is easier.

This condition is called "heart asthma". When hydrostatic pressure is over 150_200 mm Hg, fluid part of blood moves to the alveolar lumen causing development of pulmonary edema.

Pulmonary edema is divided into interstitial and alveolar edema.

Interstitial edema is a condition during which serous part of stagnated in the pulmonary circuit blood infiltrates the lung tissue, including peribronchial and perivascular spaces.

During alveolar edema not only the plasma, but also blood components (red and white blood cells, platelets) get out from the vessels. During the respiratory act blood mixes with the air creating large amount of “foam”, which violates gas exchange. This way, in addition to circulatory hypoxia, hypoxic hypoxia appears.

Condition of the patient gets worse quickly. Sitting position is optimal, but not as helping as previously. Respiratory rate is nearly 30_35 breathes per minute, but attacks of breathlessness are constant. Skin is pale with acrocyanosis. Hypoxia of central nervous system usually causes psychomotor agitation. Respiratory acts are noisy; during cough pink blood_tinged phlegm is released. Auscultation allows you to hear different wet rales, sometimes it's even possible to hear them standing aside the patient without phonendoscope.

Pulmonary edema can be also divided according to the blood pressure level: the one with elevated pressure is caused by a hypertonic disease, aorta valve insufficiency or disorders of cerebral perfusion; another one is caused by total myocardial infarction, acute inflammation of myocardial muscle, terminal valve defects, severe pneumonia and is characterized with normal or low blood pressure.

Immediate aid

- make sure patient is sitting with his legs down (orthopnea)
- provide oxygenation through nasal catheter (before placing oil it with glycerin, insert it to the depth of 10_12 cm – distance from the wing of the nose to auricle) or face mask. Do not use Vaseline, because it can burn in atmosphere with high concentration of oxygen. However if catheter is not deep enough patient will suffer from an unpleasant “burning” feeling, because oxygen flow will dry mucosa layer of the nasal cavity; also in this situation concentration of oxygen will be lower than expected.
- put venous tourniquets on the limbs in order to reduce amount of blood returning to heart: venous bed of limbs can reserve up to 1,7 liters of blood;
- constantly control heart and kidney activity (ECG, SaO₂ , and blood pressure are checked automatically through the monitor; to

control diuresis you should insert Foley catheter;

- catheterize central vein, because amount of infusions should be based on central venous pressure;

- use medical “defoamers” if they are available (ethyl alcohol or antiphomsylan solution) combined with oxygen inhalation;

- medical treatment: 1% morphine solution (decreases intravascular pressure of pulmonary circuit, inhibits respiration center in medulla oblongata preventing dyspnea progress, sedates patient);

- solutions of diuretics are used to decrease the circulating blood volume (6_12 ml of 1% furosemid solution, solution of ethacrynic acid), however be careful with them in case of low blood pressure; diuretic effect will last up to 3 hours after i/v infusion, the expected diuresis is 2_3 liters;

- if blood pressure allows you can try to use nitroglycerin to reduce intravascular pressure of pulmonary circuit (1 or 2 tablets with 10 minutes interval);

- cardiac glycosides for improvement of the heart action (0,025% digoxin solution, 0,05% strophanthin solution, 0,06% corglicon solution);

- in case of high pressure (over 150 mm Hg) use ganglionic blocking agents (1 ml of 5% pentamin solution diluted in 150 ml of saline, give i/v slowly; diluted with saline 250 mg of trimethaphan solution), because they reduce pressure in pulmonary circuit and lower the amount of blood getting to right half of the heart, however be careful with the dosage and monitor blood pressure level carefully;

- never use osmotic diuretics in case of pulmonary edema – they will increase blood volume and thus heart load!!!;

- when everything listed above failed and patient is worsening with every second you should intubate him and start artificial ventilation with positive end expiratory pressure (begin with 4_6 cm H2O).

Right_sided heart failure is an inability of right ventricle to pump blood from systemic circuit to the pulmonary circuit due to its weakness or an obstruction to the blood flow.

It occurs in case of pulmonary embolism, right ventricle infarction, excessive infusion therapy (especially including citrated blood) in patients with heart insufficiency, lung diseases (bronchial asthma, emphysema, pneumosclerosis) which cause increase of right ventricular load.

Patients have acrocyanosis, tachycardia, dyspnea, pronounced neck veins, ankle swelling, enlarged liver, ascytis. Central venous pressure is highly increased (up to 20_25 cm H₂O), however pulmonary edema does not appear.

Intensive treatment is mostly pathogenetic:

- limit the infusions (give only life_necessary solutions, check the water balance of the patient and reduce drinking water if necessary);
- in case of citrated blood transfusions use 5_10 ml of 10% calcium gluconate solution per every 500 ml of blood to prevent hypocalcaemia;
- in case of bronchial spasm use bronchial spasmolytics;
- to remove excessive fluid from the body use diuretics (furosemide solution for example);
- metabolic acidosis is corrected with 4% solution of sodium bicarbonate (i/v slowly with acid_base state control);
- in case of pulmonary embolism anticoagulants are used – fraxiparine 0,6 mg subcutaneously; heparin solution – 5000 IU every 4 hours; fibrinolytic drugs (streptokinase, fibrinolysin, urokinase, etc.)

Shock is a pathological state which can be described as a tissue hypoxia caused by hypoperfusion. Pathogenetic basis of shock depends on its reason (trauma, toxins, thermal injury) and at the same time on reactivity of the organism (level of defense mechanisms mobilization).

Stimulation of sympathetic nervous system – production of catecholamines and other vasoactive substances by hypothalamus and adrenal glands is the universal response of the body to the stress. Those mediators interact with the receptors of peripheral vessels causing their constriction and at the same time they dilatate the

vascular bed of life_important organs. This is so called “centralization of the flow”: rational decrease of blood flow in less important tissues (skin, organs of abdominal cavity, kidneys) in case of aggressive external influence for the protection of life itself (brain, heart, lungs). However influence of shock agents (pain, hypovolemia, destroyed cells, toxic metabolites), extended microcirculation violations (vascular spasm, microthrombosis and sludge) and caused by them tissue ischemia lead to hypoxic affection and cellular death of the internal organs. Further it can bring multiple organ dysfunction syndrome.

Collapse is a vascular failure. It occurs when body is not able to provide blood flow according to the new level of its needs (either because reaction is not fast enough or because sympathetic activation fails). Vascular bed volume and circulating blood volume are disproportional: too much blood gets to the microcirculation vascular reserve and the amount, which returns to the heart is not enough for the systemic needs (so called “decentralization” of the blood flow). Cardiac output and blood pressure decrease, that causes hypoperfusion of the central nervous system and thus unconsciousness and life_threatening complications.

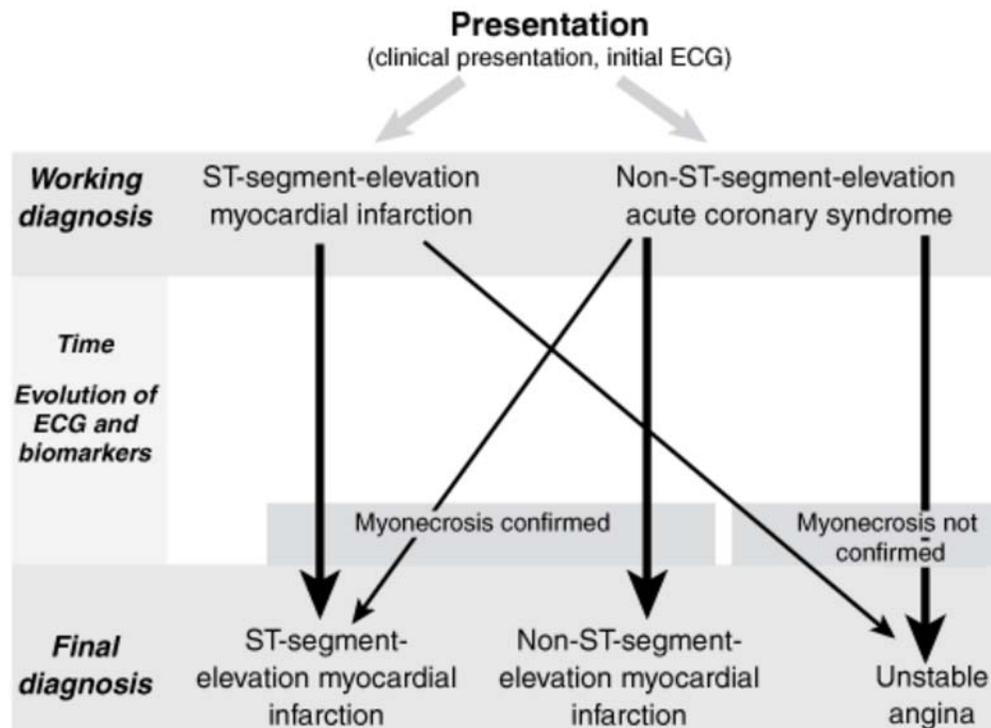
Collapse definition is a bit nominal, because if such reaction extends in time the state of shock develops. Shock itself can equally run as a vascular failure or as a sudden clinical death.

New acute coronary syndromes terminology and implications for diagnosis

The terminology used to describe ACS continues to evolve, with the emergence of the term “non-ST-segment-elevation acute coronary syndrome” (NSTEMACS). This reflects a shift away from establishing a definitive diagnosis at presentation, and towards a more clinically appropriate strategy of forming a rapid working diagnosis with its implications for initial clinical decision making.

At presentation, the initial diagnostic nomenclature focuses on risk stratification to direct treatment strategies. Establishing a definitive diagnosis often requires time, particularly for evidence of myocardial necrosis to emerge, and has important implications pertaining to prognosis, diagnostic coding, and social issues such as insurance and licensure.

1 Defining acute coronary syndromes over time: presentation to final diagnosis



ECG = electrocardiogram.

Initial working diagnosis

The initial working diagnosis is based on the clinical presentation and the initial electrocardiogram (ECG) findings and, in particular, the presence or absence of ST-segment elevation. As the vast majority of patients who present with initial ST-segment elevation develop biochemical evidence of myonecrosis, the term “ST-segment-elevation myocardial infarction” (STEMI) is often used from the outset in these patients.

ACS without ST-segment elevation on the presenting ECG represent a broad spectrum of risk, but are collectively referred to as NSTEMACS. This grouping is useful because emergency reperfusion therapy is not indicated (unless ST-segment elevation develops later), and further investigation is required to classify the patient’s risk and determine the most suitable treatment (see sections on [Investigations](#) and [Management of patients with STEMI](#) for further details).

Final diagnosis

The final diagnostic attribution (ie, clinical label) has important and persisting implications for the patient, both prognostically and socially. Current international criteria for the diagnosis of myocardial infarction have a strong emphasis on biomarkers, specifically troponin, given its high sensitivity and, in particular, specificity for myonecrosis.⁵ The diagnostic criteria for acute, evolving or recent myocardial infarction are defined as:

Typical rise in the serum level of troponin or a more rapid rise in the serum level of the MB isoenzyme of creatine kinase (CK-MB) with at least one of the following:

Ischaemic symptoms;

Development of pathological Q waves on the ECG;

ECG changes indicative of ischaemia (ST-segment elevation or depression); and

Coronary artery intervention (eg, coronary angioplasty or coronary bypass surgery);

or

Pathological findings of an acute myocardial infarction.

This definition requires a temporal appreciation of the cardiac markers, and therefore differentiation between non-ST-segment-elevation myocardial infarction and unstable angina (without evidence of myonecrosis) must be delayed.

Acute management of chest pain

Getting to hospital

Chest discomfort at rest or for a prolonged period (more than 10 minutes, not relieved by sublingual nitrates), recurrent chest discomfort, or discomfort associated with syncope or acute heart failure are considered medical emergencies. Other presentations of ACS may include back, neck, arm or epigastric pain, chest tightness, dyspnoea, diaphoresis, nausea and vomiting. Very atypical pain, including sharp and pleuritic pain, is more common in women, people with diabetes and older people.^{3,7,8}

People experiencing such symptoms should seek help promptly and activate emergency medical services to enable transport to the nearest appropriate health care facility for urgent assessment (grade D recommendation). Ideally, transport should be by ambulance. However, where ambulance response times are long, alternatives may need to be considered. Patients should be strongly discouraged from driving themselves because of the risk to other road users.

The most important initial requirement is access to a defibrillator to avoid early cardiac death from reversible arrhythmias. All Australian ambulances now carry defibrillators, and there is promise in further exploring public access defibrillation opportunities. In the case of cardiac arrest occurring in a setting where a defibrillator is not immediately available, cardiopulmonary resuscitation should be commenced immediately.

Actions in transit

Aspirin (300 mg) should be given unless already taken or contraindicated (grade A recommendation), and should preferably be given early (eg, by emergency or ambulance personnel) (grade D recommendation). Oxygen should also be given (grade D recommendation).

Glyceryl trinitrate and intravenous morphine should be given as required (grade D recommendation).

Where appropriate, a 12-lead ECG should be taken en route and transmitted to a medical facility (grade B recommendation).

Receiving medical facilities should be given warning of incoming patients in whom there is a high suspicion of ACS, particularly STEMI, or those whose condition is unstable (grade B recommendation).

Where formal protocols are in place, prehospital treatment should be given, including fibrinolysis in appropriate cases (grade A recommendation). See [section on management of patients with STEMI](#) for further discussion of prehospital fibrinolysis.

On arrival

All patients presenting with suspected ACS should be subject to ongoing surveillance and have an ECG completed within 5 minutes of arrival at the medical facility (grade A recommendation). The ECG should be assessed promptly by an appropriately qualified person (grade D recommendation).

Oxygen and pain control should be given as required (grade D recommendation).

People experiencing symptoms of ACS should seek help promptly and activate emergency medical services.

The most important initial requirement is access to a defibrillator to avoid early cardiac death from reversible arrhythmias.

Aspirin should be given early (eg, by emergency or ambulance personnel) unless already taken or contraindicated.

Oxygen should be given, as well as glyceryl trinitrate and intravenous morphine as required.

As a minimum, receiving medical facilities should be given warning of incoming patients in whom there is a high suspicion of ACS, particularly STEMI, or whose condition is unstable.

Where appropriate, a 12-lead ECG should be taken en route and transmitted to a medical facility.

Where formal protocols are in place, prehospital treatment (including fibrinolysis in appropriate cases) should be facilitated.

Patients presenting with a suspected ACS should undergo immediate electrocardiography. Further investigations may be necessary, but should not delay treatment.

While other serious diagnoses can present similarly to ACS (eg, pulmonary embolism, aortic dissection, pericarditis), once these have been excluded and ACS is considered the most likely diagnosis further delay in treatment is unnecessary and inappropriate.

Investigations and invasive vascular access techniques should not delay reperfusion therapy if indicated on the basis of ST-segment elevation on the ECG.

Patients whose condition is unstable should have early consultation with a cardiologist.

Cardiac biomarkers are becoming increasingly important to the diagnosis of myocardial infarction. See [Box 2](#) for recommendations and rationale regarding their measurement.

Electrocardiography

Electrocardiography is necessary to detect ischaemic changes or arrhythmias. It should be noted that the initial ECG has a low sensitivity for ACS, and a normal ECG does not rule out ACS. However, the ECG is the sole test required to select patients for emergency reperfusion (fibrinolytic therapy or direct PCI). Patients with STEMI who present within 12 hours of the onset of ischaemic symptoms should have a reperfusion strategy implemented promptly (grade A recommendation) — see the [section on management of patients with STEMI](#) for recommendations.

Accurate ECG interpretation in a patient with chest pain is critical. Basically, there can be three types of problems - ischemia is a relative lack of blood supply (not yet an infarct), injury is acute damage occurring right now, and finally, infarct is an area of dead myocardium. It is important to realize that certain leads represent certain areas of the left ventricle; by noting which leads are involved, you can localize the process. The prognosis often varies depending on which area of the left ventricle is involved (i.e. anterior wall myocardial infarct generally has a worse prognosis than an inferior wall infarct).

[Video: MI site and coronary thrombosis](#)

V1-V2	anteroseptal wall
V3-V4	anterior wall
V5-V6	anterolateral wall
II, III, aVF	inferior wall
I, aVL	lateral wall
V1-V2	posterior wall (reciprocal)

Infarct	
1. Ischemia	Represented by symmetrical T wave inversion (upside down). The definitive leads for ischemia are: I, II, V2 - V6.
2. Injury	Acute damage - look for elevated ST segments. (Pericarditis and cardiac aneurysm can also cause ST elevation; remember to correlate it with the patient.

3. Infarct	Look for significant "pathologic" Q waves. To be significant, a Q wave must be at least one small box wide or one-third the entire QRS height. Remember, to be a Q wave, the initial deflection must be down; even a tiny initial upward deflection makes the apparent Q wave an R wave.
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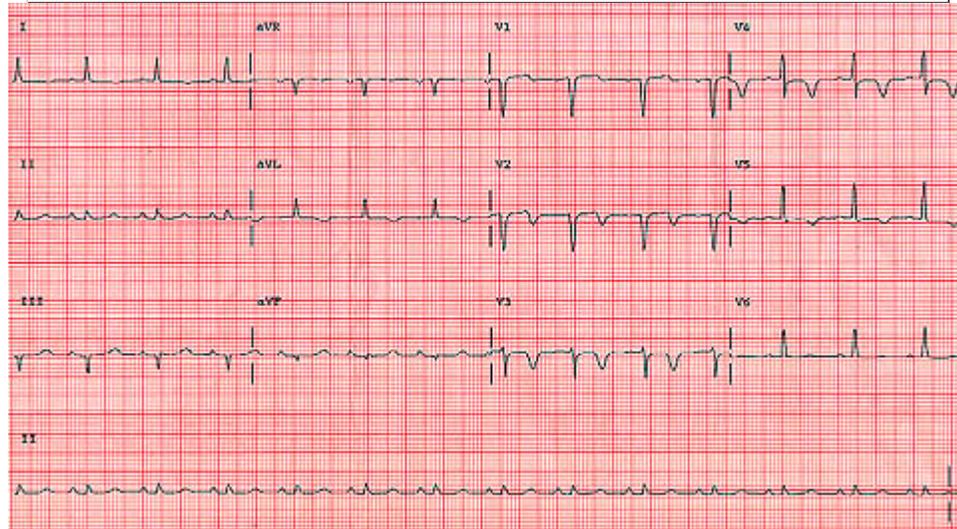


Figure: Ischemia: Note symmetric T wave inversions in leads I, V2-V5.

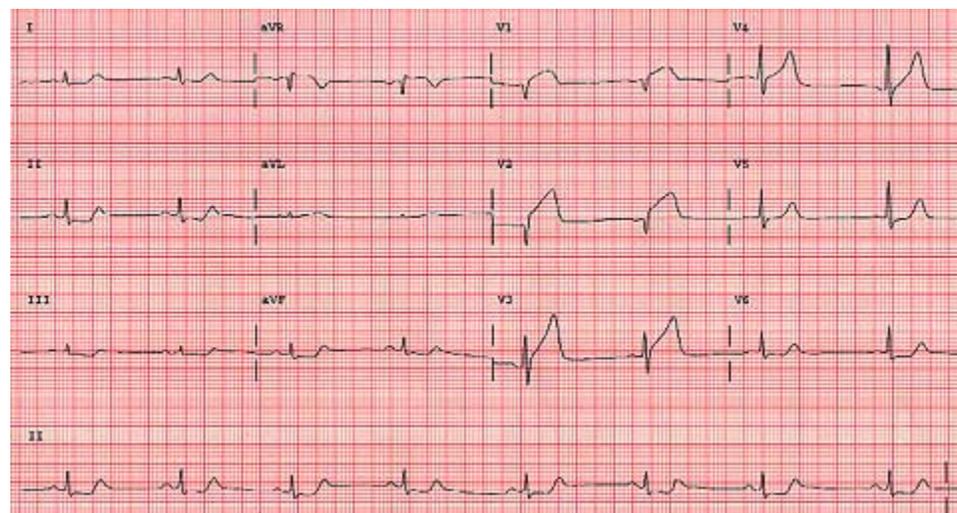


Figure: Injury: Note ST segment elevation in leads V2-V3 (anteroseptal/anterior wall).

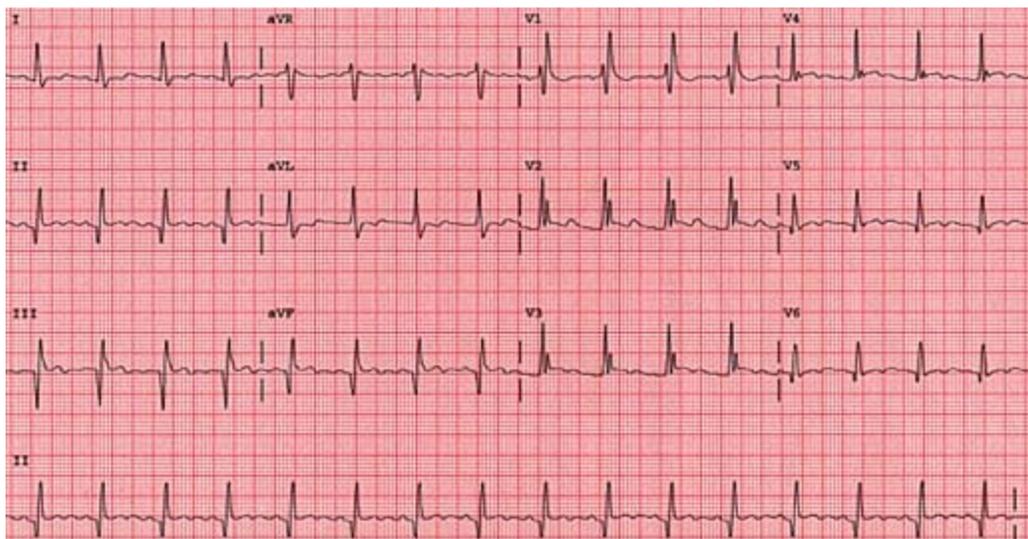


Figure 36: Infarct: Note Q waves in leads II, III, and aVF (inferior wall).

For the posterior wall, remember that vectors representing depolarization of the anterior and posterior portion of the left ventricle are in opposite directions. So, a posterior process shows up as opposite of an anterior process in V1. Instead of a Q wave and ST elevation, you get an R wave and ST depression in V1.

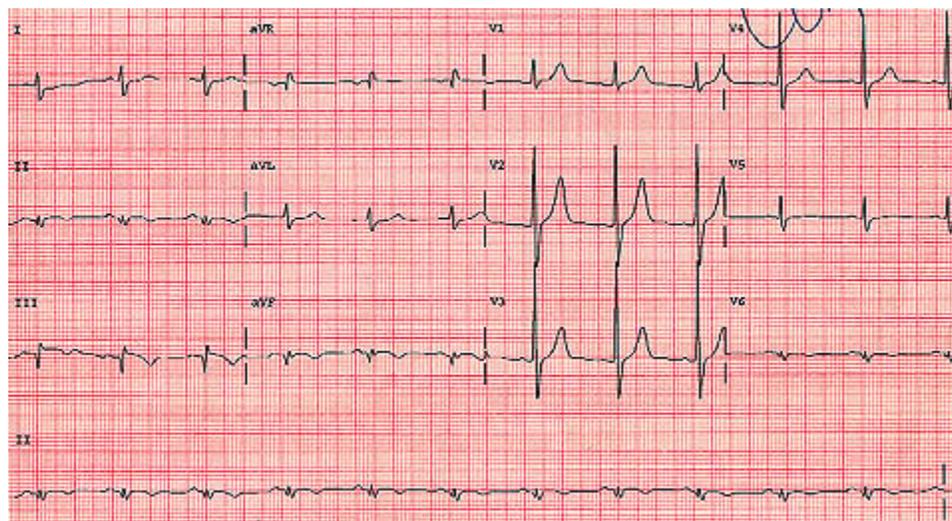


Figure: Posterior wall infarct. Notice tall R wave in V1. Posterior wall infarcts are often associated with inferior wall infarcts (Q waves in II, III and aVF).

Two other caveats: One is that normally the R wave gets larger as you go to V1 to V6. If there is no R wave "progression" from V1 to V6 this can also mean infarct. The second caveat is that, with a left bundle branch block, you cannot evaluate "infarct" on that ECG. In a patient with chest pain and left bundle branch block, you must rely on cardiac enzymes (blood tests) and the history.

Blood tests

Measurements should include:

Serum troponin I or T levels (or CK-MB if troponin is not available).

Full blood count.

Serum creatinine and electrolyte levels, particularly potassium concentration, as hypokalaemia is associated with an increased risk of arrhythmias, especially ventricular fibrillation¹⁰ (grade B recommendation). Knowledge of kidney function (expressed as estimated glomerular filtration rate) is strongly encouraged (grade B recommendation) given the association between renal impairment and adverse outcomes (evidence level III).¹¹

Serum creatine kinase (CK) level.

Serum lipid levels (fasting levels of total cholesterol, low-density-lipoprotein cholesterol, high-density-lipoprotein cholesterol and triglycerides) within 24 hours.

Blood glucose level.

Chest x-ray

A chest x-ray is useful for assessing cardiac size, evidence of heart failure and other abnormalities (grade D recommendation), but should not delay reperfusion treatment where indicated.

Further investigations

Patients without ST-segment elevation on the initial ECG should be further observed and investigated to promptly identify patients suitable for an emergency reperfusion strategy (based on ECG changes) and/or determine the best management protocol for NSTEMI based on risk stratification (see section on [Management of patients with NSTEMI](#)).

The ECG is the sole test required to select patients for emergency reperfusion (fibrinolytic therapy or direct PCI).

Patients with STEMI who present within 12 hours of the onset of ischaemic symptoms should have a reperfusion strategy implemented promptly.

Patients with a suspected ACS without ST-segment elevation on the ECG should undergo further observation and investigation to rule out other diagnoses, enable risk stratification and determine the most appropriate treatment strategy.

Patients with a normal ECG and cardiac biomarker levels after an appropriate period of observation should, where practicable, undergo provocative testing (eg, stress test) before discharge.

2 Recommendations and rationale for measuring cardiac biomarker levels

Cardiac

Recommendation Rationale

biomarker

Troponin level(Cardiac troponin I or T)

On arrival

Troponin rise indicates myonecrosis, and is a high-risk feature in NSTEMACS. Troponin is the preferred marker because about a third of patients with elevated troponin, but normal CK and CK-MB levels, will develop an adverse outcome.⁹

Not repeated if positive

Troponin remains elevated for 5–14 days, and therefore may not be useful for identifying early re-infarction.

Repeated > 8 hours after last episode of pain or other symptoms of coronary insufficiency if initially negative

Troponin elevation is often delayed by 4–6 hours. Therefore, repeat troponin testing is

necessary to identify patients at high risk who may benefit from aggressive therapy and an early invasive strategy.

Serial troponin measurements of typical in patients with rise of NSTEMI/ACS troponin suspected to be indicates high risk NSTEMI/ACS and may be an indication for more aggressive therapy.

Total level CK Serial measurements performed for 48 hours in patients with myocardial infarction Can be remeasured to confirm a second event if re-infarction is suspected later.

CK-MB level Should be measured in all patients with an ACS if troponin assay unavailable While troponin is the preferred marker of myocardial damage, if it is unavailable CK-MB is more specific

than CK for myocardial injury. CK-MB may also be used to confirm a re-infarction.

NSTEACS = non-ST-elevation acute coronary syndromes. CK = creatine kinase. CK-MB = creatine kinase-MB isoenzyme. ACS = acute coronary syndrome

Management of patients with ST-segment-elevation myocardial infarction

STEMI is defined as presentation with clinical symptoms consistent with an acute coronary syndrome with ECG features including any of:

- § **Persistent ST-segment elevation of ≥ 1 mm in two contiguous limb leads;**
- § **ST-segment elevation of ≥ 2 mm in two contiguous chest leads; or**
- § **New left bundle branch block (LBBB) pattern.⁶ (Note that LBBB is presumed new unless there is evidence otherwise; echocardiography may be useful to detect regional wall contraction abnormalities.)**

Patients with STEMI usually have a completely occluded coronary artery with thrombus at the site of a ruptured plaque. Restoring coronary patency as promptly as possible is a key determinant of short-term and long-term outcomes (level I evidence).¹⁴⁻¹⁸

Patients with STEMI who present within 12 hours of the onset of ischaemic symptoms should have a reperfusion strategy implemented promptly (grade A recommendation).

Reperfusion therapy

Reperfusion may be obtained with fibrinolytic therapy or PCI. A combination of fibrinolysis and PCI may also be used (facilitated or rescue PCI). Coronary artery bypass graft (CABG) surgery may occasionally be more appropriate — particularly in patients who have suitable anatomy and are not candidates for fibrinolysis or PCI. CABG surgery may also be considered in patients with cardiogenic shock¹⁹ or in association with mechanical repair.¹²

Antiplatelet therapy

Aspirin (300 mg) should be given to all patients with STEMI unless contraindicated and, in the absence of significant side effects, low-dose therapy should be continued in the long term (grade A recommendation).[16,20](#)

There is evidence that clopidogrel (300–600 mg loading dose) should be prescribed in addition to aspirin for patients undergoing PCI with a stent.[21-23](#) In patients selected for fibrinolytic therapy, clopidogrel (300 mg) should be given in addition to aspirin, unless contraindicated (grade B recommendation).[24](#) Note, however, that if it is thought that the patient is likely to require CABG acutely, clopidogrel should be withheld.

Clopidogrel (75 mg daily) should be continued for at least a month after fibrinolytic therapy, and for up to 12 months after stent implantation, depending on the type of stent and circumstances of implantation (level II evidence; grade B recommendation).[25](#)

Antithrombin therapy

With PCI: Antithrombin therapy should be used in conjunction with PCI (grade A recommendation). The dose of unfractionated heparin therapy will depend on concomitant use of glycoprotein (GP) IIb/IIIa inhibitors. The aim should be to obtain an activated clotting time (ACT) between 200 and 300 seconds if using GP IIb/IIIa inhibitors, or between 300 and 350 seconds if these drugs are not used (grade B recommendation). It may be advisable to give a bolus of heparin while the patient is in transit to the catheterisation laboratory (grade D recommendation).

The role of enoxaparin in acute STEMI in conjunction with PCI remains to be determined, but it appears to be safe and effective at a dose of 0.75 mg/kg (grade D recommendation).

With fibrinolysis: Antithrombin therapy should be used with fibrin-specific fibrinolytic agents (grade A recommendation).[26,27](#)

Unfractionated heparin should be given as an initial bolus dose of 60 units per kilogram of body weight (with a maximum dose of 4000 units) followed by an initial infusion of 12 units per kilogram per hour (maximum units 1000 per hour), adjusted to attain the activated partial thromboplastin time (APTT) at 1.5 to 2 times control (about 50–70 seconds; grade B recommendation).[12](#)

Enoxaparin may be used in conjunction with fibrin-specific fibrinolytic agents in patients under the age of 75 years, provided they do not have significant renal dysfunction. An intravenous bolus dose of 30 mg followed by a 1 mg/kg subcutaneous injection every 12 hours in combination with tenecteplase is the most comprehensively studied therapy.[12](#) Care should be taken in patients who are aged over 75 years, or who have renal dysfunction, as dose adjustment is required.[12](#)

The use of antithrombin therapy in conjunction with streptokinase therapy is optional.[28](#)

Glycoprotein IIb/IIIa inhibitors

It is reasonable to use abciximab with primary PCI, although there are conflicting data (grade B

recommendation). It appears the earlier it is used, the greater the advantage.²⁹ When used in patients with STEMI undergoing primary PCI, the timing of administration of abciximab is a matter of clinical judgement.^{30,31}

Full-dose GP IIb/IIIa inhibitors should be avoided with fibrinolytic therapy (grade B recommendation) as there is evidence of excessive bleeding (including intracranial haemorrhage) with this combination.³² It is unclear how early full-dose GP IIb/IIIa inhibitors can be safely given after fibrinolysis, but it is probably at least 4 hours after administration of fibrin-specific fibrinolytic agents and 24 hours after administration of streptokinase.³²

The combination of GP IIb/IIIa inhibitors with reduced doses of fibrinolytic therapy is not recommended. There is no significant advantage over full-dose fibrinolytic therapy alone, and the risk of bleeding is increased, particularly in the elderly.³² This combination has been used for facilitated PCI.³²

§ All patients undergoing reperfusion therapy (PCI or fibrinolysis) for STEMI should be given aspirin and clopidogrel unless contraindicated.

§ Antithrombin therapy should be given in combination with PCI or fibrinolytic therapy with fibrin-specific fibrinolytic agents, but its use in conjunction with streptokinase is optional.

§ It is reasonable to use abciximab with primary PCI, but GP IIb/IIIa inhibitors should generally be avoided with full or reduced doses of fibrinolytic therapy.

3 Summary of adjuvant therapy associated with reperfusion

Medication	Primary percutaneous coronary intervention	Fibrin-specific fibrinolytic
Aspirin	Yes	Yes
Clopidogrel	Yes (unless the need for acute CABG is likely)	Yes (unless the need for acute CABG is likely)
Heparin	Unfractionated heparin (ACT 200–300 s if using glycoprotein IIb/IIIa inhibitors, 300–350 s if not) or Enoxaparin*	Unfractionated heparin (APTT 1.5–2 times control [approx 50–70 s]) or Enoxaparin*
Glycoprotein IIb/IIIa inhibitors	Abciximab optional	No

CABG = coronary artery bypass graft. ACT = activated clotting time. APTT = activated partial thromboplastin time.

* Care should be taken in patients aged over 75 years, or those who have significant renal dysfunction — dose adjustment is required.

Choice of reperfusion therapy

§ Choice of reperfusion strategy depends on a number of factors, with time delay (both to presentation and potential PCI or fibrinolytic therapy) playing a major role in determining best management.

§ In general, PCI is the treatment of choice, provided it can be performed promptly by a qualified interventional cardiologist in an appropriate facility.

§ In general, the maximum acceptable delay from presentation to balloon inflation is:

- 60 minutes if a patient presents within 1 hour of symptom onset; and
- 90 minutes if a patient presents later.

Note: for patients who present late (3–12 hours after symptom onset) to a facility without PCI capability, it is appropriate to consider transfer for primary PCI if balloon inflation can be achieved within 2 hours (including transport time).

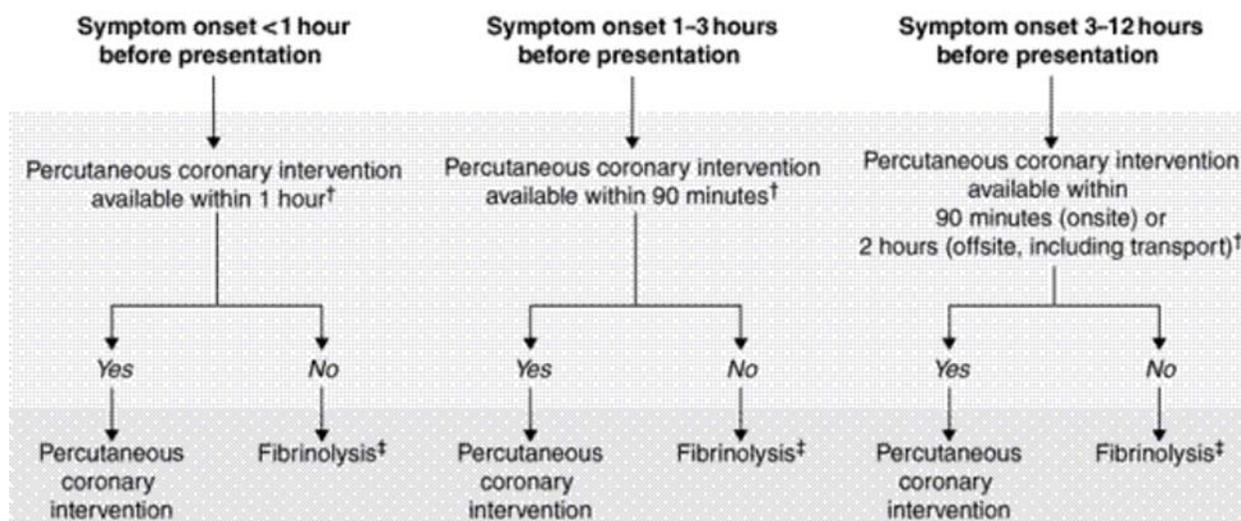
§ All PCI facilities should be able to perform angioplasty within 90 minutes of patient presentation.

§ Fibrinolysis should be considered early if PCI is not readily available, particularly in rural and remote areas.

§ When there are major delays to hospitalisation (more than 30 minutes), prehospital fibrinolysis should be considered.

§ Reperfusion is not routinely recommended in patients who present more than 12 hours after symptom onset and who are asymptomatic and haemodynamically stable.

4 Hospital management of ST-segment-elevation myocardial infarction*



Assuming no contraindications to fibrinolytic therapy — see [Box 5](#). [†] Time delay refers to time from first medical contact to balloon. [‡] Patients with ongoing symptoms or instability should be transferred for PCI.

Note: Reperfusion after 12 hours is indicated for cardiogenic shock, ongoing pain or

haemodynamic instability (see text).

5 Contraindications and cautions for fibrinolysis use in ST-segment-elevation myocardial infarction*

Absolute contraindications

Risk of bleeding

- § Active bleeding or bleeding diathesis (excluding menses)
- § Significant closed head or facial trauma within 3 months
- § Suspected aortic dissection (including new neurological symptoms)[50](#)

Risk of intracranial haemorrhage

- § Any prior intracranial haemorrhage
- § Ischaemic stroke within 3 months
- § Known structural cerebral vascular lesion (eg, arteriovenous malformation)
- § Known malignant intracranial neoplasm (primary or metastatic)

Relative contraindications

Risk of bleeding

- § Current use of anticoagulants: the higher the international normalised ratio (INR), the higher the risk of bleeding
- § Non-compressible vascular punctures
- § Recent major surgery (< 3 weeks)
- § Traumatic or prolonged (> 10 minutes) cardiopulmonary resuscitation
- § Recent (within 4 weeks) internal bleeding (eg, gastrointestinal or urinary tract haemorrhage)
- § Active peptic ulcer

Risk of intracranial haemorrhage

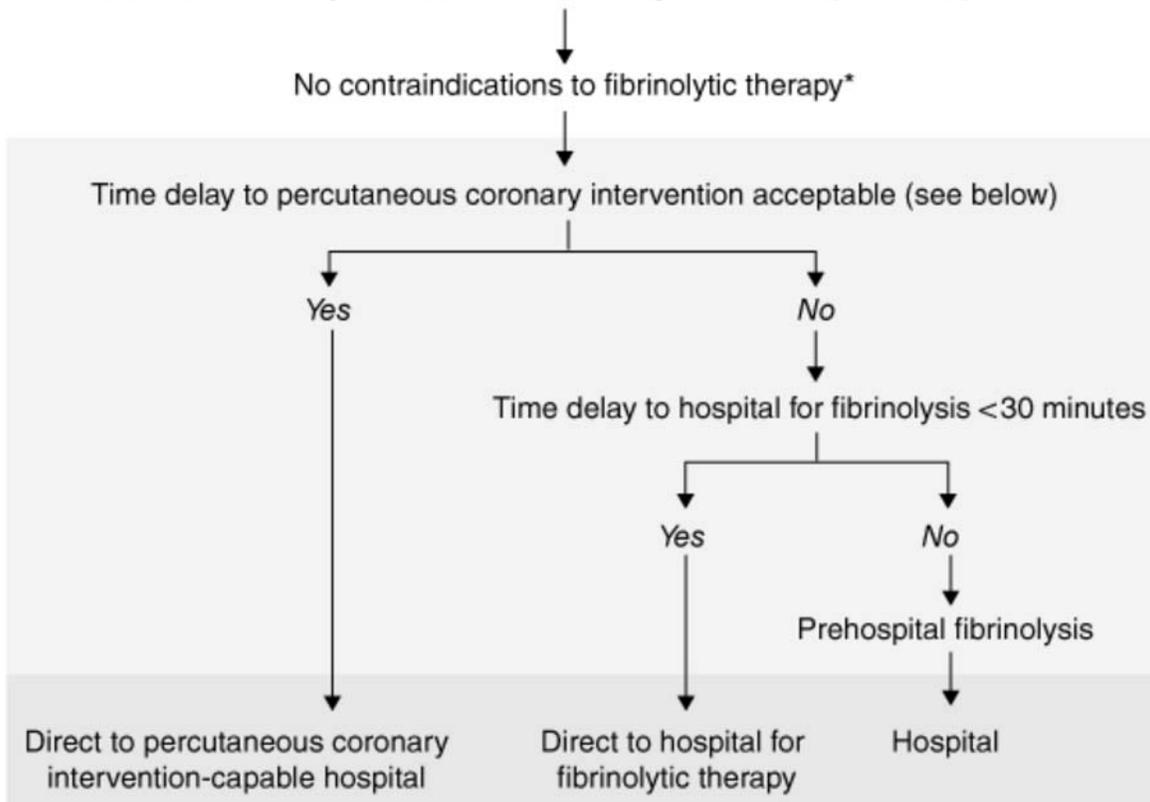
- § History of chronic, severe, poorly controlled hypertension
- § Severe uncontrolled hypertension on presentation (> 180 mmHg systolic or > 110 mmHg diastolic)
- § Ischaemic stroke more than 3 months ago, dementia, or known intracranial abnormality not covered in contraindications

Other

- § Pregnancy
-

6 Prehospital management of ST-segment-elevation myocardial infarction (STEMI)

STEMI confirmed by 12-lead electrocardiogram with expert interpretation



Time since onset of symptoms

	< 1 hour	1–3 hours	3–12 hours	> 12 hours
Acceptable delay to percutaneous coronary intervention (from first medical contact to balloon inflation)	60 minutes	90 minutes	120 minutes	Not routinely recommended (see text)

*If fibrinolysis is contraindicated, it is important that an attempt to reperfuse is made, even if there is a long delay (up to 12 hours).

Choice of fibrinolytic agent

There are four fibrinolytic agents currently available in Australia; streptokinase and the tissue

fibrin-specific fibrinolytic agents alteplase, reteplase and tenecteplase. The properties of these agents are summarised in [Box 7](#).

Fibrin-specific fibrinolytic agents have been shown to reduce mortality compared with streptokinase in patients with STEMI who present within 6 hours of symptom onset (level I evidence).⁵⁵ Fibrin-specific fibrinolytic agents also lack the significant acute side effects of hypotension and allergy caused by streptokinase (level I evidence). Streptokinase may be associated with a lower incidence of intracranial haemorrhage, particularly in older people (level I evidence), but the overall mortality is still lower with the use of fibrin-specific fibrinolytic agents (level II evidence).⁵⁶ Tenecteplase is associated with a lower rate of bleeding than alteplase (level II evidence).⁵⁷

Second-generation fibrin-specific fibrinolytic agents can be given as either single or double bolus injections, which makes them significantly easier to use than streptokinase.

In combination therapy, PCI combined with fibrin-specific fibrinolytic agents appears to have greater efficacy and results in fewer complications than PCI combined with streptokinase (level III evidence, grade B recommendation).⁵⁸

Streptokinase should not be given to patients with previous exposure (more than 5 days ago) to the drug (grade B recommendation). There is also evidence that streptokinase may be less effective in Aboriginal and Torres Strait Islander peoples because of the high levels of skin infection (and thus streptococcal antibodies), particularly in remote populations.^{45,59} It is therefore an inappropriate choice of agent in these populations (level III evidence, grade B recommendation). Making second-generation fibrin-specific fibrinolytic agents the standard choice is likely to decrease inequalities of care between Indigenous and non-Indigenous populations, in addition to providing superior reperfusion.

Therefore, second-generation fibrin-specific fibrinolytic agents which are available as a bolus (ie, reteplase, tenecteplase) are the fibrinolytics of choice (grade A recommendation). These agents should be available at all centres where fibrinolysis may be required (grade D recommendation).

§ Second-generation fibrin-specific fibrinolytic agents that are available as a bolus (ie, reteplase, tenecteplase) are the fibrinolytics of choice.

§ These agents should be available at all centres where fibrinolysis may be required.

§ Streptokinase is a particularly inappropriate choice for Aboriginal and Torres Strait Islander patients or patients with previous exposure to the drug.

§

7 Fibrinolytic agents — prescribing information and properties

Details	Tenecteplase (TNK)	Reteplase (r-PA)	Alteplase (rt-PA)	Streptokinase
Dose (also see product)	Up to 10 000 international	to 10 units	30 minutes	1.5 million international
		30 minutes	2, For patients > 65 kg:	

information) units (50 mg) on basis of apart (give 15 mg bolus; units over
 body weight: < 60 kg — each bolus then 50 mg 30–60 minutes
 6000 units 60–70 kg — slowly over no over 30
 7000 units 70–80 kg — more than minutes and
 8000 units 80–90 kg — 2 minutes) 35 mg over
 9000 units > 90 kg — the next
 10 000 units 60 minutes
 For patients ≤
 65 kg: 15 mg
 bolus; then
 0.75 mg/kg
 over
 30 minutes
 and 0.5 mg/kg
 over the next
 60 minutes.

Bolus administration	Yes	Yes	No	No
Antigenic ¹²	No	No	No	Yes
Systemic fibrinogen depletion ¹²	Minimal	Moderate	Mild	Marked
Lives saved per 1000 patients treated (approximate number at 30 days)	3553	3554	3555	2514

§ Patients who have had STEMI should be considered for early transfer to a tertiary cardiac centre with PCI facilities and links to cardiac surgical facilities.

§ If early transfer is not possible, all patients should be transferred or referred as soon as is practicable for assessment of the need for revascularisation through PCI or CABG.

Management of patients with non-ST-segment-elevation acute coronary syndromes. Risk stratification

The initial objective of evaluation is to define the likelihood of an ACS as the cause of a patient's presentation. Most patients will present with prolonged or recurrent central chest

discomfort but others, particularly the elderly, people with diabetes and women, may present with atypical symptoms. These include neck, jaw, back or epigastric discomfort or dyspnoea, diaphoresis, nausea and vomiting. Age is an important risk factor, and the presence (or absence) of coronary risk factors adds little to the accuracy of the diagnosis in middle-aged or elderly patients, but is more useful in making a diagnosis in younger patients. A history of physical or emotional stress before symptom onset increases the likelihood of an ACS. Most patients with NSTEMACS are normal on physical examination. An abnormal ECG, particularly dynamic ST-segment deviation (≥ 0.5 mm) or new T-wave inversion (≥ 2 mm) will confirm the diagnosis, but the ECG may be normal or show minor changes in up to 50% of cases.

The second objective of evaluation is to determine the risk of short-term adverse outcomes, which will direct the management strategy. [Box 8](#) provides a paradigm for the risk stratification of patients presenting with suspected NSTEMACS, and a simplified risk assessment algorithm is shown in [Box 9](#). Most patients admitted to hospital with possible NSTEMACS will have intermediate-risk or high-risk features ([Box 8](#)), and these patients are best managed with a structured clinical pathway (see [Investigation](#) section). Patients with clinical features consistent with NSTEMACS and high-risk features are best managed with aggressive medical and invasive therapy (detailed later). Patients with diabetes or chronic kidney disease with typical symptoms of ACS would be considered to be at high risk, but those with atypical symptoms and normal ECGs and cardiac biomarker levels may initially be considered at intermediate risk until a diagnosis is made. Patients with low-risk unstable angina may be managed with upgraded medical therapy and outpatient cardiac referral.

8 Features associated with high-risk, intermediate-risk and low-risk non-ST-segment-elevation acute coronary syndromes (NSTEMACS)

High-risk features

Presentation with clinical features consistent with acute coronary syndromes (ACS) and any of the following high-risk features:

- § Repetitive or prolonged (> 10 minutes) ongoing chest pain or discomfort;
- § Elevated level of at least one cardiac biomarker (troponin or creatine kinase-MB isoenzyme);
- § Persistent or dynamic electrocardiographic changes of ST-segment depression ≥ 0.5 mm or new T-wave inversion ≥ 2 mm;
- § Transient ST-segment elevation (≥ 0.5 mm) in more than two contiguous leads;
- § Haemodynamic compromise — systolic blood pressure < 90 mmHg, cool peripheries, diaphoresis, Killip Class $> I$, and/or new-onset mitral regurgitation;
- § Sustained ventricular tachycardia;
- § Syncope;
- § Left ventricular systolic dysfunction (left ventricular ejection fraction < 0.40);
- § Prior percutaneous coronary intervention within 6 months or prior coronary artery bypass surgery;

- § Presence of known diabetes (with typical symptoms of ACS); or
- § Chronic kidney disease (estimated glomerular filtration rate < 60 mL/minute) (with typical symptoms of ACS).

Intermediate-risk features

Presentation with clinical features consistent with ACS and any of the following intermediate risk features AND NOT meeting the criteria for high-risk ACS:

- § Chest pain or discomfort within the past 48 hours that occurred at rest, or was repetitive or prolonged (but currently resolved);
- § Age > 65 years;
- § Known coronary heart disease — prior myocardial infarction with left ventricular ejection fraction ≥ 0.40 , or known coronary lesion more than 50% stenosed;
- § No high-risk changes on electrocardiography (see above);
- § Two or more of the following risk factors: known hypertension, family history, active smoking or hyperlipidaemia;
- § Presence of known diabetes (with atypical symptoms of ACS);
- § Chronic kidney disease (estimated glomerular filtration rate < 60 mL/minute) (with atypical symptoms of ACS); or
- § Prior aspirin use.

Low-risk features

Presentation with clinical features consistent with an acute coronary syndrome without intermediate-risk or high-risk features. This includes onset of anginal symptoms within the last month, or worsening in severity or frequency of angina, or lowering of anginal threshold.

Treatment of NSTEMI/ACS

Aspirin is recommended (unless contraindicated) in all low-risk, intermediate-risk and high-risk patients (grade A recommendation).

High-risk patients should be treated with aggressive medical management (level I evidence, grade A recommendation) (see below) and arrangements should be made for coronary angiography and revascularisation (level I evidence, grade A recommendation), except in those with severe comorbidities, including general frailty (grade A recommendation). Age alone should not be a barrier to aggressive therapy.

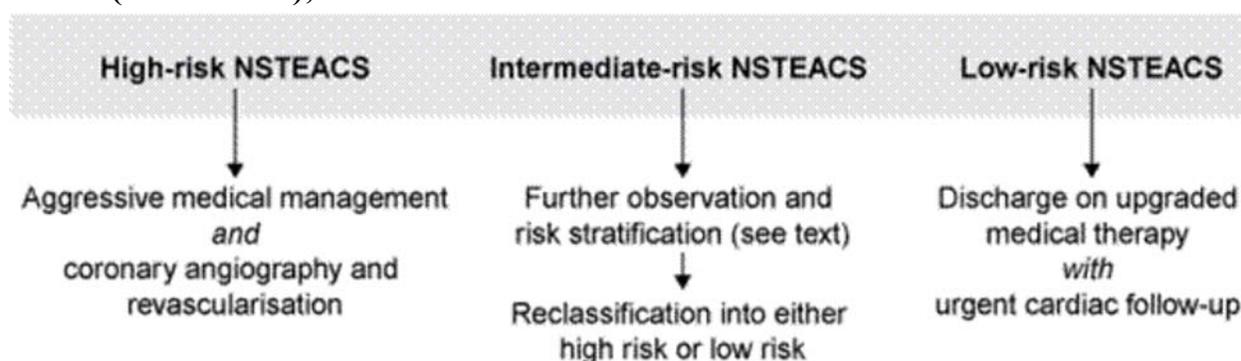
Patients at intermediate risk should be observed by staff trained in cardiac care practice and should undergo an accelerated diagnostic evaluation and further risk stratification (level III evidence, grade B recommendation). Accurate assessment can be improved by the use of structured forms for admission and continuing evaluation.⁷⁹⁻⁸² During the evaluation process, intermediate-risk patients are observed as described in the [Investigations](#) section, with frequent

electrocardiography (with or without continuous ST-segment monitoring), repeat troponin testing and provocative testing if a repeat troponin assay is negative.

Low-risk patients may be discharged on upgraded medical therapy after an appropriate period of observation and assessment (see [Investigations](#) section). These patients (including those manifesting anginal symptoms for the first time within the previous month or with a change in the tempo of their angina) are considered unstable, as some will have atherothrombotic disease with a definite risk of progression to myocardial infarction. These patients should be treated with β -blockers and aspirin, and cardiac assessment should be obtained urgently.

Treatment of patients with NSTEMACS on the basis of risk is summarised in [Box 10](#).

10 Treatment strategies for patients with non-ST-segment-elevation acute coronary syndromes (NSTEMACS), based on risk stratification



Medical management of high-risk patients

Antiplatelet therapy: Early treatment should be initiated with aspirin⁸³⁻⁸⁶ (grade A recommendation) and clopidogrel (300 mg loading dose and 75 mg daily)⁸⁷ (grade B recommendation), with the following considerations:

- § Clopidogrel should be avoided in patients likely to require emergency coronary bypass surgery (those with severe widespread ST-segment depression or haemodynamic instability);
- § If possible, clopidogrel should be discontinued 5 days before coronary bypass surgery;
- § Clopidogrel should be given (preferably more than 6 hours) before planned percutaneous coronary intervention (level I evidence, grade A recommendation),^{21,22} but may be omitted if coronary angiography is planned immediately;
- § If relevant, warfarin therapy should be discontinued and heparin given along with the recommended antiplatelet therapy (grade D recommendation).

Antithrombin therapy: Unfractionated heparin or subcutaneous enoxaparin should be given until angiography or for 48–72 hours (level I evidence, grade A recommendation).⁸⁸⁻⁹⁰ The enoxaparin dose must be reduced in patients with impaired renal function.

GP IIb/IIIa inhibitors: Intravenous tirofiban or eptifibatid is particularly recommended in high-risk patients in whom an invasive strategy is planned (level I evidence, grade A

recommendation). Administration should commence as soon as a high-risk feature is identified.^{91,92} Intravenous tirofiban or eptifibatid are also recommended if patients continue to have ischaemia while receiving enoxaparin or unfractionated heparin (level III evidence, grade B recommendation).

Concomitant tirofiban is particularly beneficial and recommended in patients with diabetes (level I evidence, grade A recommendation).

Other: A β -blocker should be given unless contraindicated (level I evidence, grade A recommendation).⁹³ Intravenous glyceryl trinitrate can be given for refractory pain (grade D recommendation).

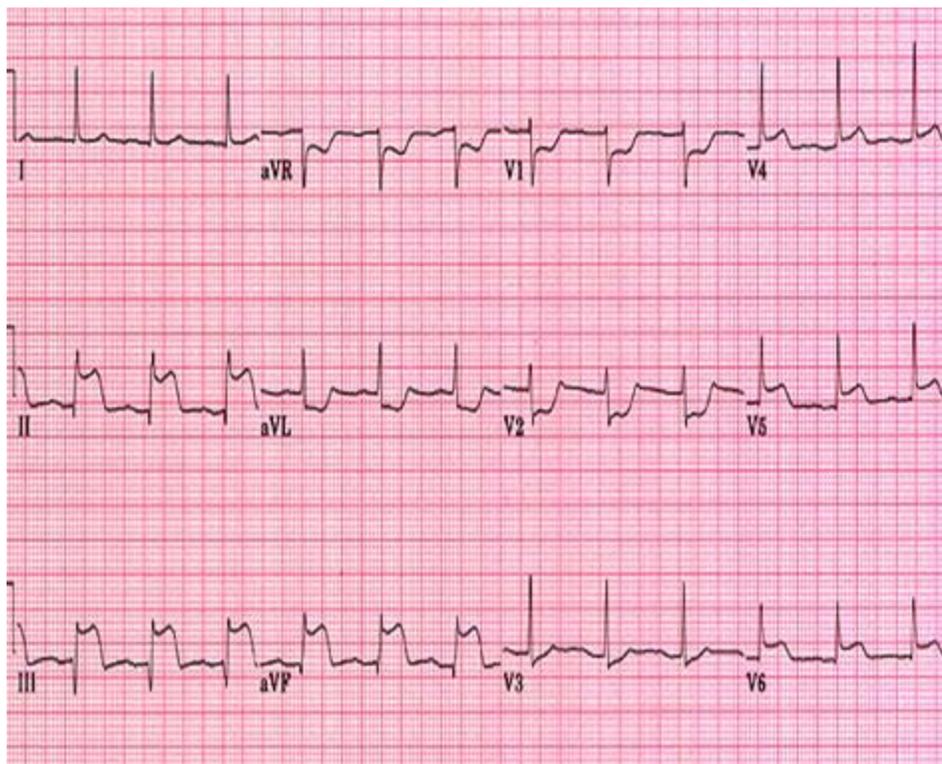
In patients with diabetes, good glycaemic control should be targeted in hospital and after discharge. This may require considering an insulin-based regimen in hospital and for 3 or more months after discharge in selected patients (grade B recommendation).⁹⁴

Early medical management of NSTEMACS is summarised in [Box 11](#).

Early coronary angiography (within 48 hours) and revascularisation is recommended in patients with NSTEMACS and high-risk features (grade A recommendation), except in patients with severe comorbidities. In addition to the features listed in [Box 8](#), pain or ischaemia refractory to medical therapy and high-risk features on early exercise testing can also identify patients suitable for early invasive therapy.

A risk score devised by the TIMI (Thrombolysis In Myocardial Infarction) study group⁹⁵ has been validated as a valuable measure of early risk in NSTEMACS.^{66,91,96} It uses a seven-point score derived from:

- § age greater than or equal to 65 years;
- § more than three coronary risk factors;
- § prior angiographic coronary obstruction;
- § ST-segment deviation;
- § more than two angina events within 24 hours;
- § use of aspirin within 7 days; and
- § elevated levels of cardiac biomarkers.



§

§

Fig. ECG with MI

Additional risk stratification on the basis of a TIMI risk score of greater than three for deciding which patients might be transferred for early invasive management may be considered where funding is constrained, but it must be remembered that 14-day cardiac event rates are still considerable, even for those with low scores (see [Box 12](#)). Appropriate patients should be transferred for angiography within 48 hours, and aggressive medical therapy with initial stabilisation of symptoms does not mitigate the need for early angiography.

12 TIMI risk scores and 14-day cardiac event rates

14-day adverse cardiac event
TIMI score rate95

0/1	4.7%
2	8.3%
3	13.2%
4	19.9%
5	26.2%
6/7	40.9%

TIMI = Thrombolysis In Myocardial Infarction study group.

§ All patients with NSTEMI should have their risk stratified to direct management decisions.

§ All patients with NSTEMI should be given aspirin unless contraindicated.

§ Patients with high-risk NSTEMI should be treated with aggressive medical management (including aspirin and clopidogrel, unfractionated heparin or subcutaneous enoxaparin, intravenous tirofiban or eptifibatid, and a β -blocker), and arrangements should be made for coronary angiography and revascularisation, except in those with severe comorbidities.

§ Patients with intermediate-risk NSTEMI should undergo an accelerated diagnostic evaluation and further assessment to allow reclassification into low-risk or high-risk categories.

§ Patients with low-risk NSTEMI, after an appropriate period of observation and assessment, may be discharged on upgraded medical therapy for urgent outpatient cardiac follow-up.

Long-term management after control of myocardial ischaemia

§ Before discharge of patients who have had an ACS, therapy with an appropriate medication regimen should be initiated, including antiplatelet agent(s), β -blocker, angiotensin-converting enzyme inhibitor, statin and other therapies as appropriate.

§ Implantable cardiac defibrillators should be considered in some patients who, despite optimal medical therapy, have persistently depressed left ventricular function more than 6 weeks after STEMI.

§ Patients should be given advice on lifestyle changes that will reduce the risk of further coronary heart disease events, including smoking cessation, good nutrition, moderate alcohol intake, regular physical activity and weight management, as appropriate.

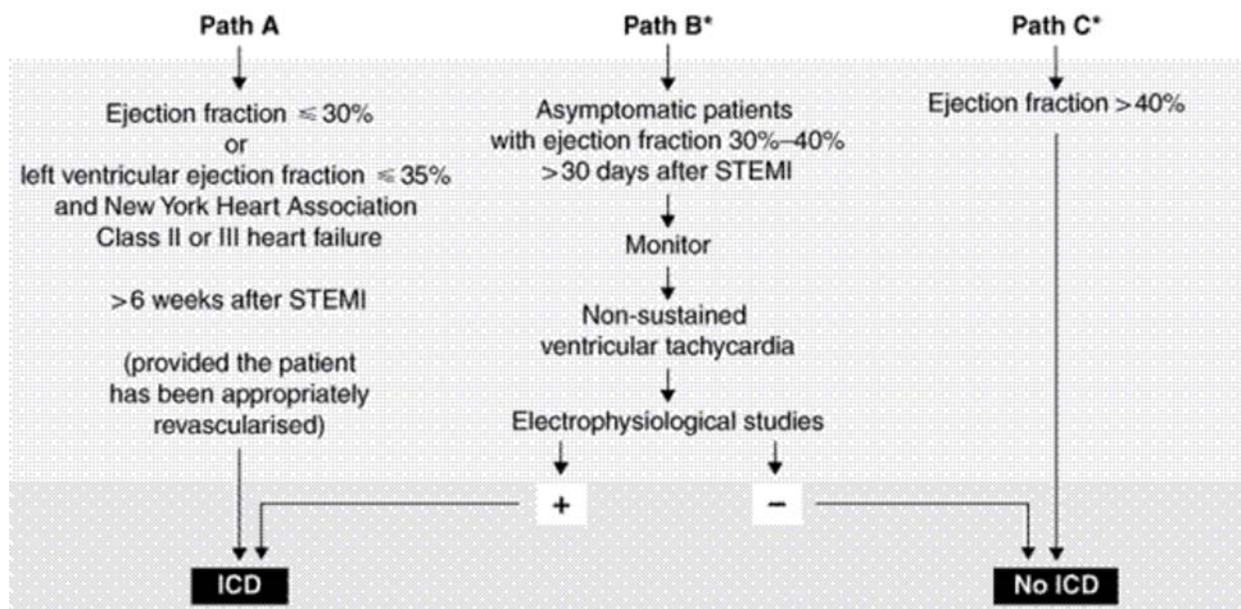
§ All patients should have access, and be actively referred, to comprehensive ongoing prevention and cardiac rehabilitation services.

§ All patients should be provided with a written action plan for chest pain.

§ Depression and coronary heart disease frequently coexist, and in patients with heart disease, depression, social isolation and lack of social support are more likely to lead to poorer outcomes. All patients with coronary heart disease should be assessed for depression and level of social support.

Appendix: Implantable cardiac defibrillator (ICD) implantation after ST-segment-

elevation myocardial infarction (STEMI): proposed management algorithm^{104,105}



This algorithm is for suggested management in an area which is still evolving. There may be considerable resource issues that will need to be explored, and cost-effectiveness data are currently lacking. Other factors such as comorbidities and conditions that significantly shorten life expectancy and reduce quality of life should be considered before ICD implantation. The evidence for benefit is strongest in patients with a left-ventricular ejection fraction $\leq 30\%$ and New York Heart Association Class II or III heart failure.

* Patients with sustained ventricular tachyarrhythmias or unexplained syncope after STEMI and an ejection fraction $> 35\%$ should also be considered for electrophysiological evaluation.

Overview of keypoints

Acute coronary syndrome (ACS) refers to a group of clinical conditions caused by myocardial ischemia including unstable angina pectoris (UA), non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI) (Fig. 1).

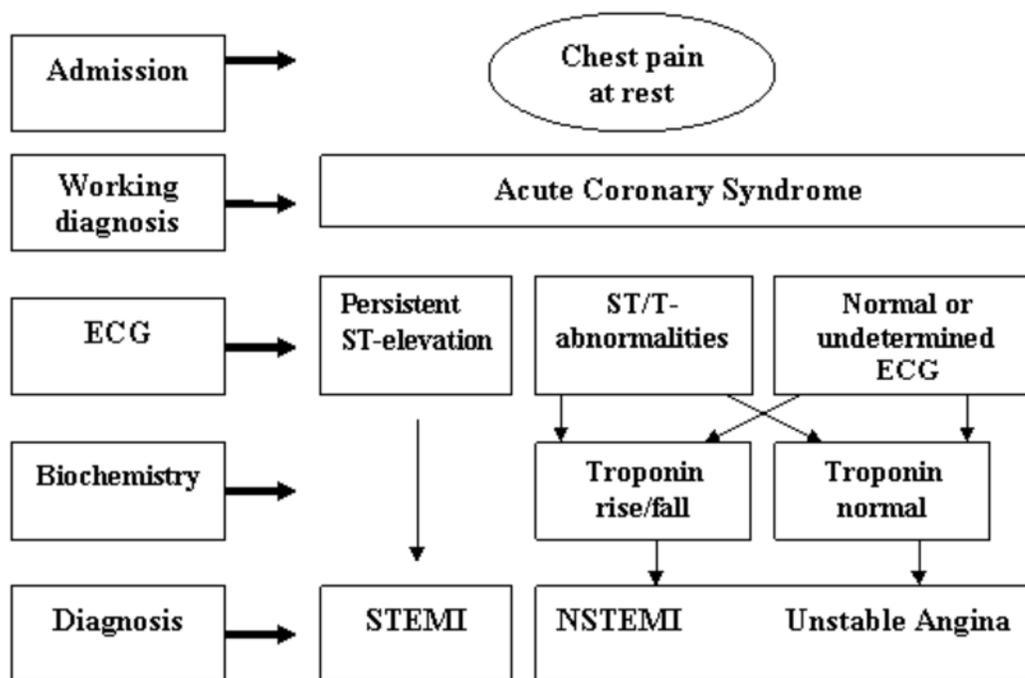


Figure 1. The spectrum of acute coronary syndrome.

ECG = electrocardiogram; NSTEMI = non-ST-elevation myocardial infarction; STEMI = ST-elevation myocardial infarction

From Hamm et al. *Eur Heart J* 2011;32:2999-3054.

Definition and terminology

Patients presenting with acute chest pain and persistent (>20 min.) ST-elevation on electrocardiogram (ECG) will have the working diagnosis of ST-elevation ACS. Most of these patients will ultimately develop STEMI and progress to Q wave myocardial infarction (MI). Patients with acute chest pain, but without persistent ST-segment elevation (presenting with persistent or transient ST depression or T-wave inversion, flat T waves, pseudo-normalization of T waves or normal ECG) will have the working diagnosis of non-ST-elevation ACS. On the basis of troponin measurements patients with non-ST-elevation ACS will be further qualified as having NSTEMI or UA.

The distinction between non-ST segment elevation ACS and ST-segment elevation ACS is clinically important because rapid recanalization therapy is critical for improving the outcome in ST elevation ACS/STEMI, but is less urgent in non-ST-segment elevation ACS.

MI is defined pathologically as myocardial cell death due to prolonged ischemia. In the clinical setting, these conditions are met when the following criteria are present: detection of a rise and/or fall of cardiac biomarkers with at least one value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischemia as recognized by at least one of the following:

- symptoms of ischemia;
- ECG changes of new ischemia (ST-elevation or depression) or development of pathologic Q waves; or
- imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Pathophysiology

Acute coronary syndrome represents a life-threatening manifestation of atherosclerosis. ACS most frequently develops as a result of plaque rupture or erosion with overlying thrombosis. A thin fibrous cap is more likely to rupture than a thick one. Factors such as the lipid and tissue factor content of the plaque, the severity of the plaque rupture, the degree of inflammation at the site, the blood flow in the area, and the patient's antithrombotic and prothrombotic balance are important in determining whether a given plaque rupture will result in ACS.

Less frequent pathophysiological causes of ACS include dynamic obstruction due to coronary vasospasm as in Prinzmetal angina or in cocaine use, coronary dissection, secondary UA in patients with fever, thyrotoxicosis, severe anemia, hypoxemia or hypotension.

Clinical presentation

The typical clinical presentation of ACS is prolonged (>20 min.) retrosternal pressure or heaviness radiating to the left arm, neck or jaw. These complaints may be accompanied by other symptoms such as diaphoresis, nausea, abdominal pain and syncope (Table 1).

Table 1. Likelihood that signs and symptoms represent an ACS

Feature	Likelihood		
	High	Intermediate	Low
	Any of the following:	Absence of high-likelihood features and presence of any of the following:	Absence of high- or intermediate-likelihood features and presence of any of the following:
History	- Chest or left arm pain reproducing previously documented angina; - known history of CAD, including MI	- Chest or left arm pain or discomfort; - age > 70 years; - male gender; - diabetes mellitus	- Probable ischemic symptoms in absence of any of the intermediate-likelihood characteristics; - recent cocaine use
Examination	Transient MR murmur, hypotension, diaphoresis, pulmonary edema, râles	Extracardiac vascular disease	Chest discomfort reproduced by palpation
ECG	New, or presumably new ST-segment elevation ($\geq 0,1$ mV) or T wave inversion ($\geq 0,2$ mV) in multiple precordial leads	- Fixed Q-waves; - ST-segment depression 0,05-0,1 mV or T wave inversion $> 0,1$ mV	- T wave flattening or inversion $< 0,1$ mV in leads with dominant R waves; - normal ECG
Cardiac markers	Elevated cardiac TnI, TnT, or CK-MB	Normal	Normal

CAD = coronary artery disease; ECG = electrocardiogram; MI = myocardial infarction; MR = mitral regurgitation; Tn = troponin; CK-MB = creatine kinase MB isoenzyme
 From Sabatine M.S., Cannon C.P. Approach to patient with chest pain. In: Bonow R.O., Mann D.L., Zipes D.P., Libby P., eds. *Braunwald's Heart Disease: a textbook of cardiovascular medicine*. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2011:chap. 53.

Atypical presentations include epigastric pain, neck, jaw, ear, arm discomfort. Some patients, especially those who are elderly or who have diabetes, present with no pain, complaining only of episodic shortness of breath, severe weakness, light-headedness, diaphoresis, or nausea and vomiting. Elderly persons may also present only with altered mental status (Table 2).

Table 2. Killip Classification in acute MI

Class	Definition	Mortality (%)
I	No congestive heart failure signs	6
II	+S ₃ and/or basilar rales	17
III	Pulmonary edema	30-40
IV	Cardiogenic shock	60-80
From Killip T. 3rd, Kimball J.T. <i>Am J Cardiol</i> 1967;20:457		

The physical examination is frequently normal. Worrisome findings include hypotension, new jugular venous distension, pulmonary edema, or a new systolic murmur. An important goal of the physical examination is to exclude non-cardiac causes of chest pain (e.g., pulmonary embolism, aortic dissection, pericarditis, valvular heart disease, pneumothorax, pneumonia, or pleural effusion). The Killip classification system (Table 2) and TIMI risk score (Table 3) are helpful in using physical exam findings to assess a patient's 30-day mortality.

Table 3. TIMI risk score for ST-elevation MI

Risk factor (weight)	Risk score/30-day mortality (%)
Age 65-74 years (2 points)	0 (0.8)
Age >75 years (3 points)	1 (1.6)
Diabetes mellitus/hypertension or angina (1 point)	2 (2.2)
Systolic BP <100 mmHg (3 points)	3 (4.4)
Heart rate >100 bpm (2 points)	4 (7.3)
Killip class II-IV (2 points)	5 (12.4)
Weight <67 kg (1 point)	6 (16.1)
Anterior ST-elevation or left bundle branch block	7 (23.4)
Time to Rx >4 hours (1 point)	8 (26.8)
Risk score = total points (0-14)	>8 (35.9)
From Morrow D.A., Antman E.M., Charlesworth A, et al. <i>Circulation</i> 2000;102:2031-2037	

Diagnosis

The diagnosis is based on a combination of history, ECG, and biochemical markers of cardiac injury.

ECG. ST segment elevation of ≥ 2 mm in two adjacent chest leads and of ≥ 1 mm in adjacent limb leads suggests of STEMI. Other associated findings may include tall peaked T waves and reciprocal ST-segment depression. Pathological Q waves indicating significant transmural myocardial damage should be $\geq 25\%$ of the R wave, ≥ 0.04 sec in duration. The infarction site can be localized from ECG changes (Table 4).

Patients with UA/NSTEMI may have a normal ECG or may have changes ranging from new T wave inversions, ST segment depression, or normalization of previously inverted T waves (pseudonormalization).

Table 4. Localization of infarcts from ECG changes

Infarct localization	ECG signs	Coronary artery
Anterior	ST elevation and/or Q waves in V ₁ -V ₄ /V ₅	LAD
Antero-septal	ST elevation and/or Q waves in V ₁ -V ₃	LAD
Antero-lateral	ST elevation and/or Q waves in V ₁ -V ₆ and in I and aVL	Proximal LAD
Lateral	ST elevation and/or Q waves in V ₅ -V ₆ and T wave inversion/ST elevation/Q waves in I and aVL	Diagonal branch of LAD or proximal LCx
Inferolateral	ST elevation and/or Q waves in II, III, aVF, and V ₅ -V ₆ (sometimes in I and aVL)	RCA or LCx, if left dominant
Inferior	ST elevation and/or Q waves in II, III, aVF	RCA or LCx, if left dominant
True posterior	Tall R waves in V ₁ -V ₂ with ST depression in V ₁ -V ₃ *. Usually occurs in conjunction with inferior or lateral infarct	RCA or obtuse marginal branch of LCx
RV infarction	ST elevation in the right precordial leads V _{3R} -V _{4R} . Usually found in conjunction with inferior or lateral infarction.	RCA
LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery; RV = right ventricular		
*The "mirror-test" is useful here to demonstrate that ST depressions are actually ST elevations of the posterior wall, and tall R waves are actually posterior Q waves.		

Biochemical markers of cardiac injury

Creatine kinase (CK). Levels twice the upper limit of normal are taken as being abnormal. Serum levels rise within 4-8 hours post-STEMI and fall to normal within 3-4 days. CK-MB isoenzyme is more specific for myocardial disease.

Cardiac troponins (TnT, TnI). Serum levels start to rise by 3 hours post-MI, and elevation may persist up to 7-14 days. Troponins can also be elevated in non-ischaemic myocardial

damage such as myocarditis, cardiomyopathy, pericarditis.

Treatment

Stabilizing measures are similar in all patients with ACS. Patients should be placed in an environment with continuous ECG monitoring and defibrillation capacity. Patients should receive aspirin 300 mg orally, analgesia, secure i.v. access and oxygen. Intramuscular injections and not be given as they cause rise in total CK and risk of bleeding with thrombolysis/anticoagulation.

STEMI

Patients presenting with ST-segment elevation or left bundle branch block on ECG benefit significantly from immediate reperfusion and are treated as one group under the term STEMI.

1. Relief of pain, breathlessness and anxiety.

Titrated **i.v. opioids** (e.g. morphine) are the analgesics most commonly used in this context. Repeated doses may be necessary. Side-effects include nausea and vomiting, hypotension with bradycardia, and respiratory depression. Anti-emetics (e.g., metoclopramide 10 mg i.v.) may be administered concurrently with opioids to minimize nausea.

Nitrates (sublingual or intravenous) may lessen pain and can be given, providing the patient is not hypotensive.

Oxygen (at 2-5 L/min for at least 2-3 hours by mask or nasal prongs) should be administered to those who are breathless, hypoxic ($\text{SaO}_2 < 95\%$), or who have heart failure. Non-invasive monitoring of blood oxygen saturation greatly helps when deciding on the need to administer oxygen or ventilatory support.

Anxiety is a natural response to the pain and the circumstances surrounding a heart attack. Reassurance of patients and those closely associated with them is of great importance. If the patient becomes excessively disturbed, it may be appropriate to administer a tranquillizer, but opioids are frequently all that is required.

2. Correction of electrolytes. Potassium and magnesium should be supplemented as both low potassium and magnesium may be arrhythmogenic.

3. Strategies to limit infarct size (beta-blockade, ACE-inhibitors, reperfusion).

Beta-blockade. Metoprolol i.v. 1-2 mg at a time repeated at 1-2 min. intervals to a maximum dose of 15-20 mg under continuous ECG and BP monitoring. Aim at a pulse rate of 60 bpm and systolic BP 100-110 mmHg.

ACE-inhibitors. After receiving aspirin, beta-blockade (if appropriate), and reperfusion, patients with STEMI should receive an ACE-inhibitor within the first 24 hours of presentation.

Reperfusion.

Primary percutaneous coronary intervention (PCI) is the current gold standard reperfusion strategy for the treatment of STEMI and demonstrates superior outcome in comparison with thrombolysis. In settings where primary PCI cannot be performed within 120 min of first medical contact by an experienced team, thrombolysis should be considered, particularly if it

can be given pre-hospital (e.g. in the ambulance) and within the first 120 min of symptom onset. As an adjunct to thrombolysis, rescue PCI should be reserved for patients who remain symptomatic post thrombolysis or develop cardiogenic shock.

Thrombolysis (fibrinolysis). Fibrinolytic therapy (Table 5) is recommended within 12 h of symptom onset if primary PCI cannot be performed within 90 min of being able to administer fibrinolysis and within 120 min from first medical contact and there are no contraindications (Table 6). The later the patient presents (particularly after 6 h), the more consideration should be given to transfer for primary PCI (in preference to fibrinolytic therapy) as the efficacy and clinical benefit of fibrinolysis decrease over time, which, in later presentations, has the effect of increasing the acceptable time delay before transfer for primary PCI.

Table 5. Dose and administration of thrombolytic agents

Streptokinase (SK)	Give as 1.5 million units in 100 mL normal saline over 1 hour.
Recombinant tissue-type plasminogen activator (rt-PA, alteplase)	Give 15 mg bolus i.v., then 0.75 mg/kg over 30 min. (not to exceed 50 mg), then 0.5 mg/kg over 60 min (not to exceed 35 mg). This should be followed by i.v. heparin
Reteplase (r-PA)	Give two i.v. bolus of 10 units 30 minutes apart
Tenecteplase (TNK-tPA)	Give as injection over 10 seconds at 30-50 mg according to body weight (500-600 mcg/kg). Maximum dose is 50 mg
APSAC (anistreplase)	Give as i.v. bolus of 30 mg over 2-5 minutes

Streptokinase is the first-generation fibrinolytic agent which is an indirect plasminogen activator and therefore relatively nonspecific for fibrin. In comparative trials, it had the lowest rate of intracranial hemorrhage. streptokinase is antigenic and allergic reactions occur on 5 to 6 % of patients.

Alteplase represents a second-generation fibrinolytic. Of all current fibrinolytics, it has the shortest half-life (4 to 8 minutes) and therefore is administered as a bolus followed by an infusion over 90 minutes or longer.

Reteplase and tenecteplase are the third-generation fibrinolytics. They are the mutants of alteplase with a longer half-life and offer the convenience of bolus administration.

Table 6. Contraindications for use of thrombolytic agents in acute MI

<p>Absolute contraindications</p> <ul style="list-style-type: none"> - previous hemorrhagic stroke at any time; any other cerebrovascular events within 1 year; - known intracranial neoplasm; - active internal bleeding (not including menses); - suspected aortic dissection
<p>Relative contraindications</p> <ul style="list-style-type: none"> - severe, uncontrolled hypertension (BP>180/110 mmHg); - history of cerebrovascular accident or known intracerebral pathologic condition not covered in contraindications; - current use of anticoagulants in therapeutic doses (INR>2); known bleeding diathesis;

- recent trauma (within 2-4 weeks), prolonged CPR (>10 min.), or major surgery (within 3 weeks);
- noncompressible vascular punctures;
- recent internal bleeding (within 2-4 weeks);
- pregnancy;
- active peptic ulcer disease;
- for streptokinase or anistreplase: prior exposure to either agent or prior allergic reaction

4. Additional therapies

Heparin. Unfractionated heparin (UFH) should be used routinely with rt-PA and its derivatives for 24-48 hours for a target aPTT (activated partial thromboplastin time) 50-70 sec or 1.5-2.0 times control (to be monitored at 3, 6, 12 and 24 h). Low molecular weight heparin (LMWH) can be used as an alternative to UFH.

Clopidogrel (loading dose of 600 mg orally followed by a maintenance dose of 75 mg/day) should be administered to all patients undergoing primary PCI. If coronary stents are used, patients should remain on clopidogrel for at least 1 month in bare-metal stents and 12 months in coated stents.

Glycoprotein IIb/IIIa inhibitors (abciximab, eptifibatide, tirofiban) are recommended routinely in the context of STEMI treated with primary PCI.

NSTEMI/UA

Timing of angiography and revascularization should be based on patient risk profile. The GRACE risk score provides the most accurate stratification of risk both on admission and at discharge due to its good discriminative power. However, the complexity of the estimation requires the use of computer or personal digital assistant software for risk calculations, which can also be performed online:

Cardiac catheterization followed by revascularization has been shown to prevent recurrent ischaemia and/or improve short- and long-term outcomes. Several risk factors (troponin elevation, diabetes, ST-segment depression, renal insufficiency, etc.) have been identified to predict the long-term benefit of an invasive strategy. Depending on the acuteness of risk, the timing of angiography can be tailored, according to four categories:

- **invasive** (<72 h);
- **urgent invasive** (<120 min);
- **early invasive** (<24 h);
- primarily **conservative**.

Urgent invasive strategy (<120 min after first medical contact)

This should be undertaken for **very high risk** patients. These patients are characterized by:

- refractory angina (indicating evolving MI without ST abnormalities);

- recurrent angina despite intense antianginal treatment, associated with ST depression (2 mm) or deep negative T waves;
- clinical symptoms of heart failure or haemodynamic instability ('shock');
- life-threatening arrhythmias (ventricular fibrillation or ventricular tachycardia).

Early invasive strategy (<24 h after first medical contact)

Most patients initially respond to the antianginal treatment, but are at increased risk and need angiography followed by revascularization. High risk patients as identified by a GRACE risk score >140 and/or the presence of at least one primary high risk criterion should undergo invasive evaluation within 24 h. High risk criteria include:

- relevant rise or fall in troponin;
- dynamic ST- or T-wave changes (symptomatic or silent);
- diabetes mellitus;
- renal insufficiency (eGFR < 60 mL/min/1.73 m²);
- early postinfarction angina;
- recent PCI;
- prior CABG;
- intermediate to high GRACE risk score

Invasive strategy (<72 h after first medical contact)

In lower risk subsets with a GRACE risk score of <140 but with at least one high risk criterion, the invasive evaluation can be delayed without increased risk but should be performed during the same hospital stay, preferably within 72 h of admission. Thus, such patients should undergo elective invasive evaluation at the first opportunity depending on the local circumstances.

Conservative strategy (no or elective angiography)

Patients that fulfill all of the following criteria may be regarded as low risk and should not routinely be submitted to early invasive evaluation:

- No recurrence of chest pain.
- No signs of heart failure.
- No abnormalities in the initial ECG or a second ECG (at 6–9 h).
- No rise in troponin level (at arrival and at 6–9 h).
- No inducible ischaemia.

In other low risk patients without recurrent symptoms a non-invasive assessment of inducible ischaemia should be performed before hospital discharge. Coronary angiography should be performed if the results are positive for reversible ischaemia.

Medical management

All patients should be treated with adequate analgesia, i.v. nitrates, beta-blockers and statins (if not contraindicated). Other agents can also be added depending on the clinical picture.

1. Anti-ischaemic agents

Anti-ischaemic drugs either decrease myocardial oxygen demand (by decreasing heart rate, lowering blood pressure, reducing preload, or reducing myocardial contractility) or increase myocardial oxygen supply (by inducing coronary vasodilatation).

Beta-blockers competitively inhibit the myocardial effects of circulating catecholamines and reduce myocardial oxygen consumption by lowering heart rate, blood pressure, and contractility. Beta-blockers should be started on presentation, initially using a short-acting agent (e.g., metoprolol 12.5-50 mg t.i.d) which, if tolerated, may be converted to a longer-acting agent. Aim for HR of \approx 50-60 bpm.

Nitrates

E.g., glyceryl trinitrate (GTN) infusion (50 mg in 50 mL 1 M saline at 1-10 mL/h). Tolerance to continuous infusion develops within 24 hours, therefore the lowest efficacious dose should be used.

The use of nitrates in unstable angina is largely based on pathophysiological considerations and clinical experience. The therapeutic benefits of nitrates and similar drug classes such as sydnonimines are related to their effects on the peripheral and coronary circulation. The major therapeutic benefit is probably related to the venodilator effects that lead to a decrease in myocardial preload and LV end-diastolic volume, resulting in a decrease in myocardial oxygen consumption. In addition, nitrates dilate normal as well as atherosclerotic coronary arteries and increase coronary collateral flow.

Nitrates should not be given to patients on phosphodiesterase-5 inhibitors (sildenafil, vardenafil, or tadalafil) because of the risk of profound vasodilatation and critical blood pressure drop.

Calcium channel blockers

Calcium channel blockers are vasodilating drugs. In addition, some have direct effects on atrioventricular conduction and heart rate. There are three subclasses of calcium blockers, which are chemically distinct and have different pharmacological effects: dihydropyridines (such as nifedipine), benzothiazepines (such as diltiazem), and phenylethylamines (such as verapamil). The agents in each subclass vary in the degree to which they cause vasodilatation, decrease myocardial contractility, and delay atrioventricular conduction. Atrioventricular block may be induced by non-dihydropyridines. Nifedipine and amlodipine produce the most marked

peripheral arterial vasodilatation, whereas diltiazem has the least vasodilatory effect. All subclasses cause similar coronary vasodilatation. Therefore, calcium channel blockers are the preferred drugs in vasospastic angina. Diltiazem and verapamil show similar efficacy in relieving symptoms and appear equivalent to beta-blockers.

E.g., diltiazem 60-360 mg orally, verapamil 40-120 mg orally t.i.d. Amlodipine/felodipine 5-10 mg PO can also be used.

Other antianginal drugs

Nicorandil, a potassium channel opener, reduced the rate of the primary composite endpoint in patients with stable angina, but was never tested in ACS patients. Ivabradine selectively inhibits the primary pacemaker current in the sinus node and may be used in selected patients with β -blocker contraindications.

Ranolazine exerts antianginal effects by inhibiting the late sodium current. It was not effective in reducing major cardiovascular events in a clinical trial, but reduced the rate of recurrent ischaemia.

Statins (e.g., atorvastatin 80 mg qd) have been shown to reduce mortality and recurrent MI in the acute setting.

2. Antiplatelet agents

Platelet activation and subsequent aggregation play a dominant role in the propagation of arterial thrombosis and consequently are the key therapeutic targets in the management of ACS. Antiplatelet therapy should be instituted as early as possible when the diagnosis of NSTEMI-ACS is made in order to reduce the risk of both acute ischaemic complications and recurrent atherothrombotic events. Platelets can be inhibited by three classes of drugs, each of which has a distinct mechanism of action.

Aspirin (acetylsalicylic acid) targets cyclo-oxygenase (COX-1), inhibiting thromboxane A_2 formation and inducing a functional permanent inhibition in platelets. However, additional complementary platelet aggregation pathways must be inhibited to ensure effective treatment and prevention of coronary thrombosis. ADP binding to the platelet P2Y₁₂ receptor plays an important role in platelet activation and aggregation, amplifying the initial platelet response to vascular damage. The antagonists of the P2Y₁₂ receptor are major therapeutic tools in ACS.

The prodrug thienopyridines such as clopidogrel and prasugrel are actively biotransformed into molecules that bind irreversibly to the P2Y₁₂ receptor. A new class of drug is the pyrimidine derivative ticagrelor, which without biotransformation binds reversibly to the P2Y₁₂ receptor, antagonizing ADP signalling and platelet activation. I.v. GP IIb/IIIa receptor antagonists (abciximab, eptifibatid, and tirofiban) target the final common pathway of platelet aggregation.

Aspirin

A daily maintenance dose of 75–100 mg should be continued indefinitely. It has the same efficacy as higher doses and carries a lower risk of gastrointestinal intolerance.

P2Y₁₂ receptor inhibitors include thienopyridines (clopidogrel, prasugrel) and triazolopyrimidine (ticagrelor). Of note, ticlopidine was the first thienopyridine investigated in ACS, but was replaced by clopidogrel because of side effects. Today ticlopidine may still be used in patients who are allergic to clopidogrel, although cross-reactions are possible. A loading dose of **clopidogrel** 300-600 mg followed by 75 mg daily maintenance for 9–12 months in addition to aspirin should be given to all patients with NSTEMI-ACS. The 600 mg loading dose of clopidogrel has a more rapid onset of action and more potent inhibitory effect than the 300 mg dose. Clopidogrel should be withheld for 5-7 days in patients requiring CABG to reduce hemorrhagic complications.

Alternative regimens:

prasugrel loading dose of 60 mg followed by a maintenance dose 10 mg daily

ticagrelor loading dose of 180 mg followed by a maintenance dose 90 mg b.i.d.

Glycoprotein IIb/IIIa receptor inhibitors

The three GP IIb/IIIa receptor inhibitors approved for clinical use are i.v. agents belonging to different classes: abciximab is a monoclonal antibody fragment; eptifibatid is a cyclic peptide; and tirofiban is a peptidomimetic molecule.

these agents should be used in conjunction with aspirin, clopidogrel and LMWH or UFH.

Eptifibatid or tirofiban should be used in high-risk patients with ongoing ischemia and elevated troponins in whom invasive strategy is not planned/available (<24 hours). In patients with an early invasive strategy, all GP IIb/IIIa receptor inhibitors can be used. Infusion is generally continued for 12 hours post-PCI.

Administration regimens:

Abciximab (ReoPro) - bolus of 0.25 mg/kg i.v. over 1 minute followed by i.v. infusion 0.125 mcg/kg/min (maximum 10 mcg/min) for 12 hours;

Eptifibatid (Integrilin) - bolus 180 mcg/kg followed by i.v. infusion 2 mcg/kg/min;

Tirofiban (Aggrastat) - 400 ng/kg/min for 30 minutes followed by i.v. infusion 100 ng/kg/min.

3. Anticoagulants

Several anticoagulants, which act at different levels of the coagulation cascade, have been investigated or are under investigation in NSTEMI-ACS:

Indirect inhibitors of coagulation (need antithrombin for their full action)

Indirect thrombin inhibitors: UFH

LMWHs

Indirect factor Xa inhibitors: LMWHs

fondaparinux

Direct inhibitors of coagulation

Direct factor Xa inhibitors: apixaban, rivaroxaban, otamixaban

Direct thrombin inhibitors (DTIs): bivalirudin, dabigatran

All patients should be given a LMWH or fondaparinux or UFH.

LMWH should be used in conjunction with aspirin and clopidogrel and continued for 2-5 days after the last episode of pain and ischemic ECG changes.

E.g., enoxaparin 1 mg/kg bid (100 U/kg twice daily).

Fondaparinux is the only selective activated factor X (factor Xa) inhibitor available for clinical use. Fondaparinux is a synthetic pentasaccharide structurally similar to the antithrombin-binding sequence common to all forms of heparin. In ACS, a 2.5 mg fixed daily dose of fondaparinux is recommended.

UFH should be used as an alternative to LMWH and fondaparinux in conjunction with aspirin and clopidogrel. A weight-adjusted dose of UFH is recommended, at an initial bolus of 60–70 IU/kg with a maximum of 5000 IU, followed by an initial infusion of 12–15 IU/kg/h, to a maximum of 1000 IU/h. The therapeutic window is narrow, requiring frequent monitoring of aPTT, with an optimal target level of 50–75 s, corresponding to 1.5–2.5 times the upper limit of normal. At higher aPTT values, the risk of bleeding complications is increased, without further antithrombotic benefits.

In the PCI setting, UFH is given as an i.v. bolus either under activated clotting time (ACT) guidance (ACT in the range of 250–350 s, or 200–250 s if a GP IIb/IIIa receptor inhibitor is given) or in a weight-adjusted manner (usually 70–100 IU/kg, or 50–60 IU/kg in combination with a GP IIb/IIIa receptor inhibitors).

Bivalirudin plus a provisional GP IIb/IIIa receptor inhibitor showed similar efficacy to heparin/LMWHs plus systematic GP IIb/IIIa receptor inhibitors, while significantly lowering the risk of major haemorrhagic complications. However, no significant difference in short- or long-term outcomes was observed between these two anticoagulation strategies. Bivalirudin is currently approved for urgent and elective PCI at a dose of 0.75 mg/kg bolus followed by 1.75 mg/kg/h. In NSTEMI-ACS patients, bivalirudin is recommended at a dose of 0.1 mg/kg i.v. bolus followed by an infusion of 0.25 mg/kg/h until PCI. Bivalirudin plus provisional GP IIb/IIIa receptor inhibitors are recommended as an alternative to UFH plus GP IIb/IIIa receptor inhibitors in patients with an intended urgent or early invasive strategy, particularly in patients with a high risk of bleeding.

At discharge from hospital, the following measures are necessary:

1. **Aspirin:** continue life long;
2. **P2Y₁₂ inhibitor:** continue for 12 months (unless at high risk of bleeding);
3. **Beta-Blocker:** if LV function is depressed;
4. **ACE inhibitor/ARB:** if LV function is depressed. Consider for patients devoid of depressed LV function;
5. **Aldosterone antagonist/ eplerenone:** if depressed LV function (LVEF \leq 35%) and either diabetes or heart failure, without significant renal dysfunction;

6. **Statin:** titrate to achieve target LDL-C levels <1.8 mmol/L (<70 mg/dL)
7. **Lifestyle:** risk-factor counselling, referral to cardiac rehabilitation / secondary prevention programme.

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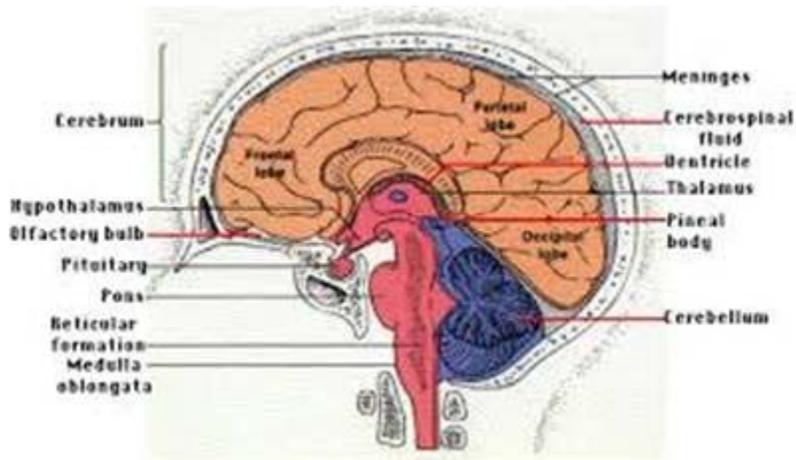
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Comatous states

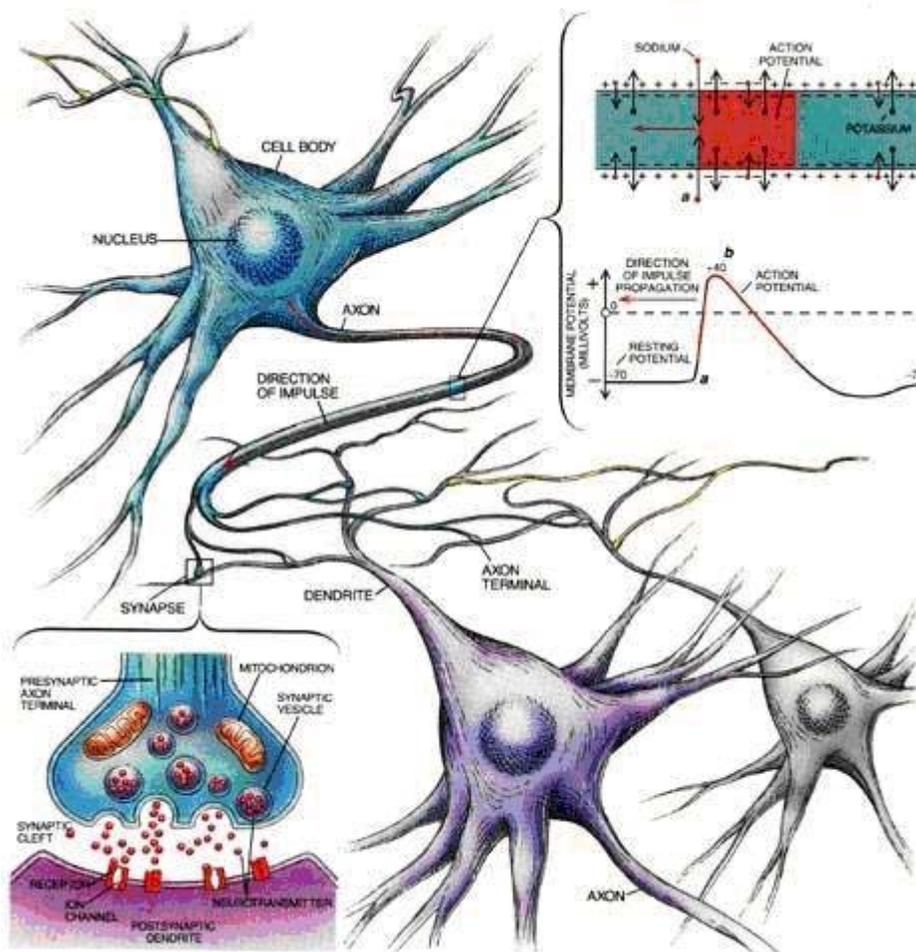


1 Anatomy and physiology of central nervous system.

Nervous system consists of two parts: central nervous system and peripheral nervous system.

The brain (central nervous system, higher vegetative centres and endocrine regulation centres) is located in the skull - reliable bony container, which protects it from the most dangerous environment factors.

As a central regulator of all body functions brain requires a very high level of metabolic processes. Its weight is just 2 % of the body weight (nearly 1500 grams), however it takes nearly 14-15% of circulating blood volume (700-800 ml) every minute. Brain consumes 20% of total oxygen, which gets to the organism every minute. However its main limitation factor is glucose, because it is the only source of energy for the brain (75 milligrams per minute or 100 grams per day).



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Thus, physiologically functions of the central nervous system depend on an adequate perfusion (oxygen and glucose supply) and adequate blood outflow (wastes transportation) from the cranial cavity.

Serious self-regulation system provides uninterrupted brain functioning and even during massive blood loss brain gets its amount of oxygen through the mechanism of blood flow centralization (of course this causes ischemic injury of visceral organs, but their affection evolution quite reasonably considered less harmful). There is also a mechanism which protects brain in case of hypoglycaemia: blood flow increases and thus increases amount of glucose getting to the nervous cells. On the other hand hyperventilation (hypocapnia) decreases blood flow and hypoventilation (hypercapnia) increases it to eliminate acid metabolites.

When brain tissue is much damaged and regulation fails, brain is not able to change its volume according to the needs. So when blood flow and outflow get into the imbalance the protective cranial “bone box” (the skull) actually becomes brain’s main enemy, because it can’t be extended. For example 5% increasing of blood volume (haematoma, tumour, overhydration, liquor hypertension, etc.) causes unconsciousness. Excessive blood flow brings liquor hyperproduction and this causes brain compression and results in brain tissue

edema and functional disorders.

Brain injuries, vascular disorders, cerebral oedema, increase of intracranial pressure, liquor circulation violations result in cellular hypoxia of central nervous system. Alerted level of consciousness is the first sign of cerebral hypoxia.



5.2 Classification, clinic and diagnostics of coma.

Coma is a complete depression of consciousness with lack of pain sensitivity and reflexes, general muscular relaxation and violated vital functions.

Classification of consciousness levels according to Bogolepow, 1982.

Conscious (normal)

Confused

Stuporous

Soporose

Comatose: moderate

deep

terminal

Each level has its own diagnostic criteria; you can find them in the table below.

Levels of consciousness	Main symptoms	General symptoms
Conscious	Alert, absolutely oriented to self, place and time patients.	Attention is active, speech is fluent, answers are correct and full, commands are obeyed, eyes are opened spontaneously.
Confused	Moderate somnolence or euphoria, partial disorientation with the place and time, but complete self orientation.	Thinking and attention are impaired. Verbal contact is slowed; questions should be repeated to receive answers. Commands are obeyed, however slowly, especially complex commands.
Stuporous	Deep somnolence, time and place disorientation, only simple commands are obeyed after awakening.	Most of the time patient sleeps, sometimes however there are periods of restlessness and agitation. Answers are curt. Defensive reaction to pain stimuli is maintained. Pelvic organs control is deficient.
Soporose	Pathologic somnolence; time, space and self disorientation.	Opens eyes in response to painful stimuli, localizes painful stimuli; reflexes of cerebral nerves and vital functions are preserved.
Moderate comatose	Unconscious	No reaction to external stimuli. To painful stimuli answers with withdrawal. Corneal and pupillar reflexes are overactive, abdominal

		reflexes are reduced. There are pathologic plantar reflexes. Pelvic organs control is violated. Vital functions are preserved.
Deep comatose	Unconscious	Extension to painful stimuli. Skin, tendon, corneal and pupillar reflexes are reduced or absent. Skeletal muscles are rigid or hypotonic. There are respiratory and cardiac disorders.
Deepest comatose	Unconscious	Areflexia, bilateral fixed midriasis, muscle atony; great violations of vital functions. Blood pressure less than 60 mmHg.

Classification of coma according to etiology and pathogenesis.

1. Coma of central genesis (epileptic, traumatic, apoplectic).
2. Coma caused by visceral organs dysfunctions and endocrine glands disorders (diabetic, hypoglycaemic, thyrotoxic, myxedemic, hypopituitary, hypocorticoid, hepatic, uremic, hypochloremic, anaemic, alimentary-distrophic).
3. Infectious coma (in case of pneumonia, malaria, neuronal infections, etc.).
4. Acute toxic coma (poisonings with alcohol and its surrogates, medicinal poisonings, carbon monoxide poisonings).
5. Coma caused by physical factors (caused by electric injury, hypothermic and hyperthermia coma, radiation coma, etc.).

Sometimes it is very hard to find the reason of the coma, so detailed anamnesis

(interview relatives and witnesses) and clinical observations are very important.

First of all ask about the duration of unconsciousness and was it lost suddenly or there were other levels of altered consciousness. Ask about the events prior to incident: did the patient fell and was there a chance of head injury? Did he have fever, flu or jaundice? Are there any sings of chronic diseases like diabetes, epilepsy, hypertonic disease? Have the patient ever suffered from similar unconsciousness episodes? Were there any suicide attempts? If the comatose condition was not sudden, have the patient complained of vomiting, convulsions? Pay attention to the personal things of the patient: you can find medical documents, medicine packages, diabetic bracelets or necklaces, etc.

If the anamnesis failed to reveal the coma etiology, concentrate on objective symptoms.

Skin colour: extreme paleness can be a sign of great blood loss, circulatory collapse, blood diseases. Cyanosis is a symptom of hypercapnic coma caused by respiratory failure or asphyxia (hanging, drowning, convulsions). Hyperaemic face allows you to think about atropine and carbon monoxide poisonings, hyperglycaemic coma or infectious disease.

Head position: tilted back head is a sign of meningitis, tetanus, hysteria; head turned aside can be the symptom of stroke.

Pathological types of breathing are also helpful in coma diagnostics: Cheyne-Stokes breathing (periods of apnoea, which are followed with chaotic frequent breathing) and Biot's breathing (periods of apnoea which are followed with breathing of equal amplitude) show deep affection of central nervous system. Kussmaul breathing (deep and laboured) proves accumulation of acid metabolites (metabolic acidosis) of exogenous (in case of poisonings) or endogenous (diabetic ketoacidosis) nature. Fever and rapid deep breathing are probable signs of infectious coma. Remember that per each excess body temperature degree there are 5-7 excess breathes and 10 excess heart beats.

Generally when you are coming to the patient with consciousness disorders try to do this from the side of patient's head (nape): this way you can easily improve airways patency if necessary (thrust the jaw forward for example) and avoid accidental injuries from the patient if his moves are uncontrolled.

You can always identify simulation or hysteric coma by opening the eyes of the patient: in case of simulating conscious patients or hysteric patients you will feel the resistance of the eyelids. Unconscious patients never resist when you try to open their eyes.

Pressing the eyeballs toy can evaluate their tone: "soft" eyeballs with decreased tone are the symptom of hypovolemia (bleeding, dehydration) or shock condition. However they can also be part of hyperglycaemic coma clinic.

The depth of coma is assessed with the reflexes reduction. If patient reacts to the

external stimuli - it's a moderate coma. If corneal reflexes are preserved with depressed other reflexes – it's a deep coma. Lack of photoreaction is a symptom of terminal coma.

Pupil's diameter varies: pupils are narrowed in case of sleeping pills overdose or organophosphate poisonings; terminally narrow pupils you can see in patients after drugs admission; pupils are dilated in case of hypoxia, neuroleptic poisonings, antihistamine medicines intoxications; the widest pupils you can observe in atropine poisoning.

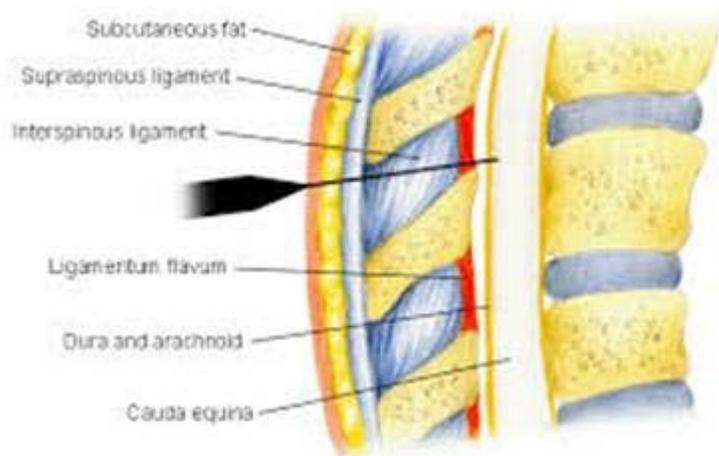
Anisocoria (pupils of different size) is a symptom of local CNS injuries. In most cases you notice this sign when a trauma causes intracranial haematoma. Examine those patients carefully: look for bruises and scratches on the head (including parts covered with hair). Sometimes you can even notice eyeballs deviation to the side of injury. Lack of patellar and heelstring reflexes proves deep CNS affection. Pathological Babinski's reflex shows organic CNS injury. Muscular tone asymmetry is a sign of stroke, tumour, haematoma, etc.

Don't forget to assess other systems and organs, because brain reaction can be only the response to a serious visceral pathology. For example unconsciousness can be a consequence of cardiac arrhythmia (Morgagni-Adams-Stokes syndrome). And on the contrary: CNS violations can cause rhythm disorders.

Localization of the brain pathology	Cardiac symptoms
Damage of frontal lobes and orbital zones	Bradycardia, complete AV block, auricular extrasystole
Damage of midbrain	Extrasystole, atrioventricular block, VF
Medulla oblongata pathology	Node and ventricular extrasystole, fluttering arrhythmia
Hypothalamic zone damage	Extrasystole, paroxysmal tachycardia
Affection of vascular center	Severe vascular tone decrease, hypotension, bradycardia, cardiac arrest.

Pericardial murmur and pleural rub can indicate uraemic coma. Changes of liver size are features of hepatic coma. Enlarged spleen is a symptom of bone marrow or liver diseases.

In case of comatose patients use all your laboratory and instrumental possibilities: perform head X-ray, CT, liquor tests, encephalography, angiography, usual blood tests, etc.



Pic. 5.1 Lumbar puncture.

For assessment of central nervous system condition Glasgow Coma Scale was proposed in 1974.

Clinical signs	Points
Eyes opening	
Spontaneous	4
In response to voice	3
In response to pain	2
Does not open the eyes	1
Verbal response	
Converses normally	5
Confused, disoriented answers	4
Inappropriate words	3
Incomprehensible sounds	2
No sounds	1
Motor response	
Obeys commands	6

Localizes painful stimuli	5
Withdrawal to painful stimuli	4
Abnormal flexion to painful stimuli	3
Extension to painful stimuli	2
No movements	1

Count the points and assess the result according to this:

13-14 points – moderate confusion

11-12 points – deep confusion

8-10 points – sopor

6-7 points - moderate coma

4-5 points - deep coma

3 points - deepest coma

Immediate aid for comatose patients:

- turn the patient aside
- thrust the jaw forward
- assess respiration (skin and mucous membranes colour, respiration rate and character, pathological noises during breathing act, intercostal spaces breathing retraction);
 - in the inspiration is difficult or oral cavity is filled with gastric contents, saline or blood provide airways patency (remove foreign bodies or liquids);
 - in case of ineffective respiration begin artificial ventilation;
 - check the pulse over peripheral and central arteries;
 - check photoreaction by raising the eyelids;
 - call for help (actually this is your primary task, but try to use help of witnesses and don't leave patient alone, especially if you are uncertain about his breathing ability).

To place patient into “recovery position” you should:

- a. place the arm nearest to you at a right angle, flex the opposite knee

b. turn patient aside by pulling the flexed knee and shoulder on the same side; then press the hand of the upper arm to the chick; make sure breathing is not difficult.

Some comatose patients can be hyperreflexive, hyperkinetic and disposed to cramps.

In case of cramps you should:

- lay patient in a supine position on a flat surface (to avoid injuries);
- prevent tongue biting inserting the gag or any available items (like stick or spoon wrapped with cloth);
- keep the airways open (usual methods described above) and hold the head in order to prevent trauma and asphyxia;
- provide oxygenation with the mask;
- between the attacks get an i/v access and give the patient 10-20 ml of 25% magnesium sulphate solution, 2 ml of 0,5% Diazepam solution;
- clean the oral cavity from blood, saliva, gastric contents;
- if patient's breathing is inadequate provide artificial ventilation.



5.3 Diabetic comas.

Diabetic ketoacidosis.

Diabetic ketoacidosis is a type of hyperglycaemic diabetic coma. It appears in case of diabetes decompensation, which can be caused by inadequate insulinotherapy (wrong dosage), violations of diet (overeating, alcohol abuse), other diseases (usually of infectious etiology – pneumonia for example). Sometimes coma is a first symptom of diabetes.

One more time pay attention to the detailed anamnesis: ask the relatives about the onset of the episode. Did the patient feel unwell few days before decompensation?

Was he complaining of weakness, thirst, nausea, vomiting, frequent urination, acetone-like breath odor?

The biochemical foundation of diabetic coma is within the mechanisms of cellular glucose consumption: when cells are not able to absorb glucose it is being accumulated in the blood and thus increases blood osmolarity. Due to osmotic pressure laws fluid will flow from the tissues to the vessels according to the gradients of concentration. This is the mechanism of thirst in case of diabetic patients. When glucose concentration is over 10 mmol per liter kidneys fail to reabsorb it completely from the urine and thus it begins to exude with the urine. The water follows glucose according to the already mentioned osmosis rules and this is the reason of diabetic polyuria and dehydration of diabetic patients. Simultaneously cells are suffering from the lack of energy caused by glucose consumption disorders. In order to maintain their vitality cells begin to use other than glucose energetic metabolites: proteins, fatty acids, etc. This ends up with ketosis (accumulation of ketone bodies) and acidosis. Cells begin to lose potassium and this brings hypokalaemia (potassium is not returning from the urine to the blood).

Your clinical and laboratory findings can be next:

- dehydration symptoms: body weight loss; dry skin and mucous membranes; decrease of skin turgor, eyeballs tone, blood pressure and central venous pressure, urinary output;
- metabolic acidosis signs: acetone-like breath odour, Kussmaul breathing (deep noisy breathing), face hyperaemia (acid substances in blood cause dilatation of small vessels and they become overfilled with blood- hyperaemia appears);
- nausea and vomiting: caused by accumulation of acids within the gastrointestinal tract - increased concentration of acid metabolites in the blood induces compensatory mechanisms of organism protection, among them mucous membranes secretion is one of the most effective;
- laboratory tests: glucose over 6,6 mmol/l, sometimes even more than 30-40 mmol/l; pH is 7,2 and less (uncompensated acidosis); hyperkalemia is caused by cellular potassium lost (with corresponding cellular deficiency of this cation); urine contains ketone bodies; blood tests prove dehydration (hemoconcentration signs: increase of hematocrit, haemoglobin, protein, red blood cells);

First aid: provide general methods of comatose patients treatment – control airways patency, adequacy of ventilation, call for help.

Intensive treatment.

1. Provide oxygenation with the oxygen mask or nasal catheter. If it is necessary perform the intubation and begin artificial ventilation.
2. Get an i/v access, insert urinary catheter and gastric probe; if you have enough time take care about the central venous line.
3. Begin the infusions: saline 1 000 ml during first hour, then 500-1000 ml per hour. Patients with coexistent cardiac diseases should be controlled very carefully: check their central venous pressure and diuresis (should not be less than 40 ml/hour).
4. Insulinotherapy: initial bolus of 10-20 Units of simple insulin (or 0,2-0,3 U/kg/hour in prolonged infusion); supportive dose is 0,1 U/kg/hour. If in 3-4 hours glucose concentration is not 30 % less dose should be doubled; if in 4-5 hours glucose concentration is not 50% less – dose is doubled again. But the speed of glycemia reduction should not be too high: make sure it is not over 5,5 mmol/l. During first 10 hours don't try to reduce to the level less than 14 mmol/l. When glucose concentration will reach 14 mmol/l reduce the insulin infusion to 0,05-0,1 U/kg/hour or change the way of insulin admission into subcutaneous injections every 3-4 hours. Usually 5-10 % solutions of glucose are used with insulin infusions to stop pathological fatty acids metabolism.
5. Control the potassium level according to the blood tests results: in case of hypokalemia add 40 mmols of potassium per each liter of solutions you give i/v, but not faster than 20 mmol/hour.
6. In case of prolonged treatment use antibiotics for infectious diseases prevention. In case if diabetes was decompensated by infection this will be your aetiological treatment of coma.
7. Take care about DIC-syndrome prophylactic: 5000 U of heparin s/c under the coagulation tests control or low molecular weight heparins s/c (0,6ml of fraxiparine once a day).
8. If acid-base disorders are serious (pH<7,1) use 100 ml of 4% sodium bicarbonate solution during first hour and control acid-base balance.

You can calculate the necessary volume of sodium bicarbonate according to the formula:

$$V_{\text{NaHCO}_3} (\text{mmol}) = 0,3 * \text{BE} * \text{BW},$$

BE- base excess (with “-”)

BW – body weight

9. Symptomatic treatment: if necessary use nootropics, heart medicines and hepatic protectors.

10. Control vital functions carefully and don't forget that decompensation of diabetes probably had curable external reason. So your task is to find it and provide proper treatment.

Parameters	Ketoacidotic coma	Hypoglycemic coma
Cause	Undiagnosed diabetes, irregularity of food intake and treatment, infections, traumas	Overdosage of insulin or peroral glucose-reducing drugs. Understated carbohydrates intake after insulin introduction, excess physical activity
Onset	Gradual (a day and more)	Acute (minutes, hours)
Arterial pressure	Reduced	At first increased, then reduced
Color of skin and mucosa membranes	Pale	Hyperemia at the beginning
Respiration	Infrequent, deep, loud	Normal
Skin humidity	Very dry	Wet
Skin turgor	Decreased	Normal
Muscle tonus	Reduced	At first increased, then reduced
Tendon reflexes	Areflexia	Inhibited at first, then increased, areflexia in coma
Glycemia	Increased	Decreased abruptly
Smell of expired air	Acetone	Non specific
Ketonemia	Significantly increased	Normal

Hyperosmolar nonketotic coma.

Hyperosmolar coma is a type of hyperglycaemic coma, caused by high glycemia without ketosis. This complication appears as a consequence of dehydration in elderly patients (in case of diarrhoea, vomiting, burns, excessive perspiration or diuresis). Hyperosmolar coma is diagnosed through the laboratory tests showing plasma hyperosmolality (check chapter 9 for details). If plasma osmolality is over 320 mOsm/l hyperosmolar coma is proved.

Intensive treatment is much alike the therapy of usual hyperglycaemic coma, however rehydration with hypotonic solutions is the main key to success in this situation (use 0,45% saline solution or 2,5% glucose solution). Us usual schemes for insulin therapy and treat the reason of dehydration. Remember: you should never use 4% sodium bicarbonate solution: its osmotic concentration is 3 times higher than the plasma osmotic concentration!

Hyperlactacidemic coma

Hyperlactacidemic coma is a diabetic coma, which appears in decompensated diabetic patients who suffer from hypoxia (disorders of respiration or oxygen transportation). In case of oxygen deficiency intracellular respiration is blocked and acid metabolites (including lactic acid) are accumulated. This brings acidosis and diabetic decompensation.

The most common reasons of hypoxia are pneumonia, shocks and anaemia.

Intensive treatment includes usual steps of hyperglycaemia therapy, but main accent is made over the liquidation hypoxia:

- check the efficiency of respiration and improve it if necessary (mask oxygenation or even artificial ventilation);
- provide shock treatment if the it is the reason of patient's comatose condition;
- transfuse blood components if the cause was anaemia;
- inhibit excessive metabolic reactions (normalize body temperature, use sedative medicines or neuroleptics if patient has psychomotor excitement and agitation);
- use antihypoxants (cytochrome c for example).

Hypoglycaemic coma

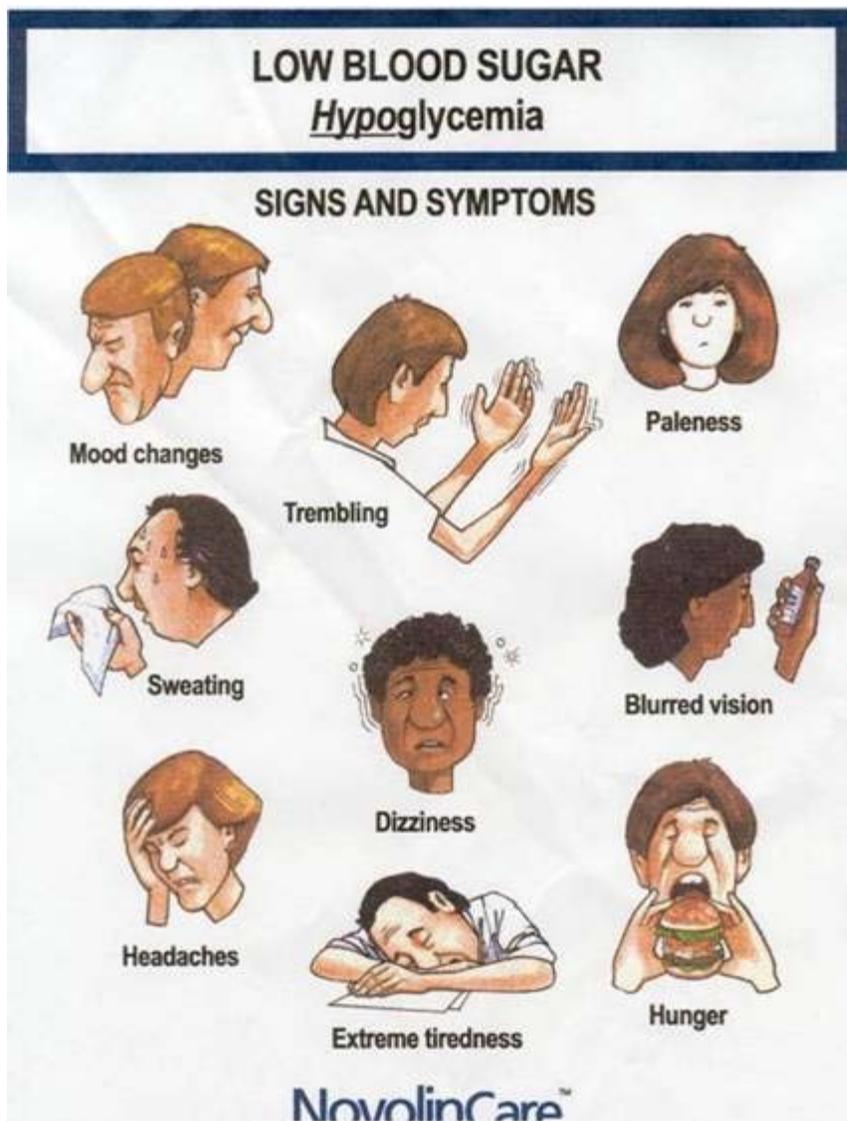
As you can see from the name hypoglycaemic coma is a condition of low glucose concentration in the blood. Unlike most of the previous types of diabetic coma it develops really quickly: within 30-60 minutes. The most common reason for this coma is improper insulin admission or diet violations (insulin overdose or lack of meal after insulin injection). Among the other reasons are: overdose of oral hypoglycaemic drugs (sulfonylurea), physical exercises without insulin dose correction, chronic alcoholism (violated glycogenolysis), hepatic diseases, postoperative gastric diseases (influence speed of glucose absorption).



Your clinical findings will be mostly connected with brain energetic deficiency: weakness, dizziness, coordination violations, dysphoria and inadequate behaviour, unconsciousness and cramps appear as a result of neuroglycopenia (as you remember glucose is the only energetic substrate for neurons, unlike other organs brain is 100% dependable on this monosaccharide).

Hypoglycaemia is classified according to the severity of the symptoms as mild (consciousness preserved, patient is able to take care of himself and hypoglycaemia is liquidated by oral admission of simple carbohydrates), moderate (patient needs help, but his condition is improving after oral admission of simple carbohydrates) and severe (patient is unconscious, needs help and his hypoglycaemia is liquidated only with parenteral glucose infusion).

When glucose concentration is 3,33-2,77 mmol/l you observe only mild hypoglycaemic symptoms. When it diminishes to the level 2,77-1,66 mmol/l the clinical picture is complete (except the unconsciousness). Glucose concentration less than 1,66 mmol/l induces coma. Hypoglycaemia lasting 4-6 hours can end up with irreversible cellular damages and even brain death.



Clinical picture is characterized with: pale skin wet from sweat; hemodynamics (central venous pressure, blood pressure) and other systems might be not involved in the initial stages; eyeballs tone and skin turgor are normal, mucous membranes are moist. Breathing is normal, not pathological Kussmaul breathing of ketoacidosis. There is no acetone-like breath odour. Urinary output is normal.

Those clinical features are quite typical, but to prove hypoglycaemia you should wait laboratory results (blood tests will show low glucose concentration).

Immediate aid.



In case of mild hypoglycaemia patient should eat 10-20 grams of simple carbohydrates (1-2 slices of bread, 2-4 candies, 200 ml of juice). If glucose level is not normalized within 20 minutes patient should take 10-20 grams of complex carbohydrates (few spoons of cereal for example) to prevent hypoglycaemic ricochet. Moderate hypoglycaemic should be treated simultaneously with simple and complex carbohydrates (10-20 grams of each).

Intensive therapy.

In case of severe hypoglycaemia use usual algorithms:

- provide oxygenation and adequate ventilation;
- get an i/v access as soon as possible;
- give 20-40-60-80-100 ml of 40% glucose solution (actually lately it is recommended to use 20% solutions because they cause less irritation of the peripheral veins); the precise dose you will choose according to clinical improvement of CNS condition (but not more than 120 ml);
 - in glucose solutions are not effective prescribe 1 mg of glukagon s/c; 1 ml of 0,1% adrenaline solution s/c, 3060 mg of prednisolone solution i/v;
 - additionally you can use: 100 mg of cocarboxylase i/v, 5 ml of 5% ascorbic acid solution,- or any required symptomatic medicines;
 - in case of brain oedema use 15% mannitol solution (0,5-1,0 g/kg) i/v;
 - even after clinical improvement patients should stay in the ITU for a

while, because his condition is still unstable and encephalopathy is still probable.

In children: when you are out of hospital use glukagon subcutaneously (0,5 mg with children under 5 years and 1 mg with elder kids). When you are in the ITU use i/v infusions of 20% glucose (1 ml/kg) during first 3 minutes, then 10% solution of glucose (2-4 ml/kg), check the glycemia and if you will not see the CNS improvement continue with 10-20% solutions of glucose and keep glucose concentration at the level of 7-11 mmol/l. Check glycemia level every half an hour or hour.

Control tests.

1. According to the GCS deep coma is characterized with:
 - A. 4-5 points
 - B. 13-14 points
 - C. 11-12 points
 - D. 3 points
 - E. complete lack of reaction to external stimuli
2. What is the condition of normal CNS functioning?
 - A. getting of 20-25 % of circulation blood volume every minute;
 - B. glycemia level not less than 3 mmol/l;
 - C. normal concentration of blood proteins and lipids;
 - D. brain should get at least 10% of total blood oxygen;
 - E. plasma osmolality should be 185-210 mOsm/l
3. Hyperglycaemic ketoacidotic coma is not characterized with:
 - A. metabolic acidosis;
 - B. overhydration;
 - C. low central venous pressure;
 - D. respiratory alkalosis;
 - E. severe paleness.
4. Hypoglycaemic coma is characterized with:
 - A. critically low blood pressure;
 - B. decreased tone of eyeballs;
 - C. wet skin;

- D. acute cardiac failure;
- E. noisy frequent breathing.

Task 1.

Andrew, the doctor, is examining Patience, the patient of 52. Patience is unconscious. His breathing is noisy and frequent; he has acetone-like breath odor; his skin and mucous membranes are dry; his blood pressure is low.

1. What symptoms will help Andrew to make a proper diagnosis?
2. Write the algorithm of Andrew's actions: clinical examination and laboratory diagnostics.

Task 2.

Choose the symptoms typical for:

- a. hyperglycaemic coma;
- b. hypoglycaemic coma.

1. Acetone-like breath odour. 2. Paleness. 3. Dry skin and mucous membranes. 4. Wet skin. 5. Hyperaemic face. 6. Tachycardia. 7. Normal heart rate. 8. Decreased tone of eyeballs. 9. Normal eyeballs tone. 10. Usual breathing rate. 11. Deep breathing. 12. Normal blood pressure. 13. Hypotension. 14. Normal urine output. 15. Oliguria. 16. Hyperkalemia. 17. Normal potassium concentration. 18. Hypokalemia. 19. Increased hematocrit. 20. Unchanged hematocrit. 21. Hyperglycaemia. 22. Hypoglycemia. 23. Central venous pressure over 4 cm H₂O. 24. Central venous pressure 6-10 cm H₂O.

Task 3. What medicines are not allowed for patients with hyperosmolar nonketotic coma and why?

a. Insulin; b. saline solution; c. 2,5 % glucose solution; d. 4% sodium bicarbonate solution; e. 10% calcium chloride solution; f. 0,45% sodium chloride solution; g. mannitol solution; h. heparin; i. spironolactone; j. prednisolone.

Task 4.

Andrew, the student of medicine, is a witness of such situation: in the street man suddenly felt and in few seconds cramps began.

What should Andrew do? What is the probable reason of this situation?

Task 5.

Patience, the patient of 50, was found unconscious by the ambulance workers at 4 o'clock in the morning. During the admission to the hospital: patient is not reacting to

pain stimuli. Respiration is normal, however in supine position airways loose patency. Alcohol odour is quite evident. Pupils are asymmetric, right is wider than the left, photoreaction is inhibited. Haemodynamics is stable. Abdomen is soft, peristalsis is active. Urination is not controlled.

What is the probable diagnosis? What treatment should be prescribed?

Acute poisonings.



Acute poisoning is a chemical injury, which occurs when chemical substance gets to the organism and violates its vital functions. If the substance is aggressive enough and proper treatment is not provided on time poisoning will bring death.

Although there are over 500 toxic substances which may cause acute poisoning, clinical picture is made up of quite similar syndromes. Proper diagnostics of these syndromes allows avoiding life-threatening complications and gets the chance to make correct preliminary conclusions about the nature of poisoning.



8.1 Main clinical syndromes of poisonings.

Affection of central nervous system manifests as excitation or depression of patient's mental activity. Depression of CNS has different stages: confusion, stupor, spoor

and toxic coma. One of the coma's deepness criteria is reaction of the patient to the painful stimuli (you can also check reaction to smell of ammonium chloride). Lack of reaction is a sign of coma. Don't live such patient without observation, because his condition is severe and at any moment life-threatening complications may appear: those patients have tendency to bradypnoea up to complete respiratory arrest. Also you should not forget that decreased tone of soft palate and tongue in a supine position will violate patency of the airways and patient can die of asphyxia. In addition comatose condition influences reflexes and lack of swallowing can lead to entrance of saline and gastric contents into the airways (and thus to development of aspiration pneumonia).

Usually CNS depression is caused by excessive alcohol consumption or admission of its surrogates, overdose of neuroleptics, sleeping pills, drugs, sedative medicines, antidepressants and carbon monoxide poisoning. You can remind intensive treatment of comatose patients in the chapter 5.

Some poisoning are followed with acute intoxication psychosis (mental disorders, hallucinations, time and space disorientation, inadequate behavior). This clinical picture you can observe in case of atropine poisoning (and also atropine-like agents: tincture of dope, henbane, amanita) or cocaine, tubazid, antihistaminic drugs and organophosphates poisoning.

In case of psychosis you will have to immobilize patient in the bed for his own good and safety (to avoid injuries both patient's and those of the stuff). You will also have to do this in order to maintain i/v lines for antidotes and sedative infusions (aggressive patient will try to remove everything he or she considers irritating). Constantly observe the patient's vital functions.

Toxic affection of the respiratory system can progress as the violation of:

a. external respiration - neurogenic form, aspirations and obstructions of the airways which bring hypoxic hypoxia;

b. hemoglobin's function – aniline and nitrobenzene create methemoglobin, carbon monoxide connected with hemoglobin creates carboxyhemoglobin and neither the first nor the second is capable of normal oxygen transportation; heavy metal, organic acids and arsenic poisonings lead to destruction of the red blood cells and emission of the free hemoglobin into the plasma;

c. oxygen transportation due to the decrease of circulating blood volume – exotoxic shock;

d. cellular respiration – tissue hypoxia occurs when cytochromes are blocked with toxins like cyanides.

Practically all severe poisonings earlier or later lead to hypoxia, because they violate oxygen supply, transportation and consumption.

Your immediate therapy actions in this situation will be:

- to assess of respiratory system (described above);
- to provide of airways patency (cleaning of the oral cavity, aspiration of the saline and gastric contents, conicotomy if necessary, etc.);
- to begin oxygen supply (face mask, nasal catheter);
- to start artificial ventilation if necessary;
- to prescribe antidotes if they are available (methylene-blue in case of nitro compounds poisonings, unithiolum in case of heavy metals and arsenic poisonings, cytochrome c in case of tissue hypoxia);
- to start hyperbaric oxygenation in case of carbon monoxide poisoning;
- to begin infusion therapy in order to stabilize the hemodynamics;
- to start general detoxification;
- to prescribe symptomatic treatment and provide prevention of the complications (for example prescription of antibiotics).

Affection of the cardiovascular system manifests as inability of the heart and vascular bed to provide adequate blood supply of the organs, which leads to metabolic disorders and in the worst case to death. Severe toxic damage of cardiovascular system brings acute cardiovascular failure: primary toxic collapse, exotoxic shock, secondary somatogenic collapse.

Primary toxic collapse appears in case of massive poison admission, when compensatory mechanisms are not quick enough to resist the chemical aggression. Immediately or minutes after poisoning patients begin to suffer from reduced cardiac output and thus from decreased blood flow in the tissues. Peripheral pulse is weak or absent, blood pressure critically lowers and cardiac arrest can appear. In most cases of primary toxic collapse ambulance is not able to save the life of the patient due to the fulminant development of life-threatening complication. However you should remember that such collapse occurs only in 5 % of the cases.

Exotoxic shock is the reason of death for 70 % of poisoning victims. Violations of hemodynamics on one hand are caused by direct heart and vessels damage and on the other hand by compensatory sympathetic and adrenal reactions. On the background of CNS and gastrointestinal system violations you will observe disorders of systemic hemodynamics and

microcirculation: arrhythmias, decrease of blood pressure, central venous pressure, cardiac output and diuresis. The peripheral vascular tone changes: toxins induce spasm or dilation of arterioles with the ischemia of one and hyperemia of other tissues. Depending on the body reaction on intensive treatment shock can be compensated, decompensated reversible and decompensated irreversible.

In case of toxic shock you should:

- get an i/v line (preferably several, including central venous access);
- start infusions of colloids (albumin, rheopolyglucin, hydroxyethylstarch solutions) and crystalloids (saline, glucose solutions, polarizing solution) in order to normalize blood pressure, heart rate and diuresis; sometimes infusion dose is up to 100-150 ml/kg, (7-10 l/day);
- constantly control patient's condition: monitor the heart action, blood pressure and central venous pressure;
- provide antidote treatment and detoxification; remember that extracorporeal detoxification is possible only after the stabilization of the hemodynamics (systolic blood pressure >90 mm Hg).

Secondary somatogenic collapse is the reason of death in 25% of the cases. It can occur few days after poisoning, when the toxin is already eliminated from the body, however the tissue changes (in the lungs, liver, kidneys, heart) are irreversible. Necessary treatment: hemodynamics stabilization, improvement of microcirculation, intensive therapy of functional disorders and organic changes (artificial lung ventilation, hemodialysis, cardiac support, etc.).

Toxic affection of gastrointestinal tract.

Usually poisoning provokes usual protective reactions: nausea, vomiting, diarrhoea. Chemically aggressive agents can cause "corrosive" effects: concentrated acids and bases can burn the mucous membrane of oral cavity, oesophagus and stomach (vomit is coloured with blood). Profuse vomiting and diarrhoea bring dehydration, electrolytes loss and acid-base imbalance. Especially rapid those complications appear in children.

After recovery patients with chemical burns of gastrointestinal mucous membranes may suffer from cicatrization and stenosis of digestive tract.

Be aware of the fact, that intensive usage of narcotic pain killers and sleeping pills can lead to inhibition of peristalsis and thus to constipation and slow elimination of toxins.

Immediate aid actions:

- clean the stomach. If the patient is conscious stimulate vomiting with the pressing on the root of the tongue or with 2-4 liters of slightly salted fluid. Don't you ever do this in case of chemical burns!
- if you have the skills and possibility insert the stomach probe and evacuate gastric contents with the help of 10-15 liters of water; if necessary give also antidotes through the probe; you can also use probes with several channels;
- after the gastric lavage in order to bind toxins use enterosorbents (activated charcoal for example);
- stimulate stool with saline laxatives (150-200 ml of 33% magnesium sulfate solution) in order to remove connected with the toxin sorbent from the intestines;
- cleansing enemas also help to eliminate toxins from the body.

Toxic affection of liver and kidneys.

This syndrome is caused by primary toxic damage of the liver and kidneys (nephrotoxic and hepatotoxic poisons) or by the secondary disorders of their functions due to violations of blood flow and oxygenation.

Liver is the main detoxification and biotransformation center of the organism, so it takes the "main blow" during intoxication. Intensive detoxification increases metabolic activity and oxygen consumption of the liver several times. Hepatocytes become very sensitive to hypoxia.

Light forms of toxic and hypoxic affections can develop without clinical manifestation. However they will be noticeable in laboratory tests (elevation of transaminases, bilirubin, phosphates). Severe poisoning will bring to toxic hepatitis and even hepatic coma.

Among the hepatotoxic substances are: heavy metals salts, dichloroethane, ethylene glycol, deadly amanita toxins.

To protect the liver you should:

- eliminate the toxic substance from the gastrointestinal tract;
- give antidotes if they exist (unithiol for heavy metals salts, lipoic acid for deadly amanita);
- prescribe cleansing enemas 2-4 times a day (to prevent intoxication with the wastes accumulated in the intestine);
- use extracorporeal detoxification (hemisorbtion, plasmapheresis, artificial liver);

- provide adequate oxygenation and blood supply of the liver;
- prescribe symptomatic treatment.

Kidneys are very important for the elimination of the poisons circulating in the blood. So in many cases they are also the “target” of the toxin. They can be damaged primary (poison affect their tissues directly) and secondary through the violations of vital functions (for example hemodynamics in case of exotoxic shock). Their condition you can control with the help of urine output per hour, which normally is not less than 0,5 ml/kg.

To prevent the renal failure you should:

- eliminate the poison as soon as possible (gastric lavage and enemas for gastrointestinal tract; hemodialysis, hemosorbition, plasmapheresis for blood);
- give antidotes if they exist (unithiol for heavy metals salts, sodium bicarbonate for hemolytic poisons, ethylic alcohol for ethylene glycol and methanol);
- treat disorders of hemodynamics (therapy against exotoxic shock);
- stimulate the urine output with the diuretics on the background of previous rehydration: this will allow you to eliminate diluted in the plasma toxins faster and to prevent renal failure; kidney is an organ which functions normally only if works intense;

Clinical observations tell us, that incredibly massive infusions (10-20-30 l/day) with diuresis stimulation really help patient to dilute and eliminate the toxin without kidneys damage.

In case of acute renal failure you should treat the patient according to the principles described in chapter 6.

8.2. Ethylic alcohol poisoning.

This type of poisoning appears in case of excessive alcohol consumption. It is one of the most common poisonings, as well as one of the lightest and prognostically the most favourable (organic damage is rare). However combined with comorbidities and complications it becomes one of the first reasons of death in toxicology, so don't underestimate it.

Alcohol poisoning, unlike drunkenness, has the characteristic signs of intoxication: vomiting, inhibition of CNS, disorders of cardiovascular system and breathing. Patients usually are in comatose condition. Pay attention to their appearance: clothes are untidy, you can notice sings of involuntary urination or defecation. There is alcohol breathing odour. Skin of the face is hyperaemic and dry. Cyanosis is a sign of respiratory insufficiency, grey

shade of skin is a symptom of cardiac disorders. Wet skin might be the symptom of hypoglycaemic coma, hypercapnia or organophosphate poisoning, which are “covered” with the obvious clinic of alcohol poisoning. In case of moderate coma vital functions are usually not involved. Pupils are narrowed or dilated, photoreaction is preserved. Objective criterion of alcohol poisoning is alcohol concentration of the blood:

- less than 1,5‰ – light inebriation
- 1,5‰-3,5‰ – moderate inebriation
- 3,5‰ and more – severe inebriation

Lethal concentration of alcohol is 5-6‰.

The most common complications of alcohol poisoning are next:

- obstruction of the airways with the tongue, soft palate or biological fluids (vomit, saline, sputum, blood) in supine position;
- regurgitation of the gastric contents and development of aspiration pneumonia; lethality is nearly 70%;
- head traumas with brain injuries: patients usually fell and hurt themselves; the problem with such injuries is that the clinic of hematoma (subdural, epidural, intracerebral) is quite often “covered” with alcohol intoxication and this is why you should always remember about the differential diagnostics. To make a correct diagnosis in case of coma you should check the specific symptoms such as anisocoria (there is no poisoning which causes pupil’s asymmetry!), signs of head injury (scratches, bruises, skull deformations, oto-liquorrhea and nasal liquorrhea, nasal and ear bleeding), asymmetric tendon reflexes and muscle tone, disparity between the amount of alcohol and deepness of coma, prolonged unconsciousness (alcohol coma even without proper treatment lasts only 3-4 hours);
- other traumatic injuries (rib fractures which violate external breathing, spleen or liver ruptures with haemorrhagic shock, ruptures of hollow organs with peritonitis; limb fractures);
- compartment syndrome appears when certain enclosed space within the body for several hours suffers from the decreased blood flow (for example when patients spends few hours in one inconvenient position); even when blood supply will be restored necrotic products will continue pathological process through toxic affection of the life-important organs (for example free myoglobin can cause renal failure).

There is always possibility of chronic diseases exacerbations on the background of alcohol poisoning (stroke, myocardial infarction). Remember about the necessity of complete examination (inspection, palpation, percussion and auscultation of undressed

patient) of such alcohol victims – it is the only way to find all the “diagnostic mysteries” patients hide!

Intensive treatment:

- evaluate CNS condition (deepness of the coma);
- provide airways patency and adequate respiration (described above; if necessary – intubate the patient and begin artificial ventilation);
- check the cardiovascular system: heart rate, pulse, blood pressure;
- in case of severe hemodynamic disorders provide infusion therapy;
- insert the stomach probe and remove its contents using lavage with water;
- take blood samples and check blood alcohol level (obligatory!);
- prescribe intravenously: 60-80 ml of 40% glucose solution, 60-80 ml of 4% sodium bicarbonate solution, 5-10 ml of 5% ascorbic acid, 1-2 ml of vitamin B₁ solution;
- if there are no comorbidities add analeptic solutions i/m (2-3 ml of caffeine or 2 ml of cordiamin);
- in case of severe intoxication begin forced diuresis.

8.3 Poisonings with alcohol surrogates.

Patients can be poisoned with: home-distilled vodka, Cologne water, denatured alcohol, methyl alcohol, lotion, brake fluid, etc. The peculiarity of such poisonings is complex effect of the alcohol and other toxic components of the “drink”.

The most toxic are methyl alcohol and antifreeze (ethylene glycol) – their lethal dose is 60-100 ml. Lower doses cause neuritis of optical nerve and thus blindness (methyl alcohol), acute renal and liver failures (ethylene glycol).

In case of these poisonings detailed anamnesis and blood identification of the poison (gas chromatography) play the most significant role in the diagnostics. However for the prognosis volume of the poisonous fluid, duration of its influence, functional condition of the liver and effectiveness of the antidote therapy and detoxification are the most important.

In the body methanol and ethylene glycol are metabolised according to so called “lethal-synthesis”: during the breakdown of the poison in the liver there are created substances much more toxic than the parent compounds.

Intensive therapy:

1. Gastric lavage with potassium permanganate (oxidizes methyl alcohol).
2. Give antidote: 50 ml of 40% ethylic alcohol solution every 3 hours orally or 100 ml of 5% ethanol solution intravenously slowly combined with glucose solution during 2 days. Antidote will block the process of their biotransformation in the liver until the poisons will not be eliminated from the body.
3. Actively eliminate the poison from the body through repeated gastric lavages, forced diarrhoea, extracorporeal methods of detoxification (hemodialysis, hemosorbtion, plasmapheresis).
4. In order to treat optic neuritis you should perform retrobulbar inject of steroids.
5. Symptomatic treatment.

8.4 Drug poisonings.

In civilized countries these poisoning are the main reason (65-70% of cases) of hospitalization in toxicology units. Usually patients overdose sleeping pills, narcotic painkillers, antihistamine drugs, hypotensive medicines. Among the reasons are suicide tries, drug abuse, toxicomania and accidental overdose due to hectic pace of modern life, etc.

Clinical picture is usually connected with CNS affection. There are phases of somnolence, sleep and coma. Depending on involvement of other systems coma can be complicated or uncomplicated. Usually respiratory complications appear: inspiration centre depression, violations of airways patency due to soft tissues (tongue, soft palate) or biologic fluids (blood, sputum, saline), pneumonia. In 15-20% of cases the poisoning development is complicated with the exotoxic shock. The peculiarities of this shock are next: circulatory disorders with blood stagnation in the pulmonary circuit, toxic affection of the myocardium and decrease of energy metabolism of the organism.

To indicate the poison you should ask relatives and witnesses and check things of the patient (for example you can find medicine packages). Evaluate the size of the pupils: extremely narrowed pupils ("poppy seeds") are the effect of narcotics admission; narrowed pupils might be the sign of sleeping or sedative medicines overdose; dilated pupils are the symptom of clofelin, antidepressants or neuroleptics administration; wide pupils covering the whole iris are usually the sign of atropine poisoning (or a poisoning with atropine-like substances: dope, henbane, amanita).

Principles of the intensive treatment in the toxicology unit:

- clean the gastrointestinal tract as soon as possible (gastric and intestinal lavage, enterosorbption, cleansing enemas) and as often as necessary;
- provide adequate respiration (check airways patency);
- in case of comatose patients intubate the trachea and begin artificial ventilation (sometimes it is necessary for weeks);
- control hemodynamics and treat its violations (infusion therapy and adrenergic agonists or antagonists if necessary);
- stimulate diuresis: patients with barbiturate poisoning should be treated with alkaline forced diuresis in order to eliminate the toxin (add to the infusion 400-600 ml of 4% sodium bicarbonate solution and prescribe diuretics);
- use antidotes: naloxone for opiates, pharmacological antagonists for anticholinergic and cholinomimetic agents; don't you ever prescribe central analeptics for comatose patients with drugs poisoning – cordiamin, caffeine, bemegride, cytiton, lobelinum can cause “cerebral blood flow stealing effect” and thus they deepen the hypoxia of brain cells!
- provide extracorporeal detoxification to eliminate toxins (hemodialysis, hemosorbption, plasmapheresis);
- prescribe antibiotics for infectious diseases prevention (for example in case of prolonged artificial ventilation);
- symptomatic treatment.

8.5 Alkali and acid poisonings.

These poisonings are among the most severe and difficult to treat. Accidentally or intentionally (suicide) victim can take mineral acids (hydrochloric, sulphuric, nitric acids), organic acids (acetic or oxalic acid), alkali (ammonium chloride, battery fluid, etc.).

When corrosive substance gets into the body along its way inside chemical burn appears: mucous membranes of oral cavity, throat, oesophagus, stomach are injured. Together they make nearly 14-15% of the body surface. Patients suffer from unbearable pain, eating and drinking are disabled. In case of acid burn coagulation necrosis appears; alkali burn is more severe, because colliquative necrosis penetrates deeper into the tissues ruining the vessels and causing bleedings. Organic acids easily get into the blood. Sometimes chemical substances or their vapours also get into the airways and thus oedema and risk of asphyxia appear.

Usually part of aggressive substance is spilled over the chin and you can notice

the burn. Systemic disorders are characterized with exotoxic shock which develops as burn shock (unbearable pain, dehydration, toxic affection of the heart, decrease of cardiac output, spasm of arterioles and microcirculation block). Organic acids also provoke hemolysis of red blood cells: free haemoglobin transforms into hydrochloric haematin and obturates renal tubules, causing acute renal failure.

Intensive treatment principles:

- evaluate patient's condition: external respiration, consciousness, cardiovascular system;
- adequate pain relief with narcotic painkillers and non-steroidal anti-inflammatory drugs (1-2 ml of 1% morphine solution; 2 ml of 50% analgin solution);
- liquidate the spasm of gastric cardia and oesophagus (1 ml of 0,1% atropine solution i/m, 5 ml of baralgin solution);
- clean the stomach during first 10 hours after poisoning; insert the stomach probe (cover it thickly with Vaseline and don't push too hard); use water for lavage and don't try to perform chemical inactivation of the poison, because during the reaction carbon dioxide can exude and acute expansion of the stomach leads to its rupture;
- provide treatment of shock (sometimes up to 10-12 liters/day of infusions);
- in case of organic acids poisonings (acetic acid, oxalic acid get into the blood) give 1500-2000 of 4% sodium bicarbonate solution intravenously slowly with diuretics; these actions will help to remove hemoglobin (released due to red blood cells hemolysis) and thus to prevent acute renal failure;
- in case of obstructive breathing disorders (mucous membrane edema) use steroids (90-120 mg of prednisolone), antihistamine drugs (2 ml of 1% dimedrol solution intravenously), sedative medicines (2 ml of 0,5% diazepam solution); perform tracheostomy or conicotomy if necessary;
- prescribe antibiotics for infectious diseases prevention (for example in case of prolonged artificial ventilation);
- symptomatic treatment.

During the recovering period patient may need surgeries for restoration of gastrointestinal tract: the most common practice is the bouginage of the oesophagus or, if necessary, oesophagus plastic.

8.6. Poisonings with toxic gases.

Among the toxic gases are carbon monoxide, car exhausts, propane and butane, ammoniac gases. The last one is the most toxic: few inhales are enough to cause unconsciousness.

The foundation of the pathology lies within the atypical haemoglobin – carboxyhaemoglobin – combination of normal haemoglobin and toxic gas. Oxygen transportation is violated (in case of severe poisonings there are nearly 70-80% of changed haemoglobin) and thus haemic hypoxia appears. In addition within the tissues cytochromes are connected with toxic substances and this leads to tissue hypoxia.

Clinical findings in case of carbon monoxide poisoning depend on the severity of the poisoning. In case of mild intoxication they are: headache, nausea, vomiting. Moderate intoxication shows unconsciousness for 12-16 hours and severe intoxication is characterized with coma, central disorders of breathing, toxic affection of heart and other organs, etc.

If intoxication advances changes of central nervous system become irreversible (brain death is possible).



Intensive treatment.

In case of mild and moderate poisonings you should carry the patient out of the toxic atmosphere as soon as possible. In hospital conditions you should provide oxygen supply, get an intravenous line for crystalloids infusion and prescribe vitamins.

In case of severe intoxication begin artificial ventilation with high oxygen flow. Luckily there is an antidote for carbon monoxide poisoning: hyperbaric oxygenation. Connection with oxygen is more natural for haemoglobin and when the pressure of

oxygen is higher than its usual partial pressure carbon monoxide is replaced from the haemoglobin. Usually in case of comatose patients 40-50 minutes sessions every 6-12 hours are enough.

To normalize tissue metabolism prescribe antihypoxants: 20% solution of sodium oxybutirate (20-40 mg/kg i/v) and cytochrome c (2-3 ml i/v) every 4-6 hours. To improve microcirculation dilute the blood with crystalloid infusions (check the level of hemodilution with hematocrit – stop when it will reach 0,3-0,35 l/l).

Prevent the infectious complications and brain oedema with standard methods.

8.7 Organophosphate poisonings.

These are the poisonings with insecticides, acaricides, herbicides, fungicides, rodenticides, desiccants, defoliants and with chemical warfare agents such as sorin, soman, V-x.

Organophosphate substances are fats and water soluble and thus they penetrate easily through the skin and mucous membranes (gastrointestinal tract, airways, etc.). In the blood they block an enzyme – cholinesterase – responsible for the breakdown of acetylcholine. As you remember acetylcholine is a universal synaptic mediator of nervous impulses and thus its accumulation on the post-synaptic membranes will cause continuous stimulation of vegetative nervous system and cross-striated muscles.

Clinically you will see: nausea, vomiting, cramps; unconsciousness in severe cases. Sometimes in the place of penetration you can see muscle fasciculation (if the poison was administered orally – tongue twitching). Stimulated parasympathetic nervous system shows wet skin, increased salivation and bronchial secretion (sometimes you can even see white phlegm in the mouth – don't mistake it with the pink phlegm of pulmonary oedema), narrowed pupils, bradycardia (heart rate 40-30 beats per minute). In addition to the obvious clinic you can always check the environment of the patient for the signs of organophosphate poisons (specific smell, containers with toxins, etc.).

One third of patients suffer from exotoxic shock, which primary causes hypertension and then hypotension, unconsciousness and depression of respiration.

Immediate aid:

- take patient out of the dangerous environment (if you suspect that the mechanism of poisoning is inhalation);
 - clean the stomach with large amounts of cold water; repeat it several times, because these substances are excreted through the mucous membranes of gastrointestinal tract;
 - give saline laxative;

- if the poison affected skin – wash it with alkaline solution.

Antidotes:

a. use peripheral m-anticholinergic drug – atropine: during the first few hours 2-3 ml of 0,1% atropine solution (up to 30-35 ml during the whole period of intensive atropinization); pay attention to the signs of atropine administration as they are the measure of your antidote treatment effectiveness: termination of excessive bronchial secretion, dilation of the pupils, tachycardia (90-110/min). During next 3-5 days continue atropine prescription (from 10-15 mg to 100-150 mg/day – period of supportive atropinization). Control clinically the level of atropinization.

b. use cholinesterase reactivators: 1-2 ml of 15% dipiroxim solution i/m, up to 600 mg; 3 ml of 40% izonotrozin solution i/m up to 3-4 grams. However remember, that cholinesterase reactivators can be used only 24 hours after poisoning. Later administered reactivators will ne not only ineffective, but also toxic for the patient.

You should also provide usual treatment as soon as possible: infusion therapy, forced diuresis, hemosorbtion, plasmapheresis, hemodialysis and antibiotics for infection preventions.

In case of ineffective external respiration and comatose patient's condition intubate the patient and start artificial respiration. Convulsions in case of organophosphate poisoning are treated with sodium oxybate (75-100 mg/kg i/v every 4 hours). Cardiac glycosides, calcium chloride, euphillinum are forbidden in case of organophosphate poisonings, because they induce toxic heart affection.

Be aware of the possibility of “second poisoning wave”: even 4-8 days after the stabilization of the patient's condition clinical picture of the poisoning might return and this time hemodynamics will decompensate quickly.

8.8 *Mushroom poisoning.*

There are edible, non-edible and relatively edible mushrooms. Non-edible or poisonous mushrooms can contain toxins harmful for central nervous system, liver, kidneys and gastrointestinal tract (according to A. Lokay, 1 968). The most dangerous poisoning is caused by deadly amanita. The poison of this mushroom – amanitotoxin- is not destroyed during cooking and there is no way to detect it in usual conditions. In case of severe poisoning the lethality is 80%.

Specific feature of amanita poisoning is prolonged latent period. Sometimes 6-12 hours pass before the first symptoms of the poisoning appear. All the other relatively edible mushrooms reveal clinical signs of the poisoning much earlier – 1-2

hours after consumption.

After the latent period is over on the background of complete health nausea, profuse vomiting and diarrhoea appear. Those symptoms begin second phase of the poisoning – gastroenterocolitic phase. Liver enlarges; patients suffer from pain in the right subcostal area, weakness, and consciousness disorders. Stool becomes watery and contains mucous. Patients loose up to 4 liters of the fluid during the day. Unlike bacterial food poisonings, mushroom poisonings are not characterized with high fever.

Liver failure and acute kidneys injury are the third phase of the poisoning, which begins on the second or third day of disease. Those failures are characterized with hepatic encephalopathy, jaundice, gastrointestinal bleedings, hepatic breath odour and oligoanuria. The level of alanine and aspartate aminotransferases is very high. When liver returns to its usual size and consciousness is changed into coma hepatargia is stated and prognosis for the disease becomes rather unfavourable.

The forth stage is a stage of recovery and is characterized with gradual regression of the clinical picture and normalization of the laboratory results during several weeks. However survival is possible only for those patients, who ate small amounts of the poisonous mushroom.

Knowing about the high lethality and severity of the amanita poisoning prevention methods become very important. Mushroomers should know the difference between deadly amanita and other mushrooms: deadly amanita is a gill-bearing mushroom with olive or green cap. Its gills white are not connected with the stem. Stem has a bulbus with white volva from one side and a white annulus from another side, under the cap.

Patients should be treated in a special toxicology units or intensive treatment unit. General principles of intensive therapy are:

- gastric and intestinal lavage, enterosorbition and saline laxatives;
- infusion therapy (necessary to liquidate electrolyte deficiency and provide forced diuresis);
- support of the liver functioning with lipoic or thioctic acid (1000-2000 mg/day), concentrated glucose solutions, steroids (up to 40 mg of dexamethasone per day) and silibin (50 mg/day);
- extracorporeal detoxification (hemodialysis, hemosorbition, plasmapheresis, artificial spleen or liver) as soon as possible;
- antibiotics if necessary (penicillin); vitamins (B,C,E);
- external drainage of thoracic duct (decreases intoxication through

elimination of toxic lymph).

8.9 Medical operations and manipulations.

Gastric lavage

Indications: necessity to remove poisons or toxins from the stomach, to clean it before operation or to liquidate stagnation during period after the operation.

Equipment required: gastric probe (with two channels if possible), Janet syringe, water for lavage (15-20 liters of room temperature water), gloves and watertight apron.

Procedure: gastric lavage of comatose patients is a procedure for doctors. In our country nurses are not allowed to do this without control of the doctor: unconscious patients have inhibited reflexes and thus probe can be easily inserted into the trachea instead of oesophagus. In this situation neither cyanosis nor cough may not appear and everything looks just fine, however feeding or lavage try can end with fatal complications (asphyxia and death).

Put on the gloves, choose the probe of necessary size and oil it with Vaseline. Patient should lie on the left side (ask nurse to hold patient's arms to limit his movements during this unpleasant procedure).

If patient is conscious you can previously use lidocaine spray to anaesthetize mucous membranes. Probe can be inserted through the nose (of course in this case size of the probe is limited) or through the mouse. Don't push too hard, especially when you are using nasal passage: you can cause bleeding. Ask patient to bow his head to the chest – this will increase chances of correct probe insertion (oesophagus, not trachea). The length of probe you insert can be measured in advance as a distance between earlobe, nose and xiphoid.

Confirmations of correct probe placement:

- auscultate the epigastrium and simultaneously infuse some air with the syringe- you will hear typical "bubble" noises;
- if you made a mistake and probe is in the trachea you can notice air released from the distant end of the tube according to the respiratory movements.

One-time water dose is nearly 200 ml: it will flow out when you will lower the probe or you will have to evacuate the water with the syringe. Repeat these actions until the water wash out the stomach will be clean (usually it takes nearly 10 liters of water).

Forced diuresis.

Indications: intoxications of different origin (poisonings, infectious diseases, endogenous intoxications).

Medicines required: normal saline (3-5 liters); detoxification solutions; polarizing solution (400 ml of 10% glucose solution, 10 ml of 7,5% potassium chloride solution, 12 units of insulin), osmotic diuretics (mannitol in the dose 1 g/kg), furosemid solution (40-80 mg)

Procedure: get and i/v line (central or peripheral) and insert urinary catheter. During the first phase of forced diuresis “water” the patient with crystalloids and detoxification solutions (30-40 ml/kg). During the second phase infuse osmotic diuretics and furosemide solution. Excessive urine output brings potassium loss, which you should treat with polarizing solution. Balance the speed of infusion with the speed of diuresis: generally per 5-7 liters of infused solutions you should receive at least 5 liters of urine.

Constantly control hemodynamics and blood electrolytes.

Control tests.

1. What medicine should be used as an antidote in case of severe soporific drugs poisoning?

- A. bemegrade
- B. cordiamin
- C. there is no such medicine
- D. unithiol
- E. cytiton

2. What is the most common death reason in case of alcohol poisoning?

- A. acute liver failure
- B. acute respiratory failure
- C. acute renal failure
- D. acute heart insufficiency
- E. collapse

3. What is necessary for the patient, who took 60 ml of acetic essence?

- A. to give 4% sodium bicarbonate solution i/v
- B. to clean the stomach with alkaline solution in order to neutralize the acid
- C. to use unithiol as an antidote

- D. to stimulate intestinal cleansing with saline laxatives
 - E. central analeptics
4. What is the symptom of organophosphate poisoning?
- A. pale and dry skin
 - B. maximal pupil's dilation
 - C. tachycardia
 - D. muscle fasciculation
 - E. acute liver failure
5. What is used as an antidote in case of severe carbon monoxide poisoning?
- A. cytochrome c
 - B. cordiamin solution
 - C. unithiol
 - D. prednisolone
 - E. antidotes are not used
6. What is typical for deadly amanita poisonings?
- A. first symptoms appear in 2-3 hours after mushroom meal;
 - B. low-grade fever
 - C. latent period of 6-12 hours
 - D. first symptoms are haemostatic disorders (bleeding)
 - E. early unconsciousness
7. What is the necessary aid in case of narcotic painkillers overdose?
- A. artificial ventilation
 - B. cordiamin
 - C. oxygen supply
 - D. immobilization of the patient in order to avoid self-injuries during the excitement-phase;
 - E. cardiac medicines

Task 1.

Patience, the patient of 19 is transported to the ITU by witnesses from the street.

Clinical findings: unconscious, cyanotic wet skin, 6 breathes per minute, respiration shallow, blood pressure 70/40 mm Hg, heart rate 112/minute. Name the reason of vital disorder and write the principles of intensive care (algorithm).

Task 2.

Patience, the patient of 19, was found unconscious in his own apartment by the ambulance workers. Clinical findings: total cyanosis, shallow breathing, respiration rate 5/min, pupils extremely narrowed, photoreaction is absent, blood pressure 80/40 mm Hg. In the elbow area there are noticeable signs of injections. Name the reason of vital disorder and write the principles of intensive care (algorithm).

Task 3.

Patience, the patient of 23 was hospitalized into ITU with the diagnosis: mushroom poisoning. It turned out, that yesterday she was eating cooked champignons. 8 hours later she noticed vomiting and diarrhoea. Clinical findings: scleral icterus, dry coated white tongue; painful abdomen (epigastrium and right hypogastric area), painful and enlarged liver (2 cm); hemodynamics and respiration are not violated.

Write the diagnosis and the phase of the disease.

Task 4.

Patience, the patient of 18, is transported to the toxicology unit with delirium. Clinical findings: hyperaemic face, dry skin, dilated pupils. Blood pressure 140/70 mm Hg, heart rate 127/minute. It is known, that 2 hours ago she took 10 unknown tablets during the suicidal attempt. What is the diagnosis and what are your actions?

Task 5.

Patience, the patient of middle age, was found by the ambulance workers in his own kitchen unconscious. Clinical findings: specific smell of organophosphates in the room, signs of vomit on the clothes, miosis, cold clammy skin with cyanotic shade, foamy white sputum in the mouth, blood pressure 90/50 mm Hg, heart rate 54/minute. What is the diagnosis? What immediate aid should be provided during pre-hospital stage?

Task 6.

Adams family were celebrating the New Year Eve near the fireplace. In the morning came the carol singers and found hosts in a condition of deep sleep. "Dream team", the ambulance workers, who came to the place of the accident, stated: 2 adults and their children in comatose condition, vomit signs on the clothes, rapid breathing, pupils dilated with weak photoreaction; heart rate is 110-120 per minute, rhythmic; blood pressure is high.

Name the reason of the accident and describe the actions of the "Dream team".

Task 7.

Name the poisoning agent for each antidote: a. atropine, b. unithiol, c. naloxone, d. tetacinum, e. ethylic alcohol, f. dipiroximum, g. lipoic acid?

Student should repeat next questions

Among the natural vasoconstrictors (agents, which cause constriction of the blood vessel) are epinephrine, norepinephrine, serotonin, angiotensin II. Stress enhances the secretion of catecholamines, their blood concentration increases and arterioles constrict. Spasm of the arterioles is the basis of blood flow centralization: peripheral flow is disregarded in order to provide brain with the oxygenated blood as long as possible. To the group of vasodilators (agents, which provide dilatation of the vessels) belong “acid” metabolites (lactate, pyruvate, adenylic acid, inosinic acid), bradykinin, acetylcholine, different medicines (neuroleptics, α -adrenergic antagonists, peripheral vasodilators, ganglionic blocking agents, etc.), some exogenous poisons. All of them cause blood flow decentralization: opening of arterioles and distribution of the blood from central vessels to the capillary bed.

Capillaries are the interweaving network of the smallest body vessels with the general length of 90-100 thousands of kilometers. However simultaneously work only 20-25% of them. They provide metabolic exchange bringing oxygen and nutrients to the tissues and take back wastes of metabolism. Periodically, every 30-40 seconds one of them get closed and others open (vasomotion effect). Capillaries contain 12% of the whole circulating blood volume, but different pathological conditions can increase this amount even 3 and more times.

“Used” blood from the capillaries flows to the venous system. Veins are the blood reservoir, which contains 70% of the total circulating blood volume. Unlike arteries they are capable of volume control and thus they influence the amount of blood, which returns to the heart.

The most important haemodynamic index of venous system is central venous pressure. CVP represents the pressure which blood causes to the walls of cava veins and right atrium. This parameter is an integral index of circulating blood volume, systemic vascular resistance and pump function of the heart. It can be measure with a special device called “phlebotonometer” (pic. 4.9) or with a usual infusion set and a ruler. Normally CVP measured from the sternum point is 0-14 cm H₂O and from midaxillary line is 8-15 cm H₂O.

Central venous pressure decreases (sometimes even to negative) in case of:

- blood loss
- excessive water loss (dehydration)

- distributive shock (decrease of peripheral resistance due to venous and arterial dilatation)

In those conditions decreases volume of blood returning to the heart and thus suffers cardiac output. In case of negative CVP cardiac arrest is highly probable.

Central venous pressure increases in case of:

- heart failure (insufficiency of left or right ventricle)
- hypervolemia (excessive blood infusion, improper infusion therapy)
- obstructions to blood flow (pulmonary embolism, cardiac tamponade, etc.)

When CVP over 15-16 cm H₂O is combined with left ventricle insufficiency the risk of pulmonary edema is very high.

Blood pressure is an integral index of arterial part of systemic haemodynamics. Talking about blood pressure we may refer to systolic, diastolic, pulse and mean arterial pressure. Systolic (P_{syst}) and diastolic (P_{diast}) pressures are measured with the manometer (method with the usage of phonendoscope was invented by M. Korotkoff). Pulse pressure (PP) is a difference between systolic and diastolic blood pressure.

Mean arterial pressure (MAP) is calculated according to the formula:

$$\text{MAP} = P_{\text{dias}} + 1/3 \text{ PP} \quad \text{mm Hg}$$

MAP defines the level of pressure necessary for the metabolic exchange in the tissues. Its measurement allows the evaluation of tissue perfusion level.

Blood pressure depends on different factors, but the most important are cardiac output and vascular resistance (mostly arterioles). This dependence is direct, thus you can increase blood pressure using:

- infusion of vasoconstrictors - solutions of epinephrine, phenylephrine (mesaton), etc. (they will increase the vascular resistance);
- infusion of hydroxyethyl starch solutions or saline (they will increase circulating blood volume)
- infusion of cardiac glycosides or other medicine which stimulate myocardium

General volume of blood in the body of a healthy adult is nearly 7% from the body weight: 70 ml per kilogram for male and 65 ml per kilogram of body weight for female. Of

course circulating blood volume is lower, because part of blood is out of metabolic processes as a reserve. CBV can be measured with the infusion of coloring substance to the blood flow (Evans blue, polyglucin) and later evaluation of its dissolution degree.

Therefore measurement of CVP, BP, cardiac output and circulating blood volume allow to evaluate condition of circulation system of the patients and to provide adequate correction.

4.2 Acute heart failure; shock and collapse.

Acute cardiovascular failure is a state of cardiac and vascular inability to provide adequate supply of tissue metabolic needs with oxygenated blood and nutrients. This, earlier or later, causes cellular death.

The reasons of the failure vary greatly: mechanic injuries, blood loss, burns, dehydration, exogenous and endogenous intoxications, immediate hypersensitivity reaction, ischemic heart disease, neural and humoral regulation disorders of vascular tone.

Acute cardiac failure is a disorder of heart pumping action. It develops due to primary heart problems or secondary, under the influence of extracardiac factors such as infection or intoxication. There are two types of heart failure: left-sided and right-sided.

Left-sided heart failure is an inability of left ventricle to pump blood from the pulmonary circuit to the systemic circuit. The most common reasons of it are myocardial infarction, mitral insufficiency, left AV valve stenosis, aortic valve stenosis, aortal insufficiency, hypertonic disease, coronary sclerosis, acute pneumonia.

Coronary circulation is possible only during the diastole and in those conditions every violation of coronary passability decreases cardiac output. This way during the systole part of the blood is not injected into aorta, but stays in the left ventricle. Diastolic pressure in the left ventricle increases and blood is literally forced to stagnate in the left atrium. At the same time right ventricle functions normally and continues to pump usual amounts of blood to the pulmonary circuit. Thus hydrostatic pressure in the vessels of pulmonary circulation increases, fluid part of the blood moves first to the lung tissue and then, through alveolar-capillary membrane, to the alveolar lumen.

Clinically pulmonary edema begins with dyspnea (during physical activity or rest). Later attacks of dyspnea are connected with persistent cough with white or pink blood-tinged phlegm. During the attack patient tries to sit as in this position breathing is easier. This condition is called "heart asthma". When hydrostatic pressure is over 150-200 mm Hg, fluid part of blood moves to the alveolar lumen causing development of pulmonary edema.

Pulmonary edema is divided into interstitial and alveolar edema.

Interstitial edema is a condition during which serous part of stagnated in the pulmonary circuit blood infiltrates the lung tissue, including peribronchial and perivascular spaces.

During alveolar edema not only the plasma, but also blood components (red and white blood cells, platelets) get out from the vessels. During the respiratory act blood mixes with the air creating large amount of “foam”, which violates gas exchange. This way, in addition to circulatory hypoxia, hypoxic hypoxia appears.

Condition of the patient gets worse quickly. Sitting position is optimal, but not as helping as previously. Respiratory rate is nearly 30-35 breathes per minute, but attacks of breathlessness are constant. Skin is pale with acrocyanosis. Hypoxia of central nervous system usually causes psychomotor agitation. Respiratory acts are noisy; during cough pink blood-tinged phlegm is released. Auscultation allows you to hear different wet rales, sometimes it's even possible to hear them standing aside the patient without phonendoscope.

Pulmonary edema can be also divided according to the blood pressure level: the one with elevated pressure is caused by a hypertonic disease, aorta valve insufficiency or disorders of cerebral perfusion; another one is caused by total myocardial infarction, acute inflammation of myocardial muscle, terminal valve defects, severe pneumonia and is characterized with normal or low blood pressure.

Immediate aid

- make sure patient is sitting with his legs down (orthopnea)
- provide oxygenation through nasal catheter (before placing oil it with glycerin, insert it to the depth of 10-12 cm – distance from the wing of the nose to auricle) or face mask. Do not use Vaseline, because it can burn in atmosphere with high concentration of oxygen.

However if catheter is not deep enough patient will suffer from an unpleasant “burning” feeling, because oxygen flow will dry mucosa layer of the nasal cavity; also in this situation concentration of oxygen will be lower than expected.

- put venous tourniquets on the limbs in order to reduce amount of blood returning to heart: venous bed of limbs can reserve up to 1,7 liters of blood;
- constantly control heart and kidney activity (ECG, SaO_2 , and blood pressure are checked automatically through the monitor; to control diuresis you should insert Foley catheter;
- catheterize central vein, because amount of infusions should be based on central venous pressure;
- use medical “defoamers” if they are available (ethyl alcohol or antiphomsylan solution) combined with oxygen inhalation

Scheme of oxygenation set connected to “defoamer” container

- a. oxygen source (cylinder with oxygen)
 - b. tube with numerous holes sunk into container with defoamer
 - c. tube for humidified oxygen (its opening should be over the level of fluid);
 - d. patient
- medical treatment: 1% morphine solution (decreases intravascular pressure of pulmonary circuit, inhibits respiration center in medulla oblongata preventive dyspnea progress, sedates patient);
 - solutions of diuretics are used to decrease the circulating blood volume (6-12 ml of 1% furosemid solution, solution of ethacrynic acid), however be careful with them in case of low blood pressure; diuretic effect will last up to 3 hours after i/v infusion, the expected diuresis is 2-3 liters
 - if blood pressure allows you can try to use nitroglycerin to reduce intravascular pressure of pulmonary circuit (1 or 2 tablets with 10 minutes interval)
 - cardiac glycosides for improvement of heart action (0,025% digoxin solution, 0,05% strophanthin solution, 0,06% corglicon solution);
 - in case of high pressure (over 150 mm Hg) use ganglionic blocking agents (1 ml of 5% pentamin solution diluted in 150 ml of saline, give i/v slowly; diluted with saline 250 mg of trimethaphan solution), because they reduce pressure in pulmonary circuit and lower the amount of blood getting to right half of the heart, however be careful with the dosage and monitor blood pressure level carefully;
 - never use osmotic diuretics in case of pulmonary edema – they will increase blood volume and thus heart load!!!
 - when everything listed above failed and patient is worsening with every second you should intubate him and start artificial ventilation with positive end expiratory pressure (begin with 4-6 cm H₂O)

Right-sided heart failure is an inability of right ventricular to pump blood from systemic circuit to the pulmonary circuit due to its weakness or an obstruction to the blood flow.

It occurs in case of pulmonary embolism, right ventricular infarction, excessive infusion therapy (especially including citrated blood) for patients with heart insufficiency, lung

diseases (bronchial asthma, emphysema, pneumosclerosis) which cause increase of right ventricular load.

Patients have acrocyanosis, tachycardia, dyspnea, pronounced neck veins, ankle swelling, enlarged liver, ascytis. Central venous pressure is highly increased (up to 20-25 cm H₂O), however pulmonary edema does not appear.

Intensive treatment is mostly pathogenetic:

- limit the infusions (give only life-necessary solutions, check the water balance of the patient and reduce drinking water if necessary);
- in case of citrated blood transfusions use 5-10 ml of 10% calcium gluconate solution per every 500 ml of blood to prevent hypocalcaemia;
- in case of bronchial spasm use bronchial spasmolytics;
- to remove excessive fluid from the body use diuretics (furosemide solution for example);
- metabolic acidosis is corrected with 4% solution of sodium bicarbonate (i/v slowly with acid-base state control);
- in case of pulmonary embolism anticoagulants are used – fraxiparine 0,6 mg subcutaneously; heparin solution – 5000 IU every 4 hours; fibrinolytic drugs (streptokinase, fibrinolysin, urokinase, etc.)

Shock is a pathological state which can be described as a tissue hypoxia caused by hypoperfusion. Pathogenetic basis of shock depends on its reason (trauma, toxins, thermal injury) and at the same time on reactivity of the organism (level of defense mechanisms mobilization).

Stimulation of sympathetic nervous system - production of catecholamines and other vasoactive substances by hypothalamus and adrenal glands are the universal response of the body to the stress. Those mediators interact with the receptors of peripheral vessels causing their constriction and at the same time they dilatate the vascular bed of life-important organs. This is so called “centralization of the flow”: rational decrease of blood flow in less important tissues (skin, organs of abdominal cavity, kidneys) in case of aggressive external influence for protecting life itself (brain, heart, lungs).

However influence of shock agents (pain, hypovolemia, destroyed cells, toxic metabolites), extended microcirculation violations (vascular spasm, microthrombosis and sludge) and caused by them tissue ischemia lead to hypoxic affection and cellular death of the internal organs. Further it can bring multiple organ dysfunction syndrome.

Collapse is a vascular failure. It occurs when body is not able to provide blood flow according to the new level of its needs (either because reaction is not fast enough or because sympathetic activation fails). Vascular bed volume and circulating blood volume are disproportional: too much blood gets to the microcirculation vascular reserve and the amount, which returns to the heart is not enough for the systemic needs (so called “decentralization” of the blood flow). Cardiac output and blood pressure decrease, that causes hypoperfusion of the central nervous system and thus unconsciousness and life-threatening complications.

Collapse definition is a bit nominal, because if such reaction extends in time the state of shock develops. Shock itself can equally run as a vascular failure or as a sudden clinical death.

Pathogenetic classification of shock (according to P. Marino, 1998):

- hypovolemic
- cardiogenic
- distributive
- mixed (two and more factors).

Clinical classification of shock:

- traumatic shock;
- haemorrhagic shock;
- dehydration shock;
- burn shock;
- septic shock;
- anaphylactic shock;
- cardiogenic shock;
- exotoxic shock.

4 Shock caused by dehydration

It is a type of hypovolemic shock, which occurs during excessive body fluid loss (not blood, because hemorrhagic shock is another shock type).

Its reasons vary greatly:

- gastrointestinal diseases (profuse vomiting, diarrhea, loss of intestinal fluid through fistula);
- polyuria (uncontrolled diuretic treatment, diabetes mellitus and insipidus, diuretic phase of acute renal failure);
- fluid loss through skin and wound surface (burns, high fever);
- inadequate infusion treatment of postoperative or comatose patients;
- hyperventilation (rapid breathing, Kussmaul breathing, inadequate artificial ventilation parameters in case of apparatus without air humidification).

However not only the complete fluid loss can be the reason of shock, but also it's pathological distribution into the extracellular space (intestinal cavity in case of intestinal paralysis, abdominal cavity in case of ascites, pleural cavity in case of pleurisy). This way will can also act prolonged heavy tissue inflammations (peritonitis) or massive injuries (crush-syndrome).

In cases described above electrolytes are also lost (cations of sodium, potassium, calcium, magnesium; anions of chlorine, hydrocarbonate). It causes complex osmolar, acid-base and electrolytic disorders.

Stage of dehydration shock is evaluated according to the actual fluid loss:

less than 5% of body weight – mild dehydration

5-10% of body weight – moderate dehydration

over 10% of body weight – severe dehydration

Water deficiency brings lowering of cardiac output, blood pressure and central venous pressure (through decrease of blood volume returning to the heart, which leads to compensatory adrenergic vasoconstriction).

Dehydration causes body weight loss, skin and mucosa dryness, decrease of subcutaneous turgor and eyeballs tone, hypothermia, tachycardia, oliguria, thirst. While dehydration progresses compensatory mechanisms weaken and central nervous system suffers: patients become sluggish, confused; hallucinations, cramps and unconsciousness are also possible. Laboratory tests show blood concentration.

One of the most important things in treatment of dehydrated patients is daily balance of fluid: check it carefully trough measuring of daily received and lost fluids (food, infusions, stool and urine output). In case of fever or tachypnea make necessary corrections. Balance should be calculated every 12-24 hours (special paper forms make this easier).

Daily fluid balance is calculated by adding all the received fluids (both enteral and parenteral ways) and deducting urine output, stool, perspiration and breathing water loss.

You should remember, that perspiration depends on body temperature: in case of normal temperature (36,6°C) patient loses 0,5 ml/kg of water during every hour; 1 degree of temperature elevation adds 0,25 ml/kg to normal value of 0,5 ml/kg.

According to the fluid balance infusion therapy is divided into positive (for dehydrated patients), negative (for overhydrated patients) and “zero” (for patients without balance disorders).

Water deficiency is calculated according to the formula:

$$W_{\text{def}} = (Ht_p - Ht_n) * 0,2 \text{ BW} / Ht_n,$$

W_{def} – water deficiency, l;

Ht_p – hematocrit of the patient, l/l;

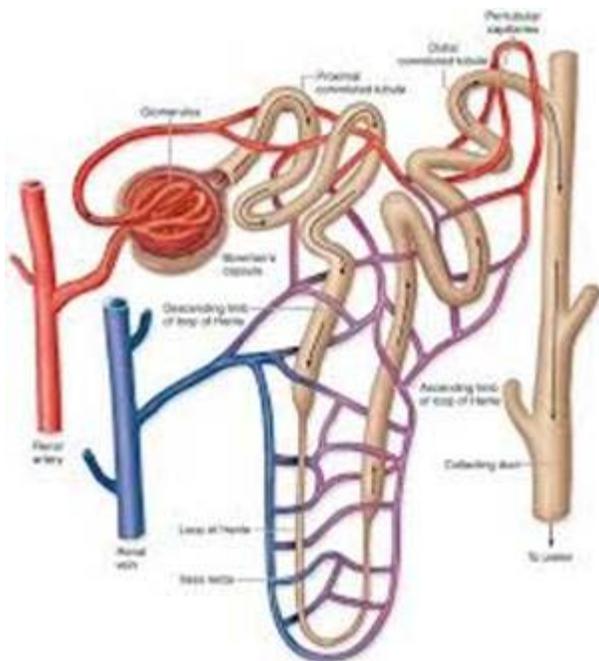
Ht_n - normal hematocrit, l/l;

BW – body weight, kg.

Use crystalloids to treat water deficiency: saline solution, Ringer’s solution, Ringer-lactate solution, electrolytic solutions, 5%, 10, 20% glucose solution. To control potassium concentration (during dehydration this cation is widely lost) prescribe polarizing GIK mixture (pic.9.4), but don’t you ever infuse concentrated potassium solutions quickly – it can cause cardiac arrest (not more than 400 of GIK solution ml per hour).

Acute kidney and hepatic failure

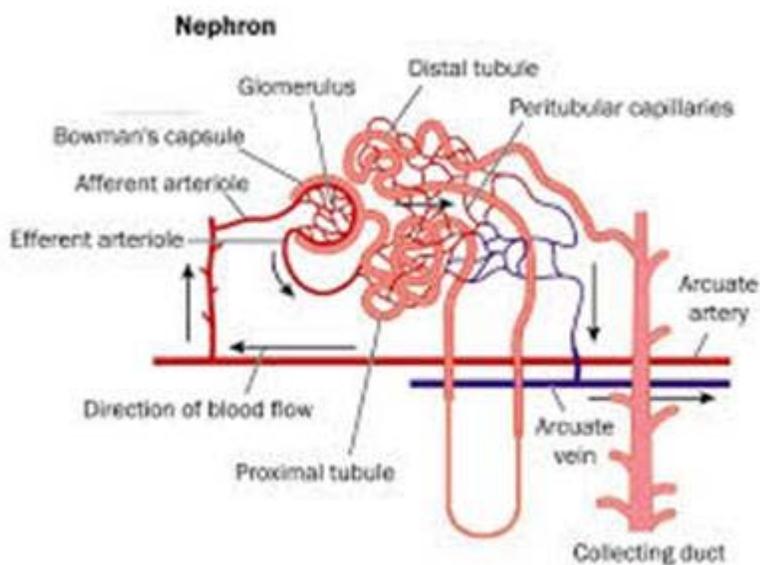
Acute renal failure.



Anatomy and physiology.

Kidneys are the pair organs located in the retroperitoneum (at the level of 12-th rib in the loin region). Their main function is elimination of the metabolic wastes from the organism. They are supplied with blood by paired renal arteries, which are direct arterial branches of abdominal aorta.

Nephron is the basic functional unit of the kidneys. Its structure is quite complicated: it consists of renal corpuscle (Bowman-Shumlanski's capsule and glomerulus), proximal convoluted tubule, loop of Henle, distal convoluted tubule and direct tubule.



Kidneys are exclusively “hard-working” organ. Their total weigh is hardly 0,4% of the total body weight, however they receive 25 % of cardiac output. 10% of total inhaled oxygen are used for their metabolic needs. During the day nearly 150 liters of primary urine are ultrafiltrated out of the blood. Ultrafiltration is possible only when effective filtration pressure is not less than 12 mm Hg. It is defined as a difference between hydrostatic (47 mm Hg), oncotic (25 mm Hg) and intracapsular pressure (10 mm Hg).

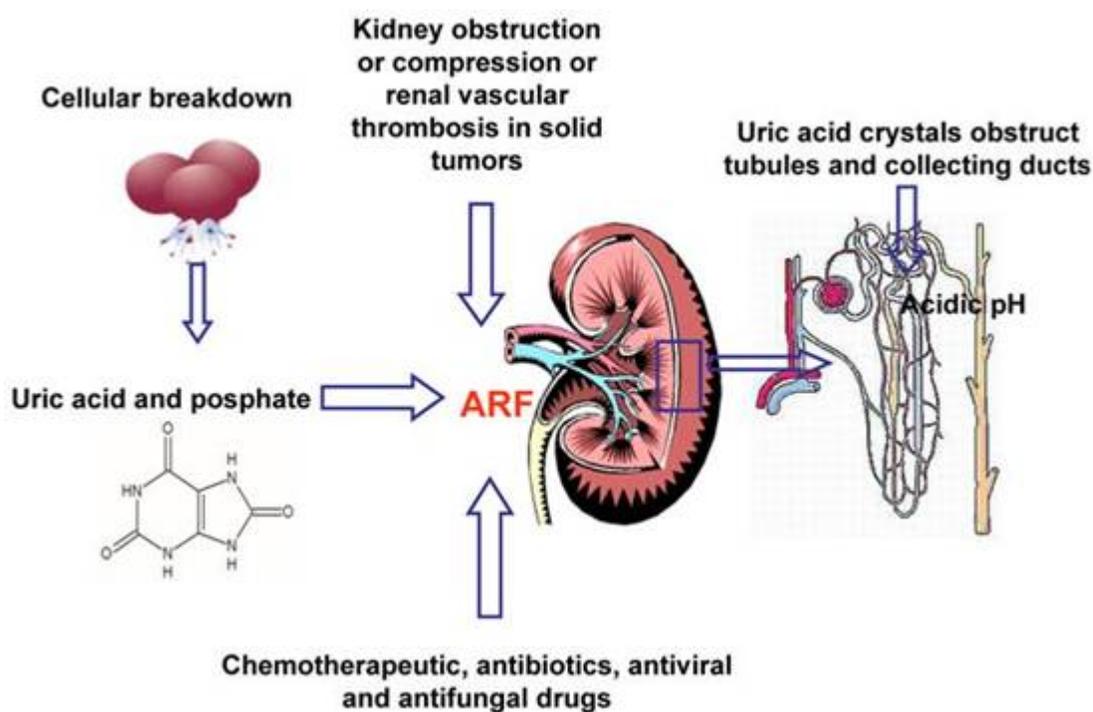
$$EFP = H_p(47) - O_p(25) - I_Cp(10) = 12 \text{ mm Hg}$$

So when hydrostatic pressure (mean arterial pressure) is decreasing or when intracapsular pressure is reaching critical values filtration stops and renal failure appears.

In tubules water, electrolytes and glucose are reabsorbed and metabolic wastes are secreted to the urine. Eventually during one day only 1 % of primary urine is evacuated from the organism in the form of secondary urine with high concentration of toxic substances.

Kidneys participate in haematopoiesis, regulation of fluid balance, electrolytes metabolism and acid-base balance.

Kidneys are also organs of secretion: their parenchyma produces rennin – substance very important for vascular tone regulation.



Etiology and pathogenesis of acute renal failure (acute kidney injury).

Acute kidney injury is a syndrome appearing due to sudden and progressive affection of nephrones, which causes violation of renal function and induces life-threatening homoeostasis disorders.

The reasons of acute kidney injury are divided into 3 groups:

1. Prerenal: pathological conditions, which lower renal blood flow, such as hypovolemia, hypotension, thrombosis, renal artery embolism, renal artery spasm, haemolysis, myolysis (muscle desintegration). Thus the prerenal failure can be connected with the massive blood loss, shock (traumatic, anaphylactic, cardiac), dehydration (burns, pancreatitis, peritonitis, vomiting, diarrhoea), crush-syndrome, transfusion of incompatible blood, acute respiratory failure.

2. Intrinsic: the primary damage is caused to the renal parenchyma by exogenous toxins (alcohol surrogates, acetic acid, ethylene glycol, heavy metal salts), nephrotoxic antibiotics (aminoglycosides), bacterial toxins (in case of sepsis), acute glomerulonephritis, eclampsia.

3. Postrenal: acute renal failure appears due to complications of urine outflow (tumours and calculi of renal pelvis and ureter, prostate, accidental ligation of the ureters during operation).

In 70% of the cases acute renal injury appears as a result of prerenal cause. In stress conditions (massive blood loss, multiple injuries) adrenals intensively produce catecholamines: arterioles of skin, smooth muscles, intestines and kidneys spasm. As you probably remember this helps to save the brain and heart (additional blood for circulating blood volume), however for the rest organs this situation, lasting over 3-4 hours will bring ischemia and even necrosis.

Another mechanism for acute renal failure is connected with the acute vascular insufficiency (collapse, endotoxicosis). Hydrostatic pressure decreases and thus filtration of the blood lowers.

The mechanism of this pathological process is next: hypoperfusion-renal ischemia-hypoxia-coagulation of blood in glomerular vessels-termination of plasma filtration-affectation of tubules membranes- compression of the nephron and capillaries-renal necrosis.

Death of over 75% of nephrons finds its clinical manifestation in acute kidneys injury. All the functions of the kidneys- ultrafiltration, reabsorption, secretion, bioactive substances production – are violated.

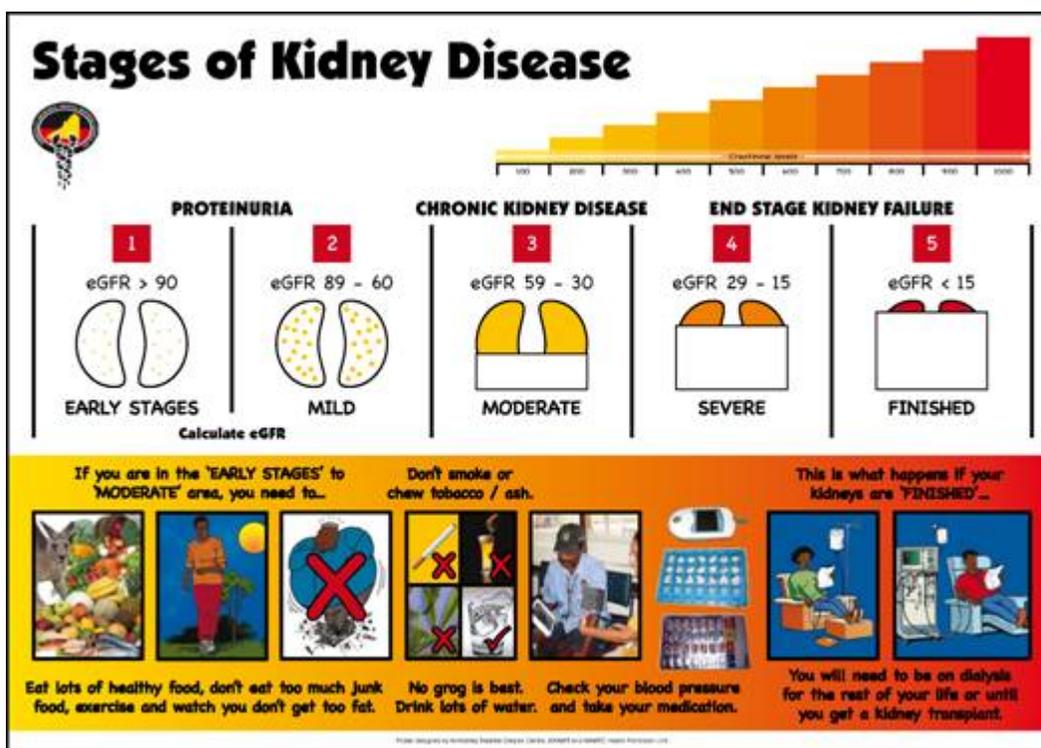
Kidney failure

Acute renal failure (ARF)	A sudden loss of kidney function caused by an illness, an injury, or a toxin that stresses the kidneys (kidney function may recover)
Chronic kidney disease (CKD)	A long and usually slow process where the kidneys lose their ability to function
End-stage renal disease (ESRD)	When the kidneys have completely and permanently shut down

Stages of acute kidneys injury: clinic and treatment.

Clinically in acute kidneys injury we differentiate 5 stages:

I. First stage (initial) is the stage of shock: depends on the initial aggressive agent and duration of its influence. It can last several hours or several days (2-3). Clinic also depends on the causing factor, however first of all you should observe carefully the haemodynamics and urine output, because correct evaluation of patient's condition and proper treatment may prevent the development of next stages.



Warning symptoms are:

- arterial hypotension (systolic blood pressure less than 70 mm Hg if it stays during few hours);
- decrease of urine output;
- hyposthenuria (low specific gravity- less than 1006-1008)

Intensive treatment depends on the primary disease. So you have next possibilities:

1. Hypovolemic shock: profuse bleeding, plasma loss, dehydration.

a. restore the circulating blood volume (blood components transfusions, colloids and crystalloids infusions, glucose solutions) – on time provided correction of circulating blood volume (and you will notice it through normalization of blood pressure, pulse, haemoconcentration indexes and especially central venous pressure) – in most cases means restore of the diuresis.

b. if there is no effect of infusion therapy: to liquidate renal arteries spasm and restore microcirculation give α -adrenoceptor antagonists (droperidol, aminazin, ganglionic blockers, epidural anesthesia); stimulate diuresis with 30% mannitol solution 1 g/kg i/v with the speed 80-100 drops per minute (with 40% glucose solution), 1% furosemid solution (beginning with 2-4 ml every 7-10 minutes and up to 40-50 ml); use additionally spasmolytics (10 ml of 2% euphillin solution i/v with saline). This “triple shot” combined with infusions can liquidate the functional renal failure.

2. Acute vascular insufficiency: anaphylaxis, toxic collapse, orthostatic collapse caused by overdose of ganglionic agents or α -adrenoceptor antagonists; “warm phase” of bacterial shock; reflectory cardiogenic shock.

a. stabilize vascular tone and perfusion pressure with adrenomimetics (epinephrine, dopamine, mezaton i/v, best of all with infusion pump).

b. use steroids, colloids and crystalloids, steroids and then – stimulate the diuresis (described above).

3. Hemolysis (reactions after blood transfusions, hemolytic poisonings, true drowning in sweet water, some snakebites and insect bites, myolysis during crash-syndrome):

a. increase the circulating blood volume providing hemodilution through infusion therapy;

b. increase blood pH infusing 4% solution of sodium bicarbonate (however don't forget to control its level);

c. liquidate spasm of renal arteries;

d. stimulate diuresis.

In case of intense hemolysis, injuries with massive muscle disintegration it would be wise to begin preventive hemodialysis.

4. Renal diseases: normalize hemodynamic indexes, give spasmolytics, stimulate diuresis and use antihypoxic treatment.

5. Postrenal reasons of acute kidneys injury demands immediate consultation of

urologist and decision about operative liquidation of urine flow obstruction (insertion of catheters into the bladder, ureters; epicystostomy, prostatectomy, lithotomy, etc.). Do not stimulate urine output with diuretics until the reason of obturation is not removed!!!

Lack of diuretic effects after stimulation states severe organic injury of nephrons and you should make a diagnosis acute renal failure, stage of oligoanuria.

II. Second stage- oligoanuria- lasts from several days to 3 weeks (duration depends on the degree of the damage and regeneration ability of the nephrons). It can manifest as oliguria (urine output less than 500 ml/day) or anuria (urine output less than 50 ml/day).

The severity of this stage is determined with its symptoms:

1. Overhydration: quite often it is caused by iatrogenic reasons – doctors try to “overfill” patient with the water to stimulate diuresis (outdated and dangerous conception!) or simply calculate the daily fluid balance in a wrong way (too “positive” balance). In addition this phase is characterized with intensive catabolism (tissues destruction) and thus excessive endogenous water production (up to 1500 ml/day). Clinical findings: increase of the body weight, circulating blood volume, blood pressure, central venous pressure, peripheral oedema, possibly pulmonary oedema.

2. Electrolytic disorders: hyperkalemia, hypermagnesemia, hypocalcemia. Clinical findings: depression, somnolence, hyporeflexia, respiratory and cardiac disorders.

3. Metabolic acidosis is caused by accumulation of hydrogen ions due to disorders of their renal secretion. Clinical findings: noisy rapid deep breathing (compensatory Kussmaul breathing), vomiting, evident haemodynamic disorders.

4. Uraemic intoxication. Clinical findings: consciousness disorders (up to coma), ammonia breath odour, uraemic polyserositis (pleuritis, pericarditis), ulceration of oesophageal mucous membrane and gastric mucous membrane, diarrhoea.

5. Disorders of synthetic renal function. Clinical findings: anaemia due to erythropoietin deficiency and hypertension (connected with renin-angiotensin disorders).



Intensive treatment.

Your therapeutic tactic will differ a lot in comparing with the previous phase.

1. Fight with overhydration. First of all medical staff should control carefully body weight gain: ideally correct treatment excludes increase of the weight. Everyday patient loses 400-500 ml of water with respiration. Don't forget to count water loss with vomiting and diarrhea. Infusion therapy should not exceed this total fluid loss and the only solutions you should use are normal saline and 20-40% glucose solutions (with insulin).

2. Control and treat electrolyte disorders: prescribe calcium chloride or calcium gluconate solutions (40-50 ml of 10% solution intravenously). Calcium acts as potassium and magnesium antagonist and thus lowers their plasma concentrations.

3. Treat metabolic acidosis with 4% sodium bicarbonate solution (up to 400-500 ml per day with acid-base control tests). Normalization of blood pH also normalizes potassium level.

4. Catabolism inhibition: to prevent tissues destruction use anabolic hormones (Nerobol, Retabolil) which prevent muscle disintegration and breakdown of the proteins. This helps to reduce endogenous water production and toxins production. For this purpose we are also using concentrated glucose solutions.

5. To eliminate the wastes from the body there are different methods: enterosorbption (enterodez, polisorb, activated charcoal, etc.), intestinal lavage (cleansing enemas 4-6 times a day), extracorporeal detoxification methods (hemodialysis, plasmapheresis, hemosorbption), peritoneal dialysis.

6. Symptomatic treatment: prescribe hypotensive medicines if necessary, preparations for cardiac support, transfuse washed red blood cells. Prevent infectious complications.

Don't forget, that in case of acute kidneys injury withdrawal of many drugs is delayed or interrupted and thus accumulation effects are possible!



Hemodialysis is the process of extracorporeal waste products removal with the help of “artificial kidney”. The principle of its work is quite simple: special pump pushes the blood through the tubes to the dialyzer. Dialyzer itself is a system of capillaries, made of semipermeable membrane (kuprofan, cellophane), which are “immersed” into the dializing solution (something chemically very close to plasma). When blood passes through the capillaries dialysis, osmosis and ultrafiltration take place. During this process toxic wastes (creatinine, urea, uric acid, phosphates, potassium and hydrogen ions) and excessive water move to the dializing solution. Simultaneously ions of sodium and calcium move from the dializing solution into the blood.

According to the method of blood taking and blood return dialysis is conducted

through arterial-venous access or venous-venous access. Aseptic conditions are obligatory; anticoagulants are used in most cases (heparin solution). Average blood speed of 200-250 ml/minute determines duration of dialysis up to 4-5 hours.

Absolute indications to haemodialysis are:

- overhydration (CVP over 15 cm H₂O);
- hyperkalaemia (potassium more than 7 mmol/l);
- creatinine more than 0,3 mmol/l;
- decompensated metabolic acidosis (pH<7,2);
- daily growth of urea > 5 mmol/l, urea level over 35 mmol/l.

Contraindications to haemodialysis:

- unstable haemodynamics (blood pressure less than 90 mm Hg);
- haemorrhagic syndrome;
- decompensated cardiac and respiratory insufficiencies;
- CNS damages (stroke, intracranial haematoma).

III. Third stage is a stage of diuretic function restoring. It begins when the daily urine output becomes more than 500 ml and lasts for 3-5 days. Due to regeneration of the glomeruli blood filtration is gradually restored. Tubules epithelium regenerates a bit slower and thus water reabsorption is still inadequate. Diuresis grows every day and at the end of this phase reaches 1500-2000 ml per day. However urine has low specific gravity and contains large amount of red blood cells and protein. Hyperkalaemia and uraemic intoxication are still dangerous, because wastes elimination is not adequate.

So the intensive treatment is quite similar to the previous stage. You can change the volume of infusions (but again – control the fluid daily balance). When daily urine output will reach physiologic point of 2-3 liters next stage starts.

IV. Fourth stage is a diuretic stage (polyuria). It lasts up to 2 weeks. Daily diuresis increase is 800-1000 ml and daily urine output reaches 7-9 liters. Biochemical blood tests get to the norm (urea, creatinine). Due to excessive diuresis dehydration and electrolyte imbalance develop: potassium and magnesium ions are lost in large amounts and this can bring life-threatening complications.

Intensive treatment is changed: now your task is to restore the circulating blood volume and lost electrolytes reserves. Correction is made according to the laboratory tests and special formulas.

Gradually concentrating ability of kidneys is returned and diuresis is normalized (specific gravity is normalized, electrolytes are reabsorbed).

V. The fifth stage is a stage of recovery. It takes few months or few years to gain complete recovery and for some patients everything will end with a chronic renal failure. In this stage your task is to prescribe symptomatic treatment, proper diet and resort therapy.

Control tests.

1. What is the normal renal blood flow?
 - A. 5-10% of circulating blood volume
 - B. 500 ml/ minute
 - C. $\frac{1}{4}$ of cardiac output
 - D. depends on renal activity
 - E. 40-50% of cardiac output.
2. During the first stage of acute renal failure you should:
 - A. begin hemodialysis;
 - B. provide intensive therapy of shock, normalize microcirculation and stimulate diuresis;
 - C. stop infusions due to overhydration risk;
 - D. perform paranephral block;
 - E. prescribe high doses of furosemide
3. What is a contraindication to hemodialysis?
 - A. potassium level more than 7,0 mmol/l
 - B. CVP>13 cm H₂O
 - C. pH<7,2;
 - D. acute renal failure of III or IV stages;
 - E. prolonged compression of the kidneys
4. Name the symptom atypical for uraemic intoxication:
 - A. metabolic alkalosis
 - B. overhydration
 - C. azotemia;
 - D. anemia;

E. hypocalcaemia

5. In case of III stage of acute renal failure you should:

A. obligatory prescribe furosemide

B. give infusions in the rate 5-10 ml/kg

C. treat respiratory alkalosis

D. first of all begin with the dialysis

E. avoid diuretics

6. The forth stage of acute renal failure:

A. is terminal

B. shows overhydration and hyperkalemia;

C. is characterized with potassium ions loss;

D. needs hemodialysis;

E. shows metabolic acidosis and compensated dyspnea.

Task 1.

What medicines should not be used for treatment of patient with the II stage of acute renal failure?

a. mannitol; b. concentrated glucose solutions; c. heparin; d. plasma; e. panangin; f. normal saline; g. calcium chloride; h. enterodez; i. rheopolyglucin j. magnesium chloride; k.nerobol.

Task 2.

Here are the clinical and laboratory findings of the patient, who is suffering from the lack of urine for 5 days:

Creatinine 0,64 mmol/l; potassium 7,1 mmol/l; urea 41 mmol/l; CVP 14,5 cm H₂O; blood pressure 170/110 mm Hg; heart rate 96/minute.

What treatment should be prescribed?

Task 3.

One week after restore of the diuresis patient with acute renal failure shows such laboratory results: hematocrit 0,5 l/l, plasma sodium 148 mmol/l, plasma potassium 3 mmol/l, chlorine 98 mmol/l. What is the reason for these changes? What changes should be done in the prescriptions?

Violations of homeostasis and their correction.

The importance of the water to the organism.

Life on earth was born in the water environment. Water is a universal solvent for all the biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the atrial walls, baroreceptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals reveal into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution). This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.

Importance of osmolarity for homoeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hyposmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.

9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobinconcentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (N_{ap} - 140) / 140,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, 0,6*BW volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 \text{ BW},$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists

(spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K- potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

$0,6 * \text{BW}$ – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 \text{ BW},$$

Na- sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

0,2*BW – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonicoverhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 \text{ BW},$$

Cl_d- chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

0,2*BW – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chlorine solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis).

You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

$0,6 * BW$ – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d – calcium deficiency, mmol

Ca_p – plasma calcium concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorbtion.

Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO₂. Normally pCO₂ of arterial blood is 36-44 mm Hg. Hypercapnia (pCO₂ increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO₂ less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H⁺ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is 0±1,5. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolonged alkalosis brings decrease of BE due to compensatory retention of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patient breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H⁺ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H⁺ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patient, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patient, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patient, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patient, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patient, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration 88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

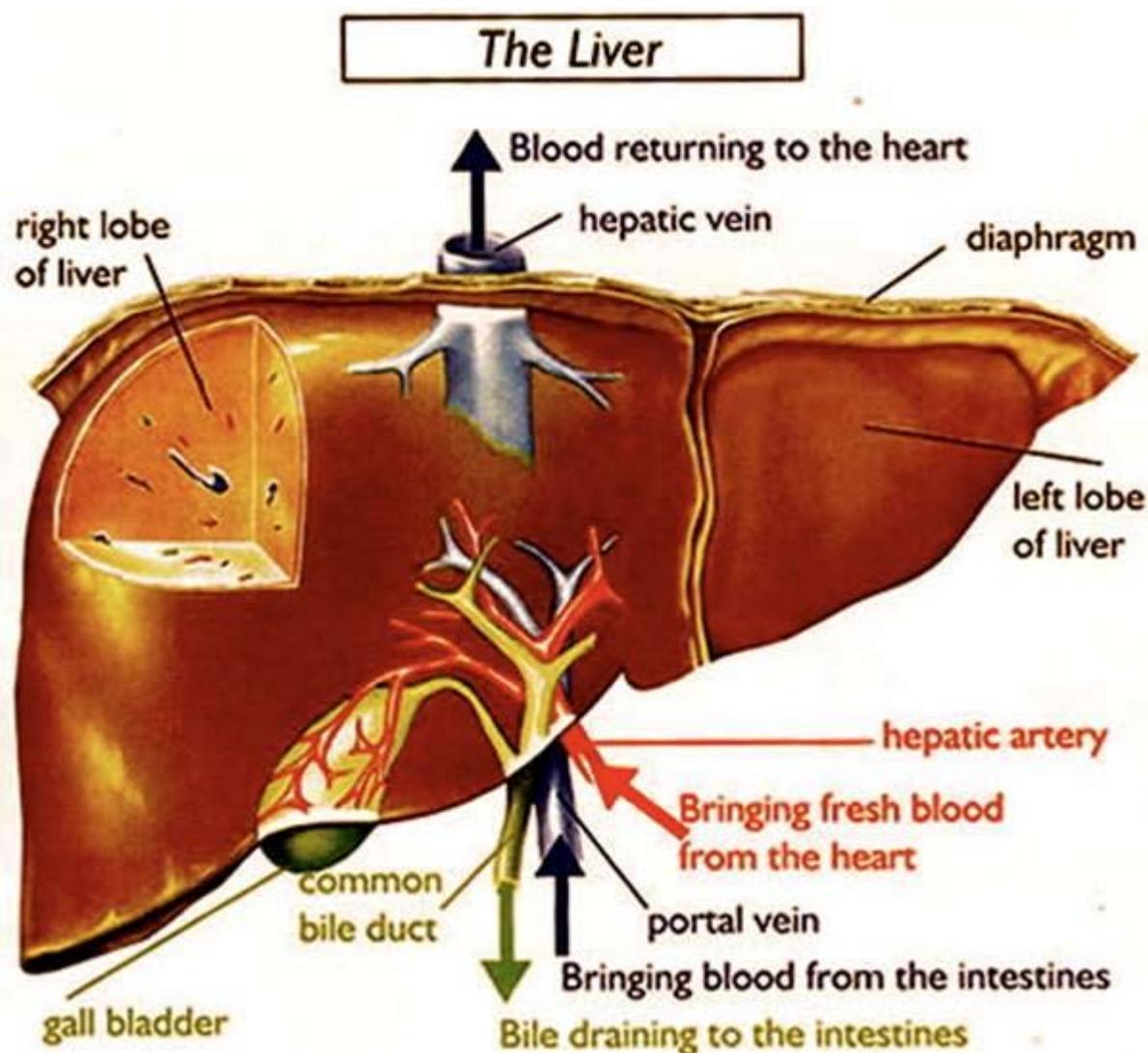
Task 7.

Patient, the patient of 50 with body weight 75 kg, was transported to the admission unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow breathing. Blood tests show: pH 7,18, pCO_{2a} 78 mm Hg, pO_{2A} – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patient, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, pCO_{2a} 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

Acute liver failure.



Anatomy and physiology.

Liver is the largest internal organ, unpaired triangular gland located in the right upper quadrant of the abdominal cavity, below the diaphragm. It is extremely important to the organism because of the variety of activities it performs. Liver is a digestive gland (produces and excretes bile necessary for lipids consumption), a detoxification centre (microsomal oxidation allows detoxification of exogenous and endogenous toxic substances) and a synthetic center, where proteins, lipids and carbohydrates are metabolized. Liver is also an organ of haematopoiesis and a blood reservoir. Additionally it helps to control acid-base balance.

To provide its metabolic needs with the oxygen organism gives nearly 25% of total consumed oxygen; in case of severe intoxication this number growth up to 40% of the total oxygen. Blood flow of the liver is for 25% provided by hepatic arteries and for 80% by the portal vein. Thus the blood liver receives is poorly oxygenated and any hypoxic condition

will bring oxygenation disorders first of all to the tissues of the liver. This evolutionary resulted in unique regeneration abilities of the liver: death of 70% of cells will end up with a failure; however after a certain adaptation period hepatic tissues will restore their quantity and quality.

Cause	Examples	Comment
Drugs and Toxins	<ul style="list-style-type: none"> ❖ Acetaminophen ❖ Amanita phalloides ❖ Isoniazid ❖ Halothane 	Acetaminophen poisoning is the overall leading cause of ALF in the US
Viral Infection	<ul style="list-style-type: none"> ❖ Hepatitis A ❖ Hepatitis B (+/-D) ❖ Hepatitis E ❖ Herpes simplex virus 	Hepatitis C is a very rare cause of ALF
Vascular problems	<ul style="list-style-type: none"> ❖ Shock ❖ Heat stroke ❖ Tumor infiltrating the liver 	Most often seen after cardiac arrest, major blood loss, or iatrogenic ligation of the major blood vessels feeding the liver
Metabolic/Miscellaneous	<ul style="list-style-type: none"> ❖ Wilson Disease ❖ Acute fatty liver of pregnancy ❖ Alpha-1 antitrypsin deficiency ❖ Autoimmune hepatitis 	Family screening is appropriate for many metabolic/genetic causes of ALF
Indeterminate	<ul style="list-style-type: none"> ❖ Unknown 	Approximately 15%-20% of adult ALF cases, and up to 50% of ALF in children, cannot be attributed to a specific cause.

Acute liver failure: aetiology and pathogenesis.

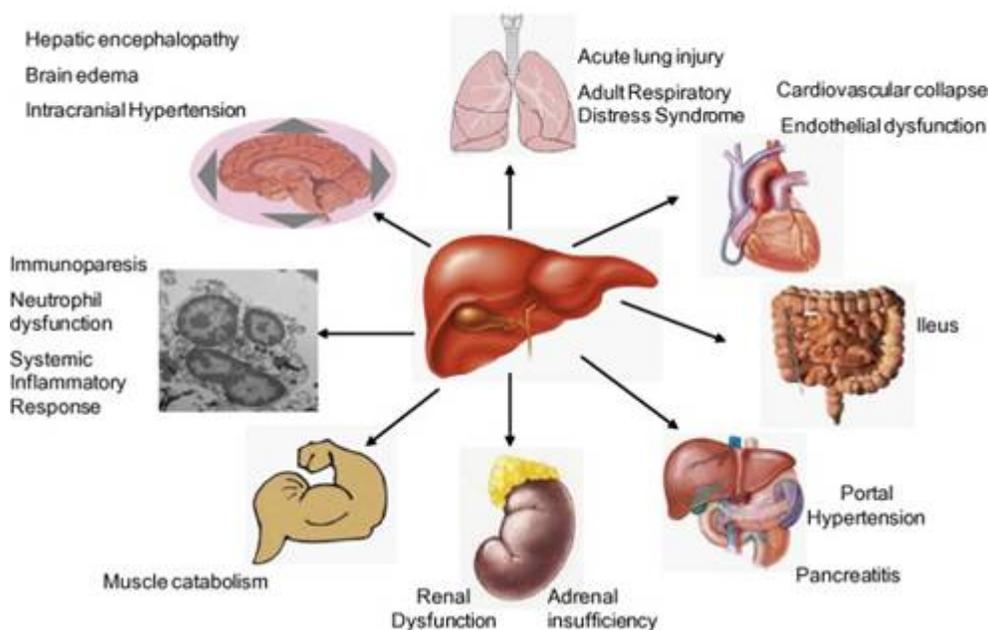
Acute liver failure is a state of hepatic cells dysfunction, caused by unknown earlier liver disease, resulting in general intoxication, coagulation violations, neurological and mental disorders. Its etiology is usually connected with: viral hepatitis (hepatitis B virus, hepatitis A virus), poisonings (mushrooms, dichlorethane, phosphorus, carbon tetrachloride, arsenic), eclampsia, burn disease, anaesthetic gas, antibiotics, sulfanilamides, massive bacterial pneumonia, cirrhosis, hepatic tumours and metastasis.

Advanced liver failure manifests in coma. Hepatic coma is divided into endogenous (“destructive”, hepatocellular) and exogenous (“shunt”, porto-caval). Toxic damage of 70% of liver cells will cause endogenous coma. In case of liver cirrhosis high portal pressure antagonizes portal blood flow and thus most of the blood moves to the caval venous system and is not detoxified – exogenous coma appears. Clinically we usually observe mixed comas.

Central nervous system in case of lever failure is affected in various ways. Ammonia

encephalopathy appears because of violations of uric acid synthesis (it is made from ammonia and without this process ammonia concentration increases several times). Food rich with proteins stimulates ammonia encephalopathy onset, as well as gastrointestinal bleedings, hypnotic medicines and opiates, alcohol, surgeries, infections and metabolic alkalosis. In the CNS tissues false mediators like octopamine, amino acids and their toxic metabolites are accumulated. On the background of hypoproteinemia interstitial edema appears and this brings respiratory hypoxia of tissues. At the same time violated synthesis of enzymes, disordered metabolism of carbohydrates and lipids, metabolic alkalosis with hypokalaemia just advance the encephalopathy.

Systemic Manifestations of Acute Liver Failure



Clinical findings in case of liver failure.

Liver failure has several forms:

1. Excretory form (disorders are mostly connected with bile production, jaundice is the main characteristic).
2. Vascular form (clinically portal hypertension is the most noticeable).
3. Hepatocellular form (most clinical signs are caused by disorders of synthetic metabolism in liver cells).

According to the duration of the process we define acute and chronic liver failure, according to the compensation level - compensated, subcompensated and decompensated failure.

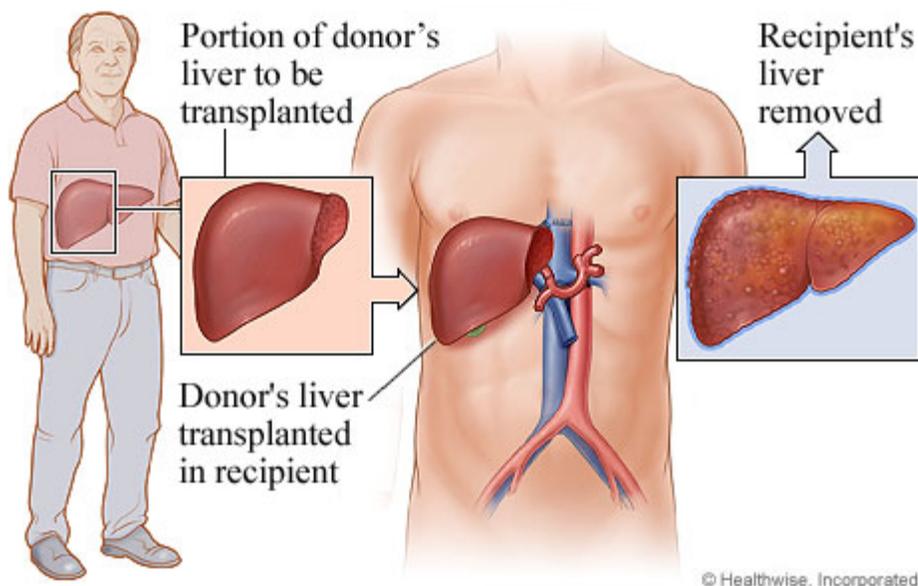
Central nervous system is damaged gradually: it begins with precoma and progresses into moderate and deep coma.

Clinical findings:

- skin: jaundice, vascular spiders, “hepatic” palm, extension of small superficial face vessels;
- fever;
- hepatic breath odour, hepatic smell of sweat and urine (this smell occurs due to transformation of methionine into methyl mercaptan);
- digestion disorders (nausea, hiccups, inappetence, smooth red tongue, abdominal pain, meteorism, defecation disorders);
- obstructive and diffuse respiration disorders – hypoxic hypoxia;
- cardiovascular disorders (arterial hypotension, tachycardia, extrasystoles);
- haemorrhagic syndrome, anemia (due to interruption of coagulation factors synthesis and bleeding of gastric or oesophageal erosions and ulcers);
- frequent additional complications: renal failure, hepatorenal syndrome is prognostically dangerous.

If liver failure progresses CNS damage deepens and you can clinically observe: weakness, headache, sluggishness, apathy, inversion of sleep and awakening. Disorientation develops gradually, there is possibility of excitement periods and cramps. You can also find overactive tendon reflexes, foot clonus, Babinski’s sign. One of the most significant symptoms is flapping: trepidation of limbs and face, especially of hands in prone position (arms extended). In case of deepest coma you will see dilated pupils, eyeballs are fixed, tendon reflexes are absent.

Progressive and quick decrease of liver size is a prognostically bad sign. However when the disease is chronic and fibrous changes took place this symptom is not noticed (liver stays enlarged).



Intensive treatment.

The basic principle of liver failure treatment is etiologically aimed therapy: you should treat the reason of the failure. Two other important components are prevention and treatment of liver failure complications during 10-14 days necessary for the regeneration of the hepatocytes.

Necessary treatment measures:

1. Patient should follow strictly bed regiment in isolated ward. Medical staff should follow aseptic and antiseptic rules.

2. Eliminate animal fats and proteins from the patient's diet to prevent encephalopathy.

3. Liquidate hepatotoxic factors (hypoxia, hypovolemia, haemorrhagic syndrome, intoxication):

- provide oxygen supply (nasal catheter, face mask with the flow 3-4 l/min); sometimes hyperbaric oxygenation and even intestinal oxygenation (0,2-0,3 ml/kg/min) are possible;

- to increase hepatic blood flow restore the circulating blood volume, improve rheological properties of the blood, restore the peristalsis. To achieve this you should: infuse crystalloids and glucose solutions, spasmolytics, 2 % eufhyllin solution (20-30 ml/day). 10% albumin solution (200-300 ml) and mannitol solution (1 g/kg) increase oncotic blood pressure and thus help to prevent interstitial oedema of the liver;

- prevent ulceration of stomach and gastrointestinal bleeding by prescription of famotidine or omeprazole (40 mg twice a day); oesophageal bleedings are stopped with Blackmore probe;

- if you suspect stagnated blood in the intestines – remove it, because otherwise intoxication will get more intense;

- use only “fresh” blood stabilized with heparin for transfusions.

Prevent and treat intoxication with:

- intestinal lavage and enemas;

- antibiotics which are not toxic to the liver (for example ampicillin 1,0 every 4 hours);

- extracorporeal blood detoxification (plasmapheresis, hemosorbition or hemodialysis; usage of artificial liver or artificial spleen);

- prescribe antagonists of ammonium (40-50 ml of 1% glutamic acid solution with glucose 3 times a day; 2,0 of alfa-arginine solution i/v every 8 hours).

4. To stimulate energetic metabolism in hepatocytes prescribe concentrated glucose solutions (10-20% solutions, up to 5g/kg/day). This will also prevent proteins breakdown and thus wastes accumulation.

5. To stabilize the membranes of the hepatocytes prescribe steroids (10-15 mg/kg of hydrocortisone).

6. To stabilize the energetic exchange and stimulate transportation of the lipids prescribe choline chloride (10 ml of 10% solution with 200 ml of glucose solution after previous atropine admission, twice a day).

7. Additionally prescribe vitamins (ascorbic acid, B₁, B₂, B₆, K, E, B₁₂, folic and nicotinic acids in doses 2-3 times higher than daily needs), cardiac glycosides, panangin, antioxidants (cytochrome c, sodium gamma-hydroxybutyrate).

8. Symptomatic treatment helps to stabilize homoeostasis if not to treat failure itself: if necessary use anticonvulsive medicines, antipyretics, etc.

Control tests.

1. The reason of exogenous liver failure is:

A. acute exogenous poisoning

B. exogenous shock

C. hepatitis B

D. hepatic cirrhosis

E. hyperbilirubinemia

2. The reason of endogenous liver failure is:

- A. oesophageal bleeding
- B. acute carbon monoxide poisoning
- C. infectious shock
- D. damage of hepatocytes
- E. endocrinological diseases

Task 1.

What is typical for exogenous (a.) and endogenous (b.) liver failure?

a. unconsciousness; b. rapid noisy breathing; c. bleeding varicose veins of oesophagus; d. ascites; e. anaemia f. elevated transaminases; g. acute exogenous poisonings; h. viral hepatitis i. alcohol abuse.

Task 2.

Patience, the patient with liver failure, receives:

a. enteral nutrition reached with proteins; b. oxygenation; c. albumin infusion; d. blood transfusion; e. sodium thiopental for cramps treatment f. steroids; g. vitamins h. gentamicin i. diuretics.

Acute poisonings.



Acute poisoning is a chemical injury, which occurs when chemical substance gets to the organism and violates its vital functions. If the substance is aggressive enough and proper treatment is not provided on time poisoning will bring death.

Although there are over 500 toxic substances which may cause acute poisoning, clinical picture is made up of quite similar syndromes. Proper diagnostics of these

syndromes allows avoiding life-threatening complications and gets the chance to make correct preliminary conclusions about the nature of poisoning.



8.1 Main clinical syndromes of poisonings.

Affection of central nervous system manifests as excitation or depression of patient's mental activity. Depression of CNS has different stages: confusion, stupor, spoor and toxic coma. One of the coma's deepness criteria is reaction of the patient to the painful stimuli (you can also check reaction to smell of ammonium chloride). Lack of reaction is a sign of coma. Don't live such patient without observation, because his condition is severe and at any moment life-threatening complications may appear: those patients have tendency to bradypnoea up to complete respiratory arrest. Also you should not forget that decreased tone of soft palate and tongue in a supine position will violate patency of the airways and patient can die of asphyxia. In addition comatose condition influences reflexes and lack of swallowing can lead to entrance of saline and gastric contents into the airways (and thus to development of aspiration pneumonia).

Usually CNS depression is caused by excessive alcohol consumption or admission of its surrogates, overdose of neuroleptics, sleeping pills, drugs, sedative medicines, antidepressants and carbon monoxide poisoning. You can remind intensive treatment of comatose patients in the chapter 5.

Some poisoning are followed with acute intoxication psychosis (mental disorders, hallucinations, time and space disorientation, inadequate behavior). This clinical picture you can observe in case of atropine poisoning (and also atropine-like agents: tincture of dope, henbane, amanita) or cocaine, tubazid, antihistaminic drugs and organophosphates poisoning.

In case of psychosis you will have to immobilize patient in the bed for his own good and safety (to avoid injuries both patient's and those of the stuff). You will also have to do this in order to maintain i/v lines for antidotes and sedative infusions (aggressive patient will try to remove everything he or she considers irritating). Constantly observe the

patient's vital functions.

Toxic affection of the respiratory system can progress as the violation of:

a. external respiration - neurogenic form, aspirations and obstructions of the airways which bring hypoxic hypoxia;

b. hemoglobin's function – aniline and nitrobenzene create methemoglobin, carbon monoxide connected with hemoglobin creates carboxyhemoglobin and neither the first nor the second is capable of normal oxygen transportation; heavy metal, organic acids and arsenic poisonings lead to destruction of the red blood cells and emission of the free hemoglobin into the plasma;

c. oxygen transportation due to the decrease of circulating blood volume – exotoxic shock;

d. cellular respiration – tissue hypoxia occurs when cytochromes are blocked with toxins like cyanides.

Practically all severe poisonings earlier or later lead to hypoxia, because they violate oxygen supply, transportation and consumption.

Your immediate therapy actions in this situation will be:

- to assess of respiratory system (described above);
- to provide of airways patency (cleaning of the oral cavity, aspiration of the saline and gastric contents, conicotomy if necessary, etc.);
- to begin oxygen supply (face mask, nasal catheter);
- to start artificial ventilation if necessary;
- to prescribe antidotes if they are available (methylene-blue in case of nitro compounds poisonings, unithiolum in case of heavy metals and arsenic poisonings, cytochrome c in case of tissue hypoxia);
- to start hyperbaric oxygenation in case of carbon monoxide poisoning;
- to begin infusion therapy in order to stabilize the hemodynamics;
- to start general detoxification;
- to prescribe symptomatic treatment and provide prevention of the complications (for example prescription of antibiotics).

Affection of the cardiovascular system manifests as inability of the heart and

vascular bed to provide adequate blood supply of the organs, which leads to metabolic disorders and in the worst case to death. Severe toxic damage of cardiovascular system brings acute cardiovascular failure: primary toxic collapse, exotoxic shock, secondary somatogenic collapse.

Primary toxic collapse appears in case of massive poison admission, when compensatory mechanisms are not quick enough to resist the chemical aggression. Immediately or minutes after poisoning patients begin to suffer from reduced cardiac output and thus from decreased blood flow in the tissues. Peripheral pulse is weak or absent, blood pressure critically lowers and cardiac arrest can appear. In most cases of primary toxic collapse ambulance is not able to save the life of the patient due to the fulminant development of life-threatening complication. However you should remember that such collapse occurs only in 5 % of the cases.

Exotoxic shock is the reason of death for 70 % of poisoning victims. Violations of hemodynamics on one hand are caused by direct heart and vessels damage and on the other hand by compensatory sympathetic and adrenal reactions. On the background of CNS and gastrointestinal system violations you will observe disorders of systemic hemodynamics and microcirculation: arrhythmias, decrease of blood pressure, central venous pressure, cardiac output and diuresis. The peripheral vascular tone changes: toxins induce spasm or dilation of arterioles with the ischemia of one and hyperemia of other tissues. Depending on the body reaction on intensive treatment shock can be compensated, decompensated reversible and decompensated irreversible.

In case of toxic shock you should:

- get an i/v line (preferably several, including central venous access);
- start infusions of colloids (albumin, rheopolyglucin, hydroxyethylstarch solutions) and crystalloids (saline, glucose solutions, polarizing solution) in order to normalize blood pressure, heart rate and diuresis; sometimes infusion dose is up to 100-150 ml/kg, (7-10 l/day);
- constantly control patient's condition: monitor the heart action, blood pressure and central venous pressure;
- provide antidote treatment and detoxification; remember that extracorporeal detoxification is possible only after the stabilization of the hemodynamics (systolic blood pressure >90 mm Hg).

Secondary somatogenic collapse is the reason of death in 25% of the cases. It can occur few days after poisoning, when the toxin is already eliminated from the body, however the tissue changes (in the lungs, liver, kidneys, heart) are irreversible. Necessary treatment: hemodynamics stabilization, improvement of microcirculation, intensive therapy of functional disorders and organic changes (artificial lung

ventilation, hemodialysis, cardiac support, etc.).

Toxic affection of gastrointestinal tract.

Usually poisoning provokes usual protective reactions: nausea, vomiting, diarrhoea. Chemically aggressive agents can cause “corrosive” effects: concentrated acids and bases can burn the mucous membrane of oral cavity, oesophagus and stomach (vomit is coloured with blood). Profuse vomiting and diarrhoea bring dehydration, electrolytes loss and acid-base imbalance. Especially rapid those complications appear in children.

After recovery patients with chemical burns of gastrointestinal mucous membranes may suffer from cicatrization and stenosis of digestive tract.

Be aware of the fact, that intensive usage of narcotic pain killers and sleeping pills can lead to inhibition of peristalsis and thus to constipation and slow elimination of toxins.

Immediate aid actions:

- clean the stomach. If the patient is conscious stimulate vomiting with the pressing on the root of the tongue or with 2-4 liters of slightly salted fluid. Don't you ever do this in case of chemical burns!
- if you have the skills and possibility insert the stomach probe and evacuate gastric contents with the help of 10-15 liters of water; if necessary give also antidotes through the probe; you can also use probes with several channels;
- after the gastric lavage in order to bind toxins use enterosorbents (activated charcoal for example);
- stimulate stool with saline laxatives (150-200 ml of 33% magnesium sulfate solution) in order to remove connected with the toxin sorbent from the intestines;
- cleansing enemas also help to eliminate toxins from the body.

Toxic affection of liver and kidneys.

This syndrome is caused by primary toxic damage of the liver and kidneys (nephrotoxic and hepatotoxic poisons) or by the secondary disorders of their functions due to violations of blood flow and oxygenation.

Liver is the main detoxification and biotransformation center of the organism, so it takes the “main blow” during intoxication. Intensive detoxification increases metabolic activity and oxygen consumption of the liver several times. Hepatocytes become very

sensitive to hypoxia.

Light forms of toxic and hypoxic affections can develop without clinical manifestation. However they will be noticeable in laboratory tests (elevation of transaminases, bilirubin, phosphates). Severe poisoning will bring to toxic hepatitis and even hepatic coma.

Among the hepatotoxic substances are: heavy metals salts, dichloroethane, ethylene glycol, deadly amanita toxins.

To protect the liver you should:

- eliminate the toxic substance from the gastrointestinal tract;
- give antidotes if they exist (unithiol for heavy metals salts, lipoic acid for deadly amanita);
- prescribe cleansing enemas 2-4 times a day (to prevent intoxication with the wastes accumulated in the intestine);
- use extracorporeal detoxification (hemosorbition, plasmapheresis, artificial liver);
- provide adequate oxygenation and blood supply of the liver;
- prescribe symptomatic treatment.

Kidneys are very important for the elimination of the poisons circulating in the blood. So in many cases they are also the “target” of the toxin. They can be damaged primary (poison affect their tissues directly) and secondary though the violations of vital functions (for example hemodynamics in case of exotoxic shock). Their condition you can control with the help of urine output per hour, which normally is not less than 0,5 ml/kg.

To prevent the renal failure you should:

- eliminate the poison as soon as possible (gastric lavage and enemas for gastrointestinal tract; hemodialysis, hemosorbition, plasmapheresis for blood);
- give antidotes if they exist (unithiol for heavy metals salts, sodium bicarbonate for hemolytic poisons, ethylic alcohol for ethylene glycol and methanol);
- treat disorders of hemodynamics (therapy against exotoxic shock);
- stimulate the urine output with the diuretics on the background of previous rehydration: this will allow you to eliminate diluted in the plasma toxins faster and to prevent renal failure; kidney is an organ which functions normally only if works intense;

Clinical observations tell us, that incredibly massive infusions (10-20-30 l/day)

with diuresis stimulation really help patient to dilute and eliminate the toxin without kidneys damage.

In case of acute renal failure you should treat the patient according to the principles described in chapter 6.

Ethylic alcohol poisoning.

This type of poisoning appears in case of excessive alcohol consumption. It is one of the most common poisonings, as well as one of the lightest and prognostically the most favourable (organic damage is rare). However combined with comorbidities and complications it becomes one of the first reasons of death in toxicology, so don't underestimate it.

Alcohol poisoning, unlike drunkenness, has the characteristic signs of intoxication: vomiting, inhibition of CNS, disorders of cardiovascular system and breathing. Patients usually are in comatose condition. Pay attention to their appearance: clothes are untidy, you can notice signs of involuntary urination or defecation. There is alcohol breathing odour. Skin of the face is hyperaemic and dry. Cyanosis is a sign of respiratory insufficiency, grey shade of skin is a symptom of cardiac disorders. Wet skin might be the symptom of hypoglycaemic coma, hypercapnia or organophosphate poisoning, which are "covered" with the obvious clinic of alcohol poisoning. In case of moderate coma vital functions are usually not involved. Pupils are narrowed or dilated, photoreaction is preserved. Objective criterion of alcohol poisoning is alcohol concentration of the blood:

- less than 1,5‰ – light inebriation
- 1,5‰-3,5‰ – moderate inebriation
- 3,5‰ and more – severe inebriation

Lethal concentration of alcohol is 5-6‰.

The most common complications of alcohol poisoning are next:

- obstruction of the airways with the tongue, soft palate or biological fluids (vomit, saline, sputum, blood) in supine position;
- regurgitation of the gastric contents and development of aspiration pneumonia; lethality is nearly 70%;
- head traumas with brain injuries: patients usually fell and hurt themselves; the problem with such injuries is that the clinic of hematoma (subdural, epidural, intracerebral) is quite often "covered" with alcohol intoxication and this is why you should always remember about the differential diagnostics. To make a correct diagnosis in case of coma you should check the specific symptoms such

as anisocoria (there is no poisoning which causes pupil's asymmetry!), signs of head injury (scratches, bruises, skull deformations, oto-liquorrhea and nasal liquorrhea, nasal and ear bleeding), asymmetric tendon reflexes and muscle tone, disparity between the amount of alcohol and deepness of coma, prolonged unconsciousness (alcohol coma even without proper treatment lasts only 3-4 hours);

- other traumatic injuries (rib fractures which violate external breathing, spleen or liver ruptures with haemorrhagic shock, ruptures of hollow organs with peritonitis; limb fractures);

- compartment syndrome appears when certain enclosed space within the body for several hours suffers from the decreased blood flow (for example when patients spends few hours in one inconvenient position); even when blood supply will be restored necrotic products will continue pathological process through toxic affection of the life-important organs (for example free myoglobin can cause renal failure).

There is always possibility of chronic diseases exacerbations on the background of alcohol poisoning (stroke, myocardial infarction). Remember about the necessity of complete examination (inspection, palpation, percussion and auscultation of undressed patient) of such alcohol victims – it is the only way to find all the “diagnostic mysteries” patients hide!

Intensive treatment:

- evaluate CNS condition (deepness of the coma);
- provide airways patency and adequate respiration (described above; if necessary – intubate the patient and begin artificial ventilation);
- check the cardiovascular system: heart rate, pulse, blood pressure;
- in case of severe hemodynamic disorders provide infusion therapy;
- insert the stomach probe and remove its contents using lavage with water;
- take blood samples and check blood alcohol level (obligatory!);
- prescribe intravenously: 60-80 ml of 40% glucose solution, 60-80 ml of 4% sodium bicarbonate solution, 5-10 ml of 5% ascorbic acid, 1-2 ml of vitamin B₁ solution;
- if there are no comorbidities add analeptic solutions i/m (2-3 ml of caffeine or 2 ml of cordiamin);
- in case of severe intoxication begin forced diuresis.

Poisonings with alcohol surrogates.

Patients can be poisoned with: home-distilled vodka, Cologne water, denatured alcohol, methyl alcohol, lotion, brake fluid, etc. The peculiarity of such poisonings is complex effect of the alcohol and other toxic components of the “drink”.

The most toxic are methyl alcohol and antifreeze (ethylene glycol) – their lethal dose is 60-100 ml. Lower doses cause neuritis of optical nerve and thus blindness (methyl alcohol), acute renal and liver failures (ethylene glycol).

In case of these poisonings detailed anamnesis and blood identification of the poison (gas chromatography) play the most significant role in the diagnostics. However for the prognosis volume of the poisonous fluid, duration of its influence, functional condition of the liver and effectiveness of the antidote therapy and detoxification are the most important.

In the body methanol and ethylene glycol are metabolised according to so called “lethal-synthesis”: during the breakdown of the poison in the liver there are created substances much more toxic than the parent compounds.

Intensive therapy:

1. Gastric lavage with potassium permanganate (oxidizes methyl alcohol).
2. Give antidote: 50 ml of 40% ethylic alcohol solution every 3 hours orally or 100 ml of 5% ethanol solution intravenously slowly combined with glucose solution during 2 days. Antidote will block the process of their biotransformation in the liver until the poisons will not be eliminated from the body.
3. Actively eliminate the poison from the body through repeated gastric lavages, forced diarrhoea, extracorporeal methods of detoxification (hemodialysis, hemosorbtion, plasmapheresis).
4. In order to treat optic neuritis you should perform retrobulbar inject of steroids.
5. Symptomatic treatment.

8.4 Drug poisonings.

In civilized countries these poisoning are the main reason (65-70% of cases) of hospitalization in toxicology units. Usually patients overdose sleeping pills, narcotic painkillers, antihistamine drugs, hypotensive medicines. Among the reasons are suicide tries, drug abuse, toxicomania and accidental overdose due to hectic pace of modern life,

etc.

Clinical picture is usually connected with CNS affection. There are phases of somnolence, sleep and coma. Depending on involvement of other systems coma can be complicated or uncomplicated. Usually respiratory complications appear: inspiration centre depression, violations of airways patency due to soft tissues (tongue, soft palate) or biologic fluids (blood, sputum, saline), pneumonia. In 15-20% of cases the poisoning development is complicated with the exotoxicshock. The peculiarities of this shock are next: circulatory disorders with blood stagnation in the pulmonary circuit, toxic affection of the myocardium and decrease of energy metabolism of the organism.

To indicate the poison you should ask relatives and witnesses and check things of the patient (for example you can find medicine packages). Evaluate the size of the pupils: extremely narrowed pupils (“poppy seeds”) are the effect of narcotics admission; narrowed pupils might be the sign of sleeping or sedative medicines overdose; dilated pupils are the symptom of clofelin, antidepressants or neuroleptics administration; wide pupils covering the whole iris are usually the sign of atropine poisoning (or a poisoning with atropine-like substances: dope, henbane, amanita).

Principles of the intensive treatment in the toxicology unit:

- clean the gastrointestinal tract as soon as possible (gastric and intestinal lavage, enterosorbition, cleansing enemas) and as often as necessary;
- provide adequate respiration (check airways patency);
- in case of comatose patients intubate the trachea and begin artificial ventilation (sometimes it is necessary for weeks);
- control hemodynamics and treat its violations (infusion therapy and adrenergic agonists or antagonists if necessary);
- stimulate diuresis: patients with barbiturate poisoning should be treated with alkaline forced diuresis in order to eliminate the toxin (add to the infusion 400-600 ml of 4% sodium bicarbonate solution and prescribe diuretics);
- use antidotes: naloxone for opiates, pharmacological antagonists for anticholinergic and cholinomimetic agents; don't you ever prescribe central analeptics for comatose patients with drugs poisoning – cordiamin, caffeine, bemegrade , cytiton, lobelinum can cause “cerebral blood flow steeling effect” and thus they deepen the hypoxia of brain cells!
- provide extracorporeal detoxification to eliminate toxins (hemodialysis, hemosorbition, plasmapheresis);
- prescribe antibiotics for infectious diseases prevention (for example in case of prolonged artificial ventilation);

- symptomatic treatment.

8.5 Alkali and acid poisonings.

These poisonings are among the most severe and difficult to treat. Accidentally or intentionally (suicide) victim can take mineral acids (hydrochloric, sulphuric, nitric acids), organic acids (acetic or oxalic acid), alkali (ammonium chloride, battery fluid, etc.).

When corrosive substance gets into the body along its way inside chemical burn appears: mucous membranes of oral cavity, throat, oesophagus, stomach are injured. Together they make nearly 14-15% of the body surface. Patients suffer from unbearable pain, eating and drinking are disabled. In case of acid burn coagulation necrosis appears; alkali burn is more severe, because colliquative necrosis penetrates deeper into the tissues ruining the vessels and causing bleedings. Organic acids easily get into the blood. Sometimes chemical substances or their vapours also get into the airways and thus oedema and risk of asphyxia appear.

Usually part of aggressive substance is spilled over the chin and you can notice the burn. Systemic disorders are characterized with exotoxic shock which develops as burn shock (unbearable pain, dehydration, toxic affection of the heart, decrease of cardiac output, spasm of arterioles and microcirculation block). Organic acids also provoke hemolysis of red blood cells: free haemoglobin transforms into hydrochloric haematin and obturates renal tubules, causing acute renal failure.

Intensive treatment principles:

- evaluate patient's condition: external respiration, consciousness, cardiovascular system;
- adequate pain relief with narcotic painkillers and non-steroidal anti-inflammatory drugs (1-2 ml of 1% morphine solution; 2 ml of 50% analgin solution);
- liquidate the spasm of gastric cardia and oesophagus (1 ml of 0,1% atropine solution i/m, 5 ml of baralgin solution);
- clean the stomach during first 10 hours after poisoning; insert the stomach probe (cover it thickly with Vaseline and don't push too hard); use water for lavage and don't try to perform chemical inactivation of the poison, because during the reaction carbon dioxide can exude and acute expansion of the stomach leads to its rupture;
- provide treatment of shock (sometimes up to 10-12 liters/day of infusions);

- in case of organic acids poisonings (acetic acid, oxalic acid get into the blood) give 1500-2000 of 4% sodium bicarbonate solution intravenously slowly with diuretics; these actions will help to remove hemoglobin (released due to red blood cells hemolysis) and thus to prevent acute renal failure;
- in case of obstructive breathing disorders (mucous membrane edema) use steroids (90-120 mg of prednisolone), antihistamine drugs (2 ml of 1% dimedrol solution intravenously), sedative medicines (2 ml of 0,5% diazepam solution); perform tracheostomy or conicotomy if necessary;
- prescribe antibiotics for infectious diseases prevention (for example in case of prolonged artificial ventilation);
- symptomatic treatment.

During the recovering period patient may need surgeries for restoration of gastrointestinal tract: the most common practice is the bouginage of the oesophagus or, if necessary, oesophagus plastic.

8.6. Poisonings with toxic gases.

Among the toxic gases are carbon monoxide, car exhausts, propane and butane, ammoniac gases. The last one is the most toxic: few inhales are enough to cause unconsciousness.

The foundation of the pathology lies within the atypical haemoglobin – carboxyhemoglobin – combination of normal haemoglobin and toxic gas. Oxygen transportation is violated (in case of severe poisonings there are nearly 70-80% of changed haemoglobin) and thus haemic hypoxia appears. In addition within the tissues cytochromes are connected with toxic substances and this leads to tissue hypoxia.

Clinical findings in case of carbon monoxide poisoning depend on the severity of the poisoning. In case of mild intoxication they are: headache, nausea, vomiting. Moderate intoxication shows unconsciousness for 12-16 hours and severe intoxication is characterized with coma, central disorders of breathing, toxic affection of heart and other organs, etc.

If intoxication advances changes of central nervous system become irreversible (brain death is possible).

Intensive treatment.

In case of mild and moderate poisonings you should you should carry the patient out of the toxic atmosphere as soon as possible. In hospital conditions you should provide oxygen supply, get an intravenous line for crystalloids infusion and prescribe

vitamins.

In case of severe intoxication begin artificial ventilation with high oxygen flow. Luckily there is an antidote for carbon monoxide poisoning: hyperbaric oxygenation. Connection with oxygen is more natural for haemoglobin and when the pressure of oxygen is higher than its usual partial pressure carbon monoxide is replaced from the haemoglobin. Usually in case of comatose patients 40-50 minutes sessions every 6-12 hours are enough.

To normalize tissue metabolism prescribe antihypoxants: 20% solution of sodium oxybutirate (20-40 mg/kg i/v) and cytochrome c (2-3 ml i/v) every 4-6 hours. To improve microcirculation dilute the blood with crystalloid infusions (check the level of hemodilution with hematocrit – stop when it will reach 0,3-0,35 l/l).

Prevent the infectious complications and brain oedema with standard methods.

8.7 Organophosphate poisonings.

These are the poisonings with insecticides, acaricides, herbicides, fungicides, rodenticides, desiccants, defoliants and with chemical warfare agents such as sorin, soman, V-x.

Organophosphate substances are fats and water soluble and thus they penetrate easily through the skin and mucous membranes (gastrointestinal tract, airways, etc.). In the blood they block an enzyme – cholinesterase – responsible for the breakdown of acetylcholine. As you remember acetylcholine is a universal synaptic mediator of nervous impulses and thus its accumulation on the post-synaptic membranes will cause continuous stimulation of vegetative nervous system and cross-striated muscles.

Clinically you will see: nausea, vomiting, cramps; unconsciousness in severe cases. Sometimes in the place of penetration you can see muscle fasciculation (if the poison was administered orally – tongue twitching). Stimulated parasympathetic nervous system shows wet skin, increased salivation and bronchial secretion (sometimes you can even see white phlegm in the mouth – don't mistake it with the pink phlegm of pulmonary oedema), narrowed pupils, bradycardia (heart rate 40-30 beats per minute). In addition to the obvious clinic you can always check the environment of the patient for the signs of organophosphate poisons (specific smell, containers with toxins, etc.).

One third of patients suffer from exotoxic shock, which primary causes hypertension and then hypotension, unconsciousness and depression of respiration.

Immediate aid:

- take patient out of the dangerous environment (if you suspect that the mechanism of poisoning is inhalation);

- clean the stomach with large amounts of cold water; repeat it several times, because these substances are excreted through the mucous membranes of gastrointestinal tract;
- give saline laxative;
- if the poison affected skin – wash it with alkaline solution.

Antidotes:

a. use peripheral m-anticholinergic drug – atropine: during the first few hours 2-3 ml of 0,1% atropine solution (up to 30-35 ml during the whole period of intensive atropinization); pay attention to the signs of atropine administration as they are the measure of your antidote treatment effectiveness: termination of excessive bronchial secretion, dilation of the pupils, tachycardia (90-110/min). During next 3-5 days continue atropine prescription (from 10-15 mg to 100-150 mg/day – period of supportive atropinization). Control clinically the level of atropinization.

b. use cholinesterase reactivators: 1-2 ml of 15% dipiroxim solution i/m, up to 600 mg; 3 ml of 40% izonotrozin solution i/m up to 3-4 grams. However remember, that cholinesterase reactivators can be used only 24 hours after poisoning. Later administered reactivators will ne not only ineffective, but also toxic for the patient.

You should also provide usual treatment as soon as possible: infusion therapy, forced diuresis, hemosorbtion, plasmapheresis, hemodialysis and antibiotics for infection preventions.

In case of ineffective external respiration and comatose patient's condition intubate the patient and start artificial respiration. Convulsions in case of organophosphate poisoning are treated with sodium oxybate (75-100 mg/kg i/v every 4 hours). Cardiac glycosides, calcium chloride, euphillinum are forbidden in case of organophosphate poisonings, because they induce toxic heart affection.

Be aware of the possibility of “second poisoning wave”: even 4-8 days after the stabilization of the patient's condition clinical picture of the poisoning might return and this time hemodynamics will decompensate quickly.

8.8 *Mushroom poisoning.*

There are edible, non-edible and relatively edible mushrooms. Non-edible or poisonous mushrooms can contain toxins harmful for central nervous system, liver, kidneys and gastrointestinal tract (according to A. Lokay, 1 968). The most dangerous poisoning is caused by deadly amanita. The poison of this mushroom – amanitotoxin- is not destroyed during cooking and there is no way to detect it in usual conditions. In case

of severe poisoning the lethality is 80%.

Specific feature of amanita poisoning is prolonged latent period. Sometimes 6-12 hours pass before the first symptoms of the poisoning appear. All the other relatively edible mushrooms reveal clinical signs of the poisoning much earlier – 1-2 hours after consumption.

After the latent period is over on the background of complete health nausea, profuse vomiting and diarrhoea appear. Those symptoms begin second phase of the poisoning – gastroenterocolitic phase. Liver enlarges; patients suffer from pain in the right subcostal area, weakness, and consciousness disorders. Stool becomes watery and contains mucous. Patients loose up to 4 liters of the fluid during the day. Unlike bacterial food poisonings, mushroom poisonings are not characterized with high fever.

Liver failure and acute kidneys injury are the third phase of the poisoning, which begins on the second or third day of disease. Those failures are characterized with hepatic encephalopathy, jaundice, gastrointestinal bleedings, hepatic breath odour and oligoanuria. The level of alanine and aspartateaminotransferases is very high. When liver returns to its usual size and consciousness is changed into coma hepatargia is stated and prognosis for the disease becomes rather unfavourable.

The forth stage is a stage of recovery and is characterized with gradual regression of the clinical picture and normalization of the laboratory results during several weeks. However survival is possible only for those patients, who ate small amounts of the poisonous mushroom.

Knowing about the high lethality and severity of the amanita poisoning prevention methods become very important. Mushroomers should know the difference between deadly amanita and other mushrooms: deadly amanita is a gill-bearing mushroom with olive or green cap. Its gills white are not connected with the stem. Stem has a bulbus with white volva from one side and a white annulus from another side, under the cap.

Patients should be treated in a special toxicology units or intensive treatment unit. General principles of intensive therapy are:

- gastric and intestinal lavage, enterosorbition and saline laxatives;
- infusion therapy (necessary to liquidate electrolyte deficiency and provide forced diuresis);
- support of the liver functioning with lipoic or thioctic acid (1000-2000 mg/day), concentrated glucose solutions, steroids (up to 40 mg of dexamethasone per day) and silibin (50 mg/day);

- extracorporeal detoxification (hemodialysis, hemosorbition, plasmapheresis, artificial spleen or liver) as soon as possible;
- antibiotics if necessary (penicillin); vitamins (B,C,E);
- external drainage of thoracic duct (decreases intoxication through elimination of toxic lymph).

8.9 Medical operations and manipulations.

Gastric lavage

Indications: necessity to remove poisons or toxins from the stomach, to clean it before operation or to liquidate stagnation during period after the operation.

Equipment required: gastric probe (with two channels if possible), Janet syringe, water for lavage (15-20 liters of room temperature water), gloves and watertight apron.

Procedure: gastric lavage of comatose patients is a procedure for doctors. In our country nurses are not allowed to do this without control of the doctor: unconscious patients have inhibited reflexes and thus probe can be easily inserted into the trachea instead of oesophagus. In this situation neither cyanosis nor cough may not appear and everything looks just fine, however feeding or lavage try can end with fatal complications (asphyxia and death).

Put on the gloves, choose the probe of necessary size and oil it with Vaseline. Patient should lie on the left side (ask nurse to hold patient's arms to limit his movements during this unpleasant procedure).

If patient is conscious you can previously use lidocaine spray to anaesthetize mucous membranes. Probe can be inserted through the nose (of course in this case size of the probe is limited) or through the mouse. Don't push too hard, especially when you are using nasal passage: you can cause bleeding. Ask patient to bow his head to the chest – this will increase chances of correct probe insertion (oesophagus, not trachea). The length of probe you insert can be measured in advance as a distance between earlobe, nose and xiphoid.

Confirmations of correct probe placement:

- auscultate the epigastrium and simultaneously infuse some air with the syringe- you will hear typical "bubble" noises;
- if you made a mistake and probe is in the trachea you can notice air released from the distant end of the tube according to the respiratory movements.

One-time water dose is nearly 200 ml: it will flow out when you will lower the probe or you will have to evacuate the water with the syringe. Repeat these actions until the water wash out the stomach will be clean (usually it takes nearly 10 liters of water).

Forced diuresis.

Indications: intoxications of different origin (poisonings, infectious diseases, endogenous intoxications).

Medicines required: normal saline (3-5 liters); detoxification solutions; polarizing solution (400 ml of 10% glucose solution, 10 ml of 7,5% potassium chloride solution, 12 units of insulin), osmotic diuretics (mannitol in the dose 1 g/kg), furosemid solution (40-80 mg)

Procedure: get and i/v line (central or peripheral) and insert urinary catheter. During the first phase of forced diuresis "water" the patient with crystalloids and detoxification solutions (30-40 ml/kg). During the second phase infuse osmotic diuretics and furosemide solution. Excessive urine output brings potassium loss, which you should treat with polarizing solution. Balance the speed of infusion with the speed of diuresis: generally per 5-7 liters of infused solutions you should receive at least 5 liters of urine.

Constantly control hemodynamics and blood electrolytes.

Control tests.

1. What medicine should be used as an antidote in case of severe soporific drugs poisoning?

- A. bemegrade
- B. cordiamin
- C. there is no such medicine
- D. unithiol
- E. cytiton

2. What is the most common death reason in case of alcohol poisoning?

- A. acute liver failure
- B. acute respiratory failure
- C. acute renal failure
- D. acute heart insufficiency

E. collapse

3. What is necessary for the patient, who took 60 ml of acetic essence?

A. to give 4% sodium bicarbonate solution i/v

B. to clean the stomach with alkaline solution in order to neutralize the acid

C. to use unithiol as an antidote

D. to stimulate intestinal cleansing with saline laxatives

E. central analeptics

4. What is the symptom of organophosphate poisoning?

A. pale and dry skin

B. maximal pupil's dilation

C. tachycardia

D. muscle fasciculation

E. acute liver failure

5. What is used as an antidote in case of severe carbon monoxide poisoning?

A. cytochrome c

B. cordiamin solution

C. unithiol

D. prednisolone

E. antidotes are not used

6. What is typical for deadly amanita poisonings?

A. first symptoms appear in 2-3 hours after mushroom meal;

B. low-grade fever

C. latent period of 6-12 hours

D. first symptoms are haemostatic disorders (bleeding)

E. early unconsciousness

7. What is the necessary aid in case of narcotic painkillers overdose?

A. artificial ventilation

B. cordiamin

C. oxygen supply

D. immobilization of the patient in order to avoid self-injuries during the

excitement-phase;

E. cardiac medicines

Task 1.

Patience, the patient of 19 is transported to the ITU by witnesses from the street. Clinical findings: unconscious, cyanotic wet skin, 6 breathes per minute, respiration shallow, blood pressure 70/40 mm Hg, heart rate 112/minute. Name the reason of vital disorder and write the principles of intensive care (algorithm).

Task 2.

Patience, the patient of 19, was found unconscious in his own apartment by the ambulance workers. Clinical findings: total cyanosis, shallow breathing, respiration rate 5/min, pupils extremely narrowed, photoreaction is absent, blood pressure 80/40 mm Hg. In the elbow area there are noticeable signs of injections. Name the reason of vital disorder and write the principles of intensive care (algorithm).

Task 3.

Patience, the patient of 23 was hospitalized into ITU with the diagnosis: mushroom poisoning. It turned out, that yesterday she was eating cooked champignons. 8 hours later she noticed vomiting and diarrhoea. Clinical findings: scleral icterus, dry coated white tongue; painful abdomen (epigastrium and righthypogastric area), painful and enlarged liver (2 cm); hemodynamics and respiration are not violated.

Write the diagnosis and the phase of the disease.

Task 4.

Patience, the patient of 18, is transported to the toxicology unit with delirium. Clinical findings: hyperaemic face, dry skin, dilated pupils. Blood pressure 140/70 mm Hg, heart rate 127/minute. It is know, that 2 hours ago she took 10 unknown tablets during the suicidal attempt. What is the diagnosis and what are your actions?

Task 5.

Patience, the patient of middle age, was found by the ambulance workers in his own kitchen unconscious. Clinical findings: specific smell of organophosphates in the room, signs of vomit on the clothes, miosis, cold clammy skin with cyanotic shade, foamy white sputum in the mouth, blood pressure 90/50 mm Hg, heart rate 54/minute. What is the diagnosis? What immediate aid should be provided during pre-hospital stage?

Task 6.

Adams family were celebrating the New Year Eve near the fireplace. In the

morning came the carol singers and found hosts in a condition of deep sleep. “Dream team”, the ambulance workers, who came to the place of the accident, stated: 2 adults and their children in comatose condition, vomit signs on the clothes, rapid breathing, pupils dilated with weak photoreaction; heart rate is 110-120 per minute, rhythmic; blood pressure is high.

Name the reason of the accident and describe the actions of the “Dream team”.

Task 7.

Name the poisoning agent for each antidote: a. atropine, b. unithiol, c. naloxone, d. tetacinum, e. ethylic alcohol, f. dipiroximum, g. lipoic acid?

The essential components of the human cardiovascular system are the heart, blood, and blood vessels. It includes: the pulmonary circulation, a "loop" through the lungs where blood is oxygenated; and the systemic circulation, a "loop" through the rest of the body to provide oxygenated blood. An average adult contains five to six quarts (roughly 4.7 to 5.7 liters) of blood, accounting for approximately 7% of their total body weight.

While it is convenient to describe the flow of the blood through the right side of the heart and then through the left side, it is important to realize that both atria contract at the same time and that both ventricles contract at the same time. The heart works as two pumps, one on the right and one on the left that works simultaneously. The right pump pumps the blood to the lungs or the pulmonary circulation at the same time that the left pump pumps blood to the rest of the body or the systemic circulation. Venous blood from systemic circulation (deoxygenated) enters the right atrium through the superior and inferior vena cava. The right atrium contracts and forces the blood through the tricuspid valve (right atrioventricular valve) and into the right ventricles. The right ventricles contract and force the blood through the pulmonary semilunar valve into the pulmonary trunk and out the pulmonary artery. This takes the blood to the lungs where the blood releases carbon dioxide and receives a new supply of oxygen. The new blood is carried in the pulmonary veins that take it to the left atrium. The left atrium then contracts and forces blood through the left atrioventricular, bicuspid, or mitral, valve into the left ventricle. The left ventricle contracts forcing blood through the aortic semilunar valve into the ascending aorta. It then branches to arteries carrying oxygen rich blood to all parts of the body.

Blood Flow Through Capillaries

From the arterioles, the blood then enters one or more capillaries. The walls of capillaries are so thin and fragile that blood cells can only pass in single file. Inside the

capillaries, exchange of oxygen and carbon dioxide takes place. Red blood cells inside the capillary release their oxygen which passes through the wall and into the surrounding tissue. The tissue then releases waste, such as carbon dioxide, which then passes through the wall and into the red blood cells.

The Circulatory System

The circulatory system is extremely important in sustaining life. Its proper functioning is responsible for the delivery of oxygen and nutrients to all cells, as well as the removal of carbon dioxide, waste products, maintenance of optimum pH, and the mobility of the elements, proteins and cells, of the immune system. In developed countries, the two leading causes of death, myocardial infarction and stroke are each direct results of an arterial system that has been slowly and progressively compromised by years of deterioration.

Arteries

Arteries are muscular blood vessels that carry blood away from the heart, oxygenated and deoxygenated blood. The pulmonary arteries will carry deoxygenated blood to the lungs and the systemic arteries will carry oxygenated blood to the rest of the body. Arteries have a thick wall that consists of three layers. The inside layer is called the endothelium, the middle layer is mostly smooth muscle and the outside layer is connective tissue. The artery walls are thick so that when blood enters under pressure the walls can expand.

Arterioles

An arteriole is a small artery that extends and leads to capillaries. Arterioles have thick smooth muscular walls. These smooth muscles are able to contract (causing vessel constriction) and relax (causing vessel dilation). This contracting and relaxing affects blood pressure; the higher number of vessels dilated, the lower blood pressure will be. Arterioles are just visible to the naked eye.

Capillaries

Capillaries are the smallest of a body's vessels; they connect arteries and veins, and most closely interact with tissues. They are very prevalent in the body; total surface area is about 6,300 square meters. Because of this, no cell is very far from a capillary, no more than 50 micrometers away. The walls of capillaries are composed of a single layer of cells, the endothelium, which is the inner lining of all the vessels. This layer is so thin that molecules such as oxygen, water and lipids can pass through them by diffusion and enter the tissues. Waste products such as carbon dioxide and urea can diffuse back into the blood to be carried away for removal from the body.

The "capillary bed" is the network of capillaries present throughout the body. These beds are able to be "opened" and "closed" at any given time, according to need. This process is called autoregulation and capillary beds usually carry no more than 25% of the amount of blood it could hold at any time. The more metabolically active the cells, the more capillaries it will require to supply nutrients.

Veins

Veins carry blood to the heart. The pulmonary veins will carry oxygenated blood to the heart while the systemic veins will carry deoxygenated to the heart. Most of the blood volume is found in the venous system; about 70% at any given time. The veins outer walls have the same three layers as the arteries, differing only because there is a lack of smooth muscle in the inner layer and less connective tissue on the outer layer. Veins have low blood pressure compared to arteries and need the help of skeletal muscles to bring blood back to the heart. Most veins have one-way valves called venous valves to prevent backflow caused by gravity. They also have a thick collagen outer layer, which helps maintain blood pressure and stop blood pooling. If a person is standing still for long periods or is bedridden, blood can accumulate in veins and can cause varicose veins. The hollow internal cavity in which the blood flows is called the lumen. A muscular layer allows veins to contract, which puts more blood into circulation. Veins are used medically as points of access to the blood stream, permitting the withdrawal of blood specimens (venipuncture) for testing purposes, and enabling the infusion of fluid, electrolytes, nutrition, and medications (intravenous delivery).

Venules

A venule is a small vein that allows deoxygenated blood to return from the capillary beds to the larger blood veins, except in the pulmonary circuit where the blood is oxygenated. Venules have three layers; they have the same makeup as arteries with less smooth muscle, making them thinner.

The double circulatory system of blood flow refers to the separate systems of pulmonary circulation and the systemic circulation in amphibians, birds and mammals (including humans.) In contrast, fishes have a single circulation system. For instance, the adult human heart consists of two separated pumps, the right side with the right atrium and ventricle (which pumps deoxygenated blood into the pulmonary circulation), and the left side with the left atrium and ventricle (which pumps oxygenated blood into the systemic circulation). Blood in one circuit has to go through the heart to enter the other circuit. Blood circulates through the body two to three times every minute. In one day, the blood travels a total of 19,000 km (12,000 miles), or four times the distance across the U.S. from coast to coast.

The Pulmonary Circuit

In the pulmonary circuit, blood is pumped to the lungs from the right ventricle of the heart. It is carried to the lungs via pulmonary arteries. At lungs, oxygen in the alveolae diffuses to the capillaries surrounding the alveolae and carbon dioxide inside the blood diffuses to the alveolae. As a result, blood is oxygenated which is then carried to the heart's left half -to the left atrium via pulmonary veins. Oxygen rich blood is prepared for the whole organs and tissues of the body. This is important because mitochondria inside the cells should use oxygen to produce energy from the organic compounds.

The Systemic Circuit

The systemic circuit supplies oxygenated blood to the organ system. Oxygenated blood from the lungs is returned to the left atrium, then the ventricle contracts and pumps blood into the aorta. Systemic arteries split from the aorta and direct blood into the capillaries. Cells consume the oxygen and nutrients and add carbon dioxide, wastes, enzymes and hormones. The veins drain the deoxygenated blood from the capillaries and return the blood to the right atrium.

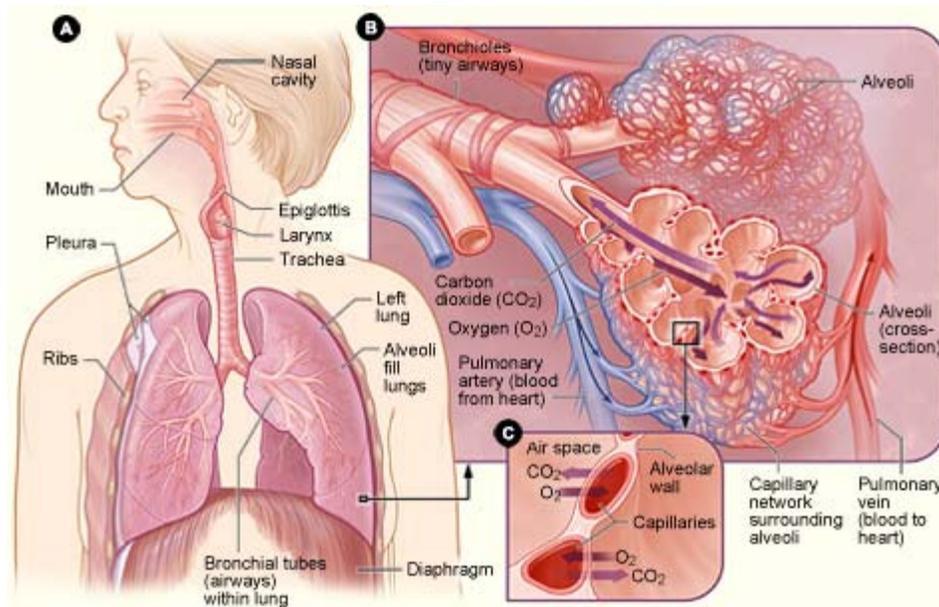
Pic. 4.1 Distribution of blood in the body:

- a. hear cavity itself 0 3% (% of blood volume)
- b. arteries -15%
- c. capillares -12%
- d. venous system – 70%

Cardiac cycle is the term used to describe the relaxation and contraction that occur, as a heart works to pump blood through the body. Heart rate is a term used to describe the frequency of the cardiac cycle. It is considered one of the four vital signs. Usually it is calculated as the number of contractions (heart beats) of the heart in one minute and expressed as "beats per minute" (bpm). When resting, the adult human heart beats at about 70 bpm (males) and 75 bpm (females), but this rate varies between people. However, the reference range is nominally between 60 bpm (if less termed bradycardia) and 100 bpm (if greater, termed tachycardia). Resting heart rates can be significantly lower in athletes, and significantly higher in the obese. The body can increase the heart rate in response to a wide variety of conditions in order to increase the cardiac output (the amount of blood ejected by the heart per unit time). Exercise, environmental stressors or psychological stress can cause the heart rate to increase above the resting rate. The pulse is the most straightforward way of measuring the heart rate, but it can be deceptive when some strokes do not lead to much cardiac output. In these cases (as happens in some arrhythmias), the heart rate may be considerably higher than the pulse. Every single 'beat' of the heart involves three major stages: atrial systole, ventricular systole and complete cardiac diastole. Throughout the cardiac cycle, the blood pressure increases and decreases. As ventricles contract the pressure rise, causing the AV valves

to slam shut.

Acute respiratory failure.



3.1 Anatomy and physiology of respiratory system.

One of the most important conditions of living is continuous gas exchange. Every day a human body consumes nearly 700 l of oxygen and produces 600 liters of carbon dioxide.

Oxygen is responsible for the mitochondrial and microsomal oxidation, peroxidation of unsaturated fatty acids and oxydase reactions. But the most significant role it plays when provides oxidative phosphorylation during energetic metabolism of the cell.

There are two types of breathing: external or lung respiration provides inflow of oxygen and carbon dioxide elimination; internal respiration is a complex process, which consists of haemoglobin transformation (connection and disconnection with oxygen), transportation of oxygenated haemoglobin through circulatory system to the tissues and local cellular metabolism.

External breathing is being regulated by centres in medulla and pons. Increasing of carbon dioxide concentration, as well as acidosis of cerebrospinal fluid, stimulates inspiration. At the end of expiration inspiration centre is being stimulated automatically and thus respiratory act begins again. In addition receptors of aorta arch and carotid sinus are also involved into respiratory act regulation: they respond to the lowering of oxygen concentration in the arterial blood.

The act of ventilation itself is being done through contraction of chest muscles and diaphragm. Muscles, responsible for respiration, enlarge chest and thus create negative pressure inside of it, what makes air move through the airways to the alveoli. Expiration is usually passive: chest falls down to its previous position and “used” air gets back to the atmosphere.

Tidal volume (TV) is 450-800 ml for male and 400-700 for female. It can be measured

by spirometer or volumeter. But not the whole air of one respiratory effort gets to the alveoli: part of it stays in oral cavity, nasal cavity, pharynx, trachea, bronchi and thus does not participate in the act of respiration itself. It is so called “dead space”- volume of airways which are ventilated, but are not involved in gas exchange. Dead space makes nearly 30% of the tidal volume (2,22 ml/kg of body weight).

Normal respiration rate (RR) is 14-20 per minute. Thus minute ventilation is calculated according to the formula:

$$\text{Minute ventilation} = \text{Tidal volume} * \text{Respiratory rate} \quad (\text{ml})$$

However the parameter, which helps to characterize the efficiency of breathing, is alveolar ventilation (AV): it shows the exact amount of the air which gets to the alveoli during the minute. The difference between minute ventilation and alveolar ventilation is the volume of dead space:

$$\text{Alveolar ventilation} = (\text{Tidal volume} - \text{Dead space volume}) * \text{Respiratory rate} \quad (\text{ml})$$

According to this formula breathing with lower frequency and higher tidal volume is more effective. In addition efficiency of breathing is increased through reduction of dead space volume. Thus endotracheal tube or tracheostomy make it two times less.

Oxygen gets to the arterial blood due to the difference of gas partial pressures. If the atmospheric pressure is 740-760 mm of Mercury and the amount of oxygen in the air is 20-21% partial pressure of Oxygen (pO_2) will be 160-150 mm of Mercury. In the airways air is being mixed with “used gases” and water steam, so the partial pressure of oxygen inside the alveoli is 95-85 mm of Mercury. With such values pO_{2a} , 3 ml of oxygen will be dissolved in one liter of blood (plasma).

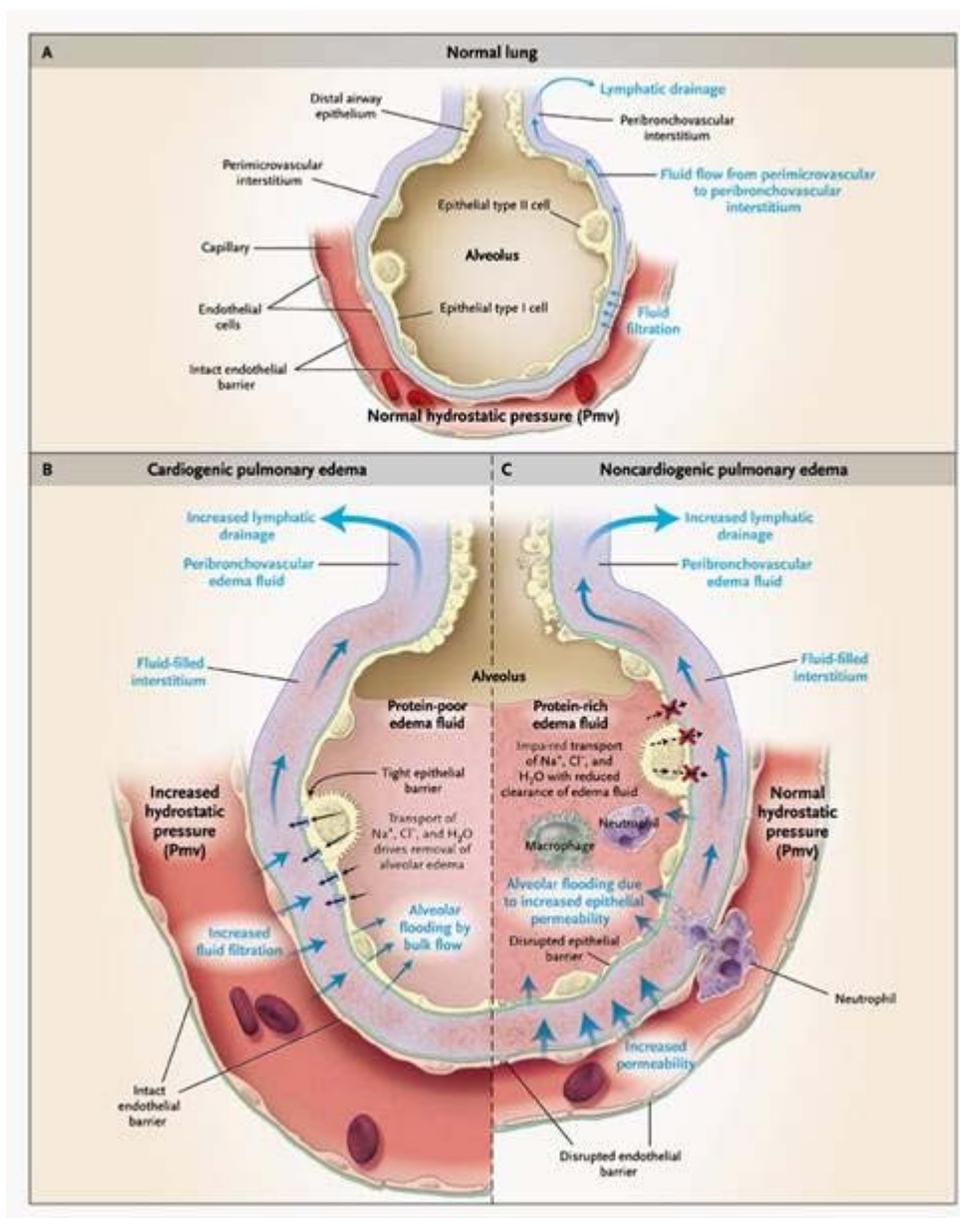
The main amount of oxygen in blood is being bound to the haemoglobin. One gram of haemoglobin bounds 1,34-1,39 ml of oxygen. In normal conditions haemoglobin of arterial blood (HbO_{2a}) is saturated to the extent of 96%. So, with the concentration of haemoglobin 120-140 grams per liter, one liter of arterial blood contains 170-190 ml of oxygen (VO_{2a}).

From every litre of blood only one third of oxygen amount (nearly 50 ml) is being used for the needs of the tissues. Thus venous blood contains nearly 120-140 ml of oxygen (VO_{2v}) and its HbO_{2v} is 70-75% and pO_{2v} 45-55 mm of Mercury.

Partial pressure of carbon dioxide in the arterial blood (pCO_{2a}) is an important index of ventilation adequacy. Normally it is 36-46 mm of Mercury. Worsening of lung ventilation causes increasing of pCO_{2a} over 44 mm of Mercury – hypercapnia appears. Excessive ventilation, on the contrary, helps in elimination of carbon dioxide and appearance of

hypocapnia: $p\text{CO}_{2a}$ is lower than 35 mm of Mercury.

Respiratory insufficiency is a condition in which metabolic needs of the body can not be satisfied with adequate oxygen admission, blood transportation, cellular consumption and carbon dioxide elimination.



Classification of respiratory insufficiency according to L. Usenko (1993):

A. Primary (caused by disorders of external respiration). Reasons:

1. airway patency disorders (obstruction with tongue, vomit, mucus, gastric contents, foreign bodies; laryngeal spasm, etc.)
2. central nervous system disorders (intoxication, brain injuries, haemorrhages, inflammations, etc.)
3. disorders of respiratory muscles activity (myasthenia, botulism, tetanus,

muscle relaxants, etc.)

4. defects of chest structure or functional chest disorders (chest injuries, limited diaphragm mobility – for example due to enteroparesis)
5. disorders of pulmonary compliance (pneumonia, bronchiolitis, atelectasis, “shock lung” syndrome, etc.)
6. disorders of ventilation-perfusion system (irregularity of ventilation and perfusion during artificial ventilation, pulmonary oedema, etc.)

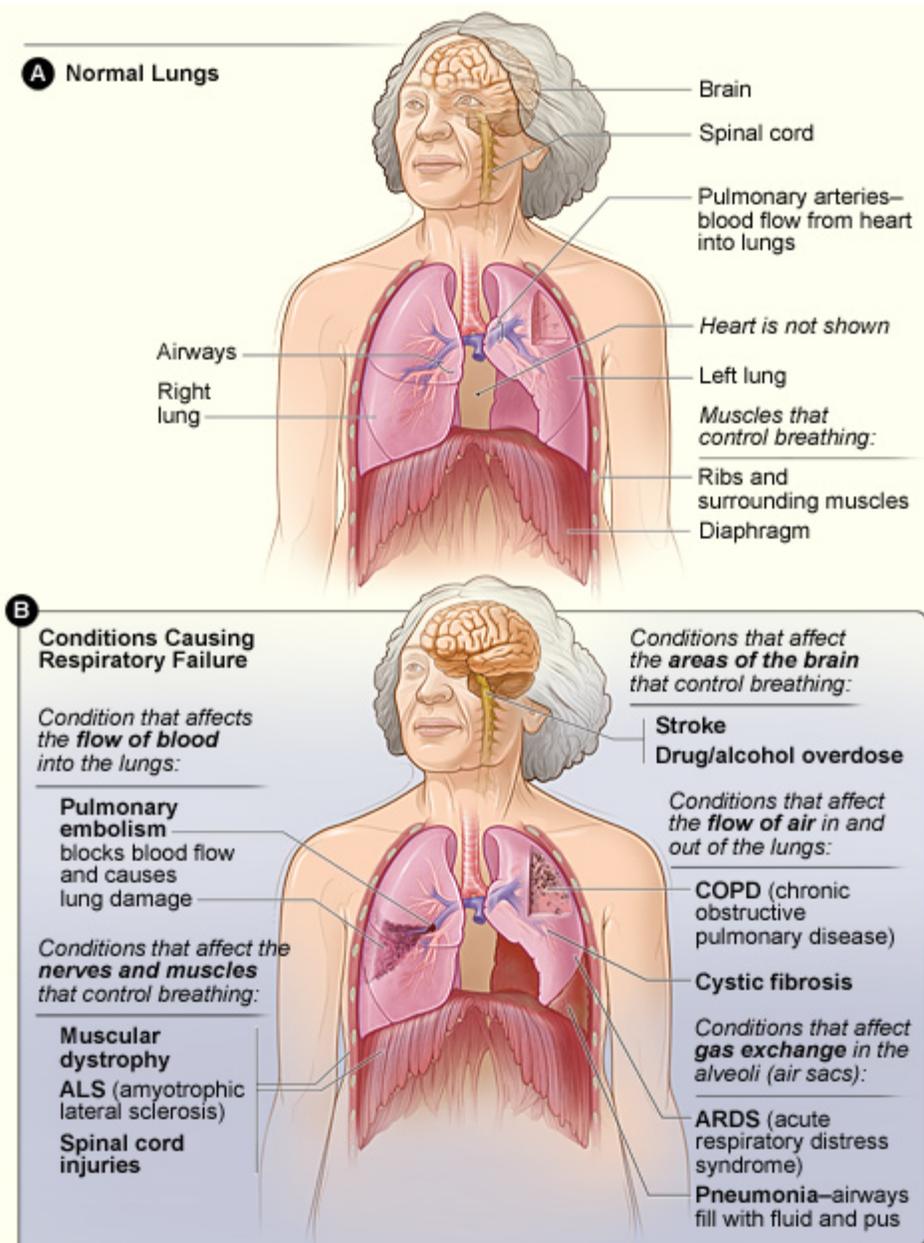
B. Secondary (caused by inability of blood to contain enough oxygen for metabolic needs of tissues or disorders of transportation and cellular consumption).

Respiratory insufficiency is characterized with hypoxia (“lowering of the oxygen”). Hypoxia occurs in case of:

1. Inability of external respiration to provide arterial blood with enough amount of oxygen (hypoxic hypoxia)
2. Lowering of oxygen amount due to anaemia - decreased level of haemoglobin, red blood cells or appearance of their atypical forms (haemic hypoxia).
3. Inability of cardiovascular system to provide systemic and pulmonary circulation enough for metabolic needs of the body (circulatory hypoxia).
4. Disorders of oxidative phosphorylation on cellular level of different organs and systems (tissue hypoxia).

Hypoxia itself can be accompanied with hypercapnia and hypocapnia. In case of tissue hypoxia there is also a possibility of hyperoxia (excessive oxygen amount) as its background.

In clinical conditions we usually deal with mixed gas exchange disorders.



Clinical symptoms of acute respiratory insufficiency

There are two types of respiratory insufficiency according to its duration: acute and chronic. Acute respiratory insufficiency develops in minutes or hours, can be life-threatening and thus needs urgent intensive treatment.

Symptoms of acute respiratory failure are really various.

Central nervous system. Conscious patients can complain of breathlessness (feeling of the air-lack), difficult inspiration or expiration. Due to development of hypoxia patients become restless, anxious, and sometimes euphoric; they can not evaluate critically their condition or environment. Terminal stages of insufficiency show total CNS inhibition – comatose state (hypoxic or hypercapnic). Quite often, especially in children, convulsions appear.

Skin and mucosa are mirror of respiratory insufficiency: their changes are quite

illustrative.

In case of external respiration disorders the amount of oxygenated haemoglobin is decreased and thus quantity of reduced haemoglobin. Arterial blood becomes “venous” (dark): skin and mucosa become cyanotic (blue, purple). First of all their colour change lips, nail plates, earlobes, afterwards face and other body parts. In case of anaemic patients with haemoglobin level 60 grams per litre and lower skin stays pale even in terminal stages of respiratory insufficiency. In case of cyanic and carbon monoxide intoxication skin, nail plates and mucosa turn bright pink, although patients are greatly suffering from hypoxia. Hydrosis is quite significant feature of hypercapnia. Terminal respiratory insufficiency is characterized with dark-grey color cold skin covered with clammy sweat.

Disorders of external respiration are the most obvious symptoms of respiratory insufficiency. Clinically in case of those patients next symptoms might be observed:

complete breathing arrest (apnea);

low respiratory rate – less than 12 per minute (bradypnea);

high respiratory rate – more than 20 per minute (tachypnea);

shallow breathing (respiratory volume less than 5 ml per kilogram of body weight);

respiratory “anarchy” (irregular breathing with pauses and uneven amplitude of respiratory movements);

pathological types of respiration:

a. Cheyne-Stokes breathing (periods of apnea, which are followed with chaotic frequent breathing);

b. Biot’s breathing (periods of apnea which are followed with breathing of equal amplitude);

c. Difficult breathing (noticeable at a distance, correlation between inspiration and expiration is violated, with active participation of additional muscles):

1. inspiratory dyspnea (difficult inspiration) – inspiration is prolonged, intercostals spaces, jugular fossa and subclavian fossa are retracted; sometimes stridorous noise can be heard

2. expiratory dyspnea (difficult expiration) – patients should make a great physical effort in order to exhale; exhalation is prolonged, noisy, heard at a distance; chest is enlarged, becomes barrel shaped

Cardiovascular system at the beginning reacts through compensative hyperdynamic reaction. Cardiac output, heart rate, systolic and diastolic pressure increase. Tissue perfusion

improves, what makes cellular oxygen supply and carbon dioxide elimination more effective. But due to hypoxia development vessel tone decreases and heart action is depressed. Blood pressure critically lowers and in case of inadequate treatment cardiac arrest appears.



Indications for artificial ventilation:

- apnoea
- central nervous system disorders - coma
- gray-cyanotic colour of skin, covered with clammy sweat
- low breathing frequency (less than 6 per minute) or high breathing frequency (more than 40 per minute) in case of normal thermal conditions, which causes great lowering of alveolar ventilation
 - excessive chest excursion, participation of additional muscles
 - pathological types of breathing
 - critical lowering of haemodynamic indexes (systolic blood pressure lower than 60 mm Hg), life-threatening arrhythmias

Decreasing of pO_{2a} to 60 mm Hg and less or increasing of pCO_{2a} mm Hg



Main types of external respiration disorders

Disorders of respiratory centre.

These disorders occur in case of narcotic intoxication, hypnotic and anaesthetic medicine usage. Injuries of the head, haemorrhages, brain strokes, inflammatory processes of the brain and meninges can violate breathing centre directly or through brain oedema. In these cases respiratory centre loses the ability of adequate reaction and thus acidosis, hypercapnia and hypoxia are not any more regulating ventilation.

In those patients we can see shallow, sometimes pathological breathing (Cheyne-Stokes, Biot's), in the most severe cases respiratory arrest occurs. The concentration of oxygen decreases and the level of carbon dioxide increases and thus hypoxia and hypercapnia damage cells on central nervous system, myocardium, other organs and systems, causing cardiac arrest.

Toxic or hypoxic damage of the brain, in turn, causes or deepens the coma and thus brings obstructive respiratory disorders. The muscle tone is decreasing, so the tongue blocks upper airways. In this situation only immediate actions, providing normal air passage, can save the patient's life.

It is quite often observed that in case of unconscious patient's oral cavity and pharynx are overfilled with saline and trachea, bronchi filled with mucus, which normally should be swallowed or spat out. Mucus gets infected quickly and thus inflammatory reactions begin, causing complications such as purulent bronchitis and tracheitis, pneumonia, which may lead coma patient to death.

Respiratory disorders of unconscious patients can be explained with passive outflow of gastric contents from stomach to the oral cavity and its further aspiration into airways (trachea

and bronchi). According to A. Zilber (1989) aspiration of 10-15 ml of acid gastric contents in most cases end with the death of the patient.

Prevention and treatment of this disease belong to the most urgent.

Urgent help:

1. Evaluate central nervous system condition – deepness of coma (chapter 5)
2. To prevent aspiration turn patient into save position (side position with head turned aside and if possible lower upper part of the body – thus gastric contents will probably flow out rather than into trachea).
3. Open the mouth of the patient and clean the oral cavity, if necessary, using your finger or forceps with surgical drape).
4. Titled the head backwards (use roll or small cushion if possible). Thus airways should stay clear.
5. Thrust the jaw forward: place your thumbs over the chin and the rest of your fingers put on the jaw (little fingers on the angles of the jaw), push the jaw forward and up, in a position of malocclusion (lower teeth in front of upper). After this action is done further providing of airways patency is quite simple and can be achieved with one finger.
6. You can also use simple airway adjuncts such as oropharyngeal and nasopharyngeal airways. Guedel tube is inserted between teeth with its curved distal end turned up, than moved toward soft palate and turned 180° and placed over the tongue.

In case of inadequate breathing or complete lack of it artificial ventilation should be provided (mouth to mouth or with apparatus for ventilation).

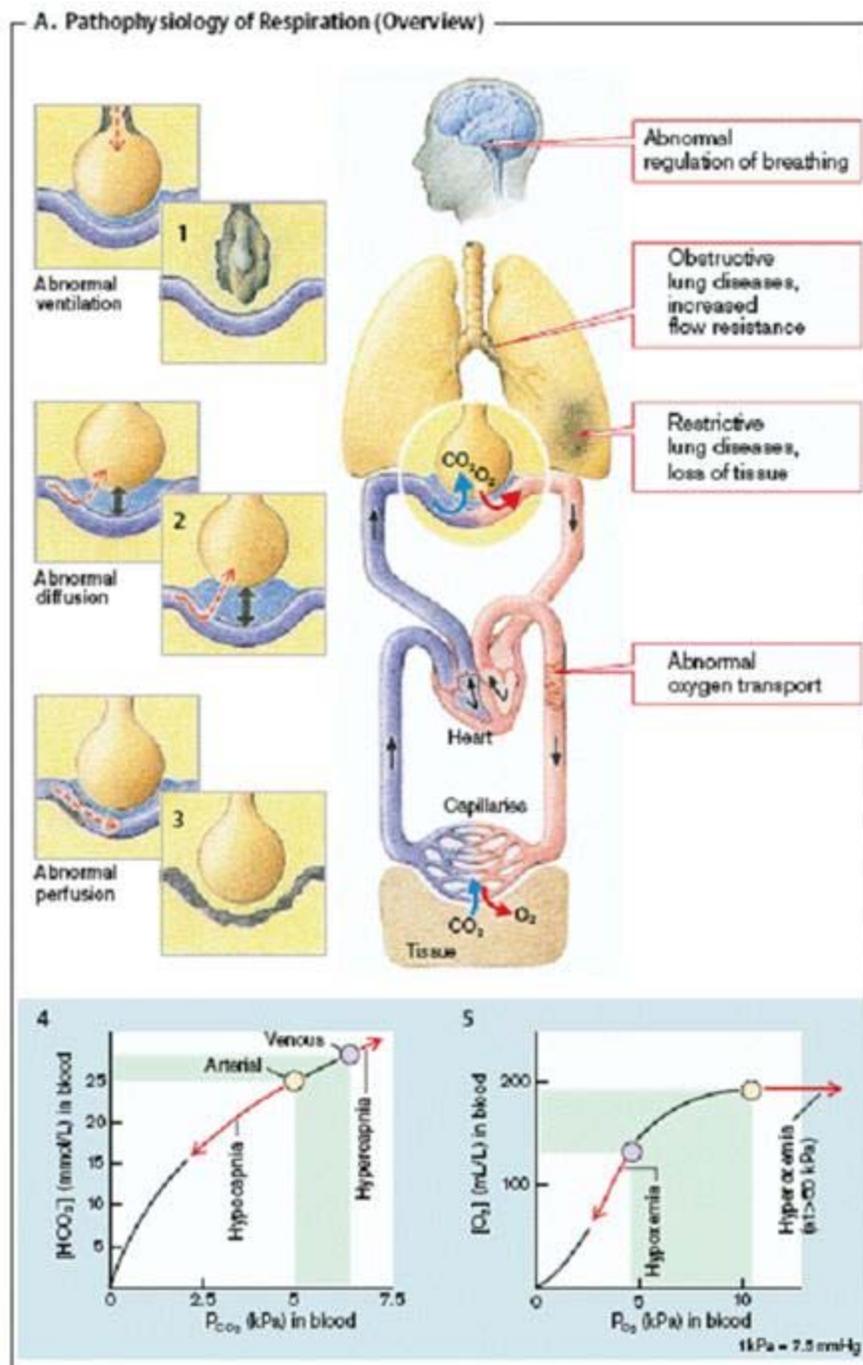
Constantly check patient's condition: control the heart and respiratory rate, blood pressure, body temperature, ECG. When there will be a possibility take biological material for the laboratory tests.

Immediate intensive treatment:

Perform direct laryngoscopy in order to check airways patency.

1. clean the oral cavity, if necessary, from foreign bodies and liquids such as vomit, sputum, blood cloths, gastric contents using electric vacuum suction
2. Provide oxygen supply of the patient: 8-10 liters per minute
3. In case of ineffective breathing begin artificial ventilation with Ambu bag or stationary respiratory apparatus.

4. Get the intravenous access, peripheral or central and start the infusion.
5. In case of further inadequacy of breathing give 0,5 ml of 0,1% of atropine intravenous and intubate the patient, because probably artificial ventilation will be prolonged.
6. Insert gastric sound and aspirate its contents.
7. Write down all the current data about patient's condition and performed treatment actions.
8. Continue observation using laboratory and instrumental techniques.
9. Continue etiological and symptomatic treatment.



Alveolar hypoventilation caused by respiratory muscles violations.

This pathology appears when chest muscles are not able to perform a complete respiratory act. Hypoventilation is the most common reason of vital disorders in case of patients with polyneuropathies, myasthenia, botulism, tetanus, poliomyelitis, organophosphate poisoning. In case of patients after operations this type of hypoventilation may occur due to relaxants used for anaesthesia, hypokalemia or metabolic alkalosis.

Excursion of the chest is reduced and thus volumes of inspiration and expiration are lower. Conscious patients feel breathless and anxious; cyanosis, clammy sweat, tachycardia, hypertension, restlessness appear. If treatment is not provided, CNS is depressed and earlier or

later cardiovascular system fails. Those patients should be ventilated with the help of respiratory apparatus immediately, as in their case oxygenation itself will not help (hypoxia will be corrected, but never the less elimination of carbon dioxide will not be improved and hypercapnia will cause cardiac arrest).

Residual relaxants effect is eliminated with anticholinergic drugs (1-2 ml of 0,05% proserin solution intravenous with previous administration of 0,1% atropine sulphate solution).

For hypokalemia correction special mix glucose, potassium and insulin is used (so called polarizing solution).

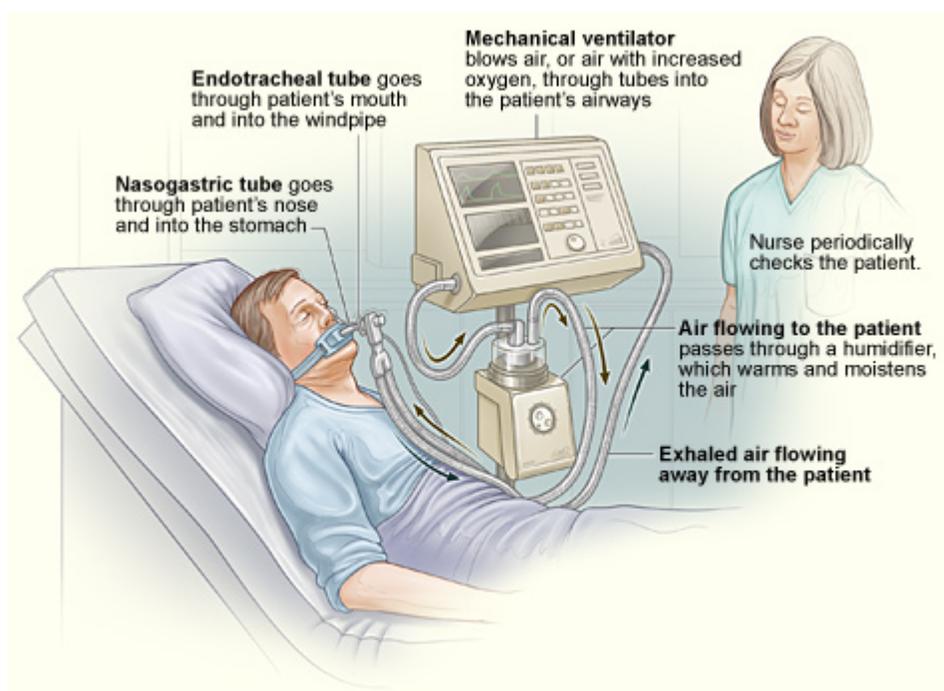
Low cholinesterase activity of plasma is compensated with fresh frozen plasma.

Organophosphate poisonings are treated with special antidotes (8.2); for elimination of convulsions specific medicines are used (5.3).

Brief lung ventilation (up to 30 minutes) can be provided with the mask. Ventilation up to 3 days needs intubation. If self-breathing is still inadequate after this time tracheostomy is recommended.

History of medicine knows cases, when patients were artificially ventilated for 18-20 years.

Team of medical professionals, responsible for treatment of patient with hypoventilation should follow all the asepsis and antiseptic rules and should be very careful and attentive to the patient's condition and apparatus work.



Rules of care: patients on prolonged ventilation

1. Constantly check the condition of the patient (vital functions should be

monitored), record in the papers parameters of ventilation, heart rate, blood pressure and central venous pressure, consciousness, physiologic and pathological liquid loss and medicines administered.

2. All the time control work of the respiratory apparatus (respiratory rate, volumes of respiration, minute ventilation, pressure in the airways during inspiration and expiration). If self-breathing is present, but inadequate it's important to achieve synchronized patient-apparatus breathing (choose the correct mode or sometimes use pharmacological sleep to "turn off" the respiratory centre of the patient if respiratory efforts are exhausting for the organism).

3. Every 30 minutes change parameters of respiration, making tidal volume 40-50% more than the necessary every 5-10 minutes. This way you prevent the development of atelectasis.

4. Every hour carry out oral, tracheal and bronchial suction using plastic catheters with side foramina and blunt end. Catheters generally should not be reused, but if it's necessary you can use them for one patient several times keeping them in antiseptic solution between suction. Procedure: doctor or nurse puts on gloves and face mask, disconnects tube from the apparatus, carefully and at the same time quickly inserts the plastic catheter, previously connected to the vacuum suction, into the tube and turns it clockwise and counterclockwise. For cleaning of principal bronchi try to turn head of the patient and to push catheter deeper, but try not to make single suction try longer than 10-12 seconds. In necessary – continue suction after a ventilation break (during this pause you can carefully clap over the chest of the patient to remove mucus from lower airways to their upper parts). After suction is done clean the catheter and place it into the antiseptic solution.

Cleaning of the trachea through tracheostomy tube has 2 main points:

- inserting the catheter keep the side foramen of it's upper end open
- during the suction shut it with your finger to create negative pressure and aspirate mucus; taking it out don't forget to scroll it.

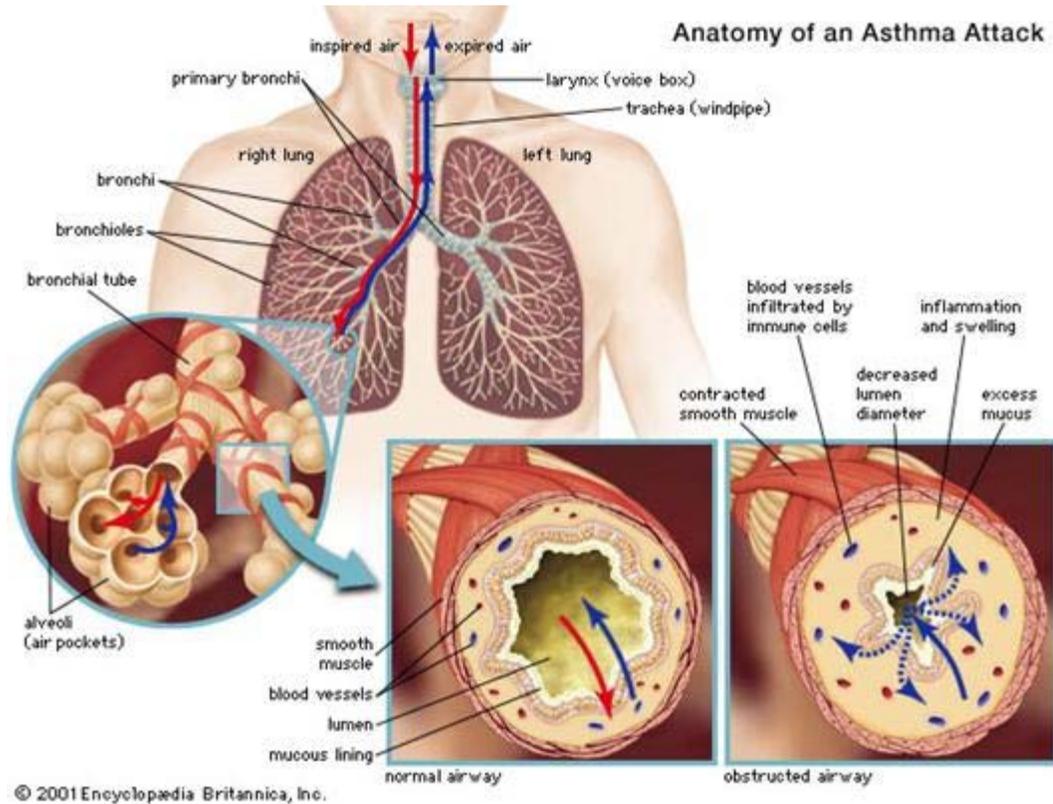
The same technique should be used for oral and nasal cavities (right and left nostril), but not the same catheter. Every catheter container with antiseptic should be signed.

5. Every hour change the position of the patient: make it lateral, supine or prone, Fowler's or Trendelenberg. Changing body position you change perfusion and ventilation correlations in the lungs and thus arterial blood becomes more oxygenated. This way appearance of bedsores is also prevented.

6. Every 12-24 hours change the endotracheal or tracheostomy tube.

7. In case of excessive mucus accumulation (worsening of auscultatory picture, x-ray negative dynamics) or atelectasis suspicion perform therapeutic bronchoscopy.

8. To prevent development of inflammatory reactions pay special attention to humidifying and warming of the ventilation mix, use aerosol inhalations, adequate infusion level, enteral and parenteral nutrition, state of the immune system and choose antibiotics rationally.



Status asthmaticus

Status asthmaticus is an acute exacerbation of asthma that is caused by bronchial spasm and does not respond to standard treatments of bronchodilators and steroids. It occurs when parasympathetic-sympathetic balance is greatly violated. Pathological stimulation of vagus causes spasms of smooth bronchi muscles, what brings increasing of resistance worsening of their patency. In addition excessive secretion and oedema of bronchial walls make airways almost not conductive.

Such exacerbation can be caused by allergens, stress or endocrinological factors.

Main clinical asthma symptom is dyspnea attack, which begins with dry cough. Those attacks are sudden; patients feel more comfortably sitting in bed with their arms based on bed. This position is called orthopnea, it help to use additional muscles (abdominal, muscles of the shoulder girdle) in respiratory act. Extreme wheezing is heard at distance. Inspiration is extremely short and expiration much prolonged and forced. Lack of mucus is rather warning

symptom. In case of complete obturation “silent lung” appears: breathing is asymmetric, there are silent parts of the lungs which do not contain any auscultative phenomena. Patients suffer from hypoxia and hypercapnia which earlier or later result in unconsciousness.

Treatment.

Most asthma attacks are successfully treated with inhalation of short acting β_2 -adrenoreceptor agonists (SABA), such as salbutamol. Anticholinergic medications, such as ipratropium bromide, might be used in addition for severe cases. Steroids are generally considered the most effective treatment available for long term control. When neither steroids, nor adrenomimetics work for treatment of a long and severe attack (or series of attacks)

In case of status asthmaticus spasm of bronchioles, inflammation and edema of bronchial wall and violation of mucus excretion and evacuation (due to abnormal cilia work) create pathogenetic base. Hyperventilation leads to greater water loss, what makes surface of bronchi dry and mucus more thick. This sick mucus can not be evacuated from airways during cough and thus it obstructs small bronchi and bronchioles, making ventilation impossible.

There are two types of status asthmaticus: anaphylactic and metabolic. Anaphylactic type is connected with allergic and immune reactions. It might appear after administration of some medicines (antibiotics, enzymes, aspirin, etc.) and develops very fast. Metabolic type of status asthmaticus is caused by metabolism disorders, which block β -adrenoreceptors. Long-term administration of sympatomimetics (asthmopent, ephedrine) can also cause metabolic type of status asthmaticus.

Status asthmaticus has certain stages:

1 stage: relative compensation. Patients are cyanotic, restless, sitting. Dyspnea has expiratory character with respiratory rate 26-40 per minute. Heart rate is 120 per minute, blood pressure in most patients is elevated. Auscultatory picture shows sibilant rales, prolonged expiration and “mosaic” breathing.

2 stage: decompensation. General condition of the patient is very serious. Patients are exhausted, sometimes excited and aggressive. Skin is cyanotic, wet. Respiratory rate might be 60 per minute, breathing is shallow, noisy at distance and extremely weak during auscultation, sometimes “silent lung” is observed. Heart rate is 120-140 per minute, blood pressure lowers to 80 mm of Mercury. Hypoxemia and hypercapnia cause mixed respiratory and metabolic acidosis.

3 stage: hypoxic coma. General condition of the patient is extremely severe. Patients are unconscious, convulsions are possible. Skin is cyanotic, covered with clammy sweat. Pupils are wide, reflexes are depressed or pathologic reflexes appear. Respiratory rate is over 60 per minute (sometimes acute bradypnea); auscultation picture – “silent lung”. Heart rate is over

140 per minute, blood pressure lowers to critical values. Biochemical blood test results show mixed decompensated acidosis ($\text{pH} < 7,2$) and serious homeostatic disorders. Acute cardiopulmonary insufficiency may lead to death.

Intensive treatment of status asthmaticus.

1. Begin oxygenation as soon as possible; if there is such possibility you should add 20%-30% of helium to the gas mix.

2. Provide constant monitoring of vital functions (ventricular fibrillation is one of possible death reasons of these patients).

3. Get intravenous access and start infusion of warm saline (polarizing solution, etc.). In case of extreme dehydration total infusion volume might reach 5-7 liters per day. In case of metabolic acidosis use 4% solution of sodium bicarbonate (100-150 ml).

4. Use steroids: solution of hydrocortisone (intravenously 150-200 mg every 2-3 hours), solution of prednisolone (60 mg every 4-6 hours), solution of dexamethason (8-16 mg every 6 hours).

5. Add to the treatment spasmolytics, antihistaminic, sedative medicines (10 ml of 2,4% ephillin solution; 2 ml of 2% no-spa solution, 1 ml of 2% dimedrol solution, 2-4 ml of 0,5% solution of diazepam or 10-20 ml of 20% solution of sodium oxybate intravenously slowly). In the past for some cases halothane-nitrous oxide mix was used to fight with bronchi spasm.

7. In case of progressive cardiac insufficiency use heart stimulating medicines such as cardiac glycosides (0,5 ml of 0,05% solution of strophanthin with saline intravenously). 7. If patient's condition is not getting worth and previously used treatment failed perform the intubation and begin artificial ventilation.

Indications for artificial ventilation in case of status asthmaticus:

- unconsciousness
- low blood pressure (< 70 mm Hg)
- tachycardia (over 140 per minute)
- $\text{pCO}_2 > 60$ mm Hg, $\text{pO}_2 < 60$ mm Hg, $\text{pH} < 7,25$

In those cases patients might receive halothane-oxygen narcosis. Therapeutic bronchoscopy should be performed with warm saline lavage (50-100 ml with its further suction, nearly 1,5 liters per one bronchoscopy). During lavage try to change patients position

in order to remove more mucus cloths.

There are many important points in general asthma treatment. Among them: rational antibiotic treatment, elimination of allergens, treatment of comorbidities and adequate asthma attacks treatment, in order to avoid development of status asthmaticus.

Chest injuries

Multiple rib fractures, especially that causing flail chest, violate greatly biomechanics of breathing. Clinically one can see reduction of chest excursion, retraction of certain chest wall parts and asymmetric breathing movements. Patients show decreasing of tidal volume, cyanosis, violations of haemodynamics, microcirculation disorders and depression of central nervous system.

Rib fractures can also cause hemothorax and pneumothorax. One of the most dangerous complications of chest injury is a tension pneumothorax, which end with a severe hypoxia, mediastinum organs dislocation and thus death.

Chest trauma patients should be treated very carefully: use all your diagnostic possibilities, including inspection, palpation, percussion, auscultation and additional laboratory and instrumental tests. Especially important is such approach with unconscious victims.

The choice of treatment depends on specific type of injury and its complications: hemothorax and pneumothorax need to be drained; tension pneumothorax should be punctuated (transformed into open pneumothorax); ineffective spontaneous ventilation should be replaced with artificial; antibiotics and painkillers are generally recommended.

“Coronary Café-syndrome“

This name is used for a sudden asphyxia, which appears when foreign body gets between the vocal cords and obturates the airways.

It is caused by a violation of swallowing and breathing. Imagine a situation in which the victim speaks loudly during the meal and then suddenly stops talking and jumps to his feet. His efforts to inhale are ineffective, hands try to free the neck rending the clothes, face gets cyanotic and swollen, eyes get filled with fear. In 3-4 minutes patient losses the consciousness, convulsions begin and finally clinical death appears (respiratory efforts cease, pulse gets weaker, defecation and urination are possible).

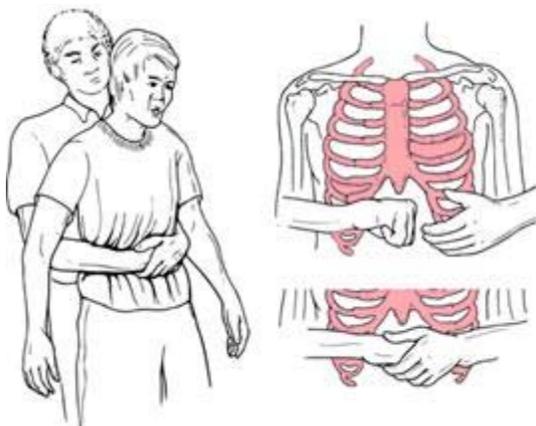
Thus, during acute foreign body obturation of glottis we can see 3 subsequent stages:

- 1- patient conscious, on his feet

- 2- patient unconscious, convulsions develop
- 3- clinical death (lasts from 5-th to 10-th minute)

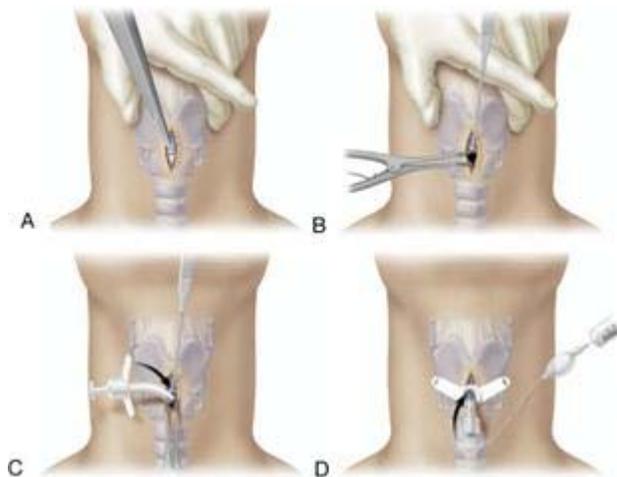
On the first stage doctor or any health care system professional (even skilled witness who has no medical education) should try to perform Heimlich manoeuvre. Stand behind the patient and wrap your arms around the victim's waist (hands on the epigastrium) and start making inwards and upwards thrusts. If the patient is still able to "collaborate" ask him to inhale during the manoeuvre.

Heimlich Manoeuvre



This manoeuvre allows to create a high pressure in the windpipe of the patient and thus exhaled air forces the foreign body to get out from the glottis. If the try will be unsuccessful only urgent conicotomy will save the life of the patient.

Procedure of conicotomy: put a roll (improvised one if necessary) under the neck of the patient, extending his neck as much as possible. In case of convulsions just ask someone to hold the patient. Then cover tightly with the first and third fingers of your left hand the thyroid cartilage and "slide" down with your index finger to the fossa, which divides thyroid and cricoid cartilages. At this place the thickness of the membrane is only few millimetres, so it can be easily pricked with a sharp object (for example kitchen knife). Limit the blade of the knife to 1cm long with the fingers of your right hand and sliding over the nail of your left hand index finger prick or cut the membrane. If it is possible insert a tube into the opening you made to prevent it's closing (you can even use your ball pen for this purpose). The faster this manipulation will be done, the higher are chances of the patient to get fully recovered.



Conicotomy consists of 2 phases:

- A. Cutting of the membrane with a scalpel with limiting its cutting length (with a plaster). It is done to save the posterior wall of trachea from injuring.
- B. Restoring of airways patency with a needle (puncture of the membrane).

If the victim is in a state of clinical death (the third stage) perform the conicotomy immediately and start CPR. You can ventilate the patient through the conicotomy tube.

Advanced medical help includes direct laryngoscopy and extraction of a foreign body with a forceps. If necessary those patients can be treated with a bronchoscopy or tracheostomy.

Laryngospasm

Laryngospasm is a pathological muscular contraction of the laryngeal cords, which manifests itself with a difficult or impossible inspiration. It appears as a consequent of pathological reflex or excessive stimulation of vagal nerve.

Usual reasons for laryngospasm are traumatic operations on reflex zones (vocal cords, trachea bifurcation, epiglottis, eye bulbs, periosteums). Irritation of vocal cords by food, saline, gastric contents, chemical or thermal stimuli as long as allergic reaction are the most common reasons for laryngospasm.

There are two types of laryngospasm: total and partial. Both cases are sudden and characterized by an inspiration dyspnea. Patient tries to inhale, his respiratory muscles contract, retracting intercostals spaces, jugular and supraclavicular fossae. During partial laryngospasm a small amount of air gets through the vocal cords and the sound of this air movement reminds cry of a rooster. Total laryngospasm is silent (aphonic). Within few seconds patient becomes cyanotic, his blood pressure and heart rate rise, later victim becomes unconscious, reflexes are inhibited. In some cases after this muscles of larynx relaxes

spontaneously and thus laryngospasm stops. But if deep hypoxia lasted for a while cardiac arrest still might appear.

Immediate aid

- begin oxygenation through the face mask as without proper oxygen supply partial laryngospasm becomes total

- whatever the reason of laryngospasm was eliminate it (remove the stimulator from the reflex zone if necessary, stop the operation for example)

- give intravenously atropine solution (0,01 mg per kilogram of body weight), ephedrine (5-10 ml of 2,4% solution, diluted in 10 ml of saline). If the initial damage was caused by thermal, chemical or allergic stimuli use antihistamine medicine (2 ml of 2,5% promethazine solution, 2 ml of 2% dimedrol solution) and steroids (60-90 mg of prednisolone).

Intensive treatment

- inhalation of bronchial spasmolytics (salbutamol);

- repeat the medicines mentioned above;

- in case of partial laryngospasm start artificial respiration through the mask with high oxygen speed and large ventilation volume;

- if your previous actions are not successful – use depolarising muscle relaxants (give 10 ml of 2% suxamethonium solution intravenously) and after total muscle relaxation perform direct laryngoscopy and trachea intubation. Of course if you have time and possibilities previously use hypnotics (for example 5-10 ml of propofol solution);

- if there are no conditions for intubation or your tries were unsuccessful, but situation is worsening into life-threatening (extreme bradycardia, unconsciousness, blood pressure less than 70 mm of Hg, general convulsions) perform conicotomy with the equipment you have and insert tube for oxygenation;

- in case of cardiac arrest start CPR according to general standards.

Bronchial spasm

Bronchial spasm is an acute disorder of external respiration caused by spasm of small bronchi smooth muscles. It can be total or partial. The reasons are the same as those for laryngospasm. Treatment for bronchial and laryngeal spasm are much alike, however muscle relaxants and conicotomy are not effective and thus death is almost unavoidable. What could be really useful is prophylactic premedication: before operations on reflex zones use atropine solution (0,01 mg per kg of body weight) in order to avoid pathological reflexes.

3.4 Medical operations and manipulations.

Usage of laryngeal mask.

Laryngeal mask is an elastic plastic tube with mask-shaped distant ending. It was invented in 1982 by British anaesthetist Archie Brain and now is popular worldwide among ambulance rescuers and anaesthesiology specialists.

Indications: providing of airways patency in case other methods failed; artificial ventilation during anaesthesia if intubation is not required.

Required equipment: laryngeal masks of different sizes, syringe (10 or 20 ml); equipment for oral cavity opening and cleaning if necessary (vacuum suction for example).

Procedure: open the mouth and clean the oral cavity if necessary. Staying on the right side of the patient use fingers of your left hand to press the chin down and thus open the mouth. Insert the laryngeal mask to the oral cavity keeping it's mask-shaped end with your index and long fingers. Glide it downwards and backwards along the tongue until you will feel a definitive resistance. After placement fill the inflatable cuff with air using the syringe: this way cuff will surround the laryngeal framework and air will be insufflated directly to the airways. The free end of the LMA can be connected to the AMBU bag or respiratory apparatus. Nowadays laryngeal masks have a variety of modifications: some of them even allow intubation, some are made with non-inflatable cuffs (i-gels), etc.

Tracheostomy

There are 3 types of tracheostomy according to anatomic placement of the incision: upper medial and low.

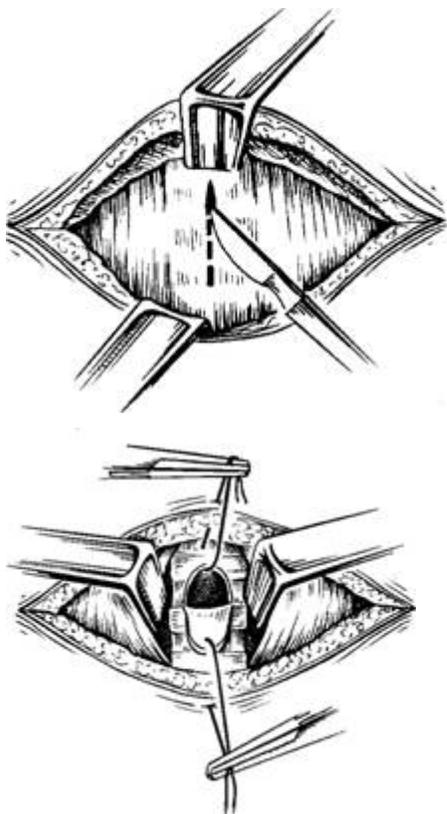
Indications: violations of airways patency in case of tumors, laryngeal stenosis; prolonged artificial ventilation of ITU patients.

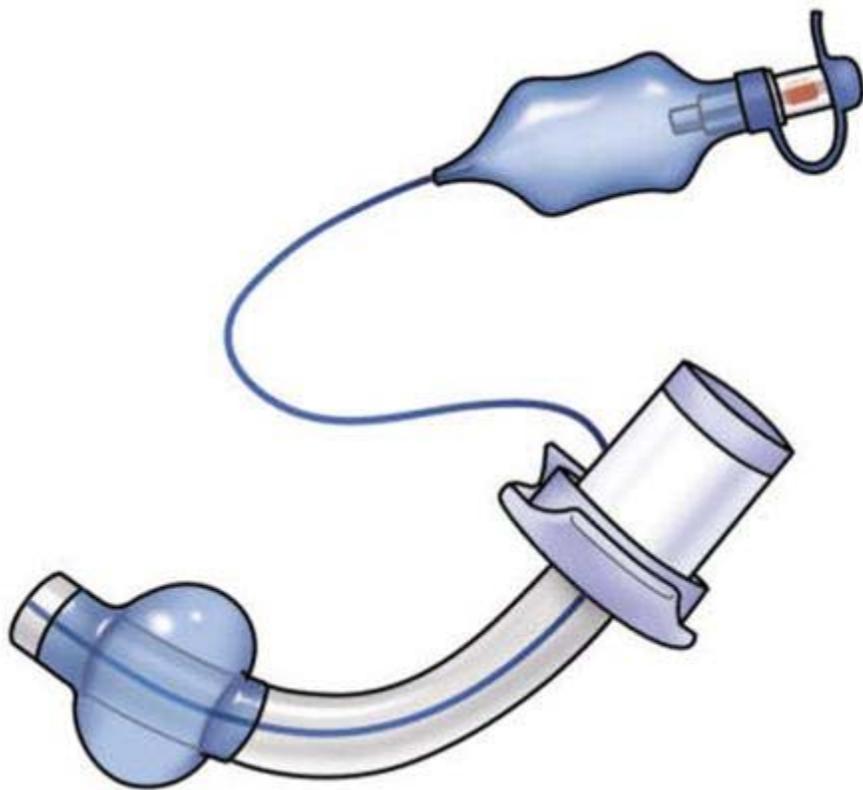
Required equipment: tracheostomy tubes of different sizes, scalpel, clamps, tissue retraction blades, stitch material and dressing, antiseptics, electric suction, respiratory apparatus, anaesthetics.

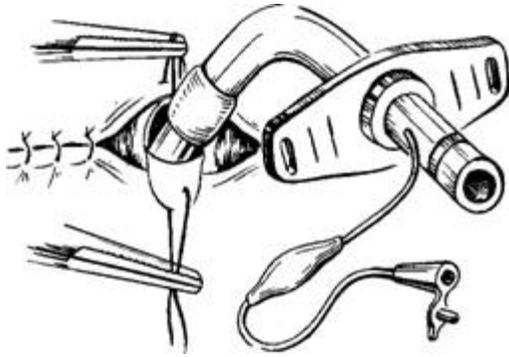
Procedure: perfect conditions for tracheostomy are those in operating room, however it can be done in ITU with specific antiseptic and aseptic measures.

After antiseptic preparation of the operative field immobilize the trachea with fingers of your left hand and cut the skin strictly along the midline of the neck right under the thyroid cartilage (the length of the incision should be 4-5 cm). After skin, hypodermic tissue and aponeurosis are cut start blunt separating of the cricoid cartilage and pull it upwards with a surgical hook. Cut the fascia, which covers the thyroid isthmus, along the lower edge of the cartilage and with a blunt hook pull the isthmus down. Make horizontal incision between the second and third or third and fourth tracheal rings, then cut one or two

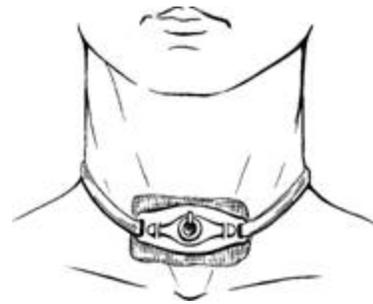
distal tracheal rings and insert the tube (use dilatator if necessary). Don't make opening too large, not more than 1/3 of trachea diameter. After tube is fixated put an aseptic bandage around the wound.







Pic.3.3



Pic.3.4

3.5 Control tests

1. Normally partial pressure of oxygen in alveoli is:

- A. equal to atmospheric pressure
- B. $80 \pm$ mm of Mercury
- C. 140 ± 5 mm of Mercury
- D. 100-110 mm of Mercury
- E. during inspiration is equal to arterial partial oxygen pressure, during expiration – equal to partial oxygen venous pressure

2. Arterial blood is saturated with oxygen for:

- A. 100%
- B. 96-97%
- C. 90-95%
- D. 85-95%
- E. percentage depends on the character of breathing

3. Partial pressure of carbon dioxide in arterial blood is:

- A. depends on partial oxygen pressure
- B. depends on intensity of tissue breathing
- C. normally is 36-44 mm of Mercury
- D. increases in case of hyperventilation

- E. depends on haemoglobin dissociation curve
4. Which pathology does not need artificial respiration?
- A. Kussmaul breathing
 - B. Biot's breathing
 - C. Cheyne-Stokse breathing
 - D. lowering of pO_{2a} to 60 mm of Mercury
 - E. respiratory rate over 40 per minute
5. What should be done for hypercapnia treatment?
- A. sodium bicarbonate solution given
 - B. artificial ventilation started
 - C. cytochrome C given
 - D. oxygenation started
 - E. blood of the same group transfused
6. What is a method of laryngospasm treatment?
- A. muscle relaxants and artificial ventilation
 - B. respiratory stimulants (cordiamin, lobelin)
 - C. sodium bicarbonate
 - D. diuretics
 - E. tracheostomy
7. What should be done to minimize the damage of prolonged (over one week) artificial ventilation?
- A. intubation
 - B. tracheostomy
 - C. conicotomy
 - D. respiratory analeptics should be given
 - E. therapeutic lavage
8. What should be done in order to prevent laryngospasm?
- A. peripheral M-cholinolitics should be given
 - B. oral cavity should be cleaned
 - C. spasmolytics should be given

- D. artificial ventilation should be started
 - E. jaw should be thrust forward
9. What should be done in case of foreign body in the airways?
- A. tracheostomy
 - B. change of body position – upper part should be lowered
 - C. mouth-to-mouth ventilation
 - D. Heimlich manoeuvre
 - E. patient should be punched between blade bones

Task 1.

Patience, the patient of 56 years, was transported by ambulance to the hospital reception with breathlessness and difficult breathing. He is restless, excited: his skin is cyanotic and dry. Respiration rate is 26, respiratory movements are deep, additional muscles participate respiration. Blood pressure is 180/110 mm of Mercury, heart rate – 106 per minute. Gasometry results are: pO_2A 67 mm of Mercury, pCO_2a – 49 mm of Mercury. Name the disorder and principles of its treatment.

Task 2.

Calculate normal spirometry indexes of Andrew, medical student of 22 years (height 170 cm, weight 70 kg): respiratory rate, tidal volume, minute ventilation, dead space volume, alveolar ventilation.

Task 3.

Patience, the patient, during cleaning of his nasal cavity suddenly felt, that inspiration became difficult. His skin gets cyanotic, blood pressure increases. Name the type of complication and describe the urgent help, which is necessary in this situation.

Task 4.

Andrew, the medical student, during a casual meeting in a café was laughing and at the same time chewing his sandwich. Suddenly he became quiet, grabbed his neck and stopped breathing. His face turns blue.

what should be done immediately

What should be done if Bonnie, student of medicine, has convulsions. He is unconscious, his pulse is weak. Describe CPR.

What will be your decision if You find Clyde, student of medicine, 3 minutes after cardiac arrest?

Task 5.

Patience, the patient, was transported to the hospital in a severe condition: unconscious, skin cyanotic, respiratory rate 64 per minute, heart rate 124 per minute, blood pressure 90/60 mm of Mercury, body temperature – normal. Choose the type of help and algorithm of your actions.

Task 6.

Patience, the patient, suffers from bronchial asthma. After regular ambulance visit he doesn't feel better, although medicines were given intravenously. His condition is critical: central nervous system depressed, skin is wet and cyanotic, expiration is prolonged greatly, sibilant rales are combined with “silent lung” parts. Blood pressure is 110/70 mm of Mercury. Heart rate is 116 per minute. Describe your immediate actions and algorithm of intensive care.

Student should repeat next questions

Violations of homeostasis and their correction.

9.1 The importance of the water to the organism.

Life on earth was born in the water environment. Water is a universal solvent for all the biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated

from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the aerial walls, bar receptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals reveal into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution). This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.

9.2 Importance of osmolarity for homoeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hypoosmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.

9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobin concentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, 0,6* BW volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

9.4 Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K- potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

$0,6 * \text{BW}$ – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na- sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

0,2*BW – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonic overhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 BW,$$

Cl_d- chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

0,2*BW – extracellular fluid volume, l.

To treat hyponatremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chloride solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p - plasma magnesium concentration of the patient, mmol/l

0,6*BW - extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d - calcium deficiency, mmol

Ca_p - plasma calcium concentration of the patient, mmol/l

0,2*BW - extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorption.

9.5 Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO₂. Normally pCO₂ of arterial blood is 36-44 mm Hg. Hypercapnia (pCO₂ increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO₂ less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H⁺ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is 0±1,5. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat

acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H⁺ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H⁺ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patience, the patient of 48 years and body weight 88 kg.

Task 2.

Patience, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patience, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patience, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patience, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patience, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration 88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

Task 7.

Patience, the patient of 50 with body weight 75 kg, was transported to the admission unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow

breathing. Blood tests show: pH 7,18, pCO_{2a} 78 mm Hg, pO_{2A} – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patience, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, pCO_{2a} 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

Life is provided through a variety of mechanisms, however all of them depend on proper circulation. Circulation itself consists of 2 parts: work of heart (pump of the body) and vessels, through which blood is pumped to the most remote organs and tissues. During every systolic contraction heart pump 70-80 ml of blood (so called stroke volume). Thus in case of heart rate 70 beats per minute heart pumps nearly 5 liters of blood, what makes more than 7 tones per day.

From the left ventricle blood gets to the arterial system of the systemic circuit. Arteries contain 15% of the whole circulating blood volume; they carry blood from the heart to their distal departments – arterioles (vessels of resistance). Arterioles themselves are defining blood distribution: in condition of constriction (spasm) they make blood supply of the capillaries impossible (ischemia appears). On the contrary, in condition of dilatation they provide maximal oxygenation. When arterioles are blocked due to the spasm blood is flowing through the arterio-venous anastomosis directly to the venous system.

Distribution of blood in the vascular bed (% of CBV).

- a. heart cavity 3%
- b. arteries 15%
- c. capillaries 12%
- d. venous system 70%

Among the natural vasoconstrictors (agents, which cause constriction of the blood vessel) are epinephrine, norepinephrine, serotonin, angiotensin II. Stress enhances the secretion of catecholamines, their blood concentration increases and arterioles constrict. Spasm of the arterioles is the basis of blood flow centralization: peripheral flow is disregarded in order to provide brain with the oxygenated blood as long as possible. To the group of vasodilators (agents, which provide dilatation of the vessels) belong “acid” metabolites (lactate, pyruvate,

adenylic acid, inosinic acid), bradykinin, acetylcholine, different medicines (neuroleptics, α -adrenergic antagonists, peripheral vasodilators, ganglionic blocking agents, etc.), some exogenous poisons. All of them cause blood flow decentralization: opening of arterioles and distribution of the blood from central vessels to the capillary bed.

Capillaries are the interweaving network of the smallest body vessels with the general length of 90-100 thousands of kilometers. However simultaneously work only 20-25% of them. They provide metabolic exchange bringing oxygen and nutrients to the tissues and take back wastes of metabolism. Periodically, every 30-40 seconds one of them get closed and others open (vasomotion effect). Capillaries contain 12% of the whole circulating blood volume, but different pathological conditions can increase this amount even 3 and more times.

“Used” blood from the capillaries flows to the venous system. Veins are the blood reservoir, which contains 70% of the total circulating blood volume. Unlike arteries they are capable of volume control and thus they influence the amount of blood, which returns to the heart.

The most important haemodynamic index of venous system is central venous pressure. CVP represents the pressure which blood causes to the walls of cava veins and right atrium. This parameter is an integral index of circulating blood volume, systemic vascular resistance and pump function of the heart. It can be measure with a special device called “phlebotonometer” (pic. 4.9) or with a usual infusion set and a ruler. Normally CVP measured from the sternum point is 0-14 cm H₂O and from midaxillary line is 8-15 cm H₂O.

Central venous pressure decreases (sometimes even to negative) in case of:

- blood loss
- excessive water loss (dehydration)
- distributive shock (decrease of peripheral resistance due to venous and arterial dilatation)

In those conditions decreases volume of blood returning to the heart and thus suffers cardiac output. In case of negative CVP cardiac arrest is highly probable.

Central venous pressure increases in case of:

- heart failure (insufficiency of left or right ventricle)
- hypervolemia (excessive blood infusion, improper infusion therapy)
- obstructions to blood flow (pulmonary embolism, cardiac tamponade, etc.)

When CVP over 15-16 cm H₂O is combined with left ventricle insufficiency the risk of pulmonary edema is very high.

Blood pressure is an integral index of arterial part of systemic haemodynamics. Talking about blood pressure we may refer to systolic, diastolic, pulse and mean arterial pressure. Systolic (P_{syst}) and diastolic (P_{diast}) pressures are measured with the manometer (method with the usage of phonendoscope was invented by M. Korotkoff). Pulse pressure (PP) is a difference between systolic and diastolic blood pressure.

Mean arterial pressure (MAP) is calculated according to the formula:

$$\text{MAP} = P_{\text{dias}} + 1/3 \text{ PP} \quad \text{mm Hg}$$

MAP defines the level of pressure necessary for the metabolic exchange in the tissues. Its measurement allows the evaluation of tissue perfusion level.

Blood pressure depends on different factors, but the most important are cardiac output and vascular resistance (mostly arterioles). This dependence is direct, thus you can increase blood pressure using:

- infusion of vasoconstrictors - solutions of epinephrine, phenylephrine (mesaton), etc. (they will increase the vascular resistance);
- infusion of hydroxyethyl starch solutions or saline (they will increase circulating blood volume)
- infusion of cardiac glycosides or other medicine which stimulate myocardium

General volume of blood in the body of a healthy adult is nearly 7% from the body weight: 70 ml per kilogram for male and 65 ml per kilogram of body weight for female. Of course circulating blood volume is lower, because part of blood is out of metabolic processes as a reserve. CBV can be measured with the infusion of coloring substance to the blood flow (Evans blue, polyglucin) and later evaluation of its dissolution degree.

Therefore measurement of CVP, BP, cardiac output and circulating blood volume allow to evaluate condition of circulation system of the patients and to provide adequate correction.

4.2 Acute heart failure; shock and collapse.

Acute cardiovascular failure is a state of cardiac and vascular inability to provide adequate supply of tissue metabolic needs with oxygenated blood and nutrients. This, earlier or later, causes cellular death.

The reasons of the failure vary greatly: mechanic injuries, blood loss, burns,

dehydration, exogenous and endogenous intoxications, immediate hypersensitivity reaction, ischemic heart disease, neural and humoral regulation disorders of vascular tone.

Acute cardiac failure is a disorder of heart pumping action. It develops due to primary heart problems or secondary, under the influence of extracardiac factors such as infection or intoxication. There are two types of heart failure: left-sided and right-sided.

Left-sided heart failure is an inability of left ventricle to pump blood from the pulmonary circuit to the systemic circuit. The most common reasons of it are myocardial infarction, mitral insufficiency, left AV valve stenosis, aortic valve stenosis, aortal insufficiency, hypertonic disease, coronary sclerosis, acute pneumonia.

Coronary circulation is possible only during the diastole and in those conditions every violation of coronary passability decreases cardiac output. This way during the systole part of the blood is not injected into aorta, but stays in the left ventricle. Diastolic pressure in the left ventricle increases and blood is literally forced to stagnate in the left atrium. At the same time right ventricle functions normally and continues to pump usual amounts of blood to the pulmonary circuit. Thus hydrostatic pressure in the vessels of pulmonary circulation increases, fluid part of the blood moves first to the lung tissue and then, through alveolar-capillary membrane, to the alveolar lumen.

Clinically pulmonary edema begins with dyspnea (during physical activity or rest). Later attacks of dyspnea are connected with persistent cough with white or pink blood-tinged phlegm. During the attack patient tries to sit as in this position breathing is easier. This condition is called “heart asthma”. When hydrostatic pressure is over 150-200 mm Hg, fluid part of blood moves to the alveolar lumen causing development of pulmonary edema.

Pulmonary edema is divided into interstitial and alveolar edema.

Interstitial edema is a condition during which serous part of stagnated in the pulmonary circuit blood infiltrates the lung tissue, including peribronchial and perivascular spaces.

During alveolar edema not only the plasma, but also blood components (red and white blood cells, platelets) get out from the vessels. During the respiratory act blood mixes with the air creating large amount of “foam”, which violates gas exchange. This way, in addition to circulatory hypoxia, hypoxic hypoxia appears.

Condition of the patient gets worse quickly. Sitting position is optimal, but not as helping as previously. Respiratory rate is nearly 30-35 breathes per minute, but attacks of breathlessness are constant. Skin is pale with acrocyanosis. Hypoxia of central nervous system usually causes psychomotor agitation. Respiratory acts are noisy; during cough pink blood-tinged phlegm is released. Auscultation allows you to hear different wet rales, sometimes it's even possible to hear them standing aside the patient without phonendoscope.

Pulmonary edema can be also divided according to the blood pressure level: the one with elevated pressure is caused by a hypertonic disease, aorta valve insufficiency or disorders of cerebral perfusion; another one is caused by total myocardial infarction, acute inflammation of myocardial muscle, terminal valve defects, severe pneumonia and is characterized with normal or low blood pressure.

Immediate aid

- make sure patient is sitting with his legs down (orthopnea)
- provide oxygenation through nasal catheter (before placing oil it with glycerin, insert it to the depth of 10-12 cm – distance from the wing of the nose to auricle) or face mask. Do not use Vaseline, because it can burn in atmosphere with high concentration of oxygen.

However if catheter is not deep enough patient will suffer from an unpleasant “burning” feeling, because oxygen flow will dry mucosa layer of the nasal cavity; also in this situation concentration of oxygen will be lower than expected.

- put venous tourniquets on the limbs in order to reduce amount of blood returning to heart: venous bed of limbs can reserve up to 1,7 liters of blood;
- constantly control heart and kidney activity (ECG, SaO₂ , and blood pressure are checked automatically through the monitor; to control diuresis you should insert Foley catheter;
- catheterize central vein, because amount of infusions should be based on central venous pressure;
- use medical “defoamers” if they are available (ethyl alcohol or antiphomsylan solution) combined with oxygen inhalation

Scheme of oxygenation set connected to “defoamer” container

- a. oxygen source (cylinder with oxygen)
 - b. tube with numerous holes sunk into container with defoamer
 - c. tube for humidified oxygen (its opening should be over the level of fluid);
 - d. patient
- medical treatment: 1% morphine solution (decreases intravascular pressure of pulmonary circuit, inhibits respiration center in medulla oblongata preventive dyspnea progress, sedates patient);
 - solutions of diuretics are used to decrease the circulating blood volume (6-12 ml of 1% furosemid solution, solution of ethacrynic acid), however be careful with

them in case of low blood pressure; diuretic effect will last up to 3 hours after i/v infusion, the expected diuresis is 2-3 liters

- if blood pressure allows you can try to use nitroglycerin to reduce intravascular pressure of pulmonary circuit (1 or 2 tablets with 10 minutes interval)
- cardiac glycosides for improvement of heart action (0,025% digoxin solution, 0,05% strophanthin solution, 0,06% corglicon solution);
- in case of high pressure (over 150 mm Hg) use ganglionic blocking agents (1 ml of 5% pentamin solution diluted in 150 ml of saline, give i/v slowly; diluted with saline 250 mg of trimethaphan solution), because they reduce pressure in pulmonary circuit and lower the amount of blood getting to right half of the heart, however be careful with the dosage and monitor blood pressure level carefully;
- never use osmotic diuretics in case of pulmonary edema – they will increase blood volume and thus heart load!!!
- when everything listed above failed and patient is worsening with every second you should intubate him and start artificial ventilation with positive end expiratory pressure (begin with 4-6 cm H₂O)

Right-sided heart failure is an inability of right ventricular to pump blood from systemic circuit to the pulmonary circuit due to its weakness or an obstruction to the blood flow.

It occurs in case of pulmonary embolism, right ventricular infarction, excessive infusion therapy (especially including citrated blood) for patients with heart insufficiency, lung diseases (bronchial asthma, emphysema, pneumosclerosis) which cause increase of right ventricular load.

Patients have acrocyanosis, tachycardia, dyspnea, pronounced neck veins, ankle swelling, enlarged liver, ascytis. Central venous pressure is highly increased (up to 20-25 cm H₂O), however pulmonary edema does not appear.

Intensive treatment is mostly pathogenetic:

- limit the infusions (give only life-necessary solutions, check the water balance of the patient and reduce drinking water if necessary);
- in case of citrated blood transfusions use 5-10 ml of 10% calcium gluconate solution per every 500 ml of blood to prevent hypocalcaemia;
- in case of bronchial spasm use bronchial spasmolytics;

- to remove excessive fluid from the body use diuretics (furosemide solution for example);
- metabolic acidosis is corrected with 4% solution of sodium bicarbonate (i/v slowly with acid-base state control);
- in case of pulmonary embolism anticoagulants are used – fraxiparine 0,6 mg subcutaneously; heparin solution – 5000 IU every 4 hours; fibrinolytic drugs (streptokinase, fibrinolysin, urokinase, etc.)

Shock is a pathological state which can be described as a tissue hypoxia caused by hypoperfusion. Pathogenetic basis of shock depends on its reason (trauma, toxins, thermal injury) and at the same time on reactivity of the organism (level of defense mechanisms mobilization).

Stimulation of sympathetic nervous system - production of catecholamines and other vasoactive substances by hypothalamus and adrenal glands are the universal response of the body to the stress. Those mediators interact with the receptors of peripheral vessels causing their constriction and at the same time they dilatate the vascular bed of life-important organs. This is so called “centralization of the flow”: rational decrease of blood flow in less important tissues (skin, organs of abdominal cavity, kidneys) in case of aggressive external influence for protecting life itself (brain, heart, lungs).

However influence of shock agents (pain, hypovolemia, destroyed cells, toxic metabolites), extended microcirculation violations (vascular spasm, microthrombosis and sludge) and caused by them tissue ischemia lead to hypoxic affection and cellular death of the internal organs. Further it can bring multiple organ dysfunction syndrome.

Collapse is a vascular failure. It occurs when body is not able to provide blood flow according to the new level of its needs (either because reaction is not fast enough or because sympathetic activation fails). Vascular bed volume and circulating blood volume are disproportional: too much blood gets to the microcirculation vascular reserve and the amount, which returns to the heart is not enough for the systemic needs (so called “decentralization” of the blood flow). Cardiac output and blood pressure decrease, that causes hypoperfusion of the central nervous system and thus unconsciousness and life-threatening complications.

Collapse definition is a bit nominal, because if such reaction extends in time the state of shock develops. Shock itself can equally run as a vascular failure or as a sudden clinical death.

Pathogenetic classification of shock (according to P. Marino, 1998):

- hypovolemic
- cardiogenic
- distributive
- mixed (two and more factors).

Clinical classification of shock:

- traumatic shock;
- haemorrhagic shock;
- dehydration shock;
- burn shock;
- septic shock;
- anaphylactic shock;
- cardiogenic shock;
- exotoxic shock.

4 Shock caused by dehydration

It is a type of hypovolemic shock, which occurs during excessive body fluid loss (not blood, because hemorrhagic shock is another shock type).

Its reasons vary greatly:

- gastrointestinal diseases (profuse vomiting, diarrhea, loss of intestinal fluid through fistula);
- polyuria (uncontrolled diuretic treatment, diabetes mellitus and insipidus, diuretic phase of acute renal failure);
- fluid loss through skin and wound surface (burns, high fever);
- inadequate infusion treatment of postoperative or comatose patients;
- hyperventilation (rapid breathing, Kussmaul breathing, inadequate artificial ventilation parameters in case of apparatus without air humidification).

However not only the complete fluid loss can be the reason of shock, but also its pathological distribution into the extracellular space (intestinal cavity in case of intestinal paralysis, abdominal cavity in case of ascites, pleural cavity in case of pleurisy). This way will can also act prolonged heavy tissue inflammations (peritonitis) or massive injuries

(crush-syndrome).

In cases described above electrolytes are also lost (cations of sodium, potassium, calcium, magnesium; anions of chlorine, hydrocarbonate). It causes complex osmolar, acid-base and electrolytic disorders.

Stage of dehydration shock is evaluated according to the actual fluid loss:

less than 5% of body weight – mild dehydration

5-10% of body weight – moderate dehydration

over 10% of body weight – severe dehydration

Water deficiency brings lowering of cardiac output, blood pressure and central venous pressure (through decrease of blood volume returning to the heart, which leads to compensatory adrenergic vasoconstriction).

Dehydration causes body weight loss, skin and mucosa dryness, decrease of subcutaneous turgor and eyeballs tone, hypothermia, tachycardia, oliguria, thirst. While dehydration progresses compensatory mechanisms weaken and central nervous system suffers: patients become sluggish, confused; hallucinations, cramps and unconsciousness are also possible. Laboratory tests show blood concentration.

One of the most important things in treatment of dehydrated patients is daily balance of fluid: check it carefully through measuring of daily received and lost fluids (food, infusions, stool and urine output). In case of fever or tachypnea make necessary corrections. Balance should be calculated every 12-24 hours (special paper forms make this easier).

Daily fluid balance is calculated by adding all the received fluids (both enteral and parenteral ways) and deducting urine output, stool, perspiration and breathing water loss.

You should remember, that perspiration depends on body temperature: in case of normal temperature (36,6°C) patient loses 0,5 ml/kg of water during every hour; 1 degree of temperature elevation adds 0,25 ml/kg to normal value of 0,5 ml/kg.

According to the fluid balance infusion therapy is divided into positive (for dehydrated patients), negative (for overhydrated patients) and “zero” (for patients without balance disorders).

Water deficiency is calculated according to the formula:

$$W_{\text{def}} = (Ht_p - Ht_n) * 0,2 \text{ BW} / Ht_n,$$

W_{def} – water deficiency, l;

Ht_p – hematocrit of the patient, l/l;

Ht_n - normal hematocrit, l/l;

BW – body weight, kg.

Use crystalloids to treat water deficiency: saline solution, Ringer's solution, Ringer-lactate solution, electrolytic solutions, 5%, 10, 20% glucose solution. To control potassium concentration (during dehydration this cation is widely lost) prescribe polarizing GIK mixture (pic.9.4), but don't you ever infuse concentrated potassium solutions quickly – it can cause cardiac arrest (not more than 400 of GIK solution ml per hour).

The essential components of the human cardiovascular system are the [heart](#), [blood](#), and [blood vessels](#). It includes: the [pulmonary circulation](#), a "loop" through the [lungs](#) where blood is oxygenated; and the [systemic circulation](#), a "loop" through the rest of the body to provide [oxygenated](#) blood. An average adult contains five to six quarts (roughly 4.7 to 5.7 liters) of blood, accounting for approximately 7% of their total body weight.

While it is convenient to describe the flow of the blood through the right side of the heart and then through the left side, it is important to realize that both atria contract at the same time and that both ventricles contract at the same time. The heart works as two pumps, one on the right and one on the left that works simultaneously. The right pump pumps the blood to the lungs or the pulmonary circulation at the same time that the left pump pumps blood to the rest of the body or the systemic circulation. Venous blood from systemic circulation (deoxygenated) enters the right atrium through the superior and inferior vena cava. The right atrium contracts and forces the blood through the tricuspid valve (right atrioventricular valve) and into the right ventricles. The right ventricles contract and force the blood through the pulmonary semilunar valve into the pulmonary trunk and out the pulmonary artery. This takes the blood to the lungs where the blood releases carbon dioxide and receives a new supply of oxygen. The new blood is carried in the pulmonary veins that take it to the left atrium. The left atrium then contracts and forces blood through the left atrioventricular, bicuspid, or mitral, valve into the left ventricle. The left ventricle contracts forcing blood through the aortic semilunar valve into the ascending aorta. It then branches to arteries carrying oxygen rich blood to all parts of the body.

Blood Flow Through Capillaries

From the arterioles, the blood then enters one or more capillaries. The walls of capillaries are so thin and fragile that blood cells can only pass in single file. Inside the capillaries, exchange of oxygen and carbon dioxide takes place. Red blood cells inside the capillary releases their oxygen which passes through the wall and into the surrounding tissue. The tissue then releases waste, such as carbon dioxide, which then passes through the wall and

into the red blood cells.

The Circulatory System

The circulatory system is extremely important in sustaining life. It's proper functioning is responsible for the delivery of oxygen and nutrients to all cells, as well as the removal of carbon dioxide, waste products, maintenance of optimum pH, and the mobility of the elements, proteins and cells, of the immune system. In developed countries, the two leading causes of death, myocardial infarction and stroke are each direct results of an arterial system that has been slowly and progressively compromised by years of deterioration.

Arteries

Arteries are muscular blood vessels that carry blood away from the heart, oxygenated and deoxygenated blood . The pulmonary arteries will carry deoxygenated blood to the lungs and the sytemic arteries will carry oxygenated blood to the rest of the body. Arteries have a thick wall that consists of three layers. The inside layer is called the endothelium, the middle layer is mostly smooth muscle and the outside layer is connective tissue. The artery walls are thick so that when blood enters under pressure the walls can expand.

Arterioles

An arteriole is a small artery that extends and leads to capillaries. Arterioles have thick smooth muscular walls. These smooth muscles are able to contract (causing vessel constriction) and relax (causing vessel dilation). This contracting and relaxing affects blood pressure; the higher number of vessels dilated, the lower blood pressure will be. Arterioles are just visible to the naked eye.

Capillaries

Capillaries are the smallest of a body's vessels; they connect arteries and veins, and most closely interact with tissues. They are very prevalent in the body; total surface area is about 6,300 square meters. Because of this, no cell is very far from a capillary, no more than 50 micrometers away. The walls of capillaries are composed of a single layer of cells, the endothelium, which is the inner lining of all the vessels. This layer is so thin that molecules such as oxygen, water and lipids can pass through them by diffusion and enter the tissues. Waste products such as carbon dioxide and urea can diffuse back into the blood to be carried away for removal from the body.

The "capillary bed" is the network of capillaries present throughout the body. These beds are able to be "opened" and "closed" at any given time, according to need. This process is called autoregulation and capillary beds usually carry no more than 25% of the amount of

blood it could hold at any time. The more metabolically active the cells, the more capillaries it will require to supply nutrients.

Veins

Veins carry blood to the heart. The pulmonary veins will carry oxygenated blood to the heart while the systemic veins will carry deoxygenated to the heart. Most of the blood volume is found in the venous system; about 70% at any given time. The veins outer walls have the same three layers as the arteries, differing only because there is a lack of smooth muscle in the inner layer and less connective tissue on the outer layer. Veins have low blood pressure compared to arteries and need the help of skeletal muscles to bring blood back to the heart. Most veins have one-way valves called venous valves to prevent backflow caused by gravity. They also have a thick collagen outer layer, which helps maintain blood pressure and stop blood pooling. If a person is standing still for long periods or is bedridden, blood can accumulate in veins and can cause varicose veins. The hollow internal cavity in which the blood flows is called the lumen. A muscular layer allows veins to contract, which puts more blood into circulation. Veins are used medically as points of access to the blood stream, permitting the withdrawal of blood specimens (venipuncture) for testing purposes, and enabling the infusion of fluid, electrolytes, nutrition, and medications (intravenous delivery).

Venules

A venule is a small vein that allows deoxygenated blood to return from the capillary beds to the larger blood veins, except in the pulmonary circuit where the blood is oxygenated. Venules have three layers; they have the same makeup as arteries with less smooth muscle, making them thinner.

The double circulatory system of blood flow refers to the separate systems of pulmonary circulation and the systemic circulation in amphibians, birds and mammals (including humans.) In contrast, fishes have a single circulation system. For instance, the adult human heart consists of two separated pumps, the right side with the right atrium and ventricle (which pumps deoxygenated blood into the pulmonary circulation), and the left side with the left atrium and ventricle (which pumps oxygenated blood into the systemic circulation). Blood in one circuit has to go through the heart to enter the other circuit. Blood circulates through the body two to three times every minute. In one day, the blood travels a total of 19,000 km (12,000 miles), or four times the distance across the U.S. from coast to coast.

The Pulmonary Circuit

In the pulmonary circuit, blood is pumped to the lungs from the right ventricle of the heart. It is carried to the lungs via pulmonary arteries. At lungs, oxygen in the alveolae diffuses to the capillaries surrounding the alveolae and carbon dioxide inside the blood diffuses to the

alveolae. As a result, blood is oxygenated which is then carried to the heart's left half -to the left atrium via pulmonary veins. Oxygen rich blood is prepared for the whole organs and tissues of the body. This is important because mitochondria inside the cells should use oxygen to produce energy from the organic compounds.

The Systemic Circuit

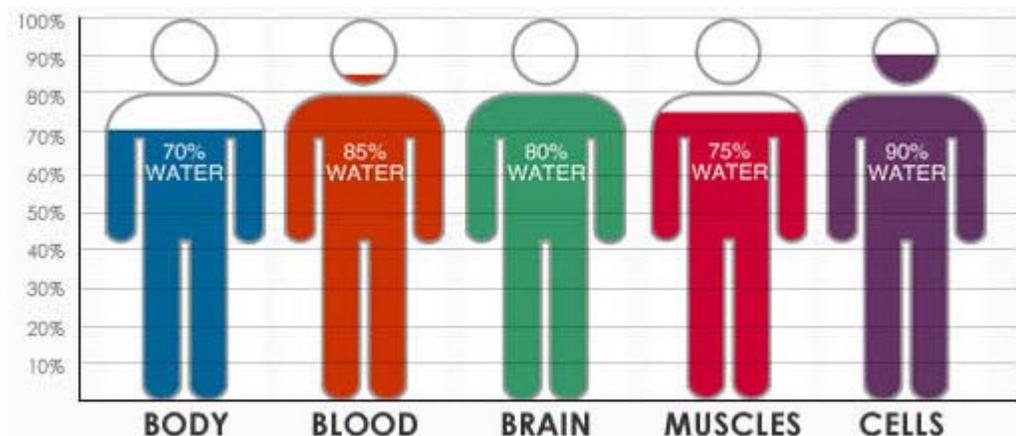
The systemic circuit supplies oxygenated blood to the organ system. Oxygenated blood from the lungs is returned to the left atrium, then the ventricle contracts and pumps blood into the aorta. Systemic arteries split from the aorta and direct blood into the capillaries. Cells consume the oxygen and nutrients and add carbon dioxide, wastes, enzymes and hormones. The veins drain the deoxygenated blood from the capillaries and return the blood to the right atrium.

Pic. 4.1 Distribution of blood in the body:

- a. hear cavity itself 0 3% (% of blood volume)
- b. arteries -15%
- c. capillares -12%
- d. venous system – 70%

Cardiac cycle is the term used to describe the relaxation and contraction that occur, as a heart works to pump blood through the body. Heart rate is a term used to describe the frequency of the cardiac cycle. It is considered one of the four vital signs. Usually it is calculated as the number of contractions (heart beats) of the heart in one minute and expressed as "beats per minute" (bpm). When resting, the adult human heart beats at about 70 bpm (males) and 75 bpm (females), but this rate varies between people. However, the reference range is nominally between 60 bpm (if less termed bradycardia) and 100 bpm (if greater, termed tachycardia). Resting heart rates can be significantly lower in athletes, and significantly higher in the obese. The body can increase the heart rate in response to a wide variety of conditions in order to increase the cardiac output (the amount of blood ejected by the heart per unit time). Exercise, environmental stressors or psychological stress can cause the heart rate to increase above the resting rate. The pulse is the most straightforward way of measuring the heart rate, but it can be deceptive when some strokes do not lead to much cardiac output. In these cases (as happens in some arrhythmias), the heart rate may be considerably higher than the pulse. Every single 'beat' of the heart involves three major stages: atrial systole, ventricular systole and complete cardiac diastole. Throughout the cardiac cycle, the blood pressure increases and decreases. As ventricles contract the pressure rise, causing the AV valves to slam shut.

Water-electrolyte and acid-basic disorders



9.1 The importance of the water to the organism.

Life on earth was born in the water environment. Water is an universal solvent for all the biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homoeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

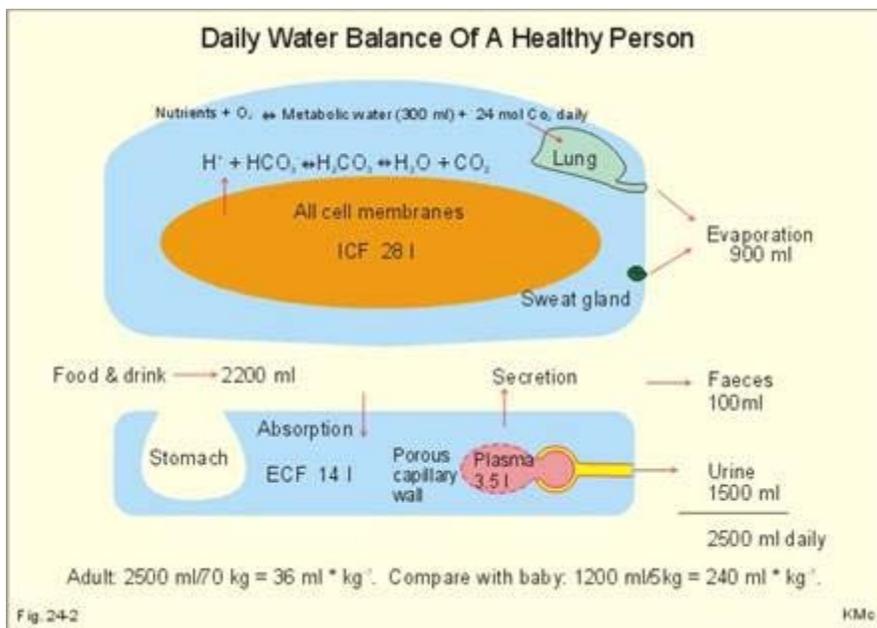
Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the atrial walls, baroreceptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals release into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution). This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.



9.2 Importance of osmolarity for homeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves

through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hyposmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

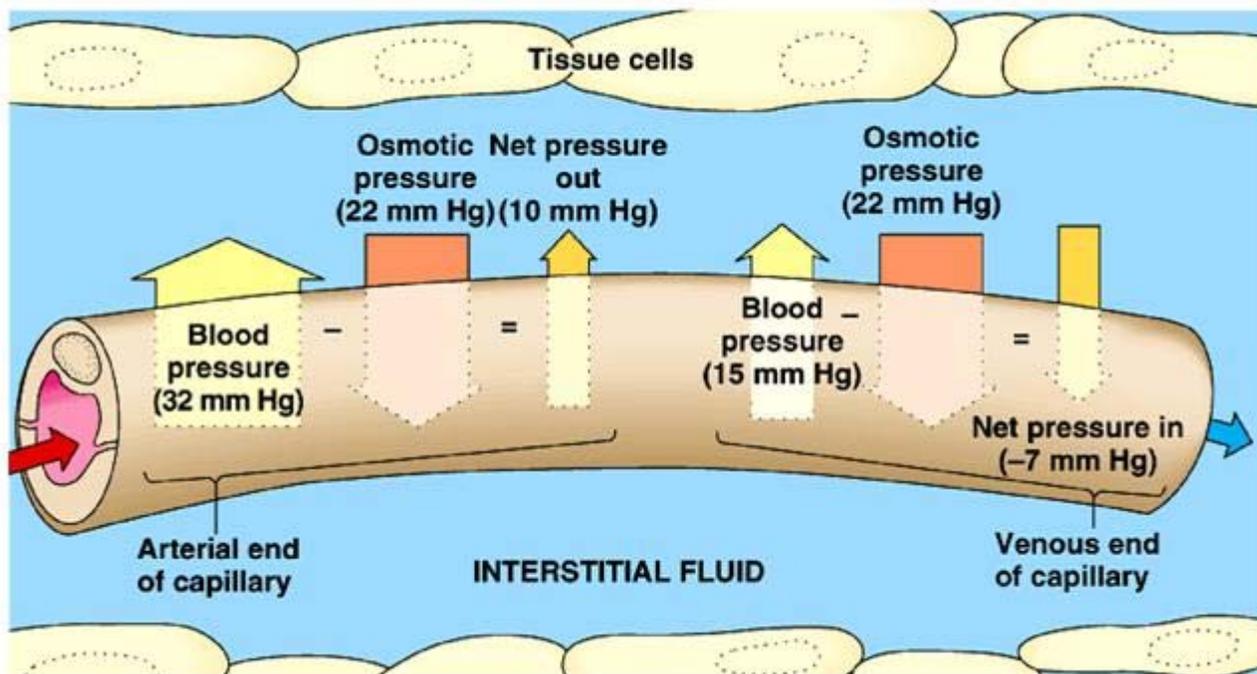
Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.



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9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution ("third space" in case of inflammation or injury).

Fluid overload ¹⁻³	Dehydration ¹⁻³
Weight gain 	Weight loss 
Swollen ankles and puffy eyes 	Dry mouth 
High blood pressure 	Low blood pressure 
Breathlessness 	Dizziness 

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobin concentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the

formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, $0,6 * BW$ volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

N_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

9.4 Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K - potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

$0,6 * BW$ – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na - sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

$0,2 * BW$ – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonic overhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 BW,$$

Cl_d - chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chloride solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6 BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

$0,6 * BW$ – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

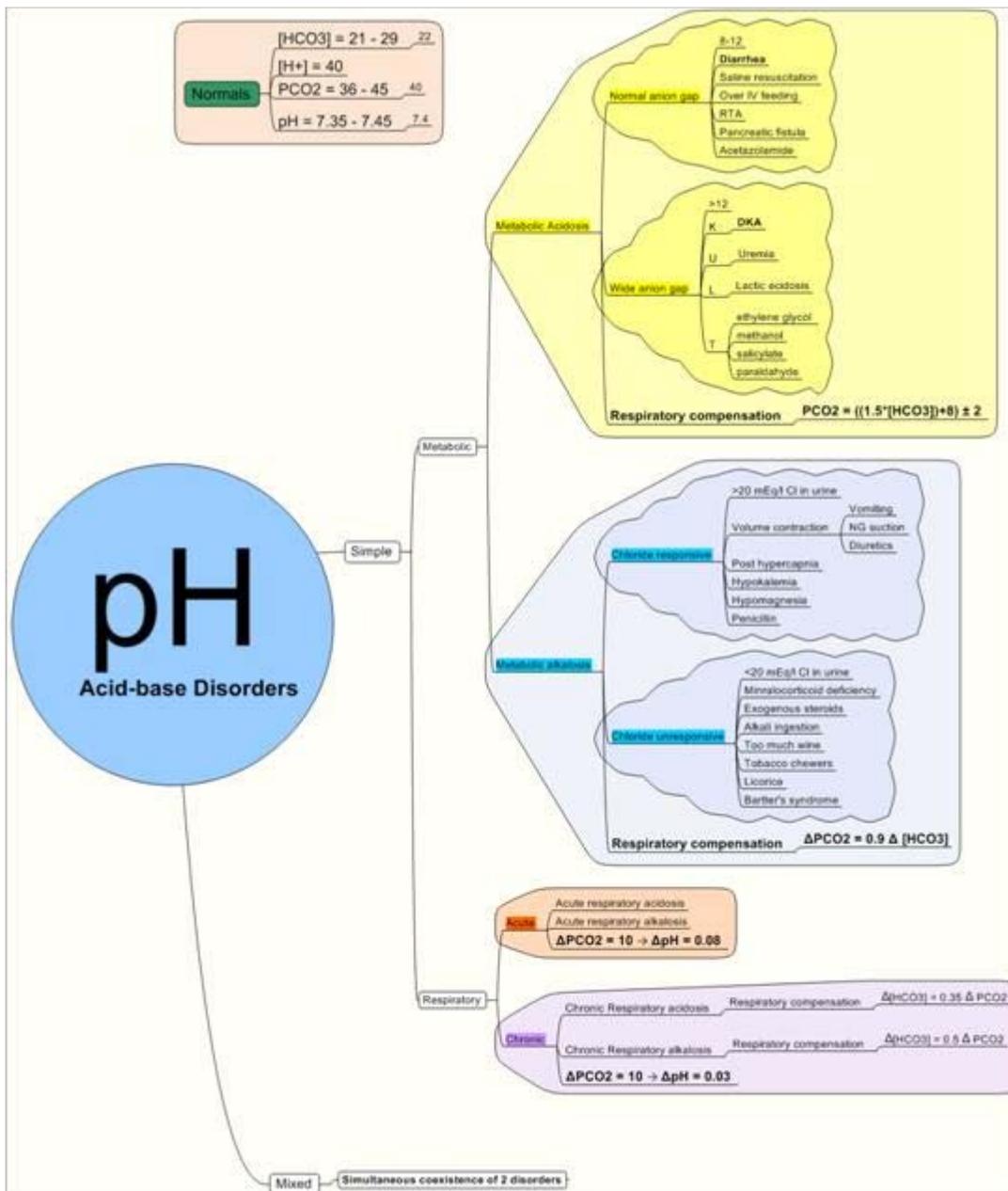
Ca_d – calcium deficiency, mmol

Ca_p – plasma calcium concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorption.



9.5 Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with $p\text{CO}_2$. Normally $p\text{CO}_2$ of arterial blood is 36-44 mm Hg. Hypercapnia ($p\text{CO}_2$ increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia ($p\text{CO}_2$ less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H^+ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is $0 \pm 1,5$. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO_2 from the body during hypoventilation. Laboratory tests show:

$\text{pH} < 7,35$,

$p\text{CO}_{2a} > 46$ mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When $p\text{CO}_2$ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

$\text{pH} > 7,45$,

$p\text{CO}_{2a} < 33$ mm Hg

BE $< +1,5$ mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H^+ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H^+ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination,

excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H⁺ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patient, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patient, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l.

Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patience, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patience, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patience, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration 88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

Task 7.

Patience, the patient of 50 with body weight 75 kg, was transported to the admission unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow breathing. Blood tests show: pH 7,18, pCO_{2a} 78 mm Hg, pO_{2A} – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patience, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, pCO_{2a} 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

Violations of homoeostasis and their correction.

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The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body

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changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

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- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

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H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, $0,6 * BW$ volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

N_{d} – sodium deficiency, mmol

N_{p} – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

9.4 Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K- potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

$0,6 * BW$ – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na - sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

$0,2 * BW$ – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonic overhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 BW,$$

Cl_d - chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chloride solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6 BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

$0,6 * BW$ – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75

mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d – calcium deficiency, mmol

Ca_p – plasma calcium concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorption.

9.5 Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

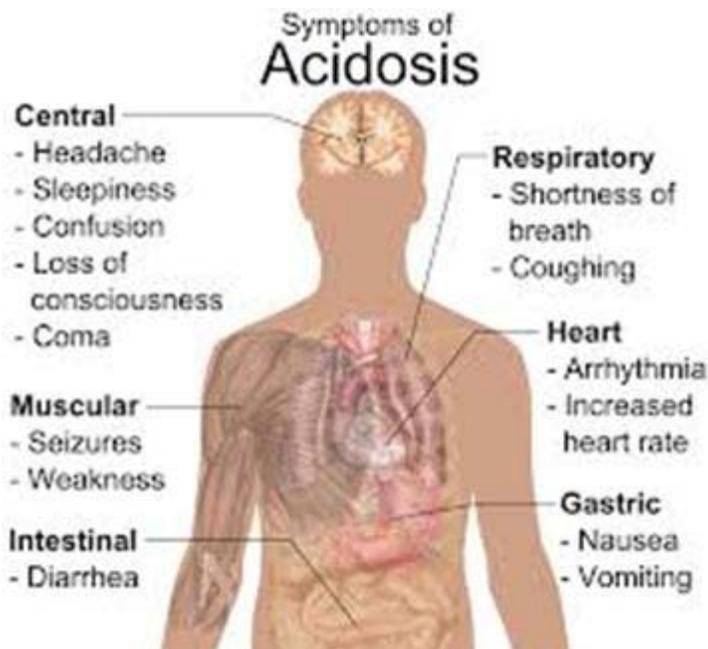
pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO_2 . Normally pCO_2 of arterial blood is 36-44 mm Hg. Hypercapnia (pCO_2 increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO_2 less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H^+ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is $0 \pm 1,5$. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency

(metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.



Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients

breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H^+ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

$$pH < 7,35,$$

$$pCO_{2a} < 35 \text{ mm Hg}$$

$$BE (-3) \text{ mmol/l.}$$

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H^+ ions concentration. Blood tests show:

$$pH > 7,45,$$

pCO_{2a} normal or insignificantly increased (compensatory reaction)

$$BE 3,0 \text{ mmol/l.}$$

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patience, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patience, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patience, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patience, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patience, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration 88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

Task 7.

Patience, the patient of 50 with body weight 75 kg, was transported to the admission unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow breathing. Blood tests show: pH 7,18, pCO_{2a} 78 mm Hg, pO_{2A} – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patience, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, pCO_{2a} 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

Student should repeat next questions

Violations of homeostasis and their correction.

9.1 The importance of the water to the organism.

Life on earth was born in the water environment. Water is a universal solvent for all the

biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homoeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the aerial walls, bar receptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals reveal into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution). This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.

9.2 Importance of osmolarity for homoeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws

of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hypoosmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.



9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobin concentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;
- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

VH₂O – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, 0,6*BW volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136 mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

Normal range	Causes of elevation	Causes of decline
Sodium (Na): 135 – 145 mEq/L	Hypernatremia: Excessive loss of water through GI system, lungs, or skin; fluid restriction, certain diuretics, hypertonic IV solutions, tube feeding; hypothalamic lesions, hyperaldosteronism, corticosteroid use, Cushing's syndrome, diabetes insipidus	Hyponatremia: Congestive heart failure, cirrhosis, nephrosis, excess fluid intake, syndrome of inappropriate antidiuretic hormone secretion (dilutional hyponatremia); sodium depletion, loss of body fluids without replacement, diuretic therapy, laxatives, nasogastric suctioning, hypoaldosteronism, cerebral salt-wasting disease
Potassium (K): 3.5 – 5.0 mEq/L	Hyperkalemia: Aldosterone deficiency, sodium depletion, acidosis, trauma, hemolysis of red blood cells, potassium-sparing diuretics	Hypokalemia: Lack of dietary intake of potassium, vomiting, nasogastric suctioning, potassium-depleting diuretics, aldosteronism, salt-wasting kidney disease, major GI surgery, diuretic therapy with inadequate potassium replacement
Calcium (Ca): 8.5 – 10.5 mg/dL	Hypercalcemia: Excessive vitamin D, immobility, hyperparathyroidism, potassium-sparing diuretics, ACE inhibitors, malignancy of bone or blood	Hypocalcemia: Hypoparathyroidism, malabsorption, insufficient or inactivated vitamin D or inadequate intake of calcium, hypoalbuminemia, diuretic therapy, diarrhea, acute pancreatitis, bone cancer, gastric surgery
Magnesium (Mg): 1.5 – 2.5 mg/dL	Hypermagnesemia: Excessive use of magnesium-containing antacids and laxatives, untreated diabetic ketoacidosis, excessive magnesium infusions	Hypomagnesemia: Malabsorption related to GI disease, excessive loss of GI fluids, acute alcoholism/cirrhosis, diuretic therapy, hyper- or hypothyroidism, pancreatitis, preeclampsia, nasogastric suctioning, fistula drainage

9.4 Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K- potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

0,6*BW – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na- sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

0,2*BW – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonic overhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 \text{ BW},$$

Cl_d - chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

$0,2 * BW$ – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chloride solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is 0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6 \text{ BW},$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

$0,6 * BW$ – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 \text{ BW},$$

Ca_d – calcium deficiency, mmol

Cl_p – plasma calcium concentration of the patient, mmol/l

$0,2 \cdot BW$ – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorbition.

9.5 Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompansated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO_2 . Normally pCO_2 of arterial blood is 36-44 mm Hg. Hypercapnia (pCO_2 increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO_2 less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H^+ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is $0 \pm 1,5$. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO_2 from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H⁺ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H^+ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patient, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patient, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patient, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patient, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patient, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine

vessel) are epinephrine, norepinephrine, serotonin, angiotensin II. Stress enhances the secretion of catecholamines, their blood concentration increases and arterioles constrict. Spasm of the arterioles is the basis of blood flow centralization: peripheral flow is disregarded in order to provide brain with the oxygenated blood as long as possible. To the group of vasodilators (agents, which provide dilatation of the vessels) belong “acid” metabolites (lactate, pyruvate, adenylic acid, inosinic acid), bradykinin, acetylcholine, different medicines (neuroleptics, α -adrenergic antagonists, peripheral vasodilators, ganglionic blocking agents, etc.), some exogenous poisons. All of them cause blood flow decentralization: opening of arterioles and distribution of the blood from central vessels to the capillary bed.

Capillaries are the interweaving network of the smallest body vessels with the general length of 90-100 thousands of kilometers. However simultaneously work only 20-25% of them. They provide metabolic exchange bringing oxygen and nutrients to the tissues and take back wastes of metabolism. Periodically, every 30-40 seconds one of them get closed and others open (vasomotion effect). Capillaries contain 12% of the whole circulating blood volume, but different pathological conditions can increase this amount even 3 and more times.

“Used” blood from the capillaries flows to the venous system. Veins are the blood reservoir, which contains 70% of the total circulating blood volume. Unlike arteries they are capable of volume control and thus they influence the amount of blood, which returns to the heart.

The most important haemodynamic index of venous system is central venous pressure. CVP represents the pressure which blood causes to the walls of cava veins and right atrium. This parameter is an integral index of circulating blood volume, systemic vascular resistance and pump function of the heart. It can be measure with a special device called “phlebotonometer” (pic. 4.9) or with a usual infusion set and a ruler. Normally CVP measured from the sternum point is 0-14 cm H₂O and from midaxillary line is 8-15 cm H₂O.

Central venous pressure decreases (sometimes even to negative) in case of:

- blood loss
- excessive water loss (dehydration)
- distributive shock (decrease of peripheral resistance due to venous and arterial dilatation)

In those conditions decreases volume of blood returning to the heart and thus suffers cardiac output. In case of negative CVP cardiac arrest is highly probable.

Central venous pressure increases in case of:

- heart failure (insufficiency of left or right ventricle)
- hypervolemia (excessive blood infusion, improper infusion therapy)

- obstructions to blood flow (pulmonary embolism, cardiac tamponade, etc.)

When CVP over 15-16 cm H₂O is combined with left ventricle insufficiency the risk of pulmonary edema is very high.

Blood pressure is an integral index of arterial part of systemic haemodynamics. Talking about blood pressure we may refer to systolic, diastolic, pulse and mean arterial pressure. Systolic (P_{syst}) and diastolic (P_{diast}) pressures are measured with the manometer (method with the usage of phonendoscope was invented by M. Korotkoff). Pulse pressure (PP) is a difference between systolic and diastolic blood pressure.

Mean arterial pressure (MAP) is calculated according to the formula:

$$\text{MAP} = P_{\text{dias}} + 1/3 \text{ PP} \quad \text{mm Hg}$$

MAP defines the level of pressure necessary for the metabolic exchange in the tissues. Its measurement allows the evaluation of tissue perfusion level.

Blood pressure depends on different factors, but the most important are cardiac output and vascular resistance (mostly arterioles). This dependence is direct, thus you can increase blood pressure using:

- infusion of vasoconstrictors - solutions of epinephrine, phenylephrine (mesaton), etc. (they will increase the vascular resistance);
- infusion of hydroxyethyl starch solutions or saline (they will increase circulating blood volume)
- infusion of cardiac glycosides or other medicine which stimulate myocardium

General volume of blood in the body of a healthy adult is nearly 7% from the body weight: 70 ml per kilogram for male and 65 ml per kilogram of body weight for female. Of course circulating blood volume is lower, because part of blood is out of metabolic processes as a reserve. CBV can be measured with the infusion of coloring substance to the blood flow (Evans blue, polyglucin) and later evaluation of its dissolution degree.

Therefore measurement of CVP, BP, cardiac output and circulating blood volume allow to evaluate condition of circulation system of the patients and to provide adequate correction.

4.2 Acute heart failure; shock and collapse.

Acute cardiovascular failure is a state of cardiac and vascular inability to provide

adequate supply of tissue metabolic needs with oxygenated blood and nutrients. This, earlier or later, causes cellular death.

The reasons of the failure vary greatly: mechanic injuries, blood loss, burns, dehydration, exogenous and endogenous intoxications, immediate hypersensitivity reaction, ischemic heart disease, neural and humoral regulation disorders of vascular tone.

Acute cardiac failure is a disorder of heart pumping action. It develops due to primary heart problems or secondary, under the influence of extracardiac factors such as infection or intoxication. There are two types of heart failure: left-sided and right-sided.

Left-sided heart failure is an inability of left ventricle to pump blood from the pulmonary circuit to the systemic circuit. The most common reasons of it are myocardial infarction, mitral insufficiency, left AV valve stenosis, aortic valve stenosis, aortal insufficiency, hypertonic disease, coronary sclerosis, acute pneumonia.

Coronary circulation is possible only during the diastole and in those conditions every violation of coronary passability decreases cardiac output. This way during the systole part of the blood is not injected into aorta, but stays in the left ventricle. Diastolic pressure in the left ventricle increases and blood is literally forced to stagnate in the left atrium. At the same time right ventricle functions normally and continues to pump usual amounts of blood to the pulmonary circuit. Thus hydrostatic pressure in the vessels of pulmonary circulation increases, fluid part of the blood moves first to the lung tissue and then, through alveolar-capillary membrane, to the alveolar lumen.

Clinically pulmonary edema begins with dyspnea (during physical activity or rest). Later attacks of dyspnea are connected with persistent cough with white or pink blood-tinged phlegm. During the attack patient tries to sit as in this position breathing is easier. This condition is called "heart asthma". When hydrostatic pressure is over 150-200 mm Hg, fluid part of blood moves to the alveolar lumen causing development of pulmonary edema.

Pulmonary edema is divided into interstitial and alveolar edema.

Interstitial edema is a condition during which serous part of stagnated in the pulmonary circuit blood infiltrates the lung tissue, including peribronchial and perivascular spaces.

During alveolar edema not only the plasma, but also blood components (red and white blood cells, platelets) get out from the vessels. During the respiratory act blood mixes with the air creating large amount of "foam", which violates gas exchange. This way, in addition to circulatory hypoxia, hypoxic hypoxia appears.

Condition of the patient gets worse quickly. Sitting position is optimal, but not as helping as previously. Respiratory rate is nearly 30-35 breathes per minute, but attacks of

breathlessness are constant. Skin is pale with acrocyanosis. Hypoxia of central nervous system usually causes psychomotor agitation. Respiratory acts are noisy; during cough pink blood-tinged phlegm is released. Auscultation allows you to hear different wet rales, sometimes it's even possible to hear them standing aside the patient without phonendoscope.

Pulmonary edema can be also divided according to the blood pressure level: the one with elevated pressure is caused by a hypertonic disease, aorta valve insufficiency or disorders of cerebral perfusion; another one is caused by total myocardial infarction, acute inflammation of myocardial muscle, terminal valve defects, severe pneumonia and is characterized with normal or low blood pressure.

Immediate aid

- make sure patient is sitting with his legs down (orthopnea)
- provide oxygenation through nasal catheter (before placing oil it with glycerin, insert it to the depth of 10-12 cm – distance from the wing of the nose to auricle) or face mask. Do not use Vaseline, because it can burn in atmosphere with high concentration of oxygen.

However if catheter is not deep enough patient will suffer from an unpleasant “burning” feeling, because oxygen flow will dry mucosa layer of the nasal cavity; also in this situation concentration of oxygen will be lower than expected.

- put venous tourniquets on the limbs in order to reduce amount of blood returning to heart: venous bed of limbs can reserve up to 1,7 liters of blood;
- constantly control heart and kidney activity (ECG, SaO₂ , and blood pressure are checked automatically trough the monitor; to control diuresis you should insert Foley catheter;
- catheterize central vein, because amount of infusions should be based on central venous pressure;
- use medical “defoamers” if they are available (ethyl alcohol or antiphomsylan solution) combined with oxygen inhalation

Scheme of oxygenation set connected to “defoamer” container

- a. oxygen source (cylinder with oxygen)
- b. tube with numerous holes sunk into container with defoamer
- c. tube for humidified oxygen (its opening should be over the level of fluid);
- d. patient

- medical treatment: 1% morphine solution (decreases intravascular

pressure of pulmonary circuit, inhibits respiration center in medulla oblongata preventive dyspnea progress, sedates patient);

- solutions of diuretics are used to decrease the circulating blood volume (6-12 ml of 1% furosemid solution, solution of ethacrynic acid), however be careful with them in case of low blood pressure; diuretic effect will last up to 3 hours after i/v infusion, the expected diuresis is 2-3 liters

- if blood pressure allows you can try to use nitroglycerin to reduce intravascular pressure of pulmonary circuit (1 or 2 tablets with 10 minutes interval)

- cardiac glycosides for improvement of heart action (0,025% digoxin solution, 0,05% strophanthin solution, 0,06% corglicon solution);

- in case of high pressure (over 150 mm Hg) use ganglionic blocking agents (1 ml of 5% pentamin solution diluted in 150 ml of saline, give i/v slowly; diluted with saline 250 mg of trimethaphan solution), because they reduce pressure in pulmonary circuit and lower the amount of blood getting to right half of the heart, however be careful with the dosage and monitor blood pressure level carefully;

- never use osmotic diuretics in case of pulmonary edema – they will increase blood volume and thus heart load!!!

- when everything listed above failed and patient is worsening with every second you should intubate him and start artificial ventilation with positive end expiratory pressure (begin with 4-6 cm H₂O)

Right-sided heart failure is an inability of right ventricular to pump blood from systemic circuit to the pulmonary circuit due to its weakness or an obstruction to the blood flow.

It occurs in case of pulmonary embolism, right ventricular infarction, excessive infusion therapy (especially including citrated blood) for patients with heart insufficiency, lung diseases (bronchial asthma, emphysema, pneumosclerosis) which cause increase of right ventricular load.

Patients have acrocyanosis, tachycardia, dyspnea, pronounced neck veins, ankle swelling, enlarged liver, ascytis. Central venous pressure is highly increased (up to 20-25 cm H₂O), however pulmonary edema does not appear.

Intensive treatment is mostly pathogenetic:

- limit the infusions (give only life-necessary solutions, check the water balance of the patient and reduce drinking water if necessary);

- in case of citrated blood transfusions use 5-10 ml of 10% calcium gluconate solution per every 500 ml of blood to prevent hypocalcaemia;
- in case of bronchial spasm use bronchial spasmolytics;
- to remove excessive fluid from the body use diuretics (furosemide solution for example);
- metabolic acidosis is corrected with 4% solution of sodium bicarbonate (i/v slowly with acid-base state control);
- in case of pulmonary embolism anticoagulants are used – fraxiparine 0,6 mg subcutaneously; heparin solution – 5000 IU every 4 hours; fibrinolytic drugs (streptokinase, fibrinolysin, urokinase, etc.)

Shock is a pathological state which can be described as a tissue hypoxia caused by hypoperfusion. Pathogenetic basis of shock depends on its reason (trauma, toxins, thermal injury) and at the same time on reactivity of the organism (level of defense mechanisms mobilization).

Stimulation of sympathetic nervous system - production of catecholamines and other vasoactive substances by hypothalamus and adrenal glands are the universal response of the body to the stress. Those mediators interact with the receptors of peripheral vessels causing their constriction and at the same time they dilate the vascular bed of life-important organs. This is so called “centralization of the flow”: rational decrease of blood flow in less important tissues (skin, organs of abdominal cavity, kidneys) in case of aggressive external influence for protecting life itself (brain, heart, lungs).

However influence of shock agents (pain, hypovolemia, destroyed cells, toxic metabolites), extended microcirculation violations (vascular spasm, microthrombosis and sludge) and caused by them tissue ischemia lead to hypoxic affection and cellular death of the internal organs. Further it can bring multiple organ dysfunction syndrome.

Collapse is a vascular failure. It occurs when body is not able to provide blood flow according to the new level of its needs (either because reaction is not fast enough or because sympathetic activation fails). Vascular bed volume and circulating blood volume are disproportional: too much blood gets to the microcirculation vascular reserve and the amount, which returns to the heart is not enough for the systemic needs (so called “decentralization” of the blood flow). Cardiac output and blood pressure decrease, that causes hypoperfusion of the central nervous system and thus unconsciousness and life-threatening complications.

Collapse definition is a bit nominal, because if such reaction extends in time the

state of shock develops. Shock itself can equally run as a vascular failure or as a sudden clinical death.

Pathogenetic classification of shock (according to P. Marino, 1998):

- hypovolemic
- cardiogenic
- distributive
- mixed (two and more factors).

Clinical classification of shock:

- traumatic shock;
- haemorrhagic shock;
- dehydration shock;
- burn shock;
- septic shock;
- anaphylactic shock;
- cardiogenic shock;
- exotoxic shock.

4 Shock caused by dehydration

It is a type of hypovolemic shock, which occurs during excessive body fluid loss (not blood, because hemorrhagic shock is another shock type).

Its reasons vary greatly:

- gastrointestinal diseases (profuse vomiting, diarrhea, loss of intestinal fluid through fistula);
- polyuria (uncontrolled diuretic treatment, diabetes mellitus and insipidus, diuretic phase of acute renal failure);
- fluid loss through skin and wound surface (burns, high fever);
- inadequate infusion treatment of postoperative or comatose patients;
- hyperventilation (rapid breathing, Kussmaul breathing, inadequate artificial ventilation parameters in case of apparatus without air humidification).

However not only the complete fluid loss can be the reason of shock, but also it's pathological distribution into the extracellular space (intestinal cavity in case of intestinal paralysis, abdominal cavity in case of ascites, pleural cavity in case of pleurisy). This way will can also act prolonged heavy tissue inflammations (peritonitis) or massive injuries (crush-syndrome).

In cases described above electrolytes are also lost (cations of sodium, potassium, calcium, magnesium; anions of chlorine, hydrocarbonate). It causes complex osmolar, acid-base and electrolytic disorders.

Stage of dehydration shock is evaluated according to the actual fluid loss:

less than 5% of body weight – mild dehydration

5-10% of body weight – moderate dehydration

over 10% of body weight – severe dehydration

Water deficiency brings lowering of cardiac output, blood pressure and central venous pressure (through decrease of blood volume returning to the heart, which leads to compensatory adrenergic vasoconstriction).

Dehydration causes body weight loss, skin and mucosa dryness, decrease of subcutaneous turgor and eyeballs tone, hypothermia, tachycardia, oliguria, thirst. While dehydration progresses compensatory mechanisms weaken and central nervous system suffers: patients become sluggish, confused; hallucinations, cramps and unconsciousness are also possible. Laboratory tests show blood concentration.

One of the most important things in treatment of dehydrated patients is daily balance of fluid: check it carefully trough measuring of daily received and lost fluids (food, infusions, stool and urine output). In case of fever or tachypnea make necessary corrections. Balance should be calculated every 12-24 hours (special paper forms make this easier).

Daily fluid balance is calculated by adding all the received fluids (both enteral and parenteral ways) and deducting urine output, stool, perspiration and breathing water loss.

You should remember, that perspiration depends on body temperature: in case of normal temperature (36,6°C) patient loses 0,5 ml/kg of water during every hour; 1 degree of temperature elevation adds 0,25 ml/kg to normal value of 0,5 ml/kg.

According to the fluid balance infusion therapy is divided into positive (for dehydrated patients), negative (for overhydrated patients) and “zero” (for patients without balance disorders).

Water deficiency is calculated according to the formula:

$$W_{\text{def}} = (Ht_p - Ht_n) * 0,2 \text{ BW} / Ht_n,$$

W_{def} – water deficiency, l;

Ht_p – hematocrit of the patient, l/l;

Ht_n - normal hematocrit, l/l;

BW – body weight, kg.

Use crystalloids to treat water deficiency: saline solution, Ringer's solution, Ringer-lactate solution, electrolytic solutions, 5%, 10, 20% glucose solution. To control potassium concentration (during dehydration this cation is widely lost) prescribe polarizing GIK mixture (pic.9.4), but don't you ever infuse concentrated potassium solutions quickly – it can cause cardiac arrest (not more than 400 of GIK solution ml per hour).

INTENSIVE THERAPY IN SURGERY

Surgical Intensive Care Unit (SICU)

Surgical Intensive Care Unit (SICU)

The Surgical Intensive Care Unit (SICU) is a 10-bed adult critical care unit designed to provide comprehensive care for critically ill surgical and trauma patients. SICU patients require highly skilled nursing care, including close observation and the use of extensive monitoring equipment, mechanical ventilation and potential end-of-life care. The level of care in an intensive care unit is greater than that available on intermediate care units. The SICU serves as the admitting unit for most trauma patients at Johns Hopkins Bayview. As a level 2 trauma center, the Medical Center ensures state-of-the-art trauma care to all the citizens of Maryland.

A Multidisciplinary Approach

The multidisciplinary team in the SICU consists of intensivists, physicians, mid-level practitioners and critical care nurses trained in critical care, surgery and trauma care of adult patients. Many of the nurses are certified through The American Association of Critical Care Nurses and are CCRN recipients. Other multidisciplinary services include:

- Respiratory therapy
- Physical and occupational therapy
- Dietary services
- Pharmacy services
- Social work
- Case management
- Ancillary support

Patient Population

The SICU's patient population includes:

- General surgery
- Trauma services
- ENT
- Orthopaedic surgery
- Vascular surgery
- Urology

- Plastic surgery
- Bariatric surgery
- OB/GYN
- Thoracic surgery

A Patient's Guide to Lung Surgery

Recovering in the ICU (Intensive Care Unit)



Waking Up in the ICU

Immediately after your surgery, you will be taken to the intensive care unit (ICU) to recover, where a team of specially trained cardiothoracic nurses will take care of you. Their goal is to help you recover as quickly and safely as possible. Along with your surgeon, members of the cardiothoracic anesthesia and surgical teams who took care of you in the operating room will continue to follow your progress in the ICU.

Because the ICU is a busy place, you can expect bright lights and a great deal of activity during the day. Many of the sounds you will hear are made by monitors and different types of equipment. Your medications, including those for pain control, will be given through intravenous (IV) tubes at very controlled rates using pumps.

To help you breathe, an endotracheal tube (breathing tube) was inserted while you were asleep. This tube is connected to a respirator that assists your breathing. Because you will not be able to talk or swallow while this tube is in place, your nurse will anticipate your needs and ask you questions that require only a yes or no answer. Nod your head to say yes, and shake your head to say no. When you are fully awake and breathing on your own, the breathing tube will be removed, and you will be able to talk.

It's normal to feel cold and to shiver for a short while after you arrive in the ICU. Your nurse will give you blankets to keep you warm. It's also normal to wake up feeling thirsty, because of

the medications you received before or during the surgery, or because you had nothing to eat or drink before your operation. Despite your thirst, you will be limited in what and how much you may drink while in the ICU.

Controlling Pain

Although all patients are concerned about the pain they will experience, pain after surgery is not as severe as most patients anticipate. To control your pain, you will be given medication that is injected, given orally or by suppository. While you are in the ICU, pain medications will be given to you as scheduled. While doses are calculated to keep you as comfortable as possible, if the medications affect your breathing and/or blood pressure, your physician may decrease the amount of pain medication given to you.

Coughing and Deep Breathing

As you recover in the ICU, the nurses will monitor your blood pressure, pulse rate and breathing. The endotracheal tube (breathing tube) will continue to help you breathe. To prevent postoperative pneumonia, the nurses and respiratory therapists will periodically remove any secretions that may have settled in your lungs during surgery. As soon as your breathing tube is removed, you should begin using your incentive spirometer, followed by coughing exercises. You should continue these exercises every hour while you are awake when you are transferred to your hospital room.

Movement and Changes in Position

While lying in bed, moving and changing position will help improve blood flow in your legs and remove secretions in your lungs. You can move your feet, wiggle your toes and point your toes up toward your head and then down toward the foot of the bed. Your nurse will help you change positions by turning you from one side to another.

Discharge from the ICU

Your surgeon, anesthesiologist and pulmonologist will determine the best time for you to be transferred from the ICU to the Cardiothoracic Unit Floor (CT Unit). Most patients are transferred the day after surgery. If you remain in the ICU for more than a day, your surgeon or a member of your surgery team will explain the specific reasons for the delay. The extra time spent in ICU is often for precautionary reasons and does not indicate any problem.

Breathing and Coughing Exercises

Incentive Spirometer



While recover from surgery, it might be uncomfortable or painful to breathe in as deeply as you normally would. But in order for your lungs to function properly, you need to use your incentive spirometer. You will be given one to take home to practice. You do not need to bring it back to the hospital.

The incentive spirometer is a small, handheld device that is used after surgery to help you expand your lungs and cough up any secretions from your lungs. It also helps maintain normal breathing pattern and measures your inspiratory volume, or how well your lungs are being filled with air when breathe in. In addition, the incentive spirometer will help exercise lungs, just as if you were going through your normal daily routine.

PEP Therapy

Positive Expiratory Pressure (PEP) therapy uses a breathing device that helps expand lungs and remove pulmonary secretions, or mucus. Therapist will assist you with this treatment. Breathing through the PEP valve, you will inhale normally, then exhale through the PEP valve, which has some resistance. This resistance will work out your lungs. After two series of ten breaths, follow with 2-3 huff coughs.

Huff Coughing

Coughing is a natural way to force mucus out of lungs. During huff coughing, gently say the word "huff". Therapist will help you with this breathing exercise.

Hepatorenal Syndrome

Introduction

Initial reports by Frerichs (1861) and Flint (1863), who had noted an association between advanced liver disease with ascites and acute oliguric renal failure in the absence of significant histological changes in the kidneys, led Heyd , and later Helwig and Schutz , to introduce the concept of the hepatorenal syndrome (HRS) to explain the increased frequency of acute renal failure after biliary surgery. However, because HRS could not be reproduced in animal models, pathophysiological concepts remained speculative and its clinical entity was not generally accepted. During the 1950s, HRS was more specifically characterised as a functional renal failure in patients with advanced liver disease, electrolyte disturbances and low urinary sodium concentrations. Hecker and Sherlock showed its temporal reversibility by norepinephrine administration. Over the next few decades, haemodynamic and perfusion studies by Epstein and other investigators identified splanchnic and systemic vasodilatation and active renal vasoconstriction as the pathophysiological hallmarks of HRS. Improved models of ascites and circulatory dysfunction contributed to therapeutic advances, including the introduction of large-volume paracentesis, vasopressin analogues, and transjugular intrahepatic stent-shunt (TIPS), which in turn have led to an improved pathophysiological understanding of HRS. Definition HRS is defined as the development of renal failure in patients with severe liver disease (acute or chronic) in the absence of any other identifiable cause of renal pathology. It is diagnosed following the exclusion of other causes of renal failure in patients with liver disease, such as hypovolaemia, drug nephrotoxicity, sepsis or glomerulonephritis. A similar syndrome can also occur in the setting of acute liver failure. In the kidney there is marked renal vasoconstriction, resulting in a low glomerular filtration rate (GFR). In the extrarenal circulation arterial vasodilatation predominates, resulting in reduction of the total systemic vascular resistance and arterial hypotension.

Diagnostic Criteria

The International Ascites Club (1996) group has defined the diagnostic criteria for HRS, and these are listed in Table 1.

Major criteria

Chronic or acute liver disease with advanced hepatic failure and portal hypertension

Low GFR, as indicated by serum creatinine > 1.5 mg/dl or 24-h creatinine clearance < 500 g/d for several days in patients with ascites without peripheral oedema or > 1000 ml in patients with peripheral oedema)

No sustained improvement in renal function (decrease of serum creatinine to 1.5 mg/dl or less or increase in 24 h creatinine clearance to 40 ml/min or more) after withdrawal of diuretics and expansion of plasma volume with 1.5 l of isotonic saline

Proteinuria

Additional criteria

Urine sodium $\lt \gt$ plasma osmolality

Urine red blood cells $\lt \gt$

Epidemiology

HRS occurs in about 4% of patients admitted to hospital with decompensated cirrhosis, the cumulative probability being 18% at 1 year, increasing to 39% at 5 years. Retrospective studies indicate that HRS is present in approximately 17% of patients admitted to hospital with ascites and in more than 50% of cirrhotic patients dying of liver failure. The most frequent cause of renal failure in cirrhosis is spontaneous bacterial peritonitis (SBP). Approximately 30% of patients with SBP develop renal failure. Type 1 HRS is characterised by rapid and progressive renal impairment and is precipitated most commonly by SBP. Type 1 HRS occurs in approximately 25% of patients with SBP, even when rapid resolution of the infection is obtained with antibiotics. Without treatment, the median survival of patients with HRS type 1 is less than 2 weeks, and virtually all patients die within 10 weeks after the onset of renal failure. Type 2 HRS is characterised by a moderate and stable reduction in GFR and commonly occurs in patients with relatively well-preserved hepatic function. The median survival is 3–6 months. Although this is markedly longer than that in type 1 HRS, it is still shorter than that of patients with cirrhosis and ascites who do not have renal failure. People of all races who have chronic liver disease are at risk of HRS, and its frequency is equal in both sexes; most patients with chronic liver disease are in the 4th–8th decade of life.

Prognosis

In a prospective study published by Gines et al., once HRS had developed the median survival was only 1.7 weeks, and it was poorer particularly in patients with apparent precipitating factors. Overall survival at 4 and 10 weeks was 20% and 10%, respectively. Patients with low urinary sodium excretion

Table 2. Risk factors for development of hepatorenal syndrome

Previous episodes of ascites

Absence of hepatomegaly

Poor nutritional status

Presence of oesophageal varices

Serum sodium $<> 553$ mosmol/l

Norepinephrine levels > 544 pg/ml

Plasma renin activity > 3.5 ng /ml

Mean arterial pressure

Pathophysiology

The hallmark of HRS is renal vasoconstriction, although the pathogenesis is not fully understood. Multiple mechanisms are probably involved and include interplay between disturbances in systemic haemodynamics, activation of vasoconstrictor systems and a reduction in activity of the vasodilator systems. The haemodynamic pattern of patients with HRS is characterised by increased cardiac output, low arterial pressure and reduced systemic vascular resistance. Renal vasoconstriction occurs in the absence of reduced cardiac output and blood volume, which is a point of contrast to most clinical conditions associated with renal hypoperfusion. Although the pattern of increased renal vascular resistance and decreased peripheral resistance is characteristic of HRS, it also occurs in other conditions, such as anaphylaxis and sepsis. Doppler studies of the brachial, middle cerebral and femoral arteries suggest that extrarenal resistance is increased in patients with HRS, while the splanchnic circulation is responsible for arterial vasodilatation and reduced total systemic vascular resistance. The renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system (SNS) are the predominant systems responsible for renal vasoconstriction. The activity of both systems is increased in patients with cirrhosis and ascites, and this effect is magnified in HRS. In contrast, an inverse relationship exists between the activity of these two systems and renal plasma flow (RPF) and the glomerular filtration rate (GFR). Endothelin is another renal vasoconstrictor that is present in increased concentration in HRS, although its role in the pathogenesis of this syndrome has yet to be identified. Adenosine is well known for its vasodilator properties, although it acts as a vasoconstrictor in the lungs and kidneys. Elevated levels of adenosine are more common in patients with heightened activity of the RAAS and may work synergistically with angiotensin II to produce renal vasoconstriction in HRS. This

effect has also been described with the powerful renal vasoconstrictor, leukotriene E4. The vasoconstricting effect of these various systems is antagonised by local renal vasodilatory factors, the most important of which are the prostaglandins. Perhaps the strongest evidence supporting their role in renal perfusion is the marked decrease in RPF and the GFR when nonsteroidal medications known to bring about a sharp reduction in PG levels are administered. Nitrous oxide (NO) is another vasodilator that is believed to play an important part in renal perfusion. Preliminary studies, predominantly based on animal experiments, have demonstrated that NO production is increased in the presence of cirrhosis, although NO inhibition does not result in renal vasoconstriction owing to a compensatory increase in PG synthesis. However, when both NO and PG production are inhibited, marked renal vasoconstriction develops. These findings demonstrate that renal vasodilators have a critical role in maintaining renal perfusion, particularly in the presence of overactivity of renal vasoconstrictors. However, we do not yet know for certain whether vasoconstrictor activity becomes the predominant system in HRS and whether a reduction in the activity of the vasodilator system contributes to this. Various theories have been proposed to explain the development of HRS in cirrhosis. The two main ones are the arterial vasodilatation theory and the hepatorenal reflex theory. The first not only describes sodium and water retention in cirrhosis, but may also be the most rational hypothesis for the development of HRS. Splanchnic arteriolar vasodilatation in patients with compensated cirrhosis and portal hypertension may be mediated by several factors, the most important of which is probably NO. In the early phases of portal hypertension and compensated cirrhosis, this underfilling of the arterial bed causes a decrease in the effective arterial blood volume and results in homeostatic reflex activation of the endogenous vasoconstrictor systems. Activation of the RAAS and SNS occurs early with antidiuretic hormone secretion, a later event when a more marked derangement in circulatory function is present. This results in vasoconstriction not only of the renal vessels, but also in the vascular beds of the brain, muscle, spleen and extremities. The splanchnic circulation is resistant to these effects because of the continuous production of local vasodilators, such as NO. In the early phases of portal hypertension, renal perfusion is maintained within normal or near-normal limits as the vasodilatory systems antagonise the renal effects of the vasoconstrictor systems. However, as liver disease progresses in severity, a critical level of vascular underfilling is achieved; renal vasodilatory systems are unable to counteract the maximal activation of the endogenous vasoconstrictors and/or intrarenal vasoconstrictors, which leads to uncontrolled renal vasoconstriction. Support for this hypothesis is provided by studies in which the administration of splanchnic vasoconstrictors in

combination with volume expanders results in improvement in arterial pressure, RPF and GFR. The alternative theory proposes that renal vasoconstriction in HRS is not related to systemic haemodynamics but is due either to a deficiency in the synthesis of a vasodilator factor or to a hepatorenal reflex that leads to renal vasoconstriction. Evidence points to the vasodilatation theory as a more tangible explanation for the development of HRS.

Histopathology of HRS

In previous definitions of HRS, changes in renal histology were reported to be absent or minimal, which reflected a rapid progression to death after development of HRS. Considering that many patients with HRS currently receive aggressive supportive treatment including renal replacement therapy to prolong survival until liver transplantation, it seems obvious that prolonged renal hypoperfusion, renal medullary hypoxia and the high frequency of infectious complications ultimately contribute to histologically detectable renal damage. However, it is increasingly recognised that structural renal damage may already be found even before renal dysfunction becomes manifest. In a series of cirrhotic patients undergoing liver transplantation, 100% of renal biopsies showed glomerular abnormalities. Tubular function is usually well preserved at the time when HRS develops, but tubular abnormalities, including increased B2 microglobulin excretion, have been reported in deeply jaundiced patients with HRS. With progressive circulatory dysfunction, prolonged renal hypoperfusion may eventually result in acute tubular necrosis by increasing the susceptibility to additional insults by radiographic contrast agents, aminoglycosides, haemorrhage, endotoxaemia or any other cause of medullary hypoxia. The presence of acute tubular necrosis could partially explain the slow or absent renal recovery in HRS type 1 even after the initiation of vasopressor support. For instance, a recent case study reports full recovery of renal function in dialysis-dependent HRS after 7 weeks of treatment with ornipressin, dopamine and intravenous albumin.

Prevention

The following measures may decrease the incidence of renal failure or HRS in patients with liver disease.

Prophylaxis Against

Bacterial Infections Bacterial infections occur in approximately 50% of patients with variceal haemorrhage, and antibiotic prophylaxis improves survival by approximately 10%. Patients who have had a previous episode of SBP have a 68% chance of recurrent infection at 1 year,

and this carries a 33% chance of developing renal failure. As bacterial infections are an important cause of renal dysfunction in cirrhotic patients, prophylaxis with antibiotics is recommended in two clinical settings, namely variceal bleeding and a history of previous SBP.

Volume Expansion

To prevent the development of renal failure in patients who develop SBP, it is now recommended that plasma volume expansion should be implemented in these patients by giving 20% albumin (1–1.5 g/kg over 1–3 days) at diagnosis to prevent circulatory dysfunction, renal impairment and mortality. Use of low-salt albumin as fluid replacement in patients undergoing large-volume paracentesis (8 g for each litre of ascitic fluid removed) is known to prevent paracentesis-induced circulatory dysfunction.

Judicious Use of Diuretics

It is important to identify the lowest effective dose of a diuretic for any individual patient, as diuretic-induced renal impairment is seen in approximately 20% of patients with ascites. It develops when the rate of diuresis exceeds the rate of ascites reabsorption, leading to intravascular volume depletion. Diuretic-induced renal impairment is usually moderate and rapidly reversible following diuretic withdrawal.

Avoidance of Nephrotoxic Drugs

Patients with cirrhosis and ascites are predisposed to the development of acute tubular necrosis during the use of aminoglycosides, with renal failure occurring in 33% of such patients as against 3–5% in the general population. Another important cause of renal failure is the use of nonsteroidal antiinflammatory drugs (NSAIDs).

Treatment

The ideal treatment for HRS is liver transplantation; however, because of the long waiting lists in the majority of transplant centres, most patients die before being offered a transplant. There is an urgent need for effective alternative therapies to increase survival chances for patients with HRS until transplantation can be performed. Treatment can be divided into initial management, pharmacological treatment and surgical manoeuvres.

Initial Management Optimise fluid management.

Renal function rarely recovers in the absence of liver recovery. The key goal in the management

of these patients is to exclude reversible or treatable lesions (mainly hypovolaemia) and to support the patient until liver recovery or liver transplantation. The treatment of HRS is directed at reversing the haemodynamic changes induced by reduced renal perfusion pressure, stimulated sympathetic nervous system and increased synthesis of humoral and renal vasoconstrictor factors. In cirrhotic patients renal failure is frequently secondary to hypovolaemia (diuretics or gastrointestinal bleeding), NSAIDs or sepsis. Precipitating factors should be recognised and treated and nephrotoxic drugs, discontinued. All patients should be challenged with up to 1.5 l of fluid, such as albumin solution or normal saline, to assess the renal response, as many patients with subclinical hypovolaemia will respond to this simple measure. This should be done with careful monitoring to avoid fluid overload. In practice, fluid overload is not usually a problem, as patients with severe liver disease function as 'fluid sumps' and their vasculature adapts to accommodate the extra fluid. This has been described by Hadengue et al., who reported increased venous compliance following fluid challenge in advanced cirrhosis. Monitor for sepsis. Evidence of sepsis should be sought in blood, ascitic, cannulae and urine cultures, and nonnephrotoxic broad-spectrum antibiotics should be started regardless of whether such evidence is found, as any delay in effective treatment of undiagnosed infection can increase mortality. In advanced cirrhosis, endotoxins and cytokines play important parts in fostering the hyperdynamic circulation and worsening renal function. Optimise blood pressure. If mean arterial pressure is low

Pharmacological Treatment

All the drugs that have been investigated in HRS have one overriding aim: to increase renal blood flow. This has been achieved either indirectly, by splanchnic vasoconstriction, or directly, using renal vasodilators. One of the principal difficulties has been the lack of agents that act purely on the splanchnic circulation. Drugs that 'spill over' into the systemic circulation may actually exacerbate the intense renal vasoconstriction already present. Currently, there is significant enthusiasm for the use of vasoconstrictor agents in HRS. However, the numbers of patients studied have been small, mortality remains high and there have been no randomised placebo-controlled trials. This deficit clearly needs to be addressed but the possibilities are limited by the relative rarity of patients with 'pure' HRS without such confounding variables as sepsis and gastrointestinal bleeding. Important aspects of the situation mentioned in these reports are the need for a pressor response to the agents used and the recurrence of abnormal renal function after the cessation of vasoconstrictor therapy. HRS is effectively a marker of poor hepatic function, and these agents are probably best utilised as a

bridge to further improvement in liver function following either cessation of alcohol abuse or liver transplantation. Thus, the decision to use vasoconstrictor agents for HRS should be based on whether the patient is a realistic transplant candidate and, if not, whether liver function might improve. Patients who do not satisfy these criteria will be tested unnecessarily, merely prolonging the process of dying when palliative care would be more appropriate.

Dopamine. Nonpressor renal doses of dopamine [2–5 $\mu\text{g kg}^{-1} \text{min}^{-1}$) are frequently prescribed to patients with acute deterioration of renal function. As shown by a recent, large scale, randomised trial, early renal dose dopamine has no role in the prevention of acute renal failure in critically ill patients and does not significantly improve renal function in patients with HRS. At higher doses, dopamine worsens the hyperdynamic circulation by exaggerating splanchnic hyperaemia and increasing portal pressure and may cause tachyarrhythmia. Thus, the use of dopamine monotherapy seems to offer no benefit in HRS. Combination therapy with dopamine and vasopressors has produced inconsistent results in HRS. Because beneficial renal effects have been reported only with vasopressor, and not with dopamine, monotherapy, it seems unlikely that dopamine contributed to renal improvement in these studies.

Misoprostol. Misoprostol, a synthetic prostaglandin E-1 analogue, has been used to reverse renal vasoconstriction in HRS. Low doses of misoprostol are vasodilatory, natriuretic and diuretic, whereas high-dose misoprostol increases renal vascular tone and inhibits sodium and water excretion. None of the five studies investigating misoprostol in HRS seems to indicate substantial benefit. Improvement of renal function occurred in 1 of these studies, but could also be explained by volume expansion.

N-Acetylcysteine. In 1999, the group at the Royal Free Hospital reported their experience with N-acetylcysteine (NAC) for the treatment of HRS. This was based on experimental models of acute cholestasis, in which the administration of NAC resulted in an improvement in renal function. Twelve patients with HRS were treated with intravenous NAC, without any adverse effects, and the survival rates were 67% and 58% at 1 month and 3 months, respectively (this included 2 patients who received liver transplantation after improvement in renal function). The mechanism of action remains unknown, but this interesting study encourages further optimism for medical treatment of a condition that once carried a hopeless diagnosis without liver transplantation. Controlled studies with longer follow-up may help answer these pressing questions.

Renal vasoconstrictor antagonists. Saralasin, an antagonist of angiotensin II receptors, was first used in 1979 in an attempt to reverse renal vasoconstriction. Because this drug inhibited the homeostatic response to hypotension commonly observed in patients with cirrhosis, it led to worsening hypotension and deterioration in renal function. Poor results were also observed with phentolamine, an alpha-

adrenergic antagonist, highlighting the importance of the sympathetic nervous system in maintaining renal haemodynamics in patients with HRS. Antagonists of endothelin A receptor. A recent case series by Soper et al. reported an improvement in GFR in patients with cirrhosis, ascites and HRS who received an endothelin A receptor antagonist. All patients showed a dose-dependent response in the form of improved inulin and para-aminohippurate excretion, RPF and GFR without changes in systemic haemodynamics. These patients were not candidates for liver transplantation and subsequently died. More work is needed to explore this therapeutic approach as a possible bridge to transplantation for patients with HRS. Systemic vasoconstrictors. These medications have shown the most promise for treatment of HRS in recent years. Hecker and Sherlock used norepinephrine in 1956 to treat patients with cirrhosis who had HRS, and they were the first to describe an improvement in arterial pressure and urine output. However, no improvement was observed in the biochemical parameters of renal function, and all patients subsequently died. Octapressin, a synthetic vasopressin analogue, was first used in 1970 to treat HRS type 1. RPF and the GFR improved in all patients, all of whom subsequently died of sepsis, gastrointestinal bleeding or liver failure. Because of these discouraging results, the use of alternative vasopressin analogues, particularly ornipressin, attracted attention. Two important studies by Lenz et al. demonstrated that short term use of ornipressin resulted in an improvement in circulatory function and a significant increase in RPF and the GFR. The combination of ornipressin and albumin was subsequently tried by Guevera in patients with HRS. This idea was based on data suggesting that the combination of plasma volume expansion and vasoconstrictors normalised renal sodium and water handling in patients who had cirrhosis with ascites. In this study, 8 patients were originally treated for 15 days with ornipressin and albumin. Treatment had to be discontinued in 4 patients after fewer than 9 days because of complications of ornipressin use that included ischaemic colitis, tongue ischaemia and glossitis. Although a marked improvement in the serum creatinine was observed during treatment, renal function deteriorated on treatment withdrawal. In the remaining 4 patients the improvement in RPF and the GFR was significant and was associated with a lowering of serum creatinine levels. These patients subsequently died, but no recurrence of HRS was observed. Owing to the high incidence of severe adverse effects with ornipressin, the same investigators used another vasopressin analogue with fewer adverse effects, namely terlipressin. In this study, nine patients were treated with terlipressin + albumin for 5–15 days. This treatment was associated with a marked fall in serum creatinine levels and an improvement in mean arterial pressure. Reversal of HRS was noted in seven of the nine patients, and HRS did not recur when treatment was discontinued. No adverse ischemic effects were reported:

according to this study, terlipressin with albumin is a safe and effective treatment for HRS. Alpha adrenergic agonists. Angeli et al. showed that long-term administration of midodrine (an alpha-adrenergic agonist) and octreotide improved renal function in patients with HRS type 1. All patients also received albumin, and the results obtained with this approach were compared against those observed with dopamine at nonpressor doses. None of the patients treated with dopamine showed any improvement in renal function, but in all the patients treated with midodrine, octreotide and volume expansion renal function did improve. No adverse effects were reported in these patients. Gulberg et al. treated seven patients who had cirrhosis and HRS type 1 with a combination of ornipressin and dopamine for infusion periods as long as 27 days, but only three of the seven patients survived. This treatment can be used as a bridge to liver transplantation. Aquaretic agents. K-Opioid antagonists inhibit antidiuretic hormone secretion by the neurohypophysis and induce water excretion. Administration of niravoline at doses ranging from 0.5 to 2 mg induced a strong aquaretic response and was well tolerated in 18 cirrhotic patients with preserved renal function, but no data are available on the use of niravoline in patients with HRS.

Surgical Manoeuvres

Transjugular intrahepatic portosystemic shunting. It is well documented that portal hypertension plays a central role in the development of refractory ascites and HRS. Earlier studies showed improved renal function after side-to-side portocaval shunting, but at the cost of a high surgical mortality in advanced cirrhosis. The transjugular intrahepatic portosystemic shunt (TIPS) was introduced as a less invasive method of reducing increased portal pressure. Guevarra et al. have investigated hepatic and renal haemodynamic changes after placement of TIPS in patients with HRS. One month after placement of TIPS a marked improvement in renal function was observed, as indicated by a significant reduction in serum creatinine and blood urea nitrogen and increased urine volume, RPF and GFR. These improvements were associated with a reduction in plasma rennin, aldosterone and norepinephrine activity. These changes were statistically significant, albeit less pronounced than observed in a similar group of patients receiving ornipressin and albumin infusions. Renal improvements were more pronounced at 30 days than at 7 days, possibly because of the deleterious effects of contrast media or the resolution of concomitant problems. After TIPS, GFR improved significantly but did not reach normal values, suggesting that TIPS does not correct all mechanisms contributing to HRS. Brensing et al. found a sustained improvement of renal function after TIPS in 31 patients with type 1 or 2 HRS, allowing the discontinuation of haemodialysis in four of seven patients. After

TIPS 3-, 6-, 12- and 18-month survival rates were 81%, 71%, 48% and 35%, respectively, in the total patient cohort, with survival in HRS type 1 patients being significantly worse than in the others. The use of TIPS to prolong survival until liver transplantation seems promising.

Other surgical shunts. Despite the theoretical benefit of improving portal hypertension and thus HRS by means of a portosystemic shunt, only a few scattered case reports have shown any benefit. Currently, particularly with the recent introduction of TIPS, portocaval shunts are not indicated in this setting.

Renal replacement therapy. Many clinicians are reluctant to institute renal replacement therapy in advanced cirrhosis, because the outcome is poor unless liver transplantation is a realistic option. Intermittent haemodialysis can be a problem because patients with HRS are prone to develop circulatory and coagulation problems, and biocompatibility is also a problematic issue. In an early study in the United Kingdom 100% mortality was observed in cirrhotic patients with HRS despite early institution of renal support. However, modern renal replacement therapies such as continuous endogenous haemofiltration (CVVH) are certainly capable of prolonging life in patients with type 1 HRS who have not responded to medical therapies or TIPS. Because the underlying hepatic problem persists, the long-term prognosis is grim and treatment should be confined to patients who are candidates for liver transplantation or have a realistic chance of hepatic recovery. The molecular adsorbent recirculating system (MARS) is a modified dialysis method that uses albumin-containing dialysate in a closed-loop secondary circuit for adsorptive removal of albumin-bound toxins. In a randomised study, short-term survival of eight HRS patients treated with MARS was superior to that of five other HRS patients treated with CVVH. In contrast to previous reports on haemodialysis, treatment was well tolerated. Unfortunately, the study was terminated after enrolment of only 13 patients, which makes evaluation of any influence on mortality difficult. Moreover, the control group seems to have received a smaller dialytic dose: creatinine levels were decreased in the MARS group only. Nonetheless, the favourable effects of this system deserve evaluation in a prospective study of adequate power.

Liver transplantation. Liver transplantation is the ideal treatment for HRS, but is completely dependent on the availability of Donors. Patients with HRS have a higher risk of postoperative morbidity, early mortality and longer hospitalisation than other transplant recipients. Gonwa et al. reported that at least one third of such patients require haemodialysis postoperatively, with a smaller proportion (5%) requiring long-term dialysis. Because renal dysfunction is common in the first few days after transplantation, avoidance nephrotoxic immunosuppressants is generally recommended until renal function is recovered. However, the GFR gradually improves to an average of 40–50 ml/min by the 6th postoperative week. The systemic and neurohumoral

abnormalities associated with HRS also resolve in the 1st postoperative month. Long-term survival rates are excellent, with the survival rate at 3 years approaching approximately 60%. This is only slightly lower than the 70–80% survival rate of transplant recipients without HRS and is markedly better than the survival rate of patients with HRS who do not receive transplants, which is virtually nil at 3 years.

VASCULAR ACCESS

PREPARING FOR VASCULAR CANNULATION

HANDWASHING

Handwashing is mandatory (and often overlooked) before the insertion of vascular devices. Scrubbing with antimicrobial cleansing solutions does not reduce the incidence of catheter-related sepsis, so a simple soap-and-water scrub is sufficient.

UNIVERSAL PRECAUTIONS

In 1985, the Centers for Disease Control introduced a strategy for blood and body fluid precautions known as universal precautions. This strategy is based on the assumption that all patients are potential sources of human immunodeficiency virus (HIV) and other blood-borne pathogens (e.g., hepatitis viruses) until proven otherwise. The following recommendations apply to the insertion of vascular catheters.

Use protective gloves for all vascular cannulations.

Use sterile gloves for all cannulations except those involving the introduction of a short catheter into a peripheral vein.

Caps, gowns, masks, and protective eyewear are not necessary unless splashes of blood are anticipated (e.g., in a trauma victim). These measures do not reduce the incidence of catheter-related sepsis.

Avoid needlestick injuries. Do not recap needles or manually remove needles from syringes.

Place all sharp instruments in puncture-resistant containers immediately after use.

If a needlestick injury is sustained during the procedure, follow the recommendations in Table 4.1.

Needlestick injuries are reported in up to 80% of medical students and interns. Therefore, in patients who are known risks for transmitting HIV or viral hepatitis, vascular cannulation should be performed only by an experienced senior-level resident or fellow.

LATEX ALLERGY

The increased use of rubber gloves (made of latex or vinyl) as protection against HIV infections has resulted in an increased recognition of allergic reactions to latex. These reactions can be manifest as a contact dermatitis (urticarial lesions of the hands and face), or as a conjunctivitis, rhinitis, or asthma. The latter three manifestations are reactions to airborne latex particles, and they do not require direct physical contact with gloves. They often appear when the affected individual enters an area where latex gloves are being used. Therefore, a latex allergy should be suspected in any ICU team member who develops atopic symptoms when in the ICU. When this occurs, a switch to vinyl gloves will eliminate the problem. Latex allergy can be manifest as anaphylaxis, so the transition to vinyl gloves for suspected latex allergy should not be delayed.

CLEANSING THE SKIN

Agents that reduce skin microflora are called antiseptics, whereas agents that reduce the microflora on inanimate objects are called disinfectants. Common antiseptic agents are listed in Table 4.2 (11,12). The most widely used antiseptic agents are alcohol and iodine, both of which have a broad spectrum of antimicrobial activity. Alcohol (commonly used as a 70% solution) may not work well on dirty skin (that is, it does not have a detergent action), so it is often used in combination with another antiseptic agent. The most popular antiseptic solution currently in use is a povidone-iodine preparation (e.g., Betadine), also known as an iodophor, a water-soluble complex of iodine and a carrier molecule. The iodine is released slowly from the carrier molecule, and this reduces the irritating effects of iodine on the skin. This preparation should be left in contact with the skin for at least 2 minutes to allow sufficient time for iodine to be

released from the carrier molecule.

HAIR REMOVAL

Shaving is not recommended for hair removal because it abrades the skin and promotes bacterial colonization. If hair removal is necessary, the hair can be clipped or a depilatory can be applied.

CATHETER INSERTION DEVICES

Vascular cannulation can be performed by advancing the catheter over a needle or guidewire that is in contact with the lumen of a blood vessel.

CATHETER-OVER-NEEDLE

A catheter-over-needle device is shown in Figure 4.1. The catheter fits snugly over the insertion needle, and has a tapered end to minimize damage to the catheter tip and soft tissues during insertion. This device can be held like a pencil (i.e., between the thumb and forefinger) as it is inserted through the skin and directed to the target vessel. When the tip of the needle enters the lumen of a patent blood vessel, blood moves up the needle by capillary action and enters the flashback chamber. When this occurs, the catheter is threaded over the needle and into the lumen of the blood vessel as the needle is withdrawn.

The advantage of a catheter-over-needle device is the ability to cannulate vessels in a simple one-step procedure. The disadvantage is the tendency for the catheter tip to become frayed as it passes through the skin and soft tissues, and to subsequently damage the vascular endothelium and promote phlebitis and thrombosis. To minimize this risk, the catheter-over-needle device is usually reserved for cannulation of superficial vessels.

CATHETER-OVER-GUIDEWIRE

Guidewire-assisted vascular cannulation was introduced in the early 1950s and is often called the Seldinger technique, after its inventor. This technique is illustrated in Figure 4.2. A small-bore needle (usually 20 gauge) is used to probe for the target vessel. When the tip of the

needle enters the vessel, a thin wire with a flexible tip (called a J-tip because of its shape) is passed through the needle and into the vessel lumen. The needle is then removed, leaving the wire in place to serve as a guide for cannulation of the vessel. When cannulating deep vessels, a rigid dilator catheter is first threaded over the guidewire and removed; this creates a tract that facilitates insertion of the vascular catheter.

The guidewire technique has the presumed advantage of minimizing damage to soft tissues and blood vessels by using a small-bore probe needle. However, the use of a rigid dilator catheter (as explained above) seems to nullify this advantage. Nevertheless, the guidewire technique is currently the preferred method for central venous and arterial cannulation.

THE CATHETERS

Vascular catheters are composed of plastic polymers impregnated with barium or tungsten salts to enhance radiopacity. Catheters intended for short-term cannulation (days) are usually made of polyurethane; catheters used for long-term venous access (weeks to months) are composed of a more flexible and less thrombogenic derivative of silicone. The silicone catheters (e.g., Hickman and Broviac catheters) are too flexible for routine percutaneous insertion, and are not appropriate for use in the ICU.

HEPARIN BONDING

Some vascular catheters are impregnated or coated with heparin to reduce thrombogenicity. However, this measure has not proven effective in reducing the incidence of catheter-associated thrombosis. Because heparin-coated catheters can be a source of heparin-induced thrombocytopenia, catheters used in the ICU should not be impregnated or coated with heparin.

CATHETER SIZE

The size of vascular catheters is commonly expressed in terms of the outside diameter, and the units of measurement are shown in Table 4.3. The French size is a metric derivative equivalent to the outside diameter in millimeters multiplied by 3; that is, French size = outside diameter (in mm) \times 3. The gauge system was developed for wires and needles, and has been adopted for catheters. There is no simple mathematical relationship between gauge size and other units of

measurement, and a table of reference values such as Table 4.3 is needed to make the appropriate conversions. Gauge sizes usually range from 14 (largest diameter) to 27 (smallest diameter).

The steady or laminar flow through a rigid tube is influenced most by the radius of the tube (see the Hagen-Poiseuille equation in the section on peripheral blood flow in Chapter 1). The influence of tube diameter on flow rate is demonstrated in Table 4.3 for gravity flow of one unit of packed red blood cells diluted with 250 mL normal saline flowing through catheters of equal length. Note that a little more than a doubling of tube diameter (from 0.7 mm to 1.65 mm) is associated with almost a quadrupling of flow rate (from 24.7 to 96.3 mL/minute). Thus, catheter size (diameter) is an important consideration if rapid flow rates are desired.

MULTILUMEN CATHETERS

Multilumen catheters were introduced for clinical use in the early 1980s, and are now used routinely for central venous cannulation. The design of a triple-lumen catheter is shown in Figure 4.3. These catheters have an outside diameter of 2.3 mm (6.9 French) and may have three channels of equal diameter (usually 18 gauge) or may have one larger channel (16 gauge) and two smaller channels of equal diameter (18 gauge). The distal opening of each channel is separated from the other by at least 1 cm to help minimize mixing of infusate solutions.

Multilumen catheters have proven to be valuable aids because they minimize the number of venipunctures needed for monitoring and infusion therapy, yet they do not increase the risk of thrombosis or infection when compared with single-lumen catheters.

INTRODUCER CATHETERS

Another valuable addition to the family of vascular catheters is the introducer catheter, shown in Figure 4.3. These large-bore catheters (8 to 9 French) can be used as conduits for insertion and removal of smaller vascular catheters (including multilumen catheters and pulmonary artery catheters) through a single venipuncture. The side-arm infusion port on the catheter provides an additional infusion line, and allows a continuous flush to prevent thrombus formation around smaller catheters that sit in the lumen of the introducer catheter. This side-arm infusion port also allows the introducer catheter to be used as a stand-alone infusion device (a rubber

membrane on the hub of the catheter provides an effective seal when fluids are infusing through the side-arm port of the catheter). The large diameter of introducer catheters makes them particularly valuable as infusion devices when rapid infusion rates are necessary (e.g., in the resuscitation of massive hemorrhage).

ACCESS ROUTES

The following is a brief description of common vascular access routes in the arm (antecubital veins and radial artery), the thoracic inlet (subclavian and jugular veins), and the groin (femoral artery and vein).

ANTECUBITAL VEINS

The veins in the antecubital fossa provide rapid and safe vascular access for acute resuscitative therapy. Although long catheters can be inserted into the antecubital veins and advanced into the superior vena cava, such peripherally inserted central venous catheters (PICC devices) are more appropriate for home infusion therapy than for treating critically ill patients. Short catheters (5 to 7 cm) are preferred for acute resuscitation via the antecubital veins because they are more easily inserted and allow more rapid infusion rates than the longer PICC catheters.

Anatomy

The surface anatomy of the antecubital veins is shown in Figure 4.4. The basilic vein runs along the medial aspect of the antecubital fossa, and the cephalic vein is situated on the opposite side. The basilic vein is preferred for cannulation because it runs a straighter and less variable course up the arm than the cephalic vein.

Insertion Technique

The patient need not be supine, but the arm should be straight and abducted. The antecubital veins can be distended by tourniquet or by inflating a blood pressure cuff to just above the diastolic pressure (this allows arterial inflow while impeding venous outflow). Once the veins are visible or palpable, a catheter-over-needle device is used to insert a short 16- or 18-gauge catheter into the basilic or cephalic vein.

Blind Insertion

If the antecubital veins are neither visible nor palpable, palpate the brachial artery pulse at a point 1 inch above the antecubital crease. The basilic vein (or brachial vein) should lie just medial to the palpated pulse at this point, and can be entered by inserting the catheter-over-needle device through the skin at a 35° to 45° angle and advancing the needle until blood return is noted. This approach has a reported success rate of 80%. Injury to the median nerve (which is also medial to the artery, but deep to the veins) can occur with excessive movement of the probe needle.

Comment

Cannulation of the antecubital veins is recommended (18,19).

For rapid venous access (e.g., cardiopulmonary resuscitation)

For thrombolytic therapy in acute myocardial infarction

For trauma victims who require thoracotomy

Remember that the shorter the catheter, the more rapid the flow rate through the catheter (see Chapter 1). Thus, insertion of short catheters into the antecubital veins permits more rapid volume resuscitation than insertion of the longer central venous catheters.

RADIAL ARTERY

The radial artery is a favored site for arterial cannulation because the vessel is superficial and accessible and the insertion site is easy to keep clean. The major disadvantage of the radial artery is its small size, which limits the success rate of cannulation and promotes vascular occlusion.

Anatomy

The surface anatomy of the radial and ulnar arteries is shown in Figure 4.4. The radial artery is usually palpable at a point just medial to the styloid process of the radius. The ulnar artery is on the opposite (medial) side of the wrist, just lateral to the pisiform bone. Although the radial artery is preferred, the ulnar artery is the larger of the two arteries and should be easier to cannulate.

The Allen Test

The Allen test evaluates the capacity of the ulnar artery to supply blood to the digits when the radial artery is occluded. The test is performed by first occluding the radial and ulnar arteries with the thumb and index finger. The patient is then instructed to raise the wrist above the head and to make a fist repeatedly until the fingers turn white. The ulnar artery is then released, and the time required for return of the normal color to the fingers is recorded. A normal response time is 7 seconds or less, and a delay of 14 seconds or greater is evidence of insufficient flow in the ulnar artery.

Although a positive Allen test (i.e., 14 seconds or longer for return of color to the digits) is often stated as a contraindication to radial artery cannulation, in numerous instances the Allen test has indicated inadequate flow in the ulnar artery, yet subsequent radial artery cannulation has been uneventful (2,21). Thus, a positive Allen test is not a contraindication to radial artery cannulation. Another limitation is the need for patient cooperation to perform the test. Therefore, this test is not worth the time it takes.

Insertion Technique

The wrist should be hyperextended to bring the artery closer to the surface. A short 20-gauge catheter is appropriate, and can be inserted by a catheter-over-needle device or by the guidewire technique. When using a catheter-over-needle device, the following through-and-through technique is recommended: When the needle tip first punctures the artery (and blood appears in the flashback chamber), the tip of the catheter is just outside the vessel. To position the catheter tip in the lumen of the vessel, the needle is passed completely through the artery and then withdrawn until blood returns again through the needle. At this point, the catheter tip should be in the lumen of the artery, and the catheter can be advanced while the needle is retracted. If two attempts at cannulation are unsuccessful, switch to an alternative site (to

reduce trauma to the vessel).

Comment

Arterial occlusion occurs in as many as 25% of radial artery cannulations, but digital ischemia is rare (2,22). Despite being well tolerated in most patients, cannulation of the radial artery (or any artery) should be reserved for monitoring blood pressure, and is not to be used as a convenience measure for monitoring blood gases or other blood components.

THE SUBCLAVIAN VEIN

More than 3 million central venous cannulations are performed yearly in the United States , and a majority of these procedures are performed via the subclavian vein. The subclavian vein is well suited for cannulation because it is a large vessel (about 20 mm in diameter) and is prevented from collapsing by its surrounding structures. The immediate risks of subclavian vein cannulation include pneumothorax (1% to 2%) and hemothorax (less than 1%). The incidence of bleeding is no different in the presence or absence of a coagulopathy ; that is, the presence of a coagulation disorder is not a contraindication to subclavian vein cannulation.

Anatomy

The subclavian vein is a continuation of the axillary vein as it passes over the first rib, and the apical pleura lies about 5 mm deep to the vein at its point of origin. As shown in Figure 4.5, the subclavian vein runs most of its course along the underside of the clavicle. The vein runs along the outer surface of the anterior scalene muscle, which separates the vein from its companion artery on the underbelly of the muscle. At the thoracic inlet, the subclavian vein meets the internal jugular vein to form the brachiocephalic vein. The convergence of the right and left brachiocephalic veins forms the superior vena cava.

Anatomic Distances

The lengths of the vascular segments involved in subclavian (and internal jugular) vein cannulation are shown in Table 4.4. The average distance from venipuncture site to the right atrium is 14.5 cm and 18.5 cm for right-sided and left-sided cannulations, respectively. These

distances are far shorter than catheter lengths recommended for right-sided (20 cm) and left-sided (30 cm) central venous cannulations, and are more consistent with a recent report showing that the average distance to the right atrium is 16.5 cm in central venous cannulation from either side in adults. Therefore, to avoid placing catheter tips in the right atrium (which can lead to cardiac perforation and fatal cardiac tamponade), all central venous catheters should be no longer than 15 or 16 cm in length.

INSERTION TECHNIQUE

The patient is placed supine, with arms at the sides and head faced away from the insertion site. A towel roll can be placed between the shoulder blades, but this is uncomfortable and unnecessary. Identify the clavicular insertion of the sternocleidomastoid muscle. The subclavian vein lies just underneath the clavicle where the muscle inserts onto the clavicle. The vein can be entered from either side of the clavicle.

Infraclavicular Approach (Insertion Site 1 in Figure 4.5). Identify the lateral margin of the sternocleidomastoid muscle as it inserts on the clavicle. The catheter is inserted in line with this margin, but below the clavicle. Insert the probing needle (18 or 20 gauge) with the bevel pointing upward (toward the ceiling) and advance the needle along the underside of the clavicle and toward the suprasternal notch. The path of the needle should be parallel to the patient's back. When the vein is entered, turn the bevel of the needle to 3 o'clock so the guidewire threads in the direction of the superior vena cava.

Supraclavicular Approach (Insertion Site 2 in Figure 4.5). Identify the angle formed by the lateral margin of the sternocleidomastoid muscle and the clavicle. The probe needle is inserted so that it bisects this angle. Keep the bevel of the needle facing upward and direct the needle under the clavicle in the direction of the opposite nipple. The vein should be entered at a distance of 1 to 2 cm from the skin surface (the subclavian vein is more superficial in the supraclavicular approach). When the vein is entered, turn the bevel of the needle to 9 o'clock so the guidewire threads in the direction of the superior vena cava.

Comment

Patient comfort and ease of insertion are the most compelling reasons to select the subclavian

vein for central venous access. Selection of the infraclavicular versus supraclavicular approach is largely a matter of personal preference. Some recommend avoiding the subclavian vein in ventilator-dependent patients because of the risk of pneumothorax. However, the risk of pneumothorax is too small to justify abandoning the subclavian vein in patients with respiratory failure.

THE INTERNAL JUGULAR VEIN

Cannulation of the internal jugular vein reduces (but does not eliminate) the risk of pneumothorax, but introduces new risks (e.g., carotid artery puncture and thoracic duct injury).

Anatomy

The internal jugular vein is located under the sternocleidomastoid muscle in the neck and, as shown in Figure 4.5, the vein follows an oblique course as it runs down the neck. When the head is turned to the opposite side, the vein forms a straight line from the pinna of the ear to the sternoclavicular joint. Near the base of the neck, the internal jugular vein becomes the most lateral structure in the carotid sheath (which contains the carotid artery sandwiched between the vein laterally and the vagus nerve medially).

Insertion Technique

The right side is preferred because the vessels run a straighter course to the right atrium. The patient is placed in a supine or Trendelenburg position, with the head turned to the opposite side. The internal jugular vein can be entered from an anterior or posterior approach.

The Anterior Approach (Insertion Site 4 in Figure 4.5). The anterior approach is through a triangular region created by two heads of the sternocleidomastoid muscle. The carotid artery is palpated in the triangle and retracted medially. The probe needle is inserted at the apex of the triangle with the bevel facing up, and the needle is advanced toward the ipsilateral nipple, at a 45° angle with the skin surface. If the vein is not encountered by a depth of 5 cm, the needle is withdrawn 4 cm and advanced again in a more lateral direction. When a vessel is entered, look for pulsations. If the blood is red and pulsating, you have entered the carotid artery. In this situation, remove the needle and tamponade the area for 5 to 10 minutes. When the carotid

artery has been punctured, no further attempts should be made on either side because puncture of both arteries can have serious consequences.

The Posterior Approach (Insertion Site 3 in Figure 4.5). The insertion site for this approach is 1 centimeter superior to the point where the external jugular vein crosses over the lateral edge of the sternocleidomastoid muscle. The probe needle is inserted with the bevel positioned at 3 o'clock. The needle is advanced along the underbelly of the muscle in a direction pointing to the suprasternal notch. The internal jugular vein should be encountered 5 to 6 cm from the skin surface with this approach.

Carotid Artery Puncture. If the carotid artery has been punctured with a probing needle, the needle should be removed and pressure should be applied to the site for at least 5 minutes (10 minutes is recommended for patients with a coagulopathy). No further attempts should be made to cannulate the internal jugular vein on either side, to avoid puncture of both carotid arteries. If the carotid artery has been inadvertently cannulated, the catheter should not be removed, as this could provoke serious hemorrhage. In this situation, a vascular surgeon should be consulted immediately.

Comment

As with the subclavian vein, cannulation of the internal jugular vein is safe and effective when performed by skilled operators. However, several disadvantages of internal jugular cannulation deserve mention. (a) Accidental puncture of the carotid artery is reported in 2 to 10% of attempted cannulations. (b) Awake patients often complain of the limited neck mobility when the internal jugular vein is cannulated. (c) In agitated patients, inappropriate neck flexion can result in thrombotic occlusion of the catheter and vein. (d) In patients with tracheostomies, the insertion site can be exposed to infected secretions that drain from the tracheal stoma.

THE EXTERNAL JUGULAR VEIN

Cannulation of the external jugular vein has two advantages: (a) There is no risk of pneumothorax, and (b) hemorrhage is easily controlled. The major drawback is difficulty advancing the catheter.

Anatomy

The external jugular vein runs along a line extending from the angle of the jaw to a point midway along the clavicle. The vein runs obliquely across the surface of the sternocleidomastoid muscle and joins the subclavian vein at an acute angle. This acute angle is the major impediment to advancing catheters that have been inserted into the external jugular vein.

Insertion Technique

The patient is placed in the supine or Trendelenburg position, with the head turned away from the insertion site. If necessary, the vein can be occluded just above the clavicle (with the forefinger of the nondominant hand) to engorge the entry site. As many as 15% of patients so not have an identifiable external jugular vein, even under optimal conditions of vein engorgement.

The external jugular vein has little support from surrounding structures, so the vein should be anchored between the thumb and forefinger when the needle is inserted. The bevel of the needle should be pointing upward when it enters the vein. The recommended insertion point is midway between the angle of the jaw and the clavicle (see Fig. 4.5). Use a 16-gauge single-lumen catheter that is 10 to 15 cm in length. If the catheter does not advance easily, do not force it, as this may result in vascular perforation at the junction between the external jugular and subclavian veins.

Comment

This approach is best reserved for temporary access in patients with a severe coagulopathy, particularly when the operator is inexperienced and does not feel comfortable cannulating the subclavian or internal jugular veins. Contrary to popular belief, cannulation of the external jugular is not always easier to accomplish than central venous cannulation because of the difficulty in advancing catheters past the acute angle at the junction of the subclavian vein.

THE FEMORAL VEIN

The femoral vein is the easiest of the large veins to cannulate and also does not carry a risk of [pneumothorax](#). The disadvantages associated with this route are venous thrombosis (10%), femoral artery puncture (5%), and limited ability to flex the hip (which can be bothersome for awake patients). Contrary to popular belief, the infection rate with femoral vein catheters is no different from that of subclavian or internal jugular vein catheters.

Anatomy

The anatomy of the femoral sheath is shown in Figure 4.6. The femoral vein is the most medial structure in the femoral sheath and is situated just medial to the femoral artery. At the inguinal ligament, the femoral vessels are just a few centimeters below the skin surface.

Insertion Technique

Palpate the femoral artery just below the inguinal crease and insert the needle (bevel up) 1 to 2 cm medial to the palpated pulse. Advance the needle at a 45° angle to the skin surface, entering the vein at a depth of 2 to 4 cm. Once in the vessel, if the catheter or guidewire will not pass beyond the tip of the needle, tilt the needle so that it is more parallel to the skin surface (this may move the needle tip away from the far side of the vessel wall and into more direct contact with the lumen of the vessel). Femoral vein catheters should be at least 15 cm long.

Blind Insertion

If the femoral artery pulse is not palpable, draw an imaginary line from the anterior superior iliac crest to the pubic tubercle, and divide the line into three equal segments. The femoral artery lies at the junction between the middle and most medial segment, and the femoral vein is 1 to 2 cm medial to this point. This method of locating the femoral vein has a reported success rate of over 90%.

Comment

Femoral vein cannulation is usually reserved for patients who are paralyzed or comatose and immobile. This approach is not recommended for cardiopulmonary resuscitation (because of the delayed transit times for bolus drug injections) or in patients with bleeding disorders.

THE FEMORAL ARTERY

Cannulation of the femoral artery is usually reserved for situations where radial artery cannulation is unsuccessful or contraindicated. Despite its reserve status, the femoral artery is larger than the radial artery, and is easier to cannulate. The complications of femoral artery cannulation are the same as for radial artery cannulation (thrombosis, bleeding, and infection). The incidence of infection is the same with radial and femoral artery catheters, and the incidence of thrombosis is lower with femoral artery cannulation. Thrombosis of the femoral artery, like that in the radial artery, only rarely results in troublesome ischemia in the distal extremity.

Localization and cannulation of the femoral artery proceeds as described in the section on femoral vein cannulation. The Seldinger technique is preferred for catheter insertion, and catheters should be 18 gauge in diameter and 15 to 20 cm long.

Comment

Femoral artery cannulation is a viable alternative and may be preferable to radial artery cannulation in patients who are paralyzed or otherwise immobile, unless they have a significant coagulopathy (in which case the radial artery is preferred). The incidence of thrombotic complications is lower in femoral artery cannulations, and the pressure in the femoral artery more closely approximates the pressure in the aorta than does the pressure in the radial artery (see Chapter 8).

IMMEDIATE CONCERNS

VENOUS AIR EMBOLISM

Inadvertent air entry is one of the most feared complications of central venous cannulation. The importance of maintaining a closed system during insertion is highlighted by the following statement:

A pressure gradient of 4 mm Hg along a 14-gauge catheter can entrain air at a rate of 90

mL/second and can produce a fatal air embolus in 1 second.

Preventive Measures

Prevention is the hallmark of reducing the morbidity and mortality of venous air embolism. The most effective method of preventing air entry is to keep the venous pressure more positive than atmospheric pressure. This is facilitated by placing the patient in the Trendelenburg position with the head 15° below the horizontal plane. Remember that the Trendelenburg position does not prevent venous air entry because patients still generate negative intrathoracic pressures while in the Trendelenburg position. When changing connections in a central venous line, a temporary positive pressure can be created by having the patient hum audibly. This not only produces a positive intrathoracic pressure, but allows clinicians to hear when the intrathoracic pressure is positive. In ventilator-dependent patients, the nurse or respiratory therapist should initiate a mechanical lung inflation when changing connections.

Clinical Presentation

The usual presentation is acute onset of dyspnea that occurs during the procedure. Hypotension and cardiac arrest can develop rapidly. Air can pass across a patent foramen ovale and obstruct the cerebral circulation, producing an acute ischemic stroke. A characteristic "mill wheel" murmur can be heard over the right heart, but this murmur may be fleeting.

Therapeutic Maneuvers

If a venous air embolism is suspected, immediately place the patient with the left side down, and attempt to aspirate air directly from the venous line. In dire circumstances, a needle should be inserted through the chest wall and into the right ventricle to aspirate the air. Unfortunately, the mortality in severe cases of venous air embolism remains high despite these maneuvers.

PNEUMOTHORAX

Pneumothorax is a concern primarily with subclavian vein cannulation but can also complicate jugular vein cannulation (2,30). This is one reason that postinsertion chest films are recommended after all central venous cannulations (or attempts). If possible, postinsertion

films should be obtained in the upright position and during expiration. Expiratory films facilitate the detection of small pneumothoraxes because expiration decreases the volume of air in the lungs, but not the volume of air in the pleural space. Thus, during expiration, the volume of air in [the pleural space](#) is a larger fraction of the total volume of the hemithorax, thereby magnifying the radiographic appearance of the pneumothorax.

Upright films are not always possible in ICU patients. When supine films are necessary, remember that pleural air does not often collect at the apex of the lung when the patient is in the supine position (32,33). In this situation, pleural air tends to collect in the subpulmonic recess and along the anteromedial border of the mediastinum (see Chapter 28).

Delayed Pneumothorax

Pneumothoraxes may not be radiographically evident until 24 to 48 hours after central venous cannulation (31,33). Therefore, the absence of a pneumothorax on an immediate postinsertion chest film does not absolutely exclude the possibility of a catheter-induced pneumothorax. This is an important consideration in patients who develop dyspnea or other signs of pneumothorax in the first few days after central venous cannulation. In the absence of signs and symptoms, there is little justification for serial chest films following central venous catheter placement.

CATHETER TIP POSITION

The properly placed central venous catheter should run parallel to the superior vena cava, and the tip of the catheter should be positioned above the junction of the superior vena cava and right atrium. The following conditions warrant corrective measures.

Tip Against the Wall of the Vena Cava

Catheters inserted from the left side must make an acute turn downward when they reach the superior vena cava. If they fail to make this turn, the catheters can end up in a position like the one shown in Figure 4.7. The tip of the catheter is up against the lateral wall of the vena cava, and in this position, the catheter tip can stab the vessel wall and perforate the vena cava. Therefore, catheters that abut the wall of the vena cava should be repositioned as soon as

possible. (The problem of vascular perforation is discussed in more detail in Chapter 5.)

Tip in the Right Atrium

The Food and Drug Administration has issued a strong warning about the risk of cardiac perforation from catheter tips that are advanced into the heart. However, cardiac perforation is a rare complication of central venous cannulation, even though over half of central venous catheters may be misplaced in the right atrium. Nevertheless, tamponade is often fatal, so cardiac placement of catheters should be avoided. A few measures help to minimize the risk of cardiac perforation. The most effective measure is to use shorter catheters, as recommended earlier. The tip of indwelling catheters should be above the third right costal cartilage (this is the level where the vena cava meets the right atrium). If the anterior portion of the third rib cannot be visualized, keep the catheter tip at or above the tracheal carina.

Cardiopulmonary resuscitation

Cardiopulmonary resuscitation (CPR) is an emergency first aid procedure for a victim of cardiac arrest. It is part of the chain of survival, which includes early access (to emergency medical services), early CPR, early defibrillation, and early advanced care. It is also performed as part of the choking protocol if all else has failed. It can be performed by trained laypersons or by health care or emergency response professionals. It is normally begun on an unbreathing unconscious person and continued until the underlying cause can be identified and a pulse is restored. CPR consists of chest compressions and rescue breaths (i.e. artificial blood circulation and lung ventilation) and is intended to maintain a flow of oxygenated blood to the brain and the heart, thereby extending the brief window of opportunity for a successful resuscitation without permanent brain damage.

Many countries have official guidelines on how CPR should be provided, and these naturally override the general description of CPR in this article.

In 2005, new CPR guidelines were published with input from International Resuscitation Councils, and was agreed at the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science. The primary goal was to simplify CPR for lay rescuers and healthcare providers alike to maximise the potential

for early resuscitation. The important changes for 2005 are as follow.

A universal compression-ventilation ratio (30:2) is recommended for all single rescuers of infant (less than one year old), child (1 year old to puberty), and adult (puberty and above) victims (excluding newborns). The only difference between the age groups is that with adults the rescuer uses two hands for the chest compressions, while with children it is only one, and with infants only two fingers (pointer and middle fingers). It is worth noting that paediatric guidelines for healthcare professionals differ from the 30:2 compression-ventilation ratio stated here.

Lay rescuers do not need to assess for pulse or signs of circulation for an unresponsive adult victim.

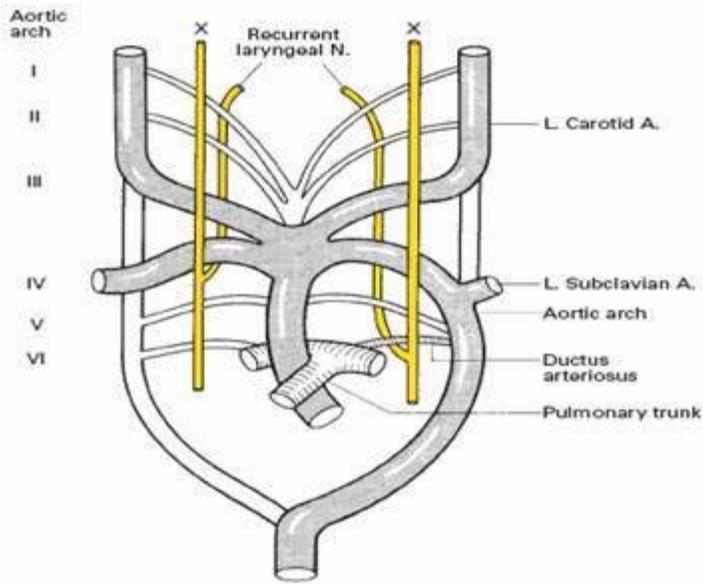
Lay rescuers do not need to provide rescue breathing without chest compressions for an adult victim.

As research has shown that lay personnel cannot accurately detect a pulse in about 40% of cases and cannot accurately discern the absence of pulse in about 10%, the pulse check step has been removed from the CPR procedure completely for lay persons and de-emphasized for healthcare professionals.

Cardiac arrest and the place of CPR

The medical term for the condition in which a person's heart has stopped is cardiac arrest (also referred to as cardiorespiratory arrest). CPR appropriate for cardiac arrest. If the patient still has a pulse, but is not breathing, this is called respiratory arrest and rescue breathing is more appropriate.

The Heart



However, since people often can't tell the difference (can't accurately feel a pulse to determine whether the heart is still beating), CPR is often recommended for both.

The most common cause of cardiac arrest outside of a hospital is ventricular fibrillation (VF), a potentially fatal arrhythmia that is usually (but not always) caused by a heart attack. Other causes of cardiac arrest include drowning, drug overdose, poisoning, electrocution.

Sudden cardiac arrest is a leading cause of death, approximately 250,000 per annum outside a hospital setting in the USA. CPR can double or triple the victim's chances of survival when commenced immediately. According to American Heart Association, only two thirds of victims of a witnessed cardiac arrest are administered CPR. Rapid access to defibrillation is also vital.

Blood circulation and lung ventilation are absolute requirements in transporting oxygen to the tissues. The brain may sustain damage after four minutes and irreversible damage after about seven minutes. The heart also rapidly loses the ability to maintain a normal rhythm. Low body temperatures as seen in drownings prolong the time the brain survives. Following cardiac arrest, effective CPR enables enough oxygen to reach the brain to delay brain death, and allows the heart to remain responsive to defibrillation attempts.

CPR is taught to the general public because they are the only ones present in the crucial few

minutes before emergency personnel are available. Simple training is the goal of the 2005 guidelines to maximise the prospect that CPR will be performed successfully.

Effectiveness

CPR is almost never effective if started more than 15 minutes after collapse because permanent brain damage has probably already occurred, especially if the person has stopped breathing, since the brain can only survive for 4-6 minutes without oxygen.[citation needed] A notable exception is cardiac arrest occurring in conjunction with exposure to very cold temperatures. Hypothermia seems to protect the victim by slowing down metabolic and physiologic processes, greatly decreasing the tissues' need for oxygen.[citation needed] There are cases where CPR, defibrillation, and advanced warming techniques have revived victims after substantial periods of hypothermia.

Used alone, few patients will make a complete recovery, and those that do survive often develop serious complications. Estimates vary, but many organizations[citation needed] stress that CPR does not "bring anyone back," it simply preserves the body for defibrillation and advanced life support. However, in the case of "non-shockable" rhythms such as Pulseless Electrical Activity (PEA), defibrillation is not indicated, and the importance of CPR rises. On average, only 5%-10% of people who receive CPR survive. The purpose of CPR is not to "start" the heart, but rather to circulate oxygenated blood, and keep the brain alive until advanced care (especially defibrillation) can be initiated. As many of these patients may have a pulse that is unpalpable by the layperson rescuer, the current consensus is to perform CPR on a patient that is not breathing. A pulse check is not required in basic CPR since it is so often missed when present, or even felt when absent, even by health care professionals

Studies have shown the importance of immediate CPR followed by defibrillation within 3–5 minutes of sudden VF cardiac arrest improve survival. In cities such as Seattle where CPR training is widespread and defibrillation by EMS personnel follows quickly, the survival rate is about 30 percent. In cities such as New York City, without those advantages, the survival rate is only 1-2 percent.

CPR is often severely misportrayed in movies and television as being highly effective in resuscitating a person who is not breathing and has no circulation. A 1996 study published in the New England Journal of Medicine showed that CPR success rates in television shows was

75%.

It is considered by a number of international bodies that in order for CPR to be effective, the guidelines must be simple and easy to remember.[citation needed]

CPR training

CPR is a practical skill and needs professional instruction followed up by regular practice on a resuscitation mannequin to gain and maintain full competency. Training is available through many commercial, volunteer and government organizations worldwide.

CPR training is not confined to the medical professionals. To be effective, CPR must be applied almost immediately after a patient's heart has stopped. Early CPR on the scene of an incident is essential to the prevention of brain damage during a cardiac arrest. Blood flow and air supply to the brain and other major organs is maintained until a defibrillator and professional medical help arrives. Almost anyone is able to perform CPR with training, and health organizations advocate the development of CPR skills throughout the general public.

It is best to obtain training in CPR before a medical emergency occurs. One needs hands-on training by experts to perform CPR safely, and guidelines change, so that training should be repeated every one or two years. Training in first aid is often available through community organizations such as the Red Cross and St. John Ambulance. In many countries in the Commonwealth of Nations, St. John Ambulance and the Medic First Aid Organization provide CPR training. In Scotland, St. Andrew's Ambulance Association provides first aid training. In the United States, the American Red Cross, American Heart Association and American CPR Training also offer CPR training. In addition, many employees at public areas or community centres are trained in CPR. Lifeguards are also trained in CPR and other first aid protocols.

In most CPR Classes a simple shortform is used for people to remember everything they need to do. The most common one used worldwide is DRABCD which stands for Danger, Response, Airway, Breathing, Compressions and Defibrillation.

History

CPR has been known in theory, if not practice, for many hundreds or even thousands of years; some claim it is described in the Bible, discerning a superficial similarity to CPR in a passage from the Books of Kings (II 4:34), wherein the Hebrew prophet Elisha warms a dead boy's body and "places his mouth over his". In the 19th century, doctor H. R. Silvester described a method (The Silvester Method) of artificial respiration in which the patient is laid on their back, and their arms are raised above their head to aid inhalation and then pressed against their chest to aid exhalation. The procedure is repeated sixteen times per minute. This type of artificial respiration is occasionally seen in movies made in the early part of the 20th century.

A second technique, described in the first edition of the Boy Scout Handbook in the United States in 1911, described a form of artificial respiration where the person was laid on their front, with their head to the side, and a process of lifting their arms and pressing on their back was utilized, essentially the Silvester Method with the patient flipped over. This form is seen well into the 1950s (it is used in an episode of Lassie during the Jeff Miller era), and was often used, sometimes for comedic effect, in theatrical cartoons of the time. This method would continue to be shown, for historical purposes, side-by-side with modern CPR in the Boy Scout Handbook until its ninth edition in 1979.

However it wasn't until the middle of the 20th century that the wider medical community started to recognise and promote it as a key part of resuscitation following cardiac arrest. Peter Safar wrote the book ABC of resuscitation in 1957. In the U.S., it was first promoted as a technique for the public to learn in the 1970s. Early marketing efforts oversold the effectiveness of CPR in rescuing heart attack and other victims, and this misperception continues even today, as the success rate for CPR is only 1/20.

Self-CPR

A form of "self-CPR" termed "Cough CPR" may help a person maintain blood flow to the brain during a heart attack while waiting for medical help to arrive and has been used in a hospital emergency room in cases where "standard CPR" was contraindicated. While this technique is not in widespread use, one researcher has recommended that it be taught broadly to the public. However, the American Heart Association (AHA), does not endorse "Cough CPR", which it terms a misnomer as it is not a recognized form of resuscitation. The AHA does recognize a limited legitimate use of the coughing technique:

This coughing technique to maintain blood flow during brief arrhythmias has been useful in the hospital, particularly during cardiac catheterization. In such cases the patient's ECG is monitored continuously, and a physician is present.

"Cough CPR" was the subject of a hoax chain e-mail entitled "How to Survive a Heart Attack When Alone" which wrongly cited "ViaHealth Rochester General Hospital" as the source of the technique. Rochester General Hospital has denied any connection with the technique.

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The ABC Of Resuscitation

1. To check an unconscious victim, place two fingers under his chin and a hand on his forehead. Tilt his head back to open his airway. Remove any obstructions from his mouth.
2. Listen and feel for victim's breathing. If he is breathing, place him in the recovery position. If he is not breathing, begin rescue breathing.
3. Check the victim's circulation by feeling for a pulse at the side of his windpipe (carotid artery). If there is no pulse, begin CPR immediately.



Fig. Steps of resuscitation

The Recovery Position

1. If a victim is unconscious but breathing, bend his near arm up at a right angle to his body. Hold the back of his far hand to his near cheek. With the near leg straight, pull the far knee toward you.
2. With the victim on his side, place his uppermost leg at a right angles to his body. His head will be supported by the hand of the uppermost arm. Tilt his head back so that he will not choke if he vomits.

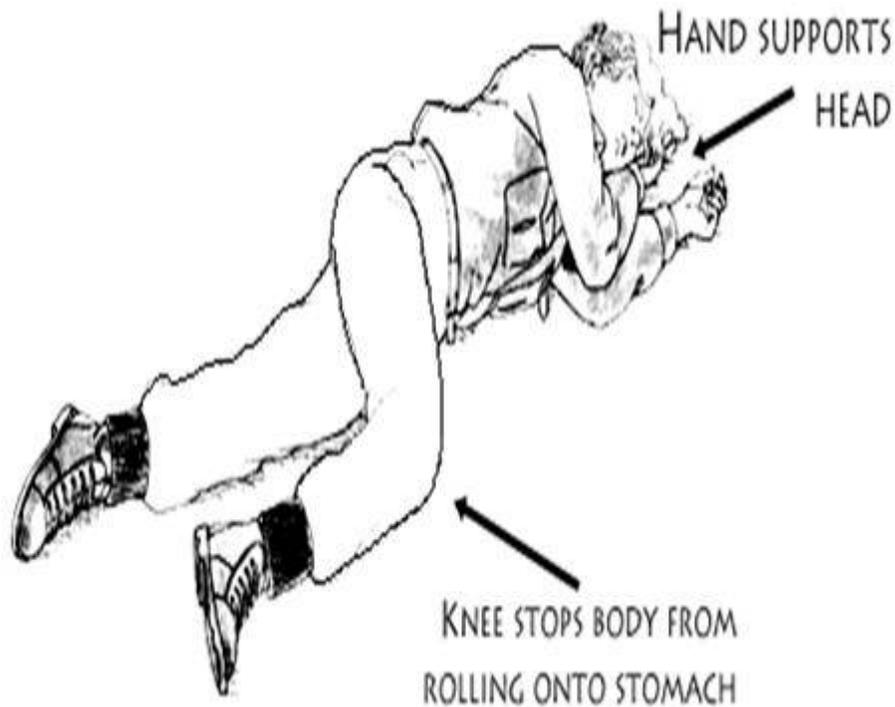


Fig. Position of patient during resuscitation

Rescue Breathing

1. To ensure an open airway, first clear the victim's mouth of obstructions, then place one hand under his chin and one on his forehead, and tilt his head back.
2. Pinching the victim's nose shut, clamp your mouth over his mouth, and blow steadily for about two seconds until his chest rises. Remove your mouth and let his chest fall, then repeat.
3. Listen for the victim's breathing and check his pulse. If he still has a pulse, give 10 breaths per minute until help arrives or the victim is breathing by himself. If the pulse has stopped, combine rescue breathing with chest compressions.



Fig. First step of resuscitation

Cardiopulmonary Resuscitation (CPR)

If a person's heart has stopped, give cardiopulmonary resuscitation (CPR). This consists of chest compressions to maintain the blood flow to the brain, combined with rescue breathing to oxygenate the blood. Give chest compressions at a rate of 80 per minute, counting "one-and-two-and..."

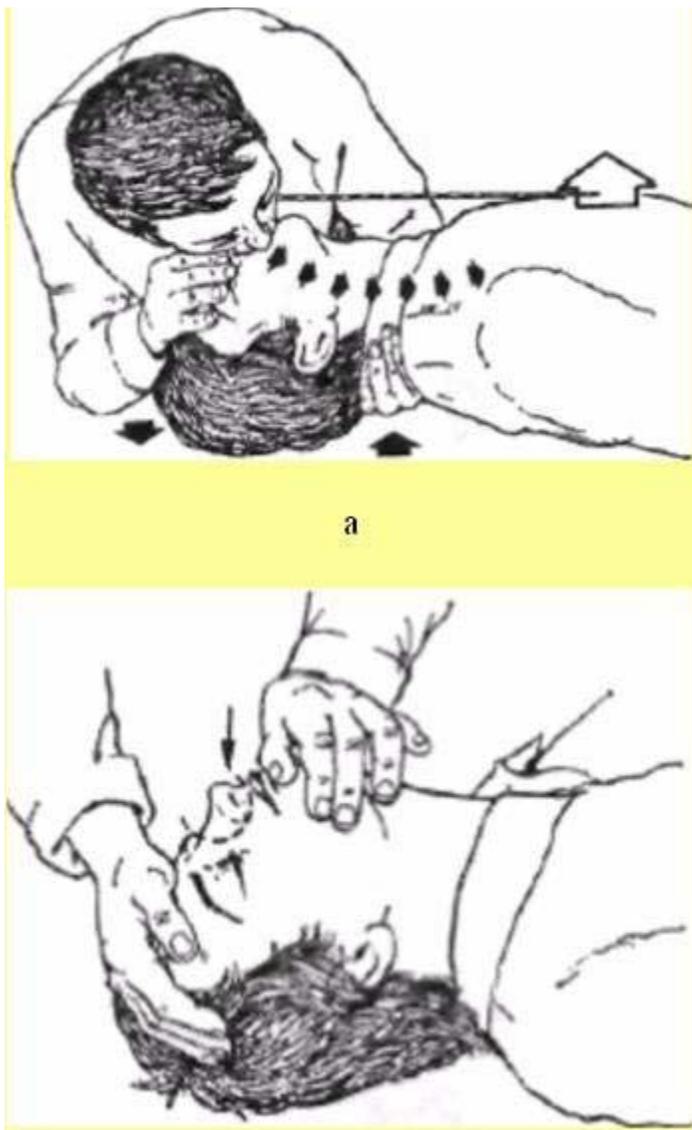


Fig. Artificial lung ventilation

1. Place the heel of your hand two finger-widths up from the end of the sternum and your other hand on top of the first. Press down firmly, then release.
2. Check for a pulse. After 15 chest compressions, give the victim two breaths of rescue breathing. Repeat until the pulse restarts, professional help arrives, or you are too exhausted to continue.

Control of the airway is the single most important task for emergency resuscitation. If the patient has inadequate oxygenation or ventilation, inability to protect the airway due to altered sensorium from illness or drugs, or external forces compromising the airway (i.e., trauma), he or she may need advanced airway techniques as described in this chapter.

CPR is now a days is called as CPCPR.

Cardiac arrest is defined as 'inability of heart to sustain effective cardiac output, impairing tissue perfusion.

Cardiac arrest may be witnessed (monitored) or unwitnessed (unmonitored). It may be inside hospital or outside hospital.

Cardiac arrest may be of cardiac origin or non cardiac.

Cardiac arrest in adults is usually of cardiac origin and in children it is usually of respiratory origin and most common cause of arrest in adults is ventricular fibrillation.

Therefore unwitnessed cardiac arrest in adults should be considered due to ventricular fibrillation until proved otherwise and in children should be considered due to asystole until proved otherwise.

Rhythms in Cardiac Arrest

1. Ventricular fibrillation (or pulseless ventricular tachycardia) - most common rhythm.
2. Asystole - most common in children.
3. Electromechanical dissociation (pulse less electrical activity).

Management guidelines are based on the recent recommendation (2005) by American Heart Association (AHA) and Emergency Cardio Vascular care (ECC) with International Lesion Committee on Resuscitation (ICCOR).

The management of CPR should be done in sequence A (Airway)—Breathing (B)—Circulation (C) and must be followed in these sequence.

Management of CPR is done under three heads:

1. Basic life support.
2. Advance life support.
3. Post resuscitation life support.

AHA and ECC have given 4 link chain of survival: 1. Early recognition and activation of emergency medical services.

2. Immediate CPR (every minute delay decreases prognosis by 7-10%).
3. Early shock (CPR + shock within 5 minutes has survival rate of 49-75%).
4. Early advance life support.

BASIC LIFE SUPPORT (BLS) (Primary A, B, C, D)

Basic life support includes:

1. Airway management (manual).
2. Breathing by mouth to mouth, mouth to nose or bag and mask ventilation.

3. Circulation by cardiac massage.

4. Defibrillation by automatic external defibrillator (AED): AED are the devices which automatically detects rhythm and give shock if rhythm is shockable. Since they detect rhythm automatically and gives shock automatically they can be used even by lay rescuer. (In manual defibrillators rescuer has to detect rhythm, therefore not included as basic life support as they can be used only by health personnel).

ADVANCE LIFE SUPPORT (ALS)

Advance life support includes:

1. Airway management by equipments like Guedel 's airway, laryngeal mask airway or endotracheal tube.
2. Breathing by advanced airways e. endotracheal tube, laryngeal mask, combitube or tracheostomy.
3. Circulation by cardiac massage.
4. Defibrillation by manual defibrillators.
5. Drugs.

AIRWAY MANAGEMENT

The most common cause of airway obstruction in unconscious patient is tongue fall (e. tongue falling

Parameter	Basic life support (Primary A,B,C,D)	Advance life support (Secondary A,B,C,D)
Airway management	Manual	With equipments like Guedel's airway, endotracheal tube laryngeal mask airway (LMA) or tracheostomy tube.
Breathing	Mouth to mouth, mouth to LMA, nose or bag & mask	Advanced methods like endotracheal tube, combitube or tracheostomy tube.
Circulation	Cardiac massage.	Cardiac massage
Defibrillation	Automatic external defibrillator	Manual defibrillator
Drugs	- back on posterior pharyngeal wall and obstructing the airway).	

This tongue fall can be managed by:

A. Manually which includes:

Open mouth and clear airways (if something is clearly visible in oral cavity).

i Tilt head backwards (e. neck extension) and chin lift. Jaw thrust e. mandible is pulled forward. In patients with cervical spine fracture head tilt and chin lift is C/I and airway should be managed only by jaw thrust

Airway insertion: Most commonly used in Guedel's, other are Safar, nasopharyngeal, laryn-

geal mask airway (LMA),

C. Endotracheal tube: It is the most definitive method to maintain airway.

Management of Airway Obstruction Due to Foreign Body

1. Infant chest thrust: 4 blows given with thrust by heel of hand between the shoulders.
2. Back blows: 4 blows on the middle of back. Again performed for infants.
3. Heimlich manoeuvre: Manual thrust with the patient standing, rescuer behind the patient and compressing the abdomen 6 to 10 times. This method is for adults and older children.
4. Chest thrust (manual compression over lower sternum): Employed in very obese or pregnant patient where abdominal compression is not possible.
5. Finger sweep method: Possible in unconscious patients only.
6. Cricothyroidotomy: As a life saving procedure to secure airway.

Airway obstruction due to other causes like laryngeal edema, acute epiglottitis and laryngotracheobronchitis may require tracheostomy.

BREATHING (Ventilation)

Breathing can be accomplished by:

1. Mouth to mouth:

Method: Open airway — pinch victim's nose — create airtight seal — give breath over 1 sec with sufficient force to move chest (not excessive force) — provide 2nd breath in the same way if rescuer is hesitant in giving direct mouth to mouth he/she can use face shields/plastic or silicone sheets or even a handkerchief, if nothing is available.

2. Mouth to airway: By Safar or Brook airway.
3. Bag and mask. Disadvantage of bag and mask

ventilation are:

Difficult and exhausting.

Increased dead space.

Increased chances of aspiration.

4. Ventilation by advanced methods:

Endotracheal tube: Intubation is most definitive and best method for ventilation. Laryngeal mask airway, Combitube. Tracheostomy tube.

5. Ventilation by automatic ventilators.

CIRCULATION

This is accomplished by cardiac massage.

Method

It is done in supine position. But in very rare cases, where supine position is not possible it can be done in prone position also.

The rescuer stand (or bend on knee if the victim is on floor) on side (usually right side), lock one hand over other and provide compression over the lower third of sternum (2 fingers above xiphoid process).

The force generated during massage should be able to depress sternum by 1.5-2 inches (approx 73 of chest wall diameter). During massage compression relaxation (chest to recoil) should be equal.

Patient should lie flat on hard surface.

Physiological Considerations of Cardiac Massage

Heart compressed between sternum and spine results in ejection of blood from heart (cardiac pump theory) but another convincing theory is thoracic pump theory which states that cardiac compression raises intrathoracic pressure forcing blood out of chest and dynamic venous compression preventing backward flow, heart acting only as a passive conduit.

Chest compression can generate a systolic BP of 80-100 mmHg but diastolic only 10-40 mmHg (which may compromise coronary flow). Effective cardiac output generated by successful massage is only 30% of normal. So all efforts to restore spontaneous cardiac activity should be started immediately.

Monitoring of CPR

1. Capnography: It is considered as most reliable and best Indicator to see the effectiveness of CPR. Successful cardiac massage should be able to produce ETCO₂ of at least 20 mmHg and ETCO₂ becoming normal (40 mmHg), is the earliest indicator of return of normal circulation.
2. Palpation of pulses (carotid): It is the most effective clinical monitor to see the effectiveness of cardiac massage. A successful cardiac massage should be able to generate a major peripheral pulse especially carotid.
3. Coronary perfusion pressure: Good guide but very difficult to measure.

COMPRESSION (C) TO VENTILATION (V) RATIO

Before advanced airway (e. by mouth to mouth, bag and mask ventilation), the ratio should be 30:2 (30 compressions followed by 2 breaths) irrespective of duration of resuscitation (previously this ratio was 15:2). The reason for change in ratio is to minimize interruption in chest compression (this is the most important goal of latest recommendations).

After advanced airway (e. endotracheal tube, LMA or combitube): Compression will be

continued at a rate of 100 compressions/minute and breathing at a rate of 8-10 breaths/min. with no synchronization e. no pause for ventilation. The aim is again same e. uninterrupted compressions. Previous ratio of 5:1 now is obsolete, rather ventilation rate should not exceed more than 12 breaths/min. as high rate (more ventilation will increase intrathoracic pressure which decreases venous return thereby decreasing cardiac output and hence coronary and cerebral perfusion). Therefore low tidal volume (6-7 ml/kg) is preferred over large tidal volume (10-12 ml/kg) and one breath should not exceed more than 1 second.

If there are 2 rescuers they should rotate after 2 minutes (or 5 cycles of 30:2) to avoid fatigue of one person providing compression (because studies have shown that after 2 minutes the performance of compression start decreasing because of fatigue), but this change over should not take more than 5 seconds.

Therefore it can concluded that key guidelines for present day CPR are (i) push hard () push fast (Hi) allow full chest recoil and (iv) minimize interruption in chest compressions.

ADVANCED LIFE SUPPORT (Secondary ABCD)

As described earlier in advanced life support airway and ventilation are maintained by advance airway method and circulation by continuing cardiac massage, recognising arrhythmias and managing them by defibrillators and drugs or any other intervention done beyond basic life support is included in advance life support. Therefore advanced life support is employed in hospital arrest.

Airway management and ventilation employed in advance life support has been described in detail in chapter on instrument (chapter no. 6).

Defibrillation: Done for ventricular fibrillation, pulseless ventricular tachycardia and polymorphic ventricular tachycardia.

Defibrillators have been classified as: Monophasic (delivers current of one polarity only) and biphasic (delivers current of two polarities), Manual or automatic (AED).

Biphasic are considered superior to monophasic and that is why most of the defibrillators which are manufactured today are biphasic.

POSITION OF DEFIBRILLATION PADDLES

The ideal position would be like that the heart is sandwiched between paddles e., one is placed anteriorly at the precordial region and second one posteriorly but this is not possible because patient lies supine so usually 1 st paddle is put on right side of chest just below the clavicle and 2nd at precordial region. Paddle should be applied with pressure equivalent to 10 kg.

Paddle size

- For adults: 13 cm.
- For children: 8 cm.
- For infants :4.5cm.

Latest Recommendations for Energy selection and shock protocol.

1. Contrary to previous recommendation of 3 successive shocks (200,300 & 360 J) now a days Only Single Shock of 360J by monophasic defibrillators, and 150-200J withbiphasic [biphasic 150-200J is equivalent to 360 J of monophasic) is given.

2. Immediately after giving shock, give 5 cycles of 30:2 (without advance airway) or 2 minutes of CPR (with advance airway) before checking rhythm [Earlier rhythm was checked just after giving shock]. The rationale for this recommendation is that it actually takes around 2 minutes for heart to recover completely after ventricular fibrillation so CPR during this 2 minutes is required to maintain coronary and cerebral perfusion.

So the protocol for advance life support maybe in continuation of basic life support (where BLS is provided by lay rescuers/paramedicals) or fresh management in hospital arrest.

Consider termination of efforts if there is no response after 20 minutes.

MANAGEMENT OF PULSELESS ELECTRICAL ACTIVITY AND ASYSTOLE

PULSELESS ELECTRICAL ACTIVITY (PEA)

Also called as electromechanical dissociation (EMD). It is the condition in which peripheral pulses are not palpable but heart shows some electrical activity on ECG other than ventricular tachycardia and ventricular fibrillation.

Causes are

1. Hypovolemia (most common cause).
2. Cardiac tamponade.
3. Tension pneumothorax.
4. Pulmonary embolism.
5. Hypoxia.
6. Hyperkalemia.
7. Hypothermia.
8. Acidosis.
9. Overdosage of digitalis, (5 blockers, calcium channel blockers.

(Conditions 6 to 9 show idioventricular rhythm).

Treatment

Treatment of PEA lies in treating the cause, adrenaline

PROTOCOL FOR ADVANCED LIFE SUPPORT

Assess responsiveness

Call for help (Activate EMS)

Manage airway and breathing⁸

Asses circulation -> if present give breaths at a rate of 10-12/min

Absent

Start

CPR with compression rate of 100/min and ventilation with 8-10 breath/min

with advance airway and 30:2 without advance airway

a. whenever possible defibrillation should precede intubation and IV access (should not delay defibrillation if ventilation is achieved without intubation, like bag & mask)

Assess Rhythm

Shockable (VF, pulseless V tachy)

1. Give one shock (360 J with monophasic & 200 J with biphasic)

Continue CPR for 2 min (or 5 cycles of 30:2 if airway is still not maintained by advance methods)

Check Rythm

Still shockable rhythm

(Asystole)

Non shockable (Asystole, PEA)

Continue CPR

Adrenaline 1 mg IV and repeat every 3-5 minutes (if IV access not possible than use intraoss-eous or endotracheal route) Consider vasopressin 40U after 1st or 2nd dose of adrenaline Atropine 1 mg, repeat every 3-5 minutes (to a max. of 3 doses) Keep on checking rhythm every 2 min, if non shockable, continue same treatment and if becomes shockable treat like shockable.

2. • Continue CPR ~* • Give adrenaline (1:10,000) 1 mg IV, endotracheal or Intraosseous

Give shock (again 360 J with monophasic & 200 J with biphasic)

Continue CPR for 2 min.

Check rhythm

Still shockable

Recovered

Non shockable

3.
 - Continue CPR
 - Repeat adrenaline 1 mg or vasopressin 40 U (as an alternative to adrenaline)

1 Shock (360 J - Monophasic, 200 J - Biphasic)

Continue CPR for 2 minutes

- Continue CPR
- Give amiodarone / lignocaine / magnesium sulphate

Shock (with 360 J - monophasic with 200 J biphasic)

- Proceed like non shockable

Recovered Shockable

Go back to stage 2 and continue same protocol

Non shockable

Treat like non shockable and pacing as immediate measures to tide over the crisis.

MANAGEMENT OF ASYSTOLE

Asystole is the terminal event of all arrhythmias.

Management

Intubate immediately and start pulmonary ventilation and cardiac massage

Consider possible causes like hyperkalemia, hypokalemia, hypothermia, hypoxia, acidosis,

I Adrenaline 1 mg IV and repeat every 3 minutes Vasopressin 40 Units after 1s' or 2nd dose of adrenaline Atropine 1 mg IV and repeat every 3 minutes to a total of 0.04 mg/kg

Transcutaneous pacing

No response Consider termination of efforts

PAEDIATRIC CPR (excluding NEWBORN)

Age classification: From CPR point of view.

- Neonate: First 4 weeks after birth.
- Infants: 4 weeks to 1 year.
- Child: 1-14 years (previously children were considered 1 -8 years) for medical staff but for lay rescuers children still mean 1-8 years.
- Adults: > 14 years for medical staff. > 8 years for lay rescuers.

Differences from Adults

- Ratio of compression to breathing without advance airway: 30:2 for one rescuer and 15:2 for 2 rescuers. (While for adults it is 30:2 irrespective of rescuers).
- With Advance airway: 100 compressions per minute (even for infants which was earlier 120/min) with 8-10 breaths/min. and unsynchronized e. no pause for ventilation (similar to adults) [contrary to previous 5:1 ratio and synchronized].
- If pulses are palpable then give breath at a rate of 12-20/min (Adults 10-12/min).
- Pulse check:
 - Infants: Brachial/Femoral.
 - Children: Carotid.
 - Adult: Carotid/Femoral.
- In emergency cuffed tubes can be used in paediatric age group if inflation pressure is less than 20 cmH₂O.
- If heart rate is <60/min with signs of poor perfusion (pallor/cyanosis) start compressions.
- Cardiac arrest in children is not usually because of ventricular fibrillation and cause is usually asphyxial (respiratory) so the difference from adults in protocol of basic life support is that after assessing responsiveness immediately start CPR and give five cycle of 30:2 and then call for Emergency medical system (EMS) while in adults (see algorithm for BLS) first step after assessing responsiveness is activation of EMS and then start CPR, further protocol for BLS remains same.
- Advance life support protocol for paediatric patient remains same with some differences like:
 1. Energy selection for shock (manual) is 2 J/kg for first shock and 4 J/kg subsequently. If AED is used it automatically select energy and do attenuation accordingly. Paediatric AED are only recommended for children < 8yrs (or <25 kg). Children > 8 years (> 25 kg) should be defibrillated with adult defibrillators.
 2. Another important difference from adults in management of asystole is that atropine and vasopressin are not recommended for paediatric age group.
 3. Endotracheal concentration of adrenaline for adults is 1:10,000 but for paediatric 1 : 1000.

NEWBORN CPR

The detailed discussion of newborn CPR is beyond the scope of this book but some of the notable differences are:

1. Rate of ventilation (breathing) is 40-60 breaths/ min (if only ventilation is given).
2. The primary measure for successful ventilation is increase in heart rate.
3. Indication of chest compression is HR (Heart rate) < 60 /min. inspite of adequate ventilation with 100% oxygen for 30 seconds.
4. 2 thumbs with encircled chest is preferred method for compression over only 2 thumb technique.
5. Compression ventilation ratio is 3:1 (90 compression with 30 breaths) and synchronized.
6. Reassessment to be made every 30 sec. and continue compression till HR > 60 /min.
7. Adrenaline indicated if HR < 60 /min.
8. Stop resuscitation if no signs of life after 10 minutes.

Lay Rescuers

1. Pulse check not expected from them therefore they can start compression even if not able to palpate pulse of an unresponsive victim.
2. Paediatric means 1-8 years (for health care professionals pead. means 1-14 years).

CPR IN PREGNANCY

1. External cardiac massage should be combined with lateral tilt.
2. Sodium bicarbonate administration is advocated early.
3. Early insertion of endotracheal tube (as pregnant patients are very vulnerable for aspiration) therefore during mask ventilation continously apply cricoid pressure till patient is intubated.
4. More prone for hypoxia so use high FIO₂.
5. Chest compression should be performed harder.
6. Emergency LSCS (cesarean section) should be considered only if:
 - Fetus > 25 week.
 - Uterine decompression is mandatory for patients life (fetus can be saved only if LSCS is performed within 5 min. of arrest).

OPEN CHEST MASSAGE

Indications are:

1. Cardiac tamponade.
2. Penetrating blunt trauma.
3. Air embolism.
4. Arrest during intrathoracic procedures.
5. Chest deformities.

COMPLICATIONS OF CPR

1. Rib fracture.
2. Pneumothorax.
3. Pneumopericardium.
4. Pneumomediastinum.
5. Injury to diaphragm.
6. Gastric injury.
7. Lung injury.
8. Injury to major vessels particularly by fractured rib.
9. Injury to abdominal organs: Liver, spleen and stomach.

OUTCOME OF CPR

Depends on the:

1. Cause.
2. The time of initiation of CPR and
3. Duration for which CPR is performed.

Survival is better, if basic life support (BLS) is

initiated within 4 minutes of arrest and ALS within 8 minutes.

Fortunately most common cause of cardiac arrest is ventricular fibrillation which if detected in time can have 50 to 60% success rate. Average survival rate of in hospital arrest is 8 to 21 %.

Survival is better if arrest time (onset of arrest to start of CPR) is less than 6 minutes and CPR time (time required for successful CPR) is less than 30 minutes.

NEWER TECHNIQUES OF CPR

1. Simultaneous abdominal compression: Limits caudal movement of diaphragm and limits dissipation of intrathoracic pressure.
2. Vest CPR: Increasing intrathoracic pressure by physically inflating the bladder around chest.
3. Simultaneous ventilation with every compression.
4. Cardiopulmonary bypass.
5. Cough CPR (applicable to conscious patient having VF): If a patient cough during VF, increase in intrathoracic pressure can maintain cerebral (perfusion (e. consciousness) for 90 sec. So ask him to cough every 1-3 sec. till he/she is defibrillated.
6. High frequency (120/min) CPR: Not better than standard CPR.

INITIAL APPROACH

The initial approach to airway management is simultaneous assessment and management of the adequacy of airway patency (the A of ABCs) and oxygenation and ventilation (the B of ABCs).

1. The patient's color and respiratory rate must be assessed; marked hypoventilation with or without cyanosis may be an indication for immediate intubation.
2. The airway should be opened with head tilt–chin lift maneuver (jaw thrust should be used if C-spine injury is suspected). If needed, the patient should be bagged with the bag-valve-mask device, including an O₂ reservoir. For a good seal, the proper size mask should be ensured. This technique may require an oral or nasal airway or two rescuers to both seal the mask (two hands) and bag the patient.
3. The patient should be placed on a cardiac monitor, pulse oximetry, and possibly capnography (end-tidal CO₂), while the remaining vitals, pulse, and blood pressure (temperature is important but can be delayed to assure the ABCs) can be collected.
4. The need for invasive airway management techniques must be determined as described later. It is essential to not wait for arterial blood gas analyses (ABG) if the initial assessment declares the need for invasive airway management. If the patient does not require immediate airway or ventilation control, he or she should be administered oxygen by face mask, as necessary, to assure an O₂ saturation of 95%. Laboratory studies should be collected as needed. Do not remove a patient from oxygen to draw an ABG unless deemed safe from the initial assessment.

OROTRACHEAL INTUBATION

The most reliable means to ensure a patent airway, prevent aspiration, and provide oxygenation and ventilation is endotracheal (ET) intubation. Many conscious patients require intubation (see the section, “Rapid Sequence Induction” later). Selection of the blade should be considered in advance, if possible. The curved blade rests in the vallecula above the epiglottis and indirectly lifts it off the larynx because of traction on the frenulum. The straight blade is used to lift the epiglottis directly. The curved blade does a better job of clearing the tongue

from view and may be less traumatic and reflex-stimulating. The straight blade is mechanically easier to insert in many patients.

Emergency Department Care and Disposition

1. Adequate ventilation must be ensured while the equipment is prepared. The patient should be preoxygenated, with or without a bag-valve-mask device, depending on the clinical need. Vital signs must be monitored and pulse oximetry used throughout the procedure.

2. The blade type and size (usually #3 or #4 curved blade, or #2 or #3 straight blade) should be selected; the blade light should be tested. The tube size (usually 7.5 to 8.0 in women, 8.0 to 8.5 in men) must be selected and the balloon cuff tested. The end of the tube should be lubricated with lidocaine jelly or similar lubricant. The use of a flexible stylet should be considered; the distal end should be bent upward if the patient's anatomy requires it. The tube and tonsillar tip suction should be placed within easy reach. If there is an assistant, he or she should be asked to pass the items when needed.

3. The patient should be positioned with the head extended and neck flexed, possibly with a rolled towel under the occiput. If C-spine injury is suspected, the head or neck should not be moved. Rapid sequence induction with in-line traction, nasotracheal intubation, or cricothyrotomy should be considered.

4. The blade should be inserted on the right and slowly advanced in search of the epiglottis. The patient should be suctioned as necessary. If the curved blade is used, the tip should be slid into the vallecula and lifted (indirectly lifting the epiglottis); if a straight blade is used, the epiglottis should be lifted directly in the direction the handle points, that is, 90° to the blade. It is important to not rock back on the teeth.

5. Once the vocal cords are visualized, it is important to not lose sight of them. The assistant should be asked to place the tube in the physician's hand. Pass the tube between the cords, avoiding force. The stylet should be removed, the balloon cuff inflated. Ventilate the patient with a bag-valve device and check for bilateral breath sounds. Placement should be confirmed with an end-tidal CO₂ detector (not reliable if the patient is in cardiac arrest) or capnography. Tube length should be checked; the usual distance (marked on the tube) from the corner of the

mouth to 2 cm above the carina is 23 cm in men and 21 cm in woman.

6.The tube should be taped in place and a bite block inserted. Correct intubation and tube placement can be verified with a portable chest x-ray.

7.If unsuccessful, reoxygenation should be performed with bag-valve-mask device. The technique can be changed by possibly using a smaller tube, different blade type or size, or repositioning the patient and reattempting intubation.

Short-term complications from orotracheal intubation (trauma to surrounding structures) are unusual, as long as correct position is confirmed. Failure to confirm position immediately can result in hypoxia and neurologic injury. Endobronchial intubation is usually on the right side and is corrected by withdrawing the tube 2 cm and listening for equal breath sounds.

NASOTRACHEAL INTUBATION

Nasotracheal intubation is indicated in situations where laryngoscopy is difficult, neuromuscular blockade is hazardous, or crico-thyrotomy unnecessary. Severely dyspneic, awake patients with congestive heart failure, chronic obstructive pulmonary disease, or asthma often cannot remain supine for other airway maneuvers but do tolerate nasotracheal intubation in the sitting position. Relative contraindications for this technique include complex nasal and massive midface fractures and bleeding disorders.

Emergency Department Care and Disposition

1.Both nares should be sprayed with a topical vasoconstrictor and anesthetic. Between 4 to 10% cocaine solution is an appropriate single agent, but may cause unwanted systemic cardiovascular effects. Topical neosynephrine is an effective vasoconstrictor, and tetracaine is a safe effective topical anesthetic.

2.The tube size must be chosen, usually between 7.0 to 7.5 in women and 7.5 to 8.0 in men. The balloon cuff of the tube should be checked for leaks. The tube should be lubricated with lidocaine jelly or similar lubricant.

3. The largest nares should be used or the right side if the nares are equal. Some operators recommend dilating the nares with a lubricated nasal airway. The patient may be sitting up or supine.

4. An assistant can immobilize the patient's neck. The physician should stand to the patient's side, with one hand on the tube and with the thumb and index finger of the other hand straddling the larynx. The tube should be advanced slowly, with steady gentle pressure. The tube should be twisted to help move past obstructions in the nose and nasopharynx. The tube should be advanced until maximal airflow is heard through the tube; this means the larynx is now close by.

5. The physician should listen carefully to the rhythm of inspiration and expiration. The tube should then be gently but swiftly advanced during the beginning of inspiration. Entrance into the larynx may initiate a cough, and most expired air should exit the tube even though the cuff is uninflated. If the tube is foggy the cuff should be inflated.

6. If intubation is unsuccessful, the physician should carefully look for a bulge lateral to the larynx (usually the tip of the tube is in the pyriform fossa on the same side as the nares used). If found, the tube must be retracted until maximal breath sounds are heard and then intubation should be reattempted by manually displacing the larynx toward the bulge. If no bulge is seen, it is possible that the tube has gone posteriorly into the esophagus. In this case, the tube should be withdrawn until maximal breath sounds are heard. Intubation should again be reattempted after the patient's head is extended and a Sellick's maneuver performed. Another option is to use a directional control tip (Endotrol) or fiberoptic laryngoscope. The head should not be moved if C-spine injury is suspected.

Complications other than local bleeding are rare. Occasionally, marked bleeding will prompt the need for orotracheal intubation or cricothyrotomy.

CRICOTHYROTOMY

Indications for immediate cricothyrotomy include severe, ongoing tracheobronchial hemorrhage, massive midface trauma, and inability to control the airway with the usual less-invasive maneuvers. Cricothyrotomy is relatively contraindicated in patients with acute

laryngeal disease due to trauma or infection or recent prolonged intubation and should not be used in children below the age of 12.

Emergency Department Care and Disposition

1. Sterile technique should be used. The cricothyroid membrane should be palpated with digital stabilization of the larynx (see With a #11 scalpel, a vertical 3 to 4 cm incision should be started at the superior border of the thyroid cartilage and incised caudally toward the suprasternal notch.

2. The membrane should be repalpated and a horizontal stab should be made through its inferior aspect. The blade should be kept temporarily in place.

3. The larynx should be stabilized by inserting the tracheal hook into the cricothyroid space and retracting upon the inferior edge of the thyroid cartilage (an assistant should hold after the hook is placed). Leaving the blade tip in the space, a slightly open hemostat should be inserted straddling the blade and spread open horizontally.

4. The scalpel should be removed and a dilator inserted (LaBorde or Trousseau). The tracheal hook can then be removed.

5. A Shiley tracheostomy tube should be introduced (or the largest tube that will fit). Alternatively, a small cuffed endotracheal tube may be used (#6 or the largest tube that will fit). The balloon should be inflated and the tube secured in place.

6. The physician should check for bilateral breath sounds. Make sure subcutaneous air is not introduced. Placement can be checked with an end-tidal CO₂ detector and chest x-ray.

RAPID SEQUENCE INDUCTION

Complex airway emergencies in select nonfasted patients may require rapid sequence induction. This technique couples sedation to induce unconsciousness (induction) with muscular paralysis. Intubation follows laryngoscopy while maintaining cricoid pressure to prevent aspiration. The principle contraindication is any condition preventing mask ventilation

or intubation.

- 1.The cardiac monitor, oximetry, and capnography should be set up, if available. Equipment should be checked.
- 2.The patient should be preoxygenated with 100% oxygen.
- 3.Lidocaine (1.5 mg/kg intravenously) should be considered in a head trauma patient to prevent increased intracranial pressure. Atropine (0.4 mg/kg intravenously) should be considered to prevent reflex bradycardia, but is not essential.
- 4.Medication for sedation or analgesia should be considered, if such agents are not being used for induction.
- 5.A defasciculating dose of a nondepolarizing agent (i.e., vecuronium at 0.02 mg/kg) is used if succinylcholine is given for paralysis.
- 6.The patient should be induced with thiopental (3 to 5 mg/kg), methohexital (1 to 2 mg/kg), or midazolam (0.1 mg/kg with 5 mg maximum dose). Barbiturates should not be used in a patient with hypotension or reactive airway disease (caution in head injury). Benzodiazepines may be inadequate for induction, however, midazolam is an excellent amnestic agent. Etomidate, 0.3 mg/kg, is an excellent alternative in a hypotensive patient. Ketamine, 1 to 2 mg/kg, should be considered for the induction of a patient who has active bronchospasm for its bronchodilator properties.
- 7.In a patient needing analgesia in addition to sedation, opiates should be considered for induction. These agents are reversible with naloxone. Fentanyl, 2 to 10 µg/kg, is commonly used.
- 8.Cricoid pressure should be applied before paralysis and maintained until intubation is accomplished.
- 9.Succinylcholine (1.0 mg/kg) is chosen for paralysis in many cases because of its rapid onset and short duration of action; it should not be used in a patient with preexisting paralysis or > 2

h after severe burns, as hyperkalemia may occur. A nondepolarizing agent such as vecuronium (0.2 mg/kg) may be chosen for a patient with increased intracranial pressure, one in status asthmaticus, or at operator discretion.

10. The trachea should be intubated and cricoid pressure released.

11. The physician should be prepared to bag the patient if intubation proves unsuccessful. Invasive airway techniques should be considered as indicated.

Alternative drugs for rapid sequence induction are listed in Chap. 15 of Emergency Medicine, A Comprehensive Study Guide, 5th ed. Airway management alternatives to the methods described earlier include retrograde tracheal intubation, translaryngeal ventilation, digital intubation, transillumination, fiberoptic assistance, and formal tracheostomy. Translaryngeal ventilation may be used to temporarily provide ventilation until a more definitive procedure is possible. When oral intubation is indicated but has been unsuccessful, and the patient can be temporarily ventilated with a bag-valve-mask unit, the following assist methods are warranted. Retrograde tracheal intubation, digital intubation, transillumination, or fiberoptic assistance may be helpful. Formal tracheostomy is reserved for those experienced in the technique when less-invasive or more-rapid methods (cricothyrotomy) are unsuccessful.

2 Dysrhythmia Management

Emergency Medicine Companion Handbook

If sustained junctional escape rhythms are producing symptoms, the underlying cause should be treated. Atropine can be used to accelerate temporarily the sinus node discharge rate and enhance AV nodal conduction.

VENTRICULAR DYSRHYTHMIAS

Aberrant Versus Ventricular Tachydysrhythmias

Differentiation between ectopic beats of ventricular origin and those of supraventricular origin that are conducted aberrantly, can be difficult, especially in sustained tachycardias with wide

QRS complexes (WCT). In general, the majority of patients with WCT have ventricular tachycardia, which should be approached as ventricular tachycardia, until proved otherwise. Several guidelines follow:

1. A preceding ectopic P wave is good evidence favoring aberrancy, although coincidental atrial and ventricular ectopic beats or retrograde conduction can occur. During a sustained run of tachycardia, AV dissociation favors a ventricular origin of the dysrhythmia.
2. Postectopic pause: A fully compensatory pause is more likely after a ventricular beat, but exceptions occur.
3. Fusion beats are good evidence for ventricular origin but, again, exceptions occur.
4. A varying bundle branch block pattern suggests aberrancy.
5. Coupling intervals are usually constant with ventricular ectopic beats, unless parasystole is present. Varying coupling intervals suggest aberrancy.
6. Response to carotid sinus massage or other vagal maneuvers will slow conduction through the AV node and may abolish reentrant SVT and slow the ventricular response in other supraventricular tachydysrhythmias. These maneuvers have essentially no effect on ventricular dysrhythmias.
7. A QRS duration of longer than 0.14 s is usually found in ventricular ectopy or tachycardia.
8. Historical criteria also have been found to be useful: a patient over 35 years old or a history of MI, CHF, or coronary artery bypass graft strongly suggest ventricular tachycardia in patients with WCT.

Emergency Department Care and Disposition

1. As with ventricular tachycardia lidocaine 1 to 1.5 mg/kg intravenously should be started and may be repeated up to 3 mg/kg.

2. Adenosine 6 mg intravenously may be tried prior to procainamide (see ventricular tachycardia management later for administration guidelines).

Premature Ventricular Contractions

Clinical Features

Premature ventricular contractions (PVCs) are due to impulses originating from single or multiple areas in the ventricles. The ECG characteristics of PVCs are (a) a premature and wide QRS complex; (b) no preceding P wave; (c) the ST-segment and T wave of the PVC are directed opposite the major QRS deflection; (d) most PVCs do not affect the sinus node, so there is usually a fully compensatory postectopic pause, or the PVC may be interpolated between 2 sinus beats; (e) many PVCs have a fixed coupling interval (within 0.04 s) from the preceding sinus beat; and (f) many PVCs are conducted into the atria, producing a retrograde P wave.

PVCs are common, occur in most patients with ischemic heart disease, and are universally found in patients with acute MI. Other common causes of PVCs include digoxin toxicity, CHF, hypokalemia, alkalosis, hypoxia, and sympathomimetic drugs.

Emergency Department Care and Disposition

1. Most acute patients with PVCs will respond to intravenous lidocaine (1 mg/kg intravenously), although some patients may require procainamide. Although single studies have suggested benefit, pooled data and meta-analysis find no reduction in mortality from either suppressive or prophylactic treatment of PVCs.

Accelerated Idioventricular Rhythm

Clinical Features

The ECG characteristics of accelerated idioventricular rhythm (AIVR) are (a) wide and regular QRS complexes; (b) rate between 40 and 100, often close to the preceding sinus rate; (c) most runs of short duration (3 to 30 beats); and (d) an AIVR often beginning with a fusion beat.

This condition is found most commonly with an acute MI.

Emergency Department Care and Disposition

1. Treatment is not necessary. On occasion, AIVR may be the only functioning pacemaker, and suppression with lidocaine can lead to cardiac asystole.

Ventricular Tachycardia

Clinical Features

Ventricular tachycardia is the occurrence of 3 or more beats from a ventricular ectopic pacemaker at a rate greater than 100. The ECG characteristics of ventricular tachycardia are (a) wide QRS complexes; (b) rate greater than 100 (most commonly 150 to 200); (c) usually regular rhythm, although there may be some beat-to-beat variation; and (d) usually constant QRS axis

Ventricular tachycardia is rare in patients without underlying heart disease. The most common causes of ventricular tachycardia are ischemic heart disease and acute MI. Ventricular tachycardia cannot be differentiated from SVT with aberrancy on the basis of clinical symptoms, blood pressure, or heart rate. Patients who are unstable should be cardioverted, which is effective for both dysrhythmias. In general, it is best to treat all wide complex tachycardias as ventricular tachycardia with lidocaine or procainamide. Adenosine appears to cause little harm in patients with ventricular tachycardia and has potential merit for the treatment of wide QRS complex tachycardias.

Emergency Department Care and Disposition

1. Unstable patients, or those in cardiac arrest, should be treated with synchronized cardioversion. Ventricular tachycardia can be converted with energies as low as 1 J, and over 90 percent can be converted with less than 10 J. ACLS guidelines recommend that pulseless ventricular tachycardia be defibrillated (unsynchronized cardioversion) with 200 J. Another alternative for unstable patients is intravenous amiodarone. (See treatment recommendations

under ventricular fibrillation.)

2. Clinically stable patients should be treated with intravenous antidysrhythmics.

a. Lidocaine 75 mg (1.0 to 1.5 mg/kg) intravenously over 60 to 90 s can be administered, followed by a constant infusion at 1 to 4 mg/min (10 to 40 $\mu\text{g}/\text{kg}/\text{min}$). A repeat bolus dose of 50 mg lidocaine may be required during the first 20 min to avoid a subtherapeutic dip in serum level due to the early distribution phase.

b. Procainamide can be administered intravenously at less than 30 mg/min until the dysrhythmia converts, the total dose reaches 15 to 17 mg/kg in normals (12 mg/kg in patients with CHF), or early signs of toxicity develop, with hypotension or QRS prolongation. The loading dose should be followed by a maintenance infusion of 2.8 mg/kg/h in normal subjects.

c. Bretylium 500 mg (5 to 10 mg/kg) intravenously over 10 min can be administered, followed by a constant infusion at 1 to 2 mg/min.

Torsades de Pointes

Atypical ventricular tachycardia (torsade de pointes, or twisting of the points) is where the QRS axis swings from a positive to negative direction in a single lead (Fig. 2-14).

FIG. 2-14 Two examples of short runs of atypical ventricular tachycardia showing sinusoidal variation in amplitude and direction of the QRS complexes: “Le torsade de pointes” (twisting of the points). Note that the top example is initiated by a late-occurring PVC (lead II).

Drugs that further prolong repolarization—quinidine, disopyramide, procainamide, phenothiazines, tricyclic antidepressants—exacerbate this dysrhythmia.

1. Reports have revealed that magnesium sulfate, 1 to 2 g intravenously over 60 to 90 s followed by an infusion of 1 to 2 g/h, is effective in abolishing torsades de pointes.

2. To date, treatment for torsades de pointes consisted of accelerating the heart rate (thereby shortening ventricular repolarization) with isoproterenol (2 to 8 $\mu\text{g}/\text{min}$), while making

arrangements for a ventricular pacemaker to overdrive the heart at rates of 90 to 120. Temporary pacing is the most effective and safest method to treat torsades de pointes and prevent its recurrence.

Ventricular Fibrillation

Clinical Features

Ventricular fibrillation is the totally disorganized depolarization and contraction of small areas of ventricular myocardium—there is no effective ventricular pumping activity. The ECG of ventricular fibrillation shows a fine-to-coarse zigzag pattern without discernible P waves or QRS complexes (Fig. 2-15). Ventricular fibrillation is never accompanied by a pulse or a measurable blood pressure.

Ventricular fibrillation is most commonly seen in patients with severe ischemic heart disease, with or without an acute MI. Primary ventricular fibrillation occurs suddenly, without preceding hemodynamic deterioration, whereas secondary ventricular fibrillation occurs after a prolonged period of left ventricular failure or circulatory shock.

Emergency Department Care and Disposition

1. Current ACLS guidelines recommend immediate electrical defibrillation with 200 J. If ventricular fibrillation persists, defibrillation should be repeated immediately, with 200 to 300 J at the second attempt, increased to 360 J at the third attempt.
2. If the initial 3 attempts at defibrillation are unsuccessful, cardiopulmonary resuscitation and intubation should be initiated.
3. Epinephrine in standard dose should be administered, 1 mg intravenously. If this is not successful, high-dose epinephrine may be subsequently given, 0.1 mg/kg, and repeated every 3 to 5 min.
4. Defibrillation should be attempted after each drug administration, at 360 J, unless lower energy levels have been previously successful.

5. Successive antidysrhythmics should then be administered with defibrillation attempted after each drug. The recommended sequence is lidocaine, 1.5 mg/kg, bretylium, 5 mg/kg, then possibly magnesium, 2 g intravenously, and procainamide (see preceding dosing guidelines).

6. Amiodarone, 150 mg over 10 min followed by 1 mg/min for 6 h, may become a preferred treatment for

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Shock Overview

The word shock is used differently by the medical community and the general public. The connotation by the public is an intense emotional reaction to a stressful situation or bad news. The medical definition of shock is much different.

Medically, shock is defined as a condition where the tissues in the body don't receive enough oxygen and nutrients to allow the cells to function. This ultimately leads to cellular death, progressing to organ failure, and finally, if untreated, whole body failure and death.

How the body works

Cells need two things to function: oxygen and glucose. This allows the cells to generate energy and do their specific jobs.

Oxygen in the air enters the body through the lungs; where oxygen molecules cross into the smallest blood vessels, the capillaries, and are picked up by red blood cells and attached to [hemoglobin](#) molecules. The red blood cells are pushed through the body by the actions of the pumping heart and deliver the oxygen to cells in all the tissues of the body. The hemoglobin then picks up carbon dioxide, the waste product of metabolism, where it is then taken back to the lungs and breathed out into the air, whereby the whole cycle begins again.

Glucose is generated in the body from the foods we eat. Glucose travels in the blood stream and uses an insulin molecule to "open the door," where it then enters the cell to provide energy for cellular metabolism.

Shock Causes

When things go wrong

If cells are deprived of oxygen, instead of using aerobic (with oxygen) metabolism to function, the cells use the anaerobic (without oxygen) pathway to produce energy. Unfortunately, lactic acid is formed as a by product of anaerobic metabolism. This acid changes the acid-base balance in the blood, making it more acidic, and this leads to situation in which cells begin to leak toxic chemicals into the bloodstream, causing blood vessel walls to become damaged. The anaerobic process ultimately leads to the death of the cell. If enough cells die, organs start to fail, and the body starts to fail and death occurs.

Think of the cardiovascular system of the body as similar to the oil pump in your car. For efficient functioning, the electrical pump needs to work to pump the oil, there needs to be enough oil, and the oil lines need to be intact. If any of these components fail, oil pressure falls and the engine may be damaged. In the body, if the heart, blood vessels, or bloodstream (circulation) fail, then the body fails.

Where things go wrong

The **oxygen delivery system** to the body's cells can fail in a variety of ways.

- The amount of oxygen in the air that is inhaled can be decreased.
- Examples include [breathing at high altitude](#) or [carbon monoxide poisoning](#).

The **lung** may be injured and not be able to transfer oxygen to the blood stream. Examples of causes include:

- pneumonia (an infection of the lung),
- [congestive heart failure](#) (the lung fills with fluid or [pulmonary edema](#)), or
- trauma with [collapse or bruising of the lung](#), or
- [pulmonary embolism](#).

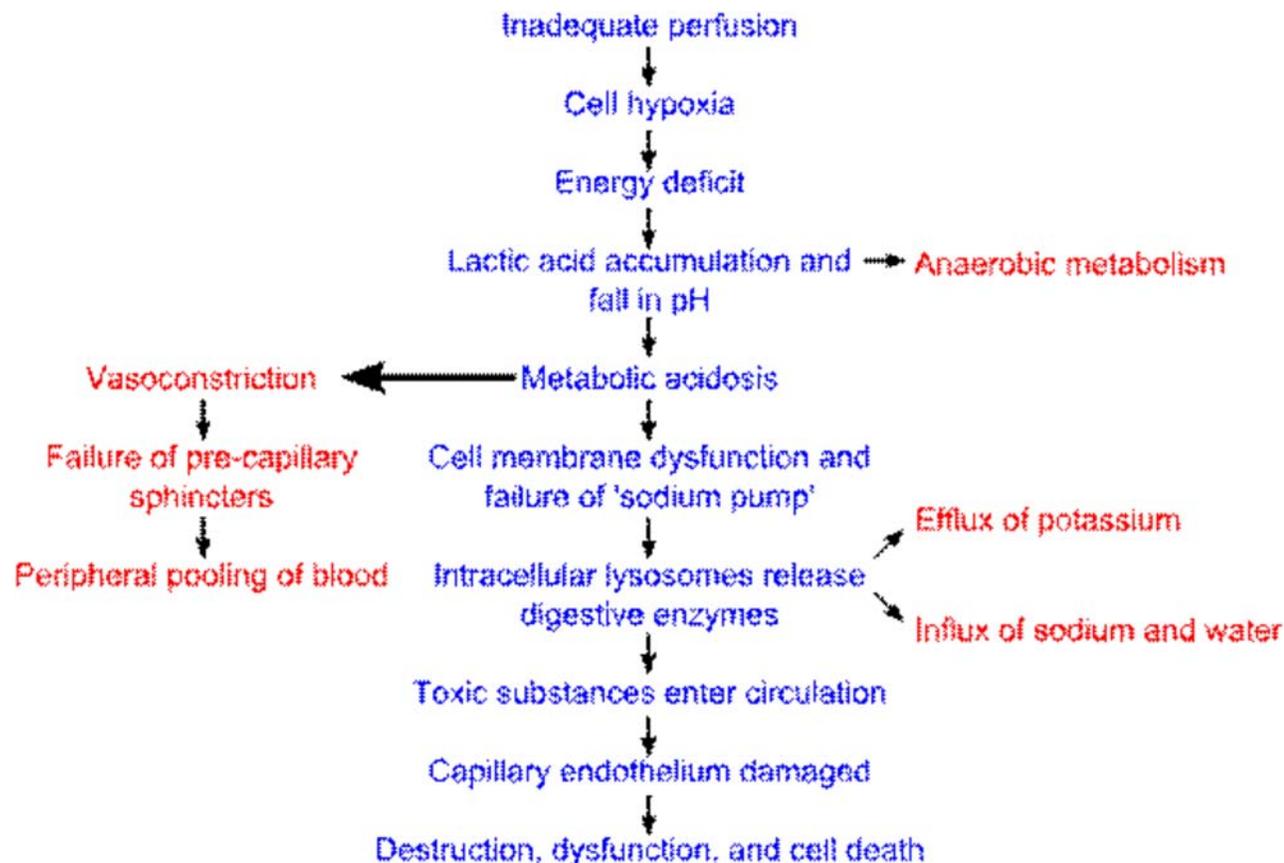


Fig. Effects of inadequate perfusion on cell function.

The **heart** may not be able to adequately pump the blood to the tissues of the body. Examples of causes examples include:

- [Heart attack](#) in which muscle tissue is lost and the heart cannot beat as strong and pump blood throughout the body.
- A [rhythm disturbance of the heart](#) occurs when the heart can't beat in a coordinated way.
- Inflammation of the sac around the heart ([pericarditis](#)) or inflammation of the heart muscle due to infections or other causes, in which the effective beating capabilities of the heart are lost.

There may **not be enough red blood cells** in the blood. If there aren't enough red blood cells ([anemia](#)), then not enough oxygen can be delivered to the tissues with each heart beat. Examples of causes may include:

- acute or chronic [bleeding](#),
- inability of the bone marrow to make red blood cells, or
- the increased destruction of red blood cells by the body (an example, [sickle cell disease](#)).

There may **not be enough other fluids** in the blood vessels. The blood stream contains the blood cells (red, white, and platelets), plasma (which is more than 90% water), and many important proteins and chemicals. Loss of body water or [dehydration](#) can cause shock.

The **blood vessels** may not be able to maintain enough pressure within their walls to allow blood to be pumped to the rest of the body. Normally, blood vessel walls have tension on them to allow blood to be pumped against gravity to areas above the level of the heart. This tension is under the control of the unconscious central nervous system, balanced between the action of two chemicals, adrenaline (epinephrine) and acetylcholine. If the adrenaline system fails, then the blood vessel walls dilate and blood pools in the parts of the body closest to the ground (lower extremities), and may have a difficult time returning to heart to be pumped around the body.

Since one of the steps in the cascade of events causing shock is damage to blood vessel walls, this loss of integrity can cause blood vessels to leak fluid, leading to dehydration which initiates a vicious circle of worsening shock.

Stages of shock

There are four stages of shock. As it is a complex and continuous condition there is no sudden transition from one stage to the next.

Initial

During this stage, the hypoperfusal state causes hypoxia, leading to the mitochondria being unable to produce adenosine triphosphate (ATP). Due to this lack of oxygen, the cell membranes become damaged, they become leaky to extra-cellular fluid, and the cells perform anaerobic respiration. This causes a build-up of lactic and pyruvic acid which results in systemic metabolic acidosis. The process of removing these compounds from the cells by the liver requires oxygen, which is absent.

Compensatory (Compensating)

This stage is characterised by the body employing physiological mechanisms, including neural, hormonal and bio-chemical mechanisms in an attempt to reverse the condition. As a result of the acidosis, the person will begin to hyperventilate in order to rid the body of carbon dioxide (CO₂). CO₂ indirectly acts to acidify the blood and by removing it the body is attempting to raise the pH of the blood. The baroreceptors in the arteries detect the resulting hypotension, and cause the release of adrenaline and noradrenaline.

- Baroreceptor reflexes
- Circulating vasoconstrictors
- Chemoreceptor reflexes
- Reabsorption of tissue fluids
- Renal reabsorption of sodium and water
- Activation of thirst mechanisms
- Cerebral ischemia
- Hemapoiesis

Fig. Compensatory mechanisms

Noradrenaline causes predominately vasoconstriction with a mild increase in heart rate, whereas adrenaline predominately causes an increase in heart rate with a small effect on the

vascular tone; the combined effect results in an increase in blood pressure. Renin-angiotensin axis is activated and arginine vasopressin is released to conserve fluid via the kidneys.

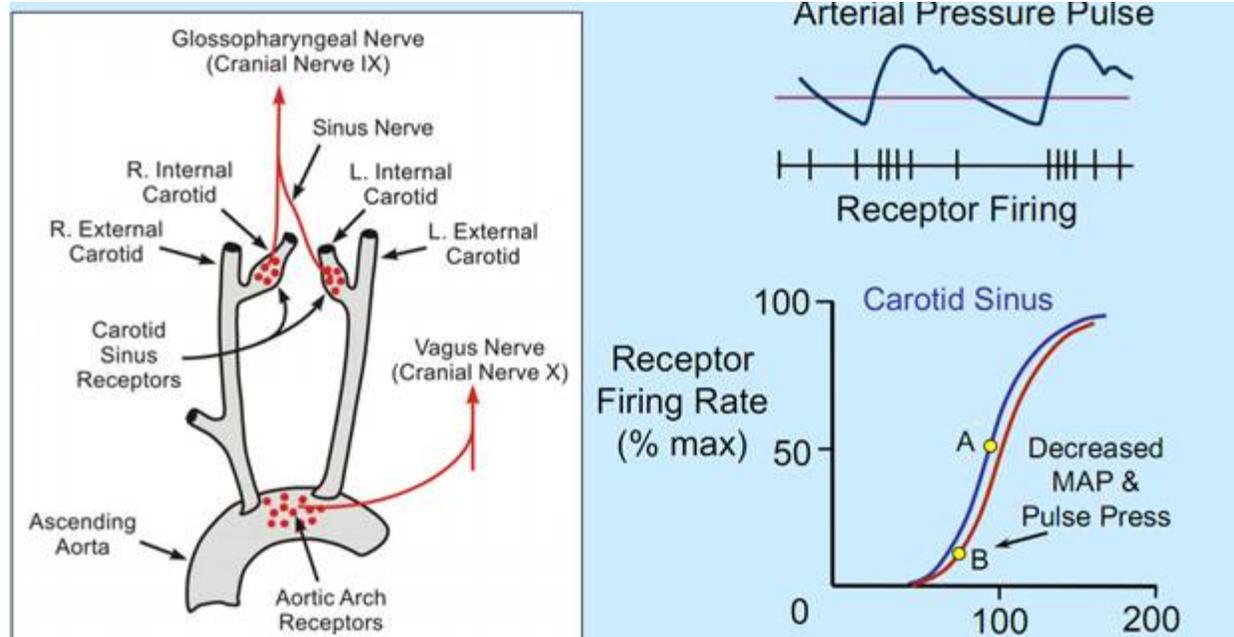


Fig. Compensatory mechanisms

Also, these hormones cause the vasoconstriction of the kidneys, gastrointestinal tract, and other organs to divert blood to the heart, lungs and brain. The lack of blood to the renal system causes the characteristic low urine production. However the effects of the Renin-angiotensin axis take time and are of little importance to the immediate homeostatic mediation of shock .

Progressive (Decompensating)

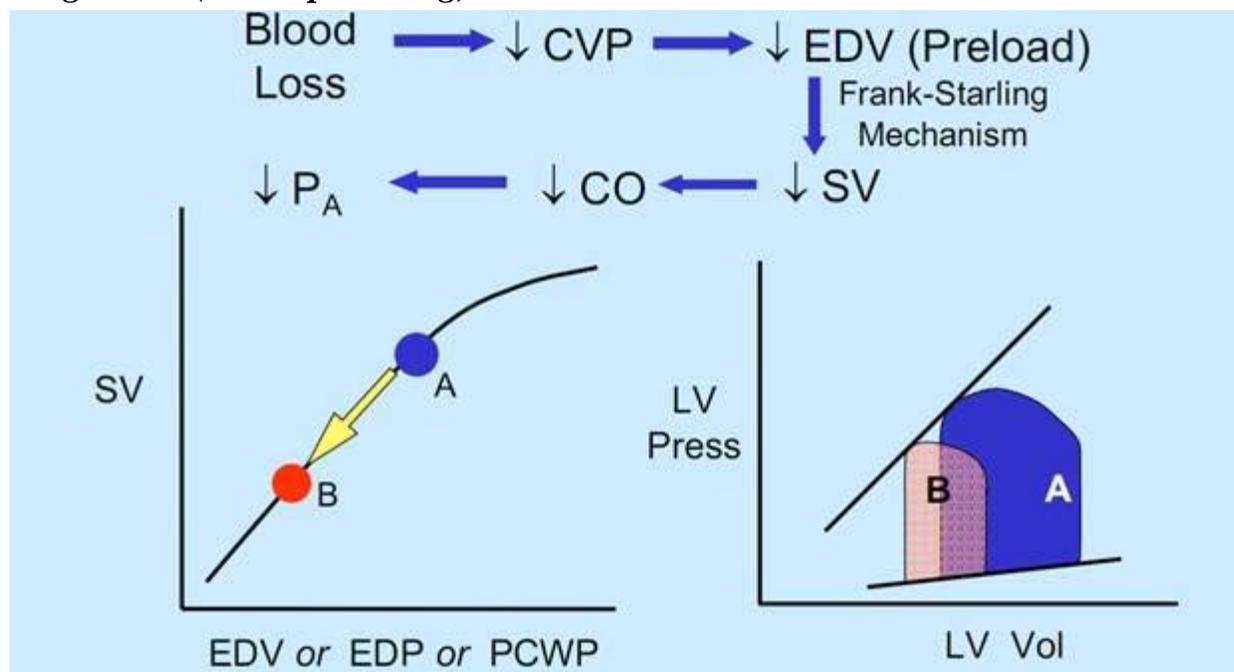


Fig. Compensatory mechanisms

Should the cause of the crisis not be successfully treated, the shock will proceed to the progressive stage and the compensatory mechanisms begin to fail. Due to the decreased perfusion of the cells, sodium ions build up within while potassium ions leak out. As anaerobic metabolism continues, increasing the body's metabolic acidosis, the arteriolar smooth muscle and precapillary sphincters relax such that blood remains in the capillaries. Due to this, the hydrostatic pressure will increase and, combined with histamine release, this will lead to leakage of fluid and protein into the surrounding tissues. As this fluid is lost, the blood concentration and viscosity increase, causing sludging of the micro-circulation. The prolonged vasoconstriction will also cause the vital organs to be compromised due to reduced perfusion. If the bowel becomes sufficiently ischemic, bacteria may enter the blood stream, resulting in the increased complication of endotoxic shock.

Refractory (Irreversible)

At this stage, the vital organs have failed and the shock can no longer be reversed. Brain damage and cell death have occurred. Death will occur imminently.

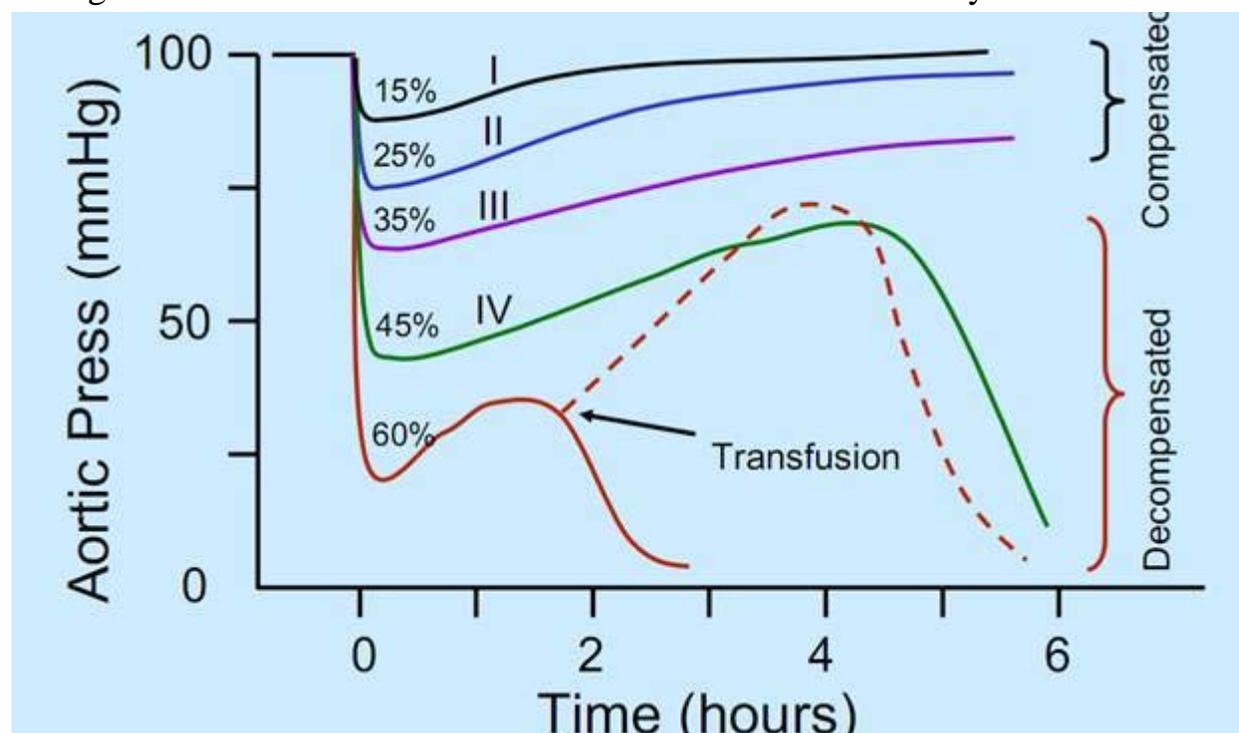


Fig. Effect of blood volume loss on arterial pressure

A medical emergency is an injury or illness that is acute and poses an immediate risk to a person's life or long term health. These emergencies may require assistance from another person, who should ideally be suitably qualified to do so, although some of these emergencies can be dealt with by the victim themselves. Dependent on the severity of the emergency, and the quality of any treatment given, it may require the involvement of multiple levels of care, from a

first aider to an emergency physician through to specialist surgeons.

Classification of shock

Shock is identified in most patients by hypotension and inadequate organ perfusion, which may be caused by either low cardiac output or low systemic vascular resistance. Circulatory shock can be subdivided into 4 distinct classes on the basis of underlying mechanism and characteristic hemodynamics, as follows:

- Hypovolemic shock
- Obstructive shock
- Distributive shock
- Cardiogenic shock

These classes of shock should be considered and systemically differentiated before establishing a definitive diagnosis of septic shock.

Hypovolemic shock results from the loss of blood volume caused by such conditions as gastrointestinal (GI) bleeding, extravasation of plasma, major surgery, trauma, and severe burns. The patient demonstrates tachycardia, cool clammy extremities, hypotension, dry skin and mucus membranes, and poor turgor.

Obstructive shock results from impedance of circulation by an intrinsic or extrinsic obstruction. Pulmonary embolism and pericardial tamponade both result in obstructive shock.

Distributive shock is caused by such conditions as direct arteriovenous shunting and is characterized by decreased resistance or increased venous capacity from the vasomotor dysfunction. These patients have high cardiac output, hypotension, large pulse pressure, a low diastolic pressure, and warm extremities with a good capillary refill. These findings on physical examination strongly suggest a working diagnosis of septic shock.

Cardiogenic shock is characterized by primary myocardial dysfunction, resulting in the inability of the heart to maintain adequate cardiac output. These patients demonstrate clinical signs of low cardiac output, while evidence exists of adequate intravascular volume. The patients have cool clammy extremities, poor capillary refill, tachycardia, narrow pulse pressure, and a low urine output.

Shock - Specific Types

Hypovolemic and Hemorrhagic Shock

Hypovolemic Shock

There needs to be enough red blood cells and water in the blood for the heart to push the fluids around within the blood vessels. When the body becomes dehydrated, there may be enough red blood cells, but the total volume of fluid is decreased, and pressure within the system decreases. Cardiac output is the amount of blood that the heart can pump out in one minute. It is calculated as the stroke volume (how much blood each heart beat can push out) multiplied by the heart rate (how fast the heart beats each minute). If there is less blood in the system to be pumped, the heart speeds up to try to keep its output steady.

Water makes up 90% of blood. If the body becomes dehydrated because water is lost or fluid intake is inadequate, the body tries to maintain cardiac output by making the heart beat faster. But as the fluid losses mount, the body's compensation mechanisms fail, and shock may ensue.

[Hypovolemic](#) (hypo=low + volemic=volume) shock due to water loss can be the endpoint of many illnesses, but the common element is the lack of fluid within the body.

[Gastroenteritis](#) can cause significant water loss from [vomiting](#) and [diarrhea](#), and is a common cause of death in third world countries. [Heat exhaustion and heat stroke](#) is caused by excessive water loss through sweating as the body tries to cool itself. Patients with infections can lose significant amounts of water from sweating. People with diabetes who have [diabetic ketoacidosis](#) lose significant water because of because of elevated blood sugar that cause excess water to be excreted in the urine.

Ultimately in hypovolemic shock, the patient cannot replace the amount of fluid that was lost by drinking enough water, and the body is unable to maintain blood pressure and cardiac output. In all shock states, when cells start to malfunction waste products build up, a downward spiral of cell death begins, increased acidosis occurs, and a worsening body environment leads to further cell death - and ultimately organ failure.

Hemorrhagic Shock

A subset of hypovolemic shock occurs when there is significant bleeding that occurs relatively quickly. Trauma is the most common example of bleeding or hemorrhage, but bleeding can

occur from medical conditions such as:

- Bleeding from the gastrointestinal tract is common; examples include [stomach or duodenal ulcers](#), [colon cancers](#) or [diverticulitis](#).
- In women, excessive bleeding can occur from the uterus.
- People with cancers or [leukemia](#) have the potential to bleed spontaneously from a variety of sources if their bone marrow does not make enough clotting factors.
- Patients who are taking blood thinners (anticoagulant medications) can bleed excessively as well.

Hemorrhage classes

Class	Blood loss	Response	Treatment
I	<15 % (0.75 l)	min. fast heart rate, normal blood pressure	minimal
II	15-30 % (0.75-1.5 l)	fast heart rate, min. low blood pressure	intravenous fluids
III	30-40 % (1.5-2 l)	very fast heart rate, low blood pressure, confusion	fluids and packed RBCs
IV	>40 % (>2 l)	critical blood pressure and heart rate	aggressive interventions

Blood loss has two effects on the body. First, there is a loss of volume within blood vessels to be pumped (see hypovolemic shock) and second, a reduced oxygen carrying capacity occurs because of the loss of red blood cells. Otherwise healthy people can lose up to 10% of their blood volume (about the amount that a person donates at a blood drive) without becoming symptomatic with weakness, lightheadedness, or shortness of breath.

The treatment of hemorrhagic shock depends on the cause. Finding and controlling the source of bleeding is of paramount importance. Intravenous fluids are used to help with resuscitation to increase the fluid volume within the blood vessel space, but blood transfusion is not always mandatory. If the bleeding is controlled and the patient becomes more stable, the bone marrow may be allowed to replenish the red blood cells that were lost.

If the red blood cell count in the blood decreases gradually over time, either because of bleeding or the inability of the body to make enough new red cells, the body can adjust to the lower levels to maintain adequate cell perfusion, but the individual's [exercise](#) tolerance may decrease. This means that they may do well in normal daily activities but find that routine exercise or household activities bring on weakness or shortness of breath. The treatment depends on the underlying diagnosis, since it isn't a total fluid problem as in hypovolemic shock.

Cardiogenic, Neurogenic, and Hypoglycemic Shock

Cardiogenic Shock

When the heart loses its ability to pump blood to the rest of the body, blood pressure decreases. Although there may be enough red blood cells and oxygen, they can't get to the cells that need them.

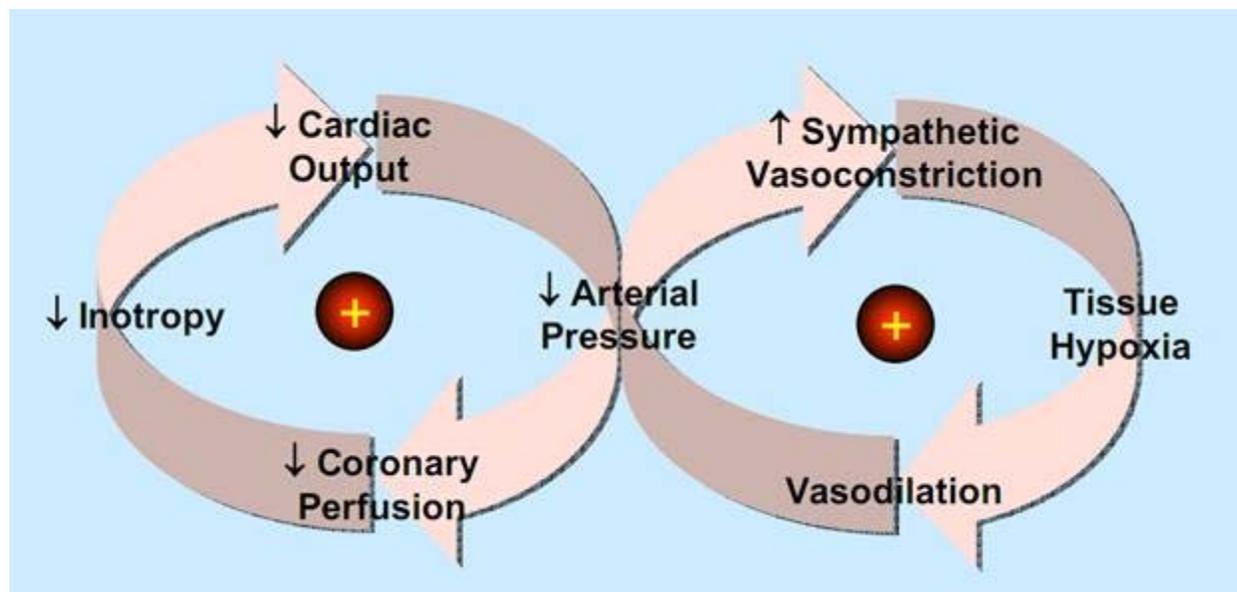


Fig. Mechanisms of cardiogenic shock

The heart is a muscle itself and needs blood supply to work. When a heart attack occurs, the blood supply to part of the heart is lost, and that can stun and irritate the heart muscle so that it isn't able to beat with an appropriate squeeze to push blood out to the rest of the body. This decreases stroke volume, and cardiac output falls.

Treatment includes trying to restore blood supply and the use of medications to support blood pressure. In more dire circumstances, machines can be used to assist the heart to support blood pressure.

Neurogenic Shock

There are involuntary muscles within blood vessel walls that maintain the squeeze so that the volume within the vessels stays constant even if the body changes position against gravity. As an analogy, is when you get up out of bed in the morning. If your blood vessels didn't squeeze a little tighter, gravity would make the blood flow to your feet, the lowest part of your body,

away from your brain, and you might pass out. The squeeze is maintained by signals from nerves in the sympathetic trunk, a long bundle of fibers running from the skull to the tailbone alongside the vertebral column.

In brain or spinal injury, the sympathetic trunk stops working and blood vessels dilate and result in blood pooling away from the heart. Since there isn't enough blood returning to the heart, the heart has a hard time pumping blood through the body.

Treatment includes fluids and medications to increase the tone in the blood vessel walls.

Hypoglycemic Shock and Hyperglycemia

High or low blood sugars are almost always associated with [diabetes](#). In people with diabetes, the body does not make enough insulin to permit glucose to enter the cells for aerobic metabolism. As treatment, insulin needs to be injected, or medication needs to be taken to boost the body's lower insulin production. There must be a balance between how much medication is taken and how much food is eaten.

If not enough food is ingested, then the blood sugar drops ([hypoglycemia](#)) and no glucose is available to enter the cells, even if there is enough insulin to permit glucose to enter the cells. The brain is very susceptible to low blood sugars, and [coma](#) has a very quick onset. Treatment is providing sugar. If the person is awake enough to swallow, a sugar solution by mouth is used, otherwise, intravenous fluids containing glucose are provided. If the lack of sugar was of short duration, the person will awaken almost immediately after treatment. If blood sugars remain low for prolonged periods of time, the brain's ability to recover is potentially lost.

When blood sugar levels spiral high out of control, there is risk of significant dehydration and shock. If there is not enough insulin in the blood stream, cells cannot use the glucose that is present, and instead turn to an alternative anaerobic metabolism to generate energy. Since glucose can't enter cells to be used, [hyperglycemia](#) (hyper= high + gly=sugar = emia) occurs as the glucose level builds up in the blood stream. The kidneys try to excrete excess sugar, but because of chemical concentration gradients between blood and urine, significant amounts of water also are lost. The body quickly becomes dehydrated and blood pressure drops, decreasing blood flow to cells. Cells which are now lacking glucose inside them are now starved of oxygen and turn to anaerobic metabolism, causing acid waste product build up. Excess acid in the body changes the metabolism for all organs, making it more difficult for oxygen to be used.

Conditions will continue worsen until insulin and significant fluids are given to the patient.

Anaphylactic Shock

When the body develops an [allergic reaction](#) to some outside chemical or substance, it can activate its immune system to combat that substance. On occasion, there can be an excess response and multiple organ systems in the body can be affected and fail. This is known as anaphylaxis. Mast cells and basophils (a type of white blood cell) that contain histamine become unstable and leak their contents to affect the muscles of the lung, heart and blood vessels. These are smooth muscles that are part of the regulatory system of the body and are not under conscious control.

- The muscles that surround bronchial tubes go into spasm and cause wheezing and shortness of breath.
- The muscles that surround blood vessels dilate, causing blood pressure to drop.
- The histamine also causes flushing of the skin, urticaria ([hives](#)), vomiting and diarrhea.
- A variety of mechanisms cause the heart muscle to pump weakly and blood vessels to leak fluid.

The combination of these effects decrease blood flow and oxygen supply to cells in the body and can result in shock.

The most common causes of anaphylactic shock include allergic reactions to foods (especially peanuts), [antibiotics](#), and [bee and wasp stings](#). Children are often allergic to eggs, [soy](#), and milk.

These allergens can cause the immune system to turn on the potential cascade to shock. Many patients have allergic reactions that are less severe and can just involve hives, but others can develop shortness of breath, wheezing, swelling of the tongue and mouth, and difficulty swallowing.

Medical interventions include injections of antihistamine like [diphenhydramine](#) (Benadryl), corticosteroids and adrenaline (epinephrine).

Patients with major allergic reactions must try to avoid the chemical trigger. They also often carry an Epipen ([epinephrine injection](#) kit) to inject themselves with epinephrine should an allergic reaction occur.

Shock Symptoms

Shock is defined as abnormal metabolism at the cellular level. Since it is not easy to directly measure cellular problems, the symptoms of shock are indirect measurements of cellular function. Shock is the end stage of all diseases, and symptoms will often be dependant on the underlying cause.

Vital signs

As the patient goes through the various stages of shock, vital signs change. In the early stages, the body tries to compensate by moving fluids around from within cells to the blood stream with an attempt to maintain blood pressure in a normal range. However, there may be a slight rise in the heart rate (tachycardia = tachy or fast + cardia or heart). For example, donating blood. A unit of blood (or about 10% of the bloods volume) is removed, yet the body compensates well, except for a little lightheadedness, which is often resolved by drinking fluids. Another example is exercising and forgetting to drink enough fluids and feeling a little tired at the end of the day.

As the body loses the ability to compensate, the breathing rate gets faster and the tachycardia increases as the body tries to pack as much oxygen onto the remaining red blood cells as possible and deliver them to the cells. Unfortunately, blood pressure starts to drop (hypotension=hypo or low + tension= pressure) as compensation mechanisms fail.

Body function

Cells don't receive enough oxygen and the organs that they comprise begin to fail. All organs may be affected.

- As the brain is affected, the patient may become confused or lose consciousness (coma).
- There may be [chest pain](#) as the heart itself doesn't get an adequate oxygen supply.
- Diarrhea may occur as the large intestine becomes irritated due to hypotension.
- [Kidneys may fail](#) and the body may stop producing urine.
- The skin becomes clammy and pale.

Shock Diagnosis

The approach to the patient in shock requires that treatment occur at the same time as the

diagnosis occurs. The source of the underlying disease needs to be found. Sometimes it is obvious, for example, a trauma victim bleeding from a wound. Other times, the diagnosis is elusive. The type of tests will depend upon the underlying condition.

The diagnosis is most often found through the medical history. A thorough physical examination will be undertaken and the patients vital signs monitored.

- **Patient vital signs monitored** might include continual blood pressure and heart rate monitoring, and oxygen measurement. Special catheters may be inserted into the large veins in the neck, chest, arm, or groin and threaded near the heart or into the pulmonary artery, to measure pressures close to the heart, which may be a better indicator of the body's fluid status. Other catheters may be inserted into arteries (arterial lines) to measure blood pressures more directly. Tubes may be placed in the bladder ([Foley catheter](#)) to measure urine output.
- **Blood laboratory tests** will be performed (the type dependent on the underlying disease or condition).
- **Radiologic tests** may be performed dependent on the underlying illness.

Differential diagnosis

Shock is a common end point of many medical conditions. It has been divided into four main types based on the underlying cause: hypovolemic, distributive, cardiogenic and obstructive. A few additional classifications are occasionally used including: endocrinologic shock.

Hypovolemic

This is the most common type of shock and is caused by insufficient circulating [volume](#). Its primary cause is hemorrhage (internal and/or external), or loss of fluid from the [circulation](#). [Vomiting](#) and [diarrhea](#) are the most common cause in children. With other causes including burns, environmental exposure and excess urine loss due to [diabetic ketoacidosis](#) and [diabetes insipidus](#).

Cardiogenic

This type of shock is caused by the failure of the heart to pump effectively. This can be due to damage to the heart muscle, most often from a large [myocardial infarction](#). Other causes of cardiogenic shock include [dysrhythmias](#), [cardiomyopathy/myocarditis](#), [congestive heart failure](#) (CHF), [contusio cordis](#), or [cardiac valve](#) problems.

Obstructive

Obstructive shock is due to obstruction of blood flow outside of the heart. Several conditions can result in this form of shock.

- [Cardiac tamponade](#) in which fluid in the pericardium prevents inflow of blood into the heart (venous return). [Constrictive pericarditis](#), in which the [pericardium](#) shrinks and hardens, is similar in presentation.
- [Tension pneumothorax](#) Through increased intrathoracic pressure, bloodflow to the heart is prevented (venous return).
- [Pulmonary embolism](#) is the result of a thromboembolic incident in the blood vessels of the [lungs](#) and hinders the return of blood to the heart.
- [Aortic stenosis](#) hinders circulation by obstructing the [ventricular outflow tract](#)

Distributive

Distributive shock is due to impaired utilization of oxygen and thus production of energy by the cell. Examples of this form of shock are:

- [Septic shock](#) is the most common cause of distributive shock. Caused by an overwhelming systemic infection resulting in [vasodilation](#) leading to hypotension. Septic shock can be caused by [Gram negative](#) bacteria such as (among others) [Escherichia coli](#), Proteus species, [Klebsiella pneumoniae](#) which release an [endotoxin](#) which produces adverse biochemical, immunological and occasionally neurological effects which are harmful to the body, and other [Gram-positive](#) cocci, such as [pneumococci](#) and [streptococci](#), and certain fungi as well as Gram-positive bacterial toxins. Septic shock also includes some elements of cardiogenic shock. In 1992, the ACCP/SCCM Consensus Conference Committee defined septic shock: ". . .sepsis-induced hypotension (systolic blood pressure <90 mm Hg or a reduction of 40 mm Hg from baseline) despite adequate fluid resuscitation along with the presence of perfusion abnormalities that may include, but are not limited to, lactic acidosis, oliguria, or an acute alteration in mental status. Patients who are receiving inotropic or vasopressor agents may have a normalized blood pressure at the time that perfusion abnormalities are identified."
- [Anaphylactic shock](#) Caused by a severe [anaphylactic reaction](#) to an [allergen](#), [antigen](#), [drug](#) or foreign protein causing the release of [histamine](#) which causes widespread vasodilation, leading to hypotension and increased capillary permeability.
- High spinal injuries may cause [neurogenic shock](#). The classic symptoms include [a slow heartrate](#) due to loss of cardiac [sympathetic tone](#) and warm skin due to dilation of the

peripheral blood vessels. (This term can be confused with [spinal shock](#) which is a recoverable loss of function of the [spinal cord](#) after injury and does not refer to the haemodynamic instability per se.)

Endocrine

Based on [endocrine](#) disturbances such as:

- [Hypothyroidism](#) (Can be considered a form of [Cardiogenic shock](#)) in critically ill patients, reduces [cardiac output](#) and can lead to [hypotension](#) and respiratory insufficiency.
- [Thyrotoxicosis](#) ([Cardiogenic shock](#))
 - may induce a reversible cardiomyopathy.
- Acute [adrenal insufficiency](#) ([Distributive shock](#)) is frequently the result of discontinuing [corticosteroid](#) treatment without tapering the dosage. However, surgery and intercurrent disease in patients on corticosteroid therapy without adjusting the dosage to accommodate for increased requirements may also result in this condition.
- Relative adrenal insufficiency ([Distributive shock](#)) in critically ill patients where present [hormone levels](#) are insufficient to meet the higher demands

Shock Treatment

Shock Self-Care at Home

If you come upon a person in shock, the initial response should be to call 101 in Ukraine (911 in Usa) and activate the emergency response system. Self-care at home is not appropriate.

Lay the person down in a safe place and try to keep them warm and comfortable.

If the patient is not awake, is not breathing, and has no heartbeat, it is appropriate to start chest compressions following the American Heart Association guidelines. It is important to send someone to get an AED if one is available.

Shock Medical Treatment

- EMS personnel are well trained in the initial assessment of the patient in shock. The first course of action is to make certain that the **ABCs** have been assessed. The so-called ABCs are:

- **Airway:** assessment of whether the patient is awake enough to try to take their own breaths and/or if there is anything blocking the mouth or nose.
- **Breathing:** assessment of the adequacy of breathing and whether it may need to be assisted with mouth-to-mouth resuscitation or more aggressive interventions like a bag and mask or intubation with an [endotracheal tube](#) and a ventilator.
- **Circulation:** assessment of the adequacy of the blood pressure and determination of whether intravenous lines are needed for delivery of fluid or medications to support the blood pressure.

- **Reducing reperfusion injury & systemic inflammatory response syndrome (SIRS)**
 - Anti-inflammatory drugs
 - NO scavenging and antioxidant drugs
- **Resuscitation fluids**
 - Crystalloid vs. non-crystalloid solutions
 - Isotonic vs. hypertonic solutions
 - Whole blood vs. packed red cells
 - Hemoglobin-based solutions
 - Perfluorocarbon-based solutions
 - Fluid volume-related issues

Fig. Shock resuscitation

- If there is bleeding that is obvious, attempts to control it with direct pressure will be attempted.
- A fingerstick blood sugar will be checked to make certain that hypoglycemia ([low blood sugar](#)) does not exist.
- In the emergency department, diagnosis and treatment will occur at the same time.
- Patients will be treated with oxygen supplementation through nasal cannulae, a face mask, or endotracheal intubation. The method and amount of oxygen will be titrated to make certain enough oxygen is available for the body to use. Again, the goal will be to pack each hemoglobin molecule with oxygen.
- Blood may be transfused if bleeding (hemorrhage) is the cause of the shock state. If

bleeding is not the case, intravenous fluids will be given to bolster the volume of fluids within the blood vessels.

- Intravenous drugs can be used to try to maintain blood pressure (vasopressors). They work by stimulating the heart to beat stronger and by squeezing blood vessels to increase the flow within them.

Shock Prognosis

Shock is a culmination of multiple organ systems in the body that have failed or are in the process of failing. Even with the best of care, there is a significant risk of death. The mortality rate for shock depends upon the type and reason for the shock, and the age and underlying health condition of the patient.

Hemorrhagic Shock

Background

Shock is a state of inadequate perfusion, which does not sustain the physiologic needs of organ tissues. Many conditions, including blood loss but also including nonhemorrhagic states such as dehydration, sepsis, impaired autoregulation, obstruction, decreased myocardial function, and loss of autonomic tone, may produce shock or shocklike states.

Pathophysiology

In hemorrhagic shock, blood loss exceeds the body's ability to compensate and provide adequate tissue perfusion and oxygenation. This frequently is due to trauma, but it may be caused by spontaneous hemorrhage (eg, GI bleeding, childbirth), surgery, and other causes.

Most frequently, clinical hemorrhagic shock is caused by an acute bleeding episode with a discrete precipitating event. Less commonly, hemorrhagic shock may be seen in chronic conditions with subacute blood loss.

- Class I hemorrhage (loss of 0-15%)
 - Little tachycardia
 - Usually no significant change in BP, pulse pressure, respiratory rate
- Class II hemorrhage (loss of 15-30%)
 - HR >100 beats per minute, tachypnea, decreased pulse pressure
- Class III hemorrhage (loss of 30-40%)
 - Marked tachycardia and tachypnea, decreased systolic BP, oliguria
- Class IV hemorrhage (loss of >40%)
 - Marked tachycardia and decreased systolic BP, narrowed pulse pressure, markedly decreased (or no) urinary output
 - Immediately life threatening

Fig. Classes of hemorrhagic shock

Physiologic compensation mechanisms for hemorrhage include initial peripheral and mesenteric vasoconstriction to shunt blood to the central circulation. This is then augmented by a progressive tachycardia. Invasive monitoring may reveal an increased cardiac index, increased oxygen delivery (ie, DO_2), and increased oxygen consumption (ie, VO_2) by tissues. Lactate levels, acid-base status, and other markers also may provide useful indicators of physiologic status. Age, medications, and comorbid factors all may affect a patient's response to hemorrhagic shock.

Failure of compensatory mechanisms in hemorrhagic shock can lead to death. Without intervention, a classic trimodal distribution of deaths is seen in severe hemorrhagic shock. An initial peak of mortality occurs within minutes of hemorrhage due to immediate exsanguination. Another peak occurs after 1 to several hours due to progressive decompensation. A third peak occurs days to weeks later due to sepsis and organ failure.

Epidemiology

Frequency

United States

Accidental injuries remain the leading cause of death in individuals aged 1-44 years. Hemorrhagic shock is a leading cause of death among trauma patients.

History

History taking should address the following:

- Specific details of the mechanism of trauma or other cause of hemorrhage are essential.
- Inquire about a history of bleeding disorders and surgery.
- Prehospital interventions, especially the administration of fluids, and changes in vital signs should be determined. Emergency medical technicians or paramedics should share this information.

Physical

Findings at physical examination may include the following:

- Head, ears, eyes, nose, and throat
 - Sources of hemorrhage usually are apparent.
 - The blood supply of the scalp is rich and can produce significant hemorrhage.
 - Intracranial hemorrhage usually is insufficient to produce shock, except possibly in very young individuals.
- Chest
 - Hemorrhage into the thoracic cavities (pleural, mediastinal, pericardial) may be discerned at physical examination. Ancillary studies often are required for confirmation.
 - Signs of hemothorax may include respiratory distress, decreased breath sounds, and dullness to percussion.
 - Tension hemothorax, or hemothorax with cardiac and contralateral lung compression, produces jugular venous distention and hemodynamic and respiratory decompensation.
 - With pericardial tamponade, the classic triad of muffled heart sounds, jugular venous distention, and hypotension often is present, but these signs may be difficult to appreciate in the setting of an acute resuscitation.
- Abdomen
 - Injuries to the liver or spleen are common causes of hemorrhagic shock. Spontaneous rupture of abdominal aortic aneurysm (AAA) may also cause severe intra-abdominal hemorrhage and shock.
 - Blood irritates the peritoneal cavity; diffuse tenderness and peritonitis are common

when blood is present. However, the patient with altered mental status or multiple concomitant injuries may not have the classic signs and symptoms at physical examination.

- Progressive abdominal distention in hemorrhagic shock is highly suggestive of intra-abdominal hemorrhage.
- Pelvis
 - Fractures can produce massive bleeding. Retroperitoneal bleeding must be suspected.
 - Flank ecchymosis may indicate retroperitoneal hemorrhage.
- Extremities
 - Hemorrhage from extremity injuries may be apparent, or tissues may obscure significant bleeding.
 - Femoral fractures may produce significant blood loss.
- Nervous system
 - Agitation and combativeness may be seen in the initial stages of hemorrhagic shock.
 - These signs are followed by a progressive decline in level of consciousness due to cerebral hypoperfusion or concomitant head injury.

Differential Diagnoses

- Abdominal Trauma, Blunt
- Abdominal Trauma, Penetrating
- Abortion, Complications
- Anemia, Acute
- Anemia, Chronic
- Blast Injuries
- Disseminated Intravascular Coagulation
- Pneumothorax, Tension and Traumatic
- Pregnancy, Ectopic
- Pregnancy, Postpartum Hemorrhage
- Pregnancy, Trauma
- Shock, Cardiogenic
- Shock, Hypovolemic
- Shock, Septic
- Spinal Cord Injuries

Laboratory Studies

- Laboratory studies are essential in management of many forms of hemorrhagic shock. Baseline levels are determined frequently, but these infrequently change the initial management after trauma. Serial evaluations of the following can help guide ongoing therapy.
 - CBC
 - Prothrombin time and/or activated partial thromboplastin time
 - Urine output rate can help guide adequacy of perfusion.
 - ABGs (Levels reflect acid-base and perfusion status.)
 - Lactate and base deficit are used in some centers to indicate the degree of metabolic debt. Clearance of these markers over time can reflect the adequacy of resuscitation.
- Typed and crossmatched packed red blood cells should be ordered immediately based on clinical suspicion of hemorrhagic shock. Fresh frozen plasma and platelets also may be required to correct or prevent coagulopathies that develop in severe hemorrhagic shock.

Imaging Studies

Cervical spine, chest, and pelvis radiographs are the standard screening images for severe trauma. Other radiographs may be indicated for orthopedic injuries.

Computed tomography can be used to image the appropriate region of suspected injury. CT scanning frequently is the method of choice for evaluating possible intra-abdominal and/or retroperitoneal sources of hemorrhage in stable patients (see the image below). Oral contrast material may not increase the diagnostic yield of abdominal CT scanning in blunt trauma. Scanning should not be delayed to administer oral contrast material.

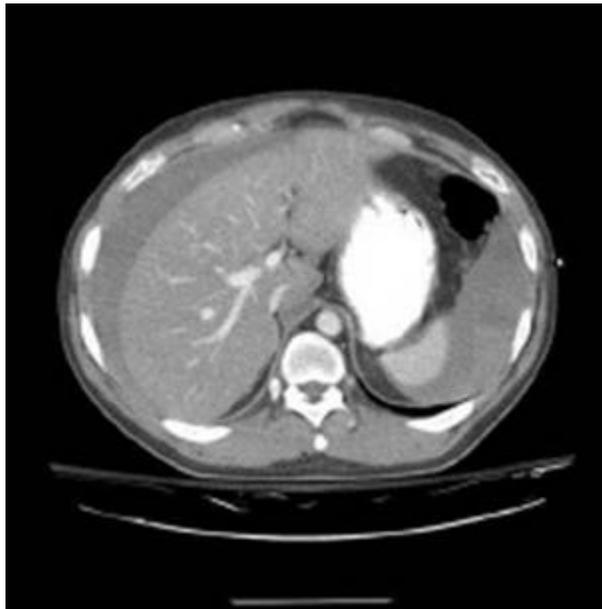


Fig. CT scan of a 26-year-old man after a motor vehicle crash shows a significant amount of intra-abdominal bleeding.

Bedside ultrasonography abdominal ultrasonography can be very useful for the rapid detection of AAA and free intra-abdominal fluid. Thoracic ultrasonographic findings can immediately confirm hemothorax or pericardial tamponade.

Directed angiography may be diagnostic and therapeutic. Interventional radiologists have had good success achieving hemostasis in hemorrhage caused by a variety of vessels and organs.

Other Tests

- An ECG can be useful for detecting dysrhythmias and cardiac sequelae of shock.
- Tissue oximetry using Near Infrared Spectroscopy (NIRS) shows promise for continuous noninvasive measurement of perfusion in hemorrhagic shock and other conditions.

Procedures

- Tube thoracostomy is necessary in significant hemothorax with or without pneumothorax.
- Central venous access facilitates fluid resuscitation and monitoring of central venous pressure and is necessary if peripheral intravenous access is inadequate or impossible to obtain.
- Diagnostic peritoneal lavage is used to detect intra-abdominal blood, fluid, and intestinal contents. It is sensitive but not specific for abdominal injury. It is not used to evaluate the retroperitoneum, which can hold significant hemorrhage, and does not identify the source

of hemorrhage.

Prehospital Care

The standard care consists of rapid assessment and expeditious transport to an appropriate center for evaluation and definitive care.

Intravenous access and fluid resuscitation are standard. However, this practice has become controversial.

- For many years, aggressive fluid administration has been advocated to normalize hypotension associated with severe hemorrhagic shock. Recent studies of urban patients with penetrating trauma have shown that mortality increases with these interventions; these findings call these practices into question.
- Reversal of hypotension prior to the achievement of hemostasis may increase hemorrhage, dislodge partially formed clots, and dilute existing clotting factors. Findings from animal studies of uncontrolled hemorrhage support these postulates. These provocative results raise the possibility that moderate hypotension may be physiologically protective and should be permitted, if present, until hemorrhage is controlled.
- These findings should not yet be clinically extrapolated to other settings or etiologies of hemorrhage. The ramifications of permissive hypotension in humans remain speculative, and safety limits have not been established yet.

Emergency Department Care

Management of hemorrhagic shock should be directed toward optimizing perfusion of and oxygen delivery to vital organs.

Diagnosis and treatment of the underlying hemorrhage must be performed rapidly and concurrently with management of shock.

Supportive therapy, including oxygen administration, monitoring, and establishment of intravenous access (eg, 2 large-bore catheters in peripheral lines, central venous access), should be initiated.

- Intravascular volume and oxygen-carrying capacity should be optimized.
- In addition to crystalloids, some colloid solutions, hypertonic solutions, and oxygen-carrying solutions (eg, hemoglobin-based and perfluorocarbon emulsions) are used or

being investigated for use in hemorrhagic shock.

- Blood products are often required in severe hemorrhagic shock. Replacement of lost components using red blood cells (RBCs), fresh frozen plasma (FFP), and platelets may be essential. The ideal ratio of RBCs to FFP remains undetermined. Recent combat experience has suggested that aggressive use of FFP may reduce coagulopathies and improve outcomes.

Determination of the site and etiology of hemorrhage is critical to guide further interventions and definitive care.

Control of hemorrhage may be achieved in the ED, or control may require consultations and special interventions.

Consultations

Consult a general or specialized surgeon, gastroenterologist, obstetrician-gynecologist, interventional radiologist, and others as required.

Medication Summary

Achievement of hemostasis, fluid resuscitation, and use of blood products are the mainstays of treatment. Pressor agents may be useful in some settings (eg, spinal shock), but these agents should not be substitutes for adequate volume resuscitation and blood product replacement.

Tranexamic acid (TXA) is an inexpensive antifibrinolytic drug that promotes blood clotting by preventing blood clots from breaking down. It has been shown to reduce mortality in trauma patients with uncontrolled hemorrhage. Further studies are planned to determine specific recommendations for TXA administration.

Vasopressors

Class Summary

These agents augment both coronary and cerebral blood flow during the low-flow state associated with shock.

[Dopamine \(Intropin\)](#)

Stimulates both adrenergic and dopaminergic receptors. Hemodynamic effect is dependent on the dose. Lower doses predominantly stimulate dopaminergic receptors that in turn produce renal and mesenteric vasodilation. Higher doses produce cardiac stimulation and renal vasodilation

Norepinephrine (Levophed)

Used in protracted hypotension following adequate fluid-volume replacement. Stimulates beta1-adrenergic and alpha-adrenergic receptors, which, in turn, increase cardiac muscle contractility and heart rate, as well as vasoconstriction; result is increased systemic BP and coronary blood flow.

Vasopressin (Pitressin)

Has vasopressor and ADH activity. Increases water resorption at distal renal tubular epithelium (ADH effect) and promotes smooth muscle contraction throughout the vascular bed of the renal tubular epithelium (vasopressor effects); however, vasoconstriction also is increased in splanchnic, portal, coronary, cerebral, peripheral, pulmonary, and intrahepatic vessels.

Epinephrine (Adrenalin, Bronitin)

Used for hypotension refractory to dopamine. Alpha-agonist effects include increased peripheral vascular resistance, reversed peripheral vasodilatation, systemic hypotension, and vascular permeability. Beta2-agonist effects include bronchodilatation, chronotropic cardiac activity, and positive inotropic effects.

Further Inpatient Care

- Admit the patient to an ICU, surgical ICU, or pediatric ICU.

- Patients with hemorrhagic shock should be admitted to an intensive care or monitored setting appropriate for the underlying condition and physiologic state.

Transfer

- In hospitals without facilities to provide definitive care, patients should be stabilized as much as possible and transferred to a facility with a higher level of care.

Complications

- Coagulopathies may occur in severe hemorrhage. Fluid resuscitation, while necessary, may exacerbate coagulopathies.
- Sepsis and multiple organ system failure occur days after acute hemorrhagic shock.
- Death is a possible complication.

Hypovolemic Shock

Hypovolemic shock refers to a medical or surgical condition in which rapid fluid loss results in multiple organ failure due to inadequate circulating volume and subsequent inadequate perfusion. Most often, hypovolemic shock is secondary to rapid blood loss (hemorrhagic shock).

Acute external blood loss secondary to penetrating trauma and severe GI bleeding disorders are 2 common causes of hemorrhagic shock. Hemorrhagic shock can also result from significant acute internal blood loss into the thoracic and abdominal cavities.

Two common causes of rapid internal blood loss are solid organ injury and rupture of an abdominal aortic aneurysm. Hypovolemic shock can result from significant fluid (other than blood) loss. Two examples of hypovolemic shock secondary to fluid loss include refractory gastroenteritis and extensive burns. The remainder of this article concentrates mainly on hypovolemic shock secondary to blood loss and the controversies surrounding the treatment of this condition. The reader is referred to other articles for discussions of the pathophysiology and treatment for hypovolemic shock resulting from losses of fluid other than blood.

The many life-threatening injuries experienced during the wars of the 1900s have significantly affected the development of the principles of hemorrhagic shock resuscitation. During World War I, W.B. Cannon recommended delaying fluid resuscitation until the cause of the hemorrhagic shock was repaired surgically. Crystalloids and blood were used extensively

during World War II for the treatment of patients in unstable conditions. Experience from the Korean and Vietnam wars revealed that volume resuscitation and early surgical intervention were paramount for surviving traumatic injuries resulting in hemorrhagic shock. These and other principles helped in the development of present guidelines for the treatment of traumatic hemorrhagic shock. However, recent investigators have questioned these guidelines, and today, controversies exist concerning the optimal treatment of hemorrhagic shock.

For more information, see Medscape's [Trauma Resource Center](#).

Pathophysiology

The human body responds to acute hemorrhage by activating the following major physiologic systems: the hematologic, cardiovascular, renal, and neuroendocrine systems.

The hematologic system responds to an acute severe blood loss by activating the coagulation cascade and contracting the bleeding vessels (by means of local thromboxane A₂ release). In addition, platelets are activated (also by means of local thromboxane A₂ release) and form an immature clot on the bleeding source. The damaged vessel exposes collagen, which subsequently causes fibrin deposition and stabilization of the clot. Approximately 24 hours are needed for complete clot fibrination and mature formation.

The cardiovascular system initially responds to hypovolemic shock by increasing the heart rate, increasing myocardial contractility, and constricting peripheral blood vessels. This response occurs secondary to an increased release of norepinephrine and decreased baseline vagal tone (regulated by the baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels). The cardiovascular system also responds by redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract.

The renal system responds to hemorrhagic shock by stimulating an increase in renin secretion from the juxtaglomerular apparatus. Renin converts angiotensinogen to angiotensin I, which subsequently is converted to angiotensin II by the lungs and liver. Angiotensin II has 2 main effects, both of which help to reverse hemorrhagic shock, vasoconstriction of arteriolar smooth muscle, and stimulation of aldosterone secretion by the adrenal cortex. Aldosterone is responsible for active sodium reabsorption and subsequent water conservation.

The neuroendocrine system responds to hemorrhagic shock by causing an increase in circulating antidiuretic hormone (ADH). ADH is released from the posterior pituitary gland in

response to a decrease in BP (as detected by baroreceptors) and a decrease in the sodium concentration (as detected by osmoreceptors). ADH indirectly leads to an increased reabsorption of water and salt (NaCl) by the distal tubule, the collecting ducts, and the loop of Henle.

The pathophysiology of hypovolemic shock is much more involved than what was just listed. To explore the pathophysiology in more detail, references for further reading are provided in the bibliography. These intricate mechanisms list above are effective in maintaining vital organ perfusion in severe blood loss. Without fluid and blood resuscitation and/or correction of the underlying pathology causing the hemorrhage, cardiac perfusion eventually diminishes, and multiple organ failure soon follows.

History

- In a patient with possible shock secondary to hypovolemia, the history is vital in determining the possible causes and in directing the workup. Hypovolemic shock secondary to external blood loss typically is obvious and easily diagnosed. Internal bleeding may not be as obvious as patients may complain only of weakness, lethargy, or a change in mental status.
- Symptoms of shock, such as weakness, lightheadedness, and confusion, should be assessed in all patients.
- In the patient with trauma, determine the mechanism of injury and any information that may heighten suspicion of certain injuries (eg, steering wheel damage or extensive passenger compartment intrusion in a motor vehicle accident).
- If conscious, the patient may be able to indicate the location of pain.
- Vital signs, prior to arrival in the ED, should also be noted.
- Chest, abdominal, or back pain may indicate a vascular disorder.
- The classic sign of a thoracic aneurysm is a tearing pain radiating to the back. Abdominal aortic aneurysms usually result in abdominal, back pain, or flank pain.
- In patients with GI bleeding, inquiry about hematemesis, melena, alcohol drinking history, excessive nonsteroidal anti-inflammatory drug use, and coagulopathies (iatrogenic or otherwise) is very important.
 - The chronology of vomiting and hematemesis should be determined.
 - The patient who presents with hematemesis after multiple episodes of forceful vomiting is more likely to have Boerhaave syndrome or a Mallory-Weiss tear,

whereas a patient with a history of hematemesis from the start is more likely to have peptic ulcer disease or esophageal varices.

- If a gynecologic cause is being considered, gather information about the following: last menstrual period, risk factors for ectopic pregnancy, vaginal bleeding (including amount and duration), vaginal passage of products of conception, and pain. All women of childbearing age should undergo a pregnancy test, regardless of whether they believe that they are pregnant. A negative pregnancy test typically excludes ectopic pregnancy as a diagnosis.

Physical

The physical examination should always begin with an assessment of the airway, breathing, and circulation. Once these have been evaluated and stabilized, the circulatory system should be evaluated for signs and symptoms of shock.

Do not rely on systolic BP as the main indicator of shock; this practice results in delayed diagnosis. Compensatory mechanisms prevent a significant decrease in systolic BP until the patient has lost 30% of the blood volume. More attention should be paid to the pulse, respiratory rate, and skin perfusion. Also, patients taking beta-blockers may not present with tachycardia, regardless of the degree of shock.

Classes of hemorrhage have been defined, based on the percentage of blood volume loss. However, the distinction between these classes in the hypovolemic patient often is less apparent. Treatment should be aggressive and directed more by response to therapy than by initial classification.

- Class I hemorrhage (loss of 0-15%)
 - In the absence of complications, only minimal tachycardia is seen.
 - Usually, no changes in BP, pulse pressure, or respiratory rate occur.
 - A delay in capillary refill of longer than 3 seconds corresponds to a volume loss of approximately 10%.
- Class II hemorrhage (loss of 15-30%)
 - Clinical symptoms include tachycardia (rate >100 beats per minute), tachypnea, decrease in pulse pressure, cool clammy skin, delayed capillary refill, and slight anxiety.
 - The decrease in pulse pressure is a result of increased catecholamine levels, which

causes an increase in peripheral vascular resistance and a subsequent increase in the diastolic BP.

- Class III hemorrhage (loss of 30-40%)
 - By this point, patients usually have marked tachypnea and tachycardia, decreased systolic BP, oliguria, and significant changes in mental status, such as confusion or agitation.
 - In patients without other injuries or fluid losses, 30-40% is the smallest amount of blood loss that consistently causes a decrease in systolic BP.
 - Most of these patients require blood transfusions, but the decision to administer blood should be based on the initial response to fluids.
- Class IV hemorrhage (loss of >40%)
 - Symptoms include the following: marked tachycardia, decreased systolic BP, narrowed pulse pressure (or immeasurable diastolic pressure), markedly decreased (or no) urinary output, depressed mental status (or loss of consciousness), and cold and pale skin.
 - This amount of hemorrhage is immediately life threatening.
- In the patient with trauma, hemorrhage usually is the presumed cause of shock. However, it must be distinguished from other causes of shock. These include cardiac tamponade (muffled heart tones, distended neck veins), tension pneumothorax (deviated trachea, unilaterally decreased breath sounds), and spinal cord injury (warm skin, lack of expected tachycardia, neurological deficits).
- The 4 areas in which life-threatening hemorrhage can occur are as follows: chest, abdomen, thighs, and outside the body.
 - The chest should be auscultated for decreased breath sounds, because life-threatening hemorrhage can occur from myocardial, vessel, or lung laceration.
 - The abdomen should be examined for tenderness or distension, which may indicate intraabdominal injury.
 - The thighs should be checked for deformities or enlargement (signs of femoral fracture and bleeding into the thigh).
 - The patient's entire body should then be checked for other external bleeding.
- In the patient without trauma, the majority of the hemorrhage is in the abdomen. The abdomen should be examined for tenderness, distension, or bruits. Look for evidence of an aortic aneurysm, peptic ulcer disease, or liver congestion. Also check for other signs of bruising or bleeding.

- In the pregnant patient, perform a sterile speculum examination. However, with third-trimester bleeding, the examination should be performed as a "double set-up" in the operating room. Check for abdominal, uterine, or adnexal tenderness.

Causes

The causes of hemorrhagic shock are traumatic, vascular, GI, or pregnancy related.

- Traumatic causes can result from penetrating and blunt trauma. Common traumatic injuries that can result in hemorrhagic shock include the following: myocardial laceration and rupture, major vessel laceration, solid abdominal organ injury, pelvic and femoral fractures, and scalp lacerations.
- Vascular disorders that can result in significant blood loss include aneurysms, dissections, and arteriovenous malformations.
- GI disorders that can result in hemorrhagic shock include the following: bleeding esophageal varices, bleeding peptic ulcers, Mallory-Weiss tears, and aortointestinal fistulas.
- Pregnancy-related disorders include ruptured ectopic pregnancy, placenta previa, and abruption of the placenta. Hypovolemic shock secondary to an ectopic pregnancy is common. Hypovolemic shock secondary to an ectopic pregnancy in a patient with a negative urine pregnancy test is rare but has been reported.

Differential Diagnoses

- Abruptio Placentae
- Aneurysm, Abdominal
- Aneurysm, Thoracic
- Fractures, Femur
- Fractures, Pelvic
- Gastritis and Peptic Ulcer Disease
- Placenta Previa
- Pregnancy, Ectopic
- Pregnancy, Postpartum Hemorrhage
- Pregnancy, Trauma
- Shock, Hemorrhagic
- Shock, Hypovolemic

- Toxicity, Iron

Laboratory Studies

- After the history is taken and the physical examination is performed, further workup depends on the probable cause of the hypovolemia, as well as on the stability of the patient's condition.
- Initial laboratory studies should include analysis of the CBC, electrolyte levels (eg, Na, K, Cl, HCO₃, BUN, creatinine, glucose levels), prothrombin time, activated partial thromboplastin time, ABGs, urinalysis (in patients with trauma), and a urine pregnancy test. Blood should be typed and cross-matched.

Imaging Studies

- Patients with marked hypotension and/or unstable conditions must first be resuscitated adequately. This treatment takes precedence over imaging studies and may include immediate interventions and immediately taking the patient to the operating room.
- The workup for the patient with trauma and signs and symptoms of hypovolemia is directed toward finding the source of blood loss.
- The atraumatic patient with hypovolemic shock requires ultrasonographic examination in the ED if an abdominal aortic aneurysm is suspected. If GI bleeding is suspected, a nasogastric tube should be placed, and gastric lavage should be performed. An upright chest radiograph should be obtained if a perforated ulcer or Boerhaave syndrome is a possibility. Endoscopy can be performed (usually after the patient has been admitted) to further delineate the source of bleeding.
- A pregnancy test should be performed in all female patients of childbearing age. If the patient is pregnant and in shock, surgical consultation and the consideration of bedside pelvic ultrasonography should be immediately performed in the ED. Hypovolemic shock secondary to an ectopic pregnancy is common. Hypovolemic shock secondary to an ectopic pregnancy in a patient with a negative pregnancy test, although rare, has been reported.
- If thoracic dissection is suspected because of the mechanism and initial chest radiographic findings, the workup may include transesophageal echocardiography, aortography, or CT scanning of the chest.
- If a traumatic abdominal injury is suspected, a focused abdominal sonography for trauma (FAST) ultrasonography examination may be performed in the stable or unstable patient.

Computed tomography (CT) scanning typically is performed in the stable patient.

- If long-bone fractures are suspected, radiographs should be obtained.

Prehospital Care

The treatment of patients with hypovolemic shock often begins at an accident scene or at home. The prehospital care team should work to prevent further injury, transport the patient to the hospital as rapidly as possible, and initiate appropriate treatment in the field. Direct pressure should be applied to external bleeding vessels to prevent further blood loss.

- Prevention of further injury applies mostly to the patient with trauma. The cervical spine must be immobilized, and the patient must be extricated, if applicable, and moved to a stretcher. Splinting of fractures can minimize further neurovascular injury and blood loss.
- Although in selected cases stabilization may be beneficial, rapid transport of sick patients to the hospital remains the most important aspect of prehospital care. Definitive care of the hypovolemic patient usually requires hospital, and sometimes surgical, intervention. Any delay in definitive care, eg, such as delayed transport, is potentially harmful.
- Most prehospital interventions involve immobilizing the patient (if trauma is involved), securing an adequate airway, ensuring ventilation, and maximizing circulation.
 - In the setting of hypovolemic shock, positive-pressure ventilation may diminish venous return, diminish cardiac output, and worsen the shock state. While oxygenation and ventilation are necessary, excessive positive-pressure ventilation can be detrimental for a patient suffering hypovolemic shock.
 - Appropriate treatment usually can be initiated without delaying transport. Some procedures, such as starting intravenous (IV) lines or splinting of extremities, can be performed while a patient is being extricated. However, procedures in the field that prolong transportation should be delayed. Benefits to giving IV fluids prior to departure from the scene are not clear; however, IV lines and fluid resuscitation should be started and continued once the patient is en route to definitive care.
- In recent years, there has been considerable debate regarding the use of military antishock trousers (MAST). MAST were introduced in the 1960s and, based mostly on anecdotal reports of success, their use became standard therapy in the prehospital treatment of hypovolemic shock in the late 1970s. By the 1980s, the American College of Surgeons Committee on Trauma included their use in the standard of care for all patients with trauma and signs or symptoms of shock. Since that time, studies have failed to show

improved outcome with the use of MAST. The American College of Surgeons Committee on Trauma no longer recommends the use of MAST.

Emergency Department Care

Three goals exist in the emergency department treatment of the patient with hypovolemic shock as follows: (1) maximize oxygen delivery - completed by ensuring adequacy of ventilation, increasing oxygen saturation of the blood, and restoring blood flow, (2) control further blood loss, and (3) fluid resuscitation. Also, the patient's disposition should be rapidly and appropriately determined.

Maximizing oxygen delivery

The patient's airway should be assessed immediately upon arrival and stabilized if necessary. The depth and rate of respirations, as well as breath sounds, should be assessed. If pathology (eg, pneumothorax, hemothorax, flail chest) that interferes with breathing is found, it should be addressed immediately. High-flow supplemental oxygen should be administered to all patients, and ventilatory support should be given, if needed. Excessive positive-pressure ventilation can be detrimental for a patient suffering hypovolemic shock and should be avoided.

Two large-bore IV lines should be started. The Poiseuille law states that flow is inversely related to the length of the IV catheter and directly related to its radius to the fourth power. Thus, a short large-caliber IV catheter is ideal; the caliber is much more significant than the length. IV access may be obtained by means of percutaneous access in the antecubital veins, cutdown of saphenous or arm veins, or access in the central veins by using the Seldinger technique. If central lines are obtained, a large-bore single-lumen catheter should be used. Intraosseous access has and continues to be used for hypotensive children younger than 6 years. Intraosseous access has also been used in hypotensive adults. The most important factor in determining the route of access is the practitioner's skill and experience.

Placement of an arterial line should be considered for patients with severe hemorrhage. For these patients, the arterial line will provide continuous blood pressure monitoring and also ease arterial blood gas testing.

Once IV access is obtained, initial fluid resuscitation is performed with an isotonic crystalloid, such as lactated Ringer solution or normal saline. An initial bolus of 1-2 L is given in an adult (20 mL/kg in a pediatric patient), and the patient's response is assessed.

If vital signs return to normal, the patient may be monitored to ensure stability, and blood should be sent for typed and cross-matched. If vital signs transiently improve, crystalloid infusion should continue and type-specific blood obtained. If little or no improvement is seen, crystalloid infusion should continue, and type O blood should be given (type O Rh-negative blood should be given to female patients of childbearing age to prevent sensitization and future complications).

If a patient is moribund and markedly hypotensive (class IV shock), both crystalloid and type O blood should be started initially. These guidelines for crystalloid and blood infusion are not rules; therapy should be based on the condition of the patient.

The position of the patient can be used to improve circulation; one example is raising the hypotensive patient's legs while fluid is being given. Another example of useful positioning is rolling a hypotensive gravid patient with trauma onto her left side, which displaces the fetus from the inferior vena cava and increases circulation. The Trendelenburg position is no longer recommended for hypotensive patients, as the patient is predisposed to aspiration. In addition, the Trendelenburg position does not improve cardiopulmonary performance and may worsen gas exchange.

Autotransfusion may be a possibility in some patients with trauma. Several devices that allow for the sterile collection, anticoagulation, filtration, and retransfusion of blood are available. In the trauma setting, this blood almost always is from a hemothorax collected by means of tube thoracostomy.

Controlling further blood loss

Control of further hemorrhage depends on the source of bleeding and often requires surgical intervention. In the patient with trauma, external bleeding should be controlled with direct pressure; internal bleeding requires surgical intervention. Long-bone fractures should be treated with traction to decrease blood loss.

In the patient whose pulse is lost in the ED or just prior to arrival, an emergency thoracotomy with cross-clamping of the aorta may be indicated to preserve blood flow to the brain. This procedure is palliative at best and requires immediate transfer to the operating room.

In the patient with GI bleeding, intravenous vasopressin and H₂ blockers have been used. Vasopressin commonly is associated with adverse reactions, such as hypertension, arrhythmias,

gangrene, and myocardial or splanchnic ischemia. Therefore, it should be considered secondary to more definitive measures. H₂ blockers are relatively safe but have no proven benefit.

Somatostatin and octreotide infusions have been shown to reduce gastrointestinal bleeding from varices and peptic ulcer disease. These agents possess the advantages of vasopressin without the significant side effects.

In patients with variceal bleeding, use of a Sengstaken-Blakemore tube can be considered. These devices have a gastric balloon and an esophageal balloon. The gastric one is inflated first, and then the esophageal one is inflated if bleeding continues. The use of this tube has been associated with severe adverse reactions, such as esophageal rupture, asphyxiation, aspiration and mucosal ulceration. For this reason, its use should be considered only as a temporary measure in extreme circumstances.

Virtually all causes of acute gynecological bleeding that cause hypovolemia (eg, ectopic pregnancy, placenta previa, abruptio placenta, ruptured cyst, miscarriage) require surgical intervention.

Early consultation and definitive care are the keys. The goal in the ED is to stabilize the hypovolemic patient, determine the cause of bleeding, and provide definitive care as quickly as possible. If transfer to another hospital is necessary, resources should be mobilized early.

In patients with trauma, if the emergency medical services personnel indicate potential serious injury, the surgeon (or trauma team) should be notified prior to the patient's arrival. In a 55-year-old patient with abdominal pain, for example, emergency ultrasonography of the abdomen may be necessary to identify an abdominal aortic aneurysm before the vascular surgeon is notified. Every patient should be individually evaluated, because delaying definitive care can increase morbidity and mortality.

Resuscitation

Whether crystalloids or colloids are best for resuscitation continues to be a matter for discussion and research. Many fluids have been studied for use in resuscitation; these include isotonic sodium chloride solution, lactated Ringer solution, hypertonic saline, albumin, purified protein fraction, fresh frozen plasma, hetastarch, pentastarch, and dextran 70.

Proponents of colloid resuscitation argue that the increased oncotic pressure produced with

these substances decreases pulmonary edema. However, the pulmonary vasculature allows considerable flow of material, including proteins, between the intravascular space and interstitium. Maintenance of the pulmonary hydrostatic pressure at less than 15 mm Hg appears to be a more important factor in preventing pulmonary edema.

Another argument is that less colloid is needed to increase the intravascular volume. Studies have shown this to be true. However, they still have not demonstrated any difference in outcome with colloids compared with crystalloids.

Synthetic colloid solutions, such as hetastarch, pentastarch, and dextran 70, have some advantages compared with natural colloids such as purified protein fraction, fresh frozen plasma, and albumin. They have the same volume-expanding properties, but because of their structures and high molecular weights, they remain mostly in the intravascular space, reducing the occurrence of interstitial edema. Although theoretic advantages exist, studies have failed to show a difference in ventilatory parameters, pulmonary function test results, days using a ventilator, total hospital days, or survival.

The European Society of Intensive Care Medicine (ESICM) advises against the use of colloids-hydroxyethyl starches (HES) in patients with severe sepsis or risk of acute kidney injury. Physicians should also avoid using colloids in patients with head injury and refrain from administering gelatins and HES in organ donors

The combination of hypertonic saline and dextran also has been studied because of previous evidence that it may improve cardiac contractility and circulation. Studies in the US and Japan have failed to show any difference when this combination was compared with isotonic sodium chloride solution or lactated Ringer solution. Thus, despite the many available resuscitation fluids, current recommendations still advocate the use of normal saline or lactated Ringer solution. In the US, one reason for the predominant use of crystalloids over the other resuscitative fluids is cost.

Recent literature suggests that the early administration of FFP and platelets improves survival and decreases overall PRBC need in patients undergoing a massive transfusion

Restoring normal circulating volume and BP prior to definitive control of bleeding

Although some data indicate that a systolic BP of 80-90 mm Hg may be adequate in penetrating truncal trauma without head injury, further studies are needed.

Current recommendations are for aggressive fluid resuscitation with lactated Ringer solution or normal saline in all patients with signs and symptoms of shock, regardless of underlying cause.

Medication Summary

The goals of pharmacotherapy are to reduce morbidity and prevent complications.

Antisecretory agents

Class Summary

These agents have vasoconstrictive properties and can reduce blood flow to portal systems.

Somatostatin (Zecnil)

Naturally occurring tetradecapeptide isolated from the hypothalamus and pancreatic and enteric epithelial cells. Diminishes blood flow to portal system because of vasoconstriction. Has similar effects as vasopressin but does not cause coronary vasoconstriction. Rapidly cleared from the circulation, with an initial half-life of 1-3 min.

Octreotide (Sandostatin)

Synthetic octapeptide. Compared to somatostatin, has similar pharmacological actions with greater potency and longer duration of action.

Used as adjunct to nonoperative management of secreting cutaneous fistulas of the stomach, duodenum, small intestine (jejunum and ileum), or pancreas.

Complications

- Neurologic sequelae
- Death

Prognosis

- The prognosis is dependent on the degree of volume loss.

Cardiogenic Shock

Background

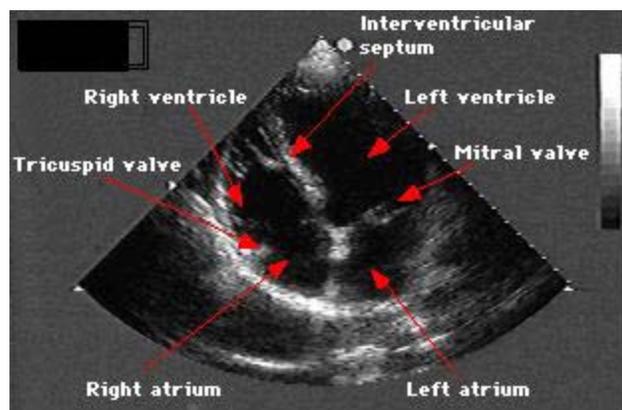
Cardiogenic shock is a physiologic state in which inadequate tissue perfusion results from cardiac dysfunction, most often systolic. It is a major, and frequently fatal, complication of a variety of acute and chronic disorders, occurring most commonly following acute [myocardial infarction \(MI\)](#). (See Pathophysiology, Etiology, and Prognosis.)

Although ST-segment elevation MI (STEMI, previously termed Q-wave MI) is encountered in most patients, cardiogenic shock may also develop in patients with non-ST-segment elevation [acute coronary syndrome](#) (NSTEMI, NSTACS, or [unstable angina](#)). (See the images below.)



Patient with an acute anterolateral myocardial infarction who developed cardiogenic shock. Coronary angiography images showed severe stenosis of the left anterior descending

coronary artery, which was dilated by percutaneous transluminal coronary angioplasty.



Echocardiogram image from a patient with cardiogenic shock shows enlarged cardiac chambers; the motion study showed poor left ventricular function. Courtesy of R. Hoeschen, MD.

The clinical definition of cardiogenic shock is decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume. Hemodynamic criteria for cardiogenic shock are sustained hypotension (systolic blood pressure < 90 mm Hg for at least 30 min) and a reduced cardiac index (< 2.2 L/min/m²) in the presence of elevated pulmonary capillary wedge pressure (>15 mm Hg). (See DDX, Workup.)

The diagnosis of cardiogenic shock can sometimes be made at the bedside by observing hypotension, absence of hypovolemia, and clinical signs of poor tissue perfusion, which include oliguria, cyanosis, cool extremities, and altered mentation. These signs usually persist after attempts have been made to correct hypovolemia, arrhythmia, hypoxia, and acidosis. (See Presentation, DDX.)

Pathophysiology

Cardiogenic shock is recognized as a low cardiac output state secondary to extensive left ventricular infarction, development of a mechanical defect (eg, ventricular septal defect or [papillary muscle rupture](#)), or right ventricular infarction.

Disorders that can result in the acute deterioration of cardiac function and lead to cardiogenic

shock include myocardial infarction (MI) or myocardial ischemia, acute myocarditis, sustained arrhythmia, severe valvular dysfunction, and decompensation of end-stage cardiomyopathy from multiple etiologies. Autopsy studies show that cardiogenic shock is generally associated with the loss of more than 40% of the left ventricular myocardial muscle.

Myocardial pathology

Cardiogenic shock is characterized by systolic and diastolic dysfunction. Patients who develop cardiogenic shock from acute MI consistently have evidence of progressive myocardial necrosis with infarct extension. Decreased coronary perfusion pressure and increased myocardial oxygen demand play a role in the vicious cycle that leads to cardiogenic shock.

Patients suffering from cardiogenic shock often have multivessel coronary artery disease with limited coronary blood flow reserve. Ischemia remote from the infarcted zone is an important contributor to shock. Myocardial diastolic function is also impaired, because ischemia causes decreased myocardial compliance, thereby increasing left ventricular filling pressure, which may lead to pulmonary edema and hypoxemia.

Cellular pathology

Tissue hypoperfusion, with consequent cellular hypoxia, causes anaerobic glycolysis, the accumulation of lactic acid, and intracellular acidosis. Also, myocyte membrane transport pumps fail, which decreases transmembrane potential and causes intracellular accumulation of sodium and calcium, resulting in myocyte swelling.

If ischemia is severe and prolonged, myocardial cellular injury becomes irreversible and leads to myonecrosis, which includes mitochondrial swelling, the accumulation of denatured proteins and chromatin, and lysosomal breakdown. These events induce fracture of the mitochondria, nuclear envelopes, and plasma membranes.

Additionally, apoptosis (programmed cell death) may occur in peri-infarcted areas and may contribute to myocyte loss. Activation of inflammatory cascades, oxidative stress, and stretching of the myocytes produces mediators that overpower inhibitors of apoptosis, thus activating the apoptosis.

Reversible myocardial dysfunction

Large areas of myocardium that are dysfunctional but still viable can contribute to the development of cardiogenic shock in patients with MI. This potentially reversible dysfunction is often described as myocardial stunning or as hibernating myocardium. Although hibernation is considered a different physiologic process than myocardial stunning, the conditions are difficult to distinguish in the clinical setting and they often coexist.

Myocardial stunning represents postischemic dysfunction that persists despite restoration of normal blood flow. By definition, myocardial dysfunction from stunning eventually resolves completely. The mechanism of myocardial stunning involves a combination of oxidative stress, abnormalities of calcium homeostasis, and circulating myocardial depressant substances.

Hibernating myocardium is a state of persistently impaired myocardial function at rest, which occurs because of the severely reduced coronary blood flow. Hibernation appears to be an adaptive response to hypoperfusion that may minimize the potential for further ischemia or necrosis. Revascularization of the hibernating (and/or stunned) myocardium generally leads to improved myocardial function.

Cardiovascular mechanics of cardiogenic shock

The main mechanical defect in cardiogenic shock is a shift to the right for the left ventricular end-systolic pressure-volume curve, because of a marked reduction in contractility. As a result, at a similar or even lower systolic pressure, the ventricle is able to eject less blood volume per beat. Therefore, the end-systolic volume is usually greatly increased in persons with cardiogenic shock.

The stroke volume is decreased, and to compensate for this, the curvilinear diastolic pressure-volume curve also shifts to the right, with a decrease in diastolic compliance. This leads to increased diastolic filling, which is associated with an increase in end-diastolic pressure. The attempt to enhance cardiac output by this mechanism comes at the cost of having a higher left ventricular diastolic filling pressure, which ultimately increases myocardial oxygen demand and causes pulmonary edema.

As a result of decreased contractility, the patient develops elevated left and right ventricular

filling pressures and low cardiac output. Mixed venous oxygen saturation falls because of the increased tissue oxygen extraction, which is due to the low cardiac output. This, combined with the intrapulmonary shunting that is often present, contributes to substantial arterial oxygen desaturation.

Systemic effects

When a critical mass of left ventricular myocardium becomes ischemic and fails to pump effectively, stroke volume and cardiac output are curtailed. Myocardial ischemia is further exacerbated by compromised myocardial perfusion due to hypotension and tachycardia.

The pump failure increases ventricular diastolic pressures concomitantly, causing additional wall stress and thereby elevating myocardial oxygen requirements. Systemic perfusion is compromised by decreased cardiac output, with tissue hypoperfusion intensifying anaerobic metabolism and instigating the formation of lactic acid, which further deteriorates the systolic performance of the myocardium.

Depressed myocardial function also leads to the activation of several physiologic compensatory mechanisms. These include sympathetic stimulation, which increases the heart rate and cardiac contractility and causes renal fluid retention, hence augmenting the left ventricular preload. The raised heart rate and contractility increases myocardial oxygen demand, further worsening myocardial ischemia.

Fluid retention and impaired left ventricular diastolic filling triggered by tachycardia and ischemia contribute to pulmonary venous congestion and hypoxemia. Sympathetically mediated vasoconstriction to maintain systemic blood pressure amplifies myocardial afterload, which additionally impairs cardiac performance. Finally, excessive myocardial oxygen demand with simultaneous inadequate myocardial perfusion worsens myocardial ischemia, initiating a vicious cycle that ultimately ends in death, if uninterrupted.

Usually, a combination of systolic and diastolic myocardial dysfunction is present in patients with cardiogenic shock. Metabolic derangements that impair myocardial contractility further compromise systolic ventricular function. Myocardial ischemia decreases myocardial compliance, thereby elevating left ventricular filling pressure at a given end-diastolic volume (diastolic dysfunction), which leads to pulmonary congestion and congestive heart failure.

Shock state

Shock state, irrespective of the etiology, is described as a syndrome initiated by acute systemic hypoperfusion that leads to tissue hypoxia and vital organ dysfunction. All forms of shock are characterized by inadequate perfusion to meet the metabolic demands of the tissues. A maldistribution of blood flow to end organs begets cellular hypoxia and end organ damage, the well-described multisystem organ dysfunction syndrome. The organs of vital importance are the brain, heart, and kidneys.

A decline in higher cortical function may indicate diminished perfusion of the brain, which leads to an altered mental status ranging from confusion and agitation to flaccid coma. The heart plays a central role in propagating shock. Depressed coronary perfusion leads to worsening cardiac dysfunction and a cycle of self-perpetuating progression of global hypoperfusion. Renal compensation for reduced perfusion results in diminished glomerular filtration, causing oliguria and subsequent renal failure.

Etiology

Cardiogenic shock can result from the following types of cardiac dysfunction:

- Systolic dysfunction
- Diastolic dysfunction
- Valvular dysfunction
- Cardiac arrhythmias
- Coronary artery disease
- Mechanical complications

The vast majority of cases of cardiogenic shock in adults are due to acute myocardial ischemia. Indeed, cardiogenic shock is generally associated with the loss of more than 40% of the left ventricular myocardium, although in patients with previously compromised left ventricular function, even a small infarction may precipitate shock. Cardiogenic shock is more likely to develop in people who are elderly or diabetic or in persons who have had a previous inferior myocardial infarction (MI).

Complications of acute MI, such as acute mitral regurgitation, large right ventricular infarction, and [rupture](#) of the interventricular septum or left ventricular free wall, can result in cardiogenic shock. Conduction abnormalities (eg, atrioventricular blocks, sinus bradycardia) are also risk factors.

Many cases of cardiogenic shock occurring after acute coronary syndromes may be due to medication administration. The use of beta blockers and angiotensin-converting enzyme (ACE) inhibitors in acute coronary syndromes must be carefully timed and monitored.

In children, preceding viral infection may cause [myocarditis](#). In addition, children and infants may have unrecognized congenital structural heart defects that are well compensated until there is a stressor. These etiologies plus toxic ingestions make up the 3 primary causes of cardiogenic shock in children.

A systemic inflammatory response syndrome–type mechanism has also been implicated in the etiology of cardiogenic shock. Elevated levels of white blood cells, body temperature, complement, interleukins, and C-reactive protein are often seen in large myocardial infarctions. Similarly, inflammatory nitric oxide synthetase (iNOS) is also released in high levels during myocardial stress. Nitric oxide production induced by iNOS may uncouple calcium metabolism in the myocardium resulting in a stunned myocardium. Additionally, iNOS leads to the expression of interleukins, which may themselves cause hypotension.

Left ventricular failure

Systolic dysfunction

The primary abnormality in systolic dysfunction is abated myocardial contractility. Acute MI or ischemia is the most common cause; cardiogenic shock is more likely to be associated with anterior MI. The causes of systolic dysfunction leading to cardiogenic shock can be summarized as follows:

- Ischemia/MI
- Global hypoxemia
- Valvular disease
- Myocardial depressant drugs - Eg, beta blockers, calcium channel blockers, and antiarrhythmics
- Myocardial contusion
- Respiratory acidosis
- Metabolic derangements - Eg, acidosis, hypophosphatemia, and hypocalcemia
- Severe myocarditis
- End-stage cardiomyopathy - Including valvular causes

- Prolonged cardiopulmonary bypass.
- Cardiotoxic drugs - Eg, doxorubicin (Adriamycin)

Diastolic dysfunction

Increased left ventricular diastolic chamber stiffness contributes to cardiogenic shock during cardiac ischemia, as well as in the late stages of hypovolemic shock and septic shock. Increased diastolic dysfunction is particularly detrimental when systolic contractility is also depressed. The causes of cardiogenic shock due primarily to diastolic dysfunction can be summarized as follows:

- Ischemia
- Ventricular hypertrophy
- Restrictive cardiomyopathy
- Prolonged hypovolemic or septic shock
- Ventricular interdependence
- External compression by pericardial tamponade

Greatly increased afterload

Increased afterload, which can impair cardiac function, can be caused by the following:

- Aortic stenosis
- Hypertrophic cardiomyopathy
- Dynamic aortic outflow tract obstruction
- Coarctation of the aorta
- Malignant hypertension

Valvular and structural abnormality

Valvular dysfunction may immediately lead to cardiogenic shock or may aggravate other etiologies of shock. Acute mitral regurgitation secondary to papillary muscle rupture or dysfunction is caused by ischemic injury. Rarely, acute obstruction of the mitral valve by a left atrial thrombus may result in cardiogenic shock by means of severely decreased cardiac output. Aortic and mitral regurgitation reduce forward flow, raise end-diastolic pressure, and aggravate shock associated with other etiologies.

Valvular and structural abnormalities associated with cardiogenic shock include the following:

- Mitral stenosis
- Endocarditis
- Mitral aortic regurgitation
- Obstruction due to atrial myxoma or thrombus
- Papillary muscle dysfunction or rupture
- Ruptured septum or free wall arrhythmias
- Tamponade

Decreased contractility

Reduced myocardial contractility can result from the following:

- Right ventricular infarction
- Ischemia
- Hypoxia
- Acidosis

Right ventricular failure

Greatly increased afterload

Afterload increase associated with right ventricular failure can result from the following:

- Pulmonary embolism
- Pulmonary vascular disease - Eg, pulmonary arterial hypertension and veno-occlusive disease
- Hypoxic pulmonary vasoconstriction
- Peak end-expiratory pressure
- High alveolar pressure
- Acute respiratory distress syndrome
- Pulmonary fibrosis
- Sleep disordered breathing
- Chronic obstructive pulmonary disease

Arrhythmias

Ventricular tachyarrhythmias are often associated with cardiogenic shock. Furthermore, bradyarrhythmias may cause or aggravate shock due to another etiology. Sinus tachycardia

and atrial tachyarrhythmias contribute to hypoperfusion and aggravate shock.

Epidemiology

International occurrence

Several multicenter thrombolytic trials in Europe reported a prevalence rate of cardiogenic shock following MI of approximately 7%.

Race-, sex-, and age-related demographics

Race-stratified mortality rates from cardiogenic shock are as follows (race-based mortality differences disappear with revascularization):

- Hispanics - 74%
- African Americans - 65%
- Whites - 56%
- Asians/others - 41%

The overall incidence of cardiogenic shock is higher in men than in women, with females accounting for 42% of patients with cardiogenic shock. This difference results from the increased prevalence of coronary artery disease in males. However, a higher percentage of female patients with MI develop cardiogenic shock than do males with MI.

Median age for cardiogenic shock mirrors the bimodal distribution of disease. For adults, the median age ranges from 65-66 years. For children, cardiogenic shock presents as a consequence of fulminant myocarditis or congenital heart disease.

Prognosis

Cardiogenic shock is the leading cause of death in acute myocardial infarction (MI). In the absence of aggressive, highly experienced technical care, mortality rates among patients with cardiogenic shock are exceedingly high (up to 70-90%). The key to achieving a good outcome is rapid diagnosis, prompt supportive therapy, and expeditious coronary artery revascularization in patients with myocardial ischemia and infarction.

Morbidity and mortality

Complications of cardiogenic shock may include the following:

- Cardiopulmonary arrest
- Dysrhythmia
- Renal failure
- Multisystem organ failure
- Ventricular aneurysm
- Thromboembolic sequelae
- Stroke
- Death

The following predictors of mortality were identified from the Global Utilization of Streptokinase and Tissue-Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial-:

- Increasing age
- Prior MI
- Altered sensorium
- Cold, clammy skin
- Oliguria

Echocardiographic findings such as left ventricular ejection fraction and mitral regurgitation are independent predictors of mortality. An ejection fraction of less than 28% is associated with a survival rate of 24% at 1 year, compared with a survival rate of 56% with a higher ejection fraction. Moderate or severe mitral regurgitation was found to be associated with a 1-year survival rate of 31%, compared with a survival rate of 58% in patients with no regurgitation.

Outcomes in cardiogenic shock significantly improve only when rapid revascularization can be achieved. The SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock?) trial demonstrated that overall mortality when revascularization occurs is 38%. When rapid revascularization is not attempted, mortality rates approach 70%. Rates vary depending on the procedure (eg, percutaneous transluminal coronary angioplasty, stent placement, thrombolytic therapy).

Physical Examination

Characteristics of patients with cardiogenic shock include the following:

- Patients in shock usually appear ashen or cyanotic and have cool skin and mottled extremities
- Peripheral pulses are rapid and faint and may be irregular if arrhythmias are present
- Jugular venous distention and crackles in the lungs are usually (but not always) present; peripheral edema also may be present.
- Heart sounds are usually distant, and third and fourth heart sounds may be present
- The pulse pressure may be low, and patients are usually tachycardic
- Patients show signs of hypoperfusion, such as altered mental status and decreased urine output

A systolic murmur is generally heard in patients with acute mitral regurgitation or ventricular septal rupture. The associated parasternal thrill indicates the presence of a ventricular septal defect, whereas the murmur of mitral regurgitation may be limited to early systole.

The systolic murmur, which becomes louder upon Valsalva and prompt standing, suggests hypertrophic obstructive cardiomyopathy (idiopathic hypertropic subaortic stenosis).

Diagnostic Considerations

Conditions to consider in the differential diagnosis of cardiogenic shock include the following:

- Systemic inflammatory response syndrome
- Acute coronary syndrome
- Aortic regurgitation
- Dilated cardiomyopathy
- Restrictive cardiomyopathy
- Congestive heart failure and pulmonary edema
- Mitral regurgitation
- Pericarditis and cardiac tamponade
- Hypovolemic shock
- Papillary muscle rupture
- Acute valvular dysfunction

Right ventricular infarction

Right ventricular infarction occurs in up to 30% of patients with inferior myocardial infarction (MI) and becomes hemodynamically unstable in 10% of these patients. The diagnosis is made by identifying an ST-segment elevation in the right precordial leads (V₃ or V₄ R) and/or typical hemodynamic findings after right heart catheterization. These are elevated right atrial and right ventricular end-diastolic pressures with normal to low pulmonary artery wedge pressure and low cardiac output.

Echocardiography findings can also be very helpful in the diagnosis of right ventricular infarction. Patients with cardiogenic shock due to this condition have a better prognosis than do patients when compared to those with cardiogenic shock due to left ventricular systolic failure.

Regarding the management of cardiogenic shock due to right ventricular infarction, supportive therapy begins with the restoration and maintenance of right ventricular preload with fluid administration. However, excessive fluid resuscitation may compromise left ventricular filling by introducing an interventricular septal shift.

Inotropic therapy with dobutamine may be effective in increasing cardiac output in patients with right ventricular infarction. Maintenance of systemic arterial pressure in order to maintain adequate coronary artery perfusion may require vasoconstricting agents, such as norepinephrine. In unstable patients, an intra-aortic balloon pump (IABP) may be useful for ensuring adequate blood supply to the already compromised right ventricle.

Revascularization of the occluded coronary artery, preferably by percutaneous transluminal coronary angioplasty (PTCA), is crucial for management and has shown to dramatically improve outcome.

Acute mitral regurgitation

Acute mitral regurgitation is usually associated with inferior MI due to ischemia or infarction of the papillary muscle. It occurs in approximately 1% of MIs, and posteromedial papillary muscle is involved more frequently than anterolateral muscle. Acute mitral regurgitation usually happens 2-7 days following acute MI and manifests with an abrupt onset of pulmonary edema, hypotension, and cardiogenic shock.

Echocardiography findings are extremely useful in making a diagnosis. The 2-dimensional (2-D) echocardiographic image shows the malfunctioning mitral valve, and findings from a

Doppler study can be used to document the severity of mitral regurgitation. Right heart catheterization is often required for stabilizing the patient. Tall V waves identified on pulmonary arterial and wedge pressure waveforms indicate acute mitral regurgitation. However, the diagnosis must be confirmed based on echocardiography or left ventriculography findings before definitive therapy or surgery is initiated.

Hemodynamic stabilization by reducing afterload, either with nitroprusside or an IABP, is often instituted. Definitive therapy requires revascularization, if ischemia is present, and/or surgical valve repair or replacement, if a structural valvular lesion is present. The mortality rate in the presurgical era was 50% in the first 24 hours, with a 2-month survival rate of 6%.

Cardiac rupture

Rupture of the free wall of the left ventricle occurs within 2 weeks of the MI and may occur within the first 24 hours. The rupture may involve the anterior, posterior, or lateral wall of the ventricle.

Cardiac rupture often presents as sudden cardiac death. Premortem symptoms include chest pain, agitation, tachycardia, and hypotension. This diagnosis should be considered in patients with electromechanical dissociation who have a history of anginal pain. Patients rarely, if ever, survive cardiac rupture.

Ventricular septal rupture

Approximately 1-3% of acute MIs are associated with ventricular septal rupture. Most septal ruptures occur within the week following MI. Patients with acute ventricular septal rupture develop acute heart failure and/or cardiogenic shock, with physical findings of a harsh holosystolic murmur and left parasternal thrill.

A left-to-right intracardiac shunt, as demonstrated by a step-up (>5% increase in oxygen saturation) between the right atrium and right ventricle, confirms the diagnosis. Alternatively, 2-D and Doppler echocardiographic findings can be used to identify the location and severity of the left-to-right shunt.

Rapid stabilization using an IABP and pharmacologic measures, followed by emergent surgical repair, is lifesaving. The timing of surgical intervention is controversial, but most experts suggest operative repair within 48 hours of the rupture.

Ventricular septal rupture portends a poor prognosis unless management is aggressive. Immediate surgical repair of patients with ventricular septal rupture is reported to be associated with survival rates of 42-75%; therefore, prompt surgical therapy is imperative as soon as possible after the diagnosis of ventricular septal rupture is confirmed.

Reversible myocardial dysfunction

Other causes of severe, reversible myocardial dysfunction are sepsis-associated myocardial depression, myocardial depression following cardiopulmonary bypass, and inflammatory myocarditis. In older literature, this presentation is often referred to as cold septic shock. In these situations, myocardial dysfunction occurs from the effects of inflammatory cytokines, such as tumor necrosis factor and interleukin 1.

Myocardial dysfunction may vary from mild to severe and may lead to cardiogenic shock. For patients in cardiogenic shock, cardiovascular support with inotropic agents may be required until recovery, which generally occurs after the underlying disease process resolves.

Differential Diagnoses

- [Myocardial Infarction](#)
- [Myocardial Ischemia](#)
- [Myocardial Rupture](#)
- [Myocarditis](#)
- [Pulmonary Edema, Cardiogenic](#)
- [Pulmonary Embolism](#)
- [Sepsis, Bacterial](#)
- [Septic Shock](#)
- [Shock, Distributive](#)
- [Shock, Hemorrhagic](#)

Resuscitation, Ventilation, and Pharmacologic Intervention

Initial management includes fluid resuscitation to correct hypovolemia and hypotension, unless pulmonary edema is present. Central venous and arterial lines are often required. Swan-Ganz catheterization and continuous percutaneous oximetry are routine.

Oxygenation and airway protection are critical; intubation and mechanical ventilation are

commonly required. However, although positive pressure ventilation may improve oxygenation, it may also compromise venous return, preload, to the heart. In any event, the patient should be treated with high-flow oxygen. Studies in patients with acute cardiogenic pulmonary edema have shown noninvasive ventilation to improve hemodynamics and reduce the intubation rate. Mortality is, however, unaffected.

A study by Shin et al suggested that patients who receive extracorporeal cardiopulmonary resuscitation (CPR) versus conventional CPR for longer than 10 minutes following in-hospital arrest have a greater chance of survival. [\[12\]](#)

Pharmacologic therapy

Patients with myocardial infarction (MI) or acute coronary syndrome are given aspirin and heparin. Both of these medications have been shown to be effective in reducing mortality in separate studies. Before initiating therapy, however, care should be taken to ensure that the patient does not have a myocardial wall rupture that is amenable to surgery.

The glycoprotein IIb/IIIa inhibitors improve the outcome of patients with non-ST-segment elevation acute coronary syndrome (NSTACS). They have been found to reduce recurrent MI following percutaneous coronary intervention (PCI) and in cardiogenic shock.

Hemodynamic Support

Dopamine, norepinephrine, and epinephrine are vasoconstricting drugs that help to maintain adequate blood pressure during life-threatening hypotension and help to preserve perfusion pressure for optimizing flow in various organs. The mean blood pressure required for adequate splanchnic and renal perfusion (mean arterial pressure [MAP] of 60 or 65 mm Hg) is based on clinical indices of organ function.

In patients with inadequate tissue perfusion and adequate intravascular volume, initiation of inotropic and/or vasopressor drug therapy may be necessary. Dopamine increases myocardial contractility and supports the blood pressure; however, it may increase myocardial oxygen demand. Dobutamine may be preferable if the systolic blood pressure is higher than 80 mm Hg; it has the advantage of not affecting myocardial oxygen demand as much as dopamine does. However, the resulting tachycardia may preclude the use of this inotropic agent in some patients.

Dopamine is usually initiated at a rate of 5-10 mcg/kg/min intravenously, and the infusion rate is adjusted according to the blood pressure and other hemodynamic parameters. Often, patients may require high doses of dopamine (as much as 20 mcg/kg/min).

If the patient remains hypotensive despite moderate doses of dopamine, a direct vasoconstrictor (eg, norepinephrine) should be started at a dose of 0.5 mcg/kg/min and titrated to maintain an MAP of 60 mm Hg. The potent vasoconstrictors (eg, norepinephrine) have traditionally been avoided because of their adverse effects on cardiac output and renal perfusion.

Vasopressor supportive therapy

Dopamine

At doses of approximately 10 mcg/kg/min, alpha-adrenergic effects lead to arterial vasoconstriction and an elevation in blood pressure. The blood pressure increases primarily as a result of the inotropic effect. The undesirable effects are tachycardia and increased pulmonary shunting, as well as the potential for decreased splanchnic perfusion and increased pulmonary arterial wedge pressure.

Norepinephrine

The dose of norepinephrine may vary from 0.2-1.5 mcg/kg/min, and large doses, as high as 3.3 mcg/kg/min, have been used because of the alpha-receptor down-regulation in persons with sepsis.

Epinephrine

Administration of this agent is associated with an increase in systemic and regional lactate concentrations. The use of epinephrine is recommended only in patients who are unresponsive to traditional agents. The undesirable effects are an increase in lactate concentration, a potential to produce myocardial ischemia, the development of arrhythmias, and a reduction in splanchnic flow.

Inotropic supportive therapy

Dobutamine

In the setting of acute myocardial infarction (MI), dobutamine use could increase the size of the infarct because of the increase in myocardial oxygen consumption that may ensue. In general, avoid dobutamine in patients with moderate or severe hypotension (eg, systolic blood pressure < 80 mm Hg) because of the peripheral vasodilation.

Phosphodiesterase inhibitors

Phosphodiesterase inhibitors (PDIs), which include inamrinone (formerly amrinone) and milrinone, are inotropic agents with vasodilating properties and long half-lives. The hemodynamic properties of PDIs are (1) a positive inotropic effect on the myocardium and peripheral vasodilation (decreased afterload) and (2) a reduction in pulmonary vascular resistance (decreased preload).

PDIs are beneficial in persons with cardiac pump failure, but they may require concomitant vasopressor administration. Unlike catecholamine inotropes, these drugs are not dependent on adrenoceptor activity; therefore, patients are less likely to develop tolerance to these medications.

Thrombolytic Therapy

Although thrombolytic therapy (TT) reduces mortality rates in patients with acute myocardial infarction (MI), its benefits for patients with cardiogenic shock secondary to MI are disappointing. When used early in the course of MI, TT reduces the likelihood of subsequent development of cardiogenic shock after the initial event.

In the Gruppo Italiano Per lo Studio Della Streptokinase Nell'Infarto Miocardio trial, 30-day mortality rates were 69.9% in patients with cardiogenic shock who received streptokinase, compared to 70.1% in patients who received a placebo.

Similarly, other studies employing a tissue plasminogen activator did not show reductions in mortality rates from cardiogenic shock. Lower rates of reperfusion of the infarct-related artery in patients with cardiogenic shock may help to explain the disappointing results from TT. Other reasons for the decreased efficacy of TT are the existence of hemodynamic, mechanical, and metabolic causes of cardiogenic shock that are unaffected by TT.

Intra-Aortic Balloon Pump

The use of the IABP reduces systolic left ventricular afterload and augments diastolic coronary perfusion pressure, thereby increasing cardiac output and improving coronary artery blood flow. The IABP is effective for the initial stabilization of patients with cardiogenic shock. However, an IABP is not definitive therapy; the IABP stabilizes patients so that definitive diagnostic and therapeutic interventions can be performed. [\[18, 19\]](#)

The IABP also may be a useful adjunct to thrombolysis for initial stabilization and transfer of patients to a tertiary care facility. Some studies have shown lower mortality rates in patients with myocardial infarction (MI) and cardiogenic shock treated with an IABP and subsequent revascularization, as previously mentioned.

Complications may be documented in up to 30% of patients who undergo IABP therapy; these relate primarily to local vascular problems, embolism, infection, and hemolysis.

The impact of treatment with an IABP on long-term survival is controversial and depends on the patient's hemodynamic status and the etiology of the cardiogenic shock. Patient selection is the key issue; inserting the IABP early, rather than waiting until full-blown cardiogenic shock has developed, may result in clinical benefit.

Ramanathan et al found that rapid and complete reversal of systemic hypoperfusion with IABP counterpulsation in the SHOCK trial and SHOCK registry was independently associated with improved inhospital, 30-day, and 1-year survival, regardless of early revascularization. This suggests that complete reversal of systemic hypoperfusion with IABP counterpulsation is an important early prognostic feature.

In the IABP-SHOCK II study, 600 patients with cardiogenic shock complicating acute myocardial infarction were randomized to intraaortic balloon counterpulsation or no intraaortic balloon counterpulsation. All patients were expected to undergo early revascularization. Use of intraaortic balloon counterpulsation did not significantly reduce 30-day mortality in these patients.

Ventricular Assist Devices

In recent years, left ventricular assist devices (LVADs) capable of providing complete short-term hemodynamic support have been developed. The application of LVAD during reperfusion, after acute coronary occlusion, causes reduction of the left ventricular preload,

increases regional myocardial blood flow and lactate extraction, and improves general cardiac function. The LVAD makes it possible to maintain the collateral blood flow as a result of maintaining the cardiac output and aortic pressure, keeping wall tension low and reducing the extent of microvascular reperfusion injury.

A pooled analysis from 17 studies showed that the mean age of this group of patients with LVADs was 59.5 ± 4.5 years and that mean support duration was 146.2 ± 60.2 hours. In 78.5% of patients (range, 53.8-100%), adjunctive reperfusion therapy, mainly percutaneous transluminal coronary angioplasty (PTCA), was used. Mean weaning and survival rates were 58.5% (range, 46-75%) and 40% (range, 29-58%), respectively.

In any case, comparing studies is difficult because important data are usually missing, mean age of patients were different, and time to treatment is not standardized. Hemodynamic presentation seems to be worse compared with data reported in the SHOCK trial, with lower cardiac index, lower systolic aortic pressure, and higher serum lactates. Taking these considerations into account, LVAD support seems to give no survival improvement in patients with cardiogenic shock complicating acute myocardial infarction (MI), compared with early reperfusion alone or in combination with IABP.

However, LVADs as a bridging option for patients with cardiogenic shock must be considered cautiously and must be avoided in patients who are unlikely to survive or are not likely to be transplant candidates. Further investigations are required to better define indications, support modalities, and outcomes.

The indications for insertion of a ventricular assist device are controversial. Such an aggressive approach to support the circulatory system in cardiogenic shock is appropriate (1) after the failure of medical treatment and an IABP, (2) when the cause of cardiogenic shock is potentially reversible, or (3) if the device can be used as a bridging option.

Percutaneous Transluminal Coronary Angioplasty

The retrospective and prospective data favor aggressive mechanical revascularization in patients with cardiogenic shock secondary to myocardial infarction (MI).

Reestablishing blood flow in the infarct-related artery may improve left ventricular function and survival following MI. In acute MI, studies show that percutaneous transluminal coronary angioplasty (PTCA) can achieve adequate flow in 80-90% of patients, compared with 50-60%

of patients after thrombolytic therapy (TT).

Several retrospective clinical trials have shown that patients with cardiogenic shock due to myocardial ischemia benefitted (reduction in 30d mortality rates) when treated with angioplasty. A study of direct (primary) PTCA in patients with cardiogenic shock reported lower mortality rates in patients treated with angioplasty combined with the use of stents than in patients treat with medical therapy.

Coronary Artery Bypass Grafting

Critical left main artery disease and 3-vessel coronary artery disease are common findings in patients who develop cardiogenic shock. The potential contribution of ischemia in the noninfarcted zone contributes to the deterioration of already compromised myocardial function.

Coronary artery bypass grafting (CABG) in the setting of cardiogenic shock is generally associated with high surgical morbidity and mortality rates. Because the results of percutaneous interventions can be favorable, routine bypass surgery is often discouraged for these patients.

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Підготував Добродієв А.В.

Shock states

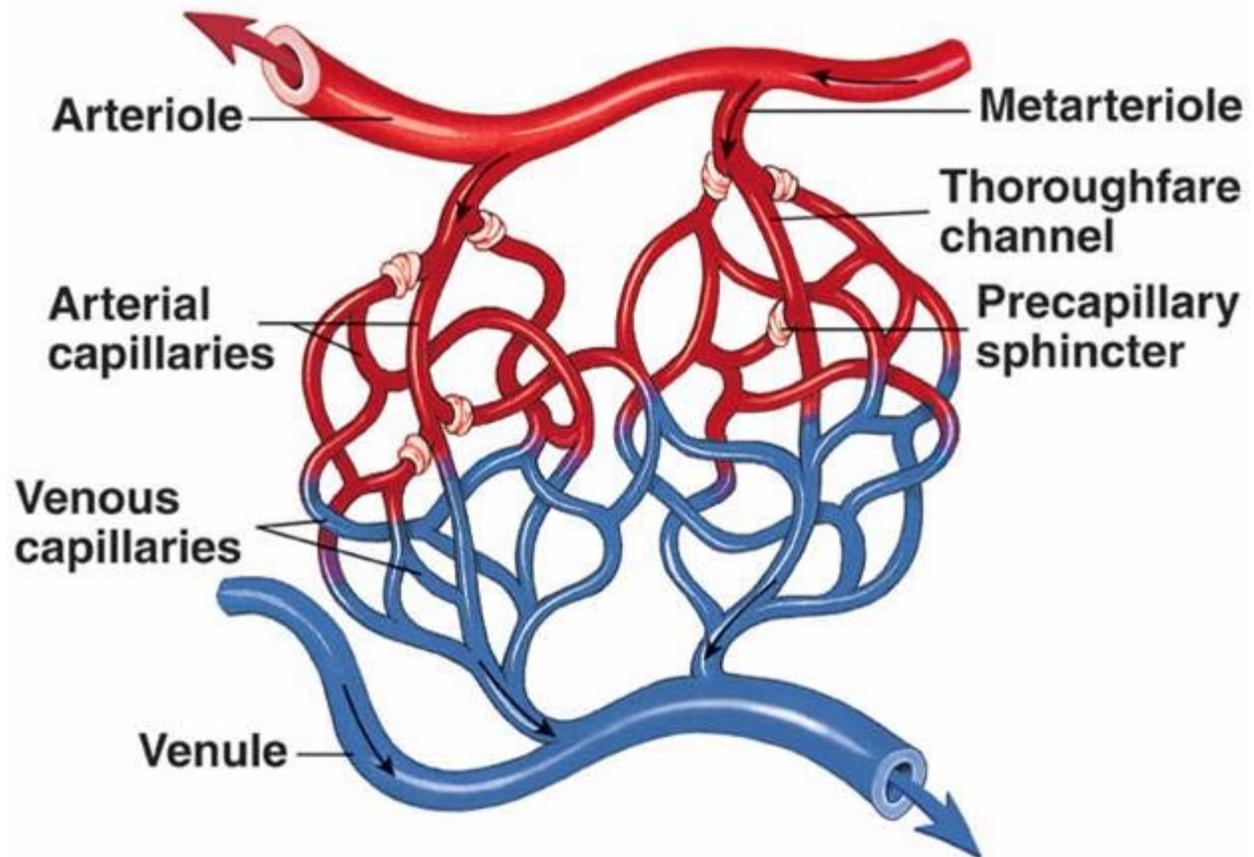
Acute circulatory insufficiency

4.1 Anatomy and physiology

Life is provided through a variety of mechanisms, however all of them depend on proper circulation. Circulation itself consists of 2 parts: work of heart (pump of the body) and vessels, through which blood is pumped to the most remote organs and tissues. During every systolic contraction heart pump 70-80 ml of blood (so called stroke volume). Thus in case of heart rate 70 beats per minute heart pumps nearly 5 liters of blood, what makes more than 7 tones per day.

From the left ventricle blood gets to the arterial system of the systemic circuit. Arteries contain 15% of the whole circulating blood volume; they carry blood from the heart to their distal departments – arterioles (vessels of resistance). Arterioles themselves are defining blood distribution: in condition of constriction (spasm) they make blood supply of the capillaries impossible (ischemia appears). On the contrary, in condition of dilatation they provide maximal oxygenation. When arterioles are blocked due to the spasm blood is flowing through the arterio-venous anastomosis directly to the venous system.

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Distribution of blood in the vascular bed (% of CBV).

- a. heart cavity 3%
- b. arteries 15%
- c. capillaries 12%
- d. venous system 70%

Among the natural vasoconstrictors (agents, which cause constriction of the blood vessel) are epinephrine, norepinephrine, serotonin, angiotensin II. Stress enhances the secretion of catecholamines, their blood concentration increases and arterioles constrict. Spasm of the arterioles is the basis of blood flow centralization: peripheral flow is disregarded in order to provide brain with the oxygenated blood as long as possible. To the group of vasodilators (agents, which provide dilatation of the vessels) belong “acid” metabolites (lactate, pyruvate, adenylic acid, inosinic acid), bradykinin, acetylcholine, different medicines (neuroleptics, α -adrenergic antagonists, peripheral vasodilators, ganglionic blocking agents, etc.), some exogenous poisons. All of them cause blood flow decentralization: opening of arterioles and distribution of the blood from central vessels to the capillary bed.

Capillaries are the interweaving network of the smallest body vessels with the general length of 90-100 thousands of kilometers. However simultaneously work only 20-25% of them. They provide metabolic exchange bringing oxygen and nutrients to the tissues and take back wastes of metabolism. Periodically, every 30-40 seconds one of them get closed and others open (vasomotion effect). Capillaries contain 12% of the whole circulating blood volume, but different pathological conditions can increase this amount even 3 and more times.

“Used” blood from the capillaries flows to the venous system. Veins are the blood reservoir, which contains 70% of the total circulating blood volume. Unlike arteries they are capable of volume control and thus they influence the amount of blood, which returns to the heart.

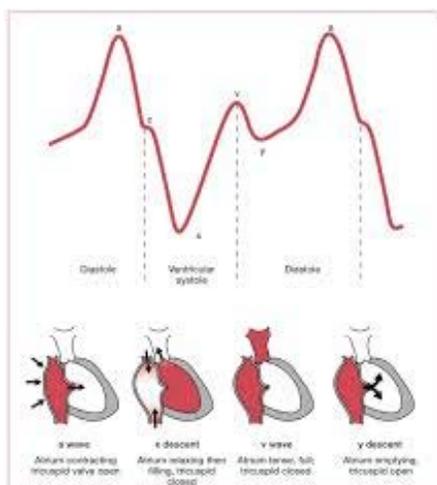
The most important haemodynamic index of venous system is central venous pressure. CVP represents the pressure which blood causes to the walls of cava veins and right atrium. This parameter is an integral index of circulating blood volume, systemic vascular resistance and pump function of the heart. It can be measure with a special device called “phlebotonometer” (pic. 4.9) or with a usual infusion set and a ruler. Normally CVP measured from the sternum point is 0-14 cm H₂O and from midaxillary line is 8-15 cm H₂O.

Central venous pressure decreases (sometimes even to negative) in case of:

- blood loss
- excessive water loss (dehydration)
- distributive shock (decrease of peripheral resistance due to venous and arterial

dilatation)

In those conditions decreases volume of blood returning to the heart and thus suffers cardiac output. In case of negative CVP cardiac arrest is highly probable.



Central venous pressure increases in case of:

- heart failure (insufficiency of left or right ventricle)
- hypervolemia (excessive blood infusion, improper infusion therapy)
- obstructions to blood flow (pulmonary embolism, cardiac tamponade, etc.)

When CVP over 15-16 cm H₂O is combined with left ventricle insufficiency the risk of pulmonary edema is very high.

Blood pressure is an integral index of arterial part of systemic haemodynamics. Talking about blood pressure we may refer to systolic, diastolic, pulse and mean arterial pressure. Systolic (P_{syst}) and diastolic (P_{diast}) pressures are measured with the manometer (method with the usage of phonendoscope was invented by M.Korotkoff). Pulse pressure (PP) is a difference between systolic and diastolic blood pressure.

Mean arterial pressure (MAP) is calculated according to the formula:

$$\text{MAP} = P_{\text{dias}} + 1/3 \text{ PP} \quad \text{mm Hg}$$

MAP defines the level of pressure necessary for the metabolic exchange in the tissues. Its measurement allows the evaluation of tissue perfusion level.

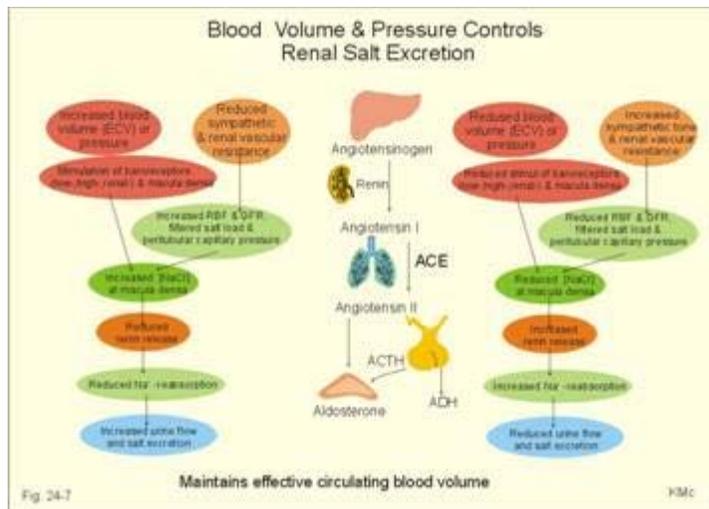
Blood pressure depends on different factors, but the most important are cardiac output and vascular resistance (mostly arterioles). This dependence is direct, thus you can increase blood pressure using:

- infusion of vasoconstrictors - solutions of

epinephrine, phenylephrine (mesaton), etc. (they will increase the vascular resistance);

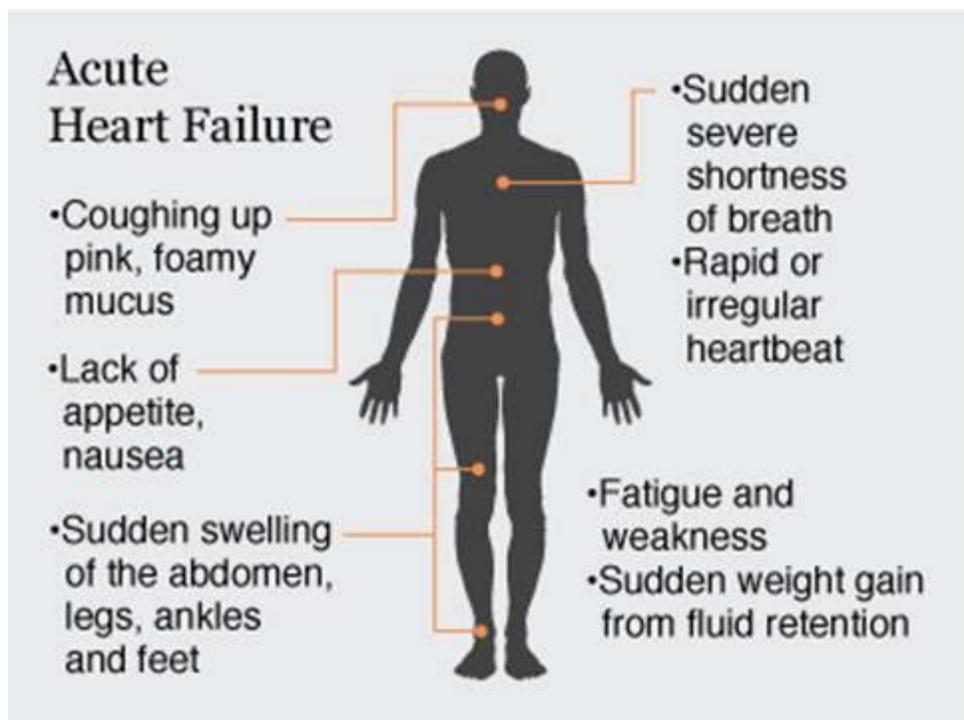
- infusion of hydroxyethyl starch solutions or saline (they will increase circulating blood volume)

- infusion of cardiac glycosides or other medicine which stimulate myocardium



General volume of blood in the body of a healthy adult is nearly 7% from the body weight: 70 ml per kilogram for male and 65 ml per kilogram of body weight for female. Of course circulating blood volume is lower, because part of blood is out of metabolic processes as a reserve. CBV can be measured with the infusion of coloring substance to the blood flow (Evans blue, polyglucin) and later evaluation of its dissolution degree.

Therefore measurement of CVP, BP, cardiac output and circulating blood volume allow to evaluate condition of circulation system of the patients and to provide adequate correction.



Acute heart failure; shock and collapse.

Acute cardiovascular failure is a state of cardiac and vascular inability to provide adequate supply of tissue metabolic needs with oxygenated blood and nutrients. This, earlier or later, causes cellular death.

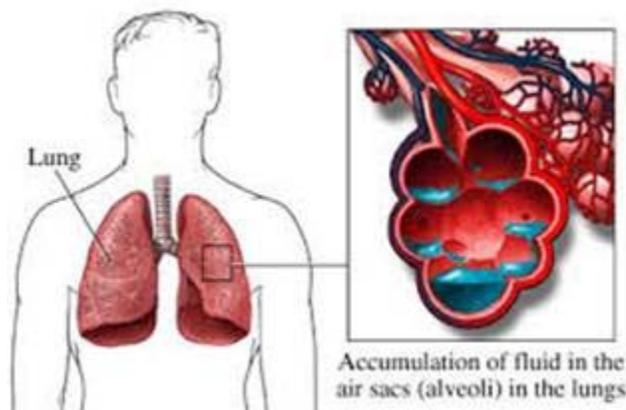
The reasons of the failure vary greatly: mechanic injuries, blood loss, burns, dehydration, exogenous and endogenous intoxications, immediate hypersensitivity reaction, ischemic heart disease, neural and humoral regulation disorders of vascular tone.

Acute cardiac failure is a disorder of heart pumping action. It develops due to primary heart problems or secondary, under the influence of extracardiac factors such as infection or intoxication. There are two types of heart failure: left-sided and right-sided.

Left-sided heart failure is an inability of left ventricle to pump blood from the pulmonary circuit to the systemic circuit. The most common reasons of it are myocardial infarction, mitral insufficiency, left AV valve stenosis, aortic valve stenosis, aortal insufficiency, hypertonic disease, coronary sclerosis, acute pneumonia.

Coronary circulation is possible only during the diastole and in those conditions every violation of coronary passability decreases cardiac output. This way during the systole part of the blood is not injected into aorta, but stays in the left ventricle. Diastolic pressure in the left ventricle increases and blood is literally forced to stagnate in the left atrium. At the same time right ventricle functions normally and continues to pump usual amounts of blood to the pulmonary circuit. Thus hydrostatic pressure in the vessels of pulmonary circulation increases, fluid part of the blood moves first to the lung tissue and then, through alveolar-capillary membrane, to the alveolar lumen.

Clinically pulmonary edema begins with dyspnea (during physical activity or rest). Later attacks of dyspnea are connected with persistent cough with white or pink blood-tinged phlegm. During the attack patient tries to sit as in this position breathing is easier. This condition is called "heart asthma". When hydrostatic pressure is over 150-200 mm Hg, fluid part of blood moves to the alveolar lumen causing development of pulmonary edema.

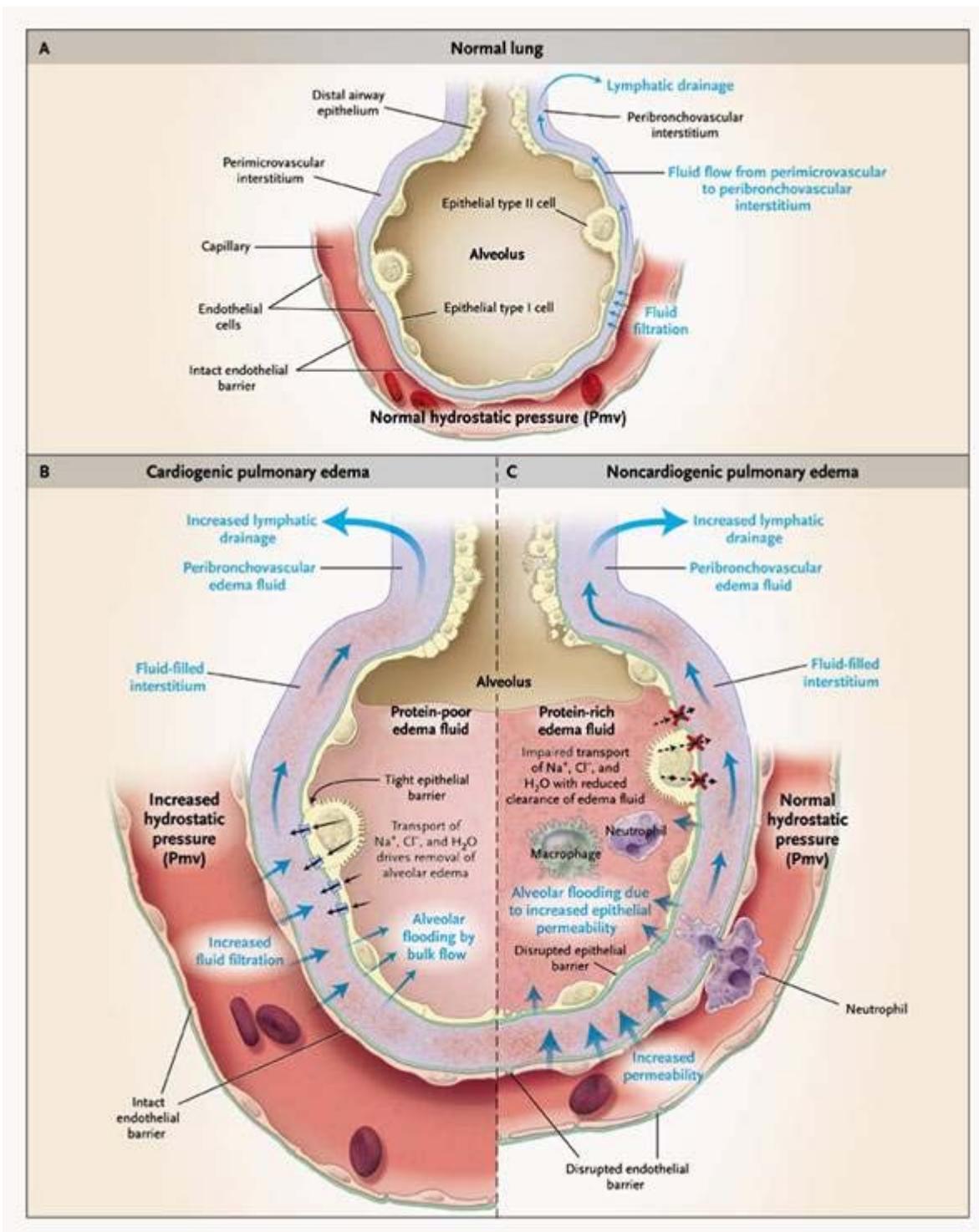


Pulmonary edema is divided into interstitial and alveolar edema.

Interstitial edema is a condition during which serous part of stagnated in the pulmonary circuit blood infiltrates the lung tissue, including peribronchial and perivascular spaces.

During alveolar edema not only the plasma, but also blood components (red and white blood cells, platelets) get out from the vessels. During the respiratory act blood mixes with the air creating large amount of “foam”, which violates gas exchange. This way, in addition to circulatory hypoxia, hypoxic hypoxia appears.

Condition of the patient gets worse quickly. Sitting position is optimal, but not as helping as previously. Respiratory rate is nearly 30-35 breathes per minute, but attacks of breathlessness are constant. Skin is pale with acrocyanosis. Hypoxia of central nervous system usually causes psychomotor agitation. Respiratory acts are noisy; during cough pink blood-tinged phlegm is released. Auscultation allows you to hear different wet rales, sometimes it's even possible to hear them standing aside the patient without phonendoscope.



Pulmonary edema can be also divided according to the blood pressure level: the one with elevated pressure is caused by a hypertonic disease, aorta valve insufficiency or disorders of cerebral perfusion; another one is caused by total myocardial infarction, acute inflammation of myocardial muscle, terminal valve defects, severe pneumonia and is characterized with normal or low blood pressure.

Immediate aid

- make sure patient is sitting with his legs down (orthopnea)
- provide oxygenation through nasal catheter (before placing oil it with

glycerin, insert it to the depth of 10-12 cm – distance from the wing of the nose to auricle) or face mask. Do not use Vaseline, because it can burn in atmosphere with high concentration of oxygen.

However if catheter is not deep enough patient will suffer from an unpleasant “burning” feeling, because oxygen flow will dry mucosa layer of the nasal cavity; also in this situation concentration of oxygen will be lower than expected.

- put venous tourniquets on the limbs in order to reduce amount of blood returning to heart: venous bed of limbs can reserve up to 1,7 liters of blood;
- constantly control heart and kidney activity (ECG, SaO₂ , and blood pressure are checked automatically trough the monitor; to control diuresis you should insert Foley catheter;
- catheterize central vein, because amount of infusions should be based on central venous pressure;
- use medical “defoamers” if they are available (ethyl alcohol or antiphomsylan solution) combined with oxygen inhalation

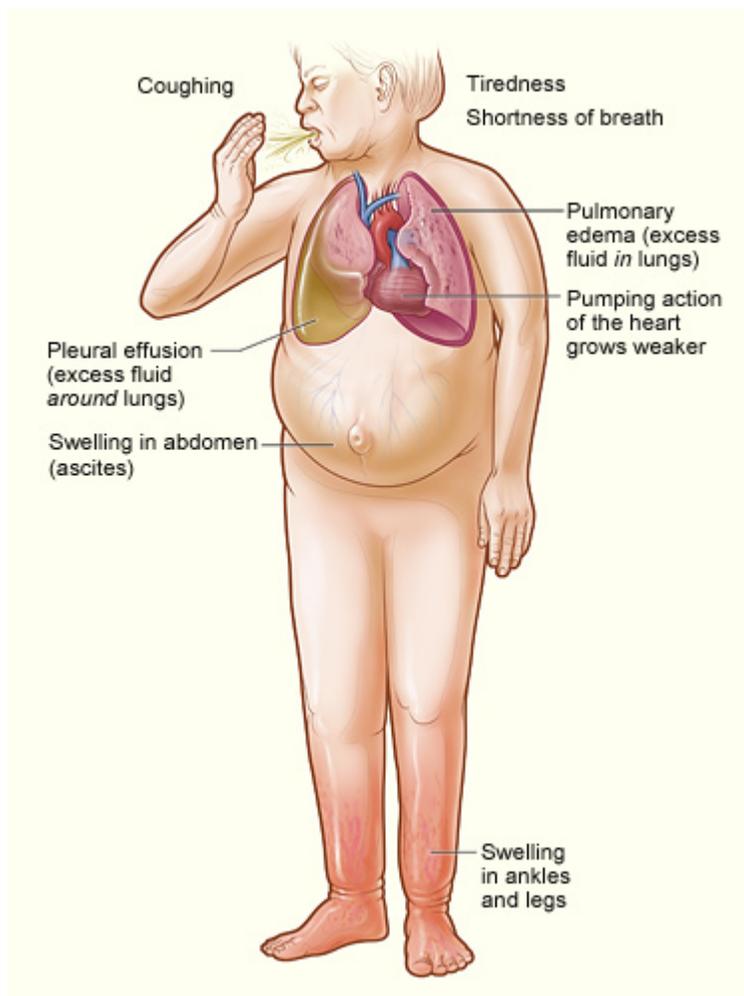
Scheme of oxygenation set connected to “defoamer” container

- a. oxygen source (cylinder with oxygen)
 - b. tube with numerous holes sunk into container with defoamer
 - c. tube for humidified oxygen (its opening should be over the level of fluid);
 - d. patient
- medical treatment: 1% morphine solution (decreases intravascular pressure of pulmonary circuit, inhibits respiration center in medulla oblongata preventive dyspnea progress, sedates patient);
 - solutions of diuretics are used to decrease the circulating blood volume (6-12 ml of 1% furosemid solution, solution of ethacrynic acid), however be careful with them in case of low blood pressure; diuretic effect will last up to 3 hours after i/v infusion, the expected diuresis is 2-3 liters
 - if blood pressure allows you can try to use nitroglycerin to reduce intravascular pressure of pulmonary circuit (1 or 2 tablets with 10 minutes interval)
 - cardiac glycosides for improvement of heart action (0,025% digoxin solution, 0,05% strophanthin solution, 0,06% corglicon solution);
 - in case of high pressure (over 150 mm Hg) use ganglionic blocking agents (1 ml of 5% pentamin solution diluted in 150 ml of saline, give i/v slowly; diluted with

saline 250 mg of trimethaphan solution), because they reduce pressure in pulmonary circuit and lower the amount of blood getting to right half of the heart, however be careful with the dosage and monitor blood pressure level carefully;

- never use osmotic diuretics in case of pulmonary edema – they will increase blood volume and thus heart load!!!

- when everything listed above failed and patient is worsening with every second you should intubate him and start artificial ventilation with positive end expiratory pressure (begin with 4-6 cm H₂O)



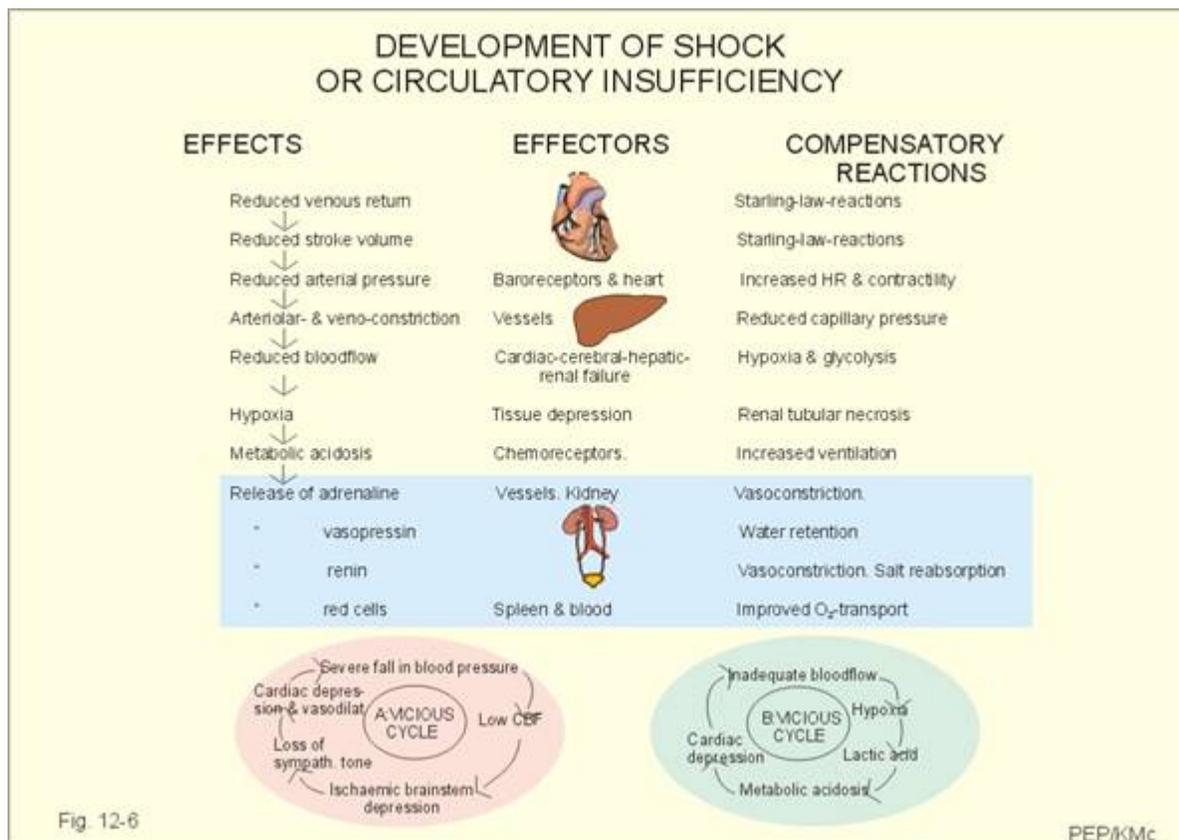
Right-sided heart failure is an inability of right ventricular to pump blood from systemic circuit to the pulmonary circuit due to its weakness or an obstruction to the blood flow.

It occurs in case of pulmonary embolism, right ventricular infarction, excessive infusion therapy (especially including citrated blood) for patients with heart insufficiency, lung diseases (bronchial asthma, emphysema, pneumosclerosis) which cause increase of right ventricular load.

Patients have acrocyanosis, tachycardia, dyspnea, pronounced neck veins, ankle swelling, enlarged liver, ascytis. Central venous pressure is highly increased (up to 20-25 cm H₂O), however pulmonary edema does not appear.

Intensive treatment is mostly pathogenetic:

- limit the infusions (give only life-necessary solutions, check the water balance of the patient and reduce drinking water if necessary);
- in case of citrated blood transfusions use 5-10 ml of 10% calcium gluconate solution per every 500 ml of blood to prevent hypocalcaemia;
- in case of bronchial spasm use bronchial spasmolytics;
- to remove excessive fluid from the body use diuretics (furosemide solution for example);
- metabolic acidosis is corrected with 4% solution of sodium bicarbonate (i/v slowly with acid-base state control);
- in case of pulmonary embolism anticoagulants are used – fraxiparine 0,6 mg subcutaneously; heparin solution – 5000 IU every 4 hours; fibrinolytic drugs (streptokinase, fibrinolysin, urokinase, etc.)



Shock is a pathological state which can be described as a tissue hypoxia caused by hypoperfusion. Pathogenetic basis of shock depends on its reason (trauma, toxins, thermal injury) and at the same time on reactivity of the organism (level of defense

mechanisms mobilization).

Stimulation of sympathetic nervous system - production of catecholamines and other vasoactive substances by hypothalamus and adrenal glands are the universal response of the body to the stress. Those mediators interact with the receptors of peripheral vessels causing their constriction and at the same time they dilate the vascular bed of life-important organs. This is so called “centralization of the flow”: rational decrease of blood flow in less important tissues (skin, organs of abdominal cavity, kidneys) in case of aggressive external influence for protecting life itself (brain, heart, lungs).

However influence of shock agents (pain, hypovolemia, destroyed cells, toxic metabolites), extended microcirculation violations (vascular spasm, microthrombosis and sludge) and caused by them tissue ischemia lead to hypoxic affection and cellular death of the internal organs. Further it can bring multiple organ dysfunction syndrome.

Collapse is a vascular failure. It occurs when body is not able to provide blood flow according to the new level of its needs (either because reaction is not fast enough or because sympathetic activation fails). Vascular bed volume and circulating blood volume are disproportional: too much blood gets to the microcirculation vascular reserve and the amount, which returns to the heart is not enough for the systemic needs (so called “decentralization” of the blood flow). Cardiac output and blood pressure decrease, that causes hypoperfusion of the central nervous system and thus unconsciousness and life-threatening complications.

Collapse definition is a bit nominal, because if such reaction extends in time the state of shock develops. Shock itself can equally run as a vascular failure or as a sudden clinical death.

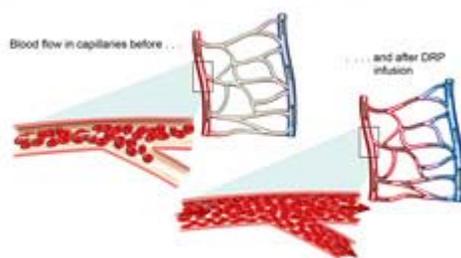
Pathogenetic classification of shock (according to P. Marino, 1998):

- hypovolemic
- cardiogenic
- distributive
- mixed (two and more factors).

Clinical classification of shock:

- traumatic shock;
- haemorrhagic shock;

- dehydration shock;
- burn shock;
- septic shock;
- anaphylactic shock;
- cardiogenic shock;
- exotoxic shock.



Traumatic and haemorrhagic shocks.

Traumatic and haemorrhagic shocks appear in case of sudden severe injuries of the body. Clinical symptoms greatly depend on the initial reason, extent of the tissue damage, blood loss volume, pain intensity, duration of pathological state and type of the body-reaction.

At the beginning patients with trauma are agitated, restless, their complaints (mostly they suffer from pain in the injured area) are numerous and intense. Further, while shock is progressing, you can observe inhibition of the central nervous system up to sopor and coma development. Breathing is rapid and deep, additional muscles participate respiratory acts. When condition of the central nervous system worsens respiration becomes shallow and weak. Comatose patients (if not helped on time) usually suffer from obstructive airways problems (fall of the tongue and soft palate, accumulation of sputum, blood, gastric contents). Skin becomes cyanotic and later mottled (especially noticeable those changes are under the knees and on hips).

In case of great blood loss (over 30% of CBV) skin is pale and clammy. Microcirculation disorders decrease the temperature of the peripheral tissues and difference between rectal and skin temperature is over 3 degrees Celsius. Another important symptom to check is capillary refill: if its over 2 seconds tissue microcirculation is violated (press with your fingertip the nail of the patient for 5 seconds and then check how long this white spot will remain).

Blood loss and tissue fluid loss decrease the subcutaneous turgor, eyeballs tone, blood filling of the subcutaneous veins. Peripheral pulse is weak, rapid and thready; blood pressure and central venous pressure are low. Hypotension and compensatory spasm of renal arteries decreases urine output through reduction of the renal blood flow. This brings state of oliguria-condition in which urine output is less than 0,5 ml per kilogram of body weight per hour.

Classification of blood loss (according to P. Marino, 1998, USA).

Class	Clinical symptoms	Blood loss volume (% of CBV)
I	Orthostatic tachycardia	15
II	Orthostatic hypotension	15-30
III	Low blood pressure in supine position, oliguria	30-40
IV	Consciousness disorders, coma	over 40

In case of traumatic shock erectile and torpid phases are described. Erectile phase is not obligatory: it begins during pre-hospital stage and doesn't last long (minutes). This phase is characterized with agitation, restlessness and stimulation of all vital systems. Torpid phase is much more dangerous, because patients suffer from CNS disorders and their lifesaving systems are as close to breakdown as possible.

To calculate the actual blood loss and thus to know class of shock you can use Allgower index (shock index).

It is a correlation between heart rate and systolic blood pressure:

$$AI = HR / P_{syst.}$$

Normally its value is 0,5-0,7 (for example in case of HR 60 and P_{syst} 120 mm Hg it will be 0,5). Shock conditions causes lowering of blood pressure which is compensated through tachycardia and thus increase of shock index.

Evaluation of blood loss according to shock index.

Shock index	Blood loss volume (% of CBV)	Stage of shock
>1	less than 20	1
1,1-1,7	20-40	2
<1,8	over 40	3

This method of blood loss evaluation is simple and allows quick evaluation of blood loss and its immediate adequate concentration of circulating blood volume. Perhaps you will better understand those calculations using this example:

patient weights 80 kilograms, his blood pressure is 80/50 mm Hg, HR 120 per minute. His perfect circulating blood volume is 70 ml per kilogram: $70 \times 80 = 5600$ ml.

$$SI = 120/80 = 1,5.$$

According to the table above he has 2 stage of shock and he lost nearly 30% (2 stage means from 20% to 40%, lets take 30%) from his CBV. So you will have to compensate 30% from 5 600 ml or: $5600 \times 0,3 = 1680$ ml of blood.

Until shock is not liquidated, even if the bleeding is stopped, lowering of CBL continues (so called relative blood loss, caused by stagnation, aggregation and sequestration of red blood cells in the microcirculation vessels).

	<u>Class I</u>	<u>Class II</u>	<u>Class III</u>	<u>Class IV</u>
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	> 100	> 120	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Decreased	Decreased	Decreased
Resp. Rate	14 – 20	20 – 30	30 – 40	> 40
UOP	> 30	20 – 30	5 – 15	negligible
Mental Status	sl. Anxious	mildly anx	confused	lethargic
Fluid	crystalloid	crystalloid	blood	blood

During shock compensatory mechanisms of the organism try to fill the vascular bed with whatever fluid they can; this includes water from intercellular space, which causes so

called “blood dilution”. The last one is detected in laboratory tests, which show decrease of blood concentration: subnormal indexes of hematocrit, hemoglobin, red blood cells and protein. This mechanism begins within first few minutes after the injury. However final evaluation of blood concentration indexes is possible only 12-24 hours after the initial trauma.

Profuse acute bleeding (over 30% of CBV during 1 hour) is life threatening. In case of chronic anemia organism is used to hemoglobin deficiency and hemorrhagic lack of even 70% of red blood cells and 30% of plasma might be acceptable (of course it does not mean, that you should not treat the bleeding or blood loss).

CHART 37-3

BEST PRACTICE for
The Client in Hypovolemic Shock

- Ensure a patent airway.
- Start an IV catheter or maintain an established catheter.
- Administer oxygen.
- Elevate the client's feet, keeping his or her head flat or elevated to a 30-degree angle.
- Examine the client for overt bleeding.
- If overt bleeding is present, apply direct pressure to the site.
- Take the client's vital signs every 5 minutes until stable.
- Administer medications as ordered.
- Increase the rate of IV fluid delivery.
- Do not leave the client.

Immediate aid.

First hour of shock is called “golden” because of its prognostic value and great therapeutic possibilities so don't waste even minute of it!

- first of all stop arterial bleeding (press artery to the bone, use special devices like tourniquets or tamponade the wound); be careful with the time of compression, because prolonged total block of blood flow will cause necrosis;
- constantly evaluate patient's condition (pulse over peripheral and central arteries, consciousness, airways patency, effectiveness of external breathing);
- if patient is unconscious and you can't control his airways all the time use the recovery position (three-quarters prone position); be careful with the neck of the patient if you suspect backbone injuries – those patients should be placed in supine position on the firm surface with immobilized neck; patients with pelvic fractures should lay on back with flexed knees;
- immobilize injured limbs with standard splints or improvised material;
- put on the wounds aseptic dressings; bandage is all you need to stop

capillary and venous bleeding; in case of pneumothorax use special Asherman seal or just a three-sided airtight dressing;

- use pain killers: either narcotic analgesics (1% promedol solution) or non-steroidal anti-inflammatory drugs like 50% analgin solution (depends on the pain intensity); patients with shock are unstable, so after giving of analgesics be very attentive to the changes in patient's condition, because pain was sympathetic system stimulator and without it blood pressure will probably lower and respiratory center might be inhibited; if you can't control the state of the patient (too many victims, dangerous environment, transportation) try to use ketamine (2-3 ml of 5% solution, i/m) instead of narcotic drugs: it has analgesic properties, however it will not influence blood pressure or breathing (but to avoid hallucinations use it with 1-2 ml of 5% diazepam solution); local anesthetic infiltration of tissues around the fracture also can be used to reduce pain (10-20 ml of 0,5% Novocain solution);

General principles of intensive trauma treatment

Constantly check the condition of main vital systems, using additional technical and laboratory possibilities. In order to implement such control you should remember about the rule of "4 catheters" for the patients with severe injuries: endotracheal tube (to provide optimal respiration and airways patency control), central venous catheter (for CVP monitoring and infusions), stomach probe (to evacuate its contents), urinary catheter (to control urine output).

Transfusion scheme for patients with blood loss (according to P. Brusov, 1997).

Level of blood compensation	Blood loss (% of CBV)	Total transfusion volume (in % of blood loss volume)	Infusion components
I	less than 10	200-300	Crystalloids
II	less than 20	200	Colloids and crystalloids
III	21-40	180	Packed red blood cells, albumin solution, colloids, crystalloids (0,3+0,1+0,3+0,3)

Patient in critical condition with an urgent need of surgical treatment should be taken to the operating room immediately: in their case intensive therapy of shock will be conducted directly on the operating table during surgery (of course general anesthesia is necessary).

In case of every open wound take care about tetanus prevention (antitetanic immunization according to the scheme).

Shock caused by dehydration

It is a type of hypovolemic shock, which occurs during excessive body fluid loss (not blood, because hemorrhagic shock is another shock type).

Its reasons vary greatly:

- gastrointestinal diseases (profuse vomiting, diarrhea, loss of intestinal fluid through fistula);
- polyuria (uncontrolled diuretic treatment, diabetes mellitus and insipidus, diuretic phase of acute renal failure);
- fluid loss through skin and wound surface (burns, high fever);
- inadequate infusion treatment of postoperative or comatose patients;
- hyperventilation (rapid breathing, Kussmaul breathing, inadequate artificial ventilation parameters in case of apparatus without air humidification).

However not only the complete fluid loss can be the reason of shock, but also it's pathological distribution into the extracellular space (intestinal cavity in case of intestinal paralysis, abdominal cavity in case of ascites, pleural cavity in case of pleurisy). This way will can also act prolonged heavy tissue inflammations (peritonitis) or massive injuries (crush-syndrome).

In cases described above electrolytes are also lost (cations of sodium, potassium, calcium, magnesium; anions of chlorine, hydrocarbonate). It causes complex osmolar, acid-base and electrolytic disorders.

Stage of dehydration shock is evaluated according to the actual fluid loss:

less than 5% of body weight – mild dehydration

5-10% of body weight – moderate dehydration

over 10% of body weight – severe dehydration

Water deficiency brings lowering of cardiac output, blood pressure and central venous pressure (through decrease of blood volume returning to the heart, which leads to compensatory adrenergic vasoconstriction).

Dehydration causes body weight loss, skin and mucosa dryness, decrease of subcutaneous turgor and eyeballs tone, hypothermia, tachycardia, oliguria, thirst. While dehydration progresses compensatory mechanisms weaken and central nervous system suffers: patients become sluggish, confused; hallucinations, cramps and unconsciousness are also possible. Laboratory tests show blood concentration.

One of the most important things in treatment of dehydrated patients is daily balance of fluid: check it carefully through measuring of daily received and lost fluids (food, infusions, stool and urine output). In case of fever or tachypnea make necessary corrections. Balance should be calculated every 12-24 hours (special paper forms make this easier).

Daily fluid balance is calculated by adding all the received fluids (both enteral and parenteral ways) and deducting urine output, stool, perspiration and breathing water loss.

You should remember, that perspiration depends on body temperature: in case of normal temperature (36,6°C) patient loses 0,5 ml/kg of water during every hour; 1 degree of temperature elevation adds 0,25 ml/kg to normal value of 0,5 ml/kg.

According to the fluid balance infusion therapy is divided into positive (for dehydrated patients), negative (for overhydrated patients) and “zero” (for patients without balance disorders).

Water deficiency is calculated according to the formula:

$$W_{\text{def}} = (Ht_p - Ht_n) * 0,2 \text{ BW} / Ht_n,$$

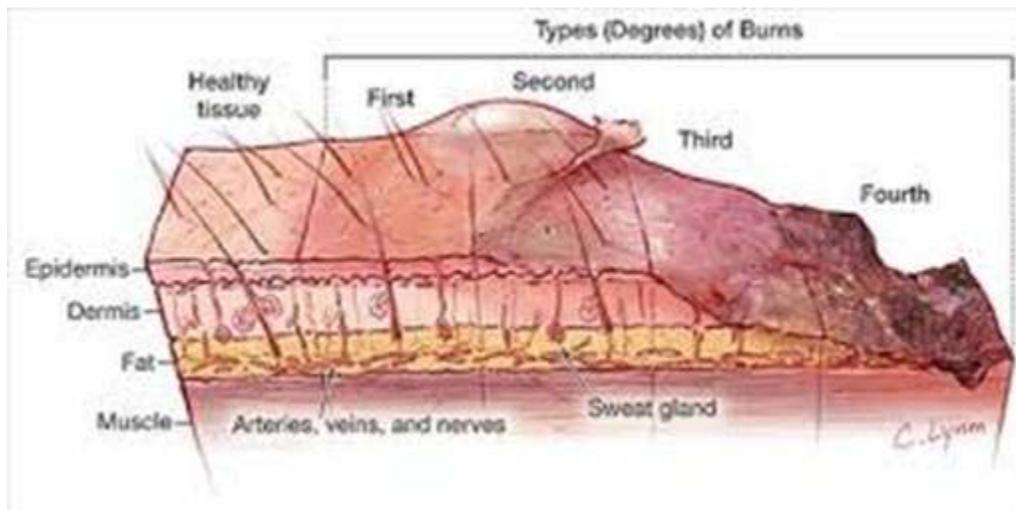
W_{def} – water deficiency, l;

Ht_p – hematocrit of the patient, l/l;

Ht_n - normal hematocrit, l/l;

BW – body weight, kg.

Use crystalloids to treat water deficiency: saline solution, Ringer’s solution, Ringer-lactate solution, electrolytic solutions, 5%, 10, 20% glucose solution. To control potassium concentration (during dehydration this cation is widely lost) prescribe polarizing GIK mixture (pic.9.4), but don’t you ever infuse concentrated potassium solutions quickly – it can cause cardiac arrest (not more than 400 of GIK solution ml per hour).



Burn shock.

Burn shock is a type of hypovolemic shock, caused by thermal or chemical agents.

It appears in adults in case if burns cover over 10-15% of the body surface. Huge plasma loss and pain stimulation of sympathetic nervous system bring hypovolemia, blood flow centralization and low cardiac output syndrome.

At the beginning patients complain of pain, nausea, vomiting, and thirst. Erectile phase of shock changes into torpid, central nervous system after stimulation becomes inhibited (stage depends on burn surface). Breathing and heart rate are increased, blood pressure is decreased. Undamaged skin is cold and mottled. If burning area covers airways asphyxia is highly possible.

Laboratory tests show concentration of the blood.

Immediate aid.

As soon as possible eliminate thermal or chemical agent (cover the fire with wet tissue, wash off chemicals with cold water). Generally use cold water (snow if you don't have an alternative) as cooling agent (not oils, not other organic substances). Take off smoldering clothes, cut them if necessary (don't try to take off something what is "soldered" with the skin by fire). Put a sterile bandage on burn areas. You should use general principles of shock treatment, including rule of 4 "catheters": central venous catheter, urinary catheter, stomach probe and endotracheal tube or nasal catheter for oxygenation – depending on condition of the patient. Take care about the proper pain relieving: prescribe narcotic analgesics (2 ml of 2% promedol solution), neuroleptics (2-3 ml of 0,25% droperidol solution). Infusion therapy is provided with crystalloids, colloids and glucose solutions (in the ratio 1:1:1 in case of mild shock, 2:1:1 in case of severe shock). Constantly monitor condition of the patient: monitor heart rate, blood pressure, central venous pressure and diuresis (make sure its not lower than 0,5 ml/kg per hour). When you suspect a thermal airways injury always intubate the patient: you will not have much time in case if edema will bring asphyxia.

Patients should be treated in special burn centers.

Anaphylactic Shock

Signs and symptoms of Anaphylaxis

Causes:

- Food
- Medication (antibiotics)
- Venom from animals (bugs, snakes, etc.)
- Allergic reactions

Treatment:

- EpiPen (Epinephrine)
- Histamine (pharmaceutical drug)
- 911

How to Prevent it:
The most common cause of this kind of shock are allergic reactions. To counteract the severity of the reaction in the future, immunotherapy is the way to go. The person is gradually vaccinated with progressively larger doses of the allergen, small enough so that the body's immune system can counteract it on its own, conditioning the body.

Glogster by Nick Termini and Zack Assenmacher

Anaphylactic shock

Anaphylactic shock is a type of distributive shock, caused by allergen induced immediate hypersensitivity reaction.

Various factors can cause anaphylactic reaction: serums, antibiotics, blood preparations, local anesthetics (Novocain), hypnotics (Sodium thiopental, Diazepam), radiocontrast agents, etc. Among non-medical allergens most common are: alimentary substances, chemical cleaning agents, cosmetics, insect poisons, fur, etc.

The main condition of anaphylactic reaction is presence of antibodies in previously sensitized organism. Antigen-antibody reaction reveals bioactive substances, such as histamine, bradykinin, slow-reacting substance of anaphylaxis. Those mediators cause vasodilatation and leakage of fluid from vessels into the tissues. The amount of blood returning to the heart decreases greatly, cardiac output reduces and thus blood pressure is lowering until the cardiac arrest appears (usually PEA).

Form of the shock depends on the time of reaction:

- fulminant form – develops within 10 minutes;
- immediate form – pre-shock phase continues 30-40 minutes;

- slow- shock appears within few hours;

Fulminant form can pass without warning signs or with them: the most common are felling of sudden hit, generalized hives and itching, pulsing headache, fear and unconsciousness.

Immediate and slow forms always have precursors (initial warning symptoms) and can develop in different types:

- skin type: itching, generalized hives, flushing;
- cerebral type: intensive headache, nausea, blindness, unconsciousness, cramps;
- asthmatic type: stridor (secondary to swelling of the throat, larynx and trachea), wheezes (caused by spasms of bronchial muscles);
- coronary type: chest pain, hypotension;
- abdominal type: crampy abdominal pain, nausea, vomiting, bloating.

Shock itself is characterized by hypotension (collapse), unconsciousness and breathing disorders. To prevent anaphylaxis you should always remember about importance of detailed allergic history of the patient and proper drug administration (local allergy tests of serums and antibiotics, Bezredko's scheme for serum injections).

In every department of any hospital should be prepared first aid kit for patients with anaphylaxis.

This kit should include:

1. 0,1% adrenaline solution (3-4 ampoules).
2. Infusion set (2) and 2, 5,10 ml syringes (2-4 items of each).
3. Saline solution 40 ml (2 bottles).
4. Polyglucin or refortan (1-2 bottles).
5. 0,5% dopamine solution 5 ml (4-6 ampoules).
6. 3% prednisolone solution (3-5 ampoules).
7. 10% calcium chloride solution (2 ampoules).
8. 2,4% ephyllin solution – 10 ml (2 ampoules).
9. Ambu bag.
10. Venous tourniquet.

11. Antihistamine solutions: 1 ml of 1% dimedrol solution (3-4 ampoules), 2 ml of 2,5% pipolfen solution (2-3 ampoules).

First aid algorithm in case of anaphylactic shock.

Evaluate patient's condition: check pulse, warning signs (precursors).

Immediately liquidate the source of the allergen, which caused anaphylaxis. Lay patient into supine position (head down, legs up). Don't you ever try to take out i/v catheter even if you know for sure, that substance in the infusion set caused anaphylaxis. Separate the set and the catheter, take some blood from the vein and than wash the catheter again, but don't remove it (at least until you don't have the new one). Time is not on your side and you might not be lucky enough to find another vein for infusions. If there was no i/v access – get one.

Give intravenously 0,5-1 ml of adrenaline solution diluted in 10 ml of saline. Check again patient's condition: consciousness, blood pressure, heart rate, and breathing. Start colloid and crystalloid infusions mixed with dopamine (refortan or stabizol with 20 ml of 0,5% dopamine solution). Give bolus of 60-90 mgprednisolone solution (2-3 ml of 3% solution). To improve airway patency you can use euphyllin (10 ml of 2,4% solution). If you notice breathing difficulties (problems with airways or respiration itself) act like in general standards – clean oral cavity and provide airway patency by opening the mouth, titling the head back and thrusting the jaw forward. Use Ambu bag if necessary and respiratory apparatus if available. Cardiac arrest requires immediate CPR.

In case of fulminant anaphylactic reaction only your calm and quick actions will save the patient: call for help, but not leave the patient alone (ask witnesses to help). Stop antigen admission (stop infusion without taking out the catheter). Start colloid and crystalloid infusions, give adrenaline and steroids. Be careful with antihistamine drugs (suprastine, dimedrol, pipolfen) when blood pressure is not yet stable, as they can deepen the hypotension.

Try to minimize the dose, which already is in the tissues: put a tourniquet over a place of injection, use local adrenaline injections around the place of antigen entrance, however those actions are not your main goal and should be provided after stabilization of the patient. Not the amount of the antigen causes shock, but the intense reaction of the organism.

Septic shock

Septic shock is a type of distributive shock, caused by generalized reaction of the organism to microorganisms and their toxins. Its etiology can be connected with

gram-positive and gram-negative bacteria, rickettsia or fungi and any mucosa can be their entrance (intestine, abdominal cavity, urinary tracts, woundsurface). Sepsis has its terminology:

SIRS – systemic inflammatory response syndrome – is a systemic answer of organism to the intensive stimuli (infection, injury, operation, etc.)

When the toxins get to the blood flow tissue macrophages answer their appearance with production of cytokines. Cytokines are mediators of systemic inflammatory response; they trigger a whole number of immune reactions (both humoral and cellular immune response). This bring to circulation and microcirculation disorders and thus to tissue hypoperfusion and tissue anoxemia. Lethality in this type of shock is 60-80%.

Disorders of circulation can be both hyperdynamic and hypodynamic. Hyperdynamic syndrome is characterized with high cardiac output and uninterrupted microcirculation. Hypodynamic reaction appears in case of low cardiac output connected with high vascular resistance; during terminal phase of shock vascular resistance drops drastically and hypotension becomes incurable.

Sepsis has its own terminology. We diagnose SIRS if two of the symptoms listed below are present:

- fever over 38°C or temperature less than 36°C;
- heart rate over 90 pre minute;
- respiration rate over 20 per minute or PaCO₂ < 32 mm HG;
- leucocytosis over $12 \cdot 10^9$ or less than $4 \cdot 10^9$ or more than 10% of young forms

Sepsis itself we state if SIRS is combined with source of infection. Severe sepsis is a sepsis connected with multiple organ dysfunction of two and more systems (for example oliguria and encephalopathy).

Components of multiple organ failure are: septic encephalopathy, polyneuropathy of terminal stages, acute respiratory distress-syndrome, septic shock, ileus and bacterial translocation, renal failure, acute tubular necrosis, DIC syndrome.

Signs of tissue dysfunction:

- arterial hypoxemia (PaO₂ / FiO₂ <300);
- acute oliguria (diuresis less than 0,5 ml/kg per hour);
- creatinine concentration over 0,5 mg/dl;
- activated partial thromboplastin time over 60 seconds;

- enteroparesis;
- thrombocytopenia (less than $100 \cdot 10^9$);
- hyperbilirubinemia (general bilirubin over 4 mg/dl or 70 $\mu\text{mol/l}$).

In those terms septic shock is defined as a condition of tissue and organ hypoperfusion caused by arterial hypotension, which can't be cured with infusions without catecholamines.

Principles of septic shock intensive treatment.

During first 6 hours your main goal is to stabilize the patient:

- central venous pressure should be 8-12 mm Hg (in case of patients with artificial ventilation: 12-15 mm Hg);
- mean arterial pressure > 65 mm Hg;
- diuresis $> 0,5$ ml/kg/hour;
- $\text{SvO}_2 > 70\%$.

1. Provide adequate infusion therapy. Volume of the infusion should be calculated according to the fluid balance and vital indexes (blood pressure, central venous pressure, heart rate). If you state hypotension give 500-1000 ml of crystalloids or 300-500 ml of colloids in 30 minutes; if necessary – repeat with the same volumes (until blood pressure is normal or you notice signs of hyperhydration). There is no evidence that colloids are more useful in this situation.

2. Vasopressors are used if hypotension is resistant to infusion therapy:

- dopamine solution 5-20 $\mu\text{g/kg/min}$
- norepinephrine 0,2-1,0 $\mu\text{g/kg/min}$
- hypophamine 0,01-0,04 U/min in case if dopamine and norepinephrine are ineffective.

3. Empirical antibiotic therapy should be started within first hour of treatment (actually from the moment you diagnosed severe sepsis or septic shock). But before you start it don't forget to take 2 blood samples (20-30 ml of 2 different peripheral veins) and send them to bacteriologist in order to receive type of microorganism and antibiotic sensitivity. The most simple scheme of empiric therapy will be single usage of meropenem or imipenem. If you suspect MRSA (Methicillin resistant Staphylococcus aureus) combine meropenem with vancomycin or linezolid.

4. Steroids are recommended for all patients, who need hemodynamic support (hydrocortisone solution i/v 200-300 mg/day).

5. Respiratory support is a part of ARDS treatment. Indications for artificial ventilation:

- $pO_{2a} < 60$ mm Hg,
- $pCO_{2a} > 50$ mm Hg or < 25 mm Hg,
- $SpO_2 < 85\%$
- respiration rate over 40 per minute.

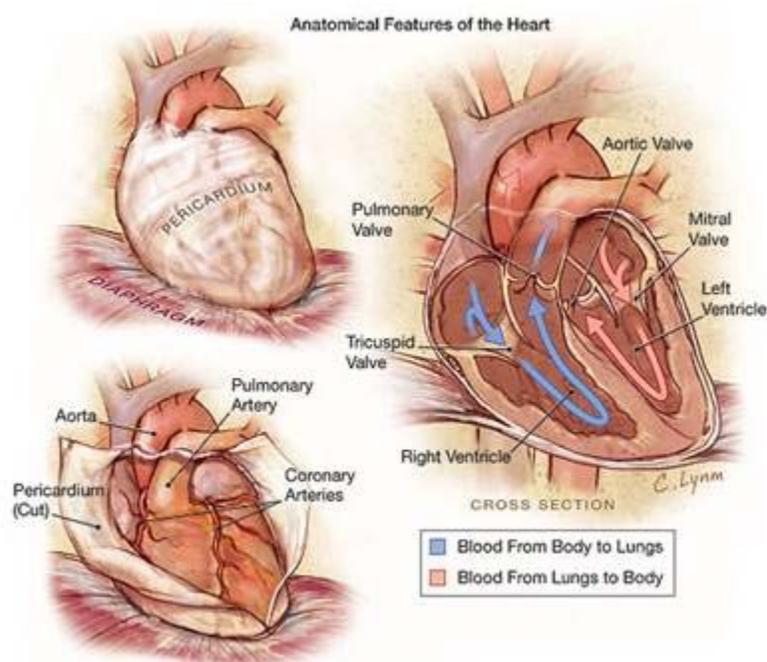
Choosing parameters of ventilation you should remember, that: tidal volume should be 6 ml/kg. P_{plato} – less than 30 cm H₂O, PEEP 5-7 cm H₂O.

6. Surgical treatment of the infection source should be done.

7. Early enteral nutrition improves treatment results and normalizes intestinal functioning. Don't forget about ulcer-prevention (omeprazole 40 mg twice a day, i/v) and peristalsis control (metoclopramide if necessary).

8. Continue syndromic treatment of renal failure, DIC, liver failure, encephalopathy; if it is possible use external methods of detoxification (hemosorbition, plasmapheresis, etc.).

Cardiogenic shock.



Cardiogenic shock is a state of acute heart failure, caused by primary “heart” affection. Usually it happens due to myocardial infarction: necrosis or heart rhythm violations influence negatively pump function of the heart and low cardiac output syndrome appears. This situation

is characterized with high central venous pressure, low blood pressure and compensatory peripheral vascular spasm.

Myocardial infarction according to modern classification is a part of wider category of acute coronary syndrome (together with unstable angina). It is divided into ST elevation MI and non-ST elevation MI (with or without elevation of ST segment on ECG). Those clinical syndromes are different forms of one pathological process: rupture of vulnerable atherosclerotic plaque in coronary artery. Ruptured plaque is filled with blood and this way it blocks blood flow in the artery (additionally affected vessel constricts and blood clot is formed in the place of rupture).

Clinical picture.

Angina pectoris is a burning chest pain or a discomfort feeling, caused by myocardial ischemia. There is also a possibility of referred pain in the jaw, one or both shoulders (usually left), back, neck area or epigastrium. Some of the patients actually never complain of chest pain, but give other pain and discomfort localizations. Unstable angina is suspected when pain pattern changes into more severe, it occurs in rest or minimal exertion and lasts over 10 minutes.

Non-ST-myocardial infarction is suspected when: pain lasts over 20-30 minutes; ST segment is not elevated on ECG, however negative T-wave and depressed ST are possible; troponin tests are positive (directly show myocardial affection). Those clinical features and “coronary” anamnesis state Non-ST-myocardial infarction. Usually those patients do not suffer of sudden occlusion of coronary arteries.

Non-ST-myocardial infarction and unstable angina together are classified as acute coronary syndrome without ST elevation and their treatment is identical.

ST-myocardial infarction is characterised with lasting angina, positive troponin tests, elevated ST segment and Q-waves on 12-leads ECG.

Diagnostics of ACS begins with detailed anamnesis. Physically you can rather differentiate different chest pain reasons than prove infarction (objective symptoms of infarction are really poor: cardiac murmur is possible; asymmetric pulse over upper limbs arteries indicates aorta dissection). ECG shows T-wave changes (sharp or negative T-wave), elevation or depression of ST segment, left bundle branch block (not chronic), heart rhythm violations. Blood tests show moderate leukocytosis $10-15 \cdot 10^9/l$, with young forms of neutrophils, rised erythrocyte sedimentation rate. There are also many MI-marker tests: troponins T and I rise in 3-4 hours after infarction onset and stay high for within 5-7 days (normal values: T_T 0-0,01 ng/l, T_i 0-0,5 ng/l); myoglobin (rises in 2-3 hours, stays within 6-10

hours; normal values 22-66 mkg/l), MB fraction of creatine phosphokinase (rises during first 3-4 hours, maximal level within first 24-36 hours; normal value 0-24 IU/l), lactate dehydrogenase-1 (rises during first 8-10 hours, stays 8-10 days; normal value 240-480 IU/l, LDH-1 is 15-25% of general LDH). There are different express-tests for infarction markers, in many countries they are available even for ambulance service. Additional information can give coagulogram, echocardiography (decrease of ejection fraction, hypokinetic and akinetic areas). Chronography is very important for further treatment tactic; it performed within first hours in case of ST-MI and 24 hours after non-ST-MI onset.

Intensive treatment.

In all cases of acute coronary syndrome are required:

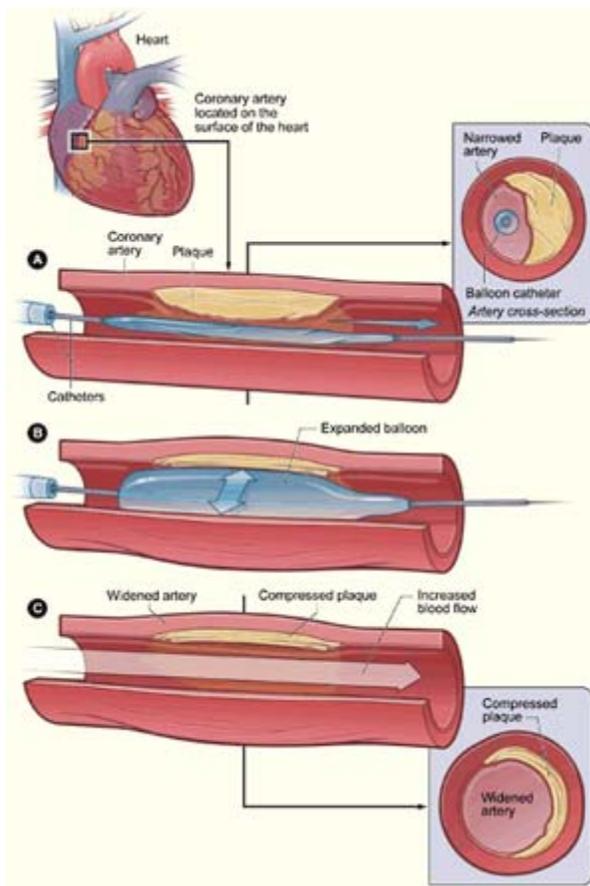
morphine (1 ml , i/v, but don't forget to dilute it with saline and give it very slowly),

oxygen (can be provided through the mask, 6-8 l/min),

nitroglycerin (tablets or 1% solution i/v with blood pressure control),

aspirin (300 mg per os as soon as possible).

The main point of therapy during the first stage of infarction (the most acute) is re-perfusion. After first 12 hours from the moment of pain onset risk of re-perfusion is overwhelming its benefits. Thrombolytics do not provide reperfusion in all patients, however they are dangerous because of haemorrhagic complications. So the choice we actually have during first hours is percutaneous coronary intervention (in case if it is not slowing re-perfusion).



Schemes of thrombolysis

- streptokinase 250 000 U, diluted in 20 ml of saline, given during 30 minutes; then 100 000 U are given i/v during 24-48 hours;
- actilyse 15 mg in bolus, then 0,75 mg/kg during 30 minutes, later 0,5 mg/kg during 60 minutes;
- urokinase 4400 U/kg during 10 minutes, then 4400 U/kg/hour during 12-24 hours.

Relative contraindications to thrombolytic therapy are: early postoperative period, recent injury or gastrointestinal bleeding, vascular cerebral pathology, hypertension with systolic blood pressure over 180 mm Hg or diastolic pressure over 110 mm Hg, high probability of left atrium clot (mitral stenosis with atrialfibrillation), acute pericarditis, subacute bacterial endocarditis, functional disorder of coagulation system, hepatic failure, pregnancy, diabetic hemorrhagic retinopathy, septic thrombophlebitis, patients, who take warfarin constantly.

Absolute contraindications: previous usage of streptokinase (within 4 years); streptococcus infection, active internal bleeding, state after cerebrovascularaccident, recent cerebral or spinal operation, intracerebral aneurism.

Treatment of patients with unstable angina.

The goal of immediate treatment is to prevent further clot formation. You can achieve this by usage of low molecular weight heparin in therapeutic doses. Another way to lower clot-risk is application of β -blockers (or diltiazem) to lower the myocardial oxygen consumption and angiotensin-converting-enzyme inhibitors to avoid left ventricle contractibility disorders or circulation failure.

Further treatment should include heparin (with coagulation control tests – aPTT every 4-6 hours). At the beginning unfractionated heparin is used in the dose of 120-160 U/kilogram, the maintenance dose is 20 U/kg/hour. The duration of such treatment is 4-5 days (if aPTT is less than 45 seconds speed of the infusion is increased by 10%; if aPTT is over 75 seconds the dose is decreased by 10 %). As an alternative you can use low molecular weight heparin (enoxaparin 1 mg/kg every 12 hours; nadroparin 86 U/kg every 12 hours).

β -blockers should be given in all cases except for those with heart rate is 50 and less, systolic blood pressure is lower than 100 mm Hg, AV blocks II-III degrees, concomitant bronchial asthma, obstructive bronchitis. Usually metoprolol is used (25-100 mg per day) intravenously during first hours, but you should control its effects when you change i/v forms into oral forms (heart rate, blood pressure).

You can also use: ACE inhibitors (captopril 6,25-50 mg/day, enalapril 2-20 mg/day, ramipril 2,5-5 mg/day); metabolic medicines (preductal 20 mg twice a day; corvitin 500 mg i/v 3 times a day during first day, twice a day on 2 and 3 day, 250 mg once a day on 4 and 5 day).

Cardiac shock itself has several forms (according to M. Chazov).

1. Reflex shock appears as a reaction to severe pain and is characterized with decrease of vascular tone and blood pressure. It is relatively easy for treatment form for shock.
2. True cardiac shock (already described).
3. Arrhythmogenic shock gives a clinical picture of serious heart rhythm violation, which worsens coronary perfusion and extends necrosis zone.
4. Inert shock is the most severe cardiac shock, hardly cured with conservative methods.

Immediate aid.

1. Place patient into semi-sitting position.
2. Free the chest (unzip shirt if it has tight collar), provide fresh air supply.

3. Check the pulse, blood pressure, consciousness.

4. If you notice pulmonary edema signs treat it appropriately (recommendations written for immediate aid of pulmonary edema you can find in chapter 3).

Intensive treatment.

1. Provide oxygen supply (flow 6-8 liters/minute). Earlier nitrous oxide narcosis was used in case of resistant pain.

2. Use pain killers as described above (1 ml of 1% morphine solution, neuroleptanalgesia: 2 ml of 0,005% fentanyl solution and 1-2 ml of 2,5%droperidol solution; if you don't have narcotic medicines or you don't have possibility to observe patients condition (respiration rate) – use 2 ml of 50%metamizol (analgin) solution intravenously).

3. In order to reduce venous hypertension use diuretics (furosemide solution 20-40 mg intravenously), venous dilators (nitroglycerin and sodiumnitroprusside, 100 mg of each diluted in 200 ml of saline, 5-10 drops per minute), however be very careful with them and control blood pressure all the time while using them.

4. Cardiac output can be regulated in a pharmaceutical way. Your choice of medicine depends on blood pressure:

a. systolic blood pressure less than 70 mm Hg - use norepinephrine (1-30 mcg/min) or dopamine (5-20 mcg/kg/min);

b. systolic blood pressure between 70-100 mm Hg - use dopamine solution (2,5-10 mcg/kg/min);

c. systolic blood pressure over 100 mm Hg – use dobutamine (2-20 mcg/kg /min);

d. in case of diastolic blood pressure is over 100 mm Hg use peripheral vasodilators (nitroglycerin, sodium nitroprusside).

For heart rhythm disorders prescribe antiarrhythmic agents. In case of bradycardia you can choose 1 ml of 0,1 % atropine solution (as adjuncts for haemodynamics control also dopamine, adrenaline in the most severe stages). Tachycardia with heart rate over 130 demands ECG topical diagnostics: atrial fibrillation and atrial flutter are treated with cardiac glycosides, β -blockers, anticoagulants; paroxysmal supraventricular tachycardia –needs vagal tests and adenosine; ventricular tachycardia is treated with lidocaine infusions (1-1,5 mg/kg every 5-10 minutes) and if its not enough – use procaine (20-30 mg/min) or cardioversion.

Exotoxic shock is a type of mixed shock and described in the chapter 8.

Medical operations and manipulations.



Peripheral intravenous access.

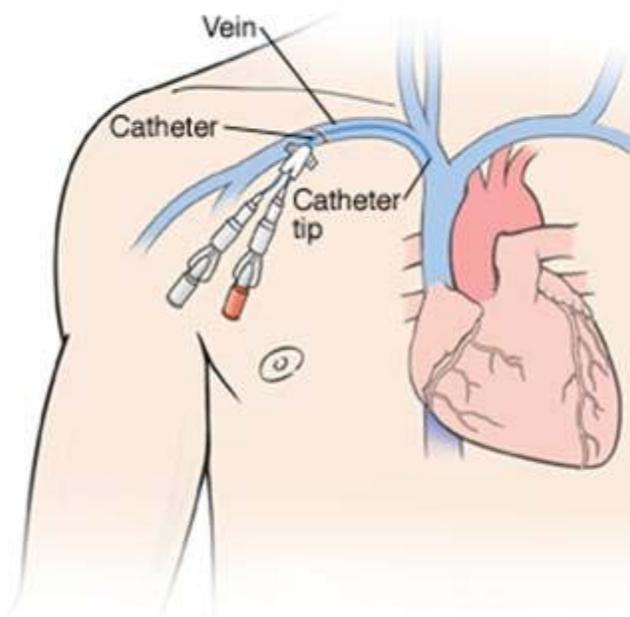
Indications: necessity of infusions.

Required equipment: i/v catheter, giving set (filled with saline), tourniquet, sterile gloves, gauze with antiseptic solution (alcohol swab); i/v dressing.

Procedure: usually i/v lines are inserted in the forearm (lower third,) or hand (try to avoid cubital veins initial catheterization, because you can use them if other possibilities will fail). Apply a tourniquet above the insertion place and disinfect the skin with antiseptic (alcohol swab, special gauze - whatever). Put on the gloves (you can put them at the beginning, but by the moment of taking catheter from the package use antiseptic to disinfect them again) and with your free hand (not the one holding catheter) tract the skin below the vein in order to stabilize its position. Now you have two possibility: direct insertion of the needle into the vein or piercing of the skin aside the vein with further movement of the needle tip over the vein and vein puncture. When blood appears in the chamber, withdraw the needle and

advance the cannula. When you are sure that the catheter is in the vein, release the tourniquet and flush the line with saline (connect the giving set with the cannula). Cover the insertion area with special dressing or simply with the plaster. Make sure everything is working properly (no extravasation signs, no blood in the giving set, quick infusion is possible) and access can't be easily removed. If you notice that growing oedema near the insertion place probably you will have to make another i/v access, because this catheter should be removed and its place should be bandaged in order to prevent further extravasation, i/v insertion of cardiac electrodes.

Central vein catheterization (subclavian vein)



Indications: necessity of permanent infusions, nutrition support, CVP and cardiac output measuring, infusion of drugs which will probably cause phlebitis if used through peripheral veins (hypertonic solutions, chemotherapy, etc).

Required equipment: central venous catheterization set (8-10 cm needle, catheter, wire guide and dilators), giving set, syringes (10 ml, 5 ml), saline (400 ml), 4 ml of 1-2% lidocaine solution, plaster or special dressing, roll (length 40-60*10-15 cm).

Procedure: first of all be aware of the fact that correct patient's position is the key to the successive central vein catheterization. Patient is lying in a supine position with a roll between blades. The arm on the side of puncture is adducted to the trunk and also supine, hand is turned into opposite direction (although some specialists consider this position wrong, because according to their observations it decreases the vein lumen). Usually we are using right subclavian vein, because the risk of complications (pneumothorax for example) is lower from this side (left lung is "higher" due to heart localization, so you can more easily to "catch" it during puncture).

Ask the nurse or assistant to disinfect the puncture area (swab with antiseptic solution 3 times). Wash your hands and put on sterile gloves according to general standards of aseptic. Take the necessary equipment from the kit and position it on a sterile surface in a way you find handy. Find the place of puncture: palpate with the fingers of your right hand point between the middle and medial third of the clavicle, 1-1,5 cm below it. Fill the 5 ml syringe with local anesthetic and infiltrate the tissues around this point. Then take the needle from the set and connect it to the syringe filled with saline. Fix the collar bone over the point of puncture with the fingers of your left hand and perforate the skin, holding the hub firmly. Move forward between collar bone and first rib, until you will feel the increase of resistance (fascia is lying few centimeters deep). After this moment move forward the needle in the direction of sternocostal articulation with continuous aspiration. When you will see the blood in the barrel disconnect the syringe from the needle (close the lumen with the fingertip of your left thumb to prevent air embolism) and insert carefully wire guide until the last of its marks. Holding the wire withdraw the needle. Try not to make unnecessary movements, because needle disconnection and wire insertion are the most “uncertain” steps of central vein cannulation (accidentally you can “loose” the vein). If it is needed use dilators: thread them over the wire and enlarge the entry site (use twisting movements pushing the dilator forward). When you will find the entry wide enough to let the catheter through without much resistance thread the catheter over the wire until the wire will exit the distal lumen of catheter (grasp it there with your left hand). Continue moving the catheter forward (simultaneously pool the wire out) until it will be at the mark your desire. Take out the wire completely and check the placement of catheter by aspiration with the syringe. If you receive venous blood you can flash the catheter with saline and connect giving set (or take blood samples, measure CVP or infuse medicines). Don't forget to fix the external part of the catheter with the special dressing or suture. After all this is done check the catheter placement with an X-ray (it should not end in the right atrium).



Pic. 4.1 Central vein access

- a. Patient's position. Place and direction of puncture.

- b. Needle is in the vein, wire is being inserted.



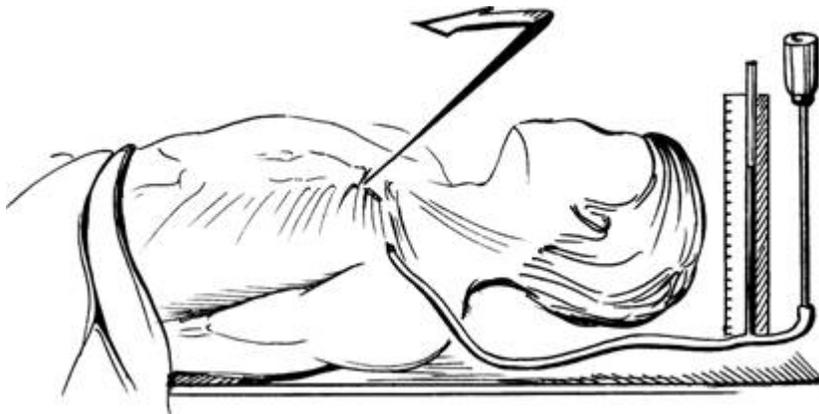
- c. Insertion of the catheter with the help of wire (rotary motion).
 d. Removal of the wire.

Measurement of central venous pressure.

Indications: haemodynamics control (circulating blood volume, vascular tone, heart activity) in case of circulatory diseases, shocks, collapse, prolonged infusion therapy.

Required equipment: central venous line, giving set filled with saline, ruler (you have the most simple method, there are also other ways, which require manometer or transducer).

Procedure: find the middle axillary line and mark it (patient is lying supine) – this is a “zero” level for all the measurements you are going to make (level of right atrium). Connect the giving set to the central venous catheter and place the tubing of the set at the “zero level”. Disconnect the bottle of the infusion and the giving set needle (thus you will connect the fluid in the tubing with atmospheric pressure). The level of the fluid will fall until the moment hydrostatic pressure of the fluid column and pressure of the blood in the catheter (we take it as pressure of the right atrium) will be equal. The height of the fluid column in the tubing is actually patient’s central venous pressure. Measure the fluid column with the ruler and you will get the numerical equivalent of this index.



Measurement of central venous pressure

Control tests.

1. Choose the wrong statement:

- A. venous vessels contain 5 times blood more than arterial
- B. central venous pressure is normally 60-120 mm of water column
- C. arterioles tone influences blood pressure
- D. serotonin constricts vessels
- E. in case of pulmonary edema, due to blood accumulation, central venous pressure lowers.

2. If blood pressure is 80/40 mm of Mercury and the heart rate is 120 per minute, what will be the shock index?

- A. could not be calculated, as we don't know the blood loss
- B. $(80+40)/120=1$
- C. $120/80=1,5$
- D. $80/120=0,67$
- E. $120/40=3$

3. Which statement is wrong in case of traumatic shock?

- A. shock index is very low
- B. low cardiac output occurs
- C. centralization of the circulation develops
- D. central venous pressure lowers
- E. erythrocytes begin to stick together

4. Choose the best treatment of fulminant anaphylactic shock:

- A. dimedrol, prednisolone, cordiamin - intravenously
- B. atropine, pipolfen, prednisolone - ointravenously
- C. poliglukin, calcium chloride, hydrocortisone - intravenously
- D. adrenalin, polyglucin, prednisolone - intravenously
- E. local usage of adrenalin in the place of injection, antihistamine medications intravenously

5. In case of cardiogenic shock you will not observe:

- A. lowering of central venous pressure
 - B. blood stagnation in the lungs
 - C. arrhythmias
 - D. lowering of blood pressure
 - E. decrease of urine output
6. Dehydration shock is characterized with:

- A. blood dilatation
- B. dilatation of the vessels
- C. hypoglycemia
- D. increasing of central venous pressure
- E. weight loss

7. To treat pulmonary edema of cardiac origin you should give:

- A. euphillin
- B. mannitol
- C. morphine
- D. verospiron
- E. calcium chloride

8. Choose the wrong statement:

- A. only in case of cardiac shock central venous pressure increases
- B. to increase blood pressure in case of traumatic shock you should use first of all adrenaline
- C. all shock states reduce cardiac output
- D. each shock lasting long enough can cause renal insufficiency
- E. in case of dehydration shock transfusion of blood is not indicated

Task 1.

Patience, the patient of 44 years, has the central venous pressure 100 mm of water column. What are probable reasons of such value? Describe the therapy.

Task 2.

During the class students of group N were checking blood volume. Results were lost. Calculate blood volume according to the parameters below:

- A. Man of 40, height 170 cm, weight 96 kg
- B. Man of 66, height 150 cm, body weight 66 kg
- C. Man of 14, height 148 cm, weight 41
- D. Man of 59, height 154 cm, weight 88 kg
- E. Man of 28, height 169 cm, body weight 61 kg.

Task 3.

After the train departure station workers found a 44 year old citizen Kane, the patient, who was lying aside the rails and suffering from a severe pain. Patience is holding with both hands his bleeding remains of the right leg (limb is amputated at middle of the thigh). The rest of the leg is lying in the pool of blood on the rails.

- A. Nurse Annie, who came immediately
- B. The “Dream team” – ambulance workers, who arrived in 10 minutes.

Task 4.

Patience, the patient of 44 with a body weight 78 kilograms, is in a shock condition due to trauma. He is sluggish. His skin is cyanotic, his breathing is rapid (24 per minute). Blood pressure is 90/40 mm Hg., heart rate – 128/minute. Central venous pressure is 0. Urinary output per hour is 20 ml. Calculate AI and evaluate the level of traumatic shock compensation.

Task 5.

Patience, the patient of 54, suddenly felt chest pain in 3 o'clock in the morning. He took one nitroglycerin tablet – it didn't help. The “Dream team” – ambulance workers who arrived later found next symptoms: gray clammy skin, shortness of breath, moist rales in lower lobes of the lungs. Heart sounds are weak, blood pressure is 100/60 mm Hg, heart rate – 100/minute. Pulse is weak and arrhythmic.

Describe the algorithm of the “Dream team”.

Task 6.

Your neighbor, granny Annie, suddenly felt ill. She is suffering from hypertonic disease for many years. During the last day her condition worsened – antihypertensive medicines are not effective any more. She has a shortness of breath, noisy breathing, cough with pink phlegm. What are your actions before the arrival of the ambulance?

Task 7.

Nurse Annie was measuring central venous pressure in the intensive care unit. Diagnoses of her patients are: a. myocardial infarction; b. traumatic shock; c. condition

after gastric resection; d. anaphylaxis. The CVP results were: 70 mm Hg, 30 mm Hg, 160 mm Hg, 0 mm Hg. Connect each disease with its probable central venous pressure.

Violations of homeostasis and their correction.

The importance of the water to the organism.

Life on earth was born in the water environment. Water is a universal solvent for all the biochemical processes of the organism. Only in case of stable quantitative and qualitative composition of both intracellular and extra cellular fluids homeostasis is remained.

The body of an adult human contains 60% of water. Intracellular water makes 40% of the body weight, the water of intercellular space makes 15% of body weight and 5% of body weight are made by the water in the vessels. It is considered that due to unlimited diffusion of water between vessels and extra vascular space the volume of extracellular fluid is 20% of body weight (15%+5%).

Physiologically insignificant amounts of water are distributed beyond the tissues in the body cavities: gastrointestinal tract, cerebral ventricles, joint capsules (nearly 1% of the body weight). However during different pathologic conditions this “third space” can cumulate large amounts of fluid: for example in case of ascites caused by chronic cardiac insufficiency or cirrhosis abdominal cavity contains up to 10 liters of fluid. Peritonitis and intestinal obstructions remove the fluid part of blood from the vessels into the intestinal cavity.

Severe dehydration is extremely dangerous for the patient. Water gets to the body with food and drinks, being absorbed by the mucous membranes of gastro-intestinal tract in total amount of 2-3 liters per day. Additionally in different metabolic transformations of lipids, carbohydrates and proteins nearly 300 of endogenous water are created. Water is evacuated from the body with urine (1,5-2 liters), stool (300 ml), perspiration and breathing (those two reasons are combined as “perspiration loss” and make from 300 to 1000 ml per day).

Water balance is regulated through complicated, but reliable mechanisms. Control over water and electrolytes excretion is realized by osmotic receptors of posterior hypothalamus, volume receptors of the atrial walls, baroreceptors of carotid sinus, juxtaglomerular apparatus of the kidneys and adrenal cortical cells.

When there is a water deficiency or electrolytes excess (sodium, chlorine) thirst appears and this makes us drink water. At the same time posterior pituitary produces antidiuretic hormone, which decreases urine output. Adrenals reveal into the blood flow aldosterone, which stimulates reabsorption of sodium ions in the tubules and thus also decreases diuresis (due to osmosis laws water will move to the more concentrated solution).

This way organism can keep precious water.

On the contrary, in case of water excess endocrine activity of glands is inhibited and water is actively removed from the body through the kidneys.

Importance of osmolarity for homoeostasis.

Water sections of the organism (intracellular and extracellular) are divided with semipermeable membrane – cell wall. Water easily penetrates through it according to the laws of osmosis. Osmosis is a movement of water through a partially permeable membrane from the solution with lower concentration to a solution with higher concentration.

Osmotic concentration (osmolarity) is the concentration of active parts in one liter of solution (water). It is defined as a number of miliosmoles per liter (mOsm/l). Normally osmotic concentration of plasma, intracellular and extracellular fluids is equal and varies between 285mOsm/l. This value is one of the most important constants of the organism, because if it changes in one sector the whole fluid of the body will be redistributed (water will move to the environment with higher concentration). Over hydration of one sector will bring dehydration of another. For example, when there is a tissue damage concentration of active osmotic parts increases and water diffuses to this compartment, causing oedema. On the contrary plasma osmolarity decreases, when there is a loss of electrolytes and osmotic concentration of the cellular fluid stays on the previous level. This brings cellular oedema, because water moves through the intracellular space to the cells due to their higher osmotic concentration.

Cerebral oedema appears when the plasma osmolarity is lower than 270 mOsm/l. Activity of central nervous system is violated and hyposmolar coma occurs. Hyperosmolar coma appears when the plasma osmolarity is over 320 mOsm/l: water leaves the cells and fills the vascular bed and this leads to cellular dehydration. The sensitive to cellular dehydration are the cells of the brain.

Plasma osmolarity is measured with osmometer. The principle of measurement is based on difference in freezing temperature between distilled water and plasma. The higher is the osmolarity (quantity of molecules) the lower is freezing temperature.

Plasma osmotic concentration can be calculated according to the formula:

$$\text{Osmotic concentration} = 1,86 * \text{Na} + \text{glucose} + \text{urea} + 10,$$

Plasma osmolarity (osmotic concentration) – mOsm/l

Na- sodium concentration of plasma, mmol/l

Glucose- glucose concentration of the plasma, mmol/l

Urea- urea concentration of the plasma, mmol/l

According to this formula sodium concentration is the main factor influencing plasma osmolarity. Normally sodium concentration is 136-144 mmol/l. Water and electrolytes balance can be violated with external fluid and electrolytes loss, their excessive inflow or wrong distribution.

9.3 Fluid imbalance and principles of its intensive treatment.

Water imbalance is divided into dehydration and overhydration.

Dehydration is caused by:

- excessive perspiration in conditions of high temperature;
- rapid breathing (dyspnea, tachypnea) or artificial ventilation without humidification of the air;
- vomiting, diarrhoea, fistulas;
- blood loss, burns;
- diuretics overdose;
- excessive urine output;
- inadequate enteral and parenteral nutrition or infusion therapy (comatose patients, postoperative care);
- pathological water distribution (“third space” in case of inflammation or injury).

Dehydration signs: weight loss, decrease of skin turgor and eyeballs tone, dry skin and mucous membranes; low central venous pressure, cardiac output and blood pressure (collapse is possible); decreased urine output and peripheral veins tone; capillary refill over 2 seconds (microcirculation disorders) and low skin temperature; intracellular dehydration is characterized with thirst and consciousness disorders. Laboratory tests show blood concentration: hematocrit, hemoglobinconcentration, protein level and red blood cells concentration increase.

Overhydration appears in case of:

- excessive water consumption, inadequate infusion therapy;

- acute and chronic renal failure, hepatic and cardiac insufficiency;
- disorders of fluid balance regulation;
- low protein edema.

Clinical findings in case of overhydration are: weight gain, peripheral oedema, transudation of the plasma into the body cavities (pleural, abdominal), high blood pressure and central venous pressure. In case of intracellular overhydration appear additional symptoms: nausea, vomiting, signs of cerebral edema (spoor, coma). Laboratory tests prove hemodilution.

According to the osmotic concentration of plasma dehydration and overhydration are divided into hypotonic, isotonic and hypertonic.

Isotonic dehydration is caused by equal loss of electrolytes and fluid from the extracellular space (without cellular disorders). Blood tests show hemoconcentration; sodium level and osmotic concentration are normal.

To treat this type of water imbalance use normal saline solution, Ringer solution, glucose-saline solutions, etc.. The volumes of infusions can be calculated according to the formula:

$$V_{H_2O} = 0,2 * BW * (H_{tp} - 0,4) / 0,4 ,$$

V_{H_2O} – volume of infusion, l

H_{tp} – patient's hematocrit, l/l,

BW – body weight, $0,2 * BW$ – volume of extracellular fluid,

0,4- normal hematocrit, l/l,

Hypertonic dehydration is caused by mostly water loss: first it appears in the vascular bed, than in the cells. Laboratory tests show hemoconcentration: elevated levels of proteins, red blood cells, hematocrit. Plasma sodium is over 155 mmol/l and osmotic concentration increases over 310 mOsm/l.

Intensive treatment: if there is no vomiting allow patients to drink. Intravenously give 0,45% saline solution and 2,5 % glucose solution, mixed with insulin. The volume of infusions is calculated according to the formula:

$$V_{H_2O} = 0,6 * BW (Na_p - 140) / 140,$$

V_{H_2O} – water deficiency, l

Na_p – plasma sodium, mmol/l

BW – body weight, 0,6*BW volume of general body fluid

140 – physiological plasma sodium concentration

Hypotonic dehydration is characterized with clinical features of extracellular dehydration. Laboratory tests show decrease of sodium and chlorine ions. Those changes cause intracellular movement of the water (intracellular overhydration). Hemoglobin, hematocrit and protein levels are increased. Sodium is lower than 136mmol/l, osmolarity is lower than 280 mOsm/l.

To treat this type of water imbalance use normal or hypertonic saline and sodium bicarbonate solution (depends on blood pH). Do not use glucose solutions!

The deficiency of electrolytes is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na_d – sodium deficiency, mmol

Na_p – plasma sodium, mmol/l

BW – body weight, 0,2 BW – volume of extracellular fluid

Isotonic overhydration is caused by excess of the water in the vascular bed and extracellular space; however intracellular homeostasis is not violated. Hemoglobin is less than 120 g/l, protein level is less than 60 g/l, plasma sodium is 136-144 mmol/l, osmotic concentration is 285-310 mOsm/l.

Treat the reason of imbalance: cardiac failure, liver insufficiency, etc. Prescribe cardiac glycosides, limit salt and water consumption. Give osmotic diuretics (mannitol solution 1,5 g/kg), saluretics (furosemide solution 2 mg/kg), aldosterone antagonists (triamterene – 200 mg), steroids (prednisolone solution 1-2 mg/kg) albumin solution if necessary (0,2-0,3 g/kg).

Hypertonic overhydration is a state of extracellular electrolytes and water excess combined with intracellular dehydration. Blood tests show decrease of hemoglobin, hematocrit, protein level, however sodium concentration is increased over 144 mmol/l, osmotic concentration is over 310 mOsm/l.

To treat this condition use solutions without electrolytes: glucose with insulin, albumin solutions and prescribe saluretics (furosemide solution), aldosterone antagonists (spironolactone). If it is necessary perform dialysis and peritoneal dialysis. Do not use crystalloids!

Hypotonic overhydration is a state of extracellular and intracellular water excess. Blood tests show decrease of haemoglobin, hematocrit, proteins, sodium and osmotic concentration. Intensive therapy of this condition includes osmotic diuretics (200-400 ml of 20% mannitol solution), hypertonic solutions (50 ml of 10% saline intravenously), steroids. When it is required use ultrafiltration to remove water excess.

Electrolytes disorders and their treatment

Potassium is a main intracellular cation. Its normal plasma concentration is 3,8-5,1 mmol/l. Daily required amount of potassium is 1 mmol/kg of body weight.

Potassium level less than 3,8 mmol/l is known as kaliopenia. Potassium deficiency is calculated according to the formula:

$$K_d = (4,5 - K_p) * 0,6 \text{ BW}$$

K - potassium deficiency, mmol;

K_p – potassium level of the patient mmol/l;

$0,6 * \text{BW}$ – total body water, l.

To treat this state use 7,5% solution of potassium chloride (1ml of this solution contains 1 mmol of potassium). Give it intravenously slowly with glucose and insulin (20-25 ml/hour). You can also prescribe magnesium preparations. Standard solution for kaliopenia treatment is:

10% glucose solution 400 ml

7,5% potassium chloride solution 20 ml

25% magnesium sulphate solution 3 ml

insulin 12 units

Give it intravenously slowly, during one hour. Forced bolus infusion of potassium solutions (10-15 ml) can bring cardiac arrest.

Potassium level over 5,2 mmol/l is a state called hyperkalemia. To treat this condition use calcium gluconate or calcium chloride solutions (10 ml of 10% solution intravenously), glucose and insulin solution, saluretics, steroids, sodium bicarbonate solution. Hyperkalemia over 7 mmol/l is an absolute indication for dialysis.

Sodium is the main extracellular cation. Its normal plasma concentration is 135-155 mmol/l. Daily required amount of potassium is 2 mmol/kg of body weight.

Sodium concentration which is lower than 135 mmol/l is known as hyponatraemia. This condition is caused by sodium deficiency or water excess. Sodium deficiency is calculated according to the formula:

$$Na_d = (140 - Na_p) * 0,2 BW,$$

Na- sodium deficiency, mmol;

Na_p – sodium concentration of the patient mmol/l;

0,2*BW – extracellular fluid volume, l.

To treat it use normal saline (1000 ml contains 154 Na mmol) or 5,8% solution of sodium chloride – your choice will depend on osmotic concentration.

Sodium concentration over 155 mmol/l is a state called hypernatremia. This condition usually appears in case of hypertonic dehydration or hypertonicoverhydration. Treatment was described in the text above.

Chlorine is the main extracellular anion. Its normal plasma concentration is 98-107 mmol/l. Daily requirement of chlorine is 215 mmol.

Hypochloremia is a condition of decreased plasma chlorine concentration (less than 98 mmol/l).

Chlorine deficiency is calculated according to the formula:

$$Cl_d = (100 - Cl_p) * 0,2 BW,$$

Cl_d- chlorine deficiency, mmol

Cl_p – plasma chlorine concentration of the patient, mmol/l

0,2*BW – extracellular fluid volume, l.

To treat hypochloremia use normal saline (1000 ml contains 154 mmol of chlorine) or 5,8% sodium chloride solution (1 ml contains 1 mmol of chlorine). The choice of solution depends on the osmotic concentration of the plasma.

Hyperchloremia is a condition of increased chlorine concentration (over 107 mmol/l). Intensive therapy of this state includes treatment of the disease, which caused it (decompensated heart failure, hyperchloremic diabetes insipidus, glomerulonephritis). You can also use glucose, albumin solutions and dialysis.

Magnesium is mostly an intracellular cation. Its plasma concentration is

0,8-1,5 mmol/l. Daily requirement of magnesium is 0,3 mmol/kg.

Hypomagnesemia is a state of decreased magnesium concentration: less than 0,8 mmol/l. Magnesium deficiency is calculated according to the formula:

$$Mg_d = (1,0 - Mg_p) * 0,6BW,$$

Mg_d - magnesium deficiency, mmol

Mg_p – plasma magnesium concentration of the patient, mmol/l

0,6*BW – extracellular fluid volume, l.

Use 25% magnesium sulphate solution to treat this state (1 ml of it contains 0,5 mmol of magnesium).

Hypermagnesemia is a state of increased magnesium concentration (more than 1,5 mmol/l). This condition appears usually in case of hyperkalemia and you should treat it as you treat hyperkalemia.

Calcium is one of the extracellular cations. Its normal concentration is 2,35-2,75 mmol/l. Daily requirement of calcium is 0,5 mmol/kg.

Calcium concentration less than 2,35 mmol/l is called hypocalcemia. Calcium deficiency is calculated according to the formula:

$$Ca_d = (2,5 - Ca_p) * 0,2 BW,$$

Ca_d – calcium deficiency, mmol

Ca_p – plasma calcium concentration of the patient, mmol/l

0,2*BW – extracellular fluid volume, l.

To treat this state use 10% calcium chloride (1 ml of the solution contains 1,1 mmol of calcium), ergocalciferol; in case of convulsions prescribe sedative medicines.

Hypercalcemia is a condition with increased calcium concentration (over 2,75 mmol/l). Treat the disease, which caused it: primary hyperparathyroidism, malignant bone tumors, etc. Additionally use infusion therapy (solutions of glucose with insulin), steroids, dialysis and hemosorbtion.

Acid-base imbalance and its treatment.

There are 2 main types of acid-base imbalance: acidosis and alkalosis.

pH is a decimal logarithm of the reciprocal of the hydrogen ion activity. It shows

acid-base state of the blood.

Normal pH of arterial blood is 7,36-7,44. Acid based imbalance is divided according to the pH level into:

pH 7,35-7,21 – subcompensated acidosis

pH < 7,2 – decompensated acidosis

pH 7,45-7,55 – subcompensated alkalosis

pH > 7,56 – decompensated alkalosis

Respiratory part of the acid-base imbalance is characterized with pCO₂. Normally pCO₂ of arterial blood is 36-44 mm Hg. Hypercapnia (pCO₂ increased over 45 mm Hg) is a sign of respiratory acidosis. Hypocapnia (pCO₂ less than 35 mm Hg) is a symptom of respiratory alkalosis.

Basis excess index is also a characteristic of metabolic processes. Normally H⁺ ions produced during metabolic reactions are neutralized with buffer system. BE of arterial blood is 0±1,5. Positive value of BE (with +) is a sign of base excess or plasma acid deficiency (metabolic alkalosis). Negative value of BE (with -) is a symptom of bases deficiency, which is caused by acid neutralization in case of metabolic acidosis.

Respiratory acidosis (hypercapnia) is a condition caused by insufficient elimination of CO₂ from the body during hypoventilation. Laboratory tests show:

pH < 7,35,

pCO_{2a} > 46 mm Hg

BE - normal values

However when the respiratory acidosis progresses renal compensation fails to maintain normal values and BE gradually increases. In order to improve this condition you should treat acute and chronic respiratory violations. When pCO₂ is over 60 mm Hg begin artificial lung ventilation (through the mask or tube; when the necessity of ventilation lasts longer than 3 days – perform tracheostomy).

Respiratory alkalosis (hypocapnia) is usually an effect of hyperventilation, caused by excessive stimulation of respiratory centre (injuries, metabolic acidosis, hyperactive metabolism, etc.) or wrong parameters of mechanical ventilation. Gasometry shows:

pH > 7,45,

pCO_{2a} < 33 mm Hg

BE < +1,5 mmol/l.

However prolong alkalosis brings decrease of BE due to compensatory retain of H⁺ ions. To improve this imbalance treat its reason: normalize ventilation parameters; if patients breathing has rate over 40 per minute – sedate the patient, perform the intubation and begin artificial ventilation with normal parameters.

Metabolic acidosis is characterized with absolute and relative increase of H⁺ ions concentration due to acid accumulation (metabolic disorders, block of acid elimination, excessive acid consumption in case of poisonings, etc.). Laboratory tests show:

pH < 7,35,

pCO_{2a} < 35 mm Hg

BE (-3) mmol/l.

Treat the main reason of acid-base disorder: diabetic ketoacidosis, renal insufficiency, poisoning, hyponatremia or hyperchloremia, etc. Normalize pH with 4% sodium bicarbonate solution. Its dose is calculated according to the formula:

$$V = 0,3 * BE * BW$$

V- volume of sodium bicarbonate solution, ml

BE – bases excess with “-”, mmol/l

BW – body weight, kg

Metabolic alkalosis is a condition of absolute and relative decrease of H⁺ ions concentration. Blood tests show:

pH > 7,45,

pCO_{2a} normal or insignificantly increased (compensatory reaction)

BE 3,0 mmol/l.

To treat this condition use “acid” solutions, which contain chlorides (saline, potassium chloride). In case of kaliopenia give potassium solutions.

Respiratory and metabolic imbalances can mix in case of severe decompensated diseases due to failure of compensatory mechanisms. Correct interpretation of these violations is possible only in case of regular and iterative gasometry blood tests.

Control tasks.

Task 1.

Calculate the total body water volume and its extracellular and intracellular volumes of the Patient, the patient of 48 years and body weight 88 kg.

Task 2.

Patient, the patient of 23 with body weight 70 kg has sodium level 152 mmol/l and hematocrit 0,49 l/l. Name the type of water balance disorder.

Task 3.

Patient, the patient of 54 with body weight 76 kg has sodium level 128 mmol/l. Calculate the volume of saline and 7,5% sodium chloride solution necessary for the treatment of this condition.

Task 4.

Patient, the patient of 60 with body weight 60 kg has sodium level 140 mmol/l and hematocrit 0,55 l/l. Name the type of disorder and prescribe infusion therapy.

Task 5.

Patient, the patient of 42 with body weight 80 kg has potassium level 2,6 mmol/l. Calculate the volume of 4% potassium chloride solution necessary for treatment of this condition.

Task 6.

Patient, the patient of 33 with body weight 67 kg and diagnosis “gastric ulcer, complicated with pylorostenosis” has potassium concentration 3 mmol/l, chlorine concentration 88 mmol/l. pH 7,49, pCO_{2a} 42 mm Hg, BE + 10 mmol/l. Name the type of disorder.

Task 7.

Patient, the patient of 50 with body weight 75 kg, was transported to the admission unit of the hospital with: unconsciousness, cyanotic skin, low blood pressure, shallow breathing. Blood tests show: pH 7,18, pCO_{2a} 78 mm Hg, pO_{2A} – 57 mm Hg, BE -4,2 mmol/l. Name the type of acid-base disorder and prescribe treatment.

Task 8.

Patient, the patient with body weight 62 kg and renal insufficiency has: potassium concentration 5,2 mmol/l, sodium concentration 130 mmol/l, calcium concentration 1,5 mmol/l, pH 7,22, pCO_{2a} 34 mm Hg, BE -9,2 mmol/l. Name the type of disorder.

Systemic inflammatory response syndrome in surgical patients. Pathogenesis, its importance in the clinical course of various diseases and traumas. Treatment tactic.

In medicine, systemic inflammatory response syndrome (SIRS) is an inflammatory state affecting the whole body, frequently in response of the immune system to infection, but not necessarily so. It is related to sepsis, a condition in which individuals both meet criteria for SIRS and have a known or highly suspected infection.



The latest finding shows that SIRS in trauma patients may be caused by immune reaction to mitochondria massively released into bloodstream from dying cells at the site of injury. [1]Contents [hide]

- 1 Classification
- 2 Definition
- 3 Complications
- 4 Causes
- 5 Treatment
- 6 See also
- 7 References

[edit]

Classification

SIRS is a serious condition related to systemic inflammation, organ dysfunction, and organ failure. It is a subset of cytokine storm, in which there is abnormal regulation of various cytokines.[citation needed] SIRS is also closely related to sepsis, in which patients satisfy criteria for SIRS and have a suspected or proven infection.[2][3][4]

[edit]

Definition

SIRS was first described by Dr. Nelson, of the University of Toronto, at the Nordic Micro Circulation meeting in Geilo, Norway in February of 1983. The intent of creating an encompassing definition was to bring together the multiple etiologies of post episode organ dysfunction (fibrin deposition, platelet aggregation, coagulopathies, leukocyte lysosomal release) into a family of negatively synergistic responses to injury and/or infection which can collectively lead to micro circulatory dysfunction. The implication of such a definition

suggested that recognition of the activation of one of the above noted humoral pathways suggests that additional processes are also active. The aggregate of such pathophysiology would lead to clinical conditions such as renal failure and/or pulmonary edema.

Criteria for SIRS were established in 1992 as part of the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference.[2] The conference concluded that the manifestations of SIRS include, but are not limited to:

Body temperature less than 36°C or greater than 38°C

Heart rate greater than 90 beats per minute

Tachypnea (high respiratory rate), with greater than 20 breaths per minute; or, an arterial partial pressure of carbon dioxide less than 4.3 kPa (32 mmHg)

White blood cell count less than 4000 cells/mm³ (4 x 10⁹ cells/L) or greater than 12,000 cells/mm³ (12 x 10⁹ cells/L); or the presence of greater than 10% immature neutrophils (band forms)

SIRS can be diagnosed when two or more of these criteria are present.[3][4][5][6]

The International Pediatric Sepsis Consensus has proposed some changes to adapt these criteria to the pediatric population.

Fever and leukocytosis are features of the acute-phase reaction, while tachycardia is often the initial sign of hemodynamic compromise. Tachypnea may be related to the increased metabolic stress due to infection and inflammation, but may also be an ominous sign of inadequate perfusion resulting in the onset of anaerobic cellular metabolism.

In children, the SIRS criteria are modified in the following fashion:

Heart rate > 2 standard deviations above normal for age in the absence of stimuli such as pain and drug administration, OR unexplained persistent elevation for greater than 30 minutes to 4 hours. In infants, also includes Heart rate < 10th percentile for age in the absence of vagal stimuli, beta-blockers, or congenital heart disease OR unexplained persistent depression for greater than 30 minutes.

Body temperature obtained orally, rectally, from Foley catheter probe, or from central venous catheter probe < 36 °C or > 38.5 °C. Temperature must be abnormal to qualify as SIRS in pediatric patients.

Respiratory rate > 2 standard deviations above normal for age OR the requirement for mechanical ventilation not related to neuromuscular disease or the administration of anesthesia.

White blood cell count elevated or depressed for age not related to chemotherapy, or greater than 10% bands + other immature forms.

Note that SIRS criteria are very non-specific, and must be interpreted carefully within the clinical context. These criteria exist primarily for the purpose of more objectively classifying critically-ill patients so that future clinical studies may be more rigorous and more easily reproducible.

As an alternative, when two or more of the systemic inflammatory response syndrome criteria are met without evidence of infection, patients may be diagnosed simply with "SIRS." Patients with SIRS and acute organ dysfunction may be termed "severe SIRS."

[edit]

Complications



SIRS is frequently complicated by failure of one or more organs or organ systems.[2][3][4] The complications of SIRS include:

- Acute lung injury
- Acute kidney injury
- Shock
- Multiple organ dysfunction syndrome

[edit]

Causes

The causes of SIRS are broadly classified as infectious or noninfectious. As above, when SIRS is due to an infection, it is considered sepsis. Noninfectious causes of SIRS include trauma, burns, pancreatitis, ischemia, and hemorrhage.[2][3][4]

Other causes include:[citation needed]

- Complications of surgery
- Adrenal insufficiency
- Pulmonary embolism
- Complicated aortic aneurysm
- Cardiac tamponade
- Anaphylaxis
- Drug overdose

[edit]

Treatment

Generally, the treatment for SIRS is directed towards the underlying problem or inciting cause (i.e. adequate fluid replacement for hypovolemia, IVF/NPO for pancreatitis, epinephrine/steroids/benadryl for anaphylaxis).[1] Selenium, glutamine, and eicosapentaenoic acid have shown effectiveness in improving

symptoms in clinical trials.[10] Other antioxidants such as vitamin E may be helpful as well.[11]

[edit]

See also

Sepsis

Septicemia

Septic shock

Acute respiratory distress syndrome

Inflammatory response

immune system

[edit]

Health science · Medicine · Medical specialities · Intensive care medicine / Critical care medicine and Critical care nursing

General terms Intensive-care unit (ICU) · Neonatal intensive care unit (NICU) · Pediatric intensive care unit (PICU) · Coronary care unit (CCU) · Critical illness insurance

Conditions Organ system failure

Shock sequence: SIRS · Sepsis · Severe sepsis · Septic shock

Organ failure: Acute renal failure · Acute respiratory distress syndrome · Acute liver failure · Respiratory failure · Multiple organ dysfunction syndrome
Polytrauma · Coma

Complications Critical illness polyneuropathy / myopathy · Critical illness–related corticosteroid insufficiency · Decubitus ulcers · Fungemia · Stress hyperglycemia · Stress ulcer

Iatrogenesis Methicillin-resistant *Staphylococcus aureus* · Oxygen toxicity · Refeeding syndrome · Ventilator-associated lung injury · Ventilator-associated pneumonia

Diagnosis Arterial blood gas · catheter (Arterial catheter, Central venous catheter, Pulmonary artery catheter) · Blood cultures · Screening cultures

Life supporting treatments Airway management · Chest tube · Dialysis · Enteral feeding · Goal-directed therapy · Induced coma · Mechanical ventilation · Therapeutic hypothermia · Total parenteral nutrition · Tracheal intubation

Drugs Analgesics · Antibiotics · Antithrombotics · Inotropes · Intravenous fluids · Neuromuscular-blocking drugs · Recombinant activated protein C · Sedatives · Stress ulcer prevention drugs · Vasopressors

ICU scoring systems APACHE II · Glasgow Coma Scale · PIM2 · SAPS II · SAPS III · SOFA

Organisations Society of Critical Care Medicine · Surviving Sepsis Campaign

Related specialties Anesthesia · Cardiology · Internal medicine · Neurology · Pediatrics · Pulmonology · Surgery · Traumatology



SEPSIS and It's Disease spectrum

- Various stages of disease
 - Bacteremia
 - SIRS
 - Sepsis syndrome
 - Sepsis shock : early and refractory



Definition

- **Infection**
 - Presence of microorganisms in a normally sterile site.
- **Bacteremia**
 - Cultivable bacteria in the blood stream.
- **Sepsis**
 - The systemic response to infection. If associated with proven or clinically suspected infection, SIRS is called "sepsis".

American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee. *Crit Care Med.* 1992;20:864-874.

SIRS

(Systemic Inflammatory Response Syndrome)

- The systemic response to a wide range of stresses.
 - Temperature $>38^{\circ}\text{C}$ (100.4°) or $<36^{\circ}\text{C}$ (96.8°F).
 - Heart rate >90 beats/min.
 - Respiratory rate >20 breaths/min or $\text{PaCO}_2 <32$ mmHg.
 - White blood cells $> 12,000$ cells/ml or $< 4,000$ cells/ml or $>10\%$ immature (band) forms.
- Note
 - Two or more of the following must be present.
 - These changes should be represent acute alterations from baseline in the absence of other known cause for the abnormalities.

American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee. *Crit Care Med.* 1992;20:864-874.

Severe Sepsis

- Sepsis with organ hypoperfusion
one of the followings :
 - $\text{SBP} < 90$ mmHg
 - Acute mental status change
 - $\text{PaO}_2 < 60$ mmHg on RA ($\text{PaO}_2 / \text{FiO}_2 < 250$)
 - Increased lactic acid/acidosis
 - Oliguria
 - DIC or Platelet $< 80,000 /\text{mm}^3$
 - Liver enzymes > 2 x normal

American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee. *Crit Care Med.* 1992;20:864-874.



MODS (Multiple Organ Dysfunction Syndrome)

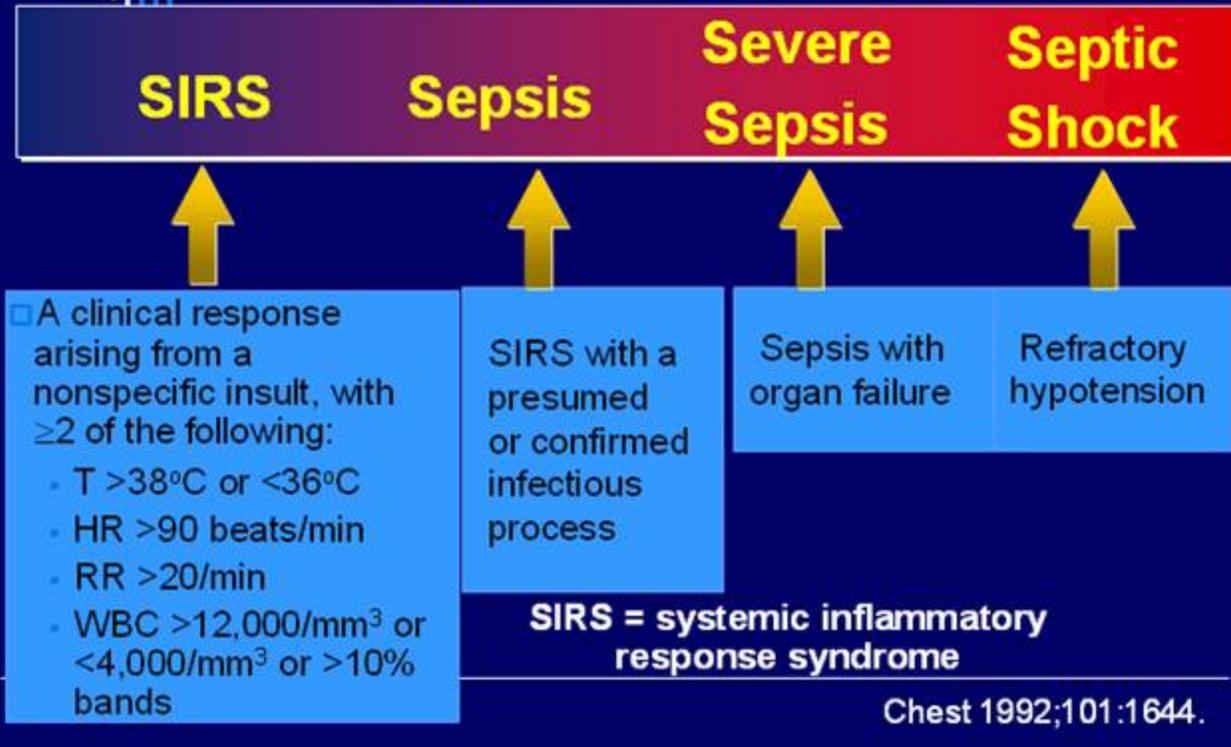
□ Sepsis with multiorgan hypoperfusion

Two or more of the followings:

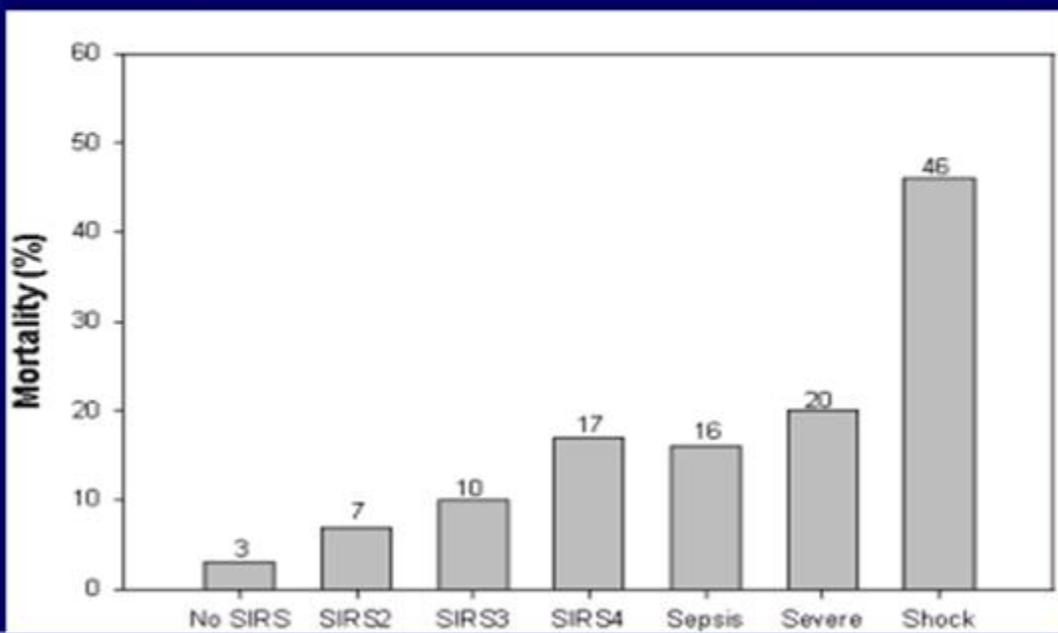
- SBP < 90 mmHg
- Acute mental status change
- PaO₂ < 60 mmHg on RA (PaO₂/FiO₂ < 250)
- Increased lactic acid/acidosis
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- DIC or Platelet < 80,000 /mm³
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American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee. Crit Care Med. 1992;20:864-874.

The Sepsis Continuum

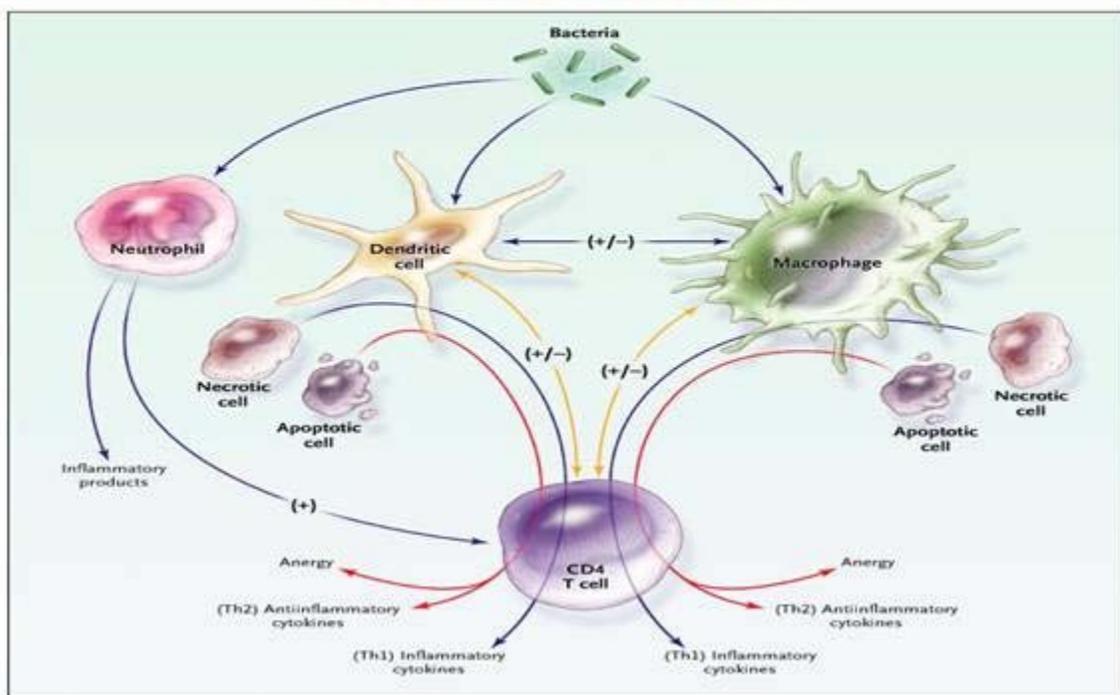


Mortality rate in SIRS

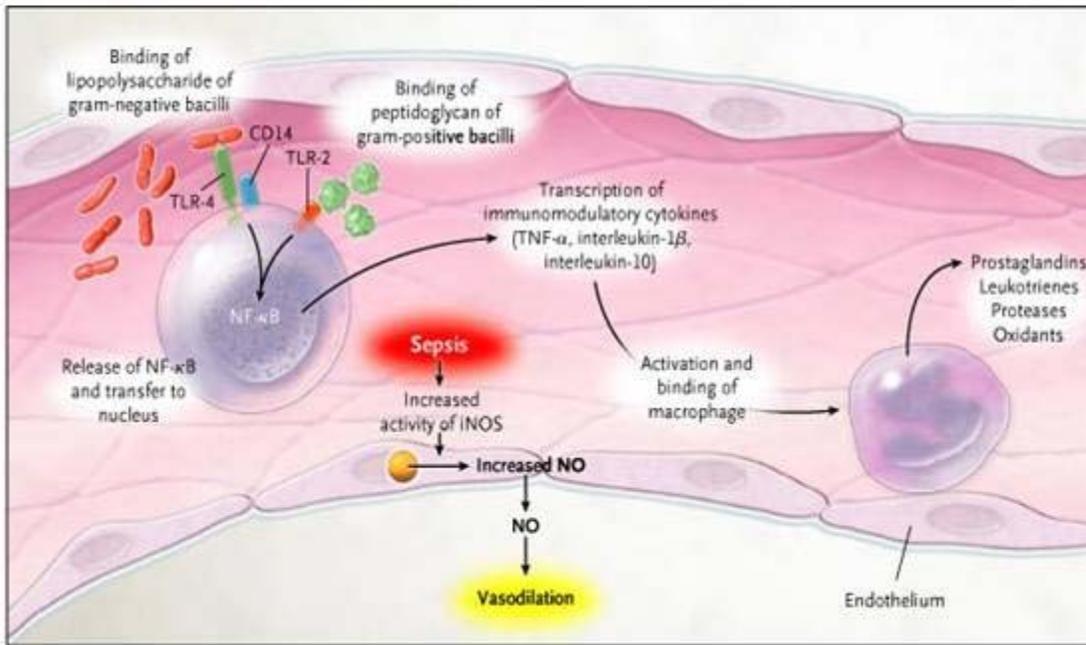


Rangel-Frausto, et al. JAMA 273:117-123, 1995.

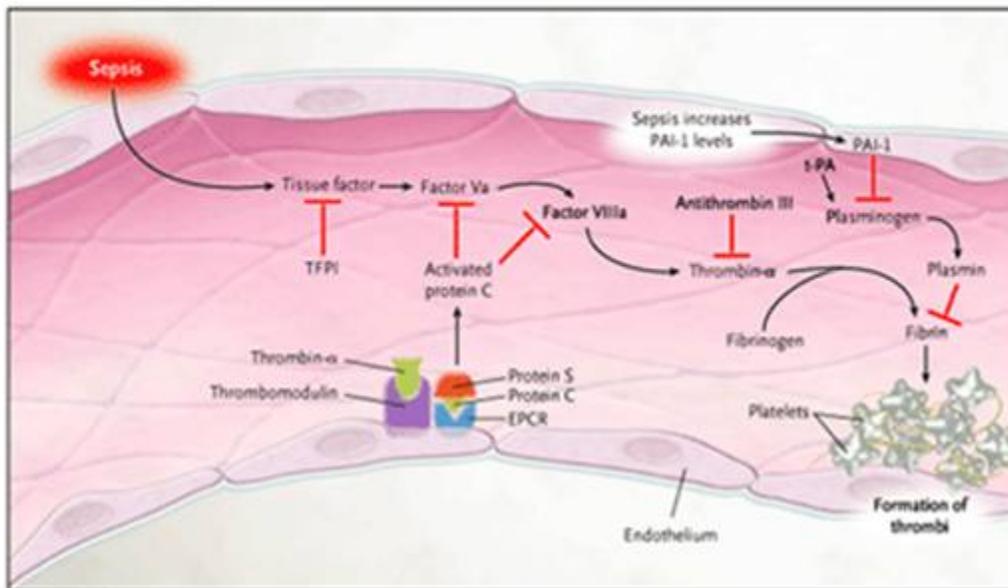
The Response to Pathogens "Cross-Talk"



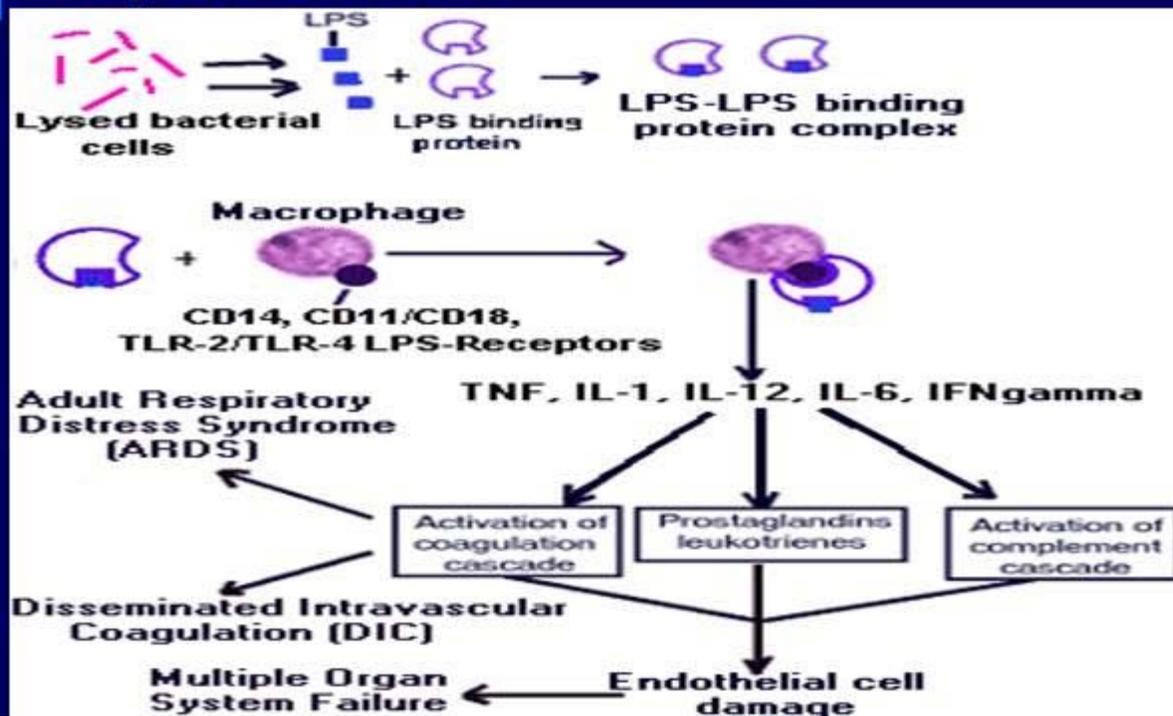
Inflammatory Response to Sepsis



Procoagulant Response in Sepsis



Pathogenesis of sepsis and septic shock



Normal Systemic Response to Infection and Injury (1)

- Leukocytosis Mobilizes neutrophils into the circulation
- Tachycardia Increases cardiac output, blood flow to injured tissue
- Fever Raises core temperature; peripheral vasoconstriction shunts blood flow to injured tissue. Occurs much more often when infection is the trigger for systemic responses

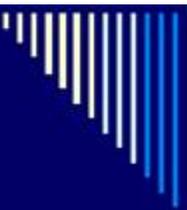


Normal Systemic Response to Infection and Injury (2)

□ Acute-Phase Responses

■ Anti-infective

- Increases synthesis of complement factors, microbe pattern-recognition molecules (mannose-binding lectin, LBP, CRP, CD14, Others)
- Sequesters iron (lactoferrin) and zinc (metallothionein)

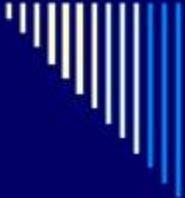


Normal Systemic Response to Infection and Injury (3)

□ Anti-inflammatory

- Releases anti-inflammatory neuroendocrine hormones (cortisol, ACTH, epinephrine, α -MSH)
 - Increases synthesis of proteins that help prevent inflammation within the systemic compartment
 - Cytokine antagonists (IL-1Ra, sTNF-Rs)
 - Anti-inflammatory mediators (e.g., IL-4, IL-6, IL-6R, IL-10, IL-13, TGF- β)
 - Protease inhibitors (e.g., α 1-antiprotease)
 - Antioxidants (haptoglobin)
- Reprograms circulating leukocytes (epinephrine, cortisol, PGE₂, ?other)

Mandell et al. Principles and Practice of Infectious Diseases 6th ed; 906:906-926.



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Normal Systemic Response to Infection and Injury (4)

- **Procoagulant**
 - Walls off infection, prevents systemic spread
- Increases synthesis or release of fibrinogen, PAI-1, C4b
- Decreases synthesis of protein C, anti-thrombin III
- **Metabolic**
 - Preserves euglycemia, mobilizes fatty acids, amino acids
 - Epinephrine, cortisol, glucagon, cytokines
- **Thermoregulatory**
 - Inhibits microbial growth
 - Fever

Risk factors of sepsis

- aggressive oncological chemotherapy and radiation therapy
- use of corticosteroid and immunosuppressive therapies for organ transplants and inflammatory diseases
- longer lives of patients predisposed to sepsis, the elderly, diabetics, cancer patients, patients with major organ failure, and with granulocytopenia.
- Neonates are more likely to develop sepsis (ex. group B Streptococcal infections).
- increased use of invasive devices such as surgical prostheses, inhalation equipment, and intravenous and urinary catheters.
- indiscriminate use of antimicrobial drugs that create conditions of overgrowth, colonization, and subsequent infection by aggressive, antimicrobial-resistant organisms.

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.

Source (usually an endogenous source of infection)

- intestinal tract
- oropharynx
- instrumentation sites
- contaminated inhalation therapy equipment
- IV fluids.
- **Most frequent sites of infection: Lungs, abdomen, and urinary tract.**
- Other sources include the skin/soft tissue and the CNS.

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.

Diagnosis

- **History**
 - community or nosocomially acquired infection
 - immunocompromised patient
 - exposure to animals, travel, tick bites, occupational hazards, alcohol use, seizures, loss of consciousness, medications
 - underlying diseases ; specific infectious agents
 - Some clues to a septic event include
 - Fever or unexplained signs with malignancy or instrumentation
 - Hypotension
 - Oliguria or anuria
 - Tachypnea or hyperpnea
 - Hypothermia without obvious cause
 - Bleeding

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.

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Specific Infectious agents

- Splenectomy (traumatic or functional)
 - *S pneumoniae*, *H influenzae*, *N meningitidis*
- Neutropenia (<500 neutrophil/ml)
 - Gram-negative, including *P aeruginosa*, gram-positives, including *S aureus*
 - Fungi, especially *Candida* species
- Hypogammaglobulinemia (e.g. CLL)
 - *S pneumoniae*, *E coli*
- Burns
 - MRSA, *P aeruginosa*, resistant gram-negatives

MacArthur RD, et al. *Mosby*, 2001:3-10.
Wheeler AP, et al. *NEJM* 1999;340:207-214.
Chaoagul W, et al. *J Infect Dis* 1989;159:890-899.

Specific Infectious agents

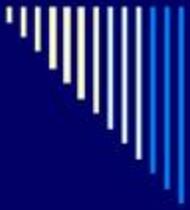
- Aids
 - *P. aeruginosa* (if neutropenic), *S. aureus*, PCP pneumonia
- Intravascular devices
 - *S. aureus*, *S. epidermidis*
- Nosocomial infections
 - MRSA, Enterococcus species, resistant gram-negative, Candida species
- Septic patients in NE of Thailand
 - *Burkholderia pseudomallei*



Diagnosis

- Physical Examination
 - essential
 - In all neutropenic patients and in patients with as suspected pelvic infection the physical exam should include rectal, pelvic, and genital examinations
 - perirectal, and/or perineal abscesses
 - pelvic inflammatory disease and/or abscesses, or prostatitis

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.



Signs and Symptoms

- Nonspecific symptoms of sepsis : not pathognomonic
 - fever
 - chills
 - constitutional symptoms of fatigue, malaise
 - anxiety or confusion
- absent symptoms in serious infections, especially in elderly individuals

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.



Complications

- Adult respiratory distress syndrome (ARDS)
- Disseminated Intravascular Coagulation (DIC)
- Acute Renal failure (ARF)
- Intestinal bleeding
- Liver failure
- Central Nervous System dysfunction
- Heart failure
- Death

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.

Diagnosis

- Before the initiation of antimicrobial therapy, at least two blood cultures should be obtained
 - At least one drawn percutaneously
 - At least one drawn through each vascular access device if inserted longer than 48 hours
- Other cultures such as urine, cerebrospinal fluid, wounds, respiratory secretions or other body fluids should be obtained as the clinical situation dictates
- Other diagnostic studies such as imaging and sampling should be performed promptly to determine the source and causative organism of the infection
 - may be limited by patient stability

[Weinstein MP. Rev Infect Dis 1983;5:35-53](#)

[Blot F. J Clin Microbiol 1999; 36: 105-109.](#)

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Weinstein MP. *Rev Infect Dis* 1983;5:35-53

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Sepsis management bundle

- Fluid resuscitation
- Appropriate cultures prior to antibiotic administration
- Early targeted antibiotics and source control
- Use of vasopressors/inotropes when fluid resuscitation optimized

Surviving Sepsis Campaign Management Guidelines Committee. *Crit Care Med* 2004; 32:858-873.



Sepsis management bundle

- ❑ Evaluation for adrenal insufficiency
- ❑ Stress dose corticosteroid administration
- ❑ Recombinant human activated protein C (xigris) for severe sepsis
- ❑ Low tidal volume mechanical ventilation for ARDS
- ❑ Tight glucose control

Surviving Sepsis Campaign Management Guidelines Committee. Crit Care Med 2004; 32:858-873.



Infection Control

- ❑ Appropriate cultures prior to antibiotic administration
- ❑ Early targeted antibiotics and source control

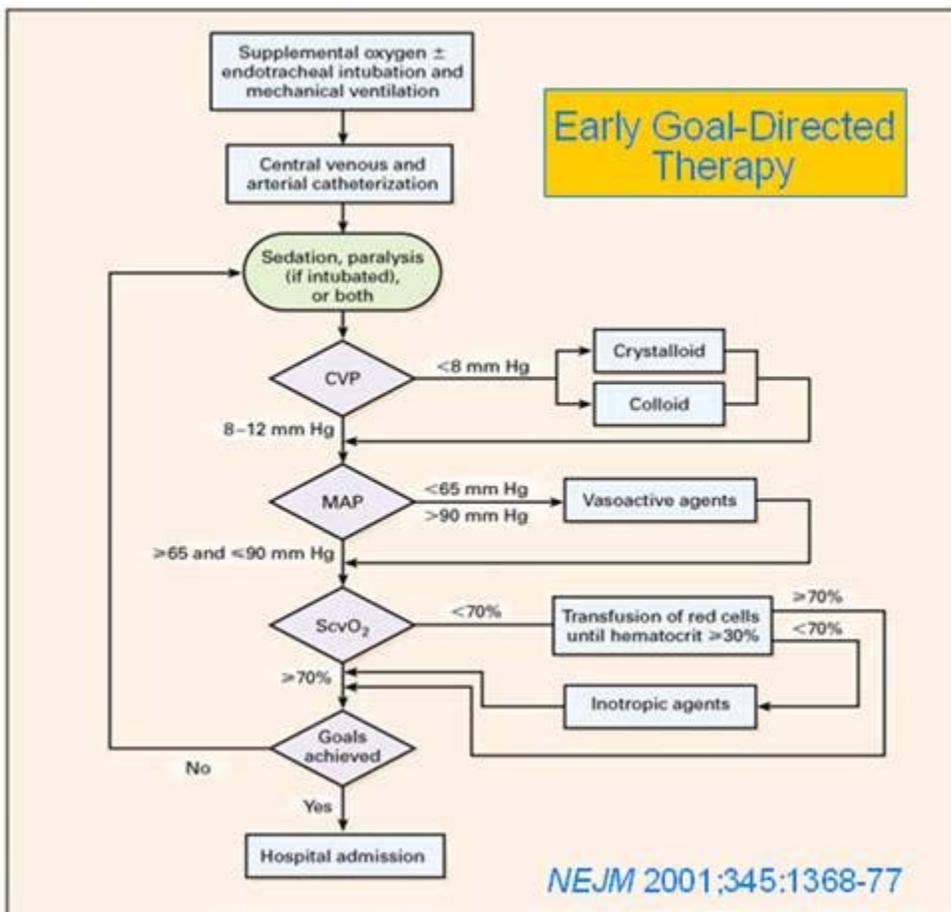
Surviving Sepsis Campaign Management Guidelines Committee. Crit Care Med 2004; 32:858-873.



CVP : central venous pressure

MAP : mean arterial pressure

ScvO₂ : central venous oxygen saturation



Antibiotic use in Sepsis (1)

- The drugs used depends on the source of the sepsis
- Community acquired pneumonia
 - third (ceftriaxone) or fourth (cefepime) generation cephalosporin is given with an aminoglycoside (usually gentamicin)
- Nosocomial pneumonia
 - Cefipime or Imipenem-cilastatin and an aminoglycoside
- Abdominal infection
 - Imipenem-cilastatin or Piperacillin-tazobactam and aminoglycoside

Angus DC, et al. Crit Care Med 2001, 29:1303-1310.



The Septic Patient

The term SIRS is used to describe the widely disseminated inflammatory reaction which can complicate a wide range of disorders eg, pancreatitis, trauma, ischaemia.

The term SEPSIS is used in those patients in whom SIRS is associated with proven infection





Systemic inflammatory response syndrome (SIRS)

- Cytokine mediators of SIRS: TNF, IL-1, IL-6, IL- 8
- Secondary inflammation mediators:
 - Arachidonic acid metabolites
 - Nitric oxide
 - Oxygen radicals
 - Platelet activating factor



Systemic inflammatory response syndrome (SIRS)

Systemic changes

- Loss of microvascular integrity
- Increased vascular permeability
- Systemic vasodilatation
- Depressed myocardial contractility
- Poor oxygen delivery
- Increased microvascular clotting



Early features of sepsis

- Fever or hypothermia
- Leucocytosis or leucopenia
- Tachycardia
- Tachypnoea
- Organ dysfunction: Brain - altered mental state
Lungs - hypoxia
Kidneys - oliguria

Nosocomial Infections (Hospital acquired infections)

(Gk: nosokomeion)

Gk: nosos- of disease; komeo – to nurse

- The patient in the ITU who has some degree of organ dysfunction is vulnerable to nosocomial infections.
- Good principles of infection control and avoidance of cross-infection by staff
- Bacteria in the GI tract of the patient is the commonest source
- Nosocomial pneumonia occurs from spillage from the upper GI tract into the lungs
- H₂ receptor antagonists encourages nosocomial infections
- Sucralfate used as stress ulcer prophylaxis is also bacteriostatic and thus reduces the incidence

Intra-abdominal sepsis Sub-phrenic abscess Management

- Resuscitation
- Confirmation of diagnosis
- Definitive treatment



Sub-phrenic abscess

“Pus somewhere, pus nowhere, pus under the diaphragm.”

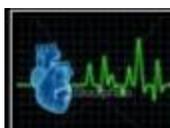
Investigations for confirmation

- Blood: Culture, FBC, CRP
- CXR
- Ultrasound
- ?CT



The Septic Patient Management

- Supportive measures:
 - Oxygenation
 - Ventilation if necessary
 - IV fluids
 - Inotropic support
 - Nutritional support
- Specific measures
 - Antibiotics
 - Drainage



Respiratory failure

Respiratory failure is defined as an arterial oxygen tension (PaO_2) at sea level of less than 8 kPa, i.e. hypoxia due inadequate gas exchange within the lung.



Emergency

- Multiple trauma (including burns)
- Leaking AAA
- Severe acute pancreatitis
- Post-operative complications:
 - Surgical
 - Cardiac
 - Respiratory
 - Renal
- Severe sepsis

Elective

- Major vascular eg, AAA
- Oesophagectomy
- Cardiac operations
- Major procedures
 - Whipple's
 - Patients in ASA 2 category or more

Indications for ICU transfer

- Potential incipient or actual organ failure in a remediable condition
- Advanced monitoring of organ function
- Treatment of organ failure:
 - Heart – use of inotropes
 - Lungs – ventilation
 - Kidneys – renal replacement therapy
- A need for 1:1 nursing

Respiratory

- **Pulse oximetry**
 - O₂ saturation of arterial blood
- **Capnography**
 - CO₂ tension in expired gas

Cardiovascular

- **Arterial lines**
- **CVP**
- **Pulmonary artery flotation catheter (PAFC)**
- **Cardiac output measurement**

Pulse oximetry

- **95% - 100% = normal**
- **93% = Warning! PaO₂ around 8.5 kPa**
- **< 90% = patient is in severe trouble**

ETCC Manual: RCSEd

Pulse oximetry

- Gives estimate of percentage saturation of oxygen binding sites
- Related to PaO_2 by oxygen dissociation curve

ETCC Manual: RCSEd

Capnography

- Infra-red absorption through gas stream
- Relies on rapid equilibration of CO_2 between alveolus and pulmonary capillary
- Useful guide to $PaCO_2$ but beware of lung disease
- Continuous measurement

Arterial line

Indications

- Continuous BP measurement
- Access for serial arterial blood gas analysis

Complications

- Bleeding
- Thrombosis
- Infection
- Pseudoaneurysm
- Accidental drug injection



Central venous catheterisation

Complications

- Inadvertent - arterial puncture
 - thoracic duct puncture
 - lung puncture
- Air embolus
- Catheter-related sepsis
- Clot formation
- Malposition and rupture of vein



ETCC Manual: RCSEd



Pulmonary artery wedge pressure (PWAP)

It is an accurate representation of the left atrial pressure which closely parallels the left ventricular end-diastolic pressure thus helping to guide fluid therapy.

ETCC Manual: RCSEd



Pulmonary artery wedge pressure

Introduced in 1970s by two cardiologists, Drs Swan and Ganz. Used to measure:

- Pressure within the pulmonary artery
- Pulmonary artery wedge pressure
- Cardiac output by thermodilution or dye dilution method
- Sampling of mixed venous blood

ETCC Manual: RCSEd



Pulmonary artery wedge pressure

Complications

- Valvular damage
- Ventricular rupture
- Pulmonary artery rupture
- Aneurysm or infarction
- Those of central venous catheterisation



Standard values

- Central venous pressure (CVP): 0-6 mm Hg
- Right ventricular pressure: 25 / 0-6mm Hg
- Pulmonary artery pressure (PAP): 25 / 6-12 mm Hg
- Wedge pressure (PAWP): 6-12 mm Hg
- Cardiac index (CI): $>2.8-3.6 \text{ L / min / m}^2$
- Systemic vascular resistance(SVR): $770-1500 \text{ dynes / sec / cm}^5$
- Oxygen delivery: $600 \text{ ml / min / m}^2$
- Oxygen consumption: $150 \text{ mls / min / m}^2$

ETCC Manual, 2005d



Respiratory failure

Respiratory distress

- Look
- Listen
- Feel

Anderson I D ed; Care of the Critically Ill Surgical Patient; RCSEng, Arnold 1999



Respiratory Failure

Type I

Hypoxia

Failed O₂ uptake

PaO₂ <8kPa (Hypoxia)

+

Normal PaCO₂ (7kPa) or low





◀ Respiratory Failure
Type II
Hypoxia + Hypercapnia

Failed O₂ uptake + Failed CO₂ removal

$$\text{PaO}_2 < 8\text{kPa} \\ + \\ \text{PaCO}_2 > 7\text{kPa}$$



Types

Factor affected

- | | |
|--------------|-----------------------------|
| ■ Hypoxic | ■ O ₂ saturation |
| ■ Anaemic | ■ Haemoglobin |
| ■ Stagnant | ■ Cardiac output |
| ■ Histotoxic | ■ Tissue utilisation |



Hypotension Causes

- Inadequate pre-load
- Decreased contractility

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Causes of inadequate pre-load in hypotension

Absolute reduction of fluid	Relative reduction of fluid
<ul style="list-style-type: none">■ Blood loss (obvious or occult)■ Dehydration with inadequate fluid replacement	<ul style="list-style-type: none">■ Venodilatation■ Mechanical interference<ul style="list-style-type: none">- tension pneumothorax- pulmonary embolism- IPPV / CPAP- tachycardia- arrhythmia

Critical states in surgical patients: acute respiratory distress syndrome, abdominal cavity syndrome, collapse.

Acute respiratory distress syndrome (ARDS), also known as respiratory distress syndrome (RDS) or adult respiratory distress syndrome (in contrast with IRDS) is a serious reaction to various forms of injuries to the lung.

ARDS is a severe lung disease caused by a variety of direct and indirect issues. It is characterized by inflammation of the lung parenchyma leading to impaired gas exchange with concomitant systemic release of inflammatory mediators causing inflammation, hypoxemia and frequently resulting in multiple organ failure. This condition is often fatal, usually requiring mechanical ventilation and admission to an intensive care unit. A less severe form is called acute lung injury (ALI).

ACUTE RESPIRATORY DISTRESS SYNDROME

Historical Perspective and Definitions

The first description of acute respiratory distress syndrome appeared in 1967, when Ashbaugh and colleagues described 12 patients with acute respiratory distress, cyanosis refractory to oxygen therapy, decreased lung compliance, and diffuse infiltrates evident on the chest radiograph. Initially called the adult respiratory distress syndrome, this entity is now termed the acute respiratory distress syndrome, since it does occur in children. Because the initial definition lacks specific criteria that could be used to identify patients systematically, there was controversy over the incidence and natural history of the syndrome and the mortality associated with it. In 1988, an expanded definition was proposed that quantified the physiologic respiratory impairment through the use of a four-point lung-injury scoring system that was based on the level of positive end-expiratory pressure, the ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen, the static lung compliance, and the degree of infiltration evident on chest radiographs. Other factors included in the assessment were the inciting clinical disorder and the presence or absence of nonpulmonary organ dysfunction

Although the lung-injury scoring system has been widely used to quantify the severity of lung injury in both clinical research and clinical trials, it cannot be used to predict the outcome during the first 24 to 72 hours after the onset of the acute respiratory distress syndrome and thus has limited clinical usefulness. When the scoring system is used four to seven days after the onset of the syndrome, scores of 2.5 or higher may be predictive of a complicated course with the need for prolonged mechanical ventilation.

In 1994, a new definition was recommended by the American–European Consensus Conference Committee. The consensus definition has two advantages. First, it recognizes that the severity of clinical lung injury varies: patients with less severe hypoxemia (as defined by a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen of 300 or less) are considered to have acute lung injury, and those with more severe hypoxemia (as defined by a ratio of 200 or less) are considered to have the acute respiratory distress syndrome. The recognition of patients with acute lung injury may facilitate earlier enrollment of affected patients in clinical trials. Second, the definition is simple to apply in the clinical setting. However, this simplicity is also a disadvantage, since factors that influence the outcome, such as the underlying cause and whether other organ systems are affected, do not need to be assessed. In addition, the criterion for the presence of bilateral infiltrates on chest radiography consistent with the presence of pulmonary edema is not sufficiently specific to be applied consistently by experienced clinicians. Nevertheless, the widespread acceptance of both the 1994 consensus definition and the 1988 lung-injury scoring system has improved the standardization of clinical research and trials. We recommend that clinicians routinely use the 1994 consensus definition to allow comparison of their patients with patients enrolled in clinical trials.

Epidemiology

Incidence

An accurate estimation of the incidence of acute lung injury and the acute respiratory distress syndrome has been hindered by the lack of a uniform definition and the heterogeneity of the causes and clinical manifestations. An early estimate by the National Institutes of Health (NIH) suggested that the annual incidence in the United States was 75 per 100,000 population. More recent studies reported lower incidences of 1.5 to 8.3 per 100,000. However, the first epidemiologic study to use the 1994 consensus definition reported considerably higher annual

incidences in Scandinavia: 17.9 per 100,000 for acute lung injury and 13.5 per 100,000 for the acute respiratory distress syndrome. On the basis of the results of screening of large numbers of patients by the NIH Acute Respiratory Distress Syndrome Network over the past three years, some investigators believe that the original estimate of 75 per 100,000 per year may be accurate. To settle this issue, a prospective epidemiologic study that is using the 1994 consensus definition is under way in Seattle.

ARDS Causes

•Direct Lung Injury:

- a) PNA and aspiration of gastric contents
or other causes of chemical pneumonitis
- b) pulmonary contusion, penetrating lung injury
- c) fat emboli
- d) near drowning
- e) inhalation injury
- f) reperfusion pulm edema after lung transplant

•Indirect lung injury

- a) sepsis
- b) severe trauma w/ shock hypoperfusion
- c) drug over dose
- d) cardiopulmonary bypass
- e) acute pancreatitis
- f) transfusion of multp blood products

ARDS was defined as the ratio of arterial partial oxygen tension (PaO₂) as fraction of inspired oxygen (FiO₂) below 200 mmHg in the presence of bilateral alveolar infiltrates on the chest x-ray. These infiltrates may appear similar to those of left ventricular failure, but the cardiac silhouette appears normal in ARDS. Also, the pulmonary capillary wedge pressure is normal (less than 18 mmHg) in ARDS, but raised in left ventricular failure.

A PaO₂/FiO₂ ratio less than 300 mmHg with bilateral infiltrates indicates acute lung injury (ALI). Although formally considered different from ARDS, ALI is usually just a precursor to ARDS. (Consensus after 1967 and 1994)

ARDS is characterized by

Acute onset

Bilateral infiltrates on chest radiograph sparing costophrenic angles

Pulmonary artery wedge pressure < 18 mmHg (obtained by pulmonary artery catheterization), if this information is available; if unavailable, then lack of clinical evidence of left ventricular failure suffices:

if PaO₂:FiO₂ < 300 mmHg (40 kPa) acute lung injury (ALI) is considered to be present

if PaO₂:FiO₂ < 200 mmHg (26.7 kPa) acute respiratory distress syndrome (ARDS) is considered to be present

To summarize and simplify, ARDS is an acute (rapid onset) syndrome (collection of symptoms) that affects the lungs widely and results in a severe oxygenation defect, but is not heart failure.

ARDS Symptoms

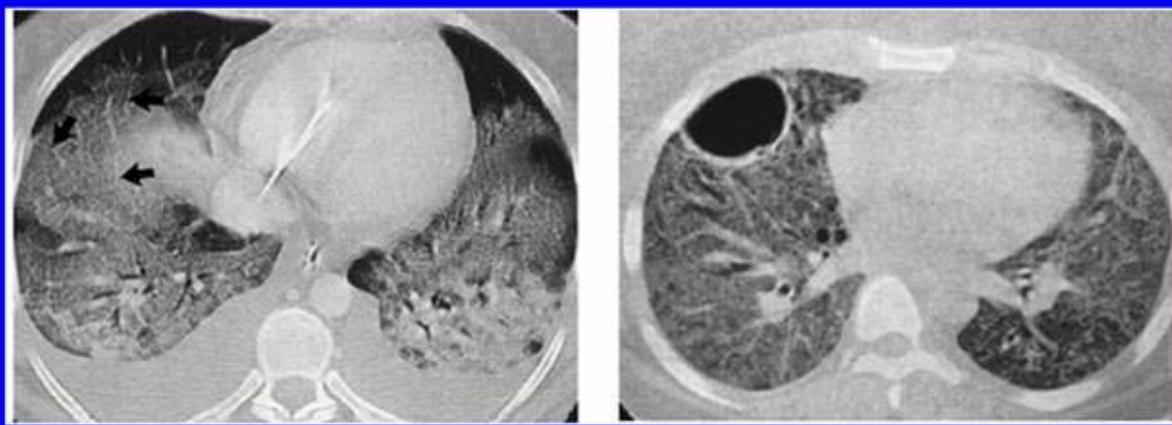
- Severe difficulty in breathing
- [Anxiety](#)
- Agitation
- [Fever](#)

Clinical, Pathological, and Radiographic Features

The definitions discussed above identify patients early in the course of acute lung injury and

the acute respiratory distress syndrome. However, the syndrome is often progressive, characterized by distinct stages with different clinical, histopathological, and radiographic manifestations. The acute, or exudative, phase is manifested by the rapid onset of respiratory failure in a patient with a risk factor for the condition. Arterial hypoxemia that is refractory to treatment with supplemental oxygen is a characteristic feature.

ARDS exudative and fibrotic phases



An arterial blood gas analysis and chest X-ray allow formal diagnosis by the aforementioned criteria. Although severe hypoxemia is generally included, the appropriate threshold defining abnormal PaO₂ has never been systematically studied. Note though, that a severe oxygenation defect is not synonymous with ventilatory support. Any PaO₂ below 100 (generally saturation less than 100%) on a supplemental oxygen fraction of 50% meets criteria for ARDS. This can easily be achieved by high flow oxygen supplementation without ventilatory support.

Any cardiogenic cause of pulmonary edema should be excluded. This can be done by placing a pulmonary artery catheter for measuring the pulmonary artery wedge pressure. However, this is not necessary and is now rarely done as abundant evidence has emerged demonstrating that the use of pulmonary artery catheters does not lead to improved patient outcomes in critical illness including ARDS.

Plain Chest X-rays are sufficient to document bilateral alveolar infiltrates in the majority of

cases. While CT scanning leads to more accurate images of the pulmonary parenchyma in ARDS, it has little utility in the clinical management of patients with ARDS, and remains largely a research tool.

Radiographically, the findings are indistinguishable from those of cardiogenic pulmonary edema. Bilateral infiltrates may be patchy or asymmetric and may include pleural effusions. Computed tomographic scanning has demonstrated that alveolar filling, consolidation, and atelectasis occur predominantly in dependent lung zones, whereas other areas may be relatively spared. However, bronchoalveolar-lavage studies indicate that even radiographically spared, nondependent areas may have substantial inflammation. Pathological findings include diffuse alveolar damage, with neutrophils, macrophages, erythrocytes, hyaline membranes, and protein-rich edema fluid in the alveolar spaces, capillary injury, and disruption of the alveolar epithelium.

Although acute lung injury and the acute respiratory distress syndrome may resolve completely in some patients after the acute phase, in others it progresses to fibrosing alveolitis with persistent hypoxemia, increased alveolar dead space, and a further decrease in pulmonary compliance. Pulmonary hypertension, owing to obliteration of the pulmonary-capillary bed, may be severe and may lead to right ventricular failure. Chest radiographs show linear opacities, consistent with the presence of evolving fibrosis. Pneumothorax may occur, but the incidence is only 10 to 13 percent and is not clearly related to airway pressures or the level of positive end-expiratory pressure. Computed tomography of the chest shows diffuse interstitial opacities and bullae. Histologically, there is fibrosis along with acute and chronic inflammatory cells and partial resolution of the pulmonary edema

The recovery phase is characterized by the gradual resolution of hypoxemia and improved lung compliance. Typically, the radiographic abnormalities resolve completely. The degree of histologic resolution of fibrosis has not been well characterized, although in many patients pulmonary function returns to normal.

Clinical Disorders and Risk Factors

The ability to identify patients at risk for acute lung injury and the acute respiratory distress syndrome is important if therapies are to be developed to prevent the disorder. The commonly

associated clinical disorders can be divided into those associated with direct injury to the lung and those that cause indirect lung injury in the setting of a systemic process.

Overall, sepsis is associated with the highest risk of progression to acute lung injury or the acute respiratory distress syndrome, approximately 40 percent. The presence of multiple predisposing disorders substantially increases the risk, as does the presence of secondary factors including chronic alcohol abuse, chronic lung disease, and a low serum pH.

Outcomes

Until recently, most studies of acute lung injury and the acute respiratory distress syndrome have reported a mortality rate of 40 to 60 percent. The majority of deaths are attributable to sepsis or multiorgan dysfunction rather than primary respiratory causes, although the recent therapeutic success of ventilation with low tidal volumes indicates that in some cases death is directly related to lung injury. Two reports suggest that mortality from this disease may be decreasing. The first, from a large county hospital in Seattle, found that the mortality rate was 36 percent in 1993 as compared with rates of 53 to 68 percent in the period from 1983 to 1987. The second, from a hospital in the United Kingdom, reported a decline in the mortality rate from 66 percent to 34 percent between 1990 to 1993 and 1994 to 1997. Possible explanations for the decrease include more effective treatments for sepsis, changes in the method of mechanical ventilation, and improvement in the supportive care of critically ill patients. The possibility that mortality is decreasing emphasizes the importance of the use of randomized control subjects rather than historical controls in clinical studies of the disorder.

Factors whose presence can be used to predict the risk of death at the time of diagnosis of acute lung injury and the acute respiratory distress syndrome include chronic liver disease, nonpulmonary organ dysfunction, sepsis, and advanced age. Surprisingly, initial indexes of oxygenation and ventilation, including the ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen and the lung-injury score, do not predict outcome. In three large studies, the mortality rate among patients with an initial ratio of partial pressure of arterial oxygen to fraction of inspired oxygen of 300 or less was similar to that among patients with a ratio of 200 or less. However, the failure of pulmonary function to improve during the first week of treatment is a negative prognostic factor.

In most patients who survive, pulmonary function returns nearly to normal within 6 to 12 months, despite the severe injury to the lung. Residual impairment of pulmonary mechanics

may include mild restriction, obstruction, impairment of the diffusing capacity for carbon monoxide, or gas-exchange abnormalities with exercise, but these abnormalities are usually asymptomatic. Severe disease and prolonged mechanical ventilation identify patients at highest risk for persistent abnormalities of pulmonary function. Those who survive the illness have a reduced health-related quality of life as well as pulmonary-disease-specific health-related quality of life.

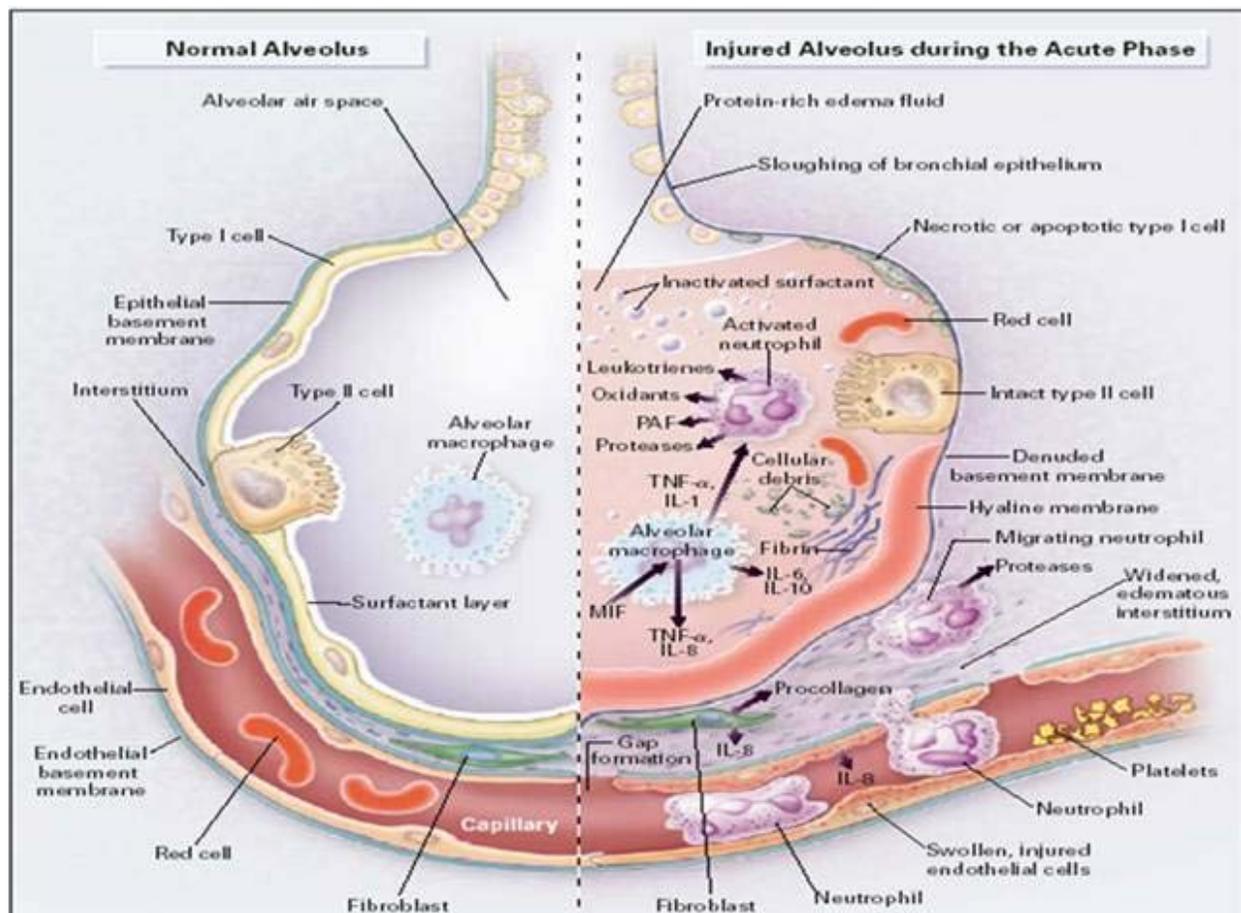
Pathogenesis

Stages of ARDS

1. Exudative (acute) phase - 0- 4 days
2. Proliferative phase - 4- 8 days
3. Fibrotic phase - >8 days
4. Recovery

Endothelial and Epithelial Injury

Two separate barriers form the alveolar–capillary barrier, the microvascular endothelium and the alveolar epithelium. The acute phase of acute lung injury and the acute respiratory distress syndrome is characterized by the influx of protein-rich edema fluid into the air spaces as a consequence of increased permeability of the alveolar–capillary barrier. The importance of endothelial injury and increased vascular permeability to the formation of pulmonary edema in this disorder has been well established.



The critical importance of epithelial injury to both the development of and recovery from the disorder has become better recognized. The degree of alveolar epithelial injury is an important predictor of the outcome. The normal alveolar epithelium is composed of two types of cells. Flat type I cells make up 90 percent of the alveolar surface area and are easily injured. Cuboidal type II cells make up the remaining 10 percent of the alveolar surface area and are more resistant to injury; their functions include surfactant production, ion transport, and proliferation and differentiation to type I cells after injury.

The loss of epithelial integrity in acute lung injury and the acute respiratory distress syndrome has a number of consequences. First, under normal conditions, the epithelial barrier is much less permeable than the endothelial barrier. Thus, epithelial injury can contribute to alveolar flooding. Second, the loss of epithelial integrity and injury to type II cells disrupt normal epithelial fluid transport, impairing the removal of edema fluid from the alveolar space. Third, injury to type II cells reduces the production and turnover of surfactant, contributing to the characteristic surfactant abnormalities. Fourth, loss of the epithelial barrier can lead to septic shock in patients with bacterial pneumonia. Finally, if injury to the alveolar epithelium is severe, disorganized or insufficient epithelial repair may lead to fibrosis.

Neutrophil-Dependent Lung Injury

Clinical and experimental studies have provided circumstantial evidence of the occurrence of neutrophil-mediated injury in acute lung injury and the acute respiratory distress syndrome. Histologic studies of lung specimens obtained early in the course of the disorder show a marked accumulation of neutrophils. Neutrophils predominate in the pulmonary edema fluid and bronchoalveolar-lavage fluid obtained from affected patients, and many animal models of acute lung injury are neutrophil-dependent. Some of the mechanisms of the sequestration and activation of neutrophils and of neutrophil-mediated lung injury are summarized in.

New evidence raises the question of whether neutrophilic inflammation is the cause or the result of lung injury. Acute lung injury and the acute respiratory distress syndrome may develop in patients with profound neutropenia, and some animal models of acute lung injury are neutrophil-independent. In clinical trials in which patients with severe pneumonia received granulocyte colony-stimulating factor in order to increase the number of circulating neutrophils, the incidence or severity of lung injury did not increase. The neutrophil has a critical role in host defense in this disorder, a factor that may explain, in part, why antiinflammatory strategies have largely been unsuccessful.

Other Proinflammatory Mechanisms

Inflammation

Inflammation alone, as in sepsis, causes endothelial dysfunction, fluid extravasation from the capillaries and impaired drainage of fluid from the lungs. Dysfunction of type II pulmonary epithelial cells may also be present, with a concomitant reduction in surfactant production. Elevated inspired oxygen concentration often becomes necessary at this stage, and they may facilitate a 'respiratory burst' in immune cells.

In a secondary phase, endothelial dysfunction causes cells and inflammatory exudate to enter the alveoli. This pulmonary edema increases the thickness of the alveolo-capillary space, increasing the distance the oxygen must diffuse to reach blood. This impairs gas exchange leading to hypoxia, increases the work of breathing, eventually induces fibrosis of the airspace.

Moreover, edema and decreased surfactant production by type II pneumocytes may cause whole alveoli to collapse, or to completely flood. This loss of aeration contributes further to the right-

to-left shunt in ARDS. As the alveoli contain progressively less gas, more blood flows through them without being oxygenated resulting in massive intrapulmonary shunting.

Collapsed alveoli (and small bronchi) do not allow gas exchange. It is not uncommon to see patients with a PaO₂ of 60 mmHg (8.0 kPa) despite mechanical ventilation with 100% inspired oxygen.

The loss of aeration may follow different patterns according to the nature of the underlying disease, and other factors. In pneumonia-induced ARDS, for example, large, more commonly causes relatively compact areas of alveolar infiltrates. These are usually distributed to the lower lobes, in their posterior segments, and they roughly correspond to the initial infected area.

In sepsis or trauma-induced ARDS, infiltrates are usually more patchy and diffuse. The posterior and basal segments are always more affected, but the distribution is even less homogeneous.

Loss of aeration also causes important changes in lung mechanical properties. These alterations are fundamental in the process of inflammation amplification and progression to ARDS in mechanically ventilated patients.

Cytokines

A complex network of cytokines and other proinflammatory compounds initiate and amplify the inflammatory response in acute lung injury and the acute respiratory distress syndrome. Proinflammatory cytokines may be produced locally in the lung by inflammatory cells, lung epithelial cells, or fibroblasts. The regulation of cytokine production by extrapulmonary factors has also been described. Macrophage inhibitory factor is a regulatory cytokine produced by the anterior pituitary that is found in high concentrations in the bronchoalveolar-lavage fluid of patients with the syndrome. This cytokine increases production of the proinflammatory cytokines interleukin-8 and tumor necrosis factor α and can override glucocorticoid-mediated inhibition of cytokine secretion.

New evidence indicates that it is not only the production of proinflammatory cytokines that is important, but also the balance between proinflammatory and antiinflammatory mediators. Several endogenous inhibitors of proinflammatory cytokines have been described, including

interleukin-1–receptor antagonist, soluble tumor necrosis factor receptor, autoantibodies against interleukin-8, and antiinflammatory cytokines such as interleukin-10 and 11. Better understanding of the role of cytokines in acute lung injury and the acute respiratory distress syndrome will be gained through studies of the biologic activity of specific cytokines, rather than by an assessment of static levels by immunologic methods.

Ventilator-Induced Lung Injury

Mechanical ventilation is an essential part of the treatment of ARDS. As loss of aeration (and the underlying disease) progress, the work of breathing (WOB) eventually grows to a level incompatible with life. Thus, mechanical ventilation is initiated to relieve respiratory muscles of their work, and to protect the usually obtunded patient's airways.

Older studies focused on the potential toxic effects of high fractions of inspired oxygen, but experimental evidence indicates that mechanical ventilation at high volumes and pressures can injure the lung, causing increased permeability pulmonary edema in the uninjured lung and enhanced edema in the injured lung. Initial theories formulated to explain these deleterious effects focused on capillary stress failure due to alveolar overdistention. More recently, cyclic opening and closing of atelectatic alveoli during mechanical ventilation have been shown to cause lung injury independently of alveolar overdistention. Alveolar overdistention coupled with the repeated collapse and reopening of alveoli can initiate a cascade of proinflammatory cytokines.

However, mechanical ventilation may constitute a risk factor for the development, or the worsening, of ARDS.

Aside from the infectious complications arising from invasive ventilation with tracheal intubation, positive-pressure ventilation directly alters lung mechanics during ARDS. The result is higher mortality, i.e. through baro-trauma, when these techniques are used.

In 1998, Amato et al. published a paper showing substantial improvement in the outcome of patients ventilated with lower tidal volumes (V_t) ($6 \text{ mL}\cdot\text{kg}^{-1}$). This result was confirmed in a 2000 study sponsored by the NIH. Although both these studies were widely criticized for several reasons, and although the authors were not the first to experiment lower-volume ventilation, they shed new light on the relationship between mechanical ventilation and ARDS.

One opinion is that the forces applied to the lung by the ventilator may work as a lever to

induce further damage to lung parenchyma. It appears that shear stress at the interface between collapsed and aerated units may result in the breakdown of aerated units, which inflate asymmetrically due to the 'stickiness' of surrounding flooded alveoli. The fewer such interfaces around an alveolus, the lesser the stress.

Indeed, even relatively low stress forces may induce signal transduction systems at the cellular level, thus inducing the release of inflammatory mediators.

This form of stress is thought to be applied by the transpulmonary pressure (gradient) (PI) generated by the ventilator or, better, its cyclical variations. The better outcome obtained in patients ventilated with lower Vt may be interpreted as a beneficial effect of the lower PI. Transpulmonary pressure, is an indirect function of the Vt setting on the ventilator, and only trial patients with plateau pressures (a surrogate for the actual PI) were less than 32 cmH₂O (3.1 kPa) had improved survival.

The way PI is applied on alveolar surface determines the shear stress to which lung units are exposed. ARDS is characterized by a usually inhomogeneous reduction of the airspace, and thus by a tendency towards higher PI at the same Vt, and towards higher stress on less diseased units.

The inhomogeneity of alveoli at different stages of disease is further increased by the gravitational gradient to which they are exposed, and the different perfusion pressures at which blood flows through them. Finally, abdominal pressure exerts an additional pressure on inferoposterior lung segments, favoring compression and collapse of those units.

The different mechanical properties of alveoli in ARDS may be interpreted as having varying time constants (the product of alveolar compliance × resistance). A long time constant indicates an alveolus which opens slowly during tidal inflation, as a consequence of contrasting pressure around it, or altered water-air interface inside it (loss of surfactant, flooding).

Slow alveoli are said to be 'kept open' using positive end-expiratory pressure, a feature of modern ventilators which maintains a positive airway pressure throughout the whole respiratory cycle. A higher mean pressure cycle-wide slows the collapse of diseased units, but it has to be weighed against the corresponding elevation in PI/plateau pressure. Newer ventilatory approaches attempt to maximize mean airway pressure for its ability to 'recruit' collapsed lung units while minimizing the shear stress caused by frequent openings and closings of aerated

units.

The prone position also reduces the inhomogeneity in alveolar time constants induced by gravity and edema. If clinically appropriate, mobilization of the ventilated patient can assist in achieving the same goal.

In patients with acute lung injury and the acute respiratory distress syndrome, ventilation at traditional tidal volumes (10 to 15 ml per kilogram of predicted body weight) may overdistend uninjured alveoli, perhaps promoting further lung injury, inhibiting resolution of the disorder, and contributing to multiorgan failure. The failure of traditional ventilatory strategies to prevent end-expiratory closure of atelectatic alveoli may also contribute to lung injury. These issues have led to a number of clinical trials of protective ventilatory strategies to reduce alveolar overdistention and increase the recruitment of atelectatic alveoli. Interestingly, a recent study found that a strategy of protective ventilation could reduce both the pulmonary and the systemic cytokine response.

Other Mechanisms of Injury

Like any form of inflammation, acute lung injury and the acute respiratory distress syndrome represent a complex process in which multiple pathways can propagate or inhibit lung injury. For example, abnormalities of the coagulation system often develop, leading to platelet–fibrin thrombi in small vessels and impaired fibrinolysis within the distal air spaces of the injured lung. Also, abnormalities in the production, composition, and function of surfactant probably contribute to alveolar collapse and gas-exchange abnormalities.

Fibrosing Alveolitis

After the acute phase of acute lung injury and the acute respiratory distress syndrome, some patients have an uncomplicated course and rapid resolution of the disorder. Others have progression to fibrotic lung injury, and such injury can be observed histologically as early as five to seven days after the onset of the disorder. The alveolar space becomes filled with mesenchymal cells and their products, along with new blood vessels. The finding of fibrosing alveolitis on histologic analysis correlates with an increased risk of death, and patients who die of the condition have a marked accumulation of collagen and fibronectin in the lung at autopsy.

The process of fibrosing alveolitis apparently begins early in the course of the disorder and may be promoted by early proinflammatory mediators such as interleukin-1. Levels of

procollagen III peptide, a precursor of collagen synthesis, are elevated in the alveolar compartment very early in the course of the illness, even at the time of intubation and the initiation of mechanical ventilation. Furthermore, the early appearance of procollagen III in the alveolar space is associated with an increased risk of death.

Resolution

Strategies that hasten the resolution of the illness may ultimately be as important as those that attenuate early inflammatory lung injury. Alveolar edema is resolved by the active transport of sodium and perhaps chloride from the distal air spaces into the lung interstitium. Water follows passively, probably through transcellular water channels, the aquaporins, located primarily on type I cells. In clinical studies, clearance of alveolar fluid can occur surprisingly early and is often apparent within the first few hours after intubation and the initiation of mechanical ventilation. Maintenance of the ability to remove alveolar fluid is associated with improved oxygenation, a shorter duration of mechanical ventilation, and an increased likelihood of survival.

A considerable quantity of both soluble and insoluble protein must also be removed from the air spaces. The removal of insoluble protein is particularly important, since hyaline membranes provide a framework for the growth of fibrous tissue. Soluble protein appears to be removed primarily by diffusion between alveolar epithelial cells. Insoluble protein may be removed by endocytosis and transcytosis by alveolar epithelial cells and by phagocytosis by macrophages

The alveolar epithelial type II cell is the progenitor for reepithelialization of a denuded alveolar epithelium. Type II cells proliferate to cover the denuded basement membrane and then differentiate into type I cells, restoring the normal alveolar architecture and increasing the fluid-transport capacity of the alveolar epithelium. This proliferation is controlled by epithelial growth factors, including keratinocyte and hepatocyte growth factors.

The mechanisms underlying the resolution of the inflammatory-cell infiltrate and fibrosis are unclear. Apoptosis (programmed cell death) is thought to be a major mechanism for the clearance of neutrophils from sites of inflammation and may be important in the clearance of neutrophils from the injured lung. However, in one study of bronchoalveolar-lavage fluid from patients with acute lung injury and the acute respiratory distress syndrome, the numbers of apoptotic neutrophils were low, perhaps because of the presence of antiapoptotic factors such as granulocyte colony-stimulating factor and granulocyte-macrophage colony-stimulating

factor. Nevertheless, high concentrations of the markers of apoptosis are present in the pulmonary edema fluid of patients, and exposure in vitro to bronchoalveolar-lavage fluids from these patients can promote epithelial-cell apoptosis. These are potentially important observations, since the mechanisms that alter epithelial integrity need to be identified. The role of proapoptotic and antiapoptotic mechanisms in both the injury and repair of the alveolar epithelium and the lung endothelium is an important area for future research.

Exams and Tests for ARDS

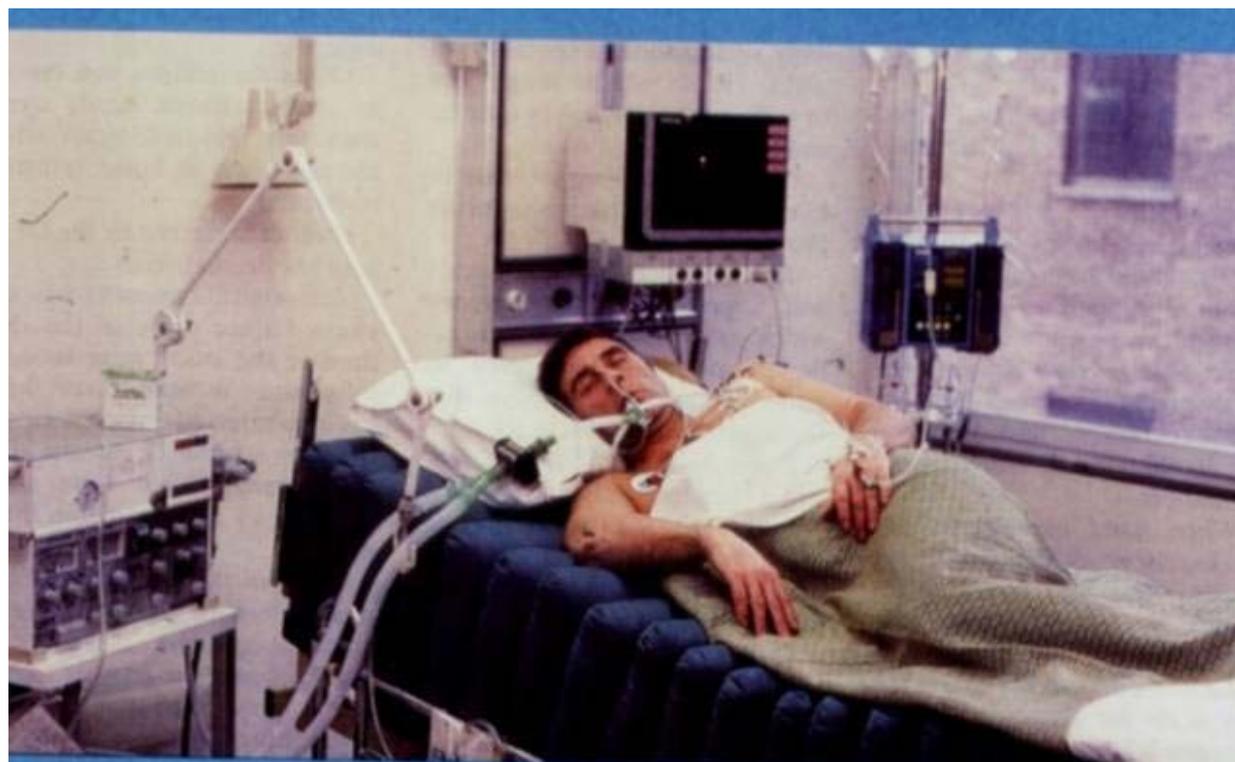
- [Arterial blood gas](#) analysis reveals hypoxemia (reduced levels of oxygen in the blood).
- A [complete blood count](#) may be taken. The number of white blood cells is increased in sepsis.
- [Chest x-ray](#) will show the presence of fluid in the lungs.
- [CT scan](#) of the chest may be required only in some situations (routine chest x-ray is sufficient in most cases).
- Echocardiogram (an [ultrasound](#) of the [heart](#)) may help exclude any heart problems that can cause fluid build-up in the lung.
- Monitoring with a [pulmonary artery catheter](#) may be done to exclude a [cardiac](#) cause for the difficulty in breathing.
- [Bronchoscopy](#) (a procedure used to look inside the [windpipe](#) and large airways of the lung) may be considered to evaluate the possibility of lung infection.

Treatment

Approach to Treatment

Improvement in the supportive care of patients with acute lung injury and the acute respiratory distress syndrome may have contributed to the recent decline in the mortality rate. There should be a careful search for the underlying cause, with particular attention paid to the possibility of

treatable infections such as sepsis or pneumonia. Abdominal infections should be treated promptly with antimicrobial agents or surgery. Prevention or early treatment of nosocomial infections is critical, since patients frequently die of uncontrolled infection. Adequate nutrition through the use of enteral feeding is preferred to parenteral nutrition since this route does not carry the serious risk of catheter-induced sepsis. Prevention of gastrointestinal bleeding and thromboembolism is also important.



An improved understanding of the pathogenesis of acute lung injury and the acute respiratory distress syndrome has led to the assessment of several novel treatment strategies. Although many specific therapies have not proved beneficial, it is encouraging that the quality of clinical trials is improving. An important advance has been the establishment of a network supported by the NIH that includes 10 centers, 24 hospitals, and 75 intensive care units and that provides the infrastructure for well-designed, multicenter, randomized trials of potential new therapies.

General

Acute respiratory distress syndrome is usually treated with mechanical ventilation in the Intensive Care Unit. Ventilation is usually delivered through oro-tracheal intubation, or tracheostomy whenever prolonged ventilation (≥ 2 weeks) is deemed inevitable.

The possibilities of non-invasive ventilation are limited to the very early period of the disease

or, better, to prevention in individuals at risk for the development of the disease (atypical pneumonias, pulmonary contusion, major surgery patients).

Treatment of the underlying cause is imperative, as it tends to maintain the ARDS picture.

Appropriate antibiotic therapy must be administered as soon as microbiological culture results are available. Empirical therapy may be appropriate if local microbiological surveillance is efficient. More than 60% ARDS patients experience a (nosocomial) pulmonary infection either before or after the onset of lung injury.

The origin of infection, when surgically treatable, must be operated on. When sepsis is diagnosed, appropriate local protocols should be enacted.

Commonly used supportive therapy includes particular techniques of mechanical ventilation and pharmacological agents whose effectiveness with respect to the outcome has not yet been proven. It is now debated whether mechanical ventilation is to be considered mere supportive therapy or actual treatment, since it may substantially affect survival.

Mechanical Ventilation

The most appropriate method of mechanical ventilation in the acute respiratory distress syndrome has been controversial since the syndrome was first described. Although the tidal volume in normal persons at rest is 6 to 7 ml per kilogram, historically a volume of 12 to 15 ml per kilogram was recommended in patients with acute lung injury and the acute respiratory distress syndrome. This comparatively high tidal volume may cause further lung injury. Interestingly, the possibility of ventilator-associated lung injury was first considered in the 1970s, leading to a study of extracorporeal membrane oxygenation in which the tidal volume was reduced to 8 to 9 ml per kilogram. However, this strategy, like extracorporeal removal of carbon dioxide in a subsequent study, failed to decrease mortality.

As described in this issue of the *Journal*, the NIH Acute Respiratory Distress Syndrome Network compared a traditional tidal volume (12 ml per kilogram of predicted body weight) with a lower tidal volume (6 ml per kilogram of predicted body weight) in 861 patients. In the group receiving lower tidal volumes, plateau pressure (airway pressure measured after a 0.5-second pause at the end of inspiration) could not exceed 30 cm of water and a detailed

protocol was used to adjust the fraction of inspired oxygen and positive end-expiratory pressure. The in-hospital mortality rate was 39.8 percent in the group treated with traditional tidal volumes and 31.0 percent in the group treated with lower tidal volumes ($P=0.007$). Thus, mortality was reduced by 22 percent in the group treated with lower tidal volumes, a finding of major importance. This large multicenter trial provides convincing evidence that a specific therapy for the acute respiratory distress syndrome can reduce mortality. It also provides evidence of the clinical significance of ventilator-associated lung injury and provides a well-defined protocol for ventilation against which future strategies can be compared.

The positive results of this trial differed from those of two previous studies of low tidal volumes, a Canadian study of 120 patients and a European study of 116 patients. There are several possible explanations for the discrepant results. First, the NIH study had the lowest tidal volume when the tidal volumes were compared with the use of the same calculation of ideal body weight. Thus, the NIH study may have been better able to show a difference between the treatment groups. Second, the study treated respiratory acidosis associated with alveolar hypoventilation and hypercapnia by allowing the respiratory rate to increase to 35 breaths per minute and by the administration of sodium bicarbonate. Conceivably, respiratory acidosis could have had deleterious effects in the groups treated with low tidal volumes in the other two studies. Finally, the other studies had many fewer patients, thus reducing the statistical power to find a treatment effect.

APRV (Airway Pressure Release Ventilation) and ARDS / ALI

No particular ventilator mode is known to improve mortality in ARDS. The landmark ARDSNet trial used a volume controlled mode and showed decrease mortality with smaller volumes. However, other modes of ventilation have not been directly compared to volume controlled ventilation.

Some practitioners favor airway pressure release ventilation (APRV). Advantages to APRV ventilation include: decreased airway pressures, decreased minute ventilation, decreased dead-space ventilation, promotion of spontaneous breathing, almost 24 hour a day alveolar recruitment, decreased use of sedation, near elimination of neuromuscular blockade, optimized arterial blood gas results, mechanical restoration of FRC (functional residual capacity), a positive effect on cardiac output (due to the negative inflection from the elevated baseline with each spontaneous breath), increased organ and tissue perfusion, potential for increased urine

output secondary to increased renal perfusion.

A patient with ARDS on average spends 8 to 11 days on a mechanical ventilator; APRV may reduce this time significantly and conserve valuable resources.

A study is needed to evaluate whether APRV will reduce patient mortality when compared to the ARDSNet protocol. However, there seems to be little political will, within the medical community, to address the need for this study, in spite of the clinical successes seen with APRV.

Positive end-expiratory pressure

There has also been considerable interest in the optimal level of positive end-expiratory pressure in patients with acute lung injury and the acute respiratory distress syndrome. It was noted early on that the use of positive end-expiratory pressure could improve oxygenation in these patients, allowing the fraction of inspired oxygen to be reduced. The best-documented effect of positive end-expiratory pressure on lung function is an increase in functional residual capacity, probably as a result of the recruitment of collapsed alveoli. Although lung injury was prevented in rats by the prophylactic use of positive end-expiratory pressure, the prophylactic use of a positive end-expiratory pressure of 8 cm of water in patients at risk for the acute respiratory distress syndrome was not successful.

Recently, Amato et al. used an “open-lung” approach to mechanical ventilation in patients with acute lung injury and the acute respiratory distress syndrome. In addition to a low tidal volume and pressure-controlled inverse-ratio ventilation, the protocol included raising the level of positive end-expiratory pressure above the lower inflection point on a pressure–volume curve for each patient in an attempt to ensure adequate recruitment of atelectatic lung. With this approach, mortality was reduced. However, the adoption of this approach cannot yet be recommended for several reasons. First, this study was small, involving only 53 patients and only a single center. Second, mortality in the group treated with conventional ventilation was unusually high (71 percent), suggesting that the high tidal volume used may have been especially injurious. Furthermore, the difference in mortality between the two groups was only apparent at 28 days; the rates of survival until hospital discharge were not significantly different between the two groups. Third, a reliable measurement of the lower inflection point of

the pressure–volume curve is technically difficult and usually requires sedation and paralysis of the patient.

Positive end-expiratory pressure (PEEP) is used in mechanically-ventilated patients with ARDS to improve oxygenation. In ARDS, three populations of alveoli can be distinguished. There are normal alveoli which are always inflated and engaging in gas exchange, flooded alveoli which can never, under any ventilatory regime, be used for gas exchange, and atelectatic or partially flooded alveoli that can be "recruited" to participate in gas exchange under certain ventilatory regimes. The recruitable alveoli represent a continuous population, some of which can be recruited with minimal PEEP, and others which can only be recruited with high levels of PEEP. An additional complication is that some or perhaps most alveoli can only be opened with higher airway pressures than are needed to keep them open. Hence the justification for maneuvers where PEEP is increased to very high levels for seconds to minutes before dropping the PEEP to a lower level. Finally, PEEP can be harmful. High PEEP necessarily increases mean airway pressure and alveolar pressure. This in turn can damage normal alveoli by overdistension resulting in DAD.

The 'best PEEP' used to be defined as 'some' cmH₂O above the lower inflection point (LIP) in the sigmoidal pressure-volume relationship curve of the lung. Recent research has shown that the LIP-point pressure is no better than any pressure above it, as recruitment of collapsed alveoli, and more importantly the overdistension of aerated units, occur throughout the whole inflation. Despite the awkwardness of most procedures used to trace the pressure-volume curve, it is still used by some to define the minimum PEEP to be applied to their patients. Some of the newest ventilators have the ability to automatically plot a pressure-volume curve. The possibility of having an 'instantaneous' tracing trigger might produce renewed interest in this analysis.

PEEP may also be set empirically. Some authors suggest performing a 'recruiting maneuver' (i.e., a short time at a very high continuous positive airway pressure, such as 50 cmH₂O (4.9 kPa), to recruit, or open, collapsed units with a high distending pressure) before restoring previous ventilation. The final PEEP level should be the one just before the drop in PaO₂ (or peripheral blood oxygen saturation) during a step-down trial.

Intrinsic PEEP (iPEEP), or auto-PEEP, first described by John Marini of St. Paul Regions

Hospital, is a potentially unrecognized contributor to PEEP in patients. When ventilating at high frequencies, its contribution can be substantial, particularly in patients with obstructive lung disease. iPEEP has been measured in very few formal studies on ventilation in ARDS patients, and its contribution is largely unknown. Its measurement is recommended in the treatment of ARDS patients, especially when using high-frequency (oscillatory/jet) ventilation.

A compromise between the beneficial and adverse effects of PEEP is inevitable.

In spite of these issues, the study by Amato et al. raises the possibility that improved alveolar recruitment with the use of higher levels of positive end-expiratory pressure than were used in the NIH study might further reduce ventilator-associated lung injury. This possibility is currently being tested in a new NIH Acute Respiratory Distress Syndrome Network ventilation trial. A number of alternative approaches to conventional mechanical ventilation have also been proposed, including prone positioning of the patient during ventilation, but have not yet been proved to be beneficial.

Prone position

Distribution of lung infiltrates in acute respiratory distress syndrome is non-uniform. Repositioning into the prone position (face down) might improve oxygenation by relieving atelectasis and improving perfusion. However, although the hypoxemia is overcome there seems to be no effect on overall survival.

Fluid and Hemodynamic Management

The rationale for restricting fluids in patients with acute lung injury and the acute respiratory distress syndrome is to decrease pulmonary edema. Studies in animals with acute lung injury indicated that the degree of edema was reduced if left atrial pressure was lowered. Some clinical studies have supported this hypothesis. Soon, a randomized trial of fluid management designed to compare restricted with liberal fluid management based on monitoring hemodynamics with either a pulmonary-artery catheter or a central venous catheter will be carried out by the NIH Acute Respiratory Distress Syndrome Network. While we await these results, a reasonable objective is to maintain the intravascular volume at the lowest level that is consistent with adequate systemic perfusion, as assessed by metabolic acid–base balance and renal function. If systemic perfusion cannot be maintained after the restoration of intravascular

volume, as is the case in patients with septic shock, treatment with vasopressors is indicated to restore end-organ perfusion and normalize oxygen delivery. However, on the basis of the negative results of clinical trials, the use of supranormal levels of oxygen delivery cannot be recommended.

Surfactant Therapy

Because of the success of surfactant-replacement therapy in infants with the neonatal respiratory distress syndrome, surfactant replacement has been proposed as a treatment for patients with acute lung injury and the acute respiratory distress syndrome. However, in one study, treatment with a synthetic surfactant had no effect on oxygenation, the duration of mechanical ventilation, or survival. There are several possible explanations for the negative results. First, the surfactant was delivered as an aerosol, and less than 5 percent may have reached the distal air spaces. Also, the product used, a protein-free phospholipid preparation, may not be the most effective for patients with acute lung injury and the acute respiratory distress syndrome. Newer preparations of surfactant that contain recombinant surfactant proteins and new approaches to their instillation, including tracheal instillation and bronchoalveolar lavage, are being evaluated in clinical trials.

Inhaled Nitric Oxide and Other Vasodilators

Nitric oxide is a potent vasodilator that can be delivered to the pulmonary vasculature by inhalation without causing systemic vasodilation. Although observational studies suggested that inhaled nitric oxide might be beneficial in patients with acute lung injury and the acute respiratory distress syndrome, the results of randomized, double-blind studies have been discouraging. In a phase 2 study, inhaled nitric oxide did not reduce mortality or reduce the duration of mechanical ventilation. The improvements in oxygenation with this treatment were small and were not sustained, and pulmonary-artery pressure decreased very little, and only on the first day of treatment. Also, a recent phase 3 study of inhaled nitric oxide showed that it had no effect on either mortality or the duration of mechanical ventilation. Thus, inhaled nitric oxide cannot be recommended for the routine treatment of acute lung injury and the acute respiratory distress syndrome, but it may be useful as a rescue therapy in patients with refractory hypoxemia. Treatment with several less selective vasodilators, including sodium nitroprusside, hydralazine, alprostadil (prostaglandin E₁), and epoprostenol (prostacyclin), has also not been shown to be beneficial.

Glucocorticoids and Other Antiinflammatory Agents

Recognition of the inflammatory nature of the lung injury in acute lung injury and the acute respiratory distress syndrome prompted interest in antiinflammatory treatments, particularly glucocorticoids. However, glucocorticoids had no benefit when they were given before the onset of the disease or early in its course. More recently, glucocorticoids have been used to treat the later, fibrosing-alveolitis phase of the disease. Encouraging results were reported in preliminary studies and in a small randomized trial of 24 patients. A larger randomized, multicenter U.S. trial of treatment with high-dose methylprednisolone for at least seven days is under way. Because treatment with high-dose methylprednisolone may increase the incidence of infection, the routine use of this drug in patients with established acute lung injury and the acute respiratory distress syndrome cannot be recommended until results of a large multicenter trial become available.

A short course of high-dose glucocorticoids could be considered as rescue therapy in patients with severe disease that is not resolving. In addition to glucocorticoids, other antiinflammatory agents designed to interrupt the process of acute lung injury have been investigated but have proved unsuccessful. The failure may reflect the complexity and redundancy of the inflammation in acute lung injury or the inability to deliver these agents early enough in the course of the illness.

Acceleration of Resolution

Recognition of the importance of the resolution phase of acute lung injury and the acute respiratory distress syndrome has stimulated interest in strategies to hasten patients' recovery from lung injury. Experimentally, removal of pulmonary edema fluid from the alveolar space can be enhanced by both catecholamine-dependent and catecholamine-independent mechanisms, including those increased by inhaled and systemic beta-agonists. Beta-agonists are appealing candidates because they are already in wide clinical use and have no serious side effects, even in critically ill patients. Treatment with beta-agonists may also increase the secretion of surfactant and perhaps exert an antiinflammatory effect, thus helping to restore vascular permeability of the lung.

Since acute injury to epithelial type I cells causes denudation of the alveolar epithelium, an additional approach to hastening the resolution of acute lung injury and the acute respiratory distress syndrome is to accelerate reepithelialization of the alveolar barrier. The proliferation of

alveolar epithelial type II cells is controlled by a number of epithelial growth factors, including keratinocyte growth factor. Experimentally, administration of keratinocyte growth factor protects against lung injury, probably in part by increasing the proliferation of alveolar type II cells and the clearance rate of alveolar fluid and by inducing antioxidant effects, and perhaps by reducing lung endothelial injury. These findings raise the possibility that an epithelium-specific growth factor could be used to accelerate the resolution of the syndrome. Overall, strategies directed at restoring the function of alveolar epithelium deserve careful evaluation.

Conclusions

In conclusion, substantial progress has been made in the understanding of acute lung injury and the acute respiratory distress syndrome. More information regarding epidemiology and pathogenesis has become available, and the importance of the resolution phase of the illness has been recognized, opening up new avenues for therapeutic intervention. Although progress in specific treatments has lagged behind basic research, the formation of the NIH Acute Respiratory Distress Syndrome Network led to a clinical trial of a ventilation strategy involving low tidal volumes, which reduced mortality by 22 percent. Large, prospective, randomized trials of new ventilatory and pharmacologic strategies may further reduce mortality from this common clinical syndrome.

General Principles of Emergency Care

General Principles of Emergency Care

Splint injured parts in the position they are found Prevent chilling, but do not add excessive heat Do not remove penetrating objects Do not try to give anything by mouth to an unconscious person or one with serious injuries Stay with the injured person until medical care or transportation arrives

Physical Examination :

The first priority: ABCs Airway, breathing, and circulation Watch chest for rhythmic breathing; listen near mouth and nose for air movement Palpate the carotid and peripheral pulses Once respiration and circulation established, assess for uncontrolled bleeding and shock If none, assess systematic head-to-toe

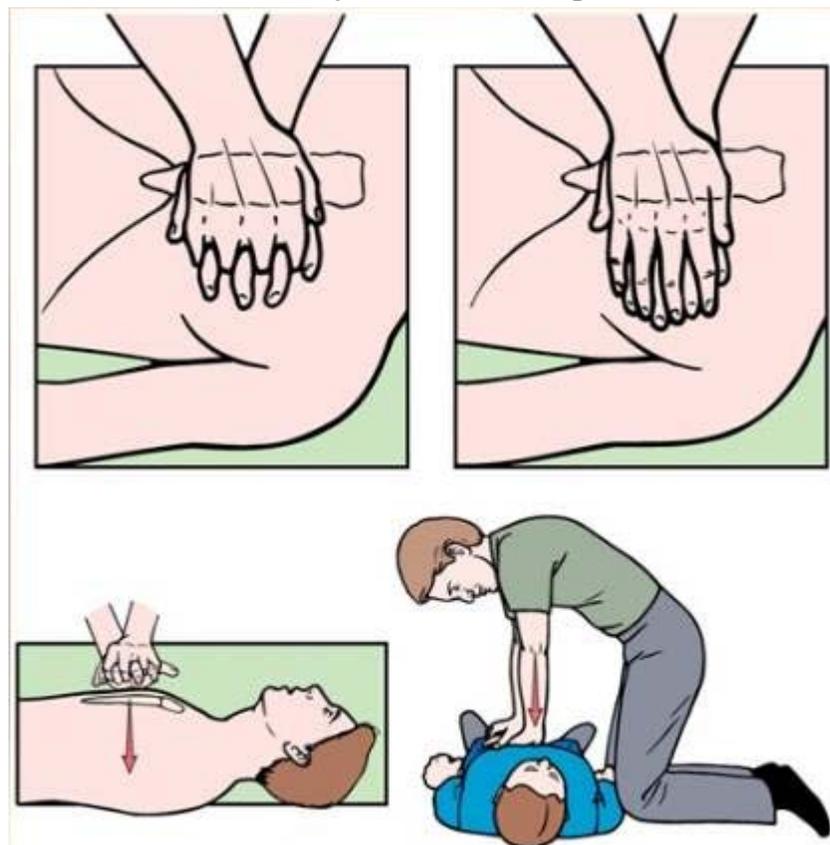
Evaluation of Accident and Emergency Patients :

Primary survey (ABCDE): Airway Breathing Circulation/hemorrhage Disability or

defibrillation Exposure Secondary survey (head-to-toe): Head, face, neck, neurologic status
Chest Abdomen/genitourinary system Limbs Log roll

Systematic Head-to-Toe Assessment

Evaluate comprehension: ask patient to follow simple commands, such as opening and closing the eyes Inspect eyes to assess pupil size, equality, and reaction to light Ask about neck pain or stiffness and the ability to swallow Inspect for chest wall movement symmetry



Systematic Head-to-Toe Assessment :

Systematic Head-to-Toe Assessment Assess breathing, dyspnea, and abnormal sounds associated with respirations Examine contour of abdomen for distention Light palpation to detect pain or tenderness Inspect the extremities for deformity or injury, and evaluate movement Assess peripheral pulses and warmth and sensation in the extremities

Cardiopulmonary Arrest :

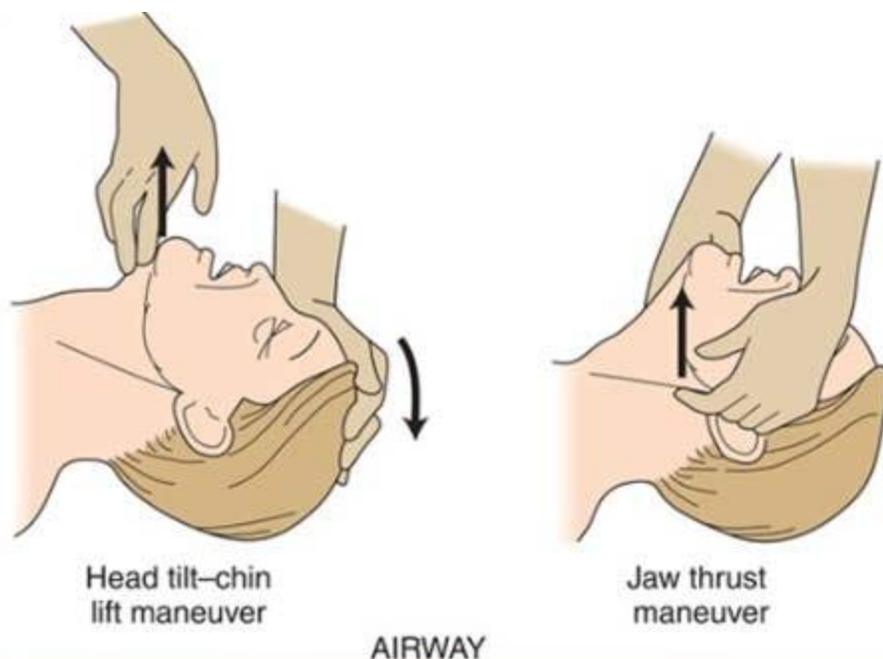
Cardiopulmonary Arrest Absence of a heartbeat and respirations Causes Myocardial infarction, heart failure, electrocution, drowning, drug overdose, anaphylaxis, and asphyxiation Signs and symptoms Collapse and quickly lose consciousness No pulse or respiration

Cardiopulmonary Arrest :

Cardiopulmonary Arrest Interventions Determine responsiveness Open airway Check for

breathing (look, listen, feel) If nonresponsive and not breathing, palpate for a pulse If no pulse in 10 seconds, begin compression:ventilation cycles of 30:2 If a pulse, deliver 10-12 rescue breaths per minute In no advanced airway, continue the 30:2 ratio With advanced airway, compressions of 100 per minute without pausing for ventilations which are done at a rate of 8-10 per minute

Figure: Airway :



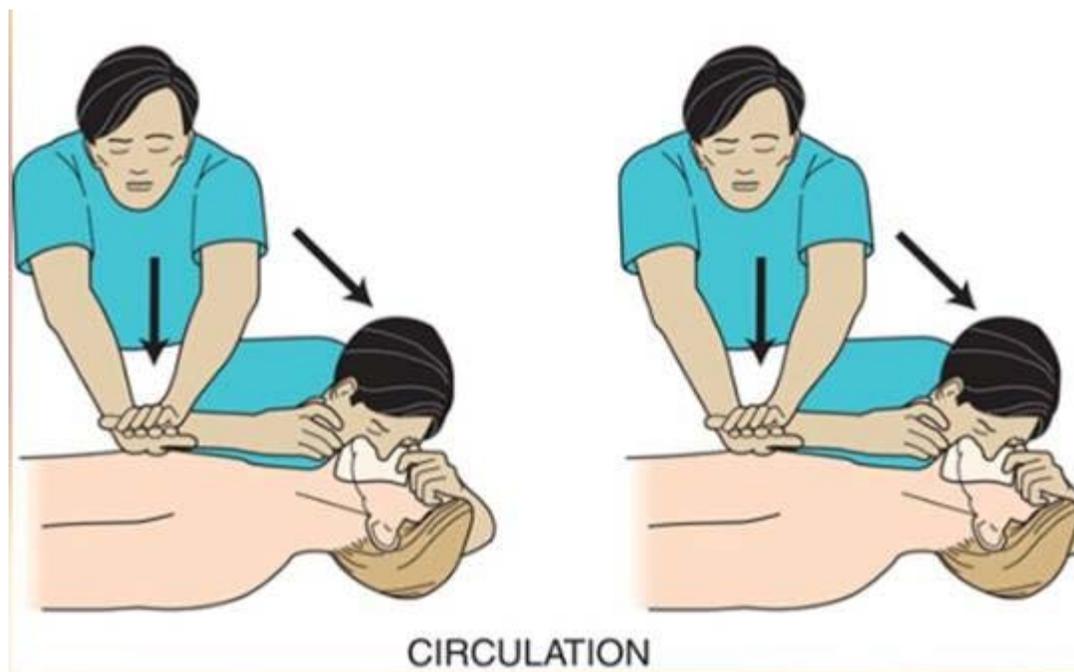
Cardiopulmonary Arrest :

Cardiopulmonary Arrest Two-rescuer CPR One rescuer compresses the chest at a rate of 100 per minute without pausing for ventilations Second rescuer ventilates with 8-10 breaths/minute Swap roles about every 2 minutes to avoid tiring Recovery position Unresponsive victim who is breathing should be log-rolled to one side if no cervical trauma is suspected



Choking or Airway Obstruction :

Choking or Airway Obstruction Assessment Universal sign of choking is grabbing the throat with one or both hands First determine if airway completely blocked If victim is able to speak, breathe, or cough with good air exchange, do nothing If unable to speak, breathe, or cough with good air exchange, act quickly to prevent suffocation



Choking or Airway Obstruction :

Choking or Airway Obstruction Victim is conscious Perform the Heimlich maneuver If effective, air expels foreign body from the airway If not, repeat maneuver until the object is expelled or victim loses consciousness



Choking or Airway Obstruction :

Choking or Airway Obstruction Victim unconscious/loses consciousness Lift the jaw and sweep a finger through the mouth to try to remove the object Tilt the head back, lift the chin, pinch the nostrils, try to ventilate If the airway is still obstructed, attempts at ventilation will fail Reposition the head and attempt once more to ventilate If unsuccessful, proceed to the next step Straddle the victim's thighs, place one hand on top of the other, and deliver up to five abdominal thrusts Repeat these three steps until the airway is clear



A

From Lewis, S.M., Hetherington, W.M., & Ditman, S.R. (Eds.) (2005). Medical-surgical nursing: Assessment

Straddle the victim's thighs, place one hand on top of the other, and deliver up to five abdominal thrusts Repeat these three steps until the airway is clear



B

Shock :

Shock Results from acute circulatory failure caused by inadequate blood volume, heart failure, overwhelming infection, severe allergic reactions, or extreme pain or fright

Hemorrhage :

Hemorrhage The loss of a large amount of blood Loss of more than 1 liter (L) of blood in an adult may lead to hypovolemic shock Death from continued uncontrolled bleeding Bleeding may be external or internal Internal bleeding is suspected if signs of shock but no external bleeding is evident

Exsanguination :

Exsanguination To Bleed Out all the Blood from the body



Hemorrhage :

Hemorrhage Apply direct, continuous pressure Elevate and immobilize the injured part (unless fracture is suspected) After bleeding stops, secure a large dressing, if available, over the wound Reinforce the dressing but do not change it If direct wound pressure and elevation fail to control bleeding, apply indirect pressure to the main artery that supplies the area

Hemorrhage Epistaxis Blood from anterior or posterior portion of the nose Most anterior nosebleeds respond to pressure Instruct the patient to sit down and lean the head forward Pinch the nostrils shut for at least 10 minutes Advise patient not to blow or pick at nose for several hours Continued bleeding or bleeding from the posterior area of the nose requires medical

treatment

Fracture :

Fracture A break in a bone Simple (closed) fracture Does not break the skin Compound (open) fracture Broken bone protrudes through the skin Complete fracture Broken ends are separated Incomplete fracture Bone ends are not separated

Fracture :

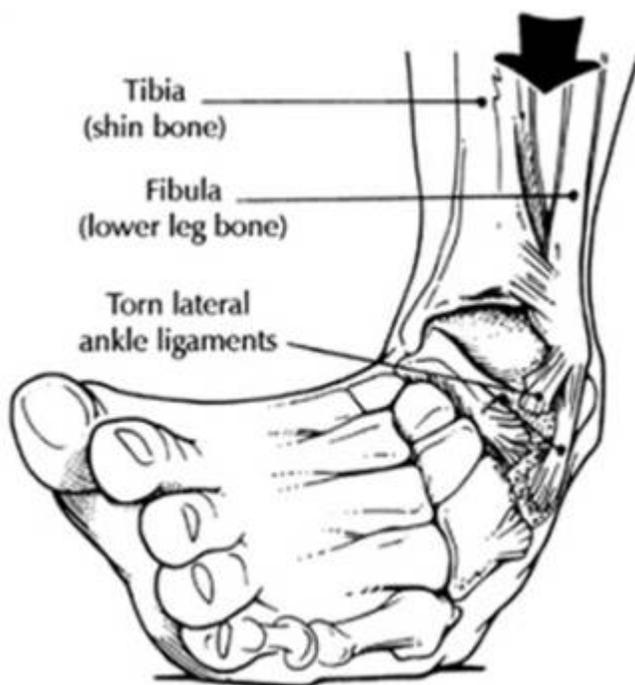
Fracture Assessment Primary symptom is pain Numbness/tingling from nerve injury and blood vessels Signs: deformity, swelling, discoloration, decreased function, and bone fragments protruding through the skin





Strains and Sprains :

Strains and Sprains Strains Injuries to muscles or tendons, or both Sprains Injuries to ligaments These injuries are painful; may be swelling Emergency treatment is: RICE- (rest, ice, compression, elevation) Victim to see physician for further evaluation



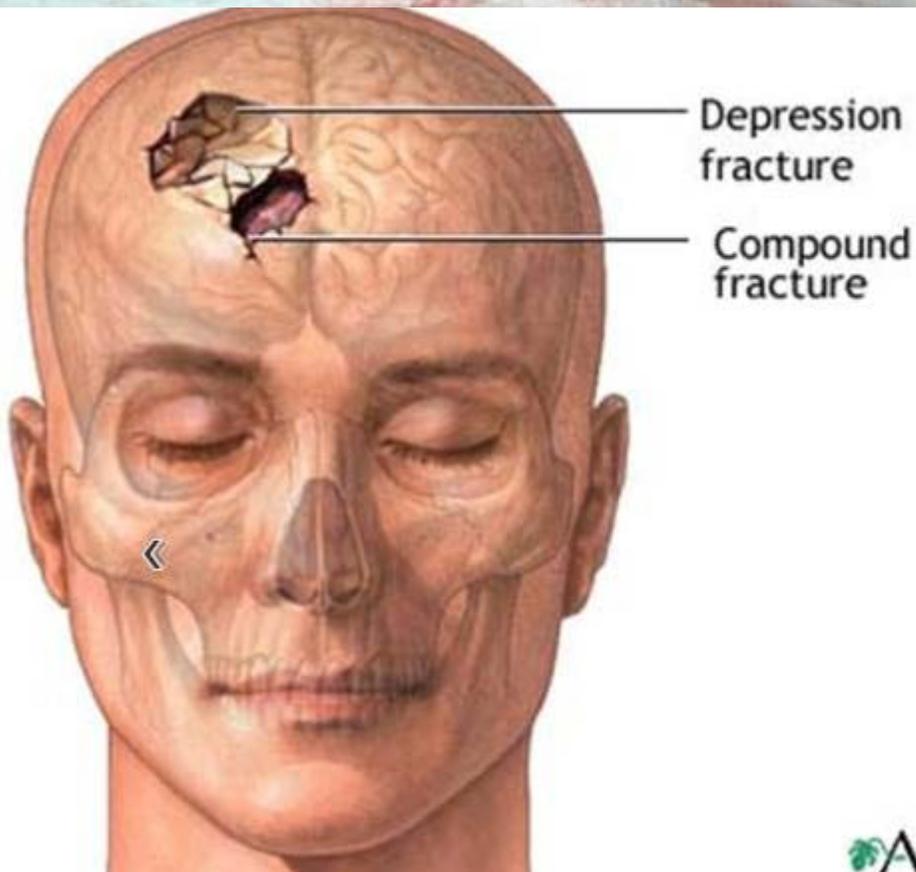
Head Injury :

Head Injury Suspected with any type of blow to the head or any unexplained loss of consciousness
Assessment Inspection and palpation of the head Evaluate for signs and symptoms of increased intracranial pressure Be alert for the leakage of cerebrospinal fluid that occurs with basilar skull fractures



Head Injury :

Head Injury Must be assessed by a physician as soon as possible Immobilize neck and keep victim flat with proper alignment of the neck and head Backboard used for transporting victim



Neck and Spinal Injuries :

Neck and Spinal Injuries Assessment Assess breathing and circulation and then begin resuscitation if needed Remember to use the jaw-thrust method to open the airway! Assess

movement and sensation in all extremities

Neck and Spinal Injuries Immediately summon expert emergency team In remote or life-threatening settings, the victim may have to be moved A rolled towel or article of clothing can be used as a collar to support the neck The victim can then be moved by log-rolling to one side and then rolling back onto a board, keeping the spine as straight as possible Throughout the movement, one rescuer supports the head while two others support the shoulders, hips, and legs

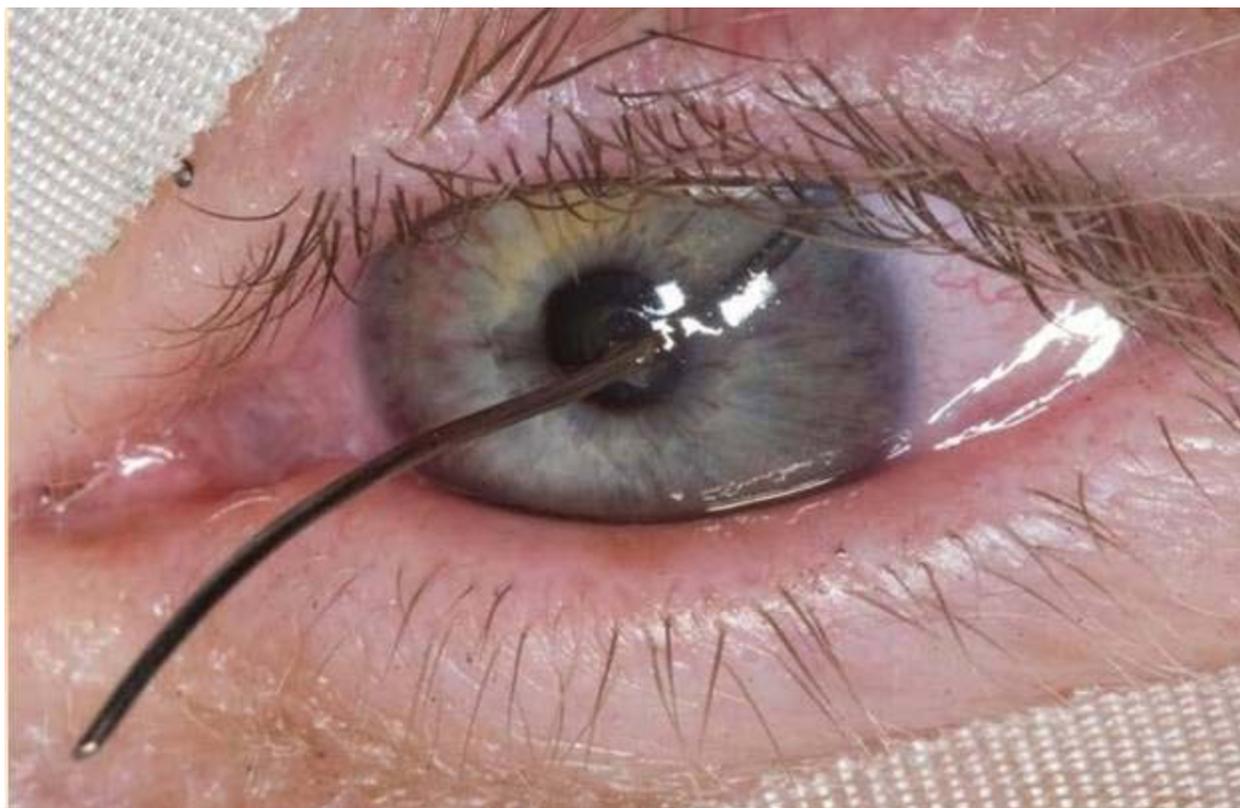
Eye Injury :

Eye Injury Assessment Inspect eyelid for trauma and the eye for redness, foreign bodies, or penetrating objects To inspect for foreign bodies, evert the eyelids



Ear Trauma :

Ear Trauma Assessment Assess extent of injury; note if any tissue is fully separated and severity of bleeding Apply direct pressure to injury to control bleeding



Ear Trauma :

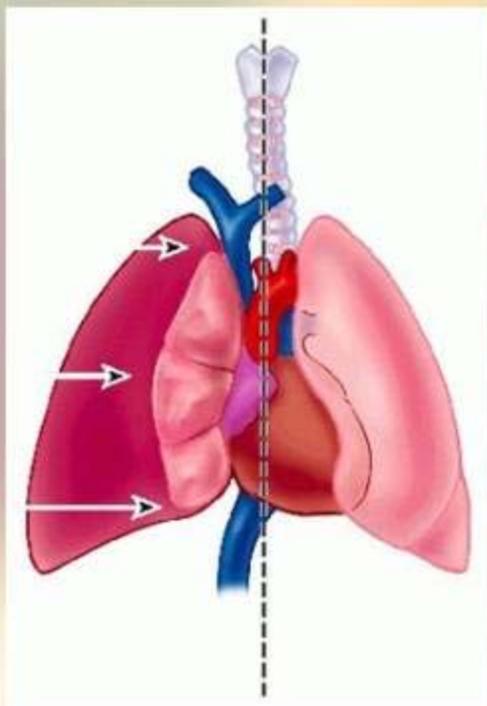
Ear Trauma If injured part is actually separated, reattachment may be possible Retrieve the tissue, wrap it in plastic, keep it cool, and transport it with the victim

Chest Injury :

Chest Injury Critical injuries: open pneumothorax, flail chest, massive hemothorax, and cardiac tamponade

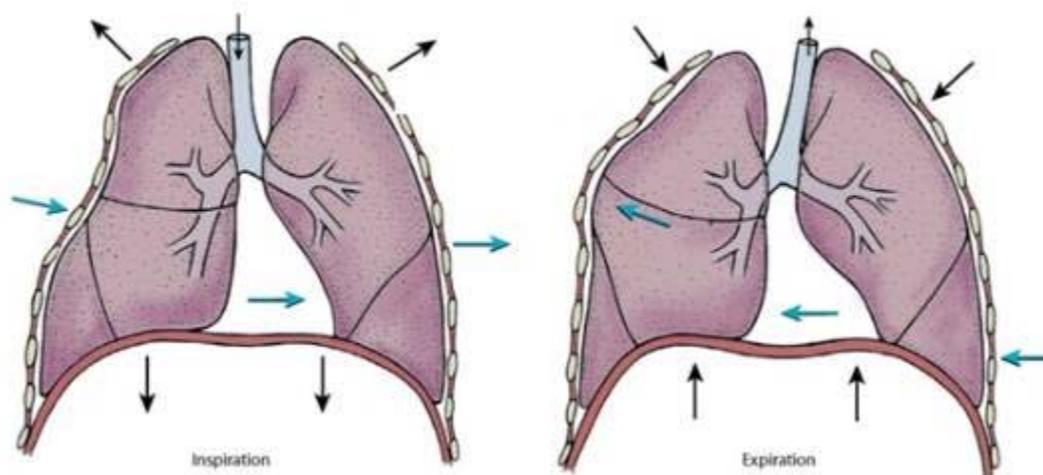
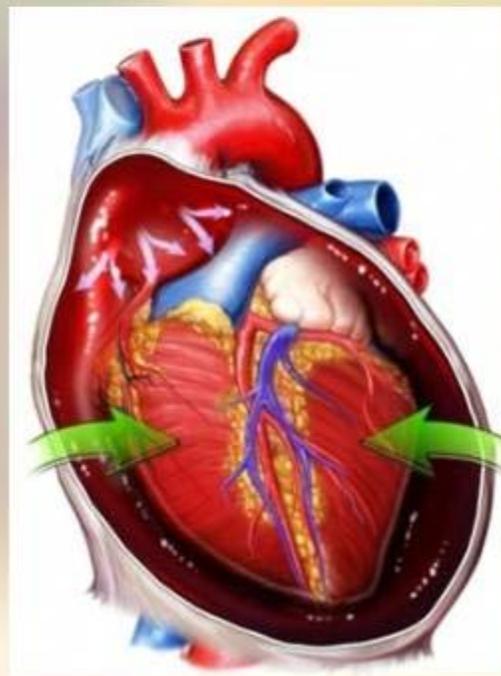
Pneumothorax

Pneumothorax



Cardiac Tamponade

Cardiac Tamponade



From Mosby's F.D. & Saunders, M. (1998). Medical-surgical nursing: Foundations for clinical practice (3rd ed.). Philadelphia: Saunders.

Assessment of Pt with Chest Injuries rate and character of respirations, skin color, pulse rate and rhythm symmetry of the chest wall movement presence of any apparent injuries to the chest
Signs and symptoms of chest injuries
Dyspnea Tachycardia Restlessness Cyanosis asymmetric or other abnormal chest wall movement, abnormal sounds of breathing Note mental state and level of consciousness



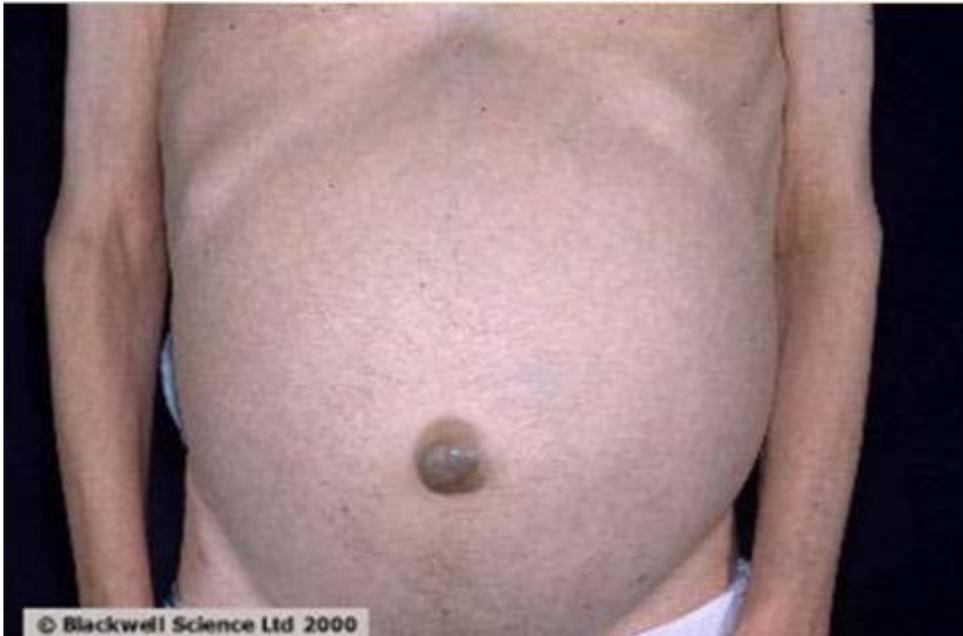
Abdominal

Injury: Assessment :

Abdominal Injury: Assessment Assess abdomen for evidence of injury Nausea/ Vomiting
Inspect Suspect internal abdominal injuries if victim complains of abdominal pain or abdomen
shows evidence of trauma or distention Protrusion of internal organs through a wound is called
evisceration

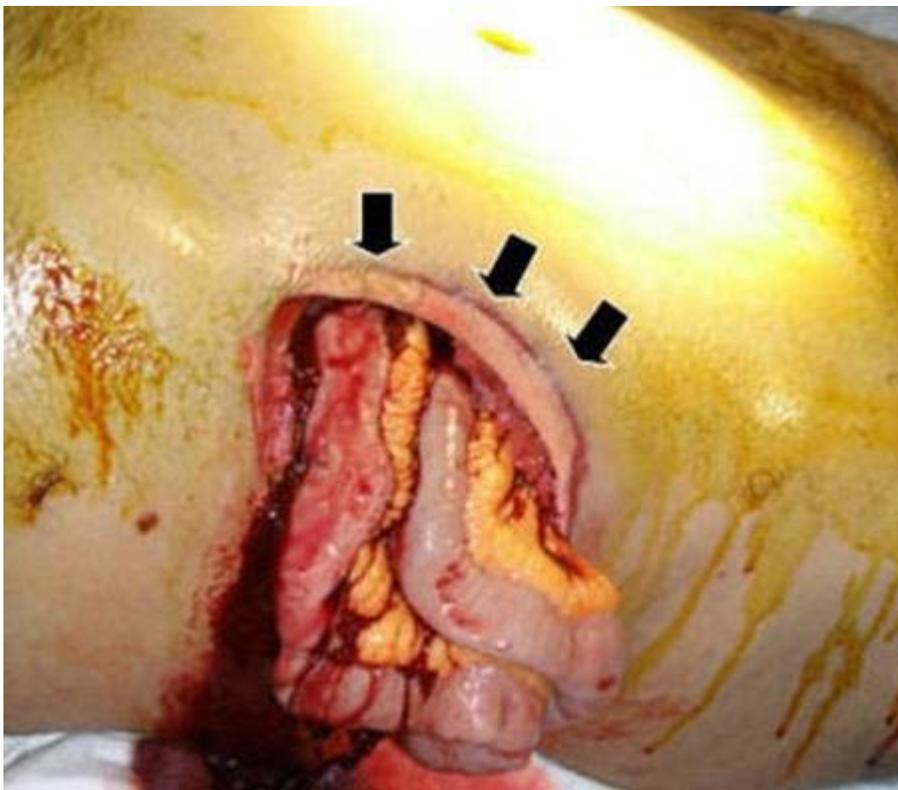
Abdominal Injury :

Abdominal Injury Cullen's Sign- Bluish tinge around umbilicus may indicates abdominal
hemorrhage



Abdominal Injury: Interventions :

Abdominal Injury: Interventions Require medical evaluation Give nothing by mouth in preparing for transport Do not attempt to replace eviscerated organs in the abdomen; this may cause additional harm Cover organs with material, such as plastic wrap or foil, to conserve moisture and warmth A saline-soaked sterile dressing is ideal but is not likely to be available on the scene of an accident Cover wound with clean cloth; transport to hospital

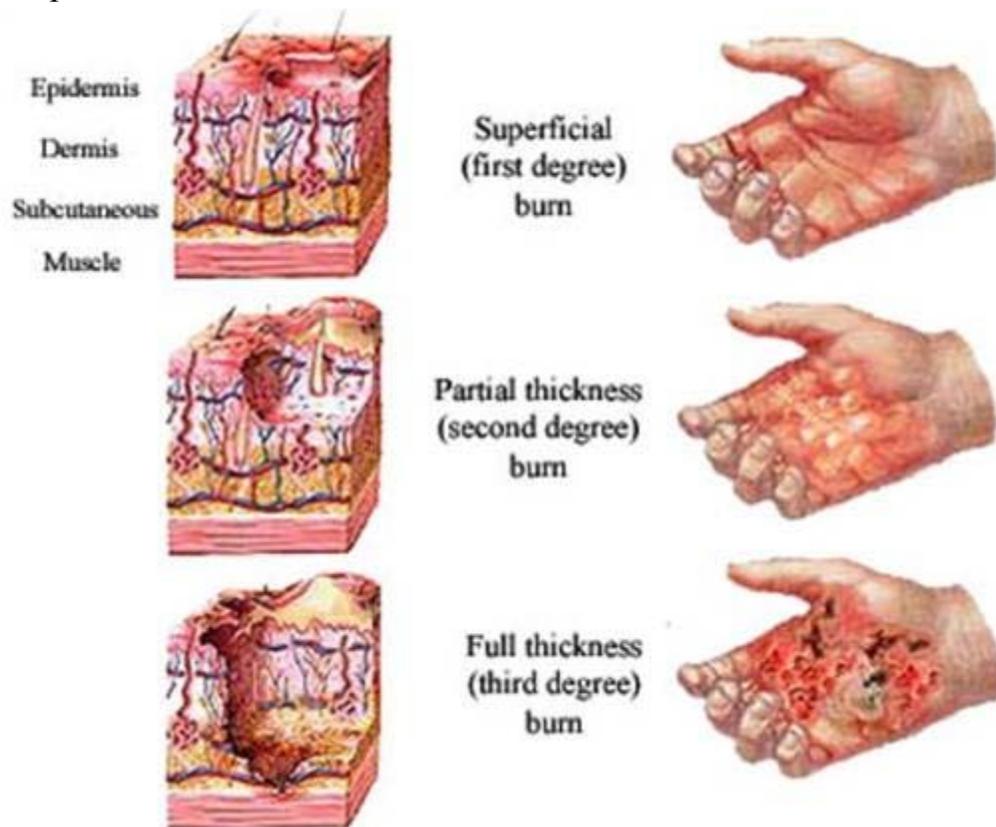


Traumatic Amputation :

Traumatic Amputation If partially/completely detached, reattachment possible Clean the wound surfaces with sterile water or saline and place the tissue in its normal position A body part that is completely detached should ideally be wrapped in sterile gauze moistened with sterile saline, placed in a watertight container such as a resealable plastic bag, and placed in an iced saline bath The tissue should not be frozen or placed in contact with ice Amputated extremities may be healthy enough for reattachment for 4-6 hours; digits as long as 8 hours

Amputated Hand :

Amputated Hand



:
Shark Bite Amputation



Burns: Assessment :

Burns: Assessment Determine the type of burn If patient has a flame burn or was in a closed, smoke-filled area, assess respirations first Determine the extent and depth of the burns Inspect skin for color, blisters, tissue destruction Superficial burns: typically pink or red and painful Deeper burns: red, white, or black; may destroy not only the skin but also the underlying tissues Electrical: difficult to assess; full extent of tissue damage may not be apparent for several days Chemical: immediately remove any remaining chemical

Burns: Interventions :

Burns: Interventions Ensure a patent airway and respirations for burn victims Rescue breathing, if needed

Hyperthermia :

Hyperthermia Body temperature $>37.2^{\circ}\text{C}$ (99°F) Heat edema and heat cramps are mild degrees of hyperthermia Can be treated by moving individual into cool place and providing fluids with electrolytes Heat exhaustion and heat stroke more serious See Table 16-5, p. 236

Hypothermia :

Hypothermia Decrease in body core temperature to $<36^{\circ}\text{C}$ (95°F) Caused by prolonged exposure to cold, extremely cold temperatures, or immersion in cold water Causes depression of vital functions, and if not corrected, death results from cardiac dysrhythmias



Hypothermia :

Hypothermia Mild stage Patient shivers in an effort to generate body heat Blood vessels in the extremities are constricted, and performing complex motor tasks is impaired Moderate hypothermia Appears dazed, poor motor coordination, slurred speech, and violent shivering May behave irrationally Severe hypothermia Waves of shivering, rigid muscles, and pale skin Pulse rate is slow and the pupils are dilated

Hypothermia



Frost Bite and Frost Nip :

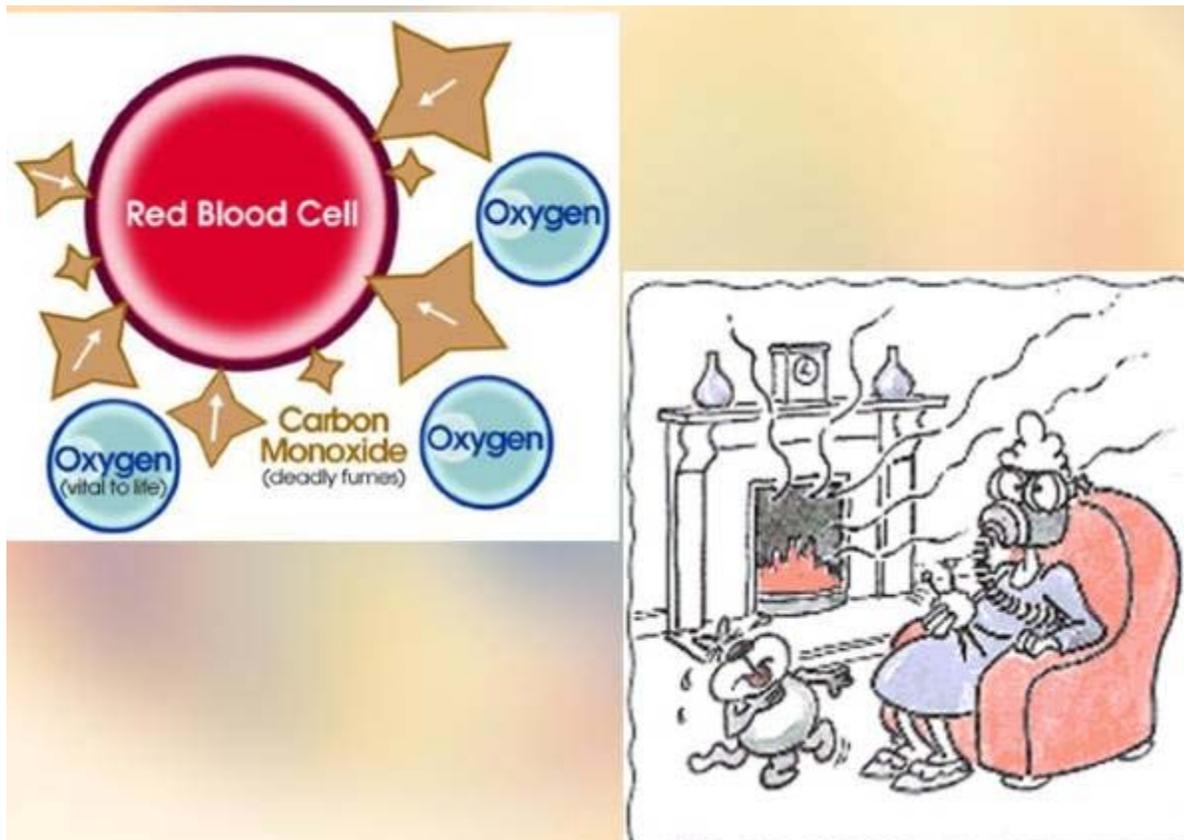
Frost Bite and Frost Nip Frost nip: mild tissue damage caused by cold Frost bite: More serious cold injury Blood vessels constrict when exposed to extreme cold. Blood clots form and circulation is impaired Tissues die as a result Pain is the first sign, followed by tingling, and numbness

Treatment for Frostbite :

Treatment for Frostbite Mild- rapid re-warming More serious- Do not attempt to thaw tissue unless warmth maintained Re-warm in heated 100-105* water-Do not rub or massage area Feet should be defrosted only if the person doesn't need to walk

Carbon Monoxide Poisoning :

Carbon Monoxide Poisoning Assessment Early signs and symptoms: headache and shortness of breath with mild exertion Then dizziness, nausea, vomiting, and mental changes As carbon monoxide in bloodstream rises, victim loses consciousness and develops cardiac and respiratory irregularities Cherry-red skin clear indicator of carbon monoxide poisoning, but skin color often found to be pale or bluish with reddish mucous membranes



Nursing Diagnosis, Goal, and Outcome Criteria :

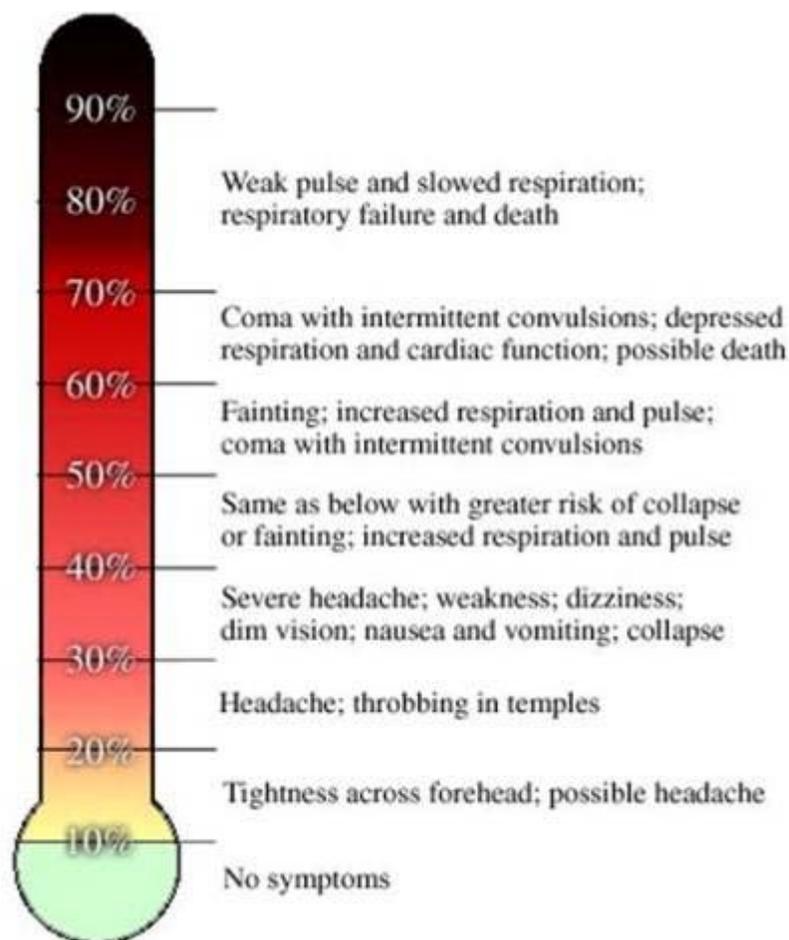
Nursing Diagnosis, Goal, and Outcome Criteria Impaired gas exchange related to carbon monoxide poisoning The goal of nursing care for the emergency treatment of the victim of carbon monoxide poisoning is normal oxygenation

Interventions :

Interventions Immediately move the victim to fresh air If person not breathing, start rescue breathing Seek emergency medical assistance immediately Give oxygen as soon as it is available At the hospital the patient may be placed in a hyperbaric oxygen chamber

Prevention :

Prevention Keep gas furnaces and stoves in proper repair Burners that use gas must be vented to the outside Don't use charcoal or wood-burning devices in closed area Never let automobile engine run in closed garage Install carbon monoxide detector alarm



Drug or Chemical Poisoning :

Drug or Chemical Poisoning Assessment History: data about relevant signs and symptoms

Name of drug or chemical. If the victim cannot provide the information, look for clues and save the container
 Amount consumed
 Length of time since substance was taken
 Last food consumed: amount, time
 Signs and symptoms that may be caused by poisons
 Victim's age and approximate weight
 Other medications, drugs, or alcohol ingested

Drug or Chemical Poisoning :

Drug or Chemical Poisoning Interventions
 Immediately call your poison center
 Some poisonings can be treated at home, others require a physician or a hospital
 Treatment of poisoning in an emergency facility may be with activated charcoal, total bowel lavage, and/or cathartics

Food Poisoning :

Food Poisoning Assessment Symptoms: nausea, vomiting, abdominal cramps, and diarrhea

Botulism caused by *Clostridium botulinum* has neurotoxic effects: difficulty breathing, seeing, and swallowing
 Clue that food poisoning is causing victim's symptoms is that all who consumed a certain food become ill
 To assist in identifying poisons, collect samples of stool or

vomited materials for possible lab analysis



Food Poisoning :

Food Poisoning Ptomaine poisoning Bacterial or chemical Cleanliness, good personal hygiene, and proper preparation and handling of foods Symptoms and treatment (Also refer to Health Promotion Points 44-2.)



Multistate Outbreak of *E. coli* O157:H7 Infections Linked to Eating Raw Refrigerated, Prepackaged Cookie Dough



Multistate Outbreak of *E. coli* O157:H7 Infections Associated with Beef from JBS Swift Beef Company

Multistate Outbreak of *E. coli* O157:H7 Infections Linked to Eating Raw Refrigerated, Prepackaged Cookie Dough Multistate Outbreak of *E. coli* O157:H7 Infections Associated with Beef from JBS Swift Beef Company



Healthy People 2010 Food Safety :

Healthy People 2010 Food Safety Focus area # 10: Food Safety. To reduce food-borne illnesses.



Food Poisoning: Interventions :

Food Poisoning: Interventions Medical care necessary if symptoms are severe or persistent The physician may order antiemetics and antidiarrheals Intravenous fluids may be prescribed with severe vomiting and diarrhea

Bites and Stings :

Bites and Stings Assessment Try to determine the type of bite Inspect bite to identify characteristics of bite site and any changes in surrounding tissue Ask about any symptoms that developed after the bite: pain, edema, numbness, tingling, nausea, fever, dizziness, and dyspnea Interventions: see Table 16-7, p. 239

Animal Bites :

Animal Bites Animal bites and Animal Control Agency: Wash area with warm soapy water for 5-10 minutes Rabies immunization or 5 IM injections given over a period of 3 weeks



Copperhead Snake :

Snakebite: :

Snakebite: Poisonous snakes and venomous snakebite Treatment Wash area Immobilize Apply suction Go to hospital Give antivenin



Insect Bites and Stings :

Remove Stinger

Health Promotion Points : Anaphylaxis Kit Individuals who have known allergies to insect bites or other common environmental allergens should carry an anaphylaxis kit. Family and friends should know how to use the contents in case the individual is unable to treat herself.

Coral Snake



Snakebite: :

Snakebite: Poisonous snakes and venomous snakebite Treatment Wash area Immobilize Apply suction Go to hospital Give antivenin

Rattlesnake



Insect Bites and Stings :

Insect Bites and Stings

Insect Bites and Stings



Remove Stinger



Health Promotion Points : Anaphylaxis Kit :

Health Promotion Points : Anaphylaxis Kit Individuals who have known allergies to insect bites or other common environmental allergens should carry an anaphylaxis kit. Family and friends should know how to use the contents in case the individual is unable to treat herself.



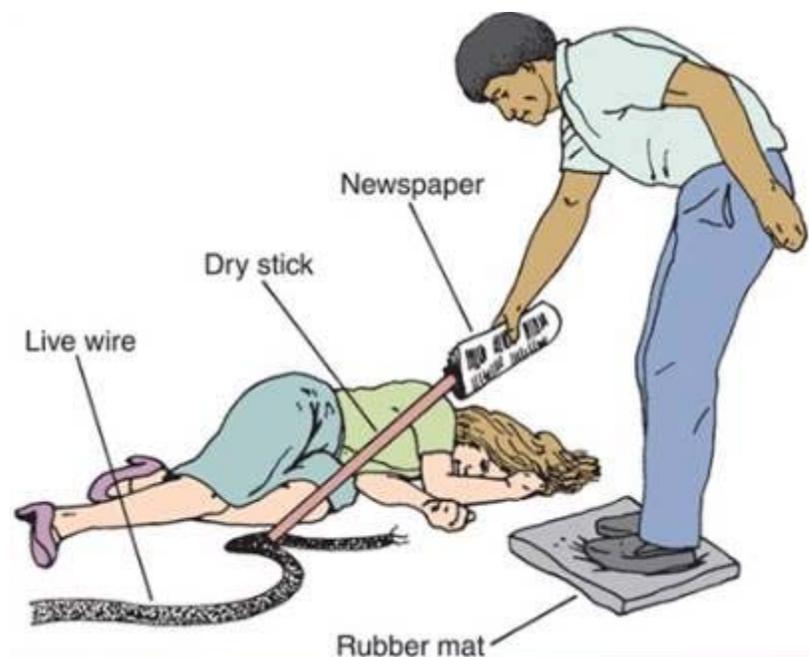
Administering Epi Pen :

Signs and Symptoms of Anaphylaxis :

Signs and Symptoms of Anaphylaxis Hives Swelling Generalized weakness Chest tightness
Abdominal cramps Constriction of throat Loss of consciousness

Safety Alert : Lightning :

Safety Alert : Lightning If outdoors when lightning occurs, avoid water, high ground, open spaces, and metal objects. Do not seek shelter under canopies, small picnic or rain shelters, or trees.



Acts of Bioterrorism Deliberate release of pathogens to kill people Anthrax, botulism, plague, smallpox, tularemia: most common biologic agents in terrorist attack Easily spread; potential to cause many deaths Health care providers must know how to protect themselves and others Staff should know where to obtain personal protective equipment and what types of precautions (i.e., patient isolation) should be taken

Disaster Planning :

Disaster Planning A challenge for the health care system is to be ready for natural disasters that often occur with short warning American Red Cross and the Salvation Army are experienced in handling these situations and quickly move in to help A call for nurse volunteers usually follows Regardless of the area of clinical expertise, there is certain to be a way each nurse can contribute

Psychological Emergencies :

Psychological Emergencies Combative patient Domestic violence/abuse Child abuse Elder abuse Psychological trauma

Legal Aspects of Emergency Care Emergency doctrine In emergencies, person may be unable to consent to care Treatment can be provided under the assumption that the patient would have consented if able Good Samaritan laws Limit liability and provide protection against malpractice claims when health care providers render first aid at the scene of an emergency These laws do not protect the nurse in the event of gross negligence or willful misconduct

Source Information

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Підготував Добродній А.В.

Ischemic heart disease: a stable and unstable angina pectoris, MI. Treatment. Differentiation of the chest pain causes.

Background: Angina pectoris is the result of myocardial ischemia caused by an imbalance between myocardial blood supply and oxygen demand. Angina is a common presenting symptom (typically, chest pain) among patients with coronary artery disease. A comprehensive approach to diagnosis and to medical management of angina pectoris is an integral part of the daily responsibilities of physicians.

Pathophysiology: Myocardial ischemia develops when coronary blood flow becomes inadequate to meet myocardial oxygen demand. This causes myocardial cells to switch from aerobic to anaerobic metabolism, with a progressive impairment of metabolic, mechanical, and electrical functions. Angina pectoris is the most common clinical manifestation of myocardial ischemia. It is caused by chemical and mechanical stimulation of sensory afferent nerve endings in the coronary vessels and myocardium. These nerve fibers extend from the first to fourth thoracic spinal nerves, ascending via the spinal cord to the thalamus, and from there to the cerebral cortex.

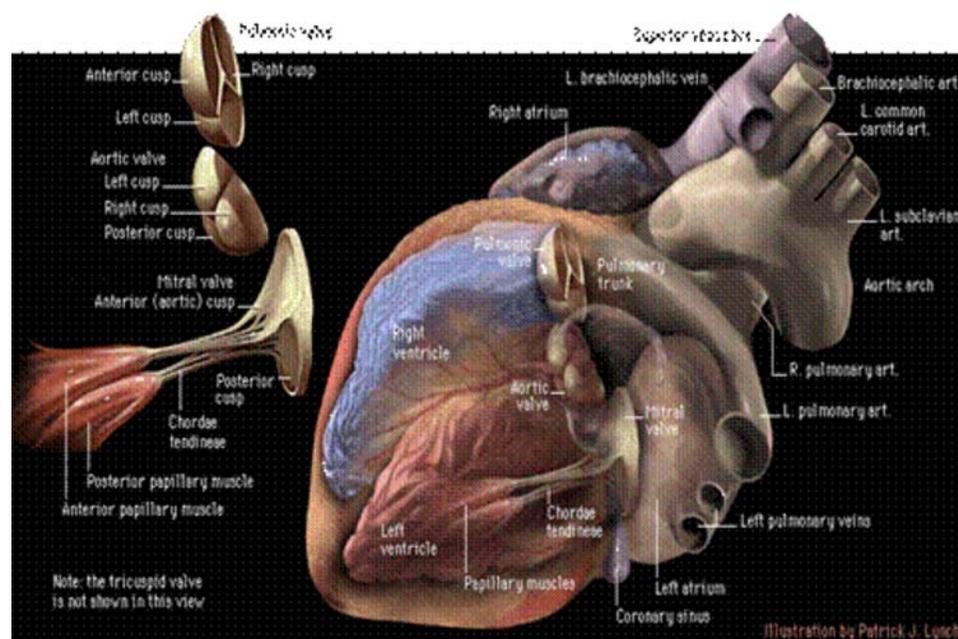


Fig. 1 Heart anatomy

Recent studies have shown that adenosine may be the main chemical mediator of anginal pain. During ischemia, ATP is degraded to adenosine, which, after diffusion to the extracellular space, causes arteriolar dilation and anginal pain. Adenosine induces angina mainly by stimulating the A₁ receptors in cardiac afferent nerve endings.

Heart rate, myocardial inotropic state, and myocardial wall tension are the major determinants

of myocardial metabolic activity and myocardial oxygen demand. Increases in the heart rate and myocardial contractile state result in increased myocardial oxygen demand. Increases in both afterload (ie, aortic pressure) and preload (ie, ventricular end-diastolic volume) result in a proportional elevation of myocardial wall tension and, therefore, increased myocardial oxygen demand. Oxygen supply to any organ system is determined by blood flow and oxygen extraction. Because the resting coronary venous oxygen saturation is already at a relatively low level (approximately 30%), the myocardium has a limited ability to increase its oxygen extraction during episodes of increased demand. Thus, an increase in myocardial oxygen demand (eg, during exercise) must be met by a proportional increase in coronary blood flow. The ability of the coronary arteries to increase blood flow in response to increased cardiac metabolic demand is referred to as coronary flow reserve (CFR). In healthy people, the maximal coronary blood flow after full dilation of the coronary arteries is roughly 4-6 times the resting coronary blood flow. CFR depends on at least 3 factors: large and small coronary artery resistance, extravascular (ie, myocardial and interstitial) resistance, and blood composition. Myocardial ischemia can result from (1) a reduction of coronary blood flow caused by fixed and/or dynamic epicardial coronary artery (ie, conductive vessel) stenosis, (2) abnormal constriction or deficient relaxation of coronary microcirculation (ie, resistance vessels), or (3) reduced oxygen-carrying capacity of the blood.

Atherosclerosis is the most common cause of epicardial coronary artery stenosis and, hence, angina pectoris. Patients with a fixed coronary atherosclerotic lesion of at least 50% show myocardial ischemia during increased myocardial metabolic demand as the result of a significant reduction in CFR. These patients are not able to increase their coronary blood flow during stress to match the increased myocardial metabolic demand, thus they experience angina. Fixed atherosclerotic lesions of at least 90% almost completely abolish the flow reserve; patients with these lesions may experience angina at rest.

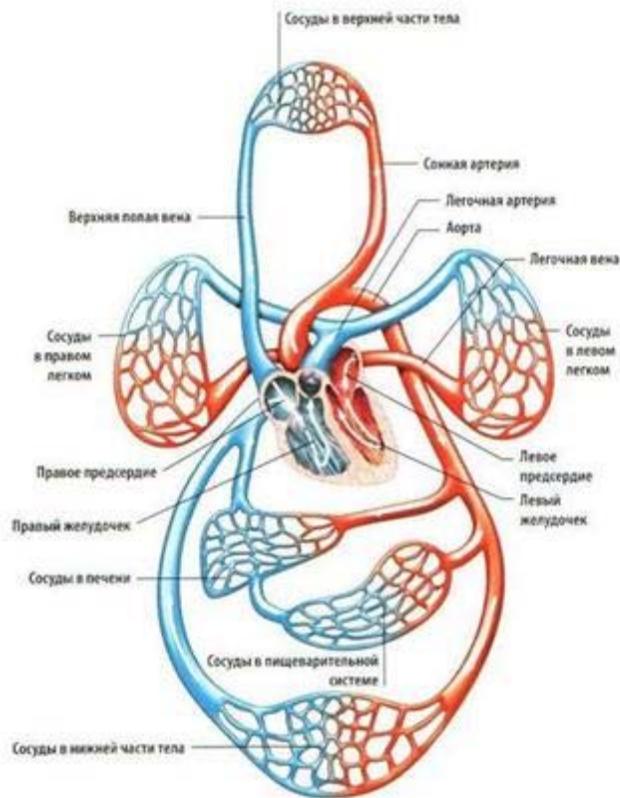


Fig 1a. Structure of cardio-vascular system

Coronary spasm can also reduce CFR significantly by causing dynamic stenosis of coronary arteries. Prinzmetal angina is defined as resting angina associated with ST-segment elevation caused by focal coronary artery spasm. Although most patients with Prinzmetal angina have underlying fixed coronary lesions, some have angiographically normal coronary arteries. Several mechanisms have been proposed for Prinzmetal angina: focal deficiency of nitric oxide production, hyperinsulinemia, low intracellular magnesium levels, smoking cigarettes, and using cocaine.

Approximately 30% of patients with chest pain referred for cardiac catheterization have normal or minimal atherosclerosis of coronary arteries. A subset of these patients demonstrates reduced CFR that is believed to be caused by functional and structural alterations of small coronary arteries and arterioles (ie, resistance vessels). Under normal conditions, resistance vessels are responsible for as much as 95% of coronary artery resistance, with the remaining

5% being from epicardial coronary arteries (ie, conductive vessels). The former is not visualized during regular coronary catheterization. Angina due to dysfunction of small coronary arteries and arterioles is called microvascular angina. Several diseases, such as diabetes mellitus, hypertension, and systemic collagen vascular diseases (eg, systemic lupus erythematosus, polyarteritis nodosa), are believed to cause microvascular abnormalities with subsequent reduction in CFR.

The syndrome that includes angina pectoris, ischemialike ST-segment changes and/or myocardial perfusion defects during stress testing, and angiographically normal coronary arteries is referred to as syndrome X. Most patients with this syndrome are postmenopausal women, and they usually have an excellent prognosis. Syndrome X is believed to be caused by microvascular angina. Multiple mechanisms may be responsible for this syndrome, including (1) impaired endothelial dysfunction, (2) increased release of local vasoconstrictors, (3) fibrosis and medial hypertrophy of the microcirculation, (4) abnormal cardiac adrenergic nerve function, and/or (5) estrogen deficiency.

A number of extravascular forces produced by contraction of adjacent myocardium and intraventricular pressures can influence coronary microcirculation resistance and thus reduce CFR. Extravascular compressive forces are highest in the subendocardium and decrease toward the subepicardium. Left ventricular (LV) hypertrophy together with a higher myocardial oxygen demand (eg, during tachycardia) cause greater susceptibility to ischemia in subendocardial layers.

Myocardial ischemia can also be the result of factors affecting blood composition, such as reduced oxygen-carrying capacity of blood, as is observed with severe anemia (hemoglobin, <8 g/dL), or elevated levels of carboxyhemoglobin. The latter may be the result of inhalation of carbon monoxide in a closed area or of long-term smoking.

Recently, ambulatory ECG monitoring has shown that silent ischemia is a common phenomenon among patients with established coronary artery disease. In one study, as many as 75% of episodes of ischemia (defined as transient ST depression of >1 mm persisting for at least 1 min) occurring in patients with stable angina were clinically silent. Silent ischemia occurs most frequently in early morning hours and may result in transient myocardial contractile dysfunction (ie, stunning). The exact mechanism(s) for silent ischemia is not known. However, autonomic dysfunction (especially in patients with diabetes), a higher pain threshold in some individuals, and the production of excessive quantities of endorphins are among the more popular hypotheses.

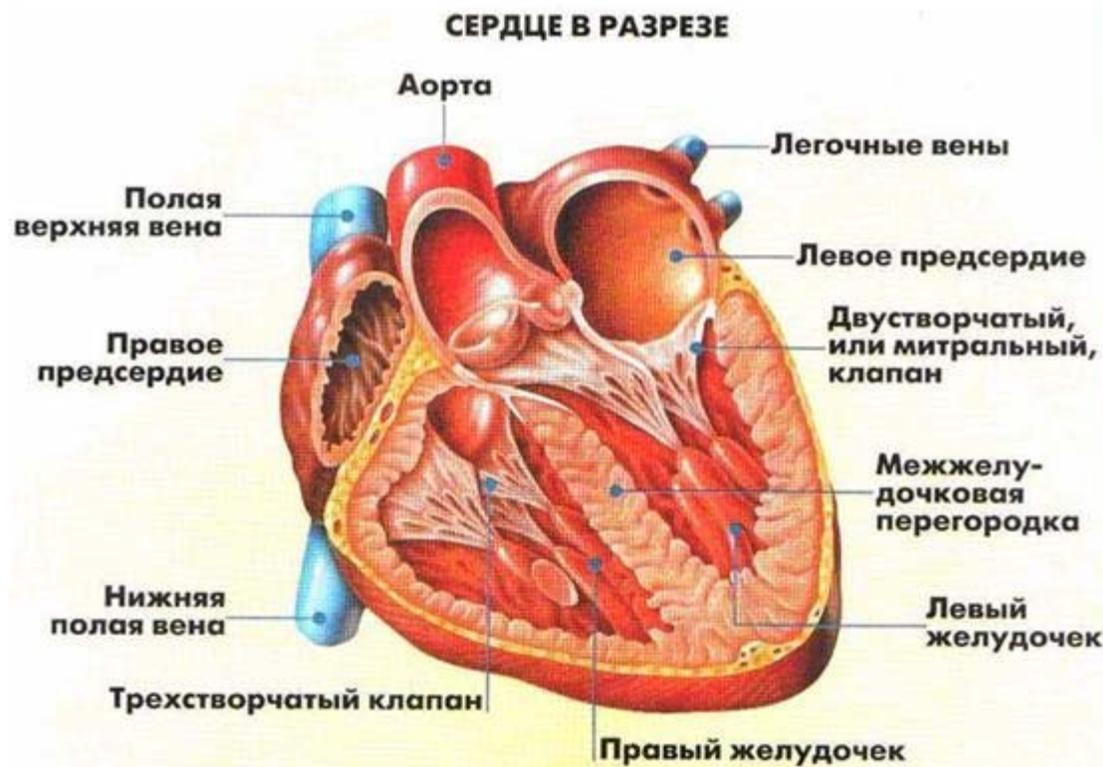


Fig. Heart structure

Frequency: In the US: Approximately 6.3 million Americans are estimated to experience angina. An estimated 350,000 new cases of angina occur every year. Each year, 1.1 million new and recurrent cases of an acute coronary event occur in this country, of which more than 40% are fatal. Roughly, more than 12 million Americans had a history of myocardial infarction (MI) and/or angina pectoris in the year 2000.

Mortality/Morbidity: Coronary artery disease is the single most common cause of death in the United States, accounting for almost one death per minute. More than half of those who die suddenly from coronary artery disease have no previous symptoms.

Race: The rate of angina pectoris in women older than 20 years ranges from 3.9% in non-Hispanic white women to 6.2% in non-Hispanic black women and 5.5% in Mexican American women. The rates of angina pectoris for men in the same ethnic groups are 2.6%, 3.1%, and 4.1%, respectively. Among American Indians aged 65-74 years, the rates (per 1000 persons) of new and recurrent heart attacks are 25.1% for men and 9.1% for women.

Sex: Angina pectoris is more often the presenting symptom of coronary artery disease in women than in men, with a female-to-male ratio of 1.7:1. It has a prevalence of 3.9 million in women and 2.3 million in men. The frequency of atypical presentations is also more common among women compared with men. Women have a slightly higher rate of mortality from coronary artery disease compared with men, in part because of an older age at presentation and

a frequent lack of classic anginal symptoms. The estimated age-adjusted prevalence of angina is greater in women than in men.

Age: The prevalence of angina pectoris increases with age. Age is a strong independent risk factor for mortality.

CLINICAL

History: Most patients with angina pectoris report of retrosternal chest discomfort rather than frank pain. The former is usually described as a pressure, heaviness, squeezing, burning, or choking sensation. Anginal pain may be localized primarily in the epigastrium, back, neck, jaw, or shoulders. Typical locations for radiation of pain are arms, shoulders, and neck. Typically, angina is precipitated by exertion, eating, exposure to cold, or emotional stress. It lasts for approximately 1-5 minutes and is relieved by rest or nitroglycerin. Chest pain lasting only a few seconds is not usually angina pectoris. The intensity of angina does not change with respiration, cough, or change in position. Pain above the mandible and below the epigastrium is rarely anginal in nature.

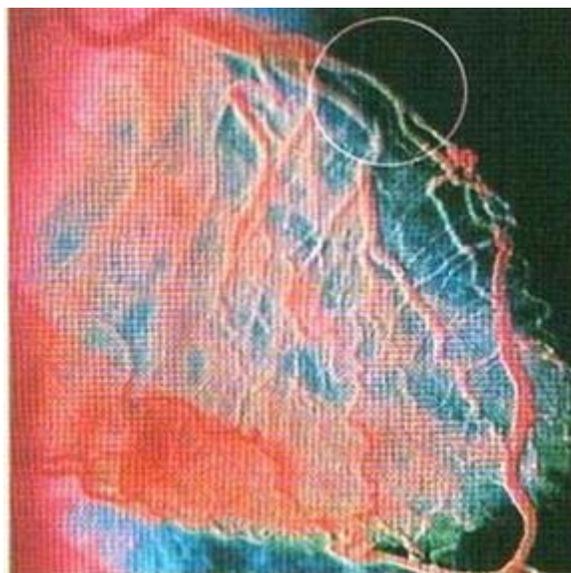


Fig.3 Coronarographia

Ask patients about the frequency of angina, severity of pain, and number of nitroglycerin pills used during angina episodes.

Angina decubitus is a variant of angina pectoris that occurs at night while the patient is recumbent. Some have suggested that it is induced by an increase in myocardial oxygen demand caused by expansion of the blood volume with increased venous return during recumbency.

The Canadian Cardiovascular Society grading scale is used for classification of angina severity, as follows:

Class I - Angina only during strenuous or prolonged physical activity

Class II - Slight limitation, with angina only during vigorous physical activity

Class III - Symptoms with everyday living activities, ie, moderate limitation

Class IV - Inability to perform any activity without angina or angina at rest, ie, severe limitation

The New York Heart Association classification is also used to quantify the functional limitation imposed by patients' symptoms, as follows:

Class I - No limitation of physical activity (Ordinary physical activity does not cause symptoms.)

Class II - Slight limitation of physical activity (Ordinary physical activity does cause symptoms.)

Class III - Moderate limitation of activity (Patient is comfortable at rest, but less than ordinary activities cause symptoms.)

Class IV - Unable to perform any physical activity without discomfort, therefore severe limitation (Patient may be symptomatic even at rest.)

Unstable angina is defined as new-onset angina (ie, within 2 mo of initial presentation) of at least class III severity, significant recent increase in frequency and severity of angina, or angina at rest.

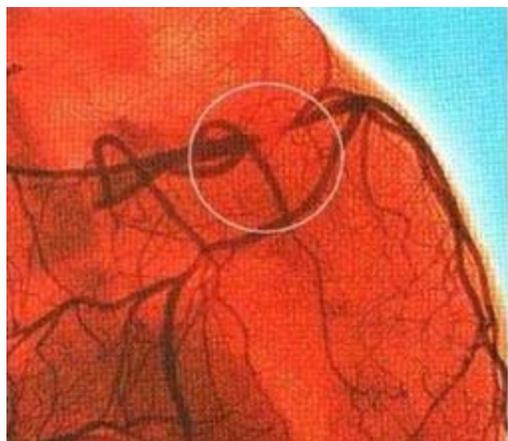


Fig. Angiography

Physical:

For most patients with stable angina, physical examination findings are normal. Diagnosing secondary causes of angina, such as aortic stenosis, is important.

A positive Levine sign (characterized by the patient's fist clenched over the sternum when describing the discomfort) is suggestive of angina pectoris.

Look for physical signs of abnormal lipid metabolism (eg, xanthelasma, xanthoma) or of diffuse atherosclerosis (eg, absence or diminished peripheral pulses, increased light reflexes or arteriovenous nicking upon ophthalmic examination, carotid bruit).

Examination of patients during the angina attack may be more helpful. Useful physical findings include third and/or fourth heart sounds due to LV systolic and/or diastolic dysfunction and mitral regurgitation secondary to papillary muscle dysfunction.

Pain produced by chest wall pressure is usually of chest wall origin.

Causes:

Decrease in myocardial blood supply due to increased coronary resistance in large and small coronary arteries

Significant coronary atherosclerotic lesion in the large epicardial coronary arteries (ie, conductive vessels) with at least a 50% reduction in arterial diameter

Coronary spasm (ie, Prinzmetal angina)

Abnormal constriction or deficient endothelial-dependent relaxation of resistant vessels associated with diffuse vascular disease (ie, microvascular angina)

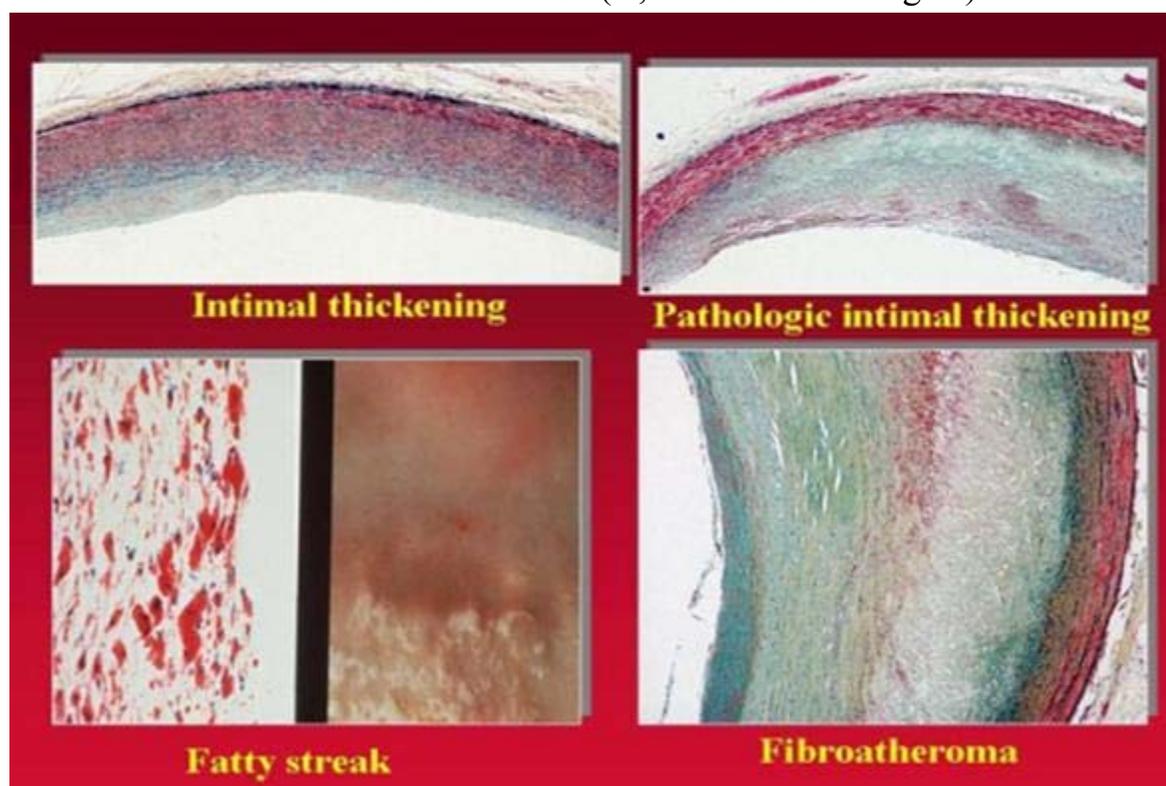


Fig. 4 Pathogenesis of atherosclerosis

Syndrome X

Systemic inflammatory or collagen vascular disease, such as scleroderma, systemic lupus erythematosus, Kawasaki disease, polyarteritis nodosa, and Takayasu arteritis

Increased extravascular forces, such as severe LV hypertrophy caused by hypertension, aortic stenosis, or hypertrophic cardiomyopathy, or increased LV diastolic pressures

Reduction in the oxygen-carrying capacity of blood, such as elevated carboxyhemoglobin or severe anemia (hemoglobin, <8 g/dL)

Congenital anomalies of the origin and/or course of the major epicardial coronary arteries

Structural abnormalities of the coronary arteries

Congenital coronary artery aneurysm or fistula

Coronary artery ectasia

Coronary artery fibrosis after chest radiation

Coronary intimal fibrosis following cardiac transplantation

Risk factors

Major risk factors for atherosclerosis: These include a family history of premature coronary artery disease, cigarette smoking, diabetes mellitus, hypercholesterolemia, or systemic hypertension.

Other risk factors: These include LV hypertrophy, obesity, and elevated serum levels of homocysteine, lipoprotein (a), plasminogen activator inhibitor, fibrinogen, serum triglycerides, or low high-density lipoprotein (HDL).

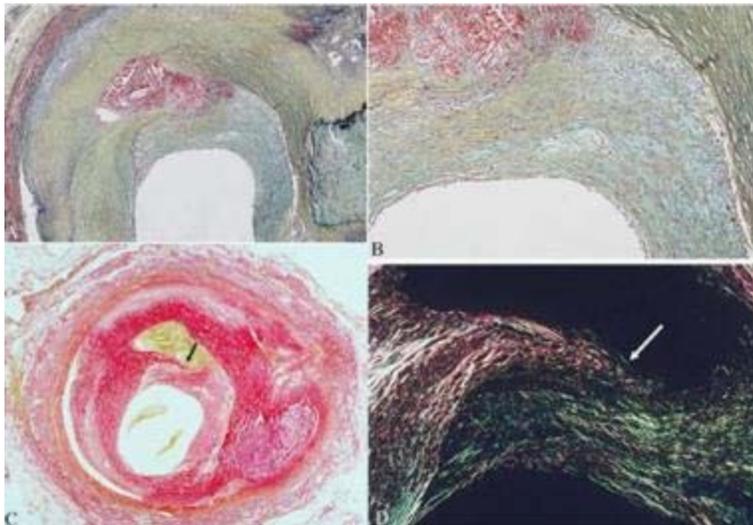


Fig. 5 Pathogenesis of atherosclerosis

Metabolic syndrome: This has recently been characterized by the presence of hyperinsulinemia (fasting glucose level, >110 mg/dL), abdominal obesity (waist circumference, >40 in for men or >35 in for women), decreased HDL cholesterol levels (<40 mg/dL for men or <50 mg/dL for women), hypertriglyceridemia (>150 mg/dL), and hypertension ($>130/85$ mm Hg). Based on data from the 2000 US census, an estimated 47 million Americans have the metabolic syndrome. Patients with the metabolic syndrome have a 3-fold increased risk for coronary atherosclerosis and stroke compared with those without this syndrome.

Precipitating factors: These include factors such as severe anemia, fever, tachyarrhythmias, catecholamines, emotional stress, and hyperthyroidism, which increase myocardial oxygen

demand.

Preventive factors: Factors associated with reduced risk of atherosclerosis are a high serum HDL cholesterol level, physical activity, estrogen, and moderate alcohol intake (1-2 drinks/d).

DIFFERENTIALS

Anemia, Anxiety Disorders , Aortic Dissection ,Aortic Stenosis ,Biliary Colic ,Cardiomyopathy, Hypertrophic ,Cholecystitis ,Coronary Artery Atherosclerosis ,Coronary Artery Vasospasm ,Diabetes Mellitus, Type 1 ,Diabetes ,ellitus, Type 2 ,Gastric Ulcers ,Gastritis, Acute ,Gastroesophageal Reflux Disease ,Hiatal Hernia ,Hypercholesterolemia, Familial ,Hypercholesterolemia, Polygenic ,Hypertension ,Hyperthyroidism ,Isolated Coronary Artery ,nomalies ,Kawasaki Disease ,Mitral Regurgitation ,Mitral Valve Prolapse ,Panic Disorder ,Pericarditis, Acute ,Pleurodynia ,Pneumothorax ,Polyarteritis Nodosa ,Pott Disease (Tuberculous Spondylitis) ,Pulmonary Embolism ,Pulmonary Hypertension, Primary ,Pulmonary Hypertension, Secondary ,Scleroderma ,Systemic Lupus Erythematosus ,Takayasu Arteritis ,Toxicity, Cocaine ,Varicella-Zoster Virus

Imaging Studies:

Chest radiograph findings are usually normal in patients with angina pectoris. However, they may show cardiomegaly in patients with previous MI, ischemic cardiomyopathy, pericardial effusion, or acute pulmonary edema. Calcification of coronary arteries frequently correlates with major coronary artery disease.

Graded exercise stress testing is the most widely used test for the evaluation of patients presenting with chest pain. In patients with established stable angina pectoris, it also can provide prognostic information about the extent of disease.

Exercise stress testing can be performed alone and in conjunction with echocardiography or myocardial perfusion scintigraphy tests. Stress echocardiography has an overall sensitivity of 78% and specificity of 86%; myocardial perfusion scintigraphy has an overall sensitivity of 83% and specificity of 77%. Exercise stress testing alone generally has somewhat lower sensitivity and specificity, but it is cheaper and therefore is a reasonable choice in those with a low probability of disease.

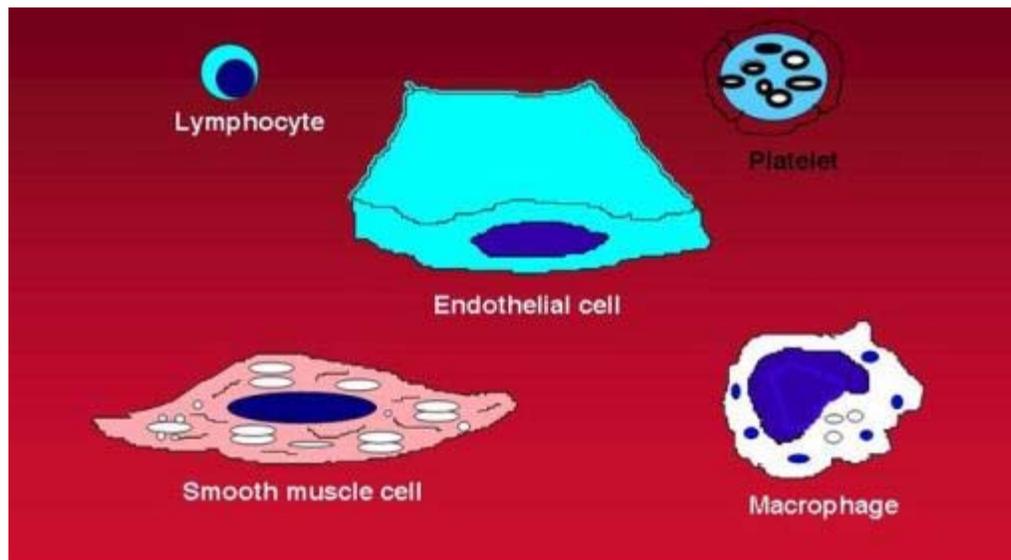


Fig. Cells which take part in atherosclerosis

These test results must be interpreted in the context of the likelihood of the presence of coronary artery disease determined from the patient's history and physical examination findings. In a population with low prevalence, the predictive abilities of these tests are low; however, in patients with a high likelihood of coronary artery disease, the predictive value is much higher.

Stress echocardiography can be used to evaluate segmental wall motion during exercise. It detects changes in regional wall motion that occur during myocardial ischemia. Normal myocardium becomes hyperdynamic during exercise; ischemic segments become hypokinetic or akinetic.

Stress echocardiography has the advantage of simultaneous evaluation of LV function, cardiac dimensions, and valvular disease. It is especially useful in patients with baseline ECG abnormalities and those with systolic murmurs suggestive of aortic stenosis or hypertrophic cardiomyopathy.

It is also helpful for localizing ischemia and evaluating its severity.

Signs of severe coronary artery disease during exercise stress echocardiography include LV dilation, a decrease in global systolic function, and new or worsening mitral regurgitation.

However, with dobutamine stress echocardiography, even in patients with severe coronary artery disease, the LV cavity may not dilate and global systolic function may improve.

A major problem with stress echocardiography is the technical difficulty with obtaining adequate images in some patients.

Thallium Tl 201 and technetium Tc 99m sestamibi are the most frequently used myocardial

perfusion scintigraphy tests. These tests are especially useful in patients with baseline ECG abnormalities, to localize the region of ischemia, and as prognostic indicators. The presence of increased lung uptake upon thallium imaging is associated with a poor prognosis. Increased lung uptake, together with poststress dilation of the LV and multiple perfusion defects, is suggestive of either left main coronary artery disease or severe 3-vessel disease. The number of affected myocardial segments is predictive of long-term survival. Smaller perfusion defects are usually associated with peripheral coronary artery lesions, which are associated with a better prognosis. The absence of perfusion defects even in the presence of symptoms indicates an excellent prognosis.

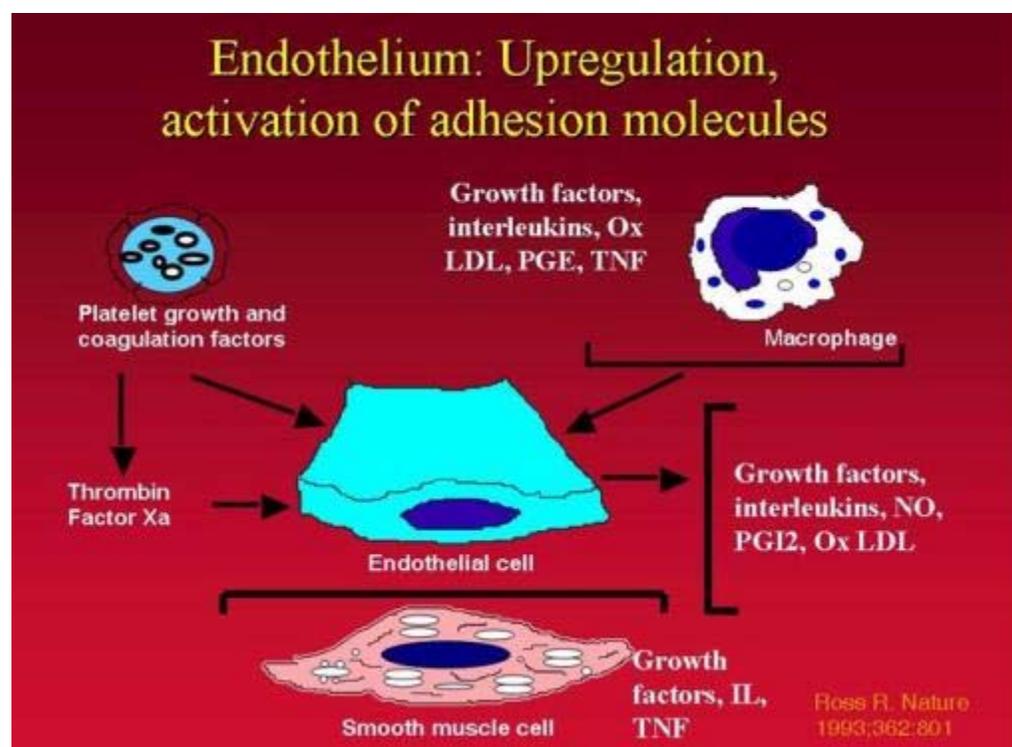


Fig. 9 Pathogenesis of atherosclerosis

The frequency of infarction or death is 1 case per 10,000 stress tests. Absolute contraindications include symptomatic cardiac arrhythmias, severe aortic stenosis, acute MI within the previous 2 days, acute myocarditis, or pericarditis. Discontinue the exercise stress test in the presence of chest pain, a drop in systolic blood pressure of more than 10 mm Hg, severe shortness of breath, fatigue, dizziness or near syncope, ST depression of more than 2 mm, ST elevation of at least 1 mm without diagnostic Q waves, or development of ventricular tachyarrhythmia.

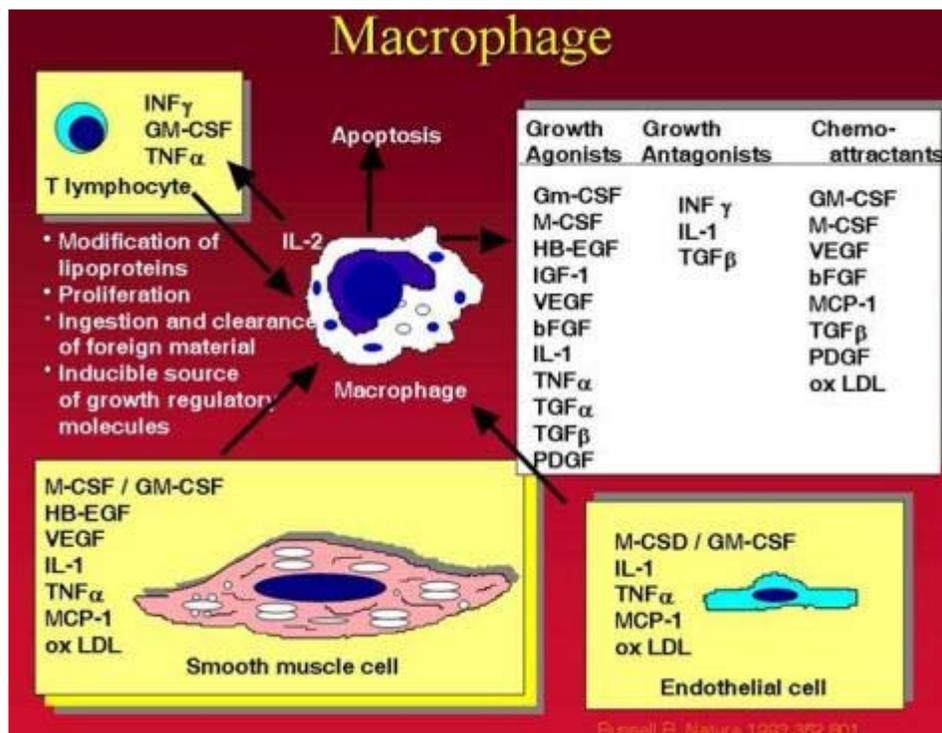


Fig. 10 Pathogenesis of atherosclerosis

Other Tests:

ECG is useful for evaluating persons with angina pectoris; however, findings are variable among patients.

Approximately 50% of patients with angina pectoris have normal findings after a resting ECG. However, abnormalities such as evidence for prior MI, intraventricular conduction delay, various degrees of atrioventricular block, arrhythmias, or ST-T-wave changes may be seen. During an attack of angina pectoris, 50% of patients with normal findings after resting ECG show abnormalities. A 1-mm or greater depression of the ST segment below the baseline, measured 80 milliseconds from the J point, is the most characteristic change. Reversible ST-segment elevation occurs with Prinzmetal angina. Some patients with coronary artery disease may show pseudonormalization of the resting ECG ST-T-wave abnormalities during episodes of chest pain.

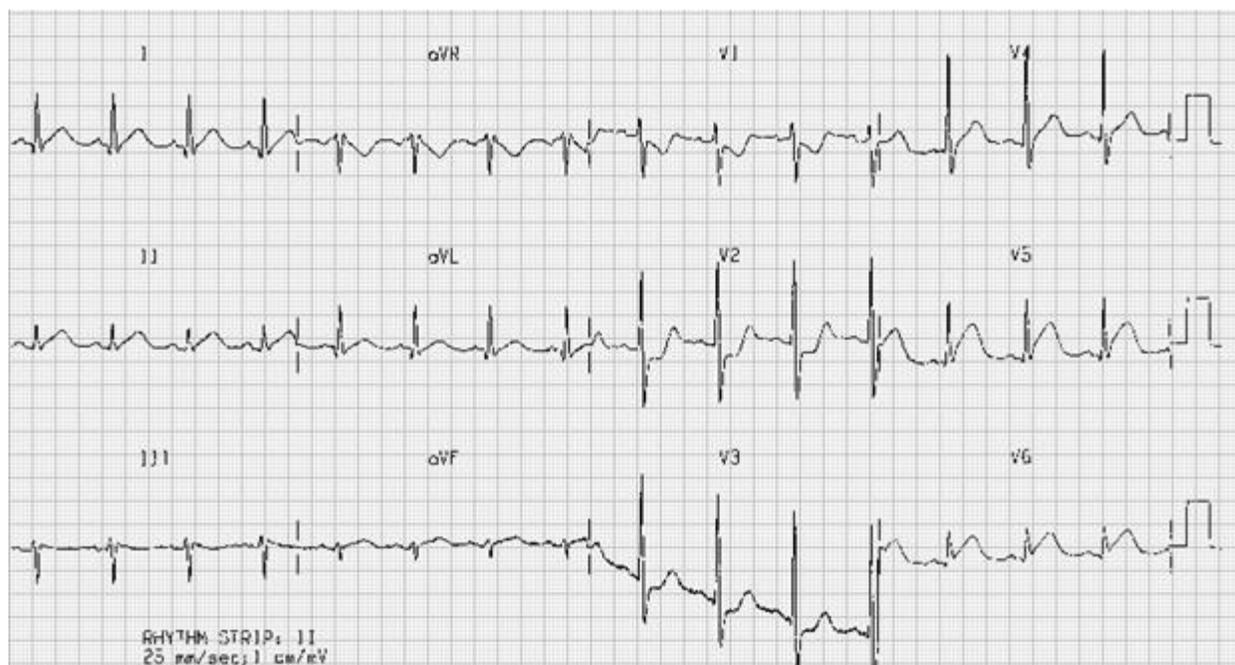


Fig 11. Subendocardial ischemia

Exercise with ECG monitoring alone is the initial procedure of choice in patients without baseline ST-segment abnormalities or in whom anatomic localization of ischemia is not a consideration.

Horizontal or down-sloping ST-segment depression of at least 1 mm, measured 80 milliseconds from the J point, is considered the characteristic ischemic response.

ST-segment depression of more than 2 mm at a low workload or that persists for more than 5 minutes after termination of exercise and a failure of blood pressure to rise or an actual drop in blood pressure are signs of severe ischemic heart disease and a poor prognosis.

Withhold beta-blockers for approximately 48 hours before the stress test, whenever possible. Patients on digoxin and those with LV hypertrophy with repolarization abnormalities more often show positive results. Exercise stress tests have lower sensitivity and specificity in women and in patients with left bundle-branch block.

Pharmacologic agents (eg, dobutamine, dipyridamole, adenosine) can be used in patients who are unable to exercise.

Ambulatory ECG monitoring can be used for diagnostic purposes in patients with chest pain suggestive of Prinzmetal angina but is primarily used to evaluate the frequency of silent ischemia. Silent ischemia has been shown to be an independent predictor of mortality in patients with angina pectoris.

Several studies have shown that calcium in the coronary arteries as detected by electron-beam computed tomography is an important indicator of coronary artery stenosis. In these studies,

the sensitivity of a positive electron-beam computed tomography scan ranged from 85-100% and the specificity varied from 41-76%, while the positive predictive value varied from 55-84% and the negative predictive value varied from 84-100%. However, several studies have shown inconsistent reproducibility in repeated measures of coronary calcium with electron-beam computed tomography. Thus, its proper role at this time remains controversial.

Procedures:

Selective coronary angiography is the definitive diagnostic test for evaluating the anatomic extent and severity of coronary artery disease.

Consider coronary angiography in symptomatic patients with inconclusive noninvasive study results, in survivors of sudden cardiac death, in those who are considered to have a poor prognosis based on the results of noninvasive studies, in those with occupational requirements for a definite diagnosis (eg, pilots), or in patients with coronary artery disease who are severely symptomatic despite maximal medical therapy.

In patients in whom Prinzmetal angina is suggested, provocative testing with ergonovine maleate during coronary angiography may be useful.

Intra-aortic balloon counterpulsation can be used in patients who continue to have unstable angina pectoris despite maximal medical treatment. This procedure should be followed promptly by coronary angiography with possible coronary revascularization.

In patients whose angina is refractory to medical therapy who are not suitable candidates for either percutaneous or surgical revascularization, enhanced external counterpulsation is a safe and noninvasive alternative therapy. It increases coronary perfusion and reduces myocardial oxygen demand by diastolic augmentation of the central aortic pressure. Several studies have shown that patients treated with enhanced external counterpulsation have a significantly reduced number of anginal episodes, improved exercise tolerance, and decreased daily use of nitroglycerin tablets. Its therapeutic effects on quality of life are noted to remain at 1-year follow-up.

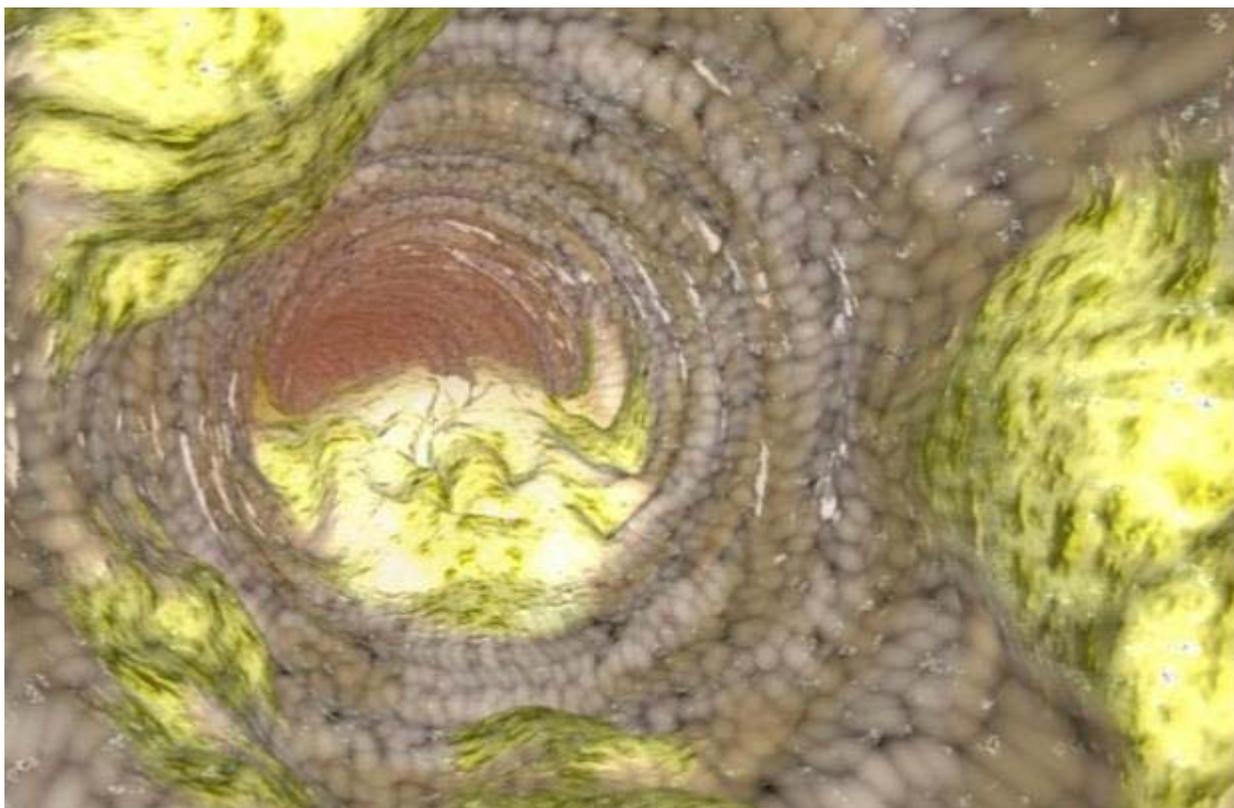


Fig. Vessels atherosclerosis

TREATMENT

Medical Care: The main goals of treatment in angina pectoris are to relieve the symptoms, slow the progression of disease, and reduce the possibility of future events, especially MI and premature death.

General measures

Smoking cessation results in a significant reduction of acute adverse effects on the heart and may reverse, or at least slow, atherosclerosis. Strongly encourage patients to quit smoking, and take an active role in helping them to achieve this goal.

Treat risk factors, including hypertension, diabetes mellitus, obesity, and hyperlipidemia.

Several clinical trials have shown that in patients with established coronary artery disease, reduction of low-density lipoprotein (LDL) level with a beta-hydroxy-beta-methylglutaryl coenzyme A reductase inhibitor (ie, statin) is associated with significant reductions in both mortality rate and major cardiac events.

These benefits are present even in patients with mild-to-moderate elevations of LDL cholesterol level.

Recent trials with cholesterol-lowering agents have confirmed the benefits of the therapeutic LDL lowering in older persons.

Angiographic studies demonstrate that a reduction of the LDL level in patients with coronary artery disease could cause slowing of progression, stabilization, or even regression of coronary artery lesions.

A recent study demonstrates a significant reduction of symptomatic myocardial ischemia in patients with unstable angina or non-Q-wave infarction with the administration of a statin during the early acute phase.

In a more recent study of 10,001 patients with stable coronary artery disease, an aggressive cholesterol-lowering approach with atorvastatin 80 mg daily (mean cholesterol level of 77 mg/dL) compared to a less-aggressive approach with atorvastatin 10 mg daily (mean cholesterol level of 101 mg/dL) resulted in a 2.2% absolute reduction and a 22% relative reduction in the occurrence of a first major cardiovascular event (defined as death from coronary heart disease; nonfatal, non-procedure-related myocardial infarction; resuscitation from cardiac arrest; or fatal or nonfatal stroke). This occurred with a greater incidence of elevated aminotransferase levels with the aggressive cholesterol-lowering approach (1.2% vs 0.2%, $p < 0.001$).

On the basis of several recent studies that have demonstrated the benefits of more aggressive LDL-lowering therapies in high-risk patients with coronary artery disease, the Committee of the National Cholesterol Education Program recently made the following modifications to the Adult Treatment Panel III (ATP III) guidelines.

In high-risk patients, a serum LDL cholesterol level of less than 100 mg/dL is the goal.

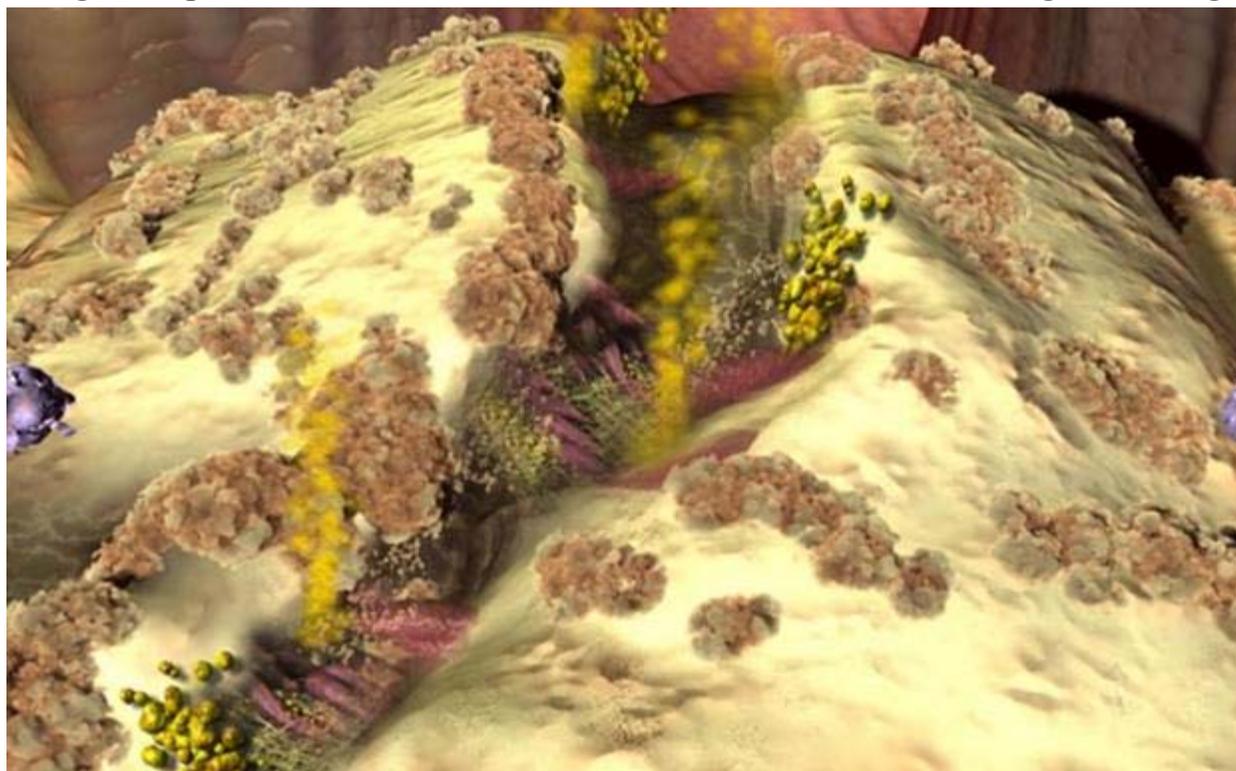


Fig. Vessels atherosclerosis

In very high-risk patients, an LDL cholesterol level goal of less than 70 mg/dL is a therapeutic option. Patients in the category of very high risk are those with established coronary artery disease with one of the following: multiple major risk factors (especially diabetes), severe and poorly controlled risk factors (especially continued cigarette smoking), multiple risk factors of the metabolic syndrome (especially high triglyceride levels [>200 mg/dL] plus non-HDL cholesterol level [>130 mg/dL] with low HDL cholesterol level [<40 mg/dL]), and patients with acute coronary syndromes.

For moderately high-risk persons (2+ risk factors), the recommended LDL cholesterol level is less than 130 mg/dL, but an LDL cholesterol level of 100 mg/dL is a therapeutic option.

Some triglyceride-rich lipoproteins, including partially degraded very LDL levels, are believed to be independent risk factors for coronary artery disease. In daily practice, non-HDL cholesterol level (ie, LDL + very LDL cholesterol [total cholesterol - HDL cholesterol]) is the most readily available measure of the total pool of these atherogenic lipoproteins. Thus, the ATP III has identified non-HDL cholesterol level as a secondary target of therapy in persons with high triglyceride levels (>200 mg/dL). The goal for non-HDL cholesterol level (for persons with serum triglyceride levels >200 mg/dL) is 30 mg/dL higher than the identified LDL cholesterol level goal.

Patients with established coronary disease and low HDL cholesterol levels are at high risk for recurrent events and should be targeted for aggressive nonpharmacological (ie, dietary modification, weight loss, physical exercise) and pharmacological treatment.

A recent study demonstrated that in patients with established coronary artery disease who have low HDL and low-risk LDL levels, drug therapy with medications that raise HDL cholesterol levels and lower triglyceride levels but have no effect on LDL cholesterol levels (eg, gemfibrozil) could significantly reduce the risk of major cardiac events.

Currently, the accepted approach to the management of patients with coronary artery disease and low HDL levels is as follows:

In all persons with low HDL cholesterol levels, the primary target of therapy is to achieve the ATP III guideline LDL cholesterol level goals with diet, exercise, and drug therapy as needed. After reaching the targeted LDL level goal, emphasis shifts to other issues. That is, in patients with low HDL cholesterol levels who have associated high triglyceride levels (>200 mg/dL), the secondary priority is to achieve the non-HDL cholesterol level goal of 30 mg/dL higher than the identified LDL cholesterol level goal. In patients with isolated low HDL cholesterol levels (triglycerides <200 mg/dL), drugs to raise the HDL cholesterol level (eg, gemfibrozil,

nicotinic acid) can be considered.

Exercise training results in improvement of symptoms, increase in the threshold of ischemia, and improvement of patients' sense of well-being. However, before enrolling a patient in an exercise-training program, perform an exercise tolerance test to establish the safety of such a program.

Consider enteric-coated aspirin at a dose of 80-325 mg/d for all patients with stable angina who have no contraindications to its use. In patients in whom aspirin cannot be used because of allergy or gastrointestinal complications, consider clopidogrel.

Although early observational studies suggested a cardiovascular protective effect with the use of hormone replacement therapy, recent large randomized trials failed to demonstrate any benefit with hormone replacement therapy in the primary or secondary prevention of cardiovascular disease.

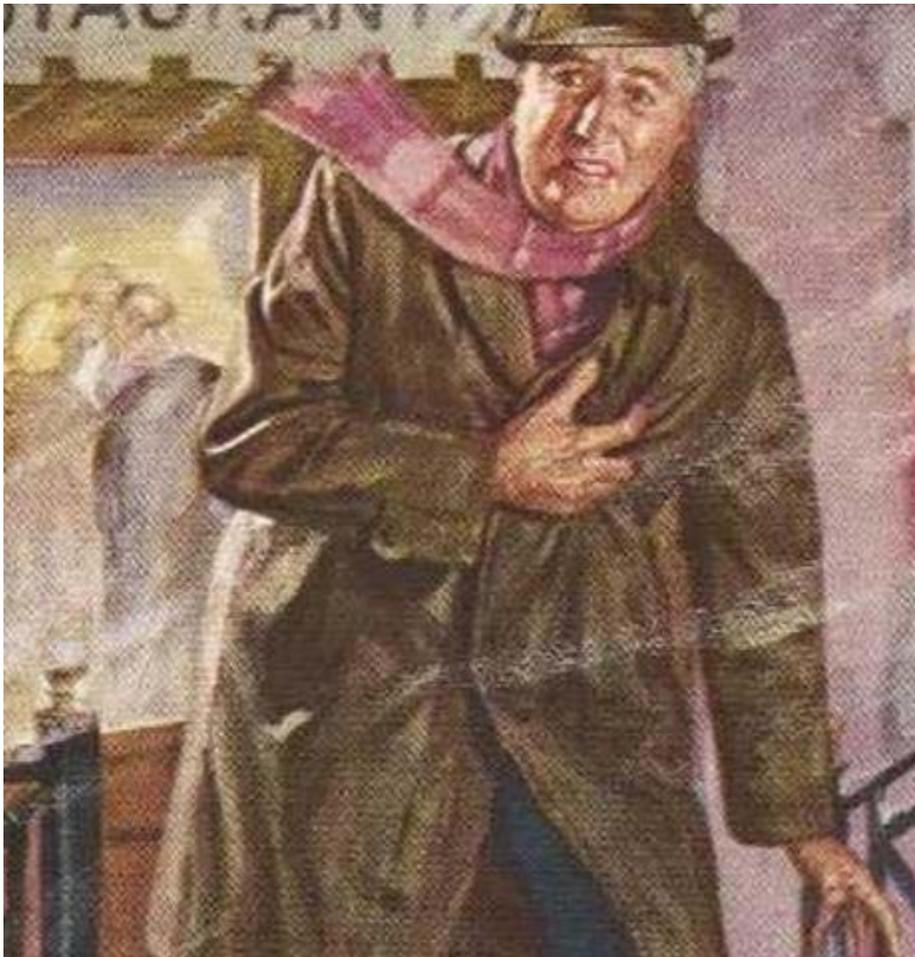


Fig. Patient with IHD

In fact, these studies even demonstrated an increased risk of coronary artery disease and stroke in patients on hormone replacement therapy.

The Women's Health Initiative study demonstrated that the use of hormone replacement therapy

for 1 year in 10,000 healthy postmenopausal women is associated with 7 more instances of coronary artery disease, 8 more strokes, 8 more pulmonary emboli, 8 more invasive breast cancers, 5 fewer hip fractures, and 6 fewer colorectal cancers.

Based on these data, the risks and benefits of hormone replacement therapy must be assessed on an individual basis for each patient.

Sublingual nitroglycerin has been the mainstay of treatment for angina pectoris. Sublingual nitroglycerin can be used for acute relief of angina and prophylactically before activities that may precipitate angina. No evidence indicates that long-acting nitrates improve survival in patients with coronary artery disease.

Beta-blockers are also used for symptomatic relief of angina and prevention of ischemic events. They work by reducing myocardial oxygen demand and by decreasing the heart rate and myocardial contractility. Beta-blockers have been shown to reduce the rates of mortality and morbidity following acute MI.

Long-acting heart rate–slowing calcium channel blockers can be used to control anginal symptoms in patients with a contraindication to beta-blockers and in those in whom symptomatic relief of angina cannot be achieved with the use of beta-blockers, nitrates, or both. Avoid short-acting dihydropyridine calcium channel blockers because they have been shown to increase the risk of adverse cardiac events.

Anginal symptoms in patients with Prinzmetal angina can be treated with calcium channel blockers with or without nitrates. In one study, supplemental vitamin E added to a calcium channel blocker significantly reduced anginal symptoms among such patients.

In patients with syndrome X and hypertension, ACE inhibitors may normalize thallium perfusion defects and increase exercise capacity.

Surgical Care:

Revascularization therapy (ie, coronary revascularization) can be considered in patients with left main artery stenosis greater than 50%, 2- or 3-vessel disease and LV dysfunction (ejection fraction, <45%), poor prognostic signs during noninvasive studies, or severe symptoms despite maximum medical therapy. The 2 main coronary revascularization procedures are percutaneous transluminal coronary angioplasty, with or without coronary stenting, and coronary artery bypass grafting.

Patients with 1- or 2-vessel disease and normal LV function who have anatomically suitable lesions are candidates for percutaneous transluminal coronary angioplasty and coronary stenting. Restenosis is the major complication, with symptomatic restenosis occurring in 20-25% of patients. Restenosis mostly occurs during the first 6 months after the procedure and

can be managed by repeat angioplasty. Several recent trials have demonstrated that the use of drug-eluting stents (eg, sirolimus-eluting stents, paclitaxel-coated stents) can remarkably reduce the rate of in-stent restenosis. Recently, with the introduction of these drug-coated stents, patients with multivessel coronary artery disease are more frequently treated with percutaneous revascularization as opposed to the surgical revascularization.

Patients with single-vessel disease and normal ventricular function treated with percutaneous transluminal coronary angioplasty show improved exercise tolerance and fewer episodes of angina compared with those who receive medical treatment. However, no difference in the frequency of MI or death has been shown between these two groups.

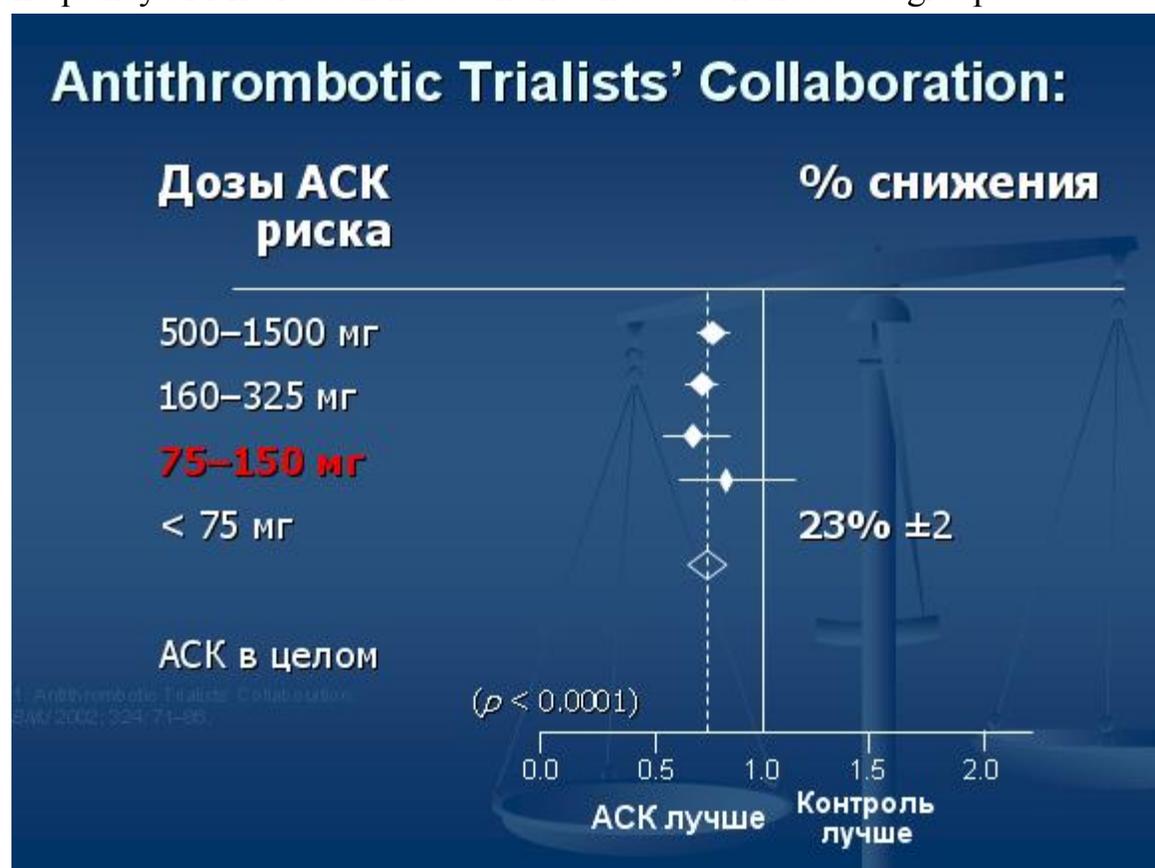


Fig. Pathogenesis of atherosclerosis

Patients with significant left main coronary artery disease, 2- or 3-vessel disease and LV dysfunction, diabetes mellitus, or lesions anatomically unsuitable for percutaneous transluminal coronary angioplasty have better results with coronary artery bypass grafting. The overall operative mortality rate for coronary artery bypass grafting is approximately 1.3%. The rate of graft patency 10 years after surgery is less than 50% for vein grafting, although more than 90% of grafts using internal mammary arteries are patent at 10 years. In recent years, interest has increased regarding surgery without cardiopulmonary bypass (ie, off-pump) in an attempt to avoid the morbidity associated with cardiopulmonary bypass. A recent randomized

study demonstrated that off-pump coronary surgery was as safe as on-pump surgery and caused less myocardial damage. However, the graft-patency rate was lower at 3 months in the off-pump group than in the on-pump group.

Recently, laser transmyocardial revascularization has been used as an experimental therapy for the treatment of severe, chronic, stable angina refractory to medical or other therapies. This technique has been performed with either an epicardial surgical technique or by a percutaneous approach. In both approaches, a series of transmural endomyocardial channels are created to improve myocardial perfusion. The surgical transmyocardial revascularization technique has been associated with symptomatic relief for end-stage chronic angina in the short term. However, no published data address the long-term efficacy of surgical transmyocardial revascularization. Nonetheless, this technique appears to provide at least symptomatic relief for end-stage chronic angina in the short term.

Diet: A diet low in saturated fat and dietary cholesterol is the mainstay of the Step I and Step II diet from the American Heart Association.

Activity: The level of activity that aggravates anginal symptoms is different for each patient. However, most patients with stable angina can avoid symptoms during daily activities simply by reducing the speed of activity.

MEDICATION

The goals of pharmacotherapy are to reduce morbidity and to prevent complications.

Drug Category: *Antiplatelet agents* -- Prevent thrombus formation by inhibiting platelet aggregation. Aspirin is proven beneficial in primary and secondary prevention of coronary artery disease. In patients with aspirin intolerance, use clopidogrel. Clopidogrel is also used in combination with aspirin after coronary stent placement. Recently, clopidogrel use in addition to aspirin has been shown to be significantly superior to aspirin alone in patients with acute coronary syndrome without ST-segment elevation MI.

Drug Name	Aspirin (Bayer, Empirin, Anacin) -- Prevents platelet aggregation by irreversible cyclooxygenase inhibition with subsequent suppression of thromboxane A ₂ . Antiplatelet effect can last as long as 7 d.
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Adult Dose	81-325 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye syndrome, do not use in children (<16 y) with flu
Interactions	Antacids and urinary alkalinizers may decrease effects; corticosteroids decrease salicylate serum levels; anticoagulants may cause additive hypoprothrombinemic effects and increased bleeding time; may antagonize uricosuric effects of probenecid and increase toxicity of phenytoin and valproic acid; doses >2 g/d may potentiate glucose-lowering effect of sulfonylurea drugs
Pregnancy	D - Unsafe in pregnancy
Precautions	May cause transient decrease in renal function and aggravate chronic kidney disease; avoid use in patients with severe anemia, history of blood coagulation defects, or taking anticoagulants; adverse effects include prolonged bleeding time, rhinitis, asthma, urticaria, and exacerbation of gout; monitor BP, BUN, and uric acid level; consider discontinuing 7 d before surgery
Drug Name	Clopidogrel (Plavix) -- Selectively inhibits ADP binding to platelet receptor and subsequent ADP-mediated activation of

	GPIIb/IIIa complex, thereby inhibiting platelet aggregation. Consider in patients with contraindication to aspirin.
Adult Dose	75 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; active pathological bleeding
Interactions	Naproxen associated with increased occult GI blood loss; prolongs bleeding time; safety of coadministration with warfarin not established
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in patients at increased risk of bleeding from trauma, surgery, or other pathological conditions; caution in patients with lesions with propensity to bleed (eg, ulcers); adverse effects include rash, diarrhea, purpura, GI ulcers, neutropenia, and rare cases of agranulocytosis; consider discontinuing 7 d before surgery

Drug Category: *Beta-adrenergic blocking agents* -- Work by competing with endogenous catecholamines for beta-adrenergic receptors. Reduce myocardial oxygen consumption via several effects, including decrease in resting and exercise heart rates and reductions in myocardial contractility and afterload. Classified as nonselective, beta-1 selective, and having intrinsic sympathomimetic effects.

Drug Name	Metoprolol (Lopressor, Toprol XL) -- Selective beta1-adrenergic receptor
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	blocker that decreases automaticity of contractions. Is lipophilic and penetrates CNS.
Adult Dose	50-200 mg PO bid
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; uncompensated CHF; bradycardia; asthma; cardiogenic shock; AV conduction abnormalities
Interactions	Aluminum salts, barbiturates, NSAIDs, penicillins, calcium salts, cholestyramine, and rifampin may decrease bioavailability and plasma levels, possibly resulting in decreased pharmacologic effects; sparfloxacin, phenothiazines, astemizole, calcium channel blockers, quinidine, flecainide, and contraceptives may increase toxicity; may increase toxicity of digoxin, flecainide, clonidine, epinephrine, nifedipine, prazosin, verapamil, and lidocaine
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Beta-adrenergic blockade may mask signs and symptoms of acute hypoglycemia and may decrease clinical signs of hyperthyroidism; abrupt withdrawal may exacerbate symptoms of hyperthyroidism, including thyroid storm; monitor patient closely and withdraw drug slowly; during IV administration, carefully monitor BP, heart rate, and ECG; adverse effects include

	hypotension, decreased libido, impotence, lethargy, depression, and decreased HDL; may cause less bronchial tree and arterial smooth muscle constriction
Drug Name	Atenolol (Tenormin) -- Selectively blocks beta-1 receptors with little or no effect on beta-2 receptors. Is hydrophilic and does not penetrate CNS.
Adult Dose	50-200 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; CHF; pulmonary edema; cardiogenic shock; AV conduction abnormalities; heart block (without pacemaker)
Interactions	Aluminum salts, barbiturates, calcium salts, cholestyramine, NSAIDs, penicillins, and rifampin may decrease effects; haloperidol, hydralazine, loop diuretics, and MAOIs may increase toxicity
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Beta-adrenergic blockade may hide symptoms of acute hypoglycemia and mask signs of hyperthyroidism; abrupt withdrawal may exacerbate symptoms of hyperthyroidism and cause thyroid storm; monitor patients closely and withdraw drug slowly; adverse effects include bradycardia, hypotension, decreased libido, impotence, and decreased HDL; beta1-selective blockers may cause less bronchial tree and arterial smooth muscle constriction; titrate

	dose carefully to level of patient tolerance and effectiveness
Drug Name	Propranolol (Inderal) -- Nonselective beta-blocker that is lipophilic (penetrates CNS). Although generally short-acting agent, long-acting preparations also available.
Adult Dose	IR: 40-160 mg PO bid SR: 60-320 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; history of bronchospasm; uncompensated CHF; bradycardia; cardiogenic shock; AV conduction abnormalities
Interactions	Aluminum salts, barbiturates, NSAIDs, penicillins, calcium salts, cholestyramine, and rifampin may decrease effects; calcium channel blockers, cimetidine, loop diuretics, and MAOIs may increase toxicity; may increase toxicity of hydralazine, haloperidol, benzodiazepines, and phenothiazines
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Beta-adrenergic blockade may mask signs of acute hypoglycemia and hyperthyroidism; abrupt withdrawal may exacerbate symptoms of hyperthyroidism, including thyroid storm; withdraw drug slowly and monitor closely; adverse effects include bronchial constriction, Raynaud phenomenon, hypotension, decreased

	libido, impotence, lethargy, depression, and decreased HDL; caution in Wolff-Parkinson-White syndrome and renal or hepatic dysfunction
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Drug Category: *Calcium channel blockers* -- Reduce transmembrane flux of calcium via calcium channels. Cause smooth muscle relaxation, resulting in peripheral arterial vasodilation and afterload reduction. Indicated when symptoms persist despite treatment with beta-blockers or when beta-blockers are contraindicated. Also indicated in patients with Prinzmetal angina with or without nitrates.

Drug Name	Amlodipine (Norvasc) -- During depolarization, inhibits calcium ions from entering slow channels and voltage-sensitive areas of vascular smooth muscle and myocardium.
Adult Dose	5-10 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe CHF; sick sinus syndrome; second- or third-degree AV block; hypotension (<90 mm Hg systolic)
Interactions	Fentanyl may increase hypotensive effects; may increase cyclosporine levels; H2 blockers (eg, cimetidine) may increase toxic effects
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Severe aortic stenosis, CHF, hepatic dysfunction; adverse effects include headache, edema, flushing, palpitation, drowsiness, and fatigue

Drug Name	Diltiazem (Cardizem CD, Dilacor) -- During depolarization, inhibits calcium ions from entering slow channels and voltage-sensitive areas of vascular smooth muscle and myocardium.
Adult Dose	IR: 120-360 mg PO divided tid/qid SR: 120-480 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe CHF; sick sinus syndrome; second- or third-degree AV block; hypotension (<90 mm Hg systolic)
Interactions	May increase carbamazepine, digoxin, cyclosporine, and theophylline levels; when administered with amiodarone, may cause bradycardia and decrease in cardiac output; when given with beta-blockers may increase cardiac depression; cimetidine may increase levels
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in impaired renal or hepatic function; may increase LFT levels, and hepatic injury may occur; adverse effects include constipation, AV conduction block, worsening of heart failure, peripheral edema, bradycardia, and AV dissociation
Drug Name	Verapamil (Calan, Covera) -- During depolarization, inhibits calcium ion from entering slow channels or voltage-sensitive areas of vascular smooth muscle and myocardium.

Adult Dose	IR: 80-120 mg PO tid/qid SR: 120-240 mg PO qd/bid
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe CHF; sick sinus syndrome; second- or third-degree AV block; hypotension (<90 mm Hg systolic)
Interactions	May increase carbamazepine, digoxin, theophylline, and cyclosporine levels; amiodarone can cause bradycardia and decrease in cardiac output; when administered concurrently with beta-blockers may increase cardiac depression; cimetidine may increase levels
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Hepatocellular injury may occur; transient elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have occurred (elevations have been transient and may disappear with continued treatment); monitor liver function periodically; adverse effects include constipation, AV dissociation, worsening heart failure, bradycardia, negative inotropism, and hypotension

Drug Category: *Short-acting nitroglycerins* -- Suitable for immediate relief of exertional or rest angina. Can also be used for prophylaxis several minutes before planned exercise to avoid angina. Reduce myocardial oxygen demand by reduction of LV and arterial pressure, primarily by reducing preload.

Drug Name	Nitroglycerin (Nitrostat, Nitro-bid, Nitrol) -- Causes relaxation of vascular smooth muscle by stimulating intracellular cyclic GMP production. Result is decrease in BP.
Adult Dose	0.3-0.6 mg SL prn 0.4 mg metered-dose spray PO prn 0.1-0.8 mg/h patch TD qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe anemia; shock; postural hypotension; head trauma; closed-angle glaucoma; cerebral hemorrhage; hypertrophic obstructive cardiomyopathy
Interactions	Concurrent sildenafil (Viagra) may cause severe hypotension and death; aspirin may increase serum concentrations; calcium channel blockers may cause markedly symptomatic orthostatic hypotension (dose adjustment of either agent may be necessary)
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in coronary artery disease and low systolic BP; adverse effects include hypotension, flushing, headache, light-headedness, and tolerance (8- to 12-h nitrate-free interval is most effective method to prevent development of tolerance); high IV doses may cause methemoglobinemia, heparin resistance, and ethanol intoxication; ischemia may worsen upon withdrawal

Drug Category: *Long-acting nitroglycerins* -- Reduce LV preload and afterload by venous and arterial dilation, which subsequently reduces myocardial oxygen consumption and relieves angina. Also cause dilation of epicardial coronary arteries, which is beneficial in patients with coronary spasm. In addition, nitroglycerin has antithrombotic and antiplatelet effects in patients with angina pectoris. No evidence suggests that nitrates improve survival or slow progression of coronary artery disease.

Drug Name	Isosorbide (Isordil, ISMO) -- Relaxes vascular smooth muscle by stimulating intracellular cyclic GMP. Decreases LV pressure (ie, preload) and arterial resistance (ie, afterload). Reduces cardiac oxygen demand by decreasing LV pressure and dilating arteries.
Adult Dose	Isosorbide dinitrate: 2.5-10 mg SL prn IR 10-30 mg PO bid/tid SR 80-120 mg PO qd IR Isosorbide mononitrate: 10-20 mg PO bid SR 30-120 mg PO qd
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe anemia; closed-angle glaucoma; postural hypotension; head trauma; cerebral hemorrhage
Interactions	Alcohol may cause severe hypotension and cardiovascular collapse; aspirin may increase serum concentrations and actions; calcium channel blockers may increase symptomatic orthostatic

	hypotension (adjust dose of either agent); may decrease effects of heparin
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Tolerance to vascular and antianginal effects of nitrates may develop; minimize tolerance by using smallest effective dose or pulse therapy (intermittent dosing) or by alternating with other coronary vasodilators (take last daily dose of short-acting agent no later than 7 pm); caution when administering to patients with glaucoma

Drug Category: *Angiotensin-converting enzyme inhibitors* -- Recently shown to reduce rates of death, MI, stroke, and need for revascularization procedures in patients with coronary artery disease or diabetes mellitus and at least one other cardiovascular risk factor, irrespective of the presence of hypertension or heart failure.

Drug Name	Ramipril (Altace) -- Prevents conversion of angiotensin I to angiotensin II, a potent vasoconstrictor, resulting in lower aldosterone secretion.
Adult Dose	2.5-5 mg PO qd; not to exceed 20 mg/d
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; history of angioedema
Interactions	May increase digoxin, lithium, and allopurinol levels; probenecid may increase levels; coadministration with diuretics increases hypotensive effects; NSAIDs may reduce hypotensive effects
Pregnancy	C - Safety for use during pregnancy has not been established.

<p>Precautions</p>	<p>Category D in second and third trimesters of pregnancy; adverse effects include persistent cough, angioedema, hypotension, and prerenal azotemia; caution in renal impairment, valvular stenosis, or severe CHF</p>
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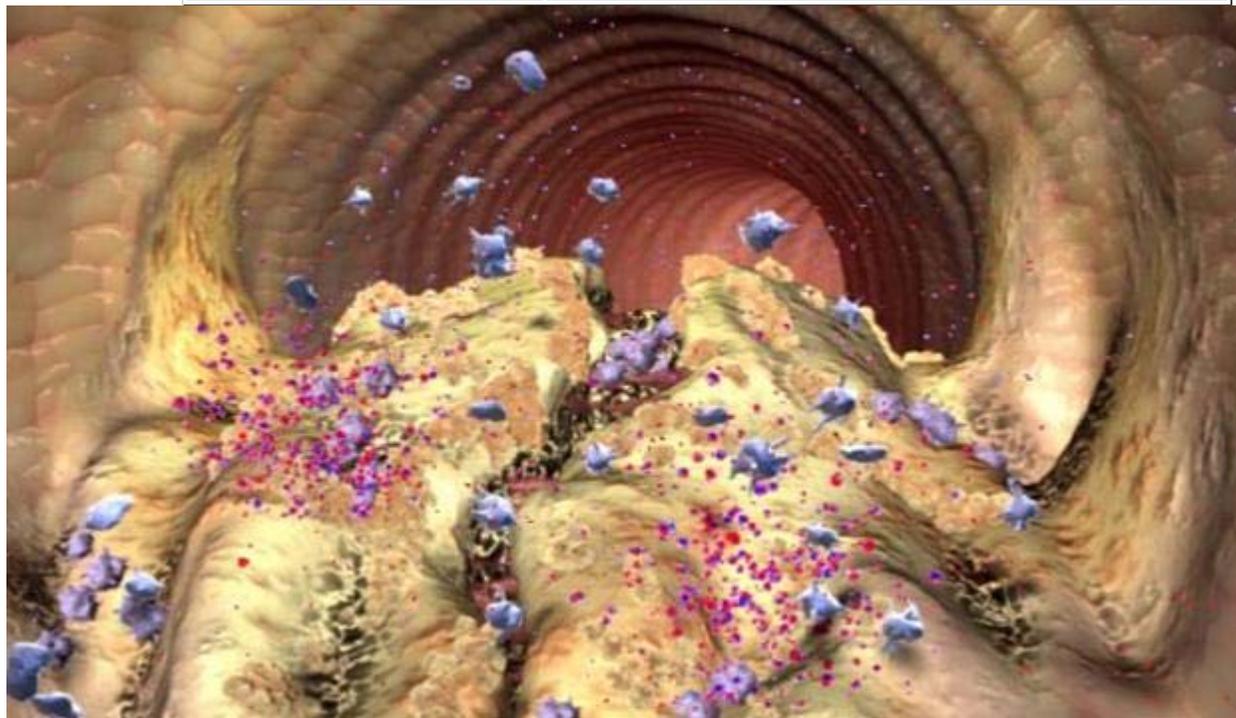


Fig Thrombosis of coronary artery

Drug Category: *Anti-ischemic agents, miscellaneous* -- Ranolazine elicits action unlike beta-blockers, calcium antagonists, or nitrates. It does not affect hemodynamics or contractile and conduction parameters.

<p>Drug Name</p>	<p>Ranolazine (Ranexa) -- Cardioselective anti-ischemic agent (piperazine derivative) that partially inhibits fatty acid oxidation. Also inhibits late sodium current into myocardial cells and prolongs QTc interval. Indicated for chronic angina unresponsive to other antianginal treatments. Used in combination with amlodipine, beta-blockers, or nitrates. Unlike beta-blockers, calcium channel blockers,</p>
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	and nitrates, does not reduce blood pressure or heart rate. Effect on angina rate or exercise tolerance appears to be smaller in women than in men. Absorption is highly variable but unaffected by food.
Adult Dose	500 mg PO bid initially; if necessary, may increase to 1000 mg PO bid
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; preexisting QT prolongation; hepatic impairment (Child-Pugh class A [mild], B [moderate], or C [severe]); QT-prolonging drugs (see Interactions); potent or moderate CYP4503A inhibitors (eg, ketoconazole, diltiazem)
Interactions	CYP4503A and P-gp substrate; potent CYP3A inhibitors (eg, ketoconazole at 200 mg bid) increase levels approximately 3.2-fold, moderate CYP3A inhibitors (eg, diltiazem at 180-360 mg/d) increase levels approximately 1.8- to 2.3-fold, and verapamil (a CYP3A and P-gp inhibitor) increases levels approximately 2-fold; caution with other P-gp inhibitors (eg, ritonavir, cyclosporine); toxicity may occur when coadministered with other drugs that increase QTc interval (eg, class I and III antiarrhythmic agents, certain macrolide and quinolone antibiotics, phenothiazines, TCAs) Inhibits CYP4503A, CYP 4502D6, and P-gp; may increase plasma levels of digoxin, simvastatin, dextromethorphan,

	TCAs, and antipsychotics
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Causes dose-related QTc-interval prolongation (obtain baseline and follow-up ECGs to monitor for torsades de pointes and potential for sudden death; mild and moderate hepatic impairment increases QTc interval compared with normal hepatic function at same plasma level; increases blood pressure by approximately 15 mm Hg in persons with severe renal impairment; common adverse effects include dizziness, headache, constipation, and nausea

FOLLOW-UP

Deterrence/Prevention:

Coronary atherosclerosis is the main preventable cause of mortality in the United States. A rigorous effort to address correctable risk factors is the mainstay of preventive cardiovascular medicine.

Smoking cessation is the single most effective preventive intervention to reduce coronary atherosclerosis prevalence. It has been associated with a coronary artery disease reduction of 7-47% in primary prevention settings.

Aggressive treatment of diabetes mellitus, hypertension, LV hypertrophy, hyperlipidemia, and obesity has an important role in the prevention of coronary artery disease.

The most important recent development in coronary atherosclerosis risk modification is the introduction of inhibitors of beta-hydroxy-beta-methylglutaryl coenzyme A reductase.

Reductions of total and LDL cholesterol levels by 25% and 35%, respectively, can achieve a similar reduction in rates of total and coronary mortality, MI, and need for coronary revascularization.

Complications:

Complications of angina pectoris include unstable angina, MI, and death.

Prognosis:

Important prognostic indicators in patients with angina pectoris include LV function, severity and location of atherosclerotic lesions, and response of symptoms to medical treatment.

LV function is the strongest predictor of long-term survival. Elevated LV end-diastolic pressure and volume along with reduced LV ejection fraction (<40%) are poor prognostic signs.

Critical lesions of left main and proximal left anterior descending coronary arteries are associated with a greater risk. Mortality rates are also directly associated with the number of epicardial arteries involved.

Unstable angina, recent MI, or both is a sign of atherosclerotic plaque instability, which is a strong predictor of increased risk of short-term coronary events.

A number of signs during noninvasive testing are predictive of a higher risk of coronary events, including ST-segment depression of more than 2 mm at a low workload, ST-segment depression that persists for more than 5 minutes after termination of exercise, and failure of blood pressure to rise or an actual drop in blood pressure.

Patients who continue to smoke after an MI have a 22-47% increased risk of reinfarction and death.

In general, Prinzmetal angina and syndrome X are associated with excellent long-term prognoses.

Patient Education:

Educating patients about the benefits of smoking cessation, a low-cholesterol diet, physical activity, and periodic screening for diabetes mellitus and hypertension is the prime component of a long-term management plan.

For excellent patient education resources, visit eMedicine's Circulatory Problems Center, Cholesterol Center, Heart Center, and Statins Center. Also, see eMedicine's patient education articles Angina Pectoris, High Cholesterol, Understanding Your Cholesterol Level, Lifestyle Cholesterol Management, Understanding Cholesterol-Lowering Medications, Chest Pain, Coronary Heart Disease, and Heart Attack.

MISCELLANEOUS**Medical/Legal Pitfalls:**

In patients with stable angina pectoris, even the most carefully performed history and physical examination have limitations. Classification of these patients solely on the basis of history and physical examination findings may lead to serious mistakes. Some type of stress testing is usually indicated to confirm the diagnosis and quantitate the severity of ischemia.

In women, elderly persons, and diabetic patients, coronary artery disease may manifest with atypical presentations other than angina pectoris, such as silent ischemia or infarction. Physicians should use a careful approach when evaluating these patients.

Classical and untypical variants of the acute myocardial infarction, criteria for diagnosis. Differential programs of treatment of the uncomplicated myocardial infarction, physical rehabilitation

Background: Myocardial infarction (MI) is the irreversible necrosis of heart muscle secondary to prolonged ischemia. This usually results from an imbalance of oxygen supply and demand. The appearance of cardiac enzymes in the circulation generally indicates myocardial necrosis. MI is considered, more appropriately, part of a spectrum referred to as acute coronary syndromes (ACSs), which also includes unstable angina and non–ST-elevation MI (NSTEMI). Patients with ischemic discomfort may or may not have ST-segment elevation. Most of those with ST-segment elevation will develop Q waves. Those without ST elevations will ultimately be diagnosed with unstable angina or NSTEMI based on the presence of cardiac enzymes.

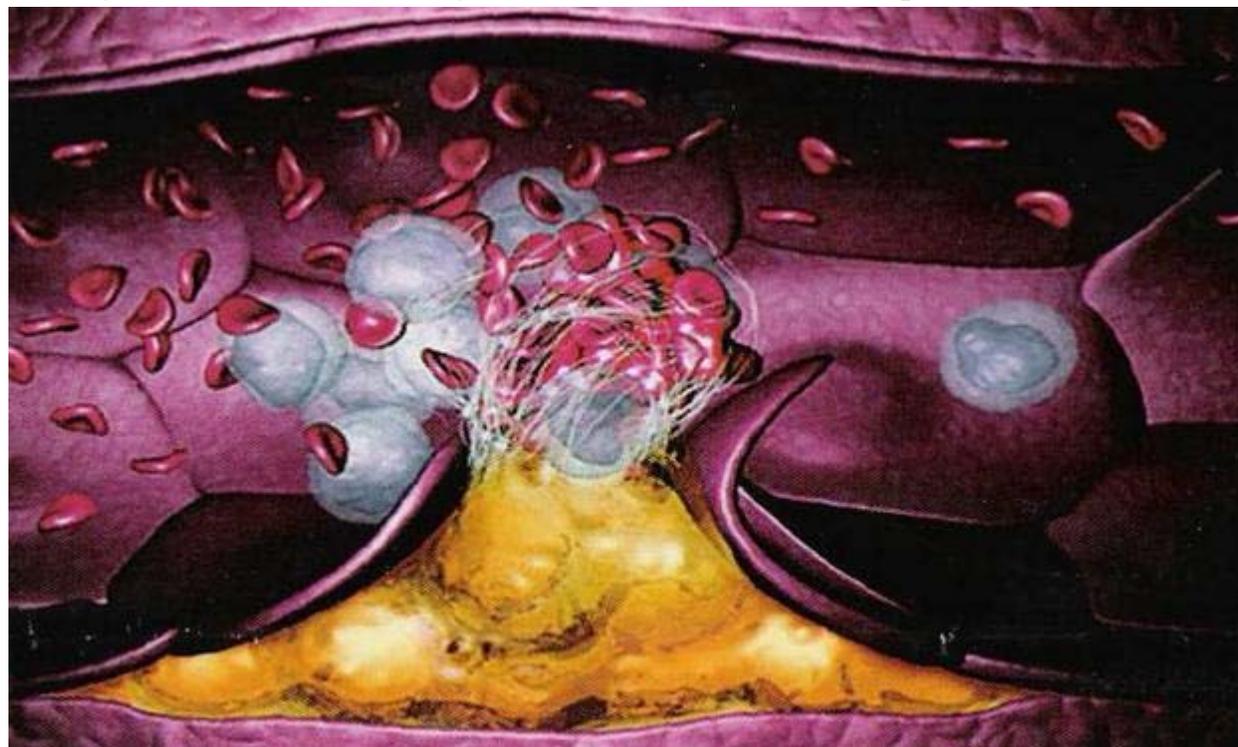


Fig. Pathogenesis of MI

MI may lead to impairment of systolic function or diastolic function and to increased predisposition to arrhythmias and other long-term complications.

Pathophysiology: Atherosclerosis is the disease primarily responsible for most ACS cases. Approximately 90% of MIs result from an acute thrombus that obstructs an atherosclerotic coronary artery. Plaque rupture is considered to be the major trigger of coronary thrombosis. Following plaque rupture, platelet activation and aggregation, coagulation pathway activation, and endothelial vasoconstriction occur and lead to coronary thrombosis and occlusion.

Atherogenesis

Consider nonatherosclerotic causes of acute MIs in younger patients or if no evidence of atherosclerosis is noted. Such causes include coronary emboli from sources such as an infected cardiac valve, coronary occlusion secondary to vasculitis, primary coronary vasospasm (variant angina), cocaine use, or other factors leading to mismatch of oxygen supply and demand, as may occur with a significant GI bleed.

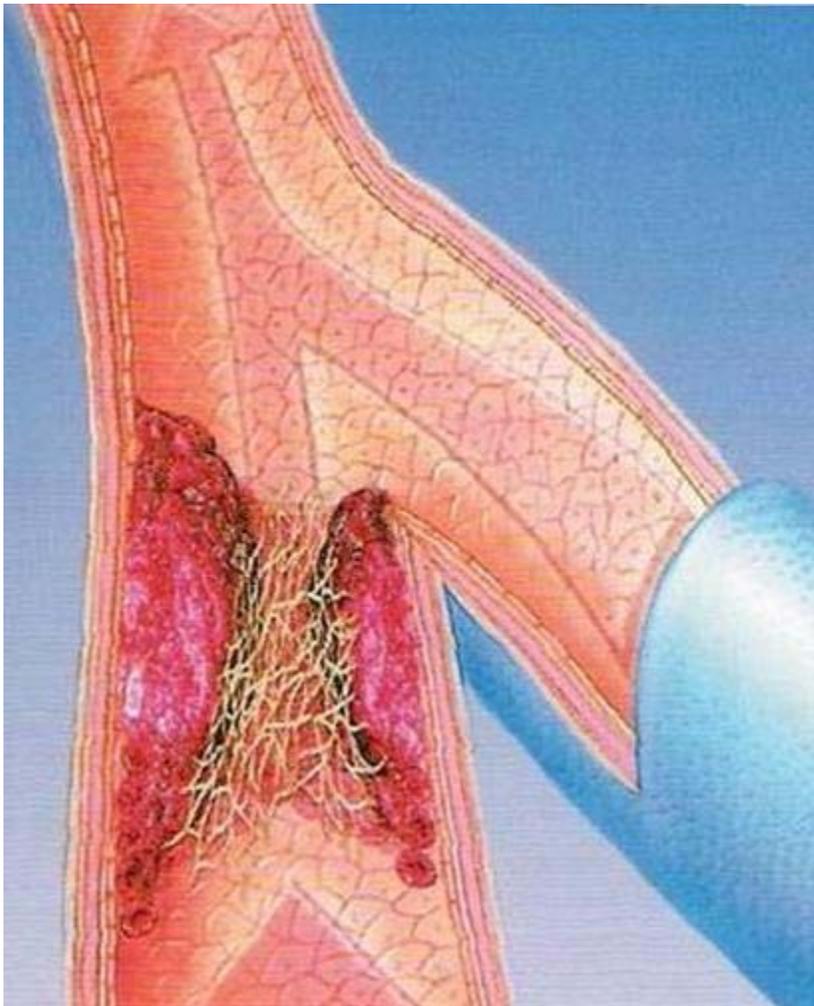


Fig. Atherogenesis

Frequency: In the US: Approximately 1.5 million cases of MI occur each year.
Internationally: Cardiovascular diseases cause 12 million deaths throughout the world each

year, according to the third monitoring report of the World Health Organization, 1991-93. They cause half of all deaths in several developed countries and are one of the main causes of death in many developing countries; they are the major cause of death in adults everywhere.

Mortality/Morbidity: Cardiovascular disease is the leading cause of death in the United States; approximately 500,000-700,000 deaths related to the coronary artery occur each year. Ischemic heart disease is the leading cause of death worldwide. Approximately 6.3 million deaths due to heart disease occurred in 1990 worldwide, which represents 29% of all deaths. The prevalence of coronary artery disease (CAD) is increasing rapidly in nonindustrialized countries.

Race: Cardiovascular disease is the leading cause of morbidity and mortality among African American, Hispanic, and white populations in the United States.

Sex: A male predominance in incidence exists up to approximately age 70 years, when the sexes converge to equal incidence.

Premenopausal women appear to be somewhat protected from atherosclerosis, possibly owing to the effects of estrogen.

Age: Incidence increases with age. Most patients who develop an acute MI are older than 60 years. Elderly people also tend to have higher rates of morbidity and mortality from their infarcts.

CLINICAL

History:

Chest pain

This is usually described as a substernal pressure sensation that also may be described as squeezing, aching, burning, or even sharp pain.

Prolonged chest discomfort lasting longer than 30 minutes is most compatible with infarction.

Radiation to the left arm or neck is common.

The sensation is precipitated by exertion and relieved by rest and nitroglycerin.

Chest pain may be associated with nausea, vomiting, diaphoresis, dyspnea, fatigue, or palpitations.

Atypical chest pain is common, especially in patients with diabetes and in elderly patients.

However, any patient may present with atypical symptoms. These symptoms are considered the anginal equivalent for that patient.

Shortness of breath

Shortness of breath may be the patient's anginal equivalent or a symptom of heart failure.

It is due to elevated end-diastolic pressures secondary to ischemia, which may then lead to

elevated pulmonary pressures.

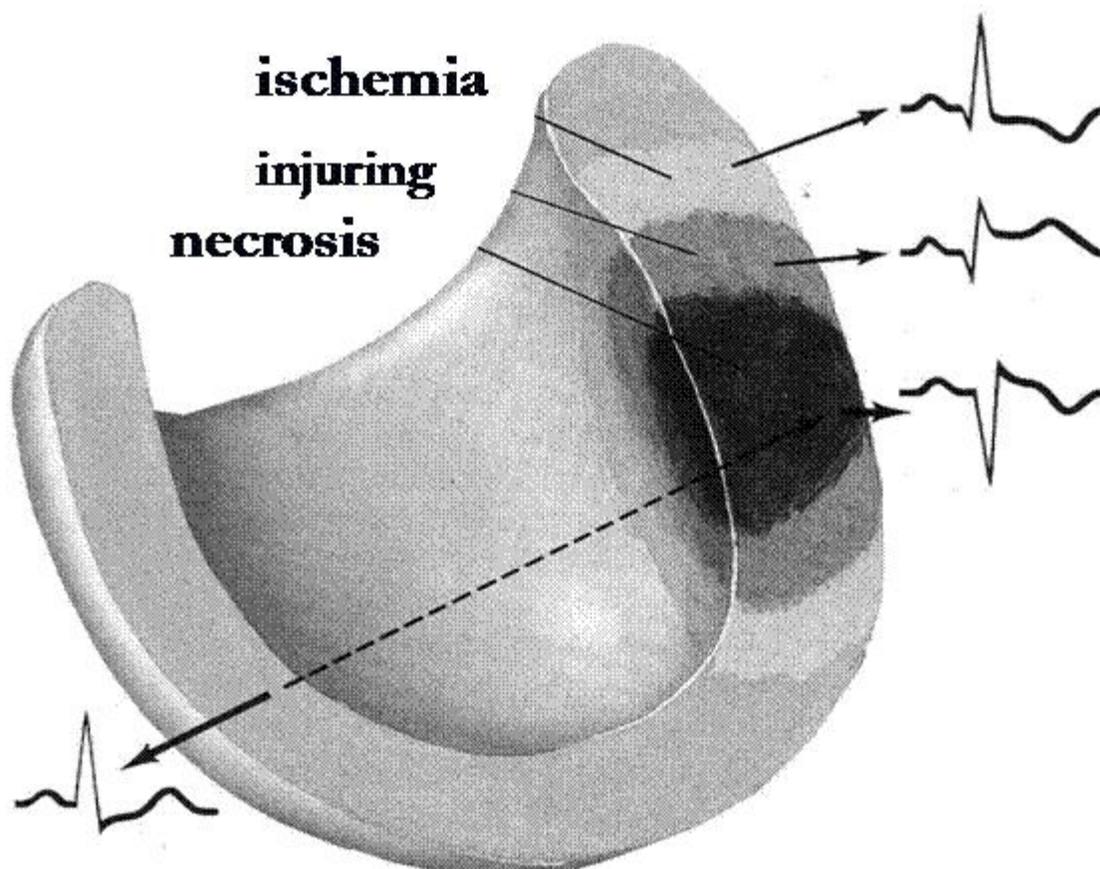


Fig. ECG changes in MI

Atypical presentations

Atypical presentations are common and frequently lead to misdiagnoses.

A patient may, for example, present with abdominal discomfort or jaw pain as his or her anginal equivalent.

An elderly patient may present with altered mental status.

Low threshold should be maintained when evaluating high- and moderate-risk patients, as their anginal equivalents may mimic other presentations.

Women tend to present more commonly with atypical symptoms such as sharp pain, fatigue, weakness, and other nonspecific complaints.

Physical: Physical examination findings can vary; one patient may be comfortable in bed, with normal examination results, while another may be in severe pain with significant respiratory distress requiring ventilatory support.

Low-grade fever may be present.

Hypotension or hypertension can be observed depending on the extent of the MI.

Fourth heart sound (S₄) may be heard in patients with ischemia. With ischemia, diastolic

dysfunction is the first physiologically measurable effect and this can then cause a stiff ventricle and an audible S4.

Dyskinetic cardiac bulge (in anterior wall MI) can occasionally be palpated.

Systolic murmur can be heard if mitral regurgitation (MR) or ventricular septal defect (VSD) develops.

Other findings include cool, clammy skin and diaphoresis.

Signs of congestive heart failure (CHF) may be found, including the following:

Third heart sound (S3) gallop

Pulmonary rales

Lower extremity edema

Elevated jugular venous pressure

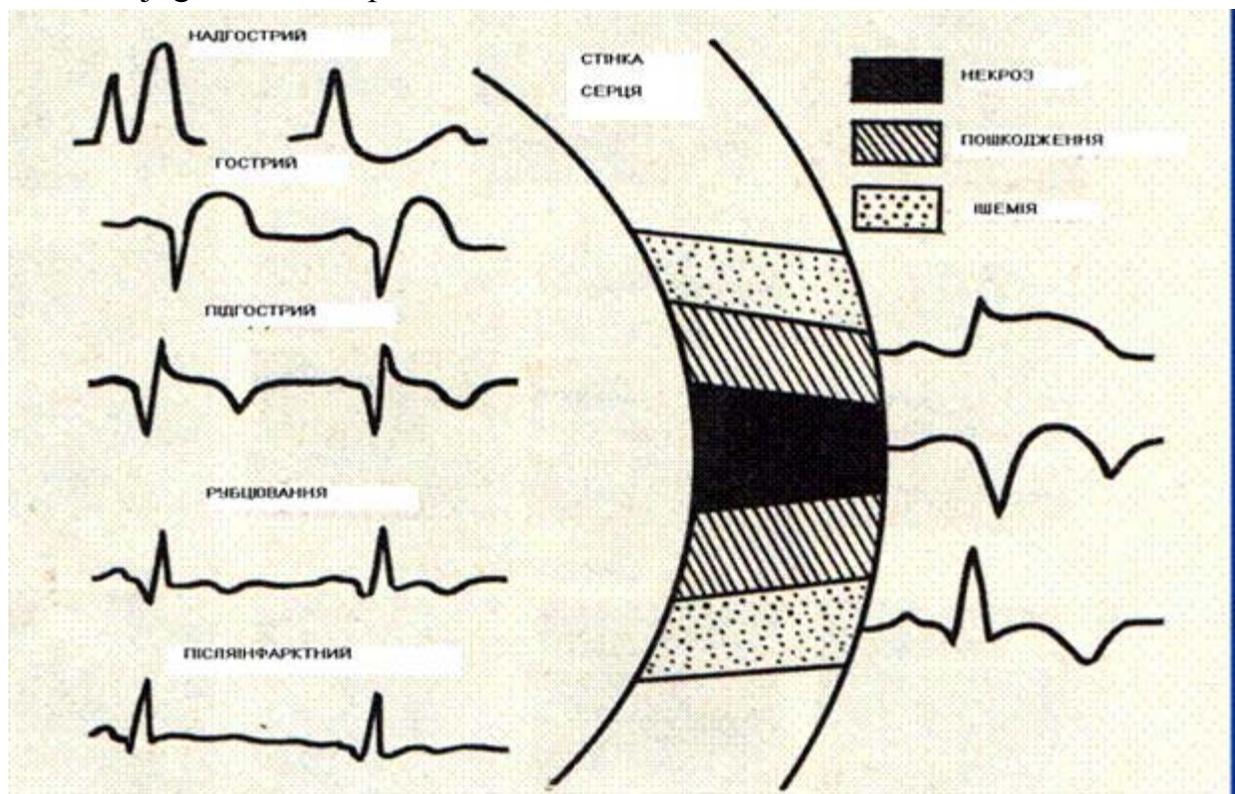


Fig Stages of MI

Causes: Atherosclerosis with occlusive or partially occlusive thrombus formation, Nonmodifiable risk factors for atherosclerosis, Age, Sex, Family history of premature coronary heart disease, Modifiable risk factors for atherosclerosis, Smoking or other tobacco use, Diabetes mellitus, Hypertension, Dyslipidemia, Obesity, New and other risk factors for atherosclerosis, Elevated homocysteine levels, Male pattern baldness. Sedentary lifestyle and/or lack of exercise, Psychosocial stress, Presence of peripheral vascular disease, Poor oral hygiene, Nonatherosclerotic causes, Vasculitis, Coronary emboli, Congenital coronary anomalies, Coronary trauma, Coronary spasm, Drug use (cocaine), Factors that increase oxygen

requirement, such as heavy exertion, fever, or hyperthyroidism , Factors that decrease oxygen delivery, such as hypoxemia of severe anemia

Other Problems to be Considered:

Pneumonia

Pancreatitis

Lab Studies:

Cardiac enzymes: In patients with suspected MI, obtain cardiac enzymes at regular intervals, starting upon admission and serially for as long as 24 hours.

Creatine kinase level

Creatine kinase comprises 3 isoenzymes, including creatine kinase with muscle subunits (CK-MM), which is found mainly in skeletal muscle; creatine kinase with brain subunits (CK-BB), predominantly found in the brain; and myocardial muscle creatine kinase (CK-MB), which is found mainly in the heart.

Serial measurements of CK-MB isoenzyme levels were previously the standard criterion for diagnosis of MI. CK-MB levels increase within 3-12 hours of onset of chest pain, reach peak values within 24 hours, and return to baseline after 48-72 hours. Levels peak earlier (wash out) if reperfusion occurs. Sensitivity is approximately 95%, with high specificity. However, sensitivity and specificity are not as high as for troponin levels, and the trend has favored using troponins for the diagnosis of MI.

Troponin levels

Troponin levels are now considered the criterion standard in defining and diagnosing MI, according to the American College of Cardiology (ACC)/American Heart Association (AHA) consensus statement on MI (Braunwald, 2000).

Cardiac troponin levels (troponin-T and troponin-I) have a greater sensitivity and specificity than CK-MB levels in detecting MI. They have important diagnostic and prognostic roles.

Positive troponin levels are considered virtually diagnostic of MI in the most recent ACC/AHA revisions, as they are without equal in combined specificity and sensitivity in this diagnosis.

Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days.

Myoglobin levels

Urine myoglobin levels rise within 1-4 hours from the onset of chest pain.

Myoglobin levels are highly sensitive but not specific, and they may be useful within the context of other studies and in early detection of MI in the ED.

Complete blood cell count

Obtain a CBC count if MI is suspected to rule out anemia as a cause of decreased oxygen supply and prior to giving thrombolytics.

Leukocytosis is also common, but not universal, in the setting of acute MI.

A platelet count is necessary if a IIb/IIIa agent is considered; furthermore, the patient's WBC count may be elevated modestly in the setting of MI, signifying an acute inflammatory state.

Chemistry profile

In the setting of MI, closely monitor potassium and magnesium levels.

Creatinine level is also needed prior to initiating treatment with an angiotensin-converting enzyme (ACE) inhibitor.

Lipid level profile: This may be helpful if obtained upon presentation because levels can change after 12-24 hours of an acute illness.

C-reactive protein (CRP) levels: Consider measuring CRP levels and other markers of inflammation upon presentation if an ACS is suspected.

Imaging Studies:

Chest radiography

Upon presentation, obtain a chest radiograph to assess the patient's heart size and the presence or absence of decompensated CHF with or without pulmonary edema.

A chest radiograph may also assist in diagnosing concomitant disease, such as pneumonia in an elderly patient, as a precipitating cause for MI.

Echocardiography

An echocardiogram may play an important role in the setting of MI.

Regional wall motion abnormalities can be identified, which are especially helpful if the diagnosis is questionable.

An echocardiogram can also define the extent of the infarction and assess overall left ventricle (LV) and right ventricle (RV) function. In addition, an echocardiogram can identify complications, such as acute MR, LV rupture, or pericardial effusion.

Myocardial perfusion imaging

Prior to discharge, obtain myocardial perfusion imaging to assess the extent of residual ischemia if the patient has not undergone cardiac catheterization. The extent of ischemia can guide further therapy as to whether to proceed with catheterization or to continue conservative therapy.

Myocardial perfusion has been shown to be a valuable method for triage of patients with chest pain in the ED. Significant variability exists among centers, and the results of the trials can be applied only to those centers with proven reliability and experience.

Cardiac angiography

Cardiac catheterization defines the patient's coronary anatomy and the extent of the disease. Most investigators recommend that all patients with MI should undergo cardiac catheterization, if it is available.

Patients with cardiogenic shock, intractable angina despite medications, or severe pulmonary congestion should undergo cardiac catheterization immediately.

Other Tests:

The electrocardiogram (ECG) is the most important tool in the initial evaluation and triage of patients in whom an ACS is suspected (see Images 1-3). It is confirmatory of the diagnosis in approximately 80% of cases.

Obtain an ECG immediately if MI is considered or suspected.

Video Localization of MI

In patients with inferior MI, record a right-sided ECG to rule out RV infarct.

Qualified personnel should review the ECG as soon as possible.

Perform ECGs serially upon presentation to evaluate progression and assess changes with and without pain.

Obtain daily serial ECGs for the first 2-3 days and additionally as needed.

Convex ST-segment elevation with upright or inverted T waves is generally indicative of MI in the appropriate clinical setting.

ST depression and T-wave changes may also indicate evolution of NSTEMI.

TREATMENT

Medical Care: Initial therapy for acute MI is directed toward restoration of perfusion in order to salvage as much of the jeopardized myocardium as possible. This may be accomplished

through medical or mechanical means, such as angioplasty or coronary artery bypass grafting. Further treatment is based on (1) restoration of the balance between the oxygen supply and demand to prevent further ischemia, (2) pain relief, and (3) prevention and treatment of any complications that may arise.

Thrombolytic therapy has been shown to improve survival rates in patients with acute MI if administered in a timely fashion in the appropriate group of patients. If PCI capability is not available or will cause a delay greater than 90 minutes, then the optimal approach is to administer thrombolytics within 12 hours of onset of symptoms in patients with ST-segment elevation greater than 0.1 mV in 2 or more contiguous ECG leads, new left bundle-branch block (LBBB), or anterior ST depression consistent with posterior infarction. Tissue plasminogen activator (t-PA) is superior to streptokinase in achieving a higher rate of coronary artery patency; however, the key to efficacy lies in the speed of the delivery of therapy. Recent trials show a high patency rate if a IIb/IIIa receptor antagonist is combined with a half dose of a thrombolytic agent as the initial reperfusion strategy. The reduced dose of a thrombolytic agent combined with a potent platelet inhibitor may prove to be the preferred method for medical reperfusion. Larger clinical trials are pending.

Aspirin and/or antiplatelet therapy Aspirin has been shown to decrease mortality and re-infarction rates after MI. Administer aspirin immediately, which the patient should chew if possible upon presentation. Continue aspirin indefinitely unless an obvious contraindication, such as a bleeding tendency or an allergy, is present. Clopidogrel may be used as an alternative in cases of a resistance or allergy to aspirin. Recent data from the CLARITY trial (CLOpidogrel as Adjunctive Reperfusion Therapy Thrombolysis in Myocardial Infarction [TIMI] 28) suggest that adding clopidogrel to this regimen is safe and effective. The clopidogrel dose used was 300 mg.

Administer a platelet glycoprotein (GP) IIb/IIIa-receptor antagonist, in addition to acetylsalicylic acid and unfractionated heparin (UFH), to patients with continuing ischemia or with other high-risk features and to patients in whom a percutaneous coronary intervention (PCI) is planned. Eptifibatid and tirofiban are approved for this use. Abciximab also can be used for 12-24 hours in patients with unstable angina or NSTEMI in whom a PCI is planned within the next 24 hours.

Beta-blockers reduce the rates of reinfarction and recurrent ischemia and possibly reduce the mortality rate if administered within 12 hours after MI. Administer routinely to all patients with MI unless a contraindication is present.

Heparin (and other anticoagulant agents) has an established role as an adjunctive agent in

patients receiving t-PA but not with streptokinase. Heparin is also indicated in patients undergoing primary angioplasty. Little data exist with regard to efficacy in patients not receiving thrombolytic therapy in the setting of acute MI. Low-molecular-weight heparins (LMWHs) have been shown to be superior to UFHs in patients with unstable angina or NSTEMI.

Nitrates have no apparent impact on mortality rate in patients with ischemic syndromes. Their utility is in symptomatic relief and preload reduction. Administer to all patients with acute MI within the first 48 hours of presentation, unless contraindicated (ie, in RV infarction).

ACE inhibitors reduce mortality rates after MI. Administer ACE inhibitors as soon as possible as long as the patient has no contraindications and remains in stable condition. ACE inhibitors have the greatest benefit in patients with ventricular dysfunction. Continue ACE inhibitors indefinitely after MI. Angiotensin-receptor blockers may be used as an alternative in patients who develop adverse effects, such as a persistent cough, although initial trials need to be confirmed.

Surgical Care:

Percutaneous coronary intervention

PCI is the treatment of choice in most patients with STEMI, assuming a door to needle time of less than 90 minutes. PCI provides greater coronary patency (>96% thrombolysis in myocardial infarction [TIMI] 3 flow), lower risk of bleeding, and instant knowledge about the extent of the underlying disease. Studies have shown that primary PCI has a mortality benefit over thrombolytic therapy.

The choice of primary PCI should be individualized to each institution and to the patient's presentation and timing.

The widespread use of stenting and adjunctive IIb/IIIa therapy are improving the results of primary PCI. A recently published trial showed that, in patients with acute MI, coronary stenting and abciximab lead to a greater degree of myocardial salvage and a better clinical outcome than fibrinolysis with thrombolytic therapy. Improvement of long- and short-term outcomes, however, depends highly on the speed with which reperfusion is achieved.

Primary PCI is also the treatment of choice in patients with cardiogenic shock, patients in whom thrombolysis failed, and those with high risk of bleeding or contraindications to thrombolytic therapy.

Only an experienced operator should perform primary PTCA, and PTCA should be performed only where the appropriate facilities are available. Operators should have at least 75 cases per year, while the center should perform at least 200 cases per year as per the recommendations of

the ACC.

Emergent or urgent coronary artery graft bypass surgery is indicated in patients in whom angioplasty fails and in patients who develop mechanical complications such as a VSD, LV, or papillary muscle rupture.

Consultations:

ED personnel should initiate evaluation and treatment, including administering a thrombolytic agent.

Obtain cardiology consultation immediately if primary PCI is considered. Otherwise, cardiology consultation may be obtained as needed and upon admission. Consultation may be obtained sooner if the patient presents with significant heart failure, mechanical complications, arrhythmias, or other complicating factors.

Diet: Initially, keep the patient on nothing by mouth (NPO) until his or her condition has been stabilized and treated. Following initial therapy and admission, a dietitian should instruct the patient regarding appropriate diet, as recommended by the AHA.

A low-salt, low-fat, and low-cholesterol diet is generally recommended.

Activity: Confine patients to bed rest to minimize oxygen consumption until reperfusion and initial therapy are complete. This usually lasts about 24-48 hours; after that, the patient's activity may be accelerated slowly as tolerated and as the clinical situation allows.

Initiate cardiac rehabilitation prior to discharge.

MEDICATION

The goals of pharmacotherapy are to reduce morbidity and to prevent complications.

Drug Category: *Salicylates* -- The antiplatelet effects of these agents may improve mortality rate.

Drug Name	Aspirin (Anacin, Ascriptin, Bayer Aspirin) -- Early administration of aspirin in patients with acute MI has been shown to reduce cardiac mortality rate by 23% in first mo.
Adult Dose	160-325 mg PO or chewed
Pediatric Dose	10-15 mg/kg/dose PO q4-6h
Contraindications	Documented hypersensitivity, liver damage; hypoprothrombinemia; vitamin K deficiency; bleeding disorders; asthma Because of association with Reye

	syndrome, do not administer to children (<16 y) who have flu
Interactions	Antacids and urinary alkalinizers can decrease pharmacologic effects; corticosteroids decrease serum levels by increasing salicylate clearance; other anticoagulants can have an additive hypoprothrombinemic effect and may increase bleeding time; may antagonize uricosuric effects of probenecid and increase free phenytoin and valproic acid levels, increasing their toxicity; in doses >2 g/d, may alter pancreatic beta-cell function and potentiate glucose-lowering effect of sulfonylurea drugs
Pregnancy	D - Unsafe in pregnancy
Precautions	Caution in patients with chronic renal insufficiency—may cause transient decrease in renal function and aggravate chronic kidney diseases; patients with severe anemia, history of blood coagulation defects, or taking anticoagulants should avoid

Drug Category: *Vasodilators* -- These agents relieve chest discomfort by improving myocardial oxygen supply, which in turn dilates epicardial and collateral vessels, improving blood supply to the ischemic myocardium.

Drug Name	Nitroglycerin (Nitro-Bid) -- Causes relaxation of vascular smooth muscle via stimulation of intracellular cyclic guanosine monophosphate production, causing decrease in BP.
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Adult Dose	400 mcg SL or spray q5min, repeat up to 3 times; if symptoms persist, 5-10 mcg/min IV infusion; titrate to 10% reduction in MAP or symptom relief, limiting adverse effects of hypotension
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe anemia; shock; postural hypotension; head trauma; closed-angle glaucoma; cerebral hemorrhage; known history of RV MI
Interactions	Aspirin may increase serum concentrations; calcium channel blockers may cause marked symptomatic orthostatic hypotension (dose adjustment of either agent may be necessary)
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Exercise caution in patients with CAD or low systolic BP

Drug Category: *Analgesics* -- Pain control is essential to quality patient care. Analgesics ensure patient comfort, promote pulmonary toilet, and have sedating properties, which are beneficial for patients who experience pain.

Drug Name	Morphine sulfate (Duramorph, Astramorph, MS Contin) -- DOC for narcotic analgesia due to its reliable and predictable effects, safety profile, and ease of reversibility with naloxone. Administered IV, may be dosed in a number of ways and commonly is titrated until desired effect.
Adult Dose	2 mg IV q5-15min, titrate to symptomatic relief or adverse effects (eg, lethargy, hypotension, respiratory depression)

Pediatric Dose	0.1-0.2 mg/kg IV q2-4h prn
Contraindications	Documented hypersensitivity; hypotension; potentially compromised airway in which establishing rapid airway control would be difficult
Interactions	Phenothiazines may antagonize analgesic effects; tricyclic antidepressants, MAOIs, and other CNS depressants may potentiate adverse effects
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Avoid using this drug in patients with hypotension, emesis, constipation, respiratory depression, nausea, or urinary retention; exercise caution in patients with atrial flutter and other supraventricular tachycardias; has vagolytic action and may increase ventricular response rate; mental status changes, respiratory depression, and shock are possible adverse effects

Drug Category: *Anticoagulants* -- Unfractionated intravenous heparin and fractionated low molecular weight subcutaneous heparins are the two choices for initial anticoagulation therapy.

Drug Name	Heparin -- Augments activity of antithrombin III and prevents conversion of fibrinogen to fibrin. Does not actively lyse but is able to inhibit further thrombus formation. Prevents reaccumulation of clot after spontaneous fibrinolysis.
Adult Dose	70 IU/kg IV bolus, followed by 15 mcg/kg/h infusion, adjust to maintain aPTT 1.5-2 times control

Pediatric Dose	<p>Loading dose: 50 IU/kg/h IV</p> <p>Maintenance infusion: 15-25 mcg/kg/h IV; increase dose by 2-4 IU/kg/h q6-8h prn using aPTT results</p>
Contraindications	<p>Documented hypersensitivity, subacute bacterial endocarditis; active bleeding; history of heparin-induced thrombocytopenia</p>
Interactions	<p>Digoxin, nicotine, tetracycline, and antihistamines may decrease effects; NSAIDs, aspirin, dextran, dipyridamole, and hydroxychloroquine may increase toxicity</p>
Pregnancy	<p>C - Safety for use during pregnancy has not been established.</p>
Precautions	<p>Observe for prolonged or excessive bleeding at venipuncture sites; some preparations contain benzyl alcohol as preservative, and benzyl alcohol used in large amounts has been associated with fetal toxicity (gasping syndrome); use of preservative-free heparin recommended in neonates; use with caution in patients with shock or severe hypotension</p>
Drug Name	<p>Enoxaparin (Lovenox) -- Enhances inhibition of factor Xa and thrombin by increasing antithrombin III activity. In addition, preferentially increases inhibition of factor Xa.</p>
Adult Dose	<p>1 mg/kg SC bid</p>
Pediatric Dose	<p>Not established</p>

Contraindications	Documented hypersensitivity; major bleeding; history of heparin-induced thrombocytopenia
Interactions	Platelet inhibitors or oral anticoagulants, such as aspirin, NSAIDs, dipyridamole, salicylates, sulfapyrazone, and ticlopidine, increase risk of bleeding and should be used with care in patients taking enoxaparin
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Caution in recent surgery, GI lesions that may be prone to bleeding, hematologic conditions, uncontrolled hypertension, diabetic retinopathy, or vitreous hemorrhage; caution in patients with renal insufficiency because elimination delayed, increasing anticoagulant effect; if thromboembolic event occurs despite LMWH prophylaxis, discontinue drug and initiate appropriate therapy; reversible elevation of hepatic transaminases seen occasionally; heparin-induced thrombocytopenia has been seen with LMWH; for significant bleeding complications, 1 mg of protamine sulfate reverses effect of approximately 1 mg of enoxaparin

Drug Category: *Thrombolytics* -- The main objective is to restore circulation through a previously occluded vessel by the rapid and complete removal of a pathologic intraluminal thrombus or embolus that has not been dissolved by the endogenous fibrinolytic system.

Drug Name	Alteplase, t-PA (Activase) -- Fibrin-specific
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	<p>agent with brief half-life of 5 min.</p> <p>Adjunctive therapy with IV heparin necessary to maintain patency of arteries recanalized by t-PA, especially during first 24-48 h.</p>
Adult Dose	<p>15 mg IV initial bolus, followed by 50 mg IV over next 30 min, and then 35 mg IV over next h; total dose not to exceed 100 mg</p>
Pediatric Dose	<p>Not established</p>
Contraindications	<p>Documented hypersensitivity; active internal bleeding; recent intracranial or intraspinal surgery or trauma; intracranial neoplasm; AV malformation or aneurysm; history of cerebrovascular accident within last 2 mo; seizure at onset of stroke; suspicion of subarachnoid hemorrhage; bleeding diathesis; serious head trauma; severe uncontrolled hypertension; do not administer to patients with history of intracranial hemorrhage</p>
Interactions	<p>Heparin, which has been administered with and after alteplase infusions to reduce risk of reocclusion, may cause bleeding complications, monitor closely for bleeding, especially at arterial puncture sites; drugs that alter platelet function (eg, aspirin, abciximab, dipyridamole) may increase risk of bleeding if administered prior to, during, or after</p>
Pregnancy	<p>C - Safety for use during pregnancy has not been established.</p>

Precautions	Adverse effects include hemorrhage, particularly intracranial; elderly patients are at greatest risk
Drug Name	Streptokinase (Kabikinase, Streptase) -- Acts with plasminogen to convert plasminogen to plasmin. Plasmin degrades fibrin clots, as well as fibrinogen and other plasma proteins. Increase in fibrinolytic activity that degrades fibrinogen levels for 24-36 h occurs with IV infusion of streptokinase. Adjunctive therapy with heparin not needed.
Adult Dose	1.5 million IU in 50 cc D5W IV over 60 min
Pediatric Dose	Administer as in adults
Contraindications	Documented hypersensitivity; active internal bleeding; intracranial neoplasm; aneurysm; bleeding diathesis; severe uncontrolled arterial hypertension
Interactions	Antifibrinolytic agents may decrease effects; antiplatelet agents and anticoagulants may increase risk of bleeding
Pregnancy	D - Unsafe in pregnancy
Precautions	Use with caution in patients with severe hypertension, those receiving medication via IM administration, and those who had trauma or surgery in previous 10 d; measure hematocrit, platelet count, aPTT, TT, PT, or fibrinogen levels before therapy initiated; either TT or aPTT should be less than twice normal control value following infusion and before instituting heparin; do

	not take BP in lower extremities, it may dislodge possible deep vein thrombus; monitor PT, aPTT, TT, or fibrinogen 4 h after initiation of therapy
Drug Name	Reteplase (Retavase) -- Recombinant plasminogen activator that forms plasmin after facilitating cleavage of endogenous plasminogen. In clinical trials, has been comparable to alteplase in achieving TIMI 2 or 3 patency at 90 min. Heparin and aspirin usually administered concomitantly and after reteplase.
Adult Dose	10 IU IV over 2 min, followed by second 10-IU IV dose after 30 min
Pediatric Dose	Not recommended
Contraindications	Documented hypersensitivity; uncontrolled hypertension; recent intracranial surgery; malformation of aneurysm; bleeding diathesis
Interactions	May increase effects of warfarin, heparin, and aspirin
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Caution in cardiovascular arrhythmias, hypotension, and perfusion arrhythmias
Drug Name	Anistreplase (Eminase) -- Non-fibrin-specific agent that activates conversion of plasminogen to plasmin and has half-life of 90 min. However, does not have any benefit over streptokinase, although has higher rate of allergic and bleeding complications. Easier to administer than

	t-PA, has lower cost (\$1500), and does not require heparinization.
Adult Dose	30 IU over 2-5 min
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; recent intracranial surgery; uncontrolled hypertension; vascular malformation; aneurysm
Interactions	Increases effects of anticoagulants and aspirin
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in cardiovascular arrhythmias, hypotension, and perfusion arrhythmias

Drug Category: *Beta-adrenergic blockers* -- This category of drugs has the potential to suppress ventricular ectopy due to ischemia or excess catecholamines. In the setting of myocardial ischemia, beta-blockers have antiarrhythmic properties and reduce myocardial oxygen demand secondary to elevations in heart rate and inotropy.

Drug Name	Metoprolol (Lopressor) -- Selective beta1-adrenergic receptor blocker that decreases automaticity and contractions. Goals of treatment are reduction in heart rate to 60-80 bpm. During IV administration, carefully monitor BP, heart rate, and ECG.
Adult Dose	5 mg IV slow infusion q5min; not to exceed 15 mg or desired heart rate 25 mg PO bid usual initial dose, up to 100 mg bid; titrate to desired effect
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; decompensated CHF; bradycardia;

	bronchial asthma; cardiogenic shock; AV conduction abnormalities
Interactions	Aluminum salts, barbiturates, NSAIDs, penicillins, calcium salts, cholestyramine, and rifampin may decrease bioavailability and plasma levels, possibly resulting in decreased pharmacologic effect; astemizole (recalled from US market), calcium channel blockers, quinidine, flecainide, and contraceptives may increase cardiotoxicity; digoxin, flecainide, acetaminophen, clonidine, epinephrine, nifedipine, prazosin, haloperidol, phenothiazines, and catecholamine-depleting agents may increase toxicity
Pregnancy	B - Usually safe but benefits must outweigh the risks.
Precautions	Do not use in cocaine-related ischemia; beta-adrenergic blockade may reduce signs and symptoms of acute hypoglycemia and may decrease clinical signs of hyperthyroidism; abrupt withdrawal may exacerbate symptoms of hyperthyroidism, including thyroid storm, monitor patient closely and withdraw drug slowly; during IV administration, carefully monitor BP, heart rate, and ECG
Drug Name	Esmolol (Brevibloc) -- Useful drug for patients at risk of experiencing complications from beta-blockers, particularly reactive airway disease, mild-to-moderate LV dysfunction, and peripheral vascular disease. Its short half-life of 8 min

	allows for titration to desired effect with ability to stop quickly if necessary.
Adult Dose	0.1 mg/kg/min IV starting maintenance dose, titrate in increments of 0.05 mg/kg/min q10-15min to total dose of 0.2 mg/kg/min
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; decompensated CHF; bradycardia; cardiogenic shock; AV conduction abnormalities
Interactions	Aluminum salts, barbiturates, NSAIDs, penicillins, calcium salts, cholestyramine, and rifampin may decrease bioavailability and plasma levels, possibly resulting in decreased pharmacologic effect; astemizole (recalled from US market), calcium channel blockers, quinidine, flecainide, and contraceptives may increase cardiotoxicity; digoxin, flecainide, acetaminophen, clonidine, epinephrine, nifedipine, prazosin, haloperidol, phenothiazines, and catecholamine-depleting agents may increase toxicity
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Do not use in cocaine-related ischemia; beta-adrenergic blockade may decrease signs and symptoms of acute hypoglycemia and clinical signs of hyperthyroidism; abrupt withdrawal may exacerbate symptoms of hyperthyroidism,

	including thyroid storm, withdraw drug slowly and monitor patient closely
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Drug Category: *Angiotensin-converting enzyme (ACE) inhibitors* -- These agents may prevent conversion of angiotensin I to angiotensin II, a potent vasoconstrictor, resulting in lower aldosterone secretion.

Drug Name	Captopril (Capoten) -- Has short half-life, which makes it important drug for initiation of ACE inhibitor therapy. Can be started at low dose and titrated upward as needed and as patient tolerates.
Adult Dose	6.25 mg PO tid initially; may titrate to total 450 mg/d
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity
Interactions	Patients receiving diuretic therapy, other vasodilator agents, agents causing renin release, agents increasing potassium, or agents affecting sympathetic activity should be monitored carefully
Pregnancy	D - Unsafe in pregnancy
Precautions	Administer with caution in patients with renal insufficiency and those with borderline low BP; may worsen renal function, especially in patients with bilateral renal artery stenosis; administer cautiously in patients with aortic stenosis because afterload reduction may worsen coronary perfusion

Drug Category: *Platelet aggregation inhibitors* -- These agents prevent acute cardiac ischemic complications in unstable angina unresponsive to conventional therapy.

Drug Name	Abciximab (ReoPro) -- Chimeric human-murine monoclonal antibody. Binds to
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	receptor with high affinity and reduces platelet aggregation by 80%. Inhibition of platelet aggregation persists for as long as 48 h after infusion stopped.
Adult Dose	0.25 mcg/kg bolus IV, followed by 0.125 mcg/kg/min infusion for 12 h
Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; bleeding diathesis; thrombocytopenia (<100,000 platelets/mL); recent trauma; intracranial tumor; severe uncontrolled hypertension; history of vasculitis; cerebrovascular accident within 2 y
Interactions	Anticoagulants, antiplatelets, and thrombolytics may increase toxicity
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Bleeding complications are rare and usually related to use of standard-dose heparin instead of weight-based dosing; severe thrombocytopenia has been associated with abciximab within first 24 h of use
Drug Name	Tirofiban (Aggrastat) -- Nonpeptide antagonist of glycoprotein IIb/IIIa receptor. Reversible antagonist of fibrinogen binding. When administered IV, >90% of platelet aggregation inhibited.
Adult Dose	0.4 mcg/kg/min IV for 30 min, then continue at 0.1 mcg/kg/min; administer half dose in patients with severe renal insufficiency (CrCl <30 mL/min)

Pediatric Dose	Not established
Contraindications	Documented hypersensitivity; severe hypertension (SBP >200 mm Hg); active internal bleeding; history of intracranial hemorrhage; intracranial neoplasm; AV malformation or aneurysm; acute pericarditis; bleeding diathesis; trauma or stroke within previous 30 d; platelet count <100,000/mL; history of thrombocytopenia following prior exposure to this product; serum creatinine >2 mg/dL (for 180-mcg/kg bolus and 2-mcg/kg/min infusion) or >4 mg/dL (for 135-mcg/kg bolus and 0.5-mcg/kg/min infusion)
Interactions	Heparin and aspirin increase risk of bleeding compared with heparin and aspirin alone; if using with other drugs that affect hemostasis (eg, warfarin), monitor patients closely
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Most common complications are bleeding events; caution in patients with platelet count <150,000/mL and in patients with hemorrhagic retinopathy; monitor platelet counts, serum creatinine, hemoglobin, hematocrit, and PT/aPTT before treatment, within 6 h after loading infusion, and at least daily thereafter (or more frequently if evidence of significant decline); because these agents inhibit platelet aggregation, exercise caution when using concurrently with drugs that affect hemostasis (eg,

	<p>thrombolytics, ticlopidine, NSAIDs, warfarin, dipyridamole, clopidogrel)</p> <p>Measure ACT and maintain aPTT at 50-70 s unless PCI necessary; maintain ACT at 250-300 s during PCI; if platelets decrease to <100,000/mL, perform additional platelet counts to exclude pseudothrombocytopenia; if thrombocytopenia confirmed, discontinue GP IIb/IIIa inhibitors and heparin and appropriately monitor and treat condition; to monitor UFH, monitor aPTT 6 h after start of heparin infusion and adjust to maintain aPTT at 1.5-2 times control</p>
Drug Name	Eptifibatide (Integrilin) -- Cyclic peptide that reversibly inhibits platelet aggregation by binding to IIb/IIIa receptor.
Adult Dose	<p>Unstable angina: 180 mcg/kg IV bolus, followed by 2 mcg/kg/min continuous infusion until discharge or surgery</p> <p>Patients undergoing PCI: 135 mcg/kg IV bolus before PCI, followed by 0.5 mcg/kg/min continuous infusion</p>
Pediatric Dose	Not established
Contraindications	<p>Documented hypersensitivity; severe hypertension (SBP >200 mm Hg); active internal bleeding; history of intracranial hemorrhage; intracranial neoplasm; AV malformation or aneurysm; acute pericarditis; bleeding diathesis; trauma or stroke within previous 30 d; platelet count <100,000/mL; history of thrombocytopenia following prior exposure to this product;</p>

	<p>serum creatinine >2 mg/dL (for 180-mcg/kg bolus and 2-mcg/kg/min infusion) or >4.0 mg/dL (for 135-mcg/kg bolus and 0.5-mcg/kg/min infusion)</p>
Interactions	<p>Heparin and aspirin increase risk of bleeding compared with heparin and aspirin alone; if using concurrently with other drugs that affect hemostasis (eg, warfarin), monitor patients closely</p>
Pregnancy	<p>C - Safety for use during pregnancy has not been established.</p>
Precautions	<p>Most common complications are bleeding events; exercise caution in patients with platelet count <150,000/mL and in patients with hemorrhagic retinopathy; because these agents inhibit platelet aggregation, exercise caution with concurrent use of drugs that affect hemostasis (eg, thrombolytic agents, ticlopidine, NSAIDs, warfarin, dipyridamole, clopidogrel)</p> <p>Measure ACT and maintain aPTT at 50-70 s unless PCI necessary; maintain ACT at 250-300 s during PCI; if platelets decrease to <100,000/mL, perform additional platelet counts to exclude pseudothrombocytopenia; if thrombocytopenia confirmed, discontinue GP IIb/IIIa inhibitors and heparin and appropriately monitor and treat condition; to monitor UFH, monitor aPTT 6 h after start of heparin infusion and adjust to maintain aPTT to 1.5-2 times control</p>

FOLLOW-UP

Further Inpatient Care: Admit patients with MI to a coronary care unit. Monitor patients carefully for arrhythmia, recurrent ischemia, and other possible complications. The patient may be transferred to a telemetry unit 24-48 hours after admission if no complications occur.

Hospitalize the patient for approximately 4-5 days after MI. Patients who undergo primary PCI or have an immediate cardiac catheterization may be discharged sooner if their hospital course is without incident.

Perform a coronary angiography on high-risk patients prior to discharge to evaluate their need for revascularization.

In the case of patients who have not had a cardiac catheterization and have no complications, perform a submaximal stress test prior to discharge to assess their subsequent risk.

Further Outpatient Care:

Arrange for follow-up within 2 weeks of discharge.

Arrange for cardiac rehabilitation.

In/Out Patient Meds:

The long-term use of aspirin in patients who have had an MI results in significant reduction in subsequent mortality rate.

Beta-blocker therapy has confirmed therapeutic benefit in survivors of acute MI. This therapy is most beneficial in patients with the highest risk.

ACE inhibitor use in patients with known CAD has been shown to reduce mortality rate.

Many trials have shown a clear benefit of lipid-lowering therapy in the secondary and primary prevention of CAD. The National Cholesterol Education Panel has set guidelines for target cholesterol levels. In general, patients who have experienced MI should achieve low-density lipoprotein (LDL) level less than 100 mg/dL, high-density lipoprotein (HDL) level greater than 40 mg/dL, and triglyceride level less than 200 mg/dL. High-risk patients should be treated to a target LDL level of less than 70 mg/dL.

Schwartz et al recently showed in the MIRACL trial that initiating atorvastatin during hospitalization for an ACS, irrespective of lipid levels, reduces the frequency of recurrent ischemic events. This treatment significantly reduced the frequency of the combined end point of death, recurrent death, MI, or worsening unstable angina requiring hospitalization.

Clopidogrel should be prescribed for a year following discharge if the patient has no contraindications and cost is not prohibitive. To reduce the risk of bleeding, the aspirin dose can be reduced to 81 mg.

Transfer: A patient in whom thrombolytic therapy fails should be transferred to a facility

where cardiac catheterization and angioplasty facilities are available.

Deterrence/Prevention:

Smoking cessation Cigarette smoking is a major risk factor for CAD. Risk of recurrent coronary events decreases 50% at 1 year after smoking cessation.

Provide all patients who smoke with guidance, education, and the support needed to avoid smoking.

Bupropion has been shown to increase the chances of patients' success in achieving smoking cessation.

Alcohol consumption

Mild alcohol consumption has been associated with a decreased risk of stroke and MI.

Cautiously consider recommending and discussing alcohol use on a case-by-case basis.

Antioxidant therapy, including vitamin E, has not shown clear benefit in the prevention of coronary events.

Do not use long-term anticoagulant (ie, warfarin) therapy routinely in post-MI patients but as an alternative in patients who cannot take antiplatelet agents. Patients with known LV thrombus, atrial fibrillation, or severe wall motion abnormalities have shown benefit from long-term anticoagulation, maintaining the international normalized ratio (INR) between 2 and 3.

Do not start post-MI patients on postmenopausal hormone therapy (HRT). Patients already taking HRT for more than 1 year may be continued on this therapy without increased risk.

COMPLICATIONS:

A number of arrhythmias occur after MI, ranging from benign to fatal. Arrhythmias are common in the setting of MI and are a major cause of morbidity and mortality. Close monitoring and immediate treatment of arrhythmias may be the most important part of the treatment of a post-MI patient within the first 48 hours. Pay close attention to exacerbating factors, such as electrolyte disturbances (especially potassium and magnesium), hypoxemia, drugs, or acidosis, and correct them accordingly.

Ventricular fibrillation and/or ventricular tachycardia occurring within the first 48 hours may be due to ischemia; however, if ventricular arrhythmias occur later, then further workup is indicated. Immediate cardioversion is the treatment of choice. Accelerated idioventricular arrhythmia is a ventricular arrhythmia that may occur in response to reperfusion. This rhythm has a benign prognosis and usually does not require therapy.

Supraventricular arrhythmias are also common. Sinus bradycardia may be due to drugs, ischemia, or a vagal response. Sinus tachycardia may be due to pain, anxiety, drugs, or other

causes. Atrial fibrillation and other atrial tachycardias may also occur. Treat any tachycardia by correcting the cause first or by pharmacotherapy, because persistent tachycardias may lead to further ischemia.

Conduction abnormalities may result from ischemia, necrosis, or chronotropic drugs, or as a vagal response. Recognition and treatment of these abnormalities are important in short- and long-term outcomes. Possible therapies include medications, such as atropine, or even placement of a transvenous pacemaker if indicated. Conduction disturbances are seen more commonly in the setting of inferior MI but are more ominous when seen with an anterior infarct.

Recurrent ischemia may be due to incomplete reperfusion. Postinfarct angina occurs in 20-30% of patients. This is an indication to proceed to cardiac catheterization followed by mechanical revascularization as needed.

CHF can be due to systolic dysfunction or diastolic dysfunction in the setting of MI. The severity of the heart failure and systolic dysfunction depends on the extent of the infarct and the presence of any other complications, such as acute mitral regurgitation. Aggressive treatment is indicated to avoid worsening of the situation. Treatment may include any or all of the following: nitrates, morphine, diuretics, ACE inhibitors, and other vasodilators if needed. Digoxin has no role in the setting of acute CHF due to ischemia.

Cardiogenic shock is defined as a systolic BP less than 90 mm Hg in the presence of organ hypoperfusion. The mortality rate due to cardiogenic shock is as high as 70% in some series. Patients usually require inotropic agents, such as dopamine or dobutamine, and occasionally an intraaortic balloon pump is required. Patients presenting with cardiogenic shock should proceed directly to the catheterization lab, if available, for mechanical revascularization. Acute MR is most common in the setting of an inferoposterior MI. This is secondary to ischemia, necrosis, or rupture of the LV papillary muscle (especially the posteromedial papillary muscle). This can lead to mild-to-severe MR with CHF. Diagnosis can be made on physical examination, but an echocardiogram is necessary to confirm the diagnosis and assess the severity, which helps in the choice of therapy. Treatment consists of aggressive afterload reduction, intraaortic balloon pump insertion, and immediate surgical repair.

[Video Rupture of chorda](#)

Ventricular rupture occurs in the interventricular septum or the LV free wall. Both are

catastrophic events with mortality rates greater than 90%. Prompt recognition, stabilization, and surgical repair are crucial to any hope of survival. Ventricular rupture is more common in women, patients with hypertension, and those receiving NSAIDs or steroids. An echocardiogram can usually define the abnormality, and a right heart catheterization can show an oxygen saturation step-up in the case of a septal rupture.

Other complications include pericarditis, ventricular aneurysms, mural thrombi, and hypertension. Recognition and treatment can be life saving.

Prognosis:

Acute MI is associated with a 30% mortality rate; half of the deaths occur prior to arrival at the hospital.

An additional 5-10% of survivors die within the first year after their MI.

Approximately half of all patients with an MI are rehospitalized within 1 year of their index event.

Overall, prognosis is highly variable and depends largely on the extent of the infarct, the residual LV function, and whether the patient underwent revascularization.

Patient Education:

Diet

Diet plays an important role in the development of CAD. Educate post-MI patients about the role of a low-cholesterol and low-salt diet.

Educate patients about the AHA dietary guidelines regarding a low-fat, low-cholesterol diet.

A dietitian should see and evaluate all post-MI patients prior to their discharge.

Smoking cessation

Educate all post-MI patients regarding the critical role of smoking in the development of CAD.

Smoking cessation classes should be offered to help patients avoid smoking after their MI.

For excellent patient education resources, visit eMedicine's Cholesterol Center. Also, see eMedicine's patient education articles High Cholesterol, Understanding Your Cholesterol Level, Lifestyle Cholesterol Management, Understanding Cholesterol-Lowering Medications, Chest Pain, Coronary Heart Disease, and Heart Attack.

MISCELLANEOUS

Medical/Legal Pitfalls:

Failure to make the diagnosis of an MI is the leading cause of litigation against ED physicians and cardiologists.

Consider atypical presentations in elderly patients, patients with diabetes, and women. Assess all patients carefully, especially if they have significant cardiac risk factors.

Review all ECGs that are obtained in a prompt fashion because time is crucial.

Obtain cardiology consultation whenever the diagnosis is questionable.

Consider an echocardiogram to assess wall motion abnormalities in difficult cases with nondiagnostic ECGs, such as with an LBBB.

Special Concerns:

Right ventricular infarction

Approximately one third of patients with inferior MI develop RV infarction. RV infarction presents a special challenge because the adjunctive therapy, other than reperfusion, is somewhat different.

A right-sided ECG with greater than 1 mm ST elevation in V3R or V4R leads describes an RV infarct. An echocardiogram may be helpful in confirming the diagnosis. On physical examination, signs of right heart failure, such as elevated jugular venous pulsation, right-sided S3, Kussmaul sign, or hypotension, may be present, and the patient may have clear lung fields. The patient becomes volume dependent to maintain adequate LV and RV filling. Occasionally, dobutamine may be needed, or even an intraaortic balloon pump for hemodynamic support. Avoid nitrates or any medications that lower preload in this setting. A pulmonary artery catheter can be helpful in guiding therapy.

Elderly patients

Elderly patients with acute MI are at increased risk of developing complications. Treat these patients aggressively.

Elderly patients have an increased risk of bleeding with thrombolytic therapy, but they also have the most to gain from this therapy.

Very elderly patients should undergo primary angioplasty if available, but they should receive thrombolytics if excessive delay is anticipated before angioplasty can be performed.

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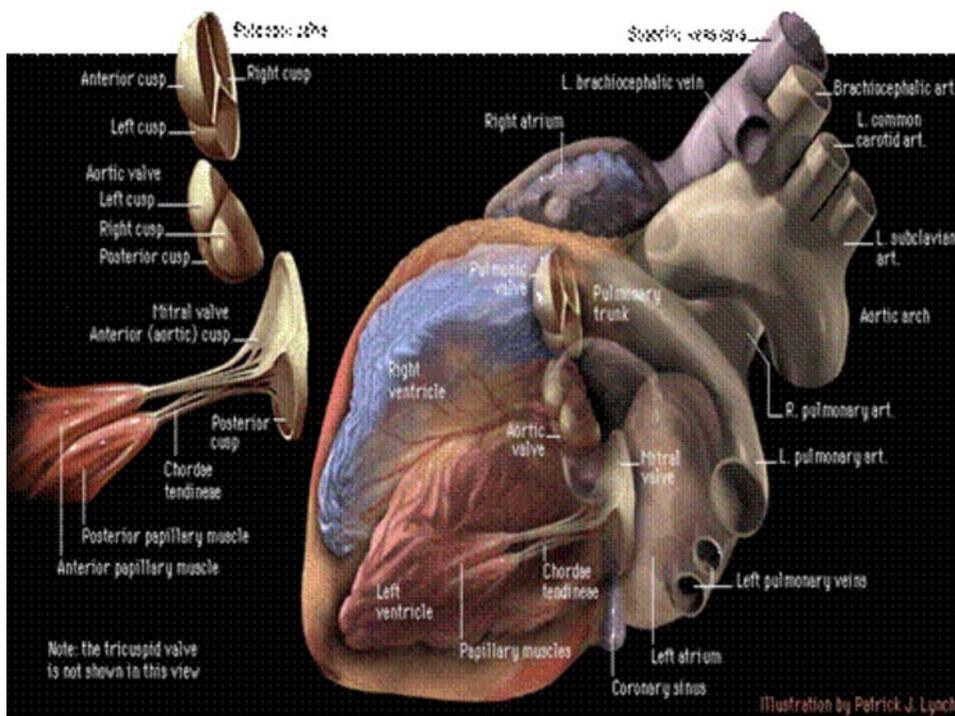
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Підготував Доброродній А.В.

Valvular Heart Disease

Primary valvular heart disease ranks well below coronary heart disease, stroke, hypertension, obesity, and diabetes as major threats to the public health. Nevertheless, it is the source of significant morbidity and mortality. Rheumatic fever is the dominant cause of valvular heart disease in developing countries. Its prevalence has been estimated to range from as low as 1.0 per 100,000 school-age children in Costa Rica to as high as 150 per 100,000 in China. Rheumatic heart disease accounts for 12–65% of hospital admissions related to cardiovascular disease and 2–10% of hospital discharges in some developing countries. Prevalence and mortality rates vary among communities even within the same country as a function of crowding and the availability of medical resources and population-wide programs for detection and treatment of Group A streptococcal pharyngitis. In economically deprived areas, tropical and subtropical climates (particularly on the Indian subcontinent), Central America, and the Middle East, rheumatic valvular disease progresses more rapidly than in more-developed nations and frequently causes serious symptoms in patients <20 years of age. This accelerated natural history may be due to repeated infections with more virulent strains of rheumatogenic streptococci.



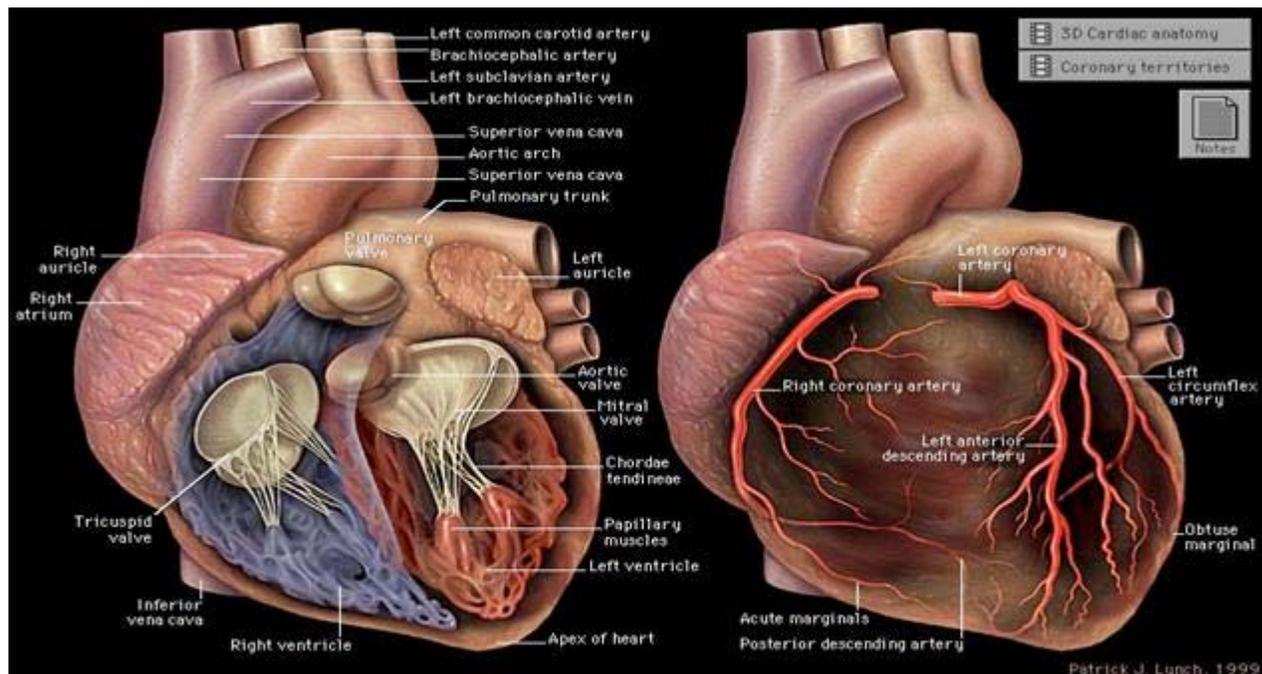


Fig. Heart anatomy

TS, a relatively uncommon valvular lesion in North America and Western Europe, is more common in tropical and subtropical climates, especially in southern Asia and in Latin America.

As of the year 2000, worldwide death rates for rheumatic heart disease approximated 5.5 per 100,000 population ($n = 332,000$), with the highest rates reported from Southeast Asia. Although there have been reports of recent isolated outbreaks of streptococcal infection in North America, valve disease in developed countries is now dominated by degenerative or inflammatory processes that lead to valve thickening, calcification, and dysfunction. The prevalence of valvular heart disease increases with age. Important left-sided valve disease may affect as many as 12–13% of adults over the age of 75.

The incidence of infective endocarditis has increased with the aging of the population, the more widespread prevalence of vascular grafts and intracardiac devices, the emergence of more virulent multidrug-resistant microorganisms, and the growing epidemic of diabetes. Infective endocarditis has become a more frequent cause of acute valvular regurgitation.

Bicuspid aortic valve disease affects as many as 1–2% of the population, and an increasing number of childhood survivors of congenital heart disease present later in life with valvular dysfunction. The past several years have witnessed significant improvements in surgical outcomes with progressive refinement of relatively less-invasive techniques. Percutaneous heart valve replacement or repair is under active clinical investigation.

Major Causes of Valvular Heart Diseases

Valve Lesion	Etiologies
Mitral stenosis	<ul style="list-style-type: none"> Rheumatic fever Congenital Severe mitral annular calcification SLE, RA
Mitral regurgitation	<ul style="list-style-type: none"> Acute <ul style="list-style-type: none"> Endocarditis Papillary muscle rupture (post-MI) Trauma Chordal rupture/Leaflet flail (MVP, IE) Chronic <ul style="list-style-type: none"> Myxomatous (MVP) Rheumatic fever Endocarditis (healed) Mitral annular calcification Congenital (cleft, AV canal) HOCM with SAM Ischemic (LV remodeling) Dilated cardiomyopathy
Aortic atenosis	<ul style="list-style-type: none"> Congenital (bicuspid, unicuspid) Degenerative calcific Rheumatic fever
Aortic regurgitation	<ul style="list-style-type: none"> Valvular <ul style="list-style-type: none"> Congenital (bicuspid) Endocarditis Rheumatic fever Myxomatous (prolapse) Traumatic Syphilis

Ankylosing spondylitis

Root disease

Aortic dissection

Cystic medial degeneration

Marfan syndrome

Bicuspid aortic valve

Nonsyndromic familial aneurysm

Aortitis

Hypertension

Tricuspid stenosis Rheumatic

Congenital

Tricuspid regurgitation Primary

Rheumatic

Endocarditis

Myxomatous (TVP)

Carcinoid

Congenital (Ebstein's)

Trauma

Papillary muscle injury (post-MI)

Secondary

RV and tricuspid annular dilatation

Multiple causes of RV enlargement (e.g., long-standing pulmonary

HTN)

Chronic RV apical pacing

Pulmonic stenosis Congenital

Carcinoid

Pulmonic regurgitation Valve disease

Congenital

Postvalvotomy

Endocarditis

- Annular enlargement
- Pulmonary hypertension
- Idiopathic dilatation
- Marfan syndrome

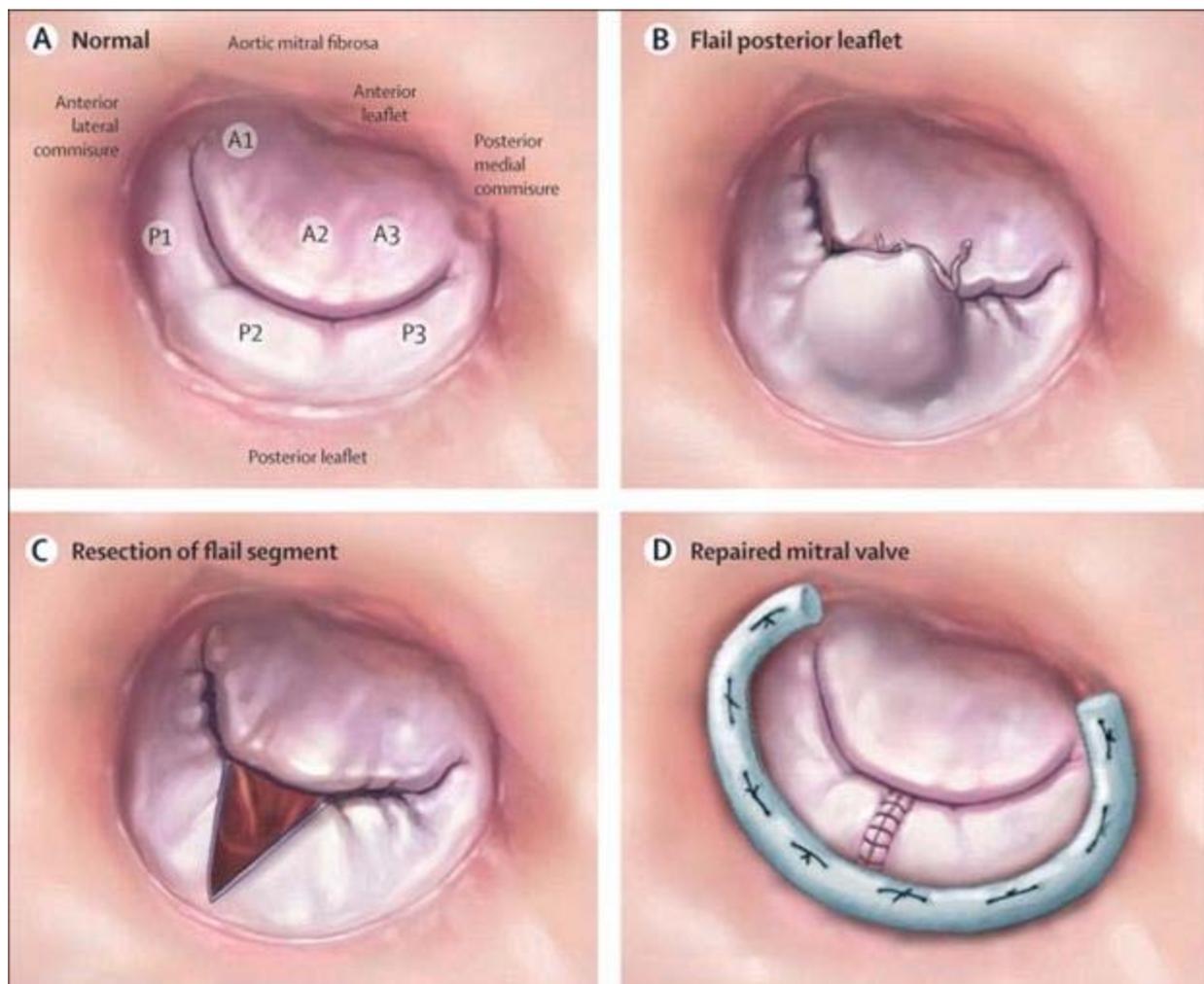
Mitral regurgitation

Mitral regurgitation affects more than 2 million people in the USA. The main causes are classified as degenerative (with valve prolapse) and ischaemic (ie, due to consequences of coronary disease) in developed countries, or rheumatic (in developing countries). This disorder generally progresses insidiously, because the heart compensates for increasing regurgitant volume by left-atrial enlargement, causes left-ventricular overload and dysfunction, and yields poor outcome when it becomes severe. Doppler-echocardiographic methods can be used to quantify the severity of mitral regurgitation. Yearly mortality rates with medical treatment in patients aged 50 years or older are about 3% for moderate organic regurgitation and about 6% for severe organic regurgitation. Surgery is the only treatment proven to improve symptoms and prevent heart failure. Valve repair improves outcome compared with valve replacement and reduces mortality of patient with severe organic mitral regurgitation by about 70%. The best short-term and long-term results are obtained in asymptomatic patients operated on in advanced repair centres with low operative mortality (<1%) and high repair rates (≥ 80 —90%). These results emphasise the importance of early detection and assessment of mitral regurgitation.

Mitral regurgitation is defined as systolic retrograde flow from the left ventricle into the left atrium. Although a trivial form of this valve disease is often seen in healthy people, epidemiological data show that moderate or severe regurgitation is the most frequent valve disease in the USA and is the second most common form of valvular heart disease needing surgery in Europe. Despite substantial reduction in the incidence of rheumatic heart disease, mitral regurgitation is a growing public health problem. Moderate or severe regurgitation is frequent, its prevalence increases with age, and it was estimated to affect 2.0—2.5 million people in the USA in 2000—a number expected to almost double by 2030 because of population ageing and growth. Although no large epidemiological studies are available, mitral regurgitation is prevalent in young adults in countries with endemic rheumatic fever. Substantial progress has been achieved to improve its diagnosis, quantification, and surgical treatment. Improved knowledge of clinical outcome of patients with mitral regurgitation

resulted in refined surgical indications. Hence, mitral regurgitation is a disease in which restoration of life expectancy can often be achieved, an encouraging outcome that emphasises the importance of early detection, assessment, and prompt consideration for treatment of patients with this condition. Challenges in management of patients with mitral regurgitation remain—elderly patients and those with disease due to ischaemic heart disease are often not offered surgery; valve repair—the preferred surgical method—is insufficiently done; new interventional techniques—minimally invasive or percutaneous—are under investigation. However, the general absence of clinical trials means evidence to guide treatment is weak.

All lesions that cause mitral regurgitation do so by reduction or elimination of the normal systolic coaptation between anterior and posterior mitral leaflets, which normally ensures mitral competence. Consistent anatomical and functional descriptors of mitral lesions are essential to assess surgical reparability but overlapping and poorly defined terminology has caused confusion. Causes and mechanisms are not synonymous and a particular cause might produce regurgitation by different mechanisms (table). Surgical correction of this valve disease is dependent on both cause and mechanism, which affect reparability. Causes are generally classified as ischaemic (mitral regurgitation due to consequences of coronary disease, not fortuitous association of both) and non-ischaemic (all other causes). Mechanisms are grossly classified as functional (mitral valve is structurally normal and disease results from valve deformation caused by ventricular remodelling) or organic (intrinsic valve lesions). They can be subclassified by leaflet movement (Carpentier's classification)—type I (normal valve movement, such as annular dilatation or leaflet perforation); type II (excessive movement); and type III (restrictive movement: IIIa—diastolic restriction such as rheumatic disease; IIIb—systolic restriction as in functional disease). Carpentier also proposed a simple lesion localisation classification.



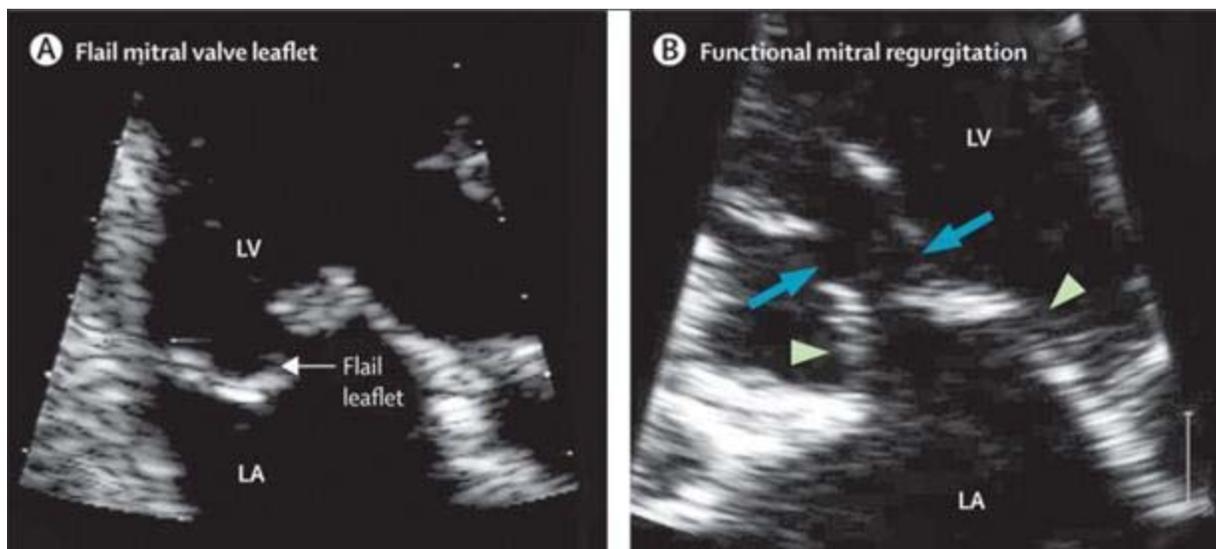
Schematic anatomical mitral-valve presentation

(A) Atrial view of a healthy mitral valve. Posterior leaflet has a shorter length but occupies a longer circumference than the anterior leaflet. Mitral annulus around the leaflet is part of the aortic-mitral fibrosa superiorly, is asymmetric, and short in its anteroposterior dimension. Leaflet segmentation starts with A1—P1 close to the anterolateral commissure, with A2—P2 centrally, and A3—P3 close to the posteromedial commissure. The normally apposing leaflets make up the mitral smile. (B) Example of a flail posterior leaflet affecting the P2 segment with ruptured chordae. Note the bulge and excess tissue of the flail segment and the annular enlargement mostly along the posterior part of its circumference. (C) Initial step of surgical valve repair. Resection of the flail segment can be triangular (as shown) or quadrangular and leaves the healthy P1 and P3 segments available for reattachment and repair. (D) Posterior leaflet has been restored by approximation of the remaining segment after resection of the flail segment and the mitral annular dimensions have been restored by an annuloplasty ring. In this example an incomplete ring has been used with its extremities sutured to the trigonal regions of the aortic-mitral fibrosa. The mitral smile and competence have been restored.

Major causes of surgical mitral regurgitation in western countries are degenerative (primary myxomatous disease, primary flail leaflets, annular calcification), representing 60—70% of cases, followed by ischaemic mitral regurgitation (20%), endocarditis (2—5%), rheumatic (2—5%), and miscellaneous causes (cardiomyopathies, inflammatory diseases, drug-induced, traumatic, congenital). Ischaemic disease probably represents a large proportion of the

non-surgical disease burden. Nomenclature and mechanisms of major causes are summarised below.

Degenerative mitral regurgitation is usually related to mitral-valve prolapse and rarely to isolated mitral annular calcification. Mitral-valve prolapse is an abnormal systolic valve movement into the left atrium (≥ 2 mm beyond saddle-shaped annular level). This excessive movement can be seen with other causes such as endocarditis. Prolapse might be of moderate magnitude (leaflet tips remain in the left ventricle—ie, billowing mitral valve) or can be severe (eversion of leaflet tip into left atrium—ie, flail leaflet—usually caused by ruptured chordae). The main phenotypes of mitral prolapse are diffuse myxomatous degeneration (mitral-valve prolapse syndrome or Barlow's disease, sometimes with posterior annular translocation into left atrium) or primary flail leaflets with ruptured chordae affecting the posterior leaflet in 70% of cases, and accompanied by myxomatous degeneration localised to the flail segment and generally normal valve morphology elsewhere. Myxomatous degeneration remodels valve tissue by increasing the spongiosa layer and valve water content and thickness, with mucopolysaccharide and matrix changes, as a functional manifestation of metalloproteinase alterations. These mitral tissue changes and prolapse might be genetically transmitted and X-chromosome linked. Degenerative mitral regurgitation is the most reparable form, warranting early and careful assessment.



Echocardiographic appearance of the two main anatomical types of mitral regurgitation from apical views centred on the mitral valve

(A) An example of a flail posterior leaflet with the tip of the leaflet floating in the left atrium. Note the otherwise grossly normal anterior leaflet. (B) An example of functional mitral regurgitation. Strut chordae (long arrows) to the anterior and posterior leaflets exert an abnormal traction on the body of the leaflets, which displaces (arrowheads) the leaflets towards the ventricular apex, creating an area of tenting above the mitral annulus and an incomplete coaptation. LA=left atrium. LV=left ventricle.

The ischaemic form of this disease rarely results from an organic mechanism (papillary-muscle rupture) and is rarely acute. Frequently, it is functional (structurally normal leaflets) and chronic, epitomising left-ventricular disease that causes valvular dysfunction. Papillary-muscle dysfunction plays little part in the generation of functional mitral regurgitation, which is mostly caused by apical and inferior-papillary-muscle displacement due to ischaemic left-ventricular remodelling. Because chordae are non-extensible, papillary-muscle displacement exerts traction on leaflets through strut chordae implanted on the body of leaflets, resulting in tethered and apically displaced leaflets (tenting). Coupled with annular flattening, enlargement, and decreased contraction, mitral valve tenting results in coaptation loss that yields functional mitral regurgitation. Asymmetric tenting due to regional scarring (inferior infarction) might explain commissural jets of ischaemic disease. Rheumatic mitral regurgitation—past the acute phase—causes chordal and leaflet retraction, which, amplified by annular dilatation, results in coaptation loss. Postinflammatory and postradiation mitral regurgitations have similar mechanisms. Retraction of tissue is a major limitation to successful valve repair.

Endocarditic mitral regurgitation might be caused by ruptured chordae or perforations. In all causes, annular enlargement is common, is located mostly or exclusively on the posterior part of the annular circumference, and surgical repair almost always requires annuloplasty.

Pathophysiology and progression

The degree of mitral regurgitation is defined by lesion severity (measured as effective regurgitant orifice [ERO] area) and the yielding volume overload (measured as regurgitant volume [RVol]), but it is also affected by the driving force (left-ventricular systolic pressure) and left-atrial compliance.⁵ Thus, in acute disease, the large regurgitant orifice converts ventricular energy mostly into potential energy (left-atrial pressure V-wave) due to non-compliant left atrium. In chronic regurgitation, the enlarged left atrium is compliant, the V-wave is often small, and ventricular energy is converted mostly into kinetic energy (large RVol). This process of atrial enlargement and increased compliance probably explains atrial pressure reduction and clinical improvement after initial heart failure caused by acute mitral regurgitation.

The ERO area is not necessarily fixed and can be dynamic. Increased loading or contractility can cause the ERO area to increase or decrease slightly. With valve prolapse, the area is very dynamic, increasing progressively during systole, and is sometimes purely end-systolic. In

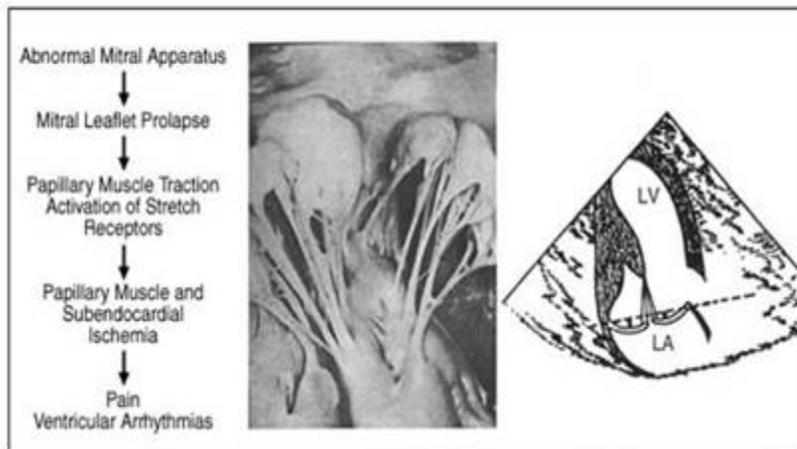
functional mitral regurgitation, ERO area is dynamic during systole, with large area during short isovolumic contraction and relaxation phases caused by lesser ventricular pressure apposing leaflets. This type of regurgitation is also dynamic with decreased loading or inotrope administration, and might disappear with these interventions, whereas exercise most often results in augmentation of ERO area. Long-term progression of organic disease is about 5—7 mL per year for RVol and is determined by ERO area progression caused by new lesions or annular enlargement. Thus, mitral regurgitation is self-sustained, causing atrial and annular enlargement, which in turn leads to increased ERO area.

The ventricular and atrial consequences of organic mitral regurgitation are initiated by volume overload with increased preload and left-ventricular and left-atrial enlargement. Impedance to ejection is reduced despite normal or increased vascular resistances, whereas myocardial afterload (end-systolic wall stress) is normal with an end-systolic volume that is normal to slightly increased. Thus, in organic disease, altered left-ventricular function might coexist with normal or high ejection fraction. Borderline normal ejection fraction, between 50—60%, already implies overt left-ventricular dysfunction. Ventricular dysfunction should be suspected when end-systolic dimensions are large but is often masked by a large ejection volume and is revealed after surgical elimination of mitral regurgitation, with a postoperative average immediate ejection fraction drop of about 10%. Diastolic ventricular dysfunction is difficult to characterise, but seems to reduce exercise capacity.

Physiology of functional mitral regurgitation is even more complex than that of organic mitral regurgitation since ventricular dysfunction predates the regurgitation. Nevertheless, functional mitral regurgitation further increases atrial pressure, which leads to pulmonary hypertension and heart failure. With increased atrial pressure and low driving force, functional regurgitation often has low RVol and can be silent.

Mitral Valve Prolapse

Long Axis View of the
Same Valve Illustrated in Figure 13



Whether functional regurgitation affects remodelling and dysfunction is uncertain but is suspected because of the high mortality associated with increased severity of mitral regurgitation.

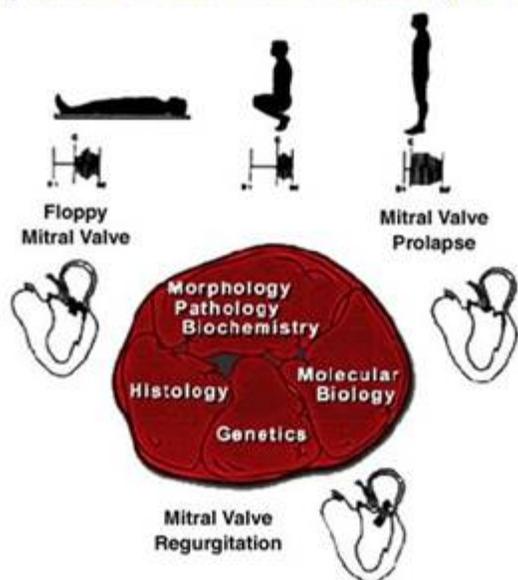
Progression or recurrence after annuloplasty is weakly related to annular enlargement but strongly to increased mitral tenting caused by ventricular remodelling, papillary-muscle displacement, and increased chordal traction; however, rates of progression are unknown.

Assessment

Initial clinical assessment looks for symptoms, signs of heart failure, and physical signs of severe mitral regurgitation—ie, displaced apical impulse, systolic thrill, loud systolic murmur, S3, early diastolic rumble, and cardiomegaly with left-atrial enlargement on chest radiography

and atrial fibrillation. These signs are important but not specific enough to rely solely on them to suggest surgery.

Spectrum of Mitral Valve Disease (1 of 2)



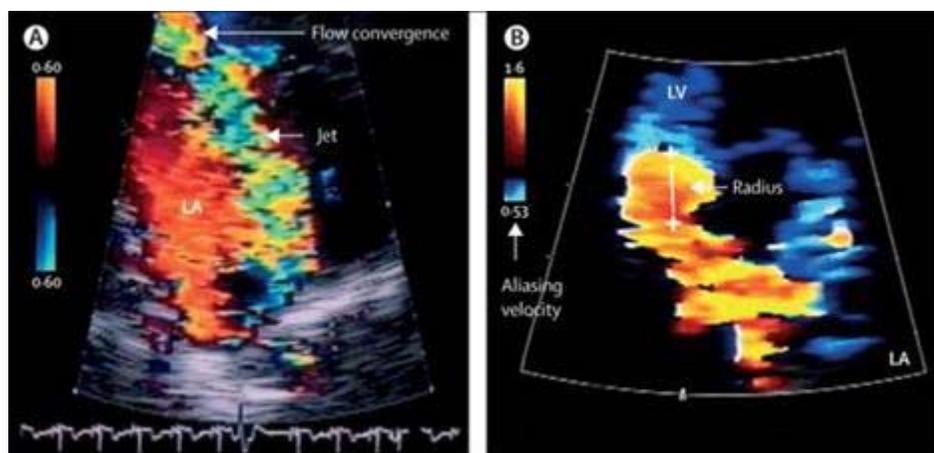
Prognosis in Mitral Valve Prolapse

- Often benign
- Rare complications
 - Endocarditis
 - Progressive MR
 - Acute
 - Chronic
 - Thromboembolism
 - Atrial and ventricular arrhythmias

Doppler echocardiography is the main method for assessment of patients with mitral regurgitation. Transthoracic or transoesophageal echocardiography provides functional anatomical information that is crucial for assessment of reparability by defining cause, mechanism, presence of calcification, and localisation of lesions. Transoesophageal echocardiography provides better imaging quality than transthoracic echocardiography but its ability to detect details such as ruptured chordae rarely changes management. Transoesophageal echocardiography essentially provides incremental clinically meaningful information (such as reparability) when transthoracic echocardiography is of poor quality or when complex,

calcified, or endocarditic lesions are suspected. Thus, transoesophageal echocardiography is rarely used on an outpatient basis and is mostly used intraoperatively for lesion verification and to monitor surgical results. Real-time three-dimensional echocardiography has at present insufficient image resolution but pilot data suggest that it allows quantitative assessment of structures that are not easily measurable by two-dimensional echocardiography, such as mitral annulus. Although emerging technologies such as transoesophageal echocardiography three-dimensional imaging have great potential, they need to be rigorously tested.

Doppler echocardiography provides crucial information about mitral regurgitation severity (table). Comprehensive integration of colour-flow imaging and pulsed and continuous wave doppler echocardiography is necessary because jet-based assessment has major limitations (figure). Quantitative assessment of regurgitation is feasible by three methods—quantitative doppler echocardiography based on mitral and aortic stroke volumes, quantitative two-dimensional echocardiography based on left-ventricular volumes, and flow-convergence analysis measuring flow with colour-flow imaging proximal to the regurgitant orifice (proximal isovelocity surface area method; figure). These methods allow measurement of ERO area and RVol and have important prognostic value. Severe mitral regurgitation is diagnosed with an ERO area of at least 40 mm² and RVol of at least 60 mL per beat; and moderate regurgitation with ERO area 20—39 mm² and RVol 30—59 mL per beat. Outcome data suggest that a smaller volume mitral regurgitation and smaller ERO area (≥ 30 mL and ≥ 20 mm², respectively) are associated with severe outcome in patients with ischaemic disease; therefore, thresholds for severe disease are cause-dependent. Consistency in all measures of mitral regurgitation severity is essential to appropriately grade disease severity (table). Haemodynamic assessment is completed with doppler measurement of cardiac index and pulmonary pressure.



Use of colour-flow imaging for assessment of mitral regurgitation

(A) Jet imaging in left atrium. The jet is eccentric and is displayed with mosaic colours, whereas the normal

flow is of uniform colour. It fills only part of the left atrium and might underestimate the regurgitation. The observation of a large flow convergence should lead to suspicion of severe regurgitation. (B) Measurement of the flow convergence with colour-flow imaging. The baseline of the colour scale has been brought down to decrease the aliasing velocity to 53 cm/s (velocity at the blue-yellow border), which allows the flow convergence (yellow) to be seen. The radius (r) of the flow convergence is used in the formula for calculation of the instantaneous regurgitant flow (258 mL/s). $\text{Flow} = 6.28 \times V_{\text{aliasing}} \times r^2 = 6.28 \times 53 \times 0.882 = 258 \text{ mL/s}$. Division of this value by the jet velocity allows calculation of the effective regurgitant orifice of mitral regurgitation.

LA=left atrium. LV=left ventricle.

Doppler echocardiography also measures left-ventricular and left-atrial consequences of mitral regurgitation. End-diastolic left-ventricular diameter and volume indicate volume overload whereas end-systolic dimension shows volume overload and ventricular function. Patients with left-ventricular ejection fraction less than 60% or end-systolic diameter of at least 40—45 mm are regarded as having overt left-ventricular dysfunction. Left-atrial diameter indicates volume overload but also conveys important prognostic information. Left-atrial volume was recommended as the preferred measure of atrial overload, (at least 40 mL/m² for severe dilatation) and predicts the occurrence of atrial fibrillation.

Exercise tests are used to define functional capacity. One in five asymptomatic patients shows severe functional limitations during cardiopulmonary exercise. Peak oxygen consumption compared with that expected for age, sex, and weight objectively measures functional limitations versus normal reference values. Other exercise modalities, such as supine-bike exercise, examine changes in severity of mitral regurgitation with activity, especially seen in ischaemic and functional disease and might reveal poor prognosis when ERO area increases. Standard postexercise echocardiography was used to detect exertional ventricular volume increase as a predictor of postoperative left-ventricular dysfunction, but difficulties in measurement of monoplane ventricular volumes hinder this approach. Other stress tests are rarely used. Dobutamine echocardiography reduces mitral regurgitation universally, but in selected patients with ischaemic disease it might reveal viability and ischaemia.

MRI shows mitral regurgitation jets, with limitations similar to those of colour-flow imaging; quantitative measurements are possible but validation studies are few. This imaging method is unique in revealing ventricular scars and in assessment of viability in ischaemic disease and is useful in measurement of ventricular volumes but its incremental diagnostic role remains unknown.

Detection of hormonal activation is important in many cardiac diseases. Atrial natriuretic peptide has little specificity for mitral regurgitation and is strongly activated by arrhythmias, irrespective of mitral regurgitation severity. B-type natriuretic peptide is of greater value than atrial natriuretic peptide in patients with regurgitation. Its activation in organic disease is

determined by the consequences—mostly left-atrial enlargement, symptoms, rhythm, and left-ventricular function—rather than the severity of regurgitation. Importantly, its activation is associated with poor outcome and should alert clinicians. Strong B-type natriuretic peptide activation is noted in functional mitral regurgitation linked to the severity of end-systolic ventricular changes and of mitral regurgitation. Subtle sympathetic activation and altered β receptors in organic disease might indicate left-ventricular dysfunction but are usually less prominent than in functional disease.

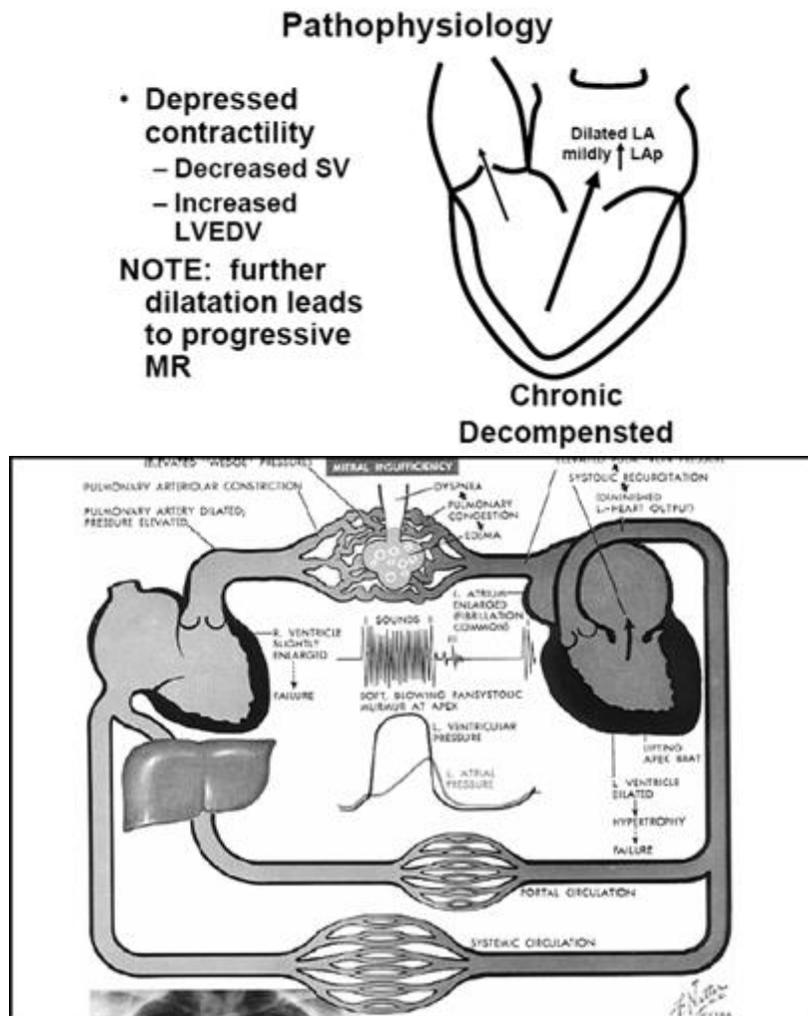
Cardiac catheterisation is not consistently used by institutions and might be overused in some.³ In academic centres, it is rarely used to define haemodynamics, which are usually provided by doppler echocardiography. Left ventriculography and right-heart catheterisation are rarely needed for assessment of mitral regurgitation. Conversely, in most patients aged 45 years or older, coronary angiography is routinely done preoperatively.

Natural history and clinical outcome

Although a few prospective studies are available, most data for mitral regurgitation outcome are extracted from observational series. Clinical outcome under medical management and after surgery is different in organic and functional disease.

Natural history of organic regurgitation has been poorly defined, largely because of limitations in severity assessment. Old studies, before echocardiography, showed a wide range in 5-year survival rates from 27% to 97%, probably related to variations in severity. Most data (table) were from studies of patients diagnosed with mitral regurgitation due to flail leaflets, most of whom had severe regurgitation. Such patients have ventricular enlargement causing the notable volume overload and incur excess mortality overall; mortality was especially high in patients with class III—IV symptoms but also notable in those with no or minimum symptoms. A sudden death rate of 1.8% per year overall varied from as high as 12.0% per year in patients with class III—IV symptoms who had not undergone surgery to 0.8% per year in asymptomatic patients with normal ejection fraction and sinus rhythm. Patients in some mitral regurgitation subsets have low mortality, such as young patients (<50 years) even with severe mitral regurgitation or those of all ages with initially a moderate disorder. Conversely, in a prospective study of asymptomatic patients with long-term follow-up, those with severe regurgitation proven by quantitative measurements showed increased mortality under medical management.⁹ Thus, older patients (≥ 50 years) with severe (defined as ERO area ≥ 40 mm²) organic mitral regurgitation are at increased risk of mortality (yearly rates of about 3% for moderate regurgitation vs 6% for the severe organic form). For morbid complications, all studies substantiated the adverse effect of severe regurgitation. Patients with flail leaflet and in general

those with severe mitral regurgitation had, under medical management, yearly cardiac event rates of 10—12%—including about 9% for heart failure and 5% for atrial fibrillation. Within 10 years of diagnosis, cardiac events arise in most patients with severe mitral regurgitation, and death occurs or cardiac surgery is needed in at least 90%, making surgery an almost unavoidable consideration in such patients. The risk of stroke is low, but in excess of that expected in old patients and is strongly linked to occurrence of atrial fibrillation, and thus to left-atrial size. Predictors of reduced survival under medical management are symptoms (class III or IV), even if transient, reduced ejection fraction, severe mitral regurgitation with ERO area of 40 mm² or more, and hormonal activation, although not as well substantiated. Predictors of cardiac events are atrial fibrillation, left-atrial enlargement of at least 40—50 mm diameter, flail leaflet or large ERO area—all markers of severe mitral regurgitation—and, during exercise, reduced peak oxygen consumption and possibly reduced right ventricular function.



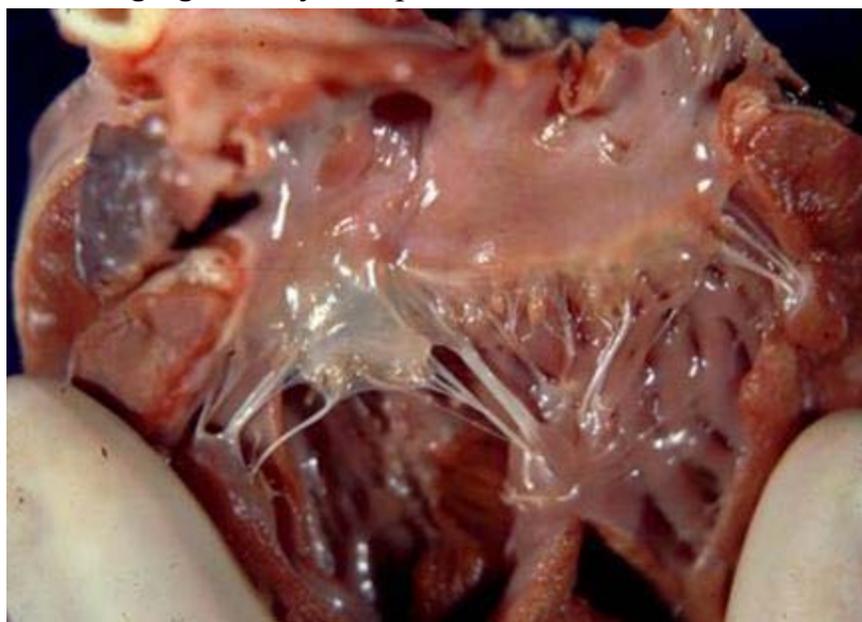
Clinical outcome after surgery depends on patient-specific, disease-related, and surgery-related factors. Early postoperative mortality is largely affected by age, but improvement of surgical results reduced the risk to about 1% for patients younger than 65 years, 2% for those aged

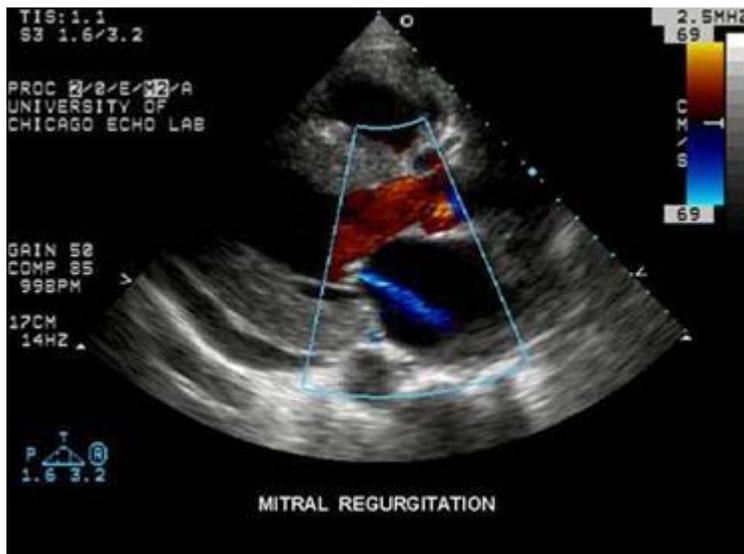
65—75 years, and 4—5% for older than 75 years. Increased surgical risk is also linked to preoperative severe symptoms or heart failure whereas ejection fraction has less effect. Surgery-related determinants of operative risk are governed by mitral reparability, which ensures reduced risk, whereas risk is increased with concomitant coronary artery bypass grafting. Other associated procedures, such as tricuspid repair or replacement, or those aimed at treatment or prevention of atrial fibrillation need a longer bypass time, which can increase risk. Long-term, patient-related factors continue to affect outcome, particularly coronary disease or reduced renal function. Age determines mortality but restoration of life expectancy is similar in young and old patients. After surgery, patients with severe symptoms before surgery continue to have increased mortality despite symptom relief, whereas in those with no or few symptoms, restoration of life expectancy can be achieved. Similarly, patients with overt preoperative ventricular dysfunction have increased postoperative mortality, especially with ejection fraction less than 50%. Generally, a 10% early postoperative reduction in ejection fraction happens after elimination of volume overload, whereas end-systolic characteristics (volume, wall stress) are unchanged. This reduction is lowest after valve repair and is minimised by preservation of subvalvular apparatus during valve replacement. Nevertheless, 25—30% of patients with mitral regurgitation present with postoperative left-ventricular dysfunction, especially those with preoperative ejection fraction of less than 60% or end-systolic diameter at least of 40—45 mm. Occasional unexpected ventricular dysfunctions arise in patients with ejection fraction greater than 60% and no perfect predictor has been identified. Hence, in some centres, prevention of postoperative left-ventricular dysfunction relies on performance of early surgery when no sign of left-ventricular alteration is present.

Coronary disease (even in the absence of angina) increases the risk of left-ventricular dysfunction despite the performance of coronary artery bypass grafting. Although no clinical trial has compared outcomes of patients randomised to repair versus replacement, observational evidence suggests that the major surgical determinant of improved long-term outcome is valve repair, which allows restoration of life expectancy and reduces the risk of heart failure after surgery. Although mitral regurgitation can recur after repair, reoperation rates do not differ after repair compared with replacement. Thus, mitral valve repair is widely regarded as the preferred mode of correction of organic mitral regurgitation, especially degenerative.

For ischaemic mitral regurgitation, the natural history of the functional form is incompletely defined whereas that of papillary-muscle rupture is known to be rapidly fatal. Whether functional regurgitation intrinsically causes poor outcome, or whether it indicates left-ventricular alterations, is still disputed. However, association of severe ischaemic mitral

regurgitation with severe outcomes, independent of ejection fraction, age, and presentation, suggests that the regurgitation is indeed causal of the poor outcome. This prognostic role of mitral regurgitation is now substantiated by results from studies of patients with acute or chronic myocardial infarction, by clinical trials and by population studies. Another important concept is that even modest regurgitation is associated with substantially increased mortality, a fact proved by quantitative data. ERO area of ischaemic mitral regurgitation independently predicts excess mortality. Patients with an area larger than 20 mm² incur about a two-fold increase in mortality risk and about a four-fold increase in the risk of heart failure compared with those with a similar ischaemic left-ventricular dysfunction but no mitral regurgitation. The better predictive value of ERO area than that of RVol is explained by the strong link between ERO area and filling pressure. Increase in ERO area with exercise might additionally affect clinical outcome, survival, and heart failure. Nevertheless, a clinical trial is needed to determine whether surgical correction of the valvular consequence (ischaemic mitral regurgitation) improves mortality and heart failure in this mainly ventricular disease. Clinical outcome of functional disease caused by cardiomyopathy is not well defined but few data suggest that mitral regurgitation yields poor outcomes.





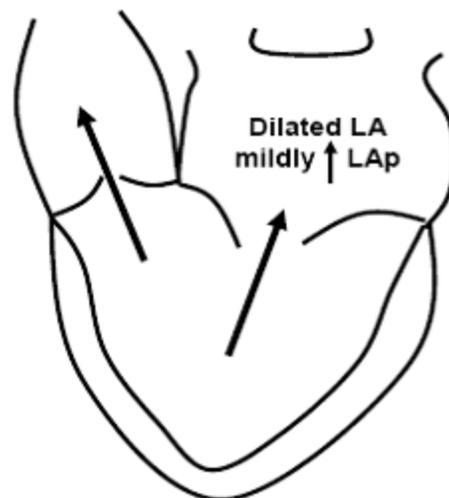
When MR is big, so are the indices:



ECHOinContext

Pathophysiology

- **Eccentric hypertrophy**
 - Increased preload
 - Increased afterload
 - Increased total stroke volume AND forward stroke volume AND LVESV returns to normal
- **Increased LA size**
 - Increased LA compliance
 - Larger volume at lower pressure



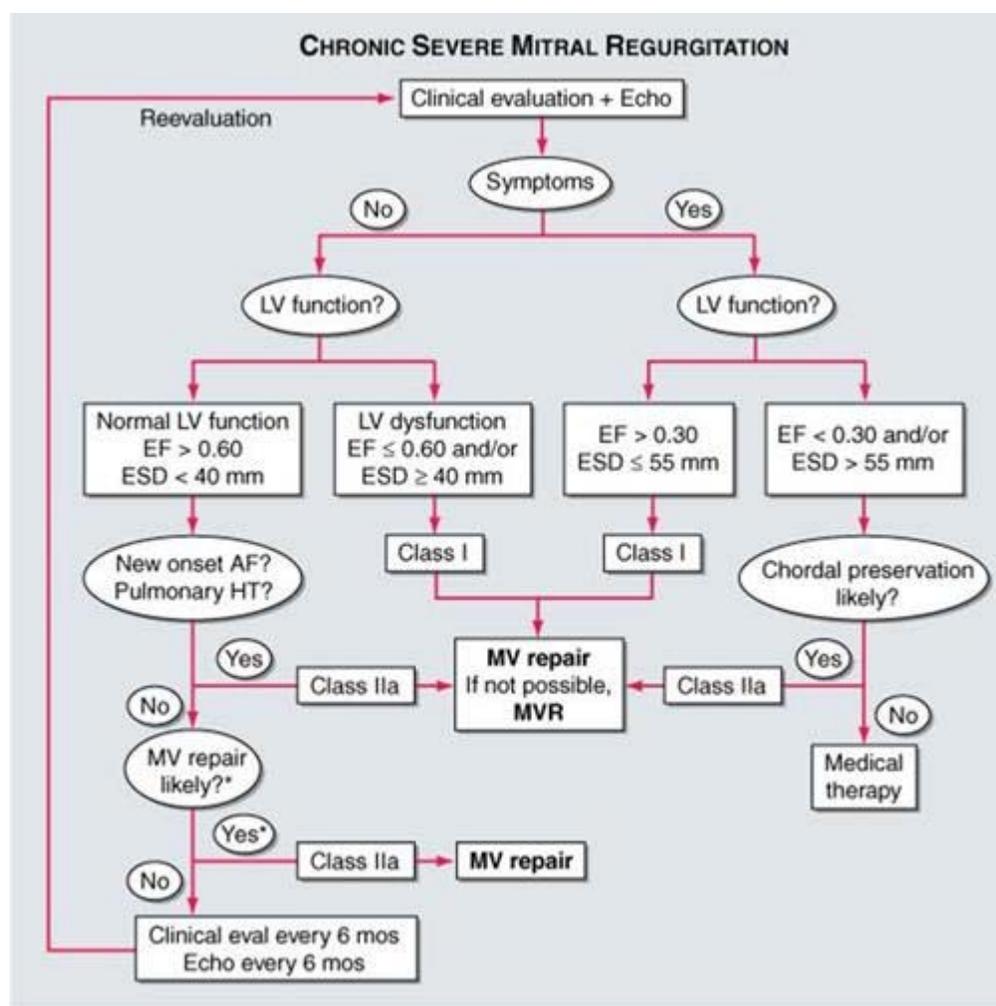
**Chronic
Compensated**

Fig Mitral regurgitation

Outcomes after surgery for functional disease remain suboptimum. Operative mortality is still high despite definite surgical improvements. Long-term mortality and heart failure rates are high, although not unexpected in patients with coronary disease, previous myocardial infarction, reduced ventricular function, and vascular comorbidity. These suboptimum outcomes explain uncertainties in surgical indications. However, with low operative mortality, postoperative heart failure and symptomatic improvements are possible.

Treatment

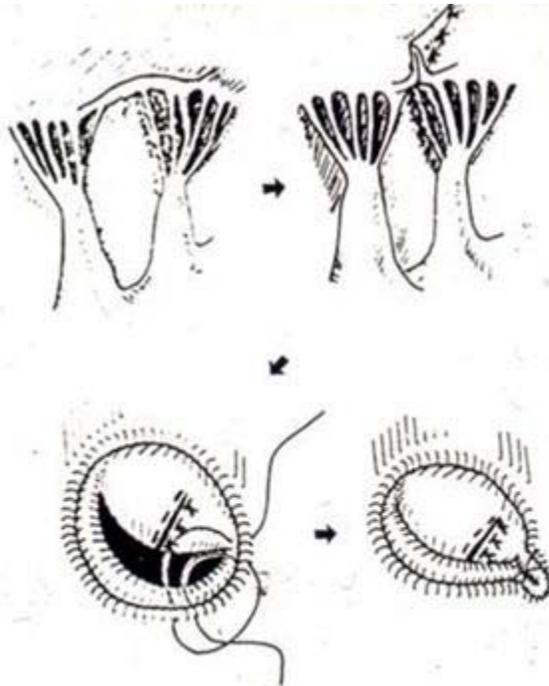
The natural history of untreated organic and functional mitral regurgitation emphasises the importance of treatment of patients with severe regurgitation. Because the effects of various treatments on survival have not been tested in randomised clinical trials, the value of any approach is estimated on the basis of outcome studies.



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: <http://www.accessmedicine.com>

Treatment of Symptomatic Mitral Regurgitation

- **Medical therapy**
 - Diuretics
 - Vasodilators
 - ACE inhibitors
- **Surgical therapy**
 - **MV replacement**
 - relief of symptoms
 - MVR survival similar to natural history
 - Operative mortality 8-10%
 - 10 year survival 50%
 - **Mitral valve repair**
 - Operative mortality 2-4%
 - 10 year survival 80%
 - Preservation of mitral apparatus!!!!!!!!!!!!!!!
 - No risk of thrombotic complications



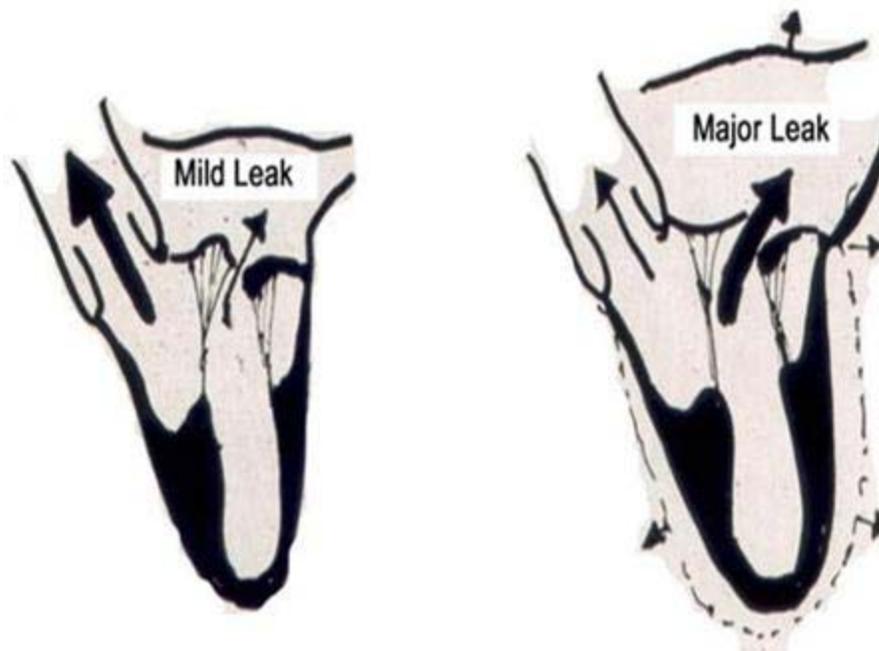
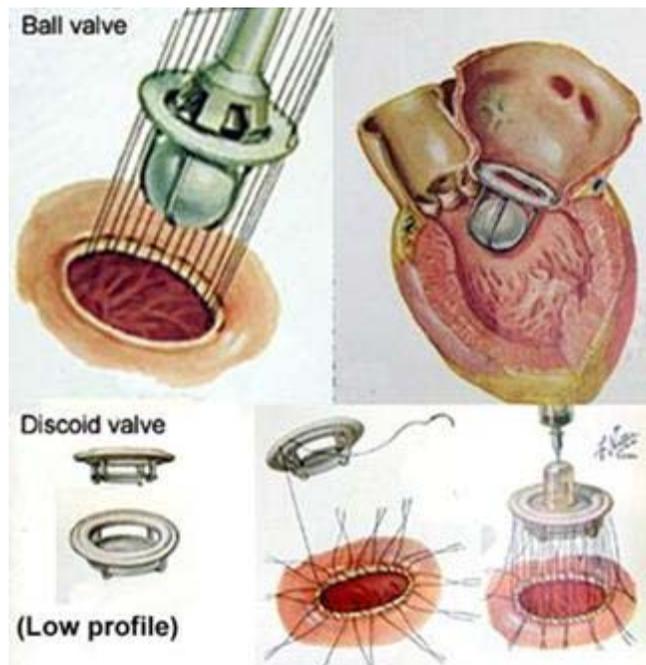


Fig Management strategy for patients with chronic severe mitral regurgitation. *Mitral valve (MV) repair may be performed in asymptomatic patients with normal left ventricular (LV) function if performed by an experienced surgical team and if the likelihood of successful MV repair is >90%. AF, atrial fibrillation; Echo, echocardiography; EF, ejection fraction; ESD, end-systolic dimension; eval, evaluation; HT, hypertension; MVR, mitral valve replacement. (From Bonow *et al.*)

Medical treatment aims to prevent progression of organic disease. Prevention of endocarditis is directed at forestalling catastrophic infectious complications and sudden mitral regurgitation progression associated with endocarditis. Diuretics often reduce or eliminate symptoms of disease but such improvement should not unduly reassure physicians. Patients who had transiently severe symptoms and improved with treatment continue to be at high risk and

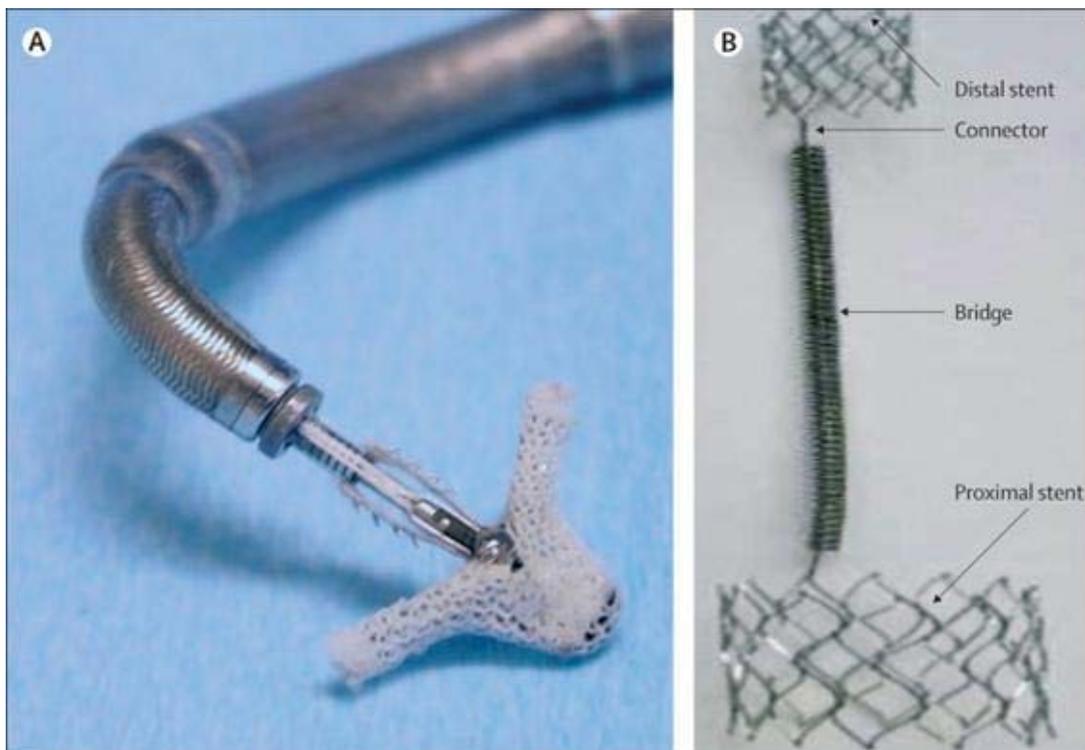
should be promptly assessed for surgery.

Treatment of organic mitral regurgitation with vasodilators has been advocated on the basis of experimental studies showing reductions in acute RVol and even ERO area with blood pressure reduction. Acutely ill patients with mitral regurgitation benefit from vasodilator treatment. However, despite some encouraging data, translation to chronic treatment of organic disease is unresolved because reported series were small, rarely randomised, and contradictory in conclusions. Activation of the tissue (not systemic) renin-angiotensin myocardial system was shown in organic mitral regurgitation. Consistent pilot studies suggest potential of drugs blocking tissue renin-angiotensin system to stabilise organic disease severity and consequences. The effect of such treatments on clinical outcome remains to be shown. β blockade in organic mitral regurgitation has only been tested in animal models and remains conjectural. Conversely, in functional disease, medical treatment has been better studied than in organic disease. Maximum medical treatment of patients with heart failure and left-ventricular dysfunction reduces functional mitral regurgitation. Specifically β blockade—with carvedilol or long-acting metoprolol—and inhibition of angiotensin-converting enzyme reduce functional mitral regurgitation severity. These therapies are recommended for treatment of left-ventricular dysfunction. Thus, non-urgent surgical indications should be reviewed after maximum medical treatment has taken effect.

Interventional treatment is not yet approved for clinical use and remains investigational. Percutaneous revascularisation of patients with ischaemic regurgitation is possible but patients are often left with residual regurgitation that affects prognosis so that more effective treatment is necessary. Resynchronisation treatment in left-ventricular dysfunction with delayed conduction might improve functional mitral regurgitation. Two specific interventional approaches to treatment are discussed here.

Valvular edge-to-edge attachment mimics the surgical procedure proposed by Alfieri and colleagues, creating a tissue bridge between anterior and posterior leaflets. Percutaneously, this technique uses a clip or sutures deployed through trans-septal catheterisation. Experimental studies have shown success and reliable clip or suture placement through the trans-septal approach ([figure](#)). Early trials also suggest safety and feasibility with close echocardiographic guidance in centres with much experience of interventional valvular procedures. Data for how well this intervention works are preliminary but encouraging,¹²⁸ suggesting that more than 80% of patients can be discharged from hospital with a clip, and mild or little mitral regurgitation. A randomised trial comparing percutaneous clip and surgery is in progress. The edge-to-edge technique has important limitations. First, the application of this technique is

restricted to localised prolapse of the central segment of the anterior and posterior leaflets. Second, annular dilatation is not addressed by the procedure and might cause residual regurgitation.



Percutaneous devices used for treatment of mitral regurgitation

(A) Percutaneous clip introduced by venous and trans-septal approach into the left atrium and through the mitral orifice. The clip then grabs both leaflets, resuspending them with prolapse. (B) Percutaneous coronary sinus cinching device introduced through the jugular vein into the coronary sinus. The distal stent (smallest) then the proximal stent are deployed. With time the bridge shrinks and cinches the annulus.

Annuloplasty aimed at reduction of annular dilatation is under investigation mostly with coronary sinus cinching. Technically, stabilisation of material with sufficient constraining force to obtain more than 20% diameter reduction is a challenge. Most devices are composed of anchoring devices placed in the distal and proximal coronary sinus and an intermediate tensioning or supporting element. Experimentally, reduction of mitral regurgitation is achievable, but clinical results are preliminary. Feasibility through a jugular approach and safety seem to be acceptable. Potential limitations are those of annuloplasty (incomplete valve tenting correction) and those of coronary sinus approach that might reduce only part of the annular circumference with an effectiveness limited by the 1–2 cm sinus-annular distance. Because of safety concerns related to proximity of the coronary sinus and circumflex artery with potential artery compression, non-coronary sinus approaches to annuloplasty and

percutaneous ventricular remodelling-constraint devices are being investigated.

On the basis of the success of balloon valvuloplasty for mitral stenosis, percutaneous treatment of mitral regurgitation is expected to be successful but this success will necessitate complex development that needs strong cardiologist—engineer collaboration and rigorous assessment.

Surgical treatment of mitral regurgitation is the only approach with defined clinical success, providing sustained relief of symptoms or heart failure. However, no randomised trial has been done to prove mortality or cardiac event reduction. The standard surgical approach is a median sternotomy, but sometimes only partial sternotomy or minimally invasive surgery through thoracoscopic approach can be used.

Valve repair includes an array of valvular, subvalvular, and annular procedures aimed at restoration of leaflet coaptation (ie, valvular normal function) and elimination of mitral regurgitation. These surgical techniques are more successful with redundant than with retracted or calcified leaflets. For valve prolapse, typical repair is resection (triangular or quadrangular) of the prolapsed posterior leaflet segment whereas the anterior leaflet is rarely resected. Subvalvular support can be obtained by chordal transfer or artificial chords rather than chordal shortening. Annuloplasty is routinely used with annular bands or flexible or rigid rings. Many additional technical procedures might be used at the surgeon's discretion to restore coaptation and valve competence. Conversely, in functional mitral regurgitation, valve repair is rather uniform with restrictive annuloplasty substantially reducing the anteroposterior annular diameter. New rings aimed at annular reshaping, specific to each cause of functional regurgitation (ischaemic disease or cardiomyopathy) are now available but their incremental value (compared with traditional rings) is not defined. Valve repair is done in about half of patients who undergo surgery for mitral regurgitation in the USA and Europe. In centres with surgeons proficient in valve repair, more than 80—90% repair rates are achieved. A failed repair is caused rarely by systolic anterior motion of the mitral valve due to excessively redundant tissue or by stenosis, but more often by insufficient correction of a prolapse, recurrence of ruptured chords, and excessive tissue retraction or resection. Overall, reoperation after 10 years is necessary in 5% of patients with repaired posterior leaflet prolapse and 10% of those with anterior leaflet interventions. 20—30% of patients with repaired functional mitral regurgitation are estimated to have recurrent regurgitation. Reoperation rate is not greater after valve repair than after replacement and because of the morbidity and mortality advantages, valve repair is the preferred method of surgical correction of mitral regurgitation.

Valve replacement involves insertion of a biological or mechanical prosthesis. Bioprosthetic valve replacement is associated with low embolic risk but shorter durability, whereas

mechanical valve replacement is associated with high risk of embolism and haemorrhagic complications (due to intensive warfarin treatment) but has potential for long-lasting durability. Results of randomised trials showed that within 10 years of surgery these risks are balanced. Older age determines the probability that bioprosthetic durability will be longer than life expectancy, and is the main bioprosthesis insertion indication (usually >65 years of age). Ability to achieve high-quality anticoagulation and patient's desire also affect the choice of prosthesis. Irrespective of the prosthesis selected, conservation of subvalvular apparatus is essential for preservation of ventricular function. The risk of prosthetic complications makes surgical indications more restrictive when valve replacement is likely.

Controversies and guidelines for treatment

In view of the experimental nature of medical and interventional treatments for mitral regurgitation, surgery is the only treatment recommended by management guidelines. Because surgery is associated with small but definite risks, those patients with a higher risk of spontaneous complications than of surgery-related complications are selected. Guidelines should, in our opinion, be interpreted as a minimum to be applied by all physicians but should not deter centres with better results than those of other centres from providing advanced care to patients with mitral regurgitation. Furthermore, the absence of clinical trials and few prospective studies create ample controversy, which should be addressed in future studies.

Although approaches to surgical indications are detailed in clinical guidelines, they are summarised here. Rescue surgical indications—class I by guidelines—are compulsory. Patients with organic mitral regurgitation who have developed severe symptoms (class III or IV), heart failure, or signs of overt left-ventricular dysfunction (ejection fraction <60% or end-systolic dimension ≥ 40 –45 mm) have an immediate high risk and therefore prompt surgery—repair (preferable) or replacement—is indicated. Even with advanced heart failure or ventricular dysfunction, contraindications to surgery are rare as long as mitral regurgitation remains severe, emphasising the importance of quantitative assessment of disease. Such rescue surgery is indispensable, but is not the preferred timing for surgery in organic disease. Indeed, patients who need to be operated on at such a late stage of their disease have increased mortality after surgery. This outcome emphasises the importance of early detection and assessment of mitral regurgitation. In functional regurgitation, rescue surgery is the most frequent surgical indication, but consideration should be given to surgery in symptomatic patients before heart failure becomes intractable.

Restorative surgical indications—class II by guidelines—are optional. Patients with no or minimum symptoms at baseline cannot expect substantial symptomatic improvement. Those with functional mitral regurgitation are rarely candidates for restorative surgery while asymptomatic but might be suitable for valve repair if coronary artery bypass grafting is necessary independently of the mitral regurgitation. In organic regurgitation, postoperative outcome studies in patients with no or minimum symptoms before surgery show restoration of life expectancy, emphasising the importance of this approach. Patients who are asymptomatic but had either reduced functional capacity by objective exercise testing, hormonal activation, or paroxysmal atrial fibrillation are specific but not exclusive candidates for restorative surgery.

Mitral stenosis

Etiology and Pathology

Rheumatic fever is the leading cause of mitral stenosis (MS) (Table). Other less common etiologies of obstruction to left atrial outflow include congenital mitral valve stenosis, cor triatriatum, mitral annular calcification with extension onto the leaflets, systemic lupus erythematosus, rheumatoid arthritis, left atrial myxoma, and infective endocarditis with large

vegetations.

Mitral Stenosis

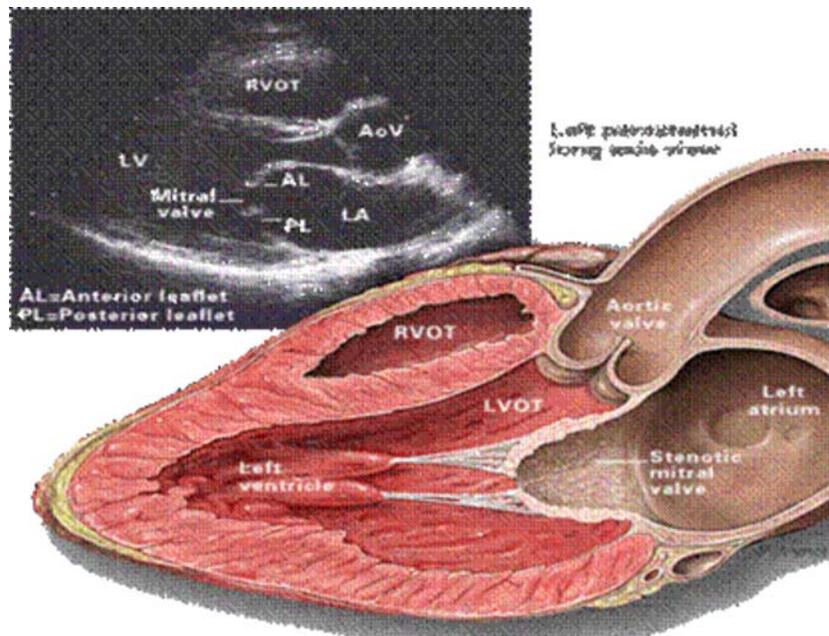
Etiologies

- **Rheumatic** – almost all cases in adults
- **Mitral Annular Ca+ - massive (rare)**
- **Congenital** – rare

60% of pts don't have a history of ARC
50% of pts who have ARC don't develop VHD

Pure or predominant MS occurs in approximately 40% of all patients with rheumatic heart disease and a history of rheumatic fever. In other patients with rheumatic heart disease, lesser degrees of MS may accompany mitral regurgitation (MR) and aortic valve disease. With reductions in the incidence of acute rheumatic fever, particularly in temperate climates and developed countries, the incidence of MS has declined considerably over the past few decades. However, it remains a major problem in developing nations, especially in tropical and

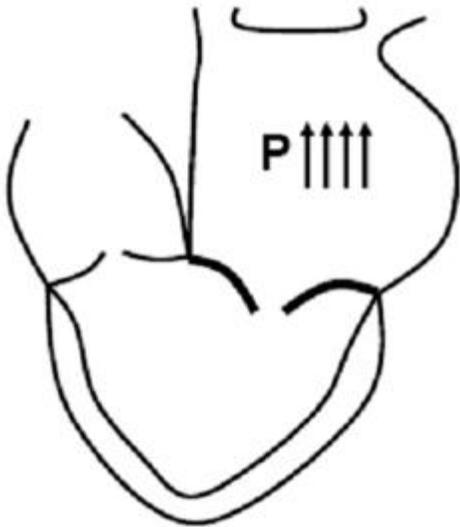
semiotropical climates.



In rheumatic MS, the valve leaflets are diffusely thickened by fibrous tissue and/or calcific deposits. The mitral commissures fuse, the chordae tendineae fuse and shorten, the valvular cusps become rigid, and these changes, in turn, lead to narrowing at the apex of the funnel-shaped ("fish-mouth") valve. Although the initial insult to the mitral valve is rheumatic, the later changes may be a nonspecific process resulting from trauma to the valve caused by altered flow patterns due to the initial deformity. Calcification of the stenotic mitral valve immobilizes the leaflets and narrows the orifice further. Thrombus formation and arterial embolization may arise from the calcific valve itself, but in patients with atrial fibrillation (AF), thrombi arise more frequently from the dilated left atrium (LA), particularly the left atrial appendage.

Pathophysiology

In normal adults, the area of the mitral valve orifice is 4–6 cm². In the presence of significant obstruction, i.e., when the orifice area is reduced to < ~2 cm², blood can flow from the LA to the left ventricle (LV) only if propelled by an abnormally elevated left atrioventricular pressure gradient, the hemodynamic hallmark of MS.



Normal MV area = 4-6cm²
Symptoms begin = < 2cm²
Critical MS = < 1cm²

When the mitral valve opening is reduced to $<1 \text{ cm}^2$, often referred to as "severe" MS, a LA pressure of $\sim 25 \text{ mmHg}$ is required to maintain a normal cardiac output (CO). The elevated pulmonary venous and pulmonary arterial (PA) wedge pressures reduce pulmonary compliance, contributing to exertional dyspnea. The first bouts of dyspnea are usually precipitated by clinical events that increase the rate of blood flow across the mitral orifice, resulting in further elevation of the LA pressure (see below).

Symptoms of Mitral Stenosis

- **Dyspnea**
 - Pulmonary venous congestion
- **Fatigue**
 - Diminished cardiac output
- **Inability to tolerate increased volume**
- **Inability to tolerate increased HR**
 - Decreased filling
 - Increased LA pressure/PV congestion
- **Hemoptysis**

Pulmonary Hypertension

The clinical and hemodynamic features of MS are influenced importantly by the level of the PAP. Pulmonary hypertension results from: (1) passive backward transmission of the elevated LA pressure; (2) pulmonary arteriolar constriction, which presumably is triggered by LA and pulmonary venous hypertension (reactive pulmonary hypertension); (3) interstitial edema in the walls of the small pulmonary vessels; and (4) organic obliterative changes in the pulmonary vascular bed. Severe pulmonary hypertension results in RV enlargement, secondary tricuspid regurgitation (TR) and pulmonic regurgitation (PR), as well as right-sided heart failure.

Symptoms

In temperate climates, the latent period between the initial attack of rheumatic carditis (in the increasingly rare circumstances in which a history of one can be elicited) and the development of symptoms due to MS is generally about two decades; most patients begin to experience disability in the fourth decade of life. Studies carried out before the development of mitral valvotomy revealed that once a patient with MS became seriously symptomatic, the disease progressed continuously to death within 2–5 years.

In patients whose mitral orifices are large enough to accommodate a normal blood flow with only mild elevations of LA pressure, marked elevations of this pressure leading to dyspnea and cough may be precipitated by sudden changes in the heart rate, volume status, or CO, as for

example with severe exertion, excitement, fever, severe anemia, paroxysmal AF and other tachycardias, sexual intercourse, pregnancy, and thyrotoxicosis. As MS progresses, lesser stresses precipitate dyspnea, and the patient becomes limited in daily activities, and orthopnea and paroxysmal nocturnal dyspnea develop. The development of permanent AF often marks a turning point in the patient's course and is generally associated with acceleration of the rate at which symptoms progress.

Pulmonary Changes

In addition to the aforementioned changes in the pulmonary vascular bed, fibrous thickening of the walls of the alveoli and pulmonary capillaries occurs commonly in MS. The vital capacity, total lung capacity, maximal breathing capacity, and [oxygen](#) uptake per unit of ventilation are reduced. Pulmonary compliance falls further as pulmonary capillary pressure rises during exercise.

Physical Findings

Inspection and Palpation

In patients with severe MS, there may be a malar flush with pinched and blue facies. In patients with sinus rhythm and severe pulmonary hypertension or associated tricuspid stenosis (TS), the jugular venous pulse reveals prominent *a* waves due to vigorous right atrial systole. The systemic arterial pressure is usually normal or slightly low. An RV tap along the left sternal border signifies an enlarged RV. A diastolic thrill may be present at the cardiac apex, with the patient in the left lateral recumbent position.

Auscultation

The first heart sound (S_1) is usually accentuated and slightly delayed. The pulmonic component of the second heart sound (P_2) also is often accentuated, and the two components of the second heart sound (S_2) are closely split. The opening snap (OS) of the mitral valve is most readily audible in expiration at, or just medial to the cardiac apex. This sound generally follows the sound of aortic valve closure (A_2) by 0.05–0.12 s. The time interval between A_2 and OS varies inversely with the severity of the MS. The OS is followed by a low-pitched, rumbling, diastolic murmur, heard best at the apex with the patient in the left lateral recumbent position. It is accentuated by mild exercise (e.g., a few rapid sit-ups) carried out just before auscultation. In general, the duration of this murmur correlates with the severity of the stenosis

in patients with preserved CO. In patients with sinus rhythm, the murmur often reappears or becomes louder during atrial systole (presystolic accentuation). Soft grade I or II/VI systolic murmurs are commonly heard at the apex or along the left sternal border in patients with pure MS and do not necessarily signify the presence of MR. Hepatomegaly, ankle edema, ascites, and pleural effusion, particularly in the right pleural cavity, may occur in patients with MS and RV failure.

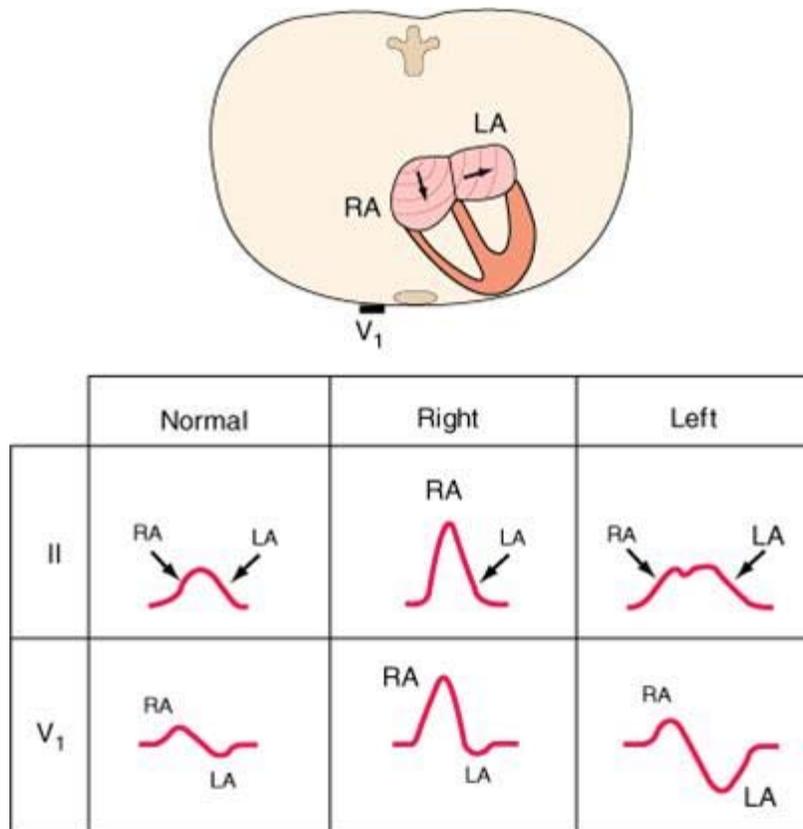
Associated Lesions

With severe pulmonary hypertension, a pansystolic murmur produced by functional TR may be audible along the left sternal border. This murmur is usually louder during inspiration and diminishes during forced expiration (Carvallo's sign). When the CO is markedly reduced in MS, the typical auscultatory findings, including the diastolic rumbling murmur, may not be detectable (silent MS), but they may reappear as compensation is restored. The *Graham Steell murmur* of PR, a high-pitched, diastolic, decrescendo blowing murmur along the left sternal border, results from dilatation of the pulmonary valve ring and occurs in patients with mitral valve disease and severe pulmonary hypertension. This murmur may be indistinguishable from the more common murmur produced by aortic regurgitation (AR), though it may increase in intensity with inspiration and is accompanied by a loud P2.

Laboratory Examination

ECG

In MS and sinus rhythm, the P wave usually suggests LA enlargement. It may become tall and peaked in lead II and upright in lead V₁ when severe pulmonary hypertension or TS complicates MS and right atrial (RA) enlargement occurs. The QRS complex is usually normal. However, with severe pulmonary hypertension, right axis deviation and RV hypertrophy are often present.



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J. *Harrison's Principles of Internal Medicine*, 17th Edition: <http://www.accessmedicine.com>

Echocardiogram

Transthoracic two-dimensional echocardiography (TTE) with color flow Doppler imaging provides critical information, including an estimate of the transvalvular peak and mean gradients and of mitral orifice size, the presence and severity of accompanying MR, the extent of restriction of valve leaflets and their thickness, the degree of distortion of the subvalvular apparatus, and the anatomic suitability for percutaneous mitral balloon valvotomy (PMBV; see below). In addition, TTE provides an assessment of the size of the cardiac chambers, an estimation of LV function, an estimation of the pulmonary artery pressure (PAP), and an indication of the presence and severity of associated valvular lesions. Transesophageal echocardiography (TEE) provides superior images and should be employed when TTE is inadequate for guiding therapy. TEE is especially indicated to exclude the presence of left atrial thrombi prior to PMBV.

Chest X-Ray

The earliest changes are straightening of the upper left border of the cardiac silhouette,

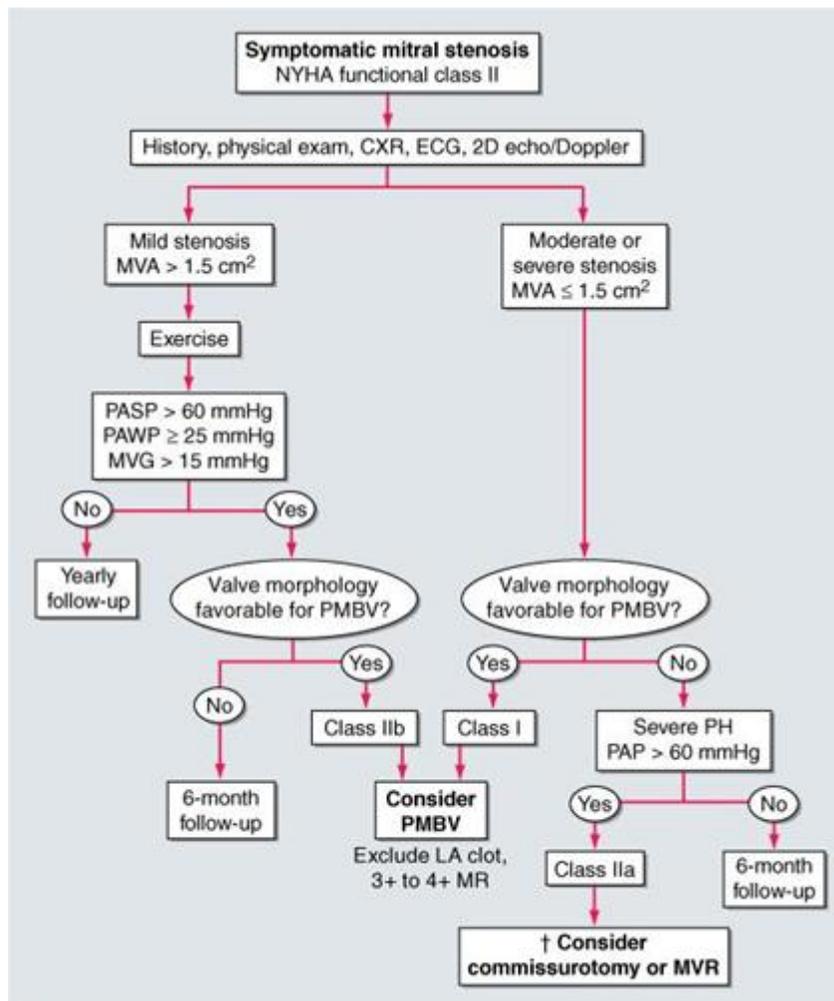
prominence of the main pulmonary arteries, dilatation of the upper lobe pulmonary veins, and posterior displacement of the esophagus by an enlarged LA. Kerley B lines are fine, dense, opaque, horizontal lines that are most prominent in the lower and mid-lung fields and that result from distention of interlobular septae and lymphatics with edema when the resting mean LA pressure exceeds approximately 20 mmHg.

Differential Diagnosis

Like MS, significant MR may also be associated with a prominent diastolic murmur at the apex due to increased flow, but in MR this diastolic murmur commences slightly later than in patients with MS, and there is often clear-cut evidence of LV enlargement. An apical pansystolic murmur of at least grade III/VI intensity as well as an S₃ suggests significant associated MR. Similarly, the apical mid-diastolic murmur associated with severe AR (*Austin Flint murmur*) may be mistaken for MS but can be differentiated from it because it is not intensified in presystole. TS, which occurs rarely in the absence of MS, may mask many of the clinical features of MS or be clinically silent.

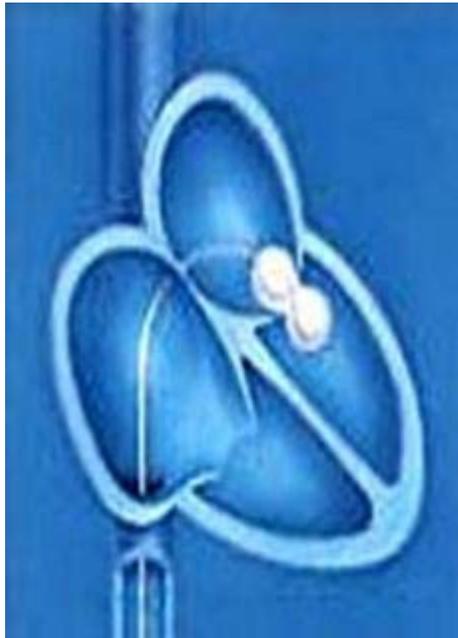
Treatment

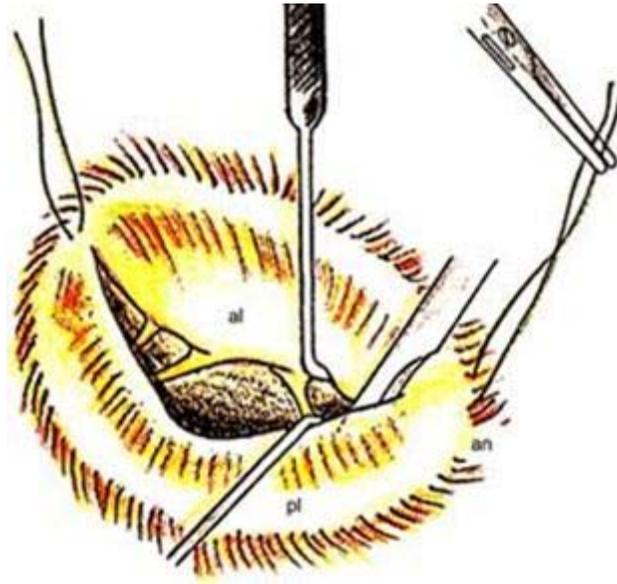
Penicillin prophylaxis of Group A β -hemolytic streptococcal infections to prevent rheumatic fever is important for at-risk patients with MS. Recommendations for infective endocarditis prophylaxis have recently changed. In symptomatic patients, some improvement usually occurs with restriction of sodium intake and maintenance doses of oral diuretics. Digitalis glycosides usually do not benefit patients with MS and sinus rhythm, but they are helpful in slowing the ventricular rate of patients with AF. Beta blockers and nondihydropyridine calcium channel blockers (e.g., [verapamil](#) or [diltiazem](#)) are also useful in this regard.



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: <http://www.accessmedicine.com>

[Warfarin](#) to an international normalized ration (INR) of 2–3 should be administered indefinitely to patients with MS who have AF or a history of thromboembolism. The routine use of warfarin in patients in sinus rhythm with LA enlargement (maximal dimension >5.5 cm) with or without spontaneous echo contrast is more controversial.





Management strategy for patients with mitral stenosis (MS) and mild symptoms. There is controversy as to whether patients with severe MS (MVA $<1.0 \text{ cm}^2$) and severe pulmonary hypertension (PH) (PASP $>60 \text{ mmHg}$) should undergo percutaneous mitral balloon valvotomy (PMBV) or mitral valve replacement (MVR) to prevent right ventricular failure. CXR, chest x-ray; ECG, electrocardiogram; echo, echocardiography; LA, left atrial; MR, mitral regurgitation; MVA, mitral valve area; MVG, mean mitral valve pressure gradient; NYHA, New York Heart Association; PASP, pulmonary artery systolic pressure; PAWP, pulmonary artery wedge pressure; 2D, 2-dimensional. (*From Bonow et al.*)

Medical Therapy of Valvular Heart Disease

Lesion	Symptom Control	Natural History
Mitral stenosis	Beta blockers, nondihydropyridine calcium channel blockers, or <u>digoxin</u> for rate control of AF; cardioversion for new-onset AF and HF; diuretics for HF	Warfarin for AF or thromboembolism; PCN for RF prophylaxis
Mitral regurgitation	Diuretics for HF Vasodilators for acute MR	<u>Warfarin</u> for AF or thromboembolism Vasodilators for HTN
Aortic stenosis	Diuretics for HF	No proven therapy
Aortic regurgitation	Diuretics and vasodilators for HF	Vasodilators for HTN

Note: Antibiotic prophylaxis is recommended according to current American Heart Association guidelines. For patients with these forms of valvular heart disease, prophylaxis is indicated for a prior history of endocarditis. HF is an indication for surgical or percutaneous treatment, and the recommendations here pertain to short-term therapy prior to definitive correction of the valve lesion. For patients whose comorbidities prohibit surgery, the medical therapies listed can be continued according to available guidelines for the management of HF. See text.

Abbreviations: AF, atrial fibrillation; HF, heart failure; HTN, systemic hypertension; PCN, penicillin; RF, rheumatic fever.

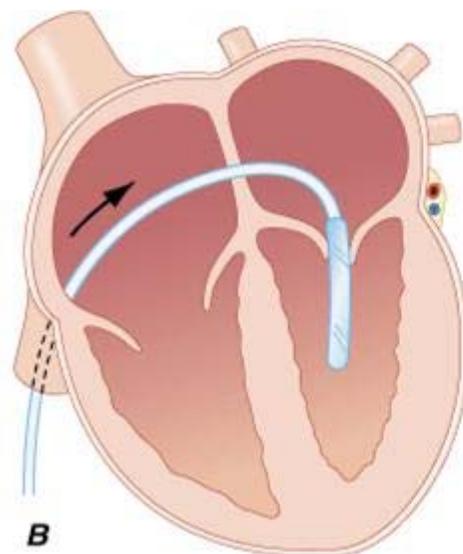
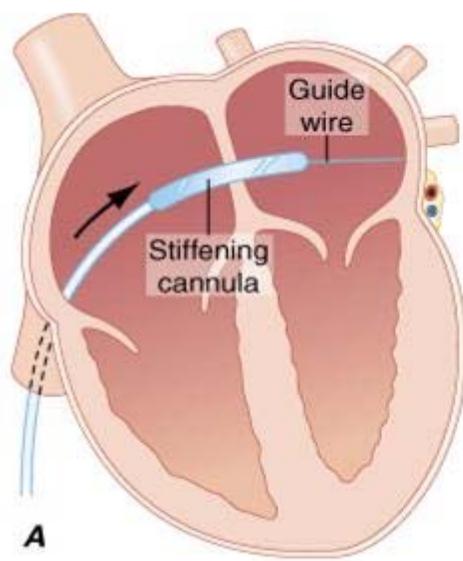
Source: Adapted from NA Boon, P Bloomfield: The medical management of valvular heart disease. Heart 87:395, 2002

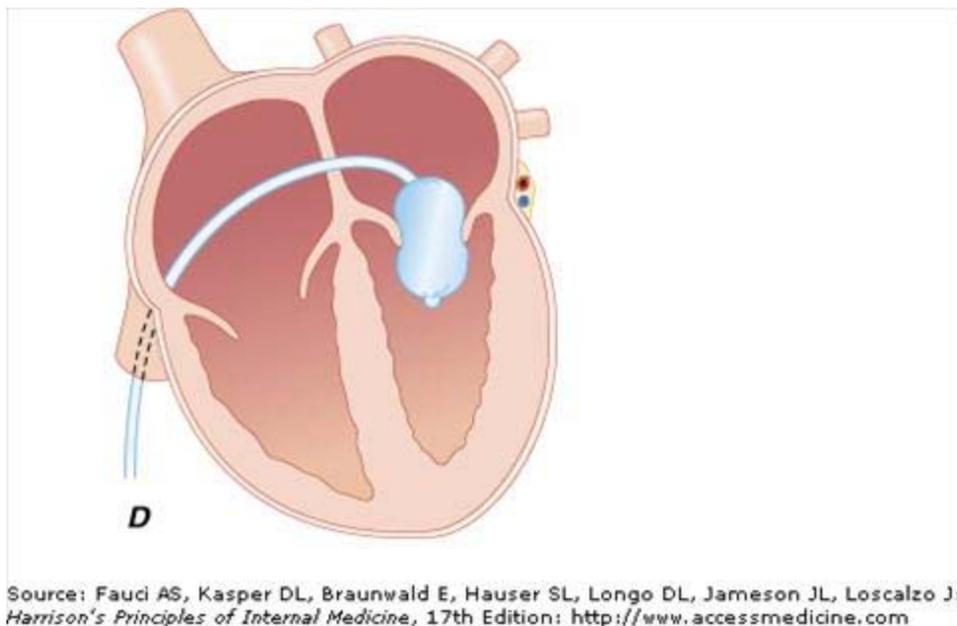
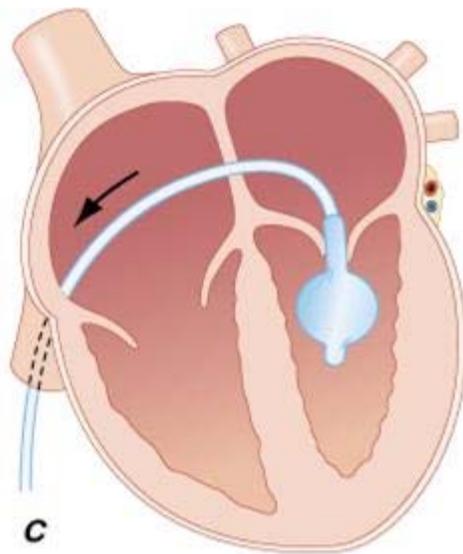
If AF is of relatively recent onset in a patient whose MS is not severe enough to warrant PMBV or surgical commissurotomy, reversion to sinus rhythm pharmacologically or by means of electrical countershock is indicated. Usually, cardioversion should be undertaken after the patient has had at least 3 consecutive weeks of [anticoagulant](#) treatment to a therapeutic INR. If cardioversion is indicated more urgently, then intravenous [heparin](#) should be provided and a TEE performed to exclude the presence of left atrial thrombus before the procedure. Conversion to sinus rhythm is rarely successful or sustained in patients with severe MS, particularly those in whom the LA is especially enlarged or in whom AF has been present for more than 1 year.

Mitral Valvotomy

Unless there is a contraindication, mitral valvotomy is indicated in symptomatic [New York Heart Association (NYHA) Functional Class II–IV] patients with isolated MS whose effective orifice (valve area) is $< \sim 1.0 \text{ cm}^2/\text{m}^2$ body surface area, or $< 1.5 \text{ cm}^2$ in normal-sized adults. Mitral valvotomy can be carried out by two techniques: PMBV and surgical valvotomy. In PMBV a catheter is directed into the LA after transseptal puncture, and a single balloon is directed across the valve and inflated in the valvular orifice. Ideal patients have relatively pliable leaflets with little or no commissural calcium. In addition, the subvalvular structures should not be significantly scarred or thickened and there should be no left atrial thrombus. The short- and long-term results of this procedure in appropriate patients are similar to those of surgical valvotomy, but with less morbidity and a lower periprocedural mortality rate. Event-free survival in younger (< 45 years) patients with pliable valves is excellent, with rates

as high as 80–90% over 3–7 years. Therefore, PMBV has become the procedure of choice for such patients when it can be performed by a skilled operator in a high-volume center.





Inoue balloon technique for mitral balloon valvotomy. **A.** After transseptal puncture, the deflated balloon catheter is advanced across the inter-atrial septum, then across the mitral valve and into the left ventricle. **B-D.** The balloon is then inflated stepwise within the mitral orifice.

Successful valvotomy is defined by a 50% reduction in the mean mitral valve gradient and a doubling of the mitral valve area. Successful valvotomy, whether balloon or surgical, usually results in striking symptomatic and hemodynamic improvement and prolongs survival. However, there is no evidence that the procedure improves the prognosis of patients with slight

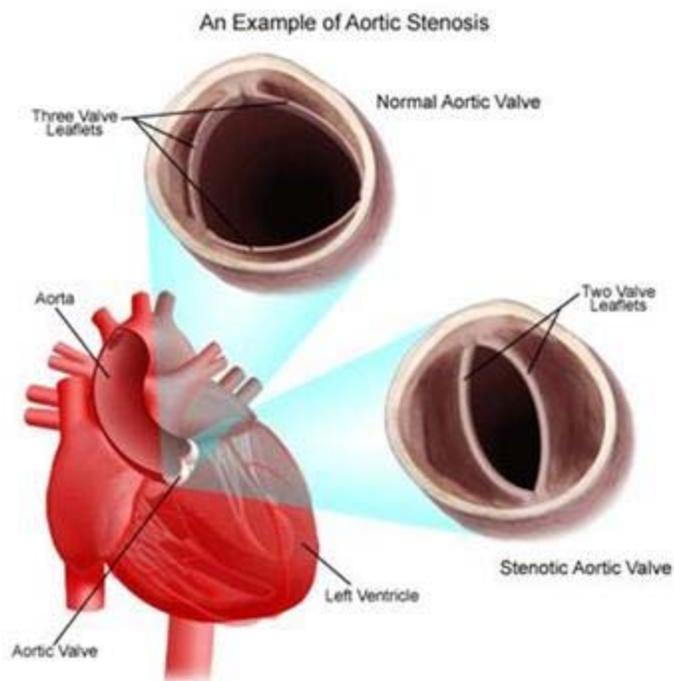
or no functional impairment. Therefore, unless recurrent systemic embolization or severe pulmonary hypertension has occurred (PA systolic pressures >50 mmHg at rest or >60 mmHg with exercise), valvotomy is *not* recommended for patients who are entirely asymptomatic and/or who have mild stenosis (mitral valve area $>1/5$ cm²). When there is little symptomatic improvement after valvotomy, it is likely that the procedure was ineffective, that it induced MR, or that associated valvular or myocardial disease was present. About half of all patients undergoing surgical mitral valvotomy require reoperation by 10 years. In the pregnant patient with MS, valvotomy should be carried out if pulmonary congestion occurs despite intensive medical treatment. PMBV is the preferred strategy in this setting and is performed with TEE and no or minimal x-ray exposure.

Mitral valve replacement (MVR) is necessary in patients with MS and significant associated MR, those in whom the valve has been severely distorted by previous transcatheter or operative manipulation, or those in whom the surgeon does not find it possible to improve valve function significantly. MVR is now routinely performed with preservation of the chordal attachments to optimize LV functional recovery. Perioperative mortality rates with MVR vary with age, LV function, the presence of CAD, and associated comorbidities. They average 5% overall but are lower in young patients and may be twice as high in older patients with comorbidities. Since there are also long-term complications of valve replacement, patients in whom preoperative evaluation suggests the possibility that MVR may be required should be operated on only if they have severe MS—i.e., an orifice area <1 cm²—and are in NYHA Class III, i.e., symptomatic with ordinary activity despite optimal medical therapy. The overall 10-year survival of surgical survivors is $\sim 70\%$. Long-term prognosis is worse in older patients and those with marked disability and marked depression of the CO preoperatively. Pulmonary hypertension and RV dysfunction are additional risk factors for poor outcome.

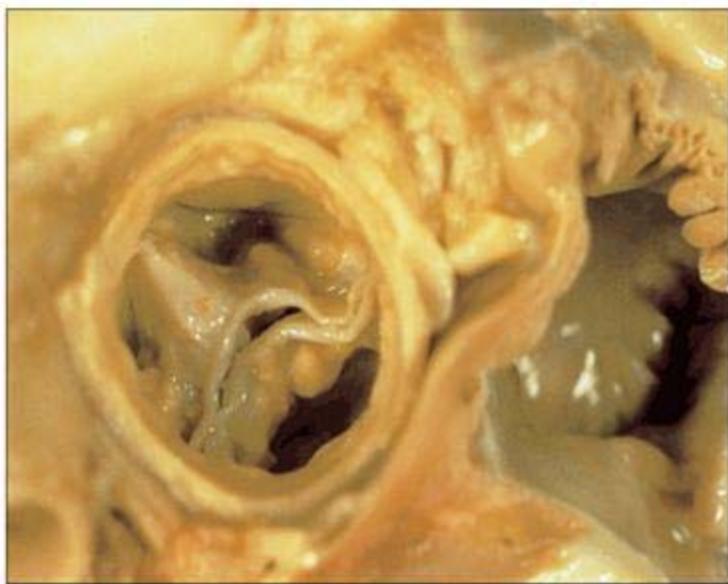
Aortic stenosis

Aortic stenosis (AS) most often is due to calcification of a congenitally bicuspid or normal trileaflet valve. Calcific changes are due to an active disease process characterized by lipid accumulation, inflammation, and calcification. Once initiated, progressive leaflet calcification and fibrosis eventually result in reduced leaflet motion with obstruction to left ventricular (LV) outflow. **Aortic stenosis** often is first diagnosed by the finding of a murmur on auscultation.

However, while a soft murmur with a physiologic split S2 reliably excludes severe **stenosis** and a grade 4 murmur with diminished carotid upstrokes confirms severe obstruction, between these extremes physical examination is not accurate for evaluation of disease severity.

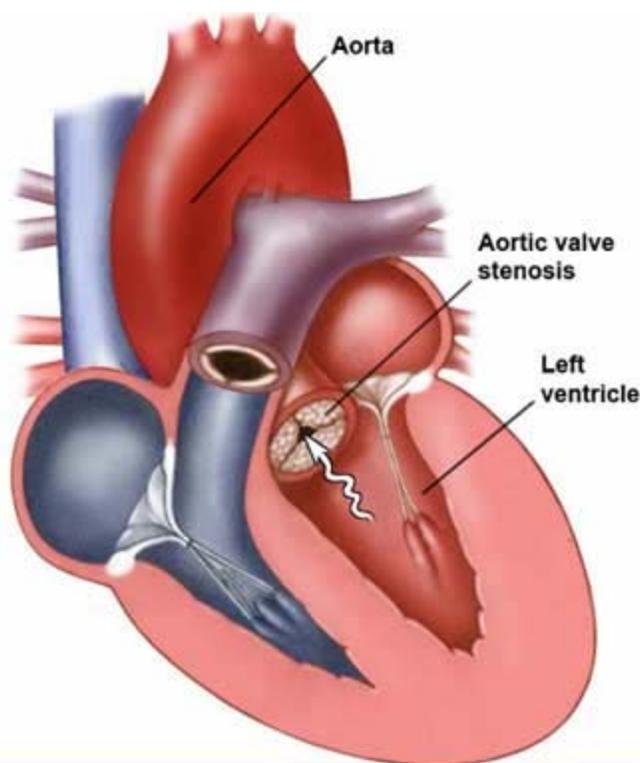


Bicuspid Aortic Valve Stenosis



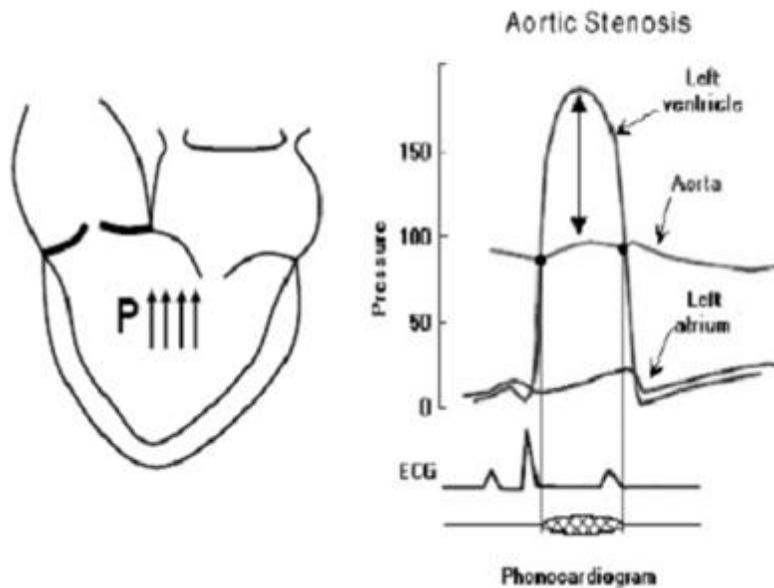
AS occurs in about one-fourth of all patients with chronic valvular heart disease; approximately 80% of adult patients with symptomatic valvular AS are male.

AS in adults may be due to degenerative calcification of the aortic cusps. It may be congenital in origin or it may be secondary to rheumatic inflammation. *Age-related degenerative calcific AS* (also known as senile or sclerocalcific AS) is now the most common cause of AS in adults in North America and Western Europe. About 30% of persons >65 years exhibit aortic valve sclerosis; many of these have a systolic murmur of AS but without obstruction, while 2% exhibit frank stenosis. Aortic sclerosis is defined echocardiographically as focal thickening or calcification of the valve cusps with a peak Doppler transaortic velocity of 2.5 m/s. Aortic sclerosis appears to be a marker for an increased risk of coronary heart disease events. On histologic examination these valves frequently exhibit changes similar to those seen with atherosclerosis and vascular inflammation. Interestingly, risk factors for atherosclerosis, such as age, male sex, smoking, diabetes mellitus, hypertension, chronic kidney disease, increased LDL, reduced HDL cholesterol, and elevated C-reactive protein are all risk factors for aortic valve calcification.



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Pathophysiology



Ventricular Compensation

- **Concentric hypertrophy**
 - Reduces wall stress
 - Reduces ventricular compliance
 - LVEDp increases
 - LAp increases

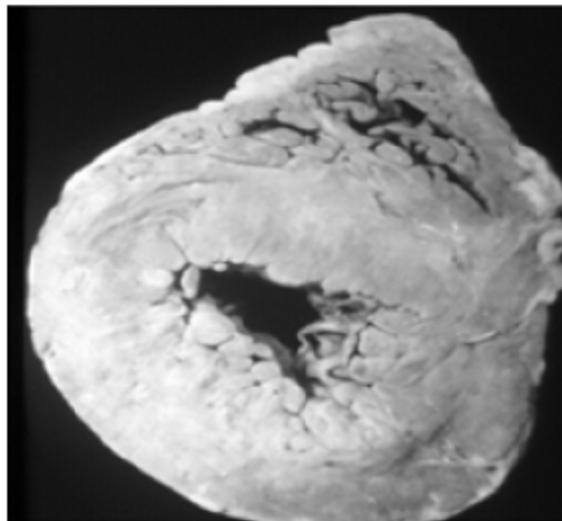


Fig. Aortic stenosis

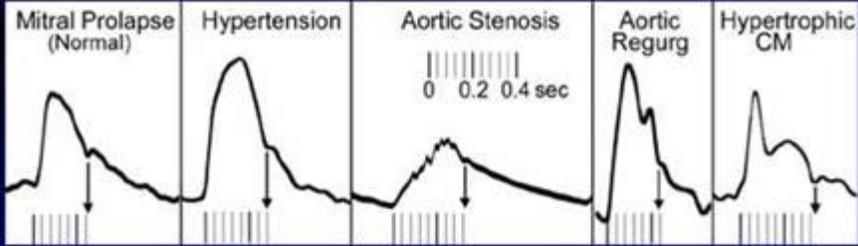
The *congenitally affected valve* may be stenotic at birth and may become progressively more fibrotic, calcified, and stenotic. In other cases the valve may be congenitally deformed, usually bicuspid [bicuspid aortic valve (BAV)], without serious narrowing of the aortic orifice during childhood; its abnormal architecture makes its leaflets susceptible to otherwise ordinary

hemodynamic stresses, which ultimately lead to valvular thickening, calcification, increased rigidity, and narrowing of the aortic orifice.

Aortic Stenosis

Physical Exam

- **Harsh Systolic Ejection Murmur – late peaking**
- **S4 gallop (from LVH)**
- **Sustained Bifid LV impulse (from LVH)**
- **Pulsus Parvus et Tardus (Carotid Impulse)**



The diagram shows five panels of pulse waveforms: Mitral Prolapse (Normal), Hypertension, Aortic Stenosis, Aortic Regurg, and Hypertrophic CM. A scale bar indicates 0, 0.2, and 0.4 seconds. The Aortic Stenosis waveform shows a low-amplitude, late-peaking systolic pulse.

Rheumatic disease of the aortic leaflets produces commissural fusion, sometimes resulting in a bicuspid-appearing valve. This condition in turn makes the leaflets more susceptible to trauma and ultimately leads to fibrosis, calcification, and further narrowing. By the time the obstruction to LV outflow causes serious clinical disability, the valve is usually a rigid calcified mass, and careful examination may make it difficult or even impossible to determine the etiology of the underlying process. Rheumatic AS is almost always associated with involvement of the mitral valve and with AR.

Pathophysiology

The obstruction to LV outflow produces a systolic pressure gradient between the LV and aorta. When severe obstruction is suddenly produced experimentally, the LV responds by dilatation and reduction of stroke volume. However, in some patients the obstruction may be present at birth and/or increase gradually over the course of many years, and LV output is maintained by the presence of concentric LV hypertrophy. Initially, this serves as an adaptive mechanism because it reduces toward normal the systolic stress developed by the myocardium, as predicted by the Laplace relation ($S = Pr/h$, where S = systolic wall stress, P = pressure, r = radius, and h = wall thickness). A large transaortic valvular pressure gradient may exist for many years

without a reduction in CO or LV dilatation; ultimately, however, excessive hypertrophy becomes maladaptive, and LV function declines.

A mean systolic pressure gradient >40 mmHg with a normal CO or an effective aortic orifice area $< \sim 1.0 \text{ cm}^2$ (or $\sim < 0.6 \text{ cm}^2/\text{m}^2$ body surface area in a normal-sized adult)—i.e., less than approximately one-third of the normal orifice—is generally considered to represent severe obstruction to LV outflow. The elevated LV end-diastolic pressure observed in many patients with severe AS signifies the presence of LV dilatation and/or diminished compliance of the hypertrophied LV wall. Although the CO at rest is within normal limits in most patients with severe AS, it usually fails to rise normally during exercise. Loss of an appropriately timed, vigorous atrial contraction, as occurs in AF or atrioventricular dissociation, may cause rapid progression of symptoms. Late in the course, the CO and LV–aortic pressure gradient decline, and the mean LA, PA, and RV pressures rise.

The hypertrophied LV elevates myocardial [oxygen](#) requirements. In addition, even in the absence of obstructive CAD, there may be interference with coronary blood flow. This is because the pressure compressing the coronary arteries exceeds the coronary perfusion pressure, often causing ischemia (especially in the subendocardium), both in the presence and in the absence of coronary arterial narrowing.

Symptoms

AS is rarely of clinical importance until the valve orifice has narrowed to approximately 1.0 cm^2 . Even severe AS may exist for many years without producing any symptoms because of the ability of the hypertrophied LV to generate the elevated intraventricular pressures required for a normal stroke volume.

Most patients with pure or predominant AS have gradually increasing obstruction for years but do not become symptomatic until the sixth to eighth decades. Exertional dyspnea, angina pectoris, and syncope are the three cardinal symptoms. Often there is a history of insidious progression of fatigue and dyspnea associated with gradual curtailment of activities. *Dyspnea* results primarily from elevation of the pulmonary capillary pressure caused by elevations of LV diastolic pressures secondary to reduced left ventricular compliance. *Angina pectoris* usually develops somewhat later and reflects an imbalance between the augmented myocardial [oxygen](#) requirements and reduced oxygen availability; the former results from the increased myocardial mass and intraventricular pressure, while the latter may result from accompanying CAD, which

is not uncommon in patients with AS, as well as from compression of the coronary vessels by the hypertrophied myocardium. Therefore, angina may occur in severe AS even without obstructive epicardial CAD. *Exertional syncope* may result from a decline in arterial pressure caused by vasodilatation in the exercising muscles and inadequate vasoconstriction in nonexercising muscles in the face of a fixed CO, or from a sudden fall in CO produced by an arrhythmia.

Since the CO at rest is usually well maintained until late in the course, marked fatigability, weakness, peripheral cyanosis, cachexia, and other clinical manifestations of a low CO are usually not prominent until this stage is reached. Orthopnea, paroxysmal nocturnal dyspnea, and pulmonary edema, i.e., symptoms of LV failure, also occur only in the advanced stages of the disease. Severe pulmonary hypertension leading to RV failure and systemic venous hypertension, hepatomegaly, AF, and TR are usually late findings in patients with isolated severe AS.

When AS and MS coexist, the reduction in CO induced by MS lowers the pressure gradient across the aortic valve and thereby masks many of the clinical findings produced by AS.

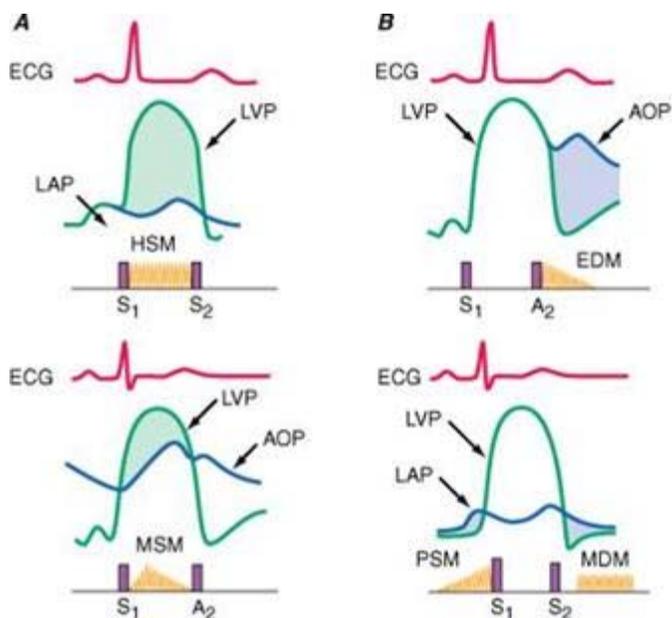
Physical Findings

The rhythm is generally regular until late in the course; at other times, AF should suggest the possibility of associated mitral valve disease. The systemic arterial pressure is usually within normal limits. In the late stages, however, when stroke volume declines, the systolic pressure may fall and the pulse pressure narrow. The peripheral arterial pulse rises slowly to a delayed sustained peak (*pulsus parvus et tardus*). In the elderly, the stiffening of the arterial wall may mask this important physical sign. In many patients the *a* wave in the jugular venous pulse is accentuated. This results from the diminished distensibility of the RV cavity caused by the bulging, hypertrophied interventricular septum.

Auscultation

An early systolic ejection sound is frequently audible in children and adolescents with congenital noncalcific valvular AS. This sound usually disappears when the valve becomes calcified and rigid. As AS increases in severity, LV systole may become prolonged so that the aortic valve closure sound no longer precedes the pulmonic valve closure sound, and the two components may become synchronous, or aortic valve closure may even follow pulmonic valve

closure, causing paradoxical splitting of S₂. The sound of aortic valve closure can be heard most frequently in patients with AS who have pliable valves, and calcification diminishes the intensity of this sound. Frequently, an S₄ is audible at the apex and reflects the presence of LV hypertrophy and an elevated LV end-diastolic pressure; an S₃ generally occurs late in the course, when the LV dilates.



A. Schematic representation of ECG, aortic pressure (AOP), left ventricular pressure (LVP), and left atrial pressure (LAP). The shaded areas indicate a transvalvular pressure difference during systole. HSM, holosystolic murmur; MSM, midsystolic murmur. **B.** Graphic representation of ECG, aortic pressure (AOP), left ventricular pressure (LVP), and left atrial pressure (LAP) with shaded areas indicating transvalvular diastolic pressure difference. EDM, early diastolic murmur; PSM, presystolic murmur; MDM, middiastolic murmur.

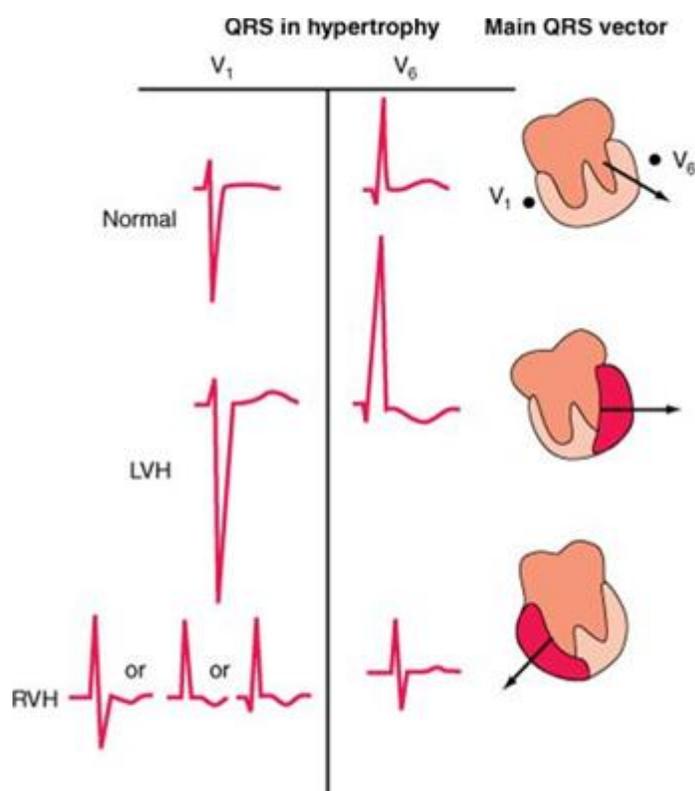
The murmur of AS is characteristically an ejection (mid) systolic murmur that commences shortly after the S₁, increases in intensity to reach a peak toward the middle of ejection, and ends just before aortic valve closure. It is characteristically low-pitched, rough and rasping in character, and loudest at the base of the heart, most commonly in the second right intercostal space. It is transmitted upward along the carotid arteries. Occasionally it is transmitted downward and to the apex, where it may be confused with the systolic murmur of MR (Gallavardin effect). In almost all patients with severe obstruction and preserved CO, the murmur is at least grade III/VI. In patients with mild degrees of obstruction or in those with

severe stenosis with heart failure in whom the stroke volume and therefore the transvalvular flow rate are reduced, the murmur may be relatively soft and brief.

Laboratory Examination

ECG

In most patients with severe AS there is LV hypertrophy.

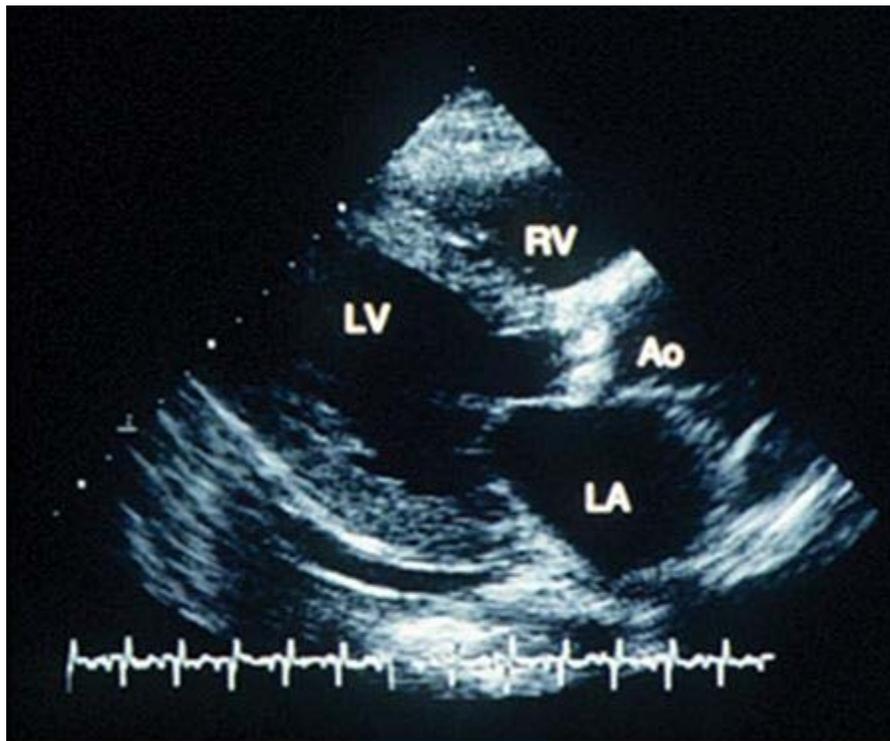


Left ventricular hypertrophy (LVH) increases the amplitude of electrical forces directed to the left and posteriorly.

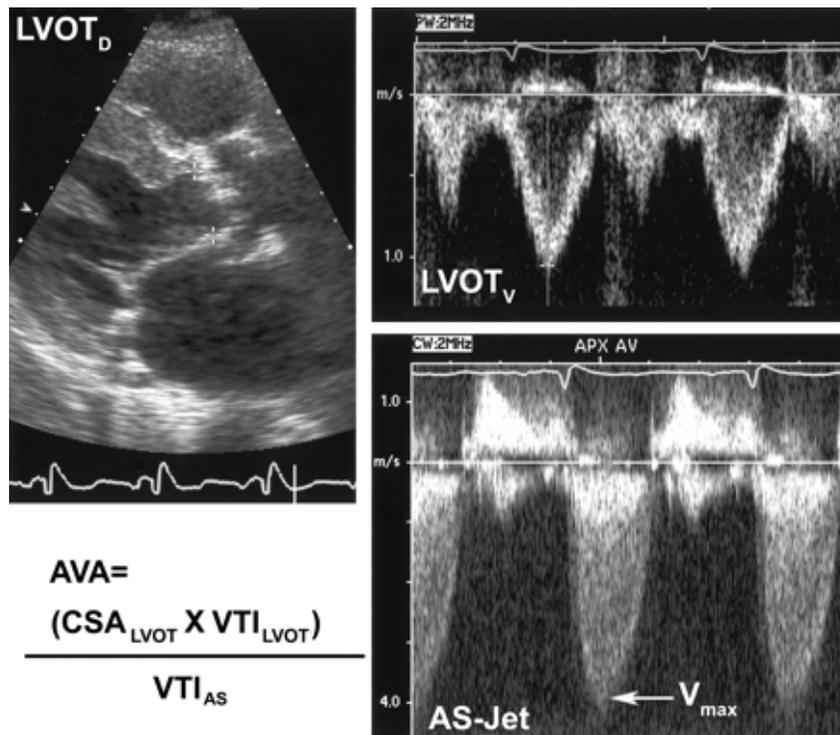
In advanced cases, ST-segment depression and T-wave inversion (LV "strain") in standard leads I and aVL and in the left precordial leads are evident. However, there is no close correlation between the ECG and the hemodynamic severity of obstruction, and the absence of ECG signs of LV hypertrophy does not exclude severe obstruction.

Echocardiogram

The key findings are LV hypertrophy and, in patients with valvular calcification (i.e., most adult patients with symptomatic AS), multiple, bright, thick, echoes from the valve.



Still frame two-dimensional echocardiographic image from the parasternal long axis view of a patient with aortic stenosis. The aortic valve is calcified with restricted opening during systole. Ao, aorta; RV, right ventricle; LA, left atrium; LV, left ventricle.



Standard evaluation of aortic stenosis (AS) severity is based on measurement of left ventricular outflow tract (LVOT) diameter (D) in a parasternal long-axis view for calculation of a circular cross-sectional area (CSA), outflow tract velocity (V) from an apical approach using pulsed Doppler, and the maximum aortic jet (AS-Jet, V_{\max}) from the continuous-wave Doppler recording. Either velocity-time integrals (VTIs) or maximum velocities can be used in the continuity equation for aortic valve area (AVA).

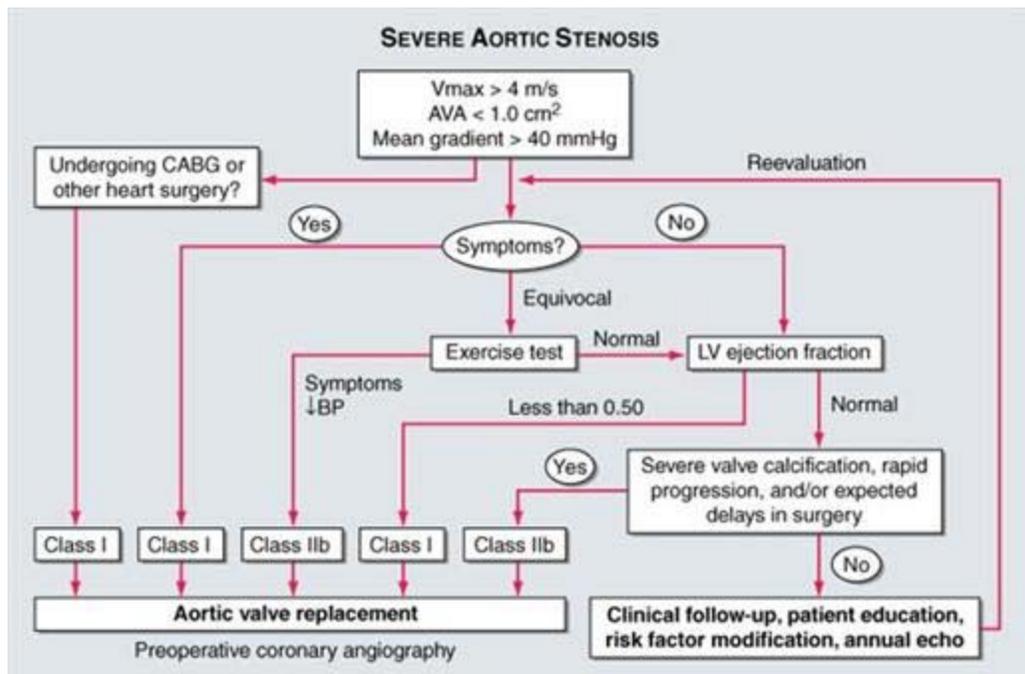
. The valve gradient and aortic valve area can be estimated by Doppler measurement of the transaortic velocity. Severe AS is defined by a valve area $<1.0 \text{ cm}^2$, whereas moderate AS is defined by a valve area of $1.0\text{--}1.5 \text{ cm}^2$ and mild AS by a valve area of $1.5\text{--}2.0 \text{ cm}^2$. LV dilatation and reduced systolic shortening reflect impairment of LV function.

Echocardiography is useful for identifying coexisting valvular abnormalities such as MS and AR, which sometimes accompany AS; for differentiating valvular AS from other forms of outflow obstruction; and for measurement of the aortic root. Aneurysmal enlargement (maximal dimension $>4.5 \text{ cm}$) of the root or ascending aorta can occur in up to 20% of patients with bicuspid aortic valve disease, independent of the severity of the valve lesion. [Dobutamine](#) stress echocardiography is useful for the evaluation of patients with severe AS and severe LV systolic dysfunction (EF <0.35).

Chest X-Ray

The chest x-ray may show no or little overall cardiac enlargement for many years. Hypertrophy without dilatation may produce some rounding of the cardiac apex in the frontal projection and slight backward displacement in the lateral view; severe AS is often associated with poststenotic dilatation of the ascending aorta. As noted above, however, aortic enlargement may be an independent process and mediated by the same type of structural changes that occur in patients with Marfan syndrome. Aortic calcification is usually readily apparent on fluoroscopic examination or by echocardiography; the absence of valvular calcification in an adult suggests that severe valvular AS is *not* present. In later stages of the disease, as the LV dilates there is increasing roentgenographic evidence of LV enlargement, pulmonary congestion, and enlargement of the LA, PA, and right side of the heart.

Medical Treatment



Management strategy for patients with severe aortic stenosis. Preoperative coronary angiography should be performed routinely as determined by age, symptoms, and coronary risk factors. Cardiac catheterization and angiography may also be helpful when there is discordance between clinical findings and echocardiography. *AVA*, aortic valve area; *BP*, blood pressure; *CABG*, coronary artery bypass graft surgery; *echo*, echocardiography; *LV*, left ventricle; *Vmax*, maximal velocity across aortic valve by Doppler echocardiography. (From Bonow et al. Modified from CM Otto: *J Am Coll Cardiol* 47:2141, 2006.)

In patients with severe AS ($<1.0 \text{ cm}^2$), strenuous physical activity should be avoided, even in the asymptomatic stage. Care must be taken to avoid dehydration and hypovolemia to protect against a significant reduction in CO. Medications used for the treatment of hypertension or CAD, including beta blockers and ACE inhibitors, are generally safe for asymptomatic patients with preserved left ventricular systolic function. [Nitroglycerin](#) is helpful in relieving angina pectoris. Retrospective studies have shown that patients with degenerative calcific AS who receive [HMG-CoA reductase inhibitors](#) ("statins") exhibit slower progression of leaflet calcification and aortic valve area reduction than those who do not. One prospective randomized clinical trial using high-dose [atorvastatin](#) failed to show a measurable benefit, although a more recent trial using [rosuvastatin](#) did show a beneficial effect. The role of [statin](#) medications may be more clearly defined with further study.

Surgical Treatment

Asymptomatic patients with calcific AS and severe obstruction should be followed carefully for the development of symptoms and by serial echocardiograms for evidence of deteriorating

LV function. Operation is indicated in patients with severe AS (valve area $<1.0 \text{ cm}^2$ or $0.6 \text{ cm}^2/\text{m}^2$ body surface area) who are symptomatic, those who exhibit LV dysfunction (EF $<50\%$), as well as those with an aneurysmal or expanding aortic root (maximal dimension $>4.5 \text{ cm}$ or annual increase in size $>0.5 \text{ cm/year}$), even if they are asymptomatic. In patients without heart failure, the operative risk of AVR is approximately 3%. It is prudent to postpone operation in patients with severe calcific AS who are truly asymptomatic and who exhibit normal LV function, i.e., EF $>50\%$, since they may continue to do well for years. However, some advocate AVR in patients with severe valve calcification and rapid progression of obstruction. The risk of surgical mortality exceeds that of sudden death in asymptomatic patients. Exercise testing is employed in many centers to assess objectively the functional capacity of asymptomatic patients for whom the history is ambiguous. As many as one-third of patients will show signs of functional impairment during exercise for which AVR should be considered. AVR is carried out in asymptomatic patients with severe or moderately severe stenosis who undergo coronary artery bypass grafting. AVR is also routinely performed in patients with moderate AS who are undergoing coronary bypass grafting or aortic root reconstruction.

Operation should, if possible, be carried out before frank LV failure develops; at this late stage, the aortic valve pressure gradient declines as the CO, stroke volume, and EF decline (low gradient, low output AS). In such patients the perioperative risk is high (15–20%), and evidence of myocardial disease may persist even when the operation is technically successful. Furthermore, long-term postoperative survival also correlates inversely with preoperative LV dysfunction. Nonetheless, in view of the even worse prognosis of such patients when they are treated medically, there is usually little choice but to advise surgical treatment, especially in patients in whom contractile reserve can be demonstrated by [dobutamine](#) echocardiography (defined by a 20% increase in stroke volume after dobutamine challenge). In patients in whom severe AS and CAD coexist, relief of the AS and revascularization of the myocardium by means of aortocoronary bypass grafting may result in striking clinical and hemodynamic improvement.

Because many patients with calcific AS are elderly, particular attention must be directed to the adequacy of hepatic, renal, and pulmonary function before AVR is recommended. Age alone is not a contraindication to AVR for AS. The mortality rate depends to a substantial extent on the patient's preoperative clinical and hemodynamic state. The 10-year survival rate of patients with AVR is approximately 60%. Approximately 30% of bioprosthetic valves evidence primary valve

failure in 10 years, requiring re-replacement, and an approximately equal percentage of patients with mechanical prostheses develop significant hemorrhagic complications as a consequence of treatment with anticoagulants.

Percutaneous Balloon Aortic Valvuloplasty

This procedure is preferable to operation in children and young adults with congenital, noncalcific AS. It is not commonly used in adults with severe calcific AS because of a very high restenosis rate and the risk of procedural complications, but on occasion it has been used successfully as a "bridge to operation" in patients with severe LV dysfunction and shock who are too ill to tolerate surgery.

Aortic Regurgitation

In approximately two-thirds of patients with valvular AR, the disease is rheumatic in origin, resulting in thickening, deformity, and shortening of the individual aortic valve cusps, changes that prevent their proper opening during systole and closure during diastole. A rheumatic origin is much less common in patients with isolated AR who do not have associated mitral valve disease. Patients with congenital BAV disease may develop predominant AR. Congenital fenestrations of the aortic valve occasionally produce mild AR. Membranous subaortic stenosis often leads to thickening and scarring of the aortic valve leaflets with secondary AR. Prolapse of an aortic cusp, resulting in progressive chronic AR, occurs in approximately 15% of patients with ventricular septal defect but may also occur as an isolated phenomenon or as a consequence of myxomatous degeneration sometimes associated with mitral and/or tricuspid valve involvement.

AR may result from infective endocarditis, which can develop on a valve previously affected by rheumatic disease, a congenitally deformed valve, or, rarely, on a normal aortic valve, and may lead to perforation or erosion of one or more leaflets. The aortic valve leaflets may become scarred and retracted during the course of syphilis or ankylosing spondylitis and contribute further to the AR that derives primarily from the associated root disease. Although traumatic rupture or avulsion of the aortic valve is an uncommon cause of acute AR, it does represent the most frequent serious lesion in patients surviving nonpenetrating cardiac injuries. The coexistence of hemodynamically significant AS with AR usually excludes all the rarer forms of AR because it occurs almost exclusively in patients with rheumatic or congenital AR. In patients with AR due to primary valvular disease, dilatation of the aortic annulus may occur

secondarily and intensify the regurgitation.

Pathophysiology

The total stroke volume ejected by the LV (i.e., the sum of the effective forward stroke volume and the volume of blood that regurgitates back into the LV) is increased in patients with AR. In patients with wide-open (free) AR, the volume of regurgitant flow may equal the effective forward stroke volume. In contrast to MR, in which a fraction of the LV stroke volume is delivered into the low-pressure LA, in AR the entire LV stroke volume is ejected into a high-pressure zone, the aorta. An increase in the LV end-diastolic volume (increased preload) constitutes the major hemodynamic compensation for AR. The dilatation and eccentric hypertrophy of the LV allow this chamber to eject a larger stroke volume without requiring any increase in the relative shortening of each myofibril. Therefore, severe AR may occur with a normal effective forward stroke volume and a normal left ventricular EF [total (forward plus regurgitant) stroke volume/end-diastolic volume], together with an elevated LV end-diastolic pressure and volume. However, through the operation of Laplace's law, LV dilatation increases the LV systolic tension required to develop any given level of systolic pressure. Chronic AR is thus a state in which LV preload and afterload are both increased. Ultimately, these adaptive measures fail. As LV function deteriorates, the end-diastolic volume rises further and the forward stroke volume and EF decline. Deterioration of LV function often precedes the development of symptoms. Considerable thickening of the LV wall also occurs with chronic AR, and at autopsy the hearts of these patients may be among the largest encountered, sometimes weighing >1000 g.

The reverse pressure gradient from aorta to LV, which drives the AR flow, falls progressively during diastole, accounting for the decrescendo nature of the diastolic murmur. Equilibration between aortic and LV pressures may occur toward the end of diastole in patients with chronic severe AR, particularly when the heart rate is slow. In patients with acute severe AR, the LV is unprepared for the regurgitant volume load. LV compliance is normal or reduced, and LV diastolic pressures rise rapidly, occasionally to levels >40 mmHg. The LV pressure may exceed the LA pressure toward the end of diastole, and this reversed pressure gradient closes the mitral valve prematurely.

In patients with chronic severe AR, the effective forward CO usually is normal or only slightly reduced at rest, but often it fails to rise normally during exertion. Early signs of LV dysfunction include reduction in the EF. In advanced stages there may be considerable

elevation of the LA, PA wedge, PA, and RV pressures and lowering of the forward CO at rest.

Physical Findings

In chronic severe AR, the jarring of the entire body and the bobbing motion of the head with each systole can be appreciated, and the abrupt distention and collapse of the larger arteries are easily visible. The examination should be directed toward the detection of conditions predisposing to AR, such as Marfan syndrome, ankylosing spondylitis, and ventricular septal defect.

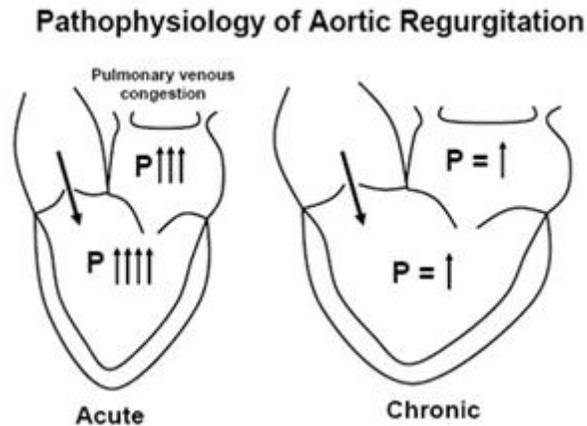
Arterial Pulse

A rapidly rising "water-hammer" pulse, which collapses suddenly as arterial pressure falls rapidly during late systole and diastole (Corrigan's pulse), and capillary pulsations, an alternate flushing and paling of the skin at the root of the nail while pressure is applied to the tip of the nail (Quincke's pulse), are characteristic of free AR. A booming "pistol-shot" sound can be heard over the femoral arteries (Traube's sign), and a to-and-fro murmur (Duroziez's sign) is audible if the femoral artery is lightly compressed with a stethoscope.

The arterial pulse pressure is widened, and there is an elevation of the systolic pressure, sometimes to as high as 300 mmHg, and a depression of the diastolic pressure. The measurement of arterial diastolic pressure with a sphygmomanometer may be complicated by the fact that systolic sounds are frequently heard with the cuff completely deflated. However, the level of cuff pressure at the time of muffling of the Korotkoff sounds (Phase IV) generally corresponds fairly closely to the true intraarterial diastolic pressure. As the disease progresses and the LV end-diastolic pressure rises, the arterial diastolic pressure may actually rise as well, since the aortic diastolic pressure cannot fall below the LV end-diastolic pressure. For the same reason, acute severe AR may also be accompanied by only a slight widening of the pulse pressure. Such patients are invariably tachycardic as the heart rate increases in an attempt to preserve the CO.

Auscultation

In patients with severe AR, the aortic valve closure sound (A_2) is usually absent. An S_3 and systolic ejection sound are frequently audible, and occasionally an S_4 also may be heard. The murmur of chronic AR is typically a high-pitched, blowing, decrescendo diastolic murmur, heard best in the third intercostal space along the left sternal border.



A. Schematic representation of ECG, aortic pressure (AOP), left ventricular pressure (LVP), and left atrial pressure (LAP). The shaded areas indicate a transvalvular pressure difference during systole. HSM, holosystolic murmur; MSM, midsystolic murmur. **B.** Graphic representation of ECG, aortic pressure (AOP), left ventricular pressure (LVP), and left atrial pressure (LAP) with shaded areas indicating transvalvular diastolic pressure difference. EDM, early diastolic murmur; PSM, presystolic murmur; MDM, middiastolic murmur.

In patients with mild AR, this murmur is brief but, as the severity increases, generally becomes louder and longer, indeed holodiastolic. When the murmur is soft, it can be heard best with the diaphragm of the stethoscope and with the patient sitting up, leaning forward, and with the breath held in forced expiration. In patients in whom the AR is caused by primary valvular disease, the diastolic murmur is usually louder along the left than the right sternal border. However, when the murmur is heard best along the right sternal border, it suggests that the AR is caused by aneurysmal dilatation of the aortic root. "Cooing" or musical diastolic murmurs suggest eversion of an aortic cusp vibrating in the regurgitant stream.

A mid-systolic ejection murmur is frequently audible in isolated AR. It is generally heard best at the base of the heart and is transmitted along the carotid vessels. This murmur may be quite loud without signifying aortic obstruction. A third murmur frequently heard in patients with severe AR is the *Austin Flint murmur*, a soft, low-pitched, rumbling mid-diastolic murmur. It is probably produced by the diastolic displacement of the anterior leaflet of the mitral valve by the AR stream but does not appear to be associated with hemodynamically significant mitral obstruction. The auscultatory features of AR are intensified by strenuous handgrip, which augments systemic resistance.

In acute severe AR, the elevation of LV end-diastolic pressure may lead to early closure of the mitral valve, an associated mid-diastolic sound, a soft or absent S₁, a pulse pressure that is not particularly wide, and a soft, short diastolic murmur of AR.

Laboratory Examination

ECG

In patients with chronic severe AR, the ECG signs of LV hypertrophy become manifest. In addition, these patients frequently exhibit ST-segment depression and T-wave inversion in leads I, aVL, V₅, and V₆ ("LV strain"). Left axis deviation and/or QRS prolongation denote diffuse myocardial disease, generally associated with patchy fibrosis, and usually signify a poor prognosis.

Echocardiogram

The extent and velocity of wall motion are normal or even supernormal, until myocardial contractility declines. A rapid, high-frequency fluttering of the anterior mitral leaflet produced by the impact of the regurgitant jet is a characteristic finding. The echocardiogram is also useful in determining the cause of AR, by detecting dilatation of the aortic annulus and root or aortic dissection. Thickening and failure of coaptation of the leaflets also may be noted. Color flow Doppler echocardiographic imaging is very sensitive in the detection of AR, and Doppler echocardiography is helpful in assessing its severity. With severe AR, the central jet width exceeds 65% of the left ventricular outflow tract, the regurgitant volume is 60 ml/beat, the regurgitant fraction is 50%, and there is diastolic flow reversal in the proximal descending thoracic aorta. The continuous wave Doppler profile shows a rapid deceleration time in patients with acute severe AR, due to the rapid increase in LV diastolic pressure. Serial two-dimensional echocardiography is valuable in assessing LV performance and in detecting progressive myocardial dysfunction.

Chest X-Ray

In chronic severe AR, the apex is displaced downward and to the left in the frontal projection. In the left anterior oblique and lateral projections, the LV is displaced posteriorly and encroaches on the spine. When AR is caused by primary disease of the aortic root, aneurysmal dilatation of the aorta may be noted, and the aorta may fill the retrosternal space in the lateral view. Echocardiography and CT angiography are more sensitive than the chest x-ray for the

detection of aortic root enlargement.

Table 1. Classification of the Severity of Aortic Regurgitation.*				
Variable	Aortic Regurgitation			
	Mild	Moderate‡		Severe
Width of vena contracta (mm)†	<3.0	3.0–5.9		≥6.0
Ratio of width of aortic regurgitant jet to left ventricular outflow (%)	<25	25–44	45–64	≥65
Regurgitant volume (ml per beat)	<30	30–44	45–59	≥60
Regurgitant fraction (%)	<30	30–39	40–49	≥50
Effective regurgitant orifice (mm ²)	<10	10–19	20–29	≥30

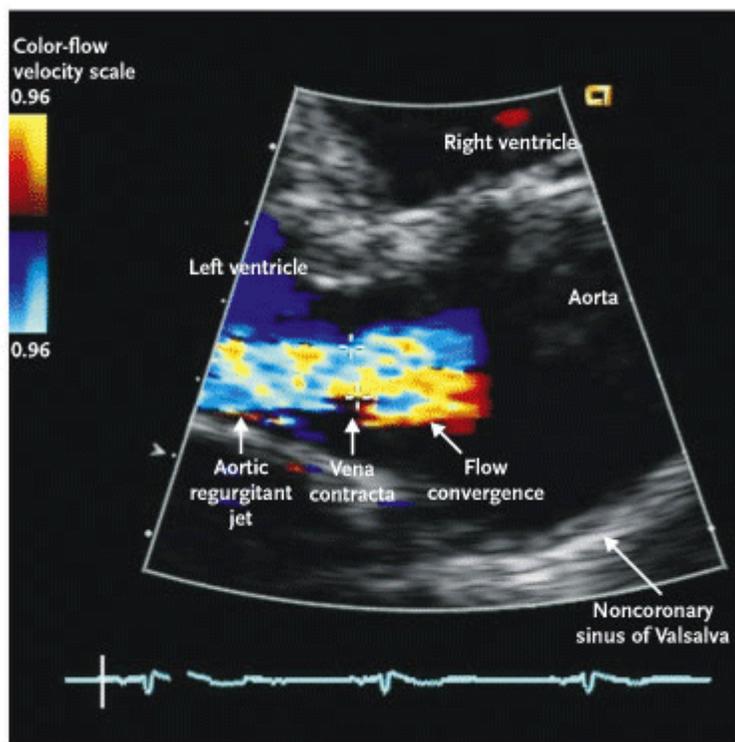
* The classification is from the American Society of Echocardiography.¹¹

† The vena contracta is the regurgitant flow at the orifice on color-flow imaging.

‡ The subdivisions of the moderate class correspond to the subcategories of “mild to moderate” and “moderately severe.”

Clinically, bounding arterial pulses, a widened pulse pressure, a loud diastolic murmur, and a third heart sound are signs of severe regurgitation but are not always specific. Doppler echocardiography has become the mainstay of the assessment of the severity of aortic regurgitation. Suggestive of severe regurgitation are signs of a broad jet width on color-flow imaging, steep jet velocity deceleration (reflecting equalization of aortic and ventricular pressure), and prolonged diastolic flow reversal in the aorta. The use of Doppler echocardiography makes it possible to quantify the effective regurgitant orifice (severe if 0.30 cm^2) and regurgitant volume (severe if 60 ml per beat)

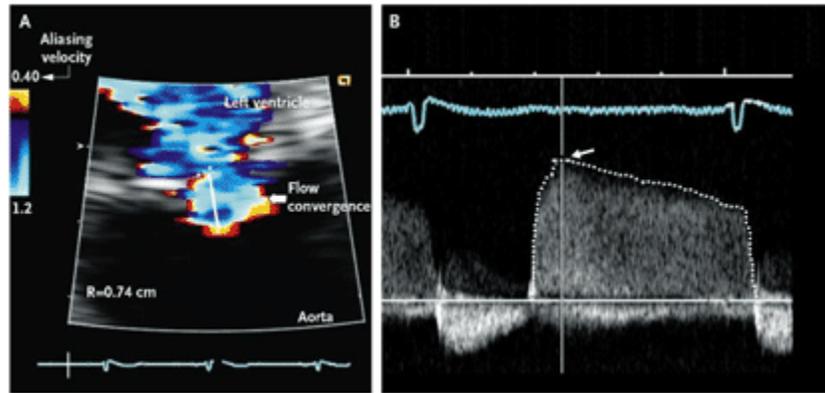
[Echo-Doppler imaging of aortic regurgitation \(video\)](#)



Example of a Jet of Aortic Regurgitation, as Shown by Color-Flow Imaging.

The three components of the regurgitant flow (flow convergence above the orifice, vena contracta through the orifice, and the jet below the orifice) are shown. The width of the vena contracta (as indicated by crosses) can be measured as a surrogate for the regurgitant orifice.

A simple, reliable measurement is the "vena contracta" — that is, the width of the regurgitant flow at the orifice, a surrogate measurement for the size of the orifice. Measurements that are 0.5 cm or more have a high sensitivity for the diagnosis of severe regurgitation, and measurements that are 0.7 cm or more have a high specificity for the diagnosis. On rare occasions, this approach is inconclusive, and either transesophageal echocardiography or angiography of the aortic root is necessary to determine the severity of aortic regurgitation. Left ventricular size and function (particularly, the end-systolic diameter and ejection fraction) should be routinely assessed, as should dilatation of the ascending aorta. If transthoracic imaging is suboptimal for the latter, transesophageal echocardiography, computed tomography, or magnetic resonance imaging can be used. Exercise testing may be warranted in asymptomatic patients with limited physical activity to evaluate functional limitations and may also provide information about changes of left ventricular function with stress.



Example of Quantitation of Aortic Regurgitation by the Convergence of the Proximal Flow. Panel A is a color-flow image of the aortic valve; the measured radius of the proximal flow convergence (R) is 0.74 cm, and the regurgitant flow is calculated as 138 ml per second. The "aliasing" velocity of 0.40 m per second (modified by baseline displacement) is the blood velocity at the junction of the orange and blue flows. Panel B shows a continuous-wave Doppler measurement of regurgitant blood velocity, at 455 cm per second (arrow). The effective regurgitant orifice area is determined by dividing the flow by the velocity, which in this case is 0.30 cm^2 .

Treatment

Acute Aortic Regurgitation

Patients with acute severe AR may respond to intravenous diuretics and vasodilators (such as [sodium nitroprusside](#)), but stabilization is usually short-lived and operation is indicated urgently. Intraaortic balloon counterpulsation is contraindicated. Beta-blockers are also best avoided so as not to reduce the CO further or slow the heart rate, which might allow proportionately more time in diastole for regurgitation to occur. Surgery is the treatment of choice.

Chronic Aortic Regurgitation

Early symptoms of dyspnea and effort intolerance respond to treatment with diuretics and vasodilators (ACE inhibitors, dihydropyridine calcium channel blockers, or [hydralazine](#)) may be useful as well. Surgery can then be performed in more controlled circumstances. The use of vasodilators to extend the compensated phase of chronic severe AR before the onset of symptoms or the development of LV dysfunction is more controversial. Expert consensus is strong regarding the need to control systolic blood pressure (goal $<140 \text{ mmHg}$) in patients with

chronic AR, and vasodilators are an excellent first choice as antihypertensive agents. It is often difficult to achieve adequate control in such patients because of the increased stroke volume. Cardiac arrhythmias and systemic infections are poorly tolerated in patients with severe AR and must be treated promptly and vigorously. Although [nitroglycerin](#) and long-acting nitrates are not as helpful in relieving anginal pain as they are in patients with ischemic heart disease, they are worth a trial. Patients with syphilitic aortitis should receive a full course of penicillin therapy. Beta blockers may be useful to retard the rate of aortic root enlargement in young patients with Marfan syndrome and aortic root dilatation with no or only mild AR. Patients with severe AR should avoid isometric exercises.

Surgical Treatment

In deciding on the advisability and proper timing of surgical treatment, two points should be kept in mind: (1) patients with chronic severe AR usually do not become symptomatic until *after* the development of myocardial dysfunction; and (2) when delayed too long (defined as >1 year from onset of symptoms or LV dysfunction), surgical treatment often does not restore normal LV function. Therefore, in patients with chronic severe AR, careful clinical follow-up and noninvasive testing with echocardiography at approximately 6-month intervals are necessary if operation is to be undertaken at the optimal time, i.e., *after* the onset of LV dysfunction but *prior* to the development of severe symptoms. Operation can be deferred as long as the patient both remains asymptomatic and retains normal LV function.

AVR is indicated for the treatment of severe AR in symptomatic patients irrespective of LV function. In general, operation should be carried out in asymptomatic patients with severe AR and progressive LV dysfunction defined by an LVEF <50%, an LV end-systolic dimension >55mm or end-systolic volume >55 mL/m², or an LV diastolic dimension >75 mm. Smaller dimensions may be appropriate thresholds in individuals of smaller stature. Patients with severe AR without indications for operation should be followed by clinical and echocardiographic examination every 3–12 months.

Surgical options for management of aortic valve and root disease have expanded considerably over the past decade. AVR with a suitable mechanical or tissue prosthesis is generally necessary in patients with rheumatic AR and in many patients with other forms of regurgitation. Rarely, when a leaflet has been perforated during infective endocarditis or torn from its attachments to the aortic annulus by thoracic trauma, primary surgical repair may be

possible. When AR is due to aneurysmal dilatation of the annulus and ascending aorta rather than to primary valvular involvement, it may be possible to reduce the regurgitation by narrowing the annulus or by excising a portion of the aortic root without replacing the valve. Resuspension of the native aortic valve leaflets is possible in approximately 50% of patients with acute AR in the setting of Type A aortic dissection. In other conditions, however, regurgitation can be eliminated only by replacing the aortic valve, excising the dilated or aneurysmal ascending aorta responsible for the regurgitation, and replacing it with a graft. This formidable procedure entails a higher risk than isolated AVR.

As in patients with other valvular abnormalities, both the operative risk and the late mortality are largely dependent on the stage of the disease and on myocardial function at the time of operation. The overall operative mortality for isolated AVR is about 3%. However, patients with marked cardiac enlargement and prolonged LV dysfunction experience an operative mortality rate of approximately 10% and a late mortality rate of approximately 5% per year due to LV failure despite a technically satisfactory operation. Nonetheless, because of the very poor prognosis with medical management, even patients with LV failure should be considered for operation.

Patients with acute severe AR require prompt surgical treatment, which may be lifesaving.

Tricuspid stenosis

TS, much less prevalent than MS in North America and Western Europe, is generally rheumatic in origin and is more common in females than in males. It does not occur as an isolated lesion and is usually associated with MS. Hemodynamically significant TS occurs in 5–10% of patients with severe MS; rheumatic TS is commonly associated with some degree of TR. Nonrheumatic causes of TS are rare.

Pathophysiology

A diastolic pressure gradient between the RA and RV defines TS. It is augmented when the transvalvular blood flow increases during inspiration and declines during expiration. A mean diastolic pressure gradient of 4 mmHg is usually sufficient to elevate the mean RA pressure to levels that result in systemic venous congestion. Unless sodium intake has been restricted and diuretics administered, this venous congestion is associated with hepatomegaly, ascites, and edema, sometimes severe. In patients with sinus rhythm, the RA *a* wave may be extremely tall

and may even approach the level of the RV systolic pressure. The y descent is prolonged. The CO at rest is usually depressed, and it fails to rise during exercise. The low CO is responsible for the normal or only slightly elevated LA, PA, and RV systolic pressures despite the presence of MS. Thus, the presence of TS can mask the hemodynamic and clinical features of the MS, which usually accompanies it.

Symptoms

Since the development of MS generally precedes that of TS, many patients initially have symptoms of pulmonary congestion. Spontaneous improvement of these symptoms should raise the possibility that TS may be developing. Characteristically, patients complain of relatively little dyspnea for the degree of hepatomegaly, ascites, and edema that they have. However, fatigue secondary to a low CO and discomfort due to refractory edema, ascites, and marked hepatomegaly are common in patients with TS and/or TR. In some patients, TS may be suspected for the first time when symptoms of right-sided failure persist after an adequate mitral valvotomy.

Physical Findings

Severe TS is associated with marked hepatic congestion, often resulting in cirrhosis, jaundice, serious malnutrition, anasarca, and ascites. Congestive hepatomegaly and, in cases of severe tricuspid valve disease, splenomegaly are present.

On auscultation, an OS of the tricuspid valve may occasionally be heard approximately 0.06 s after pulmonic valve closure. The diastolic murmur of TS has many of the qualities of the diastolic murmur of MS, and since TS almost always occurs in the presence of MS, the less-common valvular lesion may be missed. However, the tricuspid murmur is generally heard best along the left lower sternal margin and over the xiphoid process, and it is most prominent during presystole in patients with sinus rhythm. The murmur of TS is augmented during inspiration, and it is reduced during expiration and particularly during the strain phase of the Valsalva maneuver, when tricuspid blood flow is reduced.

Laboratory Examination

The ECG features of RA enlargement include tall, peaked P waves in lead II, as well as prominent, upright P waves in lead V₁. The *absence* of ECG evidence of right ventricular hypertrophy (RVH) in a patient with right-sided heart failure who is believed to have MS

should suggest associated tricuspid valve disease. The chest x-ray in patients with combined TS and MS shows particular prominence of the RA and superior vena cava without much enlargement of the PA and with less evidence of pulmonary vascular congestion than occurs in patients with isolated MS. On echocardiographic examination, the tricuspid valve is usually thickened and domes in diastole; the transvalvular gradient can be estimated by Doppler echocardiography. TTE provides additional information regarding mitral valve structure and function, LV and RV size and function, and PA pressure.

Tricuspid Stenosis: Treatment

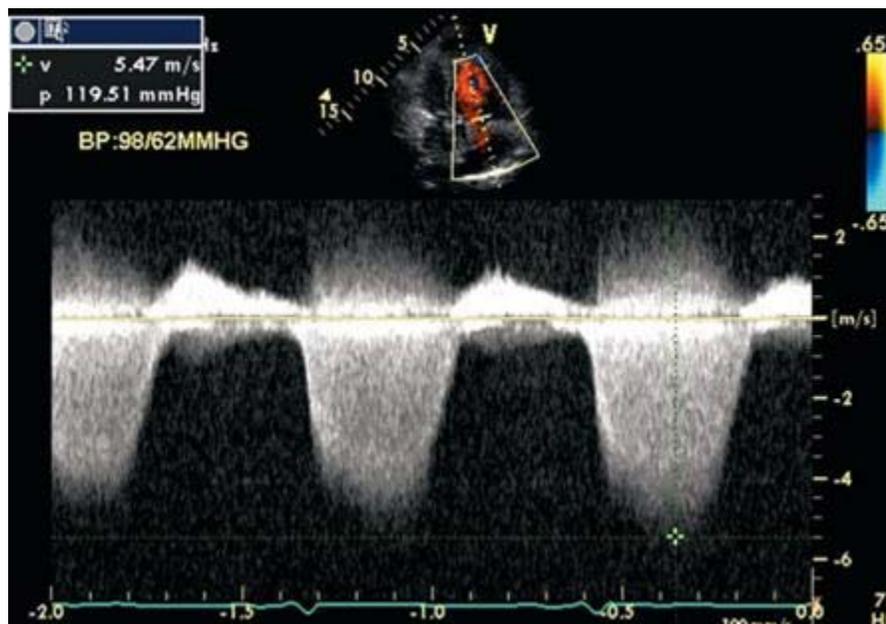
Patients with TS generally exhibit marked systemic venous congestion; intensive salt restriction, bed rest, and diuretic therapy are required during the preoperative period. Such a preparatory period may diminish hepatic congestion and thereby improve hepatic function sufficiently so that the risks of operation, particularly bleeding, are diminished. Surgical relief of the TS should be carried out, preferably at the time of surgical mitral valvotomy or MVR, in patients with moderate or severe TS who have mean diastolic pressure gradients exceeding ~ 4 mmHg and tricuspid orifice areas $< 1.5\text{--}2.0\text{ cm}^2$. TS is almost always accompanied by significant TR. Operative repair may permit substantial improvement of tricuspid valve function. If repair cannot be accomplished, the tricuspid valve may have to be replaced with a prosthesis, preferably a large bioprosthetic valve. Mechanical valves in the tricuspid position are more prone to thromboembolic complications than in other positions.

Tricuspid regurgitation

Most commonly, TR is functional and secondary to marked dilatation of the tricuspid annulus. Functional TR may complicate RV enlargement of any cause, including inferior wall infarcts that involve the RV. It is commonly seen in the late stages of heart failure due to rheumatic or congenital heart disease with severe pulmonary hypertension (pulmonary artery systolic pressure > 55 mmHg), as well as in ischemic heart disease and dilated cardiomyopathy. It is reversible in part if pulmonary hypertension is relieved. Rheumatic fever may produce organic (primary) TR, often associated with TS. Infarction of RV papillary muscles, tricuspid valve prolapse, carcinoid heart disease, endomyocardial fibrosis, infective endocarditis, and trauma all may produce TR. Less commonly, TR results from congenitally deformed tricuspid valves, and it occurs with defects of the atrioventricular canal, as well as with Ebstein's malformation of the tricuspid valve. TR also develops eventually in patients with chronic RV apical pacing.

As is the case for TS, the clinical features of TR result primarily from systemic venous congestion and reduction of CO. With the onset of TR in patients with pulmonary hypertension, symptoms of pulmonary congestion diminish, but the clinical manifestations of right-sided heart failure become intensified. The neck veins are distended with prominent *v* waves and rapid *y* descents, marked hepatomegaly, ascites, pleural effusions, edema, systolic pulsations of the liver, and a positive hepatojugular reflux. A prominent RV pulsation along the left parasternal region and a blowing holosystolic murmur along the lower left sternal margin, which may be intensified during inspiration and reduced during expiration or the strain of the Valsalva maneuver (Carvalho's sign), are characteristic findings; AF is usually present.

The ECG usually shows changes characteristic of the lesion responsible for the enlargement of the RV that leads to TR, e.g., inferior wall myocardial infarction or severe RVH. Echocardiography may be helpful by demonstrating RV dilatation and prolapsing, flail, scarred, or displaced tricuspid leaflets; the diagnosis of TR can be made by color flow Doppler echocardiography, and its severity can be estimated by Doppler examination.



Continuous-wave Doppler of tricuspid regurgitation in a patient with pulmonary hypertension. There is an increase in the velocity of flow from the right ventricle into the right atrium to 5.4 m/s. Using the modified Bernoulli equation, the peak pressure gradient between the right ventricle and right atrium during systole is 120 mmHg. Assuming a right atrial pressure of 10 mmHg, the right ventricular systolic pressure is 130 mmHg. In the absence of right ventricular outflow tract obstruction, this indicates there is severe pulmonary hypertension with a pulmonary artery systolic pressure of 130 mmHg.

Severe TR is accompanied by hepatic vein systolic flow reversal. Continuous wave Doppler is also useful in estimating PA pressure. Roentgenographic examination usually reveals enlargement of both the RA and RV.

In patients with severe TR, the CO is usually markedly reduced, and the RA pressure pulse may exhibit no *x* descent during early systole but a prominent *c-v* wave with a rapid *y* descent. The mean RA and the RV end-diastolic pressures are often elevated.

Tricuspid Regurgitation: Treatment

Isolated TR, in the absence of pulmonary hypertension, such as that occurring as a consequence of infective endocarditis or trauma, is usually well tolerated and does not require operation. Indeed, even total excision of an infected tricuspid valve may be well tolerated for several years if the PA pressure is normal. Treatment of the underlying cause of heart failure usually reduces the severity of functional TR, by reducing the size of the tricuspid annulus. In patients with mitral valve disease and TR secondary to pulmonary hypertension and massive RV enlargement, effective surgical correction of the mitral valvular abnormality results in lowering of the PA pressures and gradual reduction or disappearance of the TR without direct treatment of the tricuspid valve. However, recovery may be much more rapid in patients with severe secondary TR if, at the time of mitral valve surgery, and especially when there is measurable enlargement of the tricuspid valve annulus, tricuspid annuloplasty (generally with the insertion of a plastic ring), open tricuspid valve repair, or, in the rare instance of severe organic tricuspid valve disease, tricuspid valve replacement is performed. Tricuspid annuloplasty or replacement may be required for severe TR with primary involvement of the valve.

Ebstein's Anomaly

Ebstein's anomaly is an abnormality of the tricuspid valve in which the septal leaflets and often the posterior leaflets are displaced into the right ventricle and the anterior leaflet is usually malformed, excessively large, and abnormally attached or adherent to the right ventricular free wall. Thus, a portion of the right ventricle is "atrialized" in that it is located on the atrial side of the tricuspid valve, and the remaining functional right ventricle is small (Figure 6). The tricuspid valve is usually regurgitant but may be stenotic. Eighty percent of patients with

Ebstein's anomaly have an interatrial communication (atrial septal defect or patent foramen ovale) through which right-to-left shunting of blood may occur.

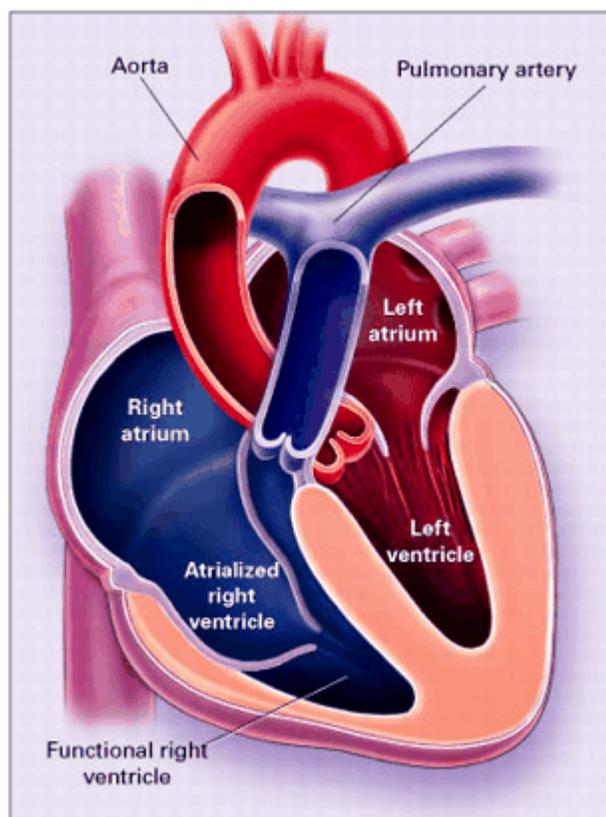


Figure Ebstein's Anomaly.

In patients with Ebstein's anomaly, a portion of the right ventricle is atrialized (i.e., located on the atrial side of the tricuspid valve), and as a result, the functional right ventricle is small. In addition, most patients have an interatrial communication (atrial septal defect or patent foramen ovale), through which right-to-left shunting may occur.

The severity of the hemodynamic derangements in patients with Ebstein's anomaly depends on the degree of displacement and the functional status of the tricuspid-valve leaflets. Patients with mild apical displacement of the tricuspid leaflets have normal valvular function, whereas those with severe tricuspid-leaflet displacement or abnormal anterior leaflet attachment, with valvular dysfunction, have elevated right atrial pressure and right-to-left interatrial shunting. Similarly, the clinical presentation of Ebstein's anomaly varies from severe heart failure in a fetus or neonate to the absence of symptoms in an adult in whom it is discovered incidentally.

When Ebstein's anomaly is discovered during fetal life, the rate of intrauterine mortality is

high. Neonates with severe disease have cyanosis, with heart failure and a murmur noted in the first days of life. Transient improvement may occur as pulmonary vascular resistance falls, but the condition worsens after the ductus arteriosus closes, thereby decreasing pulmonary blood flow. Older children with Ebstein's anomaly often come to medical attention because of an incidental murmur, whereas adolescents and adults present with a supraventricular arrhythmia. In adults with Ebstein's anomaly, the most important predictors of outcome are the New York Heart Association (NYHA) functional class, the heart size, the presence or absence of cyanosis, and the presence or absence of paroxysmal atrial tachycardias. Such tachycardias may lead to cardiac failure, worsening cyanosis, and even syncope. Patients with Ebstein's anomaly and an interatrial communication are at risk for paradoxical embolization, brain abscess, and sudden death.

On physical examination, the severity of cyanosis depends on the magnitude of right-to-left shunting. The first and second heart sounds are widely split, and a third or fourth heart sound is often present, resulting in a "triple" or "quadruple" rhythm. A systolic murmur caused by tricuspid regurgitation is usually present at the left lower sternal border. Hepatomegaly (resulting from passive hepatic congestion due to elevated right atrial pressure) may be present.

Tall and broad P waves are common on the electrocardiogram, as is right bundle-branch block. First-degree atrioventricular block occurs frequently. Since about 20 percent of patients with Ebstein's anomaly have ventricular preexcitation by way of an accessory electrical pathway between the atrium and ventricle (Wolff–Parkinson–White syndrome), a delta wave may be present. The radiographic findings depend on the severity of the anatomical abnormality. In mild cases, the heart size and pulmonary vasculature are normal. In more severe cases, marked cardiomegaly, which is largely due to right atrial enlargement, is present. In severe cases (with little functional right ventricle and marked right-to-left shunting), pulmonary vascular markings are decreased. Echocardiography is used to assess right atrial dilatation, anatomical displacement and distortion of the tricuspid-valve leaflets, and the severity of tricuspid regurgitation or stenosis; in addition, the presence and magnitude of interatrial shunting can be determined (by color Doppler imaging or bubble study), as can the presence of associated cardiac abnormalities. Electrophysiologic evaluation is warranted in patients with atrial tachyarrhythmias.

The management of Ebstein's anomaly centers on the prevention and treatment of complications. Prophylaxis against infective endocarditis is recommended. Patients with symptomatic heart failure are given diuretic agents and digoxin. Those with atrial arrhythmias may be treated pharmacologically or with catheter ablation (if an accessory pathway is present). Ablation of accessory pathways has a lower rate of success in patients with Ebstein's anomaly than in those with structurally normal hearts, and the risk of recurrence of arrhythmia is higher. In severely ill infants with Ebstein's anomaly, an arterial shunt from the systemic circulation to the pulmonary circulation is created to increase pulmonary blood flow, thereby decreasing cyanosis. Further surgery to create a univentricular heart (i.e., by the Fontan procedure) may also be considered in neonates.

Repair or replacement of the tricuspid valve in conjunction with closure of the interatrial communication is recommended for older patients who have severe symptoms despite medical therapy. When possible, valve repair is preferable to valve replacement, because it is associated with lower mortality and has fewer long-term complications. The complications of surgery to correct Ebstein's anomaly include complete heart block, persistence of supraventricular arrhythmias, residual tricuspid regurgitation after valve repair, and prosthetic-valve dysfunction.

Ebstein's Anomaly

Ebstein's anomaly is an abnormality of the tricuspid valve in which the septal leaflets and often the posterior leaflets are displaced into the right ventricle and the anterior leaflet is usually malformed, excessively large, and abnormally attached or adherent to the right ventricular free wall. Thus, a portion of the right ventricle is "atrialized" in that it is located on the atrial side of the tricuspid valve, and the remaining functional right ventricle is small (Figure 6). The tricuspid valve is usually regurgitant but may be stenotic. Eighty percent of patients with Ebstein's anomaly have an interatrial communication (atrial septal defect or patent foramen ovale) through which right-to-left shunting of blood may occur.

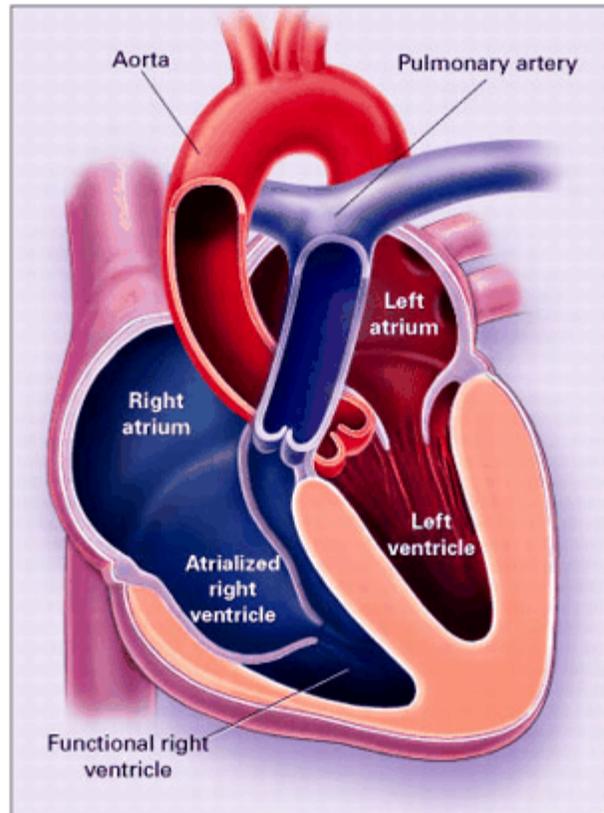


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Repair or replacement of the tricuspid valve in conjunction with closure of the interatrial communication is recommended for older patients who have severe symptoms despite medical therapy. In addition, repair or replacement should be considered for patients with less severe symptoms who have cardiac enlargement, since this condition has a poor prognosis. When possible, valve repair is preferable to valve replacement, because it is associated with lower mortality and has fewer long-term complications. However, when valve replacement is required, a bioprosthesis is preferable to a mechanical prosthesis. The complications of surgery to correct Ebstein's anomaly include complete heart block, persistence of supraventricular arrhythmias, residual tricuspid regurgitation after valve repair, and prosthetic-valve dysfunction.

Transposition of the Great Arteries

With d-transposition of the great arteries (also known as complete transposition), the aorta arises in an anterior position from the right ventricle and the pulmonary artery arises from the left ventricle (Figure 7A). Therefore, there is complete separation of the pulmonary and systemic circulations: systemic venous blood traverses the right atrium, right ventricle, aorta, and systemic circulation, whereas pulmonary venous blood traverses the left atrium, left ventricle, pulmonary artery, and pulmonary circulation. In order for an infant with this condition to survive, there must be a communication between the two circuits. In about two thirds of patients, no other associated cardiac defect is present, so that the ductus arteriosus and foramen ovale allow communication between the two circuits. Infants with this condition have severe cyanosis. The one third of patients with other associated defects that permit

intracardiac mixing (e.g., atrial septal defect, ventricular septal defect, or patent ductus arteriosus) are less critically ill, since they have less severe cyanosis. However, they are at risk for left ventricular failure due to volume overload caused by left-to-right shunting.

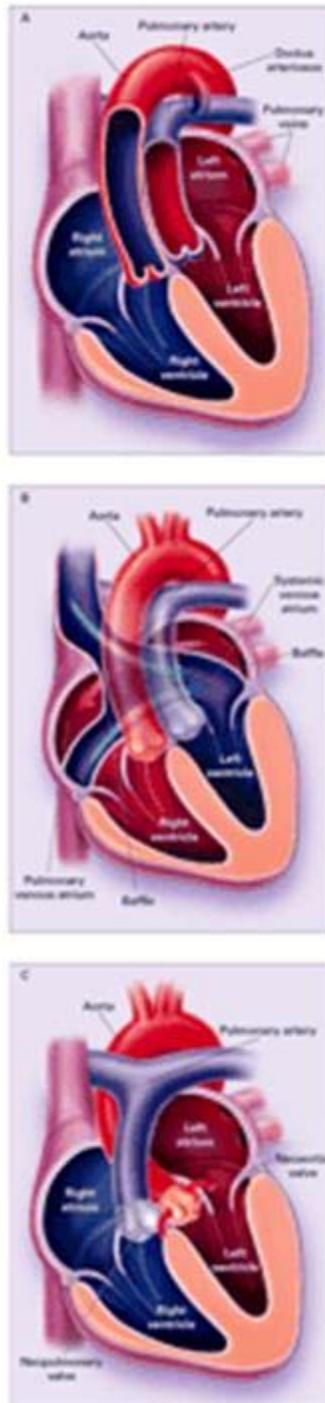


Figure 7. Transposition and Switching of the Great Arteries.

In d-transposition of the great arteries (complete transposition) (Panel A), systemic venous blood returns to the right atrium, from which it goes to the right ventricle and then to the aorta. Pulmonary venous blood returns to the left atrium, from which it goes to the left ventricle and then to the pulmonary artery. Survival is possible only if there is a communication between the two circuits, such as a patent ductus arteriosus. With the "atrial switch" operation (Panel B), a pericardial baffle is created in the atria, so that blood returning from the systemic venous circulation is directed into the left ventricle and then the pulmonary artery (blue arrows), whereas blood returning from the pulmonary venous circulation is directed into the right ventricle and then the aorta (red arrow). With the "arterial switch" operation (Panel C), the pulmonary artery and ascending aorta are transected above the semilunar valves and coronary arteries, then switched (neoaortic and neopulmonary valves).

Patients with complete transposition have cyanosis from birth and often have heart failure in the newborn period. The findings on physical examination are nonspecific. Infants have cyanosis and tachypnea. The second heart sound is single and loud (due to the anterior position of the aorta). In patients with mild cyanosis, a holosystolic murmur caused by a ventricular septal defect may be heard. Likewise, a soft systolic ejection murmur (due to pulmonary stenosis, ejection into the anteriorly located aorta, or both) may be audible. The electrocardiogram shows right-axis deviation and right ventricular hypertrophy (since the right ventricle is the systemic ventricle). Patients with a large ventricular septal defect or patent ductus arteriosus, as well as those with pulmonary stenosis, have left ventricular hypertrophy. The chest radiograph shows cardiomegaly with increased pulmonary vascularity. Classically, the cardiac silhouette is described as being egg-shaped, with a narrow "stalk."

Without intervention, patients with complete transposition have a poor prognosis. Unless intracardiac mixing is improved, progressive hypoxemia and acidosis develop; the mortality rate is 90 percent by six months of age. Infants who have less severe cyanosis (because of a sizable ventricular septal defect or patent ductus arteriosus) fare better in the neonatal period, but pulmonary vascular obstructive disease (due to increased pulmonary blood flow) is more likely to develop than in infants with more severe cyanosis; infants with less severe cyanosis are also more likely to have higher operative mortality and are less likely to have complete repair of their defect.

The immediate management of complete transposition involves creating intracardiac mixing or increasing its extent. This can be accomplished with an infusion of prostaglandin E (to maintain or restore patency of the ductus arteriosus), the creation of an atrial septal defect by means of balloon atrial septostomy (the Rashkind procedure), or both. In addition, oxygen is given to most patients (to decrease pulmonary vascular resistance and to increase pulmonary blood flow), as are digoxin and diuretic drugs (to treat heart failure).

Two surgical procedures have been used in patients with complete transposition of the great arteries. With the initial approach, known as the "atrial switch" operation (the Mustard or Senning operation), the atrial septum was excised, then a "baffle" within the atria was constructed to direct systemic venous blood across the mitral valve into the left ventricle and pulmonary venous blood across the tricuspid valve into the right ventricle (Figure 7B). Thus, physiologic circulation was restored; however, after this procedure was performed, the right ventricle continued to function as the "systemic ventricle." This operation corrected cyanosis and improved survival. The complications associated with it were leakage of the atrial baffle (often clinically inconsequential); obstruction of the baffle (often insidious and frequently asymptomatic); sinus-node dysfunction and atrial arrhythmias, particularly atrial flutter; right (systemic) ventricular dysfunction; and an increased risk of sudden death.

The atrial-switch operation has been replaced by the arterial-switch operation, in which the pulmonary artery and ascending aorta are transected above the semilunar valves and coronary arteries and then switched, so that the aorta is connected to the neo-aortic valve (formerly the pulmonary valve) arising from the left ventricle, and the pulmonary artery is connected to the neopulmonary valve (formerly the aortic valve) arising from the right ventricle (Figure 7C). The coronary arteries are relocated to the neo-aorta to restore normal coronary circulation. This operation can be performed in neonates and is associated with a low operative mortality and an excellent long-term outcome.

Eisenmenger's Syndrome

A patient with Eisenmenger's syndrome has a large left-to-right shunt that causes severe pulmonary vascular disease and pulmonary hypertension, with resultant reversal of the direction of shunting (Figure 8). With substantial left-to-right shunting, the exposure of the

pulmonary vasculature to increased blood flow as well as increased pressure often results in pulmonary vascular obstructive disease. The initial morphologic alterations (medial hypertrophy of the pulmonary arterioles, intimal proliferation and fibrosis, and occlusion of capillaries and small arterioles) are potentially reversible. However, as the disease progresses, the more advanced morphologic changes (plexiform lesions and necrotizing arteritis) are irreversible. The result is obliteration of much of the pulmonary vascular bed, leading to increased pulmonary vascular resistance. As the pulmonary vascular resistance approaches or exceeds systemic resistance, the shunt is reversed.

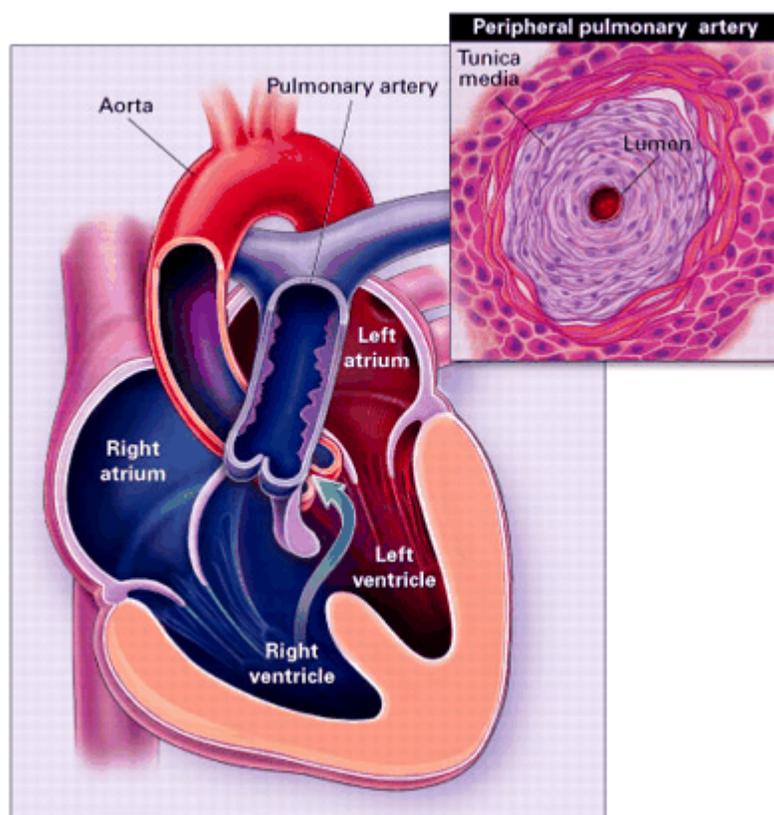


Figure 8. Eisenmenger's Syndrome.

In patients with Eisenmenger's syndrome, in response to substantial left-to-right shunting, morphologic alterations occur in the small pulmonary arteries and arterioles (inset), leading to pulmonary hypertension and the resultant reversal of the intracardiac shunt (arrow). In the small pulmonary arteries and arterioles, medial hypertrophy, intimal cellular proliferation, and fibrosis lead to narrowing or closure of the vessel lumen. With sustained pulmonary hypertension, extensive atherosclerosis and calcification often develop in the large pulmonary arteries. Eisenmenger's syndrome may occur in association with a ventricular septal defect (as

shown), but it also may occur in association with an atrial septal defect or patent ductus arteriosus.

The morphologic changes in the pulmonary vasculature that occur with Eisenmenger's syndrome usually begin in childhood, but symptoms may not appear until late childhood or early adulthood. In many patients, pulmonary congestion in early infancy (a result of the large left-to-right shunt) resolves in later infancy or early childhood as pulmonary vascular resistance increases and the magnitude of shunting decreases. Likewise, the patient may have a murmur in early childhood that disappears (as the pulmonary disease progresses and the magnitude of shunting decreases), leading to the mistaken assumption that the intracardiac communication has closed. Occasionally, patients have no history of pulmonary congestion or a murmur in childhood.

As right-to-left shunting develops, cyanosis appears. Most patients will have impaired exercise tolerance and exertional dyspnea, but these symptoms may be well compensated for years. Palpitations are common and are most often due to atrial fibrillation or flutter. As erythrocytosis due to arterial desaturation develops in patients with Eisenmenger's syndrome, symptoms of hyperviscosity (visual disturbances, fatigue, headache, dizziness, and paresthesias) may appear. Hemoptysis may occur, as a result of pulmonary infarction or rupture of dilated pulmonary arteries, arterioles, or aortopulmonary collateral vessels. Since patients with arterial desaturation have abnormal hemostasis, they are at risk for both bleeding and thrombosis. Cerebrovascular accidents may occur as a result of paradoxical embolization, venous thrombosis of cerebral vessels, or intracranial hemorrhage. In addition, patients with this condition are at risk for brain abscess. Patients with Eisenmenger's syndrome may have syncope owing to inadequate cardiac output or, less commonly, an arrhythmia. Symptoms of heart failure, which are uncommon until the disease is far advanced, portend a poor prognosis. Finally, these patients are at risk for sudden death.

On physical examination, patients have digital clubbing and cyanosis, the severity of which depends on the magnitude of right-to-left shunting. The jugular venous pressure may be normal or elevated, and prominent V waves are seen if tricuspid regurgitation is present. Arterial pulses are small in volume. A right parasternal heave (due to right ventricular hypertrophy) is present,

and the pulmonary component of the second heart sound is loud (and often palpable). The murmur caused by a ventricular septal defect, patent ductus arteriosus, or atrial septal defect disappears when Eisenmenger's syndrome develops. Many patients have a decrescendo diastolic murmur caused by pulmonary regurgitation or a holosystolic murmur caused by tricuspid regurgitation. A right-sided fourth heart sound is usually present. The lungs are clear. Peripheral edema and hepatic congestion are absent unless there is substantial right ventricular dysfunction.

The electrocardiogram shows right ventricular hypertrophy. Atrial arrhythmias may be present, particularly in patients with atrial septal defect. The chest film reveals prominent central pulmonary arteries and decreased vascular markings ("pruning") of the peripheral vessels. The size of the heart is normal in patients with a ventricular septal defect or patent ductus arteriosus, but cardiomegaly (due to right ventricular enlargement) is usually seen in those with atrial septal defect. On transthoracic echocardiography, there is evidence of right ventricular pressure overload and pulmonary hypertension. The underlying cardiac defect can usually be visualized, although shunting across the defect may be difficult to demonstrate by color Doppler imaging because of the low velocity of the jet. Contrast echocardiography permits the location of the shunt to be determined. Catheterization should be performed in any patient with suspected Eisenmenger's syndrome in order to assess the severity of pulmonary vascular disease and to quantify the magnitude of intracardiac shunting. Pulmonary vasodilators — such as oxygen, inhaled nitrous oxide, or intravenous adenosine or epoprostenol — should be administered to permit assessment of the reversibility of pulmonary hypertension.

The rate of survival among patients with Eisenmenger's syndrome is 80 percent 10 years after diagnosis, 77 percent at 15 years, and 42 percent at 25 years. Death is usually sudden, presumably caused by arrhythmias, but some patients die of heart failure, hemoptysis, brain abscess, or stroke. A history of syncope, clinically evident right ventricular systolic dysfunction, low cardiac output, and severe hypoxemia portend a poor outcome.

Patients with Eisenmenger's syndrome should avoid intravascular volume depletion, heavy exertion, high altitude, and the use of vasodilators. Because of high maternal and fetal morbidity and mortality, pregnancy should be avoided. Although no therapy has been proved to

reduce pulmonary vascular resistance, intravenous epoprostenol may be beneficial. Phlebotomy with isovolumic replacement should be performed in patients with moderate or severe symptoms of hyperviscosity; it should not be performed in asymptomatic or mildly symptomatic patients (regardless of the hematocrit). Repeated phlebotomy may result in iron deficiency, which may worsen symptoms of hyperviscosity, since iron-deficient erythrocytes are less deformable than iron-replete erythrocytes.

Patients with Eisenmenger's syndrome who are undergoing noncardiac surgery require meticulous management of anesthesia, with attention to the maintenance of systemic vascular resistance, the minimization of blood loss and intravascular volume depletion, and the prevention of iatrogenic paradoxical embolization. In preparation for noncardiac surgery, prophylactic phlebotomy (usually of 1 to 2 units of blood, with isovolumic replacement) is recommended for patients with a hematocrit above 65 percent in order to reduce the likelihood of perioperative hemorrhagic and thrombotic complications. In general, anticoagulants and antiplatelet agents should be avoided, since they exacerbate the hemorrhagic diathesis.

Lung transplantation with repair of the cardiac defect or combined heart–lung transplantation is an option for patients with Eisenmenger's syndrome who have markers of a poor prognosis (syncope, refractory right-sided heart failure, a high NYHA functional class, or severe hypoxemia). Because of the somewhat limited success of transplantation and the reasonably good survival among patients treated medically, careful selection of patients for transplantation is imperative.

For Guidelines on the Management of Adults With Congenital Heart Disease, please visit [here](#).

Atrial septal defect: case 1

A 43-year-old businessman presents to the accident and emergency department having had palpitations for 12 h. He admits to having increasing shortness of breath over the past two years, and having to rest after climbing three flights of stairs. On examination, his pulse was irregular at a rate of 110 beats per minute, and his jugular venous pulsation (JVP) 6 cm over the sternal angle. He also had a mild right ventricular impulse, a fixed split of S2 (second heart sound) and an ejection murmur 2/6 best heard at the second left intercostal space. Chest radiography showed a dilated right ventricle and increased pulmonary blood flow. The electrocardiogram showed atrial fibrillation. Transthoracic echocardiography confirmed the

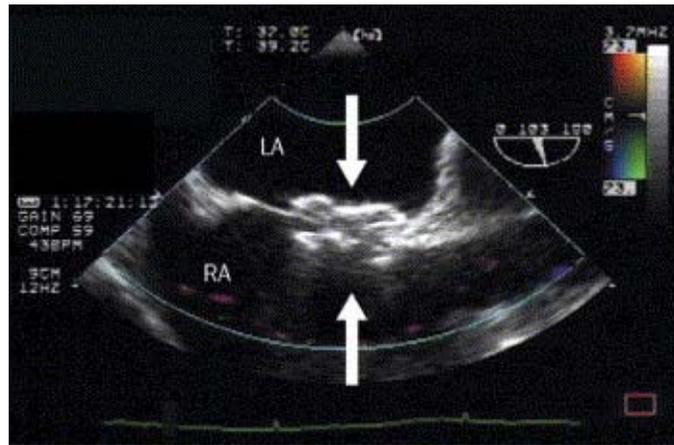
presence of a secundum atrial septal defect with moderate right ventricular dilation.

Atrial septal defects are some of the most common congenital cardiac malformations in adults, representing up to 40% of acyanotic shunt lesions in patients older than 40 years. They arise from either excessive resorption of the septum primum or from deficient growth of the septum secundum, and are occasionally associated with anomalous pulmonary venous connection (about 10%). The degree of left-to-right atrial shunting depends on the size of the defect and the diastolic filling properties of the two ventricles. A substantial shunt ($Q_p/Q_s > 1.5/1.0$) will probably cause symptoms over time, and the movement of such patients will become progressively more restricted with age. Effort dyspnoea is seen in about 30% of patients by their third decade whereas supraventricular arrhythmias (atrial fibrillation or flutter) and right heart failure develop in about 10% of patients by age 40 years.

Treatment

Haemodynamically unimportant atrial septal defects ($Q_p/Q_s < 1.5$) do not need closure, except for the prevention of paradoxical emboli in patients who have had cryptogenic stroke. In the absence of clinically significant pulmonary hypertension, closure is recommended for severe defects ($Q_p/Q_s > 1.5$, or those associated with right ventricular volume overload). In patients without symptoms, indications for closure are somewhat controversial. In those younger than 40 years, severe defects should probably be closed. The appropriate treatment for patients who are older than 40 years is in some dispute, although results of one randomised clinical trial which showed an overall survival benefit among surgical patients would suggest that closure is advisable.

Surgical closure of these defects can be done by primary suture closure or by an autologous pericardial or synthetic patch. Surgical mortality in adults with no pulmonary hypertension should be less than 1%, with a low morbidity related mainly to the development of perioperative arrhythmias (atrial flutter or fibrillation, or junctional rhythm). The use of devices to close defects percutaneously under fluoroscopy and transoesophageal echocardiographic guidance is gaining popularity.



Transoesophageal echocardiographical image of a secundum atrial septal defect after closure with Amplatzer device

LA=left atrium. RA=right atrium. Arrows point at both sides of the Amplatzer device after insertion.

Indications for closure by devices are the same as for surgical closure but selection criteria are stricter than for surgery. Devices are available only for patients with one secundum atrial septal defect and with an adequate septal margin for proper device support. Anomalous pulmonary venous connection precludes the use of this technique. This procedure is safe and effective when done by a skilled cardiologist, and major complications (eg, device embolisation, atrial perforation) arise in less than 1% of patients. Complete closure is achieved in 80% or more of patients. Long-term follow-up data, however, are not available. Notwithstanding, closure by device can be appealing to a patient wishing to avoid the consequences of surgery (general anaesthesia, cardiopulmonary bypass, pain, scar, and time for convalescence), or to a patient believed to be at high surgical risk.

Patent ductus arteriosus: case 2

A 72-year-old woman is referred by her family doctor for atypical chest pain. Risk factors for coronary artery disease are few. Physical examination revealed a grade 1/6 continuous murmur in the left subclavian area. Transthoracic echo confirmed a small colour flow jet area seen between the aorta and the pulmonary artery in the suprasternal view, suggestive of a small patent ductus arteriosus. Left-heart chambers are of normal size and function.

The incidence of isolated persistent patency of the ductus arteriosus is estimated at one in 2000 to one in 5000 births. The ductus arteriosus derives from the left sixth primitive aortic arch and

connects the proximal left pulmonary artery to the descending aorta, distal to the left subclavian artery. Occasionally, the ductus fails to close at birth and presents as a potential clinical problem.

Physiological consequences of a patent ductus arteriosus depend on the degree of left-to-right shunting, which is determined by both the size of the duct and the difference between systemic and pulmonary vascular resistances. A small ductus accompanied by a small shunt does not cause significant haemodynamic disruption but might predispose to endarteritis, especially if accompanied by an audible murmur. A moderate sized duct and shunt put a volume load on the left atrium and ventricle that results in dilation and often dysfunction of the left ventricle, and development of a trial fibrillation, or both. A large duct results initially in left-ventricular volume overload (which disappears after pulmonary hypertension develops), with a progressive rise in pulmonary artery pressure. This rise in pressure leads to high pulmonary vascular resistance and eventually, irreversible pulmonary vascular changes and systemic pulmonary pressures.

Treatment

Closure of a clinically detectable patent ductus arteriosus, in the absence of irreversible pulmonary hypertension, is usually recommended, although controversial, to avoid infective endarteritis. The risk of endarteritis in a patient with a small silent patent ductus arteriosus is regarded as negligible, and closure of such small ducts is therefore not recommended. Over the past 20 years, the effectiveness and safety of transcatheter device closure for ducts smaller than 8 mm has been established. Complete ductal closure is achieved in more than 85% of patients, 1 year after device placement, with a mortality rate of less than 1%. Patients who undergo transcatheter closure avoid general anaesthesia, thoracotomy, postoperative pain, and long convalescence, which makes this procedure very appealing. In centres with appropriate resources and experience, transcatheter device occlusion should be the method of choice for ductal closure.

Surgical closure, by ductal ligation or division, or both, has been done for over 50 years with a slightly better closure rate, but greater morbidity and mortality, than that for closure with device. Surgical closure of a duct should be reserved for patients with ducts larger than 8 mm in diameter or done in centres with no cardiologists specialising in such interventional techniques. Patients who undergo duct closure by device or surgery should be examined periodically and have an echocardiogram to assess possible recanalisation. The risk of late endarteritis from a clinically silent residual shunt, after device implantation or surgical closure,

is unknown,²⁴ and appropriate clinical management of such patients is unclear. Some would recommend closure by device in this patient to eliminate the risk of endarteritis. In our view however, there is no evidence for that approach. Others might choose a non-interventional approach with the use of prophylactic treatment for subacute bacterial endocarditis when needed.

Bicuspid aortic valve: case 3

A 25-year-old woman (Gravida one, pregnancy none, abortion none) is referred at 18 weeks' pregnancy for a heart murmur. The patient is asymptomatic. Her blood pressure is 90/60 mm Hg. On examination she had an ejection click best heard at the apex, a grade 3/6 systolic ejection murmur best heard at the second right intercostal space, with radiation to the carotids with a soft diastolic murmur grade 2/6 heard clearest along the left sternal border. A transthoracic echocardiogram confirmed the presence of a bicuspid aortic valve with moderate aortic stenosis (aortic valve area 1.0 cm^2), and mild-to-moderate aortic regurgitation. Cardiac MRI showed a dilated ascending aorta measuring 50 mm and no evidence of coarctation.



MRI of a dilated ascending aorta in a patient with a bicuspid aortic valve

Arrows indicate the sides of the dilated ascending aorta.

Bicuspid aortic valve has a male preponderance of 4 to 1. This lesion accounts for about half the cases of surgically important isolated aortic stenosis in adults. A bicuspid aortic valve consists of two cusps, often of unequal size, the larger usually containing a false raphe. This lesion generally arises in isolation but is associated with other abnormalities in 20% of patients, the most common of which is coarctation of the aorta and patent ductus arteriosus.

At least half the patients with a bicuspid aortic valve have no complications, although there is always the risk of endocarditis. Mild aortic stenosis or regurgitation from bicuspid aortic valve generally progresses as the patient ages, but the rate is variable. Enlargement of the aortic root resulting from cystic medial changes in patients with bicuspid aortic valve has a high prevalence, and occurs irrespective of altered haemodynamics or age. Aortic dissection is rare.

Treatment

Bicuspid aortic valves need intervention for stenosis when symptoms (exertional dyspnoea, angina, pre-syncope, or syncope) are present and should be considered before pregnancy when the stenosis is severe. Intervention for asymptomatic severe aortic stenosis to allow safe pregnancy is controversial, but severe aortic stenosis has proved to be a risk factor for cardiac decompensation during pregnancy.³⁰ Bicuspid aortic stenosis can be treated with balloon valvuloplasty if the patient is younger than 30 years and if the valve is not calcified. Other treatment options include open aortic valvotomy, or valve replacement with a mechanical valve, a biological valve, or a pulmonary autograft (Ross procedure).

Prophylactic surgery for proximal aortic dilation (>55 mm), in the context of bicuspid aortopathy, seems better than waiting for the aorta to dissect or rupture, although there is no agreement about the diameter at which referral for surgery is appropriate. With the added haemodynamic load of pregnancy (50% increase in cardiac output) and the high oestrogen concentrations that tend to lessen the strength of the aortic wall, any patient with a dilated ascending aorta should be closely monitored during their pregnancy.

This patient would be best treated with close medical follow-up and echocardiography every 3 months or so, to look for signs of progressive aortic root dilation. Treatment with β blockers (if blood pressure is normal) and bedrest, with or without early delivery, should be considered when rapid progression of the ascending aortopathy is seen or the ascending aorta reaches more than 55 mm. Vaginal delivery at term with a rapid (forceps-assisted) second stage labour would

be acceptable if her ascending aortopathy remained stable during pregnancy. Prophylactic replacement of the ascending aortic root with a bioprosthetic aortic valve should be considered before other pregnancies in this patient if her ascending aorta is 55 mm or more or if she has severe aortic stenosis, or both.

Coarctation of aorta: case 4

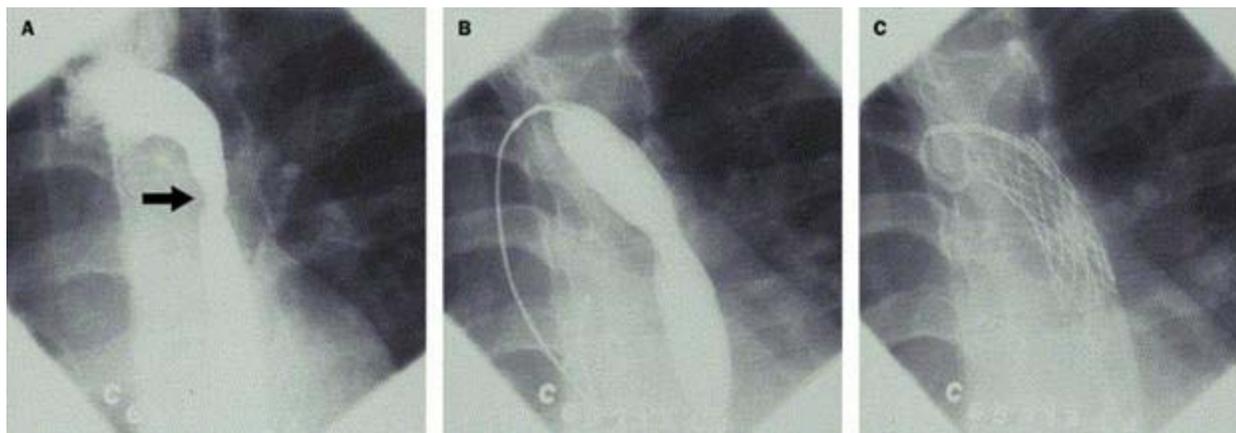
A 44-year-old man with a history of coarctation repair in his childhood is referred for persistent mild systolic hypertension. The patient is otherwise asymptomatic. The blood pressure is 145/80 mm Hg on the right arm, 120/80 mm Hg on the left arm, 120/80 mm Hg on the right leg with a mild radiofemoral delay. MRI confirms the presence of a mild discrete narrowing at the site of previous coarctation repair and cardiac catheter shows a withdrawal gradient of 20 mm Hg between the proximal and distal descending aorta.

Coarctation of the aorta is seen most frequently in men with a ratio of almost 3 to 1. It usually resides in the region of the ligamentum arteriosum. It might be discrete or associated with hypoplasia of the aortic arch and isthmus. Related abnormalities include bicuspid aortic valve in 50-85% of cases. Despite initial successful correction of coarctation, the risk of late systemic hypertension (due to residual or recurrent coarctation, or merely to abnormal aortic wall compliance) in these patients is fairly high. If inadequately controlled, hypertension leads to an increased risk of premature death from late heart failure. Other causes of premature death might be coronary artery disease with or without aortic rupture or dissection. Careful long-term follow-up and aggressive management of patients with hypertension is therefore recommended. Local complications such as re-coarctation and aneurysm formation at the site of previous transcatheter or surgical correction should be identified through periodic chest radiography, echocardiogram, MRI, or spiral CT examinations.

Treatment

All patients with serious coarctation or recoarctation having proximal hypertension, a withdrawal gradient greater than 20 mm Hg at angiography or an echo peak gradient of more than 20 mm Hg in the absence of extensive collaterals or less than 20 mm Hg in their presence, warrant treatment to eliminate the gradient and reduce the risk of long-term complications. Balloon dilatation with stent insertion in patients with native coarctation and recoarctation can be done with good immediate and medium-term results in adolescents and adults and should probably be the procedure of choice when the anatomy is suitable and expert skills are

available. If the anatomy is not suitable (ie, long tunnel-like stenosis) surgery might be needed. After surgical repair of isolated aortic coarctation, the obstruction is usually relieved with minimum mortality (<2%). However, mortality is increased for reoperation (5-15%).

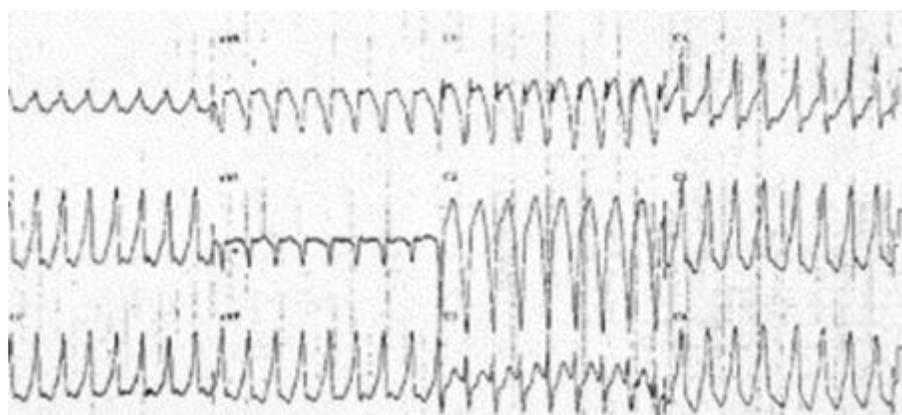


Angiograms of mild re-coarctation of the aorta (A); balloon dilatation and stenting of the re-coarctation segment of the descending aorta (B); and stented descending aorta (C)
Arrow in (A) indicates re-coarctation segment.

This patient has mild re-coarctation of the aorta (peak gradient >20 mm Hg) at the site of previous surgical repair with superimposed mild systolic hypertension. Balloon dilatation and stenting of the re-coarctation segment (if the anatomy is suitable) should be done to reduce proximal systolic blood pressure. The procedure should only be done, however, by experienced cardiologists. Surgical revision or antihypertensive medication, or both, might also be appropriate.

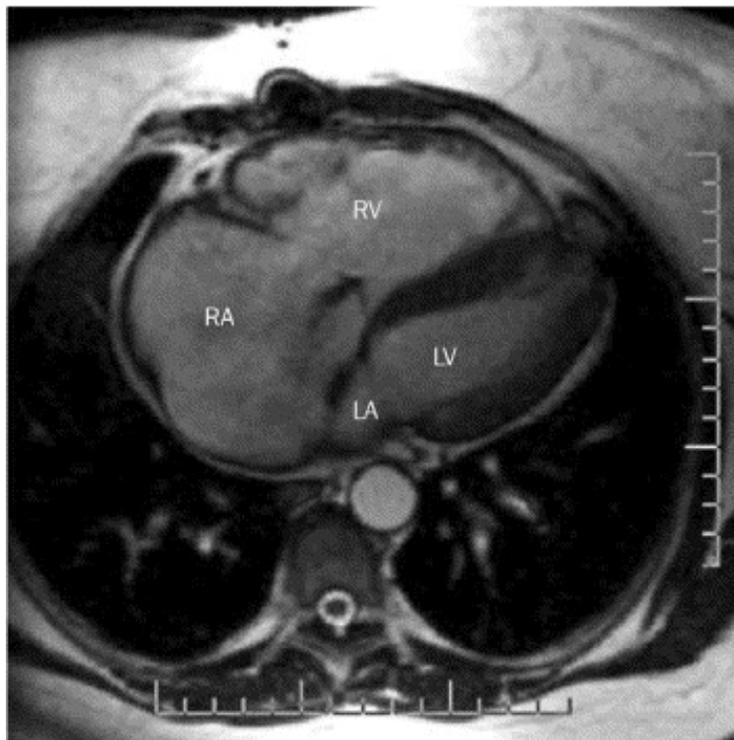
Tetralogy of Fallot: case 5

A 46-year-old woman is brought to the emergency room by ambulance after a witnessed collapse. Sustained ventricular tachycardia was ended and the patient successfully revived.



Electrocardiogram showing ventricular tachycardia in a patient after repair of tetralogy of Fallot

The patient has a history of repaired tetralogy of Fallot in childhood. She was lost to follow-up. Physical examination showed a right ventricular impulse and a low-pitched diastolic murmur grade 3/6 best heard along the left sternal border. Transthoracic echo showed a dilated right ventricle and severe pulmonary regurgitation. Cardiac MRI confirmed a severely dilated right ventricle (right ventricular end-diastolic volume 350 mL—normal range up to 108 mL/m²), severe pulmonary regurgitation, and fairly normal right ventricular systolic function.



MRI showing right right ventricular dilation in a patient after repair of tetralogy of Fallot
LA=left atrium. LV=left ventricle. RA=right atrium. RV=right ventricle.

Electrophysiological studies confirmed the presence of easily inducible ventricular tachycardia from the right ventricular outflow tract, and angiography shows normal pulmonary and coronary arteries.

Tetralogy of Fallot is the most common form of cyanotic congenital heart disease after 1 year of age, with a frequency of almost 10% of all congenital heart disease. The defect is caused by anterocephalad deviation of the outlet septum resulting in four features: (1) a non-restrictive

ventricular septal defect; (2) an overriding aorta (<50% override); (3) obstruction of the right ventricular outflow tract which may be infundibular, valvar, or (usually) both, with or without supra-valvar or branch pulmonary artery stenosis; and (4) consequent right ventricular hypertrophy.

Reparative surgery is usually done early in infancy, by closure of the ventricular septal defect with a Dacron patch and relief of the right ventricular outflow tract obstruction. Obstruction relief might include resection of infundibular muscle and insertion of a right ventricular outflow tract or transannular patch—a patch across the pulmonary valve annulus that disrupts the integrity of the pulmonary valve and causes important pulmonary regurgitation. Significant pulmonary regurgitation is almost always encountered when the transannular patch repair technique is used. Pulmonary regurgitation is usually well-tolerated indefinitely, if mild to moderate. Severe chronic pulmonary regurgitation might be well tolerated for 20 years or more, but could then lead to symptomatic right ventricular dysfunction, and increase the susceptibility to ventricular tachycardia and sudden cardiac death.

Residual obstruction of the right ventricular outflow tract can arise in the infundibulum, the pulmonary valve, and the main pulmonary trunk or branches of the left and right pulmonary arteries, or both branches. Right ventricular dilation in this setting is usually due to longstanding free pulmonary regurgitation, but might result from operative injury. Substantial tricuspid regurgitation might occur because of right ventricular dilation, which then leads to further dilation of the right ventricle.

Atrial tachyarrhythmia arises in about a third of adults and contributes to late morbidity and even mortality. It usually takes the form of atrial flutter or intra-atrial re-entrant tachycardia. Often indicative of haemodynamic trouble (significant right ventricular dilation and dysfunction, increased tricuspid regurgitation), the substrate is most likely a surgical scar in the atria and the trigger, atrial dilation.

Sustained monomorphic ventricular tachycardia is rare, compared with other types, and is highly associated with pronounced right ventricular dilation. The QRS duration from the standard surface ECG has been shown to correlate well with right ventricle size in these patients. A maximum QRS duration of 180 ms or more is a highly sensitive marker for sustained ventricular tachycardia and sudden cardiac death in adults with previous tetralogy repair, although its positive predictive value is low. Dilation of the right ventricle is thought to trigger ventricular tachycardia, whereas surgical scars (near the right ventricular outflow tract or ventricular septal defect patch) form the arrhythmia substrate. The reported frequency of

sudden death, presumably due to arrhythmia, in late follow-up series is 0.5—6% over 30 years, accounting for about a third to a half of late deaths. Older age at repair, severe left ventricular dysfunction, postoperative right ventricular hypertension, transannular patching (causing free pulmonary regurgitation), and an accelerated rate of QRS prolongation are all predictors of sudden death in these patients.

Treatment of complications

Replacement of the pulmonary valve (with either a homograft or porcine bioprosthesis) might be necessary for severe pulmonary regurgitation leading to right ventricular dilation, sustained arrhythmias or symptoms, or both. Such replacement might also be needed for a grossly calcified pulmonary valve. It has a low operative risk and leads to symptomatic improvement. Timely pulmonary valve replacement, before irreversible severe right ventricular dilation and systolic right ventricular dysfunction begins, is a major clinical goal. Concomitant tricuspid valve annuloplasty might also be necessary when at least moderate tricuspid regurgitation is present.

Patients presenting with sustained atrial flutter, atrial fibrillation, or ventricular tachycardia, should undergo a thorough assessment of their haemodynamics and should have residual haemodynamic lesions repaired—eg, significant right ventricular dilation from pulmonary regurgitation with resulting tricuspid regurgitation that needs pulmonary valve replacement and tricuspid valve annuloplasty^{50, 51}. Radiofrequency ablation, after mapping for atrial re-entry tachycardia, now yields better results than before for classic atrial flutter or incisional atrial reentrant tachycardia, or both, and should be done either percutaneously (if there is no need for concomitant surgery) or intraoperatively at the time of surgical repair of underlying haemodynamic lesions. For atrial fibrillation, a biatrial maze procedure should also be considered, and ideally done at reoperation. Likewise, transcatheter (when surgery is unnecessary) or concomitant intraoperative ablative procedures of the ventricular tachycardial pathway should be done when appropriate. Antiarrhythmic medications and the new generation of atrial antitachycardia pacemakers (for supraventricular tachycardia) can be used as adjunct treatments. Patients who were resuscitated after sudden cardiac death and are in need of surgery without a haemodynamic substrate should probably receive an automated implantable cardioverter defibrillator (AICD).

This patient should probably have pulmonary valve replacement since the right ventricle is severely dilated, with repair of the ventricular septal defect patch as well as cryoablation of the ventricular tachycardia focus at the time of surgery. Pulmonary valve replacement will lead to a smaller right ventricle and cryoablation of the ventricular tachycardia focus would eliminate the

arrhythmia substrate and probably prevent a further episode of sudden cardiac death.

Transposition of the great arteries: case 6

A 32-year-old man who had the Mustard procedure for D-transposition of the great arteries presents with increasing exertional dyspnoea and fatigue. Cardiac examination revealed a right ventricular impulse, a loud single S2 (second heart sound) and a holosystolic murmur best heard at the left sternal border. Transthoracic echo confirmed the presence of severe systemic tricuspid regurgitation and severe systemic right ventricular systolic dysfunction (ejection fraction 15%).

In patients with complete transposition of the great arteries, the connections between the atria and ventricles are concordant (normal), and the connections between ventricles and great arteries are discordant. Thus, the pulmonary and systemic circulations are connected in parallel rather than the normal in-series connection. In one circuit, systemic venous blood passes to the right atrium, the right ventricle, and then to the aorta. In the other, pulmonary venous blood passes through the left atrium and ventricle to the pulmonary artery. This situation is fatal unless the two circuits mix. About half of patients with transposition of the great arteries have additional abnormalities, most often a ventricular septal defect.

The most common previously done surgical procedure seen in adults is the atrial switch operation. Patients will have had either a Mustard or a Senning procedure. Blood is redirected at atrial level with a baffle made of Dacron or pericardium (Mustard operation) or with atrial flaps (Senning operation), to achieve physiological correction. Systemic venous return is diverted through the mitral valve into the subpulmonary morphological left ventricle and the pulmonary venous return is rerouted via the tricuspid valve into the subaortic morphological right ventricle. This repair, however, leaves the morphological right ventricle to support the systemic circulation.

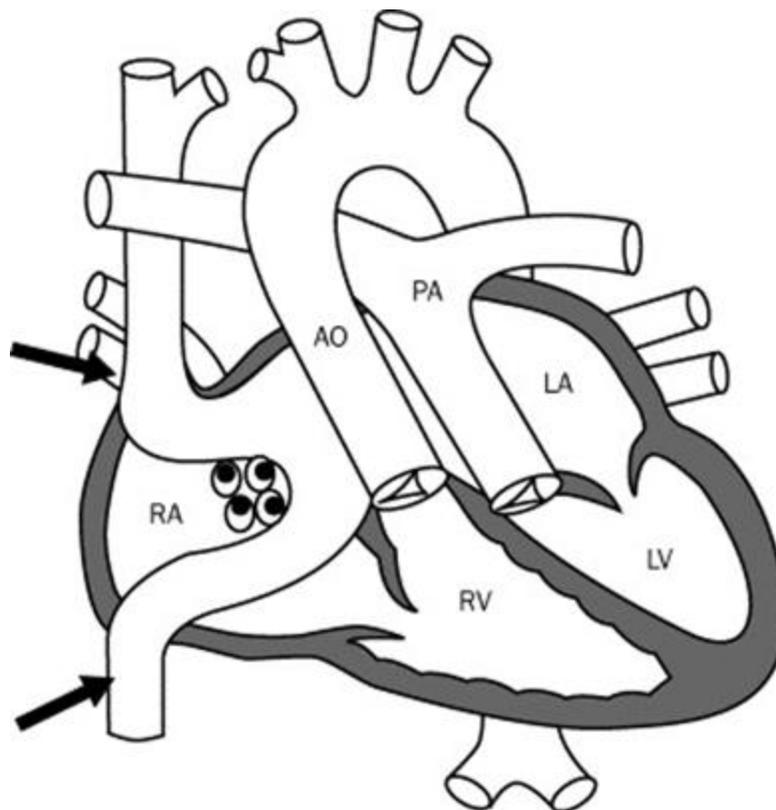


Diagram showing atrial baffle in a patient with D-transposition of the great arteries

RA=right atrium. RV=right ventricle. LA=left atrium. LV=left ventricle. PA=pulmonary artery, AO=aorta. Arrows indicate systemic venous baffle, and black dots pulmonary venous baffle.

After atrial baffle surgery, most patients reaching adulthood will be in New York Heart Association class I—II. During 25 years of follow-up, about half these patients will have moderate systemic dysfunction of the right ventricle with only a few presenting with symptoms of congestive heart failure. Severe systemic tricuspid regurgitation is present in about a third, which exacerbates right ventricular dysfunction. Atrial flutter arises in 20% of patients by age 20 and progressive sinus node dysfunction is seen in half the patients by that time. These rhythm disturbances are thought to be a result of atrial and sinus node damage at the time of atrial baffle surgery. Baffle leak or obstruction can also occur.

The atrial switch operation was gradually replaced by the arterial switch operation (Jatene) in the 1980s, but few of these patients have yet become adults. Blood is redirected at the great artery level by switching the aorta and pulmonary arteries such that the morphological left ventricle becomes the subaortic ventricle and the morphological right ventricle becomes the

subpulmonary ventricle. Reliable data for the clinical outcome in adults after the arterial switch procedure should be available over the next decade. Clinical arrhythmia promises to be less of a problem in this group of patients, but concerns about the development of supra neopulmonary artery stenosis, ostial coronary artery disease, and progressive neoaortic valve regurgitation warrant serial follow-up

Treatment

The benefits of angiotensin-converting enzyme inhibitors in patients with systemic right ventricular dysfunction after an atrial switch have not been established, and are being investigated in a double-blind multicentre randomised trial of ramipril. Occasionally, patients with a failed Mustard or Senning operation might need heart transplantation. Alternatively, a conversion procedure to an arterial switch, after re-training of the left ventricle with a pulmonary artery band, could be considered, but few data for the outcome of such a procedure are available in adults.

The degree of right ventricular dilation and systolic dysfunction should be confirmed by MRI or multiple gated acquisition examinations. An angiotensin-converting enzyme inhibitor with or without other heart failure therapies could be tried in this patient. If no clinical improvement is noted, however, a switch conversion procedure (pulmonary artery banding with left ventricular training followed by pulmonary artery debanding, and arterial switch with take down of the atrial baffle) should be used. Tricuspid valve replacement in this patient would probably lead to worsening of right ventricular function and is not recommended. Cardiac transplantation could also be considered at an appropriate time.

Fontan procedure: case 7

A 26-year-old college student with a diagnosis of tricuspid atresia palliated by the Fontan procedure goes to her local emergency room because of 6 h of continuous rapid palpitations. The patient is haemodynamically stable, and a cardiac monitor reveals atrial fibrillation with a ventricular response of ten beats per minute.

The Fontan procedure is the palliative, non-curative, surgical treatment for patients with univentricular hearts. The principle is diversion of the systemic venous return directly to the pulmonary arteries without the need for a subpulmonary ventricle. Many modifications of this procedure have been described, eg—direct atrio-pulmonary connection, total cavopulmonary connection, and extracardiac conduit. Progressive deterioration of functional status with time is

the rule, with survival at 10 years after the procedure reported to be 60—71%. The most common complications after a Fontan procedure include atrial flutter or fibrillation, right atrial thrombus formation, obstruction of the Fontan circuit, and ventricular dysfunction.

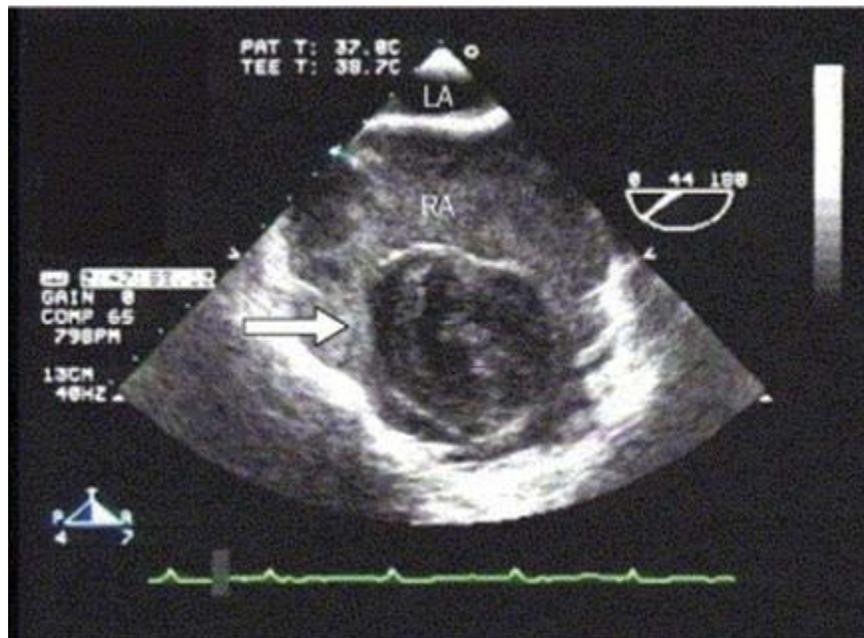
Atrial flutter or fibrillation are common (15—20% at 5-year follow-up), and increases with duration of follow-up. They are associated with serious morbidity (especially the development of atrial thrombi within hours), and can lead to profound haemodynamic deterioration. Such patients need prompt and expert medical attention. The combination of atrial incisions and multiple suture lines at the time of Fontan surgery with increased right atrial pressure and size probably accounts for the high frequency of atrial arrhythmias in these patients. Obstruction of the Fontan connection should be ruled out in all patients presenting with new onset atrial arrhythmias.

The reported frequency of thromboembolic complications in the Fontan circuit varies from 6% to 33%, dependent on the diagnostic method used and the length of follow-up. Right atrial thrombus formation relates to the presence of atrial flutter or fibrillation, right atrial dilation, right atrial smoke (spontaneous echo contrast), and the presence of artificial material used to construct the Fontan circuit.

Part obstruction of the Fontan connection leads to exercise intolerance, atrial tachyarrhythmias, and right-sided heart failure. Sudden total obstruction presents as sudden death. Protein-losing enteropathy is seen in about 2—3% of patients after the Fontan procedure. Patients present with generalised oedema, ascites, pleural effusion, or chronic diarrhoea. The diagnosis is confirmed by low serum albumin and protein and high α 1-antitrypsin stool clearance. The prognosis is poor, with a 5-year survival of 46—59%.

Treatment of complications

In patients with atrial fibrillation or flutter, prompt anticoagulation and transoesophageal echo assessment to rule out atrial thrombi before cardioversion is recommended. Long-term management with antiarrhythmic therapy is successful in less than 50%. Transcatheter atrial ablation can be done in specialised centres, with a 50% success rate. For recalcitrant cases, surgical revision with antiarrhythmic surgery is recommended.



Transoesophageal echocardiogram showing a right atrial clot
 LA=left atrium. RA=right atrium. Arrow indicates right atrial clot.

For established thrombus, thrombolytic therapy or surgical removal of the clot and conversion of the Fontan circuit have been described. Long-term anticoagulation is recommended for patients with known thrombi. Some centres anticoagulate all Fontan circuits for the rest of the patient's life. For patients with Fontan obstruction, surgical revision of the Fontan connection is usually needed. Alternatively, balloon angioplasty with or without stenting can be used when appropriate. Patients with protein-losing enteropathy might be candidates for creation of a fenestration in the atrial septum or revision of the Fontan. Alternatively, subcutaneous heparin, octreotide treatment, and prednisone treatment have also been tried with variable success. No particular treatment seems more successful than any other.

In this patient, who is haemodynamically stable, ventricular rate should be controlled with digoxin or other agents, and prompt anticoagulation with heparin should be started. The patient should be transferred to a highly specialised hospital for transoesophageal echo to rule out right atrial clot before cardioversion. Fontan obstruction as the cause of atrial fibrillation (conduit obstruction with secondary right atrial stretch) should be ruled out either at the time of transoesophageal echo or by cardiac catheterisation. Long-term coumadin is recommended for all patients with Fontan obstruction who have a history of atrial fibrillation. Maintenance of sinus rhythm after cardioversion is probably best achieved with amiodarone, although the success rate is low at 50%. Antitachycardia pacing, catheter ablation or surgical revision, or both, with concomitant biatrial maze procedure are used as second-line treatment in patients

with Fontan obstruction and chronic or paroxysmal atrial fibrillation that is unresponsive to medical management. Other causes of atrial fibrillation such as ethanol binge or hyperthyroidism should also be ruled out.

Підготував Доброродній А.В.

DISEASE OF ESOPHAGUS

ESOPHAGEAL DIVERTICULA

The esophageal diverticula are the sacciform outpouchings of the esophageal wall, which filled with mucus and undigested food.

Etiology and pathogenesis

The conducting pathogenic moment in occurrence of esophageal diverticula is the increase of intraesophageal pressure proximal to muscle sphincters, which gradually results in herniation in weak sites of the esophageal wall. Such mechanism of formation is characteristic for pulsion diverticula. Traction diverticula are formed as a result of paraesophageal inflammatory and sclerotic processes, which tract esophagus to other organs, more often - with the right bronchus. During their motions owing to a traction esophageal diverticula also are gained.

Zenker's diverticula in advanced cases are great in size. There are three stages in their development:

- 1) outpouching of mucosa;
- 2) formation of a globular sack;
- 3) enlargement of diverticulum with further descending in mediastinum.

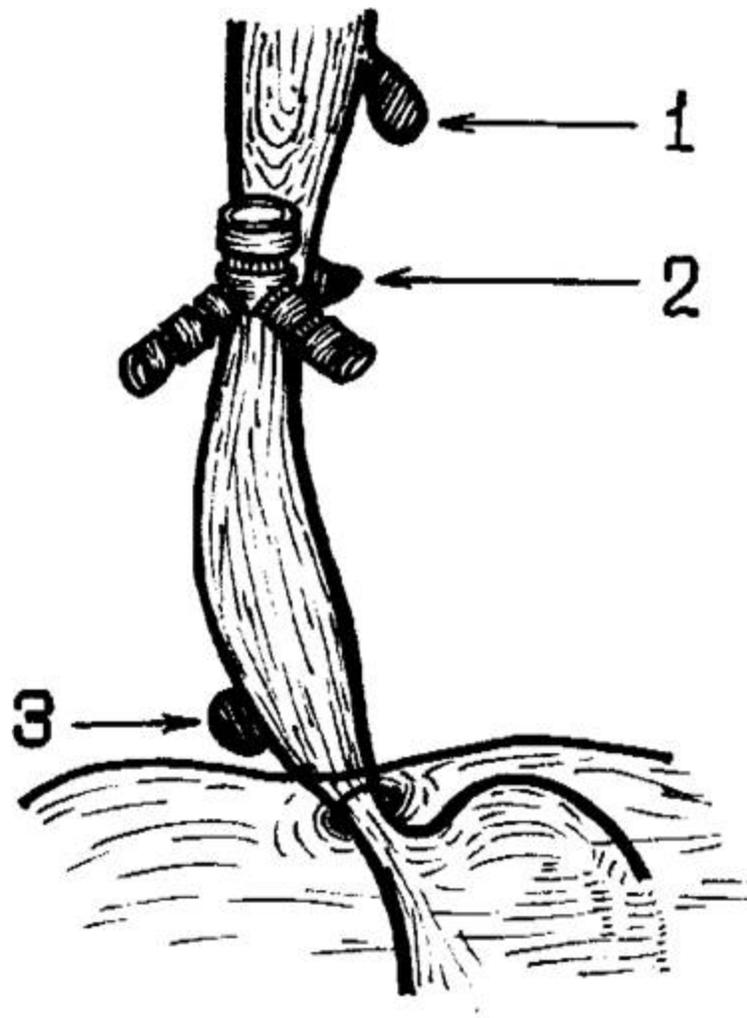
Pathology

The restricted blind herniation of the wall of esophagus could be single or multiple, ring-shaped, cylindrical, oval or sacciform-shaped. The muscle coat atrophies, that makes difficult differentiation between true and false diverticula. The latter caused by inflammatory processes. In such cases the paraesophageal scarring resulting from extrapharyngeal abscesses, mediastinitis, specific and nonspecific inflammatory processes of bifurcational lymph nodes (traction diverticula) are revealed.

The small size of the opening of pouch, for example, in globular diverticula, leads to congestion of contents with the further development of inflammation (diverticulitis erosive, catarrhal, gangrenous, purulent).

Classification

1. According to the origin:
 - a) congenital;
 - b) acquired.
2. According to number:
 - a) single;
 - b) multiple.
3. According to histological structure:
 - a) true (have all layers of esophageal wall);
 - b) false (absent muscular layer of esophageal wall).
4. According to localization:
 - a) pharyngoesophageal (Zenker's);
 - b) bifurcational;
 - c) epiphrenic.
5. According to the clinical course:
 - a) complicated;
 - b) uncomplicated.



Types of diverticula:

- 1 – pharyngoesophageal (Zenker's)
- 2 – bifurcational
- 3 – epiphrenic

Symptomatology and clinical course

Clinical manifestation of the esophageal diverticula, as a rule, connected with the occurrence of complications.

The symptomatology of Zenker's diverticula depends on the stage of development and their size.

A Zenker diverticulum, which is seen in the images below, is formed by the herniation of mucosa through an area of weakness in the posterior wall of the hypopharynx (the Killian triangle).





Sometimes Zenker diverticula are called pharyngoesophageal diverticula because of their close proximity to the cervical esophagus; however, this is somewhat of a misnomer because the diverticula actually arise from the hypopharynx rather than from the esophagus.

Of the diverticula discussed in this article, Zenker diverticula are the most common type to cause symptoms.

Zenker diverticula are an acquired pulsion-type of diverticula that probably develop because of the aging process. They form in the posterior hypopharynx at a point where a defect in the muscular wall, between the inferior pharyngeal constrictor muscle and the cricopharyngeal sphincter (Killian triangle), usually exists.

Zenker diverticula are believed to occur because of an outflow obstruction caused when loss of coordination of the buccal squirt (ie, swallowing movement of the tongue posteriorly with contraction of the oropharyngeal muscles) and opening of the cricopharyngeus (ie, the upper esophageal sphincter) occurs. The noncompliant cricopharyngeus muscle becomes

fibrotic over time.

Oropharyngeal dysphagia, usually to solids and to liquids, is the most common symptom. Retention of food material and secretions in the diverticulum, particularly when diverticula are large, can result in regurgitation of undigested food, halitosis, cough, and even aspiration pneumonia. The patient may note food on the pillow upon awakening in the morning. With very large diverticula, a mass in the neck occasionally can be detected. Cancer rarely has been reported in association with Zenker diverticula.

Salivation, cervical dysphagia, difficult swallowing and cough usually occur in advanced stages of the diverticulum.

The dysphagia is frequently caused by congestion of food in diverticulum. Also a compressible mass in the neck usually on the left side is frequently revealed. The patients should press this mass to swallow the food and sometimes make unusual movements by neck in order to empty the diverticulum. The gurgling sound when the patient is eating and foul-smell from the mouth resulting from decay of undigested food in diverticulum cause the patient to alter social activities.

The sign "of a wet pillow" results from increased salivation and nocturnal discharge of saliva and mucus from the mouth.

Diverticula of the esophageal body are relatively rare. They primarily occur in the middle and distal esophagus (see the image below).



Bifurcational diverticula are usually less 2 cm in size and therefrom rarely complicated and clinically manifested. At its greater size the complications can arise rather frequently and determine the course and manifestations of the disease.

Diverticula that occur in the distal esophagus, in the lower 6-10 cm, are termed epiphrenic diverticula (see the image below).

The epiphrenic diverticula can achieve considerably size, and more frequently complicated by diverticulitis. Being filled with food, such diverticulum can compress cervical organs, and sometimes is complicated with achalasia.



Diverticula of the mid and distal esophagus may have various etiologies. For instance, some diverticula in the mid esophagus are congenital in origin; others are of the traction variety. With the latter, diverticula develop by traction from contiguous mediastinal inflammation and adenopathy, eg, pulmonary tuberculosis and histoplasmosis. The diverticula that develop by traction and adenopathy usually are asymptomatic.

Retention of undigested food in large diverticula occasionally results in regurgitation, nocturnal cough, and aspiration pneumonia.

Occasional epiphrenic diverticula occur in the setting of long-standing peptic esophagitis and strictures, and they rarely are symptomatic. Other rare causes of diverticula of the mid and distal esophagus include iatrogenic surgical injury to the esophagus and Ehlers-Danlos syndrome (weakness of collagen). Perhaps the most common causes of mid esophageal and epiphrenic diverticula are motility disorders of the esophageal body, including achalasia, diffuse esophageal spasm, and hypertensive lower esophageal sphincter.

Dysphagia is the most common symptom associated with mid esophageal and epiphrenic diverticula, although it usually is related more to the underlying motility disturbance than to

the diverticulum per se. However, on occasion, the diverticulum may be responsible for the dysphagia, particularly if it is very large and filled with food or a bezoar. Regurgitation and aspiration may be related to large mid esophageal and epiphrenic diverticula; however, in patients with achalasia, regurgitation and aspiration are more likely to be related to poor esophageal emptying from the underlying motility disturbance (eg, hypertensive lower esophageal sphincter that fails to relax, absence of esophageal body peristalsis).

The diagnosis is confirmed by the findings of barium swallow, and also esophagoscopy.



Pharyngoesophageal (Zenker's) diverticulum)

Variants of clinical course and complications

Diverticulitis. The anginal pain, or the pain in epigastric region, which can resemble stenocardia or gastric disorders, belching, are the chief manifestations. Sometimes observed nausea and vomiting.

The perforation of diverticulum can be directed into pleural space, trachea, bronchus or pericardium. The clinical picture depends on the place of perforation. In part the perforation in trachea or bronchus results in occurrence of esophago-bronchial fistula. Clinically such complication is commonly shown by cough during meal. An everlasting esophago-bronchial fistula can cause the aspiration pneumonia with the further abscessing.

Bleeding from diverticula frequently results from erosion of esophageal mucosa on the

background of diverticulitis. Nevertheless such bleedings, as a rule, are not profuse and rather easily stopped by conservative treatment.

Malignancy rarely occurs and most often as the outcome of recurrent diverticulites.

The diagnostic program

1. Anamnesis and objective examination.
2. General blood and urine analyses.
3. Coagulogram.
4. Chest X-radiography.
5. Roentgenoscopy of esophagus and gastrointestinal tract.

Radiographic studies and upper GI endoscopy detect many esophageal diverticula incidentally because esophageal diverticula often are asymptomatic.

On standard chest radiographs and CT scans, large diverticula of the esophagus and hypopharynx also may manifest as air-filled and/or fluid-filled structures communicating with the esophagus.

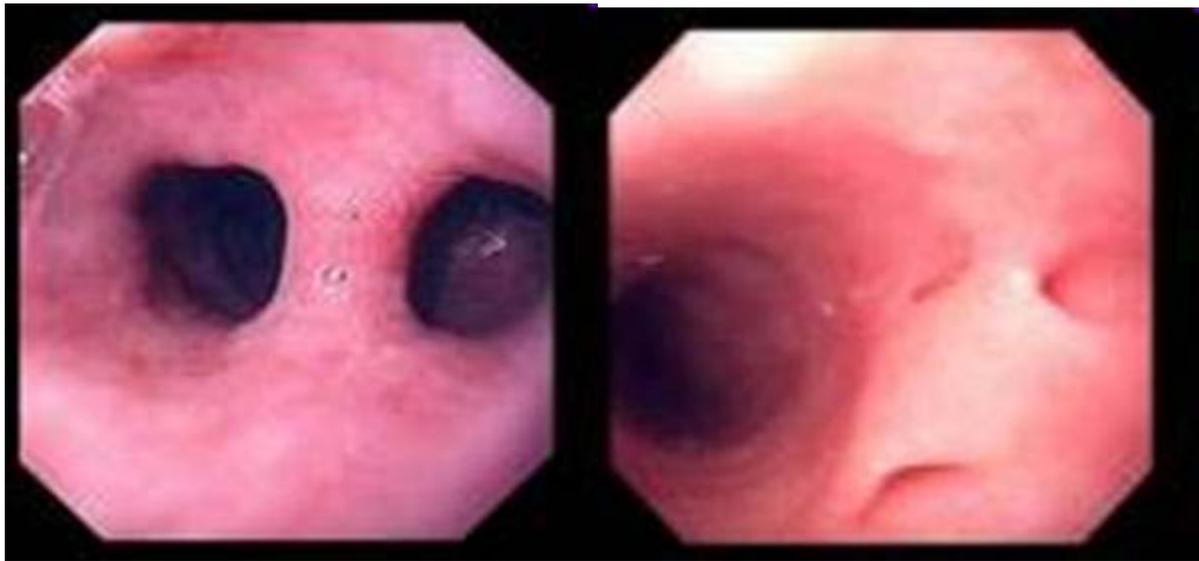
Barium radiography (ie, barium esophagography, barium swallow) generally is the diagnostic procedure of choice. In addition to being excellent at defining the structural appearance of diverticula, barium swallow also may provide clues to underlying motility disturbances that may be involved in diverticular formation. However, if the patient has dysphagia or odynophagia or has alarm symptoms, then upper endoscopy is indicated.

Barium swallow is a useful study in patients who are symptomatic and have mid esophageal and epiphrenic diverticula.

Diagnosis of esophageal intramural pseudodiverticulosis is made best using barium radiography.

Diagnosis of Zenker diverticulum is made best using barium swallow, which should include lateral views of the pharyngoesophageal junction. It also can be made using careful upper endoscopy by an experienced endoscopist.

6. Fibrogastroduodenoscopy.



Esophageal diverticula Fibrogastroduodenoscopy

Differential diagnostics

Functional diverticula (pseudodiverticula). Their clinical manifestations resemble a diffuse idiopathic esophagospasm. Intermittent dysphagia, which usually arises after meal or strong excitements, are the sings of pseudodiverticula. A retrosternal pain, which accompanied them, can result in misdiagnosis of stenocardia.

Stenocardia. It is characterized by pain attacks with irradiation in the left arm and left scapula, feeling of fear. After taking of nitroglycerin the pain, and fear, as a rule, disappear. In contrast with stenocardia, the retrosternal pain caused by a spastic stricture or diverticulum of esophagus, is characterized by feeling of compression deeply inside, which usually more expressed in the back. There is no obvious sensation of fear, irradiation of pain in arm and relief after nitroglycerin. Usually it is accompanied by disturbances of swallowing, sometimes vomiting, after that the pain frequently disappears.

Tactics and choice of treatment

The treatment of pharyngoesophageal diverticula is surgical. Conservative therapy is indicated in case of severe concomitant pathology, the patient's refusal of operation or there are no conditions for its performance. In such cases mechanically sparing diet with washing down of solid food.

Asymptomatic and minimally symptomatic esophageal body diverticula do not require treatment.

In many patients with mid esophageal and epiphrenic diverticula, dysphagia is related to underlying dysmotility; thus, treatment should be directed to the motility disorder when feasible. For instance, achalasia can be treated with pneumatic dilation, botulinum toxin injection into the lower esophageal sphincter, or surgical Heller esophagomyotomy.

Treatment of esophageal intramural pseudodiverticulosis is directed toward underlying strictures or dysmotility.

The bifurcational diverticula require operative treatment only in one patient in ten. The indications for such operation are frequently recurrent diverticulites, bleeding, perforation, esophago-bronchial fistula or suspicion on malignancy.

Treatment of Zenker diverticulum traditionally has been surgical, although the specific operation used still is controversial. Surgical options include diverticulectomy with cricopharyngeal myotomy, diverticular suspension (diverticulopexy) with cricopharyngeal myotomy, and cricopharyngeal myotomy alone.

Consider diverticulectomy when esophageal body diverticula are believed to be the cause of aspiration. An abdominal laparoscopic approach may be feasible for some patients with epiphrenic diverticula. Case reports of endoscopic treatment of giant midesophageal diverticula have been reported. However, patients who are being considered for diverticulectomy should first undergo careful study with barium swallow, flexible endoscopy, and esophageal manometry. Treatment directed at an underlying esophageal motility disorder, such as achalasia, cannot be ignored.

Diverticulectomy usually is not performed by itself because it does not correct the defect in cricopharyngeal function that usually contributes to the formation of a Zenker diverticulum.

While the transcervical approach has been used traditionally, the transoral route using a rigid esophagoscope also may be used.

Good results have been obtained by performing a diverticulotomy using a flexible endoscope and needle-knife papillotome to cut the common wall between the diverticulum and the oropharynx as well as the cricopharyngeus while the patient is consciously sedated. Data suggest that this technique offers good results with a relatively high success rate, but it should be performed in large centers with surgeons who are experienced with this technique. In some variations of this technique, the diverticulum is stapled.

Other novel techniques are being developed. Flexible endoscopic diverticulotomy approaches have been explored using various techniques, including argon plasma coagulation, monopolar coagulation forceps, and needle-knife incision. These techniques typically use a cap or hood attached to the endoscope. The goal of these techniques is the division of the septum

between the diverticulum and the esophagus, thus performing a cricopharyngeal myotomy.

Increased efforts to a laparoscopic approach to repair both epiphrenic diverticula and Zenker diverticula have been explored. The literature supports open surgery and a laparoscopic approach as appropriate methods of repair. The laparoscopic technique uses stapler closure, and multiple case reports cite wound leakage from stapler failure as a complication. With complication rates as high as 20%, a skilled surgeon with experience in this procedure is beneficial. Benefits of the laparoscopic approach include decreased morbidity because of no thoracotomy wounds and chest tubes and a less invasive approach.

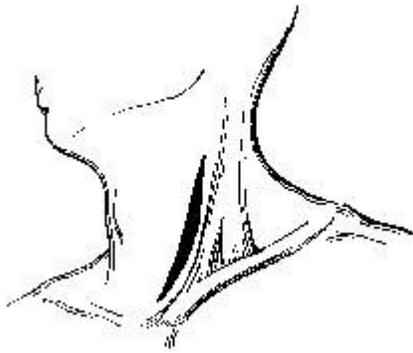
A study of 229 endoscopic diverticulotomies (in 189 patients), conducted by Kos et al, indicated that better results can be achieved using a combination of CO₂ laser and Acuspot in the endoscopic procedure than by employing endoscopic diverticulotomy with electrocautery or with a carbon dioxide (CO₂) laser alone. The investigators reported the following postsurgical results:

- Endoscopy with CO₂ laser
- Dysphagia - Absent following 78.4% of procedures
- Repeat surgery - Required following 19.6% of procedures
- Endoscopy with electrocautery
- Dysphagia - Absent following 72% of procedures
- Repeat surgery - Required following 24.3% of procedures
- Endoscopy with CO₂ laser and Acuspot
- Dysphagia - Absent following 84.6% of procedures
- Repeat surgery - Required following 13% of procedures

Accesses. In order to expose pharyngoesophageal diverticula the cervical access along the anterior border of the sternocleidomastoid muscle is applied; in case of bifurcational diverticula right-sided posterolateral thoracotomy in IV intercostal space is performed; in epiphrenal diverticula – left-sided posterolateral thoracotomy in VII intercostal space .

The essence of the operation consists of the following: the esophagus mobilized proximal and distal to diverticulum; after the exposure the latter is sutured or stapled near its basis and cut off. The line of suturing is covered by muscular layer of esophageal wall.

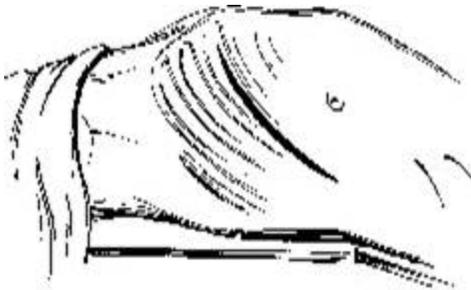
Accesses for diverticula



Cervical

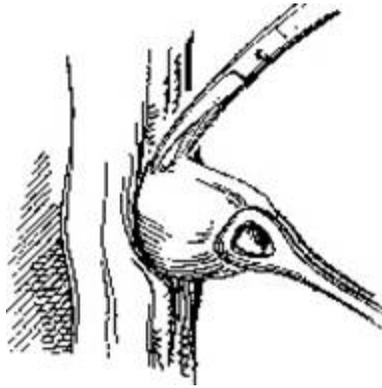


Right-sided posterolateral thoracotomy in IV intercostal space

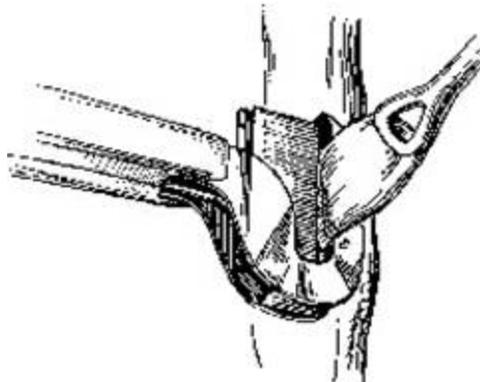


Left-sided posterolateral thoracotomy in VII intercostal space

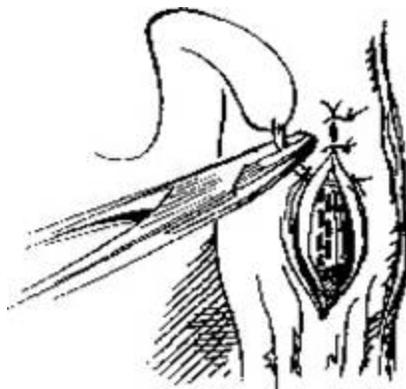
Steps of operation for esophageal diverticulum:



Mobilization of esophageal diverticulum



Suturing of diverticulum base



Suturing of esophageal muscles over the site of diverticulum

ACHALASIA OF THE CARDIA

Achalasia of the cardia is the disease, which is characterized by failure of the lower esophageal sphincter to relax with swallowing.

Etiology

The cause of this disease is still unknown. Among the underlying mechanisms are the psycho-emotional trauma, disturbance of parasympathetic and sympathetic innervation and influence of vegetotrophic substances on muscular fibers.

Pathology

Morphological changes depend on the stage of the disease, character of inflammation and mainly concern nervous and muscle fibers. Thus the phenomena of the thickening of axial cylinders of nervous fibers progressively increase, with the development of their fragmentation and vacuolization. The working hypertrophy of muscular fibers is finished by the dystrophy of myocytes and the development of sclerosis. The latter is contributed by inflammation, mainly of immune character. In final stage a mediastinal pleura, paraesophageal fat and diaphragm consolidate and knitted together.

LES pressure and relaxation are regulated by excitatory (eg, acetylcholine, substance P) and inhibitory (eg, nitric oxide, vasoactive intestinal peptide) neurotransmitters. Persons with achalasia lack nonadrenergic, noncholinergic, inhibitory ganglion cells, causing an imbalance in excitatory and inhibitory neurotransmission. The result is a hypertensive nonrelaxed esophageal sphincter.



Frequency

United States. The incidence of achalasia is approximately 1 per 100,000 people per year.

Sex

The male-to-female ratio of achalasia is 1:1.

Age

Achalasia typically occurs in adults aged 25-60 years. Less than 5% of cases occur in children.

Classification

Four stages of the disease are distinguished:

- 1) functional spasm without esophageal dilation;
- 2) constant spasm with a moderate esophageal dilation and maintained peristalsis;
- 3) cicatricial changes of the wall with expressed esophageal dilation, the peristalsis is absent;
- 4) considerable esophageal dilation with sigmoid-shaped elongation and the presence of erosive changes of esophageal mucosa.

Symptomatology and clinical course

Dysphagia in the onset of the disease wears a temporary intermittent character with further permanent interchange. The passing of food after several swallows delayed on the level of a lower part of breastbone. In some cases during meal the dysphagia arises suddenly without any cause. The majority of the patients with dysphagia swallow better warm or hot food.

Esophageal vomiting (regurgitation) is the outcome of accumulation in esophagus of two and more l. of fluid. In initial stages of the disease the regurgitation can arise during or at once after meal and is accompanied by discomfort pain sensations. In advanced stages observed regurgitation with a rotten smell. The regurgitation can occur during sleeping – the sign "of a wet pillow".

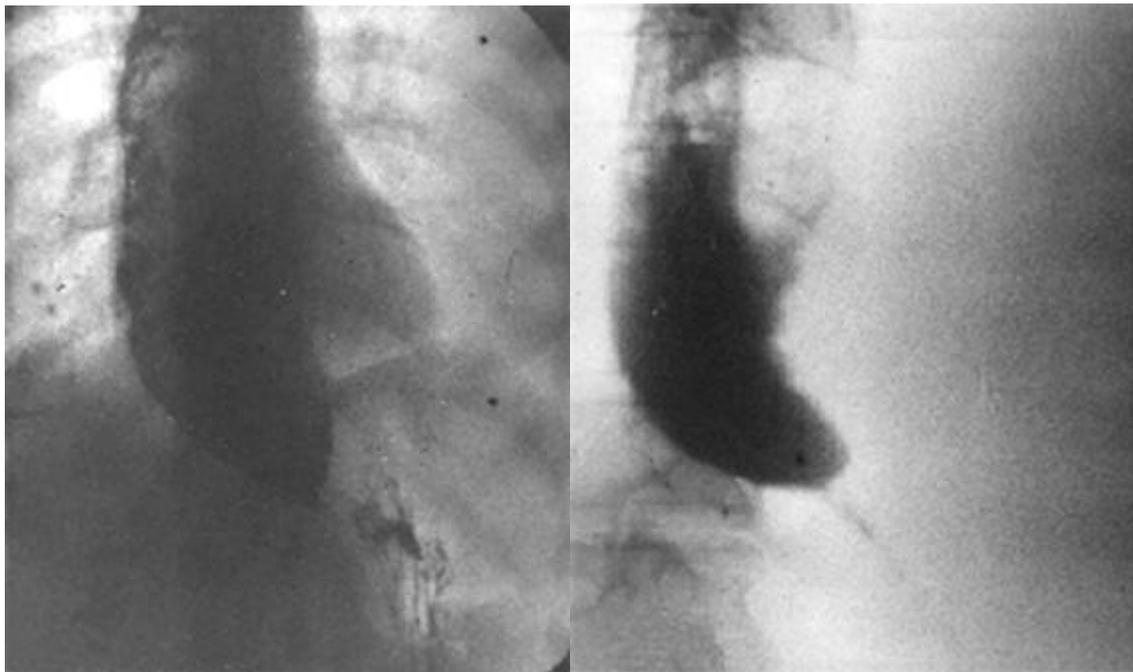
Splashing sounds and gurgling behind breastbone are rarely observed.

The sign of nocturnal cough arises owing to aspiration of fluid from esophagus into trachea. Thereby, the patients try to sleep in a sedentary position.

Pain and sense of tightness in the chest is the result of spasm and esophageal distention. With the developing of esophagitis, the pain wears a burning character.

Loss of weight is the outcome of prolonged disturbed food intake.

It is necessary to consider roentgenological contrast examination with barium swallow as the chief method, which enables to confirm the diagnosis. In the beginning of the disease revealed an inappreciable esophageal dilation and temporary delay of barium above the level of the inferior esophageal sphincter. In advanced stages of the disease observed a considerable esophageal dilation and elongation with a long delay of barium. Contours of a distal constricted part of esophagus described as the "rat tail" or "bird-beak" sign, without filling defects .



Esophageal achalasia

The endoscopic procedure reveals erosive changes of esophageal mucosa and enables to take a biopsy to rule out malignancy. Frequently in advanced stages it is failed to pass by endoscope a constricted part of esophagus and cardia.

Variants of clinical course and complications

The disease is characterized by remittent course with the change of the periods of dysphagia – from inappreciable to intensive. Even in advanced stages in minority of patients observed a latent course with complete disappearance of dysphagia in considerable esophageal dilation and cicatrical stenosis of cardia. Nevertheless later (from several months to several years) there comes an exacerbation of the disease with more severe course.

The bleeding arises owing to complications of erosive esophagitis at long duration of the disease.

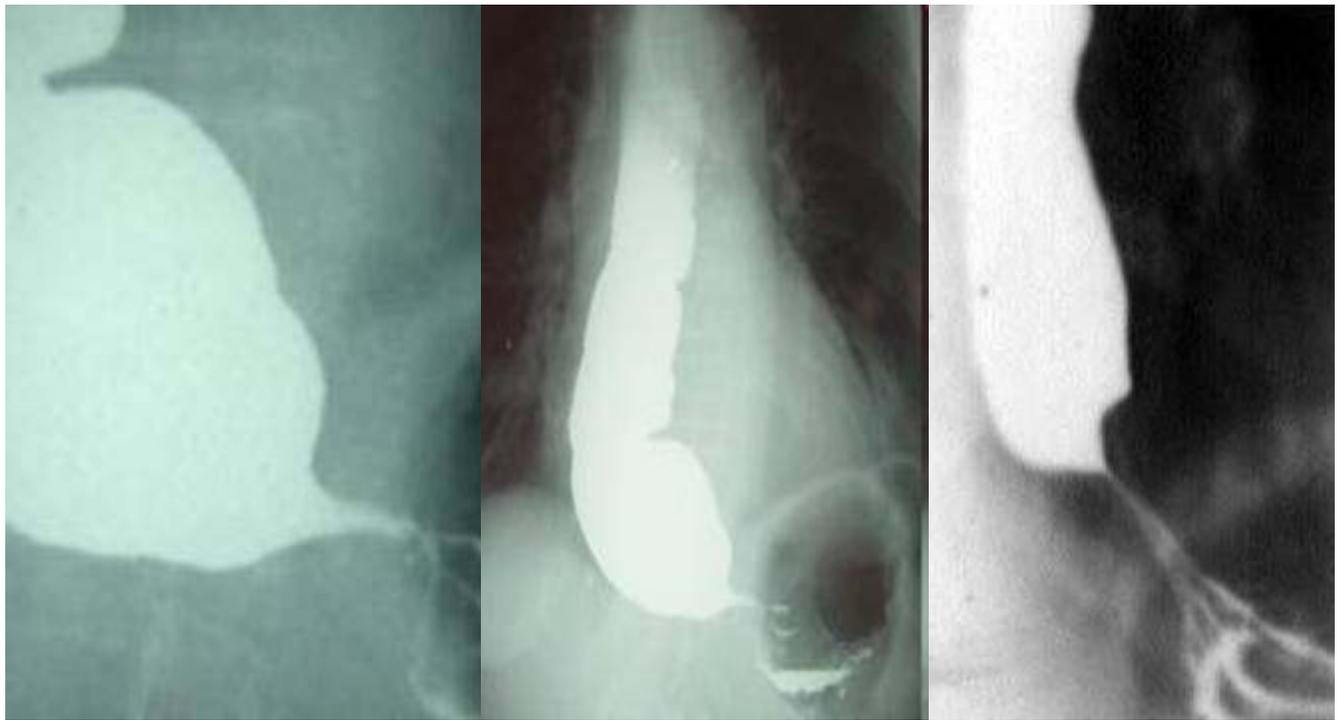
The malignancy occurs in the patients with phenomena of a chronic esophagitis and chronic character of the disease.

Pneumonia, abscesses, bronchiectases, atelectases and pneumosclerosis are frequently the outcomes of decreasing pulmonary excursion which results from compression by dilated esophagus.

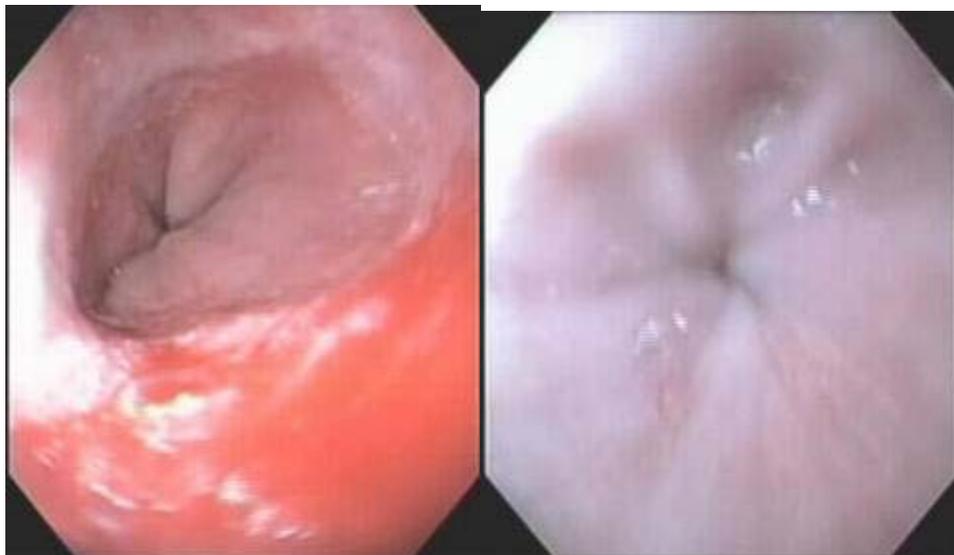
The diagnostic program

1. Anamnesis and physical findings.
2. General blood and urine analyses.
3. Chest X-radiography.
4. Esophagogastroscopy.
5. Contrast roentgenoscopy (barium swallow).

The esophagus appears dilated, and contrast material passes slowly into the stomach as the LES opens intermittently. The distal esophagus is narrowed and has been described as resembling a bird's beak



Esophageal achalasia



Esophageal achalasia

Differential diagnostics

Cancer of the lower part of esophagus and cardial part of stomach. The predominant place in differential diagnostics possesses X-ray examination. As opposed to achalasia, the cancer is characterized by irregular contours of constricted part of esophagus with filling defect. Endoscopic examination and biopsy allows to confirm the diagnosis.

Diaphragmatic hypotonia with inflection of esophagus also can be accompanied with dysphagia. However chest X-radiography enables to find out high standing of the left dome of diaphragm.

Pneumothorax. On the plain chest X-radiography the edge of dilated esophagus can resemble the edge of collapsed lung. Nevertheless in the patient with pneumothorax on the roentgenogram the lung pattern is absent.

Tactics and choice of treatment

The goal of therapy for achalasia is to relieve symptoms by eliminating the outflow resistance caused by the hypertensive and nonrelaxing LES. Once the obstruction is relieved, the food bolus can travel through the aperistaltic body of the esophagus by gravity.

Calcium channel blockers and nitrates are used to decrease LES pressure. Approximately 10% of patients benefit from this treatment. This treatment is used primarily in elderly patients who have contraindications to either pneumatic dilatation or surgery.

Endoscopic treatment includes an intrasphincteric injection of botulinum toxin to block the release of acetylcholine at the level of the LES, thereby restoring the balance between excitatory and inhibitory neurotransmitters. This treatment has limited value. Only 30% of patients treated endoscopically still have relief of dysphagia 1 year after treatment. Most patients need repeated botulinum toxin injections, with short-lasting clinical benefits. This treatment can cause an inflammatory reaction at the level of the gastroesophageal junction, making a subsequent myotomy very difficult. Compared with pneumatic dilation, botulinum toxin injection is associated with significantly higher

symptom recurrence rates at 12 months. Similarly, this treatment modality is less effective than laparoscopic Heller myotomy at 2-year follow-up. Use this treatment in elderly patients who are poor candidates for dilatation or surgery.

Pneumatic dilatation performed by a qualified gastroenterologist is the recommended treatment in those sporadic cases in which surgery is not appropriate. A balloon is inflated at the level of the gastroesophageal junction to blindly rupture the muscle fibers while leaving the mucosa intact. The success rate is 70-80%, and the perforation rate is approximately 5%. If a perforation occurs, emergency surgery is needed to close the perforation and to perform a myotomy. As many as 50% of patients may require more than 1 dilatation. The incidence of pathologic gastroesophageal reflux after the procedure is approximately 30%.

A laparoscopic Heller myotomy is considered by many to be the appropriate primary treatment of patients with achalasia (see Surgical Care). A Heller myotomy and a partial fundoplication performed from the chest (thoracoscopic) have a high incidence of gastroesophageal reflux.

Peroral endoscopic myotomy (POEM) has been recently introduced as a novel approach to achalasia. This procedure is performed under general anesthesia with endotracheal intubation. A 2-cm longitudinal mucosal incision is made on the mucosal surface to create a mucosal entry to the submucosal space. An anterior submucosal tunnel is created downwards, passing the gastroesophageal junction and about 3 cm into the proximal stomach. Once the submucosal tunnel is completed, section of the circular muscle fibers begins 2-3 cm distal to the mucosal entry, approximately 7 cm above the gastroesophageal junction. The myotomy is continued step by step distally until the gastric submucosa is reached, extending approximately 2-3 cm distal to the gastroesophageal junction. After identification and section of the circular muscle fibers of the lower esophagus and proximal stomach, the mucosal entry site is closed with hemostatic clips.

Several potential advantages of POEM compared with laparoscopic Heller myotomy have been proposed. The endoscopic approach should theoretically minimize postoperative pain. A longer myotomy can be performed, extending to the medium third of the esophagus, just below the aortic arch. A concomitant antireflux surgery may not be required because of the selective section of the circular muscle fibers without any dissection at the level of the gastroesophageal junction. In current practice, few data are available regarding clinical outcomes in small series of patients over very short

follow-up periods.

Based on the limited evidence available, POEM seems to be a promising new procedure. However, there are some concerns about this new technique. Endoscopic myotomy is a very demanding procedure, requiring major skills, with a very long learning curve. Even though several studies have reported significant reduction of LES pressure as demonstrated by manometry, the LES pressure was often between 15 and 20 mm Hg. It is known that a predictor of long-term success is an LES pressure around 10 mm Hg. Gastroesophageal reflux is reported in up to 50% of patients after POEM, replicating the results obtained when a myotomy alone was performed without an antireflux operation. Surgical revision in patients with recurrent dysphagia after POEM might be challenging. The presence of adhesions between the submucosal and longitudinal muscular layers after POEM might make the dissection at this level very difficult.

Diet. The food should be semisoft, without pungent relishes, chemically inactive and enriched with proteins, fat, carbohydrates and vitamins.

The medicament treatment should include local anesthetics, spasmolytics, and sedative drugs. Atropin and other anticholinergic agents only increase the spasm of a cardial sphincter, therefrom their usage is undesirable. The medicament treatment results only in temporary relief.

Cardiodilatation is indicated in I-II stage of achalasia. It is one of the chief methods of the treatment of this pathology. The treatment is performed as follows: under local anesthesia by aerosol or solution of anesthetic agent (lidocain, trimecain) through constricted part of the cardia under roentgenological check cardiodilatator (metal, pneumatic) is passed. The air is pumped up in balloon making pressure 200-350 mm H₂O. Repeated procedure is performed in 2-3 days. The course of dilatation includes 3-10 procedures, depending on obtained effect.

Surgical treatment is managed in III-IV stage of achalasia or in recurrence of the disease after dilatation.

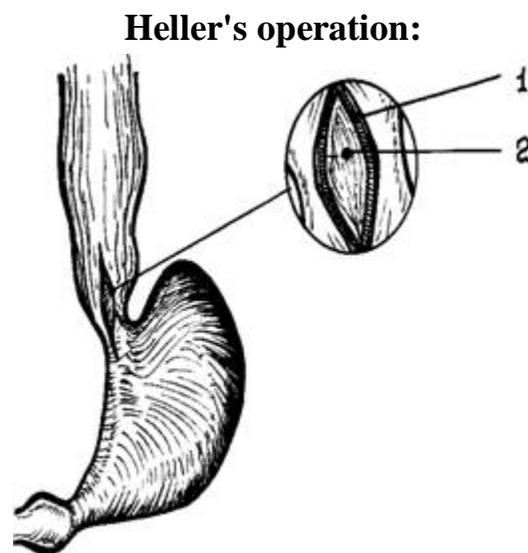
Because of excellent results, a short hospital stay, and a fast recovery time, the primary treatment is considered by many to be a laparoscopic Heller myotomy and partial fundoplication. In the author's experience and in the experience of many authors, this treatment provides a fine balance in relieving symptoms of dysphagia by performing the myotomy and in preventing gastroesophageal reflux by adding a partial wrap. A prospective, randomized study from Vanderbilt University indicated that there is significantly less risk of postoperative reflux following a Heller myotomy plus a partial fundoplication than there is after a Heller myotomy alone. The authors of this study also showed that in patients with achalasia, adding a partial fundoplication not only is more

effective in preventing postoperative reflux but also is more cost-effective at a time horizon of 10 years.

A partial fundoplication added to the myotomy entails better functional results when compared with a total fundoplication, with a lower risk of persistent or recurrent dysphagia. Recently, a multicenter, randomized controlled trial comparing partial anterior (Dor) with partial posterior (Toupet) fundoplication did not find significant differences in terms of postoperative incidence of gastroesophageal reflux. In current practice, a partial anterior fundoplication is more frequently performed since it is simpler to perform and covers the exposed esophageal mucosa.

Minimally invasive surgery for achalasia is performed under general anesthesia with the use of 5 trocars. A controlled division of the muscle fibers (myotomy) of the lower esophagus (5 cm) and the proximal stomach (1.5 cm) is carried out, followed by a partial fundoplication to prevent reflux

Heller's method (esophagomyotomy). Operation is performed through upper median laparotomy or left thoracotomy in VII intercostal space. After exposing of constricted part of esophagus and taking it on tourniquets a muscular layer of anterior wall of esophagus dissected down to mucosa. The myotomy performed from dilated part of esophagus to cardial part of stomach. The complete transection of all muscular layer of esophagus, particularly its circular fibers, is the requirement of relapse prevention.



Myotomia of the cardia (1 – muscles, 2– mucosa)



Shift of stomach fundus to the esophageal cardia



Nissen fundoplication

The defect of a muscular layer of esophageal wall is covered with a gastric fundus or by means of interrupted suture or diaphragmatic flap.

Patients remain hospitalized for 24-48 hours and return to regular activities in about 2 weeks.

The operation relieves symptoms in 85-95% of patients, and the incidence of postoperative reflux is about 20%.

For patients in whom surgery fails, they may be treated with an endoscopic dilatation first. If this fails, a second operation (extending the previous myotomy onto the anterior gastric wall) can be attempted once the cause of failure has been identified with imaging studies. The last resort is to surgically remove the esophagus (ie, esophagectomy).

Treatment options vary for patients with different degrees of illness severity. A study by

Reynoso et al suggests that among hospitalized patients with minor/moderate illness severity, laparoscopic myotomy for achalasia showed comparable or better outcomes than esophageal dilation. For major/extreme illness severity, dilation showed a comparable or better profile for hospitalized patients with achalasia.

Esophagectomy was the standard treatment in patients with achalasia and a markedly dilated or sigmoid-shaped esophagus, with Heller myotomy considered to be ineffective in such cases. However, in a study by Sweet and colleagues of 113 patients with achalasia, the investigators reported that (1) in most of the study's patients, even those with achalasia and a dilated esophagus, a laparoscopic Heller myotomy relieved dysphagia; (2) additional treatment was needed in about 20% of patients; and (3) in the end, 90% of patients had attained good swallowing ability. Esophagectomy was not required in any of the patients to maintain clinically adequate swallowing.

Cowgill et al reported on outcomes in 47 patients more than 10 years after laparoscopic Heller myotomy for achalasia. They found that notable complications were infrequent following the procedure and that no perioperative deaths had occurred. One patient underwent a second myotomy 5 years after the first, because of symptom recurrence. There were 33 surviving patients at the time of the study; the authors reported that the other patients died from causes unrelated to myotomy. Using a Likert scale and a Wilcoxon matched-pairs test to assess patients' symptoms before and after laparoscopic myotomy, Cowgill et al found significant postsurgery decreases in the frequency and severity scores for dysphagia, chest pain, vomiting, regurgitation, choking, and heartburn. They concluded that "the symptoms of achalasia are durably ameliorated by laparoscopic Heller myotomy during long-term follow-up evaluation."

Compared with pneumatic dilatation, laparoscopic Heller myotomy is associated with better results in terms of dysphagia improvement and postoperative gastroesophageal reflux rates, with a significantly lower risk of re-intervention. While the results are similar at a short-term follow-up, long-term follow-up shows that most patients after surgery are asymptomatic, compared with only 50% of patients even after multiple pneumatic dilatations.

Several studies have shown better outcomes after laparoscopic Heller myotomy than pneumatic dilatation in patients younger than 40 years. In addition, previous endoscopic treatment, such as botulinum toxin injection or pneumatic dilatation, may compromise the clinical outcome of laparoscopic Heller myotomy. Higher intraoperative complications rates and poorer long-term outcomes after laparoscopic Heller myotomy

have been reported in several series of patients previously treated with endoscopic treatments. These findings may be related to scar tissue at the level of the gastroesophageal junction, which makes surgical dissection of the anatomic planes much more difficult.

In 2011, Boeckxstaens et al[22] reported the results of a multicenter, randomized trial comparing pneumatic dilatation (95 patients) to laparoscopic Heller myotomy with Dor fundoplication (106 patients) for untreated esophageal achalasia. The perforation rate during pneumatic dilatation and laparoscopic Heller myotomy was 4% and 12%, respectively. Therapeutic success was defined as a drop in Eckardt score below 3. The study showed similar success rates after laparoscopic Heller myotomy (90%) and pneumatic dilatation (86%) over a 2-year follow-up period.

In conclusion, while pneumatic dilatation was considered the main treatment modality for patients with achalasia in the 1980s, with surgery having a secondary role in case of dilatation failure, in current practice pneumatic dilatation should be reserved for when surgical expertise is not available and for the treatment of recurrent dysphagia after myotomy.

One study only has compared in a retrospective fashion POEM and laparoscopic Heller myotomy.[28] Eighteen patients undergoing POEM were compared in a nonrandomized fashion to 55 patients treated by laparoscopic Heller myotomy. No differences were observed in terms of length of the myotomy, complication rate, and length of hospital stay. Veress needle decompression of the pneumoperitoneum was required intraoperatively in 7 (39%) patients undergoing POEM. Treatment success (Eckardt score ≤ 3) after POEM was achieved in 16 (89%) patients at median 6-month follow-up. Six weeks after POEM, routine follow-up manometry and timed-esophagram showed normalization of esophagogastric junction pressures and contrast column heights.

Only long-term follow-up and prospective trials comparing POEM with laparoscopic Heller myotomy and fundoplication will determine the role of this new technique in the treatment of esophageal achalasia.

Helerovsky's method. The operation is indicated for the patients with III-IV stage of the disease in case of considerable esophageal dilation, when performance of Heller's operation is impossible owing to cicatricial changes. However the indication for this operation should be restricted, because of frequent development of expressed esophagitis in postoperative period. The same accesses, as in Heller's operation are applied. Constricted part of esophagus to its dilation exposed and cardial part of stomach is mobilized. Dilated part of the esophagus is

anastomosed with the fundus of stomach.

ESOPHAGEAL STRICTURE

The cicatricial esophageal stenosis can arise owing to chemical, thermal and radial burns, and as a result of esophagitis or peptic ulcers. The most frequent cause of cicatricial strictures is considered to be chemical burns of esophagus, which are usually the result of accidentally or purposely (suicide) drink of acids or alkalis.

Disease processes that can produce esophageal strictures can be grouped into 3 general categories: (1) intrinsic diseases that narrow the esophageal lumen through inflammation, fibrosis, or neoplasia; (2) extrinsic diseases that compromise the esophageal lumen by direct invasion or lymph node enlargement; and (3) diseases that disrupt esophageal peristalsis and/or lower esophageal sphincter (LES) function by their effects on esophageal smooth muscle and its innervation.

Many diseases can cause esophageal stricture formation. These include acid peptic, autoimmune, infectious, caustic, congenital, iatrogenic, medication-induced, radiation-induced, malignant, and idiopathic disease processes.

The etiology of esophageal stricture can usually be identified using radiologic and endoscopic modalities and can be confirmed by endoscopic visualization and tissue biopsy. Use of manometry can be diagnostic when dysmotility is suspected as the primary process. Computed tomography (CT) scanning and endoscopic ultrasonography are valuable aids in the staging of malignant stricture. Fortunately, most benign esophageal strictures are amenable to pharmacologic, endoscopic, and/or surgical interventions.

Because peptic strictures account for 70-80% of all cases of esophageal stricture, peptic stricture is the focus of this article. A detailed discussion of possible benign and malignant processes associated with esophageal stricture and its management is beyond the scope of this article.

Frequency

United States Gastroesophageal reflux affects approximately 40% of adults. Esophageal strictures are estimated to occur in 7-23% of untreated patients with reflux disease.

Gastroesophageal reflux disease accounts for approximately 70-80% of all cases of esophageal stricture. Postoperative strictures account for about 10%, and corrosive strictures account for less than 5%.

The overall frequency of initial and subsequent dilations for peptic stricture appears to have decreased gradually since the introduction of proton pump inhibitors (PPIs) in the market

in 1989. This has been borne out by data at the author's institution and in 2 large community hospitals in Wisconsin. It is also in keeping with the general experience of gastroenterologists in the United States.

Mortality/Morbidity

The mortality rate of peptic strictures is not increased unless a procedure-related perforation occurs or the stricture is malignant. However, the morbidity for peptic strictures is significant.

Most patients undergo a chronic relapsing course with an increased risk of food impaction and pulmonary aspiration.

Frequently, coexistent Barrett esophagus and its attendant complications occur.

The need for repeated dilatation potentially increases the risk of perforation.

Race

Peptic strictures are 10-fold more common in whites than blacks or Asians. However, this is controversial as a recent retrospective study reported comparable frequencies between blacks and non-Hispanic whites. The authors reported that distribution of reflux esophagitis and grade and frequency of reflux-related esophageal ulcer and hiatal hernia were also similar in non-Hispanic whites and blacks. However, heartburn was more frequent and nausea/vomiting less frequent in non-Hispanic whites compared with blacks with erosive esophagitis or its complications.[2]

Sex

Peptic strictures are 2- to 3-fold more common in men than in women.

Age

Patients with peptic stricture tend to be older, with a longer duration of reflux symptoms.

Pathology

Peptic esophageal strictures are sequelae of gastroesophageal reflux -induced esophagitis, and they usually originate from the squamocolumnar junction and average 1-4 cm in length.

Two major factors involved in the development of a peptic esophageal stricture are as follows:

Dysfunctional lower esophageal sphincter: Mean LES pressures are lower in patients with peptic strictures compared with healthy controls or patients with milder degrees of reflux disease. A study by Ahtaridis et al showed that patients with peptic esophageal strictures had a

mean LES pressure of 4.9 mm Hg versus 20 mm Hg in control patients.[1] An LES pressure of less than 8 mm Hg appeared to correlate significantly with the presence of peptic esophageal stricture without any overlap in controls.

Disordered motility resulting in poor esophageal clearance: In the same study, Ahtaridis et al demonstrated that 64% of patients with strictures had motility disorders compared with 32% of patients without strictures.

Other possible associated factors include the following:

Presence of a hiatal hernia: Hiatal hernias are found in 10-15% of the general population, 42% of patients with reflux symptoms and no esophagitis, 63% of patients with esophagitis, and 85% of patients with peptic esophageal strictures. This suggests that hiatal hernias may play a significant role.

Acid and pepsin secretion: This does not appear to be a major factor. Patients with peptic esophageal strictures have been demonstrated to have the same acid and pepsin secretion rates as sex-matched and age-matched controls with esophagitis but no stricture formation. In fact, some authors believe that alkaline reflux may play an important role.

Gastric emptying: No good evidence suggests that delayed emptying plays a role in peptic esophageal strictures.

The morphological changes in esophageal burns pass four stages:

I – stage of acute esophagitis. Lasts from one to two months. It is characterized by edema and divestment of necrotic tissues. This stage is hazardous for erosive bleedings.

II – stage of chronic esophagitis. The ulcers of different sizes with granulating tissue in their bottom, focal constrictions of esophageal lumen are formed.

III – stage of cicatrical stricture of esophagus. Begins from 2-4th month and lasts to 2 years.

IV – stage of late complications. Develops in two years after the burn and is characterized by formed cicatrical stricture of esophagus.

Classification

According to the clinical course:

I. The period of acute manifestation has three degrees of severity:

1 - mild;

2 - moderate;

3 - severe.

II. The latent period (false improvement).

III. The period of cicatrization.

According to the depth of lesion:

I degree – superficial burn with the damage of epithelial layer of esophagus;

II degree – the burn with the damage of entire mucosa of esophagus;

III degree – the burn damage of all layers of esophagus;

IV degree – the spread of postburn necrosis on paraesophageal tissue and adjacent organs.

Symptomatology and clinical course

The clinical signs of esophageal burn directly depend on the period of lesion and degree of gravity.

Acute period

The mild degree of clinical course manifests by satisfactory general state of the patient. At swallowing the patient feels a moderate pain, sometimes salivation, hoarseness. Roentgenologically – the lumen of esophagus without changes, with free passage of barium, the mucous folds with regular contours, but in some places it is possible to observe its graduation. The esophageal peristalsis is maintained. As a rule, in 5-7 days the clinical manifestations of the burn disappear.

The moderate degree of gravity of acute period is characterized by acute substernal and pharyngeal pain at swallowing, repeated vomiting, feeling of fear and excitement. Tachycardia – 120-130 beats/min. The body temperature rises to 39°C. Oliguria develops frequently. Roentgenologically – the esophagus dilated, but in some places can be constricted as a result of edema or spasm. The lumen is filled with considerable amount of slime. The contours of mucosal folds are irregular, the peristalsis is weakened or absent at all. If there will be no complication, in 10-15 days the clinical manifestation of the disease disappear and general state of the patient is improved.

Severe degree is characterized by the clinic of shock. Pulse of weak filling and tension, expressed tachycardia, acute substernal pain. The excitement of the patient is accompanied by feeling of fear, further transmits into adynamia, frequently the patients are unconsciousness. The skin is pale, covered with cold sweat. One patient in four except esophageal burns, suffers from burn of the stomach. The clinical course of the disease is worsened by oliguria, which can

transfer into anuria, and also occurrence of other complications. It determines the unfavorable forecast.

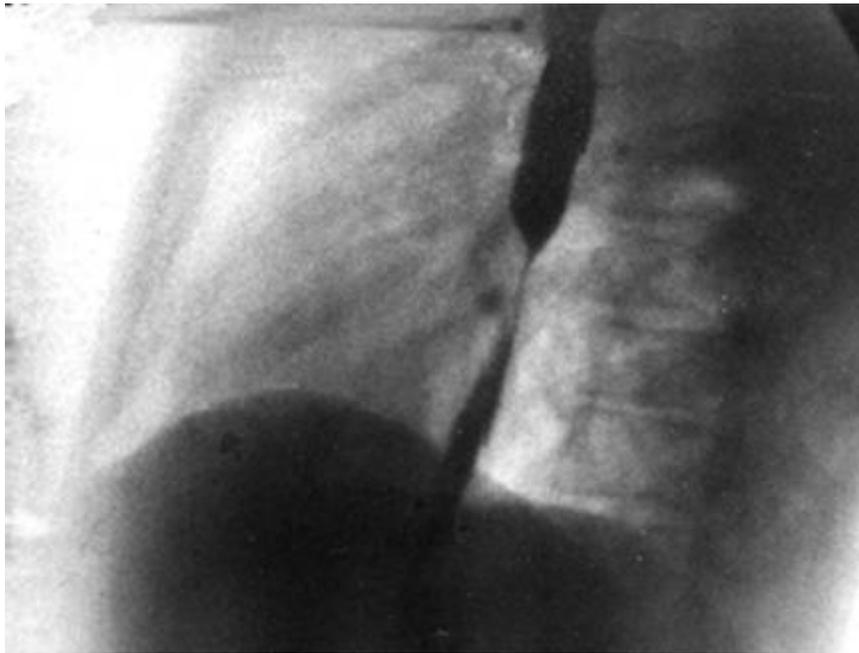
The barium swallow in the majority of patients is problematic. Nevertheless if the general state of the patient allows to carry out it, on the first day after a burn already have been observed expressed manifestations of esophagitis: the esophagus dilated, mucosal folds are failed to reveal. The deposits of destructive changed tissues in the lumen of esophagus resemble the picture of filling defects; the peristalsis is absent, complete atony.

Latent period

This period is connected with replacement of necrotic tissues by granulations. The general state of the patient is improved. The acute signs disappear. The patient swallows freely, without feeling of discomfort at passage of food.

Period of cicatrize

It frequently lasts from 1 to 12 months. It is connected with replacement of granulations by cicatricial tissue that results in progressing of esophageal stricture and disturbance of swallowing at first of solid, and further of liquid food. Such strictures develop at the orifice of esophagus, in projection of tracheal bifurcation and in the place of gastroesophageal juncture. The passage of food through the constricted regions of esophagus is possible at first only due to careful grinding and watering, but further it is inefficient. Thereafter food delay in esophagus, choking, salivation, belching and vomiting develop. If the stricture is located in the lower part of esophagus, the vomitis can be of putrefactive character. Progressing loss of weight observed, which without correction can transfer into cachexia. The level and degree of the stricture, its extension circumstantiated after X-ray examination.



Cicatricial stricture of the lower esophagus

Variants of clinical course and complications

The esophageal burns in 30 % of cases are accompanied by **disturbances of valvular function of epiglottis**. It in reinforced salivation causes the aspiration of fluid in trachea, infection of airways, development of bronchitis and pneumonia.

In 25 % of the patients the esophageal burn combined with gastric burn, mainly of its pyloric part. It can result in lot of complications, which sometimes prevail on the manifestations of esophageal burn. Especially dangerous among such complications of the early period is the gastric bleeding and perforation. The postburn stricture of pylorus belongs to the late complications.

Gastrointestinal bleedings usually occur in 3-10 days after the burn and are characteristic for the stage of formation of ulcers and granulations. Despite the rare arrosion of major vessels in such pathology, these bleedings are accompanied by considerable hemorrhage, because bleeds a considerable surface of the mucous membrane of esophagus or stomach.

Mediastinitis is mostly observed in deep burns of III-IV degree. It can be the outcome of perforation of esophagus or of hematogenic or lymphogenic spread of infection to mediastinum. The clinical manifestations mainly caused by a septic state of the patient and severe intoxication. The fever, difficult respiration, chest pain and tachycardia to 130 and more beats/min are observed. Temperature rises to 39-40°C and has hectic character.

Roentgenologically observed distention of the mediastinal shadow, sometimes detached mediastinal pleura. Pleurisy, pericarditis and lung abscesses can arise as the early complications of esophageal burns. To the late complications, except cicatricial stricture, it is necessary to regard tracheo-esophageal and broncho-esophageal fistula, and also malignancy of the cicatricial changed esophagus.

History

Patients with peptic strictures may present with heartburn, dysphagia, odynophagia, food impaction, weight loss, and chest pain.

Progressive dysphagia for solids is the most common presenting symptom. This may progress to include liquids.

Atypical presentations include chronic cough and asthma secondary to aspiration of food or acid.

The clinician cannot rely on the presence or absence of heartburn to definitely determine whether dysphagia is secondary to a peptic esophageal stricture.

- Of patients with peptic esophageal strictures, 25% have no previous history of heartburn.
- Heartburn may resolve with worsening of a peptic stricture.
- Approximately two thirds of patients with adenocarcinoma in Barrett esophagus have a history of long-standing heartburn.
- The abnormal esophageal motor activity in achalasia can produce a heartburn sensation.

Important points regarding dysphagia

- The obstruction is usually perceived at a point that is either above or at the level of the lesion.
- Dysphagia for solids and liquids simultaneously should alert the clinician to the possibility of a motility disorder such as achalasia or collagen vascular disorders.
- Dysphagia secondary to a Schatzki ring is usually intermittent and nonprogressive.
- Dysphagia for solids and liquids early in the course of disease should alert the clinician to the possibility of achalasia as an etiology of a peptic esophageal stricture.
- Benign esophageal strictures usually produce dysphagia with slow and insidious

progression (ie, months to years) of frequency and severity with minimal weight loss.

- Malignant esophageal strictures result in a rapid progression (ie, weeks to months) of severity and frequency of dysphagia and are associated frequently with significant weight loss.

Determining whether the patient takes any medications known to cause pill esophagitis is important.

Determining whether a history of collagen vascular disease or immunosuppression exists may provide clues to the underlying etiology.

Physical

Physical examination frequently does not provide clues to the cause of dysphagia.

Assessing the patient's nutritional status is important.

Patients with collagen vascular diseases may exhibit joint abnormalities, calcinosis, telangiectasias, sclerodactyly, or rashes.

The presence of atypical gastroesophageal reflux disease may be suggested by hoarse voice, posterior oropharyngeal erythema, diffuse dental erosions, wheezing, or epigastric tenderness.

Patients with adenocarcinoma of the gastroesophageal junction may have left supraclavicular lymphadenopathy (Virchow node).

Causes

Proximal or mid esophageal strictures

- Caustic ingestion (acid or alkali)
- Malignancy
- Radiation therapy[3, 4]
- Infectious esophagitis -Candida, herpes simplex virus (HSV), cytomegalovirus (CMV), human immunodeficiency virus (HIV)
- Acquired immunodeficiency syndrome (AIDS) and immunosuppression in patients who have received a transplant
- Medication-induced stricture (pill esophagitis) - Alendronate, ferrous sulfate, nonsteroidal anti-inflammatory drugs (NSAIDs), phenytoin, potassium chloride, quinidine, tetracycline, ascorbic acid.[5] Drug-induced esophagitis often occurs at

the anatomic site of narrowing, with the middle one-third behind the left atrium predominating in 75.6%. [6]

- Diseases of the skin - Pemphigus vulgaris, benign mucous membrane (cicatrical) pemphigoid, epidermolysis bullosa dystrophica
- Graft versus host disease
- Idiopathic eosinophilic esophagitis
- Extrinsic compression
- Squamous cell carcinoma
- Sequela of endoscopic submucosal dissection for superficial squamous cell neoplasms. [7]
- Miscellaneous - Trauma to the esophagus from external forces, foreign body, surgical anastomosis/postoperative stricture, congenital esophageal stenosis

Distal esophageal strictures

- Peptic stricture - Gastroesophageal reflux disease, Zollinger-Ellison syndrome
- Adenocarcinoma
- Collagen vascular disease - Scleroderma, systemic lupus erythematosus (SLE), rheumatoid arthritis
- Extrinsic compression
- Alkaline reflux following gastric resection
- Sclerotherapy and prolonged nasogastric intubation
- Crohn disease

The diagnostic program

1. Anamnesis and physical findings.

2. X-ray examination of esophagus and stomach.

- Barium esophagram provides an objective baseline record of the esophagus before medical therapy or endoscopic intervention.
- This study also provides information about the location, length, and diameter of the stricture and the smoothness or irregularity of the esophageal wall (road map).
- The information obtained can complement endoscopic findings.
- Lesions, such as diverticula and paraesophageal hernias, that potentially may lead to increased risk of complications during endoscopy can be identified.
- This study may be more sensitive than endoscopy for detection of subtle

narrowings of the esophagus such as those caused by rings and peptic strictures that are greater than 10 mm in diameter.

- This study has 100% sensitivity with luminal diameter less than 9 mm, and 90% sensitivity with luminal diameter greater than 10 mm.

3. Chest X-radiography.

- Chest radiograph, posteroanterior (PA) and lateral: Chest radiography should be used as an adjunct if extrinsic compression is considered a possible etiology of esophageal stricture.

CT scanning

- CT scans can be used to stage malignancies that produce esophageal strictures.
- Accuracy in estimating the depth of tumor invasion is 60-69%.
- Accuracy in determining spread to other organs is 82%.

4. Endoscopic examination of esophagus, stomach and duodenum.

- This procedure can be used to establish or confirm the diagnosis of esophageal stricture, to seek evidence of esophagitis, to exclude malignancy, to obtain biopsy and brush cytology specimens, and to implement therapy.
- EGD is more sensitive than barium esophagram in the identification of subtle mucosal lesions.
- Subtle strictures may be missed when smaller and thinner endoscopes are employed, especially in the setting of minimal sedation.

5. General blood analysis.

6. Coagulogram.

7. Biochemical investigation of plasma.

- Complete blood cell (CBC) count: Usually, the results of a CBC are within the reference range; however, anemia may develop due to chronic bleeding from severe esophagitis or carcinoma.
- Liver profile: Usually, the findings are within the reference range; however, the liver profile may be abnormal if metastatic disease in underlying malignancy is present.
- Complete metabolic panel: This study may allow assessment of the patient's nutritional status, especially in conjunction with weight loss.

8. Histologic Findings

- Initial histologic changes in the peptic stricture process include edema, cellular

infiltration, basal cell hyperplasia, and vascular changes with a slight increase in type III collagen deposition on healing.

- If untreated, the process can lead to progressive inflammation and ulceration involving the submucosa and muscularis mucosa. This can lead to damage of the muscular layer and the intrinsic nervous system of the esophagus, resulting in deposition of type I collagen with subsequent formation of scar tissue and stricture formation.

Differential diagnostics

It is necessary in advanced stages of esophageal and gastric strictures.

As there is the similar symptomatology, such cicatricial changes of the pyloroantral part of stomach can suggest [pylorostenosis](#) caused by peptic ulcer. The differential diagnosis is based on careful analysis of the history and endoscopic investigation of esophagus and stomach.

[Esophageal cancer](#). As this pathology can have the similar roentgenological picture, it requires thorough differential diagnostics. Besides anamnesis and clinical manifestations, the question of the diagnosis finally confirmed by histological investigation of a biopsy material, obtained during endoscopy.

Tactics and choice of treatment

The treatment of esophageal burns first of all should be guided to save the life of the patient, and also to prevent the development of esophageal strictures. The first aid must be given as soon as possible after taking of the chemical substance, which have caused the burn. In such cases by means of gastric tube and great amount of water (to 10-15 l.) immediately wash out the esophagus and stomach. It is better to use for this purpose the neutralizing solutions. If the burn is caused by acid applied 2 % solution of sodium hydrocarbonatis, and in the burns by alkalis – vinegar in the ratio 1:20 with water. For prophylaxis of shock and decreasing of psychoemotional excitement of the patient instituted anesthetizing agents. If asphyxia arise owing to edema of pharynx and epiglottis, a tracheostomy is performed.

The further aid – the treatment of shock and hypovolemia by massive intravenous infusions (up to 4-5 l. per day) of saline solutions, solutions of glucose, dextrans and blood plasma. With the purpose of detoxycation also applied forced diuresis.

Antibacterial therapy is nominated for prevention of infection complications.

In first two days after the burn the patients get parenteral feeding. Nevertheless, if the swallowing is not disturbed, it is possible to add feeding by grinding cold food. The early application of enteric feeding can be as a weak bougienage of esophagus and simultaneous prophylaxis of cicatricial strictures. The development of complications requires the treatment of their liquidation.

In the third period of the course of disease it is important not to miss a possible formation of cicatricial stenosis of esophagus. In overwhelming majority at timely and correct performance of esophageal dilatation it is possible to achieve positive effect and avoid multistep and hazardous operations. The dilatation is carried out by special [elastic thermolabile bougies](#). The first procedures of bougienage are necessary to carry out under the roentgenological check. It enables to prevent perforation of esophagus. The latter, as the complication of esophageal bougienage, can occur not only in places of cicatricial stricture, but also in the region of piriform sinus. Thereby the bougie penetrates in mediastinum and can result in mediastinitis. With the purpose of prevention of such complication the esophageal bougienage is better to carry out with conductor. It can be represented by a cord (thick thread), passed through the mouth and gastrostoma. The bougie should have the canal for conductor, nevertheless it is possible to apply the usual one with the loop, on its end.

Diet

The usual antireflux precautions and lifestyle modifications should be reinforced, although no published data exist showing that these measures are efficacious in peptic strictures.

- Patients are told to avoid fatty and spicy foods, alcohol, tobacco, chocolate, and peppermint.
- Patients should eat smaller meals, avoid eating in a hurried fashion, and chew their food well.
- Patients should be encouraged not to eat at least 2-3 hours before bedtime.
- Weight reduction should be encouraged.
- Ill-fitting dentures or poor dentition should be corrected if possible.

Traditionally, more emphasis has been placed on mechanical dilatation, and coexistent esophagitis has been relatively ignored. However, several studies have demonstrated that aggressive acid suppression using PPIs is extremely beneficial in the initial treatment of esophageal stricture, as well as long-term management.

- A dysphagia score developed by Dakkak et al in a study of 64 patients revealed that the stricture diameter only contributed to 30% of the dysphagia score and that esophagitis and other factors accounted for 70% of the score. A linear association existed between the dysphagia score only when the luminal diameter was less than 5 mm. Overall, the degree of dysphagia was worse with increasing esophagitis independent of the degree of stenosis.
- Smith et al showed in a randomized study of 366 patients that omeprazole 20 mg/d was superior to ranitidine 300 mg twice a day in preventing stricture recurrence with redilation rates of 30% and 46%, respectively, at 12 months ($P < 0.01$)
- Marks et al showed that the redilation rate in patients treated with omeprazole 20-40 mg/d was 41% versus 73% in patients treated with ranitidine 150-300 mg twice per day and almost reached significance ($P < 0.07$).[10] However, the omeprazole group showed higher rates of dysphagia relief and healing of esophagitis when compared with histamine 2 (H2) blockers.
- In contrast, 2 other studies by Swarbrick et al and Silvis et al did not show any significant differences in the redilation rates at 12 and 10 months, respectively.
- PPI treatment of patients with esophageal stricture is also more cost effective than H2 blocker therapy. Marks et al found that over a 6-month period, the cost of omeprazole therapy was \$1744 compared with \$2957 with H2 blockers.
- H2 blockers have not been shown to be any better than placebo in various trials, and no reliable data on prokinetic agents exist.

Surgical Care

In advanced cases if failed to reach the restore of esophageal patency by a bougienage, the esophagoplasty by stomach, small and large intestine is applied.

The following discussion concerns the endoscopic and surgical modalities employed for the management of peptic esophageal stricture. The choice of dilator and technique is dependent on many factors, the most important being stricture characteristics. It is also based on other factors, including patient tolerance, operator preference, and experience. No clear consensus on the optimal end point exists. In summary, dilation therapy should be tailored individually.

Endoscopic dilation dates to the 16th century, when physicians used wax wands for esophageal dilation.[13, 14] The word bougie is derived from Boujijah, an Algerian city that was the center of the medieval candle trade. The following 3 types of dilators are used:

- Mercury-filled bougies - Maloney or Hurst dilators
 - These dilators are indicated in uncomplicated strictures with diameters greater than 10-12 mm.
 - They are inexpensive and simple to perform without fluoroscopic guidance.
 - Minimal or no sedation is necessary.
 - Self-bougienage may be performed at home.
- Wire-guided polyvinyl bougies - Savary-Gilliard and American dilators
 - These dilators are relatively stiff and better suited to longer, tighter, and irregular strictures.
 - The need for fluoroscopy is variable.
 - The range is 5-20 mm, and these dilators are reusable.
 - Drawbacks include trauma to the laryngeal wall and patient discomfort.
 - American dilators are shorter, less tapered, and impregnated with barium for better fluoroscopic visualization.
- Through-the-scope (TTS) balloon dilators
 - These dilators are used through the endoscope, and they allow for direct visualization.
 - These are relatively expensive and not reusable.
 - Fluoroscopy is not mandatory, but it is useful in difficult cases.
 - Studies conflict about the benefits of balloon dilators compared with Savary dilators.

A prospective, randomized study with 17 patients in each arm comparing balloon dilators with Savary dilators was performed by Saeed et al over a 2-year period, with the end point being 45F.[15]

- Stricture recurrence was similar in the first year but lower in the second year for balloons.
- Fewer sessions were needed for balloons, 1.1 sessions +/- 0.1 versus 1.7 sessions +/- 0.2, and less procedural discomfort occurred ($P < 0.05$).
- Both devices were effective in relieving dysphagia.

Another prospective, randomized study by Scolapio et al included 251 patients with peptic strictures. Schatzki rings did not show any differences in complications, the degree of immediate relief, or the time to recurrent dysphagia.

- General rules of esophageal dilation

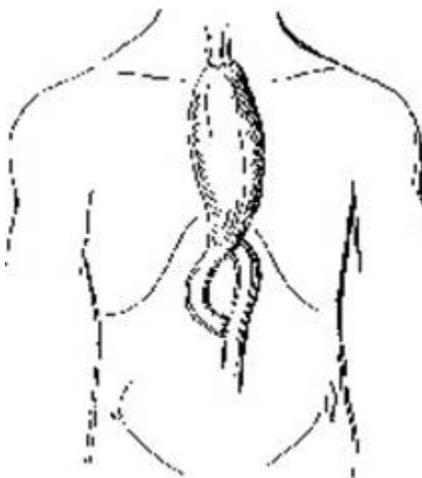
- Many authors have questioned the need for mandatory fluoroscopy, and no published data exist to advocate safety of fluoroscopy. However, one may consider using fluoroscopy in complicated strictures, especially in guiding the blind passage of a guidewire.
- Rule of 3s: The first bougie passed should be approximately equal to the estimated diameter of the stricture. Pass no more than 3 consecutive bougies of progressively increasing size after the first one that meets moderate resistance during any one dilation session. The rule of 3s has been questioned because of a lack of data verifying the increased efficacy or safety if one adheres to this rule. This rule was formulated for dilation using mercury-filled bougies resulting in dilation no greater than 1.3 mm in one session. However, polyvinyl dilators may not provide adequate tactile perception to follow this rule.
- A study by Kozarek et al showed only one perforation in 400 patients dilated with polyvinyl dilators to greater than 2 mm in one session.
- Balloon dilators frequently dilate greater than that prescribed by the rule of 3s without any increased risk of complications.
- No consensus exists regarding the end point of esophageal dilation for peptic strictures.
 - Most patients experience complete relief when dilated to 40-54F. Therefore, using this end point as a benchmark is recommended.
 - In summary, the extent of the dilatation should be individualized based on symptomatic response and technical difficulty encountered during therapy.
- Intralesional steroid injection
 - Limited anecdotal data exist showing that intralesional steroid injection of peptic strictures may be beneficial. The mechanism is unclear; it may inhibit collagen formation and enhance collagen degradation, thus increasing stricture compliance.
 - Triamcinolone 10 mg/mL in 0.5 mL aliquots was injected in 4 quadrants in 2 patients with a successful outcome as reported by Kirsch et al.
 - Lee et al showed a higher rate of achieving greater luminal diameters and duration between dilations in a nonrandomized cohort of patients with strictures of varying etiologies.[20] Similar results were obtained by Kochhar et al in 71 patients, although 8 injections of 20 mg of triamcinolone in 0.5-mL aliquots were given at the proximal margin and into the stricture itself.
 - A randomized prospective trial of Savary dilation with or without intralesional

steroids was conducted in 42 patients by Dunne et al ; it demonstrated a decreased need for second dilations in the steroid group (1.95 vs 5.5) at 1 year. Similar results were seen in a study by Ramage et al in 30 patients, but the latter study was also double blinded with a sham group. Two patients (13%) in the steroid group and 9 patients (60%) in the sham group needed repeat dilation over a 12-month period.

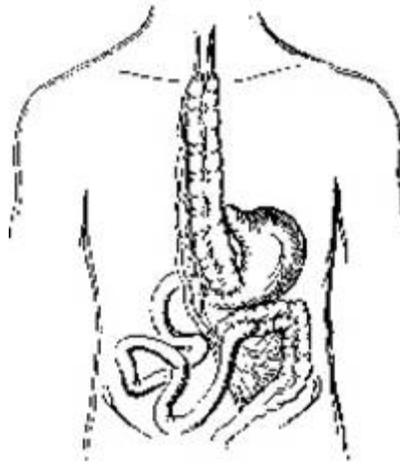
- Therefore, a trial of steroid injection may be reasonable in patients with benign strictures who experience no significant relief of dysphagia despite repeated dilations and aggressive antireflux therapy. Hishiki et al reported the use of repeated endoscopic dilatation with systemic steroids in a child with severe esophageal anastomotic stricture that did not respond to endoscopic dilatation and local steroid injection of the stricture. At 18 months follow-up, the child remained asymptomatic without any further endoscopic dilations.
- Endoscopic stricturoplasty: Two case series described a technique using a needle knife to make 4 quadrant incisions followed by Savary dilation. This was successful in 8 of 8 patients as reported by Raijman et al[25] and 5 of 6 patients as reported by Hagiwara et al.
- Pharyngoesophageal puncture is a term recently coined by Tang et al to describe the technique of endoscopic dilation of radiation-induced severe or complete pharyngoesophageal strictures. A combination of guide wires, endoscopic balloons, puncture and techniques learned from ERCP were successfully applied to 3 patients with severe/complete stenosis.
- Expandable polyester silicone-covered stent: Repici et al presented a case series of 15 patients whose condition had failed endoscopic therapy. A temporary placement of a stent for 6 weeks was successful in 12 patients over a long-term period (mean follow-up, 22.7 mo). However, the exact duration for stent placement remains unclear, as strictures can recur following stent removal. Furthermore, a variety of complications have been described following stent deployment. More recently, biodegradable stents have shown some promise in animal studies and were used to treat severe corrosive esophageal stenosis in a child.[32]
- The role of surgical treatment in peptic stricture remains in dispute. Indications include failed aggressive medical therapy or an unsuitable candidate for aggressive medical therapy. This is usually a rare occurrence in the era of PPI therapy. Various procedures advocated include the following:

- Esophageal-sparing procedures - Standard antireflux surgery (Nissen total or Belsey partial fundoplication), esophageal lengthening with antireflux surgery (Collis-Nissen or Belsey gastroplasty)
- Esophageal resection and reconstruction - Gastric or colon interposition or jejunal segment
- If the benign peptic stricture is dilatable, an esophageal-sparing operation is performed.
 - If the length of the esophagus is normal, standard antireflux surgery and postoperative dilation as necessary is recommended.
 - If the esophagus is short, performing Collis gastroplasty and postoperative dilation as necessary is recommended.
 - If the stricture is undilatable, esophageal resection and interposition is recommended.
- In the literature, some anecdotal reports exist of minimally invasive surgery, including laparoscopic transhiatal esophagectomy and laparoscopic Collis gastroplasty with Nissen fundoplication. With continuing advances in technology, whether or not minimally invasive surgery would play a major role in the surgical management of peptic stricture remains to be determined.

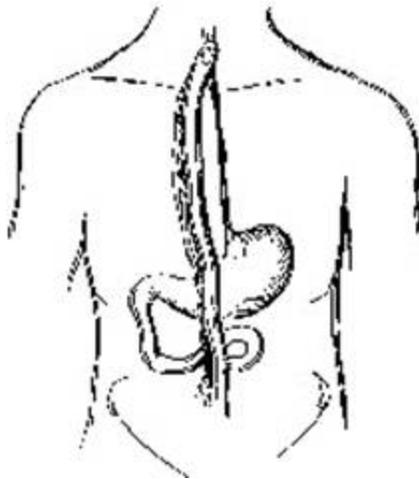
Esophageal plastic



By stomach



By large intestine



By small intestine

ESOPHAGEAL CANCER

Esophageal cancer is cancer that occurs in the esophagus — a long, hollow tube that runs from your throat to your stomach. Your esophagus carries food you swallow to your stomach to be digested.

Esophageal cancer usually begins in the cells that line the inside of the esophagus. Esophageal cancer can occur anywhere along the esophagus, but in people in the United States, it occurs most often in the lower portion of the esophagus. More men than women get esophageal cancer.

Esophageal cancer isn't common in the United States. In other areas of the world, such as Asia and parts of Africa, esophageal cancer is much more common.

Etiology

It's not clear what causes esophageal cancer. Esophageal cancer occurs when cells in your esophagus develop errors (mutations) in their DNA. The errors make cells grow and divide out of control. The accumulating abnormal cells form a tumor in the esophagus that can grow to invade nearby structures and spread to other parts of the body.

Types of esophageal cancer

Esophageal cancer is classified according to the type of cells that are involved. The type of esophageal cancer you have helps determine your treatment options. Types of esophageal cancer include:

- Adenocarcinoma. Adenocarcinoma begins in the cells of mucus-secreting glands in the esophagus. Adenocarcinoma occurs most often in the lower portion of the esophagus. Adenocarcinoma is the most common form of esophageal cancer in the United States, and it affects primarily white men.
- Squamous cell carcinoma. The squamous cells are flat, thin cells that line the surface of the esophagus. Squamous cell carcinoma occurs most often in the middle of the esophagus. Squamous cell carcinoma is the most prevalent esophageal cancer worldwide.
- Other rare types. Rare forms of esophageal cancer include choriocarcinoma, lymphoma, melanoma, sarcoma and small cell

Risk factors

It's thought that chronic irritation of your esophagus may contribute to the DNA changes that cause esophageal cancer. Factors that cause irritation in the cells of your esophagus and increase your risk of esophageal cancer include:

- Drinking alcohol
- Having bile reflux
- Chewing tobacco
- Having difficulty swallowing because of an esophageal sphincter that won't relax (achalasia)
- Drinking very hot liquids
- Eating few fruits and vegetables
- Eating foods preserved in lye, such as lutefisk, a Nordic recipe made from whitefish, and some olive recipes

- Having gastroesophageal reflux disease (GERD)
- Being obese
- Having precancerous changes in the cells of the esophagus (Barrett's esophagus)
- Undergoing radiation treatment to the chest or upper abdomen
- Smoking

Other risk factors include

- Being male
- Being between the ages of 45 and 70

Signs and symptoms

- Difficulty swallowing (dysphagia)
- Weight loss without trying
- Chest pain, pressure or burning
- Fatigue
- Frequent choking while eating
- Indigestion or heartburn
- Coughing or hoarseness

Early esophageal cancer typically causes no signs or symptoms.

If you've been diagnosed with Barrett's esophagus, a precancerous condition that increases your risk of esophageal cancer caused by chronic acid reflux, ask your doctor what signs and symptoms to watch for that may signal that your condition is worsening.

Screening for esophageal cancer isn't done routinely because of a lack of an easily identifiable high-risk group and the possible risks associated with endoscopy. If you have Barrett's esophagus, discuss the pros and cons of screening with your doctor.

Diagnostic

Using a scope to examine your esophagus (endoscopy). During endoscopy, your doctor passes a hollow tube equipped with a lens (endoscope) down your throat and into your esophagus. Using the endoscope, your doctor examines your esophagus looking for cancer or areas of irritation.

X-rays of your esophagus. Sometimes called a barium swallow, an upper gastrointestinal series or an esophagram, this series of X-rays is used to examine your esophagus. During the test, you drink a thick liquid (barium) that temporarily coats the lining of your esophagus, so

the lining shows up clearly on the X-rays.

Collecting a sample of tissue for testing (biopsy). A special scope passed down your throat into your esophagus (endoscope) or down your windpipe and into your lungs (bronchoscope) can be used to collect a sample of suspicious tissue (biopsy). What type of biopsy procedure you undergo depends on your situation. The tissue sample is sent to a laboratory to look for cancer cells.

Esophageal cancer staging

When you're diagnosed with esophageal cancer, your doctor works to determine the extent (stage) of the cancer. Your cancer's stage helps determine your treatment options. Tests used in staging esophageal cancer include computerized tomography (CT) and positron emission tomography (PET).

The stages of esophageal cancer are:

Stage I. This cancer occurs only in the top layer of cells lining your esophagus.

Stage II. The cancer has invaded deeper layers of your esophagus lining and may have spread to nearby lymph nodes.

Stage III. The cancer has spread to the deepest layers of the wall of your esophagus and to nearby tissues or lymph nodes.

Stage IV. The cancer has spread to other parts of your body.

Surgery

Surgery to remove the cancer can be used alone or in combination with other treatments. Operations used to treat esophageal cancer include:

Surgery to remove very small tumors. If your cancer is very small, confined to the superficial layers of your esophagus and hasn't spread, your surgeon may recommend removing the cancer and margin of healthy tissue that surrounds it. Surgery for very early-stage cancers can be done using an endoscope passed down your throat and into your esophagus.

Surgery to remove a portion of the esophagus (esophagectomy). Your surgeon removes the portion of your esophagus that contains the tumor and nearby lymph nodes. The remaining esophagus is reconnected to your stomach. Usually this is done by pulling the stomach up to meet the remaining esophagus. In some situations, a portion of the colon is used to replace the missing section of esophagus.

Surgery to remove part of your esophagus and the upper portion of your stomach (esophagogastrectomy). Your surgeon removes part of your esophagus, nearby lymph nodes and the upper part of your stomach. The remainder of your stomach is then pulled up and reattached to your esophagus. If necessary, part of your colon is used to help join the two.

Esophageal cancer surgery carries a risk of serious complications, such as infection, bleeding and leakage from the area where the remaining esophagus is reattached. Surgery to remove your esophagus can be performed as an open procedure using large incisions or with special surgical tools inserted through several small incisions in your skin (laparoscopically). How your surgery is performed depends on your situation and your surgeon's experience and preferences.

Surgery for supportive care

Besides treating the disease, surgery can help relieve symptoms or allow you to eat.

Relieving esophageal obstruction. A number of treatments are available to relieve esophageal obstruction. One option includes using an endoscope and special tools to widen the esophagus and place a metal tube (stent) to hold the esophagus open. Other options include surgery, radiation therapy, chemotherapy, laser therapy and photodynamic therapy.

Providing nutrition. A surgeon inserts a feeding tube (percutaneous gastrostomy) so you can receive nutrition directly into your stomach or intestine. This is usually temporary until the surgical site heals or until you're finished with chemotherapy and radiation therapy.

Chemotherapy

Chemotherapy is drug treatment that uses chemicals to kill cancer cells. Chemotherapy drugs are typically used before (neoadjuvant) or after (adjuvant) surgery in people with esophageal cancer. Chemotherapy can also be combined with radiation therapy. In people with advanced cancer that has spread beyond the esophagus, chemotherapy may be used alone to help relieve signs and symptoms caused by the cancer.

The chemotherapy side effects you experience depend on which chemotherapy drugs you receive.

Radiation therapy

Radiation therapy uses high-powered energy beams to kill cancer cells. Radiation can come from a machine outside your body that aims the beams at your cancer (external beam radiation). Or radiation can be placed inside your body near the cancer (brachytherapy).

Radiation therapy is most often combined with chemotherapy in people with esophageal cancer. It can be used before or after surgery. Radiation therapy is also used to relieve complications of advanced esophageal cancer, such as when a tumor grows large enough to stop food from passing to your stomach.

Side effects of radiation to the esophagus include sunburn-like skin reactions, painful or difficult swallowing, and accidental damage to nearby organs, such as the lungs and heart.

Combined chemotherapy and radiation

Combining chemotherapy and radiation therapy may enhance the effectiveness of each treatment. Combined chemotherapy and radiation may be the only treatment you receive, or combined therapy can be used before surgery. But combining chemotherapy and radiation treatments increases the likelihood and severity of side effects.

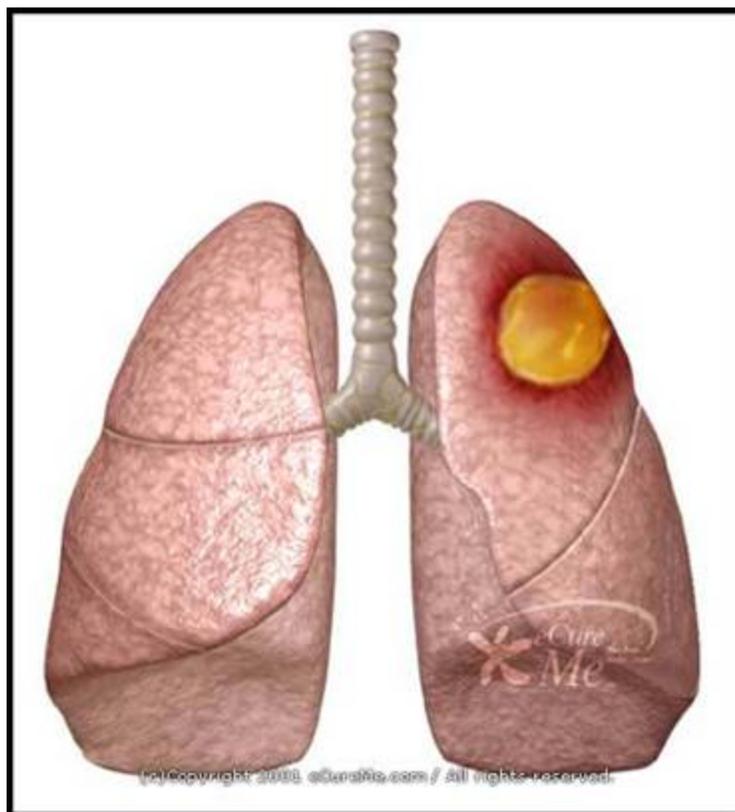
PURULENT DISEASES OF LUNGS AND PLEURA

Lung abscess is defined as necrosis of the pulmonary tissue and formation of cavities containing necrotic debris or fluid caused by microbial infection. The formation of multiple small (< 2 cm) abscesses is occasionally referred to as necrotizing pneumonia or lung gangrene. Both lung abscess and necrotizing pneumonia are manifestations of a similar pathologic process. Failure to recognize and treat lung abscess is associated with poor clinical outcome.

Lung abscess was a devastating disease in the preantibiotic era, when one third of the patients died, another one third recovered, and the remainder developed debilitating illnesses such as recurrent abscesses, chronic empyema, bronchiectasis, or other consequences of chronic pyogenic infections. In the early postantibiotic period, sulfonamides did not improve the outcome of patients with lung abscess until the penicillins and tetracyclines were available. Although resectional surgery was often considered a treatment option in the past, the role of surgery has greatly diminished over time because most patients with uncomplicated lung abscess eventually respond to prolonged antibiotic therapy.

Lung abscesses can be classified based on the duration and the likely etiology. Acute abscesses are less than 4-6 weeks old, whereas chronic abscesses are of longer duration. Primary abscess is infectious in origin, caused by aspiration or pneumonia in the healthy host; secondary abscess is caused by a preexisting condition (eg, obstruction), spread from an extrapulmonary site, bronchiectasis, and/or an immunocompromised state.

Most patients with primary lung abscess improve with antibiotics, with cure rates documented at 90-95%



Abscessing pneumonia is characterized by the multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs, which are not disposed to progression. The destruction is accompanied by expressed perifocal infiltration of a pulmonary tissue.

Abscess of lungs - purulent or ichorous destruction of necrotic sites of pulmonary tissue of one segment with formation of one or several cavities, filled by pus, and detached from adjacent parenchyma by a pyogenic capsule and expressed perifocal infiltration of surrounding pulmonary tissue. It arises at the persons with a maintained reactivity of the organism.

Gangrenous abscess is a purulent, ichorous necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma, with the liability to formation of sequestrs. Depending on reactivity of the organism it can transform into purulent abscess (after the lysis of sequestrs) or gangrene.

Gangrene of lungs – a diffuse purulent, ichorous necrosis of the tissue without the tendency to defined demarcation with prompt dynamics of spreading of necrotic zone and destruction of the parenchyma. It is characterized by a grave intoxication, liability to a pleural complications and pulmonary bleeding. If only the one lobe is affected the gangrene is considered to be limited, if affected extensive areas of lungs the gangrene is wide-spread.

Etiology and pathogenesis

Lung abscesses have numerous infectious causes. Anaerobic bacteria continue to be accountable for most

cases. These bacteria predominate in the upper respiratory tract and are heavily concentrated in areas of oral-gingival disease. Other bacteria involved in lung abscesses are gram-positive and gram-negative organisms. However, lung cavities may not always be due to an underlying infection. Some evidence suggests that individuals with cyanotic heart disorders may also be more prone to lung abscess formation. The continuous hypoperfusion of the pulmonary tissues may predispose the individuals to chronic pulmonary infections.

The predominant factors which cause the disease:

- disturbances of bronchial patency with the development of atelectasis;
- infectious inflammatory process in a pulmonary tissue;
- regional disturbances of blood supply with a further necrosis of areas of pulmonary parenchyma.

The embodiment of these factors occurs in condition of the changed reactivity of the organism.

The states which result in the aspiration of contents of the upper parts of alimentary tract (traumas of head, craniovascular disturbances, alcoholism, narcomania, narcoses, epilepsy etc.) contribute to the pulmonary abscessing. And also factors, which are capable to provoke secondary immunodeficiency and suppression of reactive processes: diabetes mellitus, irradiation, long application of corticosteroids, antineoplastic therapy, some hematological disease, AIDS favor the purulent processes.

Factors contributing to lung abscess

Oral cavity disease

- Periodontal disease
- Gingivitis

Altered consciousness

- Alcoholism
- Coma
- Drug abuse
- Anesthesia
- Seizures

Immunocompromised host

- Steroid therapy
- Chemotherapy
- Malnutrition
- Multiple trauma

Esophageal disease

- Achalasia
- Reflux disease
- Depressed cough and gag reflex
- Esophageal obstruction

Bronchial obstruction

- Tumor
- Foreign body
- Stricture

Generalized sepsis

Pathology

The abscesses mainly develop in II and VI segments of lungs. They may be single and multiple. The abscess is confined from adjacent pulmonary tissue by a capsule, which represents a granulating tissue and dense leukocytic rampart. Usually it is possible to find out a draining bronchus. Later on in the wall of the abscess the amount of connective tissue fibers is enlarged.

In gangrene the pulmonary tissue is of black color, swollen, with cavities, and in some places transfers into the sites with dark green coloring. The macropreparatus is characteristically fetid.

Classification

According to pathogenesis:

- postpneumonic;
- aspirative;
- obturative;
- posttraumatic;
- hematogenous or septic;
- lymphogenous;

- thromboembolic.

According to the character of purulent process:

- single purulent abscesses;
- multiple purulent abscesses;
- bilateral purulent abscesses;
- gangrenous abscesses (single, multiple, uni- and bilateral);
- limited gangrene;
- wide-spread gangrene.

According to stages:

- 1 stage - necrotic pneumonia;
- 2 stages - destruction and rejection;
- 3 stages - cleaning and cicatrization.

According to the term of existence:

- acute;
- chronic.

Complications:

- pulmonary bleeding;
- pyopneumothorax;
- pleural empyema;
- sepsis;

- bronchogenic dissemination.

Symptomatology and clinical course

The clinical manifestations of acute purulent destruction of lungs depend on the size of the focus and character of destruction, reactivity of the organism and stage of the disease, peculiarities of the drainage of purulent cavities and complications.

At the first stage of **acute abscess** the patients complain of general weakness, headache, malaise, suppressed appetite, moderate chest pain, dyspnea, subfebrile temperature.

At the second stage the state of the patients is worsened. The fever rises to as high as 39-40°C and has a hectic character. At the same time the chest pain increases, which associates with a troubling cough and dyspnea. The condition of the patients is worsened and the intoxication increases. One can feel a foul-smelling from the mouth at cough. The amount of sputum is small, with a rusty tone. With the beginning of the draining of destructive cavities through bronchus the daily quantity of the sputum reaches 500 ml and more. At this time is possible hemoptysis. The sputum is foul-smelling. At sedimentation it divides into three layers:

- inferior – resembling a grey mass with detrites and flaps of a pulmonary tissue;
- medial – purulent, turbid, liquid;
- upper – mucous foamy layer.

Further in favourable cases there is a considerable improvement of state of the patients. The body temperature falls, the signs of intoxication reduce and the appetite increases.

The disease grades into the third stage, which is characterized by the regress of clinical manifestations, up to their complete disappearance.

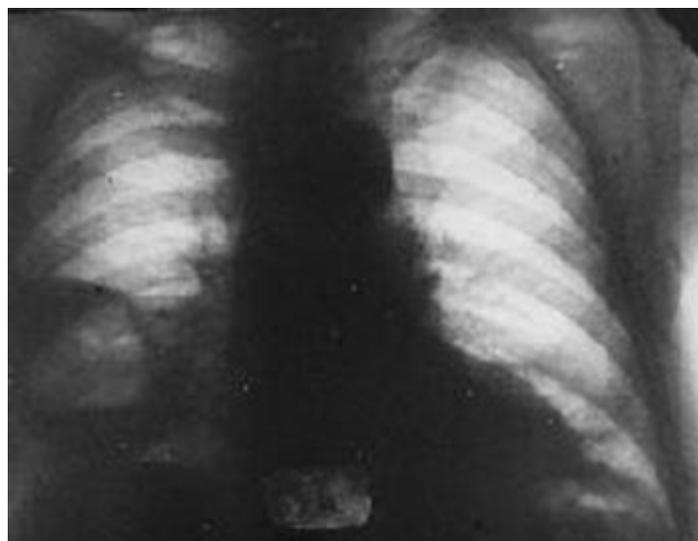
The physical signs good are well revealed at peripheral localization of the process. By palpation – weakened vocal fremitus. At percussion – a blunted sound over the site of the purulent focus and perifocal infiltration (at subpleural location of the abscess). By auscultation – tubular sound with a moist rales in the zone of purulent focus. Well-generated subpleural cavities of major sizes can be revealed by percussion by bandbox sound, on auscultation by moist rales on the background of amphoric respiration.

At X-ray of the chest at the stage of necrotic pneumonia is found out a rounded lesion with irregular contour. For the second stage is characteristic the enlightenment of the shadow with a further developing of the rounded cavity with air-fluid level (Kordin's symptom), gentle pyogenic sheath and the perifocal infiltration.

At the third stage on the place of suppuration observed expressed fibrosis, sometimes as a thin-walled annular formation.



Acute abscess of the right lower lobe before draining



Acute abscess of the right lower lobe after draining

Gangrenous abscess is characterized by a grave state of the patient, expressed purulent intoxication, cough with expectoration of a great amount (500 ml and more) of grey-green sputum with a foul-smelling, hectic body temperature. Roentgenologically the outline of the cavity is poorly defined, it contains a visible sequestrum that looks as a polymorphous shadow. The adjacent pulmonary tissue is infiltrated.



Gangrenous abscess

The clinic of a **pulmonary gangrene** differs by a terminal expression of signs. The state of the patients is critical. The patient is adynamic, exhausted, with edemas on legs. Dyspnea in rest, hemodynamic disturbances are evident. Dirty-grey or brown sputum with detrites, pieces of necrotic parenchyma and threads of blood excretes out with the cough up to 1 l. Early pleural complications are usual and represent with pulmonary bleeding, which may be profuse. Often it is associated with vital organ dysfunction and loss of consciousness.

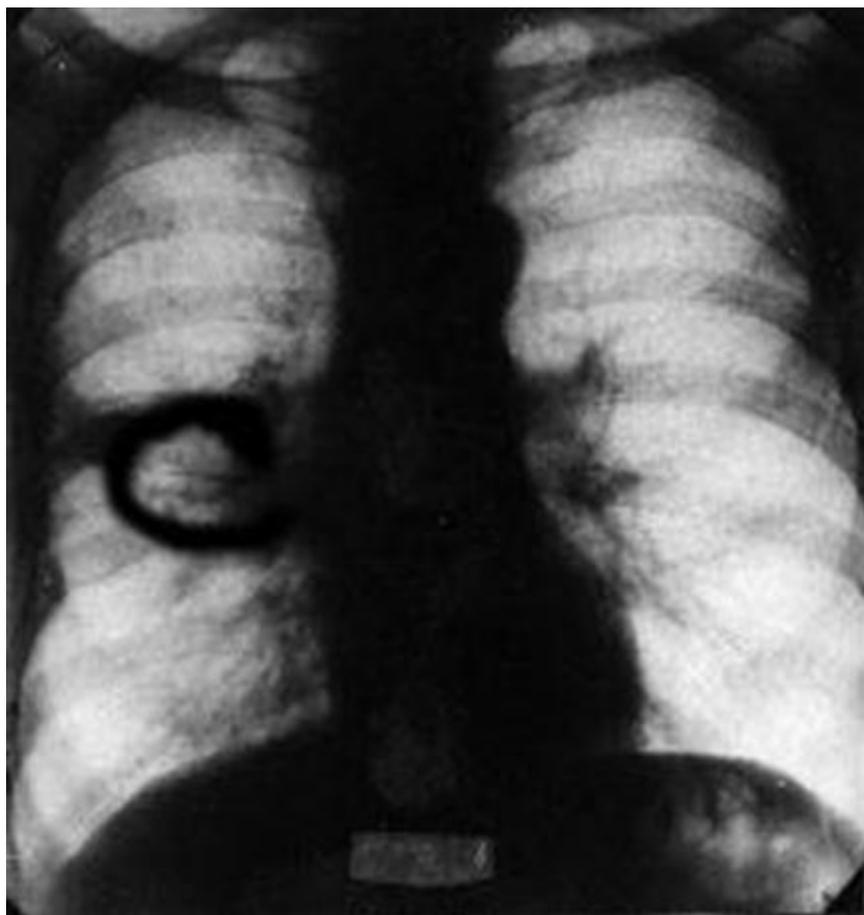
The intensive shadow which borrows a considerable area of lungs with a visible cavities, that contain sequestrum, fluid levels, is roentgenologically revealed. The shadow outline is irregular, but could be well defined if the process is within interlobar sulcus.



Pulmonary gangrene

Chronic abscess of lungs occurs at 12-15 % of cases. It is considered to be chronic at existence of a pulmonary abscess more than 6-8 weeks. It is characterized by a cyclic course. In the stage of remission the patients complain of a moderate dyspnea, cough with expectoration of a mucous or mucopurulent discharge. The exacerbation manifests by coughing up of 250-500 ml of a purulent foul sputum, chest pain, dyspnea, hectic temperature with the difference in 1,5-2°C. Dizziness, suppression of appetite, general weakness enlarges according to intoxication. The skin is pale with moderate cyanosis. The respiratory rate rises to 28-30 per min. In 6-8 months is noticed the clubbing of the fingers and deformation of the chest. The vocal fremitus is a little bit weakened on the side of lesion (particularly in peripheral localization of the process). The percussion reveals a short sound in projection of pathological process, the auscultation - a lot of moist rales on the background of amphoric respiration.

Roentgenologically chronic abscess is shown by one or several cavities of a spherical shape with a thick, dense pyogenic sheath. Exacerbation of the process manifests by a cavity with horizontal air-fluid level. The size of surrounding perifocal infiltration depends on the phase of process.



Chronic abscess of lungs

The blood analysis in pulmonary destruction is characterized by leukocytosis with deviation of the differential count to the left, lymphocytopenia, elevation of the erythrocyte sedimentation rate. Gangrenous change of the process is accompanied by a progressing anemia, sometimes by leukopenia. The hypoproteinemia arises from major losses of protein with purulent sputum. Intoxication and toxic lesion of liver leads to disproteinemia. It is associated with the enlarged concentration of mucoprotein, sialine acids, seromuroid, and fibrinogen.

The immunogram reveals the suppression of cellular and humoral immunity with a liability to hyperergy and autoaggression, depression of the mechanisms of nonspecific protection.

The cytological bronchial water lavage is characterized by expressed neutrocytosis, noncellular postdestructive insertions at lack or absence of alveolar macrophages.

Variants of clinical course and complications

According to the clinical course, there are such variants of the development of purulent diseases of lungs:

1. **Favorable course.** The adequate treatment results in prompt positive clinical, roentgenological and laboratory dynamics, and terminates by recovery.
2. **Non-progressive course.** A poor drainage of the suppurative focus and permanent purulent intoxication result in transferring of the process in chronic form.
3. **Progressing course.** Is predetermined by combination of a series of the unfavorable factors (low resistance of the organism, autoimmune aggression, high virulence of the infecting agent etc.). Characterized by diffusion of the zone of necrosis and destruction with transferring in gangrene.
4. **Incapsulated process.** Caused by the absence or complete obstruction of the draining bronchus under condition of satisfactory resistance of the organism.
5. **Complicated course.** Mostly is the result of progressive development of the pathological process.

Pulmonary bleeding arise suddenly, are associated with coughing out of a foamy, red blood and clots by portions or continuous stream. The most often source of a pulmonary bleeding are the bronchial arteries and vessels of a pulmonary tissue. The clinical manifestations of a pulmonary suppuration are accompanied by dizziness, weakness, dyspnea, chest pain. The hemodynamic disturbances depend on intensity of the bleeding. The auscultation of lungs from both sides reveals the moist rales (aspiration). If the pulmonary destruction is present the plain film of the chest shows the localization of the source of bleeding. After hospitalization of the patient with this complication the exclusive information is obtained with a fibrobronchoscopy.



According to degree the pulmonary bleedings are classified (V.Struchkov, 1985):

I degree – hemorrhage up to 300 ml.

1. Single hemoptysis.
2. Multiple hemoptysis.

II degree – hemorrhage up to 700 ml.

1. Single bleeding:
 - a) with fall in arterial pressure and decreasing of hemoglobin;
 - b) without fall in arterial pressure and decreasing of hemoglobin.
2. Multiple bleeding:
 - a) with fall in arterial pressure and decreasing of hemoglobin;
 - b) without fall in arterial pressure and decreasing of hemoglobin.

III degree - hemorrhage exceeds 700 ml.

1. Massive bleeding.
2. Fulminant, lethal bleeding.

The I degree of a pulmonary bleeding manifests by coughing out the sputum tinged with blood, the hemodynamic disturbance usually absent. At bleeding of II degree are observed decreasing of arterial pressure on 20-30 mm Hg, tachycardia to 100 beats/min, contents of hemoglobin within 60-80 g/l. The bleeding of III degree are accompanied with sharp decreasing of arterial pressure, rapid (more than 100-120 beats/min), small, sometimes thread pulse, and even its disappearance on peripheral arteries, tachypnea to 40 per 1 min, hemoglobin to 50-60 g/l. Probable the fulminant course up to the terminal state with prompt failure of cardiac activity and asphyxia by blood.

Sepsis manifests by multisystem lesions with progression of the syndrome of polyorganous failure, hematosepsis, purulent metastasizing (frequently in brain).

Characteristic complications for suppurative diseases of lungs such as pleural empyema and pyopneumothorax are described in separate parts.

The diagnostic program

1. Complaints and history of the disease.

Generally, most of the patients admitted to the hospital with a diagnosis of lung abscess have had symptoms for at least 2 weeks. These patients typically have an intermittent febrile course, productive cough, weight loss, general malaise, and night sweats. Initially, foul sputum is not observed in the course of the infection; however, after cavitation occurs, putrid expectorations are quite prevalent. The odor of the breath and sputum of a patient with an anaerobic lung abscess is often quite pronounced and noxious and may provide a clue to the diagnosis. Hemoptysis may occasionally follow the expectoration of putrid sputum.

Symptoms depend on whether the abscess is caused by anaerobic or other bacterial infection.

- Anaerobic infection in lung abscess
 - Patients often present with indolent symptoms that evolve over a period of weeks to months.
 - The usual symptoms are fever, cough with sputum production, night sweats, anorexia, and weight loss.
 - The expectorated sputum characteristically is foul smelling and bad

tasting.

- Patients may develop hemoptysis or pleurisy
- Other pathogens in lung abscess
- These patients generally present with conditions that are more emergent in nature and are usually treated while they have bacterial pneumonia.
- Cavitation occurs subsequently as parenchymal necrosis ensues.
- Abscesses from fungi, *Nocardia* species, and *Mycobacteria* species tend to have an indolent course and gradually progressive symptoms.

2. Physical findings.

The findings on physical examination of a patient with lung abscess are variable. Physical findings may be secondary to associated conditions such as underlying pneumonia or pleural effusion. The physical examination findings may also vary depending on the organisms involved, the severity and extent of the disease, and the patient's health status and comorbidities.

- Patients with lung abscesses may have low-grade fever in anaerobic infections and temperatures higher than 38.5°C in other infections.

- Generally, patients with in lung abscess have evidence of gingival disease.

- Clinical findings of concomitant consolidation may be present (eg, decreased breath sounds, dullness to percussion, bronchial breath sounds, coarse inspiratory crackles).

- The amphoric or cavernous breath sounds are only rarely elicited in modern practice.

- Evidence of pleural friction rub and signs of associated pleural effusion, empyema, and pyopneumothorax may be present. Signs include dullness to percussion, contralateral shift of the mediastinum, and absent breath sounds over the effusion.

- Digital clubbing may develop rapidly.

3. X-ray examination of chest in two planes (direct and lateral).

- A typical chest radiographic appearance of a lung abscess is an irregularly shaped cavity with an air-fluid level inside. Lung abscesses as a result of aspiration most frequently occur in the posterior segments of the upper lobes or the superior segments of the lower lobes.

- The wall thickness of a lung abscess progresses from thick to thin and from ill-defined to well-circumscribed as the surrounding lung infection resolves. The cavity wall can be smooth or ragged but is less commonly nodular, which raises the possibility of cavitating carcinoma.

- The extent of the air-fluid level within a lung abscess is often the same in posteroanterior or lateral views. The abscess may extend to the pleural surface, in which case it forms acute angles with the pleural surface.

- Anaerobic infection may be suggested by cavitation within a dense segmental consolidation in the dependent lung zones.

- Lung infection with a virulent organism results in more widespread tissue necrosis, which facilitates progression of underlying infection to pulmonary gangrene.

4. Up to one third of lung

5. Tomogram of lungs.

6. Computed tomography

- CT scanning of the lungs may help visualize the anatomy better than chest radiography. CT scanning is very useful in the identification of concomitant empyema or lung infarction.

- On CT scans, an abscess often is a rounded radiolucent lesion with a thick wall and ill-defined irregular margins.

- The vessels and bronchi are not displaced by the lesion, as they are by an empyema.

- The lung abscess is located within the parenchyma compared with loculated empyema, which may be difficult to distinguish on chest radiographs.

- The lesion forms acute angles with the pleural surface chest wall.

7. Examination of the sputum (bacteriological, cytological).

8. General blood and urine analyses.

- A complete white blood cell count with differential may reveal leukocytosis and a left shift.

- Obtain sputum for Gram stain, culture, and sensitivity.

- If tuberculosis is suspected, acid-fast bacilli stain and mycobacterial

culture is requested.

- Blood culture may be helpful in establishing the etiology.
- Obtain sputum for ova and parasite whenever a parasitic cause for lung abscess is suspected.

9. Biochemical blood analysis (protein and its fractions).

10. Immunogram.

11. Fibrobronchoscopy.

Differential diagnostics

Pneumonia

Primary bacterial pneumonias caused by single bacterial species other than the pneumococcus may account for up to 25% of community-acquired and 80% of hospital-acquired pneumonias. All of these pneumonias may have somewhat similar physical findings and x-ray evidence of pulmonary infiltration or consolidation. For proper treatment, it is crucial to identify the causative agent by blood culture and by sputum examination with stained smear and culture. Transtracheal aspiration, fiberoptic bronchoscopy, or even lung biopsy may be needed for specific diagnosis and treatment.

Streptococcal Pneumonia

Pneumonia due to hemolytic streptococci occurs usually as a sequela to viral infection of the respiratory tract, especially influenza or measles, or in persons with underlying pulmonary disease. The patients are usually in a severely toxic condition and cyanotic. Pleural effusion develops frequently and early and progresses to empyema in one-third of untreated patients. The diagnosis rests on finding large numbers of streptococci in smears of sputum and culturing hemolytic streptococci from blood and sputum.

The treatment of choice is with penicillin G in a dosage similar to that for pneumococcal pneumonia (see above). If treatment is started early, the prognosis is good.

Staphylococcal Pneumonia (picture 2)

Pneumonia caused by *Staphylococcus aureus* occurs as a sequela to viral infections of the respiratory tract (eg, influenza) and in debilitated (eg, postsurgical) patients or hospitalized infants, especially after antimicrobial drug administration. There is often a history of a mild illness with headache, cough, and generalized aches that abruptly changes to a very severe illness with high fever, chills, and exaggerated cough with purulent or blood-streaked sputum and deep cyanosis. There may be early signs of pleural effusion, empyema, or tension pneumothorax. X-ray examination reveals lung consolidation, pneumatoceles,

abscesses, empyema, and pneumothorax. The demonstration of pyopneumothorax and of cavities with air-fluid levels by x-ray is highly suggestive of Staphylococcal pneumonia. The diagnosis must be confirmed by stained smear of sputum (masses of white cells and gram-positive cocci, many intra-cellular) and culture (predominantly *S aureus*), and

also by means of cultures of pleural fluid and blood. The white count is usually more than 20,000//zL.

Initial therapy (based on sputum smear) consists of nafcillin, 6-12 g/d, or vancomycin, 2 g/d, given intravenously in divided doses as a bolus. If the staphylococcus proves to be penicillin-sensitive by laboratory test, penicillin G, 20-60 million units/d intravenously, is the antibiotic of choice. Drugs should be continued for several weeks. If empyema develops, drainage must be established. The prognosis varies with the underlying condition of the patient and the drug susceptibility of the organism.

Legionella Pneumonia

The eponym legionnaires' disease has been given to a serious pneumonia that afflicted people attending the American Legion Convention in Philadelphia in 1976. Other outbreaks have been diagnosed respectively at least since 1965, and sporadic infections have occurred at least since 1947 in many places.

Legionella pneumophila is a poorly staining gram-negative bacterium that grows slowly on special media (eg, charcoal-yeast extract) at 35 °C. There are at least 8 species of *Legionella*, some with multiple serotypes. These organisms can be recovered in human disease from sputum, bronchial washings, pleural fluid, lung biopsies, or blood. *Legionella* species occur in the environment and are acquired by humans from aerosols, dust from air-conditioning systems, water, or soil. The infection is not usually communicable from patient to contacts. Asymptomatic infection is common at all ages, whereas symptomatic infection is most often an opportunistic pneumonia in immunocompromised individuals.

Asymptomatic infection is evident only by a rise in specific antibodies. Symptomatic infection is observed mainly in elderly persons, smokers, and patients undergoing hemodialysis or renal transplant.

The incubation period is estimated to be 2-10 days. Initial symptoms are malaise, diffuse myalgias, and headache, followed in 12-48 hours by high, non-remittent fever and chills. Nausea, vomiting, and diarrhea are frequent early in the illness. On the third day a dry cough begins that is nonproductive or produces scanty mucoid, sometimes blood-streaked sputum. Dyspnea and hypoxia become marked as signs of consolidation develop. Pleuritic chest pain occurs in one-third of patients. Severe confusion or delirium may occur.

There is leukocytosis with a shift to the left, hyponatremia, abnormal liver function tests, and, occasionally, microscopic hematuria. Chest x-rays reveal patchy, often multilobar pulmonary consolidation, and, occasionally, small pleural effusions. The illness usually worsens for 4-7 days before improvement begins in those who recover. During severe outbreaks, the mortality rate has been 10% in those with manifest disease. Death is attributed to respiratory or renal failure or shock, with disseminated intravascular coagulation.

The diagnosis is based on a clinical picture compatible with the specific features of the disease and on

negative results of bacteriologic laboratory tests for other pneumonias. The organism can be identified by immunofluorescence in cultures, lung biopsy, and, rarely, sputum specimens. A retrospective diagnosis is based on a significant rise in specific serum antibodies detected by immunofluorescence.

The treatment of choice is erythromycin, 0.5-1 g every 6 hours intravenously or orally for 2-3 weeks. This usually results in improvement in 2-3 days. Rifampin, 10-20 mg/kg/d, has been suggested for patients who fail to respond to erythromycin. Assisted ventilation and management of shock are essential.

Pneumocystis carinii Pneumonia

This parasitic infection occurs in debilitated children or immunodeficient adults. It has been a prominent opportunistic infection in AIDS patients. The diagnosis is made by lung biopsy and the demonstration of typical cysts of *P. carinii* in impression smears of lung tissue stained with methenamine-silver. Early treatment with sulfamethoxazole-trimethoprim can cure the pneumonia. The same drug has been effective in prophylaxis during immunosuppression. An alternative, more toxic drug is pentamidine isethionate.

"MIXED" BACTERIAL PNEUMONIAS (Hypostatic Pneumonia, "Terminal" Pneumonia, Bronchopneumonia)

Essentials of Diagnosis

- Variable onset of fever, cough, dyspnea, expectoration.
- Symptoms and signs often masked by primary (debilitating) disease.
- Greenish-yellow sputum (purulent) with mixed flora.
- Leukocytosis (often absent in aged and debilitated patients).
- Patchy infiltration on chest x-ray.

General Considerations

Mixed bacterial pneumonias include those in which culture and smear reveal several organisms, not one of which can clearly be identified as the causative agent. These pneumonias usually appear as complications of anesthesia, surgery, aspiration, trauma, or various chronic illnesses (cardiac failure, advanced carcinoma, uremia). They are common complications of chronic pulmonary diseases such as bronchiectasis and emphysema. Old people are most commonly affected ("terminal" pneumonia). Patients treated with intermittent positive pressure breathing apparatus or immunosuppressive drugs may develop pneumonia caused by gram-negative rods.

The following findings in a debilitated, chronically ill, or aged person suggest a complicating pneumonia: (1) worsening of cough, dyspnea, cyanosis; (2) low-grade, irregular fever; (3) purulent sputum; and (4) patchy basal densities on a chest film (in addition to previously noted densities caused by a primary underlying

disease, if any), sometimes with local necrosis and cavitation.

Clinical Findings

A. Symptoms and Signs: The onset is usually insidious, with low-grade fever, cough, expectoration, and dyspnea that may become marked and lead to cyanosis. Physical findings are extremely variable and may not be impressive against a background of cardiac or pulmonary disease. The signs listed under Other Bacterial Pneumonias may also be present.

B. Laboratory Findings: The appearance of a greenish or yellowish (purulent) sputum should suggest a complicating pneumonia. Smears and cultures reveal a mixed flora, often including anaerobes. Predominant types should be noted. Leukocytosis is often absent in the aged and debilitated patient presenting with a mixed infection.

C. X-Ray Findings: X-ray (Picture 3) shows patchy, irregular infiltrations, most commonly posterior and basal (in bedridden patients). Abscess formation may be observed. Careful interpretation will avoid confusion with shadows due to preexisting heart or lung disease.

Differential Diagnosis

Mixed bacterial pneumonias must be differentiated from tuberculosis, carcinoma, and other specific mycotic, bacterial, and viral pulmonary infections (to which they may be secondary).

Treatment

Clear the airway and correct hypoxia. Unless a probably significant etiologic agent can be identified, give one of the new cephalosporins (eg, cefotaxime, 12 g/d intravenously) as initial therapy. This will be modified according to clinical and laboratory results.

Prognosis

The prognosis depends upon the nature and severity of the underlying pulmonary disease and varies with the predominating organism.

ASPIRATION PNEUMONIA

Aspiration pneumonia is an especially severe type of pneumonia, often with a high mortality rate. It results from the aspiration of gastric contents in addition to aspiration of upper respiratory flora in secretions. Important predisposing factors include impairment of the swallowing mechanism (eg, esophageal disease), inadequate cough reflex (eg, anesthesia, postoperative state, central nervous system disease, drug abuse), and impaired gastric emptying (eg, pyloric obstruction). Pulmonary injury is due in large part to the low pH (< 2.5) of gastric secretions.

Scattered areas of pulmonary edema and bronchospasm occur, and the x-ray appearance (pictures 4-5) may

be confused with that of pulmonary emboli, atelectasis, bronchopneumonia, and congestive heart failure.

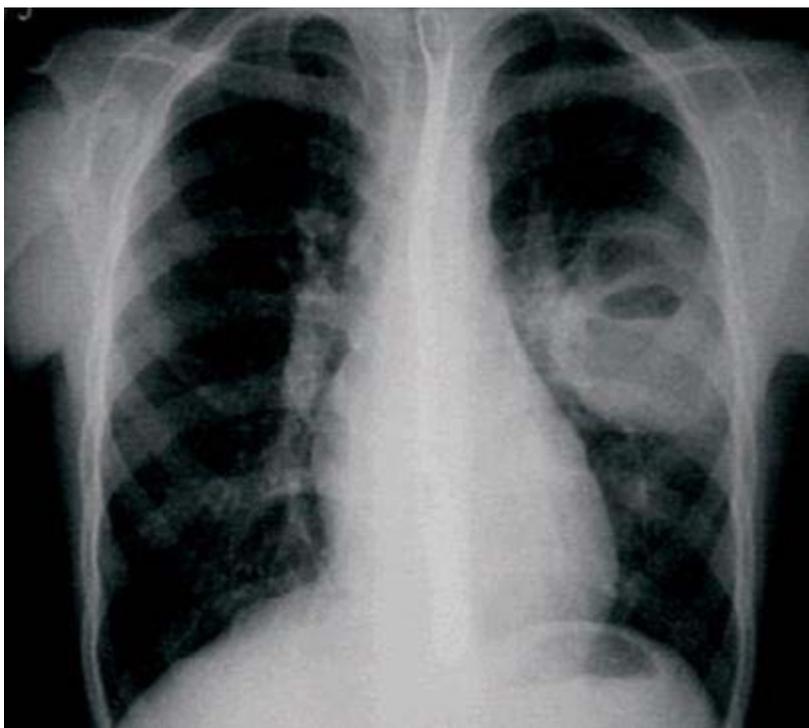
Removal of aspirated material by catheter suction or bronchoscopy may be attempted, but this usually fails to remove all aspirate completely. Corticosteroids (eg, prednisone, 100 mg orally on the first or second day) may reduce the intensity of the inflammatory reaction to acidic gastric secretion, but the value of corticosteroids in the treatment of aspiration pneumonia is not proved, and they increase the risk of superinfection. Some aspiration pneumonias have no bacterial component, but in many others a mixed bacterial flora is involved. Antimicrobial drugs directed against the latter (eg, penicillin G plus an aminoglycoside or the best available cephalosporin) are sometimes administered without waiting for evidence of progressive pulmonary infection. In doing so, however, there is a risk of favoring the development of resistant microorganisms. Therefore, administration of antimicrobials should not continue without laboratory and clinical evidence of microbial infection. Assisted ventilation and supplementary oxygen are beneficial.

The cancer of the central location due to the obturation of bronchus results in atelectasis of a segment or lobe of lungs, with probable further abscessing. For the differentiation used tomography (reveals the obturation of bronchus by tumour, lesion of central lymph nodes), cytological examination of sputum and bronchial outwashes. The determinant role belongs to fibrobronchoscopy with a biopsy and verification of the diagnosis.



Central lung carcinoma

The peripheral cancer of lungs with destruction on tomograms is characterized by cavity with irregular inner surface, which external outline connects with root of the lung because of lymphatic metastatic spreading. The central lymph nodes frequently enlarged. The diagnosis is improved by results of transthoracic puncture or catheterizing biopsy with cytological investigation, fibrobronchoscopy. If it is impossible to confirm the diagnosis the thoracotomy is indicated.



The peripheral cancer of lungs

The tubercular cavern is mainly located in the upper lobes of lungs, roentgenologically revealed on the background of characteristic changes in adjacent pulmonary tissue (calcification, dissemination), sometimes detected a draining bronchus. In the sputum mycobacteria of tuberculosis are frequently found.



The tubercular cavern

The suppurative cyst of lungs differs by a gradual onset, slow course of the suppuration, less expressed intoxication. Roentgenologically its cavity has the oval or rounded shape with a thin sheath and regular contour. Perifocal infiltration is not characteristic.

Tactics and choice of treatment

The tactics in acute pulmonary destruction should be mainly conservative.

1. The adequate antibacterial, antiinflammatory therapy consists of intravenous introduction of antibiotics of a wide spectrum activity.

ANTIBIOTICS IN LUNG ABSCESS

Anaerobic organisms[3]

- First choice - Clindamycin (Cleocin 3)
- Alternative - Penicillin
- Oral therapy - Clindamycin, metronidazole (Flagyl), amoxicillin (Amoxil)

Gram-negative organisms

- First choices - Cephalosporins, aminoglycosides, quinolones
- Alternatives - Penicillins and cephalexin (Biocef)
- Oral therapy - Trimethoprim/sulfamethoxazole (Septra)

Pseudomonal organisms: First choices include aminoglycosides, quinolones, and cephalosporin.

- Gram-positive organisms
- First choices - Oxacillin (Bactocill), clindamycin, cephalexin, nafcillin (Nafcil), and amoxicillin
- Alternatives - Cefuroxime (Ceftin) and clindamycin
- Oral therapy - Vancomycin (Lyphocin)

Nocardial organisms: First choices include trimethoprim/sulfamethoxazole and tetracycline (Sumycin).

With the purpose of maximal concentration of drugs in the pathological focus applied:

- Injection of antibiotics in the vessels of a pulmonary circulation by means of catheterization of central veins, pulmonary artery;
- Introduction of medicinal agents into respiratory tracts (in the second stage) – through the endotracheal microirrigator, nasogastric tube, during bronchoscopies, endoscopic catheterization of the abscess cavity through the draining bronchus, in aerosolic inhalations. The composition of medical admixtures includes: antibiotics, antiseptics (10 % dimexid, dioxydin, microcid etc.), enzymes;

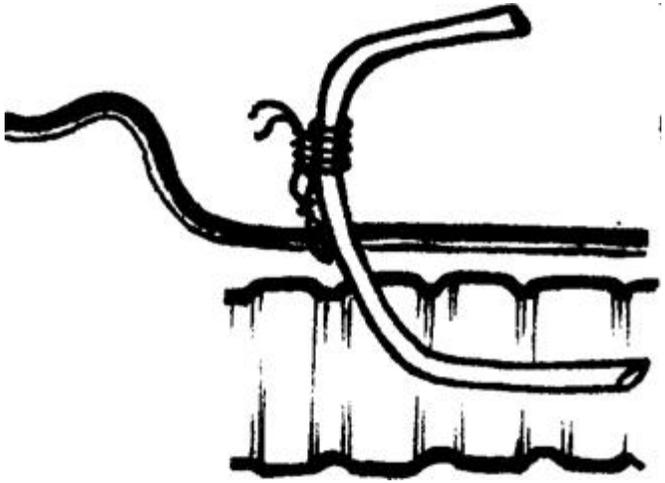
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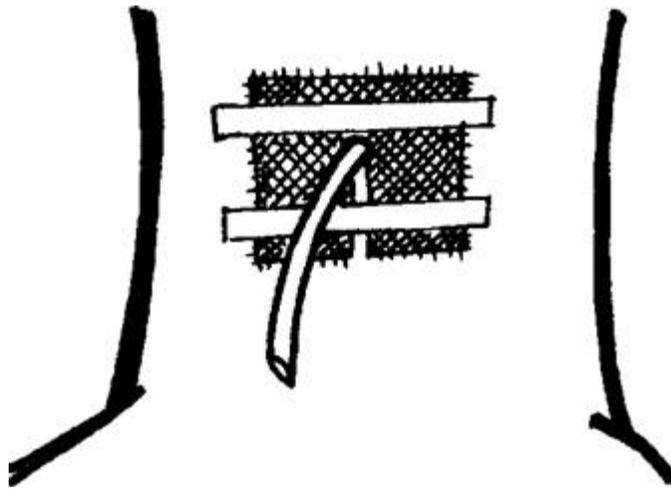
Tracheocentesis after local anesthesia



Insertion of the conductor



Incertion of the irrigator along the conductor



Skin wound

- Transcutaneous in the focus of destruction by means of puncture or draining with the usage of physical antiseptics (US, UVR, laser).
- Intrapleural;
- By means of electrophoresis.

2. Evacuation of purulent content of the cavities:

- In natural way by an active sanitation of tracheobronchial tree using repeated fibrobronchoscopies, aspirations through the endobronchial catheter, installations of medical agents through the microtracheostomy, aerosolic inhalations;
- Transthoracically by means of repeated punctures or external draining of peripheral cavities.

3. Detoxycation therapy (intra- and extracorporal).

4. Immune correction (under the control of immunogram):

- Active – staphylococcal anatoxin according to the plan;
- Passive – specific gamma-globulins, hyperimmune plasma;
- Non-specific (pyrimidine and purine derivatives, drugs of a thymus gland, splenin, levamisol,).

5. Homeostatic correction (oxygenotherapy, correction of anemia, hypoproteinemia, acidosis, microcirculatory disturbance).

6. Desensitizing, antiinflammatory therapy, regulation of activity of proteases: antihistamine, nonsteroid antiinflammatory agents, inhibitors of proteases, antioxidants.

7. Correction of dysfunction of the vital organs and systems, prevention of complications, symptomatic therapy.

Indications for operative management in acute destructive processes of lungs:

- Pulmonary bleeding of II- III degree;
- Progression of the process on the background of active and appropriate therapy;
- Tense pyopneumothorax, which is failed to liquidate by the draining of pleural space;
- Impossibility to rule out the suspicion on a malignant tumour.

Contraindications: decompensation of the vital functions and systems in the terminal stage, bilateral purulent destruction of lungs, concomitant incurable malignant tumours.

Several important factors must be considered prior to undertaking surgery. Because of the high risk of spillage of the abscess into the contralateral lung, it is almost essential that a double-lumen tube be used to protect the airway. If this is not available, surgery poses a very high risk of abscess in the other lung and a risk of ARDS. In such cases, postponing the surgery is a wise decision. Another, less-satisfactory method to deal with this problem includes positioning the patient in the prone position. The surgeon must be skilled in resecting the abscess and in rapid clamping of the bronchus to prevent spillage into the trachea. These factors are extremely important when dealing with the surgical aspects of treating a lung abscess. If doubt persists, postponing the surgery is best.

Surgical treatment is now rarely necessary and is almost never the initial choice in the treatment of lung abscesses. In current practice, fewer than 15% of patients need surgical intervention for the unchecked disease and for complications that occur in both the acute and chronic stages of the disease.

Surgical management is reserved for specific indications such as little or no response to medical treatment, inability to eliminate a carcinoma as a cause, critical hemoptysis, and complications of lung abscess (eg, empyema, bronchopleural fistula). In addition, if after 4-6 weeks of medical treatment a notable residual cavity remains and the patient is symptomatic, surgical resection is advocated.

The results of surgery are difficult to assess because of the varying patient population and the tremendous increase in illicit drug abuse, alcoholism, AIDS, and infections by

gram-negative and opportunistic organisms. These factors have increased the incidence of lung abscess and the associated morbidity.

A great deal of caution is needed during anesthesia when patients with lung abscess undergo surgery because spillage of the abscess material into the uninvolved lung can occur. Therefore, a double-lumen endotracheal tube is used in all cases.

A study by Nagasawa et al has shown that thoracoscopic surgery can lead to effective drainage of pediatric lung abscess without major complications. In addition, other benefits of thoracoscopy include rapid recovery, less pain, and minimal morbidity.

Operational incisions – anterolateral, lateral and posterolateral thoracotomy. The operation suggests segmental, polysegmental resection, lobectomy, bilobectomy combined intervention (with the decortication, pleurectomy).



Lateral access



Anterior access



Posterior access

The patients with chronic abscesses should be undergone the operative treatment after complete liquidation of exacerbation.

In a pulmonary gangrene the stabilization of the process on the background of active conservative treatment allows in future to apply conservative tactics up to recovery or making optimal conditions for operation (liquidation of intoxication, aggressive panbronchitis, diffuse infiltration of the parenchyma, pleural complications). The headlong progression of the gangrene during first days, despite the active correction,

occurrence of pulmonary bleeding requires urgent performance of operative management. Volume of the operation – pneumonectomy, bilobectomy, lobectomy.

PLEURAL EMPYEMA

The pleural empyema is a purulent inflammation of its visceral and parietal membranes, which is associated with accumulation of pus in a pleural space.

Etiology and pathogenesis

The causes of occurrence of acute pleural empyema are inflammatory, or purulent and destructive processes of lungs, abscesses of abdominal cavity (secondary pleural empyema), open and closed damages of chest, and also, in some cases, operative approaches on thoracic organs (primary pleural empyema).

A secondary pleural empyema occurs in 88 % of the patients. Thus develops fibrinous, exsudative, and then purulent pleurisy.

In case of pulmonary gangrene, purulent mediastinitis, subphrenic abscess the stage of exsudative pleurisy extremely short. The progression of the process results in transferring of focal pleural empyema into wide-spread.

Pathology

Macroscopically pleura is thickened, covered by pus with punctate hemorrhages. Microscopically it is diffusely ooazed with neutrophils. In cases, when the empyema overcome into chronic course, the pleura deposits the calcareous salts, thick pus encapsulated, sometimes with the development of fistula.

Classification

I. According to the etiological factor:

1. Specific.
2. Nonspecific.

II. According to the pathogenic factor:

1. Primary.
2. Secondary.

III. According to the clinical course:

1. Acute.
2. Chronic.

IV. According to extension of the process:

1. Focal.
2. Wide-spread.

V. According to the presence of lung destruction:

1. Empyema with destruction of pulmonary tissue.
2. Empyema without destruction of pulmonary tissue.
3. Pyopneumothorax.

VI. According to communication with environment:

1. Closed pleural empyema;
2. Open pleural empyema:
 - bronchopleural fistula;
 - thoracopleural fistula;
 - thoracopleurobronchial fistula;
 - cribrate lung.

Symptomatology and clinical course

The clinic of an acute pleural empyema depends on extension of the process, reactivity of organism and presence of complications.

The **pain** is the sign, which denote the involvement of pleural membranes in the process. Its intensity increases depending on depth of respiration and body position.

The **dyspnea** arises from accumulation of a purulent content in a pleural space and exception of particular volume of a pulmonary tissue from respiration. It's in direct ratio to amount of exudation in a pleural space.

The cough is manifestation of inflammation or purulent and destructive process in a pulmonary tissue.

Fever to 39-40°C, headache, sleeplessness, general malaise, and anorexia – all these are manifestations of intoxication.

The forced patient's position and restriction of breathing should be considered as outcomes of a pain syndrome. The extension of pleural empyema causes the swelling of thoracic wall, smoothing of intercostal spaces.

By palpation – diminished vocal fremitus on the part of lesion.

The data of percussion and auscultation depend on extension of the process and amount of pus in a pleural space. At percussion over the exudate it is possible to reveal short sound with oblique upper contour. Above the exudate – tympanic sound resulting from consolidation of pulmonary tissue. By auscultation – diminished or absent sound in a great amount of exudate.

The predominant roentgenological sign of a focal or wide-spread empyema – the presence of exudate. In localized acute pleural empyema observed a local intensive homogeneous shadow. Roentgenologically according to localization distinguished such types of a focal empyema:

- 1) apical;
- 2) paramediastinal;
- 3) parietal;
- 4) interlobar;
- 5) epiphrenic.

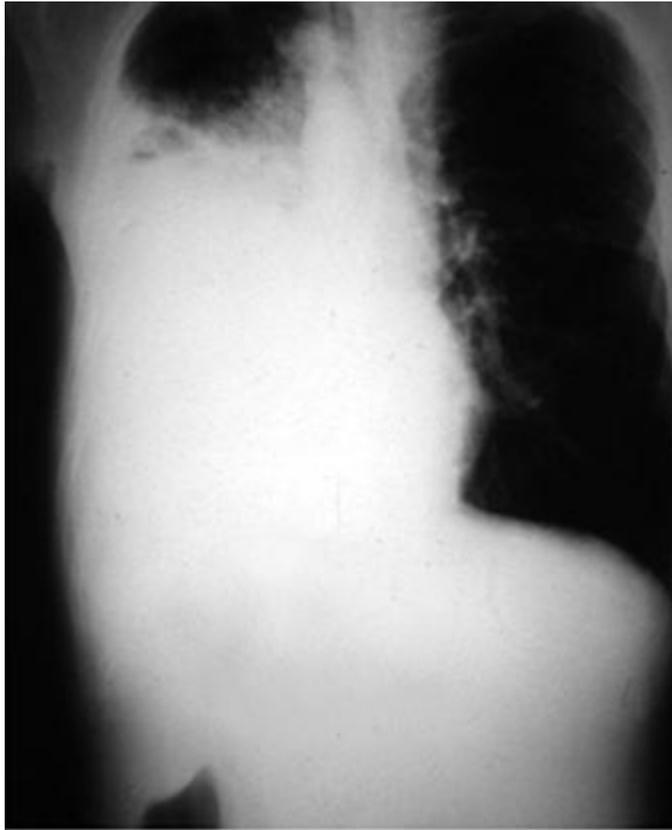
The wide-spread pleural empyema manifests by intensive homogeneous shadow in a basal parts with oblique upper contour (Damuaso' line). The diaphragmatic dome is failed to observe. The more pus contents in a pleural space, the higher the upper measure of exudate.



Left-side pleural empyema



Pleural empyema



Pleural empyema



Pleural empyema



Pleural empyema

Variants of clinical course and complications

The clinic of a focal pleural empyema depends on the site of the process. The apical empyema, due to involvement in the process of a vascular-nervous fascicle, manifests by intensive pain. The soft tissues of supraclavicular region are swelled. The percussion and auscultation has no information.

The pain syndrome in parietal (paracostal) empyema is more expressed. Thoracic excursion is restricted. The diminishing of respiratory sound can be obtained over the exudate.

The chief complaint in paramediastinal empyema is the heart pain. The location of the process in the upper mediastinum can cause the superior vena cava syndrome. The physical findings are vague.

In case of the basal (epiphrenic) empyema the patients complain of pain in subcostal area, which increases at respiration and irradiates in supraclavicular region. In some cases the pain irradiates in epigastric region. The palpation of intercostal spaces and hypochondrium is painful.

The clinical course of postoperative empyema depends on the character of operative approach (marginal resection of lung, lobectomy, pneumonectomy, operation on esophagus) and infection of the pleural space.

The clinical manifestations of posttraumatic empyema depend on the size of damage of the chest, lungs, mediastinal organs and complications (suppuration, hemothorax).

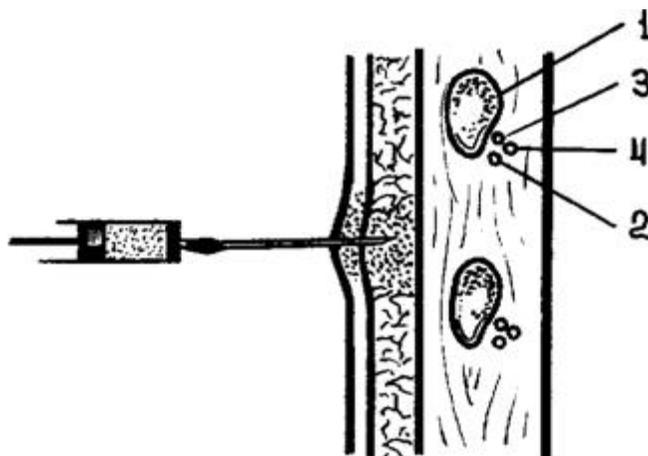
The involvement in the purulent process of a pulmonary tissue in acute empyema results in its fusion of membranes with formation of a bronchial or thoracopleural fistula (discharge of abscess through thoracic wall).

The inappropriate elimination of empyema results in chronic course, cribrate lung and pleurogenic cirrhosis of lungs.

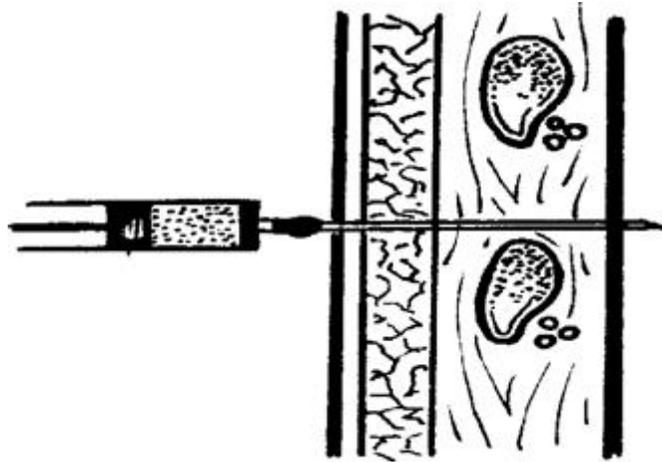
The diagnostic program

1. Complaints and history of the disease.
2. Physical findings.
3. Data of chest X-ray (in two planes, if necessary – laterography).
4. Pleural puncture.
5. The microbiological investigation of the exudate for its sensitivity to antibiotics.
6. General blood and urine analyses.
7. Biochemical blood analysis.
8. Pleurography (in transferring of the process into chronic form).

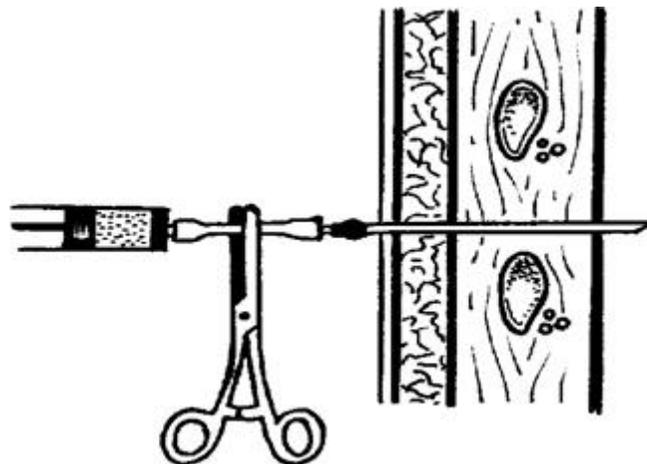
Pleural puncture:



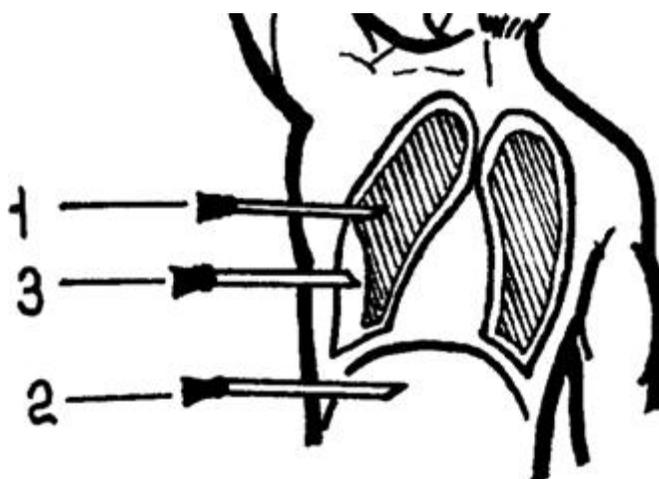
Local anesthesia



Thoracentesis



Aspiration of the fluid



Localization of the needle:

- 1 the needle damaged the lung
- 2 the needle damaged the diaphragm
- 3 the needle located in pleural sinus

Differential diagnostics

Pleuropneumonia complicated with exsudative pleurisy, in some cases resembles acute pleural empyema: a chest pain, fever, dyspnea, cough, and general weakness. The chest roentgenogram reveals hydrothorax (exsudative pleurisy, pleural empyema, hemothorax). The chief diagnostic method for differentiation is the thoracentesis. The presence of a serous (lucent, faint-yellow) exudate testifies about the pleuropneumonia, complicated with pleural effusion, and cloudy, foul-smelling exudate of white or greenish color – about acute empyema.

The major difficulties in differential diagnostics cause the limited forms of empyema.

The **Pancoast cancer** clinically and roentgenologically in most cases has almost the similar course to apical form of empyema. The transthoracic biopsy permits to confirm the diagnosis.

The **acute cholecystitis** is necessary to differentiate with the epiphrenic empyema. The pain in the right hypochondrium, fever, phrenic symptom are common for both diseases. However the objective findings, X-radiography of chest and the thoracentesis allow to differentiate these pathological processes.

The **tumour of anterior mediastinum** complicated by superior vena cava syndrome is necessary to differentiate with paramediastinal empyema. Nevertheless the body temperature in such patients, as a rule, normal. The upper cavography is possible to find out the shift of cava vein and its irregular contours (filling defect) due to growth of the mediastinal tumour.

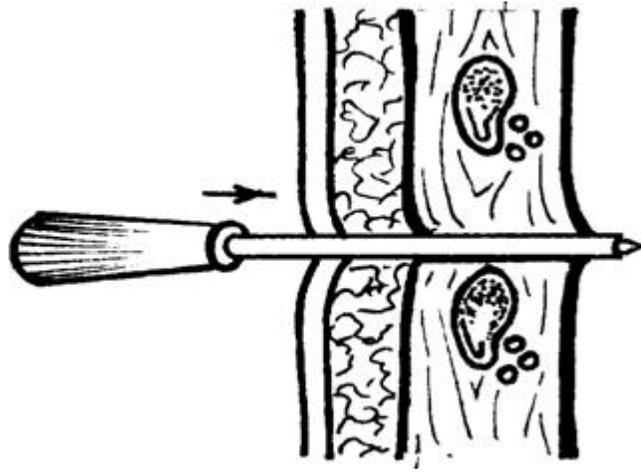
There are some difficulties in differential diagnostics of empyema with a **posttraumatic diaphragmatic hernia**. Such X-ray findings as deformation of diaphragm, additional shadows with a liquid level, intestinal loop suggest a diaphragmatic hernia. A laterography and contrast study of gastrointestinal tract is basic in differential diagnostics of this disease.

The atelectasis of a segment, and lobe of lung in some cases can cause misdiagnostics. Except X-ray chest examination (in two planes and tomography), these situations require necessity of diagnostic bronchoscopy, which reveals the cause of bronchial obturation (foreign body, endobronchial cancer etc.).

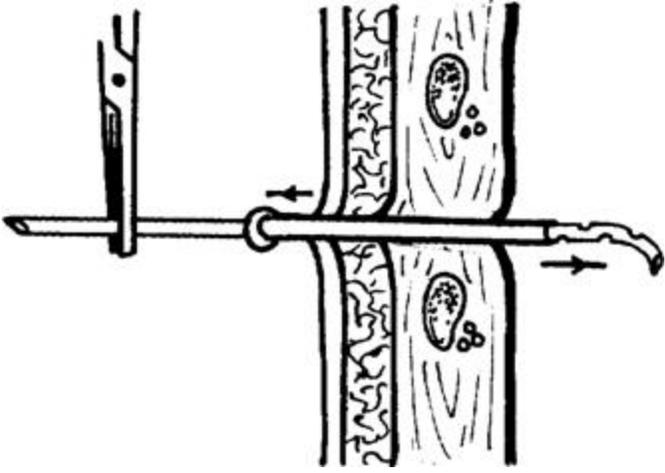
Tactics and choice of treatment

The presence of pus in a pleural space is the indication for its elimination. In the place of performed diagnostic thoracentesis carried out the draining of empyema's cavity, its sanation by means of antiseptic solutions. In a focal empyema the aspiration of pus is performed by thoracentesis and only in its inefficiency carried out a draining of pleural space.

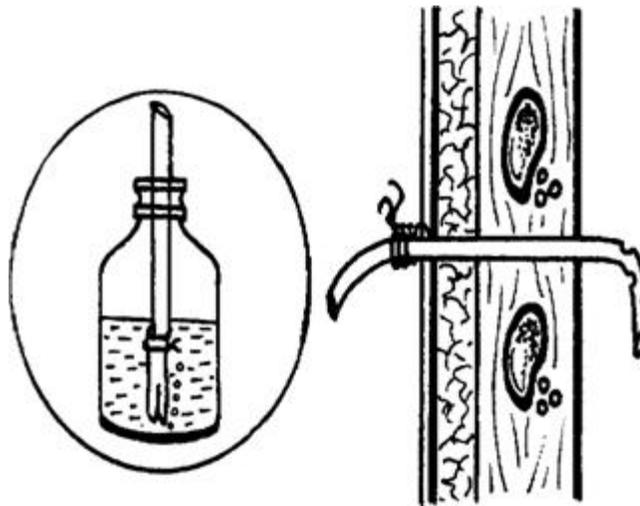
Pleural drainage:



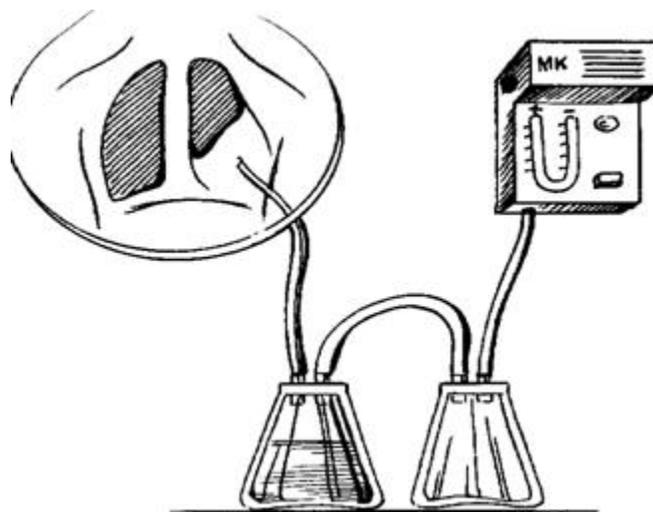
Trocar insertion



Incerption of drainage tube



Passive drainage by Bulau



Active aspiration

Intensive antibacterial and antiinflammatory therapy should be immediately instituted. For general improving used detoxication therapy (infusion of saline solutions, hemotransfusion, transfusion of proteins, solutions of dextran, haemodes, forced diuresis, hemosorption if necessary), therapy for rising up of immunological resistance of the organism.

During the empyema's sanitation decreases the amount of pus which discharges out through the drainage. The optimal variant of such course is the liquidation of empyema's cavity, then the drainage must be removed.

Transferring of the process into the chronic form (10-12 weeks) results in formation of a residual empyema's cavity, which is possible is to reveal by means of pleurography – introduction through the drainage of water-soluble contrast with the further X-radiography in 2 planes.

Operative approach is applied when the process has transferred into the chronic form, that is in case of residual empyema's cavity. Volume of the operation – pleurectomy, decortication of lung.

In some cases, when a bronchial fistula and great empyema's cavity has been formed, there is the necessity of performance of resection of lung and corrective thoracoplasty.

LUNG CYSTS

The cysts of lungs are the thin-walled cavitory formations, filled with air or liquid contents.

Inherent and acquired cyst are distinguished.

Etiology and pathogenesis

Inherent cysts arise from the abnormalities of the development of lungs under influence of the multiple chemical, physical, biological factors. Whether they can develop from a bronchial tree (bronchial) or from alveolar tissue (alveolar). Their occurrence resulting from the delay of the development of peripheral parts of bronchus with their expansion or agenesis of alveoli with dilatation of terminal bronchioles. Congenital cysts at first develop and grow as secretory formations. After communication with bronchus they finally form as air or hydroair cavities.

The acquired cysts represent fibrous cavities, which remain after abscesses, tubercular caverns, echinococci, posttraumatic intrapulmonary hematomas. The degenerative changes in the wall of bronchus with obliteration of its lumen by a cloggy secret owing to repeated inflammatory processes result in occurrence of acquired retentional cysts.

Pathology

Congenital cyst can be located in any part of lungs. The walls of bronchogenic cavities contain chaotically disposed elements of bronchus (cartilagous plates, muscle fibers, mucous glands), they are lined from inside by cylindrical or cubic epithelium. Squeezed alveolar cells line the walls of alveolar formations.

The acquired cyst are revealed in places of localization of previous diseases,. Their walls mainly consist of connective tissue. Epithelization is possible due to transferring of the epithelium from a draining bronchus at long existence.

The cysts could be uni- or multichamber, closed and open (depending on the presence of communication with bronchus).

Classification

According to parentage:

- congenital;
- acquired.

According to displacement:

- single, multiple;
- unilateral, bilateral.

Complications:

- Suppuration;
- Appearance of the valvular mechanism;
- Bleeding;
- Discharge into a pleural space (pneumothorax, pyopneumothorax, pleural empyema);
- Malignancy.

Symptomatology and clinical course

Clinical manifestations of uncomplicated pulmonary cysts are vague. Sometimes patients complain of a chest pain, periodic cough, and inflammatory diseases of respiratory tract in history. In children the signs are much expressed, the dyspnea associates with the compression of airways.

In great, superficially disposed cysts revealed delayed respiratory movements on affected side during breathing.

By palpation - weakened vocal fremitus.

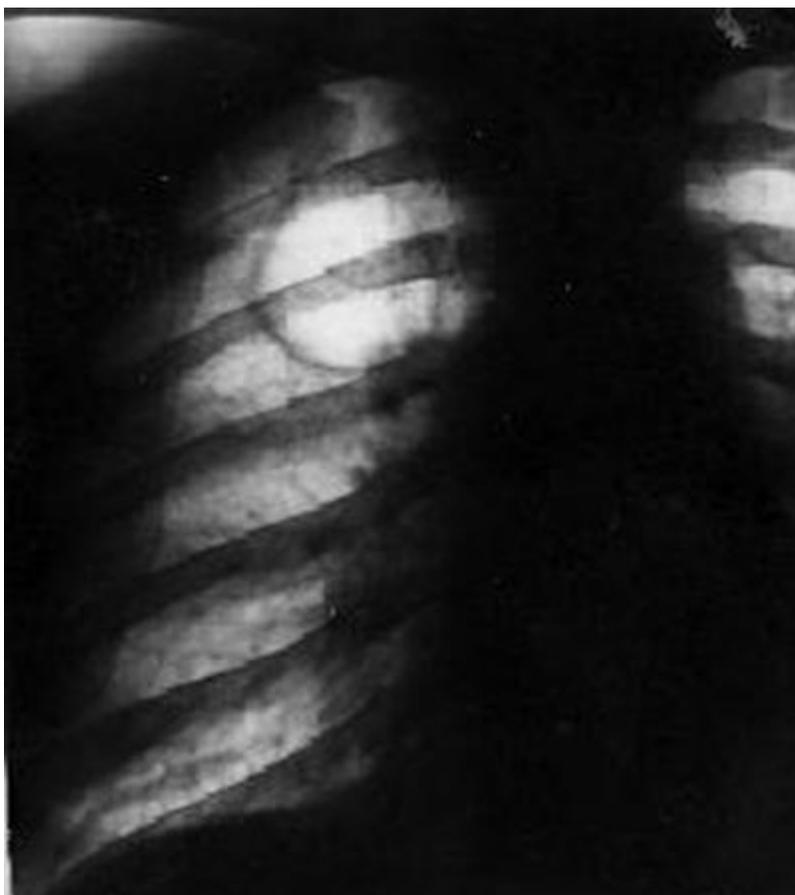
By percussion - short or bandbox sound, depending on contents.

By auscultation - weakened respiration, over the huge open cavities – amphoric.

At routine chest films the closed congenital cyst forms a homogeneous shadow of the spherical or oval shape of average intensity with rather regular edge on the background of an intact pulmonary tissue. During roentgenoscopy sometimes observed the change of its shape depending on the phase of breathing – elongation in inspiration (Escudero's syndrome). The character of the shadow is better to study on tomograms.

Acquired retentional cyst is roentgenologically shown by a shadow of irregular shape (piriform, spindle-shaped etc.), that displays the shape of the distended bronchus. The adjacent tissue, as a rule, changed due to

bronchiectases, pneumosclerosis, etc.



Cyst of the right lung

Open cysts are observed as thin-walled, good defined cavities of annular or oval shape.

On bronchograms congenital cysts are observed hypoplasia of segmental or lobar bronchus, lack of bronchial bifurcations, which tends to bone. Sometimes observed bronchiectases in the neighboring parts of lungs. A cystic cavity is infrequently defined.

The angiopulmonography reveals deformation of vascular branches, which circumflex the cyst.

Variants of clinical course and complications

The **suppuration** of the closed cysts resembles the development of a lung abscess with lesser expression of

signs of intoxication (protective role of epithelial wall).

Infection of the open cysts is characterized by a gradual long course, moderate manifestation of suppuration and late intoxication.

The tension (valvular) cysts arise more often in children. They are characterized by severe respiratory and hemodynamic disturbances, up to terminal, owing to inflexion of venous trunks, shift of mediastinum and compression of lungs. The predominant signs are increased dyspnea, cyanosis, chest pain, respiratory lag on affected side, auscultatory – absence or weakening of respiration, bandbox percussion sound over the cavity at mediastinal shift to the opposite side of the chest.

Pleural complications of pulmonary cysts, and also **bleeding** described in the relevant parts.

The malignant degeneration of pulmonary cysts occurs very rarely.

The diagnostic program

1. Complaints and history of the disease.
2. Physical methods of examination.
3. Routine chest film in two planes.
4. Lung tomography.
5. Bronchography.
6. Angiopulmonography.
7. General blood and urine analyses.

Differential diagnostics

The abscess of lungs, in contrast with a suppurative cyst, is characterized by prompt course, expressed purulent intoxication, roentgenologically its wall is irregular, with proper considerable perifocal infiltration.

The cancer with destruction differs by a thick wall with a tuberos inner surface, phenomena of lymphangitis in adjacent tissue. The determinant value has endoscopic examination or puncture with a biopsy and following morphological investigation.

The tubercular cavern displaced in the upper lobes, in adjacent tissue revealed fibrosis, petrifications,

dissemination, peribronchial lymphadenitis. In the sputum – mycobacterium of tuberculosis.

Opposite to cyst, in termed diseases, the circumflex deformation of vascular branches never observed at angiopulmonography.

The considerable difficulties can arise at differentiation of the closed congenital cysts.

The **tuberculoma** differs from them by characteristic localization. At X-ray examination the heterogeneity of the shadow with calcifications, early excentric destruction, "tubercular" background is observed.

The **benign tumours** sometimes possible to differentiate only after the results of cytological investigation of punctate or after operation.

The **echinococci cysts** on tomogram can have a double contour of chitinous and fibrous sheaths. In blood analyses observed eosinophilia. The final decision is taken out after carrying out immunological (indirect microagglutination test) allergic (Cacconi's reaction) tests.

Huge cysts, in contrast with open air cysts, are characterized by a subpleural location.

The diagnostic doubts at suspicion on a **diaphragmatic hernia** are solved by radiopaque examination of a gastrointestinal tract.

The **acquired cysts** differ from congenital by roentgenological signs of the lesion of surrounding tissues (pneumofibrosis, deforming bronchitis, secondary bronchiectases, and calcifications). Postpneumonic cavities are of irregular shape, with grooves and pockets, their walls different thick. During bronchography they filled through some small bronchi. Sometimes the verification is possible only after postoperative morphological investigation.

Tactics and choice of treatment

The pulmonary cysts require the surgical treatment. In case of complications the indication for operation becomes absolute. Contraindications: the severe respiratory disturbance, concomitant malignant nonresectable tumors, vital organs dysfunction in the stage of permanent decompensation, elderly age of the patients.

The volume of the operation: if the adjacent pulmonary tissue is intact – cystectomy, otherwise – segmental or wedged resection, lobectomy.

The conservative therapy is applied in suppuration of cysts with the purpose of preoperative preparation. It is the similar, which applied for abscesses of lungs.

SPONTANEOUS PNEUMOTHORAX

Spontaneous pneumothorax is the entry of air in a pleural space with the further lung collapse, which not associated with traumatic damage of chest or pulmonary tissue.

Etiology and pathogenesis

As a result of spontaneous disrupture of lung blebs and subpleural air cysts occurs the damage of pleural visceral membrane. It causes entry of air in pleural space. Owing to its leakage the elastic pulmonary tissue collapses. The degree of the collapse of lung depends on amount of air, that has penetrated a pleural space.

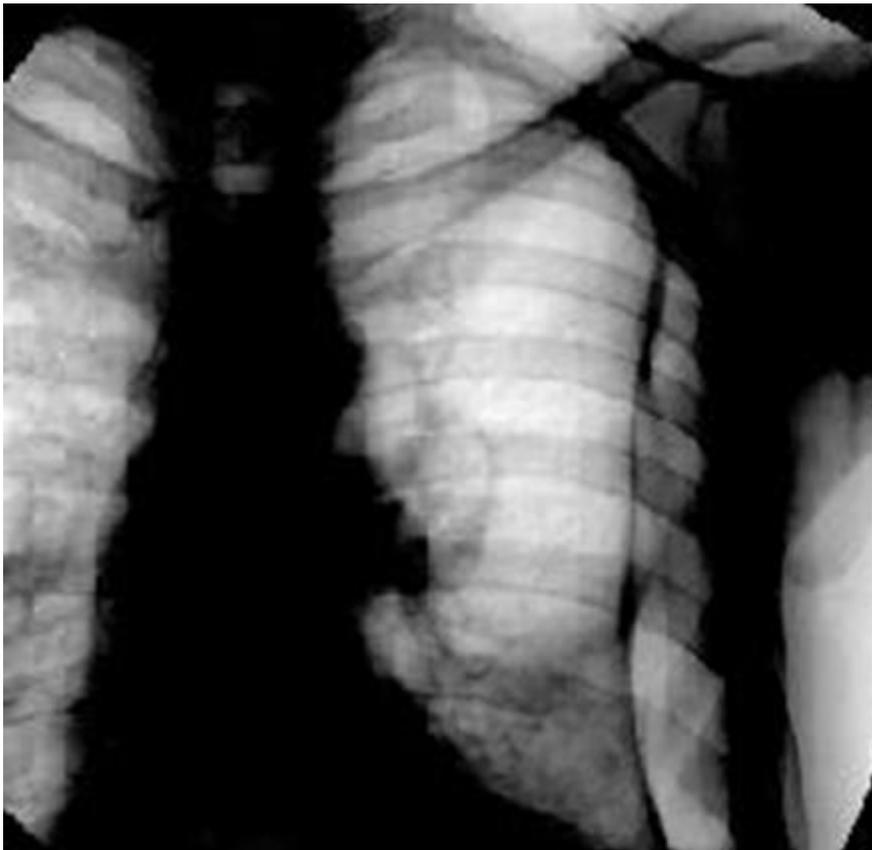
Pathology

Morphologically in spontaneous pneumothorax found out a focal bullous emphysema with disrupture of blebs, subpleural air cyst, and also disorders, which have caused disturbances of ventilating ability of bronchi. It can be bronchitis, pneumosclerosis, tuberculosis and fibrous alveolitis.

Classification

The pneumothorax can be:

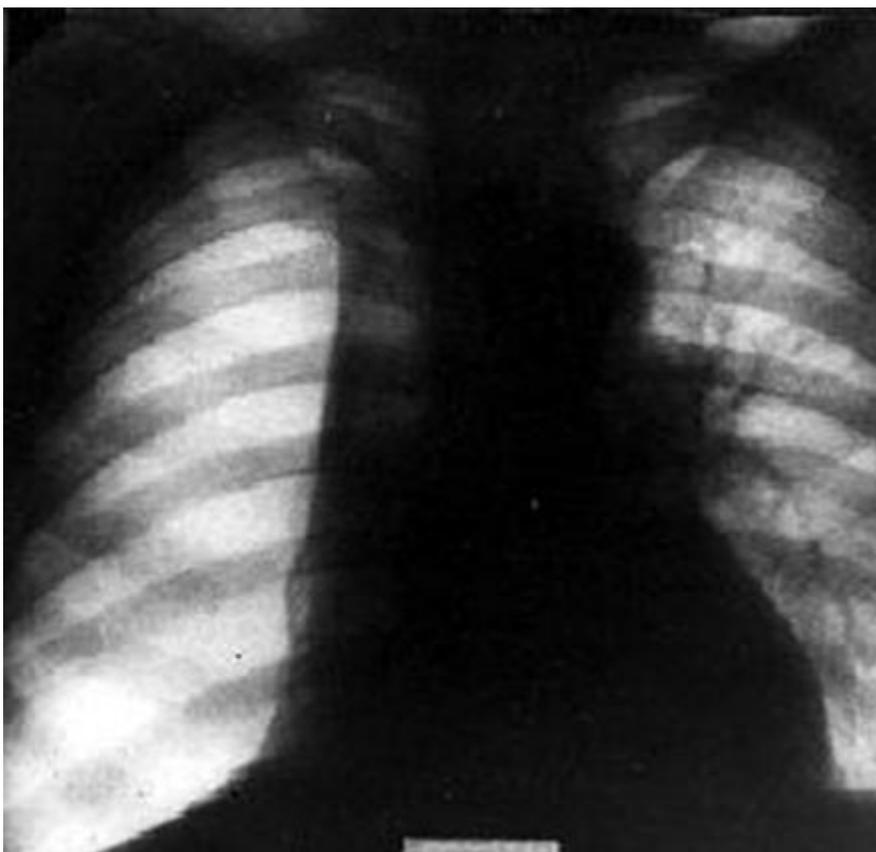
1. Unilateral or bilateral.
2. Partial (lung collapse to 1/3 of its volume).
3. Subtotal (lung collapse to 2/3 of its volume).
4. Total (lung collapse more than 2/3 of its volume).
5. Tension or valvular (complete collapse of lungs and shift of mediastinum in the opposite side).
6. Rigid (neglected pneumothorax with thickened visceral pleura).



Partial spontaneous pneumothorax



Subtotal spontaneous pneumothorax



Total spontaneous pneumothorax

Manifestation and clinical course

The onset of the disease is sudden. A state of the patient and expression of clinical manifestations depends on amount of air, which has entered the pleural space. Usually in normal conditions, and sometimes after physical activity, the patients suddenly feel acute pain on the side of lesion, dyspnea, pain in the heart region and heartbeating. An available also Acrocyanosis or total cyanosis of skin is observed. The circulatory disturbance depends on degree of hypoxia. Intensity of pain and dyspnea gradually decrease, but a dry troublesome cough appears.

Examination of the chest allows to observe expansion of intercostal spaces and restriction of respiratory excursion. By palpation - diminishing of vocal fremitus on the affected side. At percussion a chief sign of pneumothorax is the tympanic sound. Auscultation reveals weakened or sharply weakened breathing sounds. The cardiac tones are muffled, tachycardia.

The diagnosis of a spontaneous pneumothorax is confirmed by X-ray examination. On the plain roentgenogram the air is present in pleural space, and the margins of collapsed lung are found out on its background.

Thoracoscopy in pneumothorax possible to find out subpleural blebs of different sizes (0,5-3 cm), which are mainly disposed on the apex of lung.

Variants of clinical course and complications

The atypical (asymptomatic) spontaneous pneumothorax occurs in 20 % of the patients, and mainly revealed at X-ray examination. In most cases it is the partial pneumothorax.

The expressed pain syndrome and dyspnea, which resulting from collapse of lung, characterize subtotal and total pneumothorax.

The tension spontaneous pneumothorax is the most severe form of pneumothorax. It manifests by sudden onset, progressive increase of dyspnea, expressed cyanosis. The breathing is superficial, rapid, with active participation of auxiliary muscles. Mediastinal shift and flexion of vessels result in disturbance of cardiac activity up to the cardiac arrest, and requires urgent management.

Rigid pneumothorax (rigid lung). The neglected pneumothorax causes fibrinous exsudative pleurisy. On the surface of lung (visceral pleura) commissures are formed, that give no opportunity for lung expansion. The presence of residual pleural cavity and progressive development of a purulent infection result in occurrence of acute pleural empyema. In such cases the patients complain of the fever up to 38-38,5°C, general weakness and increasing of dyspnea. The phenomena of intoxication increase. Thus such patients require the treatment of pleural empyema.

The diagnostic program

1. Complaint and anamnesis of disease.
2. Physical findings.
3. Routine chest film (direct and lateral projection).
4. [Thoracentesis](#)
5. Thoracoscopy.
6. Tomography of lungs.

Differential diagnostics

Pleural effusion. This pathology manifests by more gradual onset. As opposite to pneumothorax, the complaints of chest pain predominate above the complaints of dyspnea. Frequently such patients specify on transferred undercooling.

As well as the spontaneous pneumothorax, the pleural effusion is characterized by diminishing of vocal fremitus, dullness of percussion sound, weakened or absent breathing sounds over the exudate. Nevertheless in the routine roentgenogram in such cases observed a homogeneous intensive shadow of a pleural space with oblique upper contour. The puncture of pleural space enables finally to confirm the diagnosis of pleural effusion.

Intercostal neuralgia. A predominant sign in clinical pattern is acute pain, which intensifies at physical activity, changes of body position and body movements, at deep breathing. The localization of pain coincides with zone of innervation of intercostal nerves.

Examination and chest X-radiography reveals no pathological changes.

Tactics and choice of treatment

Conservative treatment is applied in the patients with a partial pneumothorax. Thus thoracentesis in II intercostal space in the midclavicular line with aspiration of air is performed. The cases of its inefficiency, and also subtotal, total and tension pneumothorax require drainage of a pleural space with active aspiration of air.

The operative management is necessary, if there is no efficiency from active aspiration (in incomplete expansion of lung), recurrent of course of the process, presence of great subpleural blebs and rigid pneumothorax. Volume of operation depends on extension of the process: liquidation of alveolar fistula, wedged resection of lung or lobectomy.

PYOPNEUMOTHORAX

Pyopneumothorax is the discharge of lung abscess of into pleural space, which is accompanied by purulent inflammation of pleural membranes with a collapse of lung.

Etiology and pathogenesis

Peripheral placement of the purulent focus in a pulmonary tissue results in destruction (fusion) of visceral membrane. As a result of, the pus and air penetrate into a pleural space that leads to a purulent inflammation of parietal and visceral membranes of pleura. The disorder of hermeticity of a pleural space results in a lung collapse.

Among other causes of pyopneumothorax are the chest trauma, which results in collapse of lung, infection and purulent inflammation of pleural membranes.

As the basic causes of pyopneumothorax are considered:

- acute abscess of lung;
- gangrenous abscess of lung;
- gangrene of lung;
- suppurative cyst of lung;
- abscessing pneumonia;
- bronchiectatic disease;
- subphrenic abscess, which has discharged into pleural space;
- damage of esophagus;
- mediastinitis;
- chest trauma;
- operation and diagnostic manipulations on chest organs.

Pathology

Morphologically in pyopneumothorax pus and air are present in pleural space. In the lungs subpleural disposed purulent or necrotic foci, which connected with a pleural space through a pleuro-pulmonary fistula. From the outside the zone of disrapture is confined by perifocal inflammation. In the draining bronchus it is possible to see manifestations of deforming, frequently polypous bronchitis.

Classification

I. According to etiological factor:

1. Specific.
2. Nonspecific.

II. According to pathogenic factor:

1. Primary.
2. Secondary.

III. According to clinical course:

1. Asymptomatic form.
2. Mild form.
3. Acute form.

IV. According to extension of the process:

1. Localized pyopneumothorax:

- a) parietal;
- b) apical;
- c) epiphrenic;
- d) paramediastinal;
- e) polychamber.

2. Subtotal pyopneumothorax.

3. Total pyopneumothorax.

4. Tension pyopneumothorax.

Manifestation and clinical course

The clinic of pyopneumothorax depends on the size of the focus of destruction, which influences on degree of lung collapse, and on amount of purulent content in a pleural space.

The pain owing to discharge of the focus of destruction into pleural space often arises suddenly.

The dyspnea occurs as a result of collapse of lung owing to leakage of pus and air into pleural space. Its expression is in direct ratio to lung collapse. Therefore a dyspnea in rest observed in a subtotal and total pyopneumothorax. It sharply amplifies even at minor physical activity. Auxiliary muscles take part in order to force respiration.

The expectoration of sputum with ichorous smell is the outcome of destructive process in a pulmonary tissue. Its amount decreases after discharge of pus into pleural space.

Hectic fever with caused by enlargement of the area of resorption. The patients are adynamic, flaccid. Some of them are unconsciousness.

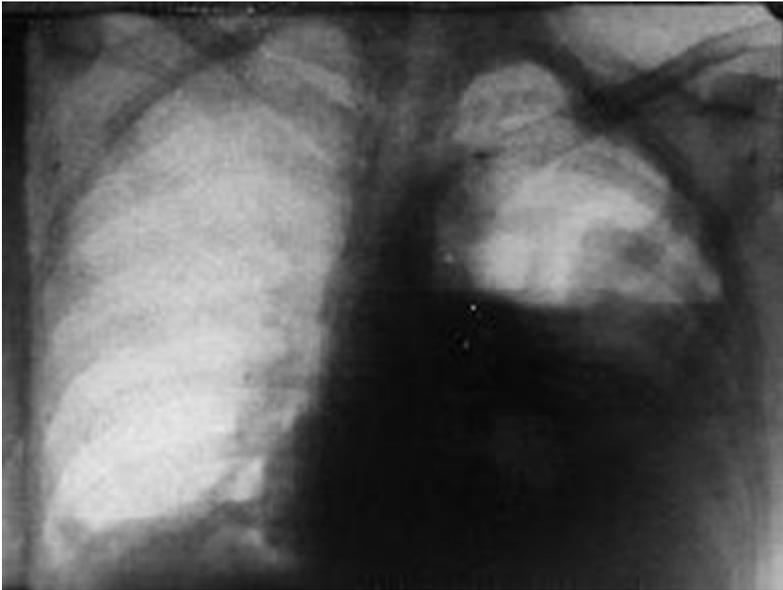
By objective examination the position of patients is forced, they sit in bed leaning upon the bed (subtotal, total pyopneumothorax). The affected hemithorax takes no part in respiration.

By palpation – diminished vocal fremitus on the side of lesion.

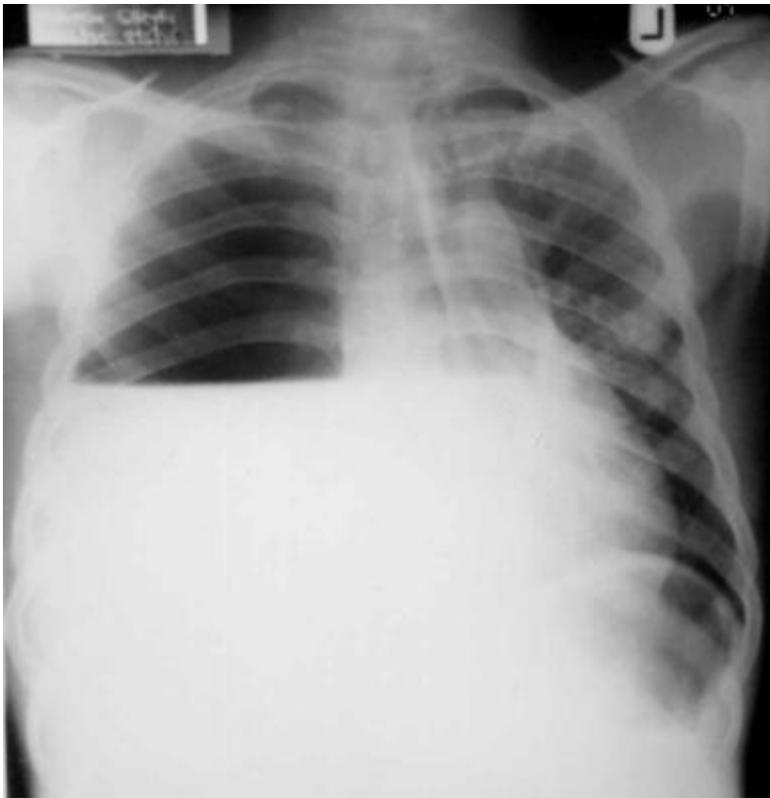
Percussion reveals a sharp shortening of sound over the zone of exudate and bandbox sound above the region of collapsed lung.

By auscultation there are no breathing sounds on the affected side. In case of localized pyopneumothorax – weakened or sharply weakened sound with a bronchial or amphoric tone.

The X-ray picture of pyopneumothorax depends on its form, but the obligatory sign is the air-fluid level in a pleural space with well-defined edge of collapsed lung on its background.



Left-side total pyopneumothorax



Pyopneumothorax



Pyopneumothorax

Variants of clinical course and complications

The clinical course of pyopneumothorax depends on adhesions between pleural membranes. It sometimes changes typical clinical course of a total lung collapse. The clinic of disease depends also on amount of a purulent exudate. Therefore according to extension of the process and size of destruction of lung distinguished acute, mild and asymptomatic forms of pyopneumothorax. Especially difficult for diagnostics is the asymptomatic form of localized pyopneumothorax. Dyspnea for such pathology not characteristic, as the adhesion of membranes prevents complete collapse of lung. Dyspnea is vague or absent at all in partial collapse. A diminished vocal fremitus on side of pathological process, shortening of percussion sound and weakened or sharply weakened breathing sounds over the collapsed lung and exudate are revealed. Roentgenological manifestations in localized pyopneumothorax not expressed and include horizontal fluid level, margin of partially collapsed lung and minor air in the pleural space.

In most cases after discharge of the destructive focus in a pleural space and collapse of lung observed the closure of bronchopleural fistula. Nevertheless the inflammatory process in a pulmonary tissue and pleural space is going on.

The diagnostic program

1. Complaint and history of disease.
2. Physical findings.
3. Chest X-radiography examination.
4. [Thoracentesis](#).
5. Pleurography.
6. Bacterial culture and antibiotic sensitivity.
7. General blood, and urine analyses.
8. Biochemical blood analysis.

Differential diagnostics

They're no special necessity for differential diagnostics of pyopneumothorax in the majority of patients. History (presence of the purulent focus in a pulmonary tissue), clinical course (acute pain and dyspnea at abscess discharge into pleural space), and also chest X-ray findings and thoracentesis frequently reliably permit to make the diagnosis.

In some cases a localized pyopneumothorax according to clinical course resembles a huge acute abscess of lungs. But the differences of roentgenological symptomatology permit to verify these pathological processes. In acute lung abscess the cavity of destruction localized in a pulmonary parenchyma, it is of rounded form with horizontal fluid level and expressed perifocal infiltration.

Tactics and choice of treatment

The purpose of treatment should include sanitation of the destructive focus in pulmonary tissue and liquidation of complications; that means elimination of pus and air from a pleural space and prompt expanding of lung.

1. Active sanitation of tracheobronchial tree by means of [tracheocentesis](#).
2. [Draining of pleural space](#), active aspiration of its content (air, pus) to expand the lungs.
3. Lavage of pleural space by antiseptic solutions.
4. Appropriate antibacterial, antiinflammatory and infusion therapy.

5. The therapy for rising up of immunological resistance of the organism (staphylococcal anatoxin according to scheme, antistaphylococcal gamma-globulin, antistaphylococcal plasma).
6. Endolymphatic introduction of immunity stimulators (thymalin, thymogen, T-activin).

The indications for operative management are the same, as in pleural empyema.

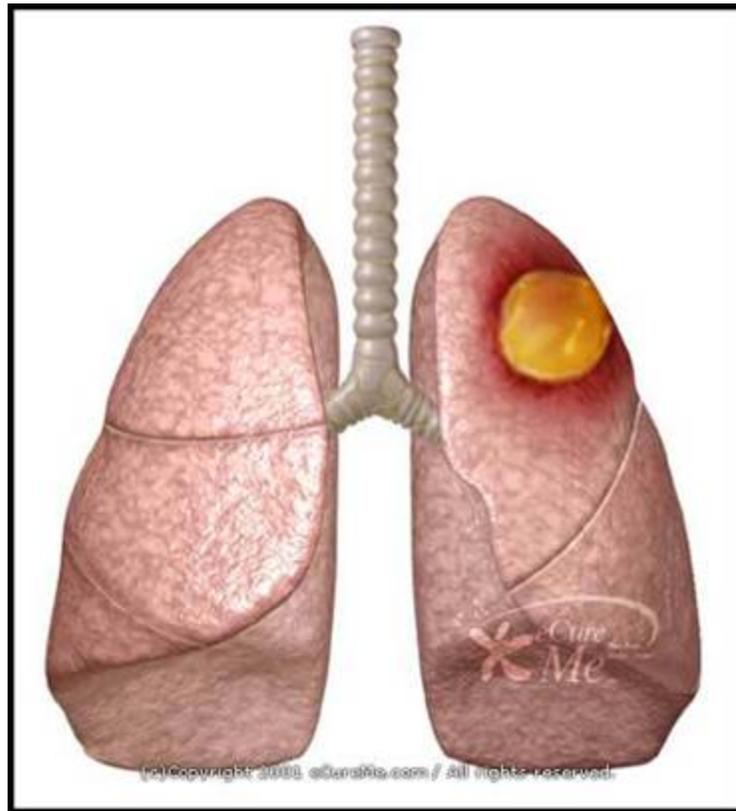
PURULENT DISEASES OF LUNGS AND PLEURA

Lung abscess is defined as necrosis of the pulmonary tissue and formation of cavities containing necrotic debris or fluid caused by microbial infection. The formation of multiple small (< 2 cm) abscesses is occasionally referred to as necrotizing pneumonia or lung gangrene. Both lung abscess and necrotizing pneumonia are manifestations of a similar pathologic process. Failure to recognize and treat lung abscess is associated with poor clinical outcome.

Lung abscess was a devastating disease in the preantibiotic era, when one third of the patients died, another one third recovered, and the remainder developed debilitating illnesses such as recurrent abscesses, chronic empyema, bronchiectasis, or other consequences of chronic pyogenic infections. In the early postantibiotic period, sulfonamides did not improve the outcome of patients with lung abscess until the penicillins and tetracyclines were available. Although resectional surgery was often considered a treatment option in the past, the role of surgery has greatly diminished over time because most patients with uncomplicated lung abscess eventually respond to prolonged antibiotic therapy.

Lung abscesses can be classified based on the duration and the likely etiology. Acute abscesses are less than 4-6 weeks old, whereas chronic abscesses are of longer duration. Primary abscess is infectious in origin, caused by aspiration or pneumonia in the healthy host; secondary abscess is caused by a preexisting condition (eg, obstruction), spread from an extrapulmonary site, bronchiectasis, and/or an immunocompromised state.

Most patients with primary lung abscess improve with antibiotics, with cure rates documented at 90-95%



Abscessing pneumonia is characterized by the multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs, which are not disposed to progression. The destruction is accompanied by expressed perifocal infiltration of a pulmonary tissue.

Abscess of lungs - purulent or ichorous destruction of necrotic sites of pulmonary tissue of one segment with formation of one or several cavities, filled by pus, and detached from adjacent parenchyma by a pyogenic capsule and expressed perifocal infiltration of surrounding pulmonary tissue. It arises at the persons with a maintained reactivity of the organism.

Gangrenous abscess is a purulent, ichorous necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma, with the liability to formation of sequestrs. Depending on reactivity of the organism it can transform into purulent abscess (after the lysis of sequestrs) or gangrene.

Gangrene of lungs – a diffuse purulent, ichorous necrosis of the tissue without the tendency to defined demarcation with prompt dynamics of spreading of necrotic zone and destruction of the parenchyma. It is characterized by a grave intoxication, liability to a pleural complications and pulmonary bleeding. If only the one lobe is affected the gangrene is considered to be limited, if affected extensive areas of lungs the gangrene is wide-spread.

Etiology and pathogenesis

Lung abscesses have numerous infectious causes. Anaerobic bacteria continue to be accountable for most

cases. These bacteria predominate in the upper respiratory tract and are heavily concentrated in areas of oral-gingival disease. Other bacteria involved in lung abscesses are gram-positive and gram-negative organisms. However, lung cavities may not always be due to an underlying infection. Some evidence suggests that individuals with cyanotic heart disorders may also be more prone to lung abscess formation. The continuous hypoperfusion of the pulmonary tissues may predispose the individuals to chronic pulmonary infections.

The predominant factors which cause the disease:

- disturbances of bronchial patency with the development of atelectasis;
- infectious inflammatory process in a pulmonary tissue;
- regional disturbances of blood supply with a further necrosis of areas of pulmonary parenchyma.

The embodiment of these factors occurs in condition of the changed reactivity of the organism.

The states which result in the aspiration of contents of the upper parts of alimentary tract (traumas of head, craniovascular disturbances, alcoholism, narcomania, narcoses, epilepsy etc.) contribute to the pulmonary abscessing. And also factors, which are capable to provoke secondary immunodeficiency and suppression of reactive processes: diabetes mellitus, irradiation, long application of corticosteroids, antineoplastic therapy, some hematological disease, AIDS favor the purulent processes.

Factors contributing to lung abscess

Oral cavity disease

- Periodontal disease
- Gingivitis

Altered consciousness

- Alcoholism
- Coma
- Drug abuse
- Anesthesia
- Seizures

Immunocompromised host

- Steroid therapy
- Chemotherapy
- Malnutrition
- Multiple trauma

Esophageal disease

- Achalasia
- Reflux disease
- Depressed cough and gag reflex
- Esophageal obstruction

Bronchial obstruction

- Tumor
- Foreign body
- Stricture

Generalized sepsis

Pathology

The abscesses mainly develop in II and VI segments of lungs. They may be single and multiple. The abscess is confined from adjacent pulmonary tissue by a capsule, which represents a granulating tissue and dense leukocytic rampart. Usually it is possible to find out a draining bronchus. Later on in the wall of the abscess the amount of connective tissue fibers is enlarged.

In gangrene the pulmonary tissue is of black color, swollen, with cavities, and in some places transfers into the sites with dark green coloring. The macropreparatus is characteristically fetid.

Classification

According to pathogenesis:

- postpneumonic;
- aspirative;
- obturative;
- posttraumatic;
- hematogenous or septic;
- lymphogenous;

- thromboembolic.

According to the character of purulent process:

- single purulent abscesses;
- multiple purulent abscesses;
- bilateral purulent abscesses;
- gangrenous abscesses (single, multiple, uni- and bilateral);
- limited gangrene;
- wide-spread gangrene.

According to stages:

- 1 stage - necrotic pneumonia;
- 2 stages - destruction and rejection;
- 3 stages - cleaning and cicatrization.

According to the term of existence:

- acute;
- chronic.

Complications:

- pulmonary bleeding;
- pyopneumothorax;
- pleural empyema;
- sepsis;

- bronchogenic dissemination.

Symptomatology and clinical course

The clinical manifestations of acute purulent destruction of lungs depend on the size of the focus and character of destruction, reactivity of the organism and stage of the disease, peculiarities of the drainage of purulent cavities and complications.

At the first stage of **acute abscess** the patients complain of general weakness, headache, malaise, suppressed appetite, moderate chest pain, dyspnea, subfebrile temperature.

At the second stage the state of the patients is worsened. The fever rises to as high as 39-40°C and has a hectic character. At the same time the chest pain increases, which associates with a troubling cough and dyspnea. The condition of the patients is worsened and the intoxication increases. One can feel a foul-smelling from the mouth at cough. The amount of sputum is small, with a rusty tone. With the beginning of the draining of destructive cavities through bronchus the daily quantity of the sputum reaches 500 ml and more. At this time is possible hemoptysis. The sputum is foul-smelling. At sedimentation it divides into three layers:

- inferior – resembling a grey mass with detrites and flaps of a pulmonary tissue;
- medial – purulent, turbid, liquid;
- upper – mucous foamy layer.

Further in favourable cases there is a considerable improvement of state of the patients. The body temperature falls, the signs of intoxication reduce and the appetite increases.

The disease grades into the third stage, which is characterized by the regress of clinical manifestations, up to their complete disappearance.

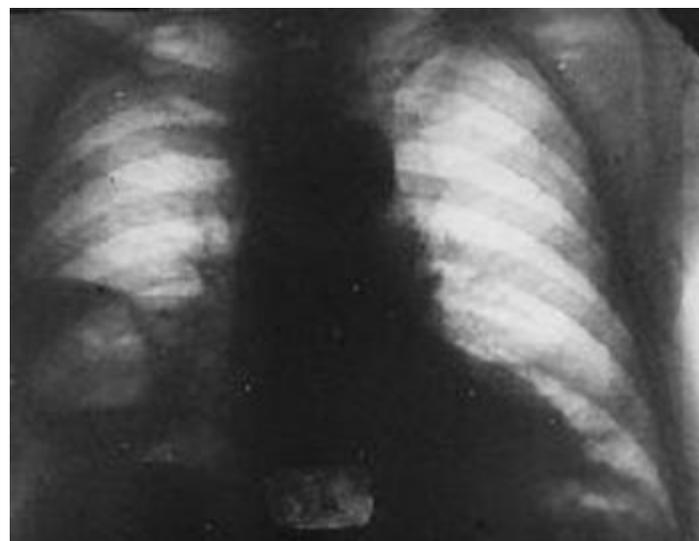
The physical signs good are well revealed at peripheral localization of the process. By palpation – weakened vocal fremitus. At percussion – a blunted sound over the site of the purulent focus and perifocal infiltration (at subpleural location of the abscess). By auscultation – tubular sound with a moist rales in the zone of purulent focus. Well-generated subpleural cavities of major sizes can be revealed by percussion by bandbox sound, on auscultation by moist rales on the background of amphoric respiration.

At X-ray of the chest at the stage of necrotic pneumonia is found out a rounded lesion with irregular contour. For the second stage is characteristic the enlightenment of the shadow with a further developing of the rounded cavity with air-fluid level (Kordin's symptom), gentle pyogenic sheath and the perifocal infiltration.

At the third stage on the place of suppuration observed expressed fibrosis, sometimes as a thin-walled annular formation.



Acute abscess of the right lower lobe before draining



Acute abscess of the right lower lobe after draining

Gangrenous abscess is characterized by a grave state of the patient, expressed purulent intoxication, cough with expectoration of a great amount (500 ml and more) of grey-green sputum with a foul-smelling, hectic body temperature. Roentgenologically the outline of the cavity is poorly defined, it contains a visible sequesters that look as a polymorphous shadow. The adjacent pulmonary tissue is infiltrated.



Gangrenous abscess

The clinic of a **pulmonary gangrene** differs by a terminal expression of signs. The state of the patients is critical. The patient is adynamic, exhausted, with edemas on legs. Dyspnea in rest, hemodynamic disturbances are evident. Dirty-grey or brown sputum with detrites, pieces of necrotic parenchyma and threads of blood excretes out with the cough up to 1 l. Early pleural complications are usual and represent with pulmonary bleeding, which may be profuse. Often it is associated with vital organ dysfunction and loss of consciousness.

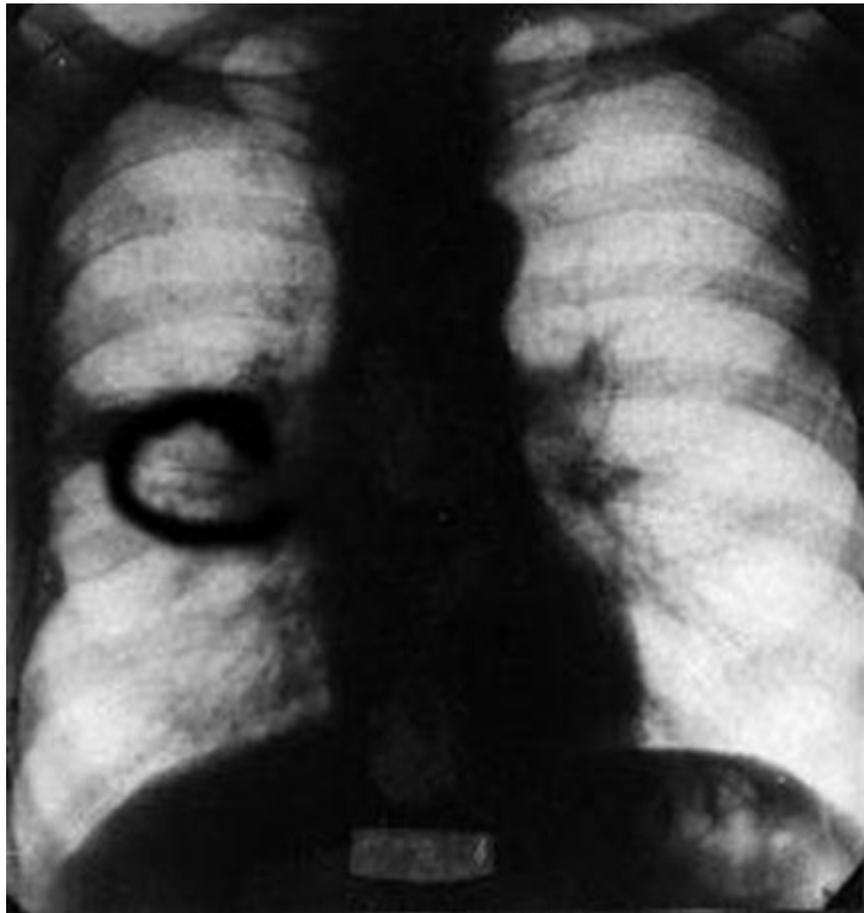
The intensive shadow which borrows a considerable area of lungs with a visible cavities, that contain sequesters, fluid levels, is roentgenologically revealed. The shadow outline is irregular, but could be well defined if the process is within interlobar sulcus.



Pulmonary gangrene

Chronic abscess of lungs occurs at 12-15 % of cases. It is considered to be chronic at existence of a pulmonary abscess more than 6-8 weeks. It is characterized by a cyclic course. In the stage of remission the patients complain of a moderate dyspnea, cough with expectoration of a mucous or mucopurulent discharge. The exacerbation manifests by coughing up of 250-500 ml of a purulent foul sputum, chest pain, dyspnea, hectic temperature with the difference in 1,5-2°C. Dizziness, suppression of appetite, general weakness enlarges according to intoxication. The skin is pale with moderate cyanosis. The respiratory rate rises to 28-30 per min. In 6-8 months is noticed the clubbing of the fingers and deformation of the chest. The vocal fremitus is a little bit weakened on the side of lesion (particularly in peripheral localization of the process). The percussion reveals a short sound in projection of pathological process, the auscultation - a lot of moist rales on the background of amphoric respiration.

Roentgenologically chronic abscess is shown by one or several cavities of a spherical shape with a thick, dense pyogenic sheath. Exacerbation of the process manifests by a cavity with horizontal air-fluid level. The size of surrounding perifocal infiltration depends on the phase of process.



Chronic abscess of lungs

The blood analysis in pulmonary destruction is characterized by leukocytosis with deviation of the differential count to the left, lymphocytopenia, elevation of the erythrocyte sedimentation rate. Gangrenous change of the process is accompanied by a progressing anemia, sometimes by leukopenia. The hypoproteinemia arises from major losses of protein with purulent sputum. Intoxication and toxic lesion of liver leads to disproteinemia. It is associated with the enlarged concentration of mucoprotein, sialine acids, seromuroid, and fibrinogen.

The immunogram reveals the suppression of cellular and humoral immunity with a liability to hyperergy and autoaggression, depression of the mechanisms of nonspecific protection.

The cytological bronchial water lavage is characterized by expressed neutrocytosis, noncellular postdestructive insertions at lack or absence of alveolar macrophages.

Variants of clinical course and complications

According to the clinical course, there are such variants of the development of purulent diseases of lungs:

1. **Favorable course.** The adequate treatment results in prompt positive clinical, roentgenological and laboratory dynamics, and terminates by recovery.
2. **Non-progressive course.** A poor drainage of the suppurative focus and permanent purulent intoxication result in transferring of the process in chronic form.
3. **Progressing course.** Is predetermined by combination of a series of the unfavorable factors (low resistance of the organism, autoimmune aggression, high virulence of the infecting agent etc.). Characterized by diffusion of the zone of necrosis and destruction with transferring in gangrene.
4. **Incapsulated process.** Caused by the absence or complete obstruction of the draining bronchus under condition of satisfactory resistance of the organism.
5. **Complicated course.** Mostly is the result of progressive development of the pathological process.

Pulmonary bleeding arise suddenly, are associated with coughing out of a foamy, red blood and clots by portions or continuous stream. The most often source of a pulmonary bleeding are the bronchial arteries and vessels of a pulmonary tissue. The clinical manifestations of a pulmonary suppuration are accompanied by dizziness, weakness, dyspnea, chest pain. The hemodynamic disturbances depend on intensity of the bleeding. The auscultation of lungs from both sides reveals the moist rales (aspiration). If the pulmonary destruction is present the plain film of the chest shows the localization of the source of bleeding. After hospitalization of the patient with this complication the exclusive information is obtained with a fibrobronchoscopy.



According to degree the pulmonary bleedings are classified (V.Struchkov, 1985):

I degree – hemorrhage up to 300 ml.

1. Single hemoptysis.
2. Multiple hemoptysis.

II degree – hemorrhage up to 700 ml.

1. Single bleeding:
 - a) with fall in arterial pressure and decreasing of hemoglobin;
 - b) without fall in arterial pressure and decreasing of hemoglobin.
2. Multiple bleeding:
 - a) with fall in arterial pressure and decreasing of hemoglobin;
 - b) without fall in arterial pressure and decreasing of hemoglobin.

III degree - hemorrhage exceeds 700 ml.

1. Massive bleeding.
2. Fulminant, lethal bleeding.

The I degree of a pulmonary bleeding manifests by coughing out the sputum tinged with blood, the hemodynamic disturbance usually absent. At bleeding of II degree are observed decreasing of arterial pressure on 20-30 mm Hg, tachycardia to 100 beats/min, contents of hemoglobin within 60-80 g/l. The bleeding of III degree are accompanied with sharp decreasing of arterial pressure, rapid (more than 100-120 beats/min), small, sometimes thread pulse, and even its disappearance on peripheral arteries, tachypnea to 40 per 1 min, hemoglobin to 50-60 g/l. Probable the fulminant course up to the terminal state with prompt failure of cardiac activity and asphyxia by blood.

Sepsis manifests by multisystem lesions with progression of the syndrome of polyorganous failure, hematosepsis, purulent metastasizing (frequently in brain).

Characteristic complications for suppurative diseases of lungs such as pleural empyema and pyopneumothorax are described in separate parts.

The diagnostic program

1. Complaints and history of the disease.

Generally, most of the patients admitted to the hospital with a diagnosis of lung abscess have had symptoms for at least 2 weeks. These patients typically have an intermittent febrile course, productive cough, weight loss, general malaise, and night sweats. Initially, foul sputum is not observed in the course of the infection; however, after cavitation occurs, putrid expectorations are quite prevalent. The odor of the breath and sputum of a patient with an anaerobic lung abscess is often quite pronounced and noxious and may provide a clue to the diagnosis. Hemoptysis may occasionally follow the expectoration of putrid sputum.

Symptoms depend on whether the abscess is caused by anaerobic or other bacterial infection.

- Anaerobic infection in lung abscess
 - Patients often present with indolent symptoms that evolve over a period of weeks to months.
 - The usual symptoms are fever, cough with sputum production, night sweats, anorexia, and weight loss.
 - The expectorated sputum characteristically is foul smelling and bad

tasting.

- Patients may develop hemoptysis or pleurisy
- Other pathogens in lung abscess
 - These patients generally present with conditions that are more emergent in nature and are usually treated while they have bacterial pneumonia.
 - Cavitation occurs subsequently as parenchymal necrosis ensues.
 - Abscesses from fungi, *Nocardia* species, and *Mycobacteria* species tend to have an indolent course and gradually progressive symptoms.

2. Physical findings.

The findings on physical examination of a patient with lung abscess are variable. Physical findings may be secondary to associated conditions such as underlying pneumonia or pleural effusion. The physical examination findings may also vary depending on the organisms involved, the severity and extent of the disease, and the patient's health status and comorbidities.

- Patients with lung abscesses may have low-grade fever in anaerobic infections and temperatures higher than 38.5°C in other infections.
- Generally, patients with in lung abscess have evidence of gingival disease.
- Clinical findings of concomitant consolidation may be present (eg, decreased breath sounds, dullness to percussion, bronchial breath sounds, coarse inspiratory crackles).
- The amphoric or cavernous breath sounds are only rarely elicited in modern practice.
- Evidence of pleural friction rub and signs of associated pleural effusion, empyema, and pyopneumothorax may be present. Signs include dullness to percussion, contralateral shift of the mediastinum, and absent breath sounds over the effusion.
- Digital clubbing may develop rapidly.

3. X-ray examination of chest in two planes (direct and lateral).

- A typical chest radiographic appearance of a lung abscess is an irregularly shaped cavity with an air-fluid level inside. Lung abscesses as a result of aspiration most frequently occur in the posterior segments of the upper lobes or the superior segments of the lower lobes.

- The wall thickness of a lung abscess progresses from thick to thin and from ill-defined to well-circumscribed as the surrounding lung infection resolves. The cavity wall can be smooth or ragged but is less commonly nodular, which raises the possibility of cavitating carcinoma.

- The extent of the air-fluid level within a lung abscess is often the same in posteroanterior or lateral views. The abscess may extend to the pleural surface, in which case it forms acute angles with the pleural surface.

- Anaerobic infection may be suggested by cavitation within a dense segmental consolidation in the dependent lung zones.

- Lung infection with a virulent organism results in more widespread tissue necrosis, which facilitates progression of underlying infection to pulmonary gangrene.

4. Up to one third of lung

5. Tomogram of lungs.

6. Computed tomography

- CT scanning of the lungs may help visualize the anatomy better than chest radiography. CT scanning is very useful in the identification of concomitant empyema or lung infarction.

- On CT scans, an abscess often is a rounded radiolucent lesion with a thick wall and ill-defined irregular margins.

- The vessels and bronchi are not displaced by the lesion, as they are by an empyema.

- The lung abscess is located within the parenchyma compared with loculated empyema, which may be difficult to distinguish on chest radiographs.

- The lesion forms acute angles with the pleural surface chest wall.

7. Examination of the sputum (bacteriological, cytological).

8. General blood and urine analyses.

- A complete white blood cell count with differential may reveal leukocytosis and a left shift.

- Obtain sputum for Gram stain, culture, and sensitivity.

- If tuberculosis is suspected, acid-fast bacilli stain and mycobacterial

culture is requested.

- Blood culture may be helpful in establishing the etiology.
- Obtain sputum for ova and parasite whenever a parasitic cause for lung abscess is suspected.

9. Biochemical blood analysis (protein and its fractions).

10. Immunogram.

11. Fibrobronchoscopy.

Differential diagnostics

Pneumonia

Primary bacterial pneumonias caused by single bacterial species other than the pneumococcus may account for up to 25% of community-acquired and 80% of hospital-acquired pneumonias. All of these pneumonias may have somewhat similar physical findings and x-ray evidence of pulmonary infiltration or consolidation. For proper treatment, it is crucial to identify the causative agent by blood culture and by sputum examination with stained smear and culture. Transtracheal aspiration, fiberoptic bronchoscopy, or even lung biopsy may be needed for specific diagnosis and treatment.

Streptococcal Pneumonia

Pneumonia due to hemolytic streptococci occurs usually as a sequela to viral infection of the respiratory tract, especially influenza or measles, or in persons with underlying pulmonary disease. The patients are usually in a severely toxic condition and cyanotic. Pleural effusion develops frequently and early and progresses to empyema in one-third of untreated patients. The diagnosis rests on finding large numbers of streptococci in smears of sputum and culturing hemolytic streptococci from blood and sputum.

The treatment of choice is with penicillin G in a dosage similar to that for pneumococcal pneumonia (see above). If treatment is started early, the prognosis is good.

Staphylococcal Pneumonia (picture 2)

Pneumonia caused by *Staphylococcus aureus* occurs as a sequela to viral infections of the respiratory tract (eg, influenza) and in debilitated (eg, postsurgical) patients or hospitalized infants, especially after antimicrobial drug administration. There is often a history of a mild illness with headache, cough, and generalized aches that abruptly changes to a very severe illness with high fever, chills, and exaggerated cough with purulent or blood-streaked sputum and deep cyanosis. There may be early signs of pleural effusion, empyema, or tension pneumothorax. X-ray examination reveals lung consolidation, pneumatoceles,

abscesses, empyema, and pneumothorax. The demonstration of pyopneumothorax and of cavities with air-fluid levels by x-ray is highly suggestive of Staphylococcal pneumonia. The diagnosis must be confirmed by stained smear of sputum (masses of white cells and gram-positive cocci, many intra-cellular) and culture (predominantly *S aureus*), and

also by means of cultures of pleural fluid and blood. The white count is usually more than 20,000//zL.

Initial therapy (based on sputum smear) consists of nafcillin, 6-12 g/d, or vancomycin, 2 g/d, given intravenously in divided doses as a bolus. If the staphylococcus proves to be penicillin-sensitive by laboratory test, penicillin G, 20-60 million units/d intravenously, is the antibiotic of choice. Drugs should be continued for several weeks. If empyema develops, drainage must be established. The prognosis varies with the underlying condition of the patient and the drug susceptibility of the organism.

Legionella Pneumonia

The eponym legionnaires' disease has been given to a serious pneumonia that afflicted people attending the American Legion Convention in Philadelphia in 1976. Other outbreaks have been diagnosed respectively at least since 1965, and sporadic infections have occurred at least since 1947 in many places.

Legionella pneumophila is a poorly staining gram-negative bacterium that grows slowly on special media (eg, charcoal-yeast extract) at 35 °C. There are at least 8 species of *Legionella*, some with multiple serotypes. These organisms can be recovered in human disease from sputum, bronchial washings, pleural fluid, lung biopsies, or blood. *Legionella* species occur in the environment and are acquired by humans from aerosols, dust from air-conditioning systems, water, or soil. The infection is not usually communicable from patient to contacts. Asymptomatic infection is common at all ages, whereas symptomatic infection is most often an opportunistic pneumonia in immunocompromised individuals.

Asymptomatic infection is evident only by a rise in specific antibodies. Symptomatic infection is observed mainly in elderly persons, smokers, and patients undergoing hemodialysis or renal transplant.

The incubation period is estimated to be 2-10 days. Initial symptoms are malaise, diffuse myalgias, and headache, followed in 12-48 hours by high, non-remittent fever and chills. Nausea, vomiting, and diarrhea are frequent early in the illness. On the third day a dry cough begins that is nonproductive or produces scanty mucoid, sometimes blood-streaked sputum. Dyspnea and hypoxia become marked as signs of consolidation develop. Pleuritic chest pain occurs in one-third of patients. Severe confusion or delirium may occur.

There is leukocytosis with a shift to the left, hyponatremia, abnormal liver function tests, and, occasionally, microscopic hematuria. Chest x-rays reveal patchy, often multilobar pulmonary consolidation, and, occasionally, small pleural effusions. The illness usually worsens for 4-7 days before improvement begins in those who recover. During severe outbreaks, the mortality rate has been 10% in those with manifest disease. Death is attributed to respiratory or renal failure or shock, with disseminated intravascular coagulation.

The diagnosis is based on a clinical picture compatible with the specific features of the disease and on

negative results of bacteriologic laboratory tests for other pneumonias. The organism can be identified by immunofluorescence in cultures, lung biopsy, and, rarely, sputum specimens. A retrospective diagnosis is based on a significant rise in specific serum antibodies detected by immunofluorescence.

The treatment of choice is erythromycin, 0.5-1 g every 6 hours intravenously or orally for 2-3 weeks. This usually results in improvement in 2-3 days. Rifampin, 10-20 mg/kg/d, has been suggested for patients who fail to respond to erythromycin. Assisted ventilation and management of shock are essential.

Pneumocystis carinii Pneumonia

This parasitic infection occurs in debilitated children or immunodeficient adults. It has been a prominent opportunistic infection in AIDS patients. The diagnosis is made by lung biopsy and the demonstration of typical cysts of *P. carinii* in impression smears of lung tissue stained with methenamine-silver. Early treatment with sulfamethoxazole-trimethoprim can cure the pneumonia. The same drug has been effective in prophylaxis during immunosuppression. An alternative, more toxic drug is pentamidine isethionate.

"MIXED" BACTERIAL PNEUMONIAS (Hypostatic Pneumonia, "Terminal" Pneumonia, Bronchopneumonia)

Essentials of Diagnosis

- Variable onset of fever, cough, dyspnea, expectoration.
- Symptoms and signs often masked by primary (debilitating) disease.
- Greenish-yellow sputum (purulent) with mixed flora.
- Leukocytosis (often absent in aged and debilitated patients).
- Patchy infiltration on chest x-ray.

General Considerations

Mixed bacterial pneumonias include those in which culture and smear reveal several organisms, not one of which can clearly be identified as the causative agent. These pneumonias usually appear as complications of anesthesia, surgery, aspiration, trauma, or various chronic illnesses (cardiac failure, advanced carcinoma, uremia). They are common complications of chronic pulmonary diseases such as bronchiectasis and emphysema. Old people are most commonly affected ("terminal" pneumonia). Patients treated with intermittent positive pressure breathing apparatus or immunosuppressive drugs may develop pneumonia caused by gram-negative rods.

The following findings in a debilitated, chronically ill, or aged person suggest a complicating pneumonia: (1) worsening of cough, dyspnea, cyanosis; (2) low-grade, irregular fever; (3) purulent sputum; and (4) patchy basal densities on a chest film (in addition to previously noted densities caused by a primary underlying

disease, if any), sometimes with local necrosis and cavitation.

Clinical Findings

A. Symptoms and Signs: The onset is usually insidious, with low-grade fever, cough, expectoration, and dyspnea that may become marked and lead to cyanosis. Physical findings are extremely variable and may not be impressive against a background of cardiac or pulmonary disease. The signs listed under Other Bacterial Pneumonias may also be present.

B. Laboratory Findings: The appearance of a greenish or yellowish (purulent) sputum should suggest a complicating pneumonia. Smears and cultures reveal a mixed flora, often including anaerobes. Predominant types should be noted. Leukocytosis is often absent in the aged and debilitated patient presenting with a mixed infection.

C. X-Ray Findings: X-ray (Picture 3) shows patchy, irregular infiltrations, most commonly posterior and basal (in bedridden patients). Abscess formation may be observed. Careful interpretation will avoid confusion with shadows due to preexisting heart or lung disease.

Differential Diagnosis

Mixed bacterial pneumonias must be differentiated from tuberculosis, carcinoma, and other specific mycotic, bacterial, and viral pulmonary infections (to which they may be secondary).

Treatment

Clear the airway and correct hypoxia. Unless a probably significant etiologic agent can be identified, give one of the new cephalosporins (eg, cefotaxime, 12 g/d intravenously) as initial therapy. This will be modified according to clinical and laboratory results.

Prognosis

The prognosis depends upon the nature and severity of the underlying pulmonary disease and varies with the predominating organism.

ASPIRATION PNEUMONIA

Aspiration pneumonia is an especially severe type of pneumonia, often with a high mortality rate. It results from the aspiration of gastric contents in addition to aspiration of upper respiratory flora in secretions. Important predisposing factors include impairment of the swallowing mechanism (eg, esophageal disease), inadequate cough reflex (eg, anesthesia, postoperative state, central nervous system disease, drug abuse), and impaired gastric emptying (eg, pyloric obstruction). Pulmonary injury is due in large part to the low pH (< 2.5) of gastric secretions.

Scattered areas of pulmonary edema and bronchospasm occur, and the x-ray appearance (pictures 4-5) may

be confused with that of pulmonary emboli, atelectasis, bronchopneumonia, and congestive heart failure.

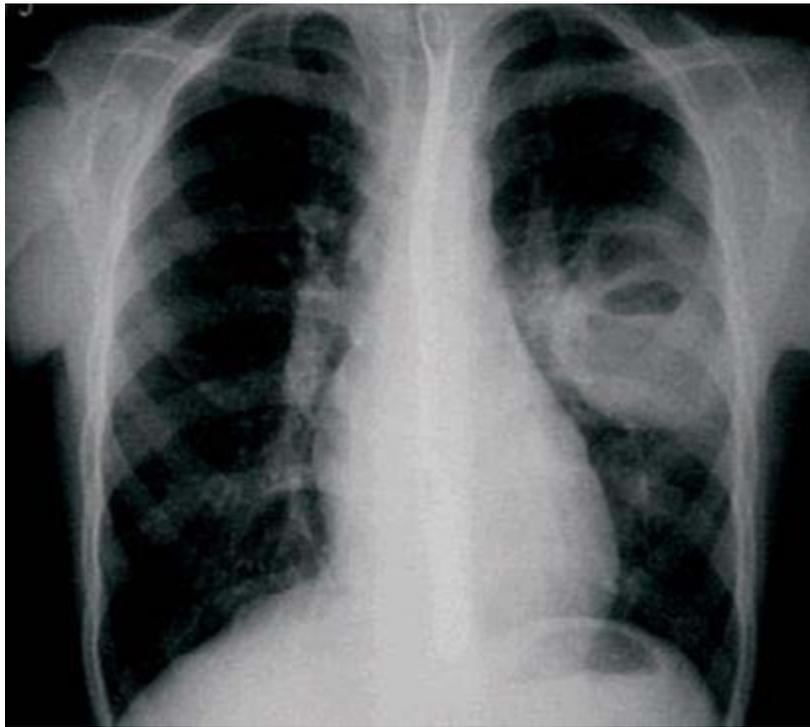
Removal of aspirated material by catheter suction or bronchoscopy may be attempted, but this usually fails to remove all aspirate completely. Corticosteroids (eg, prednisone, 100 mg orally on the first or second day) may reduce the intensity of the inflammatory reaction to acidic gastric secretion, but the value of corticosteroids in the treatment of aspiration pneumonia is not proved, and they increase the risk of superinfection. Some aspiration pneumonias have no bacterial component, but in many others a mixed bacterial flora is involved. Antimicrobial drugs directed against the latter (eg, penicillin G plus an aminoglycoside or the best available cephalosporin) are sometimes administered without waiting for evidence of progressive pulmonary infection. In doing so, however, there is a risk of favoring the development of resistant microorganisms. Therefore, administration of antimicrobials should not continue without laboratory and clinical evidence of microbial infection. Assisted ventilation and supplementary oxygen are beneficial.

The cancer of the central location due to the obturation of bronchus results in atelectasis of a segment or lobe of lungs, with probable further abscessing. For the differentiation used tomography (reveals the obturation of bronchus by tumour, lesion of central lymph nodes), cytological examination of sputum and bronchial outwashes. The determinant role belongs to fibrobronchoscopy with a biopsy and verification of the diagnosis.



Central lung carcinoma

The peripheral cancer of lungs with destruction on tomograms is characterized by cavity with irregular inner surface, which external outline connects with root of the lung because of lymphatic metastatic spreading. The central lymph nodes frequently enlarged. The diagnosis is improved by results of transthoracic puncture or catheterizing biopsy with cytological investigation, fibrobronchoscopy. If it is impossible to confirm the diagnosis the thoracotomy is indicated.



The peripheral cancer of lungs

The tubercular cavern is mainly located in the upper lobes of lungs, roentgenologically revealed on the background of characteristic changes in adjacent pulmonary tissue (calcification, dissemination), sometimes detected a draining bronchus. In the sputum mycobacteria of tuberculosis are frequently found.



The tubercular cavern

The suppurative cyst of lungs differs by a gradual onset, slow course of the suppuration, less expressed intoxication. Roentgenologically its cavity has the oval or rounded shape with a thin sheath and regular contour. Perifocal infiltration is not characteristic.

Tactics and choice of treatment

The tactics in acute pulmonary destruction should be mainly conservative.

1. The adequate antibacterial, antiinflammatory therapy consists of intravenous introduction of antibiotics of a wide spectrum activity.

ANTIBIOTICS IN LUNG ABSCESS

Anaerobic organisms[3]

- First choice - Clindamycin (Cleocin 3)
- Alternative - Penicillin
- Oral therapy - Clindamycin, metronidazole (Flagyl), amoxicillin (Amoxil)

Gram-negative organisms

- First choices - Cephalosporins, aminoglycosides, quinolones
- Alternatives - Penicillins and cephalexin (Biocef)
- Oral therapy - Trimethoprim/sulfamethoxazole (Septra)

Pseudomonal organisms: First choices include aminoglycosides, quinolones, and cephalosporin.

- Gram-positive organisms
- First choices - Oxacillin (Bactocill), clindamycin, cephalexin, nafcillin (Nafcil), and amoxicillin
- Alternatives - Cefuroxime (Ceftin) and clindamycin
- Oral therapy - Vancomycin (Lyphocin)

Nocardial organisms: First choices include trimethoprim/sulfamethoxazole and tetracycline (Sumycin).

With the purpose of maximal concentration of drugs in the pathological focus applied:

- Injection of antibiotics in the vessels of a pulmonary circulation by means of catheterization of central veins, pulmonary artery;
- Introduction of medicinal agents into respiratory tracts (in the second stage) – through the endotracheal microirrigator, nasogastric tube, during bronchoscopies, endoscopic catheterization of the abscess cavity through the draining bronchus, in aerosolic inhalations. The composition of medical admixtures includes: antibiotics, antiseptics (10 % dimexid, dioxydin, microcid etc.), enzymes;

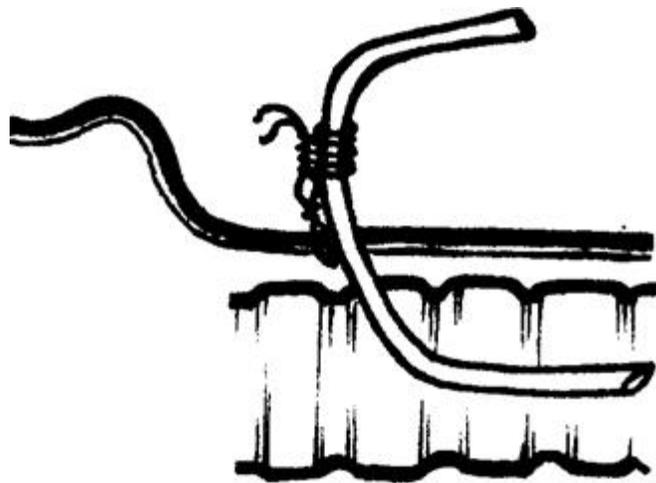
The steps of microtraheostomia:



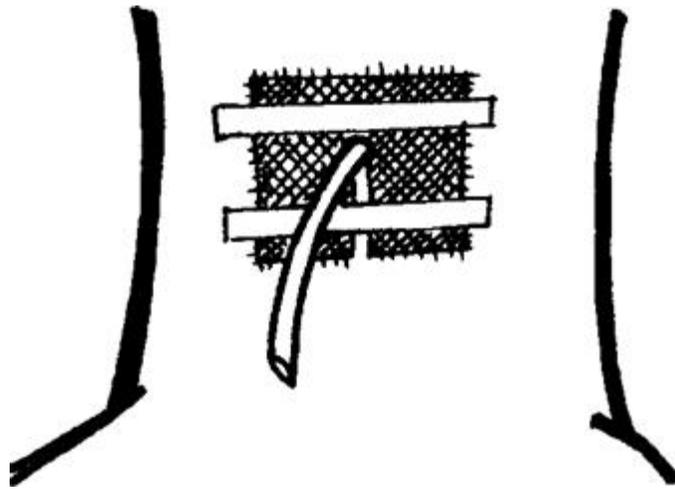
Tracheocentesis after local anesthesia



Insertion of the conductor



Incertion of the irrigator along the conductor



Skin wound

- Transcutaneous in the focus of destruction by means of puncture or draining with the usage of physical antiseptics (US, UVR, laser).
- Intrapleural;
- By means of electrophoresis.

2. Evacuation of purulent content of the cavities:

- In natural way by an active sanitation of tracheobronchial tree using repeated fibrobronchoscopies, aspirations through the endobronchial catheter, installations of medical agents through the microtracheostomy, aerosolic inhalations;
- Transthoracically by means of repeated punctures or external draining of peripheral cavities.

3. Detoxycation therapy (intra- and extracorporal).

4. Immune correction (under the control of immunogram):

- Active – staphylococcal anatoxin according to the plan;
- Passive – specific gamma-globulins, hyperimmune plasma;
- Non-specific (pirimidine and purine derivates, drugs of a thymus gland, splenin, levamisol,).

5. Homeostatic correction (oxygenotherapy, correction of anemia, hypoproteinemia, acidosis, microcirculatory disturbance).

6. Desensitizing, antiinflammatory therapy, regulation of activity of proteases: antihistamine, nonsteroid antiinflammatory agents, inhibitors of proteases, antioxidants.

7. Correction of dysfunction of the vital organs and systems, prevention of complications, symptomatic therapy.

Indications for operative management in acute destructive processes of lungs:

- Pulmonary bleeding of II- III degree;
- Progression of the process on the background of active and appropriate therapy;
- Tense pyopneumothorax, which is failed to liquidate by the draining of pleural space;
- Impossibility to rule out the suspicion on a malignant tumour.

Contraindications: decompensation of the vital functions and systems in the terminal stage, bilateral purulent destruction of lungs, concomitant incurable malignant tumours.

Several important factors must be considered prior to undertaking surgery. Because of the high risk of spillage of the abscess into the contralateral lung, it is almost essential that a double-lumen tube be used to protect the airway. If this is not available, surgery poses a very high risk of abscess in the other lung and a risk of ARDS. In such cases, postponing the surgery is a wise decision. Another, less-satisfactory method to deal with this problem includes positioning the patient in the prone position. The surgeon must be skilled in resecting the abscess and in rapid clamping of the bronchus to prevent spillage into the trachea. These factors are extremely important when dealing with the surgical aspects of treating a lung abscess. If doubt persists, postponing the surgery is best.

Surgical treatment is now rarely necessary and is almost never the initial choice in the treatment of lung abscesses. In current practice, fewer than 15% of patients need surgical intervention for the unchecked disease and for complications that occur in both the acute and chronic stages of the disease.

Surgical management is reserved for specific indications such as little or no response to medical treatment, inability to eliminate a carcinoma as a cause, critical hemoptysis, and complications of lung abscess (eg, empyema, bronchopleural fistula). In addition, if after 4-6 weeks of medical treatment a notable residual cavity remains and the patient is symptomatic, surgical resection is advocated.

The results of surgery are difficult to assess because of the varying patient population and the tremendous increase in illicit drug abuse, alcoholism, AIDS, and infections by

gram-negative and opportunistic organisms. These factors have increased the incidence of lung abscess and the associated morbidity.

A great deal of caution is needed during anesthesia when patients with lung abscess undergo surgery because spillage of the abscess material into the uninvolved lung can occur. Therefore, a double-lumen endotracheal tube is used in all cases.

A study by Nagasawa et al has shown that thoracoscopic surgery can lead to effective drainage of pediatric lung abscess without major complications. In addition, other benefits of thoracoscopy include rapid recovery, less pain, and minimal morbidity.

Operational incisions – anterolateral, lateral and posterolateral thoracotomy. The operation suggests segmental, polysegmental resection, lobectomy, bilobectomy combined intervention (with the decortication, pleurectomy).



Lateral access



Anterior access



Posterior access

The patients with chronic abscesses should be undergone the operative treatment after complete liquidation of exacerbation.

In a pulmonary gangrene the stabilization of the process on the background of active conservative treatment allows in future to apply conservative tactics up to recovery or making optimal conditions for operation (liquidation of intoxication, aggressive panbronchitis, diffuse infiltration of the parenchyma, pleural complications). The headlong progression of the gangrene during first days, despite the active correction,

occurrence of pulmonary bleeding requires urgent performance of operative management. Volume of the operation – pneumonectomy, bilobectomy, lobectomy.

PLEURAL EMPYEMA

The pleural empyema is a purulent inflammation of its visceral and parietal membranes, which is associated with accumulation of pus in a pleural space.

Etiology and pathogenesis

The causes of occurrence of acute pleural empyema are inflammatory, or purulent and destructive processes of lungs, abscesses of abdominal cavity (secondary pleural empyema), open and closed damages of chest, and also, in some cases, operative approaches on thoracic organs (primary pleural empyema).

A secondary pleural empyema occurs in 88 % of the patients. Thus develops fibrinous, exsudative, and then purulent pleurisy.

In case of pulmonary gangrene, purulent mediastinitis, subphrenic abscess the stage of exsudative pleurisy extremely short. The progression of the process results in transferring of focal pleural empyema into wide-spread.

Pathology

Macroscopically pleura is thickened, covered by pus with punctate hemorrhages. Microscopically it is diffusely oozed with neutrophils. In cases, when the empyema overcome into chronic course, the pleura deposits the calcareous salts, thick pus encapsulated, sometimes with the development of fistula.

Classification

I. According to the etiological factor:

1. Specific.
2. Nonspecific.

II. According to the pathogenic factor:

1. Primary.
2. Secondary.

III. According to the clinical course:

1. Acute.
2. Chronic.

IV. According to extension of the process:

1. Focal.
2. Wide-spread.

V. According to the presence of lung destruction:

1. Empyema with destruction of pulmonary tissue.
2. Empyema without destruction of pulmonary tissue.
3. Pyopneumothorax.

VI. According to communication with environment:

1. Closed pleural empyema;
2. Open pleural empyema:
 - bronchopleural fistula;
 - thoracopleural fistula;
 - thoracopleurobronchial fistula;
 - cribrate lung.

Symptomatology and clinical course

The clinic of an acute pleural empyema depends on extension of the process, reactivity of organism and presence of complications.

The pain is the sign, which denote the involvement of pleural membranes in the process. Its intensity increases depending on depth of respiration and body position.

The dyspnea arises from accumulation of a purulent content in a pleural space and exception of particular volume of a pulmonary tissue from respiration. It's in direct ratio to amount of exudation in a pleural space.

The cough is manifestation of inflammation or purulent and destructive process in a pulmonary tissue.

Fever to 39-40°C, headache, sleeplessness, general malaise, and anorexia – all these are manifestations of intoxication.

The forced patient's position and restriction of breathing should be considered as outcomes of a pain syndrome. The extension of pleural empyema causes the swelling of thoracic wall, smoothing of intercostal spaces.

By palpation – diminished vocal fremitus on the part of lesion.

The data of percussion and auscultation depend on extension of the process and amount of pus in a pleural space. At percussion over the exudate it is possible to reveal short sound with oblique upper contour. Above the exudate – tympanic sound resulting from consolidation of pulmonary tissue. By auscultation – diminished or absent sound in a great amount of exudate.

The predominant roentgenological sign of a focal or wide-spread empyema – the presence of exudate. In localized acute pleural empyema observed a local intensive homogeneous shadow. Roentgenologically according to localization distinguished such types of a focal empyema:

- 1) apical;
- 2) paramediastinal;
- 3) parietal;
- 4) interlobar;
- 5) epiphrenic.

The wide-spread pleural empyema manifests by intensive homogeneous shadow in a basal parts with oblique upper contour (Damuaso' line). The diaphragmatic dome is failed to observe. The more pus contents in a pleural space, the higher the upper measure of exudate.



Left-side pleural empyema



Pleural empyema



Pleural empyema



Pleural empyema



Pleural empyema

Variants of clinical course and complications

The clinic of a focal pleural empyema depends on the site of the process. The apical empyema, due to involvement in the process of a vascular-nervous fascicle, manifests by intensive pain. The soft tissues of supraclavicular region are swelled. The percussion and auscultation has no information.

The pain syndrome in parietal (paracostal) empyema is more expressed. Thoracic excursion is restricted. The diminishing of respiratory sound can be obtained over the exudate.

The chief complaint in paramediastinal empyema is the heart pain. The location of the process in the upper mediastinum can cause the superior vena cava syndrome. The physical findings are vague.

In case of the basal (epiphrenic) empyema the patients complain of pain in subcostal area, which increases at respiration and irradiates in supraclavicular region. In some cases the pain irradiates in epigastric region. The palpation of intercostal spaces and hypochondrium is painful.

The clinical course of postoperative empyema depends on the character of operative approach (marginal resection of lung, lobectomy, pneumonectomy, operation on esophagus) and infection of the pleural space.

The clinical manifestations of posttraumatic empyema depend on the size of damage of the chest, lungs, mediastinal organs and complications (suppuration, hemothorax).

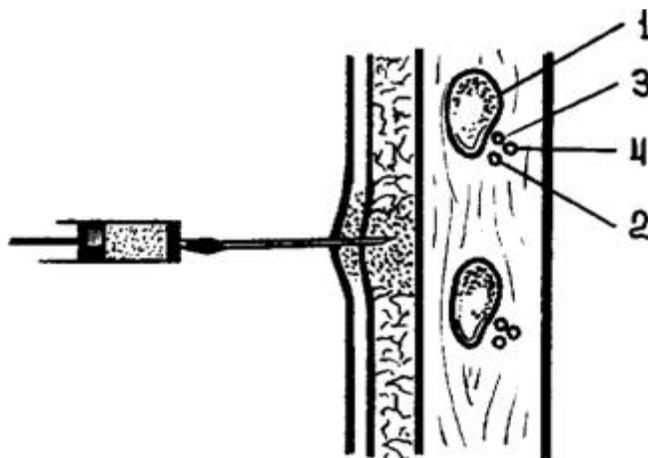
The involvement in the purulent process of a pulmonary tissue in acute empyema results in its fusion of membranes with formation of a bronchial or thoracopleural fistula (discharge of abscess through thoracic wall).

The inappropriate elimination of empyema results in chronic course, cribrate lung and pleurogenic cirrhosis of lungs.

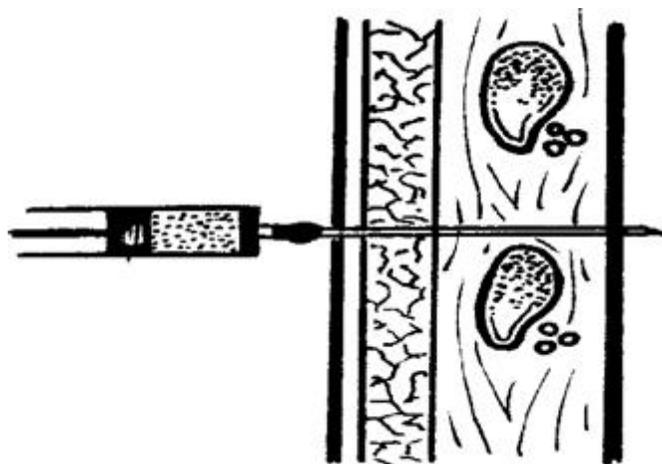
The diagnostic program

1. Complaints and history of the disease.
2. Physical findings.
3. Data of chest X-ray (in two planes, if necessary – laterography).
4. Pleural puncture.
5. The microbiological investigation of the exudate for its sensitivity to antibiotics.
6. General blood and urine analyses.
7. Biochemical blood analysis.
8. Pleurography (in transferring of the process into chronic form).

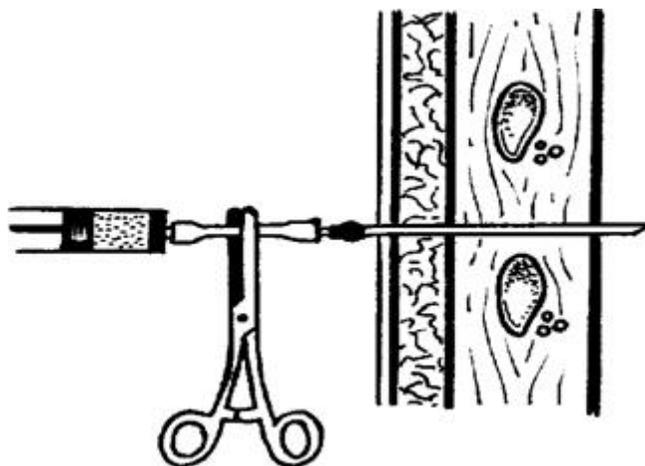
Pleural puncture:



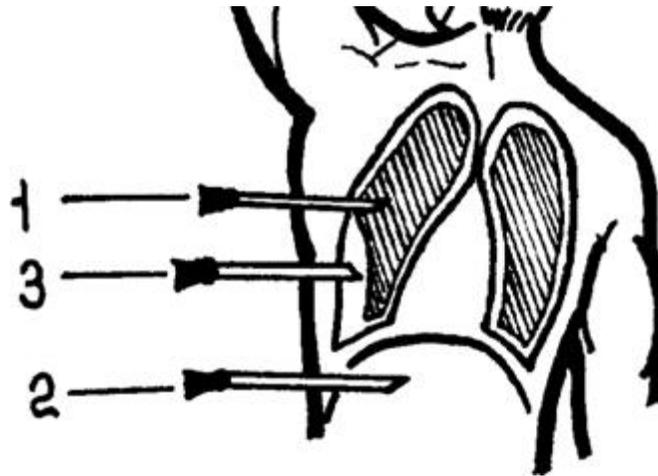
Local anesthesia



Thoracentesis



Aspiration of the fluid



Localization of the needle:

- 1 the needle damaged the lung
- 2 the needle damaged the diaphragm
- 3 the needle located in pleural sinus

Differential diagnostics

Pleuropneumonia complicated with exsudative pleurisy, in some cases resembles acute pleural empyema: a chest pain, fever, dyspnea, cough, and general weakness. The chest roentgenogram reveals hydrothorax (exsudative pleurisy, pleural empyema, hemothorax). The chief diagnostic method for differentiation is the thoracentesis. The presence of a serous (lucent, faint-yellow) exudate testifies about the pleuropneumonia, complicated with pleural effusion, and cloudy, foul-smelling exudate of white or greenish color – about acute empyema.

The major difficulties in differential diagnostics cause the limited forms of empyema.

The **Pancoast cancer** clinically and roentgenologically in most cases has almost the similar course to apical form of empyema. The transthoracic biopsy permits to confirm the diagnosis.

The **acute cholecystitis** is necessary to differentiate with the epiphrenic empyema. The pain in the right hypochondrium, fever, phrenic symptom are common for both diseases. However the objective findings, X-radiography of chest and the thoracentesis allow to differentiate these pathological processes.

The **tumour of anterior mediastinum** complicated by superior vena cava syndrome is necessary to differentiate with paramediastinal empyema. Nevertheless the body temperature in such patients, as a rule, normal. The upper cavography is possible to find out the shift of cava vein and its irregular contours (filling defect) due to growth of the mediastinal tumour.

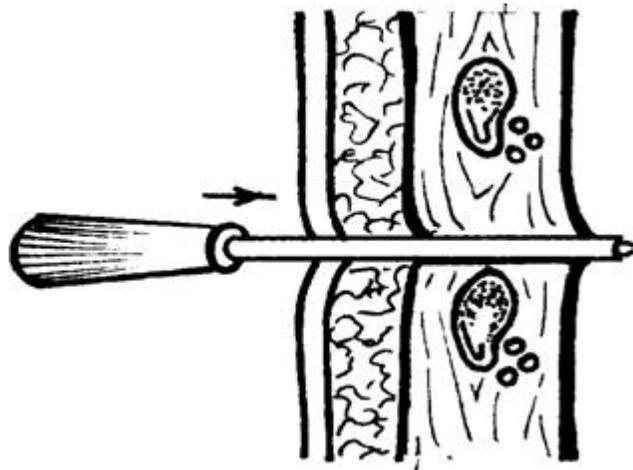
There are some difficulties in differential diagnostics of empyema with a **posttraumatic diaphragmatic hernia**. Such X-ray findings as deformation of diaphragm, additional shadows with a liquid level, intestinal loop suggest a diaphragmatic hernia. A laterography and contrast study of gastrointestinal tract is basic in differential diagnostics of this disease.

The atelectasis of a segment, and lobe of lung in some cases can cause misdiagnostics. Except X-ray chest examination (in two planes and tomography), these situations require necessity of diagnostic bronchoscopy, which reveals the cause of bronchial obturation (foreign body, endobronchial cancer etc.).

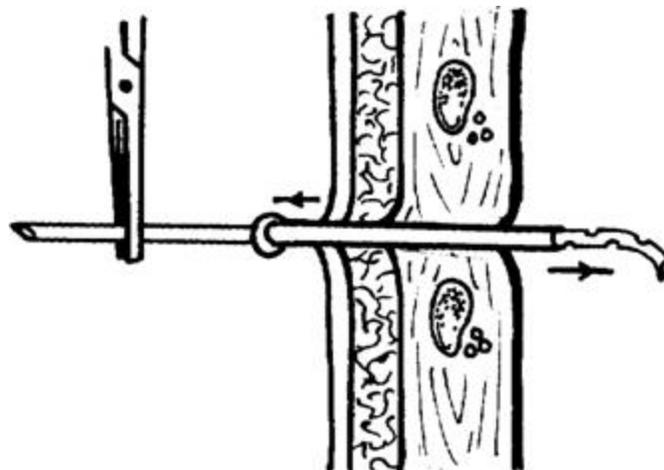
Tactics and choice of treatment

The presence of pus in a pleural space is the indication for its elimination. In the place of performed diagnostic thoracentesis carried out the draining of empyema's cavity, its sanation by means of antiseptic solutions. In a focal empyema the aspiration of pus is performed by thoracentesis and only in its inefficiency carried out a draining of pleural space.

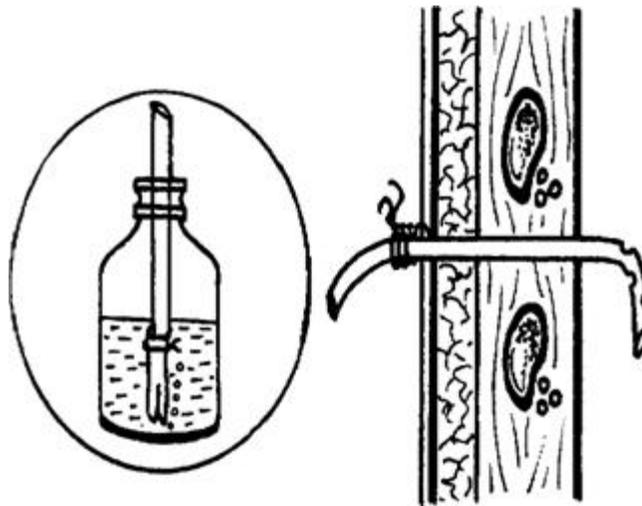
Pleural drainage:



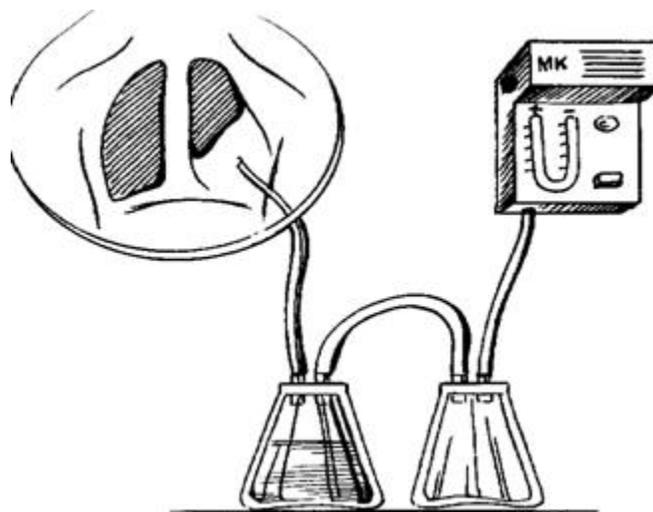
Trocar insertion



Incertion of drainage tube



Passive drainage by Bulau



Active aspiration

Intensive antibacterial and antiinflammatory therapy should be immediately instituted. For general improving used detoxication therapy (infusion of saline solutions, hemotransfusion, transfusion of proteins, solutions of dextran, haemodes, forced diuresis, hemosorption if necessary), therapy for rising up of immunological resistance of the organism.

During the empyema's sanitation decreases the amount of pus which discharges out through the drainage. The optimal variant of such course is the liquidation of empyema's cavity, then the drainage must be removed.

Transferring of the process into the chronic form (10-12 weeks) results in formation of a residual empyema's cavity, which is possible is to reveal by means of pleurography – introduction through the drainage of water-soluble contrast with the further X-radiography in 2 planes.

Operative approach is applied when the process has transferred into the chronic form, that is in case of residual empyema's cavity. Volume of the operation – pleurectomy, decortication of lung.

In some cases, when a bronchial fistula and great empyema's cavity has been formed, there is the necessity of performance of resection of lung and corrective thoracoplasty.

LUNG CYSTS

The cysts of lungs are the thin-walled cavitory formations, filled with air or liquid contents.

Inherent and acquired cyst are distinguished.

Etiology and pathogenesis

Inherent cysts arise from the abnormalities of the development of lungs under influence of the multiple chemical, physical, biological factors. Whether they can develop from a bronchial tree (bronchial) or from alveolar tissue (alveolar). Their occurrence resulting from the delay of the development of peripheral parts of bronchus with their expansion or agenesis of alveoli with dilatation of terminal bronchioles. Congenital cysts at first develop and grow as secretory formations. After communication with bronchus they finally form as air or hydroair cavities.

The acquired cysts represent fibrous cavities, which remain after abscesses, tubercular caverns, echinococci, posttraumatic intrapulmonary hematomas. The degenerative changes in the wall of bronchus with obliteration of its lumen by a cloggy secret owing to repeated inflammatory processes result in occurrence of acquired retentional cysts.

Pathology

Congenital cyst can be located in any part of lungs. The walls of bronchogenic cavities contain chaotically disposed elements of bronchus (cartilagous plates, muscle fibers, mucous glands), they are lined from inside by cylindrical or cubic epithelium. Squeezed alveolar cells line the walls of alveolar formations.

The acquired cyst are revealed in places of localization of previous diseases,. Their walls mainly consist of connective tissue. Epithelization is possible due to transferring of the epithelium from a draining bronchus at long existence.

The cysts could be uni- or multichamber, closed and open (depending on the presence of communication with bronchus).

Classification

According to parentage:

- congenital;
- acquired.

According to displacement:

- single, multiple;
- unilateral, bilateral.

Complications:

- Suppuration;
- Appearance of the valvular mechanism;
- Bleeding;
- Discharge into a pleural space (pneumothorax, pyopneumothorax, pleural empyema);
- Malignancy.

Symptomatology and clinical course

Clinical manifestations of uncomplicated pulmonary cysts are vague. Sometimes patients complain of a chest pain, periodic cough, and inflammatory diseases of respiratory tract in history. In children the signs are much expressed, the dyspnea associates with the compression of airways.

In great, superficially disposed cysts revealed delayed respiratory movements on affected side during breathing.

By palpation - weakened vocal fremitus.

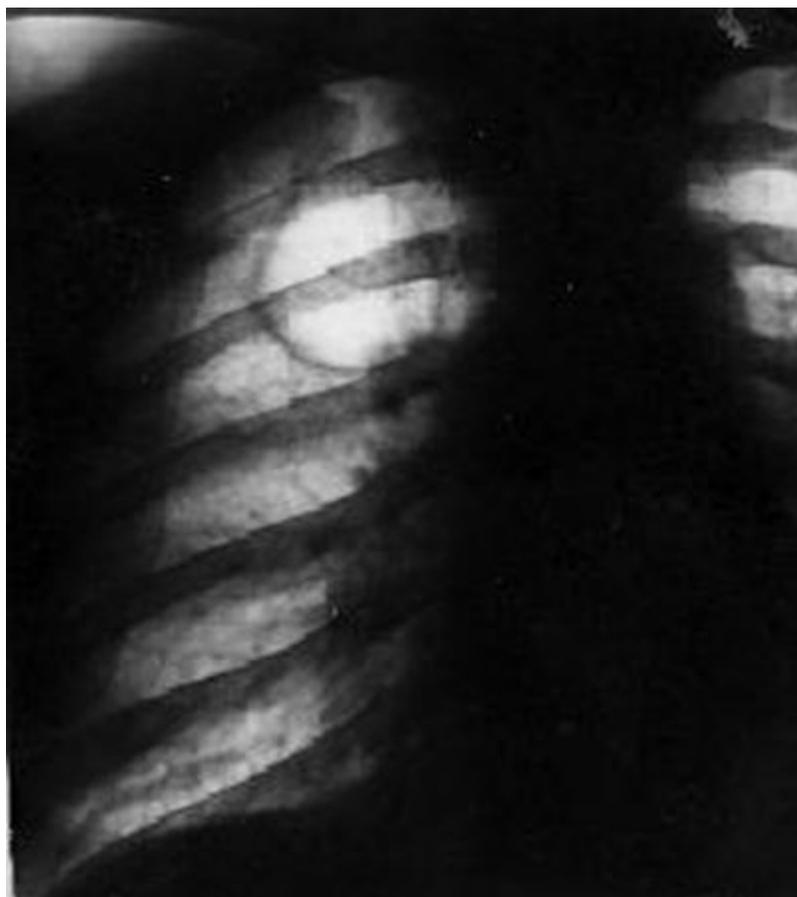
By percussion - short or bandbox sound, depending on contents.

By auscultation - weakened respiration, over the huge open cavities – amphoric.

At routine chest films the closed congenital cyst forms a homogeneous shadow of the spherical or oval shape of average intensity with rather regular edge on the background of an intact pulmonary tissue. During roentgenoscopy sometimes observed the change of its shape depending on the phase of breathing – elongation in inspiration (Escudero's syndrome). The character of the shadow is better to study on tomograms.

Acquired retentional cyst is roentgenologically shown by a shadow of irregular shape (piriform, spindle-shaped etc.), that displays the shape of the distended bronchus. The adjacent tissue, as a rule, changed due to

bronchiectases, pneumosclerosis, etc.



Cyst of the right lung

Open cysts are observed as thin-walled, good defined cavities of annular or oval shape.

On bronchograms congenital cysts are observed hypoplasia of segmental or lobar bronchus, lack of bronchial bifurcations, which tends to bone. Sometimes observed bronchiectases in the neighboring parts of lungs. A cystic cavity is infrequently defined.

The angiopulmonography reveals deformation of vascular branches, which circumflex the cyst.

Variants of clinical course and complications

The **suppuration** of the closed cysts resembles the development of a lung abscess with lesser expression of

signs of intoxication (protective role of epithelial wall).

Infection of the open cysts is characterized by a gradual long course, moderate manifestation of suppuration and late intoxication.

The tension (valvular) cysts arise more often in children. They are characterized by severe respiratory and hemodynamic disturbances, up to terminal, owing to inflexion of venous trunks, shift of mediastinum and compression of lungs. The predominant signs are increased dyspnea, cyanosis, chest pain, respiratory lag on affected side, auscultatory – absence or weakening of respiration, bandbox percussion sound over the cavity at mediastinal shift to the opposite side of the chest.

Pleural complications of pulmonary cysts, and also **bleeding** described in the relevant parts.

The malignant degeneration of pulmonary cysts occurs very rarely.

The diagnostic program

1. Complaints and history of the disease.
2. Physical methods of examination.
3. Routine chest film in two planes.
4. Lung tomography.
5. Bronchography.
6. Angiopulmonography.
7. General blood and urine analyses.

Differential diagnostics

The abscess of lungs, in contrast with a suppurative cyst, is characterized by prompt course, expressed purulent intoxication, roentgenologically its wall is irregular, with proper considerable perifocal infiltration.

The cancer with destruction differs by a thick wall with a tuberos inner surface, phenomena of lymphangitis in adjacent tissue. The determinant value has endoscopic examination or puncture with a biopsy and following morphological investigation.

The tubercular cavern displaced in the upper lobes, in adjacent tissue revealed fibrosis, petrifications,

dissemination, peribronchial lymphadenitis. In the sputum – mycobacterium of tuberculosis.

Opposite to cyst, in termed diseases, the circumflex deformation of vascular branches never observed at angiopulmonography.

The considerable difficulties can arise at differentiation of the closed congenital cysts.

The **tuberculoma** differs from them by characteristic localization. At X-ray examination the heterogeneity of the shadow with calcifications, early excentric destruction, "tubercular" background is observed.

The **benign tumours** sometimes possible to differentiate only after the results of cytological investigation of punctate or after operation.

The **echinococci cysts** on tomogram can have a double contour of chitinous and fibrous sheaths. In blood analyses observed eosinophilia. The final decision is taken out after carrying out immunological (indirect microagglutination test) allergic (Cacconi's reaction) tests.

Huge cysts, in contrast with open air cysts, are characterized by a subpleural location.

The diagnostic doubts at suspicion on a **diaphragmatic hernia** are solved by radiopaque examination of a gastrointestinal tract.

The **acquired cysts** differ from congenital by roentgenological signs of the lesion of surrounding tissues (pneumofibrosis, deforming bronchitis, secondary bronchiectases, and calcifications). Postpneumonic cavities are of irregular shape, with grooves and pockets, their walls different thick. During bronchography they filled through some small bronchi. Sometimes the verification is possible only after postoperative morphological investigation.

Tactics and choice of treatment

The pulmonary cysts require the surgical treatment. In case of complications the indication for operation becomes absolute. Contraindications: the severe respiratory disturbance, concomitant malignant nonresectable tumors, vital organs dysfunction in the stage of permanent decompensation, elderly age of the patients.

The volume of the operation: if the adjacent pulmonary tissue is intact – cystectomy, otherwise – segmental or wedged resection, lobectomy.

The conservative therapy is applied in suppuration of cysts with the purpose of preoperative preparation. It is the similar, which applied for abscesses of lungs.

SPONTANEOUS PNEUMOTHORAX

Spontaneous pneumothorax is the entry of air in a pleural space with the further lung collapse, which not associated with traumatic damage of chest or pulmonary tissue.

Etiology and pathogenesis

As a result of spontaneous disrapture of lung blebs and subpleural air cysts occurs the damage of pleural visceral membrane. It causes entry of air in pleural space. Owing to its leakage the elastic pulmonary tissue collapses. The degree of the collapse of lung depends on amount of air, that has penetrated a pleural space.

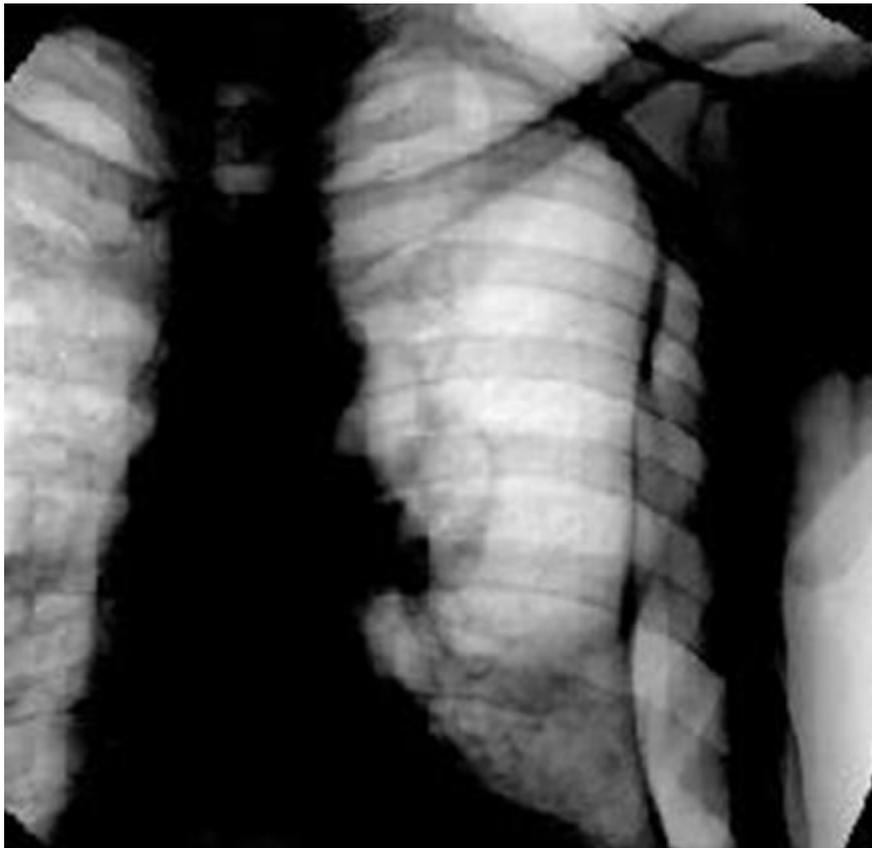
Pathology

Morphologically in spontaneous pneumothorax found out a focal bullous emphysema with disrapture of blebs, subpleural air cyst, and also disorders, which have caused disturbances of ventilating ability of bronchi. It can be bronchitis, pneumosclerosis, tuberculosis and fibrous alveolitis.

Classification

The pneumothorax can be:

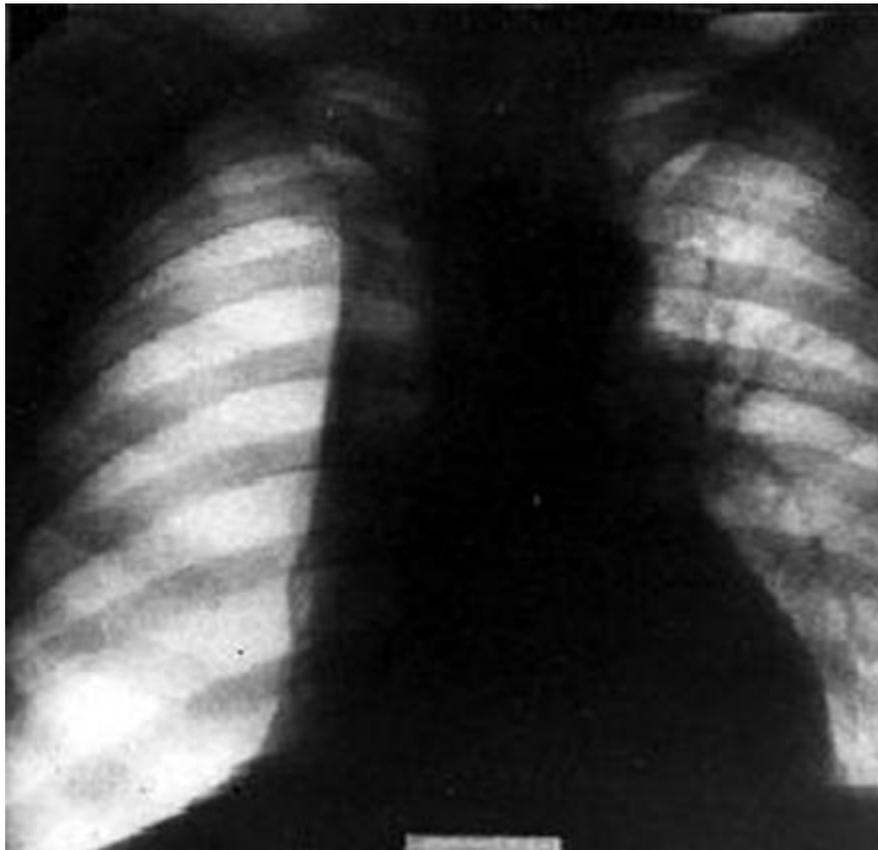
1. Unilateral or bilateral.
2. Partial (lung collapse to 1/3 of its volume).
3. Subtotal (lung collapse to 2/3 of its volume).
4. Total (lung collapse more than 2/3 of its volume).
5. Tension or valvular (complete collapse of lungs and shift of mediastinum in the opposite side).
6. Rigid (neglected pneumothorax with thickened visceral pleura).



Partial spontaneous pneumothorax



Subtotal spontaneous pneumothorax



Total spontaneous pneumothorax

Manifestation and clinical course

The onset of the disease is sudden. A state of the patient and expression of clinical manifestations depends on amount of air, which has entered the pleural space. Usually in normal conditions, and sometimes after physical activity, the patients suddenly feel acute pain on the side of lesion, dyspnea, pain in the heart region and heartbeating. An available also Acrocyanosis or total cyanosis of skin is observed. The circulatory disturbance depends on degree of hypoxia. Intensity of pain and dyspnea gradually decrease, but a dry troublesome cough appears.

Examination of the chest allows to observe expansion of intercostal spaces and restriction of respiratory excursion. By palpation - diminishing of vocal fremitus on the affected side. At percussion a chief sign of pneumothorax is the tympanic sound. Auscultation reveals weakened or sharply weakened breathing sounds. The cardiac tones are muffled, tachycardia.

The diagnosis of a spontaneous pneumothorax is confirmed by X-ray examination. On the plain roentgenogram the air is present in pleural space, and the margins of collapsed lung are found out on its background.

Thoracoscopy in pneumothorax possible to find out subpleural blebs of different sizes (0,5-3 cm), which are mainly disposed on the apex of lung.

Variants of clinical course and complications

The atypical (asymptomatic) spontaneous pneumothorax occurs in 20 % of the patients, and mainly revealed at X-ray examination. In most cases it is the partial pneumothorax.

The expressed pain syndrome and dyspnea, which resulting from collapse of lung, characterize subtotal and total pneumothorax.

The tension spontaneous pneumothorax is the most severe form of pneumothorax. It manifests by sudden onset, progressive increase of dyspnea, expressed cyanosis. The breathing is superficial, rapid, with active participation of auxiliary muscles. Mediastinal shift and flexion of vessels result in disturbance of cardiac activity up to the cardiac arrest, and requires urgent management.

Rigid pneumothorax (rigid lung). The neglected pneumothorax causes fibrinous exsudative pleurisy. On the surface of lung (visceral pleura) commissures are formed, that give no opportunity for lung expansion. The presence of residual pleural cavity and progressive development of a purulent infection result in occurrence of acute pleural empyema. In such cases the patients complain of the fever up to 38-38,5°C, general weakness and increasing of dyspnea. The phenomena of intoxication increase. Thus such patients require the treatment of pleural empyema.

The diagnostic program

1. Complaint and anamnesis of disease.
2. Physical findings.
3. Routine chest film (direct and lateral projection).
4. [Thoracentesis](#)
5. Thoracoscopy.
6. Tomography of lungs.

Differential diagnostics

Pleural effusion. This pathology manifests by more gradual onset. As opposite to pneumothorax, the complaints of chest pain predominate above the complaints of dyspnea. Frequently such patients specify on transferred undercooling.

As well as the spontaneous pneumothorax, the pleural effusion is characterized by diminishing of vocal fremitus, dullness of percussion sound, weakened or absent breathing sounds over the exudate. Nevertheless in the routine roentgenogram in such cases observed a homogeneous intensive shadow of a pleural space with oblique upper contour. The puncture of pleural space enables finally to confirm the diagnosis of pleural effusion.

Intercostal neuralgia. A predominant sign in clinical pattern is acute pain, which intensifies at physical activity, changes of body position and body movements, at deep breathing. The localization of pain coincides with zone of innervation of intercostal nerves.

Examination and chest X-radiography reveals no pathological changes.

Tactics and choice of treatment

Conservative treatment is applied in the patients with a partial pneumothorax. Thus thoracentesis in II intercostal space in the midclavicular line with aspiration of air is performed. The cases of its inefficiency, and also subtotal, total and tension pneumothorax require drainage of a pleural space with active aspiration of air.

The operative management is necessary, if there is no efficiency from active aspiration (in incomplete expansion of lung), recurrent of course of the process, presence of great subpleural blebs and rigid pneumothorax. Volume of operation depends on extension of the process: liquidation of alveolar fistula, wedged resection of lung or lobectomy.

PYOPNEUMOTHORAX

Pyopneumothorax is the discharge of lung abscess of into pleural space, which is accompanied by purulent inflammation of pleural membranes with a collapse of lung.

Etiology and pathogenesis

Peripheral placement of the purulent focus in a pulmonary tissue results in destruction (fusion) of visceral membrane. As a result of, the pus and air penetrate into a pleural space that leads to a purulent inflammation of parietal and visceral membranes of pleura. The disorder of hermeticity of a pleural space results in a lung collapse.

Among other causes of pyopneumothorax are the chest trauma, which results in collapse of lung, infection and purulent inflammation of pleural membranes.

As the basic causes of pyopneumothorax are considered:

- acute abscess of lung;
- gangrenous abscess of lung;
- gangrene of lung;
- suppurative cyst of lung;
- abscessing pneumonia;
- bronchiectatic disease;
- subphrenic abscess, which has discharged into pleural space;
- damage of esophagus;
- mediastinitis;
- chest trauma;
- operation and diagnostic manipulations on chest organs.

Pathology

Morphologically in pyopneumothorax pus and air are present in pleural space. In the lungs subpleural disposed purulent or necrotic foci, which connected with a pleural space through a pleuro-pulmonary fistula. From the outside the zone of disrapture is confined by perifocal inflammation. In the draining bronchus it is possible to see manifestations of deforming, frequently polypous bronchitis.

Classification

I. According to etiological factor:

1. Specific.
2. Nonspecific.

II. According to pathogenic factor:

1. Primary.
2. Secondary.

III. According to clinical course:

1. Asymptomatic form.
2. Mild form.
3. Acute form.

IV. According to extension of the process:

1. Localized pyopneumothorax:

- a) parietal;
- b) apical;
- c) epiphrenic;
- d) paramediastinal;
- e) polychamber.

2. Subtotal pyopneumothorax.

3. Total pyopneumothorax.

4. Tension pyopneumothorax.

Manifestation and clinical course

The clinic of pyopneumothorax depends on the size of the focus of destruction, which influences on degree of lung collapse, and on amount of purulent content in a pleural space.

The pain owing to discharge of the focus of destruction into pleural space often arises suddenly.

The dyspnea occurs as a result of collapse of lung owing to leakage of pus and air into pleural space. Its expression is in direct ratio to lung collapse. Therefore a dyspnea in rest observed in a subtotal and total pyopneumothorax. It sharply amplifies even at minor physical activity. Auxiliary muscles take part in order to force respiration.

The expectoration of sputum with ichorous smell is the outcome of destructive process in a pulmonary tissue. Its amount decreases after discharge of pus into pleural space.

Hectic fever with caused by enlargement of the area of resorption. The patients are adynamic, flaccid. Some of them are unconsciousness.

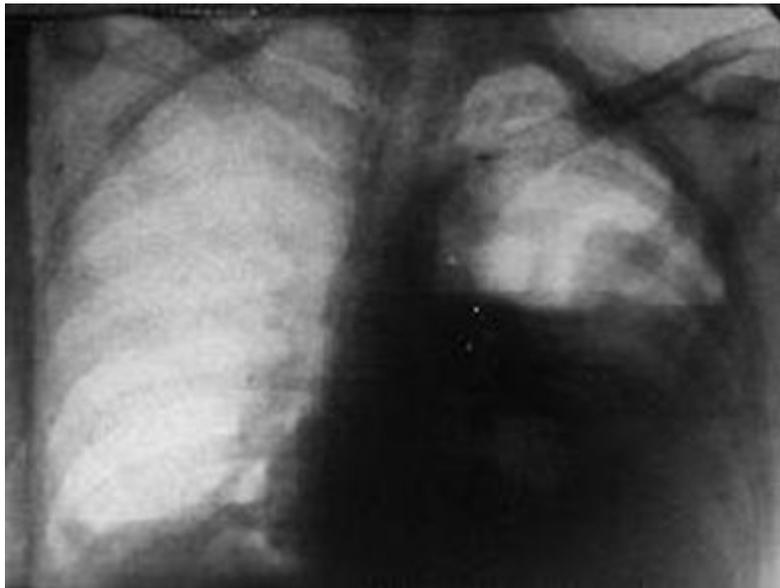
By objective examination the position of patients is forced, they sit in bed leaning upon the bed (subtotal, total pyopneumothorax). The affected hemithorax takes no part in respiration.

By palpation – diminished vocal fremitus on the side of lesion.

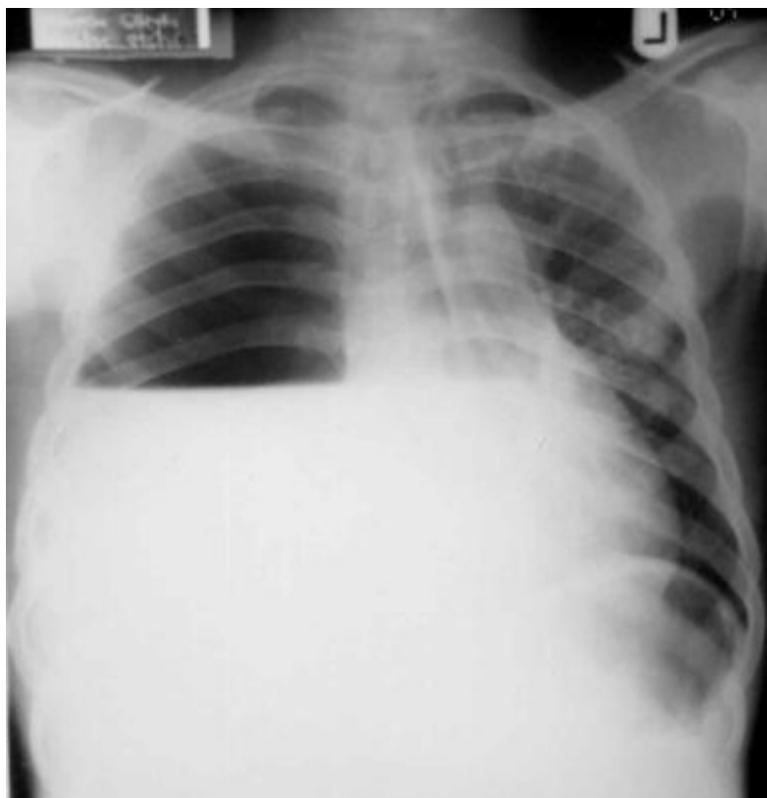
Percussion reveals a sharp shortening of sound over the zone of exudate and bandbox sound above the region of collapsed lung.

By auscultation there are no breathing sounds on the affected side. In case of localized pyopneumothorax – weakened or sharply weakened sound with a bronchial or amphoric tone.

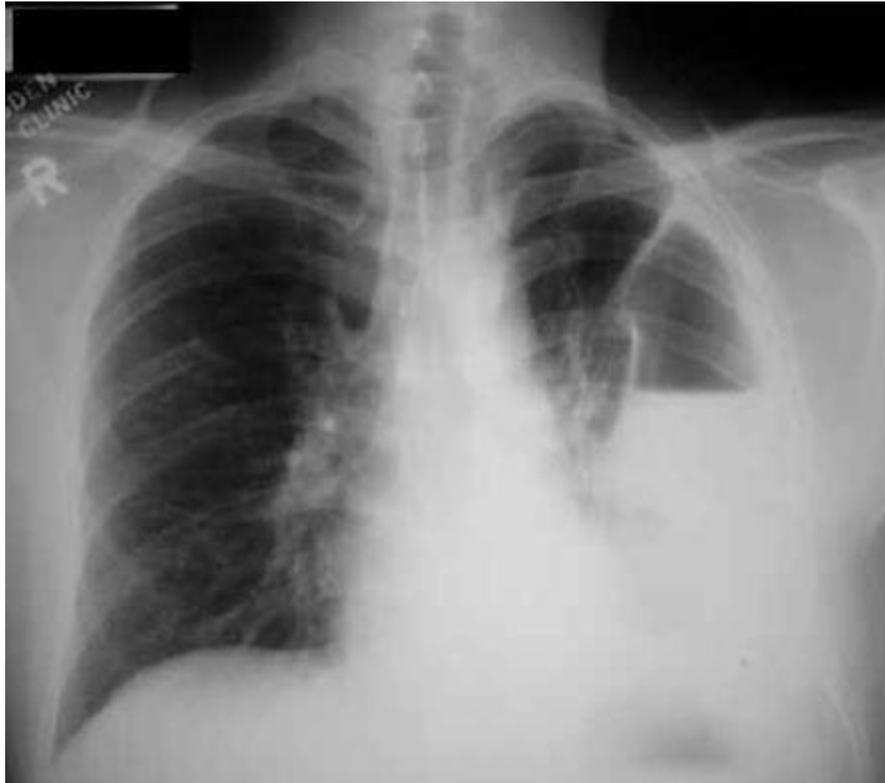
The X-ray picture of pyopneumothorax depends on its form, but the obligatory sign is the air-fluid level in a pleural space with well-defined edge of collapsed lung on its background.



Left-side total pyopneumothorax



Pyopneumothorax



Pyopneumothorax

Variants of clinical course and complications

The clinical course of pyopneumothorax depends on adhesions between pleural membranes. It sometimes changes typical clinical course of a total lung collapse. The clinic of disease depends also on amount of a purulent exudate. Therefore according to extension of the process and size of destruction of lung distinguished acute, mild and asymptomatic forms of pyopneumothorax. Especially difficult for diagnostics is the asymptomatic form of localized pyopneumothorax. Dyspnea for such pathology not characteristic, as the adhesion of membranes prevents complete collapse of lung. Dyspnea is vague or absent at all in partial collapse. A diminished vocal fremitus on side of pathological process, shortening of percussion sound and weakened or sharply weakened breathing sounds over the collapsed lung and exudate are revealed. Roentgenological manifestations in localized pyopneumothorax not expressed and include horizontal fluid level, margin of partially collapsed lung and minor air in the pleural space.

In most cases after discharge of the destructive focus in a pleural space and collapse of lung observed the closure of bronchopleural fistula. Nevertheless the inflammatory process in a pulmonary tissue and pleural space is going on.

The diagnostic program

1. Complaint and history of disease.
2. Physical findings.
3. Chest X-radiography examination.
4. [Thoracentesis](#).
5. Pleurography.
6. Bacterial culture and antibiotic sensitivity.
7. General blood, and urine analyses.
8. Biochemical blood analysis.

Differential diagnostics

They're no special necessity for differential diagnostics of pyopneumothorax in the majority of patients. History (presence of the purulent focus in a pulmonary tissue), clinical course (acute pain and dyspnea at abscess discharge into pleural space), and also chest X-ray findings and thoracentesis frequently reliably permit to make the diagnosis.

In some cases a localized pyopneumothorax according to clinical course resembles a huge acute abscess of lungs. But the differences of roentgenological symptomatology permit to verify these pathological processes. In acute lung abscess the cavity of destruction localized in a pulmonary parenchyma, it is of rounded form with horizontal fluid level and expressed perifocal infiltration.

Tactics and choice of treatment

The purpose of treatment should include sanitation of the destructive focus in pulmonary tissue and liquidation of complications; that means elimination of pus and air from a pleural space and prompt expanding of lung.

1. Active sanitation of tracheobronchial tree by means of [tracheocentesis](#).
2. [Draining of pleural space](#), active aspiration of its content (air, pus) to expand the lungs.
3. Lavage of pleural space by antiseptic solutions.
4. Appropriate antibacterial, antiinflammatory and infusion therapy.

5. The therapy for rising up of immunological resistance of the organism (staphylococcal anatoxin according to scheme, antistaphylococcal gamma-globulin, antistaphylococcal plasma).
6. Endolymphatic introduction of immunity stimulators (thymalin, thymogen, T-activin).

The indications for operative management are the same, as in pleural empyema.

CHEST TRAUMA .

Chest trauma is a significant source of morbidity and mortality in the United States. This article focuses on chest trauma caused by blunt mechanisms. Penetrating thoracic injuries are addressed in Penetrating Chest Trauma.

Blunt injury to the chest can affect any one or all components of the chest wall and thoracic cavity. These components include the bony skeleton (ribs, clavicles, scapulae, sternum), lungs and pleurae, tracheobronchial tree, esophagus, heart, great vessels of the chest, and the diaphragm. In the subsequent sections, each particular injury and injury pattern resulting from blunt mechanisms is discussed. The pathophysiology of these injuries is elucidated, and diagnostic and treatment measures are outlined.

Frequency

Trauma is responsible for more than 100,000 deaths annually in the United States. Estimates of thoracic trauma frequency indicate that injuries occur in 12 persons per million population per day. Approximately 33% of these injuries require hospital admission. Overall, blunt thoracic injuries are directly responsible for 20-25% of all deaths, and chest trauma is a major contributor in another 50% of deaths.

Pathophysiology

The major pathophysiologies encountered in blunt chest trauma involve derangements in the flow of air, blood, or both in combination. Blunt trauma commonly results in chest wall injuries (eg, rib fractures). The pain associated with these injuries can make breathing difficult, and this may compromise ventilation.

Direct lung injuries, such as pulmonary contusions, are frequently associated with major chest trauma and may impair ventilation by a similar mechanism.

Space-occupying lesions, such as pneumothoraces, hemothoraces, and hemopneumothoraces, interfere with oxygenation and ventilation by compressing otherwise healthy lung parenchyma. A situation of special concern is tension pneumothorax in which pressure continues to build in the affected hemithorax as air leaks from the pulmonary parenchyma into the pleural space. This can push mediastinal contents toward the opposite hemithorax. Distortion of the superior vena cava by this mediastinal shift can result in decreased blood return to the heart, circulatory compromise, and shock.

Relevant Anatomy

The thorax is bordered superiorly by the thoracic inlet, just cephalad to the clavicles. The major arterial blood supply to and venous drainage from the head and neck pass through the thoracic inlet.

The thoracic outlets form the superolateral borders of the thorax and transmit branches of the thoracic great vessels that supply blood to the upper extremities. The nerves that comprise the brachial plexus also access the upper extremities via the thoracic outlet. The veins that drain the arm, most importantly the axillary vein, empty into the subclavian vein, which returns to the chest via the thoracic outlet.

Inferiorly, the pleural cavities are separated from the peritoneal cavity by the hemidiaphragms. Communication routes between the thorax and abdomen are supplied by the diaphragmatic hiatuses, which allow egress of the aorta, esophagus, and vagal nerves into the abdomen and ingress of the vena cava and thoracic duct into the chest.

The chest wall is composed of layers of muscle, bony ribs, costal cartilages, sternum, clavicles, and scapulae. In addition, important neurovascular bundles course along each rib, containing an intercostal nerve, artery, and vein. The inner lining of the chest wall is the parietal pleura. The visceral pleura invests the lungs. Between the visceral and parietal pleurae is a potential space, which, under normal conditions, contains a small amount of fluid that serves mainly as a lubricant.

The lungs occupy most of the volume of each hemithorax. Each is divided into lobes. The right lung has 3 lobes, and the left lung has 2 lobes. Each lobe is further divided into segments.

The trachea enters through the thoracic inlet and descends to the carina at thoracic vertebral level 4, where it divides into the right and left mainstem bronchi. Each mainstem bronchus divides into lobar bronchi. The bronchi continue to arborize to supply the pulmonary segments and subsegments.

The heart is a mediastinal structure contained within the pericardium. The right atrium receives blood from the superior vena cava and inferior vena cava. Right atrial blood passes through the tricuspid valve into the right ventricle. Right ventricular contraction forces blood through the pulmonary valve and into the pulmonary arteries. Blood circulates through the lungs, where it acquires oxygen and releases carbon dioxide. Oxygenated blood courses through the pulmonary veins to the left atrium. The left heart receives small amounts of nonoxygenated blood via the thebesian veins, which drain the heart, and the bronchial veins.

Left atrial blood proceeds through the mitral valve into the left ventricle.

Left ventricular contraction propels blood through the aortic valve into the coronary circulation and the thoracic aorta, which exits the chest through the diaphragmatic hiatus into the abdomen. A ligamentous attachment (remnant of ductus arteriosus) exists between the descending thoracic aorta and pulmonary artery just beyond the take-off of the left subclavian artery.

The esophagus exits the neck to enter the posterior mediastinum. Through much of its course, it lies posterior to the trachea. In the upper thorax, it lies slightly to the right with the aortic arch and descending thoracic aorta to its left. Inferiorly, the esophagus turns leftward and enters the abdomen through the esophageal diaphragmatic hiatus. The thoracic duct arises primarily from the cisterna chyli in the abdomen. It traverses the diaphragm and runs cephalad through the posterior mediastinum in proximity to the spinal column. It enters the neck and veers to the left to empty into the left subclavian vein.

Clinical

The clinical presentation of patients with blunt chest trauma varies widely and ranges from minor reports of pain to florid shock. The presentation depends on the mechanism of injury and the organ systems injured.

Obtaining as detailed a clinical history as possible is extremely important in the assessment of a patient with a blunt thoracic trauma. The time of injury, mechanism of injury, estimates of MVA velocity and deceleration, and evidence of associated injury to other systems (eg, loss of consciousness) are all salient features of an adequate clinical history. Information should be obtained directly from the patient whenever possible and from other witnesses to the accident if available.

Classification

The closed damages of the chest are divided:

I. According to the injury of other organs:

1. Isolated trauma.
2. Combined trauma (craniocerebral, with damage of abdominal organs, with damage of bones).

II. According to the mechanism of trauma:

1. Contusion.
2. Compression.
3. Commotion.
4. Fracture.

III. According to the character of the chest viscerae damage:

1. Without damage of viscerae.
2. With damage of viscerae (lungs, trachea, bronchi, esophagus, heart, vessels, diaphragm etc.).

IV. According to the character of complications:

1. Uncomplicated.
2. Complicated:
 - 1) Early (pneumothorax, hemothorax, subcutaneous, mediastinal emphysema floative rib fracture, traumatic shock, asphyxia);
 - 2) Late (posttraumatic pneumonia, posttraumatic pleurisy, suppurative diseases of lungs and pleura).

V. According to the state of cardiopulmonary system:

1. Without phenomena of respiratory failure.
2. Acute respiratory failure (of I, II, III degree).
3. Without phenomena of cardiovascular failure.
4. Acute cardiovascular failure (of I, II, III degree).

VI. According to the gravity of a trauma:

1. Mild.
2. Moderate.
3. Severe.

RIB FRACTURE

Simple rib fractures are the most common injury sustained following blunt chest trauma, accounting for more than half of thoracic injuries from nonpenetrating trauma. Approximately 10% of all patients admitted after blunt chest trauma have one or more rib fractures. These fractures are rarely life-threatening in themselves but can be an external marker of more severe visceral injury inside the abdomen and the chest.

The most common mechanism of injury for rib fractures in elderly persons is a fall from height or from standing. In adults, motor vehicle accident (MVA) is the most common mechanism. Youths sustain rib fractures most often secondary to recreational and athletic activities.

Rib fractures may compromise ventilation by a variety of mechanisms. Pain from rib fractures can cause respiratory splinting, resulting in atelectasis and pneumonia. Multiple contiguous rib fractures (ie, flail chest) interfere with normal costovertebral and diaphragmatic muscle excursion, potentially causing ventilatory insufficiency. Fragments of fractured ribs can also act as penetrating objects leading to the formation of a hemothorax or a pneumothorax. Ribs commonly fracture at the point of impact or at the posterior angle (structurally their weakest area). Ribs four through nine (4-9) are the most commonly injured.

Physical

Rib fractures are the most common blunt thoracic injuries. Ribs 4-10 are most frequently involved. Patients usually report inspiratory chest pain and discomfort over the fractured rib or ribs. Physical findings include local tenderness and crepitus over the site of the fracture. If a pneumothorax is present, breath sounds may be decreased and resonance to percussion may be increased. Rib fractures may also be a marker for other associated significant injury, both intrathoracic and extrathoracic. In one report, 50% of patients with blunt cardiac injury have rib fractures. Fractures of ribs 8-12 should raise the suggestion of associated abdominal injuries. Lee and colleagues reported a 1.4- and 1.7-fold increase in the incidence of splenic and hepatic injury, respectively, in those with rib fractures.

Paradoxical chest wall excursion with inspiration is seen with flail chest. A flail chest occurs when a large segment of ribs is not attached to the spine. These ribs are broken in at least 2 places on each rib. The paradoxical movement occurs because the middle section of the rib between the 2 fracture sites moves in response to intrathoracic pressure changes not intercostal muscle contractions.

Specific signs of ventilatory insufficiency include cyanosis, tachypnea, retractions, and use of accessory muscles for ventilation.

If fracture of the lower ribs is suspected, assess the patient for abdominal tenderness and costal margin tenderness, which could raise suspicion for injury to intra-abdominal organs.[14]

Chest radiographs

Anteroposterior (AP) and lateral chest films are used routinely to assist in the diagnosis of rib fractures, yet sensitivity as low as 50% has been reported. Delayed or follow-up radiographs can be very helpful.

Chest radiographs are much more useful in the diagnosis of underlying injuries, including hemothorax, pneumothorax, lung contusion, atelectasis, pneumonia, and vascular injuries.

Findings of sternal fracture[3] or scapular fracture[15] should increase suspicion for rib fractures.

Chest CT scan

A chest CT scan is more sensitive than plain radiographs for detecting rib fractures. The modality can also provide information regarding the number of ribs involved.

If complications from rib fractures are suspected clinically or diagnosed by plain radiographs, a chest CT scan may be helpful to document specific injuries, to characterize extent of injury, and to plan for definitive management.

An associated CT scan of the abdomen with intravenous contrast should be considered in cases involving lower rib fractures with suspected or known injury to the liver and/or the spleen.



The fracture of the VI-VIII left ribs

First and second rib fractures

First and second rib fractures are considered a separate entity from other rib fractures because of the excessive energy transfer required to injure these sturdy and well-protected structures. First and second rib fractures are harbingers of associated cranial, major vascular, thoracic, and abdominal injuries. The clinician should aggressively seek to exclude the presence of these other injuries.

Pain control and pulmonary toilet are the specific treatment measures for rib fractures. First and second rib fractures do not require surgical therapy. An exception to this would be the need to excise a greatly displaced bone fragment.

Treatment

Elderly patients with 3 or more rib fractures have been shown to have a 5-fold increased mortality rate and a 4-fold increased incidence of pneumonia. Effective pain control is the cornerstone of medical therapy for patients with rib fractures. For most patients, this consists of oral or parenteral analgesic agents. Intercostal nerve blocks may be feasible for those with severe pain who do not have numerous rib fractures. A local anesthetic with a relatively long duration of action (eg, bupivacaine) can be used. Patients with multiple rib fractures whose pain is difficult to control can be treated with epidural analgesia.

Adjunctive measures in the care of these patients include early mobilization and aggressive pulmonary toilet. Rib fractures do not require surgery. Pain relief and the establishment of adequate ventilation are the therapeutic goals for this injury. Rarely, a fractured rib lacerates an intercostal artery or other vessel, which requires surgical control to achieve hemostasis acutely. In the chronic phase, nonunion and persistent pain may also require an operation.

The direct force of traumatizing factor on the chest wall results in rib fracture.

The pain localized in the zone of damage, is the chief clinical manifestation. The pain intensifies at respiration, cough and change of a body position of the patient. The overwhelming majority of the patients complain of crepitation of ribs in the fracture site.

At examination the respiratory lag on affected side is observed.

Crepitating of osseous fragment revealed by palpation, and depending on number of injured ribs – diminished

breathing sounds by auscultation.

On chest roentgenograms the break in continuity of bone fragments of ribs is observed.

Floating rib fracture (flail chest)

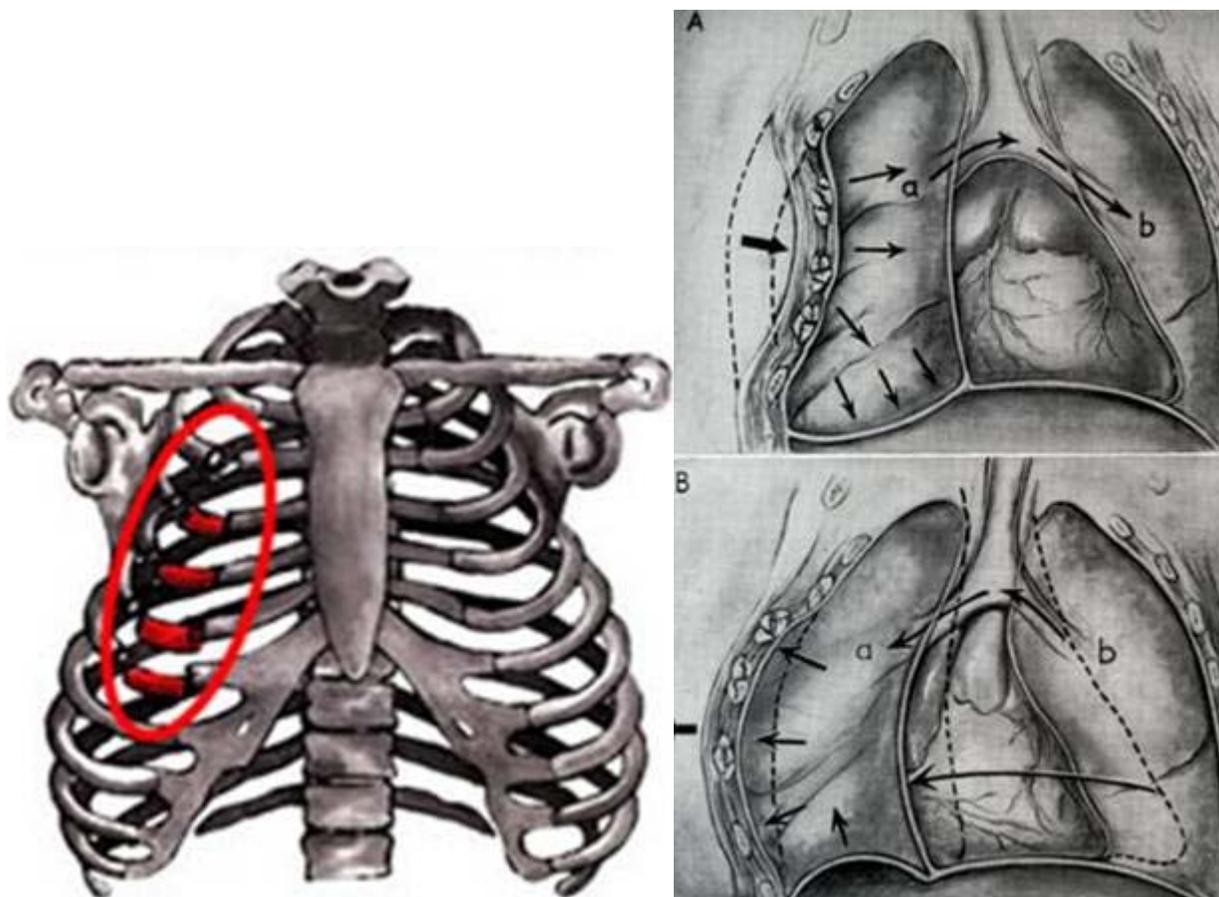
This is one of the most severe complication of the closed trauma of the chest. The floatation arises from fracture of three and more ribs along two anatomic lines. The multiple rib fractures produce an unstable segment of chest wall that moves paradoxically inward upon inspiration and balloons outward during expiration (flail chest). Thereby the respiration disturbed not only in the area of a floating segment, but also in all lungs. The permanent movement of flail chest result in rocking shift of mediastinum, which causes deviation of its organs. As a result the respiratory failure is associated with cardiovascular.

A flail chest, by definition, involves 3 or more consecutive rib fractures in 2 or more places, which produces a free-floating, unstable segment of chest wall. Separation of the bony ribs from their cartilaginous attachments, termed costochondral separation, can also cause flail chest. Patients report pain at the fracture sites, pain upon inspiration, and, frequently, dyspnea. Physical examination reveals paradoxical motion of the flail segment. The chest wall moves inward with inspiration and outward with expiration. Tenderness at the fracture sites is the rule. Dyspnea, tachypnea, and tachycardia may be present. The patient may overtly exhibit labored respiration due to the increased work of breathing induced by the paradoxical motion of the flail segment.

A significant amount of force is required to produce a flail segment. Therefore, associated injuries are common and should be aggressively sought. The clinician should specifically be aware of the high incidence of associated thoracic injuries such as pulmonary contusions and closed head injuries, which, in combination, significantly increase the mortality associated with flail chest.

All of the treatment modalities mentioned above for patients with rib fractures are appropriate for those with flail chest. Respiratory distress or insufficiency can ensue in some patients with flail chest because of severe pain secondary to the multiple rib fractures, the increased work of breathing, and the associated pulmonary contusion. This may necessitate endotracheal intubation and positive pressure mechanical ventilation. Intravenous fluids are administered judiciously because fluid overloading can precipitate respiratory failure, especially in patients with significant pulmonary contusions.

In an attempt to stabilize the chest wall and to avoid endotracheal intubation and mechanical ventilation, various operations have been devised for correcting flail chest. These include pericostal sutures, the application of external fixation devices, or the placement of plates or pins for internal fixation. With improved understanding of pulmonary mechanics and better mechanical ventilatory support, surgical therapy has not been proven superior to the supportive and medical measures discussed. However, most authors would agree that stabilization is warranted if a thoracotomy is indicated for another reason.



Flail chest

Classification

1. Central floating segment – a multiple rib fracture along parasternal or midclavicular lines.
2. Anterolateral floating segment – a multiple rib fracture along parasternal and anteaillary lines.

3. Lateral floating segment – a multiple rib fracture along anterior and posterior axillary lines.
4. Posterior floating segment – a multiple rib fracture along postaxillary and paravertebral lines.

Symptomatology and clinical course

The patient's state is grave or extremely grave. The expressed pain syndrome frequently results in traumatic shock. The patient is restless. Observed the cyanosis of skin, tachypnea, and tachycardia to 120-160 beat/min of weak filling and tension. Arterial pressure at first elevated, then its decrease observed. At examination characteristic paradoxical respiratory movements of chest, inward upon inspiration and outward during expiration, crepitus of bone fragments by palpation are revealed. Breathing sounds diminished on the side of damage by auscultation.

In case of floating rib fracture the chest X-ray examination reveals multiple, double rib fracture with deformity of the chest.



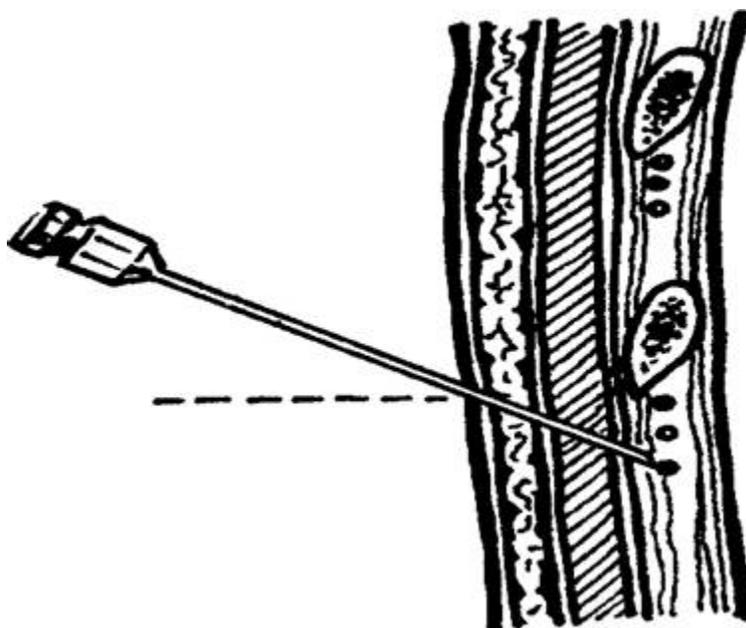
Flail chest

In 75 % of cases the multiple rib fracture is the cause of injury of lungs, pneumothorax or pneumohemothorax.

Treatment

Pain relief in closed trauma of the chest is achieved by means of different blocks:

1. Vagosympathetic block;
2. Alcohol - novocaine block of the site of fracture;
3. Paravertebral block.



Alcohol - novocaine block of the site of fracture

Except blocks, in some cases analgesics and opiates are instituted. On 2-3 day desirable the administration of electrophoresis with novocaine. For prophylaxis of congested phenomena in a pulmonary tissue used respiratory gymnastics, forced ventilation of lungs, inhalations.

The methods of reduction of the skeleton of the flail chest are divided onto three groups:

1. External fixation of a movable segment by means of suturing for intercostal muscles and traction during 2-3 weeks;
2. Intramedullary costal osteosynthesis;
3. Mechanical ventilation (often with positive end-expiratory pressure).

BREASTBONE FRACTURE

Most sternal fractures are caused by MVAs. The upper and middle thirds of the bone are most commonly affected in a transverse fashion. Patients report pain around the injured area. Inspiratory pain or a sense of dyspnea may be present. Physical examination reveals local tenderness and swelling. Ecchymosis is noted in the area around the fracture. A palpable defect or fracture-related crepitus may be present.

Associated injuries occur in 55-70% of patients with sternal fractures. The most common associated injuries are rib fractures, long bone fractures, and closed head injuries. The association of blunt cardiac injuries with sternal fractures has been a source of great debate. Blunt cardiac injuries are diagnosed in fewer than 20% of patients with sternal fractures. Caution should be used before completely excluding myocardial injury. The workup should begin with an ECG.

Most sternal fractures require no therapy specifically directed at correcting the injury. Patients are treated with analgesics and are advised to minimize activities that involve the use of pectoral and shoulder girdle muscles. The most important aspect of the care for these patients is to exclude blunt myocardial and other associated injuries. Patients who are experiencing severe pain related to the fracture and those with a badly displaced fracture are candidates for open reduction and internal fixation. Various techniques have been described, including wire suturing and the placement of plates and screws. The latter technique is associated with better outcomes.

The fracture of breastbone is commonly caused by direct forces at the site of the sternum. Usually it is the outcome of compression or result of trauma to vehicle helm.

The fracture in most cases located in the upper and medial thirds of breastbone.

The patients complain of severe pain in the site of fracture, which intensifies at respiration and movements. The pain behind the sternum and in the heart area follows the contusion of lungs and heart. Sometimes hemoptysis is observed.

Examination reveals the deformity of breastbone in the site of fracture. Displaced fragments are palpated here, that accompanied by severe pain syndrome.

By auscultation, if there are no intrapleural complications, the respiration in the first 2-3 days is vesicular from both sides. Then the fine bubbling rales are auscultated which is the first objective manifestation of a posttraumatic pneumonia.

The complete fracture of breastbone is characterized by a break in continuity of both cortical plates with a local dislocation of fragments.



Fracture of breastbone

The diagnostic program

1. Complaints and history of the disease.
2. Physical findings.
3. Chest roentgenograms in two planes.

Treatment

The sternal fracture without displacement of fragments requires conservative treatment. The fracture of the

corpus of breastbone with dislocation of fragments quite often requires operative treatment with performance of osteosynthesis.

POSTTRAUMATIC PNEUMOTHORAX

Pneumothorax is defined as the presence of air or gas in the pleural cavity, that is, in the potential space between the visceral and parietal pleura of the lung. The result is collapse of the lung on the affected side. Air can enter the intrapleural space through a communication from the chest wall (ie, trauma) or through the lung parenchyma across the visceral pleura.

Spontaneous pneumothorax is a commonly encountered problem with approaches to treatment that can vary from observation to aggressive intervention. Spontaneous pneumothorax occurs in people without underlying lung disease and in the absence of an inciting event. In other words, air is present in the intrapleural space without preceding trauma and without underlying clinical or radiologic evidence of lung disease. However, many patients whose condition is labeled as spontaneous pneumothorax have subclinical lung disease. Patients are typically between age 18 and 40 years.

Spontaneous pneumothoraces in most patients occur from the rupture of blebs and bullae. Although primary spontaneous pneumothorax (PSP) is defined as a lack of underlying pulmonary disease, these patients have asymptomatic blebs and bullae detected on computed tomography scans or upon thoracotomy. PSP is typically observed in tall, young people without parenchymal lung disease and is thought to be related to increased shear forces in the apex.

Traumatic pneumothorax results from injury, typically blunt trauma or penetrating trauma that disrupts the parietal or visceral pleura (see the images below). The mechanisms of injury are secondary to medical or surgical procedures. Pneumothoraces due to trauma are relatively straightforward and usually require tube thoracostomy.

Pathophysiology

The pleural space has a negative pressure, with the chest wall tending to spring outward and the lung's elastic recoil tending to collapse. If the pleural space is invaded by gas from a ruptured bleb, the lung collapses until equilibrium is achieved or the rupture is sealed. As the pneumothorax enlarges, the lung becomes smaller. The main physiologic consequence of this process is a decrease in vital capacity and partial pressure of oxygen.

Tension pneumothorax occurs anytime a disruption involves the visceral pleura, parietal pleura, or the tracheobronchial tree. This condition develops when injured tissue forms a 1-way valve, allowing air inflow into the pleural space and prohibiting air outflow. The volume of this nonabsorbable intrapleural air increases with each inspiration because of the 1-way valve

effect. In addition to this mechanism, the positive pressure used with mechanical ventilation therapy can cause air trapping. As a result, pressure rises within the affected hemithorax.

As the pressure increases, the ipsilateral lung collapses and causes hypoxia. Further pressure build-up causes the mediastinum to shift toward the contralateral side and impinge on and compress both the contralateral lung and the vasculature entering the right atrium of the heart. Hypoxia results as the collapsed lung on the affected side and the compressed lung on the contralateral side compromise effective gas exchange. This hypoxia and decreased venous return caused by compression of the relatively thin walls of the atria impair cardiac function. The inferior vena cava is thought to be the first to kink and restrict blood flow back to the heart. It is most evident in trauma patients who may be hypovolemic with reduced venous blood return to the heart.

Arising from numerous causes, this condition rapidly progresses to respiratory insufficiency, cardiovascular collapse, and, ultimately, death if unrecognized and untreated.

Classification

I. According to extension of process:

1. Unilateral.
2. Bilateral.

II. According to degree of a lung collapse:

1. Partial (collapse of lung to 1/3 of its volume).
2. Subtotal (collapse of lung to 2/3 of its volume).
3. Total (collapse of lung exceeding 2/3 of its volume).

Estimating the size of the pneumothorax

In evaluating the chest radiograph, first impressions of pneumothorax size can be misleading. The following methods may be used to estimate the size of the pneumothorax:

Calculate the ratio of the transverse radius of the pneumothorax (cubed) to the transverse radius of the hemithorax (cubed). To express the pneumothorax size as a percentage, multiply the fractional size by 100. This formula assumes a constant shape of the lung when it collapses and is invalid if pleural adhesions are present. The ratio of lung size to hemithorax size to

estimate pneumothorax size avoids the subjective underestimation of pneumothorax expressed as a percentage of previous lung volume.

A 2.5-cm margin of gas peripheral to the collapsing lung corresponds to a pneumothorax of about 30%. Complete collapse of the lung is a 100% pneumothorax.

A simple approach involves measuring the distance from the apex of the lung to the top margin of the visceral pleura (thoracic cupola) on the upright chest radiograph, such that a small pneumothorax is a distance to the apex that measures less than 3 cm and large pneumothorax has greater than 3 cm distance to the apex.

III. According to the mechanism of occurrence:

1. Closed.
2. Open.
3. Valvular.

The closed pneumothorax is the complication, which arises from the damage of visceral pleural membrane, which results in entry of air in a pleural space and atelectasis of lung. In chest trauma the cause of occurrence of the closed pneumothorax is the perforation of a visceral pleura and pulmonary tissue by the fragment of fractured rib.

The open pneumothorax results from formation of hole in a chest wall at massive trauma and free entry of air during inspiration inward a pleural space, and during expiration – outward.

The valvular pneumothorax occurs at damage of a pulmonary tissue or chest wall with formation of the valve, when the air during inspiration enters a pleural space, and during expiration, due to valve closure, does not exits outside. It is the most dangerous form of pneumothorax, which results in a complete pulmonary collapse, shift of mediastinum, inflection of major vessels and cardiac arrest.

Symptomatology and clinical course

The chief clinical manifestation of posttraumatic pneumothorax, which results from a pulmonary collapse, is the rest dyspnea, which amplifies at a minor exertion. This sign arises due to atelectasis of lung and its exclusion from breathing. On the background of collapsed lung only the main and lobar bronchi and pleural space are ventilated. The oxygenation of blood in collapsed lungs does not occur, therefore the shunting of a venous blood arise.

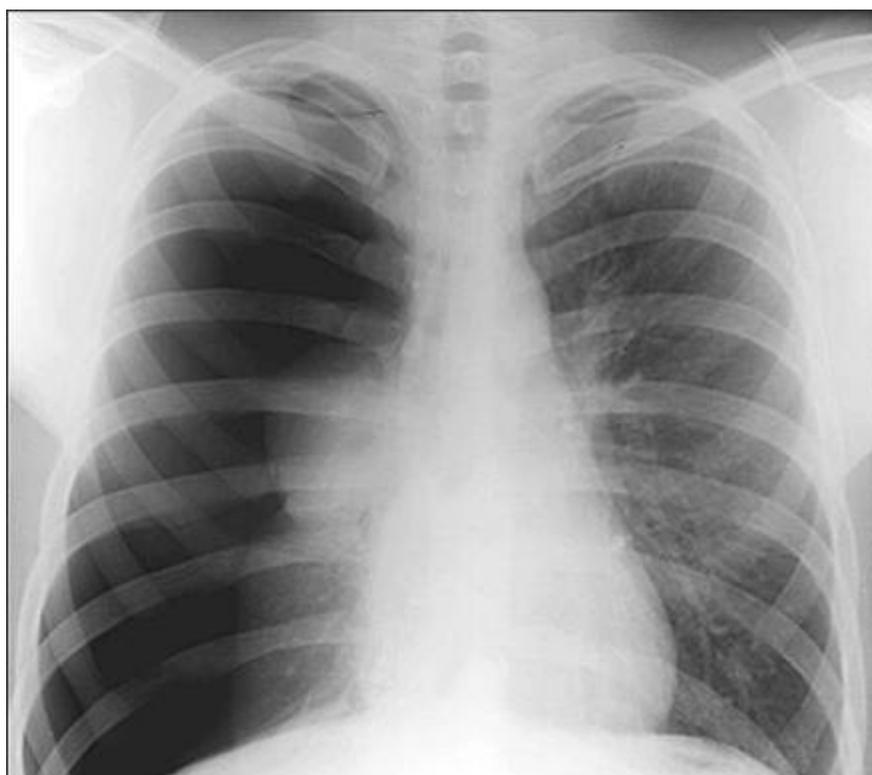
The chest pain is more characteristic manifestation for trauma with the damage of ribs, however pulmonary collapse also can associate with a pain syndrome. Nevertheless the patients promptly adapt for it and the

dyspnea finally remains the basic clinical manifestation of such complication.

On the background of severe trauma of the chest the signs of damage dominate in clinical manifestation on inappreciable entry of air in a pleural space. Pneumothorax mostly revealed during X-ray examination. Progressing of air entry in a pleural space and pulmonary collapse cause the respiratory lag on affected side. By palpation the vocal fremitus is absent. It indicates the origin of the complication – rib fracture.

Percussion obtains bandbox sound, or pulmonary sound with tympanitis. By auscultation - weak or absent breathing sounds, sometimes – amphoric respiration. The expressiveness of clinical pattern depends on degree of a pulmonary collapse.

Pulmonary atelectasis and presence of air in a pleural space are the X-ray findings that enable to establish the final diagnosis.



Right-side pneumothorax

The diagnostic program

1. Complaints and history of the disease.

The presentation of patients with pneumothorax varies depending on the type of

pneumothorax.

Spontaneous and iatrogenic pneumothorax

Despite descriptions of Valsalva maneuvers and increased intrathoracic pressures as inciting factors, spontaneous pneumothorax usually develops at rest. By definition, spontaneous pneumothorax is not associated with trauma or stress. Symptoms of iatrogenic pneumothorax are similar to those of a spontaneous pneumothorax and, depend on the age of the patient, the presence of underlying lung disease, and the extent of the pneumothorax.

Until a bleb ruptures and causes pneumothorax, no clinical signs or symptoms are present in primary spontaneous pneumothorax (PSP). Young and otherwise healthy patients can tolerate the main physiologic consequences of a decrease in vital capacity and partial pressure of oxygen fairly well, with minimal changes in vital signs and symptoms, but those with underlying lung disease may have respiratory distress. The most common underlying abnormality in secondary spontaneous pneumothorax is chronic obstructive pulmonary disease (COPD), and cystic fibrosis carries one of the highest associations, with more than 20% reporting spontaneous pneumothorax.

In one series, acute onset of chest pain and shortness of breath were present in all patients in one series; typically, both symptoms are present in 64-85% of patients. The chest pain is described as severe and/or stabbing, radiates to the ipsilateral shoulder and increases with inspiration (pleuritic). In PSP, chest often improves over the first 24 hours, even without resolution of the underlying air accumulation. Well-tolerated primary pneumothorax can take 12 weeks to resolve. In secondary pneumothorax (SSP), the chest pain is more likely to persist with more significant clinical symptoms.

Shortness of breath/dyspnea in PSP is generally of sudden onset and tends to be more severe with secondary spontaneous pneumothoraces (SSPs) because of decreased lung reserve. Anxiety, cough, and vague presenting symptoms (eg, general malaise, fatigue) are less commonly observed.

A history of previous pneumothorax is important, as recurrence is common, with rates reported between 15% and 40%. Up to 15% of recurrences can be on the contralateral side. Secondary pneumothoraces are often more likely to recur, with cystic fibrosis carrying the highest recurrence rates at 68-90%. No study has shown that the number or size of blebs and bullae found in the lung can be used to predict recurrence.

Tension pneumothorax

Signs and symptoms of tension pneumothorax are usually more impressive than those seen with a simple pneumothorax, and clinical interpretation of these is crucial for diagnosing

and treating the condition. Unlike the obvious patient presentations oftentimes used in medical training courses to describe a tension pneumothorax, actual case reports include descriptions of the diagnosis of the condition being missed or delayed because of subtle presentations that do not always present with the classically described clinical findings of this condition.

Symptoms of tension pneumothorax may include chest pain (90%), dyspnea (80%), anxiety, fatigue, or acute epigastric pain (a rare finding).

2. Physical examination.

The general appearance of the patient with pneumothorax may vary from asymptomatic to respiratory distress. It may include diaphoresis, splinting chest wall to relieve pleuritic pain, and cyanosis (in the case of tension pneumothorax). Findings on lung auscultation also vary depending on the extent of the pneumothorax. Affected patients may also reveal altered mental status changes, including decreased alertness and/or consciousness (a rare finding).

Respiratory findings may include the following:

- Respiratory distress (considered a universal finding) or respiratory arrest
- Tachypnea (or bradypnea as a preterminal event)
- Asymmetric lung expansion: A mediastinal and tracheal shift to the contralateral side can occur with a large tension pneumothorax.
- Distant or absent breath sounds: Unilaterally decreased or absent lung sounds is a common finding, but decreased air entry may be absent even in an advanced state of the disease.
- Lung sounds transmitted from the unaffected hemithorax are minimal with auscultation at the midaxillary line
- Hyperresonance on percussion: This is a rare finding and may be absent even in an advanced state of the disease.
- Decreased tactile fremitus

Adventitious lung sounds (crackles, wheeze; an ipsilateral finding)

- Cardiovascular findings may include the following:
- Tachycardia: This is the most common finding. If the heart rate is faster than 135 beats per minute (bpm), tension pneumothorax is likely.
- Pulsus paradoxus
- Hypotension: This should be considered as an inconsistently present finding; although hypotension is typically considered a key sign of a tension pneumothorax, studies suggest that hypotension can be delayed until its

appearance immediately precedes cardiovascular collapse.

- Jugular venous distention: This is generally seen in tension pneumothorax, although it may be absent if hypotension is severe.
- Cardiac apical displacement: This is a rare finding.

3. Chest X-radiography in 2 planes.

Portable chest radiography should always be included in the initial radiographic evaluation of major trauma, as significant chest injuries carry an estimated 10-50% risk of associated pneumothorax. Chest computed tomography (CT) scanning should always be performed for significant chest injuries, because they carry an estimated risk of associated pneumothorax as high as 50% and about half of these pneumothoraces may be occult.

When evaluating the chest radiograph for pneumothorax, assess rotation, which can obscure a pneumothorax and mimic a mediastinal shift. Compare the symmetry and shape of the clavicles, and look at the relative lengths of the ribs in the middle lung fields on each side on the anteroposterior (AP) or posteroanterior (PA) views. On an image with rotation, the ribs on each side often have unequal lengths.

In a nonloculated pneumothorax, air rises to the nondependent portion of the pleural cavity. Therefore, carefully examine the apices of an upright chest radiograph, and scrutinize the costophrenic and cardiophrenic angles on a supine chest radiograph.

Finding of pneumothorax on chest radiographs may include the following:

- A linear shadow of visceral pleura with lack of lung markings peripheral to the shadow may be observed, indicating collapsed lung.
- An ipsilateral lung edge may be seen parallel to the chest wall.
- In supine patients, deep sulcus sign (very dark and deep costophrenic angle) with radiolucency along costophrenic sulcus may help to identify occult pneumothorax. The anterior costophrenic recess becomes the highest point in the hemithorax, resulting in an unusually sharp definition of the anterior diaphragmatic surface due to gas collection and a depressed costophrenic angle
- Small pleural effusions commonly are present and increase in size if the pneumothorax does not reexpand.
- Mediastinal shift toward the contralateral lung may also be apparent.
- Airway or parenchymal abnormalities in the contralateral lung suggest causes of secondary pneumothorax. Evaluation of the parenchyma in the collapsed lung is less reliable.

Although expiratory images are thought to better depict subtle pneumothoraces (the volume of the pneumothorax is constant and hence proportionally higher on expiratory images), a randomized controlled trial revealed no difference in the ability of radiologists to detect pneumothoraces on inspiratory and expiratory images after procedures with the potential to cause pneumothoraces.

4. [Thoracentesis](#).
5. ECG.
6. Chest CT Scanning

Computed tomography (CT) scanning is the most reliable imaging study for the diagnosis of pneumothorax, but it is not recommended for routine use in pneumothorax. This imaging modality can help to accomplish the following:

- Distinguish between a large bulla and a pneumothorax
- Indicate underlying emphysema or emphysemalike changes (ELCs)
- Determine the exact size of the pneumothorax, especially if it is small
- Confirm the diagnosis of pneumothorax in patients with head trauma who are mechanically ventilated
- Detect occult/small pneumothoraces and pneumomediastinum (although the clinical significance of these occult pneumothoraces is unclear, particularly in the stable nonintubated patient)

CT scanning is widely used in actual clinical practice to assess the possibility of associated concurrent pulmonary disease because of the inherent superiority of CT scans to visualize the details of lung parenchyma and pleura, as can be seen in the images below.

When performed on primary spontaneous pneumothorax patients, CT detects multiple blebs and bullae in the setting of negative chest radiographic findings. This may not impact management, as there has been no correlation between number of blebs and recurrence. However, CT scanning may have a role in secondary spontaneous pneumothorax, especially to differentiate from giant bullous emphysema.

CT scanning can detect occult pneumothorax in patients who will require mechanical ventilation in trauma and emergency surgery settings. This modality has also been shown to be more sensitive than radiography for hemothorax and pulmonary contusion.

Collapse of the lung, air in the pleural cavity, and deviation of mediastinal structures are present in tension pneumothorax.

Treatment

A chest trauma, which complicated by pneumothorax with a partial pulmonary collapse (to 1/3 of volume) is indication to aspiration of air by means of thoracentesis. The cases, if the negative pressure in a pleural space is not obtained, and also subtotal, and total pneumothorax require closed [drainage of a pleural space](#).

Under the local anesthesia by solution of novocaine in II intercostal space in the midclavicular line by means of a trocar in a pleural space inserted a plastic tube, which fixed to skin. The drainage connects to [aspirative system](#) or according to method of Bulau. In the majority of patients the pneumothorax liquidates in some hours, or during 1-2 days.

The absence of effect (incomplete expansion of lung) of active aspiration, and also valvular closed pneumothorax is the indications to operative management – suturing of the pulmonary wound. In some cases a segmental resection of lung, or lobectomy is carried out.

Emergent Needle Decompression

Tension pneumothorax is a life-threatening condition that demands urgent management. If this diagnosis is suspected, do not delay treatment in the interest of confirming the diagnosis (ie, before radiologic evaluation).

The basic principle of emergent needle decompression is to introduce a catheter into the pleural space, thus producing a pathway for the air to escape and relieving the built-up pressure. Although this procedure is not the definitive treatment for tension pneumothorax, emergent needle decompression does arrest its progression and serves to restore cardiopulmonary function slightly. Needle length in persons with large pectoral muscles may be an issue, and long needles or angiocatheters may be necessary.

Immediately place the patient on 100% oxygen, ventilate the patient if necessary, and evaluate the patient for evidence of respiratory compromise, hemodynamic instability, or clinical deterioration.

Essentially, a large-bore (16 or 18 gauge) angiocatheter is introduced in the midclavicular line at the second intercostal space. Use large-bore catheters, because hemothorax can be associated with pneumothorax, and the patient may, therefore, require immediate intravenous (IV) infusion. Upright positioning, if not inappropriate due to cervical spine or trauma concerns, may be beneficial. This serves as a bridge until the definitive

treatment of tube thoracostomy. The catheter is left in place until the chest tube is placed.

The procedure is as follows:

- Locate the anatomic landmarks and quickly prepare the area to be punctured with an iodine-based solution (eg, Betadine).
- Insert a large-bore needle with a catheter into the second intercostal space, just superior to the third rib at the midclavicular line, 1-2 cm from the sternal edge (ie, to avoid injury to the internal thoracic artery). Use a catheter or needle >5 cm long, and hold it perpendicular to the chest wall when inserting; however, note that some patients may have a chest wall thickness greater than 5 cm, and failure for the symptoms to resolve may be attributed to inadequate needle length.[1, 2, 3]
- Once the needle is in the pleural space, listen for the hissing sound of air escaping to confirm the diagnosis of tension pneumothorax (note this finding on the patient's chart). In an area with high ambient noise, the escape of air may not be detected. Remove the needle while leaving the catheter in place.
- Secure the catheter in place, and install a flutter valve.

After needle decompression, immediately begin preparation to insert a thoracostomy tube. Then, reassess the patient, paying careful attention to the ABCs (ie, airway, breathing, circulation) of trauma management.

Needle Aspiration

Nonemergent needle aspiration can be used to treat a small primary spontaneous pneumothorax (PSP) or an iatrogenic pneumothorax.

The procedure is as follows:

- Palpate the rib and intercostal space intended for needle aspiration. For needle aspiration, the anterior approach at the second or third intercostal space at the midclavicular line or a lateral approach at the fifth or sixth intercostal space at the midaxillary line is appropriate. For catheter aspiration, the lateral approach is preferred.
- Prepare the skin with povidone-iodine (Betadine), alcohol scrubs, or both, and cover with sterile drapes.
- Instill a local anesthetic (eg, 1% Xylocaine solution) to skin and soft tissue down to the pleura, directing the needle over the top of the rib into the desired

intercostal space.

- Insert a large-bore Angiocath (14-gauge in an adult, 18- or 20-gauge in an infant) or ready-to-use aspiration kit into the chosen intercostal space over the top of the rib and perpendicular to the chest wall.
- For simple needle aspiration, withdraw air once the pleural cavity is entered, and when resistance is felt, withdraw the needle.
- For catheter aspiration, once the pleural cavity is entered, a soft pigtail catheter is advanced over the needle into the pleural space. A scalpel may be necessary to enlarge the entry site at the skin. Remove the needle once the pleural cavity is entered, and attach the catheter to a 3-way stopcock and large syringe (eg, 60-mL syringe) to evacuate air.
- When no more air can be aspirated (discontinue if resistance is felt, if the patient coughs excessively, or if more than 2.5 L is aspirated) or the patient suddenly coughs, the lung most likely has reexpanded.
- Close the stopcock, and secure the catheter to the chest wall.
- Obtain a chest radiograph to assess the degree of success, and obtain another radiograph 4 hours later to confirm the absence of recurring accumulation.
- If no recurrence is present, remove the catheter and massage the insertion site with sterile gauze to seal the channel into the pleural space.
- Discharge the patient with appropriate return instructions. (Some authors suggest observation for an additional 2 h after catheter removal.)
- If the pneumothorax persists, attach a Heimlich valve or a water seal and admit the patient.

Potential complications associated with needle aspiration includes pneumothorax (with potential to later tension pneumothorax), cardiac tamponade, hemorrhage (which can be life threatening), loculated intrapleural hematoma, atelectasis, pneumonia, arterial air embolism (when needle thoracostomy is performed and no tension pneumothorax is present), and pain to the patient.

Tube Thoracostomy

Tube thoracostomy is the definitive treatment for secondary spontaneous pneumothorax (SSP) (see the following image) and tension pneumothorax. Needle decompression mandates an immediate follow up with a tube thoracostomy.

The procedure is as follows:

- If the patient is hemodynamically stable, consider conscious sedation with careful titration of a short-acting narcotic and benzodiazepine.
- Place the patient in a 30-60° reverse Trendelenburg position.
- Scrub the site (centered around the fifth or sixth rib in the midaxillary line) with povidone-iodine (Betadine), alcohol, or both.
- Locally anesthetize the site with lidocaine. (Use a generous amount, and anesthetize all the way down to the pleura.)
- Create a 3- to 4-cm horizontal incision over the fifth or sixth rib in the midaxillary line.
- Use a curved hemostat, and dissect (in a controlled manner) through the soft tissue and down to the rib.
- Push the hemostat just over the superior portion of the rib, avoiding the intercostal neurovascular bundle that runs under the inferior portion of the next most superior rib. Then, puncture the intercostal muscles and parietal pleura. Spread the hemostat wide to create an adequate opening.
- Maintain the intrapleural position by inserting a finger along side of the hemostat. Assess the presence and location of pulmonary adhesions. Sweep the finger in all directions, and feel for the diaphragm and possible intra-abdominal structures. Remove the hemostat. To avoid losing the desired tract, some authors recommend keeping the finger in place until the tube is inserted.
- Insert the chest tube over the finger into the pleural space. A clamp may suffice for guiding the thoracostomy tube into place on the proximal end.
- Direct the chest tube posteriorly, and insert it until it is at least 5 cm beyond the last hole in the tube.
- Look for condensation in the tube as a sign of correct placement and air evacuation.
- Attach the tube to a water seal and vacuum device (eg, Pleur-Evac). Look for respiratory variation of the water seal and bubbling of air through the water seal. Document the amount of blood or other fluids drained.
- Connect the thoracostomy tube to an underwater seal apparatus and suction.
- Suture the tube in place, dress the wound, and tape the tube to the chest. Cover the site with Vaseline-impregnated gauze, and apply a suitable dressing. A variety

of anchoring and closure techniques exist, all of which are probably equivalent

- Obtain a follow-up chest x-ray to assess tube positioning and lung reexpansion.

Complications of tube thoracostomy include death, injury to lung or mediastinum, hemorrhage (usually from intercostal artery injury), neurovascular bundle injury, infection, bronchopleural fistula, and subcutaneous or intraperitoneal tube placement.

Indications for Surgical Intervention

- Surgical decompression (vacuum-assisted thoracostomy [VATS], open thoracotomy, etc) is indicated for the following:
 - Persistent air leak for longer than 5-7 days with a chest tube in place
 - Recurrent, ipsilateral pneumothorax
 - Contralateral or bilateral pneumothorax
 - First-time presentation in a patient with a high-risk occupation (eg, diver, pilot)
 - Patients with acquired immunodeficiency syndrome (AIDS) (often because of extensive underlying necrosis)
 - Unacceptable risk of recurrent pneumothorax for patients with plans for extended stays at remote sites
 - Lymphangiomyomatosis, a condition causing a high risk of pneumothorax.

HEMOTHORAX

Accumulation of blood within the chest, or hemothorax, is a relatively common problem, most often resulting from injury to intrathoracic structures or the chest wall. Hemothorax unrelated to trauma is considerably less common and can result from various causes. Prompt identification and treatment of traumatic hemothorax is an essential part of the care of the injured patient. In cases of hemothorax unrelated to trauma, a careful investigation for the underlying source must be performed while treatment is occurring.

Pathophysiology

Bleeding into the pleural space can occur with virtually any disruption of the tissues of the chest wall and pleura or the intrathoracic structures.

The physiologic response to the development of a hemothorax is manifested in 2 major areas: hemodynamic and respiratory. The degree of hemodynamic response is determined by the amount and rapidity of blood loss.

Normal respiratory movement may be hampered by the space-occupying effect of a large accumulation of blood within the pleural space. In trauma cases, abnormalities of ventilation and oxygenation may result, especially if associated with injuries to the chest wall. In some cases of nontraumatic origin, especially those associated with pneumothorax and a limited amount of bleeding, respiratory symptoms may predominate.

Hemodynamic changes vary depending on the amount of bleeding and the rapidity of blood loss. Blood loss of up to 750 mL in a 70-kg man should cause no significant hemodynamic change. Loss of 750-1500 mL in the same individual will cause the early symptoms of shock, ie, tachycardia, tachypnea, and a decrease in pulse pressure.

Significant signs of shock with signs of poor perfusion occur with loss of blood volume of 30% or more (1500-2000 mL). Because the pleural cavity of a 70-kg man can hold 4 or more liters of blood, exsanguinating hemorrhage can occur without external evidence of blood loss.

Blood occupying the pleural cavity takes up space that the lung would fill in normal respiratory excursion. A large enough collection causes the patient to complain of dyspnea and may produce the clinical finding of tachypnea. The volume of blood required to produce these symptoms in a given individual varies depending on a number of factors, including organs injured, severity of injury, and underlying pulmonary and cardiac reserve.

Dyspnea is a common symptom in cases in which hemothorax develops in an insidious manner, such as those secondary to metastatic disease. Blood loss in such cases is not acute as

to produce a visible hemodynamic response, and dyspnea is often the predominant complaint.

Two pathologic states are associated with the later stages of hemothorax. These include empyema and fibrothorax.

Empyema results from bacterial contamination of the retained hemothorax. If undetected or improperly treated, this can lead to bacteremia and septic shock.

Fibrothorax results when fibrin deposition develops in an organized hemothorax and coats both the parietal and visceral pleural surfaces, trapping the lung. The lung is fixed in position by this adhesive process and is unable to fully expand. Persistent atelectasis of portions of the lung and reduced pulmonary function result from this process.

Classification

I. According to extent:

1. Unilateral.
2. Bilateral.

II. According to degree of hemorrhage:

1. Small (the loss less 10 % of volume of circulating blood).
2. Moderate (loss of 10-20 % of volume of circulating blood).
3. Great (loss of 20-40 % of volume of circulating blood).
4. Total (exceeds 40 % of volume of circulating blood).

III. According to duration of bleeding:

1. With persistent hemorrhage.
2. With the stopped bleeding.

IV. According to the presence of clots in a pleural space:

1. Coagulated.
2. No- coagulated.

V. According to the presence of infection:

1. Not infected.
2. Infected (suppurative).

Symptomatology and clinical course

If hemothorax is the complication of blunt chest trauma, the clinical manifestations depend on the gravity of trauma and degree of hemorrhage. Also hemothorax by itself results in pulmonary compression and shift of mediastinum.

In case of **small hemothorax** clinical manifestations of hemorrhage are slightly expressed or absent at all.

Dyspnea, cough, general malaise and dizziness are obvious in **moderate hemothorax**. The skin is pale. The hemodynamic disturbances – tachycardia and decreased arterial pressure are observed.

The **great and total hemothorax** are associated with extremely grave condition. The patients are troubled with expressed general malaise, dizziness, dyspnea and difficult breathing. In some cases they enter medical hospitals in a terminal state. The skin is sharply pale. The peripheral pulse impaired or absent. Tachycardia, weak cardiac tones, low arterial pressure are obvious.

By percussion the dullness is revealed. On auscultation - the breathing over the site of hemothorax is sharply diminished or is not heard.

The X-ray picture of hemothorax is rather specific. The intensive homogeneous shadow on the side of the lesion with oblique upper contour (Damuaso' line) is observed. The costal sinus does not visualized. In small hemothorax, depending on the degree of intrapleural bleeding, the shadow observed only in the region of sinus. In moderate hemothorax it achieves a scapular angle (on the back surface) or V rib on anterior surface of the chest wall. In great hemothorax this shadow achieves III rib, and total hemothorax characterized by complete shadow of a pleural space, and in some cases – mediastinal shift to the healthy side.



Left side small hemothorax



Left side moderate hemothorax



Right side great hemothorax



Left side total hemothorax

The diagnostic program

1. Complaint and anamnesis of the disease.
2. Physical examination.

Symptoms and physical findings associated with hemothorax in trauma vary widely depending on the amount and rapidity of bleeding, the existence and severity of underlying pulmonary disease, the nature and degree of associated injuries, and the mechanism of injury.

Hemothorax is rarely a solitary finding in blunt trauma. Associated chest wall or pulmonary injuries are nearly always present.

Simple bony injuries consisting of one or multiple rib fractures are the most common blunt chest injuries. A small hemothorax may be associated with even single rib fractures but often remains unnoticed during the physical examination and even after chest radiography. Such small collections rarely need treatment.

Complex chest wall injuries are those in which either 4 or more sequential single rib fractures are present or a flail chest exists. These types of injuries are associated with a significant degree of chest wall damage and often produce large collections of blood within the pleural cavity and substantial respiratory impairment. Pulmonary contusion and pneumothorax are commonly associated injuries. Injuries resulting in laceration of intercostal or internal mammary arteries may produce a hemothorax of significant size and significant hemodynamic compromise. These vessels are the most common source of persistent bleeding from the chest after trauma.

Delayed hemothorax can occur at some interval after blunt chest trauma. In such cases, the initial evaluation, including chest radiography, reveals findings of rib fractures without any accompanying intrathoracic pathology. However, hours to days later, a hemothorax is seen. The mechanism is believed to be either rupture of a trauma-associated chest wall hematoma into the pleural space or displacement of rib fracture edges with eventual disruption of intercostal vessels during respiratory movement or coughing.

Large hemothoraces are usually related to injury of vascular structures. Disruption or laceration of major arterial or venous structures within the chest may result in massive or exsanguinating hemorrhage.

Hemodynamic manifestations associated with massive hemothorax are those of hemorrhagic shock. Symptoms can range from mild to profound, depending on the amount and rate of bleeding into the chest cavity and the nature and severity of associated injuries.

Because a large collection of blood will compress the ipsilateral lung, related respiratory manifestations include tachypnea and, in some cases, hypoxemia.

A variety of physical findings such as bruising, pain, instability or crepitus upon palpation over fractured ribs, chest wall deformity, or paradoxical chest wall movement may lead to the possibility of coexisting hemothorax in cases of blunt chest wall injury. Dullness to

percussion over a portion of the affected hemithorax is often noted and is more commonly found over the more dependent areas of the thorax if the patient is upright. Decreased or absent breath sounds upon auscultation are noted over the area of hemothorax.

3. Chest roentgenograms in 2 planes.

The upright chest radiograph is the ideal primary diagnostic study in the evaluation of hemothorax.

In the normal unscarred pleural space, a hemothorax is noted as a meniscus of fluid blunting the costophrenic angle or diaphragmatic surface and tracking up the pleural margins of the chest wall when viewed on the upright chest x-ray film. This is essentially the same chest radiographic appearance found with any pleural effusion.

In cases in which pleural scarring or symphysis is present, the collection may not be free to occupy the most dependent position within the thorax, but will fill whatever free pleural space is available. This situation may not create the classic appearance of a fluid layer on a chest x-ray film.

As much as 400-500 mL of blood is required to obliterate the costophrenic angle as seen on an upright chest radiograph.

In the acute trauma setting, the portable supine chest radiograph may be the first and only view available from which to make definitive decisions regarding therapy. The presence and size of a hemothorax is much more difficult to evaluate on supine films. As much as 1000 mL of blood may be missed when viewing a portable supine chest x-ray film. Only a general haziness of the affected hemithorax may be noted.

In blunt trauma cases, hemothorax is frequently associated with other chest injuries visible on the chest radiograph, such as rib fractures (see below), pneumothorax, or a widening of the superior mediastinum.

Additional studies such as ultrasonography or CT scan may sometimes be required for identification and quantification of a hemothorax noted on a plain chest radiograph

4. [Thoracentesis](#).
5. Investigation of a pleural content.
6. Test by Revilour-Greguar.
7. General blood analysis.

Hematocrit of pleural fluid. Measurement of the hematocrit is virtually never needed in a patient with a traumatic hemothorax.

This study may be needed for the analysis of a bloody effusion from a nontraumatic cause. In such cases, a pleural effusion with a hematocrit value of more than 50% of that of the circulating hematocrit is considered a hemothorax.

8. Biochemical blood analysis.
9. Determining of the blood group and Rh factor.

Variants of clinical course and complications

The coagulated hemothorax. The patient's late apply for medical aid or major bleeding results in formation of clots in a pleural space, and in some cases all blood, which has accumulated in a pleural space, forms by itself a major entire clot.

Depending on degree of bleeding and, consequently, size of clot, the patients complain of chest pain, which intensifies at respiration, dyspnea, general malaise, and dizziness. As a rule, on 3-5 day the fever to 37,5-38°C is observed.

The physical findings (diminishing and absence of vocal fremitus by palpation, dullness by percussion and sharply diminished or absent breathing by auscultation) suggest the presence of pathological process in a pleural space.

Chest roentgenogram reveals the intensive shadow, sometimes heterogeneous (with enlightenments and multiple levels).

The needle aspiration obtains small amount of a liquid hemolyzed blood and small bloody thrombi (according to inner diameter of the needle).

Suppurative hemothorax. The coagulated hemothorax in overwhelming majority is infected, that results in occurrence of a pleural empyema (clinical manifestations, diagnostics and treatment look in chapter " pleural empyema").

Treatment

A treatment of small hemothorax requires [needle aspiration](#) or [drainage](#) of pleural space and elimination of blood. The manipulation is carried out in VI-VII intercostal spaces in the postaxillary or scapular lines.

Total, great or moderate hemothorax with persistent bleeding (positive test by Revilour-Greguar) requires thoracotomy for liquidation of a bleeding source.

The bleeding wounds of lungs are sewed up by twist suture. If the pleural space contains liquid blood, the surgeon carries out its reinfusion. The clots are removed from pleural space.

Tube thoracostomy drainage

Tube thoracostomy drainage is the primary mode of treatment for hemothorax. In adult patients, large-bore chest tubes, usually 36-42F, should be used to achieve adequate drainage in adults. Smaller-caliber tubes are more likely to occlude. In pediatric patients, chest tube size varies with the size of the child. In patients older than 12 years, the chest tube size used is usually the same as that for adults. In smaller children, a 24-34F chest tube should be used, depending on the size of the child.

Thoracostomy tube placement for hemothorax should ideally be in the sixth or seventh intercostal space at the posterior axillary line. In the supine trauma victim, a common error in chest tube insertion is placement too anteriorly and superiorly, making complete drainage very unlikely.

After tube thoracostomy is performed, a repeat chest radiograph should always be obtained. This helps identify chest tube position, helps determine completeness of the hemothorax evacuation, and may reveal other intrathoracic pathology previously obscured by the hemothorax. If drainage is incomplete as visualized on the postthoracostomy chest radiograph, placement of a second drainage tube should be considered. Preferably, a video-assisted thoracic surgery (VATS) operative procedure should be undertaken to evacuate the pleural space.

In cases of hemopneumothorax, 2 chest tubes may be preferred, with the tube draining the pneumothorax placed in a more superior and anterior position.

Surgical exploration in cases of traumatic hemothorax should be performed in the following circumstances:

Greater than 1000 mL of blood is evacuated immediately after tube thoracostomy. This is considered a massive hemothorax.

Bleeding from the chest continues, defined as 150-200 mL/h for 2-4 hours.

Persistent blood transfusion is required to maintain hemodynamic stability.

In the majority of trauma cases requiring chest exploration, the bleeding source is from

the chest wall, most commonly intercostal or internal mammary arteries. Once identified, these can be easily controlled with suture ligatures in most cases. After control of obvious bleeding and evacuation of clot and blood, a rapid but thorough exploration of the entire chest cavity should be performed.

Unstable rib fractures found at the time of surgery may require some debridement of sharp rib edges to prevent further injury to the lung or adjacent chest wall structures. At some centers, flail segments or extensive rib fractures are stabilized with wires or other types of support in an attempt to improve postoperative chest wall mechanics.

A thoracic surgeon should be present or immediately available at the time of emergency thoracic exploration because control of bleeding from difficult areas such as the hilum of the lung, the heart, or the great vessels may require a surgeon with expertise in that field.

Adequate drainage of the chest after control of bleeding is very important. Because chest drainage tubes are placed under direct vision, the complication of retained hemothorax should occur with extreme infrequency. A minimum of 2 large-bore chest tubes should be used, with one positioned posteriorly and the other positioned anteriorly. Some surgeons prefer the addition of a right-angled chest tube positioned over the diaphragm.

The late sequelae of hemothorax, including residual clot, infected collections, and trapped lung, require additional treatment and, most often, surgical intervention.

SUBCUTANEOUS EMPHYSEMA

The cause of this complication of blunt chest trauma is the damage by edge of the broken rib of parietal and visceral pleural membranes with the following entering of air from a pulmonary tissue into a pleural space and through damaged chest wall (ruptured intercostal muscles) into subcutaneous fat.

In overwhelming majority the subcutaneous emphysema is the outcome of a valvular pneumothorax and pneumothorax in obliterated pleural space.

Classification

Subcutaneous emphysema is divided on:

1. Localized.
2. Widespread.
3. Total.

Symptomatology and clinical course

As the subcutaneous emphysema is the outcome of trauma complicated by a rib fracture and posttraumatic pneumothorax, the chief complaints are of chest pain and dyspnea, which intensify at respiration, movements and minor physical activity.

In **localized subcutaneous emphysema** the patients the complaints of the chest trauma are predominant in symptomatology. On examination observed a swelling of a chest wall in the place of damage. By palpation a subcutaneous crepitation is felt over this region. Percussion reveals a bandbox sound or tympanitis.

Auscultation of lungs over subcutaneous emphysema is usually impossible.

The **widespread and total subcutaneous emphysema** represents a serious moral problem for the patient. Owing to extent of air all over the chest, abdominal wall, neck (wide-spread emphysema), and also face, arms and legs (total emphysema), the patients has a specific appearance: swelling face, thick neck, enlargement of the chest, arms, and legs. Subcutaneous emphysema by itself usually causes no respiratory and cardiovascular disturbances. However the patients note the change of the quality of voice. By palpation the subcutaneous emphysema is felt in whole body ("crisping snow").

It is necessary to note, that in widespread and total emphysema the auscultation is impossible. However the

presence of subcutaneous emphysema at the closed trauma of the chest enables to suspect the presence of posttraumatic pneumothorax.

On the chest roentgenogram the enlightenment of a subcutaneous fat (presence of air) is observed.



Subcutaneous emphysema

The diagnostic program

1. Complaint and history of the disease.
2. Physical findings.
3. Chest X-radiography.

Treatment

Widespread and total subcutaneous emphysema requires the draining of subcutaneous space by plastic tubes in infra- and supraclavicular region, and also in the zone of the most expressed emphysema. Also performed the drainage of a pleural space.

The subcutaneous emphysema resolves depending on its extent from several days to 2-3,5 weeks.

TRAUMATIC INJURY OF TRACHEA AND MAIN BRONCHI

The isolated injuries of trachea and bronchi as the result of blunt trauma of the chest occur rarely and located mainly in a cervical part.

The main causes of tearing of trachea and bronchi are:

- 1) shearing forces, which arises at the moment of trauma owing to a sudden rise of intraluminal pressure against a closed glottis when the airway is compressed against the spine.;
- 2) compressing of a bronchial tree between a breastbone and vertebral column;
- 3) shift of lungs in sudden and rapid deceleration or acceleration of a body occurs with greater amplitude, than fixed bifurcation of trachea.

Such disruptions most often occurs as a result of vehicular impacts, falls from great heights, direct blows to the chest. In most cases disruption of trachea and bronchi are accompanied with the other visceral damages: lungs, skull and brain, heart, liver and flail chest.

Classification

I. According to degree of disruption:

1. Partial:
 - without damage of cartilaginous rings (I degree);
 - with fracture of cartilaginous rings (II degree).
2. Partial disruption of all layers (III degree).
3. Complete transverse disruption of all walls without disjunction of the of trachea, (bronchus) (IV degree).
4. Abruption with disjunction of the edges of trachea (bronchus) (V degree).

II. According to direction of rupture:

1. Longitudinal.
2. Oblique.

3. Transversal.

4. Mixed.

III. According to localization of the damage:

1. Tracheo-laryngeal.

2. Cervico-tracheal.

3. Mediastino-bronchial.

4. Bifurcational.

5. Bronchial.

IV. According to the size of injury:

1. Combined damages of trachea (bronchi) and adjacent organs.

2. Damage of trachea (bronchi) and other segments of the body.

3. Damage of a trachea (bronchi). adjacent organs and other segments of the body.

Symptomatology and clinical course

The clinical manifestations of the injury of trachea depend on the type of disruption, its degree and presence of concomitant damages.

Incomplete isolated disruption of trachea commonly manifests by cough and hemoptysis. Respiration is not disturbed as a rule.

The small disruptions are characterized by various clinics. If the hole is occluded by clot and mediastinal tissues, the signs, which had appeared earlier (cough, hemoptysis, mediastinal emphysema), can disappear. Nevertheless the repeated occurrence of cough, as a rule, leads to severe aggravation of the patient state.

Major and circular disruptions of trachea cause a grave state of the patients. They manifest except difficult breathing by such signs:

- 1) mediastinal emphysema or pneumothorax;
- 2) compression syndrome – compression and inflection of major vessels due to tension pneumothorax or mediastinal emphysema with transmission into acute cardiopulmonary failure;

- 3) hemorrhage syndrome;
- 4) aspiration syndrome, which is the outcome of bleeding into airways or aspirations of the gastric content;
- 5) traumatic shock.

The injuries of bronchi occur in the way of abruption of main bronchi or their disruption in the zone of bifurcation. In the zone of a tracheal bifurcation observed multiple (2-4) disruptions, which can be longitudinal, transversal or oblique.

Depending on the character of trauma, it is necessary to distinguish direct and secondary disruptions of bronchi. The direct injuries arise from the gunshot and knife wounds, penetration of rib fragments or other subjects in mediastinum or endoscopic manipulations.

The overwhelming majority of bronchial disruption is the part of blunt trauma of the chest. By the way, the damage of vessels of a lung root occurs in 41,3 %.

The predominant clinical signs of a bronchial disruption are the respiratory disturbance, gas syndrome, hemothysis and hemothorax. However these signs may be observed only in isolated injuries of lungs.

The patients state is grave. Rest dyspnea and acute pain behind a breastbone are the most troubling manifestations. The difficult swallowing, hoarseness, swelling face and subcutaneous crepitation are observed. Auscultatory on the side of trauma the breathing sounds are weak or absent at all.

The sequence of examination of the patients with injuries of trachea and bronchi depends on the character and gravity of trauma, clinical signs and concomitant damages, which threatening life.

If the state of the patient allows, a chest X-radiography is performed. Commonly it is possible to find out mediastinal emphysema, sometimes the sign of discontinuing of trachea.

The injuries of bronchi manifest by the distension of mediastinum and presence of air strips along its borders, and in some cases total or tension pneumothorax observed.

Final and most informative diagnostic method is the tracheobronchoscopy, which can be also the therapeutic method. However it is necessary to carry out decompression of a mediastinal emphysema and pneumothorax before such investigation.

Before examination the clots and liquid blood are aspirated from airways, then adjusted the localization and character of disruption. The incomplete disruptions are usually longitudinal and oblique and located on the line of membranous and cartilaginous part, circular – mainly in a cervical part of trachea. Except disruption of the wall, observed the absence of cartilaginous rings in this region and filled by blood parabronchial fat.

The open damages of trachea take place mainly in a cervical part and rarely - in thoracic. In all cases of neck

trauma it is necessary always suspect the opportunity of damage of trachea and esophagus.

Such variants of clinical course are distinguished:

- acute course (first 30 days after operation);
- chronic course (complication of trauma).

Acute course is divided onto three stages:

1. The initial stage (lasts during 2 days after the trauma with typical signs of disruption; the urgent resuscitation measures are required).
2. The stage of temporary compensation (lasts during 2 weeks; at this time it is possible to carry out diagnostic examination).
3. The stage of persistent compensation (lasts during 30 days; during this time a stenosis and other persistent complications of disruption of trachea and bronchi develops).

The diagnostic program

1. Complaint and the history of disease.
2. Physical findings.
3. Chest X-radiography.
4. Diagnostic [thoracentesis](#).
5. General blood and urine analyses.
6. Biochemical blood analysis.
7. Tracheobronchoscopy.
8. Tomography.

Treatment

There are primary operations in acute stage (first two days after the trauma) and late repairing operations (in 1 month after the trauma). The operation is based in resection of injured tissues, edges of bronchus with the further suturing of disruption, or wedge-like or circular resection with following anastomosis. In series of

cases lobe-, bilobe- or pneumonectomy is performed.

MEDIASTINAL EMPHYSEMA

Mediastinal emphysema is the complication of the blunt trauma of the chest, which is characterized by entering and accumulation of air in mediastinum.

The causes of mediastinal emphysema is partial (damage of a membranous part) or complete disruptions of trachea, bronchi, esophagus and in some cases – tension pneumothorax.

The entry of air in mediastinum leads to compressing of superior cava vein and right atrium, which results in the expressed discirculation.

Symptomatology and clinical course

The patients complain of difficult breathing and swallowing, pain behind breastbone, hoarseness, cough attacks. As a rule, the patient's position is forced – semi-sitting. The neck and face are thickened, cervical veins distended, the skin is cyanotic. By palpation – the crepitation of neck, face, and shoulder area. By auscultation heart tones are diminished with tachycardia.

On X-ray film on the background of enlightenment observed well-defined contour of a mediastinal pleura. If there is the damage of mediastinal pleural membrane a pneumothorax (mainly total or intense) is revealed.



Mediastinal emphysema with left-side pneumothorax

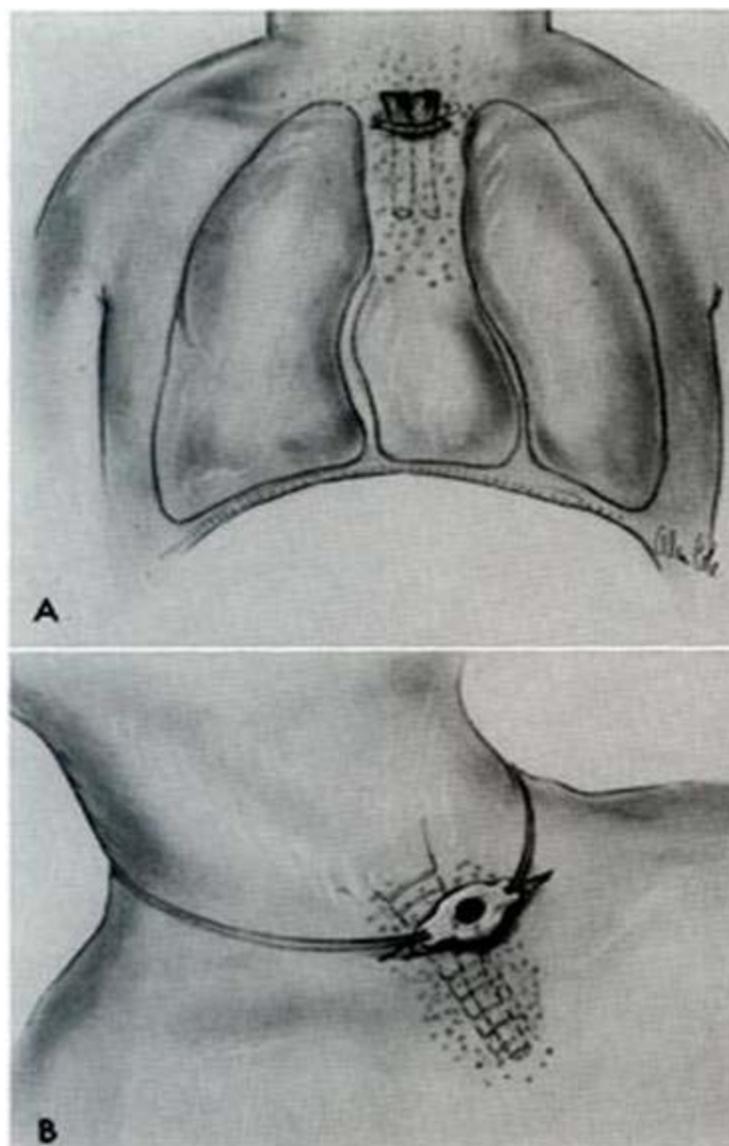
The diagnostic program

1. Complaint and history of the disease.
2. Physical findings.
3. Examination X-radiography of organs of the chest.
4. Definition of central venous pressure.
5. Control of hemodynamic data.
6. ECG.

Tactics and choice of treatment

Progressing mediastinal emphysema requires the urgent drainage of a forward mediastinum in order to prevent external cardiac tamponade.

Tension mediastinal emphysema resulting from disruption of trachea or bronchus operative management, pneumothorax, etc., requires the treatment, which is described in previous chapters.



Drainage of anterior mediastinum

SHOCK

Circulatory shock, commonly known as just shock, is a serious, life-threatening medical condition where insufficient blood flow reaches the body tissues. As the blood carries oxygen and nutrients around the body, reduced flow hinders the delivery of these components to the tissues, and can stop the tissues from functioning properly. The process of blood entering the tissues is called perfusion, so when perfusion is not occurring properly this is called a hypoperfusional (hypo = below) state.

Circulatory shock should not be confused with the emotional state of shock, as the two are not related. Medical shock is a life-threatening medical emergency and one of the most common causes of death for critically-ill people. Shock can have a variety of effects, all with similar outcomes, but all relate to a problem with the body's circulatory system. For example,

shock may lead to hypoxemia (a lack of oxygen in arterial blood) or cardiac arrest (the heart stopping)

Fig. Classes of hemorrhagic shock

- **Class I hemorrhage (loss of 0-15%)**
 - Little tachycardia
 - Usually no significant change in BP, pulse pressure, respiratory rate
- **Class II hemorrhage (loss of 15-30%)**
 - HR >100 beats per minute, tachypnea, decreased pulse pressure
- **Class III hemorrhage (loss of 30-40%)**
 - Marked tachycardia and tachypnea, decreased systolic BP, oliguria
- **Class IV hemorrhage (loss of >40%)**
 - Marked tachycardia and decreased systolic BP, narrowed pulse pressure, markedly decreased (or no) urinary output
 - Immediately life threatening

Effects of inadequate perfusion on cell function.

There are four stages of shock. As it is a complex and continuous condition there is no sudden transition from one stage to the next.

Initial

During this stage, the hypoperfused state causes hypoxia, leading to the mitochondria being unable to produce adenosine triphosphate (ATP). Due to this lack of oxygen, the cell membranes become damaged, they become leaky to extra-cellular fluid, and the cells perform anaerobic respiration. This causes a build-up of lactic and pyruvic acid which results in systemic metabolic acidosis. The process of removing these compounds from the cells by the liver requires oxygen, which is absent.

Compensatory (Compensating)

This stage is characterised by the body employing physiological mechanisms, including neural, hormonal and bio-chemical mechanisms in an attempt to reverse the condition. As a result of the acidosis, the person will begin to hyperventilate in order to rid the body of carbon dioxide (CO₂). CO₂ indirectly acts to acidify the blood and by removing it the body is attempting to raise the pH of the blood. The baroreceptors in the arteries detect the resulting hypotension, and cause the release of adrenaline and noradrenaline.

- Baroreceptor reflexes
- Circulating vasoconstrictors
- Chemoreceptor reflexes
- Reabsorption of tissue fluids
- Renal reabsorption of sodium and water
- Activation of thirst mechanisms
- Cerebral ischemia
- Hemapoiesis

Fig. Compensatory mechanisms

Noradrenaline causes predominately vasoconstriction with a mild increase in heart rate, whereas adrenaline predominately causes an increase in heart rate with a small effect on the vascular tone; the combined effect results in an increase in blood pressure. Renin-angiotensin axis is activated and arginine vasopressin is released to conserve fluid via the kidneys.

Also, these hormones cause the vasoconstriction of the kidneys, gastrointestinal tract, and other organs to divert blood to the heart, lungs and brain. The lack of blood to the renal system causes the characteristic low urine production. However the effects of the Renin-angiotensin axis take time and are of little importance to the immediate homeostatic mediation of shock .

Progressive (Decompensating)

Should the cause of the crisis not be successfully treated, the shock will proceed to the progressive stage and the compensatory mechanisms begin to fail. Due to the decreased perfusion of the cells, sodium ions build up within while potassium ions leak out. As anaerobic metabolism continues, increasing the body's metabolic acidosis, the arteriolar smooth muscle and precapillary sphincters relax such that blood remains in the capillaries[1]. Due to this, the hydrostatic pressure will increase and, combined with histamine release, this will lead to leakage of fluid and protein into the surrounding tissues. As this fluid is lost, the blood concentration and viscosity increase, causing sludging of the micro-circulation. The prolonged vasoconstriction will also cause the vital organs to be compromised due to reduced

perfusion[1]. If the bowel becomes sufficiently ischemic, bacteria may enter the blood stream, resulting in the increased complication of endotoxic shock[1].

Refractory (Irreversible)

At this stage, the vital organs have failed and the shock can no longer be reversed. Brain damage and cell death have occurred. Death will occur imminently.

A medical emergency is an injury or illness that is acute and poses an immediate risk to a person's life or long term health. These emergencies may require assistance from another person, who should ideally be suitably qualified to do so, although some of these emergencies can be dealt with by the victim themselves. Dependent on the severity of the emergency, and the quality of any treatment given, it may require the involvement of multiple levels of care, from a first aider to an emergency physician through to specialist surgeons.

Any response to an emergency medical situation will depend strongly on the situation, the patient involved and availability of resources to help them. It will also vary depending on whether the emergency occurs whilst in hospital under medical care, or outside of medical care (for instance, in the street or alone at home).

For emergencies starting outside of medical care, a key component of providing proper care is to summon the emergency medical services (usually an ambulance), by calling for help using the appropriate local emergency telephone number, such as 999, 911, 112 or 000 or 111. After determining that the incident is a medical emergency (as opposed to, for example, a police call), the emergency dispatchers will generally run through a questioning system such as AMPDS in order to assess the priority level of the call, along with the caller's name and location.

Those trained to perform first aid can act within the bounds of the knowledge they have, whilst awaiting the next level of definitive care. Those who are not able to perform first aid can also assist by remaining calm and staying with the injured or ill person. A common complaint of emergency service personnel is the propensity of people to crowd around the scene of victim, as it is generally unhelpful, making the patient more stressed, and obstructing the smooth working of the emergency services. If possible, first responders should designate a specific person to ensure that the emergency services are called. Another bystander should be sent to wait for their arrival and direct them to the proper location. Additional bystanders can be helpful in ensuring that crowds are moved away from the ill or injured patient, allowing the responder adequate space to work.

Many states of the USA have "Good Samaritan Laws" which protect civilian responders who choose to assist in an emergency. Responders acting within the scope of their knowledge

and training as a "reasonable person" in the same situation would act are often immune to liability in emergency situations. Usually, once care has begun, a first responder or first aid provider may not leave the patient or terminate care until a responder of equal or higher training (e.g., fire department or emergency medical technicians) assumes care. This can constitute abandonment of the patient, and may subject the responder to legal liability. Care must be continued until the patient is transferred to a higher level of care, the situation becomes too unsafe to continue, or the responder is physically unable to continue due to exhaustion or hazards.

The principles of the chain of survival apply to medical emergencies where the patient has an absence of breathing and heartbeat. This involves the four stages of Early access, Early CPR, Early defibrillation and Early advanced life support

Unless the situation is particularly hazardous, and is likely to further endanger the patient, evacuating an injured victim requires special skills, and should be left to the professionals of the emergency medical and fire service.

Within hospital settings, an adequate staff is generally present to deal with the average emergency situation. Emergency medicine physicians have training to deal with most medical emergencies, and maintain CPR and ACLS certifications. In disasters or complex emergencies, most hospitals have protocols to summon on-site and off-site staff rapidly.

Both emergency room and inpatient medical emergencies follow the basic protocol of Advanced Cardiac Life Support. Irrespective of the nature of the emergency, adequate blood pressure and oxygenation are required before the cause of the emergency can be eliminated. Possible exceptions include the clamping of arteries in severe hemorrhage.

While the golden hour (medicine) is a trauma treatment concept, two emergency medical conditions have well-documented time-critical treatment considerations: stroke and myocardial infarction (heart attack). In the case of stroke, there is a window of three hours within which the benefit of clot-busting drugs outweighs the risk of major bleeding. In the case of a heart attack, rapid stabilization of fatal arrhythmias can prevent sudden cardiac death. In addition, there is a direct relationship between time-to-treatment and the success of reperfusion (restoration of blood flow to the heart), including a time dependent reduction in the mortality and morbidity. In 1972 Hinshaw and Cox suggested the following classification which is still used today.[2] It uses four types of shock: hypovolemic, cardiogenic, distributive and obstructive shock:[3][4][5][8][10]

Hypovolemic shock – This is the most common type of shock and based on insufficient circulating volume. Its primary cause is loss of fluid from the circulation from either an internal

or external source. An internal source may be haemorrhage. External causes may include extensive bleeding, high output fistulae or severe burns.

DIAPHRAGMATIC HERNIA. DISEASE OF MEDIASTINUM

DIAPHRAGMATIC HERNIAS

Diaphragmatic hernia represents herniation of abdominal organs through natural openings of diaphragm, its weak places or ruptures.

A hiatal hernia occurs when a portion of the stomach prolapses through the diaphragmatic esophageal hiatus. Although the existence of hiatal hernia has been described in earlier medical literature, it has come under scrutiny only in the last century or so because of its association with gastroesophageal reflux disease (GERD) and its complications. There is also an association between obesity and the presence of hiatal hernia. By far, most hiatal hernias are asymptomatic and are discovered incidentally. On rare occasion, a life-threatening complication, such as gastric volvulus or strangulation, may present acutely.

Etiology and pathogenesis

The cause of occurrence of congenital hernia is the disturbance of embryogenesis with transformation in anomaly of diaphragm. The acquired diaphragmatic hernia more often arise owing to age-dependent involution of diaphragm, its ptosis in the people with a mainly sedentary mode of life, increase of intraperitoneal pressure, obesity, cough, overfeeding, constipation, meteorism and pregnancy. The cause of sliding hernias can be draw of esophagus upward in reflux esophagitis owing to intensive contraction of its longitudinal musculature.

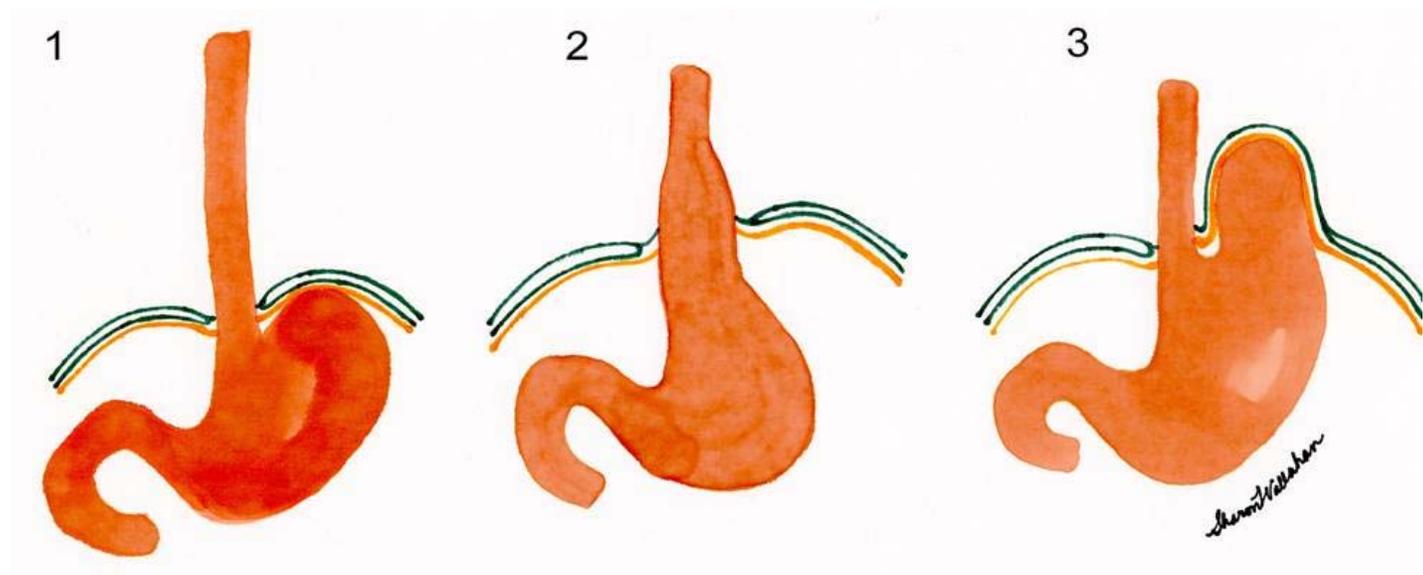
Predisposing factors include the following:

- Muscle weakening and loss of elasticity as people age is thought to predispose to hiatus hernia, based on the increasing prevalence in older people. With decreasing tissue elasticity, the gastric cardia may not return to its normal position below the diaphragmatic hiatus following a normal swallow. Loss of muscle tone around the diaphragmatic opening also may make it more patulous.
- Hiatal hernias are more common in women. This may relate to the intra-abdominal forces exerted in pregnancy.
- Burkitt et al suggest that the Western, fiber-depleted diet leads to a state of chronic constipation and straining during bowel movement, which might explain the higher incidence of this condition in Western countries.
- Obesity predisposes to hiatus hernia because of increased abdominal pressure.
- Conditions such as chronic esophagitis may cause shortening of the esophagus by causing fibrosis of the longitudinal muscles and, therefore, predispose to hiatal hernia. However, which comes first, the hiatal hernia worsening the reflux or the reflux-induced shortening of the esophagus, remains unknown.
- The presence of abdominal ascites also is associated with hiatal hernias.

Diaphragmatic hernias may be congenital or acquired. Acquired hiatal hernias are divided further into nontraumatic and traumatic hernias. The most common types of hernias are those acquired in a nontraumatic fashion. Hernias acquired in a nontraumatic fashion are divided into 2 types, (1) sliding hiatal hernia and (2) paraesophageal hiatal hernia. A mixed variety with coexisting sliding and paraesophageal components is possible.

- Sliding hiatal hernia by far is the most common type of hiatal hernia. It occurs when the gastroesophageal junction, along with a portion of the stomach, migrates into the mediastinum through the esophageal hiatus (see the image below). The majority of patients with demonstrated hiatal hernias are asymptomatic. This type of hernia interferes with the reflux barrier mechanism in several ways. As the LES moves into the chest, it no longer is exposed to positive intra-abdominal pressure and, therefore, is less effective as a sphincter. In fact, the sphincter moves into an area of low pressure, which interferes with the sphincter activity. In addition, the widening hiatus affects the competence of the diaphragmatic crura. The angle of His is lost, making regurgitation of gastric contents more likely. These changes not only predispose to reflux of gastric contents into the esophagus, but also prolong the acid contact time with the epithelium of the esophagus.

In paraesophageal hernia, also called rolling-type hiatal hernia, the widened hiatus permits the fundus of the stomach to protrude into the chest, anterior and lateral to the body of the esophagus; however, the gastroesophageal junction remains below the diaphragm (see Figure 3 of the image above). This causes the stomach to rotate in a counter-clockwise direction. As the hiatus widens, increasing amounts of the greater curvature of the stomach and, sometimes, the gastric-colic omentum, follow. The fundus eventually comes to lie above the gastroesophageal junction, with the pylorus being pulled towards the diaphragmatic hiatus. In this type of hernia, the anatomic relation of the stomach to the lower end of the esophagus (angle of His) tends to remain unchanged, so gross acid reflux does not occur.



1– norm, 2 – sliding hiatal hernia, 3 – paraesophageal hernia

Pathology

The esophagus passes through the diaphragmatic hiatus in the crural part of the diaphragm to reach the stomach. The diaphragmatic hiatus itself is approximately 2 cm in length and chiefly consists of musculotendinous slips of the right and left diaphragmatic crura arising from either side of the spine and passing around the esophagus before inserting into the central tendon of the diaphragm. The size of the hiatus is not fixed, but narrows whenever intra-abdominal pressure rises, such as when lifting weights or coughing.

The lower esophageal sphincter (LES) is an area of smooth muscle approximately 2.5-4.5 cm in length. The upper part of the sphincter normally lies within the diaphragmatic hiatus, while the lower section normally is intra-abdominal. At this level, the visceral peritoneum and the phrenoesophageal ligament cover the esophagus. The phrenoesophageal ligament is a fibrous layer of connective tissue arising from the crura, and it maintains the LES within the abdominal cavity. The A-ring is an indentation sometimes seen on barium studies, and it marks the upper part of the LES. Just below this is a slightly dilated part of the esophagus, forming the vestibule. A second ring, the B-ring, may be seen just distal to the vestibule, and it approximates the Z-line or squamocolumnar junction. The presence of a B-ring confirms the diagnosis of a hiatal hernia. Occasionally, the B-ring also is called the Schatzki ring.

Any sudden increase in intra-abdominal pressure also acts on the portion of the LES below the diaphragm to increase the sphincter pressure. An acute angle, the angle of His, is formed between the cardia of the stomach and the distal esophagus and functions as a flap at the gastroesophageal junction and helps prevent reflux of gastric contents into the esophagus (see the image below).

The gastroesophageal junction acts as a barrier to prevent reflux of contents from the stomach into the esophagus by a

combination of mechanisms forming the antireflux barrier. The components of this barrier include the diaphragmatic crura, the LES baseline pressure and intra-abdominal segment, and the angle of His. The presence of a hiatal hernia compromises this reflux barrier not only in terms of reduced LES pressure but also reduced esophageal acid clearance. Patients with hiatal hernias also have longer transient LES relaxation episodes particularly at night time. These factors increase the esophageal mucosa acid contact time predisposing to esophagitis and related complications.

Frequency

Hiatal hernias are more common in Western countries. The frequency of hiatus hernia increases with age, from 10% in patients younger than 40 years to 70% in patients older than 70 years.

Paraesophageal hernias generally tend to enlarge with time, and sometimes the entire stomach is found within the chest. The risk of these hernias becoming incarcerated, leading to strangulation or perforation, is approximately 5%. This complication is potentially lethal, and surgical intervention is necessary. Because of the high mortality associated with this condition, elective repair often is advised wherever a paraesophageal hernia is found.

Classification

There are such types of hernia:

- 1) congenital;
- 2) acquired;
- 3) posttraumatic;
- 4) true;
- 5) false.

A. Diaphragmatic hernia.

I. [Sliding](#) (axial) diaphragmatic hernia:

- 1) esophageal;
- 2) cardial;
- 3) cardiofundal.

II. Diaphragmatic hernia of [paraesophageal](#) type:

- 1) fundal;
- 2) antral;
- 3) intestinal (small and large intestine);
- 4) combined intestinal-gastric hernias;
- 5) epiploic.

III. Huge diaphragmatic hernia:

- 1) subtotal gastric;
- 2) total gastric.

IV. A short esophagus:

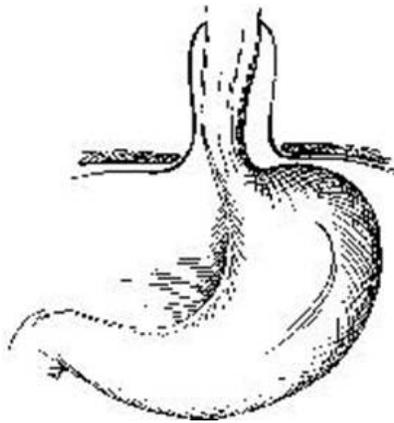
- 1) acquired short esophagus;
- 2) congenital short esophagus (thoracic stomach).

B. Parasternal hernias:

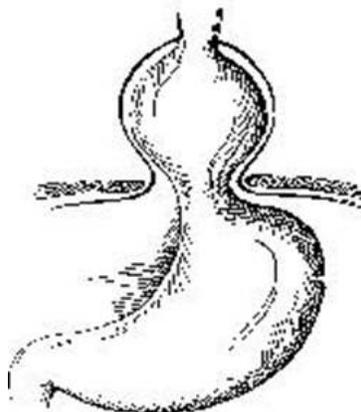
- 1) retrosternal;
- 2) retrocostosternal.

C. Lumbocostal diaphragmatic hernias.

D. Hernia of atypical localization.

Esophageal hernias:

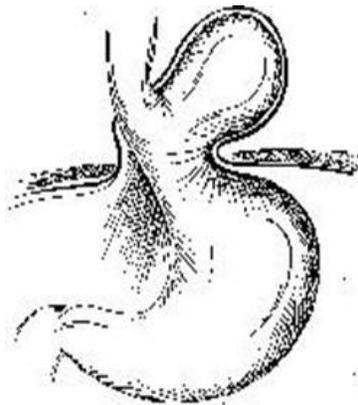
Esophageal



Cardiofundal



Mixed paraesophageal



Paraesophageal fundal

Symptomatology and clinical course

The predominant manifestations resulting from sliding diaphragmatic hernia (about 90 % of diaphragmatic hernias) are the signs of gastroesophageal reflux. It is characterized by the **pain behind breastbone** or epigastric region. It more often appears in supine position after meal or after intensive physical exertion.

Heartburn is the second according to the frequency sign and caused by the injury of esophageal mucosa by gastric juice as a result in turn of gastroesophageal reflux.

Belching by air, as a rule, observed, which commonly results in pain relief and decrease of arching feeling in epigastric region.

Regurgitation arises owing to gastroesophageal reflux, which reaches pharynx and oral cavity. More often observed regurgitation by gastric acid or bitter liquid or food.

The sign of **"lacing shoes"** is expressed when the patient bends down after liquid food, and the latter is partially poured out into the mouth. It is caused by incompetence of the lower esophageal sphincter (gastroesophageal junction).

Nausea and vomiting are rare. The latter some patients cause by themselves to achieve some relief.

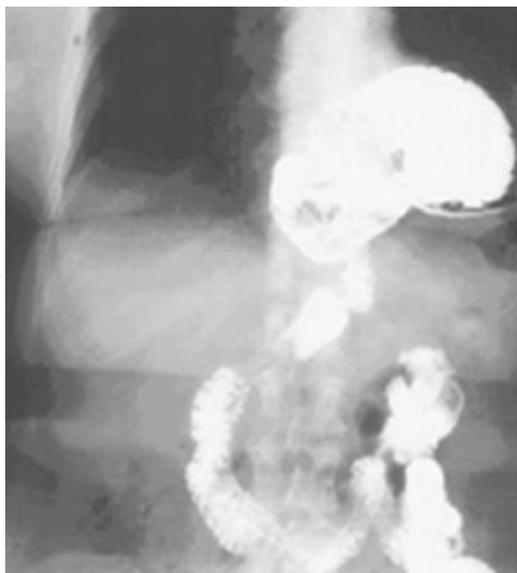
Dysphagia is rarely observed. More often it is the outcome of complications of diaphragmatic hernia (esophageal stricture, malignancy).

Roentgenological signs: 1) the sign of "bell"; 2) blunt His angle; 3) lack of air bubble of the stomach.



Sliding diaphragmatic hernia

The clinical manifestations of paraesophageal, retrosternal or lumbocostal hernias basically depend on the character of organs, which the hernial sac contents, and their compressing by hernial ring. Sometimes the clinical course even of major hernias is asymptomatic, and they are occasionally found out during X-ray examinations. For the first time the disease can manifest under the influence of physical exertion, trauma, pregnancy, labors etc.



Paraesophageal hernia

Variants of clinical course and complications

The sliding hiatal hernia commonly has typical clinical course and rather rich symptomatology, which enable to establish the diagnosis with a great degree of probability. Nevertheless occasionally gastroesophageal reflux as the sequel of a sliding hiatal hernia can result in misdiagnostics (stenocardia, acute cholelithiasis etc.).

The most often complications of sliding diaphragmatic hernia are gastric bleeding, peptic stricture of esophagus and malignancy.

The causes of the bleeding can be erosion and ulcers of stomach, which result from compression of the organ in esophageal hiatus. More often observed small bleeding, but at long-stand recurrent course they result in chronic anemia. The profuse bleeding arise rarely. The strangulation of a sliding diaphragmatic hernia never occurs.

Nevertheless for diaphragmatic hernias of other locations the most dangerous complication is naturally strangulation. Such pathology manifests by the signs of s strangulation intestinal obstruction. However the correct diagnosis frequently possible to establish only during operation.

The diagnostic program

1. Anamnesis and physical findings.

Hiatal hernias are relatively common and, in themselves, do not cause symptoms. For this reason, most people with hiatal hernias are asymptomatic. Hiatal hernias may predispose to reflux or worsen existing reflux in a minority of individuals. Physicians should resist the temptation to label hiatal hernia as a disease.

Patients can have reflux without a demonstrable hiatal hernia. When a hernia is present in a patient with symptomatic GERD, the hernia may worsen symptoms for several reasons, including the hiatal hernia acting as a fluid trap for gastric reflux and increasing the acid contact time in the esophagus. In addition, with a hiatal hernia, episodes of transient relaxation of the LES are more frequent and the length of the high-pressure zone is reduced. The main symptoms of a sliding hiatal hernia are those associated with reflux and its complications.

No clear correlation exists between the size of a hiatal hernia and the severity of the symptoms. A very large hiatal hernia may be present with no symptoms at all. Some complications are specific for a hiatal hernia.

- Esophageal complications
 - By far, the majority of hiatal hernias are asymptomatic.
 - Often, patients are left with the impression that they have a disease when a hiatal hernia is diagnosed.
 - In rare cases, however, a hiatal hernia may be responsible for intermittent bleeding from associated esophagitis, erosions (Cameron ulcers), or a discrete esophageal ulcer, leading to iron-deficiency anemia. The prevalence of large hiatal hernias in patients with iron deficiency anemia is 6-7%. This particular complication is more likely in patients who are bed-bound or those who take nonsteroidal anti-inflammatory drugs. Massive bleeding is rare.
- Nonesophageal complications
 - Incarceration of a hiatal hernia is rare and is observed only with paraesophageal hernia.
 - When this occurs, it can present abruptly, with a sudden onset of vomiting and pain, sometimes requiring immediate operative intervention.

2. X-radiography of chest and abdomen.

Although a chest radiograph may reveal a large hiatal hernia (see the first image below), and many incidentally diagnosed hiatal hernias are discovered in this manner, a barium study of the esophagus helps establish the diagnosis with greater accuracy (see the second image below).

Typical findings include an outpouching of barium at the lower end of the esophagus, a wide hiatus through which gastric folds are seen in continuum with those in the stomach, and, occasionally, free reflux of barium.

A barium study helps distinguish a sliding from a paraesophageal hernia (see the images below).

In rare cases, the entire stomach may herniate into the chest

The stomach may then undergo volvulus (see the image below) and subsequent incarceration and strangulation.

3. Esophagogastrosocopy with biopsy and histological investigation.

Hiatal hernia is diagnosed easily using upper gastrointestinal endoscopy.

The diagnosis of a hiatal hernia actually is incidental, and endoscopy is used to diagnose complications such as erosive

esophagitis, ulcers in the hiatal hernia, Barrett esophagus, or tumor.

A hiatal hernia is confirmed when the endoscope is about to enter the stomach or on retrograde view once inside the stomach (see the image below). If any doubt remains, the patient may be asked to sniff through the nose, which causes the diaphragmatic crura to approximate, seen as a pinch, closing the lumen.

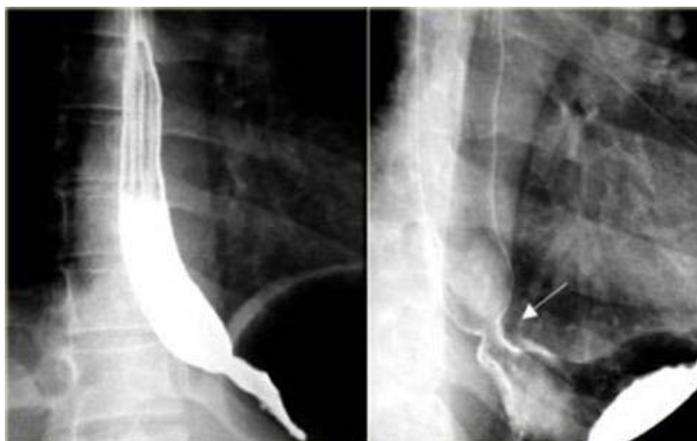
Endoscopy also permits biopsy of any abnormal or suspicious area.

Esophageal manometry has a low sensitivity for diagnosing hiatal hernia, as compared to endoscopy, and is therefore not appropriate in helping to establish a diagnosis.

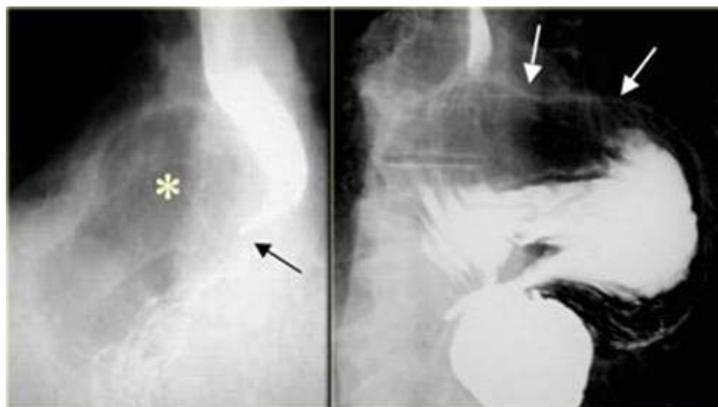
4. Contrast X-radiography of esophagus and stomach in three positions: upward, supine and upside-down position.

5. General blood and urine analyses.

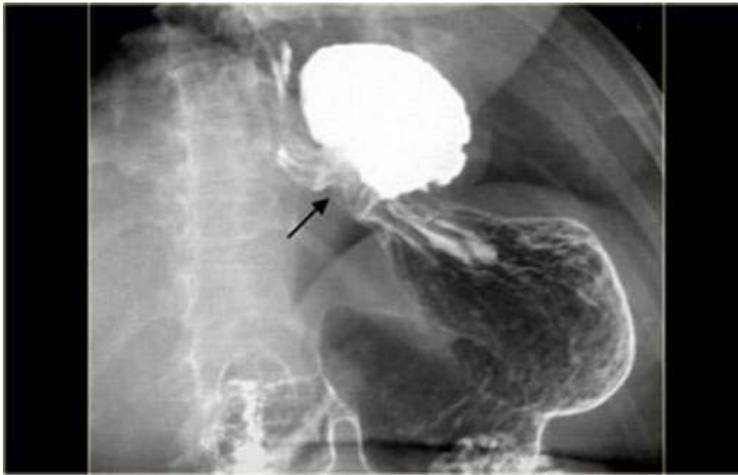
6. Coagulogram.



Sliding hernia



Paraesophageal hernia



Mixed hernia

Differential diagnostics

Stenocardia. Diaphragmatic hernias frequently cause the pain, which character not only the patient, but also doctor can identify as anginal. However in diaphragmatic hernia the pain more often is vague, spread to the stomach region and depends on body position. The pain, as a rule, arises in supine position and disappears, if the patient upward. More often it spreads to the right and anginal vice versa to the left. In diaphragmatic hernia the ECG can manifest the coronary failure, nevertheless standing up, owing to the stop of strangulation leads to disappearance of these pathological sings. The pain caused by diaphragmatic hernia does not relieve after nitroglycerin. In this case more effective and prompt is atropine.

Peptic ulcer. The pain in gastric and duodenal ulcer frequently localized in epigastric region with irradiation in the left or right hypochondrium. Nevertheless, it is characterized by periodicity, which caused by meal and disappears after the usage of soda.

Lung atelectasis, pleurisy, pneumonia are also should be differentiated with diaphragmatic hernia. Thus it is always necessary to remember, that the extrapulmonary shadow of supradiaphragmatic disposed hernia on a plain roentgenogram can resemble intrapulmonary. For correct diagnosis it is possible to recommend polypositional X-radiography, contrast roentgenography of esophagus and stomach.

Hypochromic anemia frequently associated due to repeated or permanent small bleedings. They are caused by a regional destruction a gastric mucosa. In the females of senior age if it is fail to explain genesis of the revealed anemia, it is necessary to think about the opportunity of diaphragmatic hernia and carry out appropriate X-ray examination.

Tactics and choice of treatment

The medical tactics toward diaphragmatic hernias of different localization essentially differs.

In case of sliding hiatal hernia the method of a choice is the **conservative therapy**:

- 1) the diet the same, as in peptic ulcer;

An appropriate diet maintains an ideal body mass index. Obesity predisposes to reflux disease. Burkitt et al suggest that the Western, fiber-depleted diet leads to a state of chronic constipation and straining during bowel movement, which would explain the higher incidence of this condition in Western countries.

- 2) position of the patient during sleeping – with elevated upside, during exacerbation – sedentary;
- 3) suppression of gastric secretion by administering of H₂-blockers;
- 4) neutralization of gastric acid;

- 5) intensifying of evacuation of the food from stomach;
- 6) avoidance of constipation;
- 7) anesthetics and sedative agents.

The indication for surgical treatment of sliding diaphragmatic hernia is the considerable expression of clinical signs, diminish of patient's working capacity, fail of conservative treatment, bleeding, peptic stricture, malignancy.

When hiatal hernias are symptomatic, acid reflux usually produces the symptoms. If the hernia itself is causing chest discomfort or other symptoms, surgery may be necessary.

When symptoms are due to GERD, the goals of treatment include prevention of reflux of gastric contents, improved esophageal clearance, and reduction in acid production. This is achieved in the majority of patients by a combination of the following:

- Modifying lifestyle factors
- Neutralizing acid or inhibiting acid production
- Enhancing esophageal and gastric motility

The treatment of GERD is beyond the scope of this article and is discussed in Gastroesophageal Reflux Disease.

Large hiatal hernias may cause iron deficiency anemia regardless of whether Cameron ulcers are present. This anemia responds well to PPI therapy with surgery offering no clear advantage over medical therapy.

Surgical treatment. Upper median laparotomy is mainly used. Nevertheless some surgeons prefer transthoracic accesses.

A patient with a large hiatal hernia may experience vague intermittent chest discomfort or pain. The paraesophageal hernia may strangulate and frequently is operated on prophylactically to prevent this complication. Paraesophageal hernias may present in infants or adults as a potentially life-threatening complication of strangulation, and prompt surgical repair is key. When found in asymptomatic individuals, laparoscopic repair is often undertaken, with large defects in the diaphragm being closed with mesh.

Surgery is necessary only in the minority of patients with complications of GERD despite aggressive treatment with proton pump inhibitors (PPIs). Because only a minority of patients with hiatal hernia have any problems, this represents a very small proportion of patients with sliding hiatal hernia; most patients with problems are managed medically.

By far, the majority of patients who would have undergone surgery in the past are managed successfully today with PPIs. However, young patients with severe or recurrent complications of GERD, such as strictures, ulcers, and bleeding, who cannot afford lifelong PPI treatment or would prefer to avoid taking medications long term, may be surgical candidates.

Another group of patients who are surgical candidates are those with pulmonary complications, in particular, asthma, recurrent aspiration pneumonia, chronic cough, or hoarseness linked to reflux disease.

Three major types of surgical procedures correct gastroesophageal reflux and repair the hernia in the process. They can be performed by open laparotomy or with laparoscopic approaches, which currently are being employed more frequently.

· Nissen fundoplication

○ The Nissen fundoplication performed laparoscopically has gained popularity because of its lower morbidity and shorter hospital stay compared to the open procedure performed previously. Although a relatively high incidence of postoperative complications, such as dysphagia and gas bloating, are reported, DeMeester and Peters[7] have shown that placing a larger bougie in the esophagus during this procedure, along with a shorter wrap and more complete mobilization of the stomach, have markedly reduced postoperative complications.[8, 9]

○ This procedure involves a 360° fundic wrap around the gastroesophageal junction. The diaphragmatic hiatus also is repaired.

○ A transthoracic approach may be used in patients who have had a previous Nissen wrap or those who have an irreducible hernia.

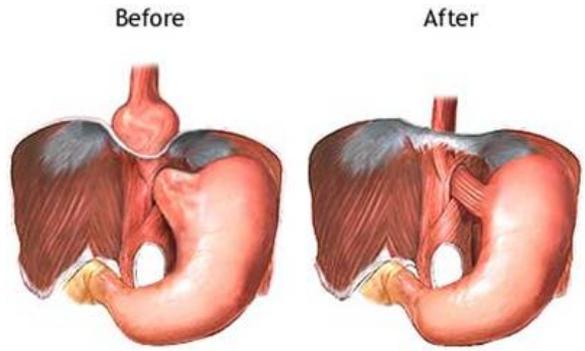
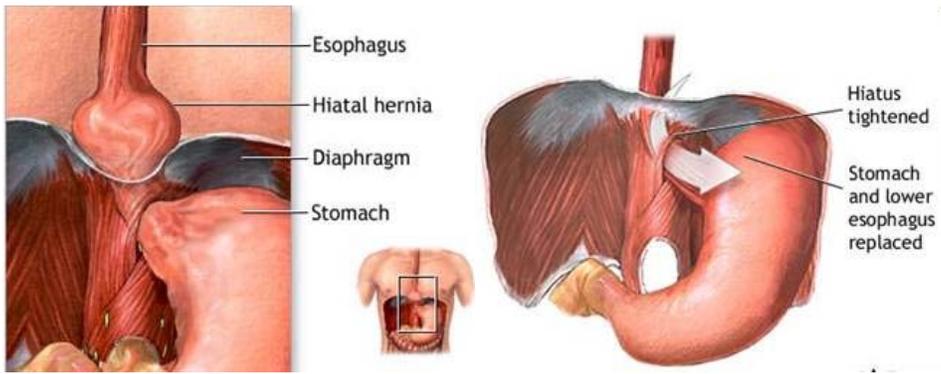
- The Toupet procedure is a variant of the Nissen wrap and involves a 180° wrap in an attempt to lessen the likelihood of postoperative dysphagia.
- Belsey (Mark IV) fundoplication: This operation involves a 270° wrap in an attempt to reduce the incidence of gas bloating and postoperative dysphagia. It also is preferred when minimal esophageal dysmotility is suspected. To complete this operation, the left and right crura of the diaphragm are approximated.
- Hill repair: In this procedure, the cardia of the stomach is anchored to the posterior abdominal areas, such as the medial arcuate ligament. This also has the effect of augmenting the angle of His and thus strengthening the antireflux mechanism.
- The antireflux procedures discussed above offer relief of symptoms in 80-90% of patients. In most cases, the procedure of choice is the one with which the surgeon is most familiar. These procedures carry low mortality and morbidity rates, lower than 15-20%. DeMeester et al found the Nissen procedure superior to the Belsey and Hill repairs with regard to symptom relief and prevention of reflux postoperatively (as judged by pH monitoring). Good long-term results have been reported for antireflux surgery, with adequate control of reflux in the range of 80% at 10 years.
- Most patients with a paraesophageal hernia remain asymptomatic. In this type of hernia, symptoms from acid reflux usually do not occur. Instead, the most common symptom is epigastric or substernal pain. Some patients complain of substernal fullness, nausea, and dysphagia.
 - A significant proportion of patients with this type of hernia develop incarceration of the hernia and possible gastric volvulus, which can lead to perforation.
 - If perforation occurs, the mortality rate is high. Because of this, many surgeons advise elective repair when the diagnosis is made.
 - The goal of surgery is to remove the hernia sac and close the abnormally wide esophageal hiatus.
 - Some surgeons then tack the stomach down in the abdomen to prevent it from migrating upwards again, or, they perform a temporary gastrostomy to help decompress the stomach and anchor it in place in the abdominal cavity.

Stages of the operation:

1. Drawing of the stomach into abdominal cavity by disjunction of adhesions in the region of its cardiac part, esophagus, excision of hernial sac.
2. The plastics of esophageal hiatus of diaphragm (cruroplasty). The most widespread cruroplasty by Hill and narrowing of esophageal ring according to Garrington.
3. Elimination of valvular failure of esophagocardial junction. The purpose of operation is to prevent gastroesophageal reflux by means of formation of His angle and esophagocardial valve. Also Nissen fundoplication is applied.
4. Gastropexia – fixation of gastric wall to parietal peritoneum.

Another tactics is applied in the patients with paraesophageal, parasternal and lumbocostal hernias. The method of choice is the surgery. Such tactics is explained by the hazard of strangulation. The essence of the operation consists of drawing down of hernial content (stomach, intestine, omentum) into abdominal cavity, removing of hernial sac and liquidation (suturing) of hernial ring.

Steps of cruroplastic



DIAPHRAGMATIC RELAXATION (DIAPHRAGMATIC EVENTRATION)

The term "diaphragmatic relaxation " was used for the first time in 1906 by Witting. It means a relaxation of diaphragm, its high standing and displacement upward of abdominal organs.

The term 'diaphragmatic eventration' is used in common practice to describe a condition of relaxation of the diaphragmatic dome. It may present at birth as a congenital condition due to a defect of diaphragmatic development or in a later stage of life as an acquired condition ('acquired diaphragmatic paralysis' or 'acquired diaphragmatic elevation').

Etiology and pathogenesis

The cause of the disease is the congenital or acquired decrease of diaphragmatic resistance, which during elevation of intraperitoneal pressure results in its outpouching. The great importance in the development of acquired relaxation belongs to the damage of diaphragmatic nerve. The cause of the latter could be inflammatory processes in chest and abdominal cavity, intoxication, poisoning, operations on chest organs and birth injury.

Pathology

In congenital form of a diaphragmatic relaxation revealed muscular aplasia, in acquired – atrophy of muscular fibers.

Classification

- 1) Complete: left-side, right-side;
- 2) Incomplete: anterior, posterior, restricted (partial).

Symptomatology and clinical course

Minor manifestation or asymptomatic course characterizes diaphragmatic relaxation. Therefrom, it is always necessary to thoroughly analyze the occurrence of multiple signs from the organs of digestive, respiratory and cardiovascular system. The clinical symptomatology basically depends on dysfunction of the diaphragm by itself and organs, which adjoin to it both in chest, and in abdominal cavity. In left-side diaphragmatic relaxation the asymptomatic course rarely occurs.

General symptomatology. The patients with diaphragmatic relaxation can feel a pain of different character, localization and intensity. The pain syndrome frequently results from gastric inflection or compression of vessels and nerves by filled stomach. Inflection of vascular bundles of pancreas, lien, kidneys, mesentery of small and large intestines as a result of shift of abdominal organs also contribute to the development of pain syndrome. Frequently patients complain of general weakness and loss of weight.

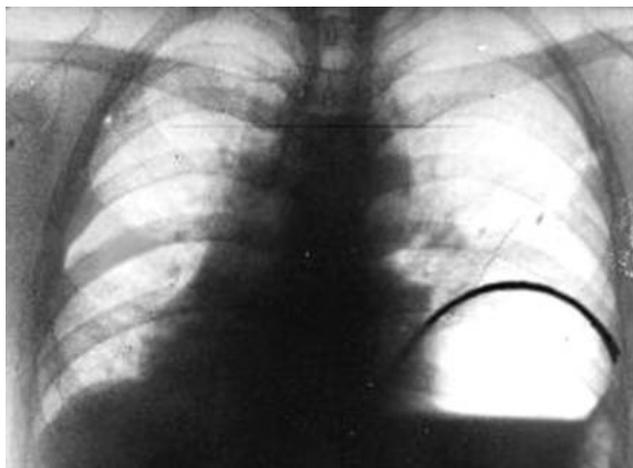
Gastrointestinal symptomatology. Dysphagia almost always arises as a result of inflection of abdominal part of esophagus. The heaviness after meal should be caused by atony of stomach and its evacuation dysfunction. Ulceration and erosive gastritis, which occurs in some patients, are the outcome of a regional ischemia from gastric inflection or torsion. Chronic constipation is basically caused by disturbance of massage influence of the diaphragm on intestine. Meanwhile heartburn, belching, nausea, vomiting and meteorism also observed.

A phrenocardiac Uden-Ramcheld's syndrome represents **cardiopulmonary signs**. It is characterized by dyspnea, discomfort in the region of heart, anginal pain, extrasystole and ECG changes (elongation of P wave, PQ interval and complex QRS).

Respiratory disturbances result from dynamic dysfunction of the diaphragm. The high standing of the diaphragm leads to compression of lung on the side of lesion and disturbed ventilation of the lower part. It causes the diminishing of vital capacity of the lungs and development of dyspnea.

Roentgenologically revealed the high standing of diaphragmatic dome (to II-III intercostal space), restriction of its excursion and reduce of the inferior

pulmonary field. Frequently observed the mediastinal shift to the opposite side. The contrast X-radiography of esophagogastric junction can find out the inflection of abdominal part of esophagus. The X-ray examination enables to establish the diagnosis with a high degree of reliability.



Diaphragmatic relaxation

Variants of clinical course and complications

Asymptomatic course of diaphragmatic relaxation in the majority of patients has caused interpretation of this pathology as "innocent disease". Nevertheless the shift and rotation of heart can cause the heart failures, and the restriction of pulmonary excursion sometimes leads to chronic pneumonia. The gastric inflection frequently may result in disturbance of the valvular mechanism of esophagogastric junction and occurrence of reflux esophagitis.

The diagnostic program

1. Anamnesis and physical findings.
2. Plain chest X-radiography.
3. Esophagogastroduodenoscopy.
4. Roentgenoscopy of esophagus and gastrointestinal tract.
5. General blood and urine analyses.

Differential diagnostics

Diaphragmatic elevation is the secondary high standing, which can arise as a result of ascites, pregnancy, expressed meteorism, peritonitis, tumours of abdomen, splenomegaly or megacolon.

Pneumothorax, pyopneumothorax, pleurisy. Such misdiagnostics in the patients with diaphragmatic relaxation frequently caused by chest pain, cough, dullness and tympanic sound revealed at percussion, and weak breathing at auscultation. Chest X-radiography rather contributes to exact diagnostics.

Diaphragmatic hernia. The differential diagnosis of diaphragmatic relaxation with this pathology is the most difficult. Nevertheless it has the important practical value, because the threat of strangulation of diaphragmatic hernia requires an active surgical tactics. During the establishment of the diagnosis it is always necessary to remember, that clinical manifestation of diaphragmatic hernia more expressed. However, the sharp inflection of abdominal organs in the patients with diaphragmatic relaxation also can associate with severe pain, which resembles strangulation. Thereafter, a reliably

differentiation of these diseases is possible only after a goal-oriented X-ray examination.

Cancer of esophagus and cardial part of stomach. A sharp gastric shift upward with inflection of abdominal part of esophagus can lead to dysphagia, substernal pain, disturbance of digestion, considerable loss of weight etc. For differential diagnostics applied a contrast X-ray examination of esophagus and stomach.

In difficult for differential diagnostics cases a pneumoperitoneum with further X-ray examination is performed. This method allows with a major degree of reliability to establish the diagnosis of diaphragmatic relaxation.

Tactics and choice of treatment

In most cases the asymptomatic course of diaphragmatic relaxation requires no special treatment.

Conservative therapy applied at presence of symptomatology:

- 1) avoidance of physical exertions, which increases intraperitoneal pressure;
- 2) diet – eating by small portions and exception of food, which form waste and gases;
- 3) therapeutic gymnastics for improving of intestinal function and decrease of the patient's weight;
- 4) symptomatic therapy for regulation of cardiovascular and respiratory systems.

The indication for operation: gastric torsion or severe cardiorespiratory dysfunction. If clinical manifestations are absent, the surgical treatment can be recommended only for women with further pregnancy and labors, because these conditions cause a sharp increase of intraperitoneal pressure with further shift of the diaphragm and abdominal organs.

Surgical treatment. By means of a lateral access in VII intercostal space a [phrenoplasty](#) is performed, which consist of incision of diaphragm from costal edge to esophageal ring with following diaphragmatic duplication.

ACUTE MEDIASTITIS

Acute mediastinitis is a purulent inflammation of mediastinum.

Etiology and pathogenesis

The penetration of pathogenic agents into mediastinum can result from perforation and chemical burns of esophagus; injuries of trachea, bronchi, operations on mediastinal organs and lungs. Also is possible the contamination from neck fat tissue and tracheobronchial lymph nodes.

Classification of mediastinitis

According to localization:

- 1) anterior;
- 2) posterior;
- 3) superior;
- 4) medial;
- 5) inferior.

According to pathogenesis:

- 1) primary;
- 2) secondary.

According to the clinical course:

- 1) acute: purulent, aseptic;
- 2) chronic.

According to the character of infection:

- 1) nonspecific;
- 2) specific.

Symptomatology and clinical course

The clinical manifestation of acute mediastinitis is characterized by prompt progressing course, dependence on extent of the process, gravity of infection and peculiarities of underlying disease.

Body temperature raises up to 39-40°C and of hectic character, the patients complain of dyspnea, cyanosis, fever and profuse sweating.

The local symptomatology of the disease depends on location of the process and involvement of esophagus, trachea, heart, n. vagus, n. phrenicus, n. recurrens, tr. sympaticus.

Also is possible dysphagia, dyspnea, constant cough, hoarseness, change of cardiac rhythm.

On percussion revealed a mediastinal widening, on auscultation – weak cardiac tones.

X-ray examination. The method of X-ray examination should be chosen according to the cause of occurrence of acute mediastinitis. If the disease is

caused by cervical phlegmon the X-radiography examination should be restricted only by chest X-ray film in three plains. In such situations observed widening of mediastinum, shadowing of its anterior part and shift of trachea. Compression of esophagus revealed by barium swallow.

The contrast X-ray examination of esophagus after its iatrogenic perforation it is possible to see penetration of barium into mediastinum, shadowing and widening of its consequent parts. Fibresophagoscopy as the method of diagnostics of esophageal perforation is not recommended due to pneumatic pressure during this manipulation.

Variants of clinical course and complications

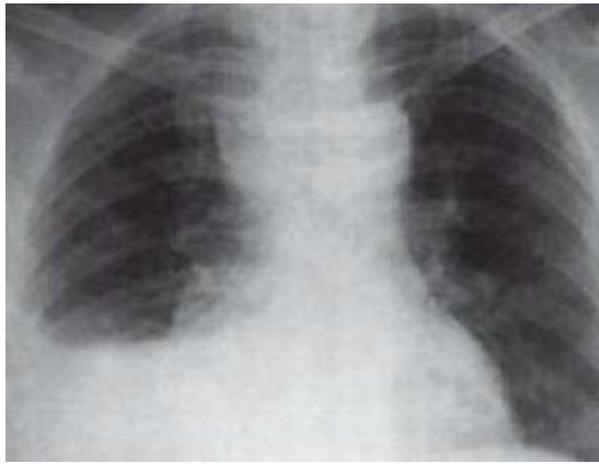
According to the features of clinical symptomatology, acute mediastinitis is divided into anterior and posterior mediastinites.

Anterior mediastinitis	Posterior mediastinitis
Throbbing substernal pain	Throbbing chest pain with irradiation in interscapular region
Intensifying of pain during percussion of breast bone	Intensifying of pain during vertebral pressing
Intensifying of pain when head is unbent back	Intensifying of pain at swallowing
Occurrence of swelling in the region of jugular fossa	Swelling above clavicle
Signs of compression of superior vena cava	Sign of compression of azygos and hemiazygos veins (distended intercostal veins, pleural effusion)

The most often complications of acute mediastinitis are: pyopneumothorax, which has arisen after the abscess discharge into pleural space, pleural empyema, purulent pericarditis, erosive bleeding and lung abscesses.

The diagnostic program

1. Complaint and history of the disease.
2. Physical findings.
3. Chest X-ray examination.
4. Contrast esophagography.
5. ECG.
6. Fibrobronchoscopy.



Acute mediastinitis

Differential diagnostics

Acute mediastinitis requires express differential diagnostics.

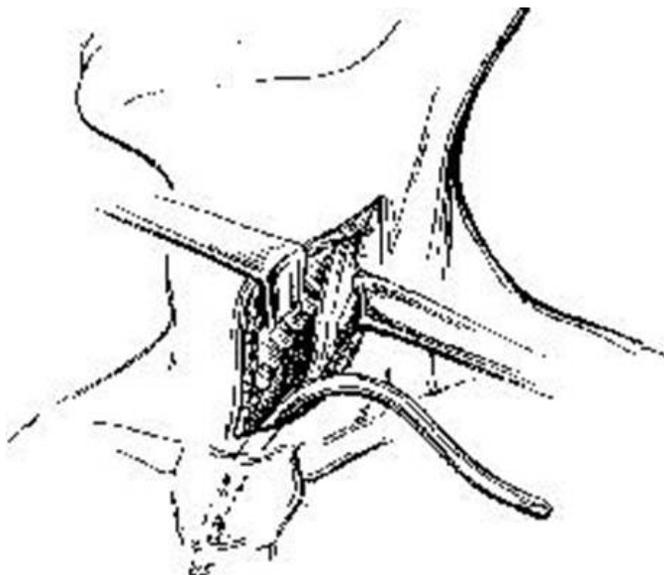
Acute pneumonia, as a rule, is the outcome of catarrhal factor, which evidence is showed by the patients. Besides, more long duration, high temperature, and cough with expectoration of mucopurulent sputum characterize the pneumonia; on auscultation –fine bubbling wet rales on the side of lesion and infiltration of pulmonary tissue at chest X-ray examination. All these findings enable to confirm or to rule out pneumonia.

Exsudative pleurisy mostly arises as the result of complication of pleuropneumonia. The process lasts, as a rule, 5-7 days. The most typical manifestations are cough and chest pain on the side of lesion, which intensifies at deep breathing. Percussion reveals a shortening of percussion sound. On auscultation – weak breathing sounds and pleural friction rub. The presence of intensive homogeneous shadow with oblique upper contour on chest X-radiography and also thoracentesis confirm the diagnosis of pleurisy.

Exsudative pericarditis. It most often results from rheumatic lesion of heart, acute myocardial infarction or polyserositis. Dyspnea, pain, heavy feeling behind breastbone, general malaise, forced sedentary patient's position are the chief signs of pericarditis. On X-ray films observed a trapezoid shape of heart, and on ECG – diminished waves. Puncture of the pericardium with obtaining of exudate finally confirms the diagnosis.

Tactics and choice of treatment

The treatment of mediastinitis is only surgical. Its character and volume significantly depend on the cause, location and extent of purulent process. Established perforation of esophagus, trachea or bronchus requires an urgent operation. The foreign bodies thus removed, and operation ends by drainage. If the process located in anterosuperior mediastinum used a cervical mediastinotomy.



Cervical mediastinotomy

Nevertheless cervical mediastinotomy is insufficient at low localization of the process. In such cases performed anterior mediastinotomy. Meanwhile, isolated posterior mediastinitis is the indication for drainage by means of posterior extrapleural mediastinotomy.

In postoperative period is necessary application of intensive antibacterial, antiinflammatory and detoxycation therapy, and also treatment direct on the increase of immunological resistance of the organism. The mortality after such operative approaches has been 26-36 %, and after conservative treatment – about 70 %.

MEDIASTINAL TUMORS

The mediastinal tumors include true tumours, cyst and masses.

Classification of tumours

1. Neurogenic tumours:

- a) ganglioneuroma;
- b) neurinoma;
- c) neurogenic sarcoma;
- d) neuroblastoma;
- e) sympathoblastoma.

2. Mesenchymal tumours:

- a) fibroma;
- b) lipoma;
- c) hemangioma;
- d) fibrosarcoma;
- e) liposarcoma;
- f) angiosarcoma.

3. Tumours originated from reticular tissue of lymph nodes:

- a) lymphosarcoma;
- b) reticulosarcoma;
- c) lymphogranulomatosis.

4. Tumour originated from thymus (thymoma), and thyroid gland (substernal, intrathoracic goiter).

5. Germ cell tumours:

- a) dermoid cyst;
- b) teratoma;
- c) mediastinal seminoma;
- d) choriocarcinoma.

6. True mediastinal cysts:

- a) mesothelial cyst;
- b) pericardial diverticula;
- c) bronchogenic cyst;
- d) enteric cyst.

7. Parasitogenic (echinococcal) cysts.

Symptomatology and clinical course

On early stages of the development the tumours are almost asymptomatic, and 40 % of mediastinal neoplasms are revealed at preventive chest X-ray examination. The patients most often complain of chest pain. The intensity of pain depends on degree of tumour compression or growth into nervous structures. In malignant growth the pain has more intensive character, than in benign. Frequently the pain precedes by feeling of heaviness, discomfort and foreign body in chest. Sometimes observed dyspnea caused by compression of airways, and major vessels both of anterior, and posterior mediastinum.

Owing to compression of the lumen of superior vena cava the syndrome of superior vena cava develops, which manifest by cyanosis of face, neck and upper half of chest, distend cervical veins, edema and dyspnea. Resulting from the rise of blood pressure and disruption of venous walls, the nasal, esophageal and pulmonary bleedings develop. As the characteristic features considered headache, loss of consciousness and hallucinations. In overwhelming cases superior vena cava syndrome results from malignant tumours of lungs and mediastinum. Only in 5-7 % of patients they are benign.

The basic method of diagnostics is a complex X-ray examination: roentgenoscopy, polypositional X-radiography, tomography, computer tomography. The examination should be start from roentgenoscopy in different plains (multiaxial roentgenoscopy). It gives the possibility to find out a pathological shadow, its location, shape, size, mobility, intensity, contours and to reveal the presence or lack of pulsation of walls. Computer tomography is also a high-grade method of diagnostics. It helps to receive the image of transversal plain of chest at any level, to confirm the location of mediastinal tumour and its communication with adjacent organs. In suspicion on a vascular nature of the process, angiography is used. It enables to rule out aneurysm of heart, aorta and its branches, reveal compression of superior vena cava and growth of the tumour into major arterial trunks.

If it is necessary to differentiate the tumour from cyst and reveal its different deposits, it is expedient to apply ultrasonic examination (sonography).

With the purpose of improvement of localization, size of mass, its communication with mediastinal organs performed pneumomediastinography (X-radiography of mediastinum with introducing of oxygen or air). For pneumomediastinography, depending on tumour locating the gas is introduced through a puncture above jugular incisure of breastbone, under xyphoid process or parasternally. Thus gas at first is spread in anterior mediastinum, and in 45-60 min. penetrates in posterior. The introduced gas achieves a good visualization of tumour contours and its growth into adjacent organs. Sometimes an artificial pneumothorax is performed on the affected side. In such patients collapse of lung gives the opportunity to differentiate pulmonary tumour from mediastinal tumours and cysts.

For morphological verification of the tumour applied such additional methods:

- thoracoscopy, which allows to examine a pleural space, to take biopsy from mediastinal lymph nodes or tumour;
- mediastinoscopy (through a small incision above the breastbone exposed trachea, and along its position performed the canal in anterior mediastinum with following insertion of a special endoscope) enables to examine anterior mediastinum, and take a biopsy from lymph nodes and tumour;
- transthoracic aspiration biopsy is performed if tumour is located near chest wall;
- transbronchial puncture of lymph nodes is carried out during bronchoscopy.

During diagnostics of mediastinal neoplasms applied according to indications bronchography, esophagography and pneumoperitoneum.

Variants of clinical course and complications

Neurogenic tumours are the most common neoplasms of mediastinum, which occur in 20 % among the tumours of this location. There arise in any age and more often benign. Their predominant localization is the posterior mediastinum. The origin of such tumours could be nervous trunks, ganglions and other nervous structures of mediastinum. From the cells of sympathetic nervous trunk arise ganglioneuromas, neuroblastomas and sympathoblastomas.

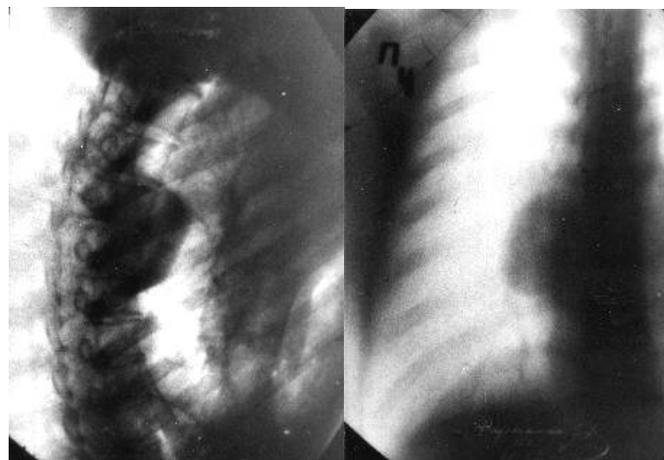
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The tumours, which arise from peripheral nerves are represent by neuromas and neurofibromas. The paragangliomas and mediastinal pheochromocytoma develop from chemoceptor cells of and according to the structure resemble the tumours of sinocarotid zone. They frequently produce hormones, and manifests by hypertension with often crises. In half of patients these tumours are malignant.

Neurogenic sarcoma is a malignant tumour of nervous sheath. It is usually solitary, or in association with von Recklinhausen's disease.

The neurogenic tumours commonly manifest by backache, hyperalgesia, pareses and paralyzes at tumour growth through the spinal canal. The pain reaction varies from slight to severe expressed neuralgias. Frequently ganglioneuroma is asymptomatic for many years. In the clinical pattern of malignant neurogenic tumours prevail general intoxication, loss of weight and pain syndrome.

Roentgenologically in neurogenic tumour of a vertebrocostal angle observed characteristic intensive rounded shadow, with vertebral and costal usuration, rib. Frequently revealed hemorrhagic pleural effusion on the side of lesion by malignant tumour.



Ganglioneuroma

Mesenchymal tumours. According to the histological origin mesenchymal benign tumours are represented by:

- from a fibrous connecting tissue – fibroma;
- from a cartilaginous and osseous tissue - chondroma, osteochondroma, osteoblastoclastoma;
- from a spinal cord – chordoma;
- from fat tissues – lipoma, hibernoma;
- the tumours, which originate from vessels – hemangioma, lymphangioma;
- from a muscular tissue - leiomyoma, rhabdomyoma.

Lipomas are the most frequent mesenchymal benign tumours with predominant location in cardiophrenic angle.

The clinical symptomatology of these tumours is atypical. At the small sizes they are usually asymptomatic. The malignant neoplasms manifest much earlier as the result of prompt infiltrative growth of the tumour and intoxication of the organism. Nevertheless, despite the malignant character, liposarcoma can grow rather slowly with late metastatic spread.

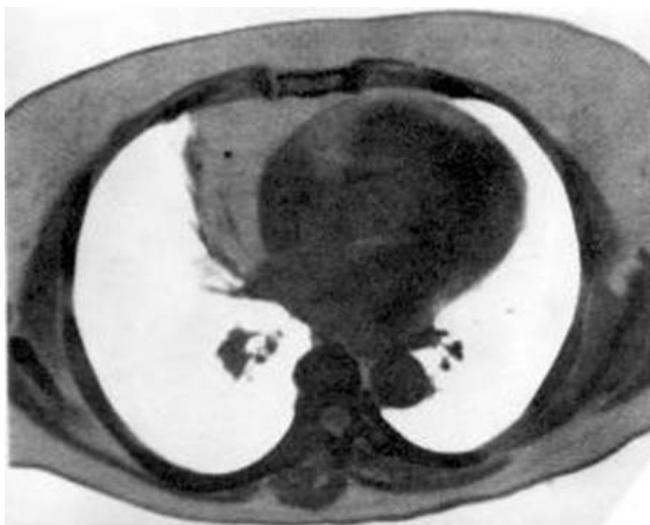
On [roentgenogram](#) such masses represented by homogeneous formations, that applies to heart shadow.

The **lymphomas** occur in 3-5 % of patients with mediastinal tumors and in 20-25 % with all malignant neoplasms of mediastinum. The lymphomas arise from mediastinal lymph nodes. Their common localization – anterior mediastinum, nevertheless lymph nodes of any part can be affected. There are three types of lymphomas: lymphosarcoma, reticulosarcoma and lymphogranulomatosis. All of them are characterized by malignant course. The initial signs of this pathology mainly caused by intoxication: malaise, subfebrile or febrile temperature with further remittent character, sweating and loss of weight. One of most typical manifestation of the disease should be considered itching of skin. Abnormally enlarged lymph nodes can compress mediastinal organs, which lead to dry cough, chest pain, and dyspnea. In lymphogranulomatosis, in contrast with other mediastinal tumours, the signs of compression are weakly expressed. It is characterized by bilateral lesion and blood changes (leukocytosis or leukopenia and elevation of erythrocyte sedimentation rate). Lymphosarcoma differs by more prompt course and considerable progression of mediastinal compression syndrome. X-ray examination, mediastinoscopy and biopsy of lymph nodes are the most valuable for diagnostics.

The **dermoid cysts and teratomas** arise owing to disturbance of embryogenesis and occur in 5-8 % of the patients with mediastinal tumors. The origin of dermoid cysts is the ectoderm, which transforms to a fibrous connecting tissue. The cystic cavity frequently contains similar to fat viscous mass of brown color with deposits of skin and hair. The teratomas arise from several germinal laminae and contain different structural tissues. They are divided on mature and immature. All mature teratomas, as a rule, are characterized by well-defined capsule, irregular rounded or oval shape, different size, and look like a cyst on slit. Immature ones look like solitary nodes, sometimes with small cavities. The structure of teratomas can include parts of glands, teeth, bones and sometimes even underdeveloped fetus. According to degree of cellular differentiation teratoma is divided on benign (80-90 %) and malignant (10-20 %). Although this disease is always congenital, it is diagnosed, mainly, in elderly age at occurrence of pain and "compression syndrome". 95 % of such cysts are located in anterior mediastinum and frequently are accompanied with cardiovascular disturbances (tachycardia, pressing pain in the region of heart). The compression of major bronchi and trachea results in occurrence of dyspnea, paroxysmal cough and hemoptysis. The infection of the tumour frequently leads to fever and increase of intoxication. The presence in sputum of hair and other tissues is considered to be the sign of bronchial fistula.

In diagnostics of teratomas the major value has a complex X-ray examination. It is possible to find out teeth, calcification of cystic capsule and its contents. Prompt growth of the tumour, disappearance of its regular contours suspects the malignancy.

Cysts of pericardium are the cavity thin-walled formations, which according to the structure resemble pericardium. They occur in 7-8 % of cases of all mediastinal tumors. Their most often location is the right cardiophrenic angle, much less often – left one. A true cyst may be single-, double- or multichamber, connected or non-connected with pericardium. The cases, when the cystic cavity communicates with the cavity of pericardium, it should be considered as diverticulum. The disease, as a rule, is asymptomatic and it is casually revealed only during prophylactic fluorography. In cases of great size of cyst the patients complain of pain in the region of heart and cardiac arrhythmia. During X-ray examination mesothelial cyst or pericardial diverticulum is observed as oval or semicircular homogeneous shadow with regular outline, which intimately applies to the shadow of heart.



Cysts of pericardium. Computer tomography

Bronchogenic and enteric cysts of mediastinum arise in the period of intrauterine development and originated from dystopic germs of bronchial or intestinal epithelium.

Bronchogenic cysts, as a rule, are single chamber, with location either in mediastinum, or in pulmonary tissue. The neighborhood of cyst with bifurcation of trachea can cause paroxysmal cough, dyspnea and respiratory disturbances. Paraesophageal location of the cyst manifests by dysphagia. The clinical manifestations of such pathology can be caused by inflammatory process in cyst or its sudden discharge into airways. If the cyst is communicated with airways, the roentgenogram reveals a fluid level, and during bronchography a contrast agent fills in the lumen of cyst. The diagnosis is possible to confirm by means of pneumomediastinography.

The enteric cyst (enterocystoma) arises from the dorsal parts of a primary intestinal tube, located more often in lower parts of posterior mediastinum and applies to esophagus. Depending on histology they are divided on esophageal, gastric and intestinal cyst. In cases, when a cystic wall is covered by gastric epithelium, which produces hydrochloric acid, an ulceration of wall, bleeding and its perforation can develop.

Frequently enteric cyst suppurates with the hazard of discharge into pleural space or pericardium, esophagus and bronchi. The most often signs of the disease is dyspnea and chest pain. Due to characteristic localization of the pathological focus in posterior mediastinum, to the right from median line, the roentgenological diagnostics is usually not difficult. In order to determine mutual relation of neoplasm to trachea, bronchi, and esophagus it is possible to apply pneumomediastinography. As there is always a danger of the development of complications, it is necessary to consider such pathology as absolute indication for operative removal.

Echinococcosis of mediastinum occurs rarely (1-2 % of all mediastinal tumors). If the parasite is of small size, the disease most often asymptomatic. The enlargement of echinococcal cyst causes pain, dyspnea, dysphagia and superior vena cava syndrome. Sometimes in such patients the cyst can discharge into bronchus or trachea. The suppuration of parasitogenic cysts is transformed into abscess and purulent mediastinitis. Roentgenological method should be considered to be predominant in the diagnostics of echinococcosis of mediastinum. The presence of homogeneous, round or oval shadow with regular outline (with further calcification) suggests echinococcosis. For confirming of the diagnosis reaction of latex-agglutination is performed. Echinococcosis of mediastinum frequently associated with anatomical lesion of lungs.

Principles of diagnostics in such situation are similar to primary mediastinal tumors.

Tactics and choice of treatment

The presence of mediastinal tumors requires surgical treatment. Expectant tactics and dynamic observation in such cases are unjustified.

In connection with a constant and substantial threat of infection, perforation and development of purulent intrapleural complications, the pericardial, bronchogenic and enteric cysts of mediastinum are the subjects to operative remove.

The operative treatment of malignant mediastinal tumors should be applied on early stages of the disease. In advanced cases it is expedient to apply antineoplastic and radiotherapy.

The benign neurogenic tumours are removed in surgical way through thoracotomy incision. If the tumour grow like a " sandglasses" a fragment of the tumour removed with following resection of vertebral arches. The malignant tumours are removed "in mass", with maximal excision of the tumour, affected ribs, paraaortic, esophageal and paratracheal lymph nodes.

SUPERIOR VENA CAVA (SVC) SYNDROME

Superior vena cava (SVC) syndrome (SVCS) is characterized by gradual, insidious compression/obstruction of the superior vena cava. Although the syndrome can be life threatening, its presentation is often associated with a gradual increase in symptomatology. For this reason, diagnosis is often delayed until significant compression of the superior vena cava has occurred.

Pathophysiology

Extrinsic compression of the superior vena cava is possible because it has a thin wall coupled with a low intravascular pressure. Because the superior vena cava is surrounded by rigid structures, it is relatively easy to compress. The low intravascular pressure also allows for the possibility of thrombus formation, such as catheter-induced thrombus.

The subsequent obstruction to flow causes an increased venous pressure, which results in interstitial edema and retrograde collateral flow.

Epidemiology

Superior vena cava syndrome is chiefly associated with malignancy. Currently, more than 90% of patients with superior vena cava syndrome have an associated malignancy as the cause. This contrasts with studies in the early 1950s in which a large proportion of cases were nonmalignant. Infectious causes (eg, syphilis, tuberculosis) have decreased because of improvements in antibiotic therapy. Of the nonmalignant causes of superior vena cava syndrome, thrombosis from central venous instrumentation (catheter, pacemaker, guidewire) is an increasingly common event, especially as these procedures become more common.

Causes

Today, the most common etiology of superior vena cava syndrome is related to malignancy.

- Prior to modern antibiotics, infectious causes including syphilis, tuberculosis, and fungi occurred with almost equal frequency.
- The most common cause of malignancy-related superior vena cava syndrome is bronchogenic carcinoma, which accounts for nearly 80% of cases.
- Lymphoma accounts for approximately 15% of cases.
- Other cases have various causes, including infectious and catheter-related etiologies. Increasingly, dialysis catheters and pacemaker leads are becoming associated with superior vena cava syndrome due to thrombosis.

Symptoms

The symptoms of SVCS are more severe if the vein becomes blocked quickly. This is because the other veins in the area do not have time to widen and take over the blood flow that cannot pass through the superior vena cava.

The most common symptoms are:

- Trouble breathing.
- Coughing.
- Swelling in the face, neck, upper body, or arms.

Less common symptoms include the following:

- Hoarse voice.
- Trouble swallowing or talking.
- Coughing up blood.
- Swollen veins in the chest or neck.
- Chest pain.
- Reddish skin color.

Laboratory Studies

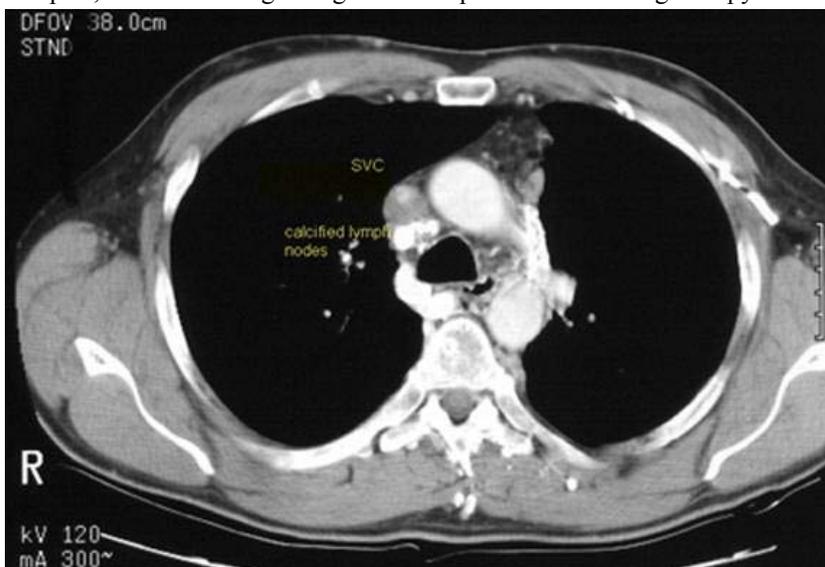
The diagnosis of superior vena cava syndrome (SVCS) is often made on clinical grounds alone, combining clinical presentation with an often-obtained history of thoracic malignancy.

Imaging Studies

Plain radiography is often helpful and reveals a mediastinal mass in most patients (as is seen in the image below).

When in doubt, venography can aid in the diagnosis, but this is usually not necessary.

Thoracic CT scanning is helpful, but the histologic diagnosis is important in initiating therapy.



Treatment

Treatment for SCVS caused by cancer depends on the following:

- The type of cancer.
- The cause of the blockage.
- How severe the symptoms are.
- The prognosis (chance of recovery).
- Whether treatment is to cure, control, or relieve the symptoms of cancer.
- The patient's wishes.

Treatment may include the following:

Watchful waiting

Watchful waiting is closely monitoring a patient's condition without giving any treatment unless symptoms appear or change.

A patient who has good blood flow through smaller veins in the area and mild symptoms may not need treatment.

The following may be used to relieve symptoms and keep the patient comfortable:

- Keeping the upper body raised higher than the lower body.
- Corticosteroids (drugs that reduce swelling).
- Diuretics (drugs that make excess fluid pass from the body in urine). Patients taking diuretics are closely watched because these drugs can cause dehydration (loss of too much fluid from the body).

Chemotherapy

Chemotherapy is the usual treatment for tumors that respond to anticancer drugs, including small cell lung cancer and lymphoma. Chemotherapy uses drugs to stop the growth of cancer cells, either by killing the cells or by stopping them from dividing. When chemotherapy is taken by mouth or injected into a vein or muscle, the drugs enter the bloodstream and can reach cancer cells throughout the body (systemic chemotherapy). When chemotherapy is placed directly into the cerebrospinal fluid, an organ, or a body cavity such as the abdomen, the drugs mainly affect cancer cells in those areas (regional chemotherapy). The way the chemotherapy is given depends on the type and stage of the cancer being treated.

Radiation therapy

If the blockage of the superior vena cava is caused by a tumor that does not usually respond to chemotherapy, radiation therapy may be given. Radiation therapy is a cancer treatment that uses high-energy x-rays or other types of radiation to kill cancer

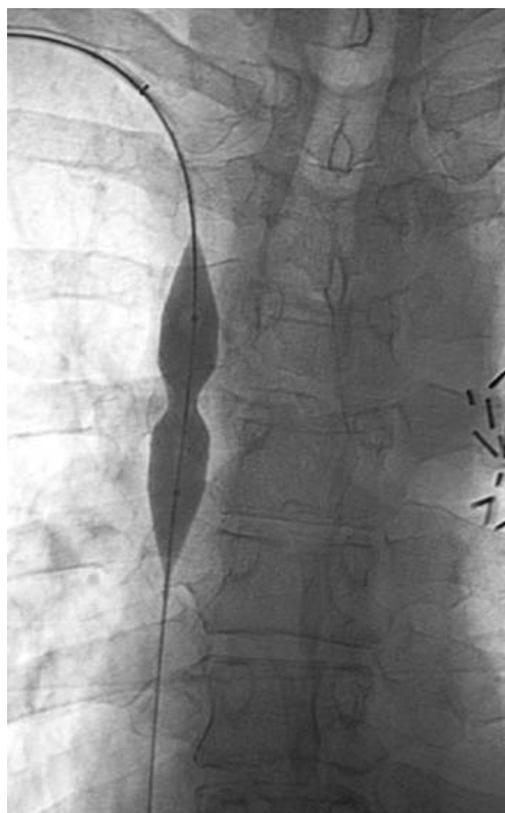
cells. External radiation therapy uses a machine outside the body to send radiation toward the cancer. The way the radiation therapy is given depends on the type and stage of the cancer being treated.

Thrombolysis

SVCS may occur when a thrombus (blood clot) forms in a partly blocked vein. Thrombolysis is a way to break up and remove blood clots. This may be done by a thrombectomy. Thrombectomy is surgery to remove the blood clot or the use of a device inserted into the vein to remove the blood clot. This may be done with or without the use of drugs to break up the clot.

Stent placement

If the superior vena cava is partly blocked by the tumor, an expandable stent (tube) may be placed inside the superior vena cava to help keep it open and allow blood to pass through. This helps most patients. Drugs to keep more blood clots from forming may also be used.



Surgery

Surgery to bypass (go around) the blocked part of the vein is sometimes used for cancer patients, but is used more often for patients without cancer.

Palliative care may be given to relieve symptoms in patients with SVCS.

Superior vena cava syndrome is serious and the symptoms can be upsetting for the patient and family. It is important that patients and family members ask questions about superior vena cava syndrome and how to treat it. This can help relieve anxiety about symptoms such as swelling, trouble swallowing, coughing, and hoarseness.

Patients with advanced cancer sometimes decide not to have any serious treatment. Palliative treatment can help keep patients comfortable by relieving symptoms to improve their quality of life.

GASTROESOPHAGEAL REFLUX DISEASE

Gastroesophageal reflux disease (GERD) is a condition in which the stomach contents (food or liquid) leak backwards from the stomach into the esophagus (the tube from the mouth to the stomach). This action can irritate the esophagus, causing heartburn and other symptoms.

Causes

When you eat, food passes from the throat to the stomach through the esophagus (also called the food pipe or swallowing tube). Once food is in the stomach, a ring of muscle fibers prevents food from moving backward into the esophagus. These muscle fibers are called the lower esophageal sphincter, or LES.

If this sphincter muscle doesn't close well, food, liquid, and stomach acid can leak back into the esophagus. This is called reflux or gastroesophageal reflux. Reflux may cause symptoms, or it can even damage the esophagus.

The risk factors for reflux include:

- Alcohol (possibly)
- Hiatal hernia (a condition in which part of the stomach moves above the diaphragm, which is the muscle that separates the chest and abdominal cavities)
- Obesity
- Pregnancy
- Scleroderma
- Smoking

Heartburn and gastroesophageal reflux can be brought on or made worse by pregnancy and many different medications. Such drugs include:

- Anticholinergics (e.g., for seasickness)
- Beta-blockers for high blood pressure or heart disease
- Bronchodilators for asthma
- Calcium channel blockers for high blood pressure
- Dopamine-active drugs for Parkinson's disease
- Progestin for abnormal menstrual bleeding or birth control
- Sedatives for insomnia or anxiety
- Tricyclic antidepressants

If you suspect that one of your medications may be causing heartburn, talk to your doctor. Never change or stop a medication you take regularly without talking to your doctor.

Symptoms

More common symptoms are:

- Feeling that food is stuck behind the breastbone
- Heartburn or a burning pain in the chest (under the breastbone)
- Increased by bending, stooping, lying down, or eating
- More likely or worse at night
- Relieved by antacids
- Nausea after eating

Less common symptoms are:

- Bringing food back up (regurgitation)
- Cough or wheezing
- Difficulty swallowing
- Hiccups

- Hoarseness or change in voice
- Sore throat

Exams and Tests

You may not need any tests if your symptoms are not severe.

If your symptoms are severe or they come back after you have been treated, one or more tests may help diagnose reflux or any complications:

- Esophagogastroduodenoscopy (EGD) is often used to find the cause and examine the esophagus (swallowing tube) for damage. The doctor inserts a thin tube with a camera on the end through your mouth. The tube is then passed into your esophagus, stomach, and small intestine.
- Barium swallow
- Continuous esophageal pH monitoring
- Esophageal manometry

A positive stool occult blood test may diagnose bleeding that is coming from the irritation in the esophagus, stomach, or intestines.

Treatment

You can make many lifestyle changes to help treat your symptoms. Avoid foods that cause problems for you. Making changes to your routine before you go to sleep may also help.

Avoid drugs such as aspirin, ibuprofen (Advil, Motrin), or naproxen (Aleve, Naprosyn). Take acetaminophen (Tylenol) to relieve pain. Take your medicines with plenty of water. When your doctor gives you a new medicine, remember to ask whether it will make your heartburn worse.

You may use over-the-counter antacids after meals and at bedtime, although they do not last very long. Common side effects of antacids include diarrhea or constipation.

Other over-the-counter and prescription drugs can treat GERD. They work more slowly than antacids but give you longer relief. Your pharmacist, doctor, or nurse can tell you how to take these drugs.

- Proton pump inhibitors (PPIs) decrease the amount of acid produced in your stomach
- H2 blockers (antagonists) lower the amount of acid released in the stomach

Anti-reflux operations (fundoplication and others) may be an option for patients whose symptoms do not go away with lifestyle changes and drugs. Heartburn and other symptoms should improve after surgery, but you may still need to take drugs for your heartburn.

There are also new therapies for reflux that can be performed through an endoscope (a flexible tube passed through the mouth into the stomach).

Outlook (Prognosis)

Most people respond to lifestyle changes and medications. However, many patients need to continue taking drugs to control their symptoms.

Possible Complications

- Asthma
- Barrett's esophagus (a change in the lining of the esophagus that can increase the risk of cancer)
- Bronchospasm (irritation and spasm of the airways due to acid)
- Chronic cough or hoarseness
- Dental problems
- Esophageal ulcer

- Stricture (a narrowing of the esophagus due to scarring)

ACUTE PERITONITIS. LIMITED PERITONITIS. SPECIFIC CLINICAL FORMS OF PERITONITIS.

Peritonitis

Peritonitis – is the acute or chronic peritoneal inflammation with characteristic local and general changes in the organism and severe dysfunction of organs and vital systems of the organism. Peritonitis is an inflammation of the peritoneum, the thin tissue that lines the inner wall of the abdomen and covers most of the abdominal organs. Peritonitis may be localized or generalised, and may result from infection (often due to rupture of a hollow organ as may occur in abdominal trauma or appendicitis) or from a non-infectious process.

Anatomy

The peritoneum is the largest and most complex serous membrane in the body. It forms a closed sac (ie, coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retroperitoneum (posterior), by covering the extraperitoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. It thereby creates a potential space between the 2 layers (ie, the peritoneal cavity).

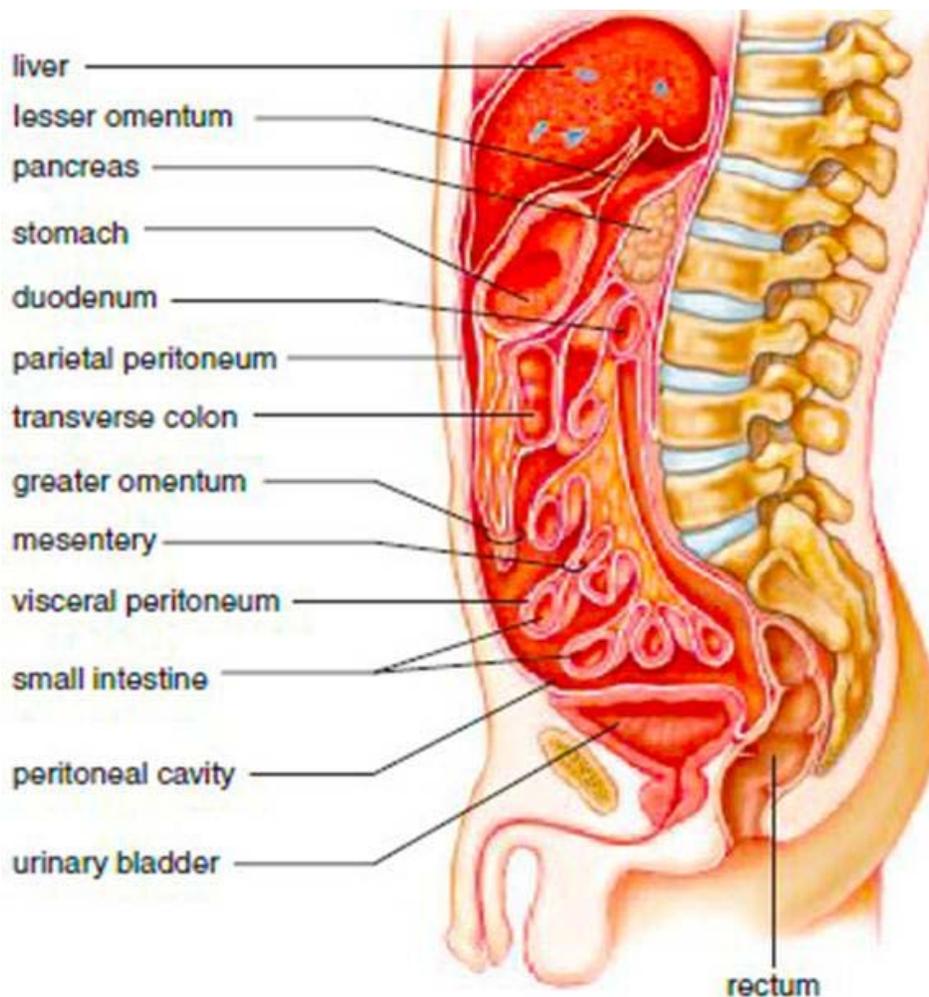
The peritoneum consists of a single layer of flattened mesothelial cells over loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillaries, nerve endings, and immune-competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by junctional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes. Pinocytotic activity of the mesothelial cells and phagocytosis by macrophages allow for clearance of macromolecules.

Normally, the amount of peritoneal fluid present is less than 50 mL, and only small volumes are transferred across the considerable surface area in a steady state each day. The peritoneal fluid represents a plasma ultrafiltrate, with electrolyte and solute concentrations similar to that of neighboring interstitial spaces and a protein content of less than 30 g/L, mainly albumin. In addition, peritoneal fluid contains small numbers of desquamated mesothelial cells and various numbers and morphologies of migrating immune cells (reference range is < 300 cells/ μ L, predominantly of mononuclear morphology).

The peritoneal cavity is divided incompletely into compartments by the mesenteric attachments and secondary retroperitonealization of certain visceral organs. A large peritoneal fold, the greater omentum, extends from the greater curvature of the stomach and the inferior aspect of the proximal duodenum

downward over a variable distance to fold upon itself (with fusion of the adjacent layers) and ascends back to the taenia omentalis of the transverse colon. This peritoneal fold demonstrates a slightly different microscopic anatomy, with fenestrated surface epithelium and a large number of adipocytes, lymphocytes, and macrophages, and it functions as a fat storage location and a mobile immune organ.

The compartmentalization of the peritoneal cavity, in conjunction with the greater omentum, influences the localization and spread of peritoneal inflammation and infections.



Etiology and pathogenesis

The main causes of peritonitis are the acute inflammation of abdominal viscera, discontinuity and disturbed permeability of their walls, open and closed traumas of the abdomen with the damage of viscera with following microbial contamination of peritoneal space.

Despite the cause of peritonitis, the disease is characterized by a typical bacterial inflammation. The infectious agents are represented by *Escherichia coli*, *Staphylococcus* and *Enterococcus*, *Proteus*, *Streptococcus* and also nonclostridial anaerobes. At least in 30 % of cases association of two or more agents occur.

Primary peritonites occur very rarely and result from pneumococcal, streptococcal and staphylococcal infection.

Besides microbial peritonites, caused by peritoneal contamination, distinguished also aseptic peritoneal inflammation, which result from entering of different chemical noninfectious agents into peritoneal cavity (blood, urine, bile, pancreatic juice, etc.). It's so called toxico-chemical peritonitis. But with the development of aseptic inflammation bacteria penetrate into peritoneal space with transformation of peritonitis into bacterial.

Chronic peritonitis is mainly caused by tuberculosis, which agents are usually located extraperitoneally (lungs, mediastinal lymph nodes) or in mesenteric lymph nodes and by hematogenous way enter the peritoneum.

Risk Factors:

The following factors may increase the risk for primary peritonitis:

- **Torn or twisted intestine**
- **Pancreatitis**
- **Inflammatory bowel disease, such as Crohn's disease or ulcerative colitis**
- **Injury caused by an operation**
- **Peritoneal dialysis**
- **Trauma**
- **Liver disease (cirrhosis)**
- **Fluid in the abdomen**
- **Weakened immune system**
- **Pelvic inflammatory disease**
- **Risk factors for secondary peritonitis include:**
- **Appendicitis (inflammation of the appendix)**
- **Stomach ulcers**

Infected peritonitis

Perforation of part of the gastrointestinal tract is the most common cause of peritonitis. Examples include perforation of the distal oesophagus (Boerhaave syndrome), of the stomach (peptic ulcer, gastric carcinoma), of the duodenum (peptic ulcer), of the

remaining intestine (e.g., appendicitis, diverticulitis, Meckel diverticulum, inflammatory bowel disease (IBD), intestinal infarction, intestinal strangulation, colorectal carcinoma, meconium peritonitis), or of the gallbladder (cholecystitis). Other possible reasons for perforation include abdominal trauma, ingestion of a sharp foreign body (such as a fish bone, toothpick or glass shard), perforation by an endoscope or catheter, and anastomotic leakage. The latter occurrence is particularly difficult to diagnose early, as abdominal pain and ileus paralyticus are considered normal in patients who have just undergone abdominal surgery. In most cases of perforation of a hollow viscus, mixed bacteria are isolated; the most common agents include Gram-negative bacilli (e.g., *Escherichia coli*) and anaerobic bacteria (e.g., *Bacteroides fragilis*). Fecal peritonitis results from the presence of faeces in the peritoneal cavity. It can result from abdominal trauma and occurs if the large bowel is perforated during surgery.

Table. Microbial Flora of Secondary Peritonitis

Type	Organism	Percentage
Aerobic	Gram negative	
	Escherichia coli	60%
	Enterobacter/Klebsiella	26%
	Proteus	22%
Gram positive	Pseudomonas	8%
	Streptococci	28%
	Enterococci	17%
	Staphylococci	7%
Anaerobic	Bacteroides	72%
	Eubacteria	24%
	Clostridia	17%
	Peptostreptococci	14%
	Peptococci	11%
Fungi	Candida	2%

Other rare, nonsurgical causes of intra-abdominal sepsis include the following:

Chlamydia peritonitis;

Tuberculosis peritonitis;

Acquired immunodeficiency syndrome (AIDS)-associated peritonitis.

Disruption of the peritoneum, even in the absence of perforation of a hollow viscus, may also cause infection simply by letting micro-organisms into the peritoneal cavity. Examples include trauma, surgical wound, continuous ambulatory peritoneal dialysis, and intra-peritoneal chemotherapy. Again, in most cases, mixed bacteria are isolated; the most common agents include cutaneous species such as *Staphylococcus aureus*, and coagulase-negative staphylococci, but many others are possible, including fungi such as *Candida*.

Spontaneous bacterial peritonitis (SBP) is a peculiar form of peritonitis occurring in the absence of an obvious source of contamination. It occurs in patients with ascites, in particular, in children. See the article on spontaneous bacterial peritonitis for more information.

Intra-peritoneal dialysis predisposes to peritoneal infection (sometimes named "primary peritonitis" in this context).

Systemic infections (such as tuberculosis) may rarely have a peritoneal localisation.

Non-infected peritonitis

Leakage of sterile body fluids into the peritoneum, such as blood (e.g., endometriosis, blunt abdominal trauma), gastric juice (e.g., peptic ulcer, gastric carcinoma), bile (e.g., liver biopsy), urine (pelvic trauma), menstruum (e.g., salpingitis), pancreatic juice (pancreatitis), or even the contents of a ruptured dermoid cyst. It is important to note that, while these body fluids are sterile at first, they frequently become infected once they leak out of their organ, leading to infectious peritonitis within 24 to 48 hours.

Sterile abdominal surgery, under normal circumstances, causes localised or minimal generalised peritonitis, which may leave behind a foreign body reaction and/or fibrotic adhesions. However, peritonitis may also be caused by the rare case of a sterile foreign body inadvertently left in the abdomen after surgery (e.g., gauze, sponge).

Much rarer non-infectious causes may include familial Mediterranean fever, TNF receptor associated periodic syndrome, porphyria, and systemic lupus erythematosus.

Classification

Peritonites are classified:

According to the character of microbial contamination:

A: primary

B: secondary.

According to clinical course:

A: acute

B: chronic.

According to the etiological agents:

A: peritonites, which caused by bacteria of digestive tract (E. colli, staphylococci, streptococci, proteus, anaerobes, etc.)

B: which caused by bacteria, which exist out of gastrointestinal tube (gonococci, pneumococci, streptococcus haemolyticus, etc.).

C: distinguished aseptic (nonbacterial peritonites), resulting from irritation by blood, bile, pancreatic juice or urine.

According to the character of exudate:

A: serous;

B: fibrinous;

C: fibrinopurulent;

D: purulent;

E: hemorrhagic;

F: "peritonitus sicca".

According to the extension of inflammatory process:

A: local;

B: diffuse;

C: generalized.

Dependent on duration of the disease and degree of pathological alterations in the clinical course of peritonitis distinguished three stages:

reactive (first 24 hours) maximal manifestation of local signs of the disease;

toxic (24-72 hours) – gradual reducing of local signs and increasing of general intoxication.

terminal (after 72 hours) – severe, often unreversable intoxication on the background of sharply expressed local manifestations of peritoneal inflammation.

Primary peritonitis.

Spontaneous bacterial peritonitis (SBP) is an acute bacterial infection of ascitic fluid. Contamination of the peritoneal cavity is thought to result from translocation of bacteria across the gut wall or mesenteric lymphatics and, less frequently, via hematogenous seeding in the presence of bacteremia.

SBP can occur as a complication of any disease state that produces the clinical syndrome of ascites, such as heart failure and Budd-Chiari syndrome. Children with nephrosis or systemic lupus erythematosus who have ascites have a high risk of developing SBP. The highest risk of SBP, however is in patients with cirrhosis who are in a decompensated state. In particular, decreased hepatic synthetic function with associated low total protein level, low complement levels, or prolonged prothrombin time (PT) is associated with maximum risk. Patients with low protein levels in ascitic fluid (< 1 g/dL) have a 10-fold higher risk of developing SBP than those with a protein level greater than 1 g/dL. Approximately 10-30% of patients with cirrhosis and ascites develop SBP. The incidence rises to more than 40% with ascitic fluid protein contents of less than 1 g/dL (which occurs 15% of patients), presumably because of decreased ascitic fluid opsonic activity.

More than 90% of cases of SBP are caused by a monomicrobial infection.

Secondary peritonitis

Common etiologic entities of secondary peritonitis (SP) include perforated appendicitis; perforated gastric or duodenal ulcer; perforated (sigmoid) colon caused by diverticulitis, volvulus, or cancer; and strangulation of the small bowel. Necrotizing pancreatitis can also be associated with peritonitis in the case of infection of the necrotic tissue.

The pathogens involved in SP differ in the proximal and distal GI tract. Gram-positive organisms predominate in the upper GI tract, with a shift toward gram-negative organisms in the upper GI tract in patients on long-term gastric acid suppressive therapy. Contamination from a distal small bowel or colon source initially may result in the release of several hundred bacterial species (and fungi); host defenses quickly eliminate most of these organisms. The resulting peritonitis is almost always polymicrobial, containing a mixture of aerobic and anaerobic bacteria with a predominance of gram-negative organisms.

As many as 15% of patients who have cirrhosis with ascites who were initially presumed to have SBP have SP. In many of these patients, clinical signs and symptoms alone are not sensitive or specific enough to reliably differentiate between the 2 entities. A thorough history, evaluation of the peritoneal fluid, and additional diagnostic tests are needed to do so; a high index of suspicion is required.

Source Regions	Causes
Esophagus	Boerhaave syndrome Malignancy

	Trauma (mostly penetrating) Iatrogenic*
Stomach	Peptic ulcer perforation Malignancy (eg, adenocarcinoma, lymphoma, gastrointestinal stromal tumor) Trauma (mostly penetrating) Iatrogenic*
Duodenum	Peptic ulcer perforation Trauma (blunt and penetrating) Iatrogenic*
Biliary tract	Cholecystitis Stone perforation from gallbladder (ie, gallstone ileus) or common duct Malignancy Choledochal cyst (rare) Trauma (mostly penetrating) Iatrogenic*
Pancreas	Pancreatitis (eg, alcohol, drugs, gallstones) Trauma (blunt and penetrating) Iatrogenic*
Small bowel	Ischemic bowel Incarcerated hernia (internal and external) Closed loop obstruction Crohn disease Malignancy (rare) Meckel diverticulum Trauma (mostly penetrating)
Large bowel and appendix	Ischemic bowel Diverticulitis Malignancy Ulcerative colitis and Crohn disease Appendicitis Colonic volvulus Trauma (mostly penetrating)

Iatrogenic

Uterus, salpinx, and ovaries Pelvic inflammatory disease (eg, salpingo-oophoritis, tubo-ovarian abscess, ovarian cyst)
Malignancy (rare)
Trauma (uncommon)

* Iatrogenic trauma to the upper GI tract, including the pancreas and biliary tract and colon, often results from endoscopic procedures; anastomotic dehiscence and inadvertent bowel injury (eg, mechanical, thermal) are common causes of leak in the postoperative period.

The most common cause of postoperative peritonitis is anastomotic leak, with symptoms generally appearing around postoperative days 5-7. After elective abdominal operations for noninfectious etiologies, the incidence of SP (caused by anastomotic disruption, breakdown of enterotomy closures, or inadvertent bowel injury) should be less than 2%. Operations for inflammatory disease (ie, appendicitis, diverticulitis, cholecystitis) without perforation carry a risk of less than 10% for the development of SP and peritoneal abscess. This risk may rise to greater than 50% in gangrenous bowel disease and visceral perforation.

After operations for penetrating abdominal trauma, SP and abscess formation are observed in a small number of patients. Duodenal and pancreatic involvement, as well as colon perforation, gross peritoneal contamination, perioperative shock, and massive transfusion, are factors that increase the risk of infection in these cases.

Peritonitis is also a frequent complication and significant limitation of peritoneal dialysis. Peritonitis leads to increased hospitalization and mortality rates.

Tertiary peritonitis.

Tertiary peritonitis (see Table 3, below) develops more frequently in immunocompromised patients and in persons with significant preexisting comorbid conditions. Although rarely observed in uncomplicated peritoneal infections, the incidence of tertiary peritonitis in patients requiring ICU admission for severe abdominal infections may be as high as 50-74%.

Tuberculous peritonitis (TP) is rare in the United States (< 2% of all causes of peritonitis), but it

continues to be a significant problem in developing countries and among patients with human immunodeficiency virus (HIV) infection. The presenting symptoms are often nonspecific and insidious in onset (eg, low-grade fever, anorexia, weight loss). Many patients with TP have underlying cirrhosis. More than 95% of patients with TP have evidence of ascites on imaging studies, and more than half of these patients have clinically apparent ascites.

In most cases, chest radiographic findings in patients with TP peritonitis are abnormal; active pulmonary disease is uncommon (< 30%). Results on Gram stain of ascitic fluid are rarely positive, and culture results may be falsely negative in up to 80% of patients. A peritoneal fluid protein level greater than 2.5 g/dL, a lactate dehydrogenase (LDH) level greater than 90 U/mL, or a predominantly mononuclear cell count of greater than 500 cells/ μ L should raise suspicion of TP but have limited specificity for the diagnosis. Laparoscopy and visualization of granulomas on peritoneal biopsy specimens, as well as cultures (requires 4-6 wk), may be needed for the definitive diagnosis; however, empiric therapy should begin immediately.

Chemical peritonitis. Chemical (sterile) peritonitis may be caused by irritants such as bile, blood, barium, or other substances or by transmural inflammation of visceral organs (eg, Crohn disease) without bacterial inoculation of the peritoneal cavity. Clinical signs and symptoms are indistinguishable from those of SP or peritoneal abscess, and the diagnostic and therapeutic approach should be the same.

Peritoneal abscess. Peritoneal abscess describes the formation of an infected fluid collection encapsulated by fibrinous exudate, omentum, and/or adjacent visceral organs. The overwhelming majority of abscesses occurs subsequent to SP. Abscess formation may be a complication of surgery. The incidence of abscess formation after abdominal surgery is less than 1-2%, even when the operation is performed for an acute inflammatory process. The risk of abscess increases to 10-30% in cases of preoperative perforation of the hollow viscus, significant fecal contamination of the peritoneal cavity, bowel ischemia, delayed diagnosis and therapy of the initial peritonitis, and the need for reoperation, as well as in the setting of immunosuppression. Abscess formation is the leading cause of persistent infection and development of tertiary peritonitis.

Symptomatology and clinical course

The clinical picture of acute peritonitis is determined by the character of primary causative lesion, duration of inflammatory process, its extension and also the stage of the disease. Predominant clinical sign is the abdominal pain, which gradually increases. Firstly it is localized in the region of the source of peritonitis and then extends all over the abdomen. Elderly patients may experience lacking pain and even pay no attention on it, but general malaise, loss of appetite, and weakness are evident. This course is also characteristic for postoperative peritonitis, which results from parting of sutures (of anastomosis or site of perforation) or leaking colon carcinoma. Simultaneously with the increase of pain also change the general appearance. The patient looks anxious, with drawn features, hollowed-eyed. Further this is accompanied by nausea and vomiting: on initial stages vomit is of gastric contents, later – duodenal and thereafter is of

intestinal contents. With progression of the disease vomiting becomes constant, effortless and overcomes into frequent regurgitation by brown fluid with foul-smelling. Patient's lips and tongue are dry, with brown fur. Respiration is of thoracic type and is shallow and rapid. In order to prevent pain the patient speaks very quite. Every change of position results in increase of pain, thus the patient lies with the knee drawn up to relax the abdominal wall.

Often the vomiting is accompanied by hiccup, which results from irritation of diaphragmatic peritoneum. This is considered to be an unfavorable prognostic sign. The patient tries to retain distended abdomen by his hands during hiccup and thus provokes increase of pain.

During examination observed restricted movements of abdominal wall, which is mainly expressed over the inflammatory focus. Abdominal percussion reveals the region of maximal painfulness, which response the site of lesion, high tympanic sound as a result of intestinal gaseous dilatation, but sometimes dullness, caused by cumulation of great amount of exudate. On palpation revealed muscular tension of abdominal wall. Especially expressed the muscular rigidity in case of perforation of hollow organs ("board-like abdomen"). Pelvic location of peritonitis usually causes less clinical manifestations. In such cases a diagnostic value has digital examination of the rectum and bimanual palpation of the pelvis and lower abdomen, which reveals overhanging and painfulness of anterior rectal wall or posterior vaginal vault owing to accumulation of the exudate.

The clinical manifestation of peritonitis is various and individual. It depends on the character of primary lesion, extension of inflammatory process, and defensive properties of the organism. The main manifestations of peritonitis are acute abdominal pain, abdominal tenderness, and abdominal guarding, which are exacerbated by moving the peritoneum, e.g., coughing (forced cough may be used as a test), flexing one's hips, or eliciting the Blumberg sign (a.k.a. rebound tenderness, meaning that pressing a hand on the abdomen elicits less pain than releasing the hand abruptly, which will aggravate the pain, as the peritoneum snaps back into place). The presence of these signs in a patient is sometimes referred to as peritonism. The localization of these manifestations depends on whether peritonitis is localized (e.g., appendicitis or diverticulitis before perforation), or generalized to the whole abdomen. In either case, pain typically starts as a generalized abdominal pain (with involvement of poorly localizing innervation of the visceral peritoneal layer), and may become localized later (with the involvement of the somatically innervated parietal peritoneal layer). Peritonitis is an example of an acute abdomen.

Collateral manifestations: diffuse abdominal rigidity ("washboard abdomen") is often present, especially in generalized peritonitis, Fever, Sinus tachycardia, Development of ileus paralyticus (i.e., intestinal paralysis), which also causes nausea, vomiting and bloating.

In reactive stage of the disease the most common are the pain, muscular rigidity and positive Shchotkin-Blumberg's symptom. The general state changed a little – the patient is active, sometimes excite. A moderate tachycardia and hypertension commonly observed.(Fig.1)



Fig.1. Shchotkin-Blumberg's symptom.

In toxic stage of the disease the pain and muscular defense tend to diminish, but on palpation the muscular tenderness and Shchotkin-Blumberg's symptom retain on the same level. More evident the signs of intestinal paresis (abdominal distension, absence of peristalsis). The general state is worsened. The patient is apathetic, the skin is blanched or cyanotic. Observed progressing of tachycardia, decreasing of blood pressure and rising of temperature. In blood analysis revealed leukocytosis and deviation of the differential

count to the left.

In terminal stage of the disease the feeling of pain disappears, but the patient suffer from the uncontrollable vomiting by congested fecal contents. The patient is adynamic, with drawn features and blanched or cyanotic skin. The pulse becomes increasingly rapid small and thready. The arterial pressure tends to diminish. No peristalsis is evident and no bowel sounds are heard on auscultation. Shchotkin-Blumberg's symptom is slightly expressed. The respiration is rapid, with congested rales, and oliguria develops. This clinical pattern resembles a septic shock. The prognosis in this stage is serious and the patient will die if the urgent treatment is not be applied.

Plain films of the chest and abdomen with the patient in both supine and the erect position are essential. The chest x-ray examination assists in identifying thoracic causes of the acute abdomen and sometimes reveals specific x-ray findings of intraabdominal catastrophes (e.g. free air under the diaphragm associated with perforation of the gastrointestinal tract).

Laparoscopy is a rapid, direct, and often definitive method of identifying the cause of peritonitis in difficult cases. Finally, for patients who have acute intraabdominal problem of unknown nature and whose symptoms, signs, and laboratory findings are suggestive of a threat of life, exploratory laparotomy remains the most prudent diagnostic procedure.

Complications:

Sequestration of fluid and electrolytes, as revealed by decreased central venous pressure, may cause electrolyte disturbances, as well as significant hypovolemia, possibly leading to shock and acute renal failure.

A peritoneal abscess may form (e.g., above or below the liver, or in the lesser omentum

Sepsis may develop, so blood cultures should be obtained.

Variants of clinical course and complications

Postoperative peritonitis is characterized by atypical and even asymptomatic course. This results from administering of analgesics, antibiotics and anesthetics. The general state of the patient after the operation is gradually worsens.

The most earliest and frequent sign of postoperative peritonitis is the increase of abdominal pain on the background of the previous satisfactory condition, tachycardia, high temperature, leukocytosis, deviation of the differential count to the left, elevation of erythrocyte sedimentation rate. The pain and muscular rigidity usually expressed slightly or absent at all. Later (on the 5-6th day) the general state continues to be worsened, which manifest by dry tongue, lack of peristalsis, expressed nausea, vomiting, tachycardia and shallow breathing. General weakness, adynamia, general intoxication and rebound tenderness symptoms progress. The outcomes of postoperative peritonitis are usually unfavorable, and they prevented by early repeated operation.

The specific complications of acute peritonitis include inflammatory infiltrates and abscesses of abdominal cavity (Fig.2) (subphrenic, subhepatic, interintestinal and pelvic), dynamic ileus, intestinal fistula, suppuration of postoperative wound, eventration, peritoneal adhesions, etc.

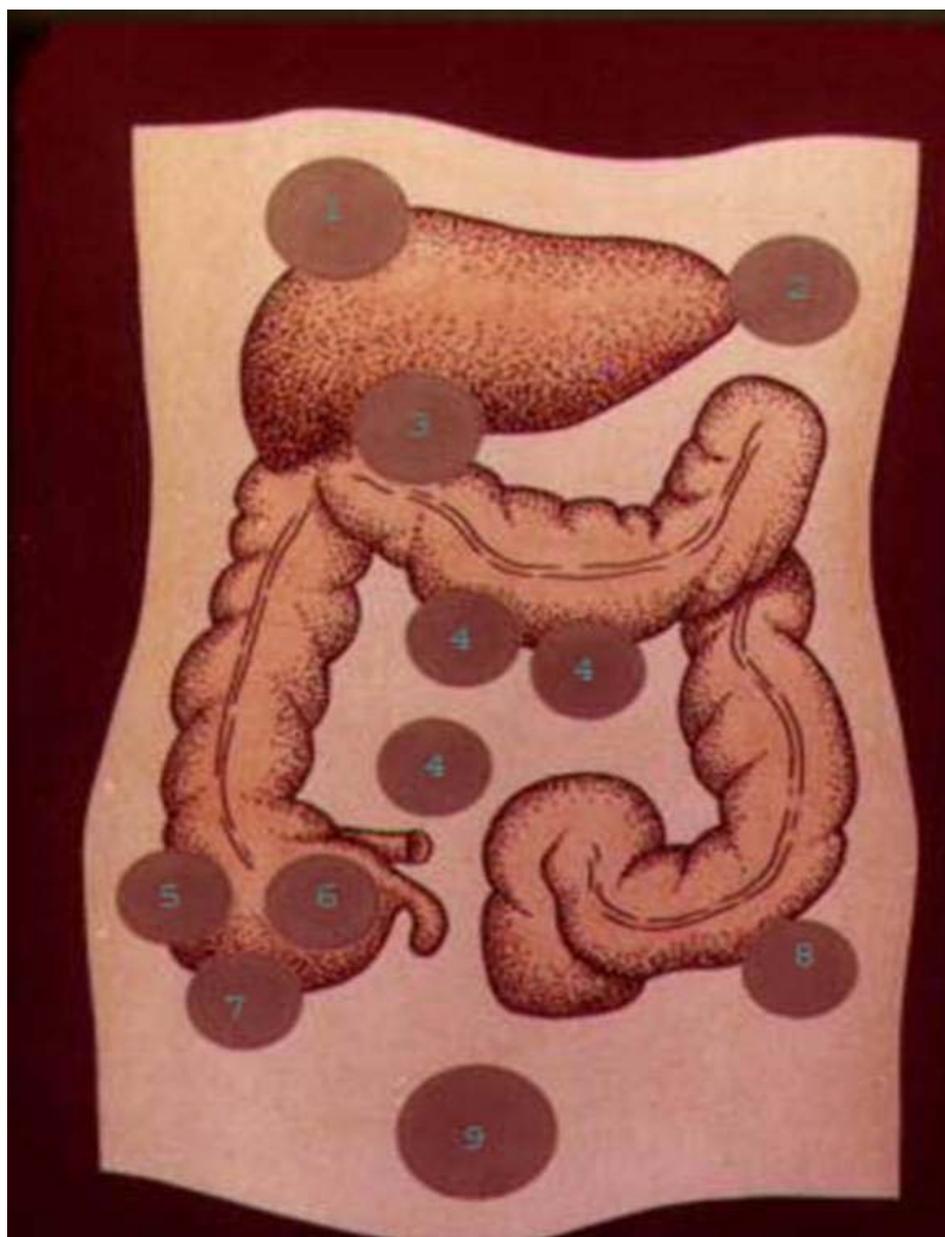


Fig.2. Localizations of abscesses of abdominal cavity

1, 2 – Right and left subdiaaphragmatic abscesses

3 - Subhepatic abscess

4 – Interintestinal abscesses

5, 6, 7 – Right iliac abscesses

8 – Left iliac abscess

9 – Abscess of the small pelvis (Douglas space abscess)

The patients with subphrenic abscess (Fig.3) as a rule complain of the pain in epigastrium and lower

chest, which irradiates into the shoulder and increases during cough and deep breathing. Sometimes revealed painfulness during digital pressing and swelling of soft tissues in the region of 7-10th Intercostals space. The patients are suffering from nausea, hiccup, and high temperature. Sometimes they must stay in forced position: supine or semisedentary. The tongue is dry, the abdomen is slightly bloated, and rebound tenderness symptoms are usually absent. In blood revealed leukocytosis, deviation of the differential count to the left. The abscess requires a surgical treatment. If the abscess is located near anterior abdominal wall, it is drained by means of oblique access under the costal arch. The abscesses, which located in posterior subphrenic space, are drained after the previous puncture through the access after resection of X rib. (Fig.4)



Fig.3. Subphrenic abscess



Fig.4. Drainage of subdiafragmatic abscess

Subhepatic abscess is characterized by the pain and presence of infiltrate below right costal arch, positive Shchotkin-Blumberg symptom. The abscess is drained through the incision along right costal arch.

The clinical pattern of interintestinal abscess is vague. It is formed mostly on the 12-14th day after appearance of peritonitis. The patients complain of the high temperature and dull pain in the site of its location. The abdomen is soft, but during palpation revealed dense, painful infiltrate. In case of localization near to abdominal wall one can observe muscular tension and positive Shchotkin-Blumberg symptom. The roentgenological or ultrasound investigation often reveals focal shadow with air-fluid level. The abscess is drained over the site of its localization, dividing the bowel loops.

Abscesses of small pelvis mostly occur as a result of appendicitis(Fig.5) or accumulation of the exudates in Douglas space in diffuse peritonitis.

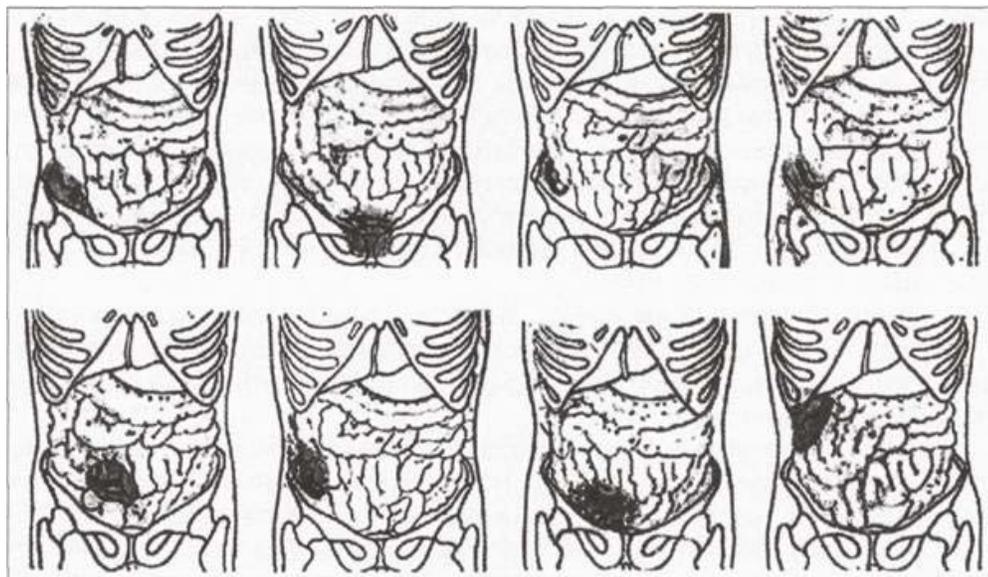
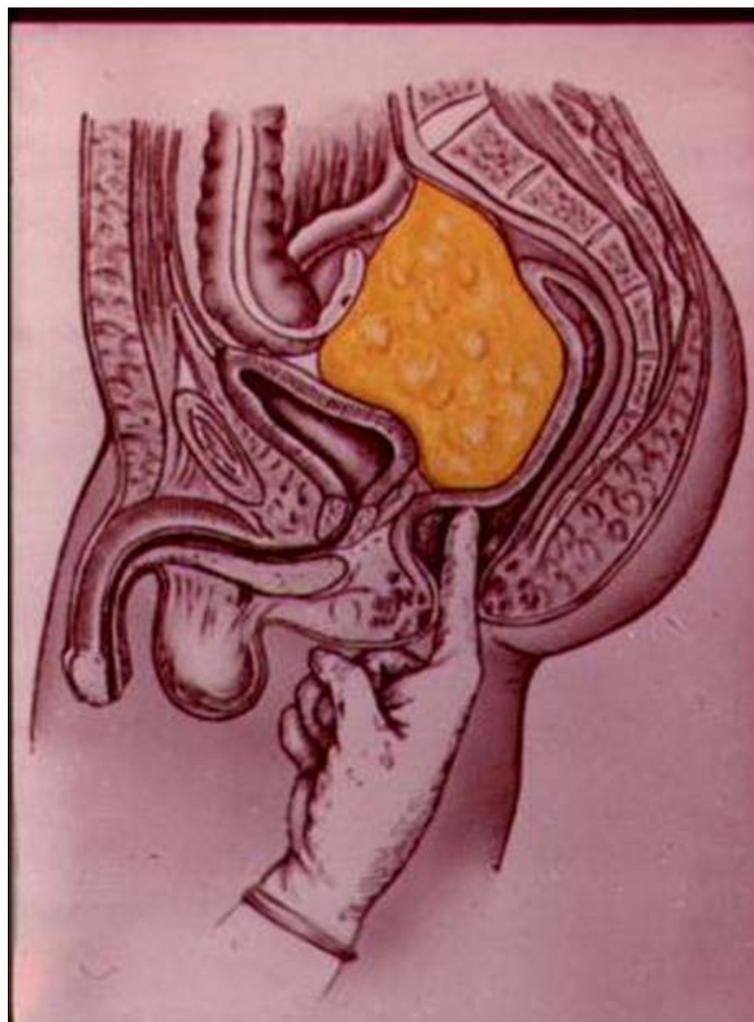
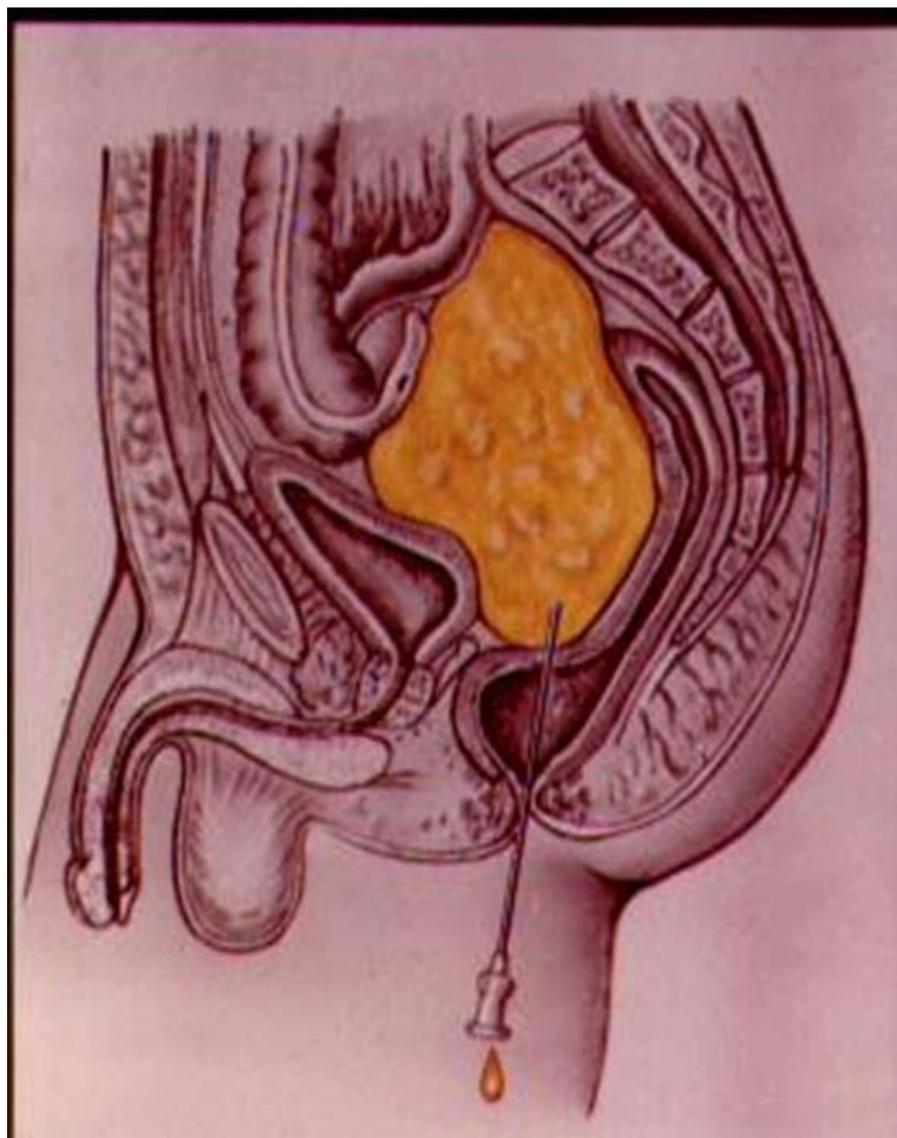


Fig.5. The localization of appendiceal abscess
depending the appendix location

Such patients complain of constant pain in the lower abdomen, high temperature, painful urinary excretion and tenesmus. The palpation of the abdomen usually reveals no pathology. But the digital rectal examination finds out a painful infiltrate that drawn into the rectum. (Fig.6) The mucosa over the infiltrate is edematous and immovable. The vaginal examination of the female patients reveals overhanging of posterior vaginal vault and painfulness of cervical shift. Often on the background of solid consistency of the infiltrate the softened regions are palpated, which respond to accumulation of pus. The purulent sites of small pelvis in males are drained through the anterior wall of the rectum and in females – through the posterior vaginal vault. For this purpose the infiltrate is punctured by thick needle and under its check the abscess is drained by means of scalpel incision. Then the incision is expanded by clamp, the pus is aspirated and the abscess cavity is drained by rubber strap, which is fixed to perineum.



A



B

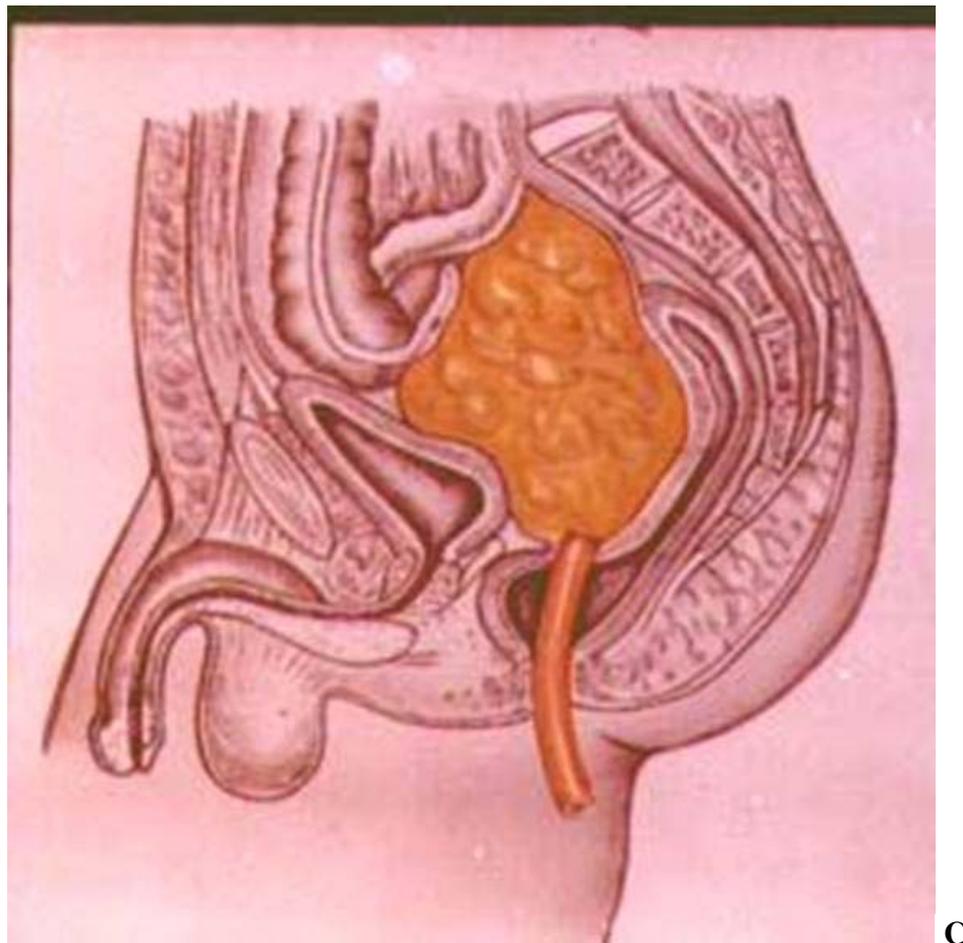


Fig. 6. Diagnostic and management of Douglas space abscess

A - Digital rectal examination

B – Diagnostic puncture through anterior rectal wall

C – The drainage is placed through anterior rectal wall

The diagnostic program

Complaints and history of the disease.

Physical findings.

General blood and urine analyses.

Biochemical blood analysis (protein and its fractions).

Examination of the exudate (bacteriological, cytological).

Laparoscopy. (Fig.7)

Plain film of the abdomen.

Laparocentesis.

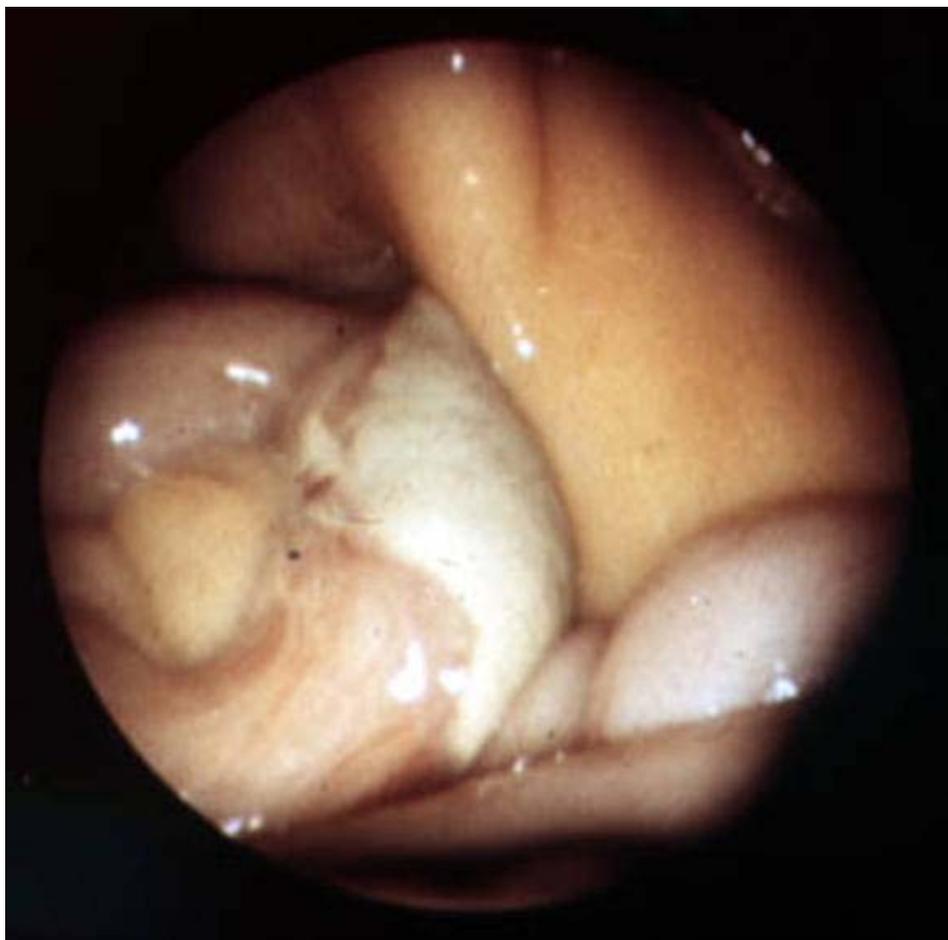


Fig.7. Purulent peritonitis(Laparoscopic picture)

Tests and diagnosis

To diagnose peritonitis, your doctor will talk with you about your medical history and perform a physical exam. When peritonitis is associated with peritoneal dialysis, your signs and symptoms, particularly cloudy dialysis fluid, may be enough for your doctor to diagnose the condition.

In cases of peritonitis in which the infection may be a result of other medical conditions (secondary peritonitis) or in which the infection arises from fluid buildup in your abdominal cavity (spontaneous peritonitis), your doctor may recommend the following tests to confirm a diagnosis:

Peritoneal fluid analysis. Using a thin needle, your doctor may take a sample of the fluid in your peritoneum (paracentesis). If you have peritonitis, examination of this fluid may show an increased white blood cell count, which typically indicates an infection or inflammation. A culture of the fluid may also reveal the presence of bacteria.

Diagnostic paracentesis should be performed in all patients who do not have an indwelling peritoneal

catheter and are suspected of having SBP. In peritoneal dialysis patients with a peritoneal catheter, fluid should be withdrawn using sterile technique. Ultrasonography may aid paracentesis if ascites is minimally detectable or questionable.

The results of aerobic and anaerobic bacterial cultures, used in conjunction with the cell count, prove the most useful in guiding therapy for those with SBP. With regard to ascitic fluid culture, direct inoculation of routine blood culture bottles at the bedside with 10 mL of ascitic fluid has been reported to significantly increase the sensitivity of microbiologic studies.

The single best predictor of SBP is an ascitic fluid neutrophil count of greater than 500 cells/ μ L, which carries a sensitivity of 86% and a specificity of 98%. By lowering the ascitic fluid neutrophil count threshold to 250 cells/ μ L, the sensitivity increases to 93% with only a minimal decrease in specificity to 94%.

The fluid should be evaluated for glucose, protein, lactate dehydrogenase (LDH), cell count, Gram stain, and aerobic and anaerobic cultures. If pancreatitis or a pancreatic leak is suspected, amylase analysis should be added to the panel. Bilirubin and creatinine levels can be analyzed as well, if a biliary or urinary leak is suspected as a possible etiology. The peritoneal/ascitic fluid characteristics or levels are then compared with their respective serum values.

The fluid in bacterial peritonitis generally demonstrates a low pH and low glucose levels with elevated protein and LDH levels. Traditionally, ascitic fluid pH of less than 7.34 was consistent with a diagnosis of SBP; however, ascitic pH is less commonly measured because it is unreliable and lacks specificity for the condition.

SBP is established when the polymorphonuclear neutrophil (PMN) count is 250 cells/ μ L or greater in conjunction with a positive bacterial culture result. In most of these cases, as mentioned previously, cultures are positive for a single organism. Obviously, these patients should receive antibiotic therapy. Although up to 30% of cultures remain negative, most of these patients are presumed to have bacterial peritonitis; they should be treated. A significantly decreased peritoneal fluid glucose level (< 50 mg/dL), a peritoneal fluid LDH level much greater than the serum LDH, a peritoneal fluid WBC count greater than 10,000 cells/ μ L, a pH lower than 7.0, high amylase levels, multiple organisms on Gram stain, or recovery of anaerobes from the culture raises the suspicion of SP in these patients. Some authors recommend repeating the paracentesis in 48-72 hours to monitor treatment success (decrease in neutrophil count to $< 50\%$ of the original value).

Culture-negative neutrocytic ascites (probable SBP) is established when the ascitic fluid culture results are negative but the PMN count is 250 cells/ μ L or greater. This may happen in as many as 50% of patients with SBP and may not actually represent a distinctly different disease entity. Rather, it may be the result of poor culturing techniques or late-stage resolving infection. Nonetheless, these patients should be treated just as aggressively as those with positive culture results.

Monomicrobial nonneutrocytic bacterascites exists when a positive culture result coexists with a PMN count 250 cells/ μ L or greater. Although this may often be the result of contamination of bacterial cultures, 38% of these patients develop SBP. Therefore, monomicrobial nonneutrocytic bacterascites may represent an early form of SBP. All study patients described who eventually developed SBP were symptomatic. For this

reason, any patient suspected clinically of having SBP in this setting must be treated.

Tuberculous peritonitis is identified by ascites with high protein content, a low glucose and low SAAG, elevated ascitic fluid WBC count, and lymphocyte predominance. In TP, the fluid Gram stain and acid-fast stain results are rarely positive, and routine culture results are falsely negative in as many as 80% of cases. A peritoneal fluid protein level greater than 2.5 g/dL, LDH level greater than 90 U/mL, and predominantly mononuclear cell count of more than 500 cells/ μ L should raise the suspicion of TP, but specificity for the diagnosis is limited. Laparoscopy with visualization of granulomas on peritoneal biopsy and specific culture (which requires 4-6 wk) may be needed for definitive diagnosis.

Peritonitis in patients receiving continuous ambulatory peritoneal dialysis (CAPD) is indicated by contamination of the dialysis catheter; cloudy effluent, total fluid WBC count of greater than 100 neutrophils/ μ L, or presence of organisms on Gram stain.

Routine intraoperative peritoneal fluid cultures in defined acute disease entities (ie, gastric or duodenal ulcer perforation, appendicitis, diverticulitis or perforation of the colon caused by obstruction or ischemia) are controversial. Several studies found no significant difference in patients with appendicitis, diverticulitis, and other common etiologies for bacterial peritonitis with regard to postoperative complication rates or overall outcomes. The antibiotic regimen was altered only 8-10% of the time based on operative culture data. In patients who had previous abdominal operations or instrumentation (eg, peritoneal dialysis catheter, percutaneous stents) and patients with prolonged antibiotic therapy, critical illness, and/or hospitalization, these cultures may reveal resistant or unusual organisms that should prompt alteration of the antibiotic strategy.

For a summary of ascitic fluid analysis, see table, below.

Table. Ascitic Fluid Analysis Summary

Routine	Optional	Unusual	Less Helpful
Cell count	Obtain culture in blood culture (BC) bottles.	Tuberculosis smear and culture	(TB) pH
Albumin	Glucose	Cytology	Lactate
Total protein	Lactate dehydrogenase (LDH)	Triglyceride	Cholesterol
	Amylase	Bilirubin	Fibronectin
	Gram stain		Alpha 1-antitrypsin

Glycosaminoglycans

Blood tests. A sample of your blood may be drawn and sent to a lab to check for a high white blood cell count. A blood culture also may be performed to determine if there are bacteria in your blood.

Most patients will have leukocytosis ($>11,000$ cells/ μ L), with a shift to the immature forms on the differential cell count. Patients who have severe sepsis, are immunocompromised, or have certain types of infections (eg, fungal, cytomegaloviral) may not have leukocytosis or leukopenia. In cases of suspected SBP, hypersplenism may reduce the polymorphonuclear leukocyte count.

Blood chemistry findings may reveal dehydration and acidosis. PT, PTT, and INR are indicated. Liver function tests may be indicated. Amylase and lipase levels should be obtained if pancreatitis is suspected. Blood culture results are positive for the offending agent in as many as 33% of patients with SBP and may help guide antibiotic therapy. Measurement of serum albumin allows calculation of the serum-to-ascites albumin gradient (SAAG). A SAAG of more than 1.1 is noted in SBP.

Imaging tests. Your doctor may want to use an X-ray to check for holes or other perforations in your gastrointestinal tract. Ultrasound may also be used. In some cases, your doctor may use a computerized tomography (CT) scan instead of an X-ray.

Radiography. Plain films of the abdomen (eg, supine, upright, and lateral decubitus positions) are often the first imaging studies obtained in patients presenting with peritonitis. Their value in reaching a specific diagnosis is limited.

Ultrasonography. Abdominal ultrasonography may be helpful in the evaluation of pathology in the right upper quadrant (eg, perihepatic abscess, cholecystitis, biloma, pancreatitis, pancreatic pseudocyst), right lower quadrant, and pelvis (eg, appendicitis, tubo-ovarian abscess, Douglas pouch abscess). However, the examination is sometimes limited because of patient discomfort, abdominal distention, and bowel gas interference. Ultrasonography may detect increased amounts of peritoneal fluid (ascites), but its ability to detect quantities of less than 100 mL is limited. The central (perimesenteric) peritoneal cavity is not visualized well with transabdominal ultrasonography. Examination from the flank or back may improve the diagnostic yield, and providing the ultrasonographer with specific information about the patient's condition and the suspected diagnosis before the examination is important. With an experienced ultrasonographer, a diagnostic accuracy of greater than 85% has been reported in several series. Ultrasonographically guided aspiration and placement of drains has evolved into a valuable tool in the diagnosis and treatment of abdominal fluid collections. Advantages of ultrasound include low cost, portability, and availability. Disadvantages are that the test is operator dependent, and there is reduced visualization in the presence of overlying bowel gas and abdominal dressings.

CT scanning. If the diagnosis of peritonitis is made clinically, a CT scan is not necessary and generally delays surgical intervention without offering clinical advantage. However, CT scanning is indicated in all

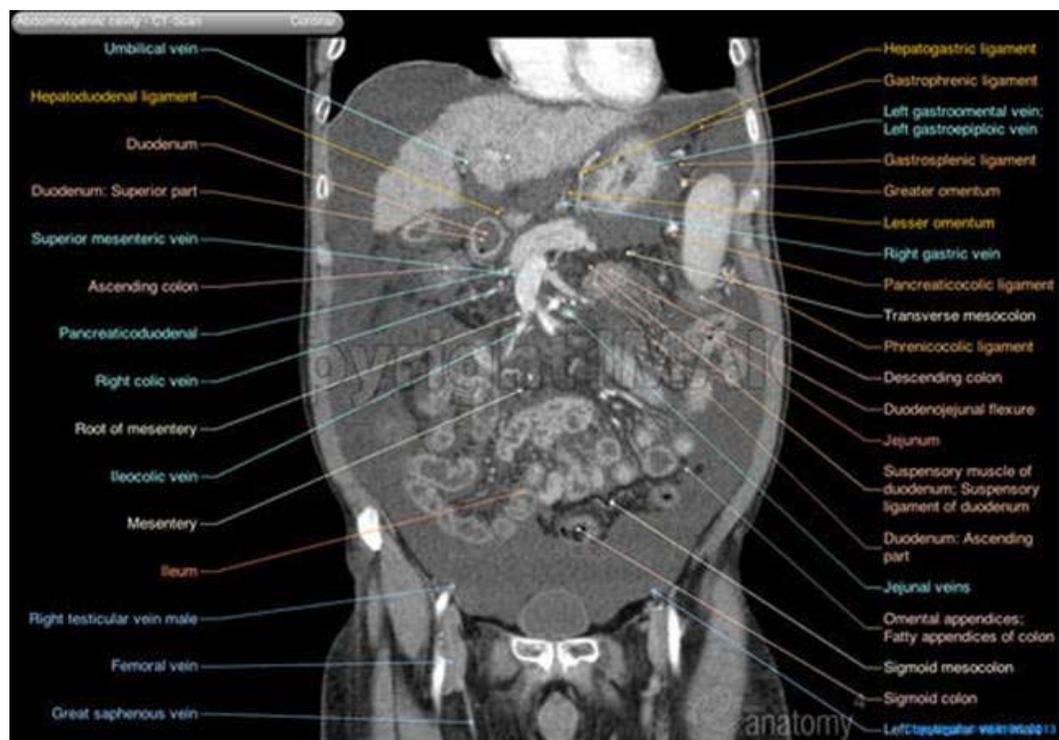
cases in which the diagnosis cannot be established on clinical grounds and findings on abdominal plain films. CT scans of the abdomen and pelvis remain the diagnostic study of choice for peritoneal abscess and related visceral pathology.

Whenever possible, the CT scan should be performed with enteral and intravenous contrast. CT scans can detect small quantities of fluid, areas of inflammation, and other GI tract pathology, with sensitivities that approach 100%. (See the image below.) CT scanning can be used to evaluate for ischemia, as well as to determine bowel obstruction. An abscess is suggested by the presence of fluid density that is not bound by the bowel or other known structures. Gas within an abdominal mass or the presence of an enhancing wall and adjacent inflammatory changes are also highly suggestive of an abscess. Ischemia can be demonstrated by a clot in a large vessel or by the absence of blood flow. Gas within the intestinal wall or in the portal vein may also suggest ischemia.

Free air is present in most cases of anterior gastric and duodenal perforation but is much less frequent with perforations of the small bowel and colon and is unusual with appendiceal perforation. Upright films are useful for identifying free air under the diaphragm (most often on the right) as an indication of a perforated viscus. Remember that the presence of free air is not mandatory with visceral perforation and that small amounts of free air are missed easily on plain films.

In abscess formation subsequent to secondary peritonitis (SP), approximately half of patients have a simple abscess without loculation, and the other half have complex abscesses secondary to fibrinous septation and organization of the abscess material. Abscess formation occurs most frequently in the subhepatic area, the pelvis, and the paracolic gutters, but it may also occur in the perisplenic area, the lesser sac, and between small bowel loops and their mesentery.

Peritoneal abscesses and other fluid collections may be aspirated for diagnosis and drained under CT guidance; this technique has become a mainstay of therapy.



MRI. MRI is an emerging imaging modality for the diagnosis of suspected intra-abdominal abscesses. Abdominal abscesses demonstrate decreased signal intensity on T1-weighted images and homogeneous or heterogeneous increased signal intensity on T2-weighted images; abscesses are observed best on gadolinium-enhanced, T1-weighted, fat-suppressed images as well-defined fluid collections with rim enhancement.

Limited availability and high cost, as well as the need for MRI-compatible patient support equipment and the length of the examination, currently limit its usefulness as a diagnostic tool in acute peritoneal infections, particularly for patients who are critically ill.

The above tests may also be necessary if you're receiving peritoneal dialysis and a diagnosis of peritonitis is uncertain after a physical exam and an examination of the dialysis fluid.\

Differential diagnostics

Thoracic processes with diaphragmatic irritation (eg, empyema), extraperitoneal processes (eg, pyelonephritis, cystitis, acute urinary retention), and abdominal wall processes (eg, infection, rectus hematoma) may mimic certain signs and symptoms of peritonitis. Always examine the patient for the presence of external hernias to rule out intestinal incarceration.

According to Adler and Gasbarra, the following should be considered in the differential diagnosis:

- Aneurysm, Abdominal
- Angioedema
- Appendicitis, Acute
- Mesenteric Ischemia
- Urinary Tract Infection in Females
- Whipple Disease
- Chemical irritants (eg, bile, blood, gastric juice, barium, enema or douche contents)
- Chronic peritoneal dialysis
- Chylous peritonitis
- Eosinophilic peritonitis
- Familial Mediterranean fever
- Fungal infections (eg, histoplasmosis, cryptococcosis, coccidioidomycosis)
- Granulomatous peritonitis (eg, parasitic infestations, sarcoidosis, tumors, Crohn disease, starch granules)
- Gynecologic disorders (Chlamydia peritonitis, salpingitis, endometriosis, teratoma, leiomyomatosis, dermoid cyst)
- HIV-associated peritonitis (from opportunistic organisms)
- Mesothelial hyperplasia and metaplasia
- Neoplasms (eg, primary mesothelioma, secondary carcinomatosis, Pseudomyxoma peritonei)
- Parasitic infections (eg, schistosomiasis, ascariasis, enterobiasis, amebiasis, strongyloidiasis)
- Perforated viscus
- Peritoneal encapsulation

Peritoneal loose bodies and peritoneal cysts

Peritoneal lymphangiectasis

Pyelonephritis

Sclerosing peritonitis

Splenosis

Vascular conditions (eg, mesenteric embolus, mesenteric nonocclusive ischemia, ischemic colitis, portal vein thrombosis, mesenteric vein thrombosis)

Vasculitis (eg, systemic lupus erythematosus, allergic vasculitis [Henoch-Schönlein purpura], Kohlmeier-Degos disease, polyarteritis nodosa)

The differential diagnostics in toxic and terminal stage of peritonitis when the typical signs of the disease are present commonly makes no difficulties. But in initial (reactive) stage the signs are similar to manifestation of causative disease (appendicitis, cholecystitis, pancreatitis, etc.). But there are variety of disorders, which according to their manifestation resemble peritonitis, renal colic for instance. A sharp pain, nausea, vomiting, intestinal paralysis, and false Shchotkin-Blumberg symptom (peritonism) frequently lead to misdiagnostics. A periodical pain attack with typical irradiation in thigh, perineum, dysuria, positive Pasternatsky's symptom, lack of inflammatory changes in blood analysis, presence of erythrocytes in urine help to make correct diagnosis. For its improvement applied x-ray film of the abdomen, urography and chromocystoscopy.

A diffuse abdominal pain, muscular tension of abdominal wall and peritonism often accompany hemorrhagic diatheses (Schonlein-Henoch's disease). This disorder mostly occurs in young people and manifests by multiple small hemorrhages on skin (forearm, chest, and thigh), mucous membranes of cheeks, tongue and peritoneum as well. The rectal examination reveals tarry stool or melena. In blood thrombocytopenia is observed.

Myocardial infarction especially in its location on posterior wall (abdominal form) usually accompanied by epigastric pain, nausea and vomiting. Also revealed abdominal wall tension with phenomena of peritonism. But ischemic heart disease in history and characteristic ECG changes can favor correct diagnostics.

Basal pleurisy and acute lower lobe pneumonia, causing the pain and muscular guard in epigastrium, also resemble peritonitis. Only thorough clinical examination leads to correct diagnostics.

Tactics and choice of treatment

Approach considerations. The current approach to peritonitis and peritoneal abscesses targets correction

of the underlying process, administration of systemic antibiotics, and supportive therapy to prevent or limit secondary complications due to organ system failure. treatment success is defined as adequate source control with resolution of sepsis and clearance of all residual intra-abdominal infection.

Early control of the septic source is mandatory and can be achieved by operative and nonoperative means.

NUTRITION AND DIETARY SUPPLEMENTS

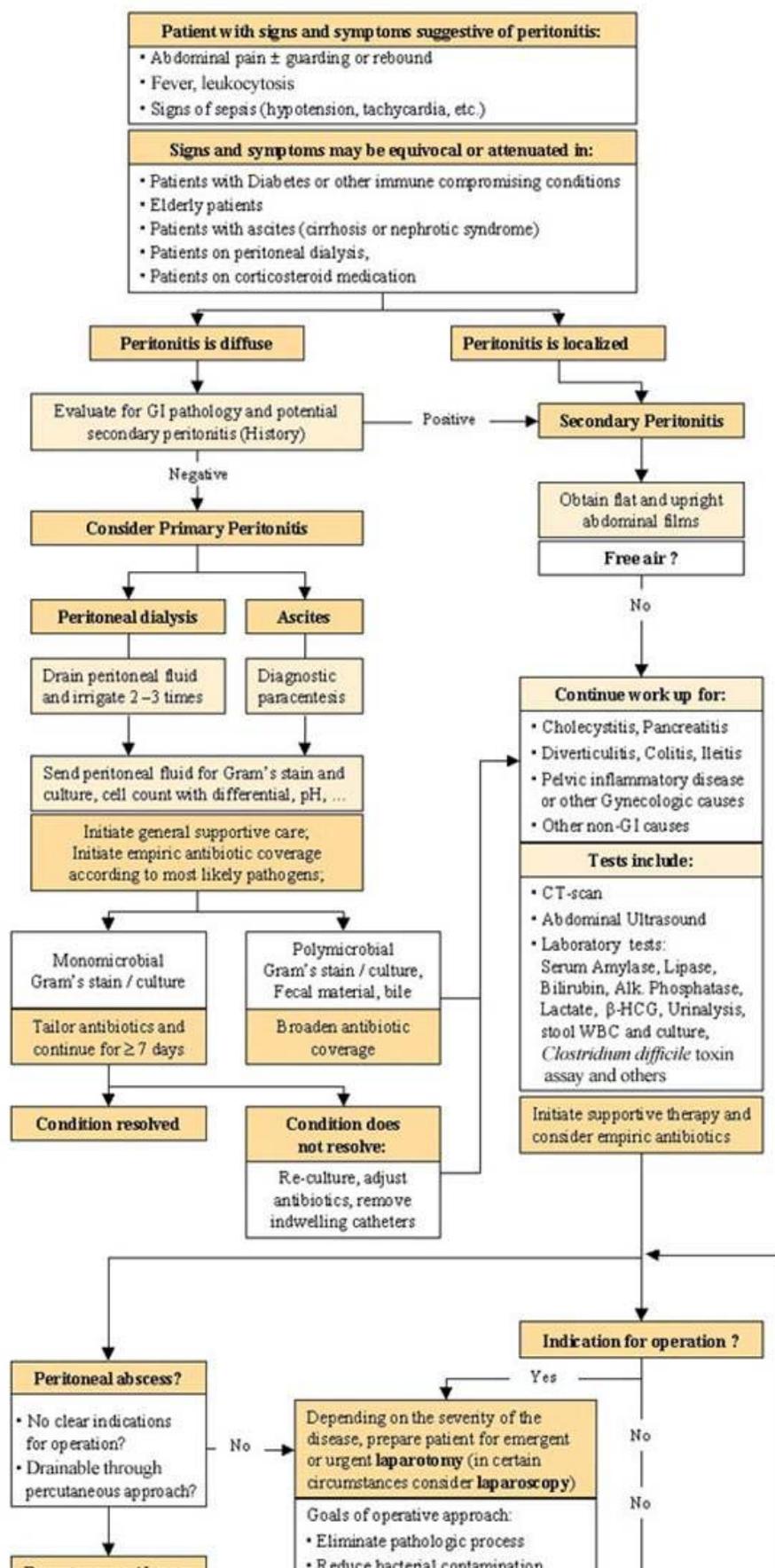
Peritonitis is a medical emergency and should be treated by a medical doctor. Do not try to treat peritonitis with herbs or supplements. However, a comprehensive treatment plan for recovering from peritonitis may include a range of complementary and alternative therapies. Ask your team of health care providers about the best ways to incorporate these therapies into your overall treatment plan. Always tell your health care provider about the herbs and supplements you are using or considering using.

These good nutrition habits may help you recover from any serious illness:

- Eat antioxidant foods, including fruits (such as blueberries, cherries, and tomatoes) and vegetables (such as squash and bell peppers).
- Eat foods high in B-vitamins and calcium, such as almonds, beans, whole grains (if no allergy), dark leafy greens (such as spinach and kale), and sea vegetables.
- Avoid refined foods, such as white breads, pastas, and especially sugar.
- Eat fewer red meats and more lean meats, cold-water fish, tofu, or beans for protein.
- Use healthy oils in foods, such as olive oil or vegetable oil.
- Avoid caffeine, alcohol, and tobacco.
- Drink 6 - 8 glasses of filtered water daily.
- Ask your doctor about taking a multivitamin daily, containing the antioxidant vitamins A, C, E, the B-complex vitamins, and trace minerals such as magnesium, calcium, zinc, and selenium.
- Probiotic supplement (containing *Lactobacillus acidophilus* among other species), 5 - 10 billion CFUs (colony forming units) a day, for gastrointestinal and immune health. Probiotics can be especially helpful when taking antibiotics, because probiotics can help restore the balance of "good" bacteria in the intestines.

Operative management addresses the need to control the infectious source and to purge bacteria and toxins.

Diagnostic and therapeutic approach to peritonitis and peritoneal abscess:



The type and extent of surgery depends on the underlying disease process and the severity of intra-abdominal infection. Definitive interventions to restore functional anatomy involve removing the source of the antimicrobial contamination and repairing the anatomic or functional disorder causing the infection. This is accomplished by surgical intervention. Occasionally, this can be achieved during a single operation; however, in certain situations, a second or a third procedure may be required. In some patients, definitive intervention is delayed until the condition of the patient improves and tissue healing is adequate to allow for a (sometimes) lengthy procedure.

To see complete information on the Surgical Approach to Peritonitis and Abdominal Sepsis, please go to the main article by clicking [here](#).

Nonoperative interventions include percutaneous abscess drainage, as well as percutaneous and endoscopic stent placements. If an abscess is accessible for percutaneous drainage and if the underlying visceral organ pathology does not clearly require operative intervention, percutaneous drainage is a safe and effective initial treatment approach. With percutaneous treatment, the definition of success includes the avoidance of further operative intervention and, in some cases, the delay of surgery until after resolution of the initial sepsis.

The general principles guiding the treatment of infections are 4-fold, as follows:

Control the infectious source

Eliminate bacteria and toxins

Maintain organ system function

Control the inflammatory process

The treatment of peritonitis is multidisciplinary, with complementary application of medical, operative, and nonoperative interventions. Medical support includes the following:

Systemic antibiotic therapy

Intensive care with hemodynamic, pulmonary, and renal support

Nutrition and metabolic support

Inflammatory response modulation therapy

Early control of the septic source is mandatory and can be achieved by operative and nonoperative means. Nonoperative interventional therapies include percutaneous drainage of abscesses and percutaneous and endoscopic stent placements.

Treatment of peritonitis and intra-abdominal sepsis always begins with volume resuscitation, correction of potential electrolyte and coagulation abnormalities, and empiric broad-spectrum parenteral antibiotic coverage.

The treatment of acute peritonitis should be always carried out with appreciation of clinical form and stage of the disease, causative factor, extension of inflammatory process, degree of metabolic disturbances

and dysfunction of vital organs of the patient.

The complex of treatment of peritonitis should include:

early operative approach in order to liquidate the source of peritonitis;

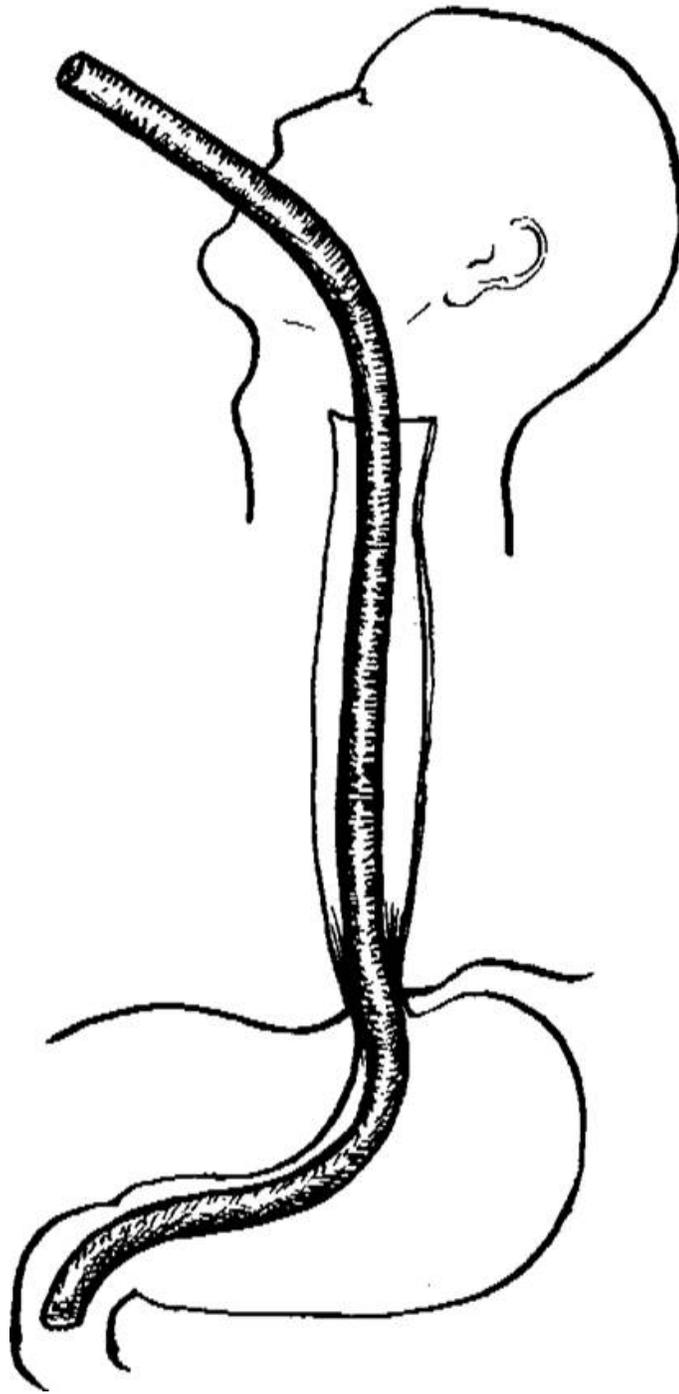
sanation of peritoneal cavity by means of lavage, adequate drainage and antibiotic therapy;

intubation and decompression of gastrointestinal tract and liquidation of paralytic ileus; (Fig.8)

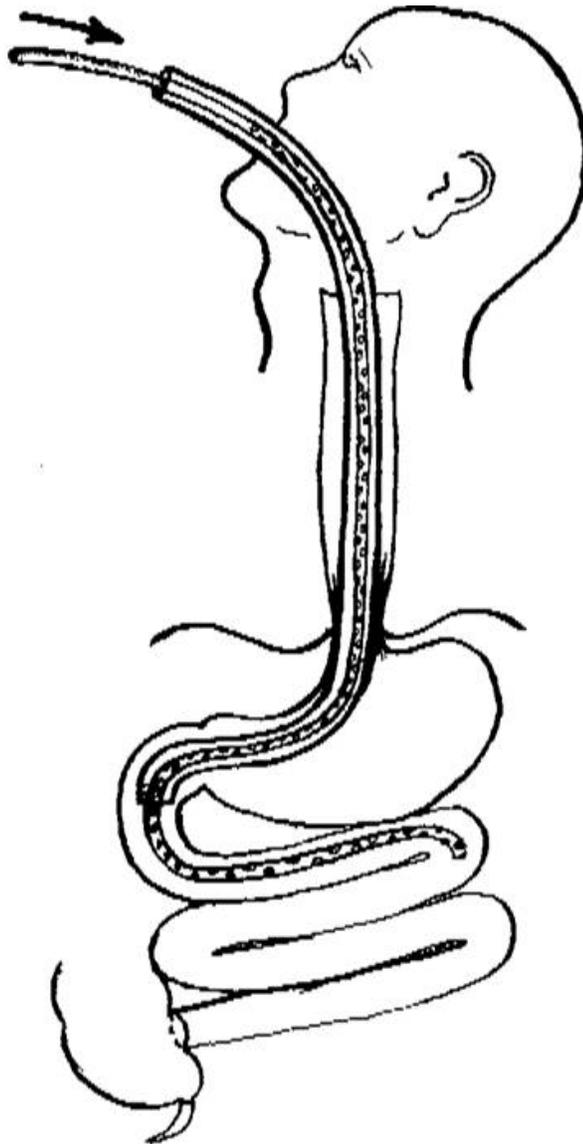
metabolic correction (acid-base balance, blood electrolytes, protein metabolism, energetic metabolism);

restore and support of visceral function (kidney, liver, heart, lung) and prevention of complications.

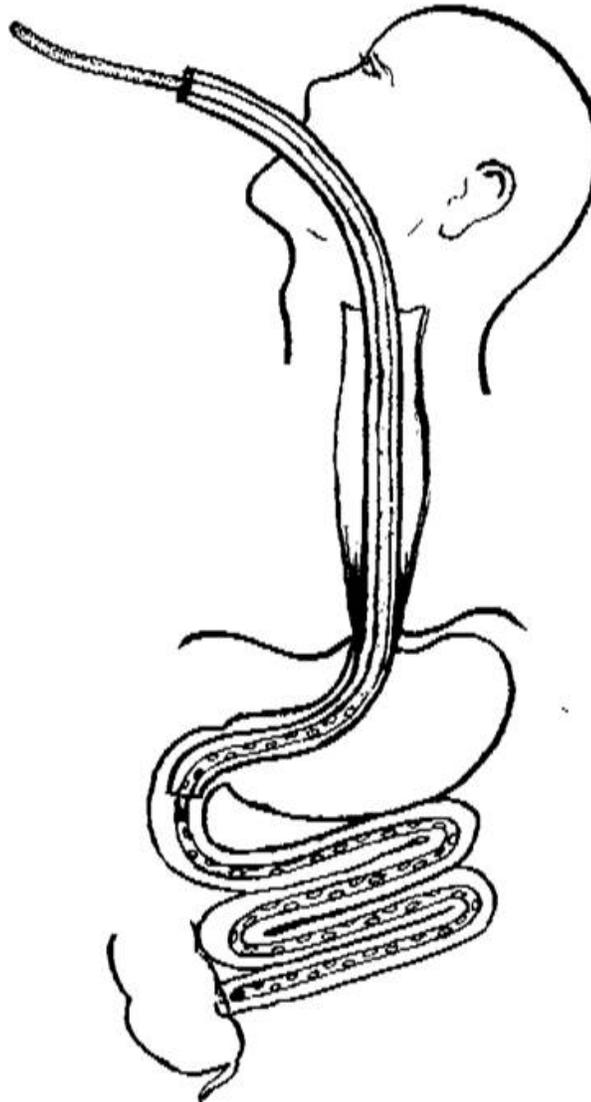
Fig.8. Principles of nasogastrintestinal intubation



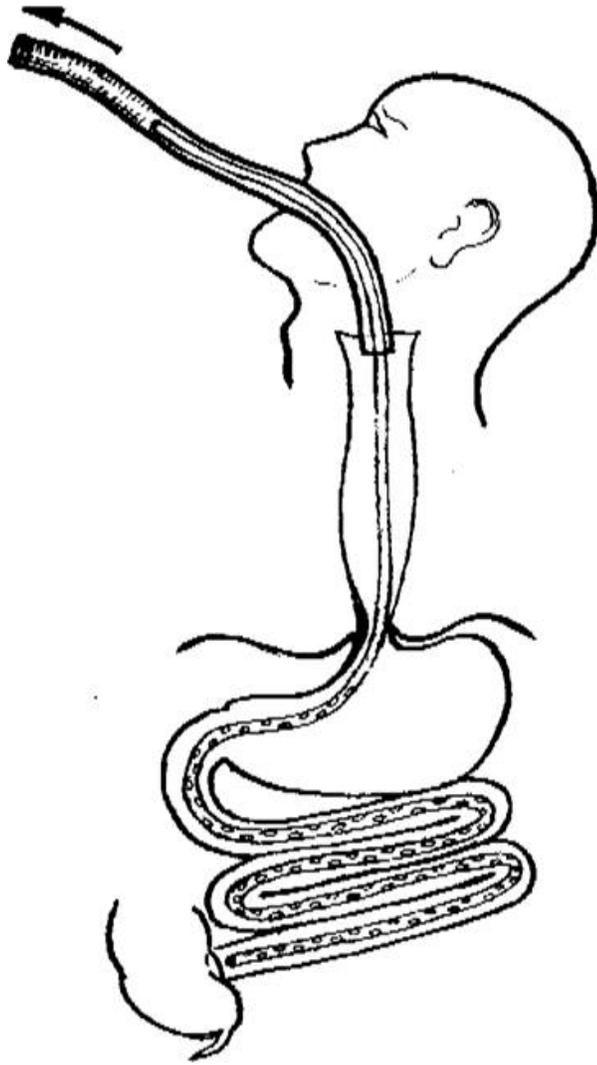
Gastric probe-guide is placed



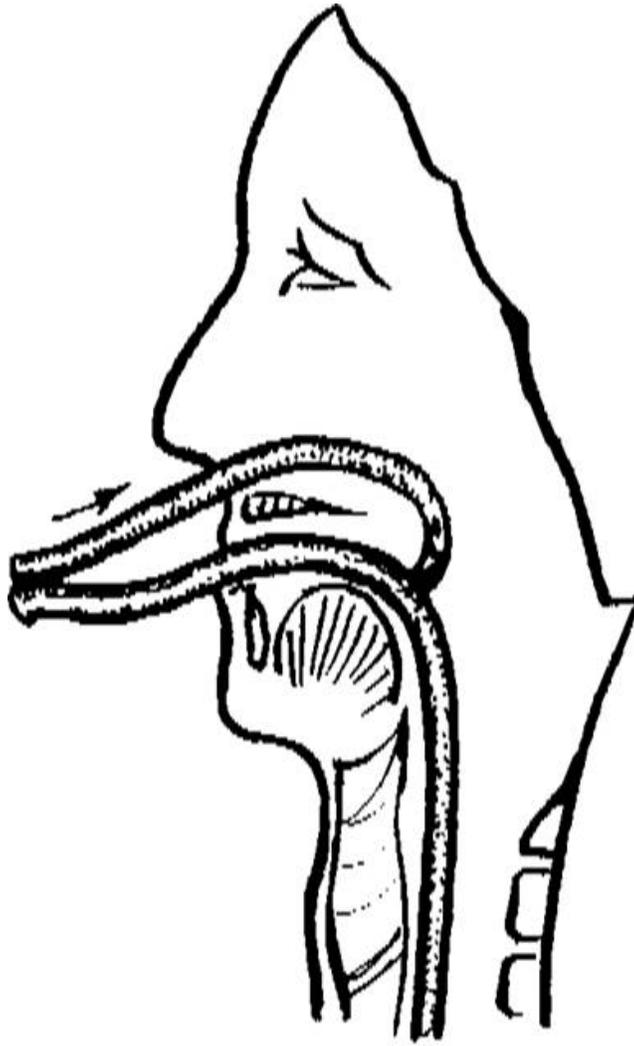
2. Beginning of intubation



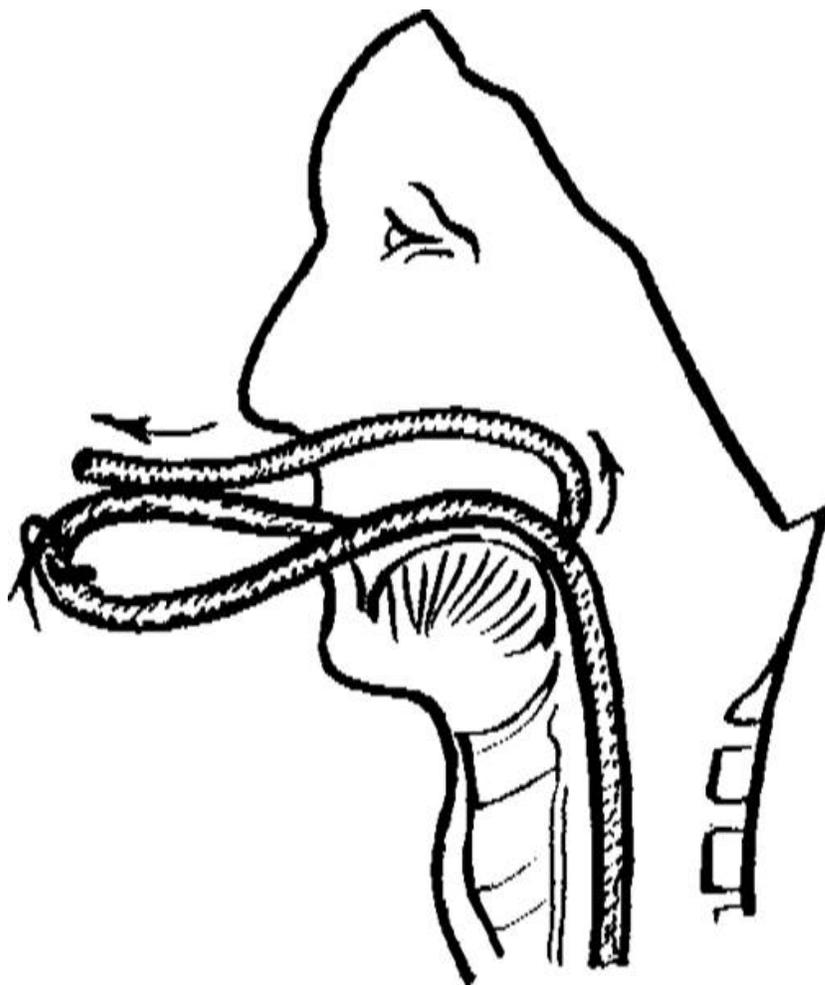
3. Intubation till caecum
through the gastric probe-guide
distally to pylorus per os



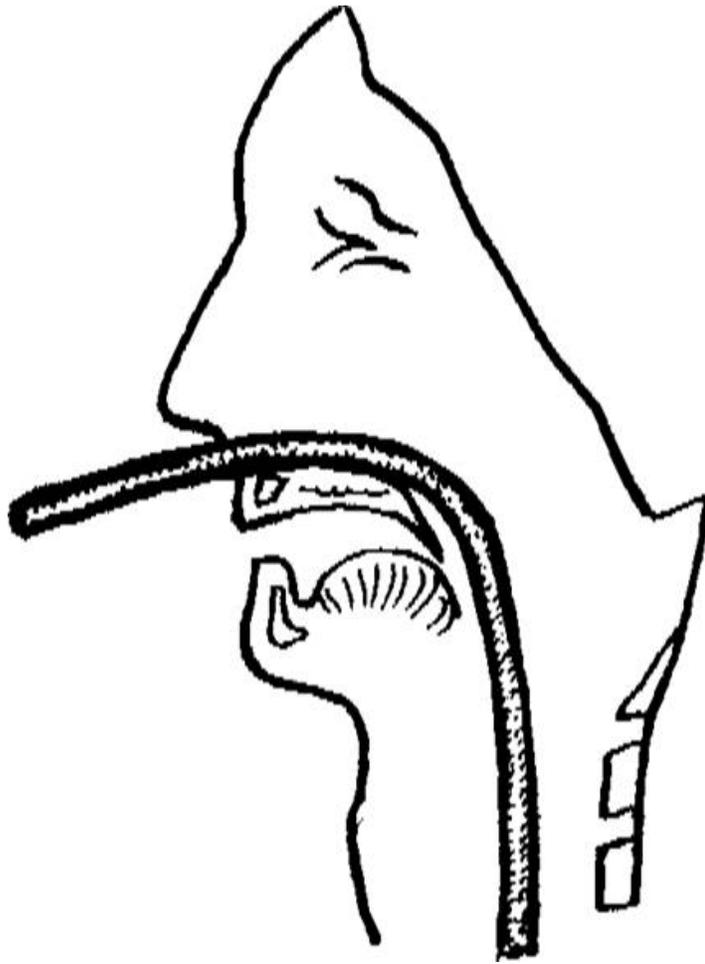
4. Removing of gastric probe



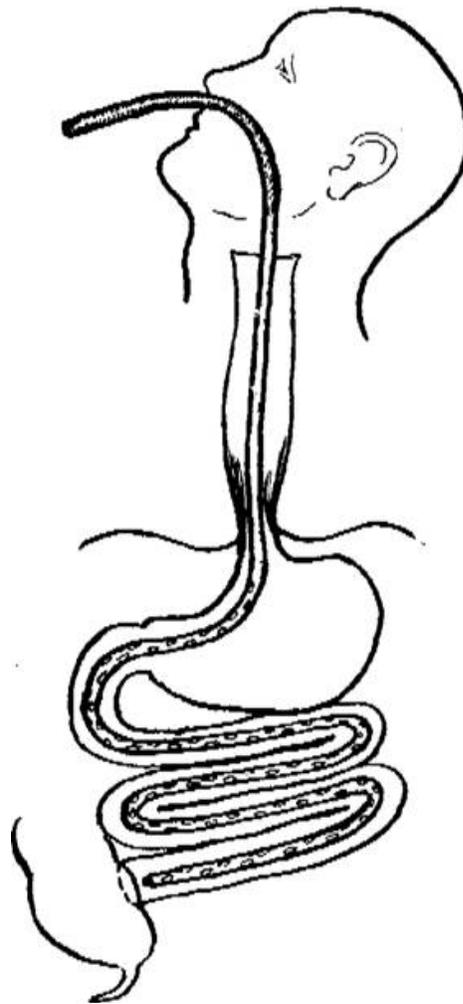
5. Fixation of proximal part
of intestinal probe to the nasal catheter



6. Removing of proximal part of intestinal



7. Proximal part is removed through the nose
probe from the oral cavity through the nose



8. Final view of nasogastrintestinal intubation

Fig.8. Principles of nasogastrintestinal intubation

The preoperative preparation in patients with peritonitis should be individual and lasted at least 2-3 hours. In extremely advanced cases, which associated with toxic shock and low arterial pressure it can last to 4-6 hours and must include nasogastric decompression of the stomach with active aspiration, catheterization of two veins, one of which is central, catheterization of bladder for diuresis control, infusion therapy.

The infusion therapy includes 5 % solution of glucose, solution of albumins, plasma, rheopolyglucin, vitamins of B and C group, solution of sodium hydrocarbonatis. The volume of fluid infusion should be at least 1.5-2 l. If there are no improvement of patient's condition before the operation, the infusion therapy must go on during operative approach.

The most common access in diffuse peritonitis is a median laparotomy, which is the most suitable for abdominal revision. In case of localized peritonitis (acute appendicitis) oblique incision may be used. The main goal of surgery must be elimination of infectious focus (appendectomy, cholecystectomy) or closure of stomach opening (perforating ulcer) or disrapture of hollow viscera. The exudate must be maximum

removed and peritoneal cavity washed up by antiseptic solutions and thereafter the intestinal decompression and draining of peritoneal space is performed.

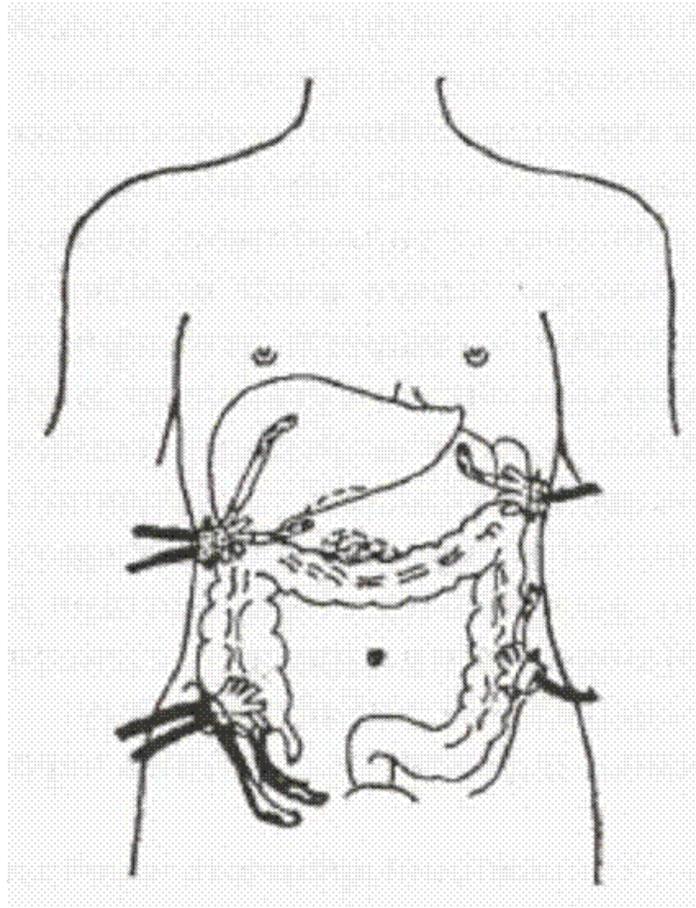


Fig.9. Scheme of the draining abdominal cavity
at extensive purulent peritonitis

In diffuse peritonitis the peritoneal cavity is drained in right and left hypochondrium and both left and right inguinal regions. It is better to use double or multiple polyethylene tubes, which are the most suitable for peritoneal dialysis. (Fig.9);(Fig.10);(Fig.11)

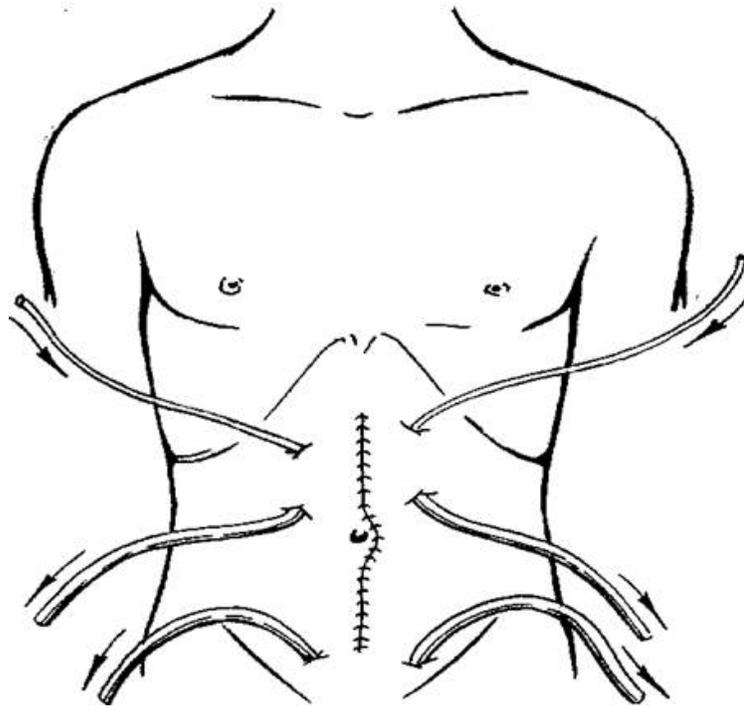


Fig.9. Localization of the drainages at peritoneal dialysis

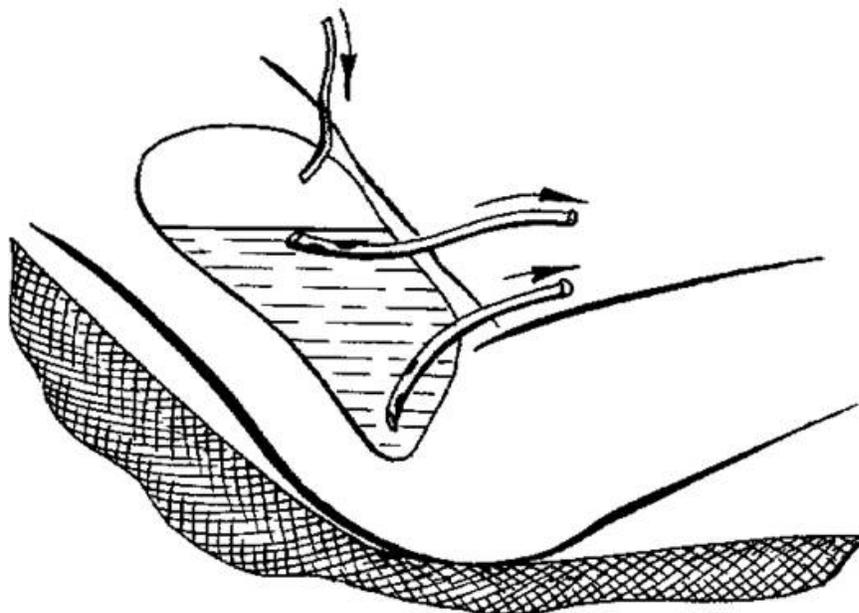


Fig.10. Scheme of localization of the drainages
at peritoneal dialysis(Lateral view)

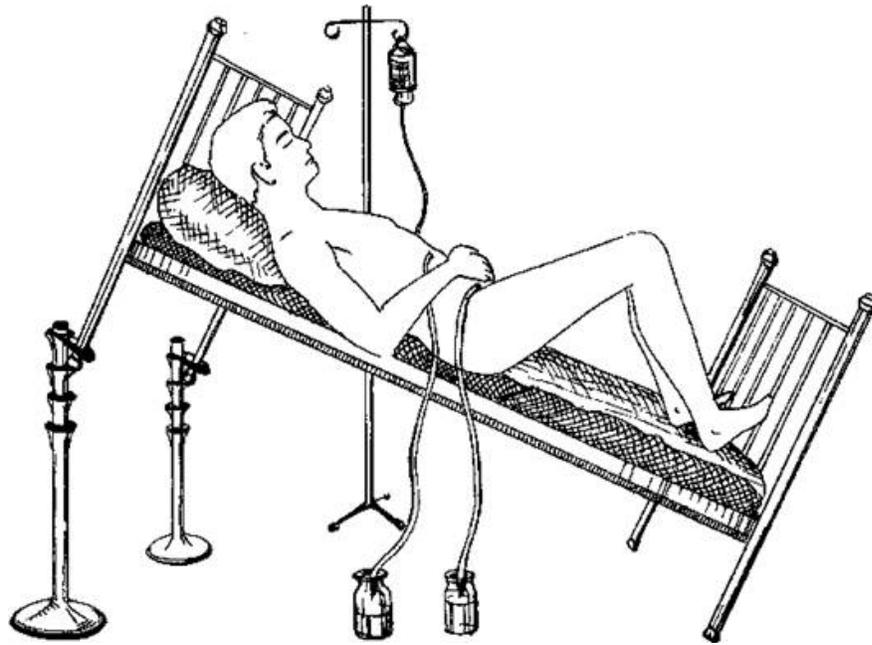


Fig.11. The position of the patient at peritoneal dialysis

Thus the infectious exudate and toxic substances are eliminated and antibiotics and antiseptic solutions are flown into the abdomen through these tubes. In 1.5-2 hours after the operation before the dialysis the patient takes a semisedentary position. Then the solutions flow in through the upper tubes and flow out through the lower. This procedure is performed as far as the solution from the lower tubes becomes clear, using for this purpose 10-25 l. of fluid.

In recent years instead of dialysis applied peritoneal lavage. Controllable peritoneostomy(Fig.12) in association with lavage, epidural anesthesia and intestinal intubation allow to rather promptly carry out sanitation of peritoneal cavity and liquidation of inflammatory process. These procedures are repeated in 1-2 days up to complete elimination of pus, fibrin and necrotic tissues. After the last sanitation the abdominal wall is closed.

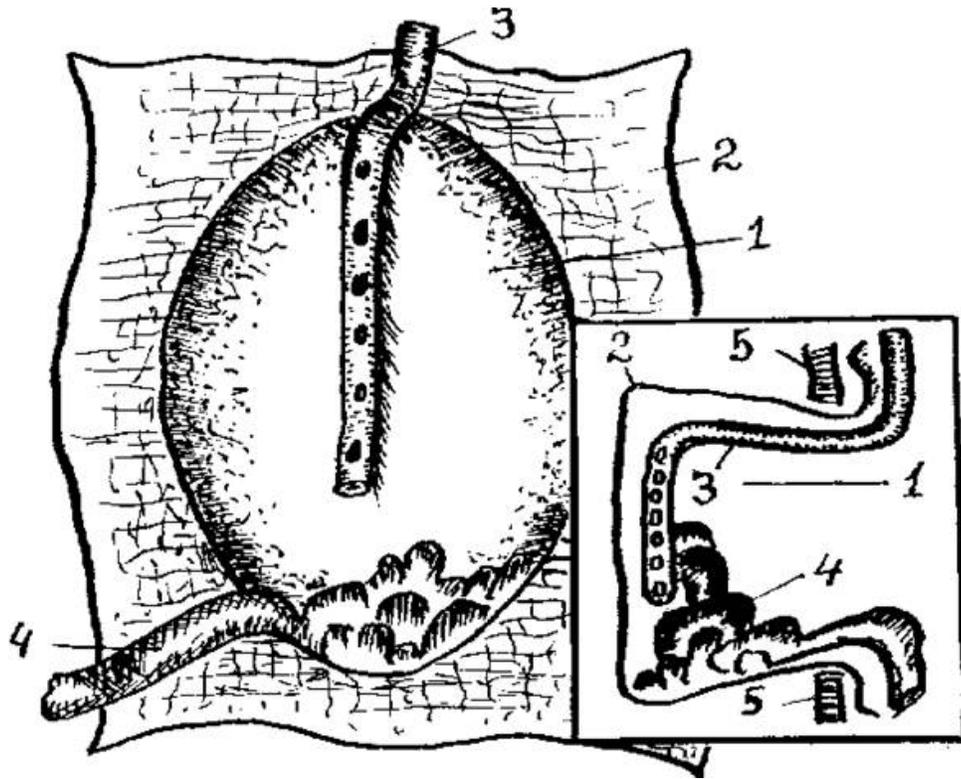


Fig.12. Wide plane drainage of abdominal cavity(begining):

bottom of operative wound

gauze(matrix)

tube for irrigation

gauze drainages

laparotomy wound margins

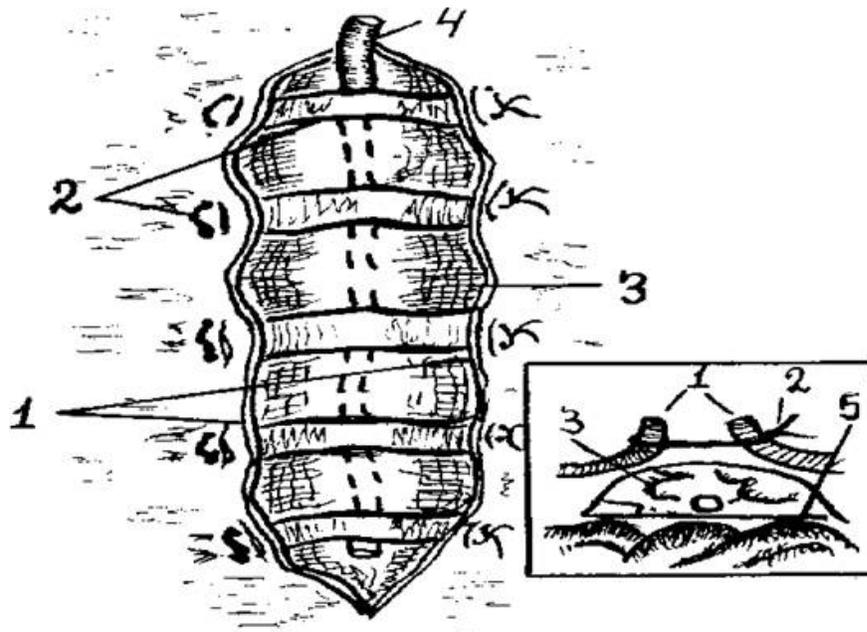


Fig.12. Wide plane drainage of abdominal cavity(finishing):

laparotomy wound margins

sutures on the skin

gauze(matrix), which covers drainages

tube for irrigation

intestines

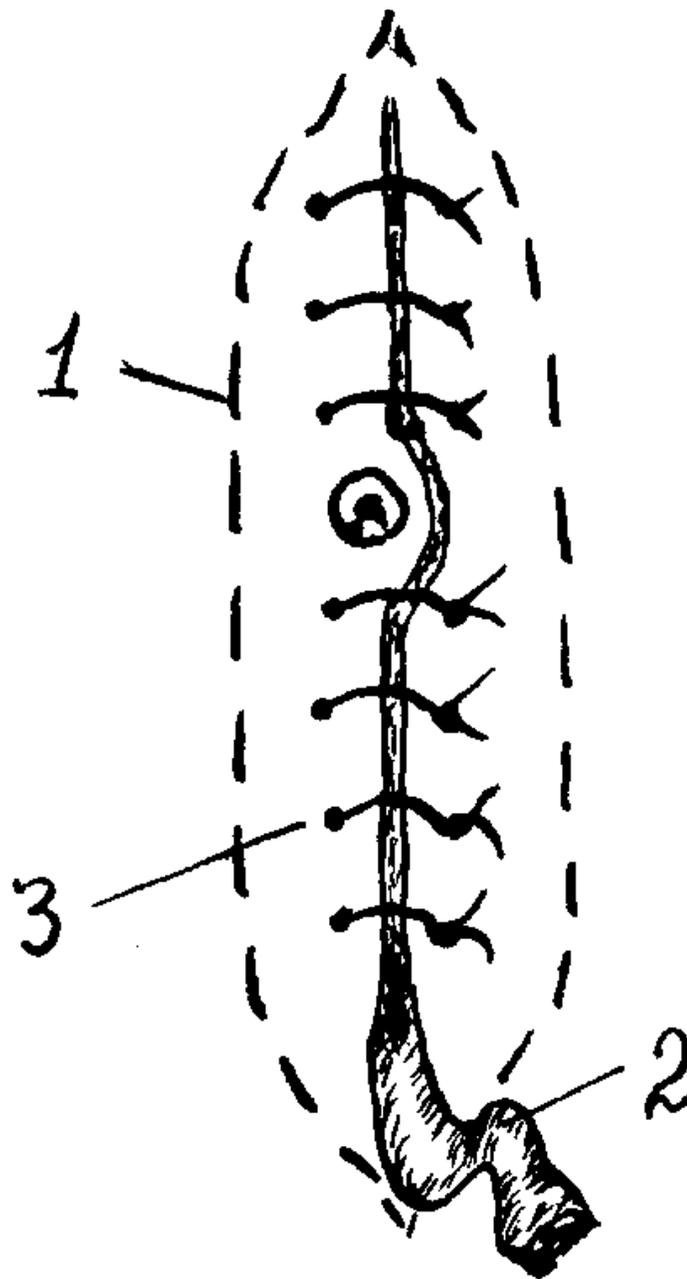


Fig.13. Reconstruction of anterior abdominal wall

laparotomy wound margins

rubber-line drainage

primary delayed suture

Antibacterial therapy is performed by means of intraabdominal and parenteral (intramuscular, intravenous, endolymphatic) administering of antibiotics. It is desirable to use broad-spectrum antibiotics and after results of antibioticogram possible to apply direct correction. The antibiotics are advisable to use in combination with sulfanilamides, metrogyl, immunostimulators.

A struggle against paralytic ileus is a very important in the complex of treatment of peritonitis. It should begin during operation by means of intestinal decompression, mesenteric blockade, gastric lavage, and

detoxycation therapy. For restitution of peristalsis used proserin, 10 % solution of sodium chloride, hypertonic enema. One of the most important factors in the treatment of peritonitis is the complete restore of the volume of circulating blood, correction of acid-base balance, blood electrolytes, protein metabolism. The total amount of fluid is calculated with account of its loss during vomiting, urinary excretion, drainage discharges and also respiration. For energetic compensation infused concentrated solution of glucose, sorbitol and lipid emulsion. Also plasma, erythrocyte and blood transfusions are used. In order to prevent hypoxia oxygenotherapy or hyperbaric oxygenation are applied.

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NEW TECHNOLOGIES IN SURGERY. MODERN METHODS OF DIAGNOSTIC AND TREATMENT

MINIMALLY INVASIVE SURGERY: INTRODUCTION

Minimally-invasive surgery describes an area of surgery that crosses all traditional disciplines, from general surgery to neurosurgery. It is not a discipline unto itself, but more a philosophy of surgery, a way of thinking. Minimally-invasive surgery is a means of performing major operations through small incisions, often using miniaturized, high-tech imaging systems, to minimize the trauma of surgical exposure. Some believe that minimal access surgery more accurately describes the small incisions generally necessary to gain access to surgical sites in high-tech surgery, but John Wickham's term minimally-invasive surgery (MIS) is widely used because it describes the paradox of postmodern high-tech surgery—small holes, big operations—and the "minimalness" of the access and invasiveness of the procedures, captured in three words.

Historical Background

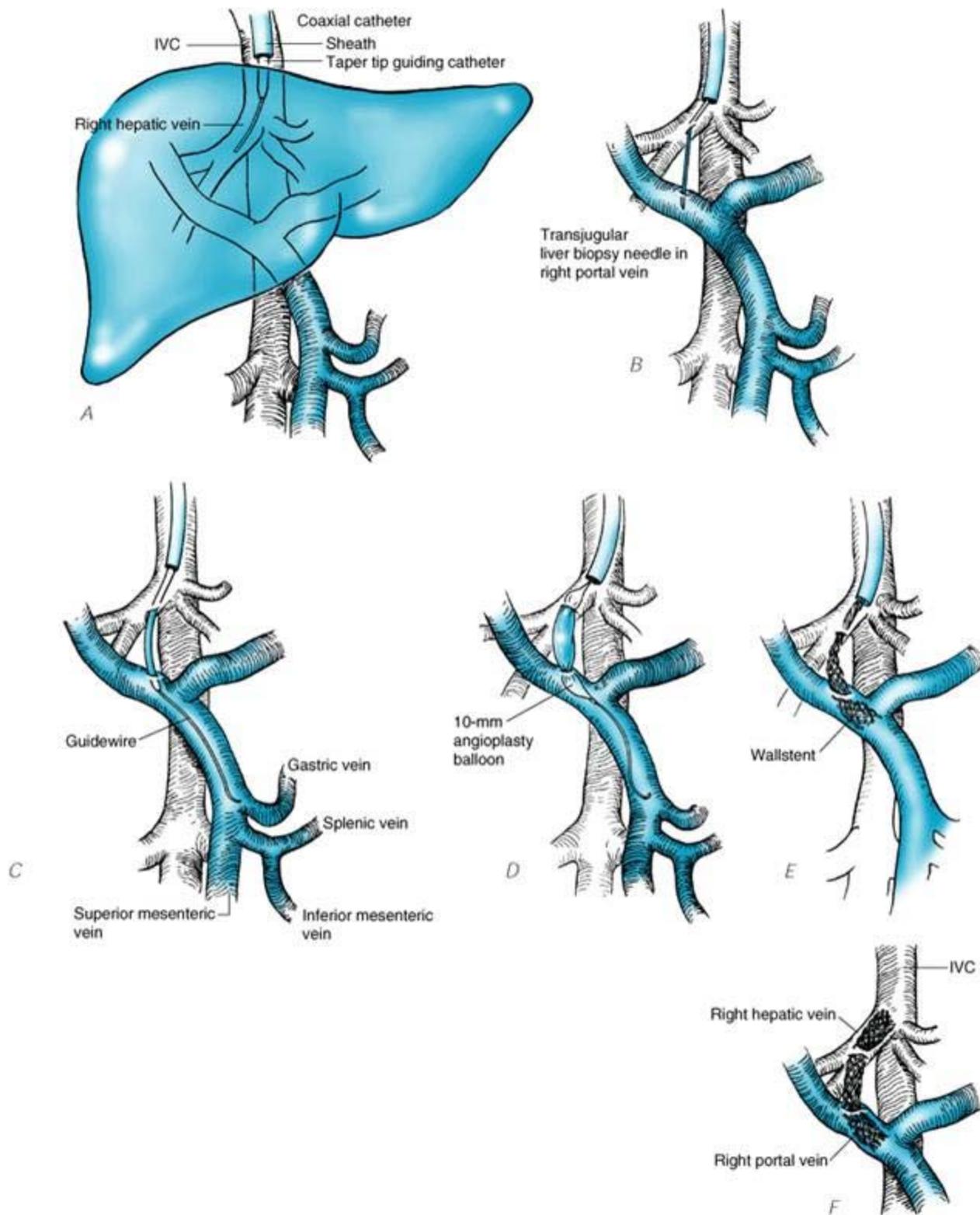
While the term *minimally-invasive surgery* is relatively recent, the history of its component parts is nearly 100 years old. What is considered the newest and most popular variety of MIS, laparoscopy, is in fact the oldest. Primitive laparoscopy, placing a cystoscope within an inflated abdomen, was first performed by Kelling in 1901. 1 Illumination of the abdomen required hot elements at the tip of the scope and was dangerous. In the late 1950s Hopkins described the rod lens, a method of transmitting light through a solid quartz rod with no heat and little light loss. 1 Around the same time, thin quartz fibers were discovered to be capable of trapping light internally and conducting it around corners, opening the field of fiberoptics and allowing the rapid development of flexible endoscopes. 2,3 In the 1970s the application of flexible endoscopy grew faster than that of rigid endoscopy except in a few fields such as gynecology and orthopedics. 4 By the mid-1970s rigid and flexible endoscopes made a rapid transition from diagnostic instruments to therapeutic ones. The explosion of video-assisted surgery in the past 10 years was a result of the development of compact, high-resolution charge-coupled devices which could be mounted on the internal end of flexible endoscopes or on the external end of a Hopkins telescope. Coupled with bright light sources, fiberoptic cables, and high-resolution video monitors, the videoendoscope has changed our understanding of surgical anatomy and reshaped surgical practice.

While optical imaging produced the majority of MIS procedures, other (traditionally radiologic) imaging technologies allowed the development of innovative procedures in the 1970s. Fluoroscopic imaging allowed the adoption of percutaneous vascular procedures, the most revolutionary of which was balloon angioplasty. Balloon-based procedures spread into all

fields of medicine, assisting in a minimally-invasive manner to open up clogged lumens. Stents were then developed that were used in many disciplines to keep the newly ballooned segment open. The culmination of fluoroscopic balloon and stent proficiency is exemplified by the transvenous intrahepatic portosystemic shunt (TIPS) (FIG. 1).

MIS procedures using ultrasound imaging have been limited to fairly crude exercises, such as fragmenting kidney stones and freezing liver tumors, because of the relatively low resolution of ultrasound devices. Newer, high-resolution ultrasound methods with high-frequency crystals may act as a guide while performing minimally-invasive resections of individual layers of the intestinal wall.

FIG. 1.



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With the transvenous intrahepatic portosystemic shunt (TIPS), percutaneous access to the superior vena cava is followed by the retrograde cannulation of the hepatic veins. Next a needle is advanced through the hepatic parenchyma until the portal venous radicle is located. A guidewire is passed across this connection, and after dilation a metallic stent is expanded with

a balloon. While not often performed by surgeons, TIPS represents a particularly creative example of minimally-invasive surgery. (Reproduced with permission from Hunter JG, Sackier JM (eds): *Minimally-Invasive Surgery*. New York: McGraw-Hill, 1993, p 271.)

Axial imaging, such as computed tomography (CT), has allowed the development of an area of MIS that is not often recognized because it requires only a CT scanner and a long needle. CT-guided drainage of abdominal fluid collections and percutaneous biopsy of abnormal tissues are minimally-invasive means of performing procedures that previously required a celiotomy. Recently, CT-guided percutaneous radiofrequency ablation has emerged as a useful treatment for primary and metastatic liver tumors. This procedure has also been performed laparoscopically under ultrasound guidance. 5

A powerful, noninvasive method of imaging that will allow the development of the least invasive—and potentially noninvasive—surgery is magnetic resonance imaging (MRI).

MRI is an extremely valuable diagnostic tool, but it is only slowly coming to be of therapeutic value. One obstacle to the use of MRI for MIS is that image production and refreshment of the image as a procedure progresses are slow. Another is that all instrumentation must be nonmetallic when working with the powerful magnets of an MRI scanner. Moreover, MRI magnets are bulky and limit the surgeon's access to the patient. Open magnets have been developed that allow the surgeon to stand between two large MRI coils, obtaining access to the portion of the patient being scanned. The advantage of MRI, in addition to the superb images produced, is that there is no radiation exposure to patient or surgeon. Some neurosurgeons are accumulating experience using MRI to perform frameless stereotactic surgery.

The Minimally-Invasive Team

From the beginning, the tremendous success of minimally-invasive surgery has been founded on the understanding that a team approach is necessary. The numerous laparoscopic procedures range from basic to advanced complexity, and require that the surgical team have an intimate understanding of the operative conduct (Table 13-1). Minimally-invasive procedures require complicated and fragile equipment that demands constant maintenance. In addition, multiple intraoperative adjustments to the equipment, camera, insufflator, monitors, and patient/surgeon position are made during these procedures. As such, a coordinated team approach is mandated in order to ensure patient safety and excellent outcomes.

A typical MIS team may consist of a laparoscopic surgeon and an operating room nurse with an interest in laparoscopic surgery. Adding dedicated laparoscopic assistants and circulating staff with an intimate knowledge of the equipment will add to and enhance the team nucleus. Studies have demonstrated that having a designated laparoscopic team reduces the conversion rate and overall operative time, which is translated into a cost savings for patient and hospital.

Physiology

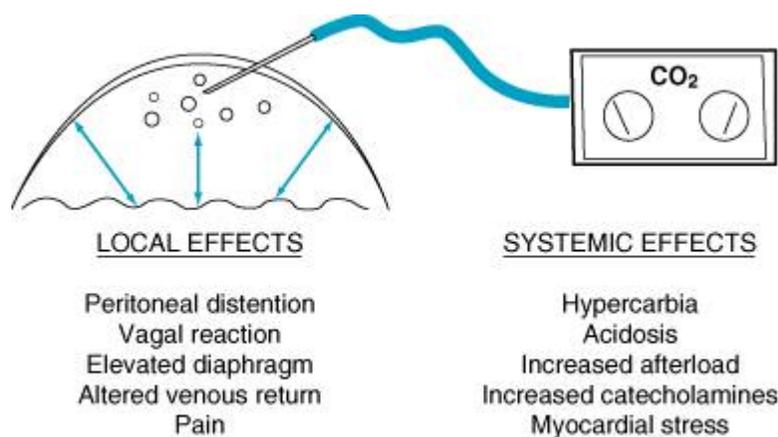
Even with the least invasive of the MIS procedures, physiologic changes occur. Many minimally-invasive procedures require minimal or no sedation, and there are few alterations to the cardiovascular, endocrinologic, or immunologic systems. The least invasive of such procedures include stereotactic biopsy of breast lesions and flexible gastrointestinal endoscopy. Minimally-invasive procedures that require general anesthesia have a greater physiologic impact because of the anesthetic agent, the incision (even if small), and the induced pneumoperitoneum.

Laparoscopy

The unique feature of endoscopic surgery in the peritoneal cavity is the need to lift the abdominal wall from the abdominal organs. Two methods have been devised for achieving this. 7 The first, used by most surgeons, is the induction of a pneumoperitoneum. Throughout the early twentieth century intraperitoneal visualization was achieved by inflating the abdominal cavity with air, using a sphygmomanometer bulb. 8 The problem with using air insufflation is that nitrogen is poorly soluble in blood and is slowly absorbed across the peritoneal surfaces. Air pneumoperitoneum was believed to be more painful than nitrous oxide pneumoperitoneum but less painful than carbon dioxide pneumoperitoneum. Subsequently, carbon dioxide and nitrous oxide were used for inflating the abdomen. N₂O had the advantage of being physiologically inert and rapidly absorbed. It also provided better analgesia for laparoscopy performed under local anesthesia when compared with CO₂ or air. 9 Despite initial concerns that N₂O would not suppress combustion, controlled clinical trials have established its safety within the peritoneal cavity. 10 In addition, nitrous oxide has recently been shown to reduce the intraoperative end-tidal CO₂ and minute ventilation required to maintain homeostasis when compared to CO₂ pneumoperitoneum. 10 The effect of N₂O on tumor biology and the development of port site metastasis are unknown. As such, caution should be exercised when performing laparoscopic cancer surgery with this agent. Finally, the safety of N₂O pneumoperitoneum in pregnancy has yet to be elucidated.

The physiologic effects of CO₂ pneumoperitoneum can be divided into two areas: (1) gas-specific effects and (2) pressure-specific effects (FIG. 2). CO₂ is rapidly absorbed across the peritoneal membrane into the circulation. In the circulation, CO₂ creates a respiratory acidosis by the generation of carbonic acid. 11 Body buffers, the largest reserve of which lies in bone, absorb CO₂ (up to 120 L) and minimize the development of hypercarbia or respiratory acidosis during brief endoscopic procedures. 11 Once the body buffers are saturated, respiratory acidosis develops rapidly, and the respiratory system assumes the burden of keeping up with the absorption of CO₂ and its release from these buffers.

FIG. 2.



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Carbon dioxide gas insufflated into the peritoneal cavity has both local and systemic effects that cause a complex set of hemodynamic and metabolic alterations. [Reproduced with permission from Hunter JG (ed): *Baillière's Clinical Gastroenterology Laparoscopic Surgery*. London/Philadelphia: Baillière Tindall, 1993, p 758.]

In patients with normal respiratory function this is not difficult; the anesthesiologist increases the ventilatory rate or vital capacity on the ventilator. If the respiratory rate required exceeds 20 breaths per minute, there may be less efficient gas exchange and increasing hypercarbia. 12 Conversely, if vital capacity is increased substantially, there is a greater opportunity for barotrauma and greater respiratory motion–induced disruption of the upper abdominal operative field. In some situations it is advisable to evacuate the pneumoperitoneum or reduce the intra-abdominal pressure to allow time for the anesthesiologist to adjust for hypercarbia. 13 While mild respiratory acidosis probably is an insignificant problem, more severe respiratory acidosis leading to cardiac arrhythmias has been reported. 14 Hypercarbia also causes tachycardia and increased systemic vascular resistance, which elevates blood pressure and increases myocardial oxygen demand. 11,14

The pressure effects of the pneumoperitoneum on cardiovascular physiology also have been studied. In the hypovolemic individual, excessive pressure on the inferior vena cava and a reverse Trendelenburg position with loss of lower extremity muscle tone may cause decreased venous return and cardiac output. 11,15 This is not seen in the normovolemic patient. The most common arrhythmia created by laparoscopy is bradycardia. A rapid stretch of the peritoneal membrane often causes a vagovagal response with bradycardia and occasionally hypotension. 16 The appropriate management of this event is desufflation of the abdomen, administration of vagolytic agents (e.g., atropine), and adequate volume replacement. 17

With the increased intra-abdominal pressure compressing the inferior vena cava, there is diminished venous return from the lower extremities. This has been well documented in the patient placed in the reverse Trendelenburg position for upper abdominal operations. Venous engorgement and decreased venous return promote venous thrombosis. 18,19 Many series of

advanced laparoscopic procedures in which deep venous thrombosis (DVT) prophylaxis was not used demonstrate the frequency of pulmonary embolus. This usually is an avoidable complication with the use of sequential compression stockings, subcutaneous heparin, or low-molecular-weight heparin. 20 In short-duration laparoscopic procedures, such as appendectomy, hernia repair, or cholecystectomy, the risk of DVT may not be sufficient to warrant extensive DVT prophylaxis.

The increased pressure of the pneumoperitoneum is transmitted directly across the paralyzed diaphragm to the thoracic cavity, creating increased central venous pressure and increased filling pressures of the right and left sides of the heart. If the intra-abdominal pressures are kept under 20 mm Hg, the cardiac output usually is well maintained. 19,20,21 The direct effect of the pneumoperitoneum on increasing intrathoracic pressure increases peak inspiratory pressure, pressure across the chest wall, and also the likelihood of barotrauma. Despite these concerns, disruption of blebs and consequent pneumothoraces are rare after uncomplicated laparoscopic surgery. 21

Increased intra-abdominal pressure decreases renal blood flow, glomerular filtration rate, and urine output. These effects may be mediated by direct pressure on the kidney and the renal vein. 22,23 The secondary effect of decreased renal blood flow is to increase plasma renin release, thereby increasing sodium retention. Increased circulating antidiuretic hormone (ADH) levels also are found during the pneumoperitoneum, increasing free water reabsorption in the distal tubules. 24 Although the effects of the pneumoperitoneum on renal blood flow are immediately reversible, the hormonally mediated changes, such as elevated ADH levels, decrease urine output for up to 1 hour after the procedure has ended. Intraoperative oliguria is common during laparoscopy, but the urine output is not a reflection of intravascular volume status; intravenous fluid administration during an uncomplicated laparoscopic procedure should not be linked to urine output. Because fluid losses through the open abdomen are eliminated with laparoscopy, the need for supplemental fluid during a laparoscopic surgical procedure is rare.

The hemodynamic and metabolic consequences of pneumoperitoneum are well tolerated by healthy individuals for a prolonged period and by most individuals for at least a short period. Difficulties can occur when a patient with compromised cardiovascular function is subjected to a long laparoscopic procedure. It is during these procedures that alternative approaches should be considered or insufflation pressure reduced. Alternative gases that have been suggested for laparoscopy include the inert gases helium, neon, and argon. These gases are appealing because they cause no metabolic effects, but are poorly soluble in blood (unlike CO₂ and N₂O) and are prone to create gas emboli if the gas has direct access to the venous system. 19 Gas emboli are rare but serious complications of laparoscopic surgery. 20,25 They should be suspected if hypotension develops during insufflation. Diagnosis may be made by listening (with an esophageal stethoscope) for the characteristic "mill wheel" murmur. The treatment of gas embolism is to place the patient in a left lateral decubitus position with the head down to trap the gas in the apex of the right ventricle. 20 A rapidly placed central venous catheter then can

be used to aspirate the gas out of the right ventricle.

In some situations minimally-invasive abdominal surgery should be performed without insufflation. This has led to the development of an abdominal lift device that can be placed through a 10- to 12-mm trocar at the umbilicus. 26 These devices have the advantage of creating little physiologic derangement, but they are bulky and intrusive. The exposure and working room offered by lift devices also are inferior to those accomplished by pneumoperitoneum. Lifting the anterior abdominal wall causes a "pinching in" of the lateral flank walls, displacing the bowel medially and anteriorly into the operative field. A pneumoperitoneum, with its well-distributed intra-abdominal pressure, provides better exposure. Abdominal lift devices also cause more postoperative pain, but they do allow the performance of MIS with standard (nonlaparoscopic) surgical instruments.

Early it was predicted that the surgical stress response would be significantly lessened with laparoscopic surgery, but this is not always the case. Serum cortisol levels after laparoscopic operations are often higher than after the equivalent operation performed through an open incision. 27 In terms of endocrine balance, the greatest difference between open and laparoscopic surgery is the more rapid equilibration of most stress-mediated hormone levels after laparoscopic surgery. Immune suppression also is less after laparoscopy than after open surgery. There is a trend toward more rapid normalization of cytokine levels after a laparoscopic procedure than after the equivalent procedure performed by celiotomy. 28

Transhiatal mobilization of the thoracic esophagus is commonly performed as a component of many laparoscopic upper abdominal procedures. Entering the posterior mediastinum transhiatally exposes the thoracic organs to positive insufflation pressure and may result in decreased venous return and a resultant decrease in cardiac output. If there is compromise of the mediastinal pleura with resultant CO₂ pneumothorax, the defect should be enlarged so as to prevent a tension pneumothorax.

Thoracoscopy

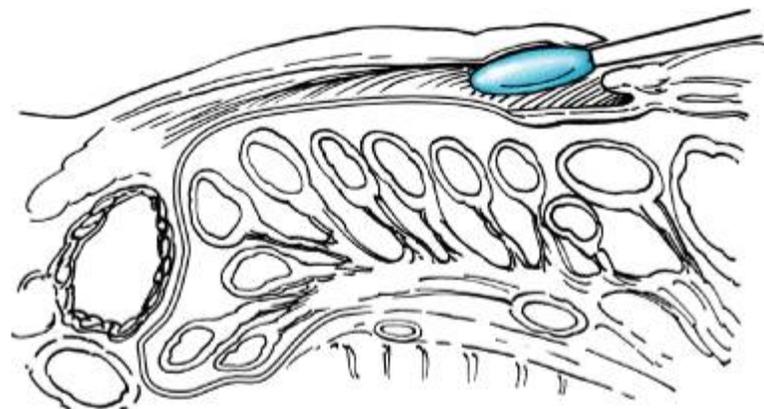
The physiology of thoracic MIS (thoracoscopy) is different from that of laparoscopy. Because of the bony confines of the thorax it is unnecessary to use positive pressure when working in the thorax. 29 The disadvantages of positive pressure in the chest include decreased venous return, mediastinal shift, and the need to keep a firm seal at all trocar sites. Without positive pressure, it is necessary to place a double-lumen endotracheal tube so that the ipsilateral lung can be deflated when the operation starts. By collapsing the ipsilateral lung, working space within the thorax is obtained. Because insufflation is unnecessary in thoracoscopic surgery, it can be beneficial to utilize standard instruments via extended port sites in conjunction with thoracoscopic instruments. This approach is particularly useful when performing advanced procedures such as thoracoscopic anatomic pulmonary resection.

Extracavitary Minimally-Invasive Surgery

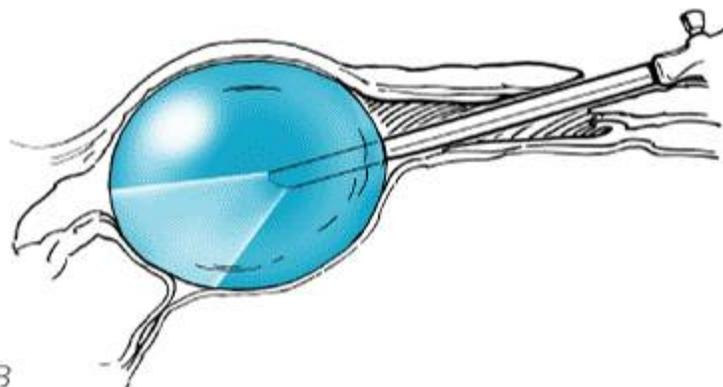
Many new MIS procedures are creating working spaces in extrathoracic and extraperitoneal locations. Laparoscopic inguinal hernia repair usually is performed in the anterior

extraperitoneal Retzius space. 30,31 Laparoscopic nephrectomy often is performed with retroperitoneal laparoscopy. Recently, an endoscopic retroperitoneal approach to pancreatic necrosectomy has been introduced. 32 Lower extremity vascular procedures and plastic surgical endoscopic procedures require the development of working space in unconventional planes, often at the level of the fascia, sometimes below the fascia, and occasionally in nonanatomic regions. 33 Some of these techniques use insufflation of gas, but many use balloon inflation to develop the space, followed by low-pressure gas insufflation or lift devices to maintain the space (FIG. 3). These techniques produce fewer and less severe adverse physiologic consequences than does the pneumoperitoneum, but the insufflation of gas into extraperitoneal locations can spread widely, causing subcutaneous emphysema and metabolic acidosis.

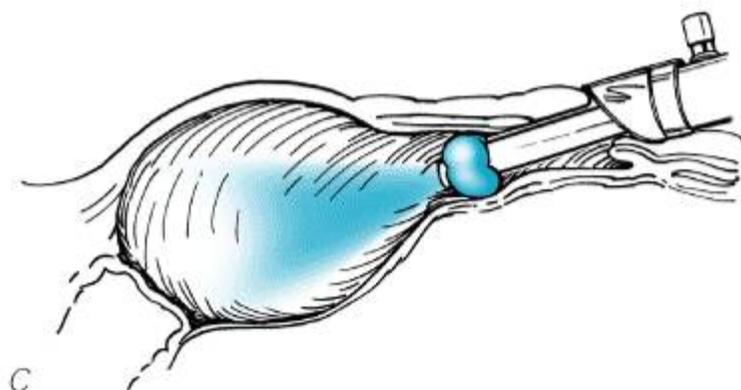
FIG. 3.



A



B



C

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Balloons are used to create extra-anatomic working spaces. In this example a balloon is introduced into the space between the posterior rectus sheath and the rectus abdominis muscle. The balloon is inflated in the preperitoneal space to create working room for extraperitoneal endoscopic hernia repair.

Anesthesia

The most important factors in appropriate anesthesia management are related to CO₂

pneumoperitoneum. 17 The laparoscopic surgeon can influence cardiovascular performance by releasing intra-abdominal retraction and dropping the pneumoperitoneum. Insensible fluid losses are negligible, and therefore intravenous fluid administration should not exceed a maintenance rate. MIS procedures usually are outpatient procedures, and short-acting anesthetic agents are preferable. Because the factors that require hospitalization after laparoscopic procedures include the management of nausea, pain, and urinary retention, the anesthesiologist should minimize the use of agents that provoke these conditions and maximize the use of medications that prevent such problems. Critical to the anesthesia management of these patients is the use of nonnarcotic analgesics (e.g., ketorolac) and the liberal use of antiemetic agents.

General Principles of Access and Equipment

The most natural ports of access for MIS are the anatomic portals of entry and exit. The nares, mouth, urethra, and anus are used to access the respiratory, gastrointestinal, and urinary systems. The advantage of using these points of access is that no incision is required. The disadvantages lie in the long distances between the orifice and the region of interest.

Access to the vascular system may be accomplished under local anesthesia by cutting down and exposing the desired vessel, usually in the groin. Increasingly, vascular access is obtained with percutaneous techniques using a small incision, a needle, and a guidewire, over which are passed a variety of different sized access devices. This approach, known as the Seldinger technique, is most frequently used by general surgeons for placement of Hickman catheters, but also is used to gain access to the arterial and venous system for performance of minimally-invasive procedures. Guidewire-assisted, Seldinger-type techniques also are helpful for gaining access to the gut for procedures such as percutaneous endoscopic gastrostomy, for gaining access to the biliary system through the liver, and for gaining access to the upper urinary tract.

In thoracoscopic surgery, the access technique is similar to that used for placement of a chest tube. In these procedures general anesthesia and split-lung ventilation are essential. A small incision is made over the top of a rib and, under direct vision, carried down through the pleura. The lung is collapsed, and a trocar is inserted across the chest wall to allow access with a telescope. Once the lung is completely collapsed, subsequent access may be obtained with direct puncture, viewing all entry sites through the videoendoscope. Because insufflation of the chest is unnecessary, simple ports that keep the small incisions open are all that is required to allow repeated access to the thorax.

Laparoscopic Access

The requirements for laparoscopy are more involved, because the creation of a pneumoperitoneum requires that instruments of access (trocars) contain valves to maintain abdominal inflation.

Two methods are used for establishing abdominal access during laparoscopic procedures. 34,35 The first, direct puncture laparoscopy, begins with the elevation of the relaxed abdominal

wall with two towel clips or a well-placed hand. A small incision is made in the umbilicus, and a specialized spring-loaded (Veress) needle is placed in the abdominal cavity (FIG. 4A and B). With the Veress needle, two distinct pops are felt as the surgeon passes the needle through the abdominal wall fascia and the peritoneum. The umbilicus usually is selected as the preferred point of access because in this location the abdominal wall is quite thin, even in obese patients. The abdomen is inflated with a pressure-limited insufflator. CO₂ gas is usually used, with maximal pressures in the range of 14 to 15 mm Hg. During the process of insufflation it is essential that the surgeon observe the pressure and flow readings on the monitor to confirm an intraperitoneal location of the Veress needle tip (FIG. 5). Laparoscopic surgery can be performed under local anesthesia, but general anesthesia is preferable. Under local anesthesia, N₂O is used as the insufflating agent, and insufflation is stopped after 2 L of gas is insufflated or when a pressure of 10 mm Hg is reached.

FIG. 4.



A

**B**

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A. Insufflation of the abdomen is accomplished with a Veress needle held at its serrated collar with a thumb and forefinger. B. Because linea alba is fused to the umbilicus, the abdominal wall is grasped with fingers or penetrating towel clip in order to elevate the abdominal wall away from the underlying structures.

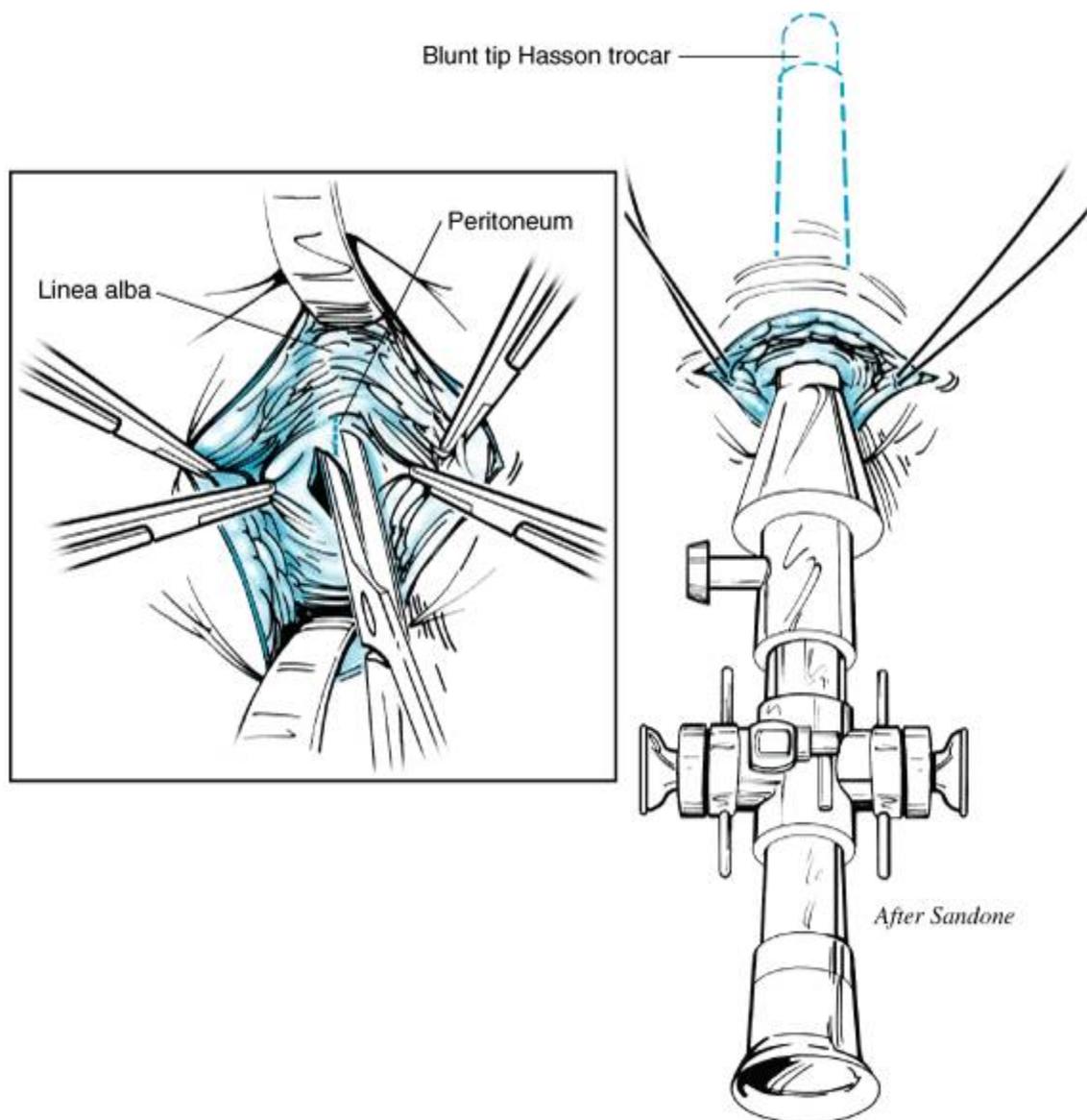
It is essential to be able to interpret the insufflator pressure readings and flow rates. These readings indicate proper intraperitoneal placement of the Veress needle.

After peritoneal insufflation, direct access to the abdomen is obtained with a 5- or 10-mm trocar. The critical issues for safe direct-puncture laparoscopy include the use of a vented stylet for the trocar, or a trocar with a safety shield or dilating tip. The trocar must be pointed away from the sacral promontory and the great vessels. 36 Patient position should be surveyed prior to trocar placement to ensure a proper trajectory. For performance of laparoscopic cholecystectomy, the trocar is angled toward the right upper quadrant.

Occasionally the direct peritoneal access (Hasson) technique is advisable. 37 With this technique, the surgeon makes a small incision just below the umbilicus and under direct vision locates the abdominal fascia. Two Kocher clamps are placed on the fascia, and with a curved Mayo scissors a small incision is made through the fascia and underlying peritoneum. A finger is placed into the abdomen to make sure that there is no adherent bowel. A sturdy suture is placed on each side of the fascia and secured to the wings of a specialized trocar, which is then passed directly into the abdominal cavity (FIG. 6). Rapid insufflation can make up for some of the time lost with the initial dissection. This technique is preferable for the abdomen of patients who have undergone previous operations in which small bowel may be adherent to the undersurface of the abdominal wound. The close adherence of bowel to the peritoneum in the previously operated abdomen does not eliminate the possibility of intestinal injury, but should

make great vessel injury extremely unlikely. Because of the difficulties in visualizing the abdominal region immediately adjacent to the primary trocar, it is recommended that the telescope be passed through a secondary trocar in order to inspect the site of initial abdominal access. 35 Secondary punctures are made with 5- and 10-mm trocars. For safe access to the abdominal cavity, it is critical to visualize all sites of trocar entry. 35,36 At the completion of the operation, all trocars are removed under direct vision and the insertion sites are inspected for bleeding. If bleeding occurs, direct pressure with an instrument from another trocar site or balloon tamponade with a Foley catheter placed through the trocar site generally stops the bleeding within 3 to 5 minutes. When this is not successful, a full-thickness abdominal wall suture has been used successfully to tamponade trocar site bleeding.

FIG. 6.



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The open laparoscopy technique involves identification and incision of the peritoneum, followed by the placement of a specialized trocar with a conical sleeve to maintain a gas seal. Specialized wings

on the trocar are attached to sutures placed through the fascia to prevent loss of the gas seal.

It is generally agreed that 5-mm trocars need no site suturing. Ten-millimeter trocars placed off the midline and above the transverse mesocolon do not require repair. Conversely, if the fascia has been dilated to allow the passage of the gallbladder, all midline 10-mm trocar sites should be repaired at the fascial level with interrupted sutures. Specialized suture delivery systems similar to crochet needles have been developed for mass closure of the abdominal wall in obese patients, in whom it is difficult to visualize the fascia through a small skin incision. Failure to close lower abdominal trocar sites that are 10 mm in diameter or larger can lead to an incarcerated hernia.

Access for Subcutaneous and Extraperitoneal Surgery

There are two methods for gaining access to nonanatomic spaces. For retroperitoneal locations, balloon dissection is effective. This access technique is appropriate for the extraperitoneal repair of inguinal hernias and for retroperitoneal surgery for adrenalectomy, nephrectomy, lumbar discectomy, pancreatic necrosectomy, or para-aortic lymph node dissection. 38,39 The initial access to the extraperitoneal space is performed in a way similar to direct puncture laparoscopy, except that the last layer (the peritoneum) is not traversed. Once the transversalis fascia has been punctured, a specialized trocar with a balloon on the end is introduced. The balloon is inflated in the extraperitoneal space to create a working chamber. The balloon then is deflated and a Hasson trocar is placed. An insufflation pressure of 10 mm Hg usually is adequate to keep the extraperitoneal space open for dissection and will limit subcutaneous emphysema. Higher gas pressures force CO₂ into the soft tissues and may contribute to hypercarbia. Extraperitoneal endosurgery provides less working space than laparoscopy, but eliminates the possibility of intestinal injury, intestinal adhesion, herniation at the trocar sites, and ileus. These issues are important for laparoscopic hernia repair because extraperitoneal approaches prevent the small bowel from sticking to the prosthetic mesh. 31

Subcutaneous surgery, the newest method of access in minimally-invasive surgery, uses the creation of working room in nonanatomic spaces. This technique has been most widely used in cardiac, vascular, and plastic surgery. 33 In cardiac surgery, subcutaneous access has been used for saphenous vein harvesting, and in vascular surgery for ligation of subfascial perforating veins (Linton procedure). With minimally-invasive techniques the entire saphenous vein above the knee may be harvested through a single incision 40,41 (FIG. 7). Once the saphenous vein is located, a long retractor that holds a 5-mm laparoscope allows the coaxial dissection of the vein and coagulation or clipping of each side branch. A small incision above the knee also can be used to ligate perforating veins in the lower leg.

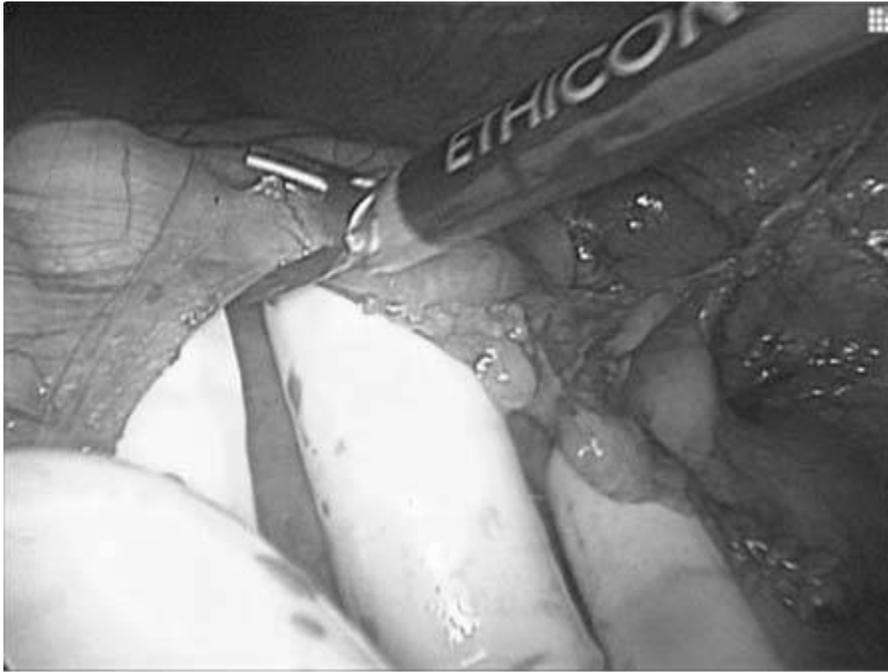
Subcutaneous access is also used for plastic surgical procedures. 41 Minimally-invasive approaches are especially well suited to cosmetic surgery, in which attempts are made to hide the incision. It is easier to hide several 5-mm incisions than one long incision. The technique of blunt dissection along fascial planes combined with lighted retractors and endoscope-holding retractors is most successful for extensive subcutaneous surgery. Some prefer gas insufflation of these soft tissue planes. The primary disadvantage of soft tissue insufflation is that

subcutaneous emphysema can be created.

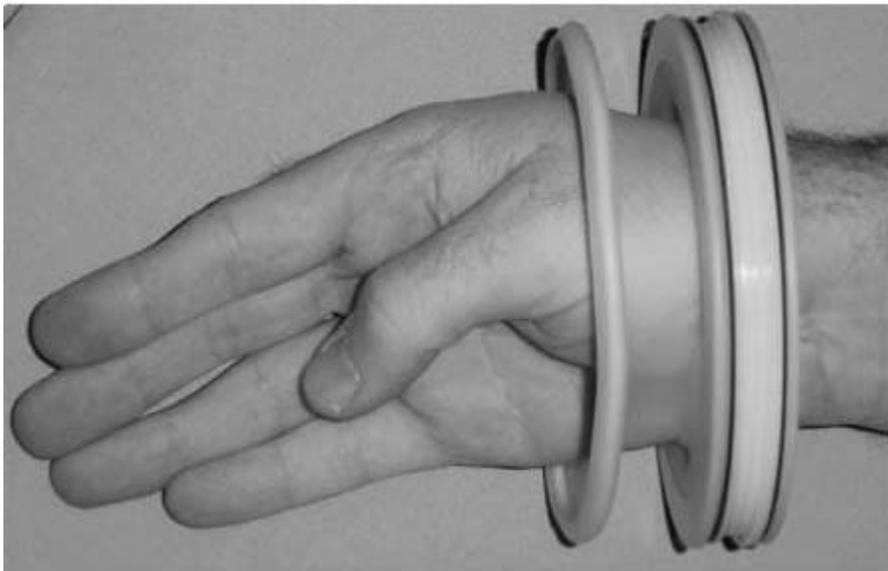
Hand-Assisted Laparoscopic Access

Hand-assisted laparoscopic surgery (HALS) is thought to combine the tactile advantages of open surgery with the minimal access of laparoscopy and thoracoscopy. This approach is commonly used to assist with difficult cases before conversion to celiotomy is necessary. Additionally, HALS is employed to help surgeons negotiate the steep learning curve associated with advanced laparoscopic procedures. 42 This technology employs a "port" for the hand which preserves the pneumoperitoneum and enables endoscopic visualization in combination with the use of minimally-invasive instruments (FIG. 8). Formal investigation of this modality has been limited primarily to case reports and small series, and has focused primarily on solid organ and colon surgery.

FIG. 8.



A
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B
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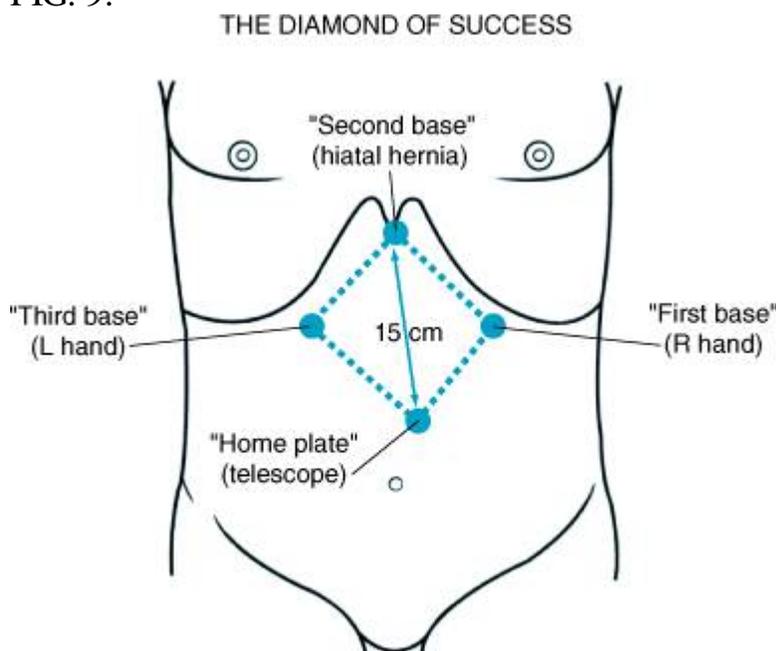
This is an example of hand-assisted laparoscopic surgery during left colectomy. The surgeon uses a hand to provide retraction and counter tension during mobilization of the colon from its retroperitoneal attachments, as well as during division of the mesocolon. This technique is particularly useful in the region of the transverse colon.

Port Placement

Trocars for the surgeon's left and right hand should be placed at least 10 cm apart. For most operations it is possible to orient the telescope between these two trocars and slightly retract

from them. The ideal trocar orientation creates an equilateral triangle between the surgeon's right hand, left hand, and the telescope, with 10 to 15 cm on each leg. If one imagines the target of the operation (e.g., the gallbladder or gastroesophageal junction) oriented at the apex of a second equilateral triangle built on the first, these four points of reference create a diamond (FIG. 9). The surgeon stands behind the telescope, which provides optimal ergonomic orientation but frequently requires that a camera operator (or robotic arm) reach between the surgeon's hands to guide the telescope.

FIG. 9.



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The diamond configuration created by placing the telescope between the left and the right hand, recessed from the target by about 15 cm. The distance between the left and the right hand is also ideally 10 to 15 cm. In this "baseball diamond" configuration, the surgical target occupies the second base position.

The position of the operating table should permit the surgeon to work with both elbows in at the sides, with arms bent 90° at the elbow. 43 It usually is necessary to alter the operating table position with left or right tilt with the patient in the Trendelenburg or reverse Trendelenburg position, depending on the operative field. 44,45

Imaging Systems

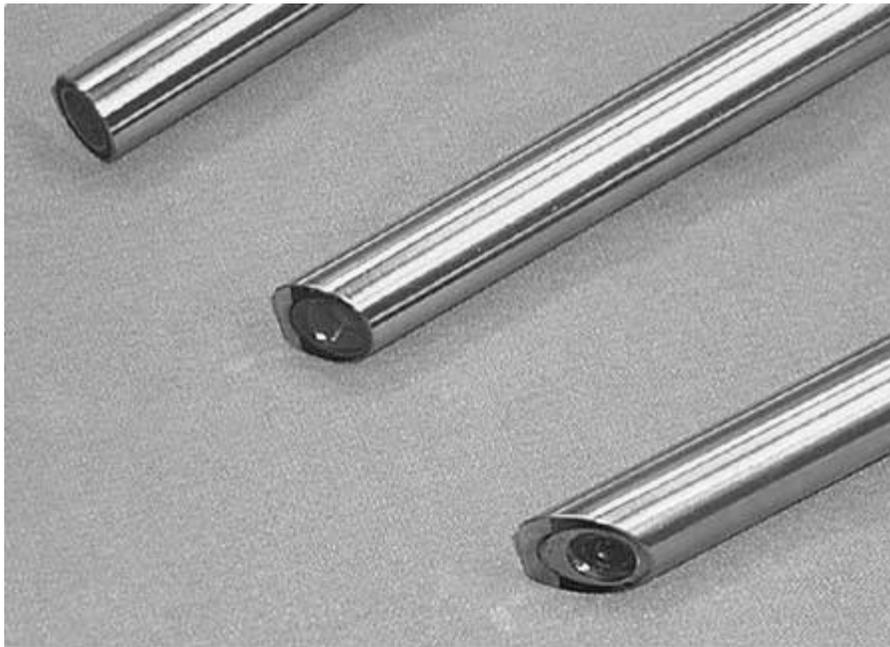
Two methods of videoendoscopic imaging are widely used. Both methods use a camera with a charge-coupled device (CCD), which is an array of photosensitive sensor elements (pixels) that convert the incoming light intensity to an electric charge. The electric charge is subsequently converted into a black-and-white image. 46 The first of these is flexible videoendoscopy, where the CCD camera is placed on the internal end of a long, flexible endoscope. In the second method, thin quartz fibers are packed together in a bundle, and the CCD camera is mounted on the external end of the endoscope. Most standard gastrointestinal endoscopes have the CCD chip at the distal end, but small, delicate choledochoscopes and

nephroscopes are equipped with fiberoptic bundles. 47 Distally-mounted CCD chips were developed for laparoscopy, but are unpopular.

Video cameras come in two basic designs. The one-chip camera has a black-and-white video chip that has an internal processor capable of converting gray scales to approximate colors. Perfect color representation is not possible with a one-chip camera, but perfect color representation is rarely necessary for endosurgery. The most accurate color representation is obtained using a three-chip video camera. A three-chip camera has red, green, and blue (RGB) input, and is identical to the color cameras used for television production. 46 RGB imaging provides the highest fidelity, but is probably not necessary for everyday use. An additional feature of newer video cameras is digital enhancement. Digital enhancement detects edges, areas where there are drastic color or light changes between two adjacent pixels. 48 By enhancing this difference, the image appears sharper and surgical resolution is improved. Digital enhancement is available on one- and three-chip cameras. Priorities in a video system for MIS are illumination first, resolution second, and color third. Without the first two attributes, video surgery is unsafe. Imaging for laparoscopy, thoracoscopy, and subcutaneous surgery uses a rigid metal telescope, usually 30 cm in length. This telescope contains a series of quartz optical rods with differing optical characteristics that provide a specific character to each telescope. 49 These metal telescopes vary in size from 2 to 10 mm in diameter. Since light transmission is dependent on the cross-sectional area of the quartz rod, when the diameter of a rod/lens system is doubled, the illumination is quadrupled. Little illumination is needed in highly-reflective, small spaces such as the knee, and a very small telescope will suffice. When working in the abdominal cavity, especially if blood is present, the full illumination of a 10-mm telescope usually is necessary.

Rigid telescopes may have a flat or angled end. The flat end provides a straight view (0°), and the angled end provides an oblique view (30 or 45°). 46 Angled scopes allow greater flexibility in viewing a wider operative field through a single trocar site (FIG. 10); rotating an angled telescope changes the field of view. The use of an angled telescope has distinct advantages for most videoendoscopic procedures, particularly in visualizing the common bile duct during laparoscopic cholecystectomy or visualizing the posterior esophagus or the tip of the spleen during laparoscopic fundoplication.

FIG. 10.



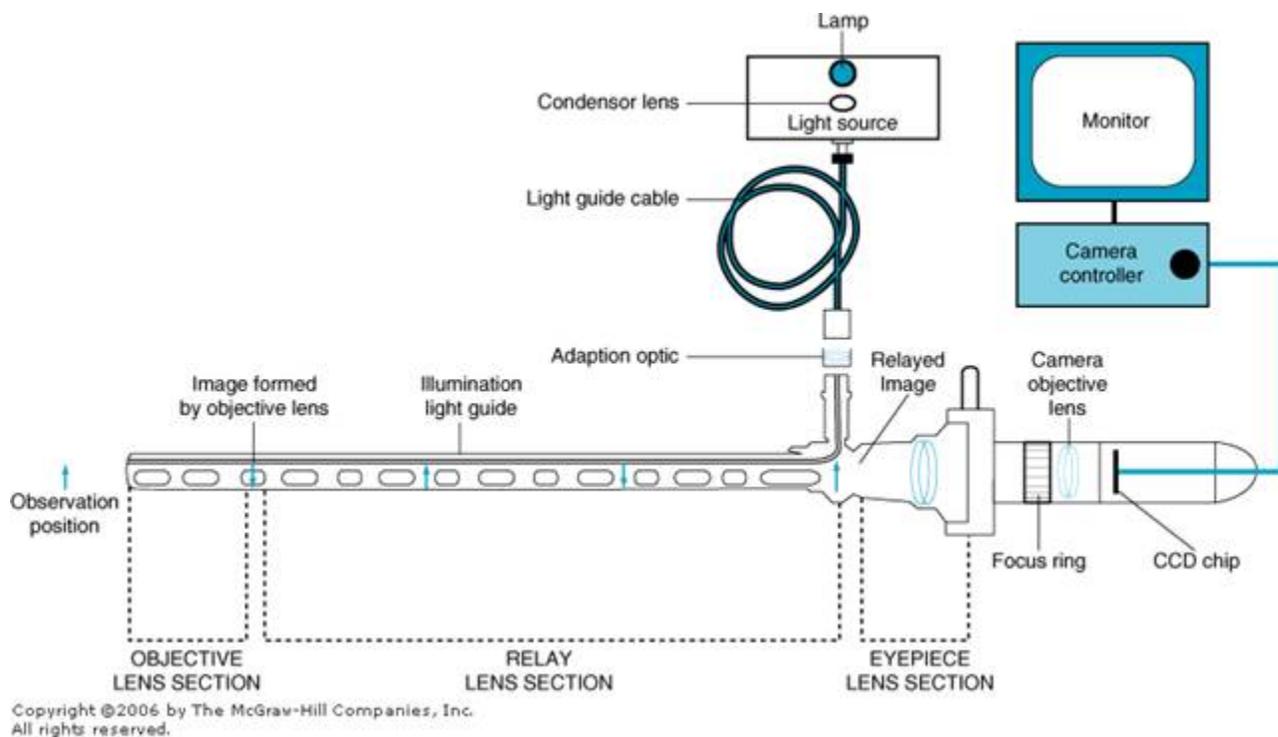
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The laparoscope tips come in a variety of angled configurations. All laparoscopes have a 70° field of view. A 30° angled scope enables the surgeon to view this field at a 30° angle to the long axis of the scope.

Light is delivered to the endoscope through a fiberoptic light cable. These light cables are highly inefficient, losing more than 90% of the light delivered from the light source. Extremely bright light sources (300 watts) are necessary to provide adequate illumination for video endosurgery.

The quality of the videoendoscopic image is only as good as the weakest component in the imaging chain (FIG. 11). Therefore it is important to use a video monitor that has a resolution equal to or greater than the camera being used. ⁴⁹ *Resolution* is the ability of the optical system to distinguish between line pairs. The larger the number of line pairs per millimeter, the sharper and more detailed the image. Most high-resolution monitors have up to 700 horizontal lines. High definition television (HDTV) can deliver up to eight times more resolution than the standard NTSC/PAL monitors; when combined with digital enhancement, a very sharp and well-defined image can be achieved. ^{46,49} A heads-up display (HUD) is a high-resolution liquid crystal monitor that is built into eyewear worn by the surgeon. ⁵⁰ This technology allows the surgeon to view the endoscopic image and operative field simultaneously. The proposed advantages of HUD include a high-resolution monocular image, which affords the surgeon mobility and reduces vertigo and eyestrain. However, this technology has not yet been widely adopted.

FIG. 11.



The Hopkins rod lens telescope includes a series of optical rods that effectively transmit light to the eyepiece. The video camera is placed on the eyepiece to provide the working image. The image is only as clear as the weakest link in the image chain. (*Reproduced with permission from Prescher et al. 46*)

There has been recent interest in three-dimensional endoscopy. Three-dimensional laparoscopy provides the additional depth of field that is lost with two-dimensional endosurgery and allows greater facility for novice laparoscopists performing complex tasks of dexterity, including suturing and knot tying. 51 The advantages of three-dimensional systems are less obvious to experienced laparoscopists. Additionally, because three-dimensional systems require the flickering of two similar images, which are resolved with special glasses, the images' edges become fuzzy and resolution is lost. The optical accommodation necessary to rectify these slightly differing images negates any advantage offered by the additional depth of field.

Energy Sources for Endoscopic and Endoluminal Surgery

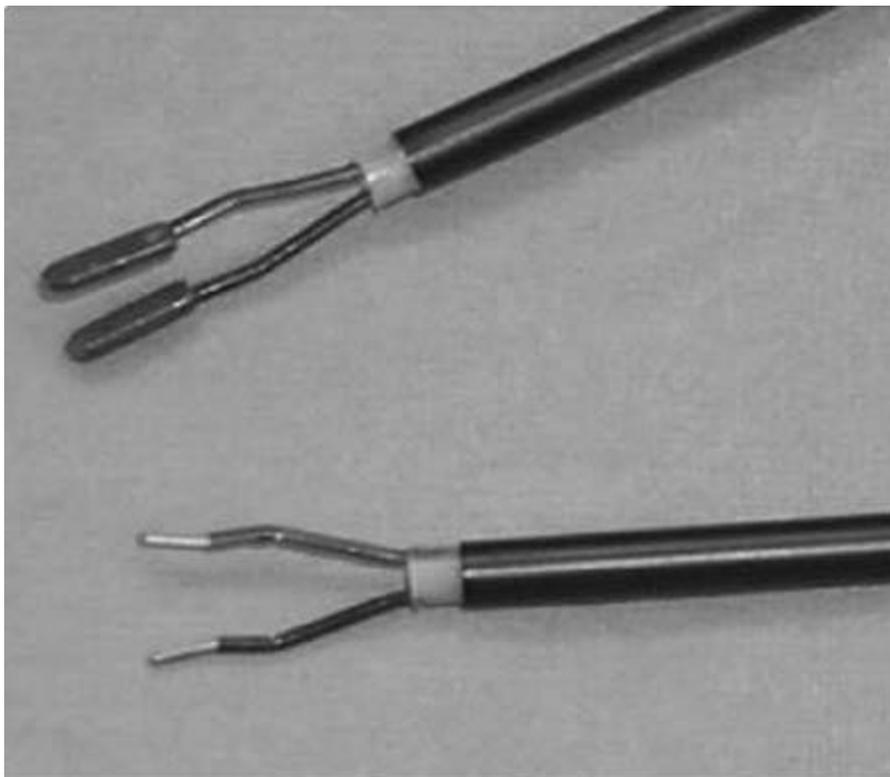
MIS uses conventional energy sources, but the requirement of bloodless surgery to maintain optimal visualization has spawned new ways of applying energy. The most common energy source is radiofrequency (RF) electrosurgery using an alternating current with a frequency of 500,000 cycles/s (Hz). Tissue heating progresses through the well-known phases of coagulation (60°C), vaporization and desiccation (100°C), and carbonization (>200°C). 52

The two most common methods of delivering RF electrosurgery are with monopolar and bipolar electrodes. With monopolar electrosurgery a remote ground plate on the patient's leg or

back receives the flow of electrons that originate at a point source, the surgical electrode. A fine-tipped electrode causes a high current density at the site of application and rapid tissue heating. Monopolar electrosurgery is inexpensive and easy to modulate to achieve different tissue effects. 53 A short-duration, high-voltage discharge of current (coagulation current) provides extremely rapid tissue heating. Lower-voltage, higher-wattage current (cutting current) is better for tissue desiccation and vaporization. When the surgeon desires tissue division with the least amount of thermal injury and least coagulation necrosis, a cutting current is used.

With bipolar electrosurgery the electrons flow between two adjacent electrodes. The tissue between the two electrodes is heated and desiccated. There is little opportunity for tissue cutting when bipolar current is used, but the ability to coapt the electrodes across a vessel provides the best method of small-vessel coagulation without thermal injury to adjacent tissues 54 (FIG. 12).

FIG. 12.



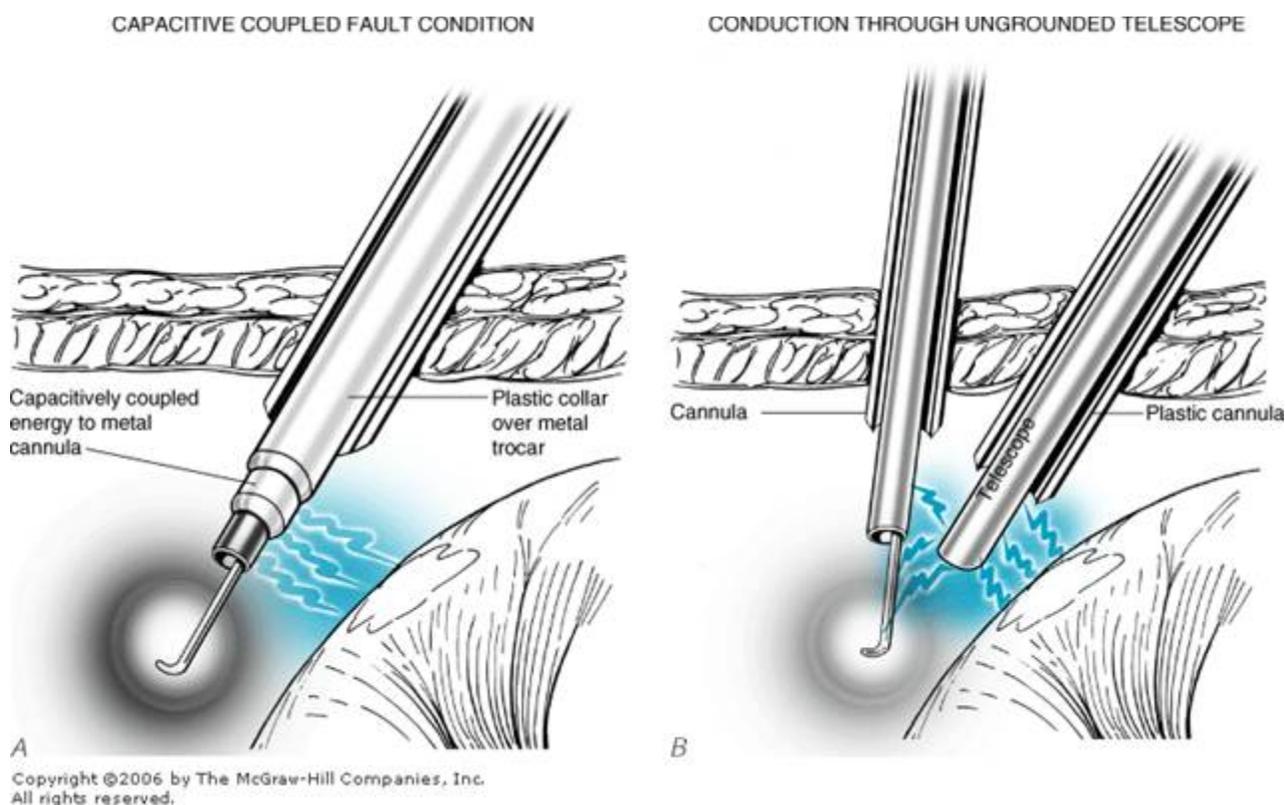
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An example of bipolar coagulation devices. The flow of electrons passes from one electrode to the other and the intervening tissue is heated and desiccated.

In order to avoid thermal injury to adjacent structures, the laparoscopic field of view must include all uninsulated portions of the electrosurgical electrode. In addition, the integrity of the insulation must be maintained and assured. Capacitive coupling occurs when a plastic trocar insulates the abdominal wall from the current; in turn the current is bled off of a metal sleeve or

laparoscope into the viscera 52 (Fig. 13–13A). This may result in thermal necrosis and a delayed fecal fistula. Another potential mechanism for unrecognized visceral injury may occur with the direct coupling of current to the laparoscope and adjacent bowel 52 (FIG. 13B).

FIG. 13.



A. Capacitive coupling occurs as a result of high current density bleeding from a port sleeve or laparoscope into adjacent bowel. B. Direct coupling occurs when current is transmitted directly from the electrode to a metal instrument or laparoscope, and then into adjacent tissue.

(Reproduced with permission from Odell. 52)

Another method of delivering radiofrequency electrosurgery is argon beam coagulation. This is a type of monopolar electrosurgery in which a uniform field of electrons is distributed across a tissue surface by the use of a jet of argon gas. The argon gas jet distributes electrons more evenly across the surface than does spray electrofulguration. This technology has its greatest application for coagulation of diffusely bleeding surfaces such as the cut edge of liver or spleen. It is of less use in laparoscopic procedures because the increased intra-abdominal pressures created by the argon gas jet can increase the chances of a gas embolus. It is paramount to vent the ports and closely monitor insufflation pressure when using this source of energy within the context of laparoscopy.

With endoscopic endoluminal surgery, radiofrequency alternating current in the form of a monopolar circuit represents the mainstay for procedures such as snare polypectomy, sphincterotomy, lower esophageal sphincter ablation, and "hot" biopsy. 55,56 A grounding ("return") electrode is necessary for this form of energy. Bipolar electrocoagulation is used primarily for thermal hemostasis. The electrosurgical generator is activated by a foot pedal so the endoscopist may keep both hands free during the endoscopic procedure.

Gas, liquid, and solid-state lasers have been available for medical application since the mid-1960s. 57 The CO₂ laser (wavelength 10.6 μm) is most appropriately used for cutting and superficial ablation of tissues. It is most helpful in locations unreachable with a scalpel such as excision of vocal cord granulomas. The CO₂ laser beam must be delivered with a series of mirrors and is therefore somewhat cumbersome to use. The next most popular laser is the neodymium yttrium-aluminum-garnet (Nd:YAG) laser. Nd:YAG laser light is 1.064 μm (1064 nm) in wavelength. It is in the near-infrared portion of the spectrum, and, like CO₂ laser light, is invisible to the naked eye. A unique feature of the Nd:YAG laser is that 1064-nm light is poorly absorbed by most tissue pigments and therefore travels deep into tissue. 58 Deep tissue penetration provides deep tissue heating (FIG. 14). For this reason the Nd:YAG laser is capable of the greatest amount of tissue destruction with a single application. 57 Such capabilities make it the ideal laser for destruction of large fungating tumors of the rectosigmoid, tracheobronchial tree, or esophagus. A disadvantage is that the deep tissue heating may cause perforation of a hollow viscus.

When it is desirable to coagulate flat lesions in the cecum, a different laser should be chosen. The frequency-doubled Nd:YAG laser, also known as the KTP laser (potassium thionyl phosphate crystal is used to double the Nd:YAG frequency), provides 532-nm light. This is in the green portion of the spectrum, and at this wavelength, selective absorption by red pigments in tissue (such as hemangiomas and arteriovenous malformations) is optimal. The depth of tissue heating is intermediate, between those of the CO₂ and the Nd:YAG lasers. Coagulation (without vaporization) of superficial vascular lesions can be obtained without intestinal perforation. 58

In flexible gastrointestinal endoscopy, the CO₂ and Nd:YAG lasers have largely been replaced by heater probes and endoluminal stents. The heater probe is a metal ball that is heated to a temperature (60 to 100°C) that allows coagulation of bleeding lesions without perforation.

Photodynamic therapy (PDT) is a palliative treatment for obstructing cancers of the gastrointestinal tract. 59 Patients are given an intravenous dose of porfimer sodium, which is a photosensitizing agent that is taken up by malignant cells. Two days after administration, the drug is endoscopically activated using a laser. The activated porfimer sodium generates oxygen free radicals, which kills the tumor cells. The tumor is later endoscopically débrided. The use of this modality for definitive treatment of early cancers is in experimental phases and has yet

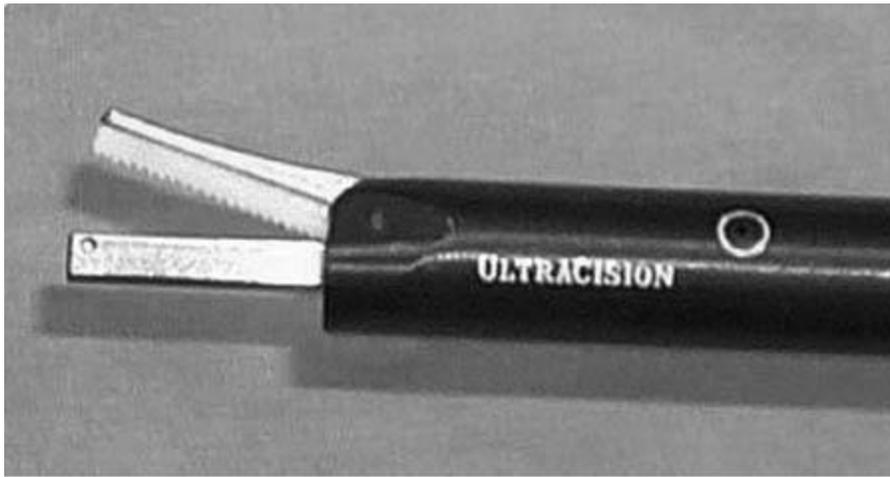
to become established.

A unique application of laser technology provides extremely rapid discharge ($<10^{-6}$ s) of large amounts of energy ($>10^3$ volts). These high-energy lasers, of which the pulsed dye laser has seen the most clinical use, allow the conversion of light energy to mechanical disruptive energy in the form of a shock wave. Such energy can be delivered through a quartz fiber, and with rapid repetitive discharges, can provide sufficient shock-wave energy to fragment kidney stones and gallstones. Shock waves also may be created with miniature electric spark-plug discharge systems known as electrohydraulic lithotriptors. These devices also are inserted through thin probes for endoscopic application. Lasers have the advantage of pigment selectivity, but electrohydraulic lithotriptors are more popular because they are substantially less expensive and are more compact.

Methods of producing shock waves or heat with ultrasonic energy are also of interest. Extracorporeal shockwave lithotripsy creates focused shock waves that intensify as the focal point of the discharge is approached. When the focal point is within the body, large amounts of energy are capable of fragmenting stones. Slightly different configurations of this energy can be used to provide focused internal heating of tissues. Potential applications of this technology include the ability to noninvasively produce sufficient internal heating to destroy tissue without an incision.

A third means of using ultrasonic energy is to create rapidly-oscillating instruments that are capable of heating tissue with friction; this technology represents a major step forward in energy technology. An example of its application is the laparoscopic coagulation shears (LCS) device (Harmonic Scalpel), which is capable of coagulating and dividing blood vessels by first occluding them and then providing sufficient heat to weld the blood vessel walls together and to divide the vessel (FIG. 15). This nonelectric method of coagulating and dividing tissue with a minimal amount of collateral damage has facilitated the performance of numerous endosurgical procedures. It is especially useful in the control of bleeding from medium-sized vessels that are too big to manage with monopolar electrocautery and require bipolar desiccation followed by cutting.

FIG. 15.



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The Harmonic Scalpel has revolutionized hemostasis and dissection in minimally-invasive surgery and has significantly facilitated the performance of advanced laparoscopic and thoracoscopic procedures. Ultrasonic energy is used to create a rapidly oscillating "working arm" which serves to heat intervening tissue with friction, which fuses the cell membranes together.

Instrumentation

Hand instruments for MIS usually are duplications of conventional surgical instruments made longer, thinner, and smaller at the tip. It is important to remember that when grasping tissue with laparoscopic instruments, a greater force is applied over a smaller surface area, which increases the risk for perforation or injury. 63

Certain conventional instruments such as scissors are easy to reproduce with a diameter of 3 to 5 mm and a length of 20 to 45 cm, but other instruments, such as forceps and clamps, cannot provide remote access. Different configurations of graspers were developed to replace the various configurations of surgical forceps and clamps. Standard hand instruments are 5 mm in diameter and 30 cm in length, but smaller and shorter hand instruments are now available for pediatric surgery, for microlaparoscopic surgery, and for arthroscopic procedures. 63 A unique laparoscopic hand instrument is the monopolar electrical hook. This device is usually configured with a suction and irrigation apparatus to eliminate smoke and blood from the operative field. The monopolar hook allows tenting of tissue over a bare metal wire with subsequent coagulation and division of the tissue.

Robotic Assistance

The term "robot" defines a device that has been programmed to perform specific tasks in place of those usually performed by people. The equipment that has been introduced under the heading of robotic assistance would perhaps be more aptly termed computer-assisted surgery, as it is controlled entirely by the surgeon for the purpose of improving team performance. An example of computer-assisted surgery includes laparoscopic camera holders, which enable the surgeon to maneuver the laparoscope either with head movements or voice activation (FIG. 16). Randomized studies with such camera holders have demonstrated a reduction in operative time, steadier image, and a reduction in the number of required laparoscope cleanings. 64 This

device has the advantage of eliminating the need for a human camera holder, which serves to free valuable operating room personnel for other duties.

FIG. 16.



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The surgeon controlled computer-assisted camera holder obviates the need for an additional assistant. This device may reduce operative times and provide a steadier image.

Another form of computer assistance involves the use of voice-activated system controls for the camera, light source, insufflators, and telephone. Studies have demonstrated a reduction in the time required to perform these tasks when compared to human intervention. 65

Room Setup and the Minimally-Invasive Suite

Nearly all MIS, whether using fluoroscopic, ultrasound, or optical imaging, incorporates a video monitor as a guide. Occasionally two images are necessary to adequately guide the operation, as in procedures such as endoscopic retrograde cholangiopancreatography (ERCP), laparoscopic common bile duct exploration, and laparoscopic ultrasonography. When two images are necessary, the images should be displayed on two adjacent video monitors or projected on a single screen with a picture-in-picture effect. The video monitor(s) should be set across the operating table from the surgeon. The patient should be interposed between the surgeon and the video monitor; ideally, the operative field also lies between the surgeon and the monitor. In pelviscopic surgery it is best to place the video monitor at the patient's feet, and in laparoscopic cholecystectomy, the monitor is placed at the 10 o'clock position (relative to

the patient) while the surgeon stands on the patient's left at the 4 o'clock position. The insufflating and patient-monitoring equipment ideally also is placed across the table from the surgeon, so that the insufflating pressure and the patient's vital signs and end-tidal CO₂ tension can be monitored.

The development of the minimally-invasive surgical suite has been a tremendous contribution to the field of laparoscopy in that it has facilitated the performance of advanced procedures and techniques (FIG. 17). By having the core equipment (monitors, insufflators, and imaging equipment) located within mobile, ceiling-mounted consoles, the surgery team is able to accommodate and make small adjustments rapidly and continuously throughout the procedure. The specifically designed minimally-invasive surgical suite serves to decrease equipment and cable disorganization, ease the movements of operative personnel around the room, improve ergonomics, and facilitate the use of advanced imaging equipment such laparoscopic ultrasound. 66 While having a minimally-invasive surgical suite available is very useful, it is not essential to successfully carry out advanced laparoscopic procedures.

FIG. 17.



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An example of a typical minimally-invasive surgery suite. All core equipment is located on easily movable consoles. These operating rooms tend to be larger in size because of the need for multiple types of equipment.

Patient Positioning

Patients usually are placed in the supine position for laparoscopic surgery. When the operative field is the gastroesophageal junction or the left lobe of the liver, it is easiest to

operate from between the legs. The legs may be elevated in Allen stirrups or abducted on leg boards to achieve this position. When pelvic procedures are performed, it usually is necessary to place the legs in Allen stirrups to gain access to the perineum. A lateral decubitus position with the table flexed provides the best access to the retroperitoneum when performing nephrectomy or adrenalectomy. For laparoscopic splenectomy, a 45°-tilt of the patient provides excellent access to the lesser sac and the lateral peritoneal attachments to the spleen. For thoracoscopic surgery, the patient is placed in the lateral position with table flexion in order to open the intercostal spaces and the distance between the iliac crest and costal margin (FIG. 18).

When the patient's knees are to be bent for extended periods or the patient is going to be placed in a reverse Trendelenburg position for more than a few minutes, deep venous thrombosis prophylaxis should be used. Sequential compression of the lower extremities during prolonged (more than 90 min) laparoscopic procedures increases venous return and provides inhibition of thromboplastin activation.

Special Considerations

Pediatric Considerations

The advantages of MIS in children may be more significant than in the adult population. 67 MIS in the adolescent is little different from that in the adult, and standard instrumentation and trocar positions can usually be used. However, laparoscopy in the infant and young child requires specialized instrumentation. The instruments are shorter (15 to 20 cm), and many are 3 mm in diameter rather than 5 mm. 68 Because the abdomen of the child is much smaller than that of the adult, a 5-mm telescope provides sufficient illumination for most operations. The development of 5-mm clippers and bipolar devices has obviated the need for 10-mm trocars in pediatric laparoscopy. 68 Because the abdominal wall is much thinner in infants, a pneumoperitoneum pressure of 8 mm Hg can provide adequate exposure. Deep venous thrombosis is rare in children, and prophylaxis against thrombosis is probably unnecessary.

Pregnancy

Concerns about the safety of laparoscopic cholecystectomy or appendectomy in the pregnant patient have been eliminated. The pH of the fetus follows the pH of the mother linearly, and therefore fetal acidosis may be prevented by avoiding a respiratory acidosis in the mother. 69 A second concern was that of increased intra-abdominal pressure, but it has been proved that midpregnancy uterine contractions provide a much greater pressure in utero than a pneumoperitoneum. Experience in well over 100 cases of laparoscopic cholecystectomy in pregnancy have been reported with uniformly good results. 70 The operation should be performed during the second trimester if possible. Protection of the fetus against intraoperative x-rays is imperative. Some believe it advisable to track fetal pulse rates with a transvaginal ultrasound probe. Access to the abdomen in the pregnant patient should take into consideration the height of the uterine fundus, which reaches the umbilicus at 20 weeks. In order not to damage the uterus or its blood supply, most surgeons feel that the open (Hasson) approach

should be used in favor of direct puncture laparoscopy. The patient should be positioned slightly on the left side in order to avoid compression of the vena cava by the uterus. Because pregnancy poses a risk for thromboembolism, sequential compression devices are essential for all procedures.

Cancer

MIS techniques have been used for many decades to provide palliation for the patient with an obstructive cancer. Laser treatment, intracavitary radiation, stenting, and dilation are outpatient techniques that can be used to reestablish the continuity of an obstructed esophagus, bile duct, ureter, or airway. MIS techniques also have been used in the staging of cancer.

Mediastinoscopy is still used occasionally before thoracotomy to assess the status of the mediastinal lymph nodes. Laparoscopy also is used to assess the liver in patients being evaluated for pancreatic, gastric, or hepatic resection. New technology and greater surgical skills allow for accurate minimally-invasive staging of cancer. ⁷¹ Occasionally it is appropriate to perform palliative measures (e.g., laparoscopic gastrojejunostomy to bypass a pancreatic cancer) at the time of diagnostic laparoscopy if diagnostic findings preclude attempts at curative resection. ⁷²

The most controversial role of MIS techniques is that of providing potentially curative surgery to the patient with cancer. It is possible to perform laparoscopy-assisted colectomy, gastrectomy, pancreatectomy, and hepatectomy in patients with intra-abdominal malignant disease, as well as thoracoscopic esophagectomy and pneumonectomy in patients with intrathoracic malignant disease. There are not yet enough data to indicate whether minimally-invasive surgical techniques provide survival rates or disease-free intervals comparable to those of conventional surgical techniques. It has been proven that in laparoscopy-assisted colectomy and gastrectomy a number of lymph nodes equal to that of an open procedure can be removed without any compromise of resection margins. A second concern centers on excessive tumor manipulation and the possibility that cancer cells would be shed during the dissection. Alarming reports of trocar site implantation with viable cancer cells have appeared in the literature.

Considerations in the Elderly and Infirm

Laparoscopic cholecystectomy has made possible the removal of a symptomatic gallbladder in many patients previously thought to be too elderly or too ill to undergo a laparotomy. Older patients are more likely to require conversion to celiotomy because of disease chronicity. ⁷³

Operations on these patients require close monitoring of anesthesia. The intraoperative management of these patients may be more difficult with laparoscopic access than with open access. The advantage of MIS lies in what happens after the operation. Much of the morbidity of surgery in the elderly is a result of impaired mobility. ⁷³ In addition, pulmonary complications, urinary tract sepsis, deep venous thrombosis, pulmonary embolism, congestive heart failure, and myocardial infarction often are the result of improper fluid management and

decreased mobility. By allowing rapid and early mobilization, laparoscopic surgery has made possible the safe performance of procedures in the elderly and infirm.

Cirrhosis and Portal Hypertension

Patients with hepatic insufficiency pose a significant challenge for any type of surgical intervention. 74 The ultimate surgical outcome in this population relates directly to the degree of underlying hepatic dysfunction. 75 Often, this group of patients has minimal reserve, and the stress of an operation will trigger complete hepatic failure or hepatorenal syndrome. These patients are at risk for major hemorrhage at all levels, including trocar insertion, operative dissection in a field of dilated veins, and secondary to an underlying coagulopathy. 75 Additionally, ascitic leak from a port site may occur, leading to bacterial peritonitis. Therefore a watertight port site closure should be carried out in all patients.

It is essential that the surgeon be aware of the Child class of severity of cirrhosis of the patient prior to intervening so that appropriate preoperative optimization can be completed. For example, if a patient has an eroding umbilical hernia and ascites, a preoperative paracentesis or transjugular intrahepatic portosystemic shunt (TIPS) procedure in conjunction with aggressive diuresis may be considered. Because these patients commonly are intravascularly depleted, insufflation pressures should be reduced in order to prevent a decrease in cardiac output and minimal amounts of low-salt intravenous fluids should be given.

Economics of Minimally-Invasive Surgery

Minimally-invasive surgical procedures reduce the costs of surgery most when length of hospital stay can be shortened. For example, shorter hospital stays can be demonstrated in laparoscopic cholecystectomy, fundoplication, splenectomy, and adrenalectomy. Procedures such as inguinal herniorrhaphy that are already performed as outpatient procedures are less likely to provide cost advantage. Procedures that still require a 4- to 7-day hospitalization, such as laparoscopy-assisted colectomy, are even less likely to deliver a lower bottom line than their open-surgery counterparts. Nonetheless, with responsible use of disposable instrumentation and a commitment to the most effective use of the inpatient setting, most laparoscopic procedures can be made less expensive than their conventional equivalents.

Robotic Surgery

With the development of advanced laparoscopic procedures, the limitations of minimally-invasive surgical techniques and instrumentation have become accentuated. For example, the mobility and positioning of a laparoscopic instrument is limited by the placement of the port site on the abdominal wall. This may prevent the surgeon from obtaining the desired instrument angle and position to perform a complex maneuver. In addition, the fine motor movements required to perform complex minimally-invasive surgical procedures may be difficult to perform with standard laparoscopic instruments and imaging. Computer-enhanced ("robotic") surgery was developed with the intent of circumventing the limitations of laparoscopy and

thoracoscopy, and to make minimally-invasive surgical techniques accessible to those without a laparoscopic background. 76 In addition, remote site surgery (telesurgery), in which the surgeon is a great distance from the patient (e.g., combat or space), has potential future applications. This was recently exemplified when a team of surgeons located in New York performed a cholecystectomy on a patient located in France. 77

These devices offer a three-dimensional view with hand- and wrist-controlled instruments that possess multiple degrees of freedom, thereby facilitating surgery with a one-to-one movement ratio that mimics open surgery (FIG. 19). Additionally, computer-enhanced surgery also offers tremor control. The surgeon is physically separated from the operating table and the working arms of the device are placed over the patient (FIG. 20). An assistant remains at the bedside and changes the instruments as needed.

FIG. 19.



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Robotic instruments and hand controls. The surgeon is in a sitting position and the arms and wrists are in an ergonomic and relaxed position.

FIG. 20.



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Room set-up and position of surgeon and assistant for robotic surgery.

Because this equipment is very costly, a primary limitation to its uniform acceptance has been attempting to achieve increased value in the form of improved clinical outcomes. There have been two randomized controlled trials that compared robotic and conventional laparoscopic approaches to Nissen fundoplication. 78,79 While there was a reduction in operative time, there was no difference in ultimate outcome. Similar results have been achieved for laparoscopic cholecystectomy. 80 Finally, it may be too early in its development (due to bulky equipment, difficulty in accessing patients, and limited instrumentation) for widespread adoption of this technology.

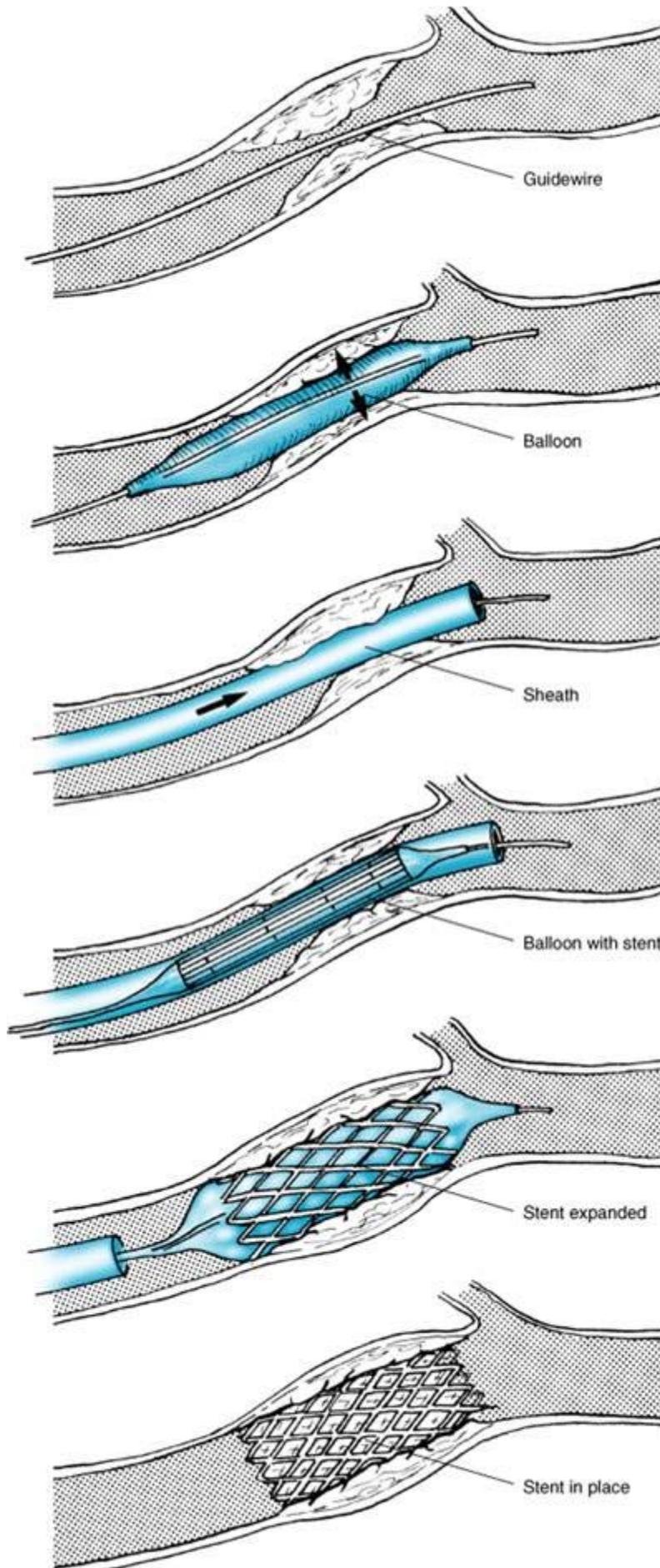
Endoluminal Surgery

The fields of vascular surgery, interventional radiology, neuroradiology, gastroenterology, general surgery, pulmonology, and urology all encounter clinical scenarios that require the urgent restoration of luminal patency of a "biologic cylinder." 81 Based on this need, fundamental techniques have been pioneered that are applicable to all specialties and virtually every organ system. As a result, all minimally-invasive surgical procedures, from coronary artery angioplasty to palliation of pancreatic malignancy, involve the use of an endoluminal balloon, dilator, prostheses, biopsy forceps, chemical agent, or thermal technique 81 (Table 13-2). Endoluminal balloon dilators may be inserted through an endoscope, or they may be

fluoroscopically guided. Balloon dilators all have low compliance—that is, the balloons do not stretch as the pressure within the balloon is increased. The high pressures achievable in the balloon create radial expansion of the narrowed vessel or orifice, usually disrupting the atherosclerotic plaque, the fibrotic stricture, or the muscular band (e.g., esophageal achalasia).
82

Once the dilation has been attained, it is frequently beneficial to hold the lumen open with a stent. 83 Stenting is particularly valuable in treating malignant lesions and in endovascular procedures (FIG. 21). Stenting usually is not applicable for long-term management of benign gastrointestinal strictures except in patients with limited life expectancy 83–85 (FIG. 22A and B).

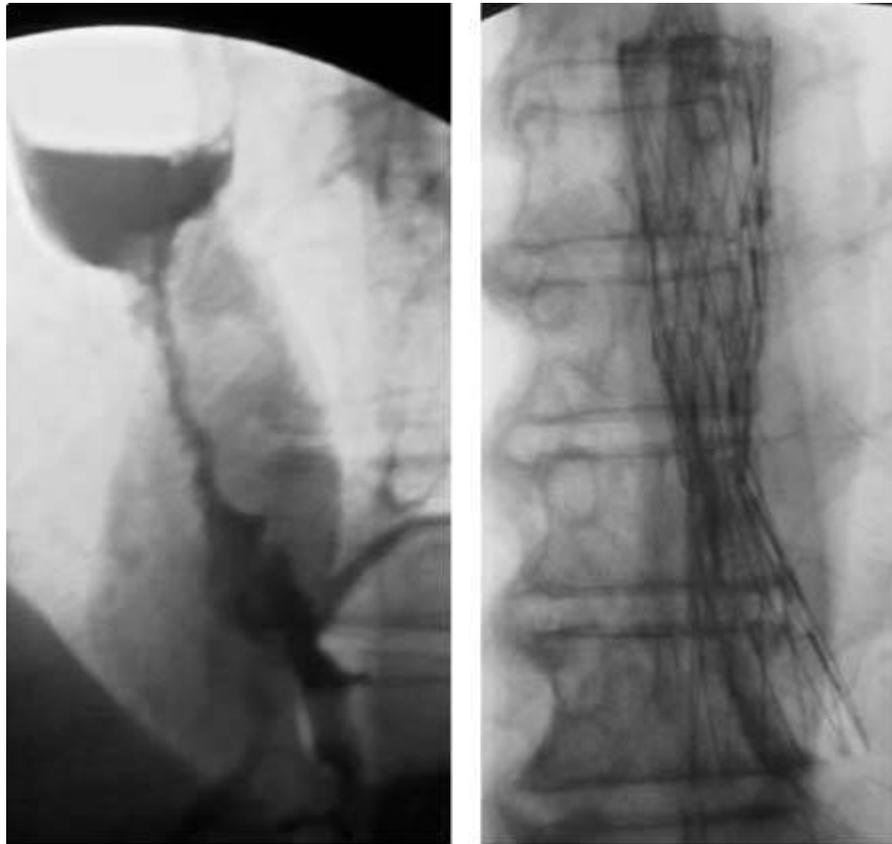
FIG. 21.



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The deployment of a metal stent across an isolated vessel stenosis is illustrated. [*Reproduced with permission from Hunter JG, Sackier JM (eds): Minimally-Invasive Surgery. New York: McGraw-Hill, 1993, p 325.*]

FIG. 22.



A

B

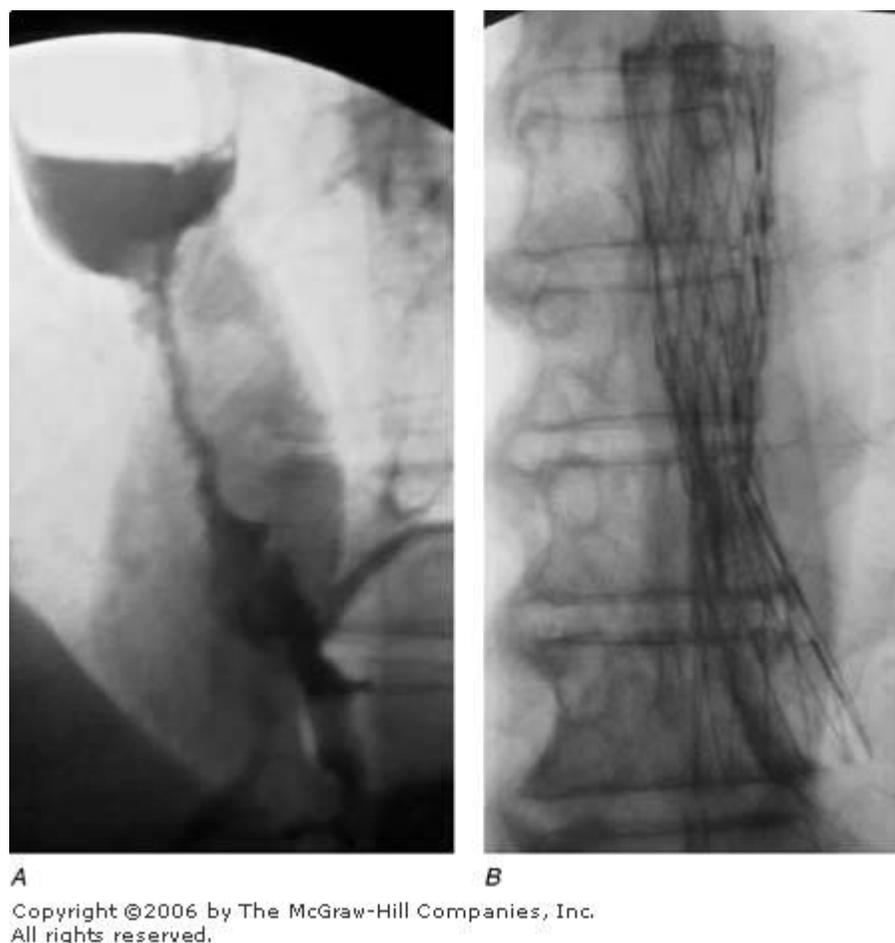
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This is an esophagram in a patient with severe dysphagia secondary to advanced esophageal cancer (A) before and (B) after placement of a covered self-expanding metal stent.

A variety of stents are available that are divided into two basic categories, plastic stents and expandable metal stents 84 (FIG. 23). Plastic stents came first and are used widely as endoprotheses for temporary bypass of obstructions in the biliary or urinary systems. Metal stents generally are delivered over a balloon and expanded with the balloon to the desired size. These metal stents usually are made of titanium or nitinol. Although great progress has been

made with expandable metal stents, two problems remain: propensity for tissue ingrowth through the interstices of the stent and stent migration. Ingrowth may be an advantage in preventing stent migration, but such tissue ingrowth may occlude the lumen and cause obstruction anew. This is a particular problem when stents are used for palliation of gastrointestinal malignant growth, and may be a problem for the long-term use of stents in vascular disease. Filling the interstices with Silastic or other materials may prevent tumor ingrowth, but also makes stent migration more likely. In an effort to minimize stent migration, stents have been incorporated with hooks and barbs.

FIG. 23.



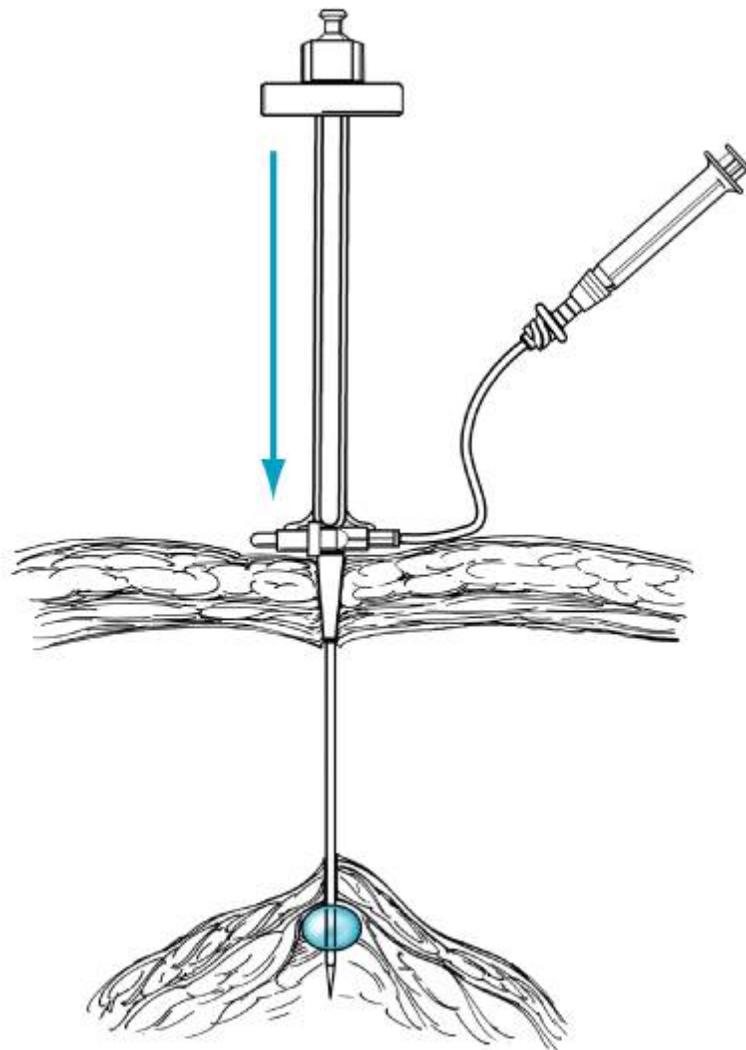
Covered self-expanding metal stents. These devices can be placed fluoroscopically or endoscopically.

Most recently, anticoagulant-eluting coronary artery stents have been placed in specialized centers. 86 This exciting technological advance may dramatically increase the long-term patency rates of stents placed in patients with coronary artery disease and peripheral atherosclerosis.

Intraluminal Surgery

The successful application of minimally-invasive surgical techniques to the lumen of the gastrointestinal tract has hinged upon the development of a port that maintains access to the gastrointestinal lumen while preventing intraperitoneal leakage of intestinal contents and facilitating adequate insufflation 87 (FIG. 24). Procedures that are gaining acceptance include resection of benign and early malignant gastric tumors, transanal resection of polyps (transanal endoscopic microsurgery), pancreatic cyst gastrostomy, and biliary sphincterotomy.

FIG. 24.



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An illustration of a radially expanding trocar used for intraluminal surgery. The stomach is insufflated using a nasogastric tube, and the anterior gastric wall is pierced with the trocar under laparoscopic guidance. A balloon is inflated and used to draw the stomach up to the anterior abdominal wall. [Reproduced with permission from Eubanks WS, Swanstrom LL, Soper NJ (eds): *Mastery of Endoscopic and Laparoscopic Surgery*. Philadelphia: Lippincott

Williams & Wilkins, 1999, p 215.]

The location of the lesion within the gastrointestinal tract is of utmost importance when considering an intraluminal approach. For example, a leiomyoma that is located on the anterior gastric wall may not be amenable to intraluminal resection because the working ports must also penetrate the anterior surface of the stomach. Preoperative endoscopy and endoscopic ultrasound should be routinely employed in order to determine resectability. 87

Education and Skill Acquisition

Surgeons in Training and Skill Acquisition

Surgeons in training acquire their skills in minimally-invasive techniques through a series of operative experiences of graded complexity. This training occurs on patients. With the recent constraints placed on resident work hours, providing adequate minimally-invasive training to future surgeons within a relatively brief time frame has become of paramount importance.

Laparoscopic surgery demands a unique set of skills that require the surgeon to function at the limit of his or her psychomotor abilities. The introduction of virtual reality training devices presents a unique opportunity to improve and enhance experiential learning in endoscopy and laparoscopy for all surgeons. This technology has the advantage of enabling objective measurement of psychomotor skills, which can be used to determine progress in skill acquisition, and ultimately technical competency. 88,89 This technology will most likely be used to create benchmarks for the performance of future minimally-invasive techniques. In addition, virtual reality training enables the surgeon to build an experience base prior to venturing into the operating room. 90 Be that as it may, no studies have demonstrated that simulator training improves overall patient outcome.

Some hospitals and training programs have established virtual reality and laparoscopic training centers that are accessible at all hours for surgeons' use.

Telementoring

In response to the Institute of Medicine's call for the development of unique technologic solutions to deliver health care to rural and underserved areas, surgeons are beginning to explore the feasibility of telementoring. Teleconsultation or telementoring is two-way audio and visual communication between two geographically separated providers. This communication can take place in the office setting, or directly in the operating room when complex scenarios are encountered. Although local communication channels may limit its performance in rural areas, the technology is available and currently being employed (FIG. 25).

FIG. 25.



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Teleconsultation and telementoring are carried out between two providers who are geographically separated. The console has a video camera, microphone, and flat screen display which can be positioned at the operating room table or in the clinic.

Innovation and Introduction of New Procedures

The revolution in minimally-invasive general surgery, which occurred in 1990, created ethical challenges for the profession. The problem was this: If competence is gained from experience, how was the surgeon to climb the competence curve (otherwise known as the learning curve) without injuring patients? If it was indeed impossible to achieve competence without making mistakes along the way, how should one effectively communicate this to patients such that they understand the weight of their decisions? Even more fundamentally important is determining the path that should be followed before one recruits the first patient for a new procedure.

Although procedure development is fundamentally different than drug development (i.e., there is great individual variation in the performance of procedures, but no difference between one tablet and the next), adherence to a process similar to that used to develop a new drug is a reasonable path for a surgical innovator. At the outset the surgeon must identify the problem that is not solved with current surgical procedures. For example, while the removal of a gallbladder through a Kocher incision is certainly effective, it creates a great deal of disability,

pain, and scarification. As a result of those issues, many patients with very symptomatic biliary colic delayed operation until life-threatening complications occurred. Clearly there was a need for developing a less invasive approach (FIG. 26).

Once the opportunity has been established, the next step involves a search through other disciplines for technologies and techniques that might be applied. Again, this is analogous to the drug industry, where secondary drug indications have often turned out to be more therapeutically important than the primary indication for drug development. The third step is in vivo studies in the most appropriate animal model. Certainly these types of studies are controversial because of the resistance to animal experimentation, and yet without such studies many humans would be injured or killed during the developmental phase of medical drugs, devices, and techniques. These steps are often called the preclinical phase of procedure development.

The decision as to when such procedures are ready to come out of the lab is a difficult one. Put simply, the procedure should be reproducible, provide the desired effect, and not have serious side effects. Once these three criteria are reached, the time for human application has arrived. Before the surgeon discusses the new procedure with his patient, it is important to achieve full institutional support. Clearly, involvement of the medical board, the chief of the medical staff, and the institutional review board are essential before commencing on a new procedure. These bodies are responsible for the use of safe, high quality medical practices within their institution, and they will demand that great caution and all possible safeguards are in place before proceeding.

The dialogue with the patient who is to be first must be thorough, brutally honest, and well documented. The psychology that allows a patient to decide to be first is quite interesting, and may under certain circumstances require psychiatric evaluation. Certainly if a dying cancer patient has a chance with a new drug, this makes sense. Similarly, if the standard surgical procedure has a high attendant morbidity and the new procedure offers a substantially better outcome, the decision to be first is understandable. On the other hand, when the benefits of the new approach are small and the risks are largely unknown, a more complete psychological profile may be necessary before proceeding.

For new surgical procedures, it is generally wise to assemble the best possible operative team, including a surgeon experienced with the old technique, and assistants who have participated in the earlier animal work. This initial team of experienced physicians and nurses should remain together until full competence with the procedure is attained. This may take 10 procedures, or it may take 50 procedures. The team will know that it has achieved competence when the majority of procedures take the same length of time, and the team is relaxed and sure of the flow of the operation. This will complete phase I of the procedure development.

In phase II, the efficacy of the procedure is tested in a nonrandomized fashion. Ideally, the outcome of new techniques must be as good or better than the procedure that is being replaced.

This phase should occur at several medical centers to prove that good outcomes are achievable outside of the pioneering institution. These same requirements may be applied to the introduction of new technology into the operating room. The value equation requires that the additional measurable procedure quality exceeds the additional measurable cost to the patient or health care system. In phase III, a randomized trial pits the new procedure against the old.

Once the competence curve has been climbed, it is appropriate for the team to engage in the education of others. During the ascension of the competence curve, other learners in the institution (i.e., surgical residents) may not have the opportunity to participate in the first case series. While this may be difficult for them to swallow, the best interest of the patient must be put before the education of the resident.

The second stage of learning occurs when the new procedure has proven its value and a handful of experts exist, but the majority of surgeons have not been trained to perform the new procedure. In this setting, it is relatively unethical for surgeons to forge ahead with a new procedure in humans as if they had spent the same amount of time in intensive study that the first team did. The fact that one or several surgical teams were able to perform an operation does not ensure that all others with the same medical degrees can perform the operation with equal skill. It behooves the learners to contact the experts and request their assistance to ensure an optimal outcome at the new center. While it is important that the learners contact the experts, it is equally important that the experts be willing to share their experience with their fellow professionals. As well, the experts should provide feedback to the learners as to whether they feel the learners are equipped to forge ahead on their own. If not, further observation and assistance from the experts are required. While this approach may sound obvious, it is fraught with difficulties. In many situations ego, competitiveness, and monetary concerns have short-circuited this process and led to poor patient outcomes. To a large extent, MIS has recovered from the black eye that it received early in development, when inadequately trained surgeons caused an excessive number of significant complications.

If innovative procedures and technologies are to be developed and applied without the mistakes of the past, surgeons must be honest when they answer these questions: Is this procedure safe? Would I consider undergoing this procedure if I developed a surgical indication? Is the procedure as good or better than the procedure it is replacing? Do I have the skills to apply this procedure safely and with equivalent results to the more experienced surgeon? If the answer to any of these questions is "no," or "I don't know," there is a professional obligation to seek another procedure or outside assistance before subjecting a patient to the new procedure.

LAPAROSCOPIC MANAGEMENT OF BILIARY STONE DISEASE

The management of biliary stones diseases has dramatically changed with the advent of the

Laparoscopic Cholecystectomy. It has now become a true outpatient laparoscopic procedure with negligible morbidity.

In the past few years, our surgical team has designed and revised numerous management protocols for various clinical settings effectively achieving impressive improvements in our surgical performance for the treatment of biliary stone diseases. This chapter will describe these management protocols and our latest technical updates.

The original Laparoscopic Cholecystectomy technique has undergone a vast maturation process over the past decade. Various technical steps has been modified and adapted to improve surgical performance and clinical outcome. As a result, nowadays, most surgeons in the Western World can safely perform a Laparoscopic Cholecystectomy with a minimal conversion rate.

ROUTINE INTRA-OPERATIVE CHOLANGIOGRAPHY

Routine operative cholangiography is recommended by most laparoscopic authors in the United States. However, recent reports demonstrate it does not significantly decrease the rate of common bile duct injury in cases where the anatomy is well-identified. Our recommendation is that routine intraoperative cholangiography should be performed by inexperienced laparoscopic surgeons and in cases where the anatomy is not well-defined.

IDENTIFYING PATIENTS WITH CHOLEDOCHOLITHIASIS

In order to achieve the level of Maximum Surgical Performance with this procedure, patients at high risk of presenting with Common Bile Duct Stones need to be identified pre-operatively. The simplest methods to initially identify these patients are:

- 1) History and Physical Examination,
- 2) Liver Function Studies,
- 3) Sonographic Findings.

Patients with a recent history of gallstone pancreatitis, jaundice, or presenting with such symptoms are at a high risk of having common bile duct pathology; the same is valid for patients with altered liver function studies. The most accurate studies are the Serum Transaminases (SGOT, SGPT). Elevations of these enzymes over 20% of their normal values are significant. But patients with severe, acute cholecystitis can occasionally generate such elevations. Also, extreme elevations of these two enzymes could represent hepatocytes necrosis as seen in hepatitis. The bilirubin level may also be elevated in certain patients with acute cholecystitis, but elevations above 2.5 or 3.0 mg/dl could identify a patient with choledocholithiasis. Finally, we find the enzymes LDH and GGTP to have no real specific value in this clinical setting.

It is interesting that in spite of our intensive efforts to identify Common Bile Duct pathology preoperatively, missed Common Bile Duct Stones are found in 1.92% of all patients.

Of these patients 76% will require additional surgical intervention (ERCP).

ROUTINE INTRA-OPERATIVE CHOLANGIOGRAPHY

This technology is being used with increasing frequency in our surgical service to identify patients with choledocholithiasis. A GE Magnetic Resonance machine was used for all studies. To date the specificity and accuracy of these studies in our services is 98.2% for common bile duct stones over 1 mm in size.

ROUTINE INTRA-OPERATIVE CHOLANGIO-SONOGRAPHY

Intra-operative cholangio-sonography is being used in many medical centers to rule out common bile duct stone. Although this modality was used on numerous occasions, we found it too time consuming to be used on a routine basis.

ANTERIOR OR SUBTOTAL LAPAROSCOPIC CHOLECYSTECTOMY

In our never-ending quest of increasing surgical performance, we meticulously analyzed when and why conversion occurred during the performance of a laparoscopic cholecystectomy. Most of them occurred in patients with acute, severe and gangrenous cholecystitis. Thus, we introduced the anterior-subtotal laparoscopic cholecystectomy to be used **ONLY** in these clinical settings when a standard laparoscopic Cholecystectomy could not be completed safely. (Refer to Technique and Surgical Performance later in chapter).

THE DECREASING IMPACT OF THE LAPAROSCOPIC CBD EXPLORATION

Significant problems have impaired the growth of Laparoscopic Common Bile Duct exploration. This technique is simply not easy to perform and good results are only achieved by experienced operators. In addition, this procedure is hardware intensive and the choledochoscopes are not as reliable as they are touted to be. For these reasons it quickly become obvious to us, the indications for this procedure were becoming more and more limited.

Our surgical team promotes the use of Endoscopic Retrograde Cholangiography and Papillotomy. When not feasible, a laparoscopic transcystic or via anterior choledochotomy CBDE is performed. It should be mentioned that some critics claim there are no studies available on the long term effects of endoscopic papillotomies and that it represents a significant additional cost. Although, this statement is correct, there are also no reports of long term adverse effects of such procedures.

CLASSIFYING THE BILIARY STONE PATIENT

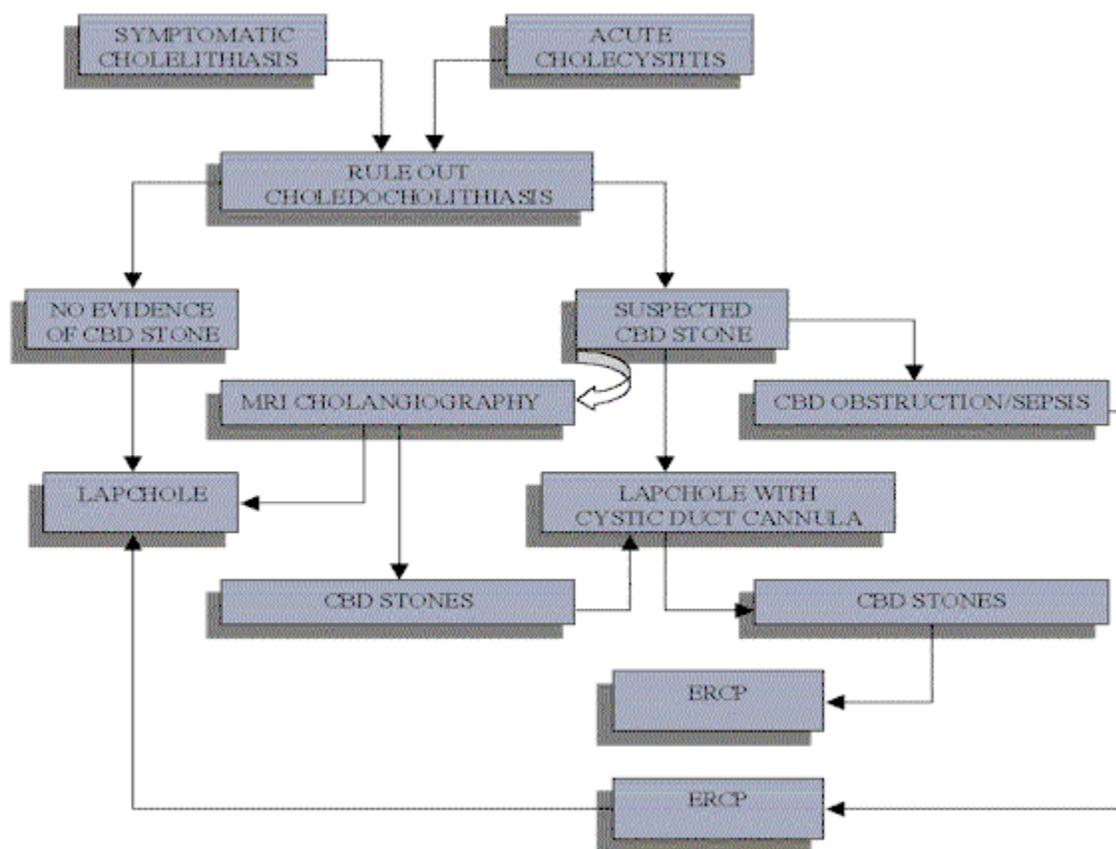
Asymptomatic Cholelithiasis	Incidental Finding on Sonogram
Acute Cholecystitis	Cholelithiasis on Sonogram, clinical Cholecystitis diagnosis or Positive Pipida Scan
Symptomatic Cholelithiasis	Positive Sonogram, normal Liver Function Tests
Cholelithiasis with Suspected Choledocholithiasis	Abnormal Liver Function Tests (Serum Transaminases elevation or Bilirubin >3.0, gallstone pancreatitis)
Cholelithiasis with Choledocholithiasis	CBD Stone on Sonogram, MR Cholangiography or Jaundice
Cholelithiasis with Resolving Gallstone Pancreatitis	Pancreatitis on Sonogram, CT or MER Cholangiography or clinically, Documented High Serum Amylase and Lipase - WITH - Decreasing Serum Pancreatic Enzymes after initial attack

IDENTIFYING PATIENTS WITH CHOLEDOCHOLITHIASIS

MANAGEMENT PROTOCOLS FOR UNCOMPLICATED BILIARY STONE DISEASES	PROPOSED MANAGEMENT
Asymptomatic Cholelithiasis	No Surgical Intervention
Asymptomatic Cholelithiasis in Diabetic Patients	No Surgical Intervention
Symptomatic Cholelithiasis or Acute Cholecystitis	LapChole
Symptomatic Cholelithiasis with Suspected Choledocholithiasis	LapChole with Insertion of Cystic Duct Cannula with Cholangiography, if Choledocholithiasis, postop ERCP
Symptomatic Cholelithiasis with Choledocholithiasis	LapChole with Insertion of Cystic Duct Cannula with Cholangiography, if Choledocholithiasis, postop ERCP
Cholelithiasis with Resolving Pancreatitis	LapChole with Insertion of Cystic Duct Cannula with Cholangiography, if Choledocholithiasis, postop ERCP

Cholelithiasis with Unresolved Pancreatitis	After acute phase subsides, MRI Cholangiography or ERC, if Choledocholithiasis ERCP followed by LapChole
Asymptomatic Gallbladder Polyps	No Surgical Intervention
Symptomatic Gallbladder Polyps	LapChole
Severe, Gangrenous Cholecystitis with Subhepatic Phlegmon	LapChole, if not safely feasible, Anterior-subtotal LapChole
Post Cholecystectomy (Lap or open) Suspected Choledocholithiasis	MR Cholangiogram or ERC
Post Cholecystectomy (Lap or open) Choledocholithiasis	ERCP, if failure Laparoscopic Common Bile Duct Exploration.

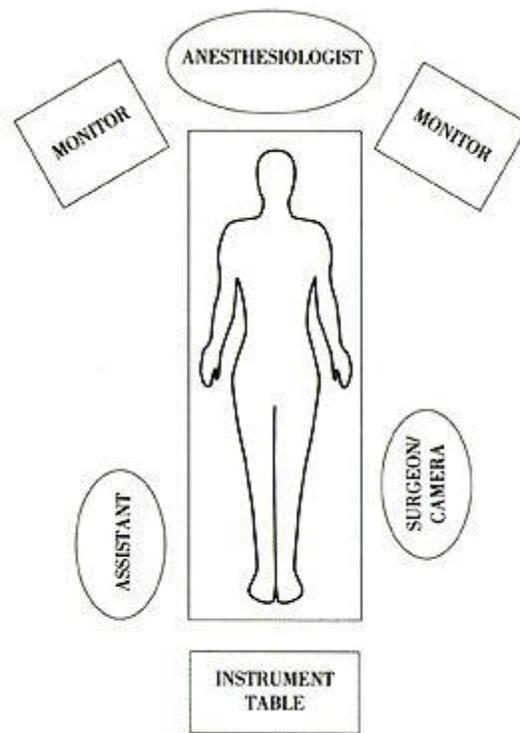
MANAGEMENT PROTOCOL DIAGRAM



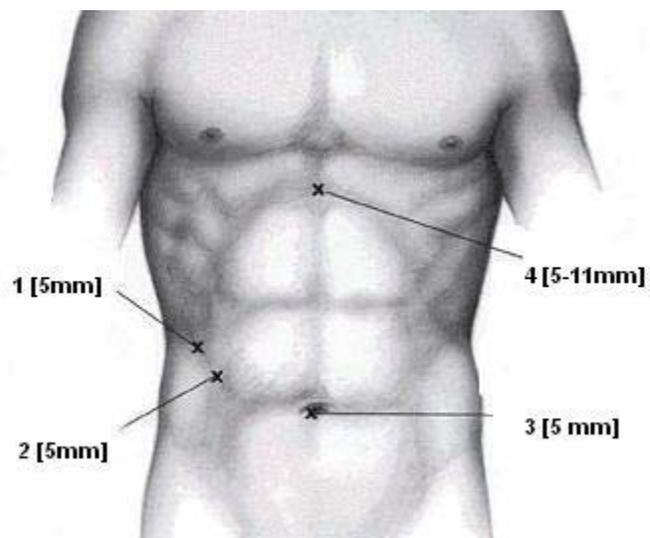
THE TECHNIQUES

- Operating Room Set-up
- Instruments
- Trocars Placement
- Additional Informed Consent
- Technique: Standard Lap- Cholecystectomy
- Technique: Intra-operative Cholangiography
- Technique: Laparoscopic Common Bile Duct Exploration - Transcystic
- Technique: Laparoscopic Common Bile Duct Exploration - Choledochotomy
- Technique: Anterior/subtotal Laparoscopic Cholecystectomy
- Technique: LapChole with Cystic Duct Cannulation
- Return to Chapter Table of Contents
-

OPERATING ROOM SET-UP



TROCARS PLACEMENT



Trocar	Type	Location
1	5 mm Trocar	<u>RUQ Lateral</u> - 4 cm below costal margin
2	5 mm Trocar	<u>RUQ Medial</u> - 4 cm below costal margin
3	5 mm Trocar	<u>Sub-umbilical</u>
4	Universal Trocar 5/10 or 5/11	<u>Epigastrium</u>

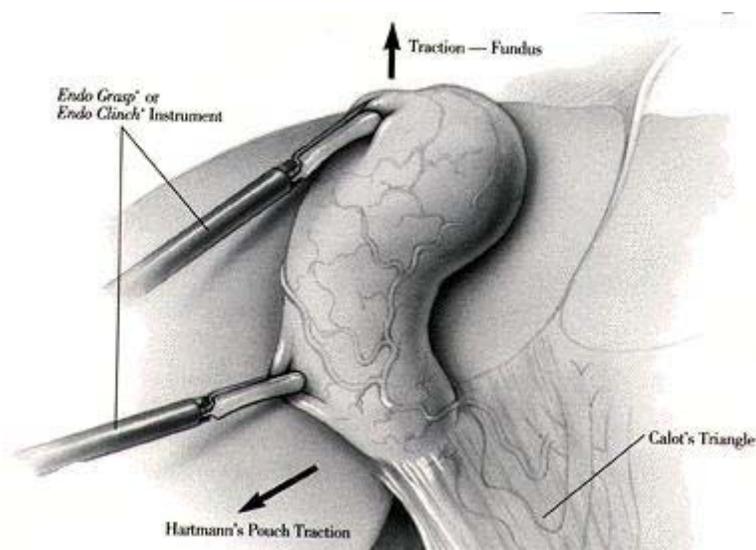
STANDARD LAPAROSCOPIC CHOLECYSTECTOMY

The pneumoperitoneum is obtained in the usual fashion. The trocars are inserted as indicated.

STEP 1: EXPOSING THE CYSTIC DUCT AND ARTERY

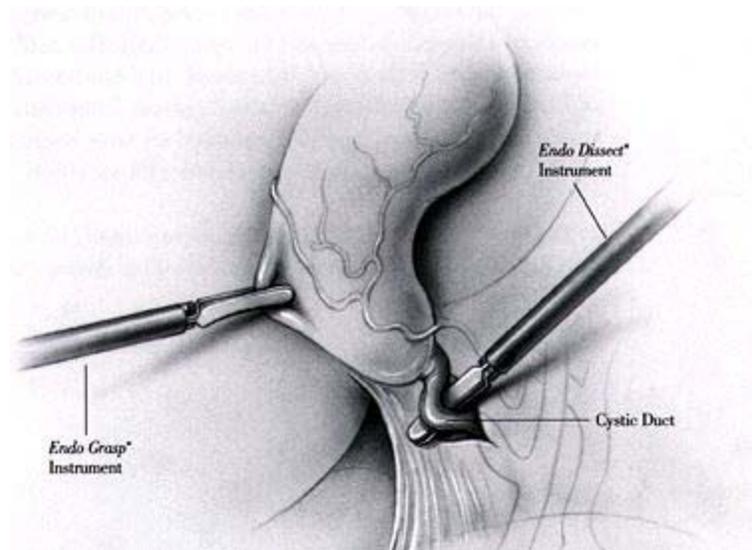
The stationary grasper [1: lateral position] is utilized to grasp the tip of the gallbladder and push it over the anterior edge of the liver by progressive traction. Hartmann's pouch is pulled upward. This exposes the cystic duct and artery as well as the common bile duct. It is important to constantly maintain this traction. In most cases, the scrub nurse or assistant hold this retractor. In difficult, longer cases, the handle of the grasper is clamped onto the skin of the abdomen or onto the protective field. The patient is now positioned head down.

CAUTION: It is not always possible to push the tip of the gallbladder (Re: cirrhotic patients) over the anterior hepatic edge. In these cases, gently push its tip against the liver, being very meticulous not to penetrate the parenchyma of the liver.



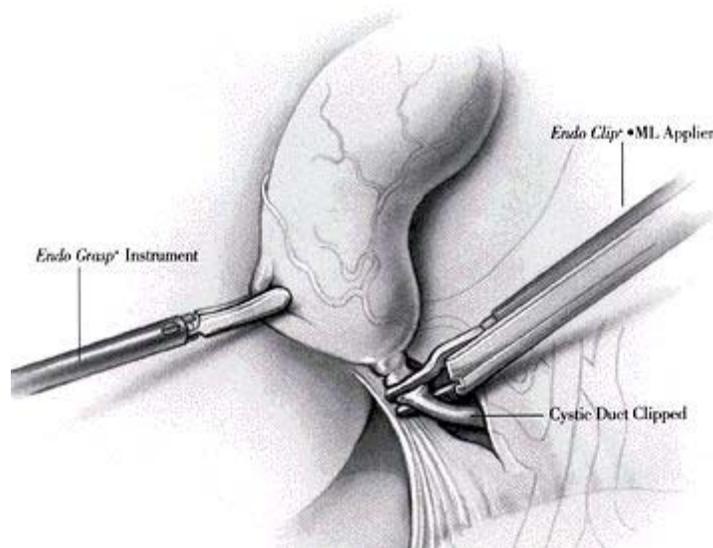
STEP 2: DISSECTING THE CYSTIC DUCT AND ARTERY

Once the field is exposed, Hartmann's pouch is grasped with the lateral working grasper and pulled laterally, further exposing Calot's triangle. The operator will then pass a dissecting grasper through the subxyphoid trocar and begin to identify the cystic duct. In acute cholecystitis, edematous layers of tissue will have to be stripped downward to expose the cystic duct.



The subxyphoid Dolphin Nose Grasper instrument is passed behind the cystic duct or actually between the cystic duct and the cystic artery. In most cases, the duct is anterior to the artery.

CAUTION : Hartmann's pouch should always be identified and visualized. The dissection of Calot's triangle can be done safely starting from the pouch and moving toward the cystic duct. This is particularly important in acute cases, when anatomical landmarks are difficult to find. It is essential to visualize Calot's triangle, which includes the cystic artery, cystic duct and the common bile duct. If visualization of this area becomes difficult, always check the tension on the stationary grasper and the intra-abdominal pressure.



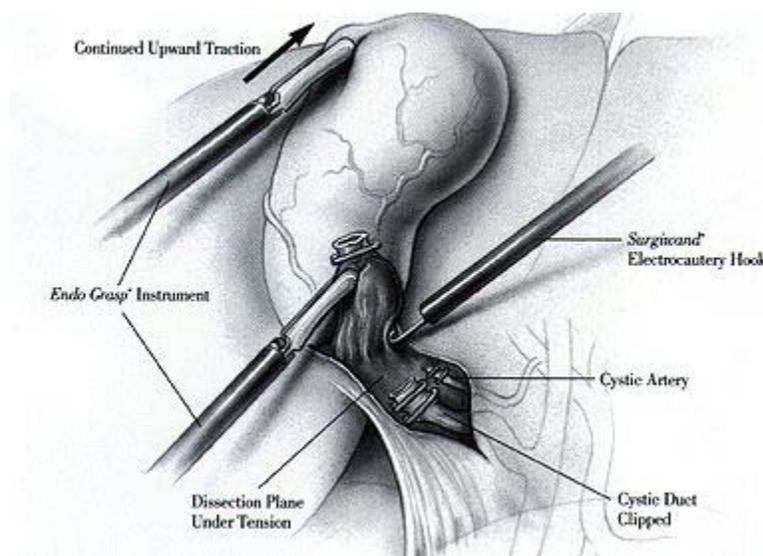
STEP 3: ROUTINE INTRA-OPERATIVE CHOLANGIOGRAM

To view the technique of Routine Intra-operative Cholangiography.

STEP 4: TRANSECTING THE CYSTIC DUCT AND ARTERY

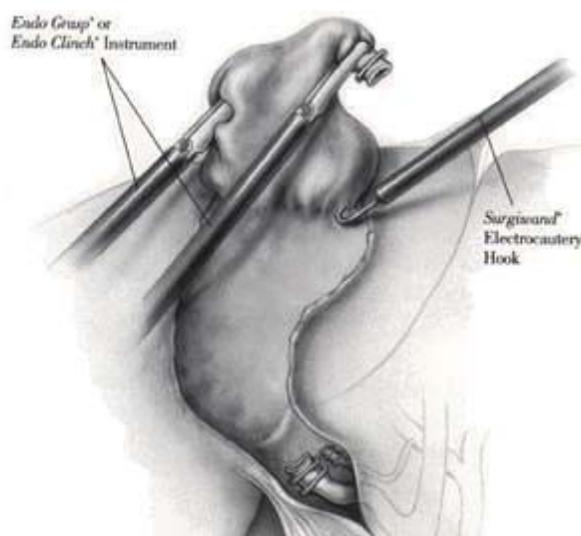
At this juncture, the cystic window is created (i.e., free space behind the cystic duct and the cystic artery). The clip applier is inserted via the subxyphoid trocar. The cystic duct and artery are clipped (three clips) as close as possible to the gallbladder. The ENDO CLIP* Applier is then withdrawn and the EndoShears™ instrument is inserted to cut them.

CAUTION: Be very careful to clearly identify the junction of the gallbladder and cystic duct and plan your transection from this anatomical landmark. In doubt, always check with an IOC.



STEP 5: DISSECTING THE BODY OF THE GALLBLADDER

Hartmann's pouch is now retracted upward. Using the EndoShears* instrument, the most lower lateral aspect of Hartmann's pouch should be dissected meticulously.



The ENDO SHEARS*instrument is withdrawn and replaced by the electrocautery hook. The gallbladder is retracted upward and tension is placed on the surgical plane between the gallbladder and its liver bed. The dissection is extended to the top of the gallbladder. Occasionally the grasper holding the cystic duct stump can be used to flip the body of the gallbladder around the stationary grasper which is still holding the fundus of the gallbladder.

In most instances, this dissection will generate smoke which can impair the surgeon's visualization. This smoke can be aspirated by opening the insufflation of the lateral trocar.

STEP 6: EXTRACTING THE GALLBLADDER

A 10 mm, large grasper is introduced via the sub-xyphoid trocar. The two lateral graspers holding the gallbladder present the gallbladder to the newly introduced large grasper. The gallbladder is pulled from the the intra-abdominal cavity through the same trocar site. This trocar site can enlarged bluntly with a peon clamp of a few millimeters. An Endocatch™ Instrument can be used to remove the specimen.

The intra-abdominal cavity is then thoroughly irrigated with normal saline. All stones that have dropped into the intra-abdominal cavity are retrieved with a morcillator or stone retrieving forceps.

The abdomen is deflated; the trocars removed, and the trocar insertion sites are closed in the usual fashion.

ROUTINE INTRA-OPERATIVE CHOLANGIOGRAM

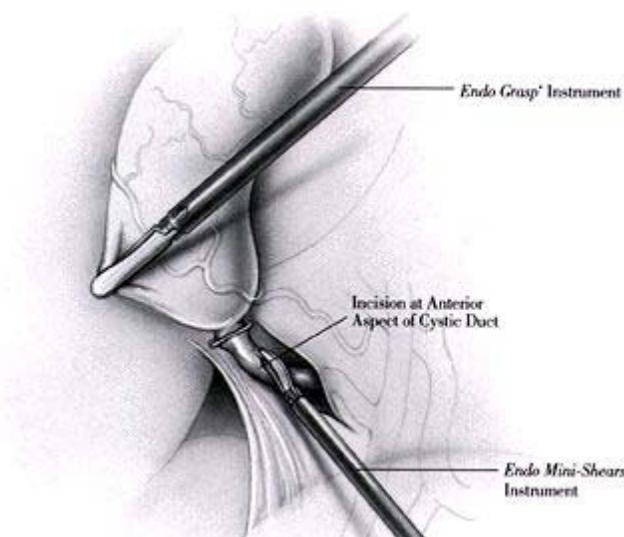
Additional Instruments:

1. Storz Cholangiograsper 5 mm
2. 1 Ureteral Catheter 4 or 5 F with Adapter
3. Dye Used: Renographin 60

STEP 1: EXPOSING THE CYSTIC DUCT AND ARTERY

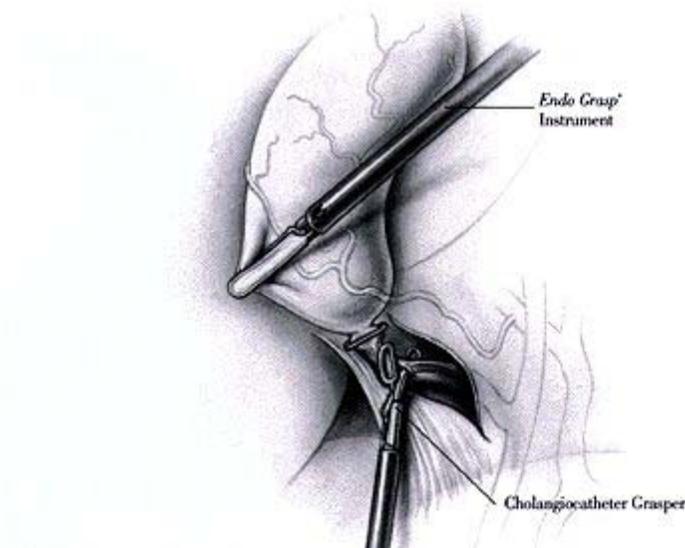
The cystic duct is dissected meticulously as close as possible to the gallbladder. The ENDO CLIP* Applier is inserted via the sub-xyphoid trocar and the cystic duct is clipped at its junction with the gallbladder. While maintaining the same exposure, the ENDO CLIP* applier is withdrawn, an ENDO SHEARS* instrument is inserted via the subxyphoid trocar.

An anterior incision is made on the cystic duct.



The ENDO SHEARS* instrument is then withdrawn and a cholangi catheter grasper with a French #4 catheter is inserted. All the side ports of the catheter have been eliminated by cutting the last 3 cm of the tip. The catheter is inserted into the duct.

In most cases, the intraluminal valves on the cystic duct will make this insertion difficult. However, with the cholangi catheter only the very tip of the catheter needs to be inserted to ensure the flow of bile enters the common bile duct. The grasper is closed around the duct; the jaws should enclose the entire width of the duct for better performance. The catheter is irrigated, and no leak should be seen around the entry site.



If the injection of dye is difficult and slow, and in most cases it is, use a 10cc syringe to inject the dye.

The cholangiogram is obtained. The following should be visible on the radiogram:

1. The cystic duct
2. The common bile duct with its hepatic bifurcation
3. Renografin in the duodenum
4. Absence of CBD stones

After completing the operative cholangiogram, the cholangiometer and grasper are removed.

LAPAROSCOPIC COMMON BILE DUCT EXPLORATION: TRANS-CYSTIC DUCT

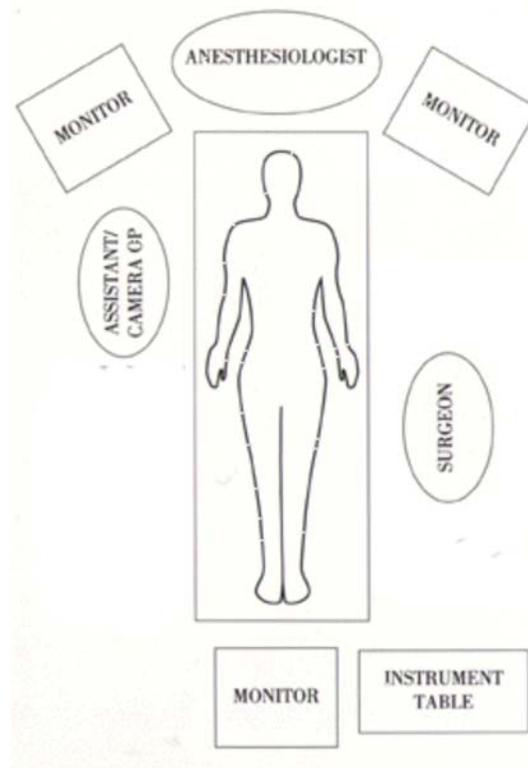
As previously mentioned, the number of laparoscopic common bile duct explorations performed on our surgical service has dramatically decreased over the past few years. These explorations are now rare and usually performed in post-cholecystectomy patients with Choledocholithiasis who have failed endoscopic retrieval. We strongly believe Choledocholithiasis is best treated by non-surgical methods such as an Endoscopic Retrograde Cholangiography and Papillotomy.

Two techniques are used to perform a common bile duct exploration via laparoscopy. These are

- 1) the cystic duct dilatation and retrieval and,
- 2) the anterior choledochotomy. Nowadays, we almost exclusively use the laparoscopic anterior choledochotomy.

Pre-exploration Work-up: A correct diagnosis should be made prior to the actual initiation of the procedure. An intraoperative cholangiogram or another imaging study should demonstrate common bile duct pathology unequivocally.

OPERATING ROOM SET-UP:



Additional Instruments and Hardware:

A second Storz Camera with a monitor

1 - 5 mm trocar (available)

Additional Instruments

1 Storz Ureteroscope- 3.0 mm or 3.5 mm with a 1.5 mm working channel

1 Phantom 5 Plus Balloon Catheter (Microvasive /75cm, 5 Fr./6 mm, 18 Fr.) with Catheter Introducer

1 LeVein Inflator 10 cc with Pressure Gauge

1 Glide Wire 0.35/150 cm with straight tip

1 Segura Stone Retrieval Stone Basket 2.4F Mini (120 cm)

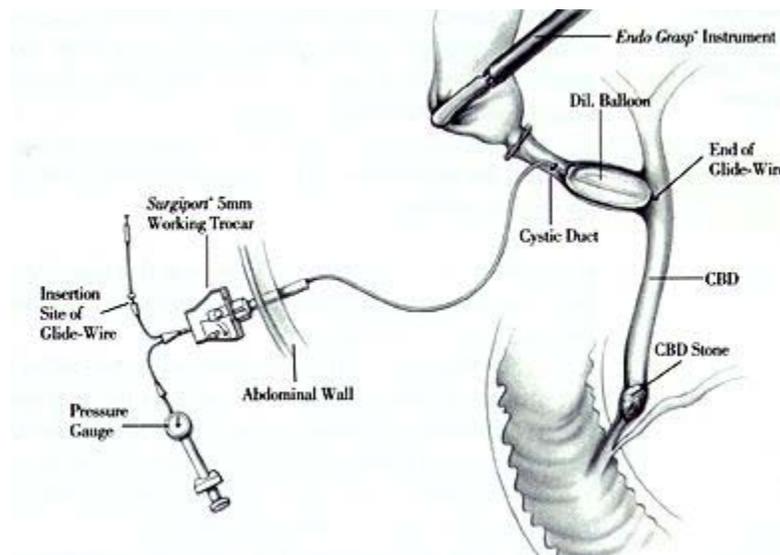
The Technique

STEP 1: THE INTRA-OPERATIVE CHOLANGIOGRAM

This technique is used at the time of a laparoscopic cholecystectomy. An operative cholangiogram has confirmed the presence of a common bile duct stone. At this point, a clip has been placed at the junction of the gallbladder and the cystic duct. The cholangio-catheter has been removed. The cystic duct should not be cut. An intact common bile duct is necessary to maintain sufficient tension for easy access into the cystic duct and the common bile duct.

STEP 2: CANNULATING THE CYSTIC DUCT

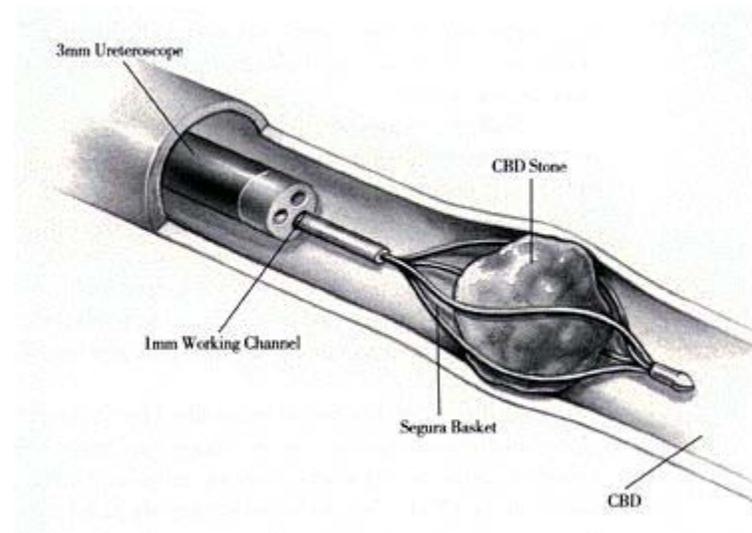
The Phantom 5 Plus Catheter is connected to the LeVeen Inflator with Pressure Gauge. The catheter is inserted via the lateral 5 mm trocar into the intraabdominal cavity. A long 4.5 mm sealed, steel shaft is used to minimize air leaks and to facilitate insertion of the catheter into the cystic duct.



A glide wire is inserted into the central channel of the Phantom 5 Plus Catheter. This glide wire is inserted into the Cystic duct and into the common bile duct using direct vision. The dilating catheter is then passed over the glide wire into the common bile duct. The balloon of the catheter entering the cystic duct is positioned at the entrance of the cystic duct. The balloon is inflated for five minutes at 12 atmospheres of pressure. The entrance of the cystic duct has now been dilated to accommodate a standard 3.0 mm ureteroscope.

STEP 3: INSERTING THE CHOLEDOCHOSCOPE

The Phantom 5 Plus Catheter is then removed and replaced by the ureteroscope. This scope is either connected to an additional camera and monitor, or to an additional camera with a image splitter. The ureteroscope is inserted into the cystic duct with a high pressure saline flow. It is pushed into the common bile duct which is visualized and fully explored.



STEP 4: RETRIEVING THE CBD STONES

Once a stone is seen, the tip of the ureteroscope is placed proximal to the stone. A Segura Basket is inserted into the working channel of the ureteroscope, advanced into the common bile duct and passed beyond the stone. It is then opened and slowly withdrawn under direct vision. When the stone is in the basket, the basket is closed and the stone grasped. The entire apparatus, including the ureteroscope and the wire basket, is pulled out of the common bile duct and the cystic duct. The stone is then released into the intraabdominal cavity and retrieved in the usual manner.

LAPAROSCOPIC COMMON BILE DUCT EXPLORATION: CHOLEDOCHOTOMY

Additional Instruments and Hardware:

A second Storz Camera with a monitor

1 - 5 mm trocar (available)

Biliary Fogarty Catheters (5, 6 F)

Zsabo-Berci Needle Driver or EndoStitch Instrument

Laparoscopic Sutures

T Tube (Sizes 12 - 18 should be available)

The Technique

This can be performed at the time of a laparoscopic cholecystectomy or in the post-cholecystectomy patient. In the latter group, the trocars used are the same as for a standard laparoscopic cholecystectomy.

STEP 1: EXPOSING THE CBD

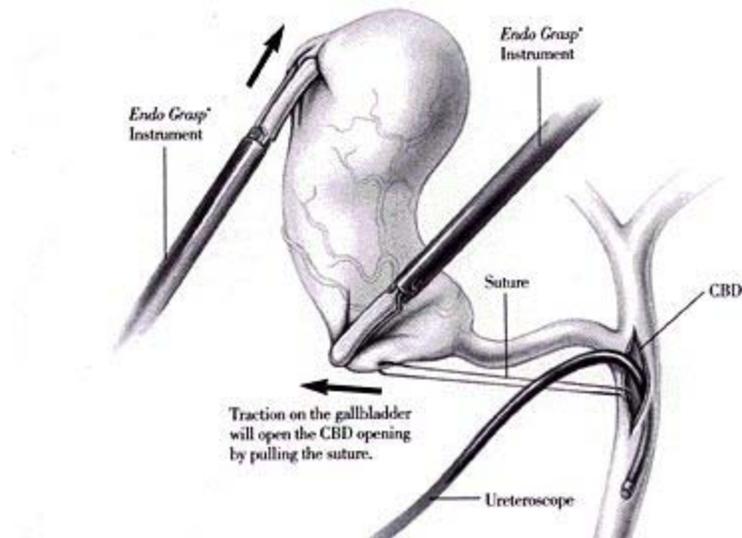
The common bile duct should be equivocally identified. We rarely proceed with a common bile duct exploration if the duct is 1cm or less in diameter. A confirmation of the diagnosis is imperative either via an intra-operative cholangiography or with an intra-operative sonographic study. A meticulous dissection of the common bile duct is performed using the ENDO SHEARS* Instrument and a non traumatic grasper from the hepatic bifurcation to the superior aspect of the pancreas. A section of the common bile duct of about 2 cm should be exposed. In some cases, the gallbladder is used to give additional retraction as demonstrated in the following picture. An endoscopic suture can be placed on the lower portion of the gallbladder and the lateral aspect of the common bile duct. In most cases however, we perform a choledochotomy without retraction sutures.

STEP 2: THE ANTERIOR CHOLEDOCHOTOMY

The anterior choledochotomy is performed by inserting the ENDO SHEARS* Instrument via the subxyphoid trocar, grasping the common bile duct with an ENDO DISSECT* Instrument (via the lateral trocar) and incising the CBD (15 to 20mm).

STEP 3: CLEARING THE CBD

Once the choledochotomy is done, the common bile duct is flushed using our high pressure irrigation device. A Biliary Fogarty Catheter is then used. It is inserted via the subxyphoid trocar and into the common bile duct, run proximally and distally. This step usually retrieves most of the common bile stones.



STEP 4: THE CHOLEDOCHOSCOPY

A Choledochoscopy is performed. An additional camera and monitor are used to connect the flexible 3 mm choledochoscope or ureteroscope. In this setting, larger ureteroscopes can be utilized as the choledochotomy can accommodate larger sizes. Stones are retrieved using a Secura Basket via the working channel of the telescope.

STEP 5: INSERTING THE T TUBE

Once the common bile duct is shown to be free of stones, a T Tube is inserted. The T Tube is usually inserted via the subxyphoid trocar after its limbs have been cut (each should be 1.0 cm in length). It is then inserted entirely into the intra-abdominal cavity. An additional 5 mm trocar is inserted in the RUQ. A grasper is inserted via this new trocar to grasp the long limb of the T Tube. The T Tube is then pulled through the anterior abdominal wall along with the trocar. The T Tube is then inserted into the common bile duct, using two graspers or ENDO DISSECT*. The common bile duct is sutured closed with endoscopic sutures. A completion Cholangiogram is then obtained.

SUBTOTAL OR ANTERIOR LAPAROSCOPIC CHOLECYSTECTOMY

Indications: Acute, severe, gangrenous Cholecystitis and the inability to complete a safe standard laparoscopic Cholecystectomy.

Operating Room Setup: Same as Standard LapChole

Hardware: Same as Standard LapChole

Instruments: Same as Standard LapChole

Additional Instruments: Two Blake drains with drainage reservoir

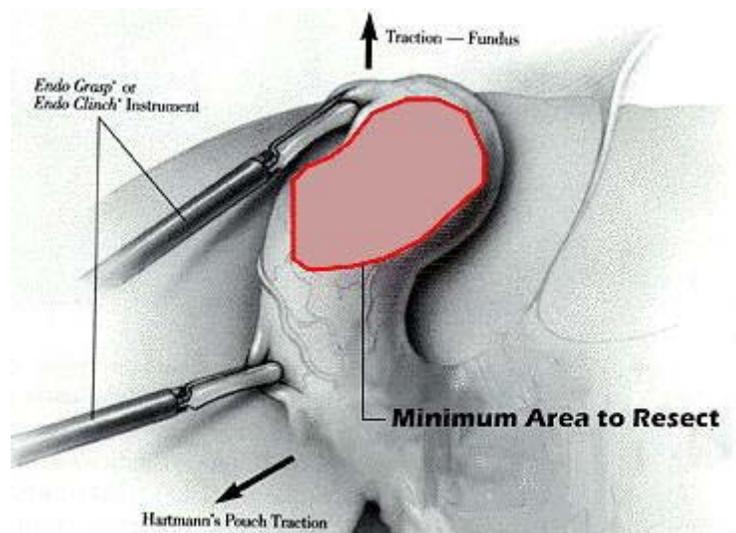
All patients are given Toradol (Roche Pharmaceuticals) and Cefizox (Fujisawa USA) during induction.

The Technique

A standard laparoscopic cholecystectomy has been initiated by the surgeon, at which time he assesses an anterior laparoscopic cholecystectomy should be performed.

Using the stationary or lateral 5 mm grasper, the tip of the fundus of the gallbladder is grasped and retracted cephalad. An ENDO SHEARS* Instrument is inserted via the sub-xyphoid trocar (with electrocautery connection). Using the other lateral grasper, the anterior aspect of the gallbladder is dissected meticulously. The dissection should be extended as low as possible toward the cystic duct without compromising the safety of the procedure.

Using the ENDO SHEARS* Instrument, the gallbladder is entered and the anterior wall of the gallbladder should be resected. Hemostasis should be controlled with the ENDO SHEARS* Instrument connected to an electrocautery source. Spilled gallstones should be retrieved and removed with a morcillator-type grasper (10 mm). The specimen should be removed via the sub-xyphoid trocar. The gallbladder fossa should be flushed thoroughly with normal saline.



Two Blake™ drains are inserted into the intra-abdominal cavity. The best method for the

insertion of these drains is to insert a 5 mm grasper via one of the lateral trocars into the intra-abdominal cavity and out through the sub-xyphoid trocar. The sub-xyphoid trocar is removed. The end of the Blake drain is grasped by the grasper outside the abdominal cavity and pulled back into the intra-abdominal cavity. The lateral grasper pulls it via the 5 mm trocar site. One drain is inserted into the open gallbladder fossa and the other into the sub-hepatic fossa.

The procedure is completed as usual.

Technical Notes

Postoperative Management: Postoperatively, the clinical behavior of these patients is the same as for all patients undergoing a minimally invasive procedure. The next day they usually are on a regular diet and ambulatory. Unless they have associated medical problems, most patients can be discharged within 48 hours. They are discharged with Blake drains in place. Interestingly, some patients will have a bile leak-drainage noticeable on postoperative day one, and others will not. This is most likely secondary to a blocked cystic duct secondary to an impacted gallstone. Nonetheless, these drains are to remain in place for two weeks or until they cease to drain.

Bile Leak-Drainage: Most patients will have significant bile drainage, as this procedure effectively creates a controlled bilio-cutaneous fistula. The average or mean bilious drainage is two days. The longest recorded drainage has been 21 days. As a rule, in the absence of a distal common bile duct obstruction, all bilious drainage or leaks will cease within three weeks.

Associated Complications: This procedure does not allow the performance of an intra-operative cholangiogram or the placement of a cystic duct cannula. One patient was found postoperatively to have a retained common bile duct stone requiring an Endoscopic Retrograde Cholangiography and Papillotomy. Another patient developed a sub-phrenic abscess and eventually required a laparotomy.

Impact of Anterior-subtotal Laparoscopic Cholecystectomy On the Conversion Rate: Prior to introducing this procedure, most conversions occurred in patients with acute, severe, and gangrenous cholecystitis. This procedure effectively decreased our conversion rate as soon as it was introduced. Actually, since its introduction, 896 LapCholes were done with only one conversion. This reduction in the conversion rate is probably the most significant advantage of this technique.

MANAGEMENT OF CHOLEDOCHOLITHIASIS WITH A CYSTIC DUCT CATHETER

Controversies in the Management of CBD Stones

In the United States, surgeons perform approximately 600,000 laparoscopic

cholecystectomies (LC) per year. LC's have largely superseded open cholecystectomies (OC) as the preferred method of gallbladder removal, accounting for 80% of such procedures in this country. One limitation of LC as compared to OC is the difficulty in dealing with common bile duct (CBD) stones. CBD stones are present in approximately 15% of patients, and are responsible for considerable morbidity and mortality (specifically pancreatitis and ascending cholangitis) which mandates the removal of such stones.

In OC, surgeons can routinely remove CBD stones via common bile duct exploration (CBDE), a natural extension of the operative procedure. In LC however, techniques for detection of CBD stones (intraoperative cholangiography or IOC) and subsequent removal are beset with pitfalls. IOC, performed by injection of dye via a cystic duct catheter placed surgically, adds significant time to the operative procedure. It also requires commitment of additional equipment and personnel to the operating room, and has a false positive rate of stone detection of up to 12%, sometimes resulting in unnecessary CBDE. Furthermore, the finding of stones on operative cholangiogram obligates the surgeon to perform CBDE, either laparoscopic or open. A laparoscopic CBDE is a time consuming, hardware intensive procedure, has a steep learning curve, is associated with up to a 50% failure rate, and risks injury to the CBD. Conversion to open CBDE negates the value of a laparoscopic procedure. Another alternative in patients with stones seen on IOC is to refer the patient postoperatively for ERCP, papillotomy, and stone removal. However, a technical failure rate of up to 15% in some series could lead to a second operative procedure, open CBDE.

A number of researchers have attempted to define parameters which could be useful in preoperative prediction of CBD stones. This includes the presence of any of several parameters: 1) Increased liver enzymes, 2) Preoperative pancreatitis, jaundice, or cholangitis, 3) A dilated CBD or intraductal stone on ultrasound, is predictive of CBD stones 25-48% of the time. Furthermore, stones can be present up to 8% of the time in the absence of such parameters or risk factors. Strategies to deal with possible CBD stones in patients with risk factors are complex. One strategy is to do preoperative ERCP with removal of stones (if present). The problem is that 50-75% of ERCP's performed because of the presence of a risk factor will show no stones. Thus, a large number of unnecessary ERCP's will be performed, with a complication rate of 5-10%, and a technical failure rate of up to 15% (i.e. failure to cannulate CBD). A second strategy is to do IOC on patients with risk factors, and to do intraoperative stone removal if stones are detected. The problem with this, as mentioned is that IOC, is time-consuming and associated with up to 12% false positive rate. Subsequent intraoperative stone removal is both time consuming and risky, and often subjects the patient to an open procedure. A third strategy is to do postoperative ERCP if the IOC shows stones. Again, the problem here is that up to a 15% failure risk associated with ERCP would subject the patient to another surgical procedure to remove the stones.

USING A CDC FOR A POST-OPERATIVE CHOLANGIOGRAM

We have developed a new and simple technique for cholangiography that we believe will largely supplant existing complicated algorithms for dealing with CBD stones. In this

laparoscopic technique, in lieu of performing IOC, we secure a standard ERCP catheter (Microvasive, tapered tip) in the cystic duct intraoperatively and leave the catheter in place after surgery.

Postoperatively, all patients undergo a cholangiogram in the x-ray department via the catheter. If no stones are demonstrated, then the catheter is pulled. If stones are present, then the endoscopist performs postoperative ERCP and papillotomy to remove the stones, and then pulls the transcystic catheter.

THE LAPCHOLE WITH CDC PLACEMENT

Operating Room Setup: Same as Standard LAPCHOLE

Hardware: Same as Standard LAPCHOLE

Instruments: Same as Standard LAPCHOLE

Additional Instruments:

- 1 Blake Drain with drainage reservoir
- 1 Ureteral 7 French Ureteral Catheter or
- 1 Fluoro Tip ERCP Cannula Tapered Tip
(210 cm - 5 French 1.7 mm with stainless steel stylet)

Technique

The procedure is initiated as described in the Standard LAPCHOLE Chapter.

1. Inserting the Cystic Duct Cannula in the Intraabdominal Cavity

The cystic duct is exposed and clipped at its junction with the gallbladder with an endoclip. Traction is maintained on Hartmann's pouch to expose the cystic duct. An anterior incision is made with the ENDO SHEARS* instrument.

The cystic duct cannula is inserted via the subxyphoid trocar site. First, the trocar is quickly removed from the subxyphoid site. The site is plugged with a finger and the cannula is inserted bluntly into the intraabdominal cavity under direct vision. When 10 to 15 cm of the cannula is in the intraabdominal cavity, the VERSAPORT* trocar is reinserted bluntly next to the cystic duct cannula. Both the cannula and the trocar are now side by side in the subxyphoid insertion site. The cannula can be advanced, withdrawn and manipulated very easily from the outside of the abdominal cavity.

2. Placing the Cannula in the Biliary Tree

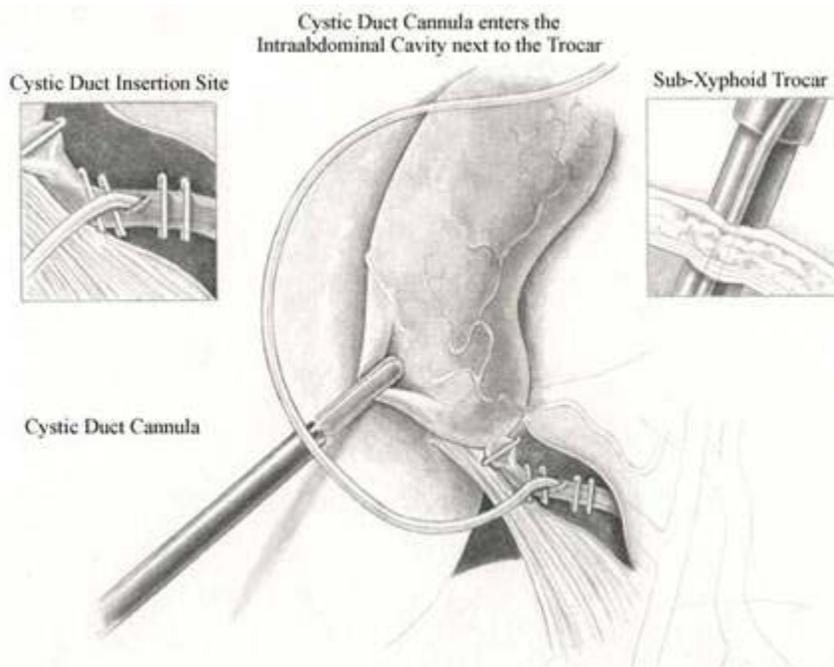
An ENDODISSECT* Instrument or an atraumatic grasper is inserted via the subxyphoid trocar and grasps the tip of the cystic duct cannula. It is inserted into the cystic duct under direct vision and advanced into the common bile duct.

We routinely advance the cannula for about 5-6 cm, and then withdraw the cannula to leave approximately 1.5 to 2 cm inside the cystic duct.

3. Securing the Cannula in the Cystic Duct

The ENDO DISSECT* or grasper is removed from the intraabdominal cavity and replaced with the ENDO CLIP* Applier. It is essential to use a USSC ENDO CLIP* or a SURGICON applier. They are the only instruments that will allow the performance of the next maneuver.

Two clips are placed on the cystic duct. It is essential NOT to close the entire clip around the cystic duct so as not to entirely obliterate the duct and cannula. The partial closing of the clip can only be performed with the USSC ENDO CLIP* applier. (The Ethicon clip Applier does not have this capability.) Another clip is tightly placed behind the cannula. If using the SURGICON clip applier, only one clip is used on the cannula and behind it.



The ENDO CLIP* applier is now replaced with the ENDO DISSECT* Grasper. The Cannula is grasped outside the cystic duct and pulled .5 cm to check that the cannula is not crushed or locked onto the cystic duct. Then additional cannula is inserted into the intraabdominal cavity to provide slack, so it can be placed laterally to allow for the completion of the laparoscopic cholecystectomy. A Blake Drain is inserted at the end of the procedure.

An intraoperative cholangiogram can be performed. If negative, the cannula is removed. We routinely do not perform an intraoperative cholangiogram. We order it a few hours after the procedure.

POST-OP ERCP

SCENARIO 1: CHOLEDOCHOLITHIASIS IS DEMONSTRATED ON THE TRANSCYSTIC CHOLANGIOGRAM: AN ERCP IS PLANNED.

ERCP Technique

The cystic duct catheter provides a portal through which a guidewire can be directed into the duodenum at the time of ERCP. The ability to place a guidewire greatly facilitates cannulation of the CBD during ERCP, especially in technically difficult cases.

Equipment

Pentax ERCP scope

Microvasive Ultratome XL

Zebra wire

Balloon Retrieval Catheters--8.5 mm. and 11 mm.(Microvasive Extractor XL)

Stone retrieval basket

Technique

STEP 1. A cholangiogram is first performed via transcystic catheter. This helps identify CBD and facilitates cannulation of papilla.

STEP 2. ERCP is then performed in the standard fashion.

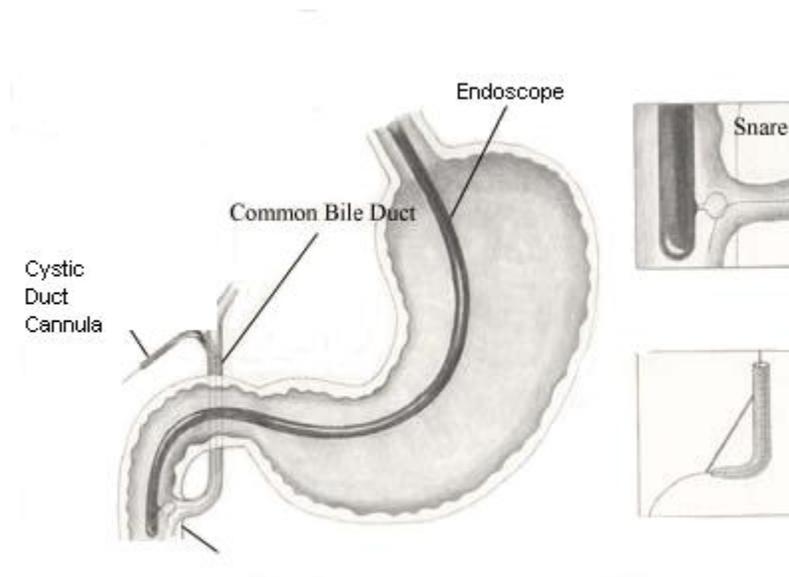
STEP 3. If cannulation takes longer than 15 minutes, then a 400 cm Zebra wire is advanced through the transcystic catheter and directed by fluoroscopy into the CBD and through the papilla.

STEP 4. The endoscopist passes a snare through the biopsy channel of the ERCP scope, snares the end of the Zebra wire, and pulls it out of the scope.

STEP 5. The papillotomy is flushed with saline and advanced over the wire, through the scope, and into position in the papilla and CBD.

STEP 6. Endoscopist performs papillotomy over the guidewire and removes guidewire/papillotomy assembly.

STEP 7. The duct is then swept with an 8.5 mm or 11 mm balloon or a stone retrieval basket to remove stone(s).



The transcystic cannula is removed by firmly pulling on it at the bedside or in the ERCP suite. The Blake drain is left in place and the patient is discharged. The Blake drain is then removed a few days later as an outpatient.

NOTE: There has been no reported leak following this protocol. However, the Blake drains are left in place should a bile leak occur.

SCENARIO 2: NO COMMON BILE DUCT STONE DEMONSTRATED.

The Cannula is removed by exerting firm traction. The Blake Drain is left in place and removed 48 hours later as an outpatient.

Advantages

This technique offers many advantages over existing strategies for dealing with CBD stones. First, ERCP's will be limited only to those patients who have a stone visualized on transcystic cholangiogram. For those surgeons or gastroenterologists who currently stratify patients' need for ERCP according to preoperative risk factors for CBD stones, the TCC approach will eliminate the need to perform ERCP on up to 80% of patients with positive risk factors but who have no stones (False Positives). The ERCP associated complications will thereby be eliminated. Second, the 15% risk of postoperative ERCP failure to cannulate or clear stones (even up to 10% in biliary referral centers) will be largely eliminated by the ability to place a transcystic, transpapillary guidewire. This safety valve will greatly facilitate endoscopic access to the bile duct, eliminate the need for a risky precut papillotomy to gain access to the CBD, and reduce the potential need for a second operation in patients in whom ERCP was a technical failure. Third, the TCC should eliminate the need for IOC and CBDE. Since the TCC/ERCP technique reduces the risks associated with ERCP and optimizes the chance of a successful outcome, the need for IOC and /or CBDE (laparoscopic or open) is greatly reduced (including those CBDE's done for false positive IOC's). Fourth, if this technique is applied to

all laparoscopic cholecystectomies, then all CBD stones will be detected including up to 8% of patients who have no preoperative risk factors for stones.

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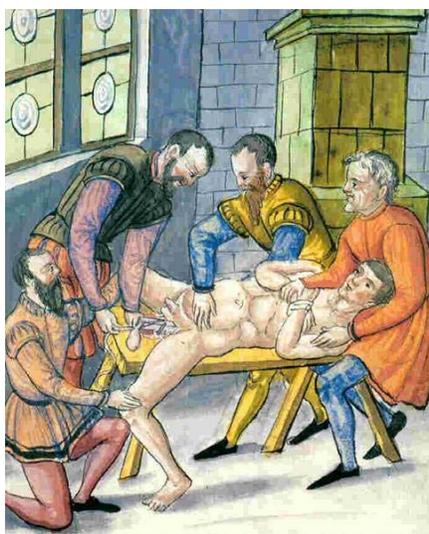
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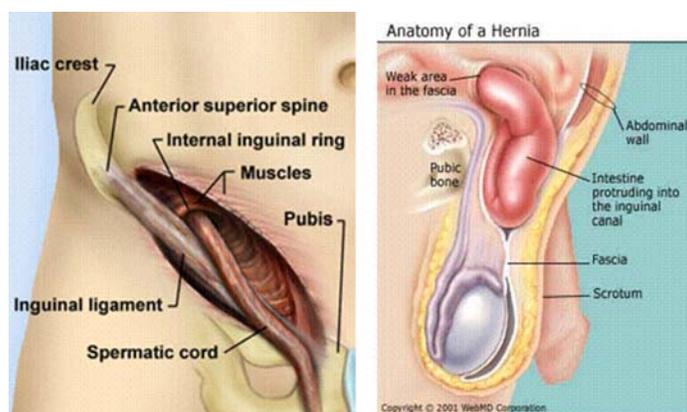
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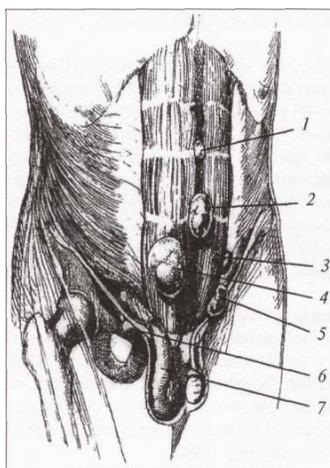
HERNIA



Hernia of the abdominal wall or external hernia (*herniae abdominalis externae*) is such surgical disease, which is characterized by outlet of the visceral organs from the place of their physiological placement through the natural channels or defects of the abdominal and pelvic wall. In such case all visceral organs covered by parietal peritoneum and skin cover are not damaged.



Internal hernia (*herniae abdominalis internae*) is such disease, visceral organs hit the peritoneum pouch. It formed in the place of natural peritoneum fold or recess and generally kept in the abdominal cavity.



Localizations of the abdominal wall hernias :1— Linea alba; 2—Umbilical; 3 —Spigelian li-nea; 4—Incisional hernia; 5—Direct inqualis hernia;6—Femoral hernia;7—Indirect inqualis hernia

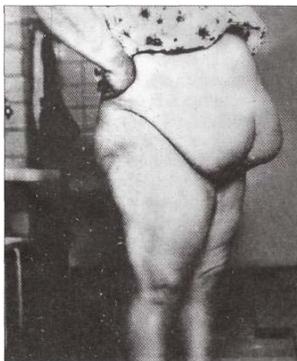
Etiology and pathogenesis

Hernias are divided into two main groups: congenital (*herniae congenitae*) and acquired (*herniae acquisitae*). The main reason of congenital hernias is malformation. Thus, inguinal hernia arose in case of noclosure of the process of peritoneum, which passes by inguinal channel during descending of the testis. On such hernias testis is located in the hernia pouch. Acquired inguinal hernia has hernia pouch and testis located outside it. Many factors are of great importance in the beginning and developing of the acquired hernia. One of them contributes, other - causes disease. The first are hereditary, anatomical inferiority of the abdominal wall, sex (inguinal area weakness in males and largeness internal femoral ring in females), age (atrophic processes in older age, anatomical inferiority of the abdominal wall in babies), weight loss, injury, postoperative scar, physical activity, pregnancy, during which abdominal wall stretched (for example, midline increased in 12 times).

Such reasons, as increased abdominal pressure and weakness of the abdominal wall, cause hernia. That arise after hard physical activity, continued cough, constipation, nerves palsy, which innervated abdominal wall, injury of muscles or aponeurosis of the abdomen.



Inguinal hernia



Postoperative hernia

Pathomorphology

Each abdominal hernia consists of hernia gate, hernia sac and hernia contents. Hernia sac forms by outpouching of parietal peritoneum and can contain any organ from abdominal cavity, but mostly – small intestine and omentum. Sometimes it contains other organs: large intestine, urinary bladder, ovary, and appendix.

The main parts of the hernia pouch are neck, body and fundus. Through the hernia's gate, peritoneum is outpouching. In the case of sliding hernia organ in the hernia pouch has mesoperitoneal disposition and not enclosed by peritoneum.

Classification

Hernia of the abdominal wall is divided:

- 1) Depends on anatomical localization: inguinal (indirect and direct), midline hernia, omphalocele, femoral hernia, lumbar hernia, sciatic hernia, (enterischiocele), lateral hernia, ischioanal [perineal] hernia (perineocele).
- 2) depends on etiology: congenital (*herniae congenitae*) and acquired (*herniae acquisitae*).
- 3) Depends on clinical presentations: complete and incomplete, reducible and nonreducible, traumatic and postoperative, complicated and noncomplicated.

Clinical management

The typical symptom of hernia is swelling, which arises on vertical position of the patient or during rise of intraperitoneal pressure. These can disappear in a state of dormancy, on vertical position of the patient or after applying small pressure. Such factors make it possible to confirm hernia.

In the case of hernia primary formation skin over the swelling almost not changed. Hernia is determined by finger examination of the inguinal channel. We can feel positive symptom of the "cough push", which is caused by cough or by the rise of intraperitoneal pressure. In the case of late stage of hernia developing evagination appear on changing body position from horizontal to vertical or after rising of the intraperitoneal pressure. If hernia sac contained small intestine than every next tension of the abdominal muscle inflated hernia sac by intestinal loop.



Symptom of the "cough push"

Diagnostics of the noncomplicated external abdominal hernias is easy. Anamnesis of patients and clinical data are enough. However, we should remember about nonreducible hernias. Such hernia's shape and dimension often does not change. Patients complicated for continuous pain in the hernia region, which irradiated to other abdominal organs. The main danger of the nonreducible hernias is jamming.

Clinical variants and complications

Inguinal hernias is developed in two ways: through the internal (middle) inguinal cavity and external (lateral). In the first case formed direct in other - indirect inguinal hernia.

Indirect hernias could be congenital and acquired. Direct hernias are only acquired and occur in older patients.

There are two main signs, which differentiate direct and indirect hernias. Direct hernia is always located medially from *a. epigastrica inf.* Indirect hernia is always located laterally from *a. epigastrica inf.* The other sign is: direct hernia located medially from deferent duct, indirect hernia located inside it

Femoral hernias are such pathological formation, which is encountered 10-20 times more often in males than females. This is explained by anatomical peculiarity of the females' pelvis, wider interval between femoral vein and lacunar [Gimbernat's] ligament and inguinal [Poupart's] ligament weakness.

There are distinguished femoral hernias, vasculo-lacunar, rural pectineal [Cloquet's] hernia, Hesselbach's hernia.

In addition, there are some kinds of femoral hernias, which can be identified only during the operation:

- 1) Medial vascular-lacunar femoral hernia, most common;
- 2) Hernia, which passed through the middle part of vascular lacuna or through the vascular sheath;
- 3) Lateral vascular-lacunar hernia, which pass outside of the femoral vessels.

Besides, there is middle or prevascular hernia.

Medial vascular -lacunar femoral hernia has three stages of developing:

- 1) Beginning femoral hernia - swelling does not pass outside internal femoral ring;
- 2) Incomplete (interstitial) hernia - swelling does not pass outside of superficial fascia;
- 3) Complete femoral hernia - swelling passed through all anatomical part of the femoral channel and outgoing to the subcutaneous cellular tissue on the anterior femoral surface below inguinal [Poupart's]

ligament.

In spite of small size of the hernia sac, femoral hernia could contain omentum, small intestine and urinary bladder. It is more difficult to diagnose femoral hernia in overweight patients because of inexpressive clinical signs.

We should differentiate femoral hernias with inguinal hernias, increased or varicose changed lymphatic nodes. In those cases, we should determine external inguinal ring and inguinal ligament.

Midline [epigastric] hernia usually has males in giving age.

There are distinguished supraumbilical, umbilical and paraumbilical hernias.

Very often, such kind of the hernia has no clinical signs and can be determined on the medical examinations. The usual clinical signs are: swelling on linea alba and intermittent pain.

Umbilical hernias occur in 2 % from all kinds of hernia. The most frequent hernias in females (the ratio is 5:1), which is explained by anatomical peculiarity of the females' umbilicus after pregnancy. Such hernia often has two- and three-chambers hernia sacs, which could contain omentum, small intestine, and sometime stomach. Clinical signs depend on those contents. However, it always characterized by pain and swelling. In some patients swelling is very large.

Diagnosis of the umbilical hernia in typical case is not very difficult. Sometimes it is arduous to differentiate incarcerated umbilical hernia and umbilical metastasis of tumor. We should remember about umbilical evagination (without organs) in the patients with liver cirrhosis because of presence ascitic fluid in the abdominal cavity.

Lumbar hernias are abdominal wall or retroperitoneal outpouchings. It does not occur very often. The area of the hernia orifice includes the superior costolumbar triangle and the inferior iliolumbar triangle. Besides that, it could be in aponeurosis slit.

Lumbar hernias could be congenital and acquired. Congenital lumbar hernias are frequently the result of aponeurosis slit or enlargement of the Pt₁ triangle or Hrunfeld interval. Acquired lumbar hernias are usually result of injury those anatomical structure or after pyoinflammatory diseases.

The most frequent clinical sign is pain. The other signs depend on hernia content. The hernia contents may include any intra- and retroperitoneal structures, e.g., the kidney, small bowel, and omentum.

Diagnosis is made by clinical examination: in the horizontal patient position on healthy side, swelling disappeared, and on the vertical patient position appeared again.

Obturator hernia is the result of wide obturator channel. In those cases hernia sac formes inside pelvic cavity, and than passes through the obturator channel, and arises on internal femoral surface.

Diagnosis of the obturator hernias is not easy, especially in the patients without swelling on the hip. In such cases, patients have complaints for pain along obturator nerve with irradiation to knee joint or hip joint. Pain increases during leg rotation or abduction. Sometime pain irradiates to the foot.

Sciatic hernias is divided into two main types: hernia of the major sciatic foramen, which passes above and under piriform muscle and hernias of the small sciatic foramen, which passes under sciatic muscle. Patients complained for pain in the sciatic region, which increased during walking. Sometime pain irradiated along sciatic nerve.

Ischiorectal [perineal] hernia is formed in the urogenital diaphragm or in the perineum muscle. Anterior and posterior hernias are distinguished depending on whether the hernia is anterior or posterior to the transverse perineal muscle and sacrospinal ligament. Hernia ring formed by rectouterine [Douglas'] pouch in the female and by retrovesical pouch [Proust's space] in the male. Anterior hernias usually contained internal genital organs or urinary bladder. Posterior hernias usually contained omentum or small intestine loops.

Diagnosis of the anterior ischiorectal hernias, which passed to perineum, usually is not difficult. Diagnostic pitfall should be on the patients with posterior hernias, which is located under large sciatic muscle and looks like sciatic hernia. In such cases, we performed vaginal and rectal examination or X-Ray examination of the urinary bladder and intestine as required.

There are three types of the *lateral abdominal hernias*:

- 1) Acquired hernia of the rectus sheath;
- 2) Acquired hernias of the Spigelian line;
- 3) Congenital hernias because of congenital hypoplasia of the abdominal wall.

Clinical signs of lateral abdominal hernias are the same as for other types of hernias, so diagnosis is not very difficult.

The most common complications for all those hernias are incarceration.

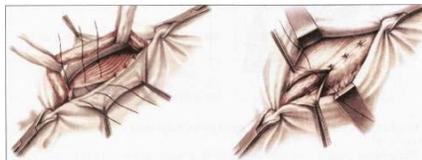
Diagnosis program

1. Anamnesis and physical examination.
2. Digital investigation of the hernia channel.
3. Sonography of the hernia pouch.
4. Common blood analysis.
5. Common urine analysis.

Tactics and choice of treatment method

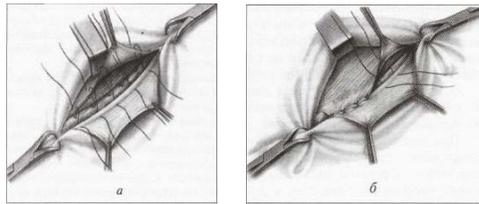
Inguinal hernia usually should be surgically repaired. On oblique inguinal hernias, we should strengthen anterior wall of the inguinal channel. On direct inguinal hernias, we should strengthen posterior wall of the inguinal channel. On recurrence hernias - we should strengthen anterior and posterior wall of the inguinal channel.

Bassini repair. After extraction of the hernia sac, we are taking spermatic duct on holders. Between the borders of transverse muscle, internal oblique muscle, transverse fascia and inguinal ligament interrupted sutures placed. Except that, couples sutures placed between border of abdominal rectus muscle sheath and pubic bone periosteum.



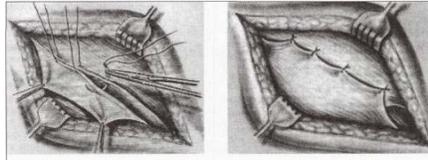
In such way, inguinal space closed and posterior wall strengthened. Spermatic duct placed on the new-formed posterior wall of the inguinal channel. Over the spermatic duct aponeurosis restored by interrupted sutures.

Girard in such kind of the operations propose to attach the edges of the internal oblique muscle and transversal muscle to the inguinal ligament over the spermatic duct. The aponeurosis of the external oblique muscle sutured by second layer of the suture. Excess of the aponeurosis is fixed to the muscle in the form of duplication.



Spasokukotsky proposed to catch the edges of the internal oblique muscle and transversus muscle with aponeurosis of the external oblique muscles by single-layer interrupted suture.

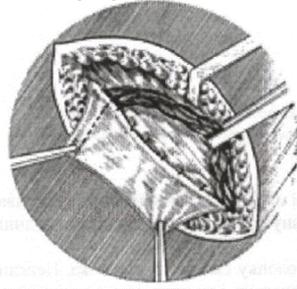
Martynov proposed the fixation to the Poupart's ligament only internal edge of the external oblique muscle aponeurosis without muscles. External edge of the aponeurosis sutured over internal in the form of duplication.



Kimbarovskyy, based on the principles of joining similar tissues, proposed special suture: Sutures placed on 1 cm from the edge of the external oblique abdominal muscle aponeurosis, grasped the part of the internal oblique and transversus muscle. After that, aponeurosis is sutured one more time from behind to the front and attached to the Poupart's ligament.

Kukudganov proposed to restore back wall of inguinal interval. Sutures are placed between the Couper's ligamentum, vagina of direct abdominal muscle and aponeurosis of the transversus muscle.

Postempysky proposed the deaf closing of inguinal interval with the lateral moving of spermatic duct.



The plastic narrowing of internal inguinal ring of to 0,8 cm is the important moment of this modification. On occasion, when internal and external inguinal rings are in one plane, a spermatic duct is displaced in lateral direction by transversal incision of the oblique and transversus muscles. Then edge of the vagina of direct muscle and aponeurosis of the internal and transversus muscles is fixed to the Couper's ligament.

LAPAROSCOPIC HERNIOPLASTY



a



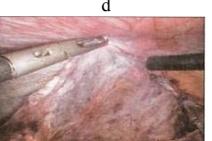
b



c



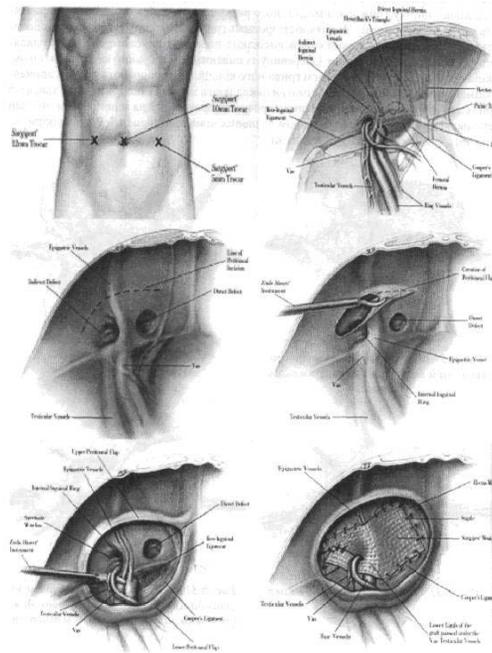
d



e

Laparoscopic hernioplasty:

a — view from inside; b — cutting of the peritoneum; c — removing the hernia; d — fixation by mesh material; e — suturing

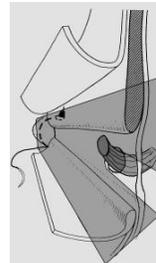


Pic. Laparoscopic hernioplasty

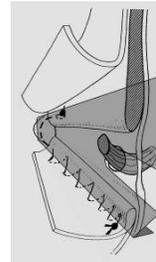
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HERNIOPLASY BY LICHTENSTEIN

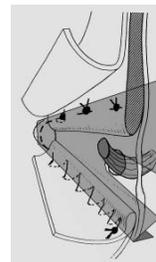
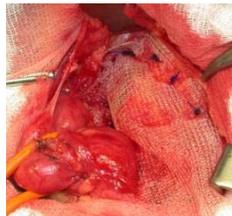
Fixation the mesh material



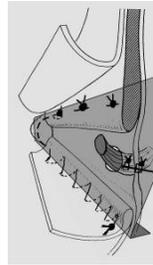
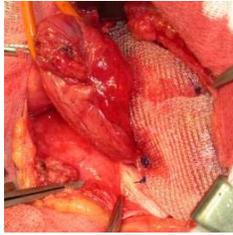
View of the knotless suture



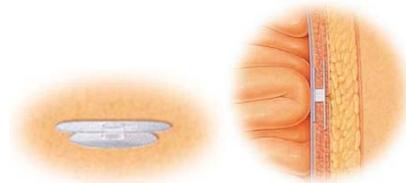
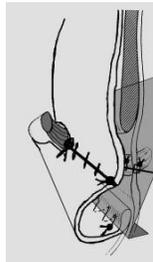
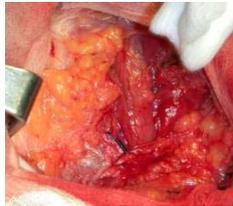
Fixation to the muscle



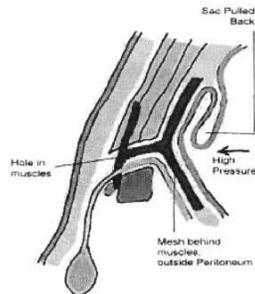
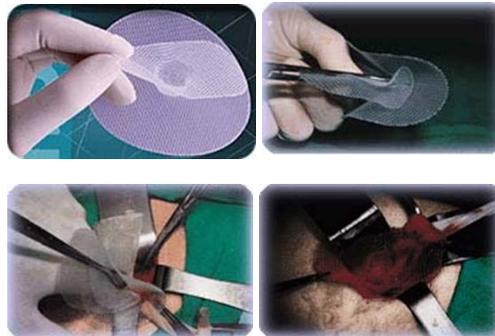
Formation of the internal inguinal ring



Knotless suture of the aponeurosis of the oblique external muscle



PROLENE
POLYPROPYLENE HERNIA SYSTEM

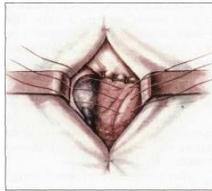


Hemioplasty by PROLENE HERNIA SYSTEM

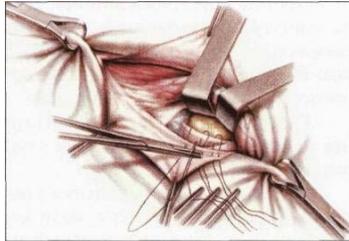
FEMORAL HERNIA

The are some methods of surgical treatment of the **femoral hernia**, when the plastic are performed intraperitoneal from the side of thigh through the inguinal channel.

The Bassini method is attributed to "femoral". It is performed from a cut, that passes under inguinal fold. After removal of hernia sack a hernia gate is liquidated by suturing of inguinal to the pectineal ligament.

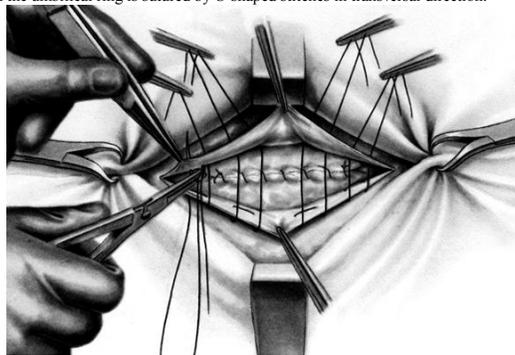


The Rudgi-Parlavecho Method. A cut passes parallel to the inguinal fold and higher it (the same as at inguinal hernia). A hernia sack is removed. After that the edges of the transversal and internal oblique muscles and inguinal ligament sutured to the periosteum of pubic bone.



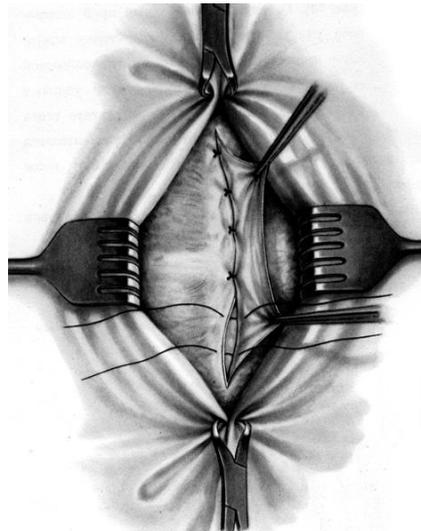
UMBILICAL HERNIA

For operative treatment of umbilical hernia a few methods are used. The Lexer operation is most widespread. It performed by imposition of sutures on an umbilical ring. After the *Meyo* method defect of anterior abdominal wall in the umbilical ring is sutured by U-shaped stitches in transversal direction.



Meyo method

Sapezhko proposed to form duplication of the abdominal white line by stitches in longitudinal direction.

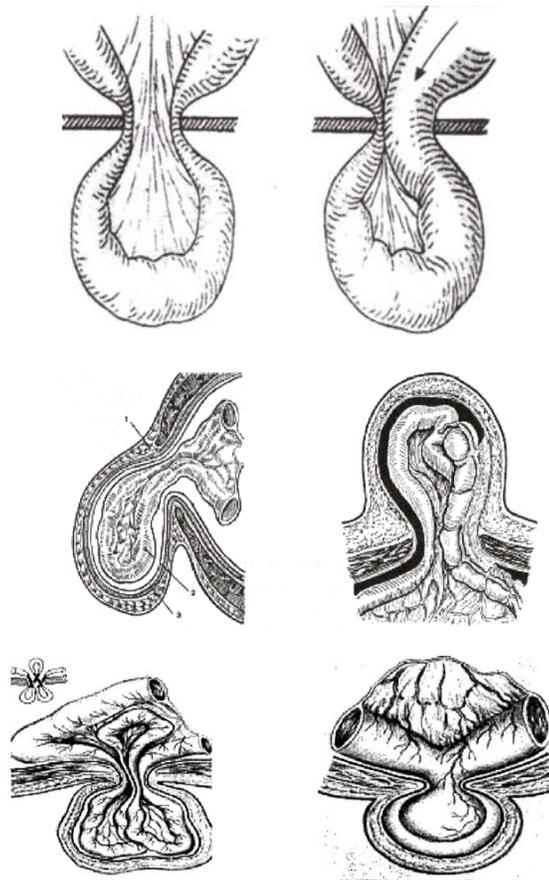


Sapezhko method

At surgical treatment of hernia of abdominal white line, abdominal lateral hernia, lumbar and obturator hernia, sciatic and ischio-rectal hernias after deleting of hernia sack it is needed to try to close a hernia orifice by suturing of fissures in aponeurosis and muscles.

INCARCERATED HERNIA

Incarcerated hernia is sudden pressing of hernia contents in a hernia orifice. Incarceration is the most frequent and most dangerous complication of hernia diseases.



Pic. Types of incarcerations

Etiology and pathogenesis

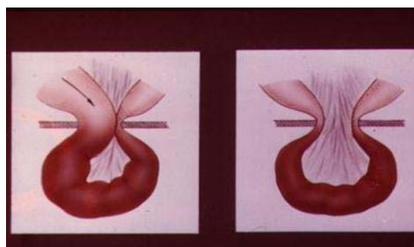
Depending on mechanism, the elastic and fecal incarceration is distinguished. At the *elastic incarceration*, after increasing intraabdominal pressure, one or a few organs relocated from an abdominal cavity to the hernia sack, where it is compressed with following ischemia and necrosis in the area of hernia gate. At the fecal incarceration in the intestinal loop which is in a hernia sack, plenty of excrement passed quickly. Proximal part of loop is overfilled, and distal is compressed in a hernia gate. So, arose its strangulation, as well as at the elastic incarceration.

Most often the loop of bowel is incarcerated. Thus three parts are distinguished in it: proximal, distal loop, central part. The heaviest pathological changes during incarceration takes place in a strangulated furrow in the central part of the incarcerated bowel.

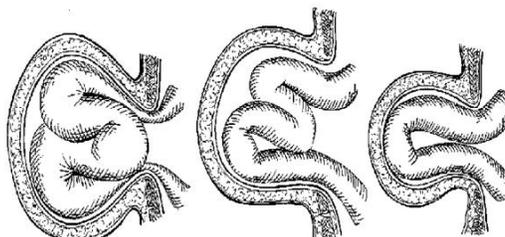
Pathomorphology

At incarcerated hernia an important role has all internal rings: inguinal, umbilical, "weak places" in a diaphragm, orifice of the omental bursa, numeral and "variant" folds of peritoneum.

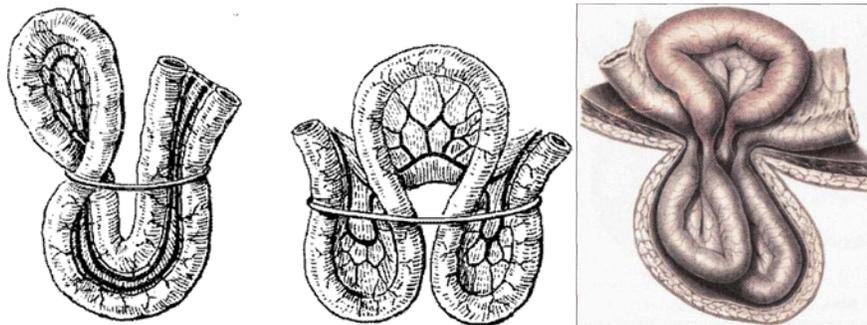
In the place of compressing of the bowels and mesentery, as a rule, it is possible to find a strangulation furrow. If circulation of blood changes, the wall of bowel cyanotic, with hemorrhages and necrosis of a different size. The loop of bowel which is located proximally the places of strangulation are extended, and distal loop mainly without changes.



Pic. Mechanizm of the incarceration.



Pic. Scheme of the incarceration.



Pic. Retrograde incarceration.

Classification of the incarcerated hernia

The incarcerated hernia is divided into the complete and incomplete. The other types of incarceration is partial (the Richter's hernia) and retrograde. The incarcerated hernia can be without the destructive changes of hernia contents and with the phlegmon of hernia sack.

Clinical management

The clinic of the incarcerated hernia depends on pulling in organ, character and duration of jamming. The clinical signs of the incarcerated hernia can be divided into three groups: 1) local changes; 2) common signs; 3) complication. From the most characteristic signs of local changes the most common is sharp pain, irreducible hernia, tension of hernia sack that and negative symptom of the "cough push".

Pain sometimes is so intensive that causes pain shock. In the case of intestinal obstruction a pain is attack-like. In case of occurring of peritonitis pain changes the character and becomes permanent.

It is necessary to mean that tensions of hernia sack and incarceration of the hernia, as signs of jamming, lose it value, if hernia was irreducible.

From other side, the isolation of hernia sack from an abdominal region during jamming is the reason of the *negative symptom of the "cough push"*.

The common signs at the incarcerated hernia has phase character. *Nausea and vomits* during first hours of disease has reflex reason, and on 2nd and 3rd days has toxic reason, that is consequence of antiperistaltic and reflux of intestinal contents to the stomach.

The *temperature of body* at first time is normal, and than rises, but usually low grade fever.

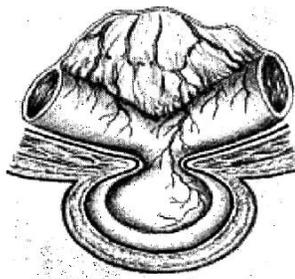
The clinic of acute intestinal obstruction and peritonitis develops at the protracted jamming of intestine. The phlegmon of hernia sack can develop in the area of the hernia swelling.

Clinical variants and complications

There are different forms of incarceration of internal organs, and accordingly — different clinical variants.

Retrograde incarceration. In such cases a hernia sack contains no less than two loops of intestine. But these loops are damaged less, than loop which is in an abdominal cavity. At this variant of jamming peritonitis arose quicker. So, surgeon during operation must always remember about the necessity of careful revision of the incarcerated loops of bowel.

Parietal incarceration (the Richter's hernia). Unlike retrograde, which has wide hernia gate, a similar pathology arises in case of narrow hernia gate. In a hernia sack in such patients located part of bowel wall, opposite it mesentery edge.



Thus, as a rule, patency of bowel is not broken. Such variant of jamming is dangerous, because there are no evident clinical signs or some of them are quite absent and intestinal patency almost is always present. Necrosis of bowel wall comes quickly and in 2-3 days the perforation with subsequent development of peritonitis begins after jamming.

The Litre's hernia. Jamming of Meckel's diverticulum can come at oblique inguinal hernia. Clinical signs of this pathology reminds the parietal incarceration. Sometimes is possible to palpate dense, short, thick tension bar in a hernia sack.

Incarceration at sliding hernia. It is observed at patients with inguinal hernia. At sliding hernia of colon, as a rule, there is the fecal incarceration. A bowel is the external wall of hernia sack in such cases. About it is necessary to remember during opening of hernia sack. Jammings of urinary bladder meet enough rarely, mainly at older-men at oblique sliding hernia of inguinal channel. It is necessary to ask before the operation, whether a patient had disorders of urination before jamming. Frequent urges, or, opposite, the reflex delay of urination is arose at the beginning of jamming already, and in urine expose macro- or microhematuria. If during operation at opening of hernia sack it medial wall has dense, doughy consistency, it is an urinary bladder.



At the incarcerated hernia the contents of hernia sack can be also omentum, appendages of colon, internal female genital organs. Sometimes combination of the incarcerated inguinal hernia with different pathological changes of testicle and deferent duct can take place.

Rough manual reduction of the incarcerated hernia can bring to pseudoreduction. Then the local signs of the incarcerated hernia disappear, and jamming of organs and its consequences is kept. There are five variants of the pseudoreduction: 1) at multicompartiment hernia sacks there is the possible moving of strangulated organs from one chamber in other, that located more deep or in a preperitoneal adipose tissue; 2) separation and reduction of hernia sack together with it content in an abdominal cavity or in a preperitoneal adipose tissue; 3) abruption of the neck from other part of hernia sack and reduction it together with content in an abdominal cavity or in a preperitoneal adipose tissue; 4) abruption of the neck from a hernia sack and from a parietal peritoneum with reduction of the incarcerated organs in an abdominal cavity; 5) break of the incarcerated bowel at the rough reduction of hernia.

Untimely operative at the incarcerated hernia, usually, is complicated by the gangrene of bowel, peritonitis or phlegmon of hernia sack. Such complications considerably worsen clinical status of patient and require other surgical tactic.

Diagnosis program

1. Anamnesis examination.

2. Physical examination.
3. Blood analysis and urine analysis.
4. Digital investigation of the rectum.
5. Survey X-Ray of abdominal cavity organs.

Differential diagnostics

As experience shows, the incarcerated hernia we should differentiate with irreducible, which as a rule, is not tense, positive symptom of the "cough push", painful on palpation. A patient complained for long duration of the disease. The incarcerated hernia needs to be differentiated with coprostasis. In such patients disorder of bowel loop patency, that is in a hernia sack, creates accumulation of excrement. Coprostasis mostly found at fecal hernia in older people, that suffer from intractable constipation. Clinically it develops gradually and slowly. The hernia swelling almost not painfully, some tense, a positive symptom of the "cough push". Beginning of coprostasis is unconnected with physical tension. Application of cleansing siphon enema washed of excrement and liquidated coprostasis.

Unreal jamming of hernia. In clinical practice there are often such situation, when during the acute surgical diseases of organs of abdominal cavity free external abdominal hernia becomes irreducible, painfully and tense, and looks like incarcerated. This is the unreal jamming of hernia, which can be observed at the acute surgical diseases of organs of abdominal cavity, ascites. During examination of such patients it is necessary to remember, that at the unreal jamming abdominal pain, vomiting, worsening of the general condition and signs of the intestine obstruction come earlier, than changes in a hernia sack.

In addition, during the operation in patients with incarcerated hernia, it is needed to make sure, whether there is a strangulation furrow, or organ, that is in a hernia sack, fixed in a hernia gate. When these signs are absent, it is possible to consider that jamming is unreal.

The incarcerated femoral hernia must be differentiated with inguinal lymphadenitis, by varicose expansion of large hypodermic vein, varicose knot and their thrombophlebitis, tumor and abscess.

From such pathology without surgical procedure it is possible to differentiate only varicose expansion of veins (varicose knot), for which the positive Valsalva test — at horizontal position of patient with the leg heaved up a knot is empty.

The incarcerated inguinal hernia needs to be differentiated also with hydrocele and orchiepididymitis, cyst of deferent duct, cyst of round ligamentum of uterus, Bartholinitis. Patients, who have with such diseases, a process usually does not spread higher external ring of inguinal channel. Also, absence of testicle in scrotum can be cryptorchidism sign.

The common clinical signs of the internal incarcerated hernia is abdominal pain and symptoms of the intestinal obstruction. A final diagnosis is set during the operation.

Differential diagnostics and clinical variants

Acute appendicitis is an inflammation of vermiform appendix caused by festering microflora.

Most frequent causes of acute appendicitis are festering microbes: intestinal stick, streptococcus, staphylococcus. Moreover, microflora can be in cavity of appendix or get there by hematogenic way, and for women — by lymphogenic one.

Factors which promote the origin of appendicitis, are the following: a) change of reactivity of organism; b) constipation and atony of intestine; c) twisting or bends of appendix; d) excrement stone in its cavity; e) thrombosis of vessels of appendix and gangrene of wall as a substance of inflammatory process (special cases).

Simple (superficial) and destructive (phlegmonous, gangrenous primary and gangrenous secondary) appendicitises which are morphological expressions of phases of acute inflammation that is completed by necrosis can be distinguished.

In simple appendicitis the changes are observed, mainly, in the distant part of appendix. There are stasis in capillaries and venule, edema and hemorrhages. Focus of festering inflammation of mucus membrane with the defect of the epithelium covering is formed in 1–2 hours (primary affect of Ashoff). This characterizes acute superficial appendicitis. The phlegmon of appendix develops to the end of the day. The organ increases, it serous tunic becomes dimmed, sanguineous, stratifications of fibrin appear on its surface, and there is pus in cavity.

In gangrenous appendicitis the appendix is thickened, its serous tunic is covered by dimmed fibrinogenous tape, differentiating of the layer structure through destruction is not succeeded.

Four phases are distinguished in clinical passing of acute appendicitis: 1) epigastric; 2) local symptoms; 3) calming down; 4) complications.

The disease begins with a sudden pain in the abdomen. It is localized in a right iliac area, has moderate intensity, permanent character and not irradiate. With 70 % of patients the pain arises in an epigastric area — it is an epigastric phase of acute appendicitis. In 2–4 hours it moves to the place of appendix existence (the Kocher's symptom). At coughing patients mark strengthening of pain in a right iliac area — it is a positive cough symptom.

Together with it, nausea and vomiting that have reflex character can disturb a patient. Often there is a delay of gases. The temperature of body of most patients rises, but high temperature can occur rarely and, mainly, it is a low grade fever. The general condition of patients gets worse only in case of growth of destructive changes in appendix.

During the examination it is possible to mark, that the right half of stomach falls behind in the act of breathing, and a patient wants to lie down on a right side with bound leg.

Painfulness is the basic and decisive signs of acute appendicitis during the examination by palpation in a right iliac area, tension of muscle of abdominal wall, positive symptoms of peritoneum irritation. About 100 pain symptoms characteristic of acute appendicitis are known, however only some of them have the real practical value.

The Blumberg's symptom. After gradual pressing by fingers on a front abdominal wall from the place of pain quickly, but not acutely, the hand is taken away. Strengthening of pain is considered as a positive symptom in that place. Obligatory here is tension of muscles of front abdominal wall.

The Voskresenskyy Symptom. By a left hand the shirt of patient is drawn downward and fixed on pubis. By the taps of 2–4 fingers of right hand epigastric area is pressed and during exhalation of patient quickly and evenly the hand slides in the direction of right iliac area, without taking the hand away. Thus there is an acute strengthening of pain.

The Bartomier's symptom is the increase of pain intensity during the palpation in right iliac area of patient in position on the left side. At such pose an omentum and loops of thin intestine is displaced to the left, and an appendix becomes accessible for palpation.

The Sitkovsky's symptom. A patient, that lies on left, feels the pain which arises or increases in a right iliac area. The mechanism of intensification of pain is explained by displacement of blind gut to the left, by drawing of mesentery of the inflamed appendix.

The Rovsing's symptom. By a left hand a sigmoid bowel is pressed to the back wall of stomach. By a right hand by ballotting palpation a descending bowel is pressed. Appearance of pain in a right iliac area is considered as a sign characteristic of appendicitis.

The Obrazcov's symptom. With the position of patient on the back by index and middle fingers the right iliac area of most painful place is pressed and the patient is asked to heave up the straightened right leg. At appendicitis pain increases acutely.

The Rozdolsky's symptom. At percussion there is painfulness in a right iliac area.

The general analysis of blood does not carry specific information, which would specify the presence of acute appendicitis. However, much leukocytosis and change of formula to the left in most cases can point to the present inflammatory process.

Acute appendicitis in children. With children of infancy acute appendicitis can be seen infrequently, but, quite often carries atypical character. All this is conditioned, mainly, by the features of anatomy of appendix, insufficient of plastic properties of the peritoneum, short omentum and high reactivity of child's organism. The inflammatory process in the appendix of children quickly makes progress and during the first half of days from the beginning of disease there can appear its destruction, even perforation. The child, more frequent than an adult, suffers vomiting. Its general condition gets worse quickly, and already the positive symptoms of irritation of peritoneum can show up during the first hours of a disease. The temperature reaction is also expressed considerably acuter. In the blood test there is high leukocytosis. It is necessary to remember, that during the examination of calmless children it is expedient to use a chloral hydrate enema.

Acute appendicitis of the people of declining and old ages can be met not so often, as of the persons of middle ages and youth. This contingent of patients is hospitalized to hospital rather late: in 2–3 days from the beginning of a disease. Because of the promoted threshold of pain sensitiveness, the intensity of pain in such patients is small, therefore they almost do not fix attention on the epigastric phase of appendicitis. More frequent are nausea and vomiting, and the temperature reaction is expressed poorly. Tension of muscles of abdominal wall is absent or insignificant through old-age relaxation of muscles. But the symptoms of irritation of peritoneum keep the diagnostic value with this group of patients. Thus, the sclerosis of vessels of appendix results in its rapid numbness, initially-gangrenous appendicitis develops. Because of such reasons the destructive forms of appendicitis prevail, often there is appendiceal infiltrate.

With pregnant women both the bend of appendix and violation of its blood flow are causes of the origin of appendicitis. Increased in sizes uterus causes such changes. It, especially in the second half of pregnancy, displaces a blind gut together with an appendix upwards, and an overdistended abdominal wall does not create adequate tension. It is needed also to remember, that pregnant women periodically can have a moderate pain in the abdomen and changes in the blood test. Together with that, psoas-symptom and the Bartomier's symptom have a diagnostic value at pregnant women.

Clinical passing of acute appendicitis at the atypical placing (not in a right iliac area) will differ from a classic vermiform appendix (Pic. 3.3.1).

Appendicitis at retrocecal and retroperitoneal location of appendiceal appendix can be with 8–20 % patients. Thus an appendix can be placed both in a free abdominal cavity and retroperitoneal. An atypical clinic arises, as a rule, at the retroperitoneal location. The patients complain at pain in lumbus or above the wing of right ilium. There they mark painfulness during palpation. Sometimes the pain irradiates to the pelvis and in the right thigh. The positive symptom of Rozanov — painfulness during palpation in the right Pti triangle is characteristic. In transition of inflammatory process on an ureter and kidney in the urines analysis red corpuscles can be found.

Appendicitis at the pelvic location of appendix can be met in 11–30 % cases. In such patients the pain is localized above the right Poupard's ligament and above pubis. At the very low placing of appendix at the beginning of disease the reaction of muscles of front abdominal wall on an inflammatory process can be absent. With transition of inflammation on an urinary bladder or rectum either the dysuric signs or diarrhea develops, mucus appears in an excrement. Distribution of process on internal genital organs provokes signs characteristic of their inflammation.

Appendicitis at the medial placing of appendix. The appendix in patients with such pathology is located between the loops of intestine, that is the large field of suction and irritation of peritoneum. At these anatomic features mesentery is pulled in the inflammatory process, acute dynamic of the intestinal obstruction develops in such patients. The pain in the abdomen is intensive, widespread, the expressed tension of muscles of abdominal wall develops, that together with symptoms of the irritation of peritoneum specify the substantial threat of peritonitis development.

For the subhepatic location of appendix the pain is characteristic in right hypocondrium. During palpation painfulness and tension of muscles can be marked.

Left-side appendicitis appears infrequently and, as a rule, in case of the reverse placing of all organs, however it can occur at a mobile blind gut. In this situation all signs which characterize acute appendicitis will be exposed not on the right, as usually, but on the left.

Among complications of acute appendicitis most value have appendiceal infiltrates and abscesses.

Appendiceal infiltrate is the conglomerate of organs and tissue not densely accrete round the inflamed vermiform appendix. It develops, certainly, on 3–5th day from the beginning of disease. Acute pain in the stomach calms down thus, the general condition of a patient gets better. Dense, not mobile, painful, with unclear contours, formation is palpated in the right iliac area. There are different sizes of infiltrate, sometimes it occupies all right iliac area. The stomach round infiltrate during palpation is soft and unpainful.

At reverse development of infiltrate (when resorption comes) the general condition of a patient gets better, sleep and appetite recommence, activity grows, the temperature of body and indexes of blood is normalized. Pain in the right iliac area calms down, infiltrate diminishes in size. In this phase of infiltrate physiotherapeutic procedure is appointed, warmth on the iliac area.

In two months after resorption of infiltrate appendectomy is conducted.

At abscessing of infiltrate the condition of a patient gets worse, the symptoms of acute appendicitis become more expressed, the temperature of body, which in most cases gains hectic character, rises, the fever appears. Next to that, pain in the right iliac area increases. Painful formation is felt there. In the blood test high leukocytosis is present with the acutely expressed change of leukocyte formula to the left.

Local abscesses of abdominal cavity, mainly, develops as a result of the atypical placing of appendix or suppuration. More frequent from other there are pelvic abscesses. Thus a patient is disturbed by pain

beneath the abcupula, there are dysuric disorders, diarrhea and tenesmus. The temperature of body rises to 38,0–39,0oC, and rectal — to considerably higher numbers. In the blood test leukocytosis, change of formula of blood is fixed to the left.

During the rectal examination the weakened sphincter of anus is found. The front wall of rectum at first is only painful, and then its overhanging is observed as dense painful infiltrate.

A subdiaphragmatic abscess develops at the high placing of appendix. The pain in the lower parts of thorax and in a upper quarter of abcupula ofn to the right, that increases at deep inhalationis except for the signs of intoxication, is characteristic of it. A patient, generally, occupies semisitting position. Swelling in an epigastric area is observed in heavy cases, smoothing and painful intercostal intervals. The abcupula ofn during palpation is soft, although tension in the area of right hypochondrium is possible. Painfulness at pressure on bottom (9–11) ribs is the early and permanent symptom of subdiaphragmatic abscess (the Krukov's symptom).

Roentgenologically the right half of diaphragm can fall behind from left one while breathing, and there is a present reactive exudate in the right pleura cavity. A gas bubble is considered the roentgenologic sign of subdiaphragmatic abscess with the horizontal level of liquid, which is placed under the diaphragm.

Interloop abscesses are not frequent complications of acute appendicitis. As well as all abscesses of abdominal cavity, they pass the period of infiltrate and abscess formation with the recreation of the proper clinic.

The poured festering peritonitis develops as a result of the timely unoperated appendicitis. Diagnostics of this pathology does not cause difficulties.

Pylephlebitis is a complication of both appendicitis and after-operative period of appendectomy.

The reason of this pathology is acute retrocecal appendicitis. At it development the thrombophlebitis process from the veins of appendix, passes to the veins of bowels mesentery, and then on to the portal vein. Patients complain at the expressed general weakness, pain in right hypochondrium, high hectic temperature of body, fever and strong sweating. Patients are adynamic, with expressed subicteritiousness of the scleras. During palpation painfulness is observed in the right half of abcupula ofn and the symptoms of irritation of peritoneum are not acutely expressed.

In case with rapid passing of disease the icterus appears, the liver is increased, kidney-hepatic insufficiency makes progress, and patients die in 7-10 days from the beginning of disease. At gradual subacute development of pathology the liver and spleen is increased in size, and after the septic state of organism ascites arises.

Acute appendicitis is differentiated with the diseases which are accompanied by pain in the abcupula ofn.

Food toxicoinfection. Complaints for pain in the epigastric area of the intermittent character, nausea, vomitings and liquid emptying are the first signs of disease. The state of patients progressively gets worse from the beginning. Next to that, it is succeeded to expect that a patient used meal of poor quality. However, here patients do not have phase passing, which is characteristic of acute appendicitis, and clear localization of pain. Defining the symptoms of irritation of peritoneum is not succeeded, the peristalsis of intestine is, as a rule, increased.

Acute pancreatitis. In anamnesis in patients with this pathology there is a gallstone disease, violation of diet and use of alcohol. Their condition from the beginning of a disease is heavy. Pain is considerably more intensive, than during appendicitis, and is concentrated in the upper half of abcupula ofn. Vomiting is frequent and does not bring to the recovery of patients.

Perforative peptic and duodenum ulcer. Diagnostic difficulties during this pathology arise up only on occasion. They can be in patients with the covered perforation, when portion of gastric juice flows out in an abdominal cavity and stays too long in the right iliac area, or in case of atypical perforations. Taking it into account, it is needed to remember, that the pain in the perforative ulcer is considerably more intensive in epigastric, instead of in the right iliac area. On the survey roentgenogram of organs of abdominal cavity under the right cupula of diaphragms free gases can be found.

The apoplexy of ovarya more frequent is with young women and, as a rule, on 10-14 day after menstruation. Pain appears suddenly and irradiate in the thigh and perineum. At the beginning of disease there can be a collapse. However, the general condition of patients suffers insignificantly. When not enough blood was passed in the abdominal cavity, all signs of pathology of abdominal cavity organs calm down after some time. Signs, which are characteristic of acute anaemia, appear at considerable hemorrhage. Abdomen more frequent is soft and painful down, (positive Kulenkampff's symptom: acute pain during palpation of stomach and absent tension of muscles of the front abdominal wall).

During paracentesis of back fornix the blood which does not convolve is got.

Extra-uterine pregnancy. A necessity to differentiate acute appendicitis with the interrupted extra-uterine pregnancy arises, when during the examination the patient complains at the pain only down in the stomach, more to the right. Taking it into account, it is needed to remember, that at extra-uterine pregnancy a few days before there can be intermittent pain in the lower part of the abdomen, sometimes excretions of "coffee" colour appear from vagina. In anamnesis often there are the present gynaecological diseases, abortions and pathological passing of pregnancy. For the clinical picture of such patient inherent sudden appearance of intensive pain in lower part of the abdomen. Often there is a brief loss of consciousness. During palpation considerable painfulness is localized lower, than at appendicitis, the abdomen is soft, the positive Kulenkampff's symptom is determined. Violations of menstrual cycle testify for pregnancy, characteristic changes are in milk glands, vagina and uterus. During the vaginal examination it is sometimes possible to palpate increased tube of uterus. The temperature of body more frequently is normal. If hemorrhage is small, the changes in the blood test are not present. The convincing proof of the broken extra-uterine pregnancy is the dark colour of blood, taken at puncture of back fornix of vagina.

Acute cholecystitis. The high placing of vermiform appendix in the right half of abdomen during its inflammation can cause the clinic somewhat similar to acute cholecystitis. But unlike appendicitis, in patients with cholecystitis the pain is more intensive, has cramp-like character, is localized in right hypochondrium and irradiate in the right shoulder and shoulder-blade. Also the epigastric phase is absent. The attack of pain can arise after the reception of spicy food and, is accompanied by nausea and frequent vomiting by bile. In anamnesis patients often have information about a gallstone disease. During examination intensive painfulness is observed in right hypochondrium, increased gall-bladder and positive symptoms Murphy's and Ortner's.

Right-side kidney colic. For this disease tormina at the level of kidney and in lumbus is inherent, hematuria and dysuric signs which can take place at the irritation of ureter by the inflamed appendix. Intensity of pain in kidney colic is one of the basic differences from acute appendicitis. Pain at first appears in lumbus and irradiate downward after passing of ureter in genital organs and front surface of the thigh. In diagnostics urogram survey is important, and if necessary — chromocystoscopy. Absence of function of right kidney to some extent allows to eliminate the diagnosis of acute appendicitis.

As experience of surgeons of the whole world testifies, in acute appendicitis timely operation is the unique effective method of treatment.

Access for appendectomy must provide implementation of operation. McBurney's incision is typical.

When during operation the appendix without the special difficulties can be shown out in a wound, antegrade appendectomy is executed. On clamps its mesentery is cut off and ligated. Near the basis the appendix is ligated and cut. Stump is processed by solution of antiseptic and peritonized by a purse-string suture (Pic. 3.3.2).

If only the basis of appendix is taken in a wound, and an apex is fixed in an abdominal cavity, more rationally retrograde appendectomy is conducted (Pic. 3.3.3). Thus the appendix near basis is cut between two ligatures. Stump is processed by antiseptic and peritonized. According to it the appendix is removed in the direction from basis to the apex. According to indication operation is concluded by draining of abdominal cavity (destructive appendicitis, exudate in an abdominal cavity, capillary hemorrhage from the bed). In recent years the laparoscopy methods of appendectomy are successfully performed.

In patients with appendiceal infiltrate it is necessary to perform conservative-temporizing tactic. Taking it into account, bed rest is appointed, protective diet, cold on the area of infiltrate, antibiotic therapy. According to resorption of infiltrate, in two months, planned appendectomy is executed.

Treatment of appendiceal abscess must be only operative. Opening and drainage of abscess, from retroperitoneal access, is performed. To delete here the appendix is not necessary, and because of denger of bleeding, peritonitis and intestinal fistula — even dangerously.

Intestinal obstruction is a complete or partial violation of passing of maintenance by the intestinal tract.

The principal reasons of intestinal obstruction can be:

- 1) commissures of abdominal cavity after traumas, wounds, previous operations and inflammatory diseases of organs of abdominal cavity and pelvis;
- 2) long mesentery of small intestine or colon, that predetermines considerable mobility of their loops;
- 3) tumours of abdominal cavity and retroperitoneal space.

Such principal reasons can cause violation of passing of intestinal maintenance, disorder of suction from the intestine and loss of plenty of electrolytes both from vomiting and in the intestine cavity as a result of disorders of bloodflow in its wall.

The morphological signs of dynamic intestinal obstruction are: small thickening of wall (at considerable paresis is thinning), friability of tissue (the bowel breaks easily) and presence of liquid maintenance and gases in cavity of bowel. At mechanical obstruction it is always possible to expose the obstacle: strang, commissures, tumours, jamnings of hernia, cicatricial strictures, wrong entered drainages, tampons and others like that. In place of compression strangulation is exposed. The bowel loop higher strangulation is extended, and distally — collapsed. In case of released invagination on small distance two strangulation furrows are observed, and distally from the second ring cylinder expansion of bowel lumen is observed.

Beginning of clinical signs of intestinal obstruction is sudden — in 1–2 hours after taking the meal. The pain in the abdomen has the intermittent character and is met in all forms of mechanical intestinal obstruction. However, some types of strangulated intestinal obstruction (node formation, volvulus of thin and colons) can be accompanied by permanent pain. It is needed to mark that at spike intestinal obstruction, invagination and obturation cramp-like pain can be considered as pathognomic sign of disease. For paralytic intestinal obstruction more frequent is inherent permanent pain which is accompanied by the progressive swelling of abdomen. At spastic obstruction of intestine the pain is mainly acute, the abdomen is not blown away, sometimes pulled in.

Nausea and vomiting are met in 75–80 % patients with the heaviest forms of high level of intestinal obstruction (node formation, volvulus of small intestine, spike obstruction). At obturation obstruction and invagination they are observed not so often.

There is a characteristic thirst which can be considered as an early symptom. Besides, the higher intestinal obstruction, the greater the thirst.

Swelling of abdomen, the delay of emptying and gases are observed in 85–90% patients, mainly, with the high forms of obstruction (volvulus of small intestine, spike intestinal obstruction).

Together with that, for invagination emptying by liquid excrement with the admixtures of mucus and blood are more characteristic.

In patients during palpation the soft abdomen is observed, sometimes — with easy resistance of front abdominal wall, and at percussion — high tympanitis. At auscultation at the beginning of disease increased peristaltic noises are present, then gradual fading of peristalsis is positive (the Mondor's symptom, "noise of beginning, quietness of end").

There are other symptoms pathognomic for intestinal obstruction.

The Vala's symptom is the limited elastic sausage-shaped formation.

The Sklarov's symptom is the noise of intestinal splash.

The Kywul's symptom is the clang above the exaggerated bowel.

The Schlang's symptom is the peristalsis of bowel, that arises after palpation of abdomen.

The Spasokukotsky's symptom is "noise of falling drop".

The Hoehenegg's symptom — incompletely closed anus in combination with balloon expansion of ampoule of rectum.

At survey roentgenoscopy or -graphy of the abdominal cavity in the loops of bowels liquids and gas are observed — the Klobjer's bowl (Pic. 3.3.4).

Strangulated obstruction. The ischemic component is the characteristic feature of this form of intestinal obstruction, that is investigation of squeezing of mesentery vessels, which determines the dynamics of pathomorphologic changes and clinical signs of disease, and the basic place among them belongs to the pain syndrome. Consequently, sudden appearance of disease, acuteness of pain syndrome and ischemic disorders in the wall of bowel cause necrosis changes of area of bowel pulling in a process. It is accompanied by the making progress worsening of the patient condition and origin of endotoxiosis.

Obturation intestinal obstruction, unlike strangulated, pass not so quickly. In its clinical picture on the first place there are the symptoms of violation of passage on the intestine (protracted intermittent pain, flatulence), instead of symptoms of bowel destruction and peritonitis.

For high, especially strangulated, intestinal obstruction progressive growth of clinical signs of disease and violation of secretory function of intestine is inherent. Thus the volume of circulatory blood diminishes, the level of haematocrit rises and leukocytosis grows. There are also deep violations of homeostasis (hypoproteinemia, hypokalemia, hyponatremia, hypoxia and others like that). In patients with low intestinal obstruction above-named signs are less expressed, and their growth is related to more protracted passing of disease. Invagination of bowel which can be characterized by the triad of characteristic signs is

the special type of intestinal obstruction with the signs of both obturation and strangulation: 1) periodicity of appearance of the intermittent attacks of pain in the abdomen; 2) presence of elastic, insignificantly painful, mobile formation in an abdominal cavity; 3) appearance of blood in the excrement or its tracks (at rectal examination).

The special forms of obturation intestinal obstruction is the obstruction caused by gall-stones. The last are got in the small intestine as a result of bedsores in the walls of gall-bladder and bowel, that adjoins to it. It is needed to mention that intestinal obstruction can be caused by concretum with considerably more small diameter than bowel lumen. The mechanism of such phenomenon is related to irritating action of bilious acids on the bowel wall. The last answers this action by a spasm with the dense wedging of stone in the bowel lumen.

Development of intestinal obstruction caused by gall-stones the attack of colic and clinic of acute cholecystitis precede always. Characteristically, that in the process of development of disease the pain caused by acute cholecystitis calms down, whereupon the new pain characteristic of other pathology — intestinal obstruction appears.

Dynamic intestinal obstruction is divided into paralytic and spastic. Paralytic obstruction often arises after different abdominal operations, inflammatory diseases of organs of abdominal cavity, traumas and poisonings. The reason of spastic intestinal obstruction can be the lead poisonings, low-quality meal, neuroses, hysterias, helminthiasis and others like that. Clinic of dynamic intestinal obstruction is always variable in signs and depends on a reason, that caused it. Disease is characterized by pain in the abdomen, delay of gases and emptying. During palpation the abdomen is blown away, painful, however soft. To diagnose this form of intestinal obstruction is not difficult, especially, if its etiology is known.

Hemostatic intestinal obstruction develops after embolism or thromboses of mesenteric arteries and thromboses of veins, there can be mixed forms. Embolism of mesenteric arteries arises in patients with heart diseases (mitral and aortic failings, heart attack of myocardium, warty endocarditis) and declared by damaging, mainly, upper mesentery arteries. Beginning of disease, certainly, is acute, with nausea, sometimes — vomiting. At first there is a picture of acute abdominal ischemic syndrome, that is often accompanied by shock (frequent pulse, decline of arterial and pulse pressure, death-damp, cyanosis of mucus membranes and acrocyanosis). Patients become excitative, uneasy, occupy the forced knee-elbow position or lie on the side with bound legs.

During the examination the abdomen keeps symmetry, abdominal wall is soft, the increased peristalsis is heard from the first minutes during 1–2 hours (hypoxic stimulation of peristalsis), which later goes out gradually (“grave quiet”). According to the phenomena of intoxication peritonitis grow quickly. At the beginning of disease the delay of gases and emptying is observed, later there is diarrhea with the admixtures of blood in an excrement. When the last is heavy to set macroscopically, it is needed to explore scourage of intestine.

Intestinal obstruction must be differentiated with the acute diseases of organs of abdominal cavity.

The perforation of gastroduodenal ulcer, as well as intestinal obstruction, passes acutely with inherent to it by sudden intensive pain and tension of muscles of abdomen. However, in patients with this pathology, unlike intestinal obstruction, the abdomen is not exaggerated, and pulled in with “wooden belly” tension of muscles of front abdominal wall. There is also characteristic ulcerous anamnesis. Roentgenologic and by percussion pneumoperitoneum is observed. Certain difficulties in conducting of differential diagnostics of intestinal obstruction can arise at atypical passing and in case of the covered perforations.

Acute pancreatitis almost always passes with the phenomena of dynamic intestinal obstruction and symptoms of intoxication and repeated vomiting, with rapid growth. During the examination in such patients, unlike intestinal obstruction, rigidity of abdominal wall and painfulness is observed in the projections of pancreas and positive Korte's symptom and Mayo-Robson's. The examination of diastase of urine and amylase of blood have important value in establishment of diagnosis.

Acute cholecystitis. Unlike intestinal obstruction, patients with this pathology complain for pain in right hypochondrium, that irradiate in the right shoulder-blade, shoulder and right subclavian area. Difficulties can arise, when the symptoms of dynamic intestinal obstruction appear on the basis of peritonitis.

The clinic of kidney colic in the signs and character of passing are similar to intestinal obstruction, however, attacks of pain in the lumbar area with characteristic irradiation in genital parts, the thigh and dysuric disorders help to set the correct diagnosis. Certain difficulties in conducting of differential diagnostics also can arise in difficult patients, at frequent vomiting which sometimes can be observed in patients with kidney colic.

During the first 1,5–2 hours after hospitalization of patient complex conservative therapy which has the differential-diagnostic value and can be preoperative preparation is conducted.

It is directed on warning of the complications related to pain shock, correction of homeostasis and, simultaneously, is the attempt of liquidation of intestinal obstruction by unoperative methods.

1. The measures directed for the fight against abdominal pain shock include conducting of neuroleptanalgesia, procaine paranephric block and introduction of spasmolytics. Patients with the expressed pain syndrome and spastic intestinal obstruction positive effect can be attained by epidural anaesthesia also.

2. Liquidation of hypovolemia with correction of electrolyte, carbohydrate and albuminous exchanges is achieved by introduction of salt blood substitutes, 5–10 % solution of glucose, gelatin, albumen and plasma of blood. There are a few methods suitable for use in the urgent surgery of calculation of amount of liquid necessary for liquidation of hypovolemia. Most simple and accessible is a calculation by the values of hematocrit. If to consider 40 % for the high bound of hematocrit norm, on each 5 % above this size it is needed to pour 1000 ml of liquids.

3. Correction of hemodynamic indexes, microcirculation and disintoxication therapy is achieved by intravenous infusion of Reopolylukline and Neohemodes.

4. Decompression of intestine tract is achieved by conducting of nasogastric drainage and washing of stomach, and also conducting of siphon enema. It is needed to underline that technically the correct conducting of siphon enema has the important value for the attempt of liquidation of intestinal obstruction by conservative facilities, therefore this manipulation must be conducted in presence of a doctor. For such enema the special device is used with the rectal tip, by a PVC pipe by a diameter of 1,5–2,0 cm and watering-can of very thin material. A liquid into the colon is brought to appearance of the pain feeling, then drop the watering-can below the level of patient who lies. The passage of gases and excrement is looked after. As a rule, this manipulation is to repeat repeatedly with the use of plenty of warm water (to 15–20 and more litres).

Liquidating of the intestinal obstruction by such conservative facilities is succeeded in 50–60 % patients with mechanical intestinal obstruction.

Patients with dynamic paralytic intestinal obstruction are expedient to stimulation of peristalsis of intestine to be conducted, besides, necessarily after infusion therapy and correction of hypovolemia. A lot of kinds of stimulation of intestine peristalsis are offered. Most common of them are:

1) hypodermic introduction of 1,0 ml of 0,05 % solution of proserin; 2) through 10 min — 60 ml intravenously stream of 10 % solution of chlorous sodium; 3) hypertensive enema.

Surgical treatment of intestinal obstruction must include such important moments:

1. According to middle laparotomy executed the novocaine blockade of mesentery of small and large intestine and operative exploration of abdominal cavity organs during which the reason of intestinal obstruction and expose viability of intestine is set.

The revision at small intestine obstruction begins from the Treitz' ligament to iliocecal corner. At large intestine obstruction the hepatic, splenic and rectosigmoid parts are observed intently. Absence of pathological processes after revision needs the examination of places of cavity and jamming of internal hernia: internal inguinal and femoral rings, obturator openings, pockets of the Treitz' ligament, Winslow's opening, diaphragm and periesophageal opening.

2. Liquidation of reasons of obstruction (scission of connection, that squeezes a bowel, violence of volvulus and node formation of loops, desinvagination, deleting of obturative tumours and others like that).

It is needed to mark that the unique method of liquidation of acute intestinal obstruction does not exist. At the lack of viability of bowel the resection of nonviable area is executed with 30–40 cm of afferent and 15–20 cm of efferent part with imposition of “side-to-side” anastomosis (Pic. 3.3.5) or “end-to-end” (Pic. 3.3.6).

3. Intubation. Decompression of intestine foresees conducting in the small intestine of elastic probe by thickness of 8–9 mm and length of 3–3,5 m with the plural openings by a diameter 2–2,5 mm along all probe, except for part, that will be in the oesophagus, pharynx and outwardly. A few methods of conducting of probe are offered in a bowel (nasogastric, through gastrostomy, ceco- or appendicostoma). Taking it into account, such procedure needs to be executed individually and according to indications.

Each of them has the advantages and failings. In connection with the threat of origin of pneumonia, entering an intubation probe to the patients of old ages is better by means of gastrostomy. Most surgeons avoid the method of introduction of probe through ceco- or appendicostoma because of technical difficulties of passing in a small intestine through a Bauhin's valve. Today the most wide clinical application has intubation of intestine extracted by the nasogastric method with the use of other thick probe as explorer of the first (by L.J. Kovalchuk, 1981). Such method not only simplifies procedure of intubation but also facilitates penetration through the piloric sphincter and duodenojejunal bend, and also warns passing of intestinal maintenance in a mouth cavity and trachea. Thus probe is tried to be conducted in the small intestine as possible farther and deleted the next day after appearance of peristalsis and passage of gases, however not later than on 7th days, because more protracted sign of probe carries the real threat of formation of bedsores in the wall of bowel.

4. Sanation and draining of abdominal cavity is executed by the generally accepted methods of washing of antiseptic. Draining of the abdominal cavity it is needed from four places: in both iliac areas and both hypochondrium, better by the coupled synthetic drainpipes.

Crohn's disease is an unspecific inflammatory process of submucosal membrane of gastrointestinal tract with propensity to the segmental lesions and recurrent passing. The local signs of disease exist in different areas of digestive tract organs, however, most frequent and most intensive they are in the distal segment of small intestine, therefore it was named terminal ileitis.

The reason of origin of the Crohn's disease for today is not finally found out. An infection and allergy are infringement factors. Together with that, granuloma, which is exposed at histological examination with present in its lymphocytic and protoplasmatic infiltrations, grounds to consider that the defined value in the origin of the Crohn's disease have immune factors. Thus inflammation begins in the submucosal membrane, and afterwards engulfs all bowel walls. The mucus membrane acquires the crimson colouring, there are deep cracks and ulcers. Combination of the damaged areas of mucus membrane with healthy creates a picture similar to the roadway. In future granuloma appears, an inflammatory process goes out outside the wall of bowel and gets to the contiguous organs (large and small intestines, urinary bladder, abdominal wall). In the eventual result there are infiltrate, abscesses and fistula. Finally, it is needed to mark that the people of young ages mainly are ill by terminal ileitis.

The morphological changes are concentrated, mainly, in the terminal part of iliac bowel, anal segment of rectum and appendix. Internal surface is hilly, thickened, swelling, deep ulcers are intermittent with the unchanged areas of mucus. The serous tunic is covered by plural, similar on tuberculosis, knots. Mesentery is sclerosed, regional lymphatic knots are hyperplastic, of whiter-rose color. By the most characteristic microscopic sign of Crohn's disease is presence of unspecific sarcoid granuloma. Hyperplasia of lymphoid elements of submucosal membrane and formation of fissured ulcers is observed also.

The Crohn's disease begins from the insignificant signs as a general weakness, increase of temperature of body, intermittent pain, that arises after the reception of meal, diarrhea without some visible features or with the admixture of blood. As this process strikes the terminal segment of small intestine, pain is concentrated in the right iliac area. Together with that, at localization of pathological focus in a colon with an anal segment pain is concentrated by its passing to the anal opening. A granuloma process takes place in the area of oesophagus, abdomen or duodenum, pain can arise up in the area of lesions. With progress of disease on endoscopy examination (proctosigmoidoscopy, fiberoptic colonoscopy, fiberoptic gastroscopy) hyperemia and deep cracks of mucus membrane, ulcers, symptom of “roadway” and stenosis are observed. At roentgenoscopy survey of organs of abdominal cavity in patients with the perforation it is possible to expose pneumoperitoneum, and at contrasting sciagraphy — stenosis of initial part of stomach, presence of ulcers or granuloma in the oesophagus. The examination of the passage on the small intestine enables to eliminate or confirm stenosis (Pic. 3.3.7). Irrigoscopy determines the defect of filling.

At acute passing of terminal ileitis, the pain appears acute in the right iliac area, sometimes intermittent, accompanied by nausea, vomitings, emptying by a liquid excrement with the admixture of blood or delay of emptying. During the examination of patient the abdomen can be exaggerated, tension of muscles and positive symptoms of irritation of peritoneum, high temperature is observed. In the general analysis of blood leukocytosis is present with the change of leukocyte formula to the left. In such difficult situation often only laparotomy helps to specify the diagnosis. The swollen segment of iliac bowel is thus observed with increased mesentery lymphatic knots. The changed area of bowel can perforate in the free abdominal cavity or penetrate in the contiguous loops of large or small intestine. It causes forming of inflammatory infiltrate, and in future — abscess formation. The unoperated abscesses are always inclined to the independent opening in surrounding organs with subsequent formation of fistula ducts.

The disease with the lesions of other parts of small and large intestine passes acutely (granulomatous enteritis, colitis). By palpation in these patients painful infiltrate is exposed, which by the character remind the clinic of invagination. Only the meticulous examination and present data analysis enable to set correct diagnosis. At granulomatous proctitis the plural cracks of mucus membrane without the signs of spasm of sphincter appear often, on the basis of which afterwards there are ulcers, that badly granulate. The same changes can develop on skin round the anal opening.

The chronic forms of disease often pass with insignificant symptoms. From the beginning of disease to establishment of diagnosis sometimes 1–2 years and more pass. Such patients periodically complain for pain, diarrhea, weight loss, increase of body temperature, nausea, vomitings and bleeding from a rectum.

Objectively in the abdominal cavity painful infiltrate is determined, and at laboratory examination — anaemia and hypoproteinemia.

Complications of the Crohn's disease can be divided into local and general. Among local, formations of fistula which arise on the front abdominal wall between the damaged bowel and surrounding organs are most characteristic (ileoileal fistula, entero-entero, enterovesical fistula). Sometimes fistulas are opened in the area of scars after the operations on the lateral wall of abdomen or in the area of anus. Next to that, stenosis inflammatory infiltrate of bowel can be transformed in acute or chronic intestinal obstruction. Some patients have the obvious threat of perforation of the changed wall of bowel or intestinal bleeding. The protracted passing of disease can be also complicated by malignization. The aphthous ulcers of tongue, node erythema, arthritises and chronic lesions of liver are general complications.

The Crohn's disease must be differentiated with the unspecific ulcerous colitis and cancer of colon.

An unspecific ulcerous colitis mainly initially strikes the mucus membrane of all colon. The disease is accompanied by the excreta with the excrement of plenty of blood and mucus. For Crohn's disease languid passing of disease is characteristic. Acute passing of disease is met considerably rarer, than chronic. The modern methods of endoscopic examination with the biopsy of mucus membrane, which helps to specify diagnosis, are helpful in differential diagnostics.

The cancer of colon is mostly accompanied by formation of deep ulcers and infiltrate. However, for the cancer process slowly progressive passing without the periods of remission is more inherent, thus the disease more frequently ends with the phenomena of intestinal obstruction. At roentgenologic examination on the background the relatively unchanged colon the lonely defect of filling is observed, and during colonoscopy — thrusting out in the lumen of bowel with an erosive surface or signs of disintegration. Histological examination of biopsy material enables to expose cancer cells.

Conservative treatment. The diet of patient, generally, must be ordinary, except for products with bad intestinal uptake. The medicine of the first row is 5-ASK (aminosalicylic acid, sulfasalazone and glucocorticoid). The medicines of the second row are: 6- mercaptopurine, azatiopurine and metronidazole. At diarrhea diphenoxilate is used — 5 ml peroral three times per days, loperamide — 2 mg peroral 3–4 times per days, smecta — 1 pack 3 times per days. At the expressed anaemia, to the considerable loss of weight, system complications, relapse of disease after operation prednisolone is applied — for 40–60 mg peroral every day during 1–2 weeks. After that its day's dose is diminished to 10–20 mg during 4–6 weeks and, in the end, stopped. For patients which are irresponsive to steroid, asatioprine is appointed (2 mg/kr) peroral. Metronidazole in a dose of 400 mg twice a days is used in the case of granulomatous disease of perineum.

The presence of external and internal fistula, stenosis of bowel, perforation and recurrent bleeding is an indication to operative treatment. The method of choice of operation is the segmental resection of the pathologically changed bowel in the distance of 30–35 cm of proximal and distal from the damaged area. The regional lymph nodes is also deleted. In case of the perforation of bowel with poured peritonitis, it is recommend not to perform primary anastomosis because of possible insolvency of stitches after the resection. In this connection, afferent and efferent loops exteriorizes on the wall of abdomen as two-channel stoma (Pic. 3.3.8). The passage by an intestine (liquidation of stoma) is restored in 2–4 months after liquidation of the peritonitis signs.

An unspecific ulcerous colitis is a diffuse inflammatory process that is accompanied by the ulcerous-necrosis changes in the mucus membrane of colon and rectums.

Etiology of unspecific ulcerous colitis to this time is not finally found out. This disease is suffered by people in the age from 20 to 40 years. An infectious factor in development of disease for today is not confirmed. However, as the exception of meal of food allergens (milk, eggs) results in the improvement of passing of disease, it is possible to consider that allergy assists to development of inflammatory process. Important significance in genesis of this pathology is also attached to immunological violations. In the blood of patient sensibilizing on the antigen of mucus membrane of colon specifically lymphocytes and immune complexes are found. The antigen-antibody reaction can cause colitis. In most patients with the chronic recurrent unspecific ulcerous colitis a stress situation causes the process of acuteing. In future, obviously, there are violations of microcirculation and cellular structures, and also the transport system of cells membranes suffers, that carries potassium and sodium ions. Taking it into account, the timely exposure of disease in which the process is localized and has a reverse tendency, can result in the positive therapeutic effect.

In patients with an unspecific ulcerous colitis the relatively isolated damages of rectum and sigmoid colon, sigmoid colon and transverse colon, so total colitis are met. The necrosis component prevails as the acute form. The wall of bowel in such cases is swollen, hyperemic, with plural erosions and ulcers of irregular form. Its infiltration by lymphocytes, plasmocytes and eosinophils with characteristic formation of granulation, crypt and abscesses are microscopically observed. In patients with a chronic process prevail, mainly, reparative-sclerotic processes. A bowel is deformed, dense, segmentally narrowed. As a result of the disfigured regeneration plural granulomatous and adenomatous pseudopolypuses appear.

Pain in the abdomen and diarrhea is one of basic signs of unspecific ulcerous colitis with emptying from 3 to 20 and more times per days. Thus during defecation the mixture of liquid excrement, mucus and blood are observed. As far as progress of disease the pain has the intermittent character and is localized by the passing of colon. By palpation it is spastic and painful. Frequent diarrhea is brought to dehydration, loss of electrolytes, albumen and anaemia. Patients are weak, there are the strongly expressed signs of intoxication, the temperature of body rises to 40 oC and the psyche is repressed. Characteristic also are tachycardia, decline of arterial pressure, avitaminosises and edema. Hypochromic anaemia is exposed in the general analysis of blood, leukocytosis, change of leukocyte formula to the left and increased ESR. In plasma of blood the decline of maintenance of potassium and sodium ions and level of general protein, especially albumen are marked. In future there are the progressive degenerative changes of parenchymatous organs.

At endoscopic examination (proctoscopy, fiberoptic colonoscopy) hyperemia of mucus membrane, swollen, contact bleeding, plural erosions, ulcers, festering and necrosis stratifications, are observed. At heavy passing of disease fiberoptic colonoscopy or irrigoscopy always has the danger of perforation or acute bowel dilatation, therefore more rationally it is to conduct it in the period of calming down of inflammatory process. During roentgenoscopy survey of organs of abdominal cavity in case of disease, complicated of acute toxic dilatation, the extended (from 10 to 20 cm and more) pneumatized bowel is exposed. During the perforation of bowel signs of pneumoperitoneum are present (air under the right cupula of diaphragm). Contrasting irrigoscopy examination in such patients enables to establish the presence of "water-pipe" symptom. Thrusting out of tailings of well-kept mucus membrane on the background of plural ulcers and cicatricial changes of bowel walls at pseudopolyposis creates roentgenologic reality of "shot target through" symptom (Pic. 3.3.9). Often in case of transformation of ulcerous colitis in the cancer on roentgenograms, stenosis cavity of bowel or defect of filling is observed (Pic. 3.3.10).

Acute, especially fulminant form of the unspecific ulcerous colitis passes the heaviest, so the prognosis is always doubtful. Taking it into account, death can come in the first days of disease. Thus an inflammatory process will strike all colon. During 1–2 days the heavy clinical picture is observed with frequent diarrhea with mucus, blood and pus, vomiting, dehydration and loss of weight of body. Next to that, deep intoxication, darkened consciousness, and the temperature of body rises to 39–40 oC is present. The expressed anaemia, tachycardia and hypovolemia are observed. The loss of albumens causes the decline of oncotic pressure and causes dehydration. The disbalance of electrolytes grows with progress of disease, microcirculation gets worse and day's diuresis goes down. In most cases this form of disease requires urgentoperative treatment (absolute indications).

A chronic recurrent unspecific ulcerous colitis is characterized by the periods of acuteing and remission. Thus in patients with the total lesions of colon in the period of acuteing the heavy degree of disease is observed, and in the period of remission — middle or even easy degree of disease, thus, such "calming down" can last 6 months and more.

A chronic continuous unspecific ulcerous colitis at the total lesions of intestine in most patients passes as middle heavy degree, and in the period of worsening the disease takes heavy shape. The easy form is met, mainly, at presence of inflammatory process in the rectum and sigmoid colon, considerably rarer it is at the lesions of left half of colon and quite rarely — at the total lesions in the period of calming down of the process. Conventionally, the unspecific ulcerous colitis begins from the rectum and engulf all parts of colon. Thus emptying are 2–3 times per days with the admixtures of mucus, sometimes blood. Thus, diarrhea can be intermittent with constipation. The temperature of body remains within the limits of norm. From the side of global and biochemical analysis of blood noticeable changes do not arise. Weight of body does not diminish. At endoscopic examination hyperemia of mucus membrane, contact bleeding, expressed vascular picture, erosions, point hemorrhages and superficial ulcers are observed. It is needed also to mark that in this situation the presence of erosions must be equated with an ulcerous process.

The middle heavy form of disease of the unspecific ulcerous colitis can be met in patients with the ulcerous colitis and proctosigmoiditis in the period of process acuteing. Thus there are the subjective feelings with considerable expression of tenesmus and heartburns.

The chronic forms of unspecific ulcerous colitis both at total and at the left-side lesions of colon, pass at the level of middle heavy degree. Frequency of emptying reaches to 5–10 times with mucus, blood and pain. Low grade fever, general weakness, nausea and loss of appetite appear, and weight of body diminishes on 5–8 kg. Moderate anaemia is exposed in the general analysis of blood, leukocytosis, increased ESR. Among the biochemical indexes of blood hypoproteinemia and hypokalemia are marked. At endoscopic examination of colon there is a considerable hyperemia and edema of mucus membrane, plural erosions, contact bleeding and superficial ulcers.

The heavy form of unspecific ulcerous colitis is at the total lesions, especially with acute, and also chronic recurrent passing of disease. The temperature of body in such patient rises to 39–40 oC, there is diarrhea (more than 10 times per days) with mucus, blood and pus, vomitings, heavy intoxication grows, weight loss on 25–30 kg, acutely expressed anaemia, leukocytosis with the change of leukocyte formula to the left, considerable changes of albuminous and electrolyte exchanges. At endoscopic examination of colon the blood is exposed in its cavity, slid, pus, fibrin incrustation, often pseudopolypuses and almost complete absence of mucus membrane. Roentgenologically some signs of complications of unspecific ulcerous colitis are confirmed.

The complications are divided into local and general. Local complications are: profuse intestinal bleeding, perforation, acute toxic dilatation, stenosis and malignization. To general the following are included: damage of liver (hepatitis, cirrhosis), stomatitis, ulcer of lower extremities, lesions of joints, eyes and skin.

Acute dysentery passes with bloody diarrheas, increased temperature of body, pain in the abdomen. Bacteriological examination of excrement enables to expose dysenteric bacillus and specify diagnosis.

Crohn's disease (granulomatous colitis) is this local process, that begins from the submucosal layer of bowel and distributes outside of walls with subsequent formation of infiltrate, abscesses and fistula. Exposure of granulomas, and during microscopic examination — accumulation of lymphocytes, neutrophils, protoplasmatic cells and the Pyrohov-Lunghans' cells are confirmed diagnosis.

The cancer of colon, in particular its enterocolitis and toxicoanaemic forms, also often can simulate an unspecific ulcerous colitis. Irrigoscopy, fiberoptic colonoscopy with biopsy and subsequent histological examination almost always help to diagnose cancer process.

Treatment of unspecific ulcerous colitis, certainly, begins with application of conservative facilities. Thus patients with easy and middle heavy forms must be under protracted conservative treatment.

The leading role is taken to the parenteral feed of patients with heavy common exhaustion (hydrolyzate of casein, aminopeptid, amynsol, vamin, alvesin, moriamin, intralipid, lipofundin, glucose and others like that). Electrolytes (chloride of sodium, sulfate of magnesium, chloride of potassium, panangin) and vitamins are entered (B6, B12, C, K, PP and others). Intensity and methods of conservative therapy always must depend on the phase of disease:

- moderately expressed passing of disease or proctitis — corticosteroid enema and sulfasalazone peroral;
- at heavy passing is parenteral introduction of liquids, nutritives, blood transfusion, system use of corticosteroids, surgical treatment;
- at chronic passing is corticosteroids peroral, asatioprine, surgical treatment;
- at remission is preparations of 5-aminosalicylic acid peroral, examination for the exception of cancer of colon.

The heavy form of passing of disease is absent of effect from the conducted conservative treatment during two weeks and progress of process testifies to the necessity of surgical treatment.

The conservative treatment must include antibacterial agent, antidiarrheal preparations, steroid hormone.

A diet is considered an important factor in treatment of such patient (diet № 4). Thus it is recommended to take a meal to 6 times per days by small portions, withdrawing milk, fruit, vegetables, wheat and rye bread from it. It is possible to appoint unfat meat and fish. Parenteral introduction of vitamins B, C, A, folic acid are helpful.

The basic antiinflammatory facilities are: sulfasalazopreparations (sulfasalazone, salazopirine), salicylazosulfanilamide (salazosulfa-pyridine, salazodimetoxine) and corticosteroids. Practice showed that sulfasalazone was one of the best antirecurrent facilities.

In patients with easy and middle heavy forms (distal or left-side lesions of colon) sulfasalazone is applied in a dose about 5 g per days, and salazopiridazine and salazodimetoxine — about 2 g during that time. The course of treatment must proceed 1–2 months. For local steroid therapy prednisolone is used as powder in a dose 60–80 mg or hydrocortisone — to 125 mg. It is dissolved in 100 ml physiologic saline and entered rectal dropwise one time per days during 3–4 weeks.

At erosive proctitis and proctosigmoiditis 5% (100,0 ml) solution of kolargole or extract of camomile in microclyster is applied.

At the heavy forms of ulcerous colitis with fulminant passing and frequent vomiting the treatment is needed to begin with intravenous introduction of 350–380 mg hydrocortisone per day. Thus procedures must proceed to appearance of positive clinical effect and realization of possibility of transition on enteral treatment. Such period lasts on the average of 6–7 days. In future it is recommended to adopt prednisolone peroral.

Sulfasalazopreparations is used in the same dose, as at the middle heavy form of flow of disease. As in patients at this form of disease water-electrolyte and albuminous exchanges are considerably violated,

there are the expressed intoxication and anaemia, it is expedient to conduct adequate therapy (intravenously - NaCl solution, glucose, chlorous potassium, albumen, hemodes, protein, whole blood), and also hemodialysis and oxygenotherapy is used.

An absolute indication for surgical treatment is the presence of such complications of unspecific ulcerous colitis as: perforation of wall of bowel, acute toxic dilatation, stenosis, profuse bleeding and malignization. By the choice of method of operation at such pathology it is needed to count coloproctectomy with exteriorization of ileostomy.

However, during the perforation of colon or toxic dilatation the operative treatment can be limited to colectomy because proctectomy will be conducted as the next stage.

For patients with total ulcerous colitis with chronic heavy passing and without the tendency to the visible improvement expedient radical operative treatment — coloproctectomy with exteriorization of ileostomy. At such tactic postoperative lethality is diminished in 5-6 times, comparative with palliative operations which were conducted earlier.

In Western Europe and North America colorectal cancer (CRC) is one of most widespread malignant neoplasm. Morbidity in these regions exceeds 20,0 on a 100000 of population. In Japan, South America, Africa and countries of Asia this tumour is met rarer (6,0:100000). There is an annual increase of frequency of cancer of colon approximately by 3 % in the developed countries. Morbidity of population of Ukraine by malignant neoplasm in 1995 was 16,3:100000.

Approximately 85 % of patients for CRC are of over 50 years old, with age frequency of cancer is increased.

The nutrition by fat and albuminous food promotes the elimination to the intestine of bile. Under the act of bacterial flora there is transformation of primary bilious acids to the secondary, which has the carcinogenic and mutagenic activity. A meal with vitamins A, C and that which contains plenty of vegetable cellulose has a braking carcinogenic influence.

The factors of risk which predetermine the origin of cancer of bowel are:

- 1) diffuse (family) polyposis, which is considered obligate precancer;
- 2) plural and single adenomatous polypses;
- 3) chronic unspecific ulcerous colitis (anamnesis more than 10 years);
- 4) Crohn's disease (granulomatous colitis).

Localization. A tumour is mostly localized in sigmoid (35–40 % cases) and blind (20–25 % cases) bowels.

Macroscopic forms. Exophytic tumours grow in the lumen of bowel as a polypus or knot and at disintegration have the appearance of ulcer with a dense bottom that is swelling by edges which come forward above the surface of the damaged mucous (saucer-shaped cancer). The endophytic (infiltrate) cancer grows in walls of bowel. The tumour spreads on the perimeter of bowel and engulfs it circular, causing narrowing of its lumen. In the right half of colon exophytic tumours grow, as a rule, in left — endophytic tumours.

Histological structure. Cancer of colon in 95 % cases has the structure of adenocarcinoma. Metastasis takes place by lymphatic and hematogenic ways in regional retroperitoneal lymphatic knots, liver, lungs.

The symptoms of cancer of colon are so numerous and various, that many authors group them in such clinical forms: toxico-anemic, dyspeptic, enterocolitic, obturation, pseudoinflammatory and tumular.

A toxico-anemic form shows up by indisposition, weakness, rapid fatigability, increase of temperature, progressive anaemia. Characteristic for the cancer of right half of colon.

The enterocolitic form is characterized by symptom of complex intestinal disorders: diarrhea, constipation, swelling, grumbling, pain.

The dyspeptic form is characterized by functional disorders of gastrointestinal tract.

An obturation form shows up by intestinal obstruction.

A pseudoinflammatory form is characterized by the symptoms of inflammatory process in the abdominal cavity.

A tumour form passes asymptomatic. A tumour is exposed by chance by a patient or doctor.

Obturation and enterocolitic forms more characteristic for the cancer of left half, other ones — of the right. For the cancer of right half of colon tendency to gradual progress is characteristic, and the tumours of left half often show up suddenly by intestinal obstruction.

Intestinal obstruction, germination in neighbouring organs and tissue, perforation, bleeding are considered as the most frequent complications of colon cancer.

Depending on the clinical signs of colon cancer, a differential diagnosis is to be conducted with appendiceal infiltrate, by different chronic specific and unspecific diseases of colon, and also other organs of abdominal cavity and retroperitoneal space (gall-bladder, pancreas, kidneys, genital organs and others like that), with the tumours of other organs of abdominal cavity and retroperitoneal space.

Radical treatment. Operative treatment is the unique method of radical treatment of colon cancer. The choice of method of operation depends on localization of tumour (Pic. 3.3.11). At cancer of right half of colon right hemicolectomy, (deleting of all right half of colon, including right third of transversal colon and distal segment of iliac bowel by length 20–25 cm) is conducted. In patients with tumours of left half of colon left hemicolectomy (segment from middle or from left third of transversal colon to overhead part of sigmoid is resected) is executed. At cancer of transversal colon, middle and distal parts of sigmoid bowels the resection of the damaged area is conducted, stepping back 5-6 cm from the edge of tumour. In patients who are hospitalized in an urgent order with the signs of intestinal obstruction, perforations and peritonitis, after intensive preoperative preparation the Hartmann operation is executed (at tumours of left half of colon). It is the resection of the damaged area of intestine and exteriorization of proximal segment on a front abdominal wall as colostomy. A distal end is sutured and is remained in the abdominal cavity. Through half-year there is a possibility of reconstructive operation. To the patients with the damage of right half of colon operation in a radical volume can be executed (right hemicolectomy).

During treatment with palliative purpose (at presence of solitary metastases) operations in a radical volume with removing of metastatic knot (in a liver) or subsequent chemotherapy (by 5- fluorouracil) can be used.

In recent years for the improvement of remote results treatment is applied by the adjuvant chemotherapy and intensive preoperative gamut-therapy.

The remote results of treatment of patients on the initial stages of CRC are fully satisfactory. At I stages the five-year survival is 85-100 %, at II — 65–70 %, at III — 25–30 %. On the whole at the I-III stages the five-year survival is 45 %.

Persons who refused from operative treatment perish in a short time. The combined treatment improves remote results approximately on 15–20%.

The gastric ulcer is the chronic disease with polycyclic passing. The main typical of peptic ulcer is the presence of ulcerous defect in a mucous tunic. One of basic places belongs among the gastroenterology diseases to this pathology. Such phenomenon explained by not only considerable distribution of disease but also those dangerous complications which always accompany gastric ulcers.

Frequency of morbidity on the peptic ulcer among the adult population is about 4 %. More frequent age in patients with gastric ulcers is 50–60 years.

To development mechanism of disease is still not enough studied. From a plenty of different theories in relation to genesis of peptic ulcer no one able to explain the disease. So, each of such factors as neurogenic, mechanical, inflammatory, vascular is present in the mechanism of development of peptic ulcer. Consider for today, that disturbance between the factors of aggression and defense of mucous tunic arose peptic ulcer. To the first factors belong: hydrochloric acid, pepsin, reverse diffusion of ions of hydrogen, products of lipid hyperoxidizing. To the second: mucus and alkaline components of gastric juice, property of epithelium of mucous tunic to permanent renewal, local blood flow of mucous tunic and submucous membrane.

In the terminal stage of mechanism of origin of gastric ulcers important role has the peptic factor and disturbance of trophism of gastric wall as a result of local ischemia. It confirmed by decreasing of blood flow in the wall of stomach at patients with ulcers on 30–35 % compared to the norm. It is proved, that a local and functional ischemia more frequent arises up on small curvature of stomach in the areas of ectopy of the antral mucous tunic in acid-forming. Exactly there ulcers appear.

Important part in ulcerogenesis is acted by duodenogastric reflux and gastritis. Also, gastrostasis can provoke hypergastrinaemia and hypersecretion and formed gastric ulcers.

Numeral scientific developments of the last years testify to the important infectious factor in the mechanism of origin of peptic ulcer conditioned, mainly, by helicobacter pylori.

Such stages of disease are distinguished: erosion, acute and chronic ulcers.

Erosions, mainly, are plural. Their bottom as a result of formation of mucriatic haematin is black, edges — infiltrated by leucocytes. A defect usually does not penetrate outside muscular tissue of the mucous tunic. If necrosis gets to more deep layers of wall of stomach, a acute ulcer develops. It has a funnel-shaped form. Bottom is also black, edges is swelled. Chronic ulcers are mainly single, sometimes arrive to the serous layer. A bottom is smooth, sometimes hilly, edges is like elevation, dense.

For today the most known classification of gastric ulcers by Johnson (1965). There are three types of gastric ulcers are distinguished: I – ulcers of small curvature (for 3 cm higher from a goalkeeper); II – double localization of ulcers simultaneously in a stomach and duodenum; III – ulcers of goalkeeper part of stomach (not farther as 3 cm from a goalkeeper). In the area of small curvature of body of stomach is localized 70,9 % ulcers, on a back wall, nearer to small curvature — 4,8 %, in the area of cardiac part — 12,9 %, in a goalkeeper part — 11,4 %. The ulcers of large curvature of stomach are casuistry and meet infrequently.

The complaints of patients with the gastric ulcer always give valuable information about the disease. The detailed analysis of their anamnesis allows to pay attention to the possible reasons of origin of ulcer, time of the first complaints, to the changes of symptoms.

Pain. A pain symptom in the peptic ulcer disease is very important. There are typical passing for this disease: hunger – pain – food intake – facilitation – again hunger – pain – food intake – facilitation (so during all days). Night pain for the gastric ulcer is not typical. The such patients rarely wake up in order to take a food. For diagnostics of ulcer localization it is important to know the time of appearance of pain. Between acceptance of food and appearance of pain it is the shorter, than the higher placed gastric ulcer. Thus, at patients with a cardiac ulcer pain arises at once after the food intake, with the ulcers of small curvature — in 50–60 minutes, at pyloric localization — approximately in two hours. However this feature it is enough relative and some patients in general do not mark dependence between food intake and pain. In other patients the pain attack is accompanied by the salivation.

A epigastric region near the xiphoid process is typical localization of pain. The irradiation of pain is not usual for gastric ulcers. Irradiation occur in patients with penetration and depended from organ, in which an ulcer penetrates.

At the examination of ulcerous patient it is expedient to determine the special pain points: Boas (pain at pressure on the left of the X–XII pectoral vertebrae), Mendel (pain at percussion on the left to epigastric region).

Vomiting, the sign of disturbance of motility function of stomach, is the second typical symptom of gastric ulcer. More frequent gastrostasis arises as a result of failure of stomach muscular, it atony which can be effect of organ ischemia. Vomiting could arises both on empty stomach and after food intake.

Heartburn is one of early symptoms of gastric ulcer, however at the prolonged passing of disease it can be hidden or quite disappear. Often it precedes of pain arising (initial heartburn) or accompanies a pain symptom. Mostly heartburn arises after the food intake, but can appear independently. It is observed not only at hypersecretion of the hydrochloric acid, but at normal secretion, even reduced acidity of gastric juice.

The belching at gastric ulcers is examined rarely, more frequent in patients with cardiac and subcardial ulcers. It is necessary to bind to disturbance of function of cardiac valve.

The general condition of patients with the uncomplicated gastric ulcer usually satisfactory, and in a period between the attacks — even good. However for most patients lost of the body weight and pallor are typical. In a epigastric region hyperpigmental spots are examined after the prolonged application of hot-water bottle. At palpation of stomach in this area sometimes appears local painful. It is needed also to check up “noise of splash”, the presence of which can be the sign of possible gastrostasis.

At the examination of mouth cavity a tongue has whiter-yellow incrustation. In patients with penetration ulcers and disturbances evacuations from a stomach examined dryness of tongue.

Stomach, as a rule, regular rounded shape, however during the pain attack is pulled in. There is antiperistalsis arises during the pylorostenosis.

The increased secretion of hydrochloric acid in patients with gastric ulcer observed rarely and, mainly, at prepyloric ulcer localizations. Mostly secretion is normal, and in some patients is even reduced.

X-Ray examination. The direct signs of ulcer at X-Ray examinations are: symptom of “Haudek's niche” (Pic. 3.2.1), ulcerous billow and convergence of folds of mucous tunic. Indirect signs: symptom of “forefinger” (circular spasm of muscles), segmental hyperperistalsis, pylorospasm, delay of evacuation from a stomach, duodenogastric reflux, disturbance of function of cardiac part (gastroesophageal reflux).

Gastroscopy can give important information about localization, sizes, kind of ulcer, dynamics of its cicatrization, and also allow to perform biopsy with subsequent histological examination.

The gastric ulcer passing can be acute and chronic. Acute ulcers arise as answer for the stress situations, related to the nervous overstrain, trauma, loss of blood, some infectious and somatic diseases. By a

diameter ulcers has from a few millimeters to centimeter, a round or oval form with even edges. Thus in most cases clinically observed clear ulcerous clinical signs. If complications is absent (bleeding, perforation) such ulcers treated and mostly heal over.

G.J. Burchynskyy (1965) such variants of clinical flow distinguished:

1. Chronic ulcer which does not heal over long time.
2. Chronic ulcer which after the conservative therapy heals over relatively easily, however inclined to the relapses after the periods of remission of a different duration.
3. Ulcers, which localization are had migrant character. Observed in people with acute ulcerous process of stomach.
4. Special form of gastric ulcer passing after the already carried disease. Passed with the expressed pain syndrome. Characterized by the presence in place of ulcerous defect of scars or deformations and absence of symptom of "niche".

There are such complications can develop in patients with gastric ulcer: penetration, stenosis, perforation, bleeding and malignization.

Chronic gastritis, as well as at a gastric ulcer, characterized by the pain syndrome, that arises after the food intake. In such patients it is possible to observe nausea and vomiting by gastric content, heartburn and belch. However, unlike an gastric ulcer, for gastritis typical symptom of "quick satiation by a food". Unsteady emptying, diarrhea also more inherent to gastritis. At gastric ulcer more frequent the delays are observed, constipation for 4–5 days.

The cancer of stomach, it is comparative with an gastric ulcer, has considerably more short anamnesis. The most typical clinical signs of this pathology are: absence of appetite, weight loss, rapid fatigability, depression, unsociability, apathy. In such patients X-Ray examination expose the "defect of filling", related to exophytic tumor and deformation of walls of organ. A final diagnosis is set after the results of multiposition biopsy of shady areas of mucous tunic of stomach.

Differential diagnostics also needs to be conducted with the so called precancerous states: gastritis with the achlorhydria; chronic, continuously recurrence ulcers, poliposis and Addison-Biermer anemia.

Conservative treatment of gastric ulcer always must be complex, individually differentiated, according to the etiology, pathogeny, localization of ulcer and character of clinical signs (disturbance of functions of gastroduodenal organs, complication, accompanying diseases).

Conservative therapy must include: a) anticholinergic drugs (atropine, methacin, platyphyllin) and myolitics (papaverine, halidor, nospanum); antacid drugs — in accordance with the results of pH-metry; b) H2- blocker histamine receptor (ranitidine) — 150 mg in the evening, famotidine — 40 mg at night, roxatidine — 150 mg in the evening, c) reparative drugs (dalargin, solcoseryl, actovegin) — for 2 ml 1–2 times per days; d) antimicrobial drugs (de-nol, metronidazole, semisynthetic antibiotic); e) vitamins of group B and symptomatic medicine.

Treatment of patient with a gastric ulcer must continues not less than 6–8 weeks.

Surgical treatment must performed in cases:

- a) at the relapse of ulcer after the course of conservative therapy;
- b) in the cases when the relapses arise during supporting antiulcer therapy;
- c) when an ulcer does not heal over during 1,5–2 months of intensive treatment, especially in families with "ulcerous anamnesis".
- d) at the relapse of ulcer in patients with complications (perforation or bleeding);
- e) at suspicion on malignization ulcers, in case of negative cytological analysis.

The choice of method of surgical treatment of gastric ulcer depended from localization and sizes of ulcer, presence of gastro- and duodenostasis, accompanying gastritis, complications of peptic ulcer (penetration, stenosis, perforation, bleeding, malignization), age of patient, general condition and accompanying diseases. In patients with cardial localization of ulcer the operation of choice is the proximal resection of stomach, which, from one side, allows to remove an ulcer, and from other — to save considerable part of organ, providing it functional ability (Pic. 3.2.2). In case with large cardial ulcers, when the vagus nerves pulled in the inflammatory infiltrate and it is impossible to save integrity even one of them, operation needs to be complemented by pyloroplasty. It will give possibility to warn pylorospasm and gastrostasis, which in an early postoperative period can be the reason of anastomosis insufficiency and other complications.

At the choice of method of surgical treatment of gastric ulcers with subcardial localization on small curvature without duodenostasis it is better to apply the methods of stomach resection with saving of passage through a duodenum.

For this purpose we are developed the method of segmental resection of stomach with addition selective proximal vagotomy. The redistribution of gastric blood flow between the functional parts of stomach as reply to medicinal vagotomy (intravenous introduction 1,0 ml 0,1 % solution of atropine of sulfate) is studied. Hyperemia of acid-forming part of stomach comes after introduction of preparation. The functional scopes of stomach parts are determined. The border between acid-forming and antral parts are the most frequent localization of gastric ulcers.

During this operation middle laparotomy is performed, intravenously entered 1,0 ml 0,1 % solution of atropine, then the scopes of functional stomach parts are identified and by stitches-holders is marked a intermedial segment. Selective proximal vagotomy is performed. After mobilization of large curvature of stomach within the limits of intermedial segment it resection is performed. After that gastro-gastro anastomosis "end-to-end" is formed (Pic. 3.2.3).

The analysis of supervisions of the patients operated by such method in postoperative period has good results. It allows to recommend this operation for clinical practice, in case of gastric ulcers of subcardial localizations, without duodenostasis, penetration, malignization or nerves Latarjet damaging.

The operation of choice in patients with subcardial ulcers and duodenostasis is gastric resection by Billroth II.

At the choice of method of surgical treatment of ulcers which are localized in upper and middle third of stomach, it is necessary to consider such factors, as absence of penetration in a small omentum and absence of the duodenostasis. In such patients is performed segmental resection of stomach with ulcer removing with selective proximal vagotomy. In case of penetration ulcer in a small omentum with involvement in infiltrate Latarjet nerves, such operation is impossible because of future spasm of pylorus and gastrostasis. If duodenostasis is absence than better to apply pylorus-saving resection by Maki-Shalimov. In patients with duodenostasis better to apply gastric resection by Billroth II.

At the border of gastric resection near pyloric sphincter can be spasm and gastrostasis in a postoperative period. Avoiding such complication is possible, if this border of gastric resection passes no more than 1,5 cm from a pyloric sphincter (M.M. Risaev, 1986). So, at a resection, that passes higher than 2,0 cm from a pylorus, integrity of both loops is kept.

Patients with antral ulcers without the duodenostasis performed the gastric resection by Billroth I (Pic. 3.2.6), and on presence of duodenostasis — Billroth II.

Prepyloric ulcers is similar to the ulcers of duodenum. Such localization of gastric ulcers without malignization allow to perform selective proximal vagotomy. However, at large prepyloric ulcers with penetration without duodenostasis is better to perform the gastric resection by Billroth I and on presence of duodenostasis — by Billroth II.

By contra-indication to operations with saving of food passing through the duodenum are also decompensated pylorostenosis, functional gastrostasis and duodenostasis. In such patients it is better to perform gastric resection by Billroth II.

The duodenal ulcer is the chronic recurrent disease which characterized by ulcerous defect on a mucous tunic of duodenum. Pathology often makes progress with complications development.

There are some etiologic factors of the duodenal ulcer: Helicobacter pylori, emotion tension and neuropsychic stress overstrain, heredity and genetic inclination, presence of chronic gastroduodenitis, disturbance of diet and harmful habits (alcohol, smoking). In pathogenesis of peptic ulcer a leading role is played disturbance of equilibrium between aggressive and projective properties of secret of stomach and it mucous tunic. The aggressive factors are vagus hyperfunctioning and hypergastrinemia; hyperproduction of hydrochloric acid and pepsin, and also reverse diffusion of the ions H⁺, action of bilious acids and isoleucine, toxins and enzymes of helicobacter pylori (HP). There are factors which are contribute to ulcerogenic action: disturbance of motility of stomach and duodenum, ischemia of duodenum, and metaplasia of the epithelium.

Morphogenesis of duodenal ulcer fundamentally does not differ from ulcer in a stomach. Chronic ulcers are mainly single, is localized on the front or back wall of bulb (bulbar ulcer) and only in 7–8 % cases — below it (postbulbar ulcer). The plural ulcers of duodenum are met in 25 % cases.

Pain in the epigastric region is the most expressed symptom of duodenal ulcer, often with displacement to the right in the projection area of bulb of duodenum and gall-bladder. Also for this pathology is typical the pain, that arises in 1,5–2 hours after food intake, "hungry" and nightly pain. As a rule, it is acute, sometimes unendurable, and is halted only after the use of food or water. Such patients complains for the seasonal exacerbation, more frequent in spring and in autumn. However exacerbation can be also in winter or in summer. In the acute period of disease heartburn often increases. However heartburn is the frequent symptom of cardial insufficiency and gastroesophageal reflux. For an duodenal ulcer the acute burning feeling of acid in a esophagus, pharynx and even in the cavity of mouth is especially typical. Often are belch by air or sour content, excessive salivation. Vomiting is not a typical symptom for duodenal ulcer. More typical sign is nausea. Sometimes for facilitation patients wilfully cause vomiting. These symptoms, arises in the late periods of passing of duodenal ulcer.

Intensity of pain and dyspepsia syndromes depends both on the depth of penetration and from distribution of ulcerous and perulcerous processes. Superficial ulceration within the mucous tunic, as a rule, does not cause the pain because it does not have sensible receptors. However, more deep layers of wall (muscular and especially serous) have plural sensible vegetative receptors. Therefore, on deepening and distribution of process arises visceral pain. At evident perulcerous processes and penetration of ulcers to neighboring organs and tissues, usually, a parietal peritoneum, that has spinal innervation, is pulled in. Pain becomes viscerosomatic, more intensive. A such pain syndrome (with an irradiation in the back) is typical for low postbulbar ulcers and bulbous ulcers of back wall, which penetrates in a pancreas and hepato-duodenal ligament. Usually such patients has good appetite. Some of them limit themselves in acceptance of ordinary food, go into to the dietary feed by small portions, and some — even hold back from a food, being afraid to provoke pain, and as a result of it weight is lost. Some of patients feeds more intensive and often.

The psychical status of patients often are changed as a asthenoneurotic syndrome: irritates, decline of working capacity, indisposition, hypochondria, abusiveness.

An inspection, as a rule, gives insignificant information. In many cases on the abdominal skin it is possible to notice hyperpigmentation after application of hot-water bottle. During the pain attack patients often occupy the forced position. At superficial palpation on the abdominal wall determined hyperesthesia in ulcer projection. In the epigastric region, during deep palpation, it is possible to define pain and muscular tension, mostly moderate intensity. There is important symptom of local percussion painful (Mendel's symptom): percussion by fingers in the symmetric epigastric areas provoke pain in the ulcer, which is increased after the deep breath. The roentgenologic and endoscopic are main diagnostic methods. The symptom of ulcerous "niche" is a classic roentgenologic sign. It is depot of contrast agent, which is corresponded to ulcerous defect, with clear contours and light bank to which converged fold mucus. Cicatricial deformation of bulb of duodenum as a shamrock, butterfly, narrowing, tube, diverticulum and other forms is the important sign of chronic ulcerous process. A roentgenologic method is especially important for determination of configuration and sizes of stomach and duodenum, and also for estimation of motility functions. X-Ray examination is the main method at the peptic ulcer complicated by stenosis, with disturbance of evacuation, duodenostasis, duodenal-gastric reflux, gastroesophageal reflux, diverticulum. But by X-Ray examination is difficult to diagnose small superficial ulcers, acute ulcers, erosions, gastritis and duodenitis. The most informing method in such cases it endoscopy.

During endoscopy examination it is possible to define localization, form, sizes and depth of ulcer. During bleeding grumes, trickle or pulsating of blood are observed. By irrigation by stypic solutions, by cryocoagulation, by laser coagulation endoscopy allows to secure hemostasis. Endoscopy allows to perform the biopsy of ulcer tissues for determination of possible malignization.

In patients with low postbulbar ulcers the clinical signs are more expressed. It characterized by late (in 2–3 hours after food intake) and intensive "hungry" and nightly pain, that often irradiate to the back and to the right hypochondrium. The postbulbar ulcers are inclined to more frequent exacerbation, and also to more frequent complications, such, as penetration, stenosis and bleeding.

The are more frequent ulcerous bleeding (the bulbous happen in 20–25 % cases, postbulbar — in 50–75 %), perforations (10–15 % cases). Penetration, stenosis and malignization in patients with duodenal ulcers are observed rarely.

Penetration is frequent complication of "low" and postbulbar ulcers of duodenum, which are placed on posterior, posterior superior and posterior inferior walls. Penetrates, usually, deep chronic ulcers, by passing through all layers of duodenum in neighboring organs and tissues (head of pancreas, hepato-duodenal ligament, small and large omentum, gall-bladder, liver). Such penetration is accompanied by development of inflammatory process in the neighboring organs and surrounding tissues and forming of cicatricial adhesions. A pain syndrome becomes more intensive, permanent and often pain irradiated in the back. Sometimes in the area of penetration it is possible to palpate painfully infiltrate.

The duodenal ulcer must be differentiated from acute and chronic cholecystitis, pancreatitis, gastroduodenitis. Endoscopy is help to diagnose duodenal ulcer.

Conservative treatment. In most patients after conservative treatment an ulcer heals over in 4–6 weeks. Warning of relapses can be carried out by only supporting therapy during many years.

The best therapy of duodenal ulcer is associated with a helicobacter infection, there is the use of antagonists of H₂- receptors of histamine (renitidine— 300 mg in the evening or 150 mg twice for days; famotidine— 40 mg in the evening or 20 mg twice for days; nizatidine — 300 mg in the evening or 150 mg twice for days; roxatidine — 150 mg in the evening) in combination with sucralfate (venter) — for 1 r three times for days and antacid (almagel, maalox or gaviscon —1 dessert-spoon in a 1 hour after food intake). To this complex it is needed to add antibacterial preparations (De-nol – 1 tabl. 4 times per a day during 4–6 weeks + oxacylline for 0,5 g 4 times per a day — 10 days + Tryhopol (metronidazole) for 0,5 g 4 times per a day — 15 days).

In treatment of duodenal ulcer used cholinotics and miolitics (atropine, methacin, platyphyllin), and also mesoprostol (200 mg 4 times per days) and omeprazole (20 or 40 mg on days).

Such treatment of patients with the duodenal ulcer must be 4–6 weeks. If complications absents there is no necessity in the special diet.

Because of appearance of new pharmaceutical preparations and modern therapeutic treatment, indication to the operative methods narrowed. But the number of acute complications of duodenal ulcer does not go down, especially bleeding and perforations which require the urgent surgery.

Indications to the elective operation:

1. Passing of duodenal ulcer with the frequent relapses which could not treated conservatively.
2. Repeated ulcerous bleeding.
3. Stenosis of outcome part of stomach.
4. Chronic penetration ulcers with the pain syndrome.
5. Suspicion for malignization ulcers.

Methods of surgical treatment.

At patients with the duodenal ulcer three types of operations are distinguished:

- organ-saving operations;
- organ-sparing operations;
- resection.

From them the better are: organ-saving operations with vagotomy, excision of ulcer and drainage operation.

Types of vagotomy: trunk (TrV) (Pic. 3.2.7), selective (SV) (Pic. 3.2.8), selective proximal (SPV) (Pic. 3.2.9). Selective proximal vagotomy is optimal in the elective surgery of duodenal ulcer. However in urgent surgery a trunk, selective or selective proximal is often used in combination with drainage operations.

Drainage of the stomach operations are: Heineke-Mikulicz pyloroplasty, Finney pyloroplasty, submucous pyloroplasty by Diver-Barden-Shalimov, gastroduodenostomy by Jaboulay, gastroenteroanastomosis.

It is necessary to mark that “clean isolated” SPV, performed in patients with duodenal ulcer, often (in 15–20 % cases) results in the relapses. The considerably less number of relapses (8–10 %) is observed after SPV in combinations with drainage operations. Especially dangerous is the relapses of the ulcers placed in the projection of large duodenal papilla, after gastroduodenostomy by Jaboulay.

The least number of relapses of duodenal ulcer is observed after organ-saving operations, that combine SPV and ulcer excision.

If ulcer localized on the anterior surface of duodenal bulb it can be performed by the method Jade (Pic. 3.2.13) with subsequent to the pyloroplasty by Heineke-Mikulich.

At patients with decompensate stenosis and expressed dilatation and by the atony of stomach it is needed to apply the classic resection of stomach depending on possible damping-syndrome by Billroth -I or Billroth -II.

The choice of subtotal resection of stomach needs to be done at suspicion for malignization or at histological confirmed malignization ulcers. In a duodenum this process happens very rarely.

Ulcerous stenosis is complication of Peptic ulcer or duodenum, which characterized by narrowing.

Stenosis of outgoing part of stomach and duodenum of ulcerous origin arises as a result of scarring and common morphological changes around an ulcer. Narrowing, disturbance of the coordinated motility of goalkeeper come as a result of it and creates the obstacle to the even moving of stomach content to the duodenum.

Such pathology in the compensation stage arises hypertrophy of the stomach walls. The pyloric ring has a 0,5–0,7 cm in diameter. The mucous tunic of pyloric part of stomach is thickened, with rough folds. Muscular fibers are hypertrophied and solid. Histological hyperplasia of pyloric glands is observed.

During decompensation the muscular layer of stomach higher stenosis becomes thinner, tone of him goes down, and a pyloric ring narrows to a few millimetres. Microscopically present atrophy of mucous tunic and muscular fibers, vessels sclerosis. A stomach collects the form of the stretched sack which goes down to the level of small pelvis.

The first signs of stenosis can be exposed already in eight-ten years from the beginning of the peptic ulcer disease.. Mainly, this is narrowing and rigidity and disturbance of retractive activity of goalkeeper, which create a barrier for transition of stomach content to the duodenum.

In the stage of the compensated stenosis hypertrophy of wall of stomach develops and tone of muscular shell rises. Hereupon gastric content, slowly, but passes through the narrowed area of stomach output. In this stage patients, usually, complained about feeling of plenitude in an epigastric area after food intake, periodic vomitings by sour gastric content. On empty a stomach by a stomach pump 200–300 ml gastric content is removed.

In the subcompensated stage muscular layer of stomach becomes thinner. Tone of him goes down, a peristalsis relaxes, and it looks like the stretched sack. Evacuation disorders is increased. Fermentation and rotting developed in stagnant gastric content. On this stage of disease development patients, usually, complain for the permanent feeling of weight in epigastric region and regurgitation with an unpleasant “rotten” smell of sulphuretted hydrogen.

Vomiting becomes systematic (once or twice on a day) up to half of liter per day. On empty a stomach it possible to aspirate from it more 500 ml of content with the food used the day before.

In the decompensation stage of the clinical signn make progress quickly. There are heavy disturbances of the general condition of patient, considerable loss of weight (to 30–40 %), acutely expressed dehydration of organism, hypoproteinemia, hypokalemia, azotemia and alkalosis. In case of the protracted neglected disease, as a result of progress of disturbances of metabolism, there can be a convulsive syndrome (gastric tetany). Vomiting in this stage not always can be considered by a typical sign, in fact patients often renounce to adopt a food, and a stomach acquires considerable sizes, overdistension form, it tone is violated and atrophy of wall comes. In such patients in an epigastric area it is possible to define the contours of the stretched stomach, with a slow peristalsis.. In the distance it is possible to hearken the splash. By a probe from a stomach to 1.5-2 litres of food with a putrid smell are removed. There can be gastric tetany at considerable disturbances of electrolyte metabolism.

A diagnosis is set according to a typical syndrome, results of sounding of stomach, rontgenoscopy, at which by contrasting of a barium expose stenosis of initial part of stomach or duodenum, determines it origin and estimate a degree.

Roentgenologically in the compensation stage stomach in normal sizes, it peristalsis deep, increased, evacuation of content proceeds no more than 6 hours. In the stage of subcompensation a stomach is megascopic, a peristalsis is loosened, evacuation stays too long to 24 hours. During decompensation a stomach is considerably extended as a sack, deformed, the waves of antiperistalsis can take place, a contrast stays too long more than 24–48 hours. The method of the double contrasting by a barium and air considerably facilitates diagnostics.

Determination of stomach motility has not only diagnostic but also prognostic value for the choice of method of operation.

In the stage of compensation motility of stomach is well-kept, often even increased. With the increasing the degree of stenosis the motility disturbance increased, up to gastroplegia.

In the biochemical blood test is marked the decline of content of albumen to 54–48 g/l; potassium — to 2,9–2,5 mmol/l; chlorides — to 85–87 mmol/l. The changes of such indexes are most expressed at patients with gastrogenous tetany.

The study of secretory function of stomach allows to define the degree of compensation of stenosis and important at the choice of adequate method of operation.

Gastroscopy with a biopsy is the enough informing method of examination of such patients. By this method is possible to determine a reason and degree of stenosis, and also state of mucous tunic of stomach.

Stenosis of the output part of stomach and duodenum of ulcerous origin it is needed to differentiate with functional gastrostasis and narrowing of tumour and chemical genesis.

Functional gastrostasis more frequent meets at women. Basic, that distinguishes it from other pathologies, there is absence of some organic changes in the area of pyloric part of stomach or in a duodenum, that can be exposed during fibergastroscopy.

Differential diagnostics of stenosis of tumour genesis, as a rule, also does not cause the special difficulties. A diagnosis is finally confirmed by histological examinations of the biopsy material taken during endoscopy.

Postburn stenosis of piloroantral area of stomach observed, from data of statistics, more than in 25 % cases of patients with the burn of esophagus. In anamnesis in each of such patients takes place by mistake or the intentionally taken an a swig at acid, alkali or other chemical matter. Some diagnostic difficulties can arise up at the isolated postburn stenosis of pyloric part of stomach. The however attentively collected anamnesis and professionally conducted endoscopic examination enable to set a correct diagnosis.

Treatment of ulcerous stenosis of piloroantral part of stomach and duodenum must be exceptionally operative. A method depend on many factors: degree of stenosis, these secretory and motility functions of stomach, age of patient, presence of accompanying diseases and others like that. In the compensated and subcompensated stages of stenosis and at enough well-kept functions of stomach it is possible to perform of organ-saving operations (vagotomy with drainage stomach operations, economy resection of stomach). At growth of the signs of stenosis and disturbance of basic functions of stomach, the volume of operation must be increased up resection by the Billroth's second method.

At the patients and older age persons with heavy accompanying pathology is performed minimum surgery —gastroenteroanastomosis.

Preoperative preparation must be strictly individual.

At patients with insignificant disturbances of gastric motor activity (stage of compensation, subcompensations) and with good level of metabolism indexes it is better to shorten preoperative preparation in time. Such patients, usually, operated on 3–4 day. Preparation before operation at patients with decompensated piloroostenosis must be directed for the correction of metabolism disturbances. Such patients must receive transfusion of liquid up to 2,5–3 l per day with content of the ions K⁺, Na⁺, Ca⁺⁺, amino acid and glucose; plasma, albumen. Twice on days performed decompensation and washing of stomach and anti-ulcerous therapy. Effective preoperative preparation in such patients requires 5–7 days, sometimes more.

The typical perforation of gastric or duodenum ulcer is strengthening of necrosis process in the area of ulcerous crater with subsequent disturbance of integrity of wall, that result to the permanent effluence of gastroduodenal content and air in a free abdominal cavity.

In 50,7 % cases perforates the ulcers of duodenum, in 42,8 % are ulcers of pyloric part of stomach, in 4,8 % are ulcers of small curvature of body of stomach and in 0,7 % are cardiac ulcers.

Ulcers, which lie on the front wall of stomach and duodenum more frequent give the perforation with general peritonitis, while ulcers on a back wall — perforation with adhesive inflammation.

The reasons of ulcers perforation are: exacerbation of peptic ulcer, harmful habits, stresses, professional, athletic overexertion, faults in the feed and abuses by strong waters.

In pathogeny of acute perforation important: progressive necrosis processes in the area of ulcerous crater with activating of virulent infection; hyperergic type of local vaculo-stromal reaction with the thrombosis of veins of stomach and duodenum; local manifestation of autoimmune conflict with accumulation of sour mucopolysaccharides on periphery of ulcer and high coefficient of plasmationization of mucous tunic (K.I. Mishkin, A.A. Frankfurt, 1971).

The clinical picture of perforation is very typical and depends on distribution of inflammatory process and infection of abdominal cavity. In clinical passing of the perforations distinguish three phases: shock, “imaginary prosperity” and peritonitis (Mondor, 1939).

For the phase of shock (to 6 hours last) typical very acute pain in epigastric region (Delafua compares it to pain from the stab with a dagger) with an irradiation in a right shoulder and collar-bone, a face is pale, with expression of strong fear, lines become (facies abdominalis) acute, a death-damp irrigates skin covers. A pulse is at first slow (vagus pulse), later becomes frequent and less filling. Sometime observed the reflex vomiting and delay of gases. Arterial pressure is reduced. On examination stomach is pulls in, does not take part in the act of breathing. At palpation is “wooden belly stomach”, especially in an upper part, where, usually, there is most pain. Positive Blumberg's sign. At percussion is disappearance of hepatic dullness (the Spizharny symptom). At rectal examination expose painful in the area of rectouterine or

rectovesical pouch (the Kulenkampf's symptom).

The phase of shock changes by the phase of "imaginary prosperity", when the reflex signs go down: the general condition of patient gets better, a pulse becomes normal, arterial pressure rises, a stomach-ache diminishes partly. However observed tension of muscles of front abdominal wall, positive Blumberg's sign.

The phase of "imaginary prosperity" in 6–12 hours from the moment of perforation changes by the phase of peritonitis: a pulse is frequent, a stomach is swollen through growing flatulence, intestinal noises are not listened, a face acquires the specific kind — facies Hippocratica — the eyes fall back, lips turn blue, a nose becomes sharp, a tongue becomes dry and furred, breathing superficial and frequent, a temperature rises.

Covered perforation (A.M. Shnieler, 1912). At this pathology the perforative hole after a perforation is closed by a fibrin, by an omentum, by the fate of liver, sometimes — piece of food. After that some amount of stomach content and air gets in an abdominal cavity. After the protection a stomach-ache diminishes, but proof tension of muscles of front abdominal wall, especially overhead quadrant of stomach is kept. At percussion hepatic dullness is doubtful. During x-Ray examination it is not always possible to mark gas in right hypocondrium (Pic. 3.2.14).

Consequences of passing of the covered perforation: the repeated perforation with development of classic clinical signs can come; at separation of process from a free abdominal cavity a subdiaphragmatic or subhepatic abscess is formed; complete closing of defect by surrounding tissue with gradual convalescence of patient.

The atypical perforation is the perforation, at which gastric or intestinal content gets not to the abdominal cavity, but in retroperitoneal space (ulcers of back wall of duodenum), large or small omentum (ulcers of small curvature of stomach), hepato-duodenal ligament.

In such patients during a perforation pain is not acutely expressed. During palpation observed insignificant rigidity of muscles of front abdominal wall. On occasion, especially on the late stages of disease, there can be hypodermic emphysema and crepitation.

The diagnosed perforated gastric and duodenal ulcer is an absolute indications to operation. Preoperative preparation must include: in I phase are antishock action; in the II and III phases — reanimation preparations, introductions of antibiotics for 2–3 hours before operation, liquidation of hypovolemia by salt blood substitutes (solution of chlorous sodium), solutions of dextran (polyhukline, reopolihukline, hemodes). Amount of liquid necessary for correction of hypovolemia, calculate after hematokrit by central vein pressure. Taking for the norm of hematokrit 40 %, on each 5 % higher norms need to be poured 1000,0 ml liquids.

Conservative treatment (method of Tejlor, 1946) can be justified at the refusal of patient from operation or in default of conditions for its implementation.

It must include:

- permanent nasogastral aspiration of gastric content;
- introduction of preparations which brake a gastric secretion (atropine, H₂- blockers and others like that);
- introduction of antibiotics;
- correction of metabolism;
- laparocentesis with drainage and closed lavage of the abdominal cavity.

In the decision of question about the choice of method of operative treatment of perforated gastroduodenal ulcers the important value has the following factors: localization of ulcer, clinico-morphological description of ulcer (perforation of acute or chronic ulcer), connected with the perforation such complications of ulcer, as bleeding, cicatricial-ulcerous stenosis, penetration, degree of risk of operation and feature of clinical situation.

Operative treatments at a perforated ulcer divide into palliative and radical.

Palliative operations are: closure of the perforative hole of ulcer, tamponade of the perforative hole by a omentum on a leg by B.A. Ooppel - P.N.Polikarpov - M.A.Pidhorbunskyy (1896, 1927, 1948) (Pic. 3.2.15). Indications and terms for their implementation are:

- perforation of acute duodenal ulcer in youth and young age without anamnesis;
- perforation of acute ulcer in the II–III phases of passing;
- perforation of callous gastric ulcer in the II–III phases of passing;
- expressed and high degrees of risk of operation.

The radical operations at perforated ulcers are: resection of stomach and excision of the perforative hole of ulcer in combination with pyloroplasty and StV, SV or SPV.

Indications and terms for implementation of resection of stomach are:

- perforation of callous gastric ulcer in I phase of clinical passing;
- repeated perforation of ulcer;
- perforation of ulcer in I phase of clinical passing in combination with stenosis and bleeding of ulcer;
- perforation of duodenal ulcer in I phase of passing in combination with a gastric ulcer;
- unexpressed and moderate degree of risk of operation;
- sufficient qualification of surgeon and material resources of operating-anaesthetic brigade.

Indications for implementation of operation of excision of the perforative hole of ulcer with pyloroplasty, StV, SV and SPV are: perforation of ulcer of front wall of duodenum or pyloric part of stomach in the I–II phases of passing;

- perforation of ulcer of front wall of duodenum in the I–II phases of passing in combination with the bleeding ulcer of back wall;
- perforation of duodenal ulcer in the I–II phases of passing in combination with the compensated stenosis of outgoing part of stomach;
- increased gastric secretion;
- insignificant and moderate degree of risk of operation;
- sufficient qualification and technical preparedness of surgeon.

Bleeding gastroduodenal ulcers are outpouring of blood in the gastrointestinal tract cavity as a result of strengthening and distribution of necrosis process in the ulcer area to vessels with the subsequent melting of their walls.

Complication of peptic or duodenal ulcer by bleeding is critical situation which threatens to life of patient and requires from the surgeon of immediate and decisive actions for clarification of reasons of bleeding and choice of tactic of treatment. The ulcerous bleeding has 60 % of the acute bleeding from the upper parts of gastrointestinal tract.

The origin of the gastrointestinal bleeding at patients with a gastric or duodenal ulcer almost is always related to exacerbation of ulcerous process. The reason of bleeding is a erosive vessel, that is on the bottom of ulcer. The expressed inflammatory and sclerotic processes round the damaged vessel embarrassed its contraction, that diminishes chances on the spontaneous stop of bleeding.

Gastric ulcers, compare with the ulcers of duodenum, complicated by bleeding more frequent. Bleeding at gastric ulcers are more expressed, profuse, with heavy passing.

At the duodenal ulcer bleeding more frequent complicate the ulcers of back wall, which penetrates in the head of pancreas.

At the men ulcer is complicated by bleeding twice more frequent, than at women. It costs to mark that 80 % patients which carried bleeding from an ulcer and treated oneself by conservative preparations, are under the permanent threat of the recurrent bleeding.

Strengthening of necrosis process are leading factors in the origin of the ulcerous bleeding in the area of ulcerous crater with distribution of this process to a vessel and subsequent melting of vascular wall; activation of fibrinolysis in tissues of stomach and duodenum; ischemia of tissues of wall of stomach.

At patients with an peptic ulcer disease, bleeding pops up, mainly at night. Vomiting can be the first sign of it, mostly, at gastric localization of ulcers. Vomiting masses, as a rule, looks like "coffee-ground". Sometimes they are as a fresh red blood or its grume.

The black tar-like emptying are the permanent symptom of the ulcerous bleeding, with an unpleasant smell ("melenä"), that can take place to a few times per days.

Bloody vomiting and emptying as "melenä" is accompanied by worsening of the general condition of patient. A acute weakness, dizziness, noise in a head and darkening in eyes, sometimes — loss of consciousness. A collapse with the signs of hemorrhagic shock can also develop. Exactly with a such clinical picture the such patients get to the hospital. It is needed to remember, that for diagnostics anamnesis is very important. Find out often, that at a patient an peptic ulcer was already diagnosed once. It appears sometimes, that bleeding is repeated or surgery concerning a perforated ulcer took place in the past. At some patients a gastric or duodenal ulcer is was not diagnosed before, the however attentively collected anamnesis exposed, that at a patient had a stomach-ache. Thus it communication with acceptance of food and seasonality is typical (more frequent appears in spring and in autumn). Patients tell, that pain in overhead part of abdomen which disturbed a few days prior to bleeding suddenly disappeared after first its displays (the Bergmann's symptom).

At patients with the ulcerous bleeding there are the typical changes of hemodynamic indexes: a pulse is frequent, weak filling and tension, arterial pressure is mostly reduced. These indexes need to be observed in a dynamics, as they can change during the short interval of time.

There is the pallor of skin and visible mucous tunics at a examination. A stomach sometimes is moderately exaggerated, but more frequent is pulled in, soft at palpation. In overhead part it is possible to notice hyperpigmental spots — tracks from the protracted application of hot-water bottle. Painful at deep palpation in the area of right hypocondrium (duodenal ulcer) or in a epigastric area (gastric ulcer) it is possible to observe at penetrated ulcers. Important symptom of Mendel also — painful at percussion in the projection of piloroduodenal area.

At the examination of patients with the gastrointestinal bleeding finger examination of rectum is obligatory. It needs to be performed at the first examination, because information about the presence of black excrement ("melenä") more frequent get according to a patient anamnesis, that can result in erroneous conclusions. Finger examination of rectum allows to expose tracks of black excrement or blood. In addition, it is sometimes possible to expose the tumour of rectum or haemorrhoidal knots which also are the source of bleeding.

The deciding value in establishment of diagnosis has the endoscopic examination. Fiber-gastroduodenoscopy enables not only to deny or confirm the presence of bleeding but also, that it is especially important, to set its reason and source. Often embarrassed the examination of stomach and duodenum present in it blood and content. In such cases it is necessary to remove blood or content, by gastric lavage, and to repeat endoscopic examination. During the examination often exposed the bleeding with fresh blood from the bottom of ulcer or ulcerous defect with one or a few erosive and thrombosed vessels (stopped bleeding). The bottom of ulcer can be covered by the package of blood.

Important information about such pathology is given by haematological indexes also. Diminishment of number of red corpuscles and haemoglobin of blood, decline of haematocrit is observed in such patients. However always needed to remember, that at first time after bleeding haematological indexes can change insignificantly. Conducting of global analysis of blood in a dynamics in every a few hours is more informing.

It is necessary always to remember that complication of peptic ulcer by bleeding happens considerably more frequent, than is diagnosed. Usually, to 50–55 % moderate bleeding (microbleeding) have the hidden passing. The massive bleeding meet considerably rarer, however almost always run across with the brightly expressed clinical signs which often carries dramatic character. In fact profuse bleeding with the loss 50–60 % to the volume of circulatory blood could stop the heart and cause the death of patient.

The clinical signs and passing of disease depend on the degree of blood (O.O. Shalimov and V.F.Saenko, 1987).

For lost of blood I degree typical there is a frequent pulse to 90–100, decline of arterial pressure to 90/60 mm Hg. The excitability of patient changes by lethargy, however clear consciousness is, breathing some frequent. After the stop of bleeding and in absent of hemorrhage compensation the expressed disturbances of circulation of blood does not observe.

At patients with the II degree of hemorrhage the general condition needs to be estimated as average. Expressed pallor of skin, sticky sweat, lethargy. Pulse — 120–130 per min., weak filling and tension, arterial pressure — 90–80/50 mm Hg. At first hours the spasm of vessels (centralization of circulation of blood) comes after bleeding, that predetermines normal or increased, arterial pressure. However, as a result of the protracted bleeding compensate mechanisms of arterial pressure are exhausted and can acutely go down at any point. Without the proper compensation of hemorrhage the such patients can survive, however

almost always there are considerable disturbances of blood circulation with disturbance of functions of liver and kidneys.

The III degree of hemorrhage characterizes heavy clinical passing. There is a pulse in such patients — 130–140 per min., and arterial pressure — from 60 to 0 mm Hg. Consciousness is almost always darkened, acutely expressed adynamy. Central vein pressure is low. Oliguria is observed, that can change by anuria. Without active and directed correction of hemorrhage a patient can die.

But, not always weight of bleeding which is conditioned by the degree of hemorrhage correspond the general condition of patient. On occasion the considerable loss of blood during the set time is accompanied by the relatively satisfactory condition of patient. And vice versa, moderate hemorrhage can bring to the considerable worsening of general condition. It can depend both on compensate possibilities of organism and from the presence of accompanying pathology.

It is needed to remember, that the ulcerous bleeding can accompanying with the perforation of ulcer. During perforation ulcers are often accompanied by bleeding. Correct diagnostics of these two complications has the important value in tactical approach and in the choice of method of surgical treatment. In fact simple suturing of perforated and bleeding ulcer can complicated in postoperative period by the profuse bleeding and cause the necessity of the repeated operation.

At wide introduction of gastroduodenoscopy of question of differential diagnostics of bleeding lost the actuality. However much a problem arises up at impossibility to execute this examination through the heavy general condition of patient or taking into account other reasons. Differential diagnostics is conducted with bleeding of ulcerous origin, which arise up in different parts of digestive tract.

For bleeding from the varicose extended veins of esophagus during portal hypertension at patients with the cirrhosis of liver the acute beginning without pain is characteristic, like during exacerbation of ulcerous disease. These bleeding differ by the special massiveness and considerable hemorrhage. Vomiting by fresh blood, expressed tachycardia, falling of arterial pressure are observed. In such patients it is possible to find the signs of cirrhosis of liver and portal hypertension (“head of jelly-fish”, hypersplenism, ascites, often is icterus).

Sliding hernia of the esophagus opening of diaphragm can be accompanied by formation of ulcers in the place of clench of the stomach by the legs of diaphragm and bleeding from them. However for this pathology are more typical microbleeding, that is hidden. In such patients often the present protracted anaemia which can achieve the critical values. Sometimes in them observe more expressed bleeding with “classic” vomiting “coffee-grounds” and melena. During the roentgenologic examination with barium is possible to expose the signs of sliding hernia of the esophagus opening: the obtuse cardial angle, absence or diminishment of gas bubble of stomach or “ringing symptom”.

The cancer tumour of stomach in the destruction stage can be also complicated by bleeding. However, such bleeding are massive, and chronic character is carried mostly with gradual growth of anaemia. For this pathology there are the inherent worsenings of the general condition of patient, loss of weight of body, decline of appetite and waiver of meat food. At the roentgenologic examination the “defect of filling” is exposed in a stomach.

The gastric bleeding can be related to the diseases of the cardio-vascular system (atherosclerosis, hypertensive disease), however such happens mainly in the older years people. Clearly, that in such patients during the endoscopic examination the source of bleeding exposing is not succeeded.

Among other diseases, with which it is necessary to differentiate the ulcerous bleeding, it is needed to remember the Mallory-Weiss syndrome, benign tumours of stomach and duodenum (more frequent leiomyoma), hemorrhagic gastritis, acute (stress) erosive defects of stomach, arteriovenous fistula of mucous tunic.

Often differential diagnostics performed according to the level of localization of source of bleeding in different parts of gastrointestinal tract. For the upper parts of digestive tract (esophagus and stomach) typical there is vomiting by grume or “coffee-grounds” content and emptying by “melena”. The farther aboral placed source of bleeding, the bloody emptying changes the more so. During the bleeding from a thin bowel excrement looks as “melena”. In case of such pathology of colon (polypuses, tumours, unspecific ulcerous colitis) emptying have the appearance of fresh red blood, mostly as packages.

The conservative therapy indicated to patients with the stopped bleeding of I degree and bleeding of the II–III degrees at patients which have heavy accompanying pathology, because of operative risk.

Conservative therapy must include:

- prescription of hemostatic preparations (intravenously the aminocaproic acid 5 % — 200–400 ml, chlorous calcium 10 % — 10,0 ml, vicasol 1 % — 3,0 ml);
- addition to the volume of circulatory blood (gelatin, poliglukine, salt blood substitutes);
- preparations of blood (fibrinogen — 2–3 r, cryoprecipitate);
- blood substitutes therapy (red corpuscles mass, washed red corpuscles, plasma of blood);
- antiulcerous preparations — blocker of H₂-receptor (ranitidine, roxatidine, nasatidine— for 150 mg 1–2 times per days);
- antacid and adsorbents (almagel, phosphalugel, maalox— for 1–2 dessert-spoons through 1 hour after food intake).

It is expedient to apply washing of stomach by water with ice and the use 5 % solution of aminocaproic acid inward for to a 1 soupsoon in every 20–30 minutes.

The endoscopic methods of stop of bleeding are used also. Among them most effective is a laser and electro-coagulation.

Absolute indications to surgical treatment are: 1) lasting bleeding I degree; 2) recurrent bleeding after hemorrhage I degree; 3) bleeding of the II–III degrees; 4) stopped bleeding with hemorrhage of the II–III degrees at the endoscopically exposed ulcerous defect with a presence on the ulcer bottom thrombosed vessels or erosive vessels covered by the package of blood.

The choice of method of surgical treatment always needs to be decided individually. On today the best tactic which gives advantage to organ-saving and organsparing methods of operations. The removing ulcer as sources of bleeding must be an obligatory condition. The methods of sewing of bleeding vessels or edging of ulcer and bandaging of vessels which feed a stomach and duodenum did not justify itself through the real threat of relapse of bleeding already in an early postoperative period (9–12 days).

Palliative operations (cutting of ulcer, forming of roundabout anastomosis) can be justified only taking into account the general condition of patient and on a necessity as possible quick and least traumatically to make off operation.

At the bleeding ulcers of duodenum it is better to apply excision of ulcer or it enteritization after methods, developed by V.Zajtsev and Velihotsky. Operation complemented by one of types of vagotomy, it is better by a selective proximal with piloroplastic. The resection of stomach on the second or first method of Biltroth can be realized only in the stable general condition of patient. During the resection of stomach in case of low bleeding duodenal ulcers it is better to execute mobilization of duodenum and suturing of its stump on transcholedoch drainage which formed as transcholedoch duodenotomy (Lapey, 1942). This method warns the possible intraoperative damages of choledoch, that are the possible at low duodenal ulcers. Transcholedoch duodenotomy by performing the decompression of stump of duodenum, warns insufficiency of its stitches, that can arise up in an early postoperative period.

In case of bleeding gastric ulcers, the resection methods of operations are also usable. Only on occasion, when patients has the grave general condition, it is possible to assume the wedge cutting of ulcer.

The origin of acute linear breaks of mucous tunic of esophagus and cardial part of stomach, which are accompanied by bleeding of a different degree of weight to the gastrointestinal tract lumen, is named the Mallory-Weiss Syndrome. First described by K. Mallory and C. Weiss in 1929. As the reason of the gastroduodenal bleeding observed in 10 % cases. Men are ill mainly in age 30–50 years.

The predetermining factors of origin of syndrome are: protracted whooping, attacks of cough, physical overstrain after the surplus food intake, alcohol with vomiting, chronic diseases of stomach, with the acute increase of intragastric pressure as a result of disordinated function of cardial and pyloric sphincter, especially at older patients with atrophy gastritis. The increase of intragastric pressure causes change of blood flow in the wall of the stretched stomach. Spontaneous break of mucous tunic of cardial part of stomach, is accompanied by bleeding in the gastrointestinal tract lumen. The break takes not only mucous tunic but also muscular layer, that weight of bleeding is predetermined. Most often the breaks are localized on small curvature, on the back wall of stomach and esophagus.

The main symptom of syndrome is “bloody” vomiting which the dyspeptic signs preceded: nausea and “unbloody” vomiting. Sometimes patients complain for pain in a epigastric area, in the lower part of thorax, which is related to sudden cardial and lower part of esophagus distension.

Weight of bleeding depends on length and depth of breaks and caliber of the damaged vessels. In one case at first the some dark blood is excreted and only at the repeated vomiting is a lot of bright red blood. In other case at once there is vomiting by a bright red blood. Sometimes bleeding looked as the tar-like emptying. The degree of hemorrhage and its weight is determined after the generally accepted chart.

Taking into account that a syndrome arises up after acceptance of a plenty of alcohol and food, the clinical forms of passing are distinguished: simple, delirious, with the signs of acute hepatic insufficiency, without the signs of acute hepatic insufficiency, that matters very much for the choice of medical tactic.

Urgent esophagogastrosopy is the basic method of diagnostics of syndrome. During it in the cardial part of stomach or esophagus single or plural fissures are diagnosed by length 0,5–4,0 cm, by width 0,5–0,8 cm which pass longitudinally, bleeding. The edges of mucus round a fissures swelled, elevated, covered by a fibrin. Often the muscular layer of stomach or esophagus is the bottom of fissure.

Conservative treatment of the Mallory-Weiss syndrome is indicated at the small rupture of mucus stomach, to the stop of bleeding, absence of bleeding. Treatment of patients is begun with active conservative therapy, which includes blood transfusion, infusion of hemostatic, application of antacid, Meulengracht's diet. At the rupture of the I–II degrees indicated endoscopy by a monopolar electrocoagulation of the fissure and covering of aerosol film-forming preparation Lifusol. The conservative method of stop of bleeding in such patients is especially perspective, because most of them has the delirious state or acute hepatic insufficiency.

Operative treatment is indicated at the deep large ruptures of mucus and muscular layers, cardial part of stomach, which are complicated by bleeding. In such cases conduct gastrotomy and suturing of ruptures by interrupted suture or 8-shaped stitch, applying nonabsorbable filaments. Sewings of ruptures of mucus stomach often supplement with vagotomy with pyloroplasty. At deep, especially plural ruptures which are accompanied by the edema of tissues, sewing of ruptures is supplement with bandaging of left gastric artery.

Hemorrhagic erosive gastritis is diffuse bleeding from mucous tunic stomach as a result of single or plural superficial defects (erosions) of mucous tunic. The gastrointestinal bleeding during erosive gastritis meet in a clinic in 13–17 % cases of acute hemorrhage in a gastrointestinal tract and take first place among bleeding of ulcerous etiology. The disease is met both at men and at women, but more frequent observe in declining years.

The spasm of large vessels in the deep layers of gastric wall, which results in disturbance of local microcirculation, hypoxia and increases of permeability of vascular wall, matters in etiology and pathogenesis of hemorrhage erosive gastritis. The local reaction causes strengthening of reverse diffusion of hydrogen ions, liberation of pepsin, histamine. Such process often is consequence of local damaging factor — action of medicinal or toxic factors for the vessels of mucus. Damaging factor could be the matters which violate a blood flow in mucus stomach (aspirin, reserpine, hormones of adrenal glands cortex). The large value in formation of erosions is had by the anatomic features of blood flow of stomach in a cardial part on small curvature. In connection with absence of submucosal vascular plexus, eventual vessels on small curvature are disposed in relation to mucus tangentially. It results in shelling of epithelium, origin of erosions. Veins damaged at first, that predetermines a hemorrhage and then bleeding. In the origin of acute hemorrhage gastritis matter also acute damage of mucus stomach by mechanical, chemical (burns) and other factors, accompanying diseases (uremia and others like that).

For hemorrhage erosive gastritis there are typical two clinical syndromes: ulcerous and hemorrhagic. The ulcerous syndrome is the most frequent sign of hemorrhage gastritis. “Typical ulcerous pain” is observed in such patients. A hemorrhagic syndrome shows up by the repeated gastric bleeding and moderately increasing anaemia. Bleeding are capillary and are not such catastrophic, as at gastric ulcers.

The clinical picture of hemorrhage gastritis is characterized by dull pain in a epigastric area, which appears at faults in a food, reception of alcohol. Patients disturbs vomiting like “coffee-grounds”, “melena”, which arise up among a complete health, symptoms of hemorrhage (dizziness, general weakness, acceleration of pulse, decline of arterial pressure). The decline of amount of red corpuscles is observed in the blood test, haemoglobin, haemathokrits, leukocytosis. During the roentgenologic examination observed the thickened winding folds of mucus stomach with the small depots of barium. At endoscopic diagnostics of bleeding the presence of single or plural erosions on mucus up to 5–7 mm in diameter are noticed, symptom of “morning dew” (“weeps” all mucus stomach).

Treatment of hemorrhage erosive gastritis, mainly, is conservative. Washing of stomach an effective by cold water or by 5 % solution of aminocaproic acid with subsequent irrigation of mucous tunic by film-forming preparations through endoscope and introduction of hemostatic. It is important the neutralization of hydrochloric acid in a stomach (antacid, additional introduction of atropine of sulfate, aspiration of gastric content), setting of preparations which stimulate reparative processes in a mucous tunic (methyluracil, sayotek, sea-buckthorn oil), antihelicoacter preparation (de-nol). If under the endoscopy control effect from conservative treatment is absent and it is the obvious threat of life of the patients, operative treatment is indicated.

Surgical treatment must be minimum. Sewing and edging of bleeding areas, selective vagotomy with pyloroplasty in most cases is effective. Only at bleeding from arising acute erosions after submucosal telangiectasia, indicated resection of stomach. It is needed to remember, that the additional focus of bleeding can be in fundal and cardial part of stomach. Without their edging and local hemostasis operation can not be radical. At the considerable damage of stomach by an erosive process, for a patient indicated resection of stomach or gastrectomy.

The hereditary hemorrhagic telangiectasia, Rendu-Osler-Weber Disease — hemorrhage angiopathy, which is characterized by focus microvascular expansion by the type of telangiectasia and angiomas with the break of which possible bleeding. Meets rarely, inherited after a autosomal-dominant type, sometimes arises up sporadically.

Teleangiectasia and angiomata develops as a result of thinning and expansion of shallow vessels. At the same time local hemostasis is violated as a result of hypoplasia of subendothelium and collagen deficiency. Bleeding is related to small resistance and easy vulnerability of vascular wall, by weak stimulation in these areas of aggregation of thrombocyte and blood coagulation. Teleangiectasia is disposed on the mucous tunics of mouth cavity, rarer is mucus of the trachea, bronchial tubes, gastrointestinal tract, urinary bladder and liver.

The disease is characterized by the frequent nose-bleeds which appear in early child's age, teleangiectasias and angiomata with certain localization. The gastrointestinal bleeding can be profuse and result in lethal termination, chronic, with the expressed anaemia. At differential diagnostics of bleeding it is necessary to observe the skin and mucous tunics. Teleangiectasias and angiomata is characterized by expansion of granulomatous vessels and is disposed, mainly, on a head, skin, mucus of the mouth, nose, on hands, finger-tips, genital organs.

Treatment of the Rendu-Osler-Weber disease is conservative, symptomatic, including hemostatic therapy. There is the indicated blood transfusion at considerable hemorrhage. Often during recrudescence and profuse gastrointestinal bleeding the resection of stomach is indicated. It is represents a problem for a surgeon, because teleangiectasias after the decline of arterial pressure, become pale and unnoticeable. A prognosis is often unfavorable, because not always it is possible to expose teleangiectasias in other organs.

Menetrier syndrome is pseudotumor gastritis. The disease rarely. Etiology and pathogenesis is unknown. During disease observed the increasing of folds of mucus stomach by the height up to 3 cm and thickness up to 2 cm. Deep cracks, which the massive bleeding are from, appear between folds. Diagnostics of bleeding is confirmed by endoscopy. Treatment is conservative, including the hemostatic therapy. If conservative treatment are ineffective, indicated resection of stomach. During operation round a stomach the megascopic lymphatic knots with soft consistency are observed.

Hemobilia is bleeding from biliary ways and liver to the intestine. Meets in 0,01 % all gastric bleeding of ulcerous genesis.

The most frequent reason of hemobilia is the traumas of liver. Among other reasons are inflammatory processes of liver, external biliary ways (abscesses, cholangitis), vascular anomalies as aneurism of hepatic artery and vein gate.

The typical signs of hemobilia: attack-like pain in right hypochondrium, moderate icterus, anaemia, presence of grume in vomiting masses and in the excrement which looks like a pencil or worm (imprints of biliary ducts). Bleeding have cyclic passing (repeat oneself in 6–8 days). A diagnosis is based on the clinical signs, information of endoscopy, at which founded the blood flow to the duodenum from a general biliary duct or bloody clot in the papilla Vateri. The most diagnostic value has selective angiography of the hepatic artery and cholangiography, which allow to expose the flowline of contrasting matter in tissues of liver.

Bleeding from biliary tracts during the damage of large vessels can be severe. So, operation is the unique treatment method in such cases. In patients with hemobilia performed opening, draining and tamponade of the haematomas with obligatory draining of general biliary channel for decompression of biliary tracts. The most radical method some surgeons count opening of haematoma with bandaging of bleeding vessel and biliary channel or resection of liver. Bandaging of hepatic artery after angiographic study of the intraorgan arterial vessels is sometimes recommended only. Better to bandage that branch of hepatic artery from which observed bleeding.

The particle of the rare extragastric diseases complicated by the acute gastrointestinal bleeding is 2 %. Among them the diseases of blood are met, blood vessels, system diseases (leukosis, haemophilia, autoimmune thrombocytopenia, hemorrhagic vasculitis, the Werlhof's disease and others like that).

Leukosis are tumours which developed from hemopoietic cells. Etiology and pathogenesis to this time is not exposed. Patients with a leukosis with the gastrointestinal bleeding is 1 % of all patients with the un ulcerous bleeding.

During leukosis in the process of extramedullary hematosis the cells of vascular wall and vessel are pulled in and from the circulatory changed into hemopoietic, that results in disturbance of permeability of vessel wall. In development of hemorrhage diathesis large part is acted the changes of thrombocytopenia, declines of growth of tissue's basophiles, which produce heparin, that shows up by wide hemorrhages in a gastrointestinal tract. Bleeding can be both insignificant and threatening to life of patient. In establishment of diagnosis sometimes there is enough simple examination of blood (hyperleukocytosis), to suspect leukosis bleeding. During endoscopy in such patients observe the presence of flat, superficial defects of mucus stomach. A final diagnosis is based on the results of biopsy and haematological examination of bone marrow.

Treatment includes complex application of hemostatic, preparations of blood and cytostatic agents, that results in the stop of bleeding and even to bring a patient into remission.

Haemophilia is the innate form of bleeding which caused by the deficit of one of three antihemophilic factors (VIII, IX, XI). The gastrointestinal bleeding is observed in 6–24 % patients with haemophilia. Absence or insufficient content in the blood of antihemophilic globulin lies in basis of disease. At diminishment of it level below 30 % there is bleeding. Haemophilia is inherited, men are ill more frequent.

Pointing in anamnesis on bleeding from babyhood allow to suspect haemophilia. Roentgenologic information and results of fibergastroscopy does not expose the substantial changes in a gastrointestinal tract. Main in diagnostics of haemophilia — examination of the system of blood coagulation. Time of blood coagulation continued to 10–30 minutes, sometimes a blood does not coagulate by hours.

Treatment is directed on compensation of insufficient components of the of blood coagulation system. In patients with haemophilia A, for which typical deficit of antihemophilic globulin, fresh blood transfusion is indicated, because in a banked blood a antihemophilic globulin collapses during a few hours. At haemophilia B and C are used dry and native plasma, cryoprecipitate, banked blood, because factors IX, XI, which predetermine the form of haemophilia, is kept in them long. ordinary hemostyptic preparation (vicasol, the C vitamin, chloride of calcium and others like that) does not give the effect. So, if form of haemophilia does not established, the treatment is necessary to begin from fresh blood transfusion, antihemophilic plasma and antihemophilic globulin transfusion.

Autoimmune thrombocytopenia, or idiopathic thrombocytopenic purpura, is accompanied by the gastrointestinal bleeding and is arisen up in 0,5–2 % patients. Often bloody vomiting and black excrement conditioned by swallowing of blood from a nose and gums.

The disease shows up by plural hypodermic hemorrhages and hemorrhages into submucous membrane. At girls and women the uterine bleeding is often observed. Thrombocytopenia on very low numbers and it is the most pathognomonic sign of disease. Typical acute increase of duration of bleeding, especially in the period of acute hemorrhage.

Fresh blood and thrombocyte mass transfusion is the most effective treatment in the case of the gastrointestinal bleeding during autoimmune thrombocytopenia. Other hemostatic preparations are indicated also. During operative treatment performed splenectomy. The absolute indications to it are frequent and protracted bleeding, threat of hemorrhage in a brain.

The Schonlein-Henoch disease is hemorrhagic vasculitis, which caused by plural microfocus microthrombovasculitis. The gastrointestinal bleeding at the Schonlein-Henoch disease is observed in 0,5–1 % cases and accompanied with great pain in a epigastric area like "abdominal colic". For this disease typical presence of purpura which has the symmetric location on the external surface of feet, legs, shoulders, buttocks, also joint syndrome with pain and edema in large joints, kidney syndrome by the type of acute or chronic glomerulonephritis. Women have the possible uterine bleeding. The intestinal bleeding can be accompanied by the edema of wall of intestine, that results in invagination or perforation of wall of bowel.

The basic and pathogenetic treatment method of patients is early application of heparin with blood transfusion, introduction of heparinized blood under the control of blood coagulation, which after adequate therapy must be increased in two times, comparative with a norm. For a patient in the initial form of disease indicated introduction of antibiotics of wide spectrum of action, hormones of adrenal glands cortex.

The diseases of the operated stomach (postgastroectomy and postvagotomy syndromes) are the diseases which arise up after surgical treatment of peptic or duodenum ulcer or other pathology of these organs.

Dumping syndrome is frequent complication of operations which are related to deleting or disturbance of function of goalkeeper (resection of stomach, vagotomy with antrectomy, vagotomy with drainage operations). It takes place in 10–30 % patients.

The rapid receipt (dumping) is considered the starting mechanism of dumping syndrome. During this concentrated, mainly carbohydrate, food passed from a stomach in an empty bowel.

In the phase changes of motility of thin bowel during dumping syndrome important part is acted by the hormones of thin bowel. In endocrine cells of APUD-системи on during dumping-syndrome observed degradation and presence of hormones of mothiline, neurotensin and enteroglucagon.

The inadequate mechanical, chemical and osmotic irritation of mucous tunic of thin bowel by chymus results for the acute increase of blood flow in a bowel. The last is accompanied by the considerable redistribution of blood, especially in heavy case of dumping syndrome: blood supply of head, lower extremities is diminishes, a blood flow in a liver is multiplied.

The numeral examinations resulted in creation of osmotic theory — the principal reason of dumping syndrome is the decline of volume of circulatory plasma as a result of coming a plenty of liquid into the lumen of thin bowel from an of circulatory system and intercellular space.

For the clinical finding of dumping syndrome typical there is the origin of attacks of general weakness during acceptance of food or during the first 15–20 minutes after it. The attack begins from feeling of plenitude in a epigastric area and is accompanied by the unpleasant feeling of heat, that "spills" in the overhead half of trunk or on all body. Thus is acutely multiplied sweating. Then there is a fatigue, appear somnolence, dizziness, noise in ears, shaking of extremities and worsening of sight. These signs sometimes achieve such intensity, that patients forced to lie down. Loss of consciousness could be in the first months after operation. The attacks are accompanied by tachycardia, sometimes by the shortness of breath, headache, paresthesia of upper and lower extremities, polyuria and vasomotor rhinitis. At the end of attack or after it patients often notice grumbling in a stomach and diarrhea.

A milk or carbohydrate food is the most frequent provoking factor of dumping syndrome. In a period between the attacks patients complain about rapid fatigueability, weakening of memory, decline of working capacity, change of mood, irritates, apathy. During roentgenologic examination after 5–15 minutes observed the increased evacuation of barium mixture through anastomosis by a wide continuous stream, expansion of efferent loop and rapid advancement of contrasting matter in the distal parts of thin bowel (Pic. 3.2.16).

By the expression of symptoms dumping syndrome is divided into three degrees of weight:

I degree is easy. Patients have the periodic attacks of weakness with dizziness, nausea, that appear after the use of carbohydrates and milk food and last no more than 15–20 min. During the attack a pulse becomes more frequent on 10–15 per min., arterial pressure rises or sometimes goes down on 1.3–2 KPa (10–15 mm Hg), the volume of circulatory blood diminishes on 200–300 ml. The deficit of mass of body of patient does not exceed 5 kg. A working capacity is well-kept. Medicinal and dietary treatment gives a good effect.

II degree — middle weight. Attacks of weakness with dizziness, pain in the region of heart, hyperhidrosis, diarrhea. Such signs last, usually, 20–40 min., arise up after the use of ordinary portions of some food. During such state a pulse becomes more frequent on 20–30 per min., arterial pressure is rises (sometimes goes down) on 2–2,7 KPa (15–20 mm Hg), the volume of circulatory blood diminishes on 300–500 ml. The deficit of mass of body of patient achieves 5–10 kg. A working capacity is reduced. Conservative treatment sometimes has a positive effect, but brief.

The III degree is hard. Patients are disturbed by the permanent, acutely expressed attacks with the collaptoid state, by a fainting fit, by diarrhea, which do not depend on character and amount of the accepted food and last about 1 hour. During the attack is multiplied frequency of pulse on 20–30 per 1 min; arterial pressure goes down on 2,7–4 KPa (20–30 mm Hg), the volume of circulatory blood diminishes more than on 500 ml. The deficit of mass of body exceeds 10 kg. Patients, as a rule, are disabled. Conservative treatment is ineffective.

The problem of treatment of patients with dumping syndrome is not easy. Before the surgical treatment, as a rule, must precede conservative. Patients with the disease of easy and middle degrees respond to conservative treatment, mainly with an enough quite good effect. At the heavy degree of disease such treatment more frequent serves as only preparation to operative treatment. If a patient does not give a consent for operation or at presence of contra-indications to operative treatment (disease of heart, livers, kidneys), conservative therapy is also applied. Such treatment must include dietotherapy, blood and plasma transfusion, correction of metabolism, hormonal preparations, symptomatic therapy, electro-stimulation of motility function of digestive tract.

The dietotherapy: using of high-calorie, various food rich in squirrel, by vitamins, by mineral salts, with normal content of fats and exception from the ration of carbohydrates which are easily assimilation (limitation of sugar, sweet drinks, honey, jam, pastry wares, kissel and fruit compotes). All it is needed to use by small portions (5–6 times per days). If the signs of dumping syndrome appear after a food, such patients it is needed to lie down and be in horizontal position not less than 1 hour. At the heavy degree of dumping syndrome patients need to eat slowly, desirably lying on left. Such position creates the best terms for evacuation of food from a stomach. Thus recommend also to repudiate from too hot and cold foods.

Medicinal treatment must include sedative, replaceable, antiserotonin, hormonal and vitamin therapy. The indications to operative treatment of patients with dumping syndrome are: heavy passing of disease, combination of dumping syndrome of middle degree with other postgastroectomy syndromes (with the syndrome of efferent loop, hypoglycemic syndrome and progressive exhaustion) and ineffective of conservative treatment of the dumping syndrome of middle degree. Most methods of operative treatment of dumping syndrome are directed on renewal of natural way of passing of food on a stomach and intestine, improvement of reservoir function of stomach and providing of proportioning receipt of food in a thin bowel.

Depending on reasons and mechanisms of development of dumping syndrome there are different methods of the repeated reconstructive operations. All of them can be divided into four basic groups: I. Operations which slow evacuation from stump of stomach. II. Redoudenization. III. Redoudenization with deceleration of evacuation from stump of stomach. IV. Operations on a thin bowel and its nerves.

Basic stages of reconstructive operations: 1) disconnection of adhesions in an abdominal cavity, releasing of gastrointestinal and interintestinal anastomosis and stump of duodenum; 2) cutting or resection of efferent and afferent loops; 3) renewal of continuity of upper part of digestive tract.

For correction of the accompany postgastroectomy pathology it is better to apply combined anti- (iso-) peristaltic gastrojejunoplasty. Thus transplant by length 20–22 cm, located between a stomach and duodenum, must consist of two parts: antiperistaltic (7–8 cm), connected with a stomach, and isoperistaltic, connected with a duodenum. An antiperistaltic segment brakes dumping of stomach stump, and isoperistaltic — hinders the reflux of duodenum content.

The attacks of weakness at a hypoglycemic syndrome arise up as a result of decline of content of sugar in a blood. It is accompanied by a acute muscular weakness, by headache, by falling of arterial pressure, by feeling of hunger and even by the loss of consciousness. It is needed to remember, that at this pathology, unlike dumping-syndrome, acceptance of food especially sweet facilitates the state of patient. However in some patients both syndromes unite and the attacks of weakness can arise up as directly after food intake, so in a few hours after it. In patients with such pathology the best results are got after antiperistaltic gastrojejunoplasty (Fink, 1976).

The postgastroectomy (agastric) asthenia arises up as a result of disturbance of digestive function of stomach, pancreas, liver and thin bowel.

In patients with such pathology stump of stomach almost fully loses ability to digest a food. It is related to the small capacity of stump and rapid evacuation of food from it, and also with the acute decline of production of hydrochloric acid and pepsin. In the mucous tunics of stump of stomach, duodenum and thin bowels as a result of fall of trophic role of gastrin and other hormones of digestive tract there are the progressive atrophy changes. Absence in gastric juice of free hydrochloric acid is the reason of acute diminishment of digestive ability of gastric juice and decline of it bactericidal. Such situation is assist in advancement to ascending direction of virulent flora, to development duodenitis, hepatitis, cholecystitis, dysbacteriosis, hypovitaminosis and decline of antitoxic function of liver. All it results in acute disturbance of evacuation from a stomach.

The clinical signs of postgastroectomy asthenia arise up after a some latent period which can last from a few months to some years. During this period patients often complain for a general weakness and bad appetite. The basic symptoms of postgastroectomy asthenia are: general weakness, edemata, acute weight loss, diarrhea, skin and endocrine abnormalities. The postgastroectomy asthenia more frequent meets at men at 40–50 years. In most cases diarrhea is the first symptom of disease, that can arise up in 2 months after operation. Diarrhea, usually, has permanent character and sometimes becomes profuse.

Weight loss appears too early, the deficit of mass of body achieves 20–30 kg. A patient quickly loses forces.

Conservative treatment is the blood, plasma and albumen transfusions. These preparations are prescribed 2–3 times per a week. Correction of disturbances of electrolyte exchange is conducted at the same time (transfusion of solutions to potassium, calcium and others like that). For the improvement of processes of albumen synthesis anabolic hormones are prescribed.

Operative treatment foresees the inclusion in the digestion process of duodenum, increase of capacity of stump of stomach and deceleration of evacuation of its content.

The afferent loop consists of part of duodenum, that stopped behind after a resection, area of empty bowel between a duodenojejunal fold and stump of stomach. The syndrome of afferent loop can arise up after the resection of stomach after the Billroth-II method. Violation of evacuation from an afferent loop and vomiting by a bile are its basic signs.

Acute and chronic obstruction of afferent loop are distinguished. The reason of acute obstruction is mechanical factors: postoperative commissure, volvulus, internal hernia, invagination, jamming behind mesentery of loop of bowel and stenosis of anastomosis.

Frequency of origin of sharp obstruction of afferent loop hesitates within the limits of 0,5–2 %. The disease can arise up in any time after operation: in a few days or a few years.

Chronic obstruction of afferent loop (actually syndrome of afferent loop), as well as acute, can arise up in any time after operation, however more often it develop after the resection of stomach with gastroenteroanastomosis on a long loop, especially when operation is performed without entero-enteroanastomosis by Brown.

The etiologic factors of syndrome of afferent loop are divided into two groups: 1) mechanical (postoperative commissure, invagination, disturbance of evacuation on a afferent loop, wrong location of afferent loop, very long afferent loop, fall of mucous tunic of afferent loop into a stomach); 2) functional (hypertensive dyskinesia of bilious ways and duodenum, damage and irritation of trunks of vagus nerves, hypotensive and spastic states of upper part of digestive tract, heightened secretion of bile and juice of pancreas under act of secretin and cholecystokinin).

For the clinical picture of acute obstruction typical is permanent, with a tendency to strengthening, pain in a epigastric area or in right hypochondrium, nausea and vomiting. At complete obstruction a bile in vomiting masses is absent. The general condition of patient progressively gets worse, the temperature of body rises, leukocytosis grows, tachycardia grows. At the objective examination painful and tension of muscles of abdominal wall is observed. In a epigastric area it is often possible to palpate tumular lump. Possible cases, when the increase of pressure in a bowel is passed on bilious ways and channels of pancreas. There can be pain and icterus in such patients. There are necrosis and perforation of duodenum with development of peritonitis during further progress of process. Acute obstruction of afferent loop in an early postoperative period can be the reason of insufficiency of stump of duodenum also.

During the roentgenologic examination of organs of abdominal cavity it is visible round form area of darkening and extended, filled by gas, bowels loop.

Patients, usually, complain for feeling of weight in a epigastric area and arching in right hypochondrium, that arises in 10–15 min. after acceptance of food and gradually grows. Together with that, appear nausea, bitter taste in a mouth, heartburn. Then there is increasing pain in a right to epigastric area. During this pain arises intensive, sometimes repeated vomiting by a bile, after which the all symptoms disappear. It could be after certain kind of food (milk, fats) or its big amount. Very rarely vomiting by bile unconnected with the feed. In heavy case patients lose up to 1 liter of bile with vomiting masses. During the objective examination observed subicteritiousness of the sclera, sign of dehydration of organism (decline of turgor of skin, dry tongue, oliguria, concentrated urine). Emptying is irregular, grey color, with considerable content of undigested fat and muscular fibres. Anaemia can develop at heavy passing of disease.

Distinguished easy, middle and heavy degrees of afferent loop syndrome. In patients with the easy degree of disease vomiting is 1–2 times per a month, and insignificant regurgitation arise up through 20 min – 2 hour after a food, more frequent after the use of milk or sweet food. At middle degree of afferent loop syndrome such attacks repeat 2–3 times per week, patients are disturbed by the considerably expressed pain syndrome, and with vomiting up to 200–300 ml of bile is lost. For a heavy degree the daily attacks of pain are typical, that is accompanied by vomiting by a bile (up to 500 ml and more).

A roentgenologic examination of the patients with the afferent loop syndrome is unspecific. Neither the passing of contrasting matter nor absence of filling of afferent loop can be considered as pathognomic signs of syndrome of afferent loop.

Treatment of acute obstruction of afferent loop is mainly operative. Essence of it is the removal of barriers of evacuation of content from an afferent loop. Adhesions are dissected, volvulus is straightened, invagination or internal hernia is liquidated. For the improvement of evacuation between afferent and efferent loops performs the entero-enteroanastomosis type “end-to-end” or after the Roux method.

Conservative treatment of syndrome of afferent loop is ineffective and, mainly, is mean the removal of hypoproteinemia and anaemia, spasmolytic preparations and vitamin are appointed. With this purpose a blood, plasma and glucose is poured with insulin, a novocaine lumbar blockade and blockade of neck-pectoral knot, washing of stomach is also done.

All operative methods of treatment of afferent loop syndrome can be divided into three groups:

I. Operations, that will liquidate the bends of afferent loop or shorten it.

II. Drainage operations.

III. Reconstructive operations.

The operations of the first group, directed on the removal of bends and invagination of afferent loop, can not be considered as radical. They need to be performed only at the grave general condition of patient.

The widest application in clinical practice at the syndrome of afferent loop has the operation offered by Roux (Pic. 3.2.17).

For the prophylaxis of afferent loop syndrome it is necessary to watch after correct imposition of anastomosis during the resection of stomach: to use for the gastroenteroanastomosis short loop of thin bowel (6–8 cm from the Treits ligament) for imposition, to sew afferent loop to small curvature for creation of spur, to fix reliably stump of stomach in peritoneum of transverse colon.

The origin of reflux after the distal resection of stomach is conditioned by some factors:

I. Traumatic factors: 1) traction of stomach during operation as reason of sprain of ligament of proximal part of stomach and mobilization of large curvature of stomach; 2) cutting of vessels of stomach and oblique muscles of it wall, in particular on small curvature; 3) vagotomy, that is accompanied by cutting of phrenico-esophageal and gastrophrenic ligaments; 4) imposition of gastrointestinal anastomosis, especially direct gastroduodenoanastomosis by Billroth-I, that results in smoothing of the Hisa corner; 5) frequent aspiration of gastric content in a postoperative period, that causes superficial esophagitis.

II. Trophic factors: 1) damage of vessels which are the reason of ischemia in the area of esophago-gastric connection, and thrombophlebitis of cardiac part of stomach; 2) disturbance of influencing of neurohumoral factors which take part in innervations of esophagus; 3) disturbance of trophism of diaphragm as a result of hypoproteinemia and weight loss; 4) ulcerous diathesis and megascopic volume of gastric secretion (especially nightly); 5) regurgitation of alkaline content of duodenum in stump of stomach which reduces tone of it muscular shell.

III. Mechanical factors: 1) gastric stasis; 2) diminishment of volume of gastric reservoir, that is accompanied by the increase of intragastric pressure.

The clinical picture of gastroesophageal reflux is conditioned by the mechanical and chemical irritations of esophagus by content of stomach or thin bowel. As a result, there is esophagitis, which can be catarrhal, erosive or ulcerous-necrotic. The symptoms of reflux are very various and can simulate different diseases of both pectoral and abdominal cavity organs.

The basic complaint of patients with this pathology is a smart behind a breastbone, especially in the area of the its lower part. It, usually, spreads upwards and can be accompanied by considerable salivation. Strengthening of pain at inclinations of trunk gave to the French authors an occasion to name this sign the “symptom of laces”. Unendurable heartburn is the second complaint, that arises up approximately in 1–2 hours after the food intake. Patients forced often to drink, somehow to decrease the unpleasant feelings, however this, certainly, does not bring them facilitation. Some of them, in addition, complain for bitter taste in a mouth.

Pain behind a breastbone often can remind the attack of stenocardia with typical irradiation. Sometimes such reflux is able to provoke real stenocardia.

Hypochromic anaemia is the frequent symptom of gastroesophageal reflux too.

The diagnosis of gastroesophageal reflux, mainly, is based on clinical information, results of roentgenologic examination, esophagoscopy.

The edema, hyperemia of mucous tunic of esophagus, easy bleeding and vulnerability it during examination, surplus of mucus and erosions covered by fibrin tape is considered the endoscopic signs of esophagitis. In doubtful case at the insignificantly expressed macroscopic changes the biopsy of mucous tunic helps to set a diagnosis.

Treatment of patients with gastroesophageal reflux is mainly conservative. Very important is diet, which avoid spicy, rough and hot food. Eating is needed often, by small portions. It is impossible also to lie down after the food intake, because the gastric content can flow in a esophagus. A supper must be not later than for 3–4 hours before sleep. Between the reception of food does not recommend to use a liquid. Next to that, it is necessary to remove factors which promote intraperitoneal pressure (carrying to the bracer, belt, constipation, flatulence). Sleeping is needed in position with a lift head and trunk. From medicinal preparations it is useful to recommend enveloping preparation.

Operative treatment of gastroesophageal reflux, that arose up after the distal resection of stomach, it is needed to recommend to the patients with the protracted passing and ineffective of conservative treatment. During operation, mainly, performed renewal of the broken Hisa angle. In addition, performed esophagoplasty, fundoplication by Nessen's and esophagofrenofundoplication.

The prophylaxis of this complication consists in the study of the state of cardiac part of stomach before and during every resection and fixing of bottom of stomach to the diaphragm and abdominal part of esophagus during leveling the Hisa angle.

Alkaline reflux-gastritis meets in 5–35 % operated patients after the resection of stomach, antrectomy, gastroenterostomy, vagotomy with pyloroplasty, and also cholecystectomy and papillosphincteroplasty.

The reason of this complication is influence of duodenum content for the mucous tunic of stomach (bilious acids, enzymes of pancreas and isolecithin). Last, forming from bile lecithin under act of phospholipase A, able to destroy the cells of superficial epithelium of mucous tunic of stomach by removing of lipid from their membranes. As a result the erosions and ulcers are formed in the patient organism. Bilious acids also has the expressed detergent's properties. As isolecithin and bilious acids, the very important bacterial flora which directly and through toxins can cause the damage of mucous tunic of stomach stump. Also, alkaline environment and disturbance of evacuation from the operated stomach influence favourably on microflora growth.

For the clinical picture of alkaline reflux-gastritis the permanent poured out pain in a epigastric area, belch and vomiting by a bile are typical. At some patients heartburn and pain is observed behind a breastbone also. In majority patients so proof loss of weight takes place, that even the protracted complex therapy and valuable feed does not provide addition to the deficit of mass of body. There are typical signs also – anaemia, hypo- or achlorhydria.

Reliable diagnostics of alkaline reflux-gastritis became possible after wide introduction in clinical practice of endoscopic examination. In such patients during gastroscopy hyperemia of mucous tunic of stomach is observed. It is often possible to observe reflux in the stomach of duodenum content. During histological examination of biopsy material a chronic inflammatory process, intestinal metaplasia, diminishment of mass of coating cells and area of hemorrhages are found. All it testifies the deep degenerative changes in the mucous tunic of stomach. The some authors underlines that the inflammatory changes, at least in the area of anastomosis, are observed in most persons which carried the resection of stomach. So, endoscopic examination can not be considered deciding in diagnostics. Even the diffuse inflammatory changes can take place in absent of clinical symptoms and, opposite, in case with expressed clinical symptoms the minimum changes of mucous tunic of stomach are sometimes observed.

Conservative treatment of reflux-gastritis (sparing diet, antacides, enveloping preparations), usually, is ineffective. Existing methods of surgical treatment, mainly, directed on the removal of reflux of duodenum content to the stomach. Most popular is operation by the Roux method. The some surgeons considers that distance from gastroenteroanastomosis to interintestinal anastomosis must be 45–50 cm.

Main reason of origin of peptic ulcer of anastomosis is leaving of the hyperacid state of stomach mucous, even after the performed operation. Such phenomenon can be consequence of many reasons: primary economy resection, wrong executed resection (when the mucous tunic of pyloric part is abandoned in stump of duodenum or stomach), heightened tone of vagus nerves and the Zollinger-Ellison syndrome.

Peptic ulcers, usually, arise up after operation during the first year. Typical signs are pain, vomiting, weight loss, bleeding, penetration and perforation.

Pain is the basic symptom of peptic ulcer. Often it has the same character and localization, as well as at peptic ulcer. However often observe it moving to the left or in the umbilical area. At first patients bind such feelings to the use of food, but then specify nightly and hungry pain. It at first is halted after a food, but in course of time is become permanent, unendurable, independent from food intake. It can increase during the flounces, the walk, can irradiate in the back, thorax or shoulder.

During the objective examination of patients is often possible to expose on a stomach hyperpigmentation from a hot-water bottle. During palpation to the left from epigastric area near a umbilicus the painful and moderate muscles tension of abdominal wall is observed. Sometimes is possible to palpate inflammatory infiltrate of different sizes. During the examination of patients with a peptic ulcer the important role has determination of gastric secretion against a background of histamine and insulin stimulation. There is a necessity also examination of basal secretion. These preoperative examinations in most patients enable to set the reason of hypersecretion which can be: 1) heightened tone of vagus nerves (positive Hollander test); 2) economy resection of stomach, often in combination with the heightened tone of vagus nerve (considerable increase of gastric secretion after histamine or pentagastrin stimulation in combination with the positive Hollander test); 3) abandoned part of mucous tunic of antral part of stomach (high basal secretion and small increase of secretion in reply to histamine and insulin stimulator); 4) the Zollinger-Ellison syndrome.

Roentgenologic diagnostics of peptic ulcer, usually, is difficult, especially at shallow, flat ulcers, bad mobility and insufficient function of anastomosis. A niche is the direct sign of a similar pathology, indirect are the expressed inflammatory changes of mucous tunic of stump of stomach and bowel, painful point in the projection of stump of stomach and anastomosis and bad function of anastomosis. The deciding value in diagnostics has endoscopic examination.

Conservative treatment of peptic ulcers, as a rule, is ineffective. So, operation must be the basic type of treatment. The choice of method of operative treatment depends on character of previous operation and from abdominal cavity pathology found during the revision. For today the most important parts of the repeated operations is vagotomy. There is obligatory also during the resection of stomach on the exception the revision of duodenum stump for liquidation of possibly abandoned mucous tunic of antral area.

Operative treatment at a peptic ulcer must consist of certain stages. Laparotomy and disconnection of adhesions through a considerable spike process (increasing of stomach, loops of intestine and liver to the postoperative scar) almost always causes large difficulties.

After the selection of anastomosis with afferent and efferent loops the last cut by the “UKL-60 appliance”, within the limits of healthy tissues with renewal of intestine continuity by “end-to-end” type anastomosis.

At patients with a peptic ulcer, that developed after gastroenterostomy, cut a duodenum and sutured its stump by one of the described methods. During it there can be the difficulties related to the presence in it active ulcer. When peptic ulcers do not cause rough deformation of stomach, apply degastroenterostomy, vagotomy and drainage operations.

In the case of the considerably expressed spike process it is possible to execute trunk subdiaphragmatic vagotomy, and in case of the insignificantly changed topography of this area — selective gastric vagotomy.

It is important to note, that stomach resected together with anastomosis, peptic ulcer and eliminated area of empty bowel by one block.

This pathology arises up as a result of perforated of peptic ulcer in a transverse colon with formation of connection between a stomach, small or large intestine.

Diagnostics of gastro-colon fistula at patients with expressed clinical signs of disease does not difficult. However, symptoms are often formed and is indicated up slowly, so such patients with different diagnoses long time treat oneself in the therapeutic or infectious parts.

The typical signs of this pathology is considered diminishment or disappearance of pain, that was before, and profuse, that does not respond to treatment, diarrhea. Patients has emptying up to 10–15 times per days and even more frequent. An excrement contains a plenty of undigested muscular fibres and fat acids (steatorrhea). In case of wide fistula an undigested food can be with an excrement.

Excrement smell from a mouth, usually, notice surrounding. The patients does not feel it. However appearance of excrement belch is indicate the hit into the stomach of excrement masses and gases, and could confirm this pathology.

The such patients very quickly lose weight (mass of body goes down on 50–60 %), their skin becomes pale with a grey tint. The protein-free edemata, ascites, hydrothorax, anasarca, signs of avitaminosis appear in non-treated case.

Through the severe losses of liquid and nonassimilable food there can be the increased appetite and unendurable thirst in such patients. However, they adopt a plenty of liquid and food but the state of them continues to get worse.

Headache, apathy and depression is observed, and at the objective examination is exhaustion (ochre colour of skin, dryness and decline of it turgor, edemata or slurred of swelling extremities, atrophy of muscles). A stomach often moderately pigmented from hot-water bottles, subinflated, with the visible peristalsis of intestine. During the changes of patient position it is possible to hear grumbling, splash and transfusion of liquid. The examination of blood can expose hypochromic anaemia.

Roentgenologic examination is a basic diagnostic method. There are three varieties of such examinations of gastro-colon fistula. During the examination with introduction of barium mixture through a mouth the hit of contrasting matter directly from a stomach into a colon is the typical roentgenologic symptom of such pathology. Irrigoscopy is more perfect and effective method. With suspicion on gastro-colon fistula it is better to perform irrigoscopy. Passing of contrasting matter to the stomach at this manipulation testifies the presence of fistula. The third method is insufflation of air in a rectum. With it help on the screen it is possible to observe the location and passing of fistula, and also, as a result, hit of air in a stomach, increase of it gas bubble. Thus there can be the belch with an excrement smell.

The important role played the tests with dyes: at peroral introduction of methylene-blue after the some time it found in excrement masses or, opposite, after an enema with methylene-blue dye appears in a stomach.

Treatment of gastro-colon fistula is exceptionally operative. It needs to be conducted after intensive preoperative preparation with correction of metabolism. All operations which can be applied at treatment of patients with gastro-colon fistula divide into palliative and radical (single-stage operation and multi-stage operation).

During the palliative operations the place of fistula of stomach, transverse colon and jejunum is disconnected and then sutured the created defects. Other variant is disconnection of stomach and transverse colon and leaving the gastroenteroanastomosis. It is necessary to remember, that during such operations the only fistula always removed and does not performed the resection of stomach. Clearly, that such situation also does not eliminate possibility of relapse of peptic ulcer and development of its complications. Taking into account it, palliative operations can be recommended in those case only, when the general condition of patient does not allow to perform radical operation.

Single-stage operation radical operations. The most widespread is degastroenterostomy with the resection of stomach. However, it is needed to remember that operation of disconnection of fistula, suturing of opening in the jejunum and transverse colon on the lines of fistula and resection of stomach applies only in case of absent of infiltrate and deformation and in the conditions of possibility to close a defect in bowels without narrowing of their lumen. This operation is the simplest, is enough easily carried by patients and it is enough radical.

Such complications appear through considerable time after operation (from 1 month to one year). Disturbances of function of gastrointestinal anastomosis can be caused by the reasons, related both to the technical mistakes during operation and with pathological processes which arose up in the area of anastomosis.

The clinical picture of disturbance of anastomosis function, mainly, depends from the degree of its closing. At complete it obstruction in patients arise up intensive vomiting, pain in a epigastric area, the symptoms of dehydration and other similar signs appear. In other words, the clinic of stenosis of the stomach output develops. Clearly, that during incomplete narrowing the clinical signs will be expressed less, and growth of them — more slow. Sometimes disturbance of evacuation can unite with the syndrome of afferent loop with a inherent clinical picture. At the roentgenologic examination of such patients expansion of stomach stump is exposed with the horizontal level of liquid and small gas bubble. Evacuation from it is absent or acutely slow.

Treatment of scar deformations and narrowing of anastomosis must be operative and directed for the disconnection of accretions and straightening of the deformed areas. In case of presence in patients large inflammatory infiltrate it does not need to perform disconnection. In such cases it is the best to apply roundabout anastomosis. If a resection by Finsterer was done in such patient, better to perform anterior gastroenteroanastomosis, and after a resection by Billroth-I — posterior. As a result of conducting of such operations the state of patient, as a rule, gets better, and often recovered the function of primary anastomosis.

Removing of all stomach and exception of duodenum from the process of digestion of food cause plural functional disturbances in an organism. Some of them meet already after the resection of stomach (dumping-syndrome, hypoglycemic syndrome), other more inherent for gastrectomy (anaemia, reflux- esophagitis and others like that).

Most patients, that carried gastrectomy, complain for a considerable physical weakness, heightened fatigueability, sometimes is complete weakness, loss of activity and acute decline of work capacity. Almost all of them notice bad sleep, worsening of memory and heightened irritates. The appearance of patients is typical. Their skin insignificantly hyperpigmented, dry, its turgor reduced, noticeable atrophy of muscles. Can be the signs of chronic coronal insufficiency in such patients, and in older-year persons is typical picture of stenocardia. Except for it, can be hypotension, bradycardia and decline of voltage on EKG; during auscultation deafness of tones is observed. From the side of the hormonal system the decline of function of sexual glands is typical: in men — declines of potency, in women — disturbances of menstrual cycle, early climax. Can be the signs of hypovitaminosis A, B, C and decline of resistibility of organism to chill, infectious diseases and tuberculosis.

The decline of mass of body is observed in 75 % patients, that carried gastrectomy. It is conditioned by the decline of power value of food as a result of disturbance of digestion, bad appetite and wrong diet. As a result of progressive hypoproteinemia there can be the protein-free edemata.

Patients with such pathology must be under the permanent clinical supervision and 1–2 times per year during a month to have the course of stationary prophylactic treatment which includes psycho-, diet-, vitaminotherapy, correcting and replaceable therapy, and also prophylaxis of anaemia.

Psychotherapy is especially indicated in the psychodepressive and asthenic states. It is performed in combination with medicinal treatment. Hypnotic preparation, bromide, tranquilizers are applied.

A food must be correctly prepared, without the protracted cooking. Patients need to feed on 6–10 times per days by small portions.

Next to dietotherapy, it is constantly necessary to apply replaceable therapy (Pancreatine, Pansinorm, Festal, Intestopan). In case of absent of esophagitis hydrochloric acid is appointed. For the improvement of albuminous exchange anabolic hormones are applied.

In case of reflux-esophagitis there are indicated feeds by small portions with predominance of liquid, ground, jelly-like foods, astringent, coating, anticholinergic preparations. Between the receptions of food does not recommend to use a liquid. In case of dysphagy appoints a sparing diet.

For the prophylaxis of iron-deficiency anaemia, that arises up in the first 2–3 years after gastrectomy, important the indication of iron preparations.

For warnings and treatments of pernicious anaemia applied cyanocobalamin for 200 mcg through a day and folic acid. Packed red blood cells is indicated in heavy case.

The relapse of ulcer is enough frequent complication of vagotomy. It meets in 8–12 % patients. The reasons of such relapses of ulcer can be: 1) inadequate decline of products of hydrochloric acid (incomplete vagotomy, reinnervation); 2) disturbance of emptying of stomach (ulcerous pylorostenosis after selective proximal vagotomy or after pyloroplasty); 3) local factors (duodenogastric reflux with development of chronic atrophy gastritis, disturbance of circulation of blood and decline of resistibility of mucous tunic); 4) exogenous factors (alcohol, smoking, medicinal preparations); 5) endocrine factors (hypergastrinaemia: hyperplasia of antral G-cells, the Zollinger-Ellison syndrome; hyperparathyroidism).

Three variants of clinical passing of relapse of ulcer are distinguished after vagotomy: 1) symptomless, when an ulcer is found during endoscopic examination; 2) recurrent with protracted lucid space; 3) persisting ulcer with typical periodicity and seasonality of exacerbation.

It is needed to underline that the clinical signs of this pathology during the relapse are less expressed, than before operation, and absence of pain does not eliminate the presence of ulcer. Sometimes bleeding can be first its sign. Complex examination, that includes roentgenologic, endoscopic examination, study of gastric secretion and determination of content of gastrin in the blood, allows not only to expose an ulcer but also, in most cases, to set its reason. The interpretation the results of gastric secretion examination in such patients are heavy. Taking into account it, it is needed to study both a basal secretion and secretion in reply to introduction of insulin and pentagastrin, and also level of pepsin.

Approximately in 35 % patients, mainly with the first two variants of clinical passing of disease, the relapses of ulcers, are treated by ordinary methods of conservative therapy. Yet in 30–40 % cicatrization of ulcers comes after application of preparations which stop a gastric secretion (cimetidine, ranitidine—150 mg for night). At other 10–20 % patients, mainly with the third variant of clinical passing, is necessary operative treatment.

The question of choice of the repeated operation in patients with the relapse of ulcer after vagotomy still does not decided. Some surgeons execute revagotomy, trunk vagotomy with drainage operation, revagotomy with antrectomy or resection of stomach. However much majority from them in case of relapse ulcer after vagotomy performed antrectomy in combination with trunk vagotomy.

Frequency of postvagotomy diarrhea hesitates from 2 to 30 %. The basic sign of complication in patient is present the liquid watery emptying about three times per days. The reasons of diarrhea are: gastric stasis and achlorhydria, denervation of pancreas, small intestine and liver, and also disturbance of motility of digestive tract. Discoordination of evacuations from a stomach, stagnation and hypochlorhydria assist to development in it different microorganisms, and it also can be the reason of diarrhea.

The clinical signs of postvagotomy diarrhea are specific. Acute beginning are typical –patient often does not have time to reach to the rest room. Such suddenness repressing operates on patients. As a result they are forced whole days to be at home, expecting the duty attack. An excrement changes colorings as a result of breeding of pigment and becomes more light.

Treatment of diarrhea must be complex. Above all things it is needed to recommend a diet with the exception of milk and other provoking products. For the removal of bacterial factor antibiotics are applied. Favourable action in case of the signs of stagnation in a stomach are had weak solutions of organic acids (lemon, apple and others like that).

Among other most distribution was got by the A.A. Kuragin and S.D. Hroismann (1971) suggestion to treat postvagotomy diarrhea by benzohexamethonium (for 1 ml 2,5 % solution 2–3 times per a day). Reported also about successful application of cholesteramine (for 4 g 3 times per a day with the subsequent decline of dose to 4 g per days).

At heavy passing of postvagotomy diarrhea, that does not respond to conservative treatment, it is needed to recommend operative treatment — degastroenterostomy with pyloroplasty. However, the type of drainage operation, as practice shows, does not influence on frequency of diarrhea origin. In this connection, some surgeons with success applied the inversion of the segment of thin bowel, located distal from the area of maximal absorption.

The cancer of stomach is a malignant formation, that develops from epithelium tissue of mucus stomach. Among the tumours of organs of digestion this pathology takes first place and is the most frequent, by the reason of death from malignant formations in many countries of world. Frequency of it at the last 30 years considerably diminished in the countries of Western Europe and North America, but yet remains high in Japan, China, countries of East Europe and South America.

Etiology of cancer of stomach is unknown. It is known that, as other diseases of gastrointestinal tract, a cancer damages a stomach. According to statistical information, it meets approximately in 40 % of all localizations of cancer.

The factors of external environment has the substantial influencing on frequency of this pathology. Above all things, feed, smoke food, salting, freezing of products and their contamination of aflatoxin. Consider that a “food factor” can be: a) by a carcinogen; b) by the solvent of carcinogens; c) to grow into a carcinogen in the process of digestion; d) to be instrumental in action of carcinogens; e) not enough to neutralize carcinogens.

In the USA and countries of Western Europe frequency of cancer of stomach in 2 times more large in the lower socio-economic groups of population. Some professional groups also can it (miners, farmers, works of rubber, woodworking and asbestine industry). High correlation communication is set between frequency of cancer of stomach and use of alcohol and smoking. The value of genetic factors (heredity, blood type) is not led to.

The cancer of stomach arises up mainly in age 60 years and above, more frequent men are ill.

Precancer. The precancer diseases of stomach are: a) chronic metaplastic disregenerator gastritis conditioned by helicobacter pylori; b) villous polypuses of stomach and chronic ulcers; c) nutritional anemia due to vitamin B12 deficiency (pernicious); d) resected stomach concerning an ulcer.

The presence of precancer changes of mucous tunic of stomach has substantial influence for frequency of stomach cancer. In those countries, where morbidity on the cancer of stomach is higher, considerably more frequent chronic gastritis are diagnosed. Lately in etiology of chronic gastritis take the important value helicobacter pylori. In Japan, where the cancer of stomach is in 40 % cases is the reason of death, chronic gastritis appears in 80 % cases of resected stomach, concerning a cancer.

Connection between polypuses, chronic gastric ulcers and possible it malignization comes into question in literature during many decades. Most authors consider that polypuses could be malignant differently. There are three histological types of polypuses: hyperplastic, villous and hamartoma. There are hyperplastic polypuses, but it not malignant.

Hamartoma is accumulation of cells of normal mucous tunic of stomach. They never becomes malignant.

Villous polypuses are potentially malignant in 40 % cases, but it happen in 10 times less, than hyperplastic. The possibility of malignization of chronic gastric ulcers is not proved. The American scientists support a hypothesis, that the cancer of stomach can be ulcerous often, but malignization of ulcers takes place rarely (no more than 3 %). From data of the Japanese scientists, on 50–70th there was higher correlation connection between chronic gastric ulcers and cancer of stomach. The frequent decline of this correlation is lately noticed (70 % on 50–70th and 10 % on 80th).

Frequency of cancer of stomach at patients with pernicious anaemia hesitates within the 5–10 %, that in 20 times higher, compare with control population. In patients with a resected stomach after peptic ulcers is multiplied the risk of origin of stomach cancer in 2–3 times (duration of latent period hesitates from 15 to 40 years). The reason of such dependence is not found out, but there is a version, that this is linked with a gastric epithelium metaplasia by an intestinal type.

From all malignant formations of the stomach in 95 % adenocarcinoma is observed. Epidermoid cancer, adeno-acanthoma and carcinoid tumours do not exceed 1 %. Frequency of leiomyosarcoma hesitates within the limits of 1–3 %. Lymphoma of gastrointestinal tract is localized in a stomach.

The prognosis of localization depends on the degree of invasion, histological variants of tumour.

The macroscopic forms of cancer of stomach in different times were described variously. More than 60 years ago the German pathologist Bermann described 5 macroscopic forms of cancer of stomach: 1) polypoid or mushroom-like; 2) saucer-shaped or with ulcerous and expressly salient edges; 3) with ulcerous and infiltration of walls of stomach; 4) diffuse -infiltrate; 5) unclassified.

American pathopsychologists is selected 4 forms. The tumours of stomach with ulcerous are the most frequent macroscopic form of cancer of stomach and arise up on soil of chronic ulcer. The signs suspicious on malignization are: the sizes of ulcer more than 2 cm in a diameter, appearance of the heightened edges.

The polypoid tumours of stomach observed only in 10 %. These tumours can achieve considerable sizes without an invasion and metastasis. Scirrhus carcinoma is the third macroscopic type. This category of tumours also does not exceed 10 %. The scirrhus carcinoma is the signs of infiltration by anaplastic cancer cells, diffusely developed connecting tissue which results in the bulge and rigidity of wall of stomach. So called “small cancers” belong to the fourth macroscopic type. It meet comparative rarely (no more than 5 %) and is characterized by superficial accumulation of cancer cells which substitute for normal mucus in such kind: a) superficial flat layer which does not rise above the level of mucus; b) salient (bursting) formation; c) erosions.

Mainly (more than 50 %) tumours arise up in a antral part or in distal (lower) third of stomach, rarer (to 15 %) — in a body and in cardia (to 25 %).

However, lately more often observed cardioesophageal cancers and diminishment of frequency of tumours of distal parts of stomach. In 2 % cases meet the multicentric focuses of growth, but from data of some authors, this percent could be multiplied in 10 times after carefully histological inspection of the resected stomachs. This assertion is based on the theory of the “tumour field” (D.I. Holovin, 1992). Especially this typically for patients which has pernicious anaemia or chronic metaplastic disregenerative gastritis.

Metastasis is carried out by lymphogenic, hematogenic and implantation ways mostly. Three (from data of some authors, four) pools of lymphogenic metastasis are selected: left gastric (knots on passing of small curvature of stomach in a gastro-subgastric ligament and pericardial); splenic (mainly, suprafrapancreatic knots); hepatic (knots in a hepato-duodenal ligament, right gastric omentum that lower pyloric groups, right gastric and suprapyloric groups, pancreatoduodenal group).

However, the such way of lymphogenic metastasis is conditional and incomplete, as at presence of block lymph flow passes retrograde metastasis, so called “jumping metastases” which predetermine the origin of remote lymphogenic metastases in left supraclavicular lymph nodes (Virhov metastasis) appear, in Lymph nodes of left axillar and inguinal areas, metastases in an umbilicus.

Direct distribution: small and large omentum, esophagus and duodenum; liver and diaphragm; pancreas, spleen, bile ducts.

Front wall of stomach: colon bowel and mesocolon; organs and tissues of retroperitoneal space.

Lymphogenic metastasis: regional lymph nodes, remote lymph nodes, left supraclavicular lymph node (Virhov), lymph node of axillar area (Irish); in a umbilicus (sisters Joseph).

Hematogenic metastasis: liver, lungs, bones, cerebrum.

Peritoneal metastasis: peritoneum, ovarium (the Krukenberg metastasis), Duglas space (the Shnieler metastasis).

All authors which are engaged in the study of problem of cancer of stomach underline absence or vagueness, no specificity of symptoms, especially on the early stages of disease. The displays of cancer of stomach are very various and depend on localization of tumour, character of its growth, morphological structure, distribution on contiguous organs and tissues. At localization of tumour in a cardiac part patient complains firstly, as a rule, for appearance of dysphagy.

At careful, purposeful collection of anamnesis it is not succeeded to expose some other, most early symptoms, which precedes to dysphagy and forces a patient to appeal to the doctor. The unpleasant feeling behind a breastbone and feeling of unpassing of hard food on a esophagus appear at the beginning of disease. After some time (as a rule, it is enough quickly, during a few weeks, sometimes even days) a hard food does not pass (it is to wash down by water or other liquid). This period can be during 1–3 months. Patients address a doctor exactly in this period. Other symptoms appear to this time: regurgitation, pain behind a breastbone, loss of mass of body, sometimes even exhaustion, the grey colouring of person, a skin is dry, quickly grows general weakness. Sometimes patients address a doctor, when already with large effort a spoon-meat passes only or complete stenosis came.

At localization of tumour in the antral part of stomach the first complaints, as a rule, are up to appearance of feeling of weight in epigastric region after the reception of food (even in a two-bit), “feeling of saturation” (after the reception of glass of water), belch (at first it is simple by air, and then with a smell). Feeling of weight grows for a day, patients forced to cause vomiting. In the morning there can be vomiting by mucus with the admixtures of “coffee-grounds” (so called “cancer” water). Patients loses weight (mass of body is lost), a weakness, anaemia grows.

Tumours localized in the body of stomach show up either a pain syndrome or syndrome of so called “small signs” (A.I. Savitsky, 1947), which is characterized by appearance of amotivational general weakness, decline of capacity, rapid fatigueability, depression (by the loss of interest to the environment), proof decline of appetite, gastric discomfort, making progress weight lost.

The carried chronic diseases of stomach, for which typical seasonality, can influence on the clinical sign of cancer of stomach. At appearance of “gastric” complaints out of season or in absent of effect from the got therapy concerning the exacerbation of “gastritis”, “ulcers” must guard a patient and doctor (symptom of “precipice” of gastric anamnesis).

In case of occurring of “gastric” symptoms first in persons in age 50 years and older it is foremost necessary to eliminate the cancer of stomach.

In parts of patients cancer of stomach shows up only the metastatic damage of other organs or complications. More than twenty so called “atypical” forms, which are characterized by “causeless” anaemia, ascites, icterus, fever, edemata, hormonal disturbances, changes of carbohydrate exchange, intestinal symptoms, are distinguished.

During the examination of patients with the cancer of stomach the pallor of skin covers (at anaemia) is observed, in neglected case is “frog” stomach (sign of ascites).

During palpation determined painful in a epigastric area, sometimes possible to palpate the tumour.

During auscultation of patients with pylorostenosis it is possible to define “noise of splash”.

Laboratory information: hypochromic anaemia, neutrophilic leukocytosis, megascopic ESR; during examination of gastric secretion: hypo- and acidity and achlorhydria.

Gastroduodenoscopy enables to diagnose a tumour even smaller 5 mm and conduct an aiming biopsy with histological examination of the taken material.

Roentgenoscopy and roentgenography examination of stomach. Basic signs: defect of filling, local absence of peristalsis, "malignant" relief of mucous tunic (Pic. 3.2.18).

Ultrasonic examination: presence of metastases in a liver, pancreas.

Computer tomography allows to estimate the basic parameters of tumour, germination in neighbouring organs and presence of metastases.

It is expedient to apply laparoscopy, mainly, for the decision of question about operable of tumour (diagnostics of metastatic defeat of organs of abdominal cavity).

At an early cancer complaints depend on the previous gastric diseases. Therefore, on the basis of clinical information, suspecting a tumour is possible only on occasion, when in patients next to clear pain symptoms an appetite goes down, appear anaemia, general weakness. In practice an early cancer is recognized at purposeful screening, and also in the process of endoscopic or roentgenologic examination of gastric patients.

A differential diagnosis is conducted with an peptic ulcer, gastritis, polyposis, other gastric and ungastric diseases. For a cancer there is typical firmness of symptoms, instead of their seasonality (typical syndrome of "precipice" of gastric anamnesis) or tendency to their gradual progress.

The row of diseases, with which the cancer of stomach is to differentiate to the doctor, depends from character of complaints of patients.

Five basic clinical syndromes are selected:

- 1) pain;
- 2) gastric discomfort;
- 3) anaemic;
- 4) dysphagic;
- 5) disturbance of evacuation from a stomach.

At patients, at what cancer of stomach shows up a pain syndrome and syndrome of gastric discomfort, a differential diagnosis is conducted with the peptic ulcer, gastritis, cancer of body of pancreas.

It is oriented on features dynamics of development of pain syndrome, ingravescence of the general condition, change of character of complaints.

A question about character of anaemia, source and nature of bleeding decides at an anaemic syndrome. In the process of examination attention is paid to the state of bottom of stomach, where bleeding malignant formations can be.

At a dysphagic syndrome a differential diagnosis is conducted with the cicatricial narrowing, achalasia of esophagus. For malignant formations testify short anamnesis, gradual progress of symptoms, signs of gastric discomfort, general weakness, weight lost.

At disturbance of evacuation from a stomach during stenosis of pyloric part, absence of ulcerous anamnesis, declining years of patients, relatively quick (weeks, months) growth of stenosis testify for tumor.

The presence of cancer of stomach is a indications for surgical treatment. However, counting on success is possible only at presence of the limited tumours (within the limits of the 0-II stages). At the III stage of disease implementation of the widespread combined operations in a radical volume is possible, however most patients die during 1–2 years. A distal or proximal subtotal resection (Pic. 3.2.19) and total gastrectomy (Pic. 3.2.20) is performed with removing of large and small omentumes and regional areas of metastasis with obligatory histological examination of stomach on the lines of resections.

During the combined operations organs which are pulled in to the pathological process are removed.

In case of IV stage of disease and satisfactory state of patient palliative operations which improve quality of life of patient are performed.

In case of presence of complications (mainly stenosis) and grave common condition of patient perform symptomatic operative treatments.

Symptomatic is operations which will liquidate one of symptoms of cancer of stomach. In this group of operations include: 1) roundabout gastrojejunostomosis (Pic. 3.2.21) and jejunostoma (in case of the stenosis tumours of stomach output); 2) gastrostoma (Pic. 3.2.22) in case of the cancer of cardiac part of stomach with disturbance of patency; 3) edging of bleeding vessels in case of complication of cancer by bleeding; 4) tamponade by omentum during the perforation of tumour.

The value of radial therapy and chemotherapy, as independent methods of treatment of cancer of stomach, is limited. Radial therapy is indicated for patients with cardiac cancer as preoperative course or as palliative treatment. Adjuvant mono- or polychemotherapy (mainly by 5-photouracil) is conducted in a postoperative period as combined therapy and in case of dissemination of the tumours.

Prognosis. The indexes of five-year survival of patients with the cancer of stomach hesitate within the limits of 5–30 %, but, from data of most authors, they do not exceed 10 %.

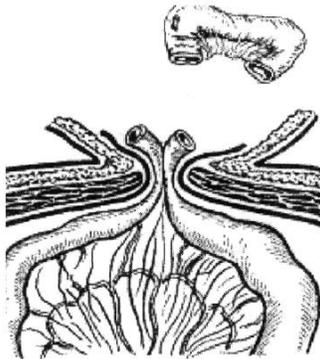
Tactics and choice of treatment method

The incarcerated hernia, regardless of time of its origin, localization and age of patient, must be operated on. However, if a patient is hospitalized already with the expressed symptoms of intestinal obstruction, than should be preoperative treatment. Such conservative therapy must be brief (1–1,5 hours), but always actively directed for correction of violations of metabolism and prophylaxis of possible pulmonary and cardiovascular complications. It is necessary also to conduct evacuation of the gastric contents and other preparatory procedures.

Patient with reduced hernia must be hospitalized and observed during 1–2 days. If a abdominal pain is contained or is growing, the signs of peritonitis and intoxication appear, than performed urgent laparotomy and necessary operation. If the symptoms of "acute" abdomen are not present, a patient examined and prepared for elective operation.

Operation at the incarcerated hernia is executed under the general anesthesia. A hernia sack is selected from surrounding tissue, cut it in the area of bottom and remove hernia water, defining its character and sending to bacterial inoculation. Retaining the damaged organs, a strangulated ring is cut. It is necessary to remember, that at the incarcerated femoral hernia ring cut up and some medially, because a femoral vein passes from a lateral side.

If a bowel is contents of hernia sack, we must estimate its viability. Remembering about possibility of the retrograde jamming, special attention must be paid to the state of strangulation furrow. About viability of the bowels testify: 1) renewal of its normal color; 2) presence or renewal of peristalsis; 3) renewal of pulsation of vessels of mesentery and bowel. If there are the certain doubting, a bowel is dipped on a holder in an abdominal cavity and in 15–20 minutes it is examined repeatedly. If one of the resulted signs of viability is absent even, it is necessary to conduct the resection of bowel. The resection is executed, receded from the strangulation furrow on a proximal loop 30–40 cm and distal — 15–20 cm. Anastomosis between proximal and distal loops it is better to impose "end-to-end". The plastic of hernia gate are conducted depending on indications after one of the surgical methods.



When the necrosis elements of omentum or fatty pendants of colon are contents of hernia sack, they must be removed within the limits of healthy tissue.

There can be necrosis of wall of colon or urinary bladder at sliding hernia. In such cases it is needed to be limited to the minimum surgical procedure: to dip a necrosis area by sutures inside the bowel or use it for forming of colostomy or epicycstostomy. These are the best to conclude operation.

In similar situations at the incarcerated parietal hernia in most patients it is possible to be limited to peritonization of displaced area of wall of bowel. If after the peritonization there is the threat of narrowing of bowel or necrosis goes outside of the strangulation furrow, it is needed to conduct the resection of bowel.

Because of insufficient blood flow of Meckel's diverticulum and, permanent threat of it necrosis, at patients with Littre's hernia it resection must be performed.

At the phlegmon of hernia sack operation is begun with hemiotomy. If the incarcerated organ is damaged by necrosis, and in a hernia sack present pus, than there is a necessity for surgeon to perform laparotomy. After that incarcerated organ resected within the limits of healthy tissue (in the generally accepted limits — 40 cm of proximal loop and 20 cm distal) and impose anastomosis. An abdominal cavity is sewn up. Incarcerated loops of bowel, together with it blind ends which located in an abdominal cavity, removed through a hernia sack, a peritoneum is sutured, the hernia sack is drained, the plastic of hernia gate are not performed. Skin is sewn up by widely spaced sutures.

[video1](#)

[video](#)

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ACUTE ILEUS. PATHOGENESIS. CLINIC. DIAGNOSTIC. TACTICS.

ACUTE DYNAMIC ILEUS.

ACUTE OBTURATIVE AND STRANGULATED INTESTINAL OBSTRUCTION.

Intestinal obstruction is a complete or partial violation of passing of maintenance by the intestinal tract.

Ileus is commonly defined simply as bowel obstruction. However, authoritative sources define it as decreased motor activity of the GI tract due to non-mechanical causes. In such sense, this does not include motility disorders that result from structural abnormalities, and, therefore, some mechanical obstructions are misnomers, such as gallstone ileus and meconium ileus, and are not true examples of ileus.

Types:

Decreased propulsive ability may be broadly classified as caused either by bowel obstruction or intestinal atony or paralysis. However, there are instances where there are symptoms and signs of a bowel obstruction, but with absence of a mechanical obstruction, mainly in *acute colonic pseudoobstruction*, also known as Ogilvie's syndrome.

Bowel obstruction. Bowel obstruction (or intestinal obstruction) is a mechanical or functional obstruction of the intestines, preventing the normal transit of the products of digestion. It can occur at any level distal to the duodenum of the small intestine and is a medical emergency. The condition is often treated conservatively over a period of 2–5 days with the patient's progress regularly monitored by an assigned physician. Surgical procedures are performed on occasion however in life-threatening cases, such as when the root cause is a fully lodged foreign object or malignant tumor.

Intestinal atony or paralysis. Paralysis of the intestine is often termed *paralytic ileus*. To be termed "paralytic ileus", the intestinal paralysis need not be complete, but it must be sufficient to prohibit the passage of food through the intestine and lead to intestinal blockage.

Paralytic ileus is a common side effect of some types of surgery, in these cases it is commonly called *postsurgical ileus*. It can also result from certain drugs and from

various injuries and illnesses, *i.e.* acute pancreatitis. Paralytic ileus causes constipation and bloating. On listening to the abdomen with a stethoscope, no bowel sounds are heard because the bowel is inactive.

A temporary paralysis of a portion of the intestines occurs typically after an abdominal surgery. Since the intestinal content of this portion is unable to move forward, food or drink should be avoided until peristaltic sound is heard from auscultation of the area where this portion lies.

Etiology and pathogenesis

The principal reasons of intestinal obstruction can be:

- 1) adhesions of abdominal cavity after traumas, wounds, previous operations and inflammatory diseases of organs of abdominal cavity and pelvis;
- 2) long mesentery of small intestine or colon, that predetermines considerable mobility of their loops;
- 3) tumours of abdominal cavity and retroperitoneal space.

Such principal reasons can cause violation of passing of intestinal maintenance, disorder of suction from the intestine and loss of plenty of electrolytes both from vomiting and in the intestine cavity as a result of disorders of bloodflow in its wall.

Most cases of ileus occur after intra-abdominal operations. Normal resumption of bowel activity after abdominal surgery follows a predictable pattern: the small bowel typically regains function within hours; the stomach regains activity in 1-2 days; and the colon regains activity in 3-5 days.

Serial abdominal radiographs mapping the distribution of radiopaque markers have shown that the colonic gradient for resolution of postoperative ileus is proximal to distal. The return of propulsive activity to the right colon occurs earlier than to the transverse or left colon.

Other causes of adynamic ileus are as follows:

- Sepsis
- Drugs (eg, opioids, antacids, warfarin, amitriptyline, chlorpromazine)
- Metabolic (eg, low potassium, magnesium, or sodium levels; anemia; hyposmolality)

- Myocardial infarction
- Pneumonia
- Trauma (eg, fractured ribs, fractured spine)
- Biliary colic and renal colic
- Head injury and neurosurgical procedures
- Intra-abdominal inflammation and peritonitis
- Retroperitoneal hematomas

Causes of Intestinal Obstruction

Causes of Intestinal Obstruction	
Location	Cause
Colon	Tumors (usually in left colon), diverticulitis (usually in sigmoid), volvulus of sigmoid or cecum, fecal impaction, Hirschsprung's disease, Crohn's disease
Duodenum	
Adults	Cancer of the duodenum or head of pancreas, ulcer disease
Neonates	Atresia, volvulus, bands, annular pancreas
Jejunum and ileum	
Adults	Hernias, adhesions (common), tumors, foreign body, Meckel's diverticulum, Crohn's disease (uncommon), Ascaris infestation, midgut volvulus, intussusception by tumor (rare)
Neonates	Meconium ileus, volvulus of a malrotated gut, atresia, intussusception

Pathophysiology. In simple mechanical obstruction, blockage occurs without vascular compromise. Ingested fluid and food, digestive secretions, and gas accumulate above the obstruction. The proximal bowel distends, and the distal segment collapses. The normal secretory and absorptive functions of the mucosa are depressed, and the bowel wall becomes edematous and congested. Severe intestinal distention is self-perpetuating and progressive, intensifying the peristaltic and secretory derangements and increasing the risks of dehydration and progression to strangulating obstruction.

According to some hypotheses, postoperative ileus is mediated via activation of inhibitory spinal reflex arcs. Anatomically, 3 distinct reflexes are involved: ultrashort reflexes confined to the bowel wall, short reflexes involving prevertebral ganglia, and long reflexes involving the spinal cord. The long reflexes are the most significant. Spinal anesthesia, abdominal sympathectomy, and nerve-cutting techniques have been demonstrated to either prevent or attenuate the development of ileus.

The surgical stress response leads to systemic generation of endocrine and inflammatory mediators that also promote the development of ileus. Rat models have shown that laparotomy, eventration, and bowel compression lead to increased numbers of macrophages, monocytes, dendritic cells, T cells, natural killer cells, and mast cells, as demonstrated by immunohistochemistry. Macrophages residing in the muscularis externa and mast cells are probably the key players in this inflammatory cascade. Calcitonin gene-related peptide, nitric oxide, vasoactive intestinal peptide, and substance P function as inhibitory neurotransmitters in the bowel nervous system. Nitric oxide and vasoactive intestinal peptide inhibitors and substance P receptor antagonists have been demonstrated to improve gastrointestinal function.

Strangulating obstruction is obstruction with compromised blood flow; it occurs in nearly 25% of patients with small-bowel obstruction. It is usually associated with hernia, volvulus, and intussusception. Strangulating obstruction can progress to infarction and gangrene in as little as 6 h. Venous obstruction occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall. The ischemic bowel becomes edematous and infarcts, leading to gangrene and perforation. In large-bowel obstruction, strangulation is rare (except with volvulus).

Perforation may occur in an ischemic segment (typically small bowel) or when marked dilation occurs. The risk is high if the cecum is dilated to a diameter ≥ 13 cm. Perforation of a tumor or a diverticulum may also occur at the obstruction site.

Pathomorphology

The morphological signs of dynamic intestinal obstruction are: small thickening of wall (at considerable paresis is thinning), friability of tissue (the bowel breaks easily) and presence of liquid maintenance and gases in cavity of bowel. At mechanical obstruction it is always possible to expose the obstacle: strang, commissures, tumours, jammings of hernia, cicatricial strictures, wrong entered drainages, tampons and others like that. In place of compression strangulation is exposed. The bowel loop higher strangulation is extended, and distally — collapsed. In case of released invagination on small distance two strangulation furrows are observed, and distally from the second ring cylinder expansion of bowel lumen is observed.

Classification

(by D.P.Chuhrienko, 1958)

Acute intestinal obstruction is divided:

I. *According to morphofunctional signs.*

1. Dynamic intestinal obstruction:

a) paralytic;

б) spastic;

в) hemostatic (embolic, thrombophlebitic).

2. Mechanical intestinal obstruction(Fig.1):

a) strangulated, volvulus, jamming;(Fig. 2)

б) obturation (closing of bowel lumen, squeezing from outside);

в) mixed (invagination, spike intestinal obstruction).

II. *According to clinical passing.*

1. Acute.

2. Chronic.

III. *According to the level of obstruction.*

1. Small intestinal.
2. Large intestinal:
 - a) high;
 - б) low.

IV. *According to the passing of intestinal maintenance.*

1. Complete.
2. Partial.

V. *According to the origin.*

1. Innate.
2. Acquired.

VI. *According to development of pathological process.*

1. Stage of acute violation of intestinal passage.
2. Stage of hemodynamic disorders of bowel wall and its mesentery.
3. Stage of peritonitis.

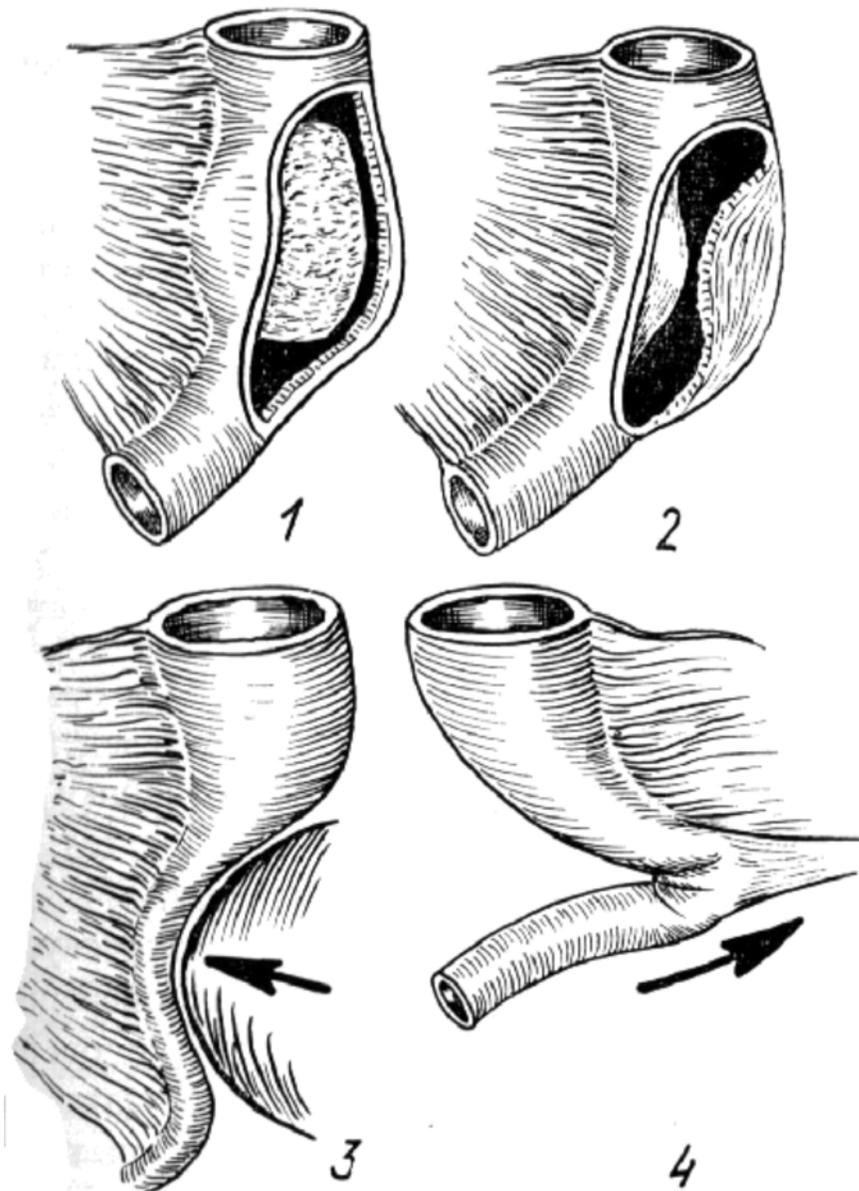


Fig.1. Types of mechanical intestinal obstruction. Obstacle reason of obturation: 1-Obturation; 2-Constriction; 3- Compression; 4- Angulation

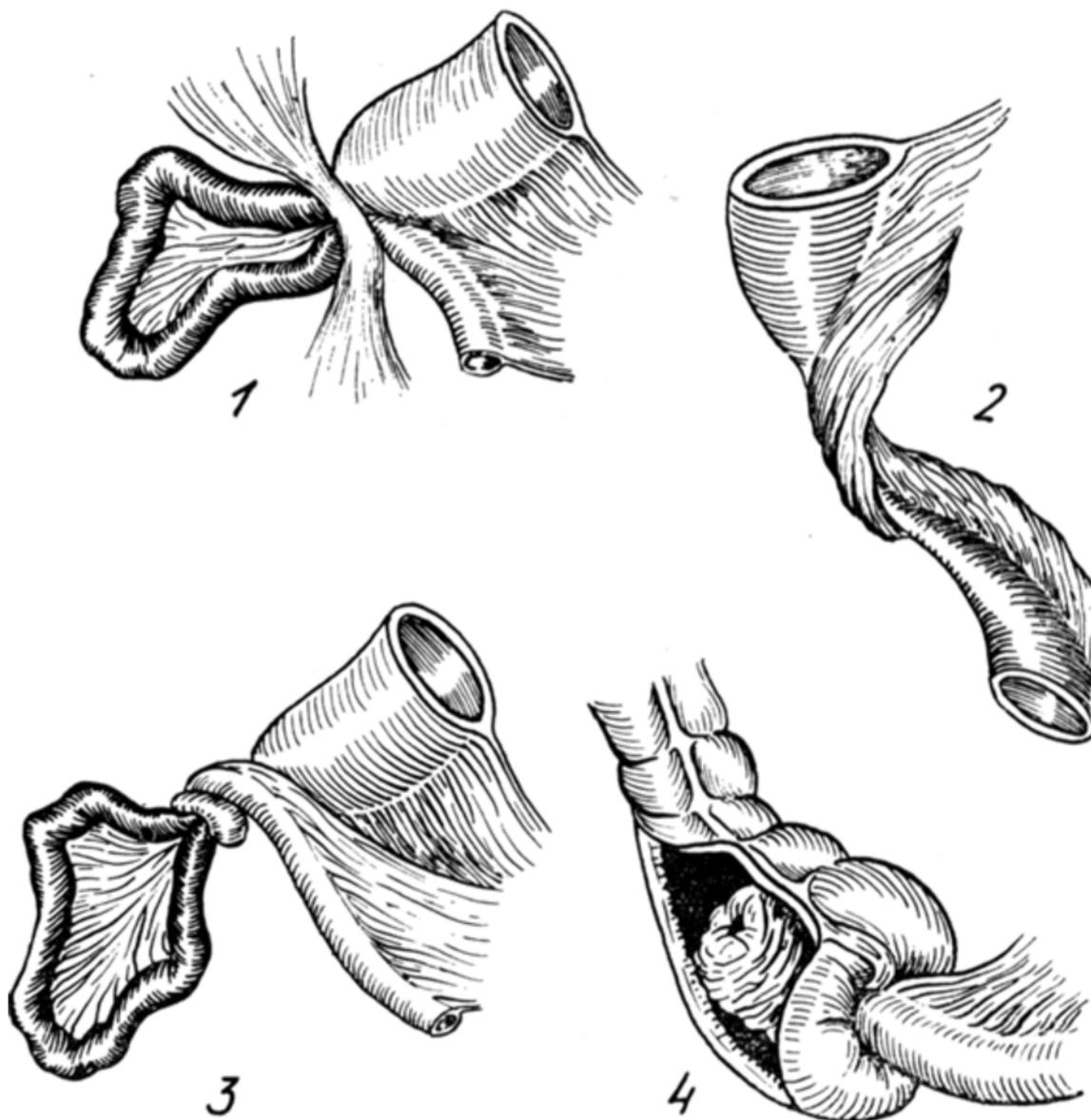


Fig.2. Mechanical intestinal obstruction. The disorders of blood circulation:
1-Strangulation; 2- Jamming; 3- Volvulus; 4- Invagination

Clinical management

Main symptoms:

- moderate, diffuse abdominal discomfort
- constipation
- abdominal distension
- nausea/vomiting, especially after meals
- lack of bowel movement and/or flatulence
- excessive belching

Beginning of clinical signs of intestinal obstruction is sudden — in 1–2 hours after taking the meal. The pain in the abdomen has the intermittent character and is met in all forms of mechanical intestinal obstruction. However, some types of strangulated intestinal obstruction (node formation, volvulus of thin and colons) can be accompanied by permanent pain. It is needed to mark that at spike intestinal obstruction, invagination and obturation cramp-like pain can be considered as pathognomic sign of disease. For paralytic intestinal obstruction more frequent is inherent permanent pain which is accompanied by the progressive swelling of abdomen. At spastic obstruction of intestine the pain is mainly acute, the abdomen is not blown away, sometimes pulled in.

Nausea and vomiting are met in 75–80 % patients with the heaviest forms of high level of intestinal obstruction (node formation, volvulus of small intestine, spike obstruction). At obturation obstruction and invagination they are observed not so often.

There is a characteristic thirst which can be considered as an early symptom. Besides, the higher intestinal obstruction, the greater the thirst.

Swelling of abdomen, the delay of emptying and gases are observed in 85–90% patients, mainly, with the high forms of obstruction (volvulus of small intestine, spike intestinal obstruction).

Together with that, for invagination emptying by liquid excrement with the admixtures of mucus and blood are more characteristic.

The abdomen may be distended and tympanic, depending on the degree of abdominal and bowel distention, and may be tender. A distinguishing feature is absent or hypoactive bowel sounds, in contrast to the high-pitched sound of obstruction. The silent abdomen of ileus reveals no discernible peristalsis or succussion splash.

In patients during palpation the soft abdomen is observed, sometimes — with easy resistance of front abdominal wall, and at percussion — high tympanitis. At auscultation at the beginning of disease increased peristaltic noises are present, then gradual fading of peristalsis is positive (the Mondor's symptom, “noise of beginning, quietness of end”).

There are other symptoms pathognomic for intestinal obstruction.

The Vala's symptom is the limited elastic sausage-shaped formation.

The Sklarov's symptom is the noise of intestinal splash.

The Kywul's symptom is the clang above the exaggerated bowel.

The Schlange's symptom is the peristalsis of bowel, that arises after palpation of abdomen.

The Spasokukotsky's symptom is "noise of falling drop".

The Hochenegg's symptom — incompletely closed anus in combination with balloon expansion of ampoule of rectum.

At survey roentgenoscopy or -graphy of the abdominal cavity in the loops of bowels liquids and gas are observed —the Klojber's bowl. (Fig.3; Fig.4)



Fig. 3. Intestinal obstruction.

X-ray examination of abdominal cavity – presence of the Klojber's bowels

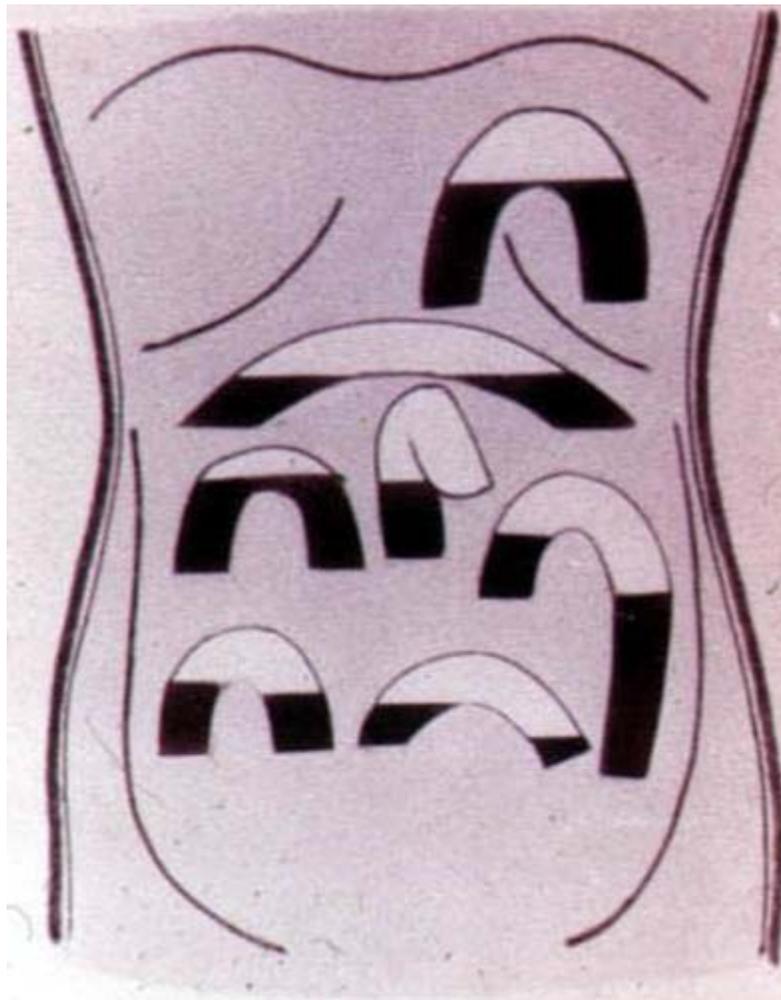


Fig. 4. Intestinal obstruction.

Mechanism of Klojbers bowels

Presence of liquid levels and air in the intestines

Variants of clinical passing and complications

Strangulated obstruction. The ischemic component is the characteristic feature of this form of intestinal obstruction, that is investigation of squeezing of mesentery vessels, which determines the dynamics of pathomorphologic changes and clinical signs of disease, and the basic place among them belongs to the pain syndrome. Consequently, sudden appearance of disease, acuteness of pain syndrome and ischemic disorders in the wall of bowel cause necrosis changes of area of bowel pulling in a process. It is accompanied by the making progress worsening of the patient

condition and origin of endotoxemia.

Obturation intestinal obstruction (Fig. 5), unlike strangulated, pass not so quickly.

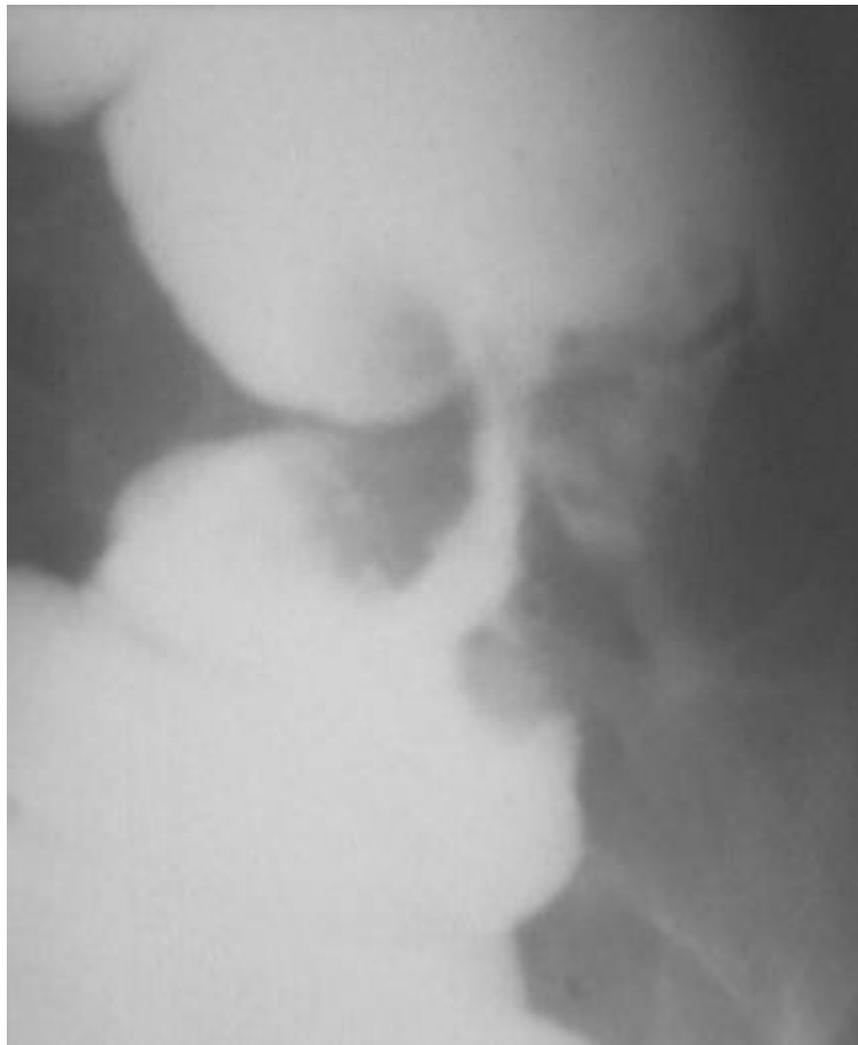


Fig. 5. Obturation mechanical obstruction by tumor.
Irrigogram

In its clinical picture on the first place there are the symptoms of violation of passage on the intestine (protracted intermittent pain, flatulence), instead of symptoms of bowel destruction and peritonitis.

For high, especially strangulated, intestinal obstruction progressive growth of clinical signs of disease and violation of secretory function of intestine is inherent. Thus the volume of circulatory blood diminishes, the level of haematocrit rises and leukocytosis grows. There are also deep violations of homeostasis (hypoproteinemia, hypokalemia, hyponatremia, hypoxia and others like that). In patients with low intestinal obstruction above-named signs are less expressed, and their growth is related

to more protracted passing of disease. Invagination of bowel which can be characterized by the triad of characteristic signs is the special type of intestinal obstruction with the signs of both obturation and strangulation: 1) periodicity of appearance of the intermittent attacks of pain in the abdomen; 2) presence of elastic, insignificantly painful, mobile formation in an abdominal cavity; 3) appearance of blood in the excrement or its tracks (at rectal examination).(Fig.6.)

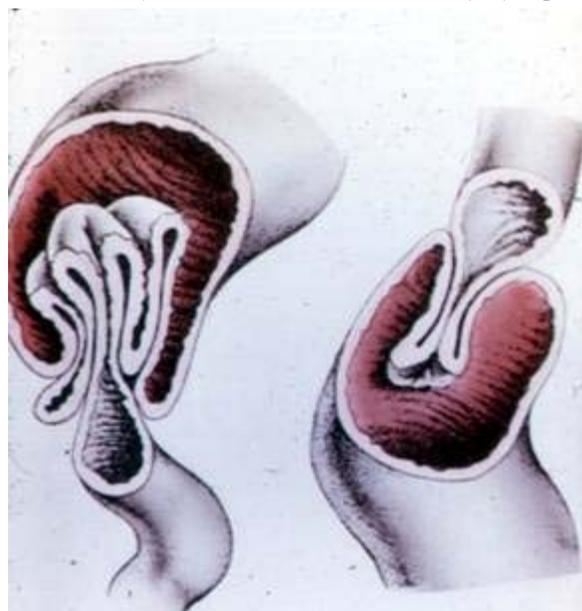


Fig. 6. Invagination of small intestine

The special forms of obturation intestinal obstruction is the obstruction caused by gall-stones. The last are got in the small intestine as a result of bed sore in the walls of gall-bladder and bowel, that adjoins to it. It is needed to mention that intestinal obstruction can be caused by concrement with considerably more small diameter than bowel lumen. The mechanism of such phenomenon is related to irritating action of bilious acids on the bowel wall. The last answers this action by a spasm with the dense wedging of stone in the bowel lumen.

Development of intestinal obstruction caused by gall-stones the attack of colic and clinic of acute cholecystitis precede always. Characteristically, that in the process of development of disease the pain caused by acute cholecystitis calms down, whereupon the new pain characteristic of other pathology — intestinal obstruction appears.

Dynamic intestinal obstruction is divided into paralytic and spastic(Fig.7).

Paralytic obstruction often arises after different abdominal operations, inflammatory diseases of organs of abdominal cavity, traumas and poisonings.



Fig.7. Spastic dynamic intestinal obstruction

The reason of spastic intestinal obstruction can be the lead poisonings, low-quality meal, neuroses, hysterias, helminthiasis and others like that. Clinic of dynamic intestinal obstruction is always variable in signs and depends on a reason, that caused it. Disease is characterized by pain in the abdomen, delay of gases and emptying. During palpation the abdomen is blown away, painful, however soft. To diagnose this form of intestinal obstruction is not difficult, especially, if its etiology is known.

Hemostatic intestinal obstruction (Fig.8) develops after embolism or thromboses of mesenteric arteries and thromboses of veins, there can be mixed forms. Embolism of mesenteric arteries arises in patients with heart diseases (mitral and aortic failings, heart attack of myocardium, warty endocarditis) and declared by damaging, mainly, upper mesentery arteries. Beginning of disease, certainly, is acute, with nausea, sometimes — vomiting. At first there is a picture of acute abdominal ischemic syndrome, that is often accompanied by shock (frequent pulse, decline of arterial and pulse pressure, death-damp, cyanosis of mucus membranes and acrocyanosis). Patients become excitative, uneasy, occupy the forced knee-elbow position or lie on the side with bound legs.



Fig.8. Hemostatic intestinal obstruction. Embolism of upper mesenteric arteries.
Necrosis of small intestines.

During the examination the abdomen keeps symmetry, abdominal wall is soft, the increased peristalsis is heard from the first minutes during 1–2 hours (hypoxic stimulation of peristalsis), which later goes out gradually (“grave quiet”). According to the phenomena of intoxication peritonitis grow quickly. At the beginning of disease the delay of gases and emptying is observed, later there is diarrhea with the admixtures of blood in an excrement. When the last is heavy to set macroscopically, it is needed to explore scourage of intestine.

Small-bowel obstruction (SBO). The most common cause of small-bowel obstruction (SBO) is postsurgical adhesions. Postoperative adhesions can be the cause of acute obstruction within 4 weeks of surgery or of chronic obstruction decades later. The incidence of SBO parallels the increasing number of laparotomies performed in developing countries. Another commonly identified cause of SBO is an incarcerated groin hernia. Other etiologies include malignant tumor (20%), hernia (10%),

inflammatory bowel disease (5%), volvulus (3%), and miscellaneous causes (2%). The causes of SBO in pediatric patients include congenital atresia, pyloric stenosis, and intussusception.

History. Obstruction can be characterized as either partial or complete versus simple or strangulated. No accurate clinical picture exists to detect early strangulation of obstruction.

Abdominal pain, often described as crampy and intermittent, is more prevalent in simple obstruction. Often, the presentation may provide clues to the approximate location and nature of the obstruction. Usually, pain that occurs for a shorter duration of time and is colicky and accompanied by bilious vomiting may be more proximal. Pain that lasts as long as several days, is progressive in nature, and is accompanied by abdominal distention may be typical of a more distal obstruction.

Changes in the character of the pain may indicate the development of a more serious complication (ie, constant pain of a strangulated or ischemic bowel).

Patients also report the following:

- Nausea
- Vomiting - Associated more with proximal obstructions
- Diarrhea - An early finding
- Constipation - A late finding, as evidenced by the absence of flatus or bowel movements
- Fever and tachycardia - Occur late and may be associated with strangulation
- Previous abdominal or pelvic surgery, previous radiation therapy, or both - May be part of the patient's medical history
- History of malignancy - Particularly ovarian and colonic malignancy

Physical Examination

Abdominal distention is present. The duodenal or proximal small bowel has less distention when obstructed than the distal bowel has when obstructed. Hyperactive bowel sounds occur early as GI contents attempt to overcome the obstruction; hypoactive bowel sounds occur late.

Exclude incarcerated hernias of the groin, femoral triangle, and obturator

foramina. Proper genitourinary and pelvic examinations are essential. Look for the following during rectal examination:

- Gross or occult blood, which suggests late strangulation or malignancy
- Masses, which suggest obturator hernia

Check for symptoms commonly believed to be more diagnostic of intestinal ischemia, including the following:

- Fever (temperature $>100^{\circ}\text{F}$)
- Tachycardia (>100 beats/min)
- Peritoneal signs

No reliable way exists to differentiate simple from early strangulated obstruction on physical examination. Serial abdominal examinations are important and may detect changes early.

Complications of SBO include the following:

- Sepsis
- Intra-abdominal abscess
- Wound dehiscence
- Aspiration
- Short-bowel syndrome (as a result of multiple surgeries)
- Death (secondary to delayed treatment)

Large-bowel obstruction (LBO) is an emergency condition that requires early identification and intervention. Approximately 60% of mechanical large-bowel obstructions (LBOs) are caused by malignancies, 20% are caused by diverticular disease, and 5% are the result of colonic volvulus. The most common causes of adult large-bowel obstruction are as follows:

- Neoplasm (benign or malignant)
- Stricture (diverticular or ischemic)
- Volvulus (eg, colonic, sigmoid, cecal)
- Incarcerated hernia
- Intussusception, usually with an identifiable anatomic abnormality in adults

but not in children

- Impaction or obstipation

Gallstone ileus

Neoplasms and diverticular disease

Obstructions caused by tumors tend to have a gradual onset and result from tumor growth narrowing the colonic lumen.

Diverticulitis is associated with muscular hypertrophy of the colonic wall. Repetitive episodes of inflammation cause the colonic wall to become fibrotic and thickened, leading to luminal narrowing.

Volvuli

A colonic volvulus results when the colon twists on its mesentery, which impairs the venous drainage and arterial inflow. Symptoms of this condition are usually abrupt.

A sigmoid volvulus typically occurs in older, debilitated individuals with a history of chronic constipation, or those living in an institutionalized setting.

A cecal volvulus is caused by a congenital defect in the peritoneum, which results in inadequate fixation of the cecum, and increased cecal mobility. Patients usually present with this disorder in the sixth decade of life.

Intussusception

Intussusception is primarily a pediatric disease; however, it is estimated that between 5% and 16% of all intussusceptions in the Western world occur in adults, of which approximately two thirds of adult intussusception cases are caused by tumors. Two main types of intussusception affect the large bowel: enterocolic and colocolic.

Enterocolic intussusceptions involve both the small bowel and the large bowel. These are composed of either ileocolic intussusceptions or ileocecal intussusceptions, depending on where the lead point is located.

Colocolic intussusceptions involve only the colon. They are classified as either colocolic or sigmoidorectal intussusceptions.

Acute colonic pseudo-obstruction/Ogilvie syndrome

Acute colonic pseudo-obstruction (ACPO), or Ogilvie syndrome, has many etiologies. This disorder is typically seen in elderly patients who are hospitalized with a

severe illness. In a retrospective review of more than 1400 cases of acute colonic pseudo-obstruction, the most common predisposing conditions were operative and nonoperative trauma (11%), infections (10%), and cardiac disease (10-18%).

History. Obtain the patient's history of bowel movements, flatus, obstipation (ie, no gas or bowel movement), and symptoms. Attempt to distinguish complete bowel obstruction from partial obstruction, which is associated with passage of some gas or stool, and from ileus. Also inquire about the patient's current and past history in an attempt to determine the most likely cause.

Major complaints in patients with large-bowel obstruction (LBO) include abdominal distention, nausea, vomiting, and crampy abdominal pain. An abrupt onset of symptoms makes an acute obstructive event (eg, cecal or sigmoid volvulus) a more likely diagnosis. A history of chronic constipation, long-term cathartic use, and straining at stools implies diverticulitis or carcinoma.

Changes in the patient's caliber of stools (eg, passage of melanotic bloody stools) strongly suggest carcinoma. When associated with weight loss, the likelihood of neoplastic obstruction increases.

A history of recurrent left lower quadrant abdominal pain over several years is more consistent with diverticulitis, a diverticular stricture, or similar problems.

A history of aortic surgery suggests the possibility of an ischemic stricture.

Diagnostic program

1. Anamnesis and physical methods of examination (auscultation of abdomen, percussion and others like that).
2. General analysis of blood, urines and biochemical blood test.
3. Survey sciagraphy of organs of abdominal cavity.
4. Coagulogramm.
5. Electrocardiography.
6. Irrigography.

Small-Bowel Obstruction Workup Approach Considerations

If the diagnosis is unclear, admission and observation are warranted to detect

early obstructions. Essential laboratory tests are needed; these include the following:

- Serum chemistries - Results are usually normal or mildly elevated
- Blood urea nitrogen (BUN) level - If the BUN level is increased, this may indicate decreased volume state (eg, dehydration)
- Creatinine level - Creatinine level elevations may indicate dehydration
- Complete blood count (CBC) - The white blood cell (WBC) count may be elevated with a left shift in simple or strangulated obstructions; increased hematocrit is an indicator of volume state (ie, dehydration)
- Lactate dehydrogenase tests
- Urinalysis
- Type and crossmatch - The patient may require surgical intervention
- Laboratory tests to exclude biliary or hepatic disease are also needed; they

include the following:

- Phosphate level
- Creatine kinase level
- Liver panels

Studies have been performed to evaluate the use of water-soluble oral contrast as a tool in the management of SBO and as a predictive tool for nonoperative resolution of adhesive SBO. It does not cause resolution of the SBO, but it may reduce the hospital stay in patients not requiring surgery.

Plain Radiography

Obtain plain radiographs first for patients in whom small-bowel obstruction (SBO) is suspected. At least 2 views, supine or flat and upright, are required. Plain radiographs are diagnostically more accurate in cases of simple obstruction. However, diagnostic failure rates of as much as 30% have been reported.

In one small study, the sensitivity of plain radiography was reported to be 75%, and specificity was reported to be 53%; similar findings were reported in a second study. In another study, plain films were more accurate in the detection of acute SBO and the accuracy was higher if interpreted by more-experienced radiologists.

Plain radiography is of little assistance in differentiating strangulation from simple obstruction. Some have used abdominal radiography to distinguish between

complete obstruction and partial or no SBO.

A study by Lappas et al proposed that 2 findings were more predictive of a higher grade or complete SBO: (1) the presence of an air-fluid differential height in the same small-bowel loop and (2) the presence of a mean level width greater than 25 mm. The study found that when the 2 findings are present, the obstruction is most likely high grade or complete. When both are absent, the authors proposed, a low-grade (partial) SBO is likely or nonexistent.

Dilated small-bowel loops with air-fluid levels indicate SBO, as does absent or minimal colonic gas. SBO is demonstrated in the radiographs below.

CT scanning

Computed tomography (CT) scanning is the study of choice if the patient has fever, tachycardia, localized abdominal pain, and/or leukocytosis.

CT scanning is useful in making an early diagnosis of strangulated obstruction and in delineating the myriad other causes of acute abdominal pain, particularly when clinical and radiographic findings are inconclusive. It also has proved useful in distinguishing the etiologies of small-bowel obstruction (SBO), that is, in distinguishing extrinsic causes (such as adhesions and hernia) from intrinsic causes (such as neoplasms and Crohn disease). In addition, CT scanning differentiates the above from intraluminal causes, such as bezoars. The modality may be less useful in the evaluation of small bowel ischemia associated with obstruction.

CT scanning is capable of revealing abscess, inflammatory process, extraluminal pathology resulting in obstruction, and mesenteric ischemia and enables the clinician to distinguish between ileus and mechanical small bowel obstruction in postoperative patients.

The modality does not require oral contrast for the diagnosis of SBO, because the retained intraluminal fluid serves as a natural contrast agent.

Obstruction is present if the small-bowel loop is greater than 2.5 cm in diameter dilated proximal to a distinct transition zone of collapsed bowel less than 1 cm in diameter. A smooth beak indicates simple obstruction without vascular compromise; a serrated beak may indicate strangulation. Bowel wall thickening, portal venous gas, or pneumatosis indicates early strangulation.

One small series reported a sensitivity of 93%, a specificity of 100%, and an accuracy of 94% for CT scanning in the detection of obstructions. Another series reported a sensitivity of 92% and specificity of 71% in the correct identification of partial or complete SBO.

Ultrasonography

Ultrasonography is less costly and invasive than CT scanning and may reliably exclude SBO in as many as 89% of patients; specificity is reportedly 100%.

In a small study by Jang et al in which the use of bedside ultrasonography by emergency physicians was compared with radiography for the detection of small-bowel obstruction (SBO), emergency physician-performed ultrasonography compared favorably with radiography. Dilated bowel on ultrasonography had a sensitivity of 91% and a specificity of 84% for SBO, while radiography had a sensitivity of 46% and a specificity of 66%.

Large-Bowel Obstruction Workup.

Approach Considerations

Laboratory studies are directed at evaluating the dehydration and electrolyte imbalance that may occur as a consequence of large-bowel obstruction (LBO) and at ruling out ileus as a diagnosis.

Routine complete blood cell count (CBC), serum chemistries, and urine specific gravity should be evaluated. A decreased hematocrit level, particularly with evidence of chronic iron-deficiency anemia, may suggest chronic lower gastrointestinal (GI) bleeding, particularly due to colon cancer. A stool guaiac test also should be performed, for similar reasons.

Obtain a prothrombin time (PT) as well as a type and crossmatch.

Although bowel obstruction, or even constipation, may mildly elevate the white blood cell (WBC) count, substantial leukocytosis should prompt reconsideration of the diagnosis. Ileus, secondary to an intra-abdominal or extra-abdominal infection or another process, is a possibility.

The suggestion of an abnormal anion gap (see the Anion Gap calculator) also should prompt an arterial blood gas (ABG) measurement and/or a serum lactate level measurement.

Plain Radiography

Obtain an upright chest radiograph to determine whether free air is present, which would suggest perforation of a hollow viscus and ileus rather than organic obstruction, as well as flat and upright abdominal radiographs, which may demonstrate dilatation of the small and/or large bowel and air-fluid levels.

Chest radiographs will demonstrate free air if perforation has occurred (see the first image below); abdominal radiographs may be diagnostic of sigmoid or cecal volvulus (ie, kidney bean appearance on the radiograph) (see the second and third images below, respectively). Intramural air is an ominous sign that suggests colonic ischemia. The absence of free air does not exclude perforation (this finding may be absent in half of all perforations).

Tracing colonic air around the colon, into the left gutter, and down into the rectum or demonstrating an abrupt cut-off in colonic air suggests the anatomic location of the obstruction.

A dilated colon without air in the rectum is more consistent with obstruction. The presence of air in the rectum is consistent with obstipation, ileus, or partial obstruction. However, this finding can be misleading, particularly if the patient has undergone rectal examinations or enemas.

Radiocontrast Radiography

Contrast studies include an enema with water-soluble contrast (ie, Gastrografin) (see the following images) or computed tomography (CT) scanning with intravenous (IV) and oral (PO) or rectal (PR) contrast. Contrast studies that reveal a column of contrast ending in a "bird's beak" are suggestive of colonic volvulus.

Indications for imaging with contrast

Radiopaque contrast material may be administered and imaging of the colon may be performed under the following circumstances:

- Perform it if the diagnosis of large bowel obstruction is suspected but not proven
- If differentiation between obstipation and obstruction is required
- If localization is required for surgical intervention

Water-soluble contrast vs barium

Water-soluble Gastrografin has important advantages over barium as a contrast agent and generally should be used first. Gastrografin usually does not cause chemical peritonitis if the patient has colonic perforation, and it has an osmotic laxative effect that may actually wash out an obstipated colon.

If large-bowel perforation is ruled out using a Gastrografin study but a more detailed anatomic definition is required (particularly of the right colon), a barium enema may be performed.

Computed Tomography Scanning

Although computed tomography (CT) scanning is useful to help rule out intra-abdominal abscess or other causes of ileus, this imaging modality is generally not used initially in patients with large-bowel obstruction (LBO), unless the diagnosis is still in question.

CT scanning, particularly with rectal contrast, may demonstrate a mass or evidence of metastatic disease. Generally, the findings do not alter management, because these patients will be explored and operatively decompressed, regardless of the CT scan findings.

CT colography may be useful in evaluating these patients, not only to delineate the source of the obstruction but also to rule out synchronous proximal lesions, which may occur in about 1% of patients and which might motivate a more extended resection if identified and if the patient's condition will tolerate the more extensive procedure.

Flexible Endoscopy

Flexible endoscopy preceded by rectal enema may be useful in evaluating left-sided colonic obstruction, including the anatomic location and pathology of the lesion. Because the cecum is not reached in such cases, the endoscopist must be alert to the possibility of incorrectly identifying anatomic landmarks and the location of the obstruction.

An abdominal radiograph with the tip of the endoscope at the site of the

obstruction may be extraordinarily helpful in identifying and documenting the location of the large-bowel obstruction (LBO).

Although flexible endoscopy is relatively comfortable for the patient and provides a better view than rigid sigmoidoscopy, rigid sigmoidoscopy may also be used, depending on the availability of resources and training of personnel.

Right-sided colonic obstruction is more difficult to evaluate without first administering an oral bowel preparation, which is contraindicated in the setting of bowel obstruction.

Differential diagnostics

- Abortion, Threatened
- Alcoholic Ketoacidosis
- Cholangitis
- Cholecystitis and Biliary Colic in Emergency Medicine
- Cholelithiasis
- Constipation
- Diverticular Disease
- Dysmenorrhea
- Endometriosis
- Inflammatory Bowel Disease
- Mesenteric Ischemia

Intestinal obstruction must be differentiated with the acute diseases of organs of abdominal cavity.

The perforation of gastroduodenal ulcer, as well as intestinal obstruction, passes acutely with inherent to it by sudden intensive pain and tension of muscles of abdomen. However, in patients with this pathology, unlike intestinal obstruction, the abdomen is not exaggerated, and pulled in with “wooden belly” tension of muscles of front abdominal wall. There is also characteristic ulcerous anamnesis. Roentgenologic and by percussion pneumoperitoneum is observed. Certain difficulties in conducting of differential diagnostics of intestinal obstruction can arise at atypical passing and in case of the covered perforations.

Acute pancreatitis almost always passes with the phenomena of dynamic

intestinal obstruction and symptoms of intoxication and repeated vomiting, with rapid growth. During the examination in such patients, unlike intestinal obstruction, rigidity of abdominal wall and painfulness is observed in the projections of pancreas and positive Korte's symptom and Mayo-Robson's. The examination of diastase of urine and amylase of blood have important value in establishment of diagnosis.

Acute cholecystitis. Unlike intestinal obstruction, patients with this pathology complain for pain in right hypochondrium, that irradiate in the right shoulder-blade, shoulder and right subclavian area. Difficulties can arise, when the symptoms of dynamic intestinal obstruction appear on the basis of peritonitis.

The clinic of kidney colic in the signs and character of passing are similar to intestinal obstruction, however, attacks of pain in the lumbar area with characteristic irradiation in genital parts, the thigh and dysuric disorders help to set the correct diagnosis. Certain difficulties in conducting of differential diagnostics also can arise in difficult patients, at frequent vomiting which sometimes can be observed in patients with kidney colic.

Table. Characteristics of Ileus, Pseudo-obstruction, and Mechanical Obstruction

	Ileus	Pseudo-obstruction	Mechanical Obstruction (Simple)
Symptoms	Mild abdominal pain, bloating, nausea, vomiting, obstipation, constipation	Crampy abdominal pain, constipation, obstipation, nausea, vomiting, anorexia	Crampy abdominal pain, constipation, obstipation, nausea, vomiting, anorexia
Physical Examination Findings	Silent abdomen, distention, tympanic	Borborygmi, tympanic, peristaltic waves, hypoactive or hyperactive bowel	Borborygmi, peristaltic waves, high-pitched bowel sounds, rushes,

		sounds, distention, localized tenderness	distention, localized tenderness
Plain Radiographs	Large and small bowel dilatation, diaphragm elevated	Isolated large bowel dilatation, diaphragm elevated	Bow-shaped loops in ladder pattern, paucity of colonic gas distal to lesion, diaphragm mildly elevated, air-fluid levels

Tactics and choice of treatment method

During the first 1,5–2 hours after hospitalization of patient complex conservative therapy which has the differential-diagnostic value and can be preoperative preparation is conducted.

Emergency Department Care (SBO)

Initial emergency department (ED) treatment consists of aggressive fluid resuscitation, bowel decompression, administration of analgesia and antiemetic as indicated clinically, early surgical consultation, and administration of antibiotics. (Antibiotics are used to cover against gram-negative and anaerobic organisms.)

Initial decompression can be performed by placement of a nasogastric (NG) tube for suctioning GI contents and preventing aspiration. Monitor airway, breathing, and circulation (ABCs).

Blood pressure monitoring, as well as cardiac monitoring in selected patients (especially elderly patients or those with comorbid conditions), is important.

Nonoperative inpatient care

Continued NG suction provides symptomatic relief, decreases the need for intraoperative decompression, and benefits all patients. No clinical advantage to using a long tube (nasointestinal) instead of a short tube (NG) has been observed.

A nonoperative trial of as many as 3 days is warranted for partial or simple

obstruction. Provide adequate fluid resuscitation and NG suctioning. Resolution of obstruction occurs in virtually all patients with these lesions within 72 hours. Good data regarding nonoperative management suggest it to be successful in 65-81% of partial small-bowel obstruction (SBO) cases without peritonitis. Nonoperative treatment for several types of SBO are as follows:

- Malignant tumor - Obstruction by tumor is usually caused by metastasis; initial treatment should be nonoperative (surgical resection is recommended when feasible)

- Inflammatory bowel disease - To reduce the inflammatory process, treatment generally is nonoperative in combination with high-dose steroids; consider parenteral treatment for prolonged periods of bowel rest, and undertake surgical treatment, bowel resection, and/or stricturoplasty if nonoperative treatment fails.

- Intra-abdominal abscess - CT scan- guided drainage is usually sufficient to relieve obstruction

- Radiation enteritis - If obstruction follows radiation therapy acutely, nonoperative treatment accompanied by steroids is usually sufficient; if the obstruction is a chronic sequela of radiation therapy, surgical treatment is indicated

- Incarcerated hernia - Initially use manual reduction and observation; advise elective hernia repair as soon as possible after reduction

- Acute postoperative obstruction - This is difficult to diagnose, because symptoms often are attributed to incisional pain and postoperative ileus; treatment should be nonoperative

- Adhesions - Decreasing intraoperative trauma to the peritoneal surfaces can prevent adhesion formation

Conservative Management (LBO)

Medical care of colonic obstruction is directed primarily at supporting the patient and treating any comorbid illnesses. This involves resuscitation, correction of fluid and electrolyte imbalances, and nasogastric decompression to temporarily treat the obstruction and to prevent vomiting and aspiration. Medications that slow colonic motility (eg, narcotics, anticholinergics) should be stopped, if possible.

Oral laxatives are contraindicated if large-bowel obstruction is suspected. If any evidence suggests simple constipation, patients should be managed with transrectal enemas. Tap water, isotonic sodium chloride solution, and a variety of other fluids may be used. In patients with renal insufficiency, the physician should be sensitive to the electrolyte content of the fluid.

If the patient's pain is sufficiently severe to merit use of significant analgesics, peritonitis, rather than large-bowel obstruction, should be considered as the first diagnosis.

For a small subset of patients, in whom the obstruction is not only malignant but also reflects substantially disseminated or even inoperable disease, consideration of completely nonoperative palliative therapy within the context of a palliative care or hospice approach may be appropriate. This might include somatostatin therapy and may or may not include nasogastric decompression.

For acute colonic pseudo-obstruction (ACPO), or Ogilvie syndrome, underlying precipitant factors must be identified and corrected. If no perforation is present, pseudo-obstruction is treated with conservative management for the first 24 hours. This includes bowel rest, hydration, and management of underlying disorders.

Pharmacologic treatment of acute colonic pseudo-obstruction with neostigmine or colonoscopic decompression may be effective in cases that do not resolve with conservative management. Colonoscopic decompression may be successful in as many as 80% of patients with acute colonic pseudo-obstruction.

Surgical intervention for acute colonic pseudo-obstruction is associated with a high mortality and morbidity. This treatment is reserved for refractory cases or cases complicated by perforation.

Cleansing enemas

Perform cleansing enemas if obstipation is suspected rather than true large-bowel obstruction. These can also be performed to prepare the distal colon for endoscopic evaluation.

Endoscopic reduction of volvulus

Endoscopic reduction is indicated for sigmoid volvulus when peritoneal signs are

absent, which would imply dead bowel or perforation. This procedure is also indicated when evidence of mucosal ischemia is not present upon endoscopy. An experienced person should perform the procedure.

Endoscopic reduction is not indicated for the less common cecal or transverse colon volvulus.

A rigid sigmoidoscope may be used if a flexible instrument is not available. The endoscopist must have sufficient experience with this technique.

Reduction of a volvulus does not imply cure. The sigmoid usually reinvaginates if definitive treatment is not carried out.

These patients are generally admitted to the hospital, subjected to mechanical bowel preparation, and managed surgically by sigmoid resection, unless contraindications are present.

Reduction of intussusception with barium enema

Barium enema for reduction of intussusception is useful and often successful in children in whom a pathologic leading point for the intussusception is unlikely. This procedure should be performed by an experienced radiologist, because the risk of perforation is significant.

In adults, typically a pathologic leading point for the intussusception is present. Success is far less likely, and patients still require surgery to deal with their pathology.

Endoscopic dilation and stenting of colonic obstruction

Endoscopic dilation and stenting of colonic obstruction is indicated for colonic near-total obstruction through which some small amount of lumen remains. The procedure may be palliative in a high-risk patient with an unresectable malignancy, accepting a risk of reobstruction of the stent, or preparatory to surgical resection.

In cases in which the stent is deployed before surgery, this procedure permits relief of the acute obstruction, resuscitation of the patient, and mechanical bowel preparation before a 1-stage colonic resection and reanastomosis, thus avoiding temporary or permanent colostomy.

Endoscopic dilation and stenting of colonic obstruction should be performed only by an endoscopist experienced in such procedures.

Surgical consultation and backup should be available, as the risk of perforation is increased during attempts at such procedures, with a potentially catastrophic result.

Although some experience with stenting has been positive, with some retrospective preference for the Ultraflex stent over the Wallstent because of ease of placement, a multicenter trial of endoscopic stenting using the Wallstent versus surgery for stage IV left-sided colorectal cancer was terminated early because of an unacceptably high incidence of perforation. Whether this finding reflects the technical aspects of the procedure in that study, the particular stent used, or a truly unacceptable incidence of this dangerous complication awaits further study.

Dietary considerations

Patients with complete large-bowel obstruction should receive nothing by mouth (NPO). Patients with a partial obstruction may tolerate minimal clear liquids, oral medications, and a gradual bowel preparation.

It is directed on warning of the complications related to pain shock, correction of homeostasis and, simultaneously, is the attempt of liquidation of intestinal obstruction by unoperative methods.

1. The measures directed for the fight against abdominal pain shock include conducting of neuroleptanalgesia, procaine paranephric block and introduction of spasmolytics. Patients with the expressed pain syndrome and spastic intestinal obstruction positive effect can be attained by epidural anaesthesia also.

2. Liquidation of hypovolemia with correction of electrolyte, carbohydrate and albuminous exchanges is achieved by introduction of salt blood substitutes, 5–10 % solution of glucose, gelatinol, albumen and plasma of blood. There are a few methods suitable for use in the urgent surgery of calculation of amount of liquid necessary for liquidation of hypovolemia. Most simple and accessible is a calculation by the values of hematocrit. If to consider 40 % for the high bound of hematocrit norm, on each 5 % above this size it is needed to pour 1000 ml of liquids.

3. Correction of hemodynamic indexes, microcirculation and disintoxication therapy is achieved by intravenous infusion of Reopolyhukine and Neohemodes.

4. Decompression of intestine tract is achieved by conducting of nasogastric

drainage and washing of stomach, and also conducting of siphon enema. It is needed to underline that technically the correct conducting of siphon enema has the important value for the attempt of liquidation of intestinal obstruction by conservative facilities, therefore this manipulation must be conducted in presence of a doctor. For such enema the special device is used with the rectal tip, by a PVC pipe by a diameter of 1,5–2,0 cm and watering-can of very thin material. A liquid into the colon is brought to appearance of the pain feeling, then drop the watering-can below the level of patient who lies. The passage of gases and excrement is looked after. As a rule, this manipulation is to repeat repeatedly with the use of plenty of warm water (to 15–20 and more litres).

Liquidating of the intestinal obstruction by such conservative facilities is succeeded in 50–60 % patients with mechanical intestinal obstruction.

Surgical Intervention

Surgical intervention is directed at relieving the obstruction.

A diverting transverse loop colostomy may be the least invasive procedure for a very ill patient with a left colonic obstruction. It permits relief of the obstruction and further resuscitation without compromising chances for a subsequent resection. A case report described the use of hand-assisted laparoscopy via the loop colostomy site for subsequent resection of the obstructing lesion.

A sigmoid colostomy without resection may be used in patients with a rectal obstruction that cannot be managed without a combined abdominoperineal approach. Cecostomy should not be performed, because the diversion is inadequate.

In younger patients without substantial comorbidity, some surgeons would consider primary anastomosis, rather than ileostomy, in the right colon, assuming no intraoperative hypotension, blood loss, or other complications are present.

If resection and proximal colostomy or ileostomy are performed, a mucous fistula is generally extracted from the distal end, unless the obstruction is rectosigmoid, in which case the distal end may be oversewn or stapled and left to drain transanally.

If the cause of the obstruction can be relieved nonsurgically, through procedures such as decompressing a volvulus, or if the obstruction is only partial, deferring surgery temporarily and supporting the patient while the large bowel is cleansed so that primary

anastomosis may be performed more safely is preferable.

A slow preoperative mechanical bowel preparation is indicated for patients who have incomplete obstruction, provided the patient can tolerate it. Some authors prefer polyethylene glycol solutions, such as GO-LYTELY, because they avoid issues of fluid and electrolyte imbalance. The fluid should be administered slowly (rather than given in the standard manner of 1 gal over 4 h), and the patient should be observed for abdominal cramping and intolerance.

Carcinoma

In most patients, the obstructing lesion is resected. Because the colon has not been cleansed, anastomosis is often risky. After resection, most surgeons perform a proximal colostomy if the obstruction is on the left side or ileostomy if it is on the right side.

In patients with substantial comorbidity and surgical risk or in the presence of an unresectable tumor, a diverting proximal colostomy or ileostomy may be performed without resection.

Left vs right colon carcinoma. Surgical treatment of left colon carcinoma includes resection without primary anastomosis or resection with primary anastomosis and intraoperative lavage. Endoscopically placed expandable metal stents can be used to relieve the large-bowel obstruction, thus allowing for a primary colorectal anastomosis.

Right colonic obstructions are treated with a right colectomy and a primary anastomosis between the ileum and the transverse colon. Patients with high-risk features for surgery (advanced age, complete obstruction, or severe comorbidities) may benefit from stent placement until patient can be optimized for a surgical procedure. Palliative colorectal stents are an option in patients who are poor surgical candidates or have advanced cancer.

Diverticulitis

Patients with persistent obstruction secondary to diverticular disease despite appropriate medical management are treated surgically. Surgical resection follows the same principles as the treatment of carcinomas. Elective colonic resection is offered to patients with recurrent disease.

Sigmoid vs cecal volvulus

Sigmoidoscopy with volvulus reduction is the procedure of choice for sigmoid volvuli. Second choice is sigmoid colectomy.

The primary treatment of cecal volvuli is also surgical. A cecopexy often needs to be performed to prevent recurrence. Second choice is colonoscopy, due to the high risk of colonic perforation.

Intussusception

Adult colonic intussusception is treated with primary colon resection without prior reduction.

Patients with dynamic paralytic intestinal obstruction are expedient to stimulation of peristalsis of intestine to be conducted, besides, necessarily after infusion therapy and correction of hypovolemia. A lot of kinds of stimulation of intestine peristalsis are offered. Most common of them are:

1) hypodermic introduction of 1,0 ml of 0,05 % solution of proserin; 2) through 10 min — 60 ml intravenously stream of 10 % solution of chlorous sodium; 3) hypertensive enema.

Surgical treatment of intestinal obstruction must include such important moments:

1. According to middle laparotomy executed the novocaine blockade of mesentery of small and large intestine and operative exploration of abdominal cavity organs during which the reason of intestinal obstruction and expose viability of intestine is set.(Fig.9)



Fig.9. Intestinal obstruction.

Overblowing of small intestine

The revision at small intestine obstruction begins from the Treitz' ligament to iliocecal corner. At large intestine obstruction the hepatic, splenic and rectosigmoid parts are observed intently. Absence of pathological processes after revision needs the examination of places of cavity and jamming of internal hernia: internal inguinal and femoral rings, obturator openings, pockets of the Treitz' ligament, Winslow's opening, diaphragm and periesophageal opening.

2. Liquidation of reasons of obstruction (scission of connection, that squeezes a bowel, violence of volvulus and node formation of loops, desinvagination, deleting of obturative tumours and others like that).

It is needed to mark that the unique method of liquidation of acute intestinal obstruction does not exist. At the lack of viability of bowel the resection of nonviable area is executed with 30–40 cm of afferent and 15–20 cm of efferent part with imposition of “side-to-side”(Fig. 10; Fig. 11) anastomosis or “end-to-end”(Fig.12).

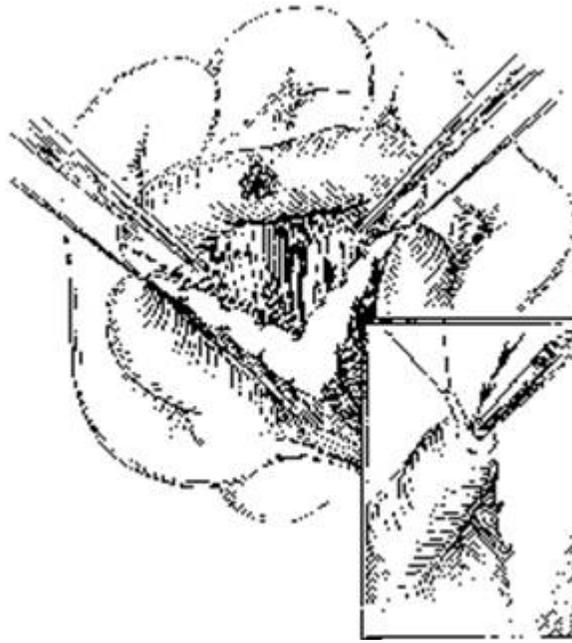


Fig 10. The resection of the small intestine with imposition of anastomosis "side – to side"

- mobilization of segment of the small intestine
- peritonization of the stump of bowel

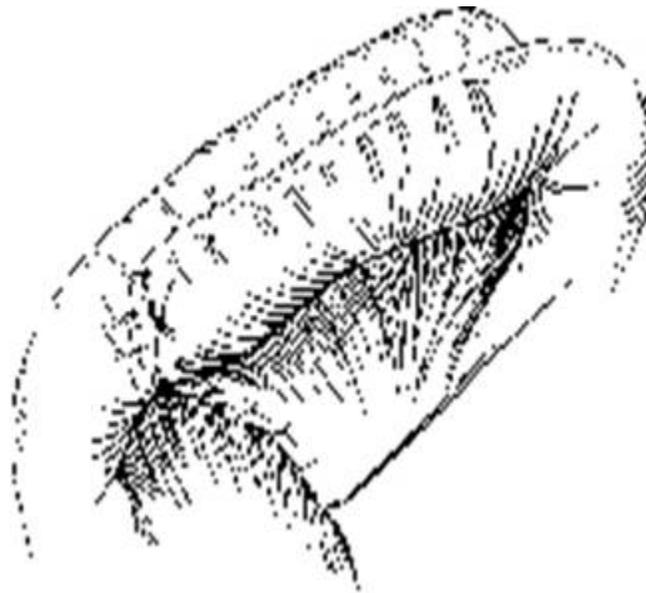


Fig 11. Enteroenteroanastomosis "side-to-side" is formed.



ABC

Fig 12. The resection of the small intestine with imposition of “end-to-end” anastomosis

A- mobilization and removing of the changed loop of the bowel

B- formation of the back lip of anastomosis

C- Final view of enteroenteronastomosis

3. Intubation. (Fig.13) Decompression of intestine foresees conducting in the small intestine of elastic probe by thickness of 8–9 mm and length of 3–3,5 m with the plural openings by a diameter 2–2,5 mm along all probe, except for part, that will be in the oesophagus, pharynx and outwardly. A few methods of conducting of probe are offered in a bowel (nasogastric, through gastrostomy, ceco- or appendicostoma). Taking it into account, such procedure needs to be executed individually and according to indications.



Fig. 13. Nasogastrintestinal probe

Each of them has the advantages and failings. In connection with the threat of origin of pneumonia, entering an intubation probe to the patients of old ages is better by means of gastrostomy. Most surgeons avoid the method of introduction of probe through ceco-appendicostoma because of technical difficulties of passing in a small intestine through a Bauhin's valve.(Fig.14)

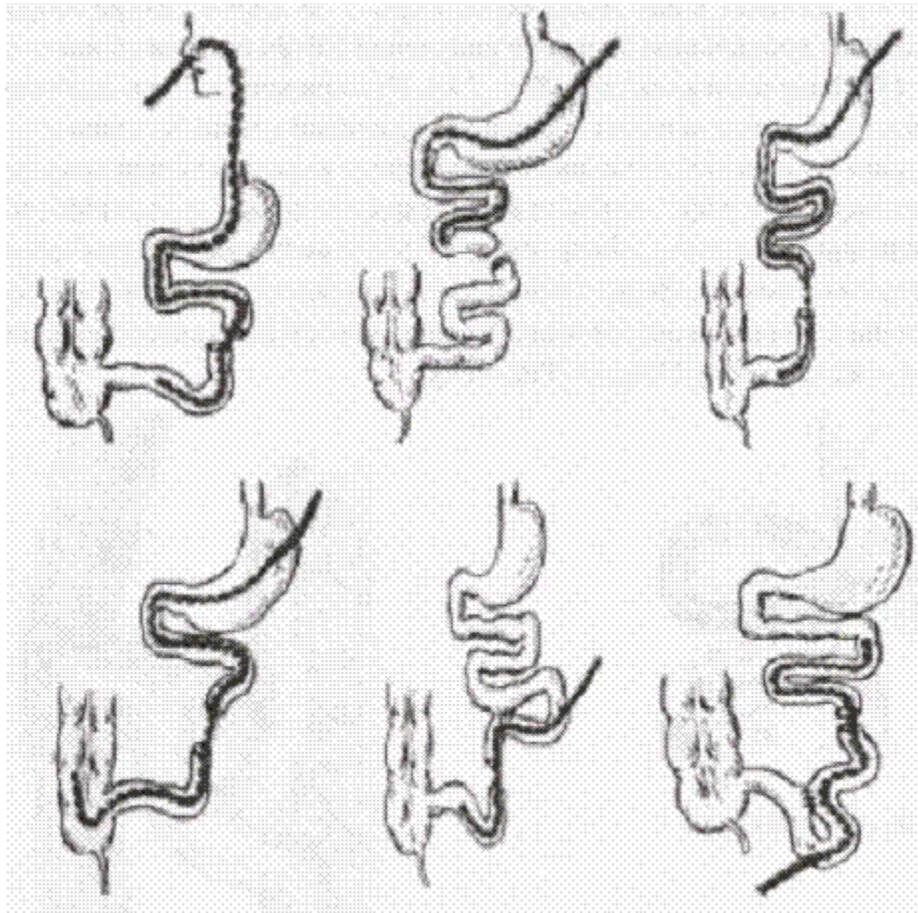
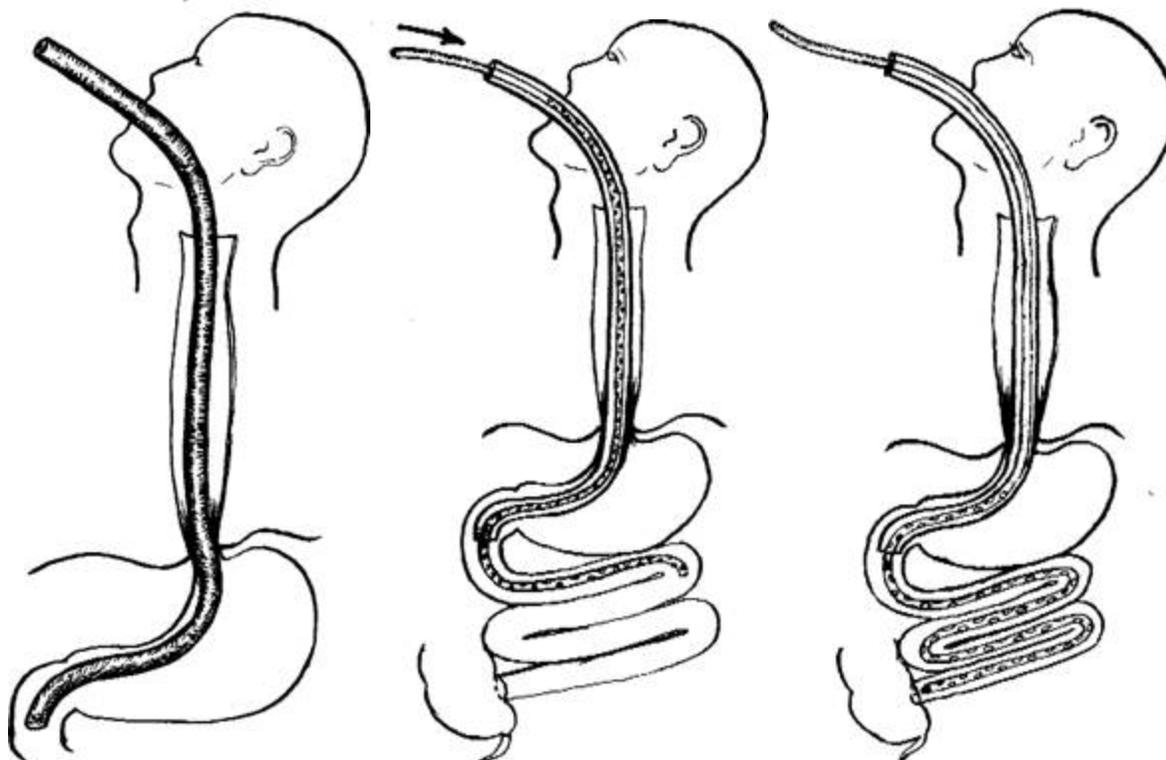
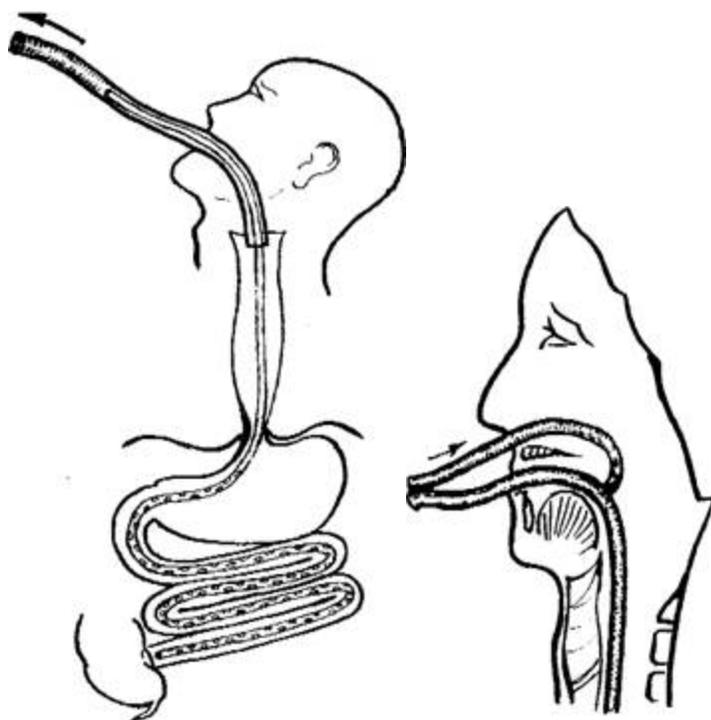


Fig.14. Types of decompression of digestive tract

Today the most wide clinical application has intubation of intestine extracted by the nasogastric method with the use of other thick probe as explorer of the first (by L.J. Kovalchuk, 1981)(Fig.15). Such method not only simplifies procedure of intubation but also facilitates penetration through the piloric sphincter and duodenojejunal bend, and also warns passing of intestinal maintenance in a mouth cavity and trachea. Thus probe is tried to be conducted in the small intestine as possible farther and deleted the next day after appearance of peristalsis and passage of gases, however not later than on 7th days, because more protracted sign of probe carries the real threat of formation of bedsores in the wall of bowel.

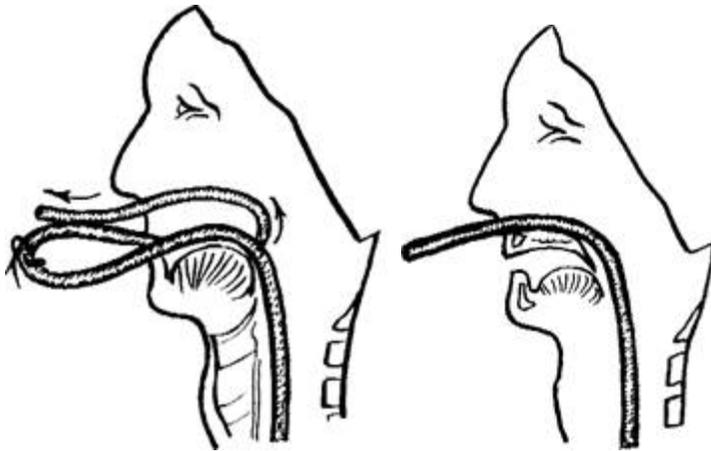


1. Gastric probe-guide is placed distally to pylorus per os
 2. Beginning of intubation
 3. Intubation till caecum

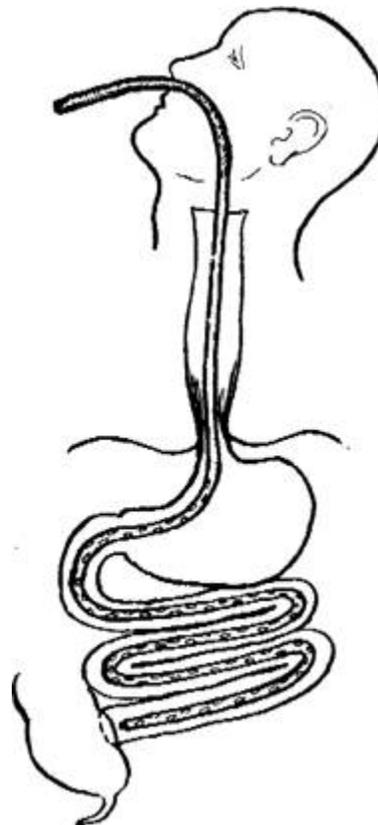


4. Removing of gastric probe
 5. Fixation of proximal part

of intestinal probe to the nasal catheter



6. Removing of proximal part of intestinal
7. Proximal part is removed through the nose probe from the oral cavity through the nose



8. Final view of nasogastric intubation

Fig. 15. Principles of nasogastric intubation

4. Sanation and draining of abdominal cavity is executed by the generally accepted methods of washing of antiseptic. Draining of the abdominal cavity it is needed from four places: in both iliac areas and both hypochondrium, better by the coupled synthetic drain pipes.

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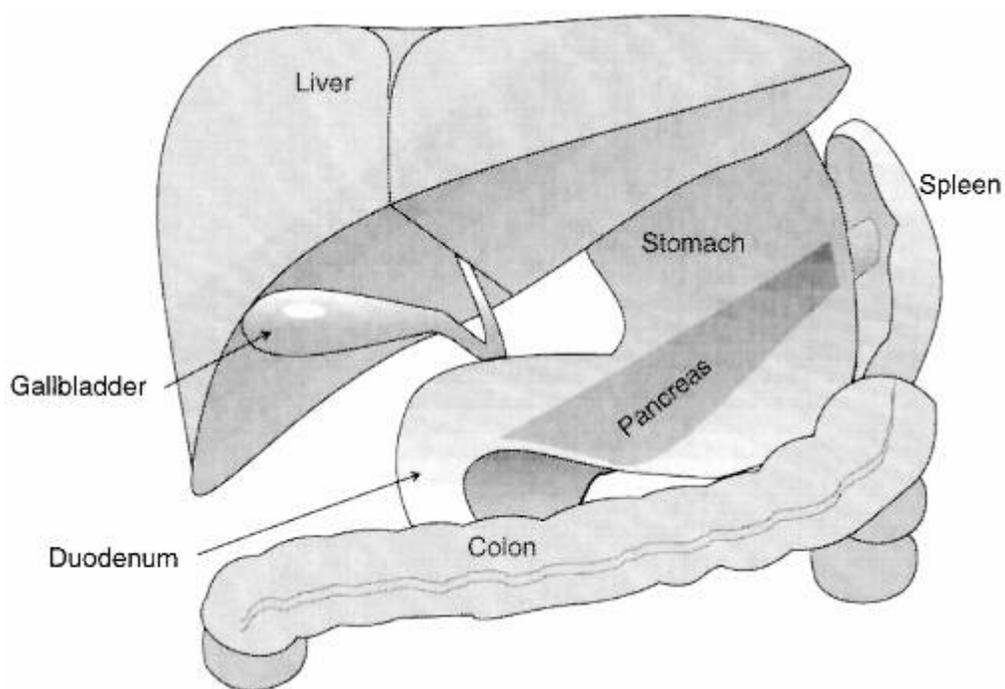
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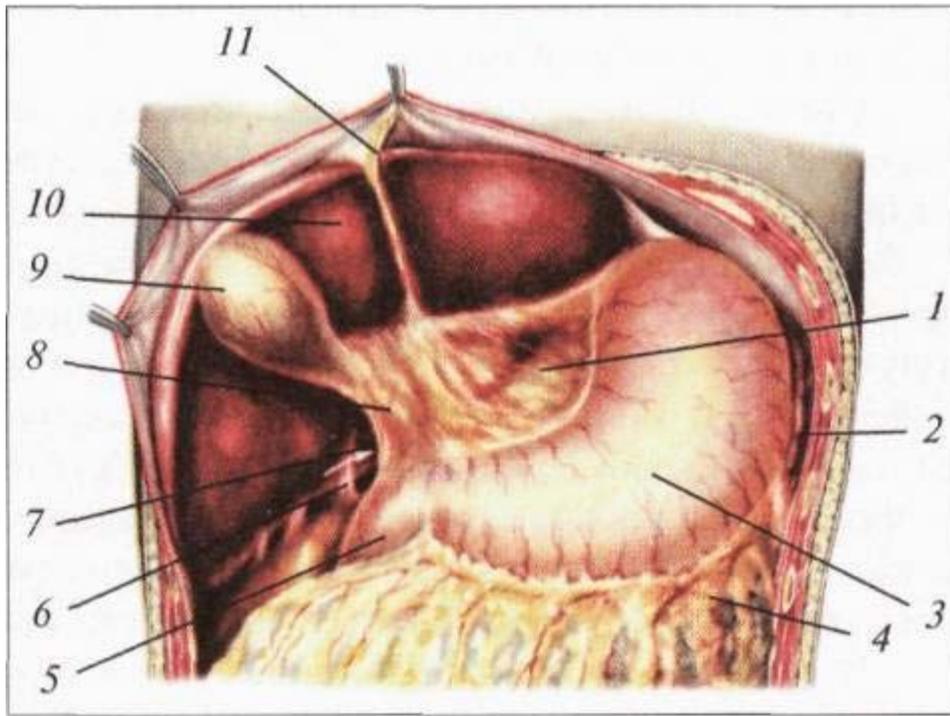
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MODERN PRINCIPLES OF DIAGNOSTICS OF ULCERATIVE DISEASE OF THE STOMACH AND THE DUODENUM. POSTRESECTION AND POSTVAGOTOMY SYNDROMES. CLASSIFICATION. CLINICS. DIAGNOSTICS. CONSERVATIVE AND SURGICAL TREATMENT. PROPHYLAXIS.

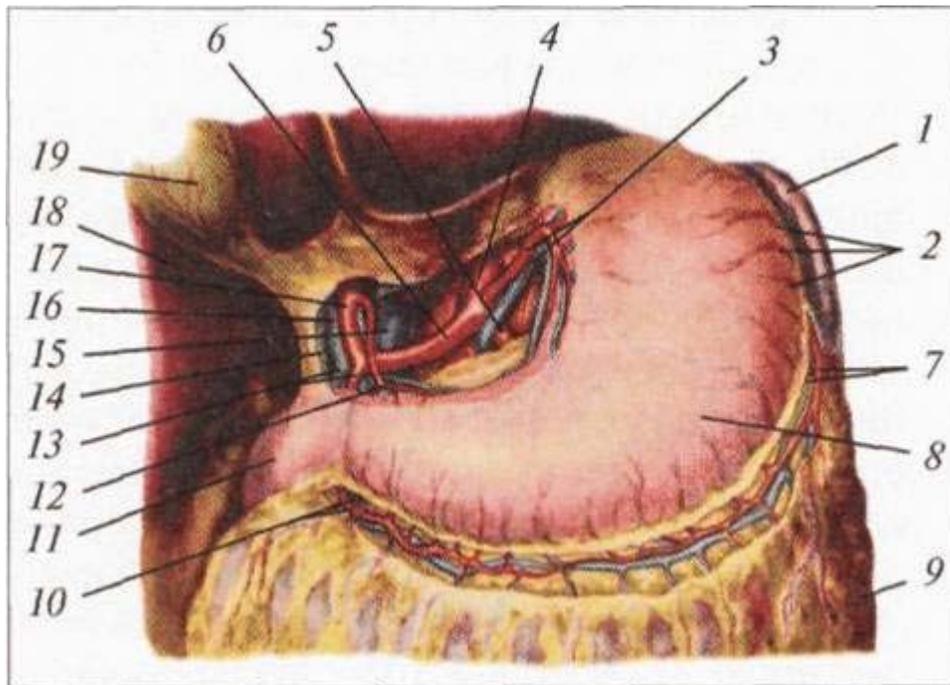
GASTRIC ANATOMY AND PHYSIOLOGY



Pic. The anatomic relationships in the upper abdomen. The stomach is bounded on its left by the spleen, posteriorly (dorsally) by the pancreas, inferiorly (caudally) by the colon, and to its right by the duodenum along the liver's edge.

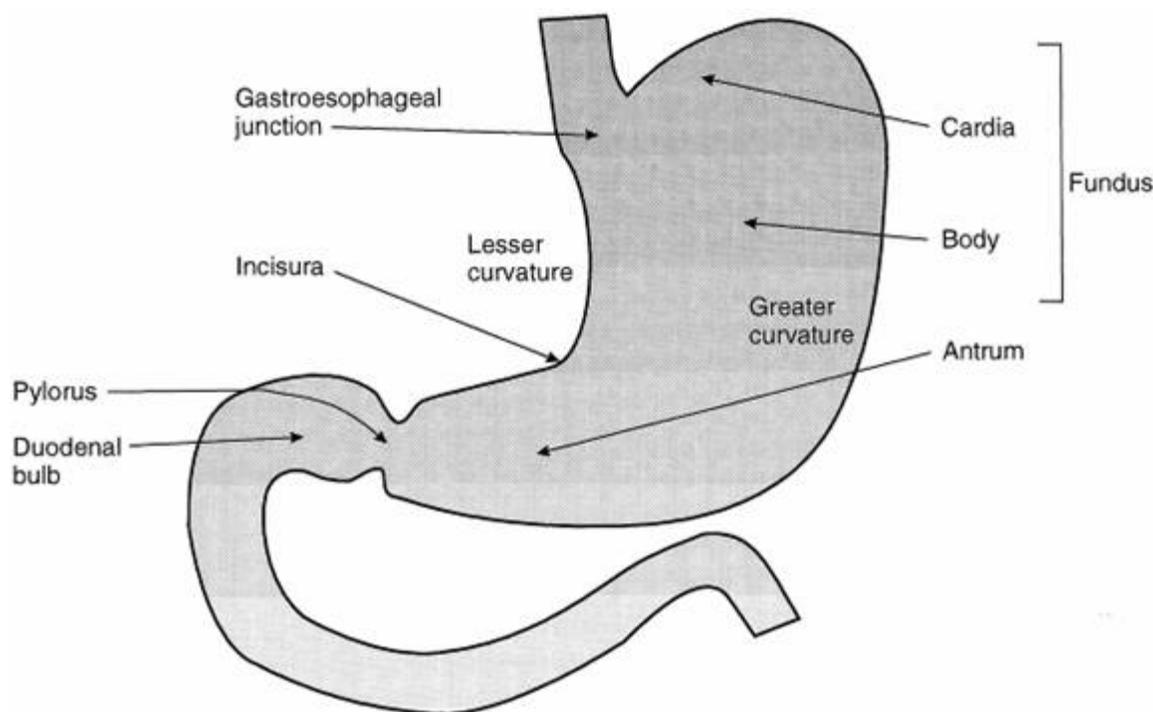


Pic. 1 — lig. hepatogastricum; 2— lien; 3— gaster; 4— lig. gastrocolicum; 5— duodenum; 6— lig. hepatorenale; 7— foramen epiploicum (Winslovi); 8 — lig. hepatoduodenale; 9 — vesica fellea; 10 — hepar; 11 — lig. teres hepatis



Pic. 1 — lien; 2 — aa. et vv. gastricae breves; 3 — a. et v. gastrica sinistra; 4 —

truncus coeliacus; 5-a. lienalis; 6-a. hepatica communis; 7 — a. et v. gastro-epiploica sinistra; 8—gaster; 9— omentum majus; 10—a. et v. gastromentalis dextra; 11 — duodenum; 12—a. et v. gastrica dextra; 13-a, et v. gastroduodenalis; 14 — ductus choledochus; 15 — v. cava inferior; 16— v. portae; 17 — a. hepatica propria; 18 — hepar; 19 — vesica fellea



Pic. Anatomically, the stomach is divided into several segments. Functionally, the cardia and the antrum differ from the body in that they contain no acid secretory properties. The incisura is an area on the lesser curvature, which marks the antrum-body junction and is often easily seen on barium upper intestinal series.

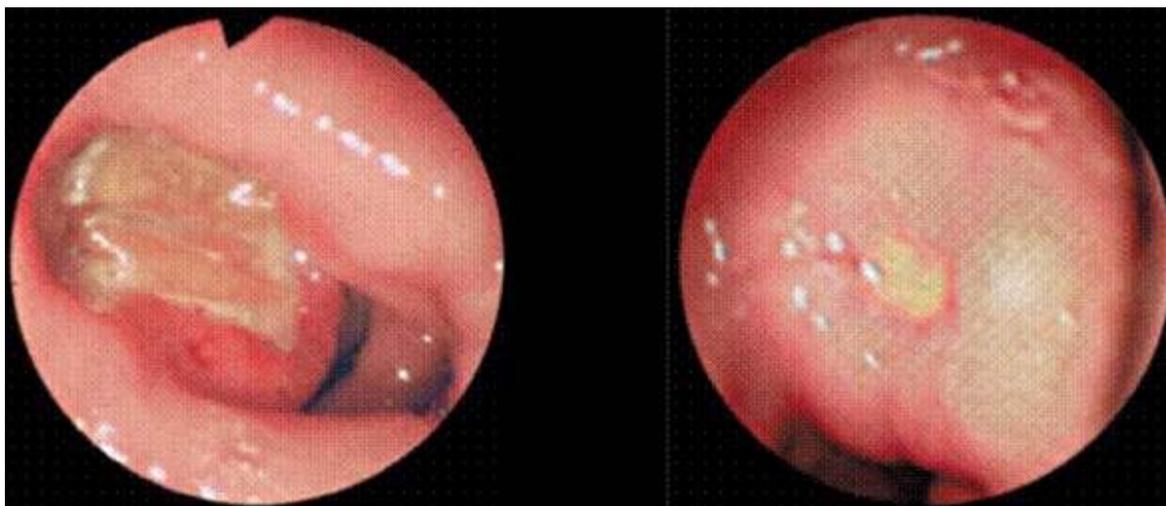
[video](#)

GASTRIC ULCER

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The gastric ulcer is the chronic disease with polycyclic passing. The main typical of peptic ulcer is the presence of ulcerous defect in a mucous tunic. One of basic places belongs among the gastroenterology diseases to this pathology. Such phenomenon

explained by not only considerable distribution of disease but also those dangerous complications which always accompany gastric ulcers.



Pic. The presence of ulcerous defect in a mucous tunic.

Etiology and pathogenesis

Frequency of morbidity on the peptic ulcer among the adult population is about 4 %. More frequent age in patients with gastric ulcers is 50–60 years.

To development mechanism of disease is still not enough studied. From a plenty of different theories in relation to genesis of peptic ulcer no one able to explain the disease. So, each of such factors as neurogenic, mechanical, inflammatory, vascular is present in the mechanism of development of peptic ulcer. Consider for today, that disturbance between the factors of aggression and defense of mucous tunic arose peptic ulcer. To the first factors belong: hydrochloric acid, pepsin, reverse diffusion of ions of hydrogen, products of lipid hyperoxidizing. To the second: mucus and alkaline components of gastric juice, property of epithelium of mucous tunic to permanent renewal, local blood flow of mucous tunic and submucous membrane.

In the terminal stage of mechanism of origin of gastric ulcers important role has the peptic factor and disturbance of trophism of gastric wall as a result of local ischemia. It confirmed by decreasing of blood flow in the wall of stomach at patients with ulcers on

30–35 % compared to the norm. It is proved, that a local and functional ischemia more frequent arises up on small curvature of stomach in the areas of ectopy of the antral mucous tunic in acid-forming. Exactly there ulcers appear.

Important part in ulcerogenesis is acted by duodenogastric reflux and gastritis. Also, gastrostasis can provoke hypergastrinaemia and hypersecretion and formed gastric ulcers.

Numeral scientific developments of the last years testify to the important infectious factor in the mechanism of origin of peptic ulcer conditioned, mainly, by helicobacter pylori.

Pathomorphology

Such stages of disease are distinguished: erosion, acute and chronic ulcers.

Erosions, mainly, are plural. Their bottom as a result of formation of muriatic haematine is black, edges — infiltrated by leucocytes. A defect usually does not penetrate outside muscular tissue of the mucous tunic. If necrosis gets to more deep layers of wall of stomach, a acute ulcer develops. It has a funnel-shaped form. Bottom is also black, edges is swelled. Chronic ulcers are mainly single, sometimes arrive to the serous layer. A bottom is smooth, sometimes hilly, edges is like elevation, dense.

Classification

For today the most known classification of gastric ulcers by Johnson (1965). There are three types of gastric ulcers are distinguished: I – ulcers of small curvature (for 3 cm higher from a goalkeeper); II– double localization of ulcers simultaneously in a stomach and duodenum; III – ulcers of goalkeeper part of stomach (not farther as 3 cm from a goalkeeper). In the area of small curvature of body of stomach is localized 70,9 % ulcers, on a back wall, nearer to small curvature — 4,8 %, in the area of cardial part – 12,9 %, in a goalkeeper part — 11,4 %. The ulcers of large curvature of stomach are casuistry and meet infrequently.

Clinical management

The complaints of patients with the gastric ulcer always give valuable information about the disease. The detailed analysis of their anamnesis allows to pay attention to the possible reasons of origin of ulcer, time of the first complaints, to the changes of symptoms.

Pain. A pain symptom in the peptic ulcer disease is very important. There are typical passing for this disease: hunger – pain – food intake – facilitation – again hunger – pain – food intake – facilitation (so during all days). Night pain for the gastric ulcer is not typical. The such patients rarely wake up in order to take a food. For diagnostics of ulcer localization it is important to know the time of appearance of pain. Between acceptance of food and appearance of pain it is the shorter, than the higher placed gastric ulcer. Thus, at patients with a cardial ulcer pain arises at once after the food intake, with the ulcers of small curvature — in 50–60 minutes, at pyloric localization — approximately in two hours. However this feature it is enough relative and some patients in general do not mark dependence between food intake and pain. In other patients the pain attack is accompanied by the salivation.

A epigastric region near the xiphoid process is typical localization of pain. The irradiation of pain is not usual for gastric ulcers. Irradiation occur in patients with penetration and depended from organ, in which an ulcer penetrates.

At the examination of ulcerous patient it is expedient to determine the special pain points: *Boas* (pain at pressure on the left of the X–XII pectoral vertebrae), *Mendel* (pain at percussion on the left to epigastric region).

Vomiting, the sign of disturbance of motility function of stomach, is the second typical symptom of gastric ulcer. More frequent gastrostasis arises as a result of failure of stomach muscular, it atony which can be effect of organ ischemia. Vomiting could arises both on empty stomach and after food intake.

Heartburn is one of early symptoms of gastric ulcer, however at the prolonged passing of disease it can be hidden or quite disappear. Often it precedes of pain arising (initial heartburn) or accompanies a pain symptom. Mostly heartburn arises after the food intake, but can appear independently. it is observed not only at hypersecretion of the

hydrochloric acid, but at normal secretion, even reduced acidity of gastric juice.

The belching at gastric ulcers is examined rarely, more frequent in patients with cardial and subcardial ulcers. It is necessary to bind to disturbance of function of cardial valve.

The general condition of patients with the uncomplicated gastric ulcer usually satisfactory, and in a period between the attacks — even good. However for most patients lost of the body weight and pallor are typical. In a epigastric region hyperpigmental spots are examined after the prolonged application of hot-water bottle. At palpation of stomach in this area sometimes appears local painful. It is needed also to check up “noise of splash”, the presence of which can be the sign of possible gastrostasis.

At the examination of mouth cavity a tongue has whiter-yellow incrustation. In patients with penetration ulcers and disturbances evacuations from a stomach examined dryness of tongue.

Stomach, as a rule, regular rounded shape, however during the pain attack is pulled in. There is antiperistalsis arises during the pylorostenosis.

The increased secretion of hydrochloric acid in patients with gastric ulcers observed rarely and, mainly, at prepyloric ulcer localizations. Mostly secretion is normal, and in some patients is even reduced.

X-Ray examination. The direct signs of ulcer at X-Ray examinations are: symptom of “Haudek's niche” (Pic. 3.2.1), ulcerous billow and convergence of folds of mucous tunic. Indirect signs: symptom of “forefinger” (circular spasm of muscles), segmental hyperperistalsis, pylorospasm, delay of evacuation from a stomach, duodenogastric reflux, disturbance of function of cardial part (gastroesophageal reflux).

Gastroscopy can give important information about localization, sizes, kind of ulcer, dynamics of its cicatrization, and also allow to perform biopsy with subsequent histological examination.

Clinical variants and complication

The gastric ulcer passing can be acute and chronic. Acute ulcers arise as answer for the stress situations, related to the nervous overstrain, trauma, loss of blood, some

infectious and somatic diseases. By a diameter ulcers has from a few millimeters to centimeter, a round or oval form with even edges. Thus in most cases clinically observed clear ulcerous clinical signs. If complications is absent (bleeding, perforation) such ulcers treated and mostly heal over.

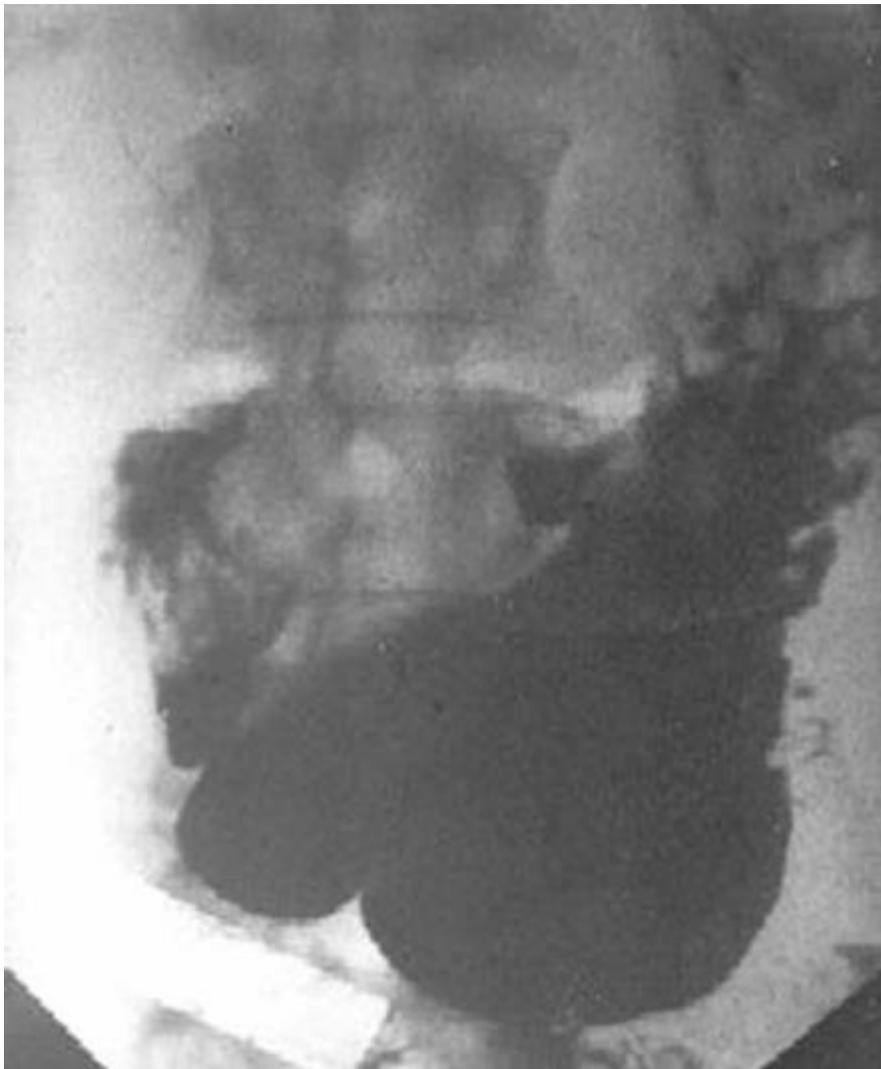
G.J. Burchynskyy (1965) such variants of clinical flow distinguished:

1. Chronic ulcer which does not heal over long time.
2. Chronic ulcer which after the conservative therapy heals over relatively easily, however inclined to the relapses after the periods of remission of a different duration.
3. Ulcers, which localization are had migrant character. Observed in people with acute ulcerous process of stomach.
4. Special form of gastric ulcer passing after the already carried disease. Passed with the expressed pain syndrome. Characterized by the presence in place of ulcerous defect of scars or deformations and absence of symptom of “niche”.

There are such complications can develop in patients with gastric ulcer: penetration, stenosis, perforation, bleeding and malignization.

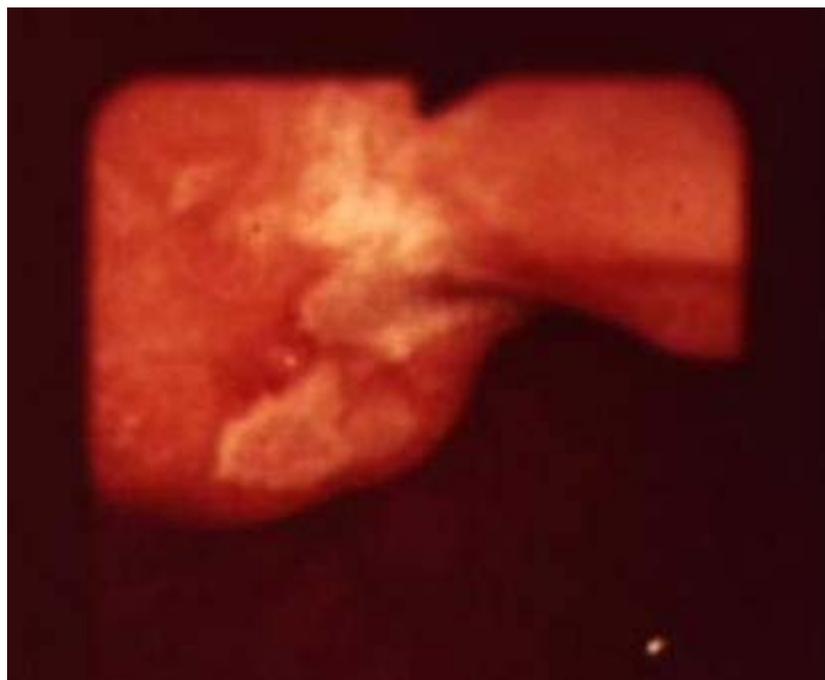
Diagnosis program

1. Anamnesis and physical examination.
2. Endoscopy.
3. X-Ray examination of stomach.
4. Examination of gastric secretion by the method of aspiration of gastric contents.
5. Gastric pH metry.
6. Multiposition biopsy of edges of ulcer and mucous tunic of stomach.
7. Gastric Dopplerography.
8. Sonography of abdominal cavity organs.
9. General and biochemical blood analysis.
10. Coagulogram.





Pic. Symptom of "Haudek's niche"



Pic. Peptic ulcer of the stomach (endoscopy)

Differential diagnostics

Chronic gastritis, as well as at an gastric ulcer, characterized by the pain

syndrome, that arises after the food intake. In such patients it is possible to observe nausea and vomiting by gastric content, heartburn and belch. However, unlike an gastric ulcer, for gastritis typical symptom of “quick satiation by a food”. Unsteady emptying, diarrhea also more inherent to gastritises. At gastric ulcer more frequent the delays are observed, constipation for 4–5 days.

The *cancer of stomach*, it is comparative with an gastric ulcer, has considerably more short anamnesis. The most typical clinical signs of this pathology are: absence of appetite, weight loss, rapid fatigability, depression, unsociability, apathy. In such patients X-Ray examination expose the “defect of filling”, related to exophytic tumor and deformation of walls of organ. A final diagnosis is set after the results of multiposition biopsy of shady areas of mucous tunic of stomach.

Differential diagnostics also needs to be conducted with the so called precancerous states: gastritis with the achlorhydria; chronic, continuously recurrence ulcers, poliposis and Addison-Biermer anemia.

Tactic and choice of treatment method

Conservative treatment of gastric ulcer always must be complex, individually differentiated, according to the etiology, pathogeny, localization of ulcer and character of clinical signs (disturbance of functions of gastroduodenal organs, complication, accompanying diseases).

Conservative therapy must include:

- Omeprazole 20 mg 2 time per day or H₂- blocker histamine receptor (ranitidine) — 150 mg in the evening, famotidine — 40 mg at night, roxatidine — 150 mg in the evening
- antacid drugs — in accordance with the results of pH-metry;
- reparative drugs (dalargin, solcoseryl, actovegin) — for 2 ml 1–2 times per days
- antimicrobial drugs (clarytromicine 500 mg twice daily, de-nol, metronidazole)

Treatment of patient with a gastric ulcer must continue not less than 6–8 weeks.

Surgical treatment must be performed in cases:

- a) at the relapse of ulcer after the course of conservative therapy;
- б) in the cases when the relapses arise during supporting antiulcer therapy;
- в) when an ulcer does not heal over during 1,5–2 months of intensive treatment, especially in families with “ulcerous anamnesis”.
- г) at the relapse of ulcer in patients with complications (perforation or bleeding);
- д) at suspicion on malignization ulcers, in case of negative cytological analysis.

The choice of method of surgical treatment of gastric ulcer depended from localization and sizes of ulcer, presence of gastro- and duodenostasis, accompanying gastritis, complications of peptic ulcer (penetration, stenosis, perforation, bleeding, malignization), age of patient, general condition and accompanying diseases. In patients with cardial localization of ulcer the operation of choice is the proximal resection of stomach, which, from one side, allows to remove an ulcer, and from other — to save considerable part of organ, providing it functional ability (Pic. 3.2.2). In case with large cardial ulcers, when the vagus nerves pulled in the inflammatory infiltrate and it is impossible to save integrity even one of them, operation needs to be complemented by pyloroplasty. It will give possibility to warn pylorospasm and gastrostasis, which in an early postoperative period can be the reason of anastomosis insufficiency and other complications.

At the choice of method of surgical treatment of gastric ulcers with *subcardial localization* on small curvature without duodenostasis it is better to apply the methods of stomach resection with saving of passage through a duodenum.

For this purpose we have developed the method of segmental resection of stomach with addition selective proximal vagotomy. The redistribution of gastric blood flow between the functional parts of stomach as reply to medicinal vagotomy (intravenous introduction 1,0 ml 0,1 % solution of atropine of sulfate) is studied. Hyperemia of acid-forming part of stomach comes after introduction of preparation. The functional scopes of stomach parts are determined. The border between acid-forming and antral parts are the most frequent localization of gastric ulcers.

During this operation middle laparotomy is performed, intravenously entered 1,0 ml 0,1 % solution of atropine, then the scopes of functional stomach parts are identified and by stitches-holders is marked a intermedial segment. Selective proximal vagotomy is performed. After mobilization of large curvature of stomach within the limits of intermedial segment it resection is performed. After that gastro-gastro anastomosis “end-to-end” is formed (Pic. 3.2.3).

The analysis of supervisions of the patients operated by such method in postoperative period has good results. It allows to recommend this operation for clinical practice, in case of gastric ulcers of subcardial localizations, without duodenostasis, penetration, malignization or nerves Latarjet damaging.

The operation of choice in patients with subcardial ulcers and duodenostasis is gastric resection by Billroth II.

At the choice of method of surgical treatment of ulcers which are localized in upper and middle third of stomach, it is necessary to consider such factors, as absence of penetration in a small omentum and absence of the duodenostasis. In such patients is performed segmental resection of stomach with ulcer removing with selective proximal vagotomy. In case of penetration ulcer in a small omentum with involvement in infiltrate Latarjet nerves, such operation is impossible because of future spasm of pylorus and gastrostasis. If duodenostasis is absence than better to apply pylorus-saving resection by Maki-Shalimov. In patients with duodenostasis better to apply gastric resection by Billroth II.

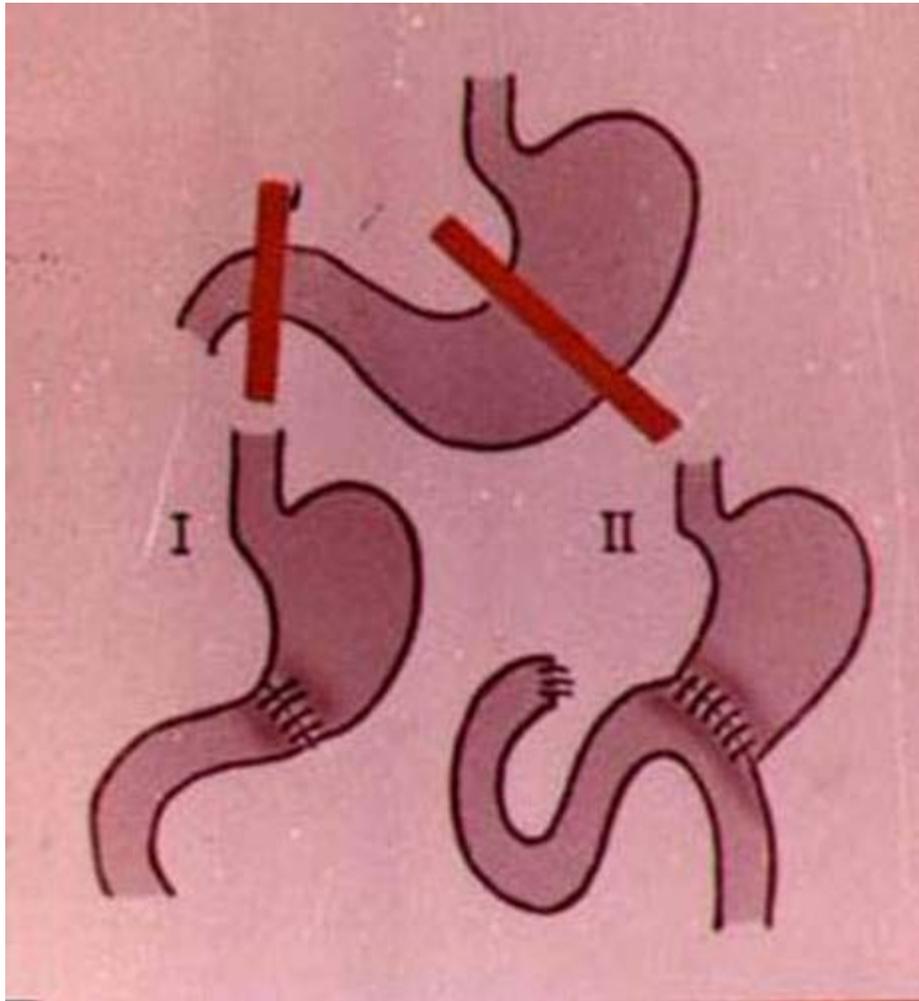
At the border of gastric resection near pyloric sphincter can be spasm and gastrostasis in a postoperative period . Avoiding such complication is possible, if this border of gastric resection passes no more than 1,5 cm from a pyloric sphincter (M.M. Risaev, 1986). So, at a resection, that passes higher than 2,0 cm from a pylorus, integrity of both loops is kept.

Patients with antral ulcers without the duodenostasis performed the gastric resection by Billroth I (Pic. 3.2.6), and on presence of duodenostasis – Billroth II.

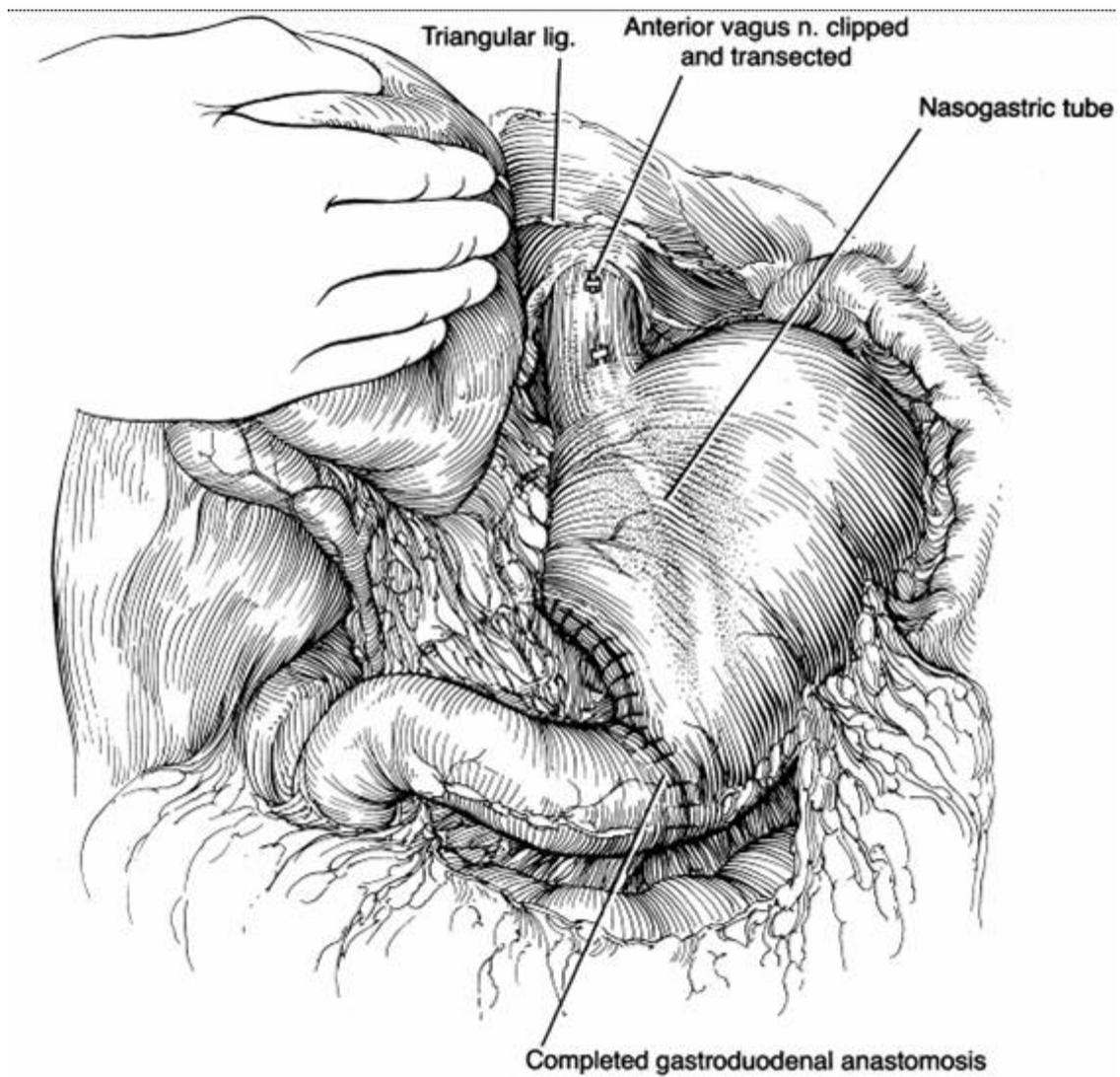
Prepyloric ulcers is similar to the ulcers of duodenum. Such localization of gastric ulcers without malignization allow to perform selective proximal vagotomy. However, at

large prepyloric ulcers with penetration without duodenostasis is better to perform the gastric resection by Billroth I and on presence of duodenostasis – by Billroth II.

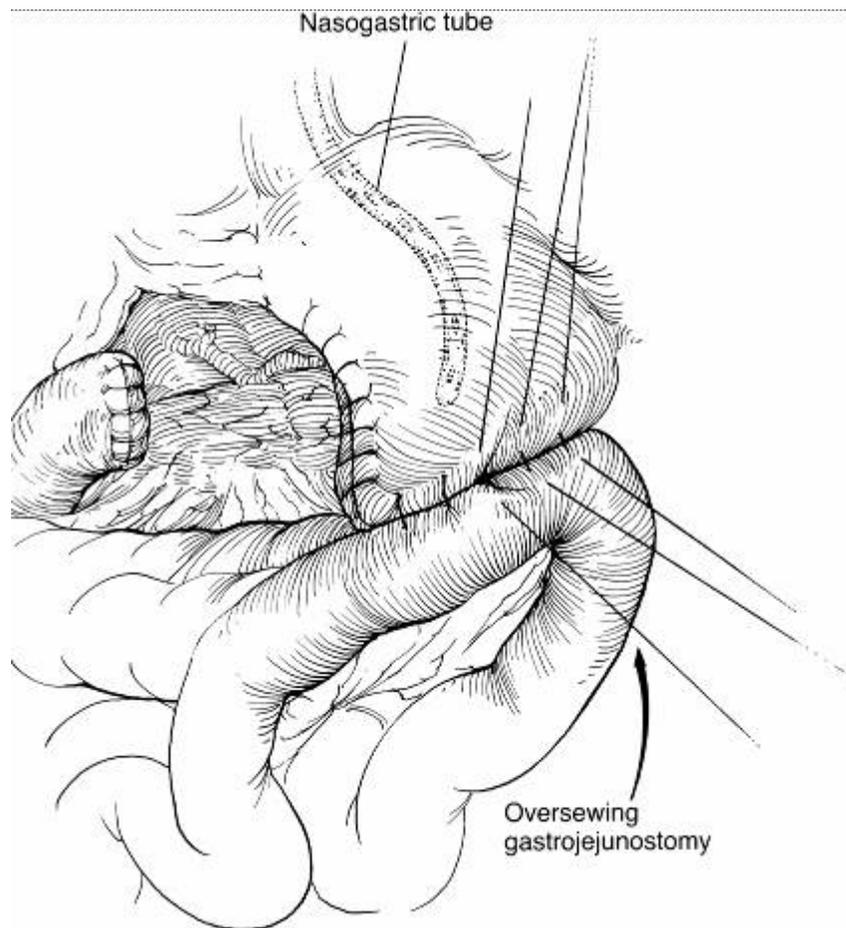
By contra-indication to operations with saving of food passing through the duodenum are also decompensated pylorostenosis , functional gastrostasis and duodenostasis. In such patients it is better to perform gastric resection by Billroth II.



Pic. Billroth I and Billroth II resection



Pic. Billroth I reconstruction



Pic. Billroth II reconstruction

DUODENAL ULCER

The duodenal ulcer is the chronic recurrent disease which characterized by ulcerous defect on a mucous tunic of duodenum. Pathology often makes progress with complications development.

Etiology and pathogenesis

There are some etiologic factors of the duodenal ulcer: *Helicobacter pylori*, emotion tension and neuropsychic stress overstrain, heredity and genetic inclination, presence of chronic gastroduodenitis, disturbance of diet and harmful habits (alcohol,

smoking). In pathogenesis of peptic ulcer a leading role is played disturbance of equilibrium between aggressive and projective properties of secret of stomach and its mucous tunic. The aggressive factors are vagus hyperfunctioning and hypergastrinemia; hyperproduction of hydrochloric acid and pepsin, and also reverse diffusion of the ions H^+ , action of biliary acids and isoleucine, toxins and enzymes of helicobacter pylori (HP). There are factors which contribute to ulcerogenic action: disturbance of motility of stomach and duodenum, ischemia of duodenum, and metaplasia of the epithelium.

Pathomorphology

Morphogenesis of duodenal ulcer fundamentally does not differ from ulcer in a stomach. Chronic ulcers are mainly single, is localized on the front or back wall of bulb (bulbar ulcer) and only in 7–8 % cases – below it (postbulbar ulcer). The plural ulcers of duodenum are met in 25 % cases.

Classification

(by A.L.Hrebenev, A.O.Sheptulin, 1989)

The duodenal ulcer is divided:

I. *By etiology:*

- A. True duodenal ulcer.
- B. Symptomatic ulcers.

II. *By passing of disease:*

- 1. Acute (first exposed ulcer).
- 2. Chronic:
 - a) with the rare exacerbation;
 - b) with the annual exacerbation;

- c) with the frequent exacerbation (2 times per a year and more frequent).

III. *By the stages of disease:*

1. Exacerbation.
2. Scarring:
 - a) stage of “red” scar;
 - b) stage of “white” scar.
3. Remission.

IV. *By localization:*

1. Ulcers of bulb of duodenum.
2. Low postbulbar ulcers.
3. Combined ulcers of duodenum and stomach.

V. *By sizes:*

1. Small ulcers up to 0,5 cm.
2. Middle — up 1,5 cm.
3. Large — up to 3 cm;
4. Giant ulcers over 3 cm.

VI. *By the presence of complications:*

1. Bleeding.
2. Perforation.
3. Penetration.
4. Organic stenosis.
5. Periduodenitis.
6. Malignization.

Clinical management

Pain in the epigastric region is the most expressed symptom of duodenal ulcer, often with displacement to the right in the projection area of bulb of duodenum and gall-bladder. Also for this pathology is typical the pain, that arises in 1,5–2 hours after food intake, “hungry” and nightly pain. As a rule, it is acute, sometimes unendurable, and is halted only after the use of food or water. Such patients complains for the seasonal exacerbation, more frequent in spring and in autumn. However exacerbation can be also in winter or in summer. In the acute period of disease heartburn often increases. However heartburn is the frequent symptom of cardial insufficiency and gastroesophageal reflux. For an duodenal ulcer the acute burning feeling of acid in a esophagus, pharynx and even in the cavity of mouth is especially typical. Often are belch by air or sour content, excessive salivation. Vomiting is not a typical symptom for duodenal ulcer. More typical sign is nausea. Sometimes for facilitation patients wilfully cause vomiting. These symptoms, arises in the late periods of passing of duodenal ulcer.

Intensity of pain and dyspepsia syndromes depends both on the depth of penetration and from distribution of ulcerous and periulcerous processes. Superficial ulceration within the mucous tunic, as a rule, does not cause the pain because it does not have sensible receptors. However, more deep layers of wall (muscular and especially serous) have plural sensible vegetative receptors. Therefore, on deepening and distribution of process arises visceral pain. At evident periulcerous processes and penetration of ulcers to neighboring organs and tissues, usually, a parietal peritoneum, that has spinal innervation, is pulled in. Pain becomes viscerosomatic, more intensive. A such pain syndrome (with an irradiation in the back) is typical for low postbulbar ulcers and bulbous ulcers of back wall, which penetrates in a pancreas and hepato-duodenal ligament. Usually such patients has good appetite. Some of them limit themselves in acceptance of ordinary food, go into to the dietary feed by small portions, and some — even hold back from a food, being afraid to provoke pain, and as a result of it weight is lost. Some of patients feeds more intensive and often.

The psychical status of patients often are changed as a asthenoneurotic syndrome: irritates, decline of working capacity, indisposition, hypochondria, abusiveness.

An inspection, as a rule, gives insignificant information. In many cases on the abdominal skin it is possible to notice hyperpigmentation after application of hot-water bottle. During the pain attack patients often occupy the forced position. At superficial palpation on the abdominal wall determined hyperesthesia in ulcer projection. In the

epigastric region, during deep palpation, it is possible to define pain and muscular tension, mostly moderate intensity. There is important symptom of local percussion painful (Mendel's symptom): percussion by fingers in the symmetric epigastric areas provoke pain in the ulcer, which is increased after the deep breath. The roentgenologic and endoscopic are main diagnostic methods. The symptom of ulcerous "niche" is a classic roentgenologic sign. It is depot of contrast agent, which is corresponded to ulcerous defect, with clear contours and light bank to which converged fold mucus. Cicatricial deformation of bulb of duodenum as a shamrock, butterfly, narrowing, tube, diverticulum and other forms is the important sign of chronic ulcerous process. A roentgenologic method is especially important for determination of configuration and sizes of stomach and duodenum, and also for estimation of motility functions. X-Ray examination is the main method at the peptic ulcer complicated by stenosis, with disturbance of evacuation, duodenostasis, duodenal-gastric reflux, gastroesophageal reflux, diverticulum. But by X-Ray examination is difficult to diagnose small superficial ulcers, acute ulcers, erosions, gastritises and duodenitises. The most informing method in such cases it endoscopy.

During endoscopy examination it is possible to define localization, form, sizes and depth of ulcer. During bleeding grumes, trickle or pulsating of blood are observed. By irrigation by styptic solutions, by cryocoagulation, by laser coagulation endoscopy allows to secure hemostasis. Endoscopy allows to perform the biopsy of ulcer tissues for determination of possible malignization.

Clinical variants and complication

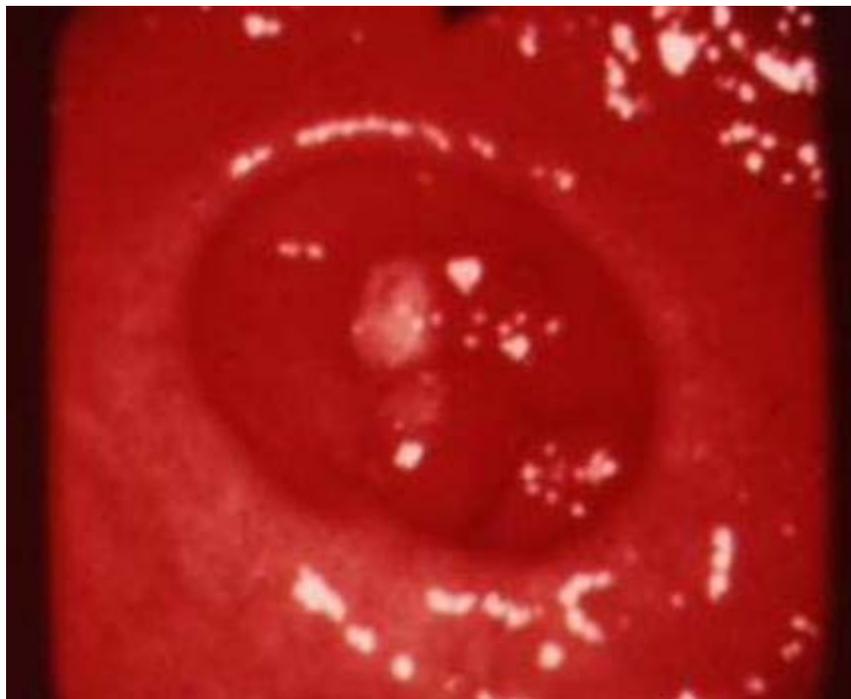
In patients with low postbulbar ulcers the clinical signs are more expressed. It characterized by late (in 2–3 hours after food intake) and intensive "hungry" and nightly pain, that often irradiate to the back and to the right hypochondrium. The postbulbar ulcers are inclined to more frequent exacerbation, and also to more frequent complications, such, as penetration, stenosis and bleeding.

The are more frequent ulcerous bleeding (the bulbous happen in 20–25 % cases, postbulbar — in 50–75 %), perforations (10–15 % cases). Penetration, stenosis and malignization in patients with duodenal ulcers are observed rarely.

Penetration is frequent complication of “low” and postbulbar ulcers of duodenum, which are placed on posterior, posterior superior and posterior inferior walls. Penetrates, usually, deep chronic ulcers, by passing through all layers of duodenum in neighboring organs and tissues (head of pancreas, hepato-duodenal ligament, small and large omentum, gall-bladder, liver). Such penetration is accompanied by development of inflammatory process in the neighboring organs and surrounding tissues and forming of cicatrical adhesions. A pain syndrome becomes more intensive, permanent and often pain irradiated in the back. Sometimes in the area of penetration it is possible to palpate painfully infiltrate.

Diagnostic program

1. Anamnesis and physical examination.
2. Endoscopy.
3. X-Ray examination of stomach and duodenum.
4. General and biochemical blood analysis.
5. Coagulogram.



Pic. Duodenoscopy

Differential diagnostics

The duodenal ulcer must be differentiated from acute and chronic cholecystitis, pancreatitis, gastroduodenitis. Endoscopy is help to diagnose duodenal ulcer.

Tactic and choice of treatment method

Conservative treatment. In most patients after conservative treatment an ulcer heals over in 4–6 weeks. Warning of relapses can be carried out by only supporting therapy during many years.

The best therapy of duodenal ulcer is associated with a helicobacter infection, there is the use of antagonists of H₂- receptors of histamine (renitidine— 300 mg in the evening or 150 mg twice for days; famotidine— 40 mg in the evening or 20 mg twice for days; nisatidine — 300 mg in the evening or 150 mg twice for days; roxatidine — 150 mg in the evening) in combination with sucralfate (venter) — for 1 г three times for days and antacid (almagel, maalox or gaviscon —1 dessert-spoon in a 1 hour after food intake). To this complex it is needed to add antibacterial preparations (De-nol – 1 tabl. 4 times per a day during 4–6 weeks + oxacylline for 0,5 g 4 times per a day — 10 days + Tryhopol (metronidazole) for 0,5 g 4 times per a day — 15 days).

In treatment of duodenal ulcer used chinolitics and miolitics (atropine, methacin, platyphyllin), and also mesoprostol (200 mg 4 times per days) and omeprasole (20 or 40 mg on days).

Such treatment of patients with the duodenal ulcer must be 4–6 weeks. If complications absents there is no necessity in the special diet.

Because of appearance of new pharmaceutical preparations and modern therapeutic treatment, indication to the operative methods narrowed. But the number of acute complications of duodenal ulcer does not go down, especially bleeding and perforations which require the urgent surgery.

Indications to the elective operation:

1. Passing of duodenal ulcer with the frequent relapses which could not be treated conservatively.
2. Repeated ulcerous bleeding.
3. Stenosis of the outlet part of the stomach.
4. Chronic penetrating ulcers with the pain syndrome.
5. Suspicion of malignization of ulcers.

Methods of surgical treatment.

At patients with the duodenal ulcer three types of operations are distinguished:

- organ-saving operations;
- organ-sparing operations;
- resection.

From them the better are: organ-saving operations with vagotomy, excision of the ulcer and drainage operation.

Types of vagotomy: trunk (TrV) (Pic. 3.2.7), selective (SV) (Pic. 3.2.8), selective proximal (SPV) (Pic. 3.2.9). Selective proximal vagotomy is optimal in the elective surgery of duodenal ulcer. However in urgent surgery a trunk, selective or selective proximal is often used in combination with drainage operations.

Drainage of the stomach operations are: Heineke-Mikulicz pyloroplasty, Finney pyloroplasty, submucous pyloroplasty by Diver-Barden-Shalimov, gastroduodenostomy by Jaboulay, gastroenteroanastomosis.

It is necessary to mark that “clean isolated” SPV, performed in patients with duodenal ulcer, often (in 15–20 % cases) results in relapses. The considerably less number of relapses (8–10 %) is observed after SPV in combination with drainage operations. Especially dangerous are the relapses of the ulcers placed in the projection of the large duodenal papilla, after gastroduodenostomy by Jaboulay.

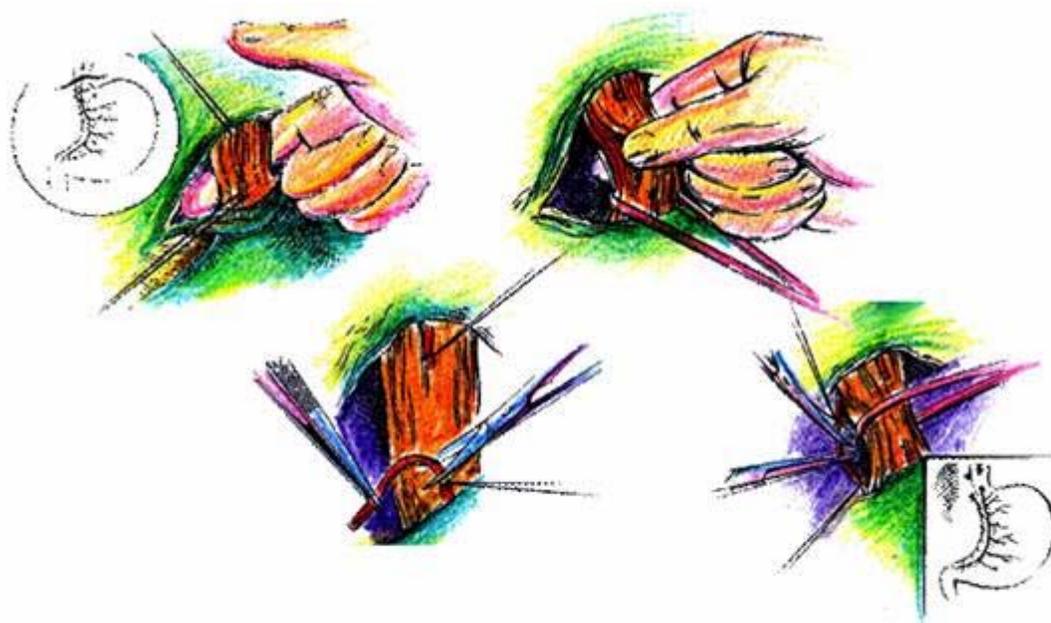
The least number of relapses of duodenal ulcer is observed after organ-saving operations, that combine SPV and ulcer excision.

If the ulcer is localized on the anterior surface of the duodenal bulb it can be performed by

the method Jade (Pic. 3.2.13) with subsequent to the pyloroplasty by Heineke-Mikulich.

At patients with decompensate stenosis and expressed dilatation and by the atony of stomach it is needed to apply the classic resection of stomach depending on possible damping-syndrome by Billroth -I or Billroth -II.

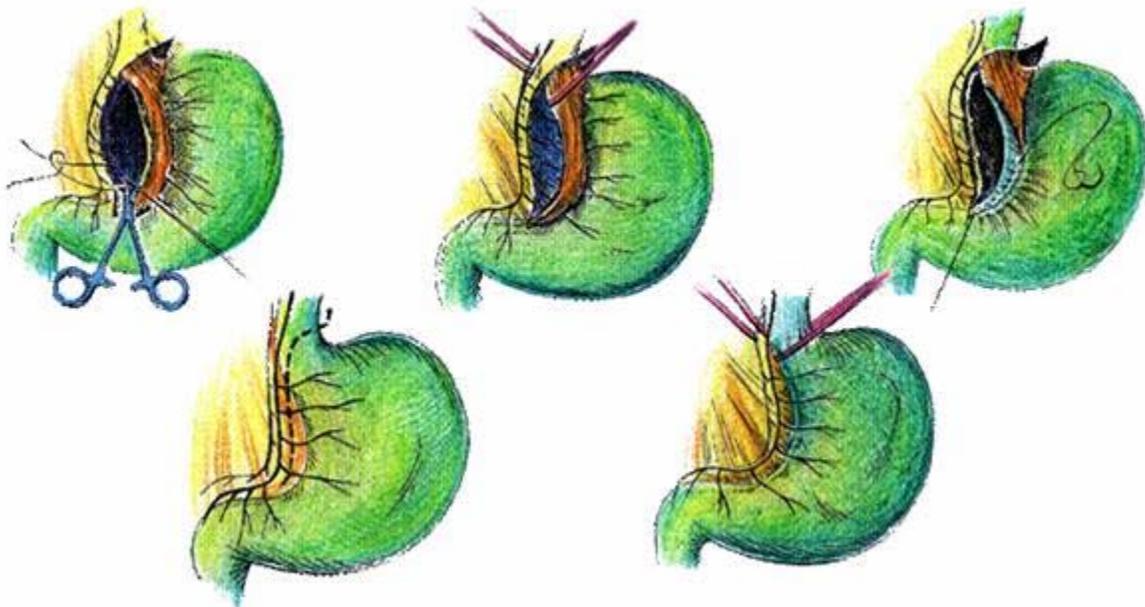
The choice of subtotal resection of stomach needs to be done at suspicion for malignization or at histological confirmed malignization ulcers. In a duodenum this process happens very rarely.



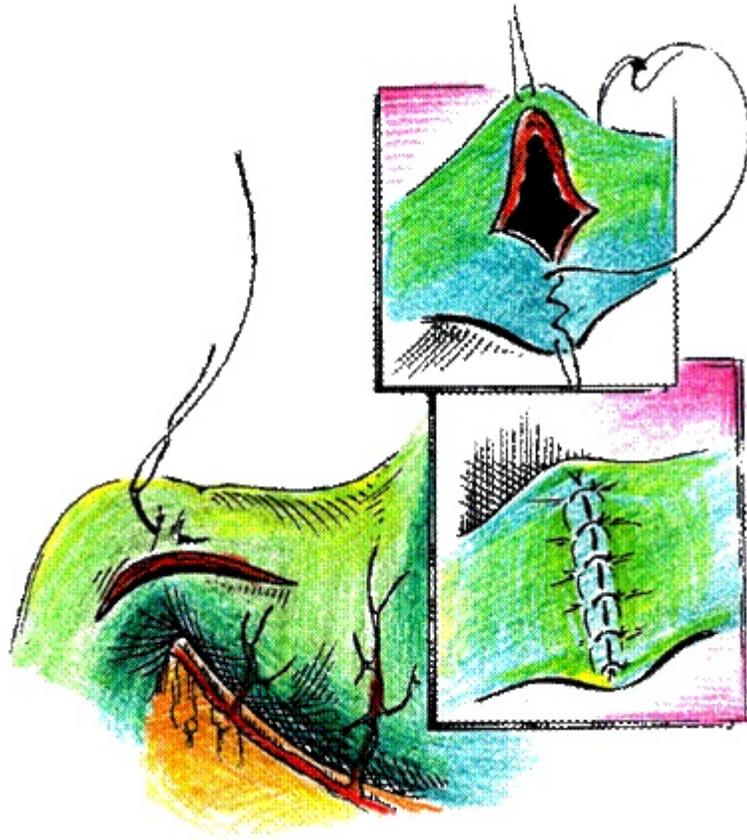
Pic. Trunk vagotomy (TrV)



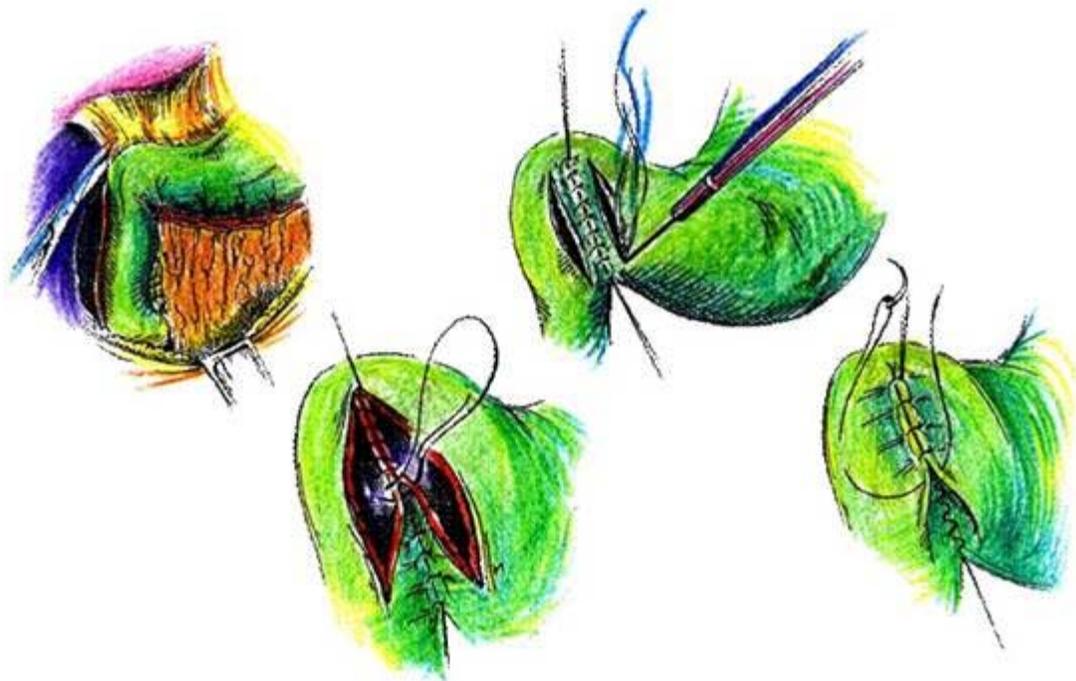
Pic. Selective vagotomy (SV)



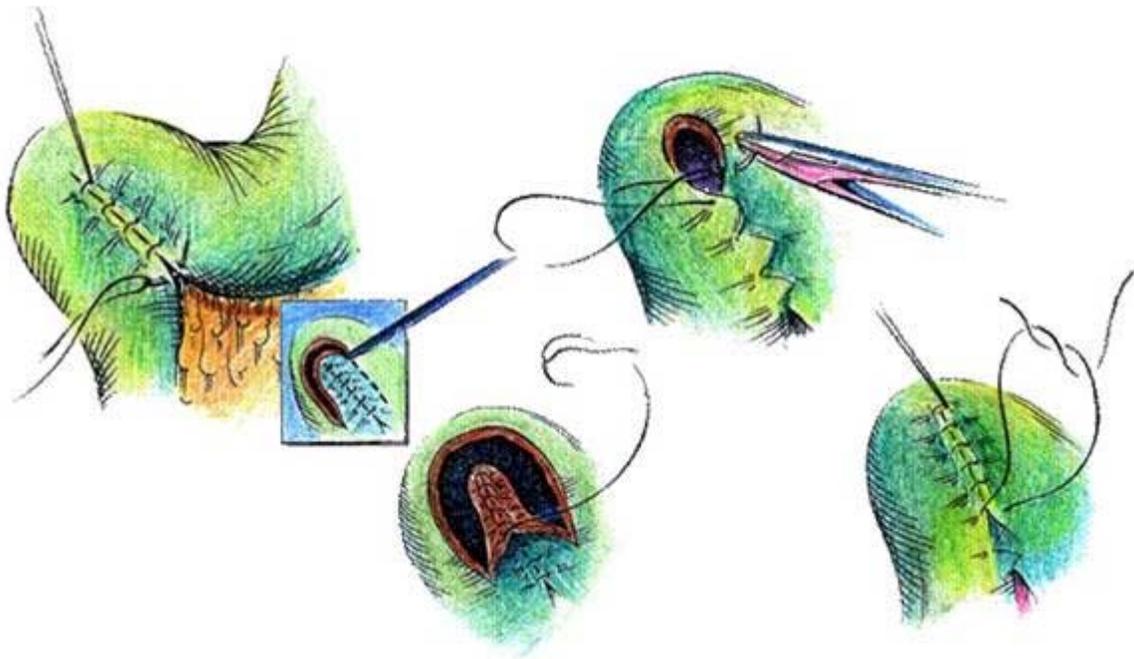
Pic. Selective proximal vagotomy (SPV)



Pic. Heineke-Mikulicz pyloroplasty



Pic. Gastroduodenostomy by Jaboulay



Pic. Finney pyloroplasty

ULCEROUS STENOSIS

Ulcerous stenosis is complication of Peptic ulcer or duodenum, which characterized by narrowing.



Pic. Ulcer stenosis.

Etiology and pathogenesis

Stenosis of outgoing part of stomach and duodenum of ulcerous origin arises as a result of scarring and common morphological changes around an ulcer. Narrowing, disturbance of the coordinated motility of gastric peristalsis come as a result of it and creates

the obstacle to the even moving of stomach content to the duodenum.

Pathomorphology

Such pathology in the compensation stage arises hypertrophy of the stomach walls. The pyloric ring has a 0,5–0,7 cm in diameter. The mucous tunic of pyloric part of stomach is thickened, with rough folds. Muscular fibers are hypertrophied and solid. Histological hyperplasia of pyloric glands is observed.

During decompensation the muscular layer of stomach higher stenosis becomes thinner, tone of him goes down, and a pyloric ring narrows to a few millimetres. Microscopically present atrophy of mucous tunic and muscular fibers, vessels sclerosis. A stomach collects the form of the stretched sack which goes down to the level of small pelvis.

Classification

(by O.O. Shalimov and V.F.Saenko, 1987)

Three clinical stages of stenosis are distinguished:

- I — compensated;
- II — subcompensated;
- III — decompensated.

The morphological changes in the initial part of stomach and duodenum are represented by the classification offered by M.I. Kusin (1985). On the basis of clinical, roentgenologic, endoscopic, electrogastrographical and intraoperative methods the examinations distinguished three stages of stenosis:

- I — inflammatory;
- II — cicatricial-ulcerous;
- III — cicatricial.

In accordance with localization, by V.F.Saenko (1988) distinguished three types of

stenosis:

- I — stenosis of goalkeeper;
- II — stenosis of bulb of duodenum;
- III — postbulbar duodenal stenosis.

First two types of stenosis are similar by functional and the organic changes. It united them by one name — pyloroduodenal, or high duodenal stenosis. The second group is postbulbar duodenal stenosis. Feature of them is that a goalkeeper does not take participation in the cicatricial- ulcerous process and its function is not broken.

Clinical management

The first signs of stenosis can be exposed already in eight-ten years from the beginning of the peptic ulcer disease. Mainly, this is narrowing and rigidity and disturbance of retractive activity of goalkeeper, which create a barrier for transition of stomach content to the duodenum.

In the stage of the *compensated stenosis* hypertrophy of wall of stomach develops and tone of muscular shell rises. Hereupon gastric content, slowly, but passes through the narrowed area of stomach output. In this stage patients, usually, complained about feeling of plenitude in an epigastric area after food intake, periodic vomitings by sour gastric content. On empty stomach by a stomach pump 200–300 ml gastric content is removed.

In the *subcompensated stage* muscular layer of stomach becomes thinner. Tone of it goes down, peristalsis relaxes, and it looks like the stretched sack. Evacuation disorders is increased. Fermentation and rotting developed in stagnant gastric content. On this stage of disease development patients, usually, complain for the permanent feeling of weight in epigastric region and regurgitation with an unpleasant “rotten” smell of sulphuretted hydrogen.

Vomiting becomes systematic (once or twice on a day) up to half of liter per day. On empty stomach it is possible to aspirate from it more 500 ml of content with the food used the day before.

In the *decompensation stage* of the clinical signs make progress quickly. There are

heavy disturbances of the general condition of patient, considerable loss of weight (to 30–40 %), acutely expressed dehydration of organism, hypoproteinemia, hypokalemia, azotemia and alkalosis. In case of the protracted neglected disease, as a result of progress of disturbances of metabolism, there can be a convulsive syndrome (gastric tetany). Vomiting in this stage not always can be considered by a typical sign, in fact patients often renounce to adopt a food, and a stomach acquires considerable sizes, overdistension form, its tone is violated and atrophy of wall comes. In such patients in an epigastric area it is possible to define the contours of the stretched stomach, with a slow peristalsis. In the distance it is possible to hear the splash. By a probe from a stomach to 1.5–2 litres of food with a putrid smell are removed. There can be gastric tetany at considerable disturbances of electrolyte metabolism.

A diagnosis is set according to a typical syndrome, results of sounding of stomach, roentgenoscopy, at which by contrasting of a barium expose stenosis of initial part of stomach or duodenum, determines its origin and estimates a degree.

Roentgenologically in the compensation stage stomach is in normal sizes, its peristalsis deep, increased, evacuation of content proceeds no more than 6 hours. In the stage of subcompensation a stomach is megascopic, its peristalsis is loosened, evacuation stays too long to 24 hours. During decompensation a stomach is considerably extended as a sack, deformed, the waves of antiperistalsis can take place, a contrast stays too long more than 24–48 hours. The method of the double contrasting by a barium and air considerably facilitates diagnostics.

Determination of stomach motility has not only diagnostic but also prognostic value for the choice of method of operation.

In the stage of compensation motility of stomach is well-kept, often even increased. With the increasing degree of stenosis the motility disturbance increased, up to gastroplegia.

In the biochemical blood test is marked the decline of content of albumen to 54–48 g/l; potassium — to 2,9–2,5 mmol/l; chlorides — to 85–87 mmol/l. The changes of such indexes are most expressed at patients with gastrogenous tetany.

The study of secretory function of stomach allows to define the degree of compensation of stenosis and is important at the choice of adequate method of operation.

Gastroscopy with a biopsy is the enough informing method of examination of such

patients. By this method is possible to determine a reason and degree of stenosis, and also state of mucous tunic of stomach.

Diagnosis program

1. Complaints of patient and anamnesis of disease.
3. Sounding of stomach and examination of gastric content.
4. Fibergastroduodenoscopy, biopsy.
5. Intragastric pH-metry.
6. Study of motility of stomach.
7. Roentgenologic examination of stomach and duodenum (structural features, passage).
8. Sonography.



Pic. X-Ray examination. Ulcer stenosis.

Differential diagnostics

Stenosis of the output part of stomach and duodenum of ulcerous origin it is needed to differentiate with functional gastrostasis and narrowing of tumour and chemical genesis.

Functional gastrostasis more frequent meets at women. Basic, that distinguishes it from other pathologies, there is absence of some organic changes in the area of pyloric part of stomach or in a duodenum, that can be exposed during fibergastroscopy.

Differential diagnostics of stenosis of *tumour genesis*, as a rule, also does not cause the special difficulties. A diagnosis is finally confirmed by histological

examinations of the biopsy material taken during endoscopy.

Postburn stenosis of piloroantral area of stomach observed, from data of statistics, more than in 25 % cases of patients with the burn of esophagus. In anamnesis in each of such patients takes place by mistake or the intentionally taken an a swig at acid, alkali or other chemical matter. Some diagnostic difficulties can arise up at the isolated postburn stenosis of pyloric part of stomach. The however attentively collected anamnesis and professionally conducted endoscopic examination enable to set a correct diagnosis.

Tactic and choice of treatment method

Treatment of ulcerous stenosis of piloroantral part of stomach and duodenum must be exceptionally operative. A method depend on many factors: degree of stenosis, these secretory and motility functions of stomach, age of patient, presence of accompanying diseases and others like that. In the compensated and subcompensated stages of stenosis and at enough well-kept functions of stomach it is possible to perform of organ-saving operations (vagotomy with drainage stomach operations, economy resection of stomach). At growth of the signs of stenosis and disturbance of basic functions of stomach, the volume of operation must be increased up resection by the Bilioth's second method.

At the patients and older age persons with heavy accompanying pathology is performed minimum surgery —gastroenteroanastomosis.

Preoperative preparation must be strictly individual.

At patients with insignificant disturbances of gastric motor activity (stage of compensation, subcompensations) and with good level of metabolism indexes it is better to shorten preoperative preparation in time. Such patients, usually, operated on 3–4 day. Preparation before operation at patients with decompensated pilorostenosis must be directed for the correction of metabolism disturbances. Such patients must receive transfusion of liquid up to 2,5–3 l per day with content of the ions K^+ , Na^+ , Ca^{++} , amino acid and glucose; plasma, albumen. Twice on days performed decompression and washing of stomach and anti-ulcerous therapy. Effective preoperative preparation in such patients requires 5–7 days, sometimes more.

PERFORATED GASTRODUODENAL ULCERS

The typical perforation of gastric or duodenum ulcer is strengthening of necrosis process in the area of ulcerous crater with subsequent disturbance of integrity of wall, that result to the permanent effluence of gastroduodenal content and air in a free abdominal cavity.

Etiology and pathogenesis

In 50,7 % cases perforates the ulcers of duodenum, in 42,8 % are ulcers of pyloric part of stomach, in 4,8 % are ulcers of small curvature of body of stomach and in 0,7 % are cardial ulcers.

Ulcers, which lie on the front wall of stomach and duodenum more frequent give the perforation with general peritonitis, while ulcers on a back wall — perforation with adhesive inflammation.

The reasons of ulcers perforation are: exacerbation of peptic ulcer, harmful habits, stresses, professional, athletic overexertion, faults in the feed and abuses by strong waters.

Pathomorphology

In pathogeny of acute perforation important: progressive necrosis processes in the area of ulcerous crater with activating of virulent infection; hyperergic type of local vaculo-stromal reaction with the thrombosis of veins of stomach and duodenum; local manifestation of autoimmune conflict with accumulation of sour mucopolysaccharides on periphery of ulcer and high coefficient of plasmatization of mucous tunic (K.I. Mishkin, A.A. Frankfurt, 1971).

Classification

(by V.S. Savelev, 1986)

Perforated gastroduodenal ulcers are divided:

1. *After etiology:*

- ulcerous;
- un ulcerous.

2. *After localization:*

- gastric (small curvature, cardial, antral, prepyloric, pyloric) ulcer, front and back walls;
- ulcers of duodenum (front and back walls).

3. *After passing:*

- perforated in an abdominal cavity;
- covered perforations;
- atypical perforations.

Clinical management

The clinical picture of perforation is very typical and depends on distribution of inflammatory process and infection of abdominal cavity. In clinical passing of the perforations distinguish three phases: shock, “imaginary prosperity” and peritonitis (Mondor, 1939).

For the phase of shock (to 6 hours last) typical very acute pain in epigastric region (Delafua compares it to pain from the stab with a dagger) with an irradiation in a right shoulder and collar-bone, a face is pale, with expression of strong fear, lines become (facies abdominalis) acute, a death-damp irrigates skin covers. A pulse is at first slow (vagus pulse), later becomes frequent and less filling. Sometime observed the reflex

vomiting and delay of gases. Arterial pressure is reduced. On examination stomach is pulled in, does not take part in the act of breathing. At palpation is “wooden belly stomach”, especially in an upper part, where, usually, there is most pain. Positive Blumberg's sign. At percussion is disappearance of hepatic dullness (the Spizharnyy symptom). At rectal examination expose painful in the area of rectouterine or rectovesical pouch (the Kulenkampff's symptom).

The phase of shock changes by the phase of “imaginary prosperity”, when the reflex signs go down: the general condition of patient gets better, a pulse becomes normal, arterial pressure rises, a stomach-ache diminishes partly. However observed tension of muscles of front abdominal wall, positive Blumberg's sign.

The phase of “imaginary prosperity” in 6–12 hours from the moment of perforation changes by the phase of peritonitis: a pulse is frequent, a stomach is swollen through growing flatulence, intestinal noises are not listened, a face acquires the specific kind — facies Hippocratica — the eyes fall back, lips turn blue, a nose becomes sharp, a tongue becomes dry and furred, breathing superficial and frequent, a temperature rises.

Variants of clinical passing and complication

Covered perforation (A.M. Shnicler, 1912). At this pathology the perforative hole after a perforation is closed by a fibrin, by a omentum, by the fate of liver, sometimes — piece of food. After that some amount of stomach content and air gets in an abdominal cavity. After the protection a stomach-ache diminishes, but proof tension of muscles of front abdominal wall, especially overhead quadrant of stomach is kept. At percussion hepatic dullness is doubtful. During x-Ray examination it is not always possible to mark gas in right hypochondrium (Pic. 3.2.14).

Consequences of passing of the covered perforation: the repeated perforation with development of classic clinical signs can come; at separation of process from a free abdominal cavity a subdiaphragmatic or subhepatic abscess is formed; complete closing of defect by surrounding tissue with gradual convalescence of patient.

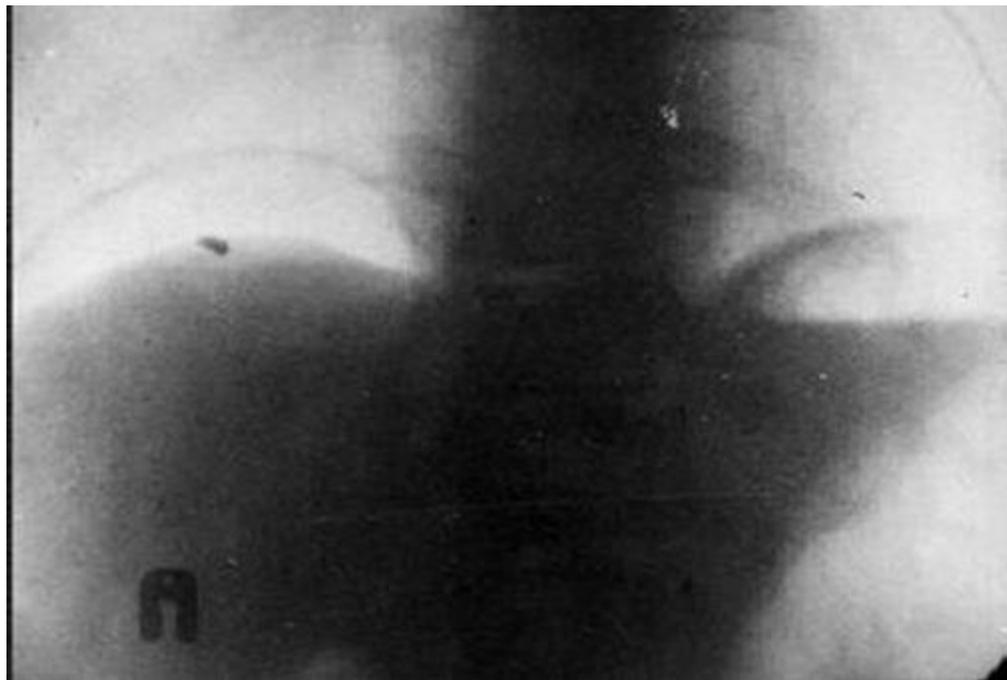
The atypical perforation is the perforation, at which gastric or intestinal content gets not to the abdominal cavity, but in retroperitoneal space (ulcers of back wall of duodenum), large or small omentum (ulcers of small curvature of stomach), hepato-

duodenal ligament.

In such patients during a perforation pain is not acutely expressed. During palpation observed insignificant rigidity of muscles of front abdominal wall. On occasion, especially on the late stages of disease, there can be hypodermic emphysema and crepitation.

Diagnosis program

1. Anamnesis and physical examination.
2. Global analysis of blood and urine, biochemical blood test, coagulogram.
3. X-Ray examination of abdominal cavity organs for presence of free gas (pneumoperitoneum).
4. Pneumogastrography, contrasting pneumogastrography.
5. Fiber-gastroduodenoscopy.
6. Sonography of abdominal cavity organs.
7. Laparocentesis with the Neymark diagnostic test (to the 2–3 ml of abdominal cavity exudate adds 4–5 drops of the 10 % solution of iodine. If the admixtures of gastric content appear in exudate, then under action of iodine gastric content gets a dirtily-dark blue color).
8. Laparoscopy.



Pic. X-Ray examination of abdominal cavity organs. Presence of free gas (pneumoperitoneum).

Tactic and choice of treatment method

The diagnosed perforated gastric and duodenum ulcer is an absolute indications to

operation. Preoperative preparation must include: in I phase are antishock action; in the II and III phases — reanimation preparations, introductions of antibiotics for 2–3 hours before operation, liquidation of hypovolemia by salt blood substitutes (solution of chlorous sodium), solutions of dextran (polyhlukine, reopolihlukine, hemodes). Amount of liquid necessary for correction of hypovolemia, calculate after hematokrit by central vein pressure. Taking for the norm of hematokrit 40 %, on each 5 % higher norms need to be poured 1000,0 ml liquids.

Conservative treatment (method of Tejlor, 1946) can be justified at the refusal of patient from operation or in default of conditions for its implementation.

It must include:

- permanent nasogastral aspiration of gastric content;
- introduction of preparations which brake a gastric secretion (atropine, H₂-blockers and others like that);
- introduction of antibiotics;
- correction of metabolism;
- laparocentesis with drainage and closed lavage of the abdominal cavity.

In the decision of question about the choice of method of operative treatment of perforated gastroduodenal ulcers the important value has the following factors: localization of ulcer, clinico-morphological description of ulcer (perforation of acute or chronic ulcer), connected with the perforation such complications of ulcer, as bleeding, cicatricial-ulcerous stenosis, penetration, degree of risk of operation and feature of clinical situation.

Operative treatments at a perforated ulcer divide into palliative and radical.

Palliative operations

Palliative operations are: closure of the perforative hole of ulcer, tamponade of the perforative hole by a omentum on a leg by B.A. Opperl - P.N.Polikarpov - M.A.Pidhorbunsky (1896, 1927, 1948) (Pic. 3.2.15). Indications and terms for their implementation are:

- perforation of acute duodenal ulcer in youth and young age without anamnesis;
- perforation of acute ulcer in the II–III phases of passing;
- perforation of callous gastric ulcer in the II–III phases of passing;
- expressed and high degrees of risk of operation.

Radical operations

The radical operations at perforated ulcers are: resection of stomach and excision of the perforative hole of ulcer in combination with pyloroplasty and StV, SV or SPV.

Indications and terms for implementation of resection of stomach are:

- perforation of callous gastric ulcer in I phase of clinical passing;
- repeated perforation of ulcer;
- perforation of ulcer in I phase of clinical passing in combination with stenosis and bleeding of ulcer;
- perforation of duodenal ulcer in I phase of passing in combination with a gastric ulcer;
- unexpressed and moderate degree of risk of operation;
- sufficient qualification of surgeon and material resources of operating-anaesthetic brigade.

Indications for implementation of operation of excision of the perforative hole of ulcer with pyloroplasty, StV, SV and SPV are: perforation of ulcer of front wall of duodenum or pyloric part of stomach in the I–II phases of passing;

- perforation of ulcer of front wall of duodenum in the I–II phases of passing in combination with the bleeding ulcer of back wall;
- perforation of duodenal ulcer in the I–II phases of passing in combination with the compensated stenosis of outgoing part of stomach;
- increased gastric secretion;
- insignificant and moderate degree of risk of operation;

— sufficient qualification and technical preparedness of surgeon.

[video](#)

BLEEDING GASTRODUODENAL ULCERS

Bleeding gastroduodenal ulcers are outpouring of blood in the gastrointestinal tract cavity as a result of strengthening and distribution of necrosis process in the ulcer area to vessels with the subsequent melting of their walls.

Complication of peptic or duodenal ulcer by bleeding is critical situation which threatens to life of patient and requires from the surgeon of immediate and decisive actions for clarification of reasons of bleeding and choice of tactic of treatment. The ulcerous bleeding has 60 % of the acute bleeding from the upper parts of gastrointestinal tract.

Etiology and pathogenesis

The origin of the gastrointestinal bleeding at patients with a gastric or duodenal ulcer almost is always related to exacerbation of ulcerous process. The reason of bleeding is a erosive vessel, that is on the bottom of ulcer. The expressed inflammatory and sclerotic processes round the damaged vessel embarrassed its contraction, that diminishes chances on the spontaneous stop of bleeding.

Gastric ulcers, compare with the ulcers of duodenum, complicated by bleeding more frequent. Bleeding at gastric ulcers are more expressed, profuse, with heavy passing.

At the duodenal ulcer bleeding more frequent complicate the ulcers of back wall, which penetrates in the head of pancreas.

At the men ulcer is complicated by bleeding twice more frequent, than at women. It costs to mark that 80 % patients which carried bleeding from an ulcer and treated oneself by conservative preparations, are under the permanent threat of the recurrent bleeding.

Pathomorphology

Strengthening of necrosis process are leading factors in the origin of the ulcerous bleeding in the area of ulcerous crater with distribution of this process to a vessel and subsequent melting of vascular wall; activation of fibrinolysis in tissues of stomach and duodenum; ischemia of tissues of wall of stomach.

Classification

Bleeding gastroduodenal ulcers after the degree of weight of loss of blood (by O.O. Shalimov and V.F.Saenko, 1987) are divided:

I degree is *easy* — observed at the loss to 20 % volume of circulatory blood (at a patient with weight of body 70 kg it is up to 1000 ml);

II degree — *middle weight* is loss from 20 to 30 % volume of circulatory blood (1000–1500 ml);

The III degree is *heavy* — is observed at loss of blood more than 30 % volume of circulatory blood (1500–2500 ml).

Clinical management

At patients with an peptic ulcer disease, bleeding pops up, mainly at night. Vomiting can be the first sign of it, mostly, at gastric localization of ulcers. Vomiting masses, as a rule, looks like “coffee-grounds”. Sometimes they are as a fresh red blood or its grume.

The black tar-like emptying are the permanent symptom of the ulcerous bleeding, with an unpleasant smell (“melena”), that can take place to a few times per days.

Bloody vomiting and emptying as “melena” is accompanied by worsening of the general condition of patient. A acute weakness, dizziness, noise in a head and darkening

in eyes, sometimes — loss of consciousness. A collapse with the signs of hemorrhagic shock can also develop. Exactly with a such clinical picture the such patients get to the hospital. It is needed to remember, that for diagnostics anamnesis is very important. Find out often, that at a patient an peptic ulcer was already diagnosed once. It appears sometimes, that bleeding is repeated or surgery concerning a perforated ulcer took place in the past. At some patients a gastric or duodenum ulcer is was not diagnosed before, the however attentively collected anamnesis exposed, that at a patient had a stomach-ache. Thus it communication with acceptance of food and seasonality is typical (more frequent appears in spring and in autumn). Patients tell, that pain in overhead part of abdomen which disturbed a few days prior to bleeding suddenly disappeared after first its displays (*the Bergmann's symptom*).

At patients with the ulcerous bleeding there are the typical changes of hemodynamic indexes: a pulse is frequent, weak filling and tension, arterial pressure is mostly reduced. These indexes need to be observed in a dynamics, as they can change during the short interval of time.

There is the pallor of skin and visible mucous tunics at a examination. A stomach sometimes is moderately exaggerated, but more frequent is pulled in, soft at palpation. In overhead part it is possible to notice hyperpigmental spots — tracks from the protracted application of hot-water bottle. Painful at deep palpation in the area of right hypochondrium (duodenal ulcer) or in a epigastric area (gastric ulcer) it is possible to observe at penetrated ulcers. Important symptom of Mendel also — painful at percussion in the projection of piloroduodenal area.

At the examination of patients with the gastrointestinal bleeding finger examination of rectum is obligatory. It needs to be performed at the first examination, because information about the presence of black excrement (“melena”) more frequent get according to a patient anamnesis, that can result in erroneous conclusions. Finger examination of rectum allows to expose tracks of black excrement or blood. In addition, it is sometimes possible to expose the tumour of rectum or haemorrhoidal knots which also are the source of bleeding.

The deciding value in establishment of diagnosis has the endoscopic examination. Fiber-gastroduodenoscopy enables not only to deny or confirm the presence of bleeding but also, that it is especially important, to set its reason and source. Often embarrassed the examination of stomach and duodenum present in it blood and content. In such cases

it is necessary to remove blood or content, by gastric lavage, and to repeat endoscopic examination. During the examination often exposed the bleeding with fresh blood from the bottom of ulcer or ulcerous defect with one or a few erosive and thrombosed vessels (stopped bleeding). The bottom of ulcer can be covered by the package of blood.

Important information about such pathology is given by haematological indexes also. Diminishment of number of red corpuscles and haemoglobin of blood, decline of haematocritis is observed in such patients. However always needed to remember, that at first time after bleeding haematological indexes can change insignificantly. Conducting of global analysis of blood in a dynamics in every a few hours is more informing.

Variants of clinical passing and complication

It is necessary always to remember that complication of peptic ulcer by bleeding happens considerably more frequent, than is diagnosed. Usually, to 50–55 % moderate bleeding (microbleeding) have the hidden passing. The massive bleeding meet considerably rarer, however almost always run across with the brightly expressed clinical signs which often carries dramatic character. In fact profuse bleeding with the loss 50–60 % to the volume of circulatory blood could stop the heart and cause the death of patient.

The clinical signs and passing of disease depend on the degree of lost of blood (O.O. Shalimov and V.F.Saenko, 1987).

For lost of blood I degree typical there is a frequent pulse to 90–100, decline of arterial pressure of to 90/60 mm Hg. The excitability of patient changes by lethargy, however clear consciousness is, breathing some frequent. After the stop of bleeding and in absent of hemorrhage compensation the expressed disturbances of circulation of blood does not observe.

At patients with the II degree of hemorrhage the general condition needs to be estimated as average. Expressed pallor of skin, sticky sweat, lethargy. Pulse — 120–130 per min., weak filling and tension, arterial pressure — 90–80/50 mm Hg. At first hours the spasm of vessels (centralization of circulation of blood) comes after bleeding, that predetermines normal or increased, arterial pressure. However, as a result of the protracted bleeding compensate mechanisms of arterial pressure are exhausted and can acutely go down at any point. Without the proper compensation of hemorrhage the such patients can survive, however almost always there are considerable disturbances of blood

circulation with disturbance of functions of liver and kidneys.

The III degree of hemorrhage characterizes heavy clinical passing. There is a pulse in such patients — 130–140 per min., and arterial pressure — from 60 to 0 mm Hg. Consciousness is almost always darkened, acutely expressed adynamy. Central vein pressure is low. Oliguria is observed, that can change by anuria. Without active and directed correction of hemorrhage a patient can die.

But, not always weight of bleeding which is conditioned by the degree of hemorrhage correspond the general condition of patient. On occasion the considerable loss of blood during the set time is accompanied by the relatively satisfactory condition of patient. And vice versa, moderate hemorrhage can bring to the considerable worsening of general condition. It can depend both on compensate possibilities of organism and from the presence of accompanying pathology.

It is needed to remember, that the ulcerous bleeding can accompanying with the perforation of ulcer. During perforation ulcers are often accompanied by bleeding. Correct diagnostics of these two complications has the important value in tactical approach and in the choice of method of surgical treatment. In fact simple suturing of perforated and bleeding ulcer can complicated in postoperative period by the profuse bleeding and cause the necessity of the repeated operation.

Diagnosis program

1. Anamnesis and physical examination.
2. Finger examination of rectum.
3. Gastroduodenoscopy.
4. Global analysis of blood.
5. Coagulogram.
6. 7. Biochemical blood test.
7. X-Ray examination of gastrointestinal tract.
8. Electrocardiography.



Pic. Endoscopy - stopped bleeding.

Differential diagnostics

At wide introduction of gastroduodenoscopy of question of differential diagnostics of bleeding lost the actuality. However much a problem arises up at impossibility to execute this examination through the heavy general condition of patient or taking into account other reasons. Differential diagnostics is conducted with bleeding of un ulcerous origin, which arise up in different parts of digestive tract.

For bleeding from the *varicose extended veins of esophagus* during portal hypertension at patients with the cirrhosis of liver the acute beginning without pain is characteristic, like during exacerbation of ulcerous disease. These bleeding differ by the special massiveness and considerable hemorrhage. Vomiting by fresh blood, expressed

tachycardia, falling of arterial pressure are observed. In such patients it is possible to find the signs of cirrhosis of liver and portal hypertension (“head of jelly-fish”, hypersplenism, ascites, often is icterus).

Sliding hernia of the esophagus opening of diaphragm can be accompanied by formation of ulcers in the place of clench of the stomach by the legs of diaphragm and bleeding from them. However for this pathology are more typical microbleeding, that is hidden. In such patients often the present protracted anaemia which can achieve the critical values. Sometimes in them observe more expressed bleeding with “classic” vomiting “coffee-grounds” and melena. During the roentgenologic examination with barium is possible to expose the signs of sliding hernia of the esophagus opening: the obtuse cardial angle, absence or diminishment of gas bubble of stomach or “ringing symptom”.

The cancer tumour of stomach in the destruction stage can be also complicated by bleeding. However, such bleeding are massive, and chronic character is carried mostly with gradual growth of anaemia. For this pathology there are the inherent worsenings of the general condition of patient, loss of weight of body, decline of appetite and waiver of meat food. At the roentgenologic examination the “defect of filling” is exposed in a stomach.

The gastric bleeding can be related to the diseases of the cardio-vascular system (atherosclerosis, hypertensive disease), however such happens mainly in the older years people. Clearly, that in such patients during the endoscopic examination the source of bleeding exposing is not succeeded.

Among other diseases, with which it is necessary to differentiate the ulcerous bleeding, it is needed to remember the Mallory-Weiss syndrome, benign tumours of stomach and duodenum (more frequent leiomyoma), hemorrhagic gastritis, acute (stress) erosive defeats of stomach, arteriovenous fistula of mucous tunic.

Often differential diagnostics performed according to the level of localization of source of bleeding in different parts of gastrointestinal tract. For the upper parts of digestive tract (esophagus and stomach) typical there is vomiting by grume or “coffee-grounds” content and emptying by “melena”. The farther aboral placed source of bleeding, the bloody emptying changes the more so. During the bleeding from a thin bowel excrement looks as “melena”. In case of such pathology of colon (polypuses,

tumours, unspecific ulcerous colitis) emptying have the appearance of fresh red blood, mostly as packages.

Tactic and choice of treatment method

The conservative therapy indicated to patients with the stopped bleeding of I degree and bleeding of the II–III degrees at patients which have heavy accompanying pathology, because of operative risk.

Conservative therapy must include:

— prescription of hemostatic preparations (intravenously the aminocapronic acid 5 % — 200–400 ml, chlorous calcium 10 % — 10,0 ml, vicasol 1 % — 3,0 ml);

— addition to the volume of circulatory blood (gelatin, poliglukine, salt blood substitutes);

— preparations of blood (fibrinogen — 2–3 r, cryoprecipitate);

— blood substitutes therapy (red corpuscles mass, washed red corpuscles, plasma of blood);

— antiulcerous preparations — blocker of H₂- receptor (ranitidine, roxatidine, nasatidine— for 150 mg 1–2 times per days);

— antacid and adsorbents (almagel, phosphalugel, maalox— for 1–2 dessertspoons through 1 hour after food intake).

It is expedient to apply washing of stomach by water with ice and the use 5 % solution of aminocapronic acid inward for to a 1 soup spoon in every 20–30 minutes.

The endoscopic methods of stop of bleeding are used also. Among them most effective is a laser and electro-coagulation.

Absolute indications to surgical treatment are: 1) lasting bleeding I degree; 2) recurrent bleeding after hemorrhage I degree; 3) bleeding of the II–III degrees; 4) stopped bleeding with hemorrhage of the II–III degrees at the endoscopically exposed ulcerous defect with a presence on the ulcer bottom thrombosed vessels or erosive vessels covered by the package of blood.

The *choice of method of surgical treatment* always needs to be decided

individually. On today the best tactic which gives advantage to organ-saving and organosparing methods of operations. The removing ulcer as sources of bleeding must be an obligatory condition. The methods of sewing of bleeding vessels or edging of ulcer and bandaging of vessels which feed a stomach and duodenum did not justify itself through the real threat of relapse of bleeding already in an early postoperative period (9–12 days).

Palliative operations (cutting of ulcer, forming of roundabout anastomosis) can be justified only taking into account the general condition of patient and on a necessity as possible quick and least traumatically to make off operation.

At the bleeding ulcers of duodenum it is better to apply excision of ulcer or it exteritirization after methods, developed by V.Zajtsev and Velihotsky. Operation complemented by one of types of vagotomy, it is better by a selective proximal with piliroplastic. The resection of stomach on the second or first method of Bilioth can be realized only in the stable general condition of patient. During the resection of stomach in case of low bleeding duodenal ulcers it is better to execute mobilization of duodenum and suturing of its stump on transcholedochus drainage which formed as transcholedochus duodenotomy (Laqey, 1942). This method warns the possible intraoperative damages of choledoch, that are the possible at low duodenal ulcers. Transcholedochus duodenotomy by performing the decompression of stump of duodenum, warns insufficiency of its stitches, that can arise up in an early postoperative period.

In case of bleeding gastric ulcers, the resection methods of operations are also usable. Only on occasion, when patients has the grave general condition, it is possible to assume the wedge cutting of ulcer.

[video](#)

MALIGNIZATION

CANCER OF STOMACH

The cancer of stomach is a malignant formation, that develops from epithelium tissue of mucus stomach. Among the tumours of organs of digestion this pathology takes

first place and is the most frequent, by the reason of death from malignant formations in many countries of world. Frequency of it at the last 30 years considerably diminished in the countries of Western Europe and North America, but yet remains high in Japan, China, countries of East Europe and South America.

Etiology and pathogenesis

Etiology of cancer of stomach is unknown. It is known that, as other diseases of gastrointestinal tract, a cancer damages a stomach. According to statistical information, it meets approximately in 40 % of all localizations of cancer.

The factors of external environment has the substantial influencing on frequency of this pathology. Above all things, feed, smoke food, salting, freezing of products and their contamination of aflatoxin. Consider that a “food factor” can be: a) by a carcinogen; b) by the solvent of carcinogens; c) to grow into a carcinogen in the process of digestion; d) to be instrumental in action of carcinogens; e) not enough to neutralize carcinogens.

In the USA and countries of Western Europe frequency of cancer of stomach in 2 times more large in the lower socio-economic groups of population. Some professional groups also can it (miners, farmers, works of rubber, woodworking and asbestine industry). High correlation communication is set between frequency of cancer of stomach and use of alcohol and smoking. The value of genetic factors (heredity, blood type) is not led to.

The cancer of stomach arises up mainly in age 60 years and above, more frequent men are ill.

Precancer. The precancer diseases of stomach are: a) chronic metaplastic disregenerator gastritis conditioned by helicobacter pylori; b) villous polypuses of stomach and chronic ulcers; c) nutritional anemia due to vitamin B12 deficiency (pernicious); d) resected stomach concerning an ulcer.

The presence of precancer changes of mucous tunic of stomach has substantial influence for frequency of stomach cancer. In those countries, where morbidity on the cancer of stomach is higher, considerably more frequent chronic gastritises are diagnosed. Lately in etiology of chronic gastritises take the important value helicobacter pylori. In Japan, where the cancer of stomach is in 40 % cases is the reason of death, chronic gastritis appears in 80 % cases of resected stomach, concerning a cancer.

Connection between polypuses, chronic gastric ulcers and possible its malignization comes into question in literature during many decades. Most authors consider that polypuses could be malignant differently. There are three histological types of polypuses: hyperplastic, villous and hamartoma. There are hyperplastic polypuses, but it is not malignant.

Hamartoma is accumulation of cells of normal mucous tunic of stomach. They never become malignant.

Villous polypuses are potentially malignant in 40 % cases, but it happens in 10 times less, than hyperplastic. The possibility of malignization of chronic gastric ulcers is not proved. The American scientists support a hypothesis, that the cancer of stomach can be ulcerous often, but malignization of ulcers takes place rarely (no more than 3 %). From data of the Japanese scientists, on 50–70th there was higher correlation connection between chronic gastric ulcers and cancer of stomach. The frequent decline of this correlation is lately noticed (70 % on 50–70th and 10 % on 80th).

Frequency of cancer of stomach at patients with pernicious anaemia hesitates within the 5–10 %, that is 20 times higher, compare with control population. In patients with a resected stomach after peptic ulcers is multiplied the risk of origin of stomach cancer in 2–3 times (duration of latent period hesitates from 15 to 40 years). The reason of such dependence is not found out, but there is a version, that this is linked with a gastric epithelium metaplasia by an intestinal type.

Pathomorphology

From all malignant formations of the stomach in 95 % adenocarcinoma is observed. Epidermoid cancer, adeno-acanthoma and carcinoid tumours do not exceed 1 %. Frequency of leiomyosarcoma hesitates within the limits of 1–3 %. Lymphoma of gastrointestinal tract is localized in a stomach.

The prognosis of localization depends on the degree of invasion, histological variants of tumour.

The macroscopic forms of cancer of stomach in different times were described variously. More than 60 years ago the German pathologist Bermann described 5

macroscopic forms of cancer of stomach: 1) polypoid or mushroom-like; 2) saucer-shaped or with ulcerous and expressly salient edges; 3) with ulcerous and infiltration of walls of stomach; 4) diffuse -infiltrate; 5) unclassified.

American pathopsychologists is selected 4 forms. The tumours of stomach with ulcerous are the most frequent macroscopic form of cancer of stomach and arise up on soil of chronic ulcer. The signs suspicious on malignization are: the sizes of ulcer more than 2 cm in a diameter, appearance of the heightened edges.

The polypoid tumours of stomach observed only in 10 %. These tumours can achieve considerable sizes without an invasion and metastasis. Scirrhus carcinoma is the third macroscopic type. This category of tumours also does not exceed 10 %. The scirrhus carcinoma is the signs of infiltration by anaplastic cancer cells, diffusely developed connecting tissue which results in the bulge and rigidity of wall of stomach. So called “small cancers” belong to the fourth macroscopic type. It meet comparative rarely (no more than 5 %) and is characterized by superficial accumulation of cancer cells which substitute for normal mucus in such kind: a) superficial flat layer which does not rise above the level of mucus; b) salient (bursting) formation; c) erosions.

Mainly (more than 50 %) tumours arise up in a antral part or in distal (lower) third of stomach, rarer (to 15 %) — in a body and in cardia (to 25 %).

However, lately more often observed cardioesophageal cancers and diminishment of frequency of tumours of distal parts of stomach. In 2 % cases meet the multicentric focuses of growth, but from data of some authors, this percent could be multiplied in 10 times after carefully histological inspection of the resected stomachs. This assertion is based on the theory of the “tumour field” (D.I. Holovin, 1992). Especially this typically for patients which has pernicious anaemia or chronic metaplastic disregenerative gastritis.

Metastasis is carried out by lymphogenic, hematogenic and implantation ways mostly. Three (from data of some authors, four) pools of lymphogenic metastasis are selected: left gastric (knots on passing of small curvature of stomach in a gastro-subgastric ligament and pericardial); splenic (mainly, suprainfrapancreatic knots); hepatic (knots in a hepato-duodenal ligament, right gastric omentum that lower pyloric groups, right gastric and suprapyloric groups, pancreatoduodenal group).

However, the such way of lymphogenic metastasis is conditional and incomplete,

as at presence of block lymph flow passes retrograde metastasis, so called “jumping metastases” which predetermine the origin of remote lymphogenic metastases in left supraclavicular lymph nodes (Virhov metastasis) appear, in Lymph nodes of left axillar and inguinal areas, metastases in a umbilicus.

Direct distribution: small and large omentum, esophagus and duodenum; liver and diaphragm; pancreas, spleen, bile ducts.

Front wall of stomach: colon bowel and mesocolon; organs and tissues of retroperitoneal space.

Lymphogenic metastasis: regional lymph nodes, remote lymph nodes, left supraclavicular lymph node (Virhov), lymph node of axillar area (Irish); in a umbilicus (sisters Joseph).

Hematogenic metastasis: liver, lungs, bones, cerebrum.

Peritoneal metastasis: peritoneum, ovarium (the Krukenberg metastasis), Duglas space (the Shnicler metastasis).

Classification (by system of TNM)

T— primary tumour.

T0 is a primary tumour is not determined.

Tx — not enough data for estimation of primary tumour.

Tis is invasive carcinoma: intraepithelial tumour without the invasion of own shell mucus (Carcinoma in situ).

T1 is a tumour infiltrate the wall of stomach to the submucous layer.

T2 is a tumour damages mucus, submucous and muscular layers.

T3 is a tumour germinates in a serous shell.

T4 is a tumour passes to the neighbouring structures.

N are regional lymphatic nodes.

Nx — not enough information for the damage assessment of lymphatic nodes.

No — metastases in regional lymph nodes are not present.

N1 are damaged perigastral lymph nodes in the distance no more than 3 cm from a primary tumour along small or large curvature of stomach.

N2 are damaged perigastral lymph nodes in the distance more than 3 cm from a primary tumour, which can be removed during operation, including lymph nodes placed along left gastric, splenic, abdominal and general hepatic arteries.

M is remote metastases.

Mx — not enough information for estimation of remote metastases.

Mo — remote metastases are not present.

M1 is presence of remote metastases.

Groupment by stages

Stage 0 T No Mo.

Stage I T1-2 No Mo.

Stage II T2-3 No Mo.

Stage III T1-4 N1-2 Mo.

Stage IV any T, any N M1.

Except for clinical classification (TNM or cTNM), for the most detailed study *pathological classification (postsurgical, posthistological) which is signed pTNM.*

G — histopathological differentiation:

G1 is the well differentiated tumour;

G2 is the moderately differentiated tumour;

G3-4 — it is badly or undifferentiated tumour.

Clinical management

All authors which are engaged in the study of problem of cancer of stomach underline absence or vagueness, no specificity of symptoms, especially on the early stages of disease. The displays of cancer of stomach are very various and depend on localization of tumour, character of its growth, morphological structure, distribution on contiguous organs and tissues. At localization of tumour in a cardial part patient complains firstly, as a rule, for appearance of dysphagy.

At careful, purposeful collection of anamnesis it is not succeeded to expose some other, most early symptoms, which precedes to dysphagy and forces a patient to appeal to the doctor. The unpleasant feeling behind a breastbone and feeling of unpassing of hard food on a esophagus appear at the beginning of disease. After some time (as a rule, it is enough quickly, during a few weeks, sometimes even days) a hard food does not pass (it is to wash down by water or other liquid). This period can be during 1–3 months. Patients address a doctor exactly in this period. Other symptoms appear to this time: regurgitation, pain behind a breastbone, loss of mass of body, sometimes even exhaustion, the grey colouring of person, a skin is dry, quickly grows general weakness. Sometimes patients address a doctor, when already with large effort a spoon-meat passes only or complete stenosis came.

At localization of tumour in the antral part of stomach the first complaints, as a rule, are up to appearance of feeling of weight in epigastric region after the reception of food (even in a two-bit), “feeling of saturation” (after the reception of glass of water), belch (at first it is simple by air, and then with a smell). Feeling of weight grows for a day, patients forced to cause vomiting. In the morning there can be vomiting by mucus with the admixtures of “coffee-grounds” (so called “cancer” water). Patients loses weight (mass of body is lost), a weakness, anaemia grows.

Tumours localized in the body of stomach show up either a pain syndrome or syndrome of so called “small signs” (A.I. Savitskyy, 1947), which is characterized by appearance of amotivational general weakness, decline of capacity, rapid fatigueability, depression (by the loss of interest to the environment), proof decline of appetite, gastric discomfort, making progress weight lost.

The carried chronic diseases of stomach, for which typical seasonality, can influence on the clinical sign of cancer of stomach. At appearance of “gastric” complaints out of season or in absent of effect from the got therapy concerning the exacerbation of “gastritis”, “ulcers” must guard a patient and doctor (symptom of “precipice” of gastric

anamnesis).

In case of occurring of “gastric” symptoms first in persons in age 50 years and older it is foremost necessary to eliminate the cancer of stomach.

In parts of patients cancer of stomach shows up only the metastatic damage of other organs or complications. More than twenty so called “atypical” forms, which are characterized by “causeless” anaemia, ascites, icterus, fever, edemata, hormonal disturbances, changes of carbohydrate exchange, intestinal symptoms, are distinguished.

During the examination of patients with the cancer of stomach the pallor of skin covers (at anaemia) is observed, in neglected case is “frog” stomach (sign of ascites).

During palpation determined painful in a epigastric area, sometimes possible to palpate the tumour.

During auscultation of patients with pylorostenosis it is possible to define “noise of splash”.

Laboratory information: hypochromic anaemia, neutrophilic leukocytosis, megascopic ESR; during examination of gastric secretion: hypo- and anacidity and achlorhydria.

Gastroduodenoscopy enables to diagnose a tumour even smaller 5 mm and conduct an aiming biopsy with histological examination of the taken material.

Roentgenoscopy and roentgenography examination of stomach. Basic signs: defect of filling, local absence of peristalsis, “malignant” relief of mucous tunic (Pic. 3.2.18).

Ultrasonic examination: presence of metastases in a liver, pancreas.

Computer tomography allows to estimate the basic parameters of tumour, germination in neighbouring organs and presence of metastases.

It is expedient to apply laparoscopy, mainly, for the decision of question about operable of tumour (diagnostics of metastatic defeat of organs of abdominal cavity).

Diagnosis program

1. Anamnesis and physical methods of examination.

2. Roentgenologic examination of stomach.
3. Endoscopic examination with a biopsy (if necessary from a few places and even repeatedly), cytologic and histological examination.
4. Sonography, computer tomography.
5. Laboratory, radioisotope methods of examination.
6. Laparoscopy.
7. Diagnostic (therapeutic) laparotomy.

Differential diagnostics

At an early cancer complaints depend on the previous gastric diseases. Therefore, on the basis of clinical information, suspecting a tumour is possible only on occasion, when in patients next to clear pain symptoms an appetite goes down, appear anaemia, general weakness. In practice an early cancer is recognized at purposeful screening, and also in the process of endoscopic or roentgenologic examination of gastric patients.

A differential diagnosis is conducted with an peptic ulcer, gastritis, polyposis, other gastric and ungastric diseases. For a cancer there is typical firmness of symptoms, instead of their seasonality (typical syndrome of “precipice” of gastric anamnesis) or tendency to their gradual progress.

The row of diseases, with which the cancer of stomach is to differentiate to the doctor, depends from character of complaints of patients.

Five basic clinical syndromes are selected:

- 1) pain;
- 2) gastric discomfort;
- 3) anaemic;
- 4) dysphagic;
- 5) disturbance of evacuation from a stomach.

At patients, at what cancer of stomach shows up a pain syndrome and syndrome of gastric discomfort, a differential diagnosis is conducted with the peptic ulcer, gastritis,

cancer of body of pancreas.

It is oriented on features dynamics of development of pain syndrome, ingravescence of the general condition, change of character of complaints.

A question about character of anaemia, source and nature of bleeding decides at an anaemic syndrome. In the process of examination attention is paid to the state of bottom of stomach, where bleeding malignant formations can be.

At a dysphagic syndrome a differential diagnosis is conducted with the cicatricial narrowing, achalasia of esophagus. For malignant formations testify short anamnesis, gradual progress of symptoms, signs of gastric discomfort, general weakness, weight lost.

At disturbance of evacuation from a stomach during stenosis of pyloric part, absence of ulcerous anamnesis, declining years of patients, relatively quick (weeks, months) growth of stenosis testify for tumor.

Tactic and choice of method of surgical treatment

The presence of cancer of stomach is a indications for surgical treatment. However, counting on success is possible only at presence of the limited tumours (within the limits of the 0–II stages). At the III stage of disease implementation of the widespread combined operations in a radical volume is possible, however most patients die during 1–2 years. A distal or proximal subtotal resection (Pic. 3.2.19) and total gastrectomy (Pic. 3.2.20) is performed with removing of large and small omentumes and regional areas of metastasis with obligatory histological examination of stomach on the lines of resections.

During the combined operations organs which are pulled in to the pathological process are removed.

In case of IV stage of disease and satisfactory state of patient palliative operations which improve quality of life of patient are performed.

In case of presence of complications (mainly stenosis) and grave common condition of patient perform symptomatic operative treatments.

Symptomatic is operations which will liquidate one of symptoms of cancer of stomach. In this group of operations include: 1) roundabout gastrojejunoanastomosis

(Pic. 3.2.21) and jejunostoma (in case of the stenosis tumours of stomach output); 2) gastrostoma (Pic. 3.2.22) in case of the cancer of cardial part of stomach with disturbance of patency; 3) edging of bleedingx vessels in case of complication of cancer by bleeding; 4) tamponade by omentum during the perforation of tumour.

The value of radial therapy and chemotherapy, as independent methods of treatment of cancer of stomach, is limited. Radial therapy is indicated for patients with cardial cancer as preoperative course or as palliative treatment. Adjuvant mono- or polychemotherapy (mainly by 5-phtoruracil) is conducted in a postoperative period as combined therapy and in case of dissemination of the tumours.

Prognosis. The indexes of five-year survival of patients with the cancer of stomach hesitate within the limits of 5–30 %, but, from data of most authors, they do not exceed 10 %.

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METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTSMETHODOLOGICAL INSTRUCTION TO LESSON №4
" ULCERATIVE DISEASE OF THE STOMACH (UDS) AND THE DUODENUM (UDD), COMPLICATED ULCER"

The aim. To master clinics, diagnostics, differential diagnostics of various clinical forms of UDS and UDD. To learn the treatment tactics, indications and contraindications for operative intervention, operative intervention type at various clinical forms of UDS and UDD, principles of postoperative period course, expertise of the working ability after operations for UDS and UDD.

Professional orientation of students. UDS and UDD is one of the most frequent surgical illnesses of organs of gastric-intestinal tract. Morbidity reaches the level of 10-15%. Major complications of UDS and UDD are perforation, bleeding, ulcerative stenosis, malignization. They are vitally dangerous and require urgent or pressing surgical interventions. Postoperative lethality after urgent operations because of UDS and UDD complications sometimes reaches 15-20%.

Methodology of Practical Class 9.00-12.00.**Algorithm of communicative skills:**

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened

Student's independent work program:**Theme №1. Modern principles of diagnostics of surgical treatment of the ulcerative disease of stomach and duodenum.**

1. Anatomic and physiologic features of the stomach and the duodenum.
2. Factors of the ulcerative disease development.
3. Pathogenesis of the ulcerative disease of the stomach and the duodenum.
4. Classification of ulcerative disease of the stomach and the duodenum.
5. Diagnosis methods of the ulcerative disease of the stomach and the duodenum.
6. Curing tactics at the ulcerative disease.
7. Modern principles of conservative and surgical treatment of UDS and UDD.
8. Choice of the operative treatment method at UDS and UDD.
9. Types of radical (organ-sparing and organ-preserving) and palliative operations for the reason of UDS and UDD.
10. Peculiarities of the postoperative period course after the operations for UDS and UDD reason.
11. Evaluation of treatment results of patients with UDS and UDD.
11. Disablement expertise after the operations for UDS and UDD reason.

Theme №2. Perforative and bleeding ulcer of stomach and duodenum.

1. Risk factors of the perforative ulcer of stomach and duodenum.
2. Pathogenesis of the perforative ulcer.
3. Classification of perforative gastroduodenal ulcers.
4. Phases of clinical course of the perforative ulcer.
5. Objective signs of the perforative gastroduodenal ulcer.
6. Differential diagnosis of the perforative ulcer with the acute appendicitis, acute pancreatitis, acute cholecystitis, acute ileus, renal colic, basal pleuropneumonia, myocardial infarction.
7. Peculiarities of the untypical and covered perforation of the gastroduodenal ulcer.
8. Roentgenic signs of perforation.
9. Laparocentesis, laparoscopy and endoscopy in the diagnostics of the perforative ulcer.
10. Curing tactics at the perforative ulcer.
11. Taylor's method in the perforative ulcer treatment
12. Indications and methods of radical and palliative operations for the reason of the perforative ulcer.
13. Peculiarities of the preoperative preparation and postoperative period course at the operative treatment of the perforating ulcer.
14. Risk factors of bleeding from the ulcer.
15. Pathogenesis of the acute gastric-intestinal bleeding.
16. Classification of gastric-intestinal bleedings.
17. Main and additional symptoms of bleeding ulcer.
18. Phases of clinical course of the bleeding ulcer.
19. Differential diagnosis of the bleeding ulcer with pulmonary bleeding, atherosclerotic, medicamentation bleedings, bleedings at blood diseases, portal hypertension, stomach tumours, Mallory-Weiss syndrome, erosive gastritis, hernias of the esophageal diaphragm foramen.
20. Subsidiary methods of the bleeding ulcer diagnosis.
21. Curing tactics at bleeding gastroduodenal ulcer.
22. Indications to conservative treatment of bleeding ulcer.
23. Principles of the conservative treatment of bleeding ulcer.
24. Indications for the operative treatment of bleeding ulcer.
25. Types of radical operations.
26. Types of palliative operations.
27. Peculiarities of the postoperative period course after the operations for bleeding ulcers.

Theme №3. Stenosing ulcer. Malignization and penetration/

1. Classification of cicatricial ulcerative stenosis of stomach outlet.
2. Clinical signs of the ulcerative stenosis depending on its stage.
3. Differential diagnosis of the cicatricial ulcerative stenosis with acute ileus, hernia of the esophageal opening of the diaphragm, achalasia of esophagus, duodenostasis (chronic duodenal obstruction), cicatricial postburn stenosis, stomach foreign body, stomach cancer.
4. Peculiarities of water-electrolytic exchange at ulcerative stenosis.
5. Verification methods of diagnosis of ulcerative stenosis of stomach and duodenum.
6. Curing tactics at stenosis of stomach outlet. Conservative and surgical treatment.
7. Peculiarities of the preoperative preparation of patients with the ulcerative stenosis of stomach outlet.
8. Methods of operative treatment of the ulcerative stenosis of stomach outlet.
9. Peculiarities of the postoperative period course.
10. Disablement expertise after the operations for ulcerative stenosis of stomach outlet.
11. Conception definition. Classification of penetration.
12. General clinical characteristics of penetrating ulcer.
13. Clinical characteristics of typical versions of stomach and duodenum penetration.
14. Clinical characteristics of untypical versions of stomach and duodenum penetration.
15. Differential diagnosis of penetrating ulcer.
16. Verification methods of penetration diagnosis.

17. Choice of curing tactics at penetrating ulcer of stomach and duodenum.
18. Peculiarities of operative treatment of patients with penetrating ulcer of stomach and duodenum.
19. Conception definition of ulcer-cancer, cancer from ulcer.
20. Malignization frequency depending on the ulcer allocation.
21. General clinical signs of ulcer malignization.
22. Verification methods of diagnosis of malignant gastric ulcer.
23. Curing tactics and peculiarities of operative treatment of patients with malignant gastric ulcer.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Anatomic and physiologic features of stomach and duodenum.
2. Modern theories of appearance and pathogenesis stages of UDS and UDD.
3. Definition and classification of various clinical forms of UDS and UDD.
4. Modern examination methods of secretory and motor-evacuative functions of stomach and duodenum.
5. Verification methods of UDS and UDD diagnosis.
6. Clinics, diagnostics, differential diagnostics and curing tactics choice at uncomplicated and complicated UDS and UDD.
7. Modern principles of complex conservative treatment of patients with UDS and UDD.
8. Modern methods of operative treatment of UDS and UDD.
9. Clinics, diagnostics, differential diagnostics, choice of curing tactics and methods of operative treatment at perforative ulcer of stomach and duodenum.
10. Clinics, diagnostics, differential diagnostics, choice of curing tactics and methods of operative treatment at bleeding ulcer of stomach and duodenum.
11. Peculiarities of postoperative period course after operations for UDS and UDD.

The student has to be able to:

1. To take the history of the patient with the suspect for UDS and UDD.
2. To reveal main clinical features and symptoms at various clinical forms of UDS and UDD.
3. To define free gas in the abdominal cavity.
4. To define the presence of free liquid in the abdominal cavity.
5. To argue and formulate the preliminary diagnosis.
6. To make a plan of the examination of the patient and explain the examination results (general blood test, examination of gastric secretion, plain roentgenogram of abdominal organs, roentgenoscopy and roentgenography of stomach and duodenum).
7. To define indications and contraindications for operative intervention, to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form of UDS and UDD.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.
(the instructor has tests & situational tasks)

1. First-line therapy for routine peptic duodenal ulcer disease includes:
 - A. Vagotomy and antrectomy.
 - B. Upper endoscopy and biopsy to rule out tumor.
 - C. Evaluation for *Helicobacter pylori*.
 - D. Serum gastrin determination.
 - E. Cream or milk-based "Sippy" diet.
2. All of the following are complications of peptic ulcer surgery except:
 - A. Duodenal stump blowout.
 - B. Dumping.
 - C. Diarrhea.
 - D. Delayed gastric emptying.
 - E. Steatorrhea.
3. The presentation of Zollinger-Ellison syndrome includes all of the following except:
 - A. Hyperparathyroidism in patients with multiple endocrine neoplasia type 1 (MEN 1) syndrome.
 - B. Diarrhea.
 - C. Migratory rash.
 - D. Jejunal ulcers.
 - E. Duodenal ulcers.
4. In patients with bleeding duodenal ulcers, the endoscopic finding associated with the highest incidence of rebleeding is:
 - A. Visible vessel.
 - B. Cherry-red spot.
 - C. Clean ulcer bed.
 - D. Duodenitis.
 - E. Shallow, 3-mm. ulcer.
5. All of the following are contraindications for highly selective vagotomy except:
 - A. Intractable duodenal ulcer disease.
 - B. Peptic ulcer disease causing gastric outlet obstruction.
 - C. Fundic peptic ulceration.
 - D. Cigarette chain smoking.
 - E. Perforated peptic ulcer disease with more than 24 hours' soilage.
6. Appropriate management of severe vomiting associated with gastric outlet obstruction from peptic ulcer disease includes all of the following except:
 - A. Nasogastric suction.
 - B. Intravenous hydration.
 - C. Nutritional assessment; upper endoscopy to rule out malignancy.
 - D. Intravenous H₂ antagonist.
 - E. Oral antacid therapy.+

7. Which of the following statements about gastric polyps is/are true?
- Like their colonic counterparts, gastric epithelial polyps are common tumors.
 - They are analogous to colorectal polyps in natural history.
 - Endoscopy can uniformly predict the histology of a polyp based on location and appearance.
 - In a given patient, multiple polyps are generally of a single histologic type.+
 - Gastric adenomatous polyps greater than 2 cm. in diameter should be excised because of the risk of malignant transformation.
8. All of the following are contraindications for highly selective vagotomy except:
- Intractable duodenal ulcer disease.+
 - Peptic ulcer disease causing gastric outlet obstruction.
 - Fundic peptic ulceration.
 - Cigarette chain smoking.
 - Perforated peptic ulcer disease with more than 24 hours' soilage.
9. Numerous epidemiologic associations have been made between (1) environmental and dietary factors and (2) the incidence of gastric cancer, including all except:
- Dietary nitrites.
 - Dietary salt.
 - Helicobacter pylori infection.
 - Dietary ascorbic acid.+
10. All of the following benign conditions are associated with increased rates of gastric cancer except:
- Pernicious anemia.
 - Multiple endocrine neoplasia type I (MEN 1).+
 - Adenomatous polyps.
 - Chronic atrophic gastritis.

The answers for the self-checking tests.

- 1 – C.
- 2 – E.
- 3 – C.
- 4 – B.
- 5 – A.
- 6 – E.
- 7 – D.
- 8 – A.
- 9 – D.
- 10 – B.

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POSTRESECTION AND POSTVAGOTOMY SYNDROMES. CLASSIFICATION. CLINICS. DIAGNOSTICS. CONSERVATIVE AND SURGICAL TREATMENT. PROPHYLAXIS.

DUMPING SYNDROME

Etiology and pathogenesis

Dumping syndrome is frequent complication of operations which are related to deleting or disturbance of function of goalkeeper (resection of stomach, vagotomy with antrectomy, vagotomy with drainage operations). It takes place in 10–30 % patients.

The rapid receipt (dumping) is considered the starting mechanism of dumping syndrome. During this concentrated, mainly carbohydrate, food passed from a stomach in an empty bowel.

In the phase changes of motility of thin bowel during dumping syndrome important part is acted by the hormones of thin bowel. In endocrine cells of APUD-системи on during dumping-syndrome observed degranulation and presence of hormones of mothiline, neurotensin and enteroglucagon.

The inadequate mechanical, chemical and osmotic irritation of mucous tunic of thin bowel by chymus results for the acute increase of blood flow in a bowel. The last is accompanied by the considerable redistribution of blood, especially in heavy case of dumping syndrome : blood supply of head, lower extremities is diminishes, a blood flow in a liver is multiplied.

The numeral examinations resulted in creation of osmotic theory –the principal reason of dumping syndrome is the decline of volume of circulatory plasma as a result of coming a plenty of liquid into the lumen of thin bowel from an of circulatory system and intercellular space.

Clinical management

For the clinical finding of dumping syndrome typical there is the origin of attacks of general weakness during acceptance of food or during the first 15–20 minutes after it. The attack begins from feeling of plenitude in a epigastric area and is accompanied by the unpleasant feeling of heat, that “spills” in the overhead half of trunk or on all body. Thus is acutely multiplied sweating. Then there is a fatigue, appear somnolence, dizziness, noise in ears, shaking of extremities and worsening of sight. These signs sometimes achieve such intensity, that patients forced to lie down. Loss of consciousness could be in the first months after operation. The attacks are accompanied by tachycardia, sometimes by the shortness of breath, headache, paresthesia of upper and lower extremities, polyuria and vasomotor rhinitis. At the end of attack or after it patients often notice grumbling in a stomach and diarrhea.

A milk or carbohydrate food is the most frequent provoking factor of dumping

syndrome. In a period between the attacks patients complain about rapid fatigueability, weakening of memory, decline of working capacity, change of mood, irritates, apathy. During roentgenologic examination after 5–15 minutes observed the increased evacuation of barium mixture through anastomosis by a wide continuous stream, expansion of efferent loop and rapid advancement of contrasting matter in the distal parts of thin bowel (Pic. 3.2.16).

By the expression of symptoms dumping syndrome is divided into three degrees of weight:

I degree is easy. Patients have the periodic attacks of weakness with dizziness, nausea, that appear after the use of carbohydrates and milk food and last no more than 15–20 min. During the attack a pulse becomes more frequent on 10–15 per min., arterial pressure rises or sometimes goes down on 1.3-2 KPa (10–15 mm Hg), the volume of circulatory blood diminishes on 200–300 ml. The deficit of mass of body of patient does not exceed 5 kg. A working capacity is well-kept. Medicinal and dietary treatment gives a good effect.

II degree — middle weight. Attacks of weakness with dizziness, pain in the region of heart, hyperhidrosis, diarrhea. Such signs last, usually, 20–40 min., arise up after the use of ordinary portions of some food. During such state a pulse becomes more frequent on 20–30 per min., arterial pressure is rises (sometimes goes down) on 2–2,7 KPa (15–20 mm Hg), the volume of circulatory blood diminishes on 300–500 ml. The deficit of mass of body of patient achieves 5–10 kg. A working capacity is reduced. Conservative treatment sometimes has a positive effect, but brief.

The III degree is hard. Patients are disturbed by the permanent, acutely expressed attacks with the collaptoid state, by a fainting fit, by diarrhea, which do not depend on character and amount of the accepted food and last about 1 hour. During the attack is multiplied frequency of pulse on 20–30 per 1 min; arterial pressure goes down on 2,7–4 KPa (20–30 mm Hg), the volume of circulatory blood diminishes more than on 500 ml. The deficit of mass of body exceeds 10 kg. Patients, as a rule, are disabled. Conservative treatment is ineffective.



Pic. 1. Dumping syndrome (quick evacuation of the contrast)

Tactic and choice of treatment method

The problem of treatment of patients with dumping syndrome is not easy. Before the surgical treatment, as a rule, must precede conservative. Patients with the disease of easy and middle degrees respond to conservative treatment, mainly with an enough quite good effect. At the heavy degree of disease such treatment more frequent serves as only preparation to operative treatment. If a patient does not give a consent for operation or at presence of contra-indications to operative treatment (disease of heart, livers, kidneys), conservative therapy is also applied. Such treatment must include dietotherapy, blood and plasma transfusion, correction of metabolism, hormonal preparations, symptomatic therapy, electro-stimulation of motility function of digestive tract.

The dietotherapy: using of high-calorie, various food rich in squirrel, by vitamins, by mineral salts, with normal content of fats and exception from the ration of carbohydrates which are easily assimilation (limitation of sugar, sweet drinks, honey, jam, pastry wares, kissel and

fruit compotes). All it is needed to use by small portions (5–6 times per days). If the signs of dumping syndrome appear after a food, such patients it is needed to lie down and be in horizontal position not less than 1 hour. At the heavy degree of dumping syndrome patients need to eat slowly, desirably lying on left. Such position creates the best terms for evacuation of food from a stomach. Thus recommend also to repudiate from too hot and cold foods.

Medicinal treatment must include sedative, replaceable, antiserotonin, hormonal and vitamin therapy. The indications to operative treatment of patients with dumping syndrome are: heavy passing of disease, combination of dumping syndrome of middle degree with other postgastrectomy syndromes (with the syndrome of efferent loop, hypoglycemic syndrome and progressive exhaustion) and uneffective of conservative treatment of the dumping syndrome of middle degree. Most methods of operative treatment of dumping syndrome are directed on renewal of natural way of passing of food on a stomach and intestine, improvement of reservoir function of stomach and providing of proportioning receipt of food in a thin bowel.

Depending on reasons and mechanisms of development of dumping syndrome there are different methods of the repeated reconstructive operations. All of them can be divided into four basic groups: I. Operations which slow evacuation from stump of stomach. II. Redoudenization. III. Redoudenization with deceleration of evacuation from stump of stomach. IV. Operations on a thin bowel and its nerves.

Basic stages of reconstructive operations: 1) disconnection of adhesions in an abdominal cavity, releasing of gastrointestinal and interintestinal anastomosis and stump of duodenum; 2) cutting or resection of efferent and afferent loops; 3) renewal of continuity of upper part of digestive tract.

For correction of the accompany postgastrectomy pathology it is better to apply combined anti- (iso-) peristaltic gastrojejunoplasty. Thus transplant by length 20–22 cm, located between a stomach and duodenum, must consist of two parts: antiperistaltic (7–8 cm), connected with a stomach, and isoperistaltic, connected with a duodenum. An antiperistaltic segment brakes dumping of stomach stump, and isoperistaltic — hinders the reflux of duodenum content.

HYPOGLYCEMIC SYNDROME

The attacks of weakness at a hypoglycemic syndrome arise up as a result of decline of content of sugar in a blood. It is accompanied by a acute muscular weakness, by headache, by falling of arterial pressure, by feeling of hunger and even by the loss of consciousness.

STAGES OF THE HYPOGLYCEVIC SYNDROME

I stage	Signs beghins after 2-2,5 h after food intake, 2-3 times per week. Patients does not feel it.
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II stage	Signs begins 2-3 times per week.
III stage	Signs begins every day. Patients always has sweet food and bread.

It is needed to remember, that at this pathology, unlike dumping-syndrome, acceptance of food especially sweet facilitates the state of patient. However in some patients both syndromes unite and the attacks of weakness can arise up as directly after food intake, so in a few hours after it. In patients with such pathology the best results are got after antiperistaltic gastrojejunoplasty (Fink, 1976).

POSTGASTRECTOMY (AGASTRIC) ASTHENIA

Etiology and pathogenesis

The postgastrectomy (agastric) asthenia arises up as a result of disturbance of digestive function of stomach, pancreas, liver and thin bowel.

In patients with such pathology stump of stomach almost fully loses ability to digest a food. It is related to the small capacity of stump and rapid evacuation of food from it, and also with the acute decline of production of hydrochloric acid and pepsin. In the mucous tunics of stump of stomach, duodenum and thin bowels as a result of fall of trophic role of gastrin and other hormones of digestive tract there are the progressive atrophy changes. Absence in gastric juice of free hydrochloric acid is the reason of acute diminishment of digestive ability of gastric juice and decline of it bactericidal. Such situation is assist in advancement to ascending direction of virulent flora, to development duodenitis, hepatitis, cholecystitis, dysbacteriosis, hypovitaminosis and decline of antitoxic function of liver. All it results in acute disturbance of evacuation from a stomach.

Clinical management

The clinical signs of postgastrectomy asthenia arise up after a some latent period which can last from a few months to some years. During this period patients often complain for a general weakness and bad appetite. The basic symptoms of postgastrectomy asthenia are: general weakness, edemata, acute weight loss, diarrhea, skin and endocrine abnormalities. The postgastrectomy asthenia more frequent meets at men at 40–50 years. In most cases diarrhea is the first symptom of disease, that can arise up in 2 months after operation. Diarrhea, usually, has permanent character and sometimes becomes profuse.

Weight loss appears too early, the deficit of mass of body achieves 20–30 kg. A patient quickly loses forces.

Tactic and choice of treatment method

Conservative treatment is the blood, plasma and albumen transfusions. These preparations are prescribed 2–3 times per a week. Correction of disturbances of electrolyte exchange is conducted at the same time (transfusion of solutions to potassium, calcium and others like that). For the improvement of processes of albumen synthesis anabolic hormones are prescribed.

Operative treatment foresees the inclusion in the digestion process of duodenum, increase of capacity of stump of stomach and deceleration of evacuation of its content.

SYNDROME OF AFFERENT LOOP

Etiology and pathogenesis

The afferent loop consists of part of duodenum, that stopped behind after a resection, area of empty bowel between a duodenojejunal fold and stump of stomach. The syndrome of afferent loop can arise up after the resection of stomach after the Bilrthoht-II method. Violation of evacuation from a afferent loop and vomiting by a bile are its basic signs.

Acute and chronic obstruction of afferent loop are distinguished. The reason of acute obstruction is mechanical factors: postoperative commissure, volvulus, internal hernia, invagination, jamming behind mesentery of loop of bowel and stenosis of anastomosis.

Frequency of origin of sharp obstruction of afferent loop hesitates within the limits of 0,5–2 %. The disease can arise up in any time after operation: in a few days or a few years.

Chronic obstruction of afferent loop (actually syndrome of afferent loop), as well as acute, can arise up in any time after operation, however more often it develop after the resection of stomach with gastroenteroanastomosis on a long loop, especially when operation is performed without entero-enteroanastomosis by Brown.

The etiologic factors of syndrome of afferent loop are divided into two groups: 1) mechanical (postoperative commissure, invagination, disturbance of evacuation on a afferent loop, wrong location of afferent loop, very long afferent loop, fall of mucous tunic of afferent loop into a stomach); 2) functional (hypertensive dyskinesia of bilious ways and duodenum, damage and irritation of trunks of vagus nerves, hypotensive and spastic states of upper part of digestive tract, heightened secretion of bile and juice of pancreas under act of secretin and cholecystokinin).

Clinical management

For the clinical picture of acute obstruction typical is permanent, with a tendency to strengthening, pain in a epigastric area or in right hypochondrium, nausea and vomiting. At complete obstruction a bile in vomiting masses is absent. The general condition of patient progressively gets worse, the temperature of body rises, leukocytosis grows, tachycardia grows.

At the objective examination painful and tension of muscles of abdominal wall is observed. In a epigastric area it is often possible to palpate tumular lump. Possible cases, when the increase of pressure in a bowel is passed on bilious ways and channels of pancreas. There can be pain and icterus in such patients. There are necrosis and perforation of duodenum with development of peritonitis during further progress of process. Acute obstruction of afferent loop in an early postoperative period can be the reason of insufficiency of stump of duodenum also.

During the roentgenologic examination of organs of abdominal cavity it is visible round form area of darkening and extended, filled by gas, bowels loop.

Patients, usually, complain for feeling of weight in a epigastric area and arching in right hypochondrium, that arises in 10–15 min. after acceptance of food and gradually grows. Together with that, appear nausea, bitter taste in a mouth, heartburn. Then there is increasing pain in a right to epigastric area. During this pain arises intensive, sometimes repeated vomiting by a bile, after which the all symptoms disappear. It could be after certain kind of food (milk, fats) or its big amount. Very rarely vomiting by bile unconnected with the feed. In heavy case patients lose up to 1 liter of bile with vomiting masses. During the objective examination observed subicteritiousness of the sclera, sign of dehydration of organism (decline of turgor of skin, dry tongue, oliguria, concentrated urine). Emptying is irregular, grey color, with considerable content of undigested fat and muscular fibres. Anaemia can develop at heavy passing of disease.

Distinguished easy, middle and heavy degrees of afferent loop syndrome. In patients with the easy degree of disease vomiting is 1–2 times per a month, and insignificant regurgitation arise up through 20 min – 2 hour after a food, more frequent after the use of milk or sweet food. At middle degree of afferent loop syndrome such attacks repeat 2–3 times per week, patients are disturbed by the considerably expressed pain syndrome, and with vomiting up to 200–300 ml of bile is lost. For a heavy degree the daily attacks of pain are typical, that is accompanied by vomiting by a bile (up to 500 ml and more).

DEGREES OF AFFERENT LOOP SYNDROME

easy	vomiting is 1–2 times per a month, and insignificant regurgitation arise up through 20 min – 2 hour after a food, more frequent after the use of milk or sweet food.
middle	attacks repeat 2–3 times per week, patients are disturbed by the considerably expressed pain syndrome, and with vomiting up to 200–300 ml of bile is lost.
heavy	the daily attacks of pain are typical, that is accompanied by vomiting by a bile (up to 500 ml and more).

A roentgenologic examination of the patients with the afferent loop syndrome is unspecific. Neither the passing of contrasting matter nor absence of filling of afferent loop can

be considered as pathognomic signs of syndrome of afferent loop.

Tactic and choice of treatment method

Treatment of acute obstruction of afferent loop is mainly operative. Essence of it is the removal of barriers of evacuation of content from an afferent loop. Adhesions are dissected, volvulus is straightened, invagination or internal hernia is liquidated. For the improvement of evacuation between afferent and efferent loops performs the entero-enteroanastomosis type “end-to-end” or after the Roux method.

Conservative treatment of syndrome of afferent loop is ineffective and, mainly, is mean the removal of hypoproteinemia and anaemia, spasmolytic preparations and vitamin are appointed. With this purpose a blood, plasma and glucose is poured with insulin, a novocaine lumbar blockade and blockade of neck-pectoral knot, washing of stomach is also done.



Рис. 2. Resection by Roux

All *operative methods* of treatment of afferent loop syndrome can be divided into three groups:

- I. Operations, that will liquidate the bends of afferent loop or shorten it.
- II. Drainage operations.
- III. Reconstructive operations. [video](#)

The operations of the first group, directed on the removal of bends and invagination of afferent loop, can not be considered as radical. They need to be performed only at the grave general condition of patient.

The widest application in clinical practice at the syndrome of afferent loop has the

operation offered by Roux.

For the prophylaxis of afferent loop syndrome it is necessary to watch after correct imposition of anastomosis during the resection of stomach: to use for the gastroenteroanastomosis short loop of thin bowel (6–8 cm from the Treits ligament) for imposition, to sew afferent loop to small curvature for creation of spur, to fix reliably stump of stomach in peritoneum of transverse colon.

REFLUX-ESOPHAGITIS

Etiology and pathogenesis

The origin of reflux after the distal resection of stomach is conditioned by some factors:

I. Traumatic factors: 1) traction of stomach during operation as reason of sprain of ligament of proximal part of stomach and mobilization of large curvature of stomach; 2) cutting of vessels of stomach and oblique muscles of it wall, in particular on small curvature; 3) vagotomy, that is accompanied by cutting of phrenico-esophageal and gastrophrenic ligaments; 4) imposition of gastrointestinal anastomosis, especially direct gastroduodenoanastomosis by Billroth-I, that results in smoothing of the Hisa corner; 5) frequent aspiration of gastric content in a postoperative period, that causes superficial esophagitis.

II. Trophic factors: 1) damage of vessels which are the reason of ischemia in the area of esophago-gastric connection, and thrombophlebitis of cardial part of stomach; 2) disturbance of influencing of neurohumoral factors which take part in innervations of esophagus; 3) disturbance of trophism of diaphragm as a result of hypoproteinemia and weight loss; 4) ulcerous diathesis and megascopic volume of gastric secretion (especially nightly); 5) regurgitation of alkaline content of duodenum in stump of stomach which reduces tone of it muscular shell.

III. Mechanical factors: 1) gastric stasis; 2) diminishment of volume of gastric reservoir, that is accompanied by the increase of intragastric pressure.

Clinical management

The clinical picture of gastroesophageal reflux is conditioned by the mechanical and chemical irritations of esophagus by content of stomach or thin bowel. As a result, there is esophagitis, which can be catarrhal, erosive or ulcerous-necrotic. The symptoms of reflux are very various and can simulate different diseases of both pectoral and abdominal cavity organs.

The basic complaint of patients with this pathology is a smart behind a breastbone, especially in the area of the its lower part. It, usually, spreads upwards and can be accompanied by considerable salivation. Strengthening of pain at inclinations of trunk gave to the French authors an occasion to name this sign the “symptom of laces”. Unendurable heartburn is the second complaint, that arises up approximately in 1–2 hours after the food intake. Patients forced often to drink, somehow to decrease the unpleasant feelings, however this, certainly, does not bring them facilitation. Some of them, in addition, complain for bitter taste in a

mouth.

Pain behind a breastbone often can remind the attack of stenocardia with typical irradiation. Sometimes such reflux is able to provoke real stenocardia.

Hypochromic anaemia is the frequent symptom of gastroesophageal reflux too.

The diagnosis of gastroesophageal reflux, mainly, is based on clinical information, results of roentgenologic examination, esophagoscopy.

The edema, hyperemia of mucous tunic of esophagus, easy bleeding and vulnerability it during examination, surplus of mucus and erosions covered by fibrin tape *is* considered *the endoscopic signs* of esophagitis. In doubtful case at the insignificantly expressed macroscopic changes the biopsy of mucous tunic helps to set a diagnosis.

Tactic and choice of treatment method

Treatment of patients with gastroesophageal reflux is mainly *conservative*. Very important is diet, which avoid spicy, rough and hot food. Eating is needed often, by small portions. It is impossible also to lie down after the food intake, because the gastric content can flow in a esophagus. A supper must be not later than for 3–4 hours before sleep. Between the reception of food does not recommend to use a liquid. Next to that, it is necessary to remove factors which promote intraperitoneal pressure (carrying to the bracer, belt, constipation, flatulence). Sleeping is needed in position with a lift head and trunk. From medicinal preparations it is useful to recommend enveloping preparation.

Operative treatment of gastroesophageal reflux, that arose up after the distal resection of stomach, it is needed to recommend to the patients with the protracted passing and uneffective of conservative treatment. During operation, mainly, performed renewal of the broken Hisa angle. In addition, performed esophagoplasty, fundoplication by Nessen's and esophagofrenofundoplication.

The prophylaxis of this complication consists in the study of the state of cardial part of stomach before and during every resection and fixing of bottom of stomach to the diaphragm and abdominal part of esophagus during leveling the Hisa angle.

ALKALINE REFLUX-GASTRITIS

Etiology and pathogenesis

Alkaline reflux-gastritis meets in 5–35 % operated patients after the resection of stomach, antrectomy, gastroenterostomy, vagotomy with pyloroplasty, and also cholecystectomy and papillosphincteroplasty.

The reason of this complication is influence of duodenum content for the mucous tunic of stomach (bilious acids, enzymes of pancreas and isolecithin). Last, forming from bile lecithin under act of phospholipase A, able to destroy the cells of superficial epithelium of mucous tunic of stomach by removing of lipid from their membranes. As a result the erosions and ulcers are formed in the patient organism. Bilious acids also has the expressed detergent's properties. As isolecithin and bilious acids, the very important bacterial flora which directly

and through toxins can cause the damage of mucous tunic of stomach stump. Also, alkaline environment and disturbance of evacuation from the operated stomach influence favourably on microflora growth.

Clinical management

For the clinical picture of alkaline reflux-gastritis the permanent poured out pain in a epigastric area, belch and vomiting by a bile are typical. At some patients heartburn and pain is observed behind a breastbone also. In majority patients so proof loss of weight takes place, that even the protracted complex therapy and valuable feed does not provide addition to the deficit of mass of body. There are typical signs also – anaemia, hypo- or achlorhydria.

Reliable diagnostics of alkaline reflux-gastritis became possible after wide introduction in clinical practice of endoscopic examination. In such patients during gastroscopy hyperemia of mucous tunic of stomach is observed. It is often possible to observe reflux in the stomach of duodenum content. During histological examination of biopsy material a chronic inflammatory process, intestinal metaplasia, diminishment of mass of coating cells and area of hemorrhages are found. All it testifies the deep degenerative changes in the mucous tunic of stomach. The some authors underlines that the inflammatory changes, at least in the area of anastomosis, are observed in most persons which carried the resection of stomach. So, endoscopic examination can not be considered deciding in diagnostics. Even the diffuse inflammatory changes can take place in absent of clinical symptoms and, opposite, in case with expressed clinical symptoms the minimum changes of mucous tunic of stomach are sometimes observed.

Tactic and choice of treatment method

Conservative treatment of reflux-gastritis (sparing diet, antacides, enveloping preparations), usually, is ineffective. Existent methods of surgical treatment, mainly, directed on the removal of reflux of duodenum content to the stomach. Most popular is operation by the Roux method. The some surgeons considers that distance from gastroenteroanastomosis to interintestinal anastomosis must be 45–50 cm.

PEPTIC ULCER OF THE ANASTOMOSIS

Etiology and pathogenesis

Main reason of origin of peptic ulcer of anastomosis is leaving of the hyperacid state of stomach mucous, even after the performed operation. Such phenomenon can be consequence of many reasons: primary economy resection, wrong executed resection (when the mucous tunic of pyloric part is abandoned in stump of duodenum or stomach), heightened tone of vagus nerves and the Zollinger-Ellison syndrome.

Clinical management

Peptic ulcers, usually, arise up after operation during the first year. Typical signs are pain, vomiting, weight loss, bleeding, penetration and perforation.

Pain is the basic symptom of peptic ulcer. Often it has the same character and

localization, as well as at peptic ulcer. However often observe it moving to the left or in the umbilical area. At first patients bind such feelings to the use of food, but then specify nightly and hungry pain. It at first is halted after a food, but in course of time is become permanent, unendurable, independent from food intake. It can increase during the flounces, the walk, can irradiate in the back, thorax or shoulder.

During the *objective examination* of patients is often possible to expose on a stomach hyperpigmentation from a hot-water bottle. During palpation to the left from epigastric area near a umbilicus the painful and moderate muscles tension of abdominal wall is observed. Sometimes is possible to palpate inflammatory infiltrate of different sizes. During the examination of patients with a peptic ulcer the important role has determination of gastric secretion against a background of histamine and insulin stimulation. There is a necessity also examination of basal secretion. These preoperative examinations in most patients enable to set the reason of hypersecretion which can be: 1) heightened tone of vagus nerves (positive Hollander test); 2) economy resection of stomach, often in combination with the heightened tone of vagus nerve (considerable increase of gastric secretion after histamine or pentagastrin stimulation in combination with the positive Hollander test); 3) abandoned part of mucous tunic of antral part of stomach (high basal secretion and small increase of secretion in reply to histamine and insulin stimulator); 4) the Zollinger-Ellison syndrome.

Roentgenologic diagnostics of peptic ulcer, usually, is difficult, especially at shallow, flat ulcers, bad mobility and insufficient function of anastomosis. A niche is the direct sign of a similar pathology, indirect are the expressed inflammatory changes of mucous tunic of stump of stomach and bowel, painful point in the projection of stump of stomach and anastomosis and bad function of anastomosis. The deciding value in diagnostics has endoscopic examination.

Tactic and choice of treatment method

Conservative treatment of peptic ulcers, as a rule, is ineffective. So, operation must be the basic type of treatment. The choice of method of operative treatment depends on character of previous operation and from abdominal cavity pathology found during the revision. For today the most important parts of the repeated operations is vagotomy. There is obligatory also during the resection of stomach on the exception the revision of duodenum stump for liquidation of possibly abandoned mucous tunic of antral area.

Operative treatment at a peptic ulcer must consist of certain stages. Laparotomy and disconnection of adhesions through a considerable spike process (increasing of stomach, loops of intestine and liver to the postoperative scar) almost always causes large difficulties.

After the selection of anastomosis with afferent and efferent loops the last cut by the “UKL-60 appliance”, within the limits of healthy tissues with renewal of intestine continuity by “end-to-end” type anastomosis.

At patients with a peptic ulcer, that developed after gastroenterostomy, cut a duodenum and sutured its stump by one of the described methods. During it there can be the difficulties related to the presence in it active ulcer. When peptic ulcers do not cause rough deformation of stomach, apply degastroenterostomy, vagotomy and drainage operations.

In the case of the considerably expressed spike process it is possible to execute trunk subdiaphragmatic vagotomy, and in case of the insignificantly changed topography of this area — selective gastric vagotomy.

It is important to note, that stomach resected together with anastomosis, peptic ulcer and eliminated area of empty bowel by one block.

GASTRIC-COLON FISTULA

This pathology arises up as a result of perforated of peptic ulcer in a transverse colon with formation of connection between a stomach, small or large intestine.

Clinical management

Diagnostics of gastro-colon fistula at patients with expressed clinical signs of disease does not difficult. However, symptoms are often formed and is indicated up slowly, so such patients with different diagnoses long time treat oneself in the therapeutic or infectious parts.

The typical signs of this pathology is considered diminishment or disappearance of pain, that was before, and profuse, that does not respond to treatment, diarrhea. Patients has emptying up to 10–15 times per days and even more frequent. An excrement contains a plenty of undigested muscular fibres and fat acids (steatorrhea). In case of wide fistula an undigested food can be with an excrement.

Excrement smell from a mouth, usually, notice surrounding. The patients does not feel it. However appearance of excrement belch is indicate the hit into the stomach of excrement masses and gases, and could confirm this pathology.

The such patients very quickly lose weight (mass of body goes down on 50–60 %), their skin becomes pale with a grey tint. The protein-free edemata, ascites, hydrothorax, anasarca, signs of avitaminosis appear in non-treated case.

Through the severe losses of liquid and nonassimilable food there can be the increased appetite and unendurable thirst in such patients. However, they adopt a plenty of liquid and food but the state of them continues to get worse.

Headache, apathy and depression is observed, and at the objective examination is exhaustion (ochre colour of skin, dryness and decline of it turgor, edemata or slurred of swelling extremities, atrophy of muscles). A stomach often moderately pigmented from hot-water bottles, subinflated, with the visible peristalsis of intestine. During the changes of patient position it is possible to hear grumbling, splash and transfusion of liquid. The examination of blood can expose hypochromic anaemia.

Roentgenologic examination is a basic diagnostic method. There are three varieties of such examinations of gastro-colon fistula. During the examination with introduction of barium mixture through a mouth the hit of contrasting matter directly from a stomach into a colon is the typical roentgenologic symptom of such pathology. Irrigoscopy is more perfect and effective method. With suspicion on gastro-colon fistula it is better to perform irrigoscopy. Passing of contrasting matter to the stomach at this manipulation testifies the presence of fistula. The third

method is insufflation of air in a rectum. With it help on the screen it is possible to observe the location and passing of fistula, and also, as a result, hit of air in a stomach, increase of it gas bubble. Thus there can be the belch with an excrement smell.

The important role played the tests with dyes: at peroral introduction of methylene-blue after the some time it found in excrement masses or, opposite, after an enema with methylene-blue dye appears in a stomach.

Tactic and choice of treatment method

Treatment of gastro-colon fistula is exceptionally operative. It needs to be conducted after intensive preoperative preparation with correction of metabolism. All operations which can be applied at treatment of patients with gastro-colon fistula divide into palliative and radical (single-stage operation and multi-stage operation).

During the *palliative operations* the place of fistula of stomach, transverse colon and jejunum is disconnected and then sutured the created defects. Other variant is disconnection of stomach and transverse colon and leaving the gastroenteroanastomosis. It is necessary to remember, that during such operations the only fistula always removed and does not performed the resection of stomach. Clearly, that such situation also does not eliminate possibility of relapse of peptic ulcer and development of its complications. Taking into account it, palliative operations can be recommended in those case only, when the general condition of patient does not allow to perform radical operation.

Single-stage operation radical operations. The most widespread is degastroenterostomy with the resection of stomach. However, it is needed to remember that operation of disconnection of fistula, suturing of opening in the jejunum and transverse colon on the lines of fistula and resection of stomach applies only in case of absent of infiltrate and deformation and in the conditions of possibility to close a defect in bowels without narrowing of their lumen. This operation is the simplest, is enough easily carried by patients and it is enough radical.

THE SCAR DEFORMATION AND NARROWING OF ANASTOMOSIS AFTER THE STOMACH RESECTION

Such complications appear through considerable time after operation (from 1 month to one year). Disturbances of function of gastrointestinal anastomosis can be caused by the reasons, related both to the technical mistakes during operation and with pathological processes which arose up in the area of anastomosis.

Clinical management

The clinical picture of disturbance of anastomosis function, mainly, depends from the degree of its closing. At complete it obstruction in patients arise up intensive vomiting, pain in a epigastric area, the symptoms of dehydration and other similar signs appear. In other words,

the clinic of stenosis of the stomach output develops. Clearly, that during incomplete narrowing the clinical signs will be expressed less, and growth of them — more slow. Sometimes disturbance of evacuation can unite with the syndrome of afferent loop with a inherent clinical picture. At the roentgenologic examination of such patients expansion of stomach stump is exposed with the horizontal level of liquid and small gas bubble. Evacuation from it is absent or acutely slow.

Tactic and choice of treatment method

Treatment of scar deformations and narrowing of anastomosis must be operative and directed for the disconnection of accretions and straightening of the deformed areas. In case of presence in patients large inflammatory infiltrate it does not need to perform disconnection. In such cases it is the best to apply roundabout anastomosis. If a resection by Finsterer was done in such patient, better to perform anterior gastroenteroanastomosis, and after a resection by Billroth-I — posterior. As a result of conducting of such operations the state of patient, as a rule, gets better, and often recovered the function of primary anastomosis.

CONSEQUENCES AND COMPLICATIONS OF GASTRECTOMY

Removing of all stomach and exception of duodenum from the process of digestion of food cause plural functional disturbances in an organism. Some of them meet already after the resection of stomach (dumping-syndrome, hypoglycemic syndrome), other more inherent for gastrectomy (anaemia, reflux- esophagitis and others like that).

Most patients, that carried gastrectomy, complain for a considerable physical weakness, heightened fatigueability, sometimes is complete weakness, loss of activity and acute decline of work capacity. Almost all of them notice bad sleep, worsening of memory and heightened irritates. The appearance of patients is typical. Their skin insignificantly hyperpigmentated, dry, its turgor reduced, noticeable atrophy of muscles. Can be the signs of chronic coronal insufficiency in such patients, and in older-year persons is typical picture of stenocardia. Except for it, can be hypotension, bradycardia and decline of voltage on EKG; during auscultation deafness of tones is observed. From the side of the hormonal system the decline of function of sexual glands is typical: in men — declines of potency, in women — disturbances of menstrual cycle, early climax. Can be the signs of hypovitaminosis A, B, C and decline of resistibility of organism to chill, infectious diseases and tuberculosis.

The decline of mass of body is observed in 75 % patients, that carried gastrectomy. It is conditioned by the decline of power value of food as a result of disturbance of digestion, bad appetite and wrong diet. As a result of progressive hypoproteinemia there can be the protein-free edemata.

Tactic and choice of treatment method

Patients with such pathology must be under the permanent clinical supervision and 1–2

times per year during a month to have the course of stationary prophylactic treatment which includes psycho-, diet-, vitaminotherapy, correcting and replaceable therapy, and also prophylaxis of anaemia.

Psychotherapy is especially indicated in the psychodepressive and asthenic states. It is performed in combination with medicinal treatment. Hypnotic preparation, bromide, tranquilizers are applied.

A food must be correctly prepared, without the protracted cooking. Patients need to feed on 6–10 times per days by small portions.

Next to dietotherapy, it is constantly necessary to apply replaceable therapy (Pancreatine, Pansinorm, Festal, Intestopan). In case of absent of esophagitis hydrochloric acid is appointed. For the improvement of albuminous exchange anabolic hormones are applied.

In case of reflux-esophagitis there are indicated feeds by small portions with predominance of liquid, ground, jelly-like foods, astringent, coating, anticholinergic preparations. Between the receptions of food does not recommend to use a liquid. In case of dysphagy appoints a sparing diet.

For the prophylaxis of iron-deficiency anaemia, that arises up in the first 2–3 years after gastrectomy, important the indication of iron preparations.

For warnings and treatments of pernicious anaemia applied cyanocobalamin for 200 mcg through a day and folic acid. Packed red blood cells is indicated in heavy case.

Differential diagnostics and clinical variants

Acute appendicitis is an inflammation of vermiform appendix caused by festering microflora.

Most frequent causes of acute appendicitis are festering microbes: intestinal stick, streptococcus, staphylococcus. Moreover, microflora can be in cavity of appendix or get there by hematogenic way, and for women – by lymphogenic one.

Factors which promote the origin of appendicitis, are the following: a) change of reactivity of organism; b) constipation and atony of intestine; c) twisting or bends of appendix; d) excrement stone in its cavity; e) thrombosis of vessels of appendix and gangrene of wall as a substance of inflammatory process (special cases).

Simple (superficial) and destructive (phlegmonous, gangrenous primary and gangrenous secondary) appendicitises which are morphological expressions of phases of acute inflammation that is completed by necrosis can be distinguished.

In simple appendicitis the changes are observed, mainly, in the distant part of appendix. There are stasis in capillaries and venule, edema and hemorrhages. Focus of festering inflammation of mucus membrane with the defect of the epithelium covering is formed in 1–2 hours (primary affect of Ashoff). This characterizes acute superficial appendicitis. The phlegmon of appendix develops to the end of the day. The organ increases, its serous tunic becomes dimmed, sanguineous, stratifications of fibrin appear on its surface, and there is pus in cavity.

In gangrenous appendicitis the appendix is thickened, its serous tunic is covered by dimmed fibrinogenous tape, differentiating of the layer structure through destruction is not

succeeded.

Four phases are distinguished in clinical passing of acute appendicitis: 1) epigastric; 2) local symptoms; 3) calming down; 4) complications.

The disease begins with a sudden pain in the abdomen. It is localized in a right iliac area, has moderate intensity, permanent character and not irradiate. With 70 % of patients the pain arises in a epigastric area - it is an epigastric phase of acute appendicitis. In 2–4 hours it moves to the place of appendix existence (the Kocher's symptom). At coughing patients mark strengthening of pain in a right iliac area – it is a positive cough symptom.

Together with it, nausea and vomiting that have reflex character can disturb a patient. Often there is a delay of gases. The temperature of body of most patients rises, but high temperature can occur rarely and, mainly, it is a low grade fever. The general condition of patients gets worse only in case of growth of destructive changes in appendix.

During the examination it is possible to mark, that the right half of stomach falls behind in the act of breathing, and a patient wants to lie down on a right side with bound leg.

Painfulness is the basic and decisive signs of acute appendicitis during the examination by palpation in a right iliac area, tension of muscle of abdominal wall, positive symptoms of peritoneum irritation. About 100 pain symptoms characteristic of acute appendicitis are known, however only some of them have the real practical value.

The Blumberg's symptom. After gradual pressing by fingers on a front abdominal wall from the place of pain quickly, but not acutely, the hand is taken away. Strengthening of pain is considered as a positive symptom in that place. Obligatory here is tension of muscles of front abdominal wall.

The Voskresenkyy Symptom. By a left hand the shirt of patient is drawn downward and fixed on pubis. By the taps of 2-4 fingers of right hand epigastric area is pressed and during exhalation of patient quickly and evenly the hand slides in the direction of right iliac area, without taking the hand away. Thus there is an acute strengthening of pain.

The Bartomier's symptom is the increase of pain intensity during the palpation in right iliac area of patient in position on the left side. At such pose an omentum and loops of thin intestine is displaced to the left, and an appendix becomes accessible for palpation.

The Sitkovsky's symptom. A patient, that lies on left, feels the pain which arises or increases in a right iliac area. The mechanism of intensification of pain is explained by displacement of blind gut to the left, by drawing of mesentery of the inflamed appendix.

The Rovsing's symptom. By a left hand a sigmoid bowel is pressed to the back wall of stomach. By a right hand by ballotting palpation a descending bowel is pressed. Appearance of pain in a right iliac area is considered as a sign characteristic of appendicitis.

The Obrazcov's symptom. With the position of patient on the back by index and middle fingers the right iliac area of most painful place is pressed and the patient is asked to heave up the straightened right leg. At appendicitis pain increases acutely.

The Rozdolsky's symptom. At percussion there is painfulness in a right iliac area.

The general analysis of blood does not carry specific information, which would specify the presence of acute appendicitis. However, much leukocytosis and change of formula to the left in most cases can point to the present inflammatory process.

Acute appendicitis in children. With children of infancy acute appendicitis can be seen infrequently, but, quite often carries atypical character. All this is conditioned, mainly, by the features of anatomy of appendix, insufficient of plastic properties of the peritoneum, short omentum and high reactivity of child's organism. The inflammatory process in the appendix of children quickly makes progress and during the first half of days from the beginning of disease there can appear its destruction, even perforation. The child, more frequent than an adult, suffers vomiting. Its general condition gets worse quickly, and already the positive symptoms of irritation of peritoneum can show up during the first hours of a disease. The temperature reaction is also expressed considerably acuter. In the blood test there is high leukocytosis. It is necessary to remember, that during the examination of calmless children it is expedient to use a chloral hydrate enema.

Acute appendicitis of the people of declining and old ages can be met not so often, as of the persons of middle ages and youth. This contingent of patients is hospitalized to hospital rather late: in 2–3 days from the beginning of a disease. Because of the promoted threshold of pain sensitiveness, the intensity of pain in such patients is small, therefore they almost do not fix attention on the epigastric phase of appendicitis. More frequent are nausea and vomiting, and the temperature reaction is expressed poorly. Tension of muscles of abdominal wall is absent or insignificant through old-age relaxation of muscles. But the symptoms of irritation of peritoneum keep the diagnostic value with this group of patients. Thus, the sclerosis of vessels of appendix results in its rapid numbness, initially-gangrenous appendicitis develops. Because of such reasons the destructive forms of appendicitis prevail, often there is appendiceal infiltrate.

With pregnant women both the bend of appendix and violation of its blood flow are causes of the origin of appendicitis. Increased in sizes uterus causes such changes. It, especially in the second half of pregnancy, displaces a blind gut together with an appendix upwards, and an overdistension abdominal wall does not create adequate tension. It is needed also to remember, that pregnant women periodically can have a moderate pain in the abdomen and changes in the blood test. Together with that, psoas-symptom and the Bartomier's symptom have a diagnostic value at pregnant women.

Clinical passing of acute appendicitis at the atypical placing (not in a right iliac area) will differ from a classic vermiform appendix (Pic. 3.3.1).

Appendicitis at retrocecal and retroperitoneal location of appendiceal appendix can be with 8–20 % patients. Thus an appendix can be placed both in a free abdominal cavity and retroperitoneal. An atypical clinic arises, as a rule, at the retroperitoneal location. The patients complain at pain in lumbus or above the wing of right ilium. There they mark painfulness during palpation. Sometimes the pain irradiates to the pelvis and in the right thigh. The positive symptom of Rozanov — painfulness during palpation in the right Pti triangle is characteristic. In transition of inflammatory process on an ureter and kidney in the urines analysis red corpuscles can be found.

Appendicitis at the pelvic location of appendix can be met in 11–30 % cases. In such patients the pain is localized above the right Poupart's ligament and above pubis. At the very low placing of appendix at the beginning of disease the reaction of muscles of front abdominal

wall on an inflammatory process can be absent. With transition of inflammation on an urinary bladder or rectum either the dysuric signs or diarrhea develops, mucus appears in an excrement. Distribution of process on internal genital organs provokes signs characteristic of their inflammation.

Appendicitis at the medial placing of appendix. The appendix in patients with such pathology is located between the loops of intestine, that is the large field of suction and irritation of peritoneum. At these anatomic features mesentery is pulled in the inflammatory process, acute dynamic of the intestinal obstruction develops in such patients. The pain in the abdomen is intensive, widespread, the expressed tension of muscles of abdominal wall develops, that together with symptoms of the irritation of peritoneum specify the substantial threat of peritonitis development.

For the subhepatic location of appendix the pain is characteristic in right hypochondrium. During palpation painfulness and tension of muscles can be marked.

Left-side appendicitis appears infrequently and, as a rule, in case of the reverse placing of all organs, however it can occur at a mobile blind gut. In this situation all signs which characterize acute appendicitis will be exposed not on the right, as usually, but on the left.

Among complications of acute appendicitis most value have appendiceal infiltrates and abscesses.

Appendiceal infiltrate is the conglomerate of organs and tissue not densely accrete round the inflamed vermiform appendix. It develops, certainly, on 3–5th day from the beginning of disease. Acute pain in the stomach calms down thus, the general condition of a patient gets better. Dense, not mobile, painful, with unclear contours, formation is palpated in the right iliac area. There are different sizes of infiltrate, sometimes it occupies all right iliac area. The stomach round infiltrate during palpation is soft and unpainful.

At reverse development of infiltrate (when resorption comes) the general condition of a patient gets better, sleep and appetite recommence, activity grows, the temperature of body and indexes of blood is normalized. Pain in the right iliac area calms down, infiltrate diminishes in size. In this phase of infiltrate physiotherapeutic procedure is appointed, warmth on the iliac area.

In two months after resorption of infiltrate appendectomy is conducted.

At abscessing of infiltrate the condition of a patient gets worse, the symptoms of acute appendicitis become more expressed, the temperature of body, which in most cases gains hectic character, rises, the fever appears. Next to that, pain in the right iliac area increases. Painful formation is felt there. In the blood test high leukocytosis is present with the acutely expressed change of leukocyte formula to the left.

Local abscesses of abdominal cavity, mainly, develops as a result of the atypical placing of appendix or suppuration. More frequent from other there are pelvic abscesses. Thus a patient is disturbed by pain beneath the umbilicus, there are dysuric disorders, diarrhea and tenesmus. The temperature of body rises to 38,0–39,0°C, and rectal — to considerably higher numbers. In the blood test leukocytosis, change of formula of blood is fixed to the left.

During the rectal examination the weakened sphincter of anus is found. The front wall of rectum at first is only painful, and then its overhanging is observed as dense painful infiltrate.

A subdiaphragmatic abscess develops at the high placing of appendix. The pain in the lower parts of thorax and in an upper quarter of abdomen to the right, that increases at deep inhalation except for the signs of intoxication, is characteristic of it. A patient, generally, occupies semisitting position. Swelling in an epigastric area is observed in heavy cases, smoothing and painful intercostal intervals. The abdomen during palpation is soft, although tension in the area of right hypochondrium is possible. Painfulness at pressure on bottom (9–11) ribs is the early and permanent symptom of subdiaphragmatic abscess (the Krukov's symptom).

Roentgenologically the right half of diaphragm can fall behind from left one while breathing, and there is a present reactive exudate in the right pleura cavity. A gas bubble is considered the roentgenologic sign of subdiaphragmatic abscess with the horizontal level of liquid, which is placed under the diaphragm.

Interloop abscesses are not frequent complications of acute appendicitis. As well as all abscesses of abdominal cavity, they pass the period of infiltrate and abscess formation with the recreation of the proper clinic.

The poured festering peritonitis develops as a result of the timely unoperated appendicitis. Diagnostics of this pathology does not cause difficulties.

Pylephlebitis is a complication of both appendicitis and after-operative period of appendectomy.

The reason of this pathology is acute retrocecal appendicitis. At its development the thrombophlebitis process from the veins of appendix, passes to the veins of bowels mesentery, and then on to the portal vein. Patients complain at the expressed general weakness, pain in right hypochondrium, high hectic temperature of body, fever and strong sweating. Patients are adynamic, with expressed subicteritiousness of the scleras. During palpation painfulness is observed in the right half of abdomen and the symptoms of irritation of peritoneum are not acutely expressed.

In case with rapid passing of disease the icterus appears, the liver is increased, kidney-hepatic insufficiency makes progress, and patients die in 7-10 days from the beginning of disease. At gradual subacute development of pathology the liver and spleen is increased in size, and after the septic state of organism ascites arises.

Acute appendicitis is differentiated with the diseases which are accompanied by pain in the abdomen.

Food toxicoinfection. Complaints for pain in the epigastric area of the intermittent character, nausea, vomitings and liquid emptying are the first signs of disease. The state of patients progressively gets worse from the beginning. Next to that, it is succeeded to expose that a patient used meal of poor quality. However, here patients do not have phase passing, which is characteristic of acute appendicitis, and clear localization of pain. Defining the symptoms of irritation of peritoneum is not succeeded, the peristalsis of intestine is, as a rule, increased.

Acute pancreatitis. In anamnesis in patients with this pathology there is a gallstone disease, violation of diet and use of alcohol. Their condition from the beginning of a disease is heavy. Pain is considerably more intensive, than during appendicitis, and is concentrated in the

upper half of abdomen. Vomiting is frequent and does not bring to the recovery of patients.

Perforative peptic and duodenum ulcer. Diagnostic difficulties during this pathology arise up only on occasion. They can be in patients with the covered perforation, when portion of gastric juice flows out in an abdominal cavity and stays too long in the right iliac area, or in case of atypical perforations. Taking it into account, it is needed to remember, that the pain in the perforative ulcer is considerably more intensive in epigastric, instead of in the right iliac area. On the survey roentgenogram of organs of abdominal cavity under the right cupula of diaphragms free gases can be found.

The apoplexy of ovary more frequent is with young women and, as a rule, on 10-14 day after menstruation. Pain appears suddenly and irradiate in the thigh and perineum. At the beginning of disease there can be a collapse. However, the general condition of patients suffers insignificantly. When not enough blood was passed in the abdominal cavity, all signs of pathology of abdominal cavity organs calm down after some time. Signs, which are characteristic of acute anaemia, appear at considerable hemorrhage. Abdomen more frequent is soft and painful down, (positive Kulenkampff's symptom: acute pain during palpation of stomach and absent tension of muscles of the front abdominal wall).

During paracentesis of back fornix the blood which does not convolve is got.

Extra-uterine pregnancy. A necessity to differentiate acute appendicitis with the interrupted extra-uterine pregnancy arises, when during the examination the patient complains at the pain only down in the stomach, more to the right. Taking it into account, it is needed to remember, that at extra-uterine pregnancy a few days before there can be intermittent pain in the lower part of the abdomen, sometimes excretions of "coffee" colour appear from vagina. In anamnesis often there are the present gynaecological diseases, abortions and pathological passing of pregnancy. For the clinical picture of such patient inherent sudden appearance of intensive pain in lower part of the abdomen. Often there is a brief loss of consciousness. During palpation considerable painfulness is localized lower, than at appendicitis, the abdomen is soft, the positive Kulenkampff's symptom is determined. Violations of menstrual cycle testify for pregnancy, characteristic changes are in milk glands, vagina and uterus. During the vaginal examination it is sometimes possible to palpate increased tube of uterus. The temperature of body more frequently is normal. If hemorrhage is small, the changes in the blood test are not present. The convincing proof of the broken extra-uterine pregnancy is the dark colour of blood, taken at puncture of back fornix of vagina.

Acute cholecystitis. The high placing of vermiform appendix in the right half of abdomen during its inflammation can cause the clinic somewhat similar to acute cholecystitis. But unlike appendicitis, in patients with cholecystitis the pain is more intensive, has cramp-like character, is localized in right hypochondrium and irradiate in the right shoulder and shoulder-blade. Also the epigastric phase is absent. The attack of pain can arise after the reception of spicy food and, is accompanied by nausea and frequent vomiting by bile. In anamnesis patients often have information about a gallstone disease. During examination intensive painfulness is observed in right hypochondrium, increased gall-bladder and positive symptoms Murphy's and Ortner's.

Right-side kidney colic. For this disease tormina at the level of kidney and in lumbus is

inherent, hematuria and dysuric signs which can take place at the irritation of ureter by the inflamed appendix. Intensity of pain in kidney colic is one of the basic differences from acute appendicitis. Pain at first appears in lumbus and irradiate downward after passing of ureter in genital organs and front surface of the thigh. In diagnostics urogram survey is important, and if necessary — chromocystoscopy. Absence of function of right kidney to some extent allows to eliminate the diagnosis of acute appendicitis.

As experience of surgeons of the whole world testifies, in acute appendicitis timely operation is the unique effective method of treatment.

Access for appendectomy must provide implementation of operation. McBurney's incision is typical.

When during operation the appendix without the special difficulties can be shown out in a wound, antegrade appendectomy is executed. On clamps its mesentery is cut off and ligated. Near the basis the appendix is ligated and cut. Stump is processed by solution of antiseptic and peritonized by a purse-string suture (Pic. 3.3.2).

If only the basis of appendix is taken in a wound, and an apex is fixed in an abdominal cavity, more rationally retrograde appendectomy is conducted (Pic. 3.3.3). Thus the appendix near basis is cut between two ligatures. Stump is processed by antiseptic and peritonized. According to it the appendix is removed in the direction from basis to the apex. According to indication operation is concluded by draining of abdominal cavity (destructive appendicitis, exudate in an abdominal cavity, capillary hemorrhage from the bed). In recent years the laparoscopy methods of appendectomy are successfully performed.

In patients with appendiceal infiltrate it is necessary to perform conservative-temporizing tactic. Taking it into account, bed rest is appointed, protective diet, cold on the area of infiltrate, antibiotic therapy. According to resorption of infiltrate, in two months, planned appendectomy is executed.

Treatment of appendiceal abscess must be only operative. Opening and drainage of abscess, from retroperitoneal access, is performed. To delete here the appendix is not necessary, and because of danger of bleeding, peritonitis and intestinal fistula — even dangerously.

Intestinal obstruction is a complete or partial violation of passing of maintenance by the intestinal tract.

The principal reasons of intestinal obstruction can be:

- 1) commissures of abdominal cavity after traumas, wounds, previous operations and inflammatory diseases of organs of abdominal cavity and pelvis;
- 2) long mesentery of small intestine or colon, that predetermines considerable mobility of their loops;
- 3) tumours of abdominal cavity and retroperitoneal space.

Such principal reasons can cause violation of passing of intestinal maintenance, disorder of suction from the intestine and loss of plenty of electrolytes both from vomiting and in the intestine cavity as a result of disorders of bloodflow in its wall.

The morphological signs of dynamic intestinal obstruction are: small thickening of wall (at considerable paresis is thinning), friability of tissue (the bowel breaks easily) and presence of liquid maintenance and gases in cavity of bowel. At mechanical obstruction it is always

possible to expose the obstacle: strang, commissures, tumours, jammings of hernia, cicatricial strictures, wrong entered drainages, tampons and others like that. In place of compression strangulation is exposed. The bowel loop higher strangulation is extended, and distally — collapsed. In case of released invagination on small distance two strangulation furrows are observed, and distally from the second ring cylinder expansion of bowel lumen is observed.

Beginning of clinical signs of intestinal obstruction is sudden — in 1–2 hours after taking the meal. The pain in the abdomen has the intermittent character and is met in all forms of mechanical intestinal obstruction. However, some types of strangulated intestinal obstruction (node formation, volvulus of thin and colons) can be accompanied by permanent pain. It is needed to mark that at spike intestinal obstruction, invagination and obturation cramp-like pain can be considered as pathognomic sign of disease. For paralytic intestinal obstruction more frequent is inherent permanent pain which is accompanied by the progressive swelling of abdomen. At spastic obstruction of intestine the pain is mainly acute, the abdomen is not blown away, sometimes pulled in.

Nausea and vomiting are met in 75–80 % patients with the heaviest forms of high level of intestinal obstruction (node formation, volvulus of small intestine, spike obstruction). At obturation obstruction and invagination they are observed not so often.

There is a characteristic thirst which can be considered as an early symptom. Besides, the higher intestinal obstruction, the greater the thirst.

Swelling of abdomen, the delay of emptying and gases are observed in 85–90% patients, mainly, with the high forms of obstruction (volvulus of small intestine, spike intestinal obstruction).

Together with that, for invagination emptying by liquid excrement with the admixtures of mucus and blood are more characteristic.

In patients during palpation the soft abdomen is observed, sometimes — with easy resistance of front abdominal wall, and at percussion — high tympanitis. At auscultation at the beginning of disease increased peristaltic noises are present, then gradual fading of peristalsis is positive (the Mondor's symptom, "noise of beginning, quietness of end").

There are other symptoms pathognomic for intestinal obstruction.

The Vala's symptom is the limited elastic sausage-shaped formation.

The Sklarov's symptom is the noise of intestinal splash.

The Kywul's symptom is the clang above the exaggerated bowel.

The Schlange's symptom is the peristalsis of bowel, that arises after palpation of abdomen.

The Spasokukotsky's symptom is "noise of falling drop".

The Hochenegg's symptom — incompletely closed anus in combination with balloon expansion of ampoule of rectum.

At survey roentgenoscopy or -graphy of the abdominal cavity in the loops of bowels liquids and gas are observed — the Klojber's bowl (Pic. 3.3.4).

Strangulated obstruction. The ischemic component is the characteristic feature of this form of intestinal obstruction, that is investigation of squeezing of mesentery vessels, which

determines the dynamics of pathomorphologic changes and clinical signs of disease, and the basic place among them belongs to the pain syndrome. Consequently, sudden appearance of disease, acuteness of pain syndrome and ischemic disorders in the wall of bowel cause necrosis changes of area of bowel pulling in a process. It is accompanied by the making progress worsening of the patient condition and origin of endotoxiosis.

Obturation intestinal obstruction, unlike strangulated, pass not so quickly. In its clinical picture on the first place there are the symptoms of violation of passage on the intestine (protracted intermittent pain, flatulence), instead of symptoms of bowel destruction and peritonitis.

For high, especially strangulated, intestinal obstruction progressive growth of clinical signs of disease and violation of secretory function of intestine is inherent. Thus the volume of circulatory blood diminishes, the level of haematocritis rises and leukocytosis grows. There are also deep violations of homeostasis (hypoproteinemia, hypokalemia, hyponatremia, hypoxia and others like that). In patients with low intestinal obstruction above-named signs are less expressed, and their growth is related to more protracted passing of disease. Invagination of bowel which can be characterized by the triad of characteristic signs is the special type of intestinal obstruction with the signs of both obturation and strangulation: 1) periodicity of appearance of the intermittent attacks of pain in the abdomen; 2) presence of elastic, insignificantly painful, mobile formation in an abdominal cavity; 3) appearance of blood in the excrement or its tracks (at rectal examination).

The special forms of obturation intestinal obstruction is the obstruction caused by gall-stones. The last are got in the small intestine as a result of bed sore in the walls of gall-bladder and bowel, that adjoins to it. It is needed to mention that intestinal obstruction can be caused by concrement with considerably more small diameter than bowel lumen. The mechanism of such phenomenon is related to irritating action of bilious acids on the bowel wall. The last answers this action by a spasm with the dense wedging of stone in the bowel lumen.

Development of intestinal obstruction caused by gall-stones the attack of colic and clinic of acute cholecystitis precede always. Characteristically, that in the process of development of disease the pain caused by acute cholecystitis calms down, whereupon the new pain characteristic of other pathology — intestinal obstruction appears.

Dynamic intestinal obstruction is divided into paralytic and spastic. Paralytic obstruction often arises after different abdominal operations, inflammatory diseases of organs of abdominal cavity, traumas and poisonings. The reason of spastic intestinal obstruction can be the lead poisonings, low-quality meal, neuroses, hysterias, helminthiasis and others like that. Clinic of dynamic intestinal obstruction is always variable in signs and depends on a reason, that caused it. Disease is characterized by pain in the abdomen, delay of gases and emptying. During palpation the abdomen is blown away, painful, however soft. To diagnose this form of intestinal obstruction is not difficult, especially, if its etiology is known.

Hemostatic intestinal obstruction develops after embolism or thromboses of mesenteric arteries and thromboses of veins, there can be mixed forms. Embolism of mesenteric arteries arises in patients with heart diseases (mitral and aortic failings, heart attack of myocardium,

warty endocarditis) and declared by damaging, mainly, upper mesentery arteries. Beginning of disease, certainly, is acute, with nausea, sometimes — vomiting. At first there is a picture of acute abdominal ischemic syndrome, that is often accompanied by shock (frequent pulse, decline of arterial and pulse pressure, death-damp, cyanosis of mucus membranes and acrocyanosis). Patients become excitative, uneasy, occupy the forced knee-elbow position or lie on the side with bound legs.

During the examination the abdomen keeps symmetry, abdominal wall is soft, the increased peristalsis is heard from the first minutes during 1–2 hours (hypoxic stimulation of peristalsis), which later goes out gradually (“grave quiet”). According to the phenomena of intoxication peritonitis grow quickly. At the beginning of disease the delay of gases and emptying is observed, later there is diarrhea with the admixtures of blood in an excrement. When the last is heavy to set macroscopically, it is needed to explore scourage of intestine.

Intestinal obstruction must be differentiated with the acute diseases of organs of abdominal cavity.

The perforation of gastroduodenal ulcer, as well as intestinal obstruction, passes acutely with inherent to it by sudden intensive pain and tension of muscles of abdomen. However, in patients with this pathology, unlike intestinal obstruction, the abdomen is not exaggerated, and pulled in with “wooden belly” tension of muscles of front abdominal wall. There is also characteristic ulcerous anamnesis. Roentgenologic and by percussion pneumoperitoneum is observed. Certain difficulties in conducting of differential diagnostics of intestinal obstruction can arise at atypical passing and in case of the covered perforations.

Acute pancreatitis almost always passes with the phenomena of dynamic intestinal obstruction and symptoms of intoxication and repeated vomiting, with rapid growth. During the examination in such patients, unlike intestinal obstruction, rigidity of abdominal wall and painfulness is observed in the projections of pancreas and positive Korte's symptom and Mayo-Robson's. The examination of diastase of urine and amylase of blood have important value in establishment of diagnosis.

Acute cholecystitis. Unlike intestinal obstruction, patients with this pathology complain for pain in right hypochondrium, that irradiate in the right shoulder-blade, shoulder and right subclavian area. Difficulties can arise, when the symptoms of dynamic intestinal obstruction appear on the basis of peritonitis.

The clinic of kidney colic in the signs and character of passing are similar to intestinal obstruction, however, attacks of pain in the lumbar area with characteristic irradiation in genital parts, the thigh and dysuric disorders help to set the correct diagnosis. Certain difficulties in conducting of differential diagnostics also can arise in difficult patients, at frequent vomiting which sometimes can be observed in patients with kidney colic.

During the first 1,5–2 hours after hospitalization of patient complex conservative therapy which has the differential-diagnostic value and can be preoperative preparation is conducted.

It is directed on warning of the complications related to pain shock, correction of homeostasis and, simultaneously, is the attempt of liquidation of intestinal obstruction by unoperative methods.

1. The measures directed for the fight against abdominal pain shock include conducting of neuroleptanalgesia, procaine paranephric block and introduction of spasmolytics. Patients with the expressed pain syndrome and spastic intestinal obstruction positive effect can be attained by epidural anaesthesia also.

2. Liquidation of hypovolemia with correction of electrolyte, carbohydrate and albuminous exchanges is achieved by introduction of salt blood substitutes, 5–10 % solution of glucose, gelatinol, albumen and plasma of blood. There are a few methods suitable for use in the urgent surgery of calculation of amount of liquid necessary for liquidation of hypovolemia. Most simple and accessible is a calculation by the values of hematocrit. If to consider 40 % for the high bound of hematocrit norm, on each 5 % above this size it is needed to pour 1000 ml of liquids.

3. Correction of hemodynamic indexes, microcirculation and disintoxication therapy is achieved by intravenous infusion of Reopolyhukine and Neohemodes.

4. Decompression of intestine tract is achieved by conducting of nasogastric drainage and washing of stomach, and also conducting of siphon enema. It is needed to underline that technically the correct conducting of siphon enema has the important value for the attempt of liquidation of intestinal obstruction by conservative facilities, therefore this manipulation must be conducted in presence of a doctor. For such enema the special device is used with the rectal tip, by a PVC pipe by a diameter of 1,5–2,0 cm and watering-can of very thin material. A liquid into the colon is brought to appearance of the pain feeling, then drop the watering-can below the level of patient who lies. The passage of gases and excrement is looked after. As a rule, this manipulation is to repeat repeatedly with the use of plenty of warm water (to 15–20 and more litres).

Liquidating of the intestinal obstruction by such conservative facilities is succeeded in 50–60 % patients with mechanical intestinal obstruction.

Patients with dynamic paralytic intestinal obstruction are expedient to stimulation of peristalsis of intestine to be conducted, besides, necessarily after infusion therapy and correction of hypovolemia. A lot of kinds of stimulation of intestine peristalsis are offered. Most common of them are:

1) hypodermic introduction of 1,0 ml of 0,05 % solution of proserin; 2) through 10 min — 60 ml intravenously stream of 10 % solution of chlorous sodium; 3) hypertensive enema.

Surgical treatment of intestinal obstruction must include such important moments:

1. According to middle laparotomy executed the novocaine blockade of mesentery of small and large intestine and operative exploration of abdominal cavity organs during which the reason of intestinal obstruction and expose viability of intestine is set.

The revision at small intestine obstruction begins from the Treitz' ligament to iliocecal corner. At large intestine obstruction the hepatic, splenic and rectosigmoid parts are observed intently. Absence of pathological processes after revision needs the examination of places of cavity and jamming of internal hernia: internal inguinal and femoral rings, obturator openings, pockets of the Treitz' ligament, Winslow's opening, diaphragm and periesophageal opening.

2. Liquidation of reasons of obstruction (scission of connection, that squeezes a bowel,

violence of volvulus and node formation of loops, desinvagination, deleting of obturative tumours and others like that).

It is needed to mark that the unique method of liquidation of acute intestinal obstruction does not exist. At the lack of viability of bowel the resection of nonviable area is executed with 30–40 cm of afferent and 15–20 cm of efferent part with imposition of “side-to-side” anastomosis (Pic. 3.3.5) or “end-to-end” (Pic. 3.3.6).

3. Intubation. Decompression of intestine foresees conducting in the small intestine of elastic probe by thickness of 8–9 mm and length of 3–3,5 m with the plural openings by a diameter 2–2,5 mm along all probe, except for part, that will be in the oesophagus, pharynx and outwardly. A few methods of conducting of probe are offered in a bowel (nasogastric, through gastrostomy, ceco- or appendicostoma). Taking it into account, such procedure needs to be executed individually and according to indications.

Each of them has the advantages and failings. In connection with the threat of origin of pneumonia, entering an intubation probe to the patients of old ages is better by means of gastrostomy. Most surgeons avoid the method of introduction of probe through ceco- or appendicostoma because of technical difficulties of passing in a small intestine through a Bauhin's valve. Today the most wide clinical application has intubation of intestine extracted by the nasogastric method with the use of other thick probe as explorer of the first (by L.J. Kovalchuk, 1981). Such method not only simplifies procedure of intubation but also facilitates penetration through the piloric sphincter and duodenojejunal bend, and also warns passing of intestinal maintenance in a mouth cavity and trachea. Thus probe is tried to be conducted in the small intestine as possible farther and deleted the next day after appearance of peristalsis and passage of gases, however not later than on 7th days, because more protracted sign of probe carries the real threat of formation of bedsores in the wall of bowel.

4. Sanation and draining of abdominal cavity is executed by the generally accepted methods of washing of antiseptic. Draining of the abdominal cavity it is needed from four places: in both iliac areas and both hypochondrium, better by the coupled synthetic drainpipes.

Crohn's disease is an unspecific inflammatory process of submucosal membrane of gastrointestinal tract with propensity to the segmental lesions and recurrent passing. The local signs of disease exist in different areas of digestive tract organs, however, most frequent and most intensive they are in the distal segment of small intestine, therefore it was named terminal ileitis.

The reason of origin of the Crohn's disease for today is not finally found out. An infection and allergy are infringement factors. Together with that, granuloma, which is exposed at histological examination with present in its lymphocytic and protoplasmatic infiltrations, grounds to consider that the defined value in the origin of the Crohn's disease have immune factors. Thus inflammation begins in the submucosal membrane, and afterwards engulfs all bowel walls. The mucus membrane acquires the crimson colouring, there are deep cracks and ulcers. Combination of the damaged areas of mucus membrane with healthy creates a picture similar to the roadway. In future granuloma appears, an inflammatory process goes out outside the wall of bowel and gets to the contiguous organs (large and small intestines, urinary bladder, abdominal wall). In the eventual result there are infiltrate, abscesses and fistula. Finally, it is

needed to mark that the people of young ages mainly are ill by terminal ileitis.

The morphological changes are concentrated, mainly, in the terminal part of iliac bowel, anal segment of rectum and appendix. Internal surface is hilly, thickened, swelling, deep ulcers are intermittent with the unchanged areas of mucus. The serous tunic is covered by plural, similar on tuberculosis, knots. Mesentery is sclerosed, regional lymphatic knots are hyperplastic, of whiter-rose color. By the most characteristic microscopic sign of Crohn's disease is presence of unspecific sarcoid granuloma. Hyperplasia of lymphoid elements of submucosal membrane and formation of fissured ulcers is observed also.

The Crohn's disease begins from the insignificant signs as a general weakness, increase of temperature of body, intermittent pain, that arises after the reception of meal, diarrhea without some visible features or with the admixture of blood. As this process strikes the terminal segment of small intestine, pain is concentrated in the right iliac area. Together with that, at localization of pathological focus in a colon with an anal segment pain is concentrated by its passing to the anal opening. A granuloma process takes place in the area of oesophagus, abdomen or duodenum, pain can arise up in the area of lesions. With progress of disease on endoscopy examination (proctosigmoidoscopy, fiberoptic colonoscopy, fiberoptic gastroscopy) hyperemia and deep cracks of mucus membrane, ulcers, symptom of "roadway" and stenosis are observed. At roentgenoscopy survey of organs of abdominal cavity in patients with the perforation it is possible to expose pneumoperitoneum, and at contrasting sciagraphy — stenosis of initial part of stomach, presence of ulcers or granuloma in the oesophagus. The examination of the passage on the small intestine enables to eliminate or confirm stenosis (Pic. 3.3.7). Irrigoscopy determines the defect of filling.

At acute passing of terminal ileitis, the pain appears acute in the right iliac area, sometimes intermittent, accompanied by nausea, vomitings, emptying by a liquid excrement with the admixture of blood or delay of emptying. During the examination of patient the abdomen can be exaggerated, tension of muscles and positive symptoms of irritation of peritoneum, high temperature is observed. In the general analysis of blood leukocytosis is present with the change of leukocyte formula to the left. In such difficult situation often only laparotomy helps to specify the diagnosis. The swollen segment of iliac bowel is thus observed with increased mesentery lymphatic knots. The changed area of bowel can perforate in the free abdominal cavity or penetrate in the contiguous loops of large or small intestine. It causes forming of inflammatory infiltrate, and in future — abscess formation. The unoperated abscesses are always inclined to the independent opening in surrounding organs with subsequent formation of fistula ducts.

The disease with the lesions of other parts of small and large intestine passes acutely (granulomatous enteritis, colitis). By palpation in these patients painful infiltrate is exposed, which by the character remind the clinic of invagination. Only the meticulous examination and present data analysis enable to set correct diagnosis. At granulomatous proctitis the plural cracks of mucus membrane without the signs of spasm of sphincter appear often, on the basis of which afterwards there are ulcers, that badly granulate. The same changes can develop on skin round the anal opening.

The chronic forms of disease often pass with insignificant symptoms. From the

beginning of disease to establishment of diagnosis sometimes 1–2 years and more pass. Such patients periodically complain for pain, diarrhea, weight loss, increase of body temperature, nausea, vomitings and bleeding from a rectum.

Objectively in the abdominal cavity painful infiltrate is determined, and at laboratory examination — anaemia and hypoproteinemia.

Complications of the Crohn's disease can be divided into local and general. Among local, formations of fistula which arise on the front abdominal wall between the damaged bowel and surrounding organs are most characteristic (ileoileal fistula, entero-entero, enterovesical fistula). Sometimes fistulas are opened in the area of scars after the operations on the lateral wall of abdomen or in the area of anus. Next to that, stenosis inflammatory infiltrate of bowel can be transformed in acute or chronic intestinal obstruction. Some patients have the obvious threat of perforation of the changed wall of bowel or intestinal bleeding. The protracted passing of disease can be also complicated by malignization. The aphthous ulcers of tongue, node erythema, arthritises and chronic lesions of liver are general complications.

The Crohn's disease must be differentiated with the unspecific ulcerous colitis and cancer of colon.

An unspecific ulcerous colitis mainly initially strikes the mucus membrane of all colon. The disease is accompanied by the excreta with the excrement of plenty of blood and mucus. For Crohn's disease languid passing of disease is characteristic. Acute passing of disease is met considerably rarer, than chronic. The modern methods of endoscopic examination with the biopsy of mucus membrane, which helps to specify diagnosis, are helpful in differential diagnostics.

The cancer of colon is mostly accompanied by formation of deep ulcers and infiltrate. However, for the cancer process slowly progressive passing without the periods of remission is more inherent, thus the disease more frequently ends with the phenomena of intestinal obstruction. At roentgenologic examination on the background the relatively unchanged colon the lonely defect of filling is observed, and during colonoscopy — thrusting out in the lumen of bowel with an erosive surface or signs of disintegration. Histological examination of biopsy material enables to expose cancer cells.

Conservative treatment. The diet of patient, generally, must be ordinary, except for products with bad intestinal uptake. The medicine of the first row is 5-ASK (aminosalicylic acid, sulfasalazone and glucocorticoid). The medicines of the second row are: 6-mercaptopurine, azatiopurine and metronidazole. At diarrhea diphenoxilate is used — 5 ml peroral three times per days, loperamide — 2 mg peroral 3–4 times per days, smecta — 1 pack 3 times per days. At the expressed anaemia, to the considerable loss of weight, system complications, relapse of disease after operation prednisolone is applied — for 40–60 mg peroral every day during 1–2 weeks. After that its day's dose is diminished to 10–20 mg during 4–6 weeks and, in the end, stopped. For patients which are irresponsive to steroid, asatioprine is appointed (2 мг/кг) peroral. Metronidazole in a dose of 400 mg twice a days is used in the case of granulomatous disease of perineum.

The presence of external and internal fistula, stenosis of bowel, perforation and recurrent bleeding is an indication to operative treatment. The method of choice of operation is the

segmental resection of the pathologically changed bowel in the distance of 30–35 cm of proximal and distal from the damaged area. The regional lymph nodes is also deleted. In case of the perforation of bowel with poured peritonitis, it is recommend not to perform primary anastomosis because of possible insolvency of stitches after the resection. In this connection, afferent and efferent loops exteriorizes on the wall of abdomen as two-channel stoma (Pic. 3.3.8). The passage by an intestine (liquidation of stoma) is restored in 2–4 months after liquidation of the peritonitis signs.

An unspecific ulcerous colitis is a diffuse inflammatory process that is accompanied by the ulcerous-necrosis changes in the mucus membrane of colon and rectums.

Etiology of unspecific ulcerous colitis to this time is not finally found out. This disease is suffered by people in the age from 20 to 40 years. An infectious factor in development of disease for today is not confirmed. However, as the exception of meal of food allergens (milk, eggs) results in the improvement of passing of disease, it is possible to consider that allergy assists to development of inflammatory process. Important significance in genesis of this pathology is also attached to immunological violations. In the blood of patient sensibilizing on the antigen of mucus membrane of colon specifically lymphocytes and immune complexes are found. The antigen-antibody reaction can cause colitis. In most patients with the chronic recurrent unspecific ulcerous colitis a stress situation causes the process of acutening. In future, obviously, there are violations of microcirculation and cellular structures, and also the transport system of cells membranes suffers, that carries potassium and sodium ions. Taking it into account, the timely exposure of disease in which the process is localized and has a reverse tendency, can result in the positive therapeutic effect.

In patients with an unspecific ulcerous colitis the relatively isolated damages of rectum and sigmoid colon, sigmoid colon and transverse colon, so total colitis are met. The necrosis component prevails as the acute form. The wall of bowel in such cases is swollen, hyperemic, with plural erosions and ulcers of irregular form. Its infiltration by lymphocytes, plasmocytes and eosinophils with characteristic formation of granulation, crypt and abscesses are microscopically observed. In patients with a chronic process prevail, mainly, reparative-sclerotic processes. A bowel is deformed, dense, segmentally narrowed. As a result of the disfigured regeneration plural granulomatous and adenomatous pseudopolypuses appear.

Pain in the abdomen and diarrhea is one of basic signs of unspecific ulcerous colitis with emptying from 3 to 20 and more times per days. Thus during defecation the mixture of liquid excrement, mucus and blood are observed. As far as progress of disease the pain has the intermittent character and is localized by the passing of colon. By palpation it is spastic and painful. Frequent diarrhea is brought to dehydration, loss of electrolytes, albumen and anaemia. Patients are weak, there are the strongly expressed signs of intoxication, the temperature of body rises to 40 oC and the psyche is repressed. Characteristic also are tachycardia, decline of arterial pressure, avitaminosises and edemata. Hypochromic anaemia is exposed in the general analysis of blood, leukocytosis, change of leukocyte formula to the left and increased ESR. In plasma of blood the decline of maintenance of potassium and sodium ions and level of general protein, especially albumen are marked. In future there are the progressive degenerative changes of parenchymatous organs.

At endoscopic examination (proctoscopy, fiberoptic colonoscopy) hyperemia of mucus membrane, swollen, contact bleeding, plural erosions, ulcers, festering and necrosis stratifications, are observed. At heavy passing of disease fiberoptic colonoscopy or irrigoscopy always has the danger of perforation or acute bowel dilatation, therefore more rationally it is to conduct it in the period of calming down of inflammatory process. During roentgenoscopy survey of organs of abdominal cavity in case of disease, complicated of acute toxic dilatation, the extended (from 10 to 20 cm and more) pneumatized bowel is exposed. During the perforation of bowel signs of pneumoperitoneum are present (air under the right cupula of diaphragm). Contrasting irrigoscopy examination in such patients enables to establish the presence of “water-pipe” symptom. Thrusting out of tailings of well-kept mucus membrane on the background of plural ulcers and cicatricial changes of bowel walls at pseudopolyposis creates roentgenologic reality of “shot target through” symptom (Pic. 3.3.9). Often in case of transformation of ulcerous colitis in the cancer on roentgenograms, stenosis cavity of bowel or defect of filling is observed (Pic. 3.3.10).

Acute, especially fulminant form of the unspecific ulcerous colitis passes the heaviest, so the prognosis is always doubtful. Taking it into account, death can come in the first days of disease. Thus an inflammatory process will strike all colon. During 1–2 days the heavy clinical picture is observed with frequent diarrhea with mucus, blood and pus, vomiting, dehydration and loss of weight of body. Next to that, deep intoxication, darkened consciousness, and the temperature of body rises to 39–40 oC is present. The expressed anaemia, tachycardia and hypovolemia are observed. The loss of albumens causes the decline of oncotic pressure and causes dehydration. The disbalance of electrolytes grows with progress of disease, microcirculation gets worse and day's diuresis goes down. In most cases this form of disease requires urgentoperative treatment (absolute indications).

A chronic recurrent unspecific ulcerous colitis is characterized by the periods of acuteening and remission. Thus in patients with the total lesions of colon in the period of acuteening the heavy degree of disease is observed, and in the period of remission— middle or even easy degree of disease, thus, such “calming down” can last 6 months and more.

A chronic continuous unspecific ulcerous colitis at the total lesions of intestine in most patients passes as middle heavy degree, and in the period of worsening the disease takes heavy shape. The easy form is met, mainly, at presence of inflammatory process in the rectum and sigmoid colon, considerably rarer it is at the lesions of left half of colon and quite rarely — at the total lesions in the period of calming down of the process. Conventionally, the unspecific ulcerous colitis begins from the rectum and engulfs all parts of colon. Thus emptying are 2–3 times per days with the admixtures of mucus, sometimes blood. Thus, diarrhea can be intermittent with constipation. The temperature of body remains within the limits of norm. From the side of global and biochemical analysis of blood noticeable changes do not arise. Weight of body does not diminish. At endoscopic examination hyperemia of mucus membrane, contact bleeding, expressed vascular picture, erosions, point hemorrhages and superficial ulcers are observed. It is needed also to mark that in this situation the presence of erosions must be equated with an ulcerous process.

The middle heavy form of disease of the unspecific ulcerous colitis can be met in

patients with the ulcerous colitis and proctosigmoiditis in the period of process acutening. Thus there are the subjective feelings with considerable expression of tenesmus and heartburns.

The chronic forms of unspecific ulcerous colitis both at total and at the left-side lesions of colon, pass at the level of middle heavy degree. Frequency of emptying reaches to 5–10 times with mucus, blood and pain. Low grade fever, general weakness, nausea and loss of appetite appear, and weight of body diminishes on 5–8 kg. Moderate anaemia is exposed in the general analysis of blood, leukocytosis, increased ESR. Among the biochemical indexes of blood hypoproteinemia and hypokalemia are marked. At endoscopic examination of colon there is a considerable hyperemia and edema of mucus membrane, plural erosions, contact bleeding and superficial ulcers.

The heavy form of unspecific ulcerous colitis is at the total lesions, especially with acute, and also chronic recurrent passing of disease. The temperature of body in such patient rises to 39–40 °C, there is diarrhea (more than 10 times per days) with mucus, blood and pus, vomitings, heavy intoxication grows, weight loss on 25–30 kg, acutely expressed anaemia, leukocytosis with the change of leukocyte formula to the left, considerable changes of albuminous and electrolyte exchanges. At endoscopic examination of colon the blood is exposed in its cavity, slid, pus, fibrin incrustation, often pseudopolypuses and almost complete absence of mucus membrane. Roentgenologically some signs of complications of unspecific ulcerous colitis are confirmed.

The complications are divided into local and general. Local complications are: profuse intestinal bleeding, perforation, acute toxic dilatation, stenosis and malignization. To general the following are included: damage of liver (hepatitis, cirrhosis), stomatitis, ulcer of lower extremities, lesions of joints, eyes and skin.

Acute dysentery passes with bloody diarrheas, increased temperature of body, pain in the abdomen. Bacteriological examination of excrement enables to expose dysenteric bacillus and specify diagnosis.

Crohn's disease (granulomatous colitis) is this local process, that begins from the submucosal layer of bowel and distributes outside of walls with subsequent formation of infiltrate, abscesses and fistula. Exposure of granulomas, and during microscopic examination — accumulation of lymphocytes, neutrophils, protoplasmatic cells and the Pyrohov-Lunghans' cells are confirmed diagnosis.

The cancer of colon, in particular its enterocolitis and toxicoanemic forms, also often can simulate an unspecific ulcerous colitis. Irrigoscopy, fiberoptic colonoscopy with biopsy and subsequent histological examination almost always help to diagnose cancer process.

Treatment of unspecific ulcerous colitis, certainly, begins with application of conservative facilities. Thus patients with easy and middle heavy forms must be under protracted conservative treatment.

The leading role is taken to the parenteral feed of patients with heavy common exhaustion (hydrolyzate of casein, aminopeptid, amynosol, vamin, alvesin, moriamin, intralipid, lipofundin, glucose and others like that). Electrolytes (chloride of sodium, sulfate of magnesium, chloride of potassium, panangin) and vitamins are entered (B6, B12, C, K, PP and others). Intensity and methods of conservative therapy always must depend on the phase of

disease:

a) moderately expressed passing of disease or proctitis — corticosteroid enema and sulfasalazone peroral;

б) at heavy passing is parenteral introduction of liquids, nutritives, blood transfusion, system use of corticosteroids, surgical treatment;

в) at chronic passing is corticosteroids peroral, asatioprine, surgical treatment;

г) at remission is preparations of 5-aminosalicylic acid peroral, examination for the exception of cancer of colon.

The heavy form of passing of disease is absent of effect from the conducted conservative treatment during two weeks and progress of process testifies to the necessity of surgical treatment.

The conservative treatment must include antibacterial agent, antidiarrheal preparations, steroid hormone.

A diet is considered an important factor in treatment of such patient (diet № 4). Thus it is recommended to take a meal to 6 times per days by small portions, withdrawing milk, fruit, vegetables, wheat and rye bread from it. It is possible to appoint unfat meat and fish. Parenteral introduction of vitamins B, C, A, folic acid are helpful.

The basic antiinflammatory facilities are: sulfasalazopreparations (sulfasalazone, salazopirine), salicylazosulfanilamide (salazosulfa-pyridine, salazodimetoxine) and corticosteroids. Practice showed that sulfasalazone was one of the best antirecurrent facilities.

In patients with easy and middle heavy forms (distal or left-side lesions of colon) sulfasalazone is applied in a dose about 5 g per days, and salazopiridazine and salazodimetoxine — about 2 g during that time. The course of treatment must proceed 1–2 months. For local steroid therapy prednisolone is used as powder in a dose 60–80 mg or hydrocortisone — to 125 mg. It is dissolved in 100 ml physiologic saline and entered rectal dropwise one time per days during 3–4 weeks.

At erosive proctitis and proctosigmoiditis 5 % (100,0 ml) solution of kolargole or extract of camomile in microclyster is applied.

At the heavy forms of ulcerous colitis with fulminant passing and frequent vomiting the treatment is needed to begin with intravenous introduction of 350–380 mg hydrocortisone per day. Thus procedures must proceed to appearance of positive clinical effect and realization of possibility of transition on enteral treatment. Such period lasts on the average of 6–7 days. In future it is recommended to adopt prednisolone peroral.

Sulfasalazopreparations is used in the same dose, as at the middle heavy form of flow of disease. As in patients at this form of disease water-electrolyte and albuminous exchanges are considerably violated, there are the expressed intoxication and anaemia, it is expedient to conduct adequate therapy (intravenously - NaCl solution, glucose, chlorous potassium, albumen, hemodes, protein, whole blood), and also hemodialysis and oxygenotherapy is used.

An absolute indication for surgical treatment is the presence of such complications of unspecific ulcerous colitis as: perforation of wall of bowel, acute toxic dilatation, stenosis, profuse bleeding and malignization. By the choice of method of operation at such pathology it is needed to count colproctectomy with exteriorization of ileostomy.

However, during the perforation of colon or toxic dilatation the operative treatment can be limited to colectomy because proctectomy will be conducted as the next stage.

For patients with total ulcerous colitis with chronic heavy passing and without the tendency to the visible improvement expedient radical operative treatment — colproctectomy with exteriorization of ileostomy. At such tactic postoperative lethality is diminished in 5-6 times, comparative with palliative operations which were conducted earlier.

In Western Europe and North America colorectal cancer (CRC) is one of most widespread malignant neoplasm. Morbidity in these regions exceeds 20,0 on a 100000 of population. In Japan, South America, Africa and countries of Asia this tumour is met rarer (6,0:100000). There is an annual increase of frequency of cancer of colon approximately by 3 % in the developed countries. Morbidity of population of Ukraine by malignant neoplasm in 1995 was 16,3:100000.

Approximately 85 % of patients for CRC are of over 50 years old, with age frequency of cancer is increased.

The nutrition by fat and albuminous food promotes the elimination to the intestine of bile. Under the act of bacterial flora there is transformation of primary bilious acids to the secondary, which has the carcinogenic and mutagenic activity. A meal with vitamins A, C and that which contains plenty of vegetable cellulose has a braking carcinogenic influence.

The factors of risk which predetermine the origin of cancer of bowel are:

- 1) diffuse (family) poliposis, which is considered obligate precancer;
- 2) plural and single adenomatous polypuses;
- 3) chronic unspecific ulcerous colitis (anamnesis more than 10 years);
- 4) Crohn's disease (granulomatous colitis).

Localization. A tumour is mostly localised in sigmoid (35–40 % cases) and blind (20–25 % cases) bowels.

Macroscopic forms. Exophytic tumours grow in the lumen of bowel as a polypus or knot and at disintegration have the appearance of ulcer with a dense bottom that is swelling by edges which come forward above the surface of the damaged mucus (saucer-shaped cancer). The endophytic (infiltrate) cancer grows in walls of bowel. The tumour spreads on the perimeter of bowel and engulfs it circular, causing narrowing of its lumen. In the right half of colon exophytic tumours grow, as a rule, in left — endophytic tumours.

Histological structure. Cancer of colon in 95 % cases has the structure of adenocarcinoma. Metastasis takes place by lymphatic and hematogenic ways in regional retroperitoneal lymphatic knots, liver, lungs.

The symptoms of cancer of colon are so numerous and various, that many authors group them in such clinical forms: toxico-anemic, dyspeptic, enterocolitic, obturation, pseudoinflammatory and tumular.

A toxico-anemic form shows up by indisposition, weakness, rapid fatigability, increase of temperature, progressive anaemia. Characteristic for the cancer of right half of colon.

The enterocolitic form is characterized by symptom of complex intestinal disorders: diarrhea, constipation, swelling, grumbling, pain.

The dyspeptic form is characterized by functional disorders of gastrointestinal tract.

An obturation form shows up by intestinal obstruction.

A pseudoinflammatory form is characterized by the symptoms of inflammatory process in the abdominal cavity.

A tumour form passes asymptomatic. A tumour is exposed by chance by a patient or doctor.

Obturation and enterocolitic forms more characteristic for the cancer of left half, other ones — of the right. For the cancer of right half of colon tendency to gradual progress is characteristic, and the tumours of left half often show up suddenly by intestinal obstruction.

Intestinal obstruction, germination in neighbouring organs and tissue, perforation, bleeding are considered as the most frequent complications of colon cancer.

Depending on the clinical signs of colon cancer, a differential diagnosis is to be conducted with appendiceal infiltrate, by different chronic specific and unspecific diseases of colon, and also other organs of abdominal cavity and retroperitoneal space (gall-bladder, pancreas, kidneys, genital organs and others like that), with the tumours of other organs of abdominal cavity and retroperitoneal space.

Radical treatment. Operative treatment is the unique method of radical treatment of colon cancer. The choice of method of operation depends on localization of tumour (Pic. 3.3.11). At cancer of right half of colon right hemicolectomy, (deleting of all right half of colon, including right third of transversal colon and distal segment of iliac bowel by length 20–25 cm) is conducted. In patients with tumours of left half of colon left hemicolectomy (segment from middle or from left third of transversal colon to overhead part of sigmoid is resected) is executed. At cancer of transversal colon, middle and distal parts of sigmoid bowels the resection of the damaged area is conducted, stepping back 5-6 cm from the edge of tumour. In patients who are hospitalized in an urgent order with the signs of intestinal obstruction, perforations and peritonitis, after intensive preoperative preparation the Hartmann operation is executed (at tumours of left half of colon). It is the resection of the damaged area of intestine and exteriorization of proximal segment on a front abdominal wall as colostomy. A distal end is sutured and is remained in the abdominal cavity. Through half-year there is a possibility of reconstructive operation. To the patients with the damage of right half of colon operation in a radical volume can be executed (right hemicolectomy).

During treatment with palliative purpose (at presence of solitary metastases) operations in a radical volume with removing of metastatic knot (in a liver) or subsequent chemotherapy (by 5- fluorouracil) can be used.

In recent years for the improvement of remote results treatment is applied by the adjuvant chemotherapy and intensive preoperative gamut-therapy.

The remote results of treatment of patients on the initial stages of CRC are fully satisfactory. At I stages the five-year survival is 85-100 %, at II — 65–70 %, at III — 25–30 %. On the whole at the I–III stages the five-year survival is 45 %.

Persons who refused from operative treatment perish in a short time. The combined treatment improves remote results approximately on 15–20%.

The basis of disease of pancreas is degenerative-inflammatory processes which are considered to be acute pancreatitis, the so called autolysis tissue by its own enzymes. In the

structure of acute pathology of organs of abdominal cavity this disease takes the third place after acute appendicitis and cholecystitis. Women suffer from acute pancreatitis 3–3,5 times more frequently than men.

Acute pancreatitis is a polyetiology disease. Its secondary forms, which arise on the background of pathologies of bile-excreting system and duodenum are closely associated with anatomic and functionally with pancreas, and are met in clinical practice.

Among the “starting” factors of origin of cholelithiasis disease (biliary pancreatitis) abuse by an alcohol and food overloads (fat and irritating products), traumas of pancreas, operating-room in particular, and also separate infectious diseases (parotitis, mononucleosis) are most frequent, especially infection of bilious ways. However, in 10–20 % of patients the reason of acute pancreatitis remains unknown (cryptogenic form).

In the basis of such damages of pancreas and enzymic toxemia lies mainly activating of pancreatic, and then the tissue enzymes (trypsin, lipase, amylase). Often the combination of the broken outflow of pancreatic secret and promoted secretion takes place, which provokes intraductal hypertension.

Among explanations of primary mechanisms of activating of pancreatic enzymes the most value belongs to: a) theory of “general duct” with reflux of bile in the ducts of pancreas; b) blockade of outflow of pancreatic juice with development of intraductal hypertension and penetration of secret in interstitial tissue; in) violation of blood flow of pancreas (vasculitis, thrombophlebitis and embolisms, cardiac insufficiency and others like that); g) toxic and allergic damages of gland. The role of alcohol in such situations can be dual: stimulation of secretion of pancreas and direct damaging action on its tissue.

The process of acute inflammation of pancreas consistently passes the stages of edema, pancreatonecrosis and festering pancreatitis. In the stage of edema there is pancreas of hyperemic, increased in volume, with the shallow hearths of necrosis or, as it is in swingeing majority of cases, without them.

Pancreatonecrosis can pass with fatty or hemorrhagic character. In the first case, as a rule, pancreas is increased, dense, cut whity-yellow hearths are selected to necrosis. Increase of crimson-black pancreas with darkly-brown infiltrate on a cut is characteristic for hemorrhagic pancreatonecrosis.

Dystrophy of parenchyma is exposed microscopically, up to necrosis, hemorrhages, thromboses of vessels and signs of inflammatory infiltration.

The disease begins suddenly, after the surplus reception of rich spicy food and use of alcohol. Pain, vomiting and phenomena of dynamic intestinal obstruction are considered the most characteristic signs of acute pancreatitis.

A stomach-ache is permanent and so strong, that can result in shock, localized in an epigastric area and left hypochondrium. Some patients feel pain in right hypochondrium with irradiation in the back, loin or breastbone.

In a short period of time after appearance of pain there is a repeated strong vomiting, that does not facilitate the state of patient.

In general vomiting is considered a frequent and characteristic symptom. It is repeated or continuous and never brings facilitation. Vomit masses contain bile, as admixture, and at the

difficult form of acute pancreatitis remind “coffee-grounds”.

Nausea, hiccup, belch and dryness in a mouth are attributed as less characteristic symptoms of this pathology.

During the examination the skin is pale, often subicterus. Some patients have cyanosis with a “marble picture” as a result of violation of microcirculation. Later the component of respiratory insufficiency can join it. At progressive general condition the patient quickly gets worse to passing of acute pancreatitis, intoxication grows. The skin takes shelter with sticky sweat.

The temperature of body of patients at the beginning of disease can be normal. It rises at resorption of products of autolysis tissue and development of inflammatory process in biliary ways.

The pulse in most cases is at first slow, then becomes frequent, notably passing ahead the increase of temperature of body.

Arterial pressure goes down.

The tongue in the first hour of disease is moist, assessed by white and grey raid. At vomiting by bile the raid has yellow or greenish tint.

The abdominal is blown away, peristaltic noises are loosened. The signs of paresis of stomach and intestine demonstrate early. They need to be included in the pathological process of mesentery root of bowel. At palpation painfulness in an epigastric area and in right, and sometimes and in left, hypochondrium is marked. However, in spite of great pain in stomach, it remains soft for a long time. A little later there is moderate tension or resistance of muscles of front abdominal wall.

Poor local symptoms during heavy intoxication are characteristic for the early period of acute pancreatitis. Later there are symptoms of irritation of peritoneum, and at percussion dulling is marked in lateral parts of abdominal as a result of accumulation of liquid, and also the sign of aseptic phlegmon of retroperitoneal cellulose as slurred or edema of lumbar area is seen. For diagnostics of acute pancreatitis there is the row of characteristic symptoms which have different clinical value.

The Mondor's symptom is violet spots on face and trunk.

The Lagermph's symptom is acute cyanosis of person.

The Halsted's Symptom is cyanosis of abdominal skin.

The Gray's symptom is cyanosis of lateral walls of abdomen.

The Kullen's symptom is the yellow colouring of skin near a belly-button.

The Korte's symptom is painful resistance as a lumbar bar in a epigastric area on 6–7 cm higher belly-button.

The Voskresynskyy's symptom is absence of pulsation of abdominal aorta in an epigastric area.

The Mayo-Robson's symptom is feeling of pain at pressure by fingers in the left costal-vertebral corner.

The Rozdolskyy's symptom — painfulness at percussion above pancreas.

The Blumberg's symptom — in patients with acute pancreatitis more frequently is low-grade. Such feature of this sign of irritation of peritoneum needs to be explained by

character of localization of pathological process, mainly in retroperitoneal spacious.

In clinical passing of pancreatonecrosis it is possible to select three periods (V.S. Saveljev, 1978).

The I period (hemodynamic violations and pancreatogenic shock) lasts during 2–3 days. Violation of central hemodynamics, diminishment of volume of circulatory blood and disorders of microcirculation, which at first arise as a result of angiospasm, are considered the most characteristic signs, and later as a result of joining of the intravascular rolling up and laying of elements of blood.

The II period (insufficiency of parenchymatous organs) lasts from 3rd to the 7th day of disease. Violation of functions of basic organs and systems, sign of cardio-vascular, hepatic and kidney insufficiency and growth of violations of breathing are thus observed. In this period there is possible damaging of the central nervous system, which is erected mainly to disorders of psyche, appearances of delirium and commas which in the eventual result are the main reasons of patients' death.

The III period (postnecrosis dystrophic and festering complications) comes in 1–2 weeks after the beginning of disease. During it, on the background of progress of necrosis processes in pancreas, the regenerative changes develop, there are parapancreatic infiltrate and cysts, cystic fibrosis of pancreas. Aseptic retroperitoneal phlegmon which strengthens intoxication can also develop. There is festering pancreatitis at joining of infection. During this period such complications, as erosive bleeding, internal or external fistula, retroperitoneal phlegmon, can develop in patients.

From laboratory information leucocytosis which at the necrosis and hemorrhagic forms of pancreatitis sometimes arrives at $25-30 \times 10^9$, lymphopenia, change of leukocytic formula to the left and the increased ESR are characteristic. Growth of activity of amylase of blood and urine is very often marked, and is the important sign of pancreatitis. For estimation of the state of other organs maintenance of general albumen and its factions, glucose of blood, bilirubin, urea, electrolytes, acid-base equilibrium (ABE), and also the state of blood coagulation are determined. It is necessary to mark that the exposure of hypocalcemia is considered a bad predictive sign of heavy passing of acute pancreatitis.

Ultrasonic examination (Pic. 3.5.1) of gall-bladder and pancreas often specifies the increase of their sizes, bulge of walls and presence or absence of concrement of gall-bladder and general bilious duct.

Computer tomography enables to describe in details the changes in pancreas and surrounding organs.

At sciagraphy survey of organs of abdominal cavity gives a possibility to expose the unfolded "horseshoe" of duodenum, pneumatization, expansion of transverse colon (the Gobia's symptom). On the 1st stage of diagnostics in the plan of differential diagnosis of acute destructive pancreatitis with other diseases of abdominal cavity, diagnostics of distribution of destructive damaging of different parts of pancreas and estimation of distribution of parapancreatitis is possible only by the method of computer tomography which depending on clinico-laboratory signs and weight of passing is needed to apply in a different period, and sometimes a few times in dynamics with interval of 4–5 days.

Laparoscopy and laparocentesis are often used for a doubtful diagnosis or necessity of taking away the exudation of abdominal cavity for biochemical or bacteriological examination.

Retrograde endoscopic cholangiopancreatography is used in case of mechanical icterus and suspicion of choledocholithiasis. The last methods are invasive and can if it is necessary transform from diagnostic to manipulation treatments: laparoscopic draining of abdominal cavity at pancreatogenic peritonitis and endoscopic papillotomy at choledocholithiasis and biliary pancreatitis.

Clinical passing of disease can be abortive, slowly or quickly progressive. At abortive passing the process is limited to acute edema of pancreas with convalescence in 7–10 days.

Rapid progress is characteristic for pancreatonecrosis. In patients expressed toxemia, impregnation by exudation of retroperitoneal cellulose and development of fermentative hemorrhagic peritonitis can be seen. Strengthening of stomachache, continuous vomiting, proflaccid paresis of intestine, positive symptoms of irritation of peritoneum and growth of hemodynamic violations are the clinical signs of necrosis of pancreas.

There is a formation of parapancreatic infiltrate at slow progress.

Among early complications of acute pancreatitis shock, peritonitis and acute cardiac, pulmonary, hepatic and kidney insufficiency can be distinguished.

Before later complications it is needed to deliver the abscesses of pancreas, subdiaphragmatic, interintestinal abscesses, pyogenic abscess omentum bag, phlegmons of retroperitoneal space and erosive bleeding.

In future formations of pseudocysts, fistula of pancreas, intestinal fistula and development of saccharine diabetes are possible.

Acute pancreatitis needs to be differentiated with the row of acute diseases of organs of abdominal cavity.

Acute mechanical intestinal obstruction. In patients with this pathology pain is of the alternated character and is accompanied by nausea, vomiting, delay of gases and emptying. It is possible to see the Kloyber bowls on the sciagram survey of organs of abdominal cavity.

Acute cholecystitis runs with characteristic localization of pain and muscular defense, with presence of increased, painful gall-bladder or infiltrate in right hypochondrium. Often acute (especially lately) pancreatitis develops on the background of gallstone disease (biliary pancreatitis).

Thrombosis or embolism of mesenteric vessels. Both for pancreatitis and for the thrombosis of mesenteric vessels great pain at soft abdomen (absence of defense muscles of front abdominal wall), that precedes to development of peritonitis, is inherent. Yet from the beginning the disease gains heavy character of passing. In anamnesis in such patients a heart disease or heart attack of myocardium rheumatic is met. As a result of gangrene of intestine, the symptoms of peritonitis appear very quickly and intoxication grows. The fragments of mucus shell are found in flushing waters of intestine at the detailed examination, which have the appearance of "meat flushing".

A perforated gastric and duodenum ulcer is distinguished by the presence of dagger pain, defense of abdominal wall, ulcerous anamnesis.

The conservative method is considered the basic one for treatment of acute pancreatitis,

but in connection with that unsuccessful conservative treatment of patients with acute pancreatitis can often put a question about the necessity of operation, therefore patients must be in permanent surgical establishment. Thus acute pancreatitis with heavy passing is necessary to be treated under the conditions of separation of intensive therapy.

Before conservative treatment hunger, bed rest, fight against pain and enzymic toxemia, conducting of acid-base state, prophylaxis of festering infection and acute ulcers of digestive duct are to be entered .

Patient's stomach is washed by cold soda solution and a cold on an epigastric area and left hypochondrium is used. Medicinal therapy is prescribed also: spasmolytics (papaverine, platyphyllin, no-shparum, baralgin, atropine); inhibitor of protease (contrical, trasilol, gordox, antagosan); cytostatic agent (5-fluorouracil, ftorafur). Positive action of inhibitor of protease is marked only in the first days of disease which are subject to conditioned application of large doses. Antibiotics of wide spectrum of action: a) tienam, which most effective in the prophylaxis of festering pancreatitis, as is selected by pancreatic juice; b) cephalosporins (kefzol, cefazoline); c) cefamizine (mefoxine).

Disintoxication therapy is conducted also (5 % but 10 % solutions of glucose, hemodes, reopolyhukine, polyhukine, plasma of blood, only from 3 to 5 liters on days, in accordance with a necessity).

For the improvement of rheological properties of blood heparine is prescribed (5 000 ODES every 4 hours).

If patients have the expressed pain syndrome and phenomena of general intoxication during all pain period plus 48 hours (by Bakulev), hunger is used. Such mode lasts on the average of 2–4 days. The parenteral feed of albuminous hydrolyzate is thus conducted, by the mixtures of amino acid and fatty emulsion. Alkaline water of to 1–2 l. and albuminous-carbohydrate diet are also appointed. Infusion therapy is complemented by plasma, by albumen, hemodes, reopolyhukine. The improvements of microcirculation in pancreas are achieved due to introduction of reopolyhukine, komplamine, trental and heparin 5000 ODES 6 times per days under the control the indexes of the coagulation system of blood. Anticholinergic drug (sulfate of atropine, methacin, platyphyllin), H₂-histamin blocker (cimetidine, ranisan, ranitidine, famotidine, omeprazol) are also applied. For the removal of pain: 1) sulfate of the atropine 0,1 % — 1 ml + promedol 2 % — 1 ml + papaverine 2 % — 2 ml + analgin 50 % — 2 ml; 2) isotonic solution of chloride of sodium — 500 ml + baralgin — 5 ml + diphenhydramine hydrochloride 1 % — 1 ml + papaverine 2 % — 2 ml + magnesium the sulfate 25 % — 5 ml + ascorbic acid — 5 ml + lipoic acid 0,5 % — 2 ml + novocaine 0,5 % — 10 ml. are used. From the first days by a nasogastral probe the permanent aspiration of gastric maintenance is conducted also. The Motility function of gastro-intestinal highway gets better at application of cerucal or primperane. With the same purpose forced diuresis (maninil, furosemide, aminophylline) is used on the background of intravenous introduction of plenty of liquid.

At uneffective conservative treatment of patients with acute pancreatitis of middle weight and heavy form it is expedient to apply surgical treatment.

Surgical treatment is carried out for patients with biliary pancreatitis (for a day long

from the beginning of disease) in combination with the destructive forms of cholecystitis, at complications of acute pancreatitis by peritonitis, abscess of omentum bag or phlegmon of retroperitoneal cellulose.

Overhead-middle laparotomy, which allows to estimate the state of pancreas, biliary ways and other organs of abdominal cavity, is the best access in this situation. In case of destructive pancreatitis the possible use of lumbar laparotomy from left to right hypochondrium through a mesogastric area is useful.

Cholecystectomy is executed at calculous cholecystitis, phlegmonous inflammation of walls of gall-bladder and biliary pancreatitis. If there are more than 0,9 cm at expansion of choledochus, presence of concrement, ointment-like bile in it, increase of concentration of bilirubin in the whey of blood over 21 mmol/L, choledocholithotomy is complemented by external draining of choledochus. Information of lithiasis of general biliary duct is absent, cholecystectomy in patients with acute pancreatitis is complemented by external draining of choledochus, better by Pikovskyy method (through stump of cystic duct).

Transduodenal sphincteroplasty is shown at fixed concrement of large duodenal papilla (Pic. 3.5.2), if they are diagnosed intraoperative, and also in the cases of papillotomy with extraction of concrement when there is no possibility to execute endoscopic operation .

Omentopancreatopexy. After laparotomy and cutting of gastro-colon and gastro-pancreatic ligament mobile part of large omentum through opening in gastro-colon ligament is conducted and fixed by separate stitches to the peritoneum along the overhead and lower edges of pancreas. Such operation needs to be considered rational at the expressed edema of pancreas and presence of necrosis in it.

Abdominisation of pancreas. A cellulose round pancreas (along the lower and overhead edges of body and tail) is infiltrated by solution of novocaine, after it parietal peritoneum is cut. Under the body and tail glands free end of omentum is conducted and is bundled by a gland. This operation is able to warn the hit of enzymes and products of disintegration in retroperitoneal space.

Sequestrectomy is deleting of necrosis part of gland within the limits of nonviable tissue. Operation is executed in a dull way.

Necrectomy (deleting of necrosis part of gland within the limits of healthy tissue) is executed by an acute way: tissue of gland is cut on verge of necrosis and bleeding vessels are carefully bandaged.

The resection of pancreas is deleting the part of organ with its transversal cutting within the limits of the unchanged (ad oculus) tissue of gland. The resections of tail and body of pancreas are distinguished (Pic. 3.5.3).

Pancreatectomy is a complete deleting of pancreas. Operative treatment is applied infrequently. After the resection of pancreas adequate draining of its bed is very responsible.

The prognosis of disease depends on character of morphological changes of parapancreatic to the cellulose in pancreas. The more difficult destructive changes, the worst the prognosis.

Chronic pancreatitis is a progressive inflammation of pancreas with the periodic acutening and remission, related to the process of autolysis, that shows up by pain, by violation

of exocrine and endocrine functions of gland with the eventual result of fibrosis of organ and high risk of malignization.

A gallstone disease is considered the most frequent reason of chronic pancreatitis. Pathogenesis of cholangiogenic pancreatitis acted in pancreatic ducts (theory of general duct) is in part of difficulty of outflow of pancreatic secret and reflux of infected bile or maintenance of duodenum. Dyskinesia, spasms and stenosis of the Vater's papilla of duodenum are instrumental in reflux. Bile or duodenal maintenance, that gets Wirsung's duct, activates the enzymes of pancreas and is instrumental in the origin of its inflammation. Development of pancreatitis potentiates infection. The last can penetrate pancreas not only due to reflux but also in a hematogenic or lymphogenic way.

Thus, chronic pancreatitis develops as a result of functional violations of pancreas, which with the flow of time pass to organic. The reasons of such violations are the attack of acute pancreatitis suffered the in past, alcoholism, traumas of gland, pathology of its vessels, gastroduodenal ulcers, gastritis or duodenostasis.

The morphological changes in pancreas at chronic pancreatitis are mainly taken to development of passionately-degenerative processes and atrophy of parenchyma. Connecting tissue in such cases develops both in the particles of gland and between them. In one case the process has diffuse character, in the other it is limited. Thus pancreas becomes dense as a result of excrescence of connecting tissue. It can be multiplied, taking shape of chronic hypertrophy pancreatitis. Atrophy of gland comes in other cases, besides, not evenly in different parts.

The inflammatory edema of parenchyma is exposed in case of acutening of process. Hemorrhages, fatty necrosis and pseudocysts are exposed on the surface of cut.

As passing of disease has cyclic character with the periodic changes of remission and acutening, the clinic of chronic pancreatitis depends on the phase of development of inflammatory process. Violation of excretory and incretory functions of pancreas influences polymorphic of symptoms which remission is especially determining in the phase.

Pain, dyspepsia phenomena and progressive loss of weight of body are the basic signs of chronic pancreatitis. Besides, pain, is permanent, changes only its intensity, mainly in epigastric region, sometimes on the left, burning, squeezing or prickly, comes forward the unique symptom of disease, complaints about it precede other symptoms. In some patients the pain feelings increase in lying position. Therefore patients occupy forced sitting position. Intensity of pain can change throughout a day. Patients explain it by acceptance of rich, fried food, boiled eggs, coffee. The last is the principal reason of acutening of process with acute pain syndrome.

It is needed to mark, that occasionally passing of chronic pancreatitis can take hidden, smooth shapes, with the moderately expressed pain syndrome or pain, that has atypical character, for example, stenocardia. In such patients the symptoms related to violations of exogenous function of pancreas come forward. They complain about absence of appetite, nausea, belch, sometimes vomiting and diarrhea with putrid smell. Thirst, general weakness and progressive loss of weight is observed also.

At palpation of abdomen pain does not arise, or it is quite insignificant. It is sometimes succeeded to palpate horizontally placed pancreas as dense, moderately painful tension bar.

The transmission of pulsation of aorta at palpation in a epigastric area count characteristic for pathology.

During intervals between the attacks the feeling of patients remains satisfactory.

Development of saccharine diabetes is the basic sign of endocrine insufficiency, hypoglycemia is rarer. The feature of this form of saccharine diabetes consists in the fact that it shows up in a few years after the beginning of disease, runs easier and often carries latent character. There can be hypoglycemia at the insufficient products of glucagon.

The syndrome of biliary hypertension with development of mechanical icterus and cholangitis determining it can develop in some patients. The reasons for such cholestasis more frequently are tubular stenosis of choledochus, choledocholithiasis or stenotic papillitis. There is duodenal obstruction in some cases.

Important information about it can be given by the laboratory and instrumental methods of examination.

Examination of excretory function of pancreas is based on establishment of level of amylase in the whey of blood and urine. In acutening period of chronic pancreatitis this level of amylase rises, the numbers of tripsin and lipase grow.

Coprologic examination. Macroscopic picture of excrement gets greyish color, in large masses — with unpleasant smell. Steatorrhea (increase of amount of neutral fat) and creatorrhea are characteristic for it (a plenty of muscular fibres).

Examination of incretory function of pancreas includes: 1) determination of sugar in blood and urine (characteristic is hyperglycemia and glycosuria); 2) radioimmunoassay of hormones (insulin, C-peptide and glucagon).

Sciagraphy survey of organs of abdominal cavity in two projections enables to expose existent concrement in ducts and calcificat in parenchyma of pancreas.

Relaxation duodenography. Thus the development of “horseshoe” of duodenum and change of relief of its mucus can be seen (Pic. 3.5.4).

Cholecystocholangiography with the purpose of diagnostics of gallstone disease and second damaging of bilious ways is conducted.

Ultrasonic examination (sonography) is one of the basic methods of diagnostics. With the help of symptoms of chronic pancreatitis it is possible to expose inequality of contours of gland, increase of closeness of its parenchyma, increase or diminishment of sizes of organ, expansion of pancreatic duct and wirsungolithiasis or presence of concrement of parenchyma. Thus it is necessary to inspect gall-bladder, liver and bile-excreting ways for diagnostics of gallstone disease and choledocholithiasis (Pic. 3.5.5).

Scintigraphy of pancreas. On early stages strengthening of scintigraphic picture is observed, on later ones — defects of accumulation to radionuclide (symptom of “sieve” or “bee honeycomb”).

Computer tomography allows to expose the increase or diminishment of sizes of gland, presence of calcificats, concrement, inequality of contours of organ, focuses or diffuse changes of its structure (Pic. 3.5.6, Pic. 3.5.7).

Endoscopic retrograde cholangiopancreatography (ERCPG). Expansion of pancreatic duct its deformation, wirsungolithiasis is marked, (Pic. 3.5.8).

It is expedient to apply laparoscopy in the phase of acutening of chronic pancreatitis at development of fatty and hemorrhagic pancreatonecrosis (“stearin name-plates”, exudation).

The puncture biopsy of pancreas under sonography control can have an important value for differential diagnostics of pancreatitis and cancer.

Percutaneous transhepatic cholangioduodenography and -stomy. This method is used both for differential diagnostics of pseudo tumor-like form of chronic pancreatitis and cancer of pancreas and with the purpose of preoperative preparation at presence of icterus. During it there is a possibility to expose expansion of intra- and out-of-hepatic ducts, localization and slowness of their stricture.

Chronic recurrent pancreatitis. The changes of periods of acuteening and remission are characteristic for it. The first period shows up by the attacks of pain of different frequency and duration, and during remission patients feel satisfactory.

Chronic pain pancreatitis. Intensive pain in the overhead half of abdomen with an irradiation in loin and region of heart is inherent for this form. Also belting pain often appears.

Chronic painless (latent) pancreatitis. In patients with this form of pathology for a long time the pain is either absent in general or arises after the reception of spicy food rich and can be insignificantly expressed. Violation of excretory or incretory function of pancreas come forward on the first plan.

Chronic pseudo tumor-like pancreatitis. Dull pain in the projection of head of pancreas, dyspepsia disorders and syndrome of biliary hypertension are clinical its signs.

Chronic cholangiogenic pancreatitis. Both clinic of chronic cholecystitis and cholelithiasis and clinic of pancreatitis are characteristic for this form.

Chronic indurative pancreatitis. In patients with this diseases symptoms of excretory and incretory insufficiency of pancreas are present. The low indexes of amylase in blood and urine are characteristic. At the expressed sclerosis of head of pancreas the with including process of general bilious duct, development of mechanical icterus is possible.

Among complications of chronic pancreatitis, fatty dystrophy and cirrhosis of liver, stricture of terminal part of general bilious duct, ulcers of duodenum, thrombosis of splenic vein, saccharine diabetes, pseudocysts of pancreas, exudation pleurisy and pericarditis and heart attack of myocardium are observed.

Disease of gall-bladder and bilious ways (gallstone disease, dyskinesia of bilious ways). For these diseases pain in right hypochondrium is inherent, that irradiates in right shoulder-blade and shoulder. At chronic pancreatitis pain is localized in epigastric area, left hypochondrium, often is of belting character. One of the basic additional methods of inspection for confirmation of diseases of gall-bladder and ducts is sonography.

Ulcerous disease of stomach and duodenum. Pain at ulcerous disease is seasonal (relapses more frequent in spring and autumn), unites with heartburn and has tendency to diminishment after vomiting. In patients with chronic pancreatitis pain arises after faults in a diet, often is of belting character. Frequent vomiting is determining, that does not bring facilitation to the patient. Also violations of excretory and incretory functions of pancreas can take place.

Abdominal ischemic syndrome. Patients with this pathology complain about pain, that

arises at once after the reception of meal, somewhat diminishes after application of spasmolytics. For the disease considerable weight loss and waiver of meal in connection with dread of pain attack can be characteristic. The basic method of examination, with a necessity for differential diagnostics, celiacography is useful, which enables to expose occlusion of abdominal trunk or its compression. During conducting of differential diagnostics with two last nosologies it is necessary to state a possibility of origin of secondary pancreatitis.

Cancer of pancreas. Mechanical icterus and presence of Courvoisier's symptom are considered the clearest and most important displays of cancer of head of pancreas, and carcinoma of body and tail is a proof pain syndrome. For the cancer the damage of pancreas, rapid progress of symptomatology are characteristic, and for chronic pancreatitis the protracted passing with proper clinical symptomatology and changes which can be exposed by the laboratory, roentgenologic and instrumental methods of examination are characteristic. The most informative among methods of diagnostics of cancer of pancreas are sonography (echo-producing formations in parenchyma of pancreas), computer tomography (tumor knots) and puncture biopsy of gland with the histological examination (reliable diagnostics of cancer).

Heart attack of myocardium. In anamnesis of patients with the heart attack of myocardium it is possible to expose pain behind breastbone, that arises at the physical activity and emotional stress, it is irradiated in left shoulder-blade and left shoulder, unrelated with the reception of meal and disappears as a result of action of coronarolytics. The typical changes of ECG confirm the diagnosis of heart attack of myocardium. In addition, no violations of external and incretions of pancreas are characteristic. The roentgenologic and instrumental methods of examination can help in differential diagnostics.

Treatment is conducted in the phase of acutening of chronic pancreatitis, as well as at its acute form. In the first days the bed rest and medical starvation is prescribed without limitation of alkaline drink (mineral water). The fight against pain syndrome includes application of anaesthetic preparations and spasmolytics (promedol, analgin, baralgin, papaverine, no-shparum, platyphyllin). Preparation action is directed on the decline of pancreatic secretion (atropine, methacin, sandostatine, dalargin, stilamine, somatostatine) or on oppression of gastric secretion: H₂-blockers (hystodil, cimetidine, hastrocepin, ranisan, tagamet and others like that), antiacides (almagel, gastropan). Appoint, next to it, and antihistaminic preparations (diphenhydramine hydrochloride, suprastine, fenkarol, tavegil). Antienzymic therapy is also important: a) inhibitor of protease (contrical, trasilol, hordox, antagosan), the dose of which must depend on the level of hyperenzymeemia; b) cytostatic agent (ftorafur, 5-fluorouracil); c) chemical inhibitor of tripsin (aminocapronic acid, pentoxil). For the improvement of microcirculation at this pathology heparin, reopolyhlucline and reohluman are applied. The ponderable value is achieved by disintoxication therapy (hemodes, hluconeodes, enterodes). With the purpose of parenteral feed 5–10–40 % glucose with insulin, plasma, albumen, alvesyn, polyamin and lipofundine are used. Normalization of agile function of organs of digestion is achieved by settings of cerucal and reglan. In complex treatment it is necessary to include vitamins (C, B₁, B₆, B₁₂) and anabolic hormones (retabolil, nerobol).

At calming down of the inflammatory phenomena a diet № 5 is prescribed in pancreas and conduct correction of excretory insufficiency of pancreas (festal, pansinorm,

pancreosymin, digestal and others like that). With the purpose of stimulation of function of pancreas it is possible to apply secretin.

Correction of endocrine insufficiency of pancreas. At development of the secondary saccharine diabetes of easy degree a diet is recommended with limitation of carbohydrates, bukaran, maninil and other peroral preparations, at middle and heavy degrees — insulinotherapy.

Physical therapy procedures. Except medical treatment it is possible to apply inductothermy, microwave therapy (high frequency) and electro-stimulation of duodenum. For spa treatment visiting of Morshyn, Husjatyn, Shidnytsja is recommend.

Indication to operation and its volume depend on the form of pancreatitis. Acutening of chronic cholangiogenic pancreatitis at presence of gallstone disease must be examined as indication to operation in first 24 hours since disease's beginning. Operative treatment is done in case of:

- 1) calcinosis pancreas with the expressed pain syndrome;
- 2) violation of patency of duct of pancreas;
- 3) presence of cyst or fistula of resistance to conservative therapy during 2–4 months;
- 4) mechanical icterus on soil of tubular stenosis of distal part of general bilious duct;
- 5) compression and thrombosis of portal vein;
- 6) gallstone disease complicated by chronic pancreatitis;
- 7) ulcerous disease of stomach and duodenum complicated by secondary pancreatitis;
- 8) duodenostasis, complicated by chronic pancreatitis;
- 9) impossibility of exception to operation tumors or violations of arterial circulation of blood of pancreas.

Cholecystectomy at presence of calculous cholecystitis and secondary pancreatitis, acute destructive cholecystitis or hydropsy of gall-bladder.

Choledocholithotomy is executed for patients with cholangiolithiasis: a) with the deaf stitch of general bilious duct (use rarely); b) with its external draining for taking of infected bile (cholangitis), decline of biliary hypertension (at the edema of head of pancreas); c) with internal draining (at tubular stenosis of distal part of general bilious duct, acute expansion of choledochus with the complete loss of elasticity of its wall (execute one of variants of choledochoduodenostomy).

Papillosphincterotomy: a) execute transduodenal with papillosphincteroplasty; b) endoscopic is recommended at the isolated or connected with choledocholithiasis stenosis of large duodenal papilla, fixed concrement of large papilla of duodenum.

Wirsungoplasty is scission of plastic arts of narrow part or distal part of main pancreatic duct (apply at patients with stricture of proximal part of duct by a slowness no more than 2 cm). Lately at the isolated stenosis of bee-entrance of main pancreatic duct endoscopic wirsungotomy is executed.

Pancreatojejunostomy: a) longitudinal (it is executed at considerable expansion of pancreatic duct);) caudal (by Duval) with the resection of distal part of pancreas (Pic. 3.5.9).

Resection of pancreas: a) distal or caudal; b) distal subtotal; c) pancreatoduodenal (PDR); e) total duodenopancreatectomy heads or bodies of gland (execute in case of fibrous-

degenerative pancreatitis).

Oklusion of ducts of pancreas by polymeric connections (cianocrylat, prolamine, neopren and others like that) results in atrophy of exocrine parenchyma, but keeps to the islet of tissue.

Operations on the nervous system are used in case of the pain forms of chronic indurative pancreatitis, resistant to conservative therapy, in default of rough morphological changes of parenchyma, stroma of gland and deformation of main pancreatic duct: a) left-side splanchnicectomy; b) bilateral pectoral splanchnicectomy and sympathectomy; c) postganglionic neurotomy of pancreas.

Cyst of pancreas is a cavity, filled by liquid (pancreatic juice, exudation, pus), intimately soldered with head, body or tail of organ, is limited by capsule, which has epithelium on internal surface.

Pseudocyst (unreal cyst) is a cavity in pancreas which appears as a result of its destruction, limited by capsule, that does not have epithelium on internal surface.

The reasons of pseudocysts are destructive pancreatitis, traumas of pancreas, oklusion of Wirsung's duct by parasite, concrement, tumors, innate anomalies of development.

To the real cysts belong: innate (dysontogenetic) cysts which are anomalic in development; acquired retention cysts which develop as a result of difficult outflow of pancreatic juice, cystadenoma and cystadenocarcinoma (by mechanism the origins belong more frequently to proliferative, sometimes — degenerative cysts).

The mechanism of development of pseudocysts consists in the focus necrosis of gland, difficult normal outflow of its secret, there is a destruction of walls of pancreatic ducts with overrun of pancreatic juice gland that causes reactive inflammation of peritoneum of surrounding organs which form the walls of pseudocyst.

Morphologically the cysts of pancreas are divided into: pseudocysts retention to the duct are innate, single and multiple.

Pseudocysts are fresh and old. The internal surface of fresh pseudocyst is rough, granulating, grey-red. The table of contents is alkaline, grey or with a brown tint. In an old pseudocyst the wall is smooth and shiny, pale-grey. The table of contents is lighter. Epithelium pseudocysts is absent. More frequently they are met in body and tail of gland and are not connected with ducts.

Retention cysts connected with an obturated duct. The cavity has smooth, grey-white surface, maintenance is transparent, watery or mucous-like. Innate cysts are mainly multiple and shallow. A simple retention cyst differ from those that are always connected with the anomalies of development of ducts and are unite with polycystosis buds and liver.

Rarely there are echinococcus cysts, which have a clear chitinous shell, liquid in cavity and daughter's blisters. They are localized in the area of head of pancreas.

According to clinical passing pseudocysts are divided into acute, subacute and chronic.

According to weight of passing — into simple (uncomplicated) and complicated.

In patients with the cystic damaging of pancreas there can be pain of different character and intensity (dull, permanent, cramp-like and belting). It is localized more frequently in right hypochondrium, epigastric area (cyst of head and body of gland), left hypochondrium (cyst of

tail of pancreas). Pain is irradiated in the back, left shoulder-blade, shoulder and spine.

Dyspepsia violations are characteristic. Nausea, vomiting and belch are observed.

The syndrome of functional insufficiency of pancreas shows up by disorders of exocrine and endocrine insufficiency and depends on the degree of damage of organ. The unsteady emptying, replacement of diarrhea of constipation, steatorrhea and creatorrhea, development of the second diabetes are marked.

Compression syndrome. Arises as a result of compression of neighbouring organs. Clinically the compression of organs of gastro-intestinal highway shows up by complete or partial obstruction of general bilious duct (mechanical icterus), vein (portal hypertension) gate, splenic vein (splenomegaly).

During the examination patients with large cysts are marked by asymmetry of abdomen in epigastric and mesogastric areas. At palpation of abdomen tumular formation of elastic consistency with an even, immobile surface is found.

Sonography examination shows echo-free formation with a clear capsule, determines localization and sizes of cyst (Pic. 3.5.10).

Contrasting roentgenologic examination of stomach and duodenum with the sulfate of barium at the cyst of head of pancreas exposes moving of pyloric part of stomach upwards and breeding of „horseshoe” duodenum (at relaxation duodenography in the conditions of low artificial blood pressure). If a cyst is localized in the area of body of gland, displacement of stomach is marked forward and upwards or downward, rapprochement of its walls, moving of duodenal transition and loops of thin bowel downward and to the right; at lateral projection the distance between stomach and spine is increased. The cyst localized in the area of tail of gland, displaces the stomach forward and upwards, to the left or to the right (Pic. 3.5.11).

Cholecystocholangiography exposes calculous cholecystitis and cholelithiasis.

Retrograde pancreatocholangiography exposes the changed and deformed, infrequently extended pancreatic duct, occasionally there can be filling of cavity of cyst by the contrasting matter.

Computer tomography shows accumulation of liquid limited by the capsule of different closeness and thickness (Pic. 3.5.12).

Laboratory examinations exposes hyperamylasemia, steatorrhea and creatorrhea, sometimes — hyperglycemia and glycosuria.

Clinical passing of cysts of pancreas depends on their kind, localization, size, stage of forming and complications.

Four stages of forming of pseudocyst are distinguished (P.G. Karaguljan, 1972).

I stage (1–1,5 months last) — in the center of inflammatory process the cavity of disintegration, which takes surrounding tissue, appears in an omentum bag.

The II stage (2–3 months) is characterized by the beginning of forming of capsule of pseudocyst. Cyst is magnificent, unformed, acute inflammatory phenomena calms down.

The III stage (3–12 months) is completion of forming of capsule of pseudocyst. Last accretes with surrounding organs.

The IV stage (begins an in year from the origin of cyst) is a separated cyst. The cyst is mobile, easily selected from connections with surrounding organs.

Retention cysts arise at closing of lumen of pancreatic duct (concrement, sclerosis). The internal surface of cyst is covered with epithelium. Pain syndrome, violation of exocrine function of gland are characteristic.

Traumatic cysts belong to the pseudocysts with similar passing and clinic, as well as inflammatory pseudocysts.

Parasite cysts (to echinococcus, cysticercotic) are met as casuistry. In such patients Kaconi test and serological Weinberg's reaction are positive.

The variants of clinical passing of the real and unreal cysts depend on their complications.

Perforation in free abdominal cavity. Clinic of the poured peritonitis is characteristic. Tormina, positive symptoms of irritation of peritoneum, possible shock state as a result of irritation of peritoneum by pancreatic juice arise.

Perforation in stomach, duodenum, small, rarer in large intestine is accompanied by diminishment of cyst in sizes or complete disappearance, sometimes diarrhea appears.

Suppuration of maintenance of cyst is accompanied by pain which becomes more intensive, temperature rises, leucocytosis grows.

The erosive bleeding appears suddenly and is accompanied by the symptoms of internal bleeding (expressed general weakness, dizziness). The pallor of skin and mucus shells, sticky death-damp, tachycardia and anemia are observed.

Mechanical icterus arises as a result of compression of cyst on the terminal part of choledochus. The icterus of skin and mucus shells, acholic excrement, dark urine, hyperbilirubinemia, increase of the ALT and AsT level are exposed.

Portal hypertension develops as a result of compression of portal vein. Ascites, varicose expansion of veins of esophagus and stomach, moderate icterus are diagnosed.

Reactive exudation pleurisy more frequently arises in left pleura cavity, where roentgenologic exudation is diagnosed with high maintenance of amylase.

At malignization the walls of cyst specific symptoms are absent, a diagnosis is set during operation (surgical biopsy of cyst wall).

The cysts of pancreas are differentiated with the tumors of abdominal cavity and of retroperitoneal space.

Cancer of pancreas. For the cancer tumor of pancreas syndrome of "small signs" (discomfort in epigastric area, loss of appetite, general weakness), permanent dull pain, unrelated with the reception and composition of meal, icterus (cancer of head of gland), Courvoisier's symptom (increased, unpainfully gall-bladder) are characteristic. Inconstant pain at cysts of pancreas is more frequently related to faults in a diet; in anamnesis destructive pancreatitis, traumas of gland are carried. Sonography examination, retrograde pancreatocholangiography and computer tomography help in establishment of diagnosis.

Tumors of retroperitoneal space are passed asymptomatic, clinic shows up by a considerable compression on neighbouring organs. Nausea, vomit, chronic intestinal obstruction, dysuric disorders arise. Clinic of cysts of pancreas, on the opposite, are expressed on early stages. Pain, dyspepsia syndromes, syndrome of exocrine and endocrine insufficiency of pancreas are characteristic. Pain is related to the reception of meal and alcohol.

Aneurism of abdominal aorta. Dull, indefinite pain in abdomen which is unrelated with the reception of meal, pulsation and pulsating formation in abdomen are characteristic, auscultatory is systolic murmur. Aortography allows to confirm a diagnosis.

The cyst of mesentery of thin bowel has painless passing, at palpation it is mobile, easily changes position in abdomen. The cysts of pancreas are practically immobile, pain, anamnesis and laboratory information are characteristic.

The cyst of liver has protracted asymptomatic passing. Pain appears at infection of cyst. For this pathology symptoms which take place at the cysts of pancreas are not typical (pain related to the reception of rich food, alcohol, hyperamylasemia). Topic diagnostics is carried out at ultrasonic examination, scintigraphy, computer tomography.

Conservative treatment. Treatment of acute or chronic pancreatitis is conducted in accordance with principles. At the unfavorable dynamics of passing the diseases hunger with the permanent sucking of gastric maintenance, parenteral feed and intravenous introduction of liquids are appointed. Puncture of cysts is used through abdominal wall under sonography control with aspiration of maintenance.

Surgical treatment is the method of choice of treatment of cysts of pancreas. The choice of treatment method depends on the stage of forming of pancreas cysts.

On the I stage operation is not used, conservative treatment of pancreatitis is conducted. On the II stage it is used at suppuration of pseudocyst (external draining of cyst). On the III — internal draining of cyst is used. More frequently cystojejunostomy on the eliminated loop of thin bowel by Roux (Pic. 3.5.13), cystojejunostomy with entero-entero anastomosis by Brawn and closing of afferent loop by Shalimov. Cystogastrostomy (Pic. 3.5.14) are executed and cystoduodenostomy is now not applied because of possible complications (infection of cyst, erosive bleeding). Marsupialization (opening and sewing down of cyst to the parietal peritoneum and skin) is used infrequently (at suppuration of cyst is seriously patientsing with the septic state). On the IV stage external and internal draining of cyst and radical operations are applied: a) enucleation of cysts (executed very rarely); b) distal resection of pancreas with a cyst.

The cancer of pancreas is a malignant tumor of epithelium tissue. Its specific gravity among all malignant tumors makes 10 %. Greater part of patients with cancer of pancreas (to 80 %) is made by the persons of capable working age.

The origin of cancer of pancreas is related to character of nutrition: with the promoted maintenance of albumens and fats in meal.

Shortage of vitamins, especially B and C, harmful habits (abuse of alcohol, smoking), presence of carcinogenic matters in food (nitrite, nitrates and others like that), tonsillectomy suffered in the past also belong to etiologic factors. The cancer tumor of pancreas can arise on the background of protracted period of chronic pancreatitis.

A cancer tumor is localized in the head. Rarer — in the area of body or tail, rarer there is a diffuse damage of pancreas.

A tumor has the appearance of a dense knot or conglomerate of knots of different sizes. It resembles epithelium of pancreatic ducts or epithelium of acinous tissue, sometimes — the Langerhans' islet.

Adenocarcinoma (50–55 %) is exposed microscopically, carcinoid (32–35 %), epidermoid cancer or skin is seldom met.

The cancer of pancreas gives metastases quickly enough which spread in lymphogenic way in parapancreatic lymphatic knots, and afterwards — in the gate of liver. The hematogenic metastases are often exposed in lungs, bones, buds and brain. Possible also remote metastases to the type of Virhov's, Shnitsler's, Krukenberg's.

The clinical signs and passing of cancer of pancreas are various. They depend both on localization of tumor in pancreas and the mutual relations of pathological process with surrounding organs or tissue.

Pain is a permanent symptom on which 60–90 % patients specify. It is conditioned by involvement in the process of nervous elements of pancreas and retroperitoneum space. The pain feelings at cancer of pancreas are unrelated with acceptance of meal, can be periodic with irradiation in the back. The insignificant loss of weight makes progress and for a short time becomes considerable enough. Such is clinic of cancer of body and tail of pancreas.

Icterus is characteristic of the cancer of head of pancreas, that arises as a result of obturation of general bilious duct and develops slowly but with steady growth. At palpation of abdomen Courvoisier's symptom is observed. Protracted, to 3–4 weeks, icterus results in piling up of products of disintegration of bile in blood and tissue, causing heavy intoxication of organism, violation of liver unction, buds and coagulation system of blood. Obturation of duct of pancreas causes dyspepsia disorders: belch, nausea, vomiting, diarrhea (“fat” emptying). Distributions of tumor on duodenum and narrowing of its lumen show up by the signs of stenosis of exit from the stomach of a different degree (feeling of plenitude in a epigastric area, periodic pain, belch and vomiting).

Bilirubinemia grows gradually, mainly due to direct bilirubin. The increase of activity of alkaline phosphatase and level of cholesterol are observed in blood. As at mechanical icterus a bile does not get the intestine, stercobilin in excrement is absent (acholic excrement). There is also no urobiliny in urine, although bilious pigments are present there (bilirubin). It is possible to expose steatorrhea and kreatorrhea in excrement as a result of obturation of pancreatic duct and exception of enzymes of pancreas from digestion.

With the help of radioimmunoassay it is sometimes succeeded to mark the increase of level of tumor markers in the whey of blood: cancer-embryo to the antigen, ferritin.

Sciagraphy of gastro-intestinal highway can expose the cancer heads of pancreas, the unfolded “horseshoe” of duodenum, and in case of localization of tumor in the body of gland — displacement of back wall of stomach forward. At duodenoscopy rigidity of mucus shell of descending part of duodenum, narrowing of its lumen are determined, and sometimes there is germination of bowel by tumor.

Scanning is an informing method of examination with the use of ^{75}Se -methionine. Such examinations can expose the hearths of reduced accumulation of isotope or its absence in tissue of gland at the damage by tumor.

During laparoscopy the cancer of pancreas is visualized infrequently, however, dissemination of peritoneum and its metastatic hearths in liver are diagnosed without difficulties.

By ultrasonic (sonography) examination it is succeeded to expose the places of promoted closeness of tissue of gland, sign of mechanical icterus at localization of tumor in the head.

Most informing among all is computer tomography (Pic. 3.5.15). It is possible to define both the tumor of gland and its size and metastatic knots. The changes of main duct of pancreas as segmental stenosis or breaking are fixed on retrograde endoscopic pancreatography.

Clinical passing of cancer of pancreas in 70 % patients is marked by the background diseases and complications. This circumstance allows to select a few clinical forms of the cancer of pancreas before appearance of icterus: pancreatitic, diabetogenic, cholangitic and gastritis-like. The names specify the feature of clinical signs of different forms of disease.

Mechanical icterus is the heaviest complication of cancer of pancreas. With the increase of duration and growth of its intensity development of such dangerous complications, as hepatic or hepatic-kidney insufficiency, cholemic bleeding is possible.

Anamnesis has an important value for differential diagnostics. The presence of attacks of pain or intermittent icterus testifies its calculous origin. A pain syndrome at the cancer of pancreas does not have such acuteness and intensity, as at gallstone disease. Icterus in cancer patients, unlike cholelithiasis, develops gradually, incessantly grows and is of proof character.

Often substantial difficulties arise during conducting the differential diagnostics of obturative and infectious icterus. It is necessary to remember, that at viral hepatitis the level of transaminase and aldolase in the whey of blood rises by 2–3 times. At obturation icterus their level does not change substantially, and the increase of activity of alkaline phosphatase and instead of that the level of cholesterol is marked.

However, most operation difficulties are met during conducting the differential diagnosis of the cancer of pancreas and chronic indurative pancreatitis. In fact both processes during examination and palpation produce similar pictures. In such cases puncture of the densest area of pancreas is executed and cytologic examination is quickly conducted.

Treatment of cancer of pancreas is mainly surgical. The choice of method and volume of operation depends on localization of tumor, stage of process, age of patient and his general condition.

Taking it into account, as practice shows, radical operations in the moment of establishment of final diagnosis are successfully executed only in 15–20 % of patients. Pancreatoduodenal resection is the method of choice of operation in patients with the damage of head of pancreas. Operation foresees deleting one block of head of pancreas, distal part of stomach, duodenum and distal part of general bilious duct. Four anastomosis are thus imposed: gastroenteroanastomosis, cholecysto-enteroanastomosis or choledochenteroanastomosis, pancreatoentero-anastomosis and enteroenteroanastomosis. Sometimes this operation is executed in two stages. On the first one biliary-enteric anastomosis is formed for taking bile and improvement of function of liver, and the second stage is carried out in 3–4 weeks. However, more frequently symptomatic operations are to be executed: cholecysto-enteroanastomosis or choledochoduodenoanastomosis. They are able to liquidate icterus and prolong the life of patients for 5–9 months. In case of damage of body and tail of pancreas the distal subtotal resection of gland with spleen is radical.

POSTVAGOTOMY SYNDROMES

RELAPSE OF ULCER

Etiology and pathogenesis

The relapse of ulcer is enough frequent complication of vagotomy. It meets in 8–12 % patients. The reasons of such relapses of ulcer can be: 1) inadequate decline of products of hydrochloric acid (incomplete vagotomy, reinnervation); 2) disturbance of emptying of stomach (ulcerous pylorostenosis after selective proximal vagotomy or after pyloroplasty); 3) local factors (duodenogastric reflux with development of chronic atrophy gastritis, disturbance of circulation of blood and decline of resistibility of mucous tunic); 4) exogenous factors (alcohol, smoking, medicinal preparations); 5) endocrine factors (hypergastrinaemia: hyperplasia of antral G-cells, the Zollinger-Ellison syndrome; hyperparathyroidism).

Clinical management

Three variants of clinical passing of relapse of ulcer are distinguished after vagotomy: 1) symptomless, when an ulcer is found during endoscopic examination; 2) recurrent with protracted lucid space; 3) persisting ulcer with typical periodicity and seasonality of exacerbation.

It is needed to underline that the clinical signs of this pathology during the relapse are less expressed, than before operation, and absence of pain does not eliminate the presence of ulcer. Sometimes bleeding can be first its sign. Complex examination, that includes roentgenologic, endoscopic examination, study of gastric secretion and determination of content of gastrin in the blood, allows not only to expose an ulcer but also, in most cases, to set its reason. The interpretation the results of gastric secretion examination in such patients are heavy. Taking into account it, it is needed to study both a basal secretion and secretion in reply to introduction of insulin and pentagastrin, and also level of pepsin.



Pic 3. Postvagotomy dilatation of the stomach.

Tactic and choice of treatment method

Approximately in 35 % patients, mainly with the first two variants of clinical passing of disease, the relapses of ulcers, are treated by ordinary methods of conservative therapy. Yet in 30–40 % cicatrization of ulcers comes after application of preparations which stop a gastric secretion (cimetidine, ranitidine—150 mg for night). At other 10–20 % patients, mainly with the third variant of clinical passing, is necessary operative treatment.

The question of choice of the repeated operation in patients with the relapse of ulcer after vagotomy still does not decided. Some surgeons execute revagotomy, trunk vagotomy with drainage operation, revagotomy with antrectomy or resection of stomach. However much majority from them in case of relapse ulcer after vagotomy performed antrectomy in combination with trunk vagotomy.

Postvagotomy diarrhea

Etiology and pathogenesis

Frequency of postvagotomy diarrhea hesitates from 2 to 30 %. The basic sign of complication in patient is present the liquid watery emptying about three times per days. The reasons of diarrhea are: gastric stasis and achlorhydria, denervation of pancreas, small intestine and liver, and also disturbance of motility of digestive tract. Discoordination of evacuations from a stomach, stagnation and hypochlorhydria assist to development in it different microorganisms, and it also can be the reason of diarrhea.

Clinical management

The clinical signs of postvagotomy diarrhea are specific. Acute beginning are typical –patient often does not have time to reach to the rest room. Such suddenness repressing operates on patients. As a result they are forced whole days to be at home, expecting the duty attack. An excrement changes colorings as a result of breeding of pigment and becomes more light.

Tactic and choice of treatment method

Treatment of diarrhea must be complex. Above all things it is needed to recommend a diet with the exception of milk and other provoking products. For the removal of bacterial factor antibiotics are applied. Favourable action in case of the signs of stagnation in a stomach are had weak solutions of organic acids (lemon, apple and others like that).

Among other most distribution was got by the A.A. Kuragin and S.D. Hroismann (1971) suggestion to treat postvagotomy diarrhea by benzohexamethonium (for 1 ml 2,5 % solution 2–3 times per a day). Reported also about successful application of cholesteramine (for 4 g 3 times per a day with the subsequent decline of dose to 4 g per days).

At heavy passing of postvagotomy diarrhea, that does not respond to conservative treatment, it is needed to recommend operative treatment — degastroenterostomy with pyloroplasty. However, the type of drainage operation, as practice shows, does not influence on frequency of diarrhea origin. In this connection, some surgeons with success applied the inversion of the segment of thin bowel, located distal from the area of maximal absorption.

Differential diagnostics and clinical variants

Menetrie syndrome is pseudotumor gastritis. The disease rarely. Etiology and pathogenesis is unknown. During disease observed the increasing of folds of mucus stomach by the height up to 3 cm and thickness up to 2 cm. Deep cracks, which the massive bleeding are from, appear between folds. Diagnostics of bleeding is confirmed by endoscopy. Treatment is conservative, including the hemostatic therapy. If conservative treatment are uneffective, indicated resection of stomach. During operation round a stomach the megascopic lymphatic knots with soft consistency are observed.

Hemobilia is bleeding from bilious ways and liver to the intestine. Meets in 0,01 % all gastric bleeding of unulcerous genesis.

The most frequent reason of hemobilia is the traumas of liver. Among other reasons are inflammatory processes of liver, external bilious ways (abscesses, cholangitis), vascular anomalies as aneurism of hepatic artery and vein gate.

The typical signs of hemobilia: attack-like pain in right hypochondrium, moderate icterus, anaemia, presence of grume in vomiting masses and in the excrement which looks like a pencil or worm (imprints of bilious ducts). Bleeding have cyclic passing (repeat oneself in

6–8 days). A diagnosis is based on the clinical signs, information of endoscopy, at which founded the blood flow to the duodenum from a general bilious duct or bloody clot in the papilla Vateri. The most diagnostic value has selective angiography of the hepatic artery and cholangiography, which allow to expose the flowline of contrasting matter in tissues of liver.

Bleeding from biliary tracts during the damage of large vessels can be severe. So, operation is the unique treatment method in such cases. In patients with hemobilia performed opening, draining and tamponade of the haematomas with obligatory draining of general bilious channel for decompression of biliary tracts. The most radical method some surgeons count opening of haematoma with bandaging of bleeding vessel and bilious channel or resection of liver. Bandaging of hepatic artery after angiographic study of the intraorgan arterial vessels is sometimes recommended only. Better to bandage that branch of hepatic artery from which observed bleeding.

The particle of the rare extragastric diseases complicated by the acute gastrointestinal bleeding is 2 %. Among them the diseases of blood are met, blood vessels, system diseases (leukosis, haemophilia, autoimmune thrombocytopenia, hemorrhagic vasculitis, the Werlhof's disease and others like that).

Leukosis are tumours which developed from hemopoietic cells. Etiology and pathogenesis to this time is not exposed. Patients with a leucosis with the gastrointestinal bleeding is 1 % of all patients with the un ulcerous bleeding.

During leucosis in the process of extramedullar hematosis the cells of vascular wall and vessel are pulled in and from the circulatory changed into hemopoietic, that results in disturbance of permeability of vessel wall. In development of hemorrhage diathesis large part is acted the changes of thrombocytopenia, declines of growth of tissue's basophiles, which produce heparin, that shows up by wide hemorrhages in a gastrointestinal tract. Bleeding can be both insignificant and threatening to life of patient. In establishment of diagnosis sometimes there is enough simple examination of blood (hyperleukocytosis), to suspect leucosis bleeding. During endoscopy in such patients observe the presence of flat, superficial defects of mucus stomach. A final diagnosis is based on the results of biopsy and haematological examination of bone marrow.

Treatment includes complex application of hemostatic, preparations of blood and cytostatic agents, that results in the stop of bleeding and even to bring a patient into remission.

Haemophilia is the innate form of bleeding which caused by the deficit of one of three antihemophilic factors (VIII, IX, XI). The gastrointestinal bleeding is observed in 6–24 % patients with haemophilia. Absence or insufficient content in the blood of antihemophilic globulin lies in basis of disease. At diminishment of it level below 30 % there is bleeding. Haemophilia is inherited, men are ill more frequent.

Pointing in anamnesis on bleeding from babyhood allow to suspect haemophilia. Roentgenologic information and results of fibergastroscopy does not expose the substantial changes in a gastrointestinal tract. Main in diagnostics of haemophilia — examination of the system of blood coagulation. Time of blood coagulation continued to 10–30 minutes, sometimes a blood does not coagulate by hours.

Treatment is directed on compensation of insufficient components of the of blood

coagulation system. In patients with haemophilia A, for which typical deficit of antihemophilic globulin, fresh blood transfusion is indicated, because in a banked blood a antihemophilic globulin collapses during a few hours. At haemophilia B and C are used dry and native plasma, cryoprecipitate, banked blood, because factors IX, XI, which predetermine the form of haemophilia, is kept in them long. ordinary hemostyptic preparation (vicasol, the C vitamin, chloride of calcium and others like that) does not give the effect. So, if form of haemophilia does not established, the treatment is necessary to begin from fresh blood transfusion, antihemophilic plasma and antihemophilic globulin transfusion.

Autoimmune thrombocytopenia, or idiopathic thrombocytopenic purpura, is accompanied by the gastrointestinal bleeding and is arisen up in 0,5–2 % patients. Often bloody vomiting and black excrement conditioned by swallowing of blood from a nose and gums.

The disease shows up by plural hypodermic hemorrhages and hemorrhages into submucous membrane. At girls and women the uterine bleeding is often observed. Thrombocytopenia on very low numbers and it is the most pathognomonic sign of disease. Typical acute increase of duration of bleeding, especially in the period of acute hemorrhage.

Fresh blood and thrombocyte mass transfusion is the most effective treatment in the case of the gastrointestinal bleeding during autoimmune thrombocytopenia. Other hemostatic preparations are indicated also. During operative treatment performed splenectomy. The absolute indications to it are frequent and protracted bleeding, threat of hemorrhage in a brain.

The Schonlein-Henoch disease is hemorrhagic vasculitis, which caused by plural microfocus microthrombovasculitis. The gastrointestinal bleeding at the Schonlein-Henoch disease is observed in 0,5–1 % cases and accompanied with great pain in a epigastric area like “abdominal colic”. For this disease typical presence of purpura which has the symmetric location on the external surface of feet, legs, shoulders, buttocks, also joint syndrome with pain and edema in large joints, kidney syndrome by the type of acute or chronic glomerulonephritis. Women have the possible uterine bleeding. The intestinal bleeding can be accompanied by the edema of wall of intestine, that results in invagination or perforation of wall of bowel.

The basic and pathogenetic treatment method of patients is early application of heparin with blood transfusion, introduction of heparinized blood under the control of blood coagulation, which after adequate therapy must be increased in two times, comparative with a norm. For a patient in the initial form of disease indicated introduction of antibiotics of wide spectrum of action, hormones of adrenal glands cortex.

The diseases of the operated stomach (postgastrectomy and postvagotomy syndromes) are the diseases which arise up after surgical treatment of peptic or duodenum ulcer or other pathology of these organs.

Dumping syndrome is frequent complication of operations which are related to deleting or disturbance of function of goalkeeper (resection of stomach, vagotomy with antrectomy, vagotomy with drainage operations). It takes place in 10–30 % patients.

The rapid receipt (dumping) is considered the starting mechanism of dumping syndrome. During this concentrated, mainly carbohydrate, food passed from a stomach in an empty bowel.

In the phase changes of motility of thin bowel during dumping syndrome important part is acted by the hormones of thin bowel. In endocrine cells of APUD-системи on during dumping-syndrome observed degranulation and presence of hormones of mothiline, neurotensin and enteroglucagon.

The inadequate mechanical, chemical and osmotic irritation of mucous tunic of thin bowel by chymus results for the acute increase of blood flow in a bowel. The last is accompanied by the considerable redistribution of blood, especially in heavy case of dumping syndrome : blood supply of head, lower extremities is diminishes, a blood flow in a liver is multiplied.

The numeral examinations resulted in creation of osmotic theory —the principal reason of dumping syndrome is the decline of volume of circulatory plasma as a result of coming a plenty of liquid into the lumen of thin bowel from an of circulatory system and intercellular space.

For the clinical finding of dumping syndrome typical there is the origin of attacks of general weakness during acceptance of food or during the first 15–20 minutes after it. The attack begins from feeling of plenitude in a epigastric area and is accompanied by the unpleasant feeling of heat, that “spills” in the overhead half of trunk or on all body. Thus is acutely multiplied sweating. Then there is a fatigue, appear somnolence, dizziness, noise in ears, shaking of extremities and worsening of sight. These signs sometimes achieve such intensity, that patients forced to lie down. Loss of consciousness could be in the first months after operation. The attacks are accompanied by tachycardia, sometimes by the shortness of breath, headache, paresthesia of upper and lower extremities, polyuria and vasomotor rhinitis. At the end of attack or after it patients often notice grumbling in a stomach and diarrhea.

A milk or carbohydrate food is the most frequent provoking factor of dumping syndrome. In a period between the attacks patients complain about rapid fatigueability, weakening of memory, decline of working capacity, change of mood, irritates, apathy. During roentgenologic examination after 5–15 minutes observed the increased evacuation of barium mixture through anastomosis by a wide continuous stream, expansion of efferent loop and rapid advancement of contrasting matter in the distal parts of thin bowel (Pic. 3.2.16).

By the expression of symptoms dumping syndrome is divided into three degrees of weight:

I degree is easy. Patients have the periodic attacks of weakness with dizziness, nausea, that appear after the use of carbohydrates and milk food and last no more than 15–20 min. During the attack a pulse becomes more frequent on 10–15 per min., arterial pressure rises or sometimes goes down on 1.3-2 KPa (10–15 mm Hg), the volume of circulatory blood diminishes on 200–300 ml. The deficit of mass of body of patient does not exceed 5 kg. A working capacity is well-kept. Medicinal and dietary treatment gives a good effect.

II degree — middle weight. Attacks of weakness with dizziness, pain in the region of heart, hyperhidrosis, diarrhea. Such signs last, usually, 20–40 min., arise up after the use of ordinary portions of some food. During such state a pulse becomes more frequent on 20–30 per min., arterial pressure is rises (sometimes goes down) on 2–2,7 KPa (15–20 mm Hg), the volume of circulatory blood diminishes on 300–500 ml. The deficit of mass of body of patient

achieves 5–10 kg. A working capacity is reduced. Conservative treatment sometimes has a positive effect, but brief.

The III degree is hard. Patients are disturbed by the permanent, acutely expressed attacks with the collaptoid state, by a fainting fit, by diarrhea, which do not depend on character and amount of the accepted food and last about 1 hour. During the attack is multiplied frequency of pulse on 20–30 per 1 min; arterial pressure goes down on 2,7–4 KPa (20–30 mm Hg), the volume of circulatory blood diminishes more than on 500 ml. The deficit of mass of body exceeds 10 kg. Patients, as a rule, are disabled. Conservative treatment is uneffective.

The problem of treatment of patients with dumping syndrome is not easy. Before the surgical treatment, as a rule, must precede conservative. Patients with the disease of easy and middle degrees respond to conservative treatment, mainly with an enough quite good effect. At the heavy degree of disease such treatment more frequent serves as only preparation to operative treatment. If a patient does not give a consent for operation or at presence of contraindications to operative treatment (disease of heart, livers, kidneys), conservative therapy is also applied. Such treatment must include dietotherapy, blood and plasma transfusion, correction of metabolism, hormonal preparations, symptomatic therapy, electro-stimulation of motility function of digestive tract.

The dietotherapy: using of high-calorie, various food rich in squirrel, by vitamins, by mineral salts, with normal content of fats and exception from the ration of carbohydrates which are easily assimilation (limitation of sugar, sweet drinks, honey, jam, pastry wares, kissel and fruit compotes). All it is needed to use by small portions (5–6 times per days). If the signs of dumping syndrome appear after a food, such patients it is needed to lie down and be in horizontal position not less than 1 hour. At the heavy degree of dumping syndrome patients need to eat slowly, desirably lying on left. Such position creates the best terms for evacuation of food from a stomach. Thus recommend also to repudiate from too hot and cold foods.

Medicinal treatment must include sedative, replaceable, antiserotonin, hormonal and vitamin therapy. The indications to operative treatment of patients with dumping syndrome are: heavy passing of disease, combination of dumping syndrome of middle degree with other postgastrectomy syndromes (with the syndrome of efferent loop, hypoglycemic syndrome and progressive exhaustion) and uneffective of conservative treatment of the dumping syndrome of middle degree. Most methods of operative treatment of dumping syndrome are directed on renewal of natural way of passing of food on a stomach and intestine, improvement of reservoir function of stomach and providing of proportioning receipt of food in a thin bowel.

Depending on reasons and mechanisms of development of dumping syndrome there are different methods of the repeated reconstructive operations. All of them can be divided into four basic groups: I. Operations which slow evacuation from stump of stomach. II. Redoudenization. III. Redoudenization with deceleration of evacuation from stump of stomach. IV. Operations on a thin bowel and its nerves.

Basic stages of reconstructive operations: 1) disconnection of adhesions in an abdominal cavity, releasing of gastrointestinal and interintestinal anastomosis and stump of duodenum; 2) cutting or resection of efferent and afferent loops; 3) renewal of continuity of upper part of digestive tract.

For correction of the accompany postgastrectomy pathology it is better to apply combined anti- (iso-) peristaltic gastrojejunoplasty. Thus transplant by length 20–22 cm, located between a stomach and duodenum, must consist of two parts: antiperistaltic (7–8 cm), connected with a stomach, and isoperistaltic, connected with a duodenum. An antiperistaltic segment brakes dumping of stomach stump, and isoperistaltic — hinders the reflux of duodenum content.

The attacks of weakness at a hypoglycemic syndrome arise up as a result of decline of content of sugar in a blood. It is accompanied by a acute muscular weakness, by headache, by falling of arterial pressure, by feeling of hunger and even by the loss of consciousness. It is needed to remember, that at this pathology, unlike dumping-syndrome, acceptance of food especially sweet facilitates the state of patient. However in some patients both syndromes unite and the attacks of weakness can arise up as directly after food intake, so in a few hours after it. In patients with such pathology the best results are got after antiperistaltic gastrojejunoplasty (Fink, 1976).

The postgastrectomy (agastric) asthenia arises up as a result of disturbance of digestive function of stomach, pancreas, liver and thin bowel.

In patients with such pathology stump of stomach almost fully loses ability to digest a food. It is related to the small capacity of stump and rapid evacuation of food from it, and also with the acute decline of production of hydrochloric acid and pepsin. In the mucous tunics of stump of stomach, duodenum and thin bowels as a result of fall of trophic role of gastrin and other hormones of digestive tract there are the progressive atrophy changes. Absence in gastric juice of free hydrochloric acid is the reason of acute diminishment of digestive ability of gastric juice and decline of it bactericidal. Such situation is assist in advancement to ascending direction of virulent flora, to development duodenitis, hepatitis, cholecystitis, dysbacteriosis, hypovitaminosis and decline of antitoxic function of liver. All it results in acute disturbance of evacuation from a stomach.

The clinical signs of postgastrectomy asthenia arise up after a some latent period which can last from a few months to some years. During this period patients often complain for a general weakness and bad appetite. The basic symptoms of postgastrectomy asthenia are: general weakness, edemata, acute weight loss, diarrhea, skin and endocrine abnormalities. The postgastrectomy asthenia more frequent meets at men at 40–50 years. In most cases diarrhea is the first symptom of disease, that can arise up in 2 months after operation. Diarrhea, usually, has permanent character and sometimes becomes profuse.

Weight loss appears too early, the deficit of mass of body achieves 20–30 kg. A patient quickly loses forces.

Conservative treatment is the blood, plasma and albumen transfusions. These preparations are prescribed 2–3 times per a week. Correction of disturbances of electrolyte exchange is conducted at the same time (transfusion of solutions to potassium, calcium and others like that). For the improvement of processes of albumen synthesis anabolic hormones are prescribed.

Operative treatment foresees the inclusion in the digestion process of duodenum, increase of capacity of stump of stomach and deceleration of evacuation of its content.

The afferent loop consists of part of duodenum, that stopped behind after a resection, area of empty bowel between a duodenojejunal fold and stump of stomach. The syndrome of afferent loop can arise up after the resection of stomach after the Bilroth-II method. Violation of evacuation from a afferent loop and vomiting by a bile are its basic signs.

Acute and chronic obstruction of afferent loop are distinguished. The reason of acute obstruction is mechanical factors: postoperative commissure, volvulus, internal hernia, invagination, jamming behind mesentery of loop of bowel and stenosis of anastomosis.

Frequency of origin of sharp obstruction of afferent loop hesitates within the limits of 0,5–2 %. The disease can arise up in any time after operation: in a few days or a few years.

Chronic obstruction of afferent loop (actually syndrome of afferent loop), as well as acute, can arise up in any time after operation, however more often it develop after the resection of stomach with gastroenteroanastomosis on a long loop, especially when operation is performed without entero-enteroanastomosis by Brown.

The etiologic factors of syndrome of afferent loop are divided into two groups: 1) mechanical (postoperative commissure, invagination, disturbance of evacuation on a afferent loop, wrong location of afferent loop, very long afferent loop, fall of mucous tunic of afferent loop into a stomach); 2) functional (hypertensive dyskinesia of bilious ways and duodenum, damage and irritation of trunks of vagus nerves, hypotensive and spastic states of upper part of digestive tract, heightened secretion of bile and juice of pancreas under act of secretin and cholecystokinin).

For the clinical picture of acute obstruction typical is permanent, with a tendency to strengthening, pain in a epigastric area or in right hypochondrium, nausea and vomiting. At complete obstruction a bile in vomiting masses is absent. The general condition of patient progressively gets worse, the temperature of body rises, leukocytosis grows, tachycardia grows. At the objective examination painful and tension of muscles of abdominal wall is observed. In a epigastric area it is often possible to palpate tumular lump. Possible cases, when the increase of pressure in a bowel is passed on bilious ways and channels of pancreas. There can be pain and icterus in such patients. There are necrosis and perforation of duodenum with development of peritonitis during further progress of process. Acute obstruction of afferent loop in an early postoperative period can be the reason of insufficiency of stump of duodenum also.

During the roentgenologic examination of organs of abdominal cavity it is visible round form area of darkening and extended, filled by gas, bowels loop.

Patients, usually, complain for feeling of weight in a epigastric area and arching in right hypochondrium, that arises in 10–15 min. after acceptance of food and gradually grows. Together with that, appear nausea, bitter taste in a mouth, heartburn. Then there is increasing pain in a right to epigastric area. During this pain arises intensive, sometimes repeated vomiting by a bile, after which the all symptoms disappear. It could be after certain kind of food (milk, fats) or its big amount. Very rarely vomiting by bile unconnected with the feed. In heavy case patients lose up to 1 liter of bile with vomiting masses. During the objective examination observed subicteritiousness of the sclera, sign of dehydration of organism (decline of turgor of skin, dry tongue, oliguria, concentrated urine). Emptying is irregular, grey color, with considerable content of undigested fat and muscular fibres. Anaemia can develop at heavy

passing of disease.

Distinguished easy, middle and heavy degrees of afferent loop syndrome. In patients with the easy degree of disease vomiting is 1–2 times per a month, and insignificant regurgitation arise up through 20 min – 2 hour after a food, more frequent after the use of milk or sweet food. At middle degree of afferent loop syndrome such attacks repeat 2–3 times per week, patients are disturbed by the considerably expressed pain syndrome, and with vomiting up to 200–300 ml of bile is lost. For a heavy degree the daily attacks of pain are typical, that is accompanied by vomiting by a bile (up to 500 ml and more).

A roentgenologic examination of the patients with the afferent loop syndrome is unspecific. Neither the passing of contrasting matter nor absence of filling of afferent loop can be considered as pathognomic signs of syndrome of afferent loop.

Treatment of acute obstruction of afferent loop is mainly operative. Essence of it is the removal of barriers of evacuation of content from an afferent loop. Adhesions are dissected, volvulus is straightened, invagination or internal hernia is liquidated. For the improvement of evacuation between afferent and efferent loops performs the entero-enteroanastomosis type “end-to-end” or after the Roux method.

Conservative treatment of syndrome of afferent loop is ineffective and, mainly, is mean the removal of hypoproteinemia and anaemia, spasmolytic preparations and vitamin are appointed. With this purpose a blood, plasma and glucose is poured with insulin, a novocaine lumbar blockade and blockade of neck-pectoral knot, washing of stomach is also done.

All operative methods of treatment of afferent loop syndrome can be divided into three groups:

- I. Operations, that will liquidate the bends of afferent loop or shorten it.
- II. Drainage operations.
- III. Reconstructive operations.

The operations of the first group, directed on the removal of bends and invagination of afferent loop, can not be considered as radical. They need to be performed only at the grave general condition of patient.

The widest application in clinical practice at the syndrome of afferent loop has the operation offered by Roux (Pic. 3.2.17).

For the prophylaxis of afferent loop syndrome it is necessary to watch after correct imposition of anastomosis during the resection of stomach: to use for the gastroenteroanastomosis short loop of thin bowel (6–8 cm from the Treits ligament) for imposition, to sew afferent loop to small curvature for creation of spur, to fix reliably stump of stomach in peritoneum of transverse colon.

The origin of reflux after the distal resection of stomach is conditioned by some factors:

- I. Traumatic factors: 1) traction of stomach during operation as reason of sprain of ligament of proximal part of stomach and mobilization of large curvature of stomach; 2) cutting of vessels of stomach and oblique muscles of it wall, in particular on small curvature; 3) vagotomy, that is accompanied by cutting of phrenico-esophageal and gastrophrenic ligaments; 4) imposition of gastrointestinal anastomosis, especially direct gastroduodenoanastomosis by Billroth-I, that results in smoothing of the Hisa corner; 5) frequent aspiration of gastric content

in a postoperative period, that causes superficial esophagitis.

II. Trophic factors: 1) damage of vessels which are the reason of ischemia in the area of esophago-gastric connection, and thrombophlebitis of cardial part of stomach; 2) disturbance of influencing of neurohumoral factors which take part in innervations of esophagus; 3) disturbance of trophism of diaphragm as a result of hypoproteinemia and weight loss; 4) ulcerous diathesis and megascopic volume of gastric secretion (especially nightly); 5) regurgitation of alkaline content of duodenum in stump of stomach which reduces tone of its muscular shell.

III. Mechanical factors: 1) gastric stasis; 2) diminishment of volume of gastric reservoir, that is accompanied by the increase of intragastric pressure.

The clinical picture of gastroesophageal reflux is conditioned by the mechanical and chemical irritations of esophagus by content of stomach or thin bowel. As a result, there is esophagitis, which can be catarrhal, erosive or ulcerous-necrotic. The symptoms of reflux are very various and can simulate different diseases of both pectoral and abdominal cavity organs.

The basic complaint of patients with this pathology is a smart behind a breastbone, especially in the area of the its lower part. It, usually, spreads upwards and can be accompanied by considerable salivation. Strengthening of pain at inclinations of trunk gave to the French authors an occasion to name this sign the “symptom of laces”. Unendurable heartburn is the second complaint, that arises up approximately in 1–2 hours after the food intake. Patients forced often to drink, somehow to decrease the unpleasant feelings, however this, certainly, does not bring them facilitation. Some of them, in addition, complain for bitter taste in a mouth.

Pain behind a breastbone often can remind the attack of stenocardia with typical irradiation. Sometimes such reflux is able to provoke real stenocardia.

Hypochromic anaemia is the frequent symptom of gastroesophageal reflux too.

The diagnosis of gastroesophageal reflux, mainly, is based on clinical information, results of roentgenologic examination, esophagoscopy.

The edema, hyperemia of mucous tunic of esophagus, easy bleeding and vulnerability it during examination, surplus of mucus and erosions covered by fibrin tape is considered the endoscopic signs of esophagitis. In doubtful case at the insignificantly expressed macroscopic changes the biopsy of mucous tunic helps to set a diagnosis.

Treatment of patients with gastroesophageal reflux is mainly conservative. Very important is diet, which avoid spicy, rough and hot food. Eating is needed often, by small portions. It is impossible also to lie down after the food intake, because the gastric content can flow in a esophagus. A supper must be not later than for 3–4 hours before sleep. Between the reception of food does not recommend to use a liquid. Next to that, it is necessary to remove factors which promote intraperitoneal pressure (carrying to the bracer, belt, constipation, flatulence). Sleeping is needed in position with a lift head and trunk. From medicinal preparations it is useful to recommend enveloping preparation.

Operative treatment of gastroesophageal reflux, that arose up after the distal resection of stomach, it is needed to recommend to the patients with the protracted passing and uneffective of conservative treatment. During operation, mainly, performed renewal of the broken Hisa

angle. In addition, performed esophagoplasty, fundoplication by Nessen's and esophagofrenofundoplication.

The prophylaxis of this complication consists in the study of the state of cardiac part of stomach before and during every resection and fixing of bottom of stomach to the diaphragm and abdominal part of esophagus during leveling the Hisa angle.

Alkaline reflux-gastritis meets in 5–35 % operated patients after the resection of stomach, antrectomy, gastroenterostomy, vagotomy with pyloroplasty, and also cholecystectomy and papillosphincteroplasty.

The reason of this complication is influence of duodenum content for the mucous tunic of stomach (bilious acids, enzymes of pancreas and isolecithin). Last, forming from bile lecithin under act of phospholipase A, able to destroy the cells of superficial epithelium of mucous tunic of stomach by removing of lipid from their membranes. As a result the erosions and ulcers are formed in the patient organism. Bilious acids also has the expressed detergent's properties. As isolecithin and bilious acids, the very important bacterial flora which directly and through toxins can cause the damage of mucous tunic of stomach stump. Also, alkaline environment and disturbance of evacuation from the operated stomach influence favourably on microflora growth.

For the clinical picture of alkaline reflux-gastritis the permanent poured out pain in a epigastric area, belch and vomiting by a bile are typical. At some patients heartburn and pain is observed behind a breastbone also. In majority patients so proof loss of weight takes place, that even the protracted complex therapy and valuable feed does not provide addition to the deficit of mass of body. There are typical signs also – anaemia, hypo- or achlorhydria.

Reliable diagnostics of alkaline reflux-gastritis became possible after wide introduction in clinical practice of endoscopic examination. In such patients during gastroscopy hyperemia of mucous tunic of stomach is observed. It is often possible to observe reflux in the stomach of duodenum content. During histological examination of biopsy material a chronic inflammatory process, intestinal metaplasia, diminishment of mass of coating cells and area of hemorrhages are found. All it testifies the deep degenerative changes in the mucous tunic of stomach. The some authors underlines that the inflammatory changes, at least in the area of anastomosis, are observed in most persons which carried the resection of stomach. So, endoscopic examination can not be considered deciding in diagnostics. Even the diffuse inflammatory changes can take place in absent of clinical symptoms and, opposite, in case with expressed clinical symptoms the minimum changes of mucous tunic of stomach are sometimes observed.

Conservative treatment of reflux-gastritis (sparing diet, antacides, enveloping preparations), usually, is ineffective. Existent methods of surgical treatment, mainly, directed on the removal of reflux of duodenum content to the stomach. Most popular is operation by the Roux method. The some surgeons considers that distance from gastroenteroanastomosis to interintestinal anastomosis must be 45–50 cm.

Main reason of origin of peptic ulcer of anastomosis is leaving of the hyperacid state of stomach mucous, even after the performed operation. Such phenomenon can be consequence of many reasons: primary economy resection, wrong executed resection (when the mucous tunic of pyloric part is abandoned in stump of duodenum or stomach), heightened tone of vagus

nerves and the Zollinger-Ellison syndrome.

Peptic ulcers, usually, arise up after operation during the first year. Typical signs are pain, vomiting, weight loss, bleeding, penetration and perforation.

Pain is the basic symptom of peptic ulcer. Often it has the same character and localization, as well as at peptic ulcer. However often observe it moving to the left or in the umbilical area. At first patients bind such feelings to the use of food, but then specify nightly and hungry pain. It at first is halted after a food, but in course of time is become permanent, unendurable, independent from food intake. It can increase during the flounces, the walk, can irradiate in the back, thorax or shoulder.

During the objective examination of patients is often possible to expose on a stomach hyperpigmentation from a hot-water bottle. During palpation to the left from epigastric area near a umbilicus the painful and moderate muscles tension of abdominal wall is observed. Sometimes is possible to palpate inflammatory infiltrate of different sizes. During the examination of patients with a peptic ulcer the important role has determination of gastric secretion against a background of histamine and insulin stimulation. There is a necessity also examination of basal secretion. These preoperative examinations in most patients enable to set the reason of hypersecretion which can be: 1) heightened tone of vagus nerves (positive Hollander test); 2) economy resection of stomach, often in combination with the heightened tone of vagus nerve (considerable increase of gastric secretion after histamine or pentagastrin stimulation in combination with the positive Hollander test); 3) abandoned part of mucous tunic of antral part of stomach (high basal secretion and small increase of secretion in reply to histamine and insulin stimulator); 4) the Zollinger-Ellison syndrome.

Roentgenologic diagnostics of peptic ulcer, usually, is difficult, especially at shallow, flat ulcers, bad mobility and insufficient function of anastomosis. A niche is the direct sign of a similar pathology, indirect are the expressed inflammatory changes of mucous tunic of stump of stomach and bowel, painful point in the projection of stump of stomach and anastomosis and bad function of anastomosis. The deciding value in diagnostics has endoscopic examination.

Conservative treatment of peptic ulcers, as a rule, is ineffective. So, operation must be the basic type of treatment. The choice of method of operative treatment depends on character of previous operation and from abdominal cavity pathology found during the revision. For today the most important parts of the repeated operations is vagotomy. There is obligatory also during the resection of stomach on the exception the revision of duodenum stump for liquidation of possibly abandoned mucous tunic of antral area.

Operative treatment at a peptic ulcer must consist of certain stages. Laparotomy and disconnection of adhesions through a considerable spike process (increasing of stomach, loops of intestine and liver to the postoperative scar) almost always causes large difficulties.

After the selection of anastomosis with afferent and efferent loops the last cut by the "UKL-60 appliance", within the limits of healthy tissues with renewal of intestine continuity by "end-to-end" type anastomosis.

At patients with a peptic ulcer, that developed after gastroenterostomy, cut a duodenum and sutured its stump by one of the described methods. During it there can be the difficulties related to the presence in it active ulcer. When peptic ulcers do not cause rough deformation of

stomach, apply degastroenterostomy, vagotomy and drainage operations.

In the case of the considerably expressed spike process it is possible to execute trunk subdiaphragmatic vagotomy, and in case of the insignificantly changed topography of this area — selective gastric vagotomy.

It is important to note, that stomach resected together with anastomosis, peptic ulcer and eliminated area of empty bowel by one block.

This pathology arises up as a result of perforated of peptic ulcer in a transverse colon with formation of connection between a stomach, small or large intestine.

Diagnostics of gastro-colon fistula at patients with expressed clinical signs of disease does not difficult. However, symptoms are often formed and is indicated up slowly, so such patients with different diagnoses long time treat oneself in the therapeutic or infectious parts.

The typical signs of this pathology is considered diminishment or disappearance of pain, that was before, and proof, profuse, that does not respond to treatment, diarrhea. Patients has emptying up to 10–15 times per days and even more frequent. An excrement contains a plenty of undigested muscular fibres and fat acids (steatorrhea). In case of wide fistula an undigested food can be with an excrement.

Excrement smell from a mouth, usually, notice surrounding. The patients does not feel it. However appearance of excrement belch is indicate the hit into the stomach of excrement masses and gases, and could confirm this pathology.

The such patients very quickly lose weight (mass of body goes down on 50–60 %), their skin becomes pale with a grey tint. The protein-free edemata, ascites, hydrothorax, anasarca, signs of avitaminosis appear in non-treated case.

Through the severe losses of liquid and nonassimilable food there can be the increased appetite and unendurable thirst in such patients. However, they adopt a plenty of liquid and food but the state of them continues to get worse.

Headache, apathy and depression is observed, and at the objective examination is exhaustion (ochre colour of skin, dryness and decline of it turgor, edemata or slurred of swelling extremities, atrophy of muscles). A stomach often moderately pigmented from hot-water bottles, subinflated, with the visible peristalsis of intestine. During the changes of patient position it is possible to hear grumbling, splash and transfusion of liquid. The examination of blood can expose hypochromic anaemia.

Roentgenologic examination is a basic diagnostic method. There are three varieties of such examinations of gastro-colon fistula. During the examination with introduction of barium mixture through a mouth the hit of contrasting matter directly from a stomach into a colon is the typical roentgenologic symptom of such pathology. Irrigoscopy is more perfect and effective method. With suspicion on gastro-colon fistula it is better to perform irrigoscopy. Passing of contrasting matter to the stomach at this manipulation testifies the presence of fistula. The third method is insufflation of air in a rectum. With it help on the screen it is possible to observe the location and passing of fistula, and also, as a result, hit of air in a stomach, increase of it gas bubble. Thus there can be the belch with an excrement smell.

The important role played the tests with dyes: at peroral introduction of methylene-blue after the some time it found in excrement masses or, opposite, after an enema with

methylene-blue dye appears in a stomach.

Treatment of gastro-colon fistula is exceptionally operative. It needs to be conducted after intensive preoperative preparation with correction of metabolism. All operations which can be applied at treatment of patients with gastro-colon fistula divide into palliative and radical (single-stage operation and multi-stage operation).

During the palliative operations the place of fistula of stomach, transverse colon and jejunum is disconnected and then sutured the created defects. Other variant is disconnection of stomach and transverse colon and leaving the gastroenteroanastomosis. It is necessary to remember, that during such operations the only fistula always removed and does not performed the resection of stomach. Clearly, that such situation also does not eliminate possibility of relapse of peptic ulcer and development of its complications. Taking into account it, palliative operations can be recommended in those case only, when the general condition of patient does not allow to perform radical operation.

Single-stage operation radical operations. The most widespread is degastroenterostomy with the resection of stomach. However, it is needed to remember that operation of disconnection of fistula, suturing of opening in the jejunum and transverse colon on the lines of fistula and resection of stomach applies only in case of absent of infiltrate and deformation and in the conditions of possibility to close a defect in bowels without narrowing of their lumen. This operation is the simplest, is enough easily carried by patients and it is enough radical.

Such complications appear through considerable time after operation (from 1 month to one year). Disturbances of function of gastrointestinal anastomosis can be caused by the reasons, related both to the technical mistakes during operation and with pathological processes which arose up in the area of anastomosis.

The clinical picture of disturbance of anastomosis function, mainly, depends from the degree of its closing. At complete it obstruction in patients arise up intensive vomiting, pain in a epigastric area, the symptoms of dehydration and other similar signs appear. In other words, the clinic of stenosis of the stomach output develops. Clearly, that during incomplete narrowing the clinical signs will be expressed less, and growth of them — more slow. Sometimes disturbance of evacuation can unite with the syndrome of afferent loop with a inherent clinical picture. At the roentgenologic examination of such patients expansion of stomach stump is exposed with the horizontal level of liquid and small gas bubble. Evacuation from it is absent or acutely slow.

Treatment of scar deformations and narrowing of anastomosis must be operative and directed for the disconnection of accretions and straightening of the deformed areas. In case of presence in patients large inflammatory infiltrate it does not need to perform disconnection. In such cases it is the best to apply roundabout anastomosis. If a resection by Finsterer was done in such patient, better to perform anterior gastroenteroanastomosis, and after a resection by Billroth-I — posterior. As a result of conducting of such operations the state of patient, as a rule, gets better, and often recovered the function of primary anastomosis.

Removing of all stomach and exception of duodenum from the process of digestion of food cause plural functional disturbances in an organism. Some of them meet already after the

resection of stomach (dumping-syndrome, hypoglycemic syndrome), other more inherent for gastrectomy (anaemia, reflux- esophagitis and others like that).

Most patients, that carried gastrectomy, complain for a considerable physical weakness, heightened fatigueability, sometimes is complete weakness, loss of activity and acute decline of work capacity. Almost all of them notice bad sleep, worsening of memory and heightened irritates. The appearance of patients is typical. Their skin insignificantly hyperpigmented, dry, its turgor reduced, noticeable atrophy of muscles. Can be the signs of chronic coronal insufficiency in such patients, and in older-year persons is typical picture of stenocardia. Except for it, can be hypotension, bradycardia and decline of voltage on EKG; during auscultation deafness of tones is observed. From the side of the hormonal system the decline of function of sexual glands is typical: in men — declines of potency, in women — disturbances of menstrual cycle, early climax. Can be the signs of hypovitaminosis A, B, C and decline of resistibility of organism to chill, infectious diseases and tuberculosis.

The decline of mass of body is observed in 75 % patients, that carried gastrectomy. It is conditioned by the decline of power value of food as a result of disturbance of digestion, bad appetite and wrong diet. As a result of progressive hypoproteinemia there can be the protein-free edemata.

Patients with such pathology must be under the permanent clinical supervision and 1–2 times per year during a month to have the course of stationary prophylactic treatment which includes psycho-, diet-, vitaminotherapy, correcting and replaceable therapy, and also prophylaxis of anaemia.

Psychotherapy is especially indicated in the psychodepressive and asthenic states. It is performed in combination with medicinal treatment. Hypnotic preparation, bromide, tranquilizers are applied.

A food must be correctly prepared, without the protracted cooking. Patients need to feed on 6–10 times per days by small portions.

Next to dietotherapy, it is constantly necessary to apply replaceable therapy (Pancreatine, Pansinorm, Festal, Intestopan). In case of absent of esophagitis hydrochloric acid is appointed. For the improvement of albuminous exchange anabolic hormones are applied.

In case of reflux-esophagitis there are indicated feeds by small portions with predominance of liquid, ground, jelly-like foods, astringent, coating, anticholinergic preparations. Between the receptions of food does not recommend to use a liquid. In case of dysphagy appoints a sparing diet.

For the prophylaxis of iron-deficiency anaemia, that arises up in the first 2–3 years after gastrectomy, important the indication of iron preparations.

For warnings and treatments of pernicious anaemia applied cyanocobalamin for 200 mcg through a day and folic acid. Packed red blood cells is indicated in heavy case.

The relapse of ulcer is enough frequent complication of vagotomy. It meets in 8–12 % patients. The reasons of such relapses of ulcer can be: 1) inadequate decline of products of hydrochloric acid (incomplete vagotomy, reinnervation); 2) disturbance of emptying of stomach (ulcerous pylorostenosis after selective proximal vagotomy or after pyloroplasty); 3) local factors (duodenogastric reflux with development of chronic atrophy gastritis, disturbance of

circulation of blood and decline of resistibility of mucous tunic); 4) exogenous factors (alcohol, smoking, medicinal preparations); 5) endocrine factors (hypergastrinaemia: hyperplasia of antral G-cells, the Zollinger-Ellison syndrome; hyperparathyroidism).

Three variants of clinical passing of relapse of ulcer are distinguished after vagotomy: 1) symptomless, when an ulcer is found during endoscopic examination; 2) recurrent with protracted lucid space; 3) persisting ulcer with typical periodicity and seasonality of exacerbation.

It is needed to underline that the clinical signs of this pathology during the relapse are less expressed, than before operation, and absence of pain does not eliminate the presence of ulcer. Sometimes bleeding can be first its sign. Complex examination, that includes roentgenologic, endoscopic examination, study of gastric secretion and determination of content of gastrin in the blood, allows not only to expose an ulcer but also, in most cases, to set its reason. The interpretation the results of gastric secretion examination in such patients are heavy. Taking into account it, it is needed to study both a basal secretion and secretion in reply to introduction of insulin and pentagastrin, and also level of pepsin.

Approximately in 35 % patients, mainly with the first two variants of clinical passing of disease, the relapses of ulcers, are treated by ordinary methods of conservative therapy. Yet in 30–40 % cicatrization of ulcers comes after application of preparations which stop a gastric secretion (cimetidine, ranitidine—150 mg for night). At other 10–20 % patients, mainly with the third variant of clinical passing, is necessary operative treatment.

The question of choice of the repeated operation in patients with the relapse of ulcer after vagotomy still does not decided. Some surgeons execute revagotomy, trunk vagotomy with drainage operation, revagotomy with antrectomy or resection of stomach. However much majority from them in case of relapse ulcer after vagotomy performed antrectomy in combination with trunk vagotomy.

Frequency of postvagotomy diarrhea hesitates from 2 to 30 %. The basic sign of complication in patient is present the liquid watery emptying about three times per days. The reasons of diarrhea are: gastric stasis and achlorhydria, denervation of pancreas, small intestine and liver, and also disturbance of motility of digestive tract. Discoordination of evacuations from a stomach, stagnation and hypochlorhydria assist to development in it different microorganisms, and it also can be the reason of diarrhea.

The clinical signs of postvagotomy diarrhea are specific. Acute beginning are typical –patient often does not have time to reach to the rest room. Such suddenness repressing operates on patients. As a result they are forced whole days to be at home, expecting the duty attack. An excrement changes colorings as a result of breeding of pigment and becomes more light.

Treatment of diarrhea must be complex. Above all things it is needed to recommend a diet with the exception of milk and other provoking products. For the removal of bacterial factor antibiotics are applied. Favourable action in case of the signs of stagnation in a stomach are had weak solutions of organic acids (lemon, apple and others like that).

Among other most distribution was got by the A.A. Kuragin and S.D. Hroismann (1971) suggestion to treat postvagotomy diarrhea by benzohexamethonium (for 1 ml 2,5 % solution

2–3 times per a day). Reported also about successful application of cholesteramine (for 4 g 3 times per a day with the subsequent decline of dose to 4 g per days).

At heavy passing of postvagotomy diarrhea, that does not respond to conservative treatment, it is needed to recommend operative treatment — degastroenterostomy with pyloroplasty. However, the type of drainage operation, as practice shows, does not influence on frequency of diarrhea origin. In this connection, some surgeons with success applied the inversion of the segment of thin bowel, located distal from the area of maximal absorption.

The cancer of stomach is a malignant formation, that develops from epithelium tissue of mucus stomach. Among the tumours of organs of digestion this pathology takes first place and is the most frequent, by the reason of death from malignant formations in many countries of world. Frequency of it at the last 30 years considerably diminished in the countries of Western Europe and North America, but yet remains high in Japan, China, countries of East Europe and South America.

Etiology of cancer of stomach is unknown. It is known that, as other diseases of gastrointestinal tract, a cancer damages a stomach. According to statistical information, it meets approximately in 40 % of all localizations of cancer.

The factors of external environment has the substantial influencing on frequency of this pathology. Above all things, feed, smoke food, salting, freezing of products and their contamination of aflatoxin. Consider that a “food factor” can be: a) by a carcinogen; b) by the solvent of carcinogens; c) to grow into a carcinogen in the process of digestion; d) to be instrumental in action of carcinogens; e) not enough to neutralize carcinogens.

In the USA and countries of Western Europe frequency of cancer of stomach in 2 times more large in the lower socio-economic groups of population. Some professional groups also can it (miners, farmers, works of rubber, woodworking and asbestine industry). High correlation communication is set between frequency of cancer of stomach and use of alcohol and smoking. The value of genetic factors (heredity, blood type) is not led to.

The cancer of stomach arises up mainly in age 60 years and above, more frequent men are ill.

Precancer. The precancer diseases of stomach are: a) chronic metaplastic disregenerator gastritis conditioned by helicobacter pylori; b) villous polypuses of stomach and chronic ulcers; c) nutritional anemia due to vitamin B12 deficiency (pernicious); d) resected stomach concerning an ulcer.

The presence of precancer changes of mucous tunic of stomach has substantial influence for frequency of stomach cancer. In those countries, where morbidity on the cancer of stomach is higher, considerably more frequent chronic gastritises are diagnosed. Lately in etiology of chronic gastritises take the important value helicobacter pylori. In Japan, where the cancer of stomach is in 40 % cases is the reason of death, chronic gastritis appears in 80 % cases of resected stomach, concerning a cancer.

Connection between polypuses, chronic gastric ulcers and possible it malignization comes into question in literature during many decades. Most authors consider that polypuses could be malignant differently. There are three histological types of polypuses: hyperplastic, villous and hamartoma. There are hyperplastic polypuses, but it not malignant.

Hamartoma is accumulation of cells of normal mucous tunic of stomach. They never becomes malignant.

Villous polypuses are potentially malignant in 40 % cases, but it happen in 10 times less, than hyperplastic. The possibility of malignization of chronic gastric ulcers is not proved. The American scientists support a hypothesis, that the cancer of stomach can be ulcerous often, but malignization of ulcers takes place rarely (no more than 3 %). From data of the Japanese scientists, on 50–70th there was higher correlation connection between chronic gastric ulcers and cancer of stomach. The frequent decline of this correlation is lately noticed (70 % on 50–70th and 10 % on 80th).

Frequency of cancer of stomach at patients with pernicious anaemia hesitates within the 5–10 %, that in 20 times higher, compare with control population. In patients with a resected stomach after peptic ulcers is multiplied the risk of origin of stomach cancer in 2–3 times (duration of latent period hesitates from 15 to 40 years). The reason of such dependence is not found out, but there is a version, that this is linked with a gastric epithelium metaplasia by an intestinal type.

From all malignant formations of the stomach in 95 % adenocarcinoma is observed. Epidermoid cancer, adeno-acanthoma and carcinoid tumours do not exceed 1 %. Frequency of leiomyosarcoma hesitates within the limits of 1–3 %. Lymphoma of gastrointestinal tract is localized in a stomach.

The prognosis of localization depends on the degree of invasion, histological variants of tumour.

The macroscopic forms of cancer of stomach in different times were described variously. More than 60 years ago the German pathologist Bermann described 5 macroscopic forms of cancer of stomach: 1) polypoid or mushroom-like; 2) saucer-shaped or with ulcerous and expressly salient edges; 3) with ulcerous and infiltration of walls of stomach; 4) diffuse-infiltrate; 5) unclassified.

American pathopsychologists is selected 4 forms. The tumours of stomach with ulcerous are the most frequent macroscopic form of cancer of stomach and arise up on soil of chronic ulcer. The signs suspicious on malignization are: the sizes of ulcer more than 2 cm in a diameter, appearance of the heightened edges.

The polypoid tumours of stomach observed only in 10 %. These tumours can achieve considerable sizes without an invasion and metastasis. Scirrhus carcinoma is the third macroscopic type. This category of tumours also does not exceed 10 %. The scirrhus carcinoma is the signs of infiltration by anaplastic cancer cells, diffusely developed connecting tissue which results in the bulge and rigidity of wall of stomach. So called “small cancers” belong to the fourth macroscopic type. It meet comparative rarely (no more than 5 %) and is characterized by superficial accumulation of cancer cells which substitute for normal mucus in such kind: a) superficial flat layer which does not rise above the level of mucus; b) salient (bursting) formation; c) erosions.

Mainly (more than 50 %) tumours arise up in a antral part or in distal (lower) third of stomach, rarer (to 15 %) — in a body and in cardia (to 25 %).

However, lately more often observed cardioesophageal cancers and diminishment of

frequency of tumours of distal parts of stomach. In 2 % cases meet the multicentric focuses of growth, but from data of some authors, this percent could be multiplied in 10 times after carefully histological inspection of the resected stomachs. This assertion is based on the theory of the “tumour field” (D.I. Holovin, 1992). Especially this typically for patients which has pernicious anaemia or chronic metaplastic disregenerative gastritis.

Metastasis is carried out by lymphogenic, hematogenic and implantation ways mostly. Three (from data of some authors, four) pools of lymphogenic metastasis are selected: left gastric (knots on passing of small curvature of stomach in a gastro-subgastric ligament and pericardial); splenic (mainly, suprainfrapancreatic knots); hepatic (knots in a hepato-duodenal ligament, right gastric omentum that lower pyloric groups, right gastric and suprapyloric groups, pancreatoduodenal group).

However, the such way of lymphogenic metastasis is conditional and incomplete, as at presence of block lymph flow passes retrograde metastasis, so called “jumping metastases” which predetermine the origin of remote lymphogenic metastases in left supraclavicular lymph nodes (Virhov metastasis) appear, in Lymph nodes of left axillar and inguinal areas, metastases in a umbilicus.

Direct distribution: small and large omentum, esophagus and duodenum; liver and diaphragm; pancreas, spleen, bile ducts.

Front wall of stomach: colon bowel and mesocolon; organs and tissues of retroperitoneal space.

Lymphogenic metastasis: regional lymph nodes, remote lymph nodes, left supraclavicular lymph node (Virhov), lymph node of axillar area (Irish); in a umbilicus (sisters Joseph).

Hematogenic metastasis: liver, lungs, bones, cerebrum.

Peritoneal metastasis: peritoneum, ovarium (the Krukenberg metastasis), Duglas space (the Shnicler metastasis).

All authors which are engaged in the study of problem of cancer of stomach underline absence or vagueness, no specificity of symptoms, especially on the early stages of disease. The displays of cancer of stomach are very various and depend on localization of tumour, character of its growth, morphological structure, distribution on contiguous organs and tissues. At localization of tumour in a cardial part patient complains firstly, as a rule, for appearance of dysphagy.

At careful, purposeful collection of anamnesis it is not succeeded to expose some other, most early symptoms, which precedes to dysphagy and forces a patient to appeal to the doctor. The unpleasant feeling behind a breastbone and feeling of unpassing of hard food on a esophagus appear at the beginning of disease. After some time (as a rule, it is enough quickly, during a few weeks, sometimes even days) a hard food does not pass (it is to wash down by water or other liquid). This period can be during 1–3 months. Patients address a doctor exactly in this period. Other symptoms appear to this time: regurgitation, pain behind a breastbone, loss of mass of body, sometimes even exhaustion, the grey colouring of person, a skin is dry, quickly grows general weakness. Sometimes patients address a doctor, when already with large effort a spoon-meat passes only or complete stenosis came.

At localization of tumour in the antral part of stomach the first complaints, as a rule, are

up to appearance of feeling of weight in epigastric region after the reception of food (even in a two-bit), “feeling of saturation” (after the reception of glass of water), belch (at first it is simple by air, and then with a smell). Feeling of weight grows for a day, patients forced to cause vomiting. In the morning there can be vomiting by mucus with the admixtures of “coffee-grounds” (so called “cancer” water). Patients loses weight (mass of body is lost), a weakness, anaemia grows.

Tumours localized in the body of stomach show up either a pain syndrome or syndrome of so called “small signs” (A.I. Savitsky, 1947), which is characterized by appearance of amotivational general weakness, decline of capacity, rapid fatigueability, depression (by the loss of interest to the environment), proof decline of appetite, gastric discomfort, making progress weight lost.

The carried chronic diseases of stomach, for which typical seasonality, can influence on the clinical sign of cancer of stomach. At appearance of “gastric” complaints out of season or in absent of effect from the got therapy concerning the exacerbation of “gastritis”, “ulcers” must guard a patient and doctor (symptom of “precipice” of gastric anamnesis).

In case of occurring of “gastric” symptoms first in persons in age 50 years and older it is foremost necessary to eliminate the cancer of stomach.

In parts of patients cancer of stomach shows up only the metastatic damage of other organs or complications. More than twenty so called “atypical” forms, which are characterized by “causeless” anaemia, ascites, icterus, fever, edemata, hormonal disturbances, changes of carbohydrate exchange, intestinal symptoms, are distinguished.

During the examination of patients with the cancer of stomach the pallor of skin covers (at anaemia) is observed, in neglected case is “frog” stomach (sign of ascites).

During palpation determined painful in a epigastric area, sometimes possible to palpate the tumour.

During auscultation of patients with pylorostenosis it is possible to define “noise of splash”.

Laboratory information: hypochromic anaemia, neutrophilic leukocytosis, megascopic ESR; during examination of gastric secretion: hypo- and anacidity and achlorhydria.

Gastroduodenoscopy enables to diagnose a tumour even smaller 5 mm and conduct an aiming biopsy with histological examination of the taken material.

Roentgenoscopy and roentgenography examination of stomach. Basic signs: defect of filling, local absence of peristalsis, “malignant” relief of mucous tunic (Pic. 3.2.18).

Ultrasonic examination: presence of metastases in a liver, pancreas.

Computer tomography allows to estimate the basic parameters of tumour, germination in neighbouring organs and presence of metastases.

It is expedient to apply laparoscopy, mainly, for the decision of question about operable of tumour (diagnostics of metastatic defeat of organs of abdominal cavity).

At an early cancer complaints depend on the previous gastric diseases. Therefore, on the basis of clinical information, suspecting a tumour is possible only on occasion, when in patients next to clear pain symptoms an appetite goes down, appear anaemia, general weakness. In practice an early cancer is recognized at purposeful screening, and also in the process of

endoscopic or roentgenologic examination of gastric patients.

A differential diagnosis is conducted with an peptic ulcer, gastritis, polyposis, other gastric and ungastric diseases. For a cancer there is typical firmness of symptoms, instead of their seasonality (typical syndrome of “precipice” of gastric anamnesis) or tendency to their gradual progress.

The row of diseases, with which the cancer of stomach is to differentiate to the doctor, depends from character of complaints of patients.

Five basic clinical syndromes are selected:

- 1) pain;
- 2) gastric discomfort;
- 3) anaemic;
- 4) dysphagic;
- 5) disturbance of evacuation from a stomach.

At patients, at what cancer of stomach shows up a pain syndrome and syndrome of gastric discomfort, a differential diagnosis is conducted with the peptic ulcer, gastritis, cancer of body of pancreas.

It is oriented on features dynamics of development of pain syndrome, ingravescent of the general condition, change of character of complaints.

A question about character of anaemia, source and nature of bleeding decides at an anaemic syndrome. In the process of examination attention is paid to the state of bottom of stomach, where bleeding malignant formations can be.

At a dysphagic syndrome a differential diagnosis is conducted with the cicatrical narrowing, achalasia of esophagus. For malignant formations testify short anamnesis, gradual progress of symptoms, signs of gastric discomfort, general weakness, weight lost.

At disturbance of evacuation from a stomach during stenosis of pyloric part, absence of ulcerous anamnesis, declining years of patients, relatively quick (weeks, months) growth of stenosis testify for tumor.

The presence of cancer of stomach is a indications for surgical treatment. However, counting on success is possible only at presence of the limited tumours (within the limits of the 0–II stages). At the III stage of disease implementation of the widespread combined operations in a radical volume is possible, however most patients die during 1–2 years. A distal or proximal subtotal resection (Pic. 3.2.19) and total gastrectomy (Pic. 3.2.20) is performed with removing of large and small omentumes and regional areas of metastasis with obligatory histological examination of stomach on the lines of resections.

During the combined operations organs which are pulled in to the pathological process are removed.

In case of IV stage of disease and satisfactory state of patient palliative operations which improve quality of life of patient are performed.

In case of presence of complications (mainly stenosis) and grave common condition of patient perform symptomatic operative treatments.

Symptomatic is operations which will liquidate one of symptoms of cancer of stomach. In this group of operations include: 1) roundabout gastrojejunoanastomosis (Pic. 3.2.21) and

jejunosoma (in case of the stenosis tumours of stomach output); 2) gastrostoma (Pic. 3.2.22) in case of the cancer of cardial part of stomach with disturbance of patency; 3) edging of bleedingx vessels in case of complication of cancer by bleeding; 4) tamponade by omentum during the perforation of tumour.

The value of radial therapy and chemotherapy, as independent methods of treatment of cancer of stomach, is limited. Radial therapy is indicated for patients with cardial cancer as preoperative course or as palliative treatment. Adjuvant mono- or polychemotherapy (mainly by 5-phtoruracil) is conducted in a postoperative period as combined therapy and in case of dissemination of the tumours.

Prognosis. The indexes of five-year survival of patients with the cancer of stomach hesitate within the limits of 5–30 %, but, from data of most authors, they do not exceed 10 %.

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ABDOMINAL PAIN, EVALUATION OF PAIN CHARACTERISTICS IN THE DIFFERENTIAL DIAGNOSTIC OF ABDOMINAL DISEASES

Abdominal pain (or stomach ache) is a common symptom associated with transient disorders or serious disease. Diagnosing the cause of abdominal pain can be difficult, because many diseases can cause this symptom. Most frequently the cause is benign and/or self-limiting, but more serious causes may require urgent intervention.

DIFFERENTIAL DIAGNOSIS

Gastrointestinal

GI tract

Inflammatory: gastroenteritis, appendicitis, gastritis, esophagitis, diverticulitis, Crohn's disease, ulcerative colitis, microscopic colitis

Obstruction: hernia, intussusception, volvulus, post-surgical adhesions, tumours, superior mesenteric artery syndrome, severe constipation, hemorrhoids

Vascular: embolism, thrombosis, hemorrhage, sickle cell disease, abdominal angina, blood vessel compression (such as celiac artery compression syndrome), Postural orthostatic tachycardia syndrome

digestive: peptic ulcer, lactose intolerance, coeliac disease, food allergies

Glands

Bile system

Inflammatory: cholecystitis, cholangitis

Obstruction: cholelithiasis, tumours

Liver

Inflammatory: hepatitis, liver abscess

Pancreatic

Inflammatory: pancreatitis

Renal and urological

Inflammation: pyelonephritis, bladder infection

Obstruction: kidney stones, urolithiasis, Urinary retention, tumours

Vascular: left renal vein entrapment

Gynaecological or obstetric

Inflammatory: pelvic inflammatory disease

Mechanical: ovarian torsion

Endocrinological: menstruation, Mittelschmerz

Tumors: endometriosis, fibroids, ovarian cyst, ovarian cancer

Pregnancy: ruptured ectopic pregnancy, threatened abortion

Abdominal wall

muscle strain or trauma

muscular infection

neurogenic pain: herpes zoster, radiculitis in Lyme disease, abdominal cutaneous nerve entrapment

syndrome (ACNES), tabes dorsalis

· Referred pain

· from the thorax: pneumonia, pulmonary embolism, ischemic heart disease, pericarditis

· from the spine: radiculitis

· from the genitals: testicular torsion

· Metabolic disturbance

· uremia, diabetic ketoacidosis, porphyria, C1-esterase inhibitor deficiency, adrenal insufficiency, lead poisoning, black widow spider bite, narcotic withdrawal

· Blood vessels

· aortic dissection, abdominal aortic aneurysm

· Immune system

· sarcoidosis

· vasculitis

· familial Mediterranean fever

· Idiopathic

· irritable bowel syndrome (affecting up to 20% of the population, IBS is the most common cause of recurrent, intermittent abdominal pain)

ACUTE ABDOMINAL PAIN

Acute abdomen can be defined as severe, persistent abdominal pain of sudden onset that is likely to require surgical intervention to treat its cause. The pain may frequently be associated with nausea and vomiting, abdominal distention, fever and signs of shock. One of the most common conditions associated with acute abdominal pain is acute appendicitis.

Selected causes of acute abdomen

· Traumatic : blunt or perforating trauma to the stomach, bowel, spleen, liver, or kidney

· Inflammatory :

· Infections such as appendicitis, cholecystitis, pancreatitis, pyelonephritis, pelvic inflammatory disease, hepatitis, mesenteric adenitis, or a subdiaphragmatic abscess

· Perforation of a peptic ulcer, a diverticulum, or the caecum

· Complications of inflammatory bowel disease such as Crohn's disease or ulcerative colitis

· Mechanical :

· Small bowel obstruction secondary to adhesions caused by previous surgeries, intussusception, hernias, benign or malignant neoplasms

· Large bowel obstruction caused by colorectal cancer, inflammatory bowel disease, volvulus, fecal impaction or hernia

· Vascular : occlusive intestinal ischemia, usually caused by thromboembolism of the superior mesenteric artery

BY LOCATION

Location

· Upper middle abdominal pain

· Stomach (gastritis, stomach ulcer, stomach cancer)

· Pancreas pain (pancreatitis or pancreatic cancer, can radiate to the left side of the waist, back, and even

shoulder)

- Duodenal ulcer, diverticulitis
- Appendicitis (starts here, after several times moves to lower right abdomen)
- Upper right abdominal pain
 - Liver (caused by hepatomegaly due to fatty liver, hepatitis, or caused by liver cancer, abscess)
 - Gallbladder and biliary tract (gallstones, inflammation, roundworms)
 - Colon pain (below the area of liver - bowel obstruction, functional disorders, gas accumulation, spasm, inflammation, colon cancer)
- Upper left abdominal pain
 - Spleen pain (splenomegaly)
 - Pancreas
 - Colon pain (below the area of spleen - bowel obstruction, functional disorders, gas accumulation, spasm, inflammation, colon cancer)
- Middle abdominal pain (pain in the area around belly button)
 - Appendicitis (starts here)
 - Small intestine pain (inflammation, intestinal spasm, functional disorders)
- Lower abdominal pain
- Lower right abdominal pain
 - Cecum (intussusception, bowel obstruction)
 - Appendix point (Appendicitis location)
- Lower left abdominal pain
 - Sigmoid colon (polyp), sigmoid volvulus, obstruction or gas accumulation)
- Pelvic pain
 - bladder (cystitis, may secondary to diverticulum and bladder stone, bladder cancer)
 - pain in women (uterus, ovaries, fallopian tubes)
- Right lumbago and back pain
 - liver pain (hepatomegaly)
 - right kidney pain (its location below the area of liver pain)
- Left lumbago and back pain
 - less in spleen pain
 - left kidney pain
- Low back pain
 - kidney pain (kidney stone, kidney cancer, hydronephrosis)
 - Ureteral stone pain

DIAGNOSTIC APPROACH

When a physician assesses a patient to determine the etiology and subsequent treatment for abdominal pain the patient's history of the presenting complaint and physical examination should derive a diagnosis in over 90% of cases.

It is important also for a physician to remember that abdominal pain can be caused by problems outside the abdomen, especially heart attacks and pneumonias which can occasionally present as abdominal pain.

Investigations that would aid diagnosis include

- Blood tests including full blood count, electrolytes, urea, creatinine, liver function tests, pregnancy test, amylase and lipase.
- Urinalysis
- Imaging including erect chest X-ray and plain films of the abdomen
- An electrocardiograph to rule out a heart attack which can occasionally present as abdominal pain

If diagnosis remains unclear after history, examination and basic investigations as above then more advanced investigations may reveal a diagnosis. These as such would include

- Computed Tomography of the abdomen/pelvis
- Abdominal or pelvic ultrasound
- Endoscopy and colonoscopy (not used for diagnosing acute pain)

ACUTE APPENDICITIS

Acute appendicitis is an inflammation of vermiform appendix caused by festering microflora.

Etiology and pathogenesis

Most frequent causes of acute appendicitis are festering microbes: intestinal stick, streptococcus, staphylococcus. Moreover, microflora can be in cavity of appendix or get there by hematogenic way, and for women – by lymphogenic one.

Factors which promote the origin of appendicitis, are the following: a) change of reactivity of organism; b) constipation and atony of intestine; c) twisting or bends of appendix; d) excrement stone in its cavity; e) thrombosis of vessels of appendix and gangrene of wall as a substance of inflammatory process (special cases).

Pathomorphology

Simple (superficial) and destructive (phlegmonous, gangrenous primary and gangrenous secondary) appendicitises which are morphological expressions of phases of acute inflammation that is completed by necrosis can be distinguished.

In simple appendicitis the changes are observed, mainly, in the distant part of appendix. There are stasis in capillaries and venule, edema and hemorrhages. Focus of festering inflammation of mucus membrane with the defect of the epithelium covering is formed in 1–2 hours (primary affect of Ashoff). This characterizes acute superficial appendicitis. The phlegmon of appendix develops to the end of the day. The organ increases, its serous tunic becomes dimmed, sanguineous, stratifications of fibrin appear on its surface, and there is pus in cavity.

In gangrenous appendicitis the appendix is thickened, its serous tunic is covered by dimmed fibrinogenous tape, differentiating of the layer structure through destruction is not succeeded.

Classification (by V.I. Kolesnikov)

1. Appendiceal colic.
2. Simple superficial appendicitis.
3. Destructive appendicitis:
 - a) phlegmonous;
 - б) gangrenous;
 - в) perforated.
4. Complicated appendicitis:
 - a) appendicular infiltrate;

- б) appendicular abscess;
- в) diffuse purulent peritonitis.

5. Other complications of acute appendicitis (pylephlebitis, sepsis, retroperitoneal phlegmon, local abscesses of abdominal cavity).



Simple superficial appendicitis



Destructive phlegmonous appendicitis:



Destructive gangrenous appendicitis:

Symptoms and clinical course

Four phases are distinguished in clinical course of acute appendicitis: 1) epigastric; 2) local symptoms; 3) calming down; 4) complications.

The disease begins with a sudden pain in the abdomen. It is localized in a right iliac area, has moderate intensity, permanent character and not irradiate. With 70 % of patients the pain arises in a epigastric area - it is an epigastric phase of acute appendicitis. In 2–4 hours it moves to the place of appendix existence (the Kocher's symptom). At coughing patients mark strengthening of pain in a right iliac area – it is a positive cough symptom.

Together with it, nausea and vomiting that have reflex character can disturb a patient. Often there is a delay of gases. The temperature of body of most patients rises, but high temperature can occur rarely and, mainly, it is a low grade fever. The general condition of patients gets worse only in case of growth of destructive changes in appendix.

During the examination it is possible to mark, that the right half of stomach falls behind in the act of breathing, and a patient wants to lie down on a right side with bound leg.

Painfulness is the basic and decisive signs of acute appendicitis during the examination by palpation in a right iliac area, tension of muscle of abdominal wall, positive symptoms of peritoneum irritation. About 100 pain symptoms characteristic of acute appendicitis are known, however only some of them have the real practical value.

The Blumberg's symptom. After gradual pressing by fingers on a front abdominal wall from the place of pain quickly, but not acutely, the hand is taken away. Strengthening of pain is considered as a positive symptom in that place. Obligatory here is tension of muscles of front abdominal wall. [Slide](#).

The Voskresensky's symptom. By a left hand the shirt of patient is drawn downward and fixed on pubis. By the taps of 2-4 fingers of right hand epigastric area is pressed and during exhalation of patient quickly and evenly the hand slides in the direction of right iliac area, without taking the hand away. Thus there is an acute strengthening of pain.

The Bartomier's symptom is the increase of pain intensity during the palpation in right iliac area of patient in position on the left side. At such pose an omentum and loops of thin intestine is displaced to the left, and an appendix becomes accessible for palpation.

The Sitkovsky's symptom. A patient, that lies on left, feels the pain which arises or increases in a right iliac area. The mechanism of intensification of pain is explained by displacement of blind gut to the left, by drawing of mesentery of the inflamed appendix.

The Rovsing's symptom. By a left hand a sigmoid bowel is pressed to the back wall of stomach. By a right hand by balloting palpation a descending bowel is pressed. Appearance of pain in a right iliac area is considered as a sign characteristic of appendicitis.

The Obrazcov's symptom. With the position of patient on the back by index and middle fingers the right iliac area of most painful place is pressed and the patient is asked to heave up the straightened right leg. At appendicitis pain increases acutely.

The Rozdolsky's symptom. At percussion there is painfulness in a right iliac area.

The general analysis of blood does not carry specific information, which would specify the presence of acute appendicitis. However, much leukocytosis and change of formula to the left in most cases can point to the present inflammatory process.

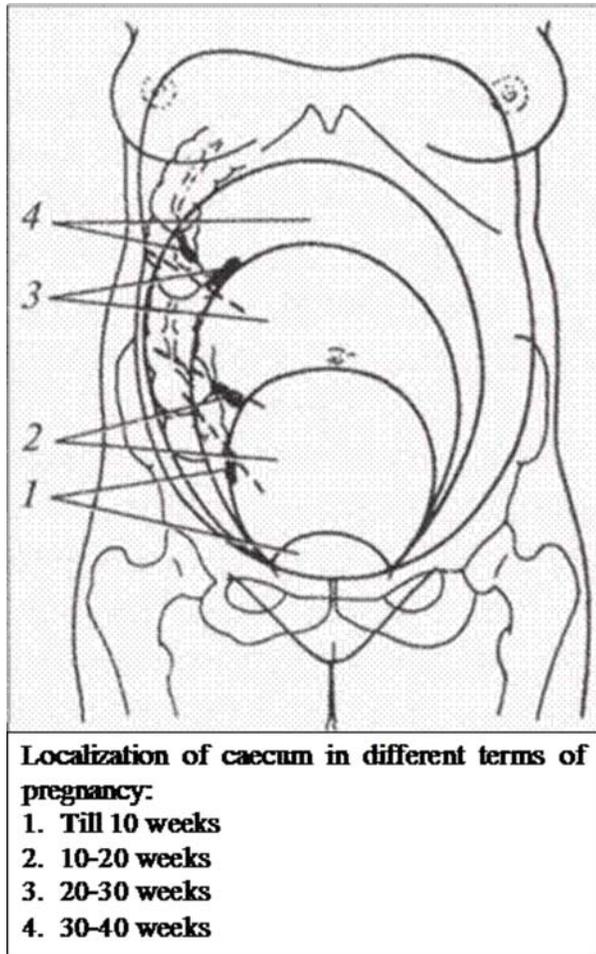
Variants of clinical course and complication

Acute appendicitis in children. With children of infancy acute appendicitis can be seen infrequently, but, quite often carries atypical character. All this is conditioned, mainly, by the features of anatomy of appendix, insufficient of plastic properties of the peritoneum, short omentum and high reactivity of child's organism. The inflammatory process in the appendix of children quickly makes progress and during the first half of days from the beginning of disease there can appear its destruction, even perforation. The child, more frequent than an adult, suffers vomiting. Its general condition gets worse quickly, and already the positive symptoms of irritation of peritoneum can show up during the first hours of a disease. The temperature reaction is also expressed considerably acuter. In the blood test there is high leukocytosis. It is necessary to remember, that during the examination of calmless children it is expedient to use a chloral hydrate enema.

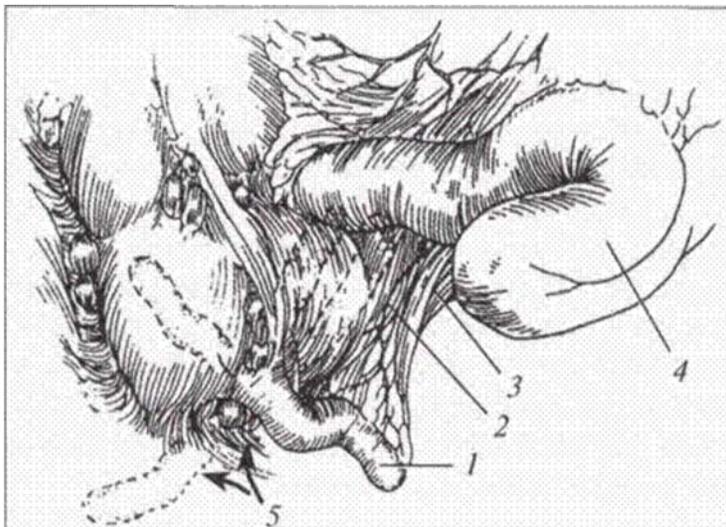
Acute appendicitis of the people of declining and old ages can be met not so often, as of the persons of middle ages and youth. This contingent of patients is hospitalized to hospital rather late: in 2–3 days from the beginning of a disease. Because of the promoted threshold of pain sensitiveness, the intensity of pain in such patients is small, therefore they

almost do not fix attention on the epigastric phase of appendicitis. More frequent are nausea and vomiting, and the temperature reaction is expressed poorly. Tension of muscles of abdominal wall is absent or insignificant through old-age relaxation of muscles. But the symptoms of irritation of peritoneum keep the diagnostic value with this group of patients. Thus, the sclerosis of vessels of appendix results in its rapid numbness, initially-gangrenous appendicitis develops. Because of such reasons the destructive forms of appendicitis prevail, often there is appendiceal infiltrate.

With **pregnant women** both the bend of appendix and violation of its blood flow are causes of the origin of appendicitis. Increased in sizes uterus causes such changes. It, especially in the second half of pregnancy, displaces a blind gut together with an appendix upwards, and an overdistension abdominal wall does not create adequate tension. It is needed also to remember, that pregnant women periodically can have a moderate pain in the abdomen and changes in the blood test. Together with that, *psoas-symptom* and the *Bartomier's* symptom have a diagnostic value at pregnant women.



Clinical course of acute appendicitis at the atypical location (not in a right iliac area) will differ from a classic vermiform appendix .



Variants of appendix localization

1. Appendix
2. Appendicular artery
3. Appendicular mesentery
4. Ilium
5. Caecum

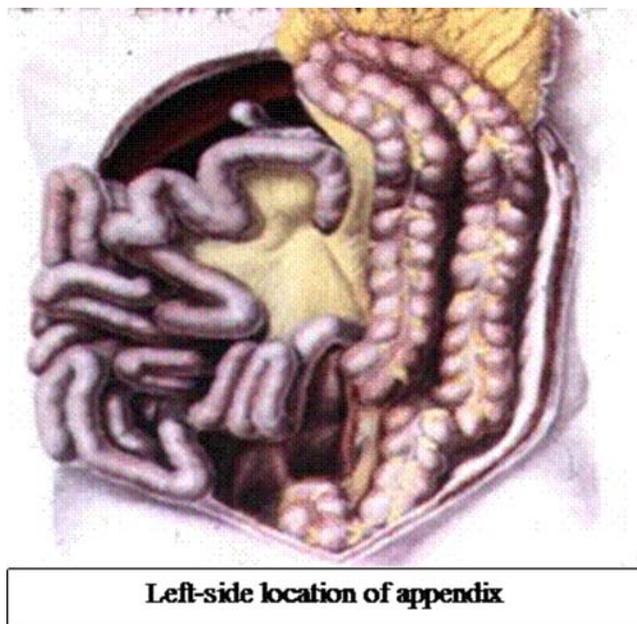
Appendicitis at retrocecal and retroperitoneal location of appendiceal appendix can be with 8–20 % patients. Thus an appendix can be placed both in a free abdominal cavity and retroperitoneal. An atypical clinic arises, as a rule, at the retroperitoneal location. The patients complain at pain in lumbus or above the wing of right ileum. There they mark painfulness during palpation. Sometimes the pain irradiates to the pelvis and in the right thigh. The positive symptom of Rozanov — painfulness during palpation in the right Pti triangle is characteristic. In transition of inflammatory process on an ureter and kidney in the urines analysis red corpuscles can be found.

Appendicitis at the pelvic location of appendix can be met in 11–30 % cases. In such patients the pain is localized above the right Poupart's ligament and above pubis. At the very low placing of appendix at the beginning of disease the reaction of muscles of front abdominal wall on an inflammatory process can be absent. With transition of inflammation on an urinary bladder or rectum either the dysuric signs or diarrhea develops, mucus appears in an excrement. Distribution of process on internal genital organs provokes signs characteristic of their inflammation.

Appendicitis at the *medial placing* of appendix. The appendix in patients with such pathology is located between the loops of intestine, that is the large field of suction and irritation of peritoneum. At these anatomic features mesentery is pulled in the inflammatory process, acute dynamic of the intestinal obstruction develops in such patients. The pain in the abdomen is intensive, widespread, the expressed tension of muscles of abdominal wall develops, that together with symptoms of the irritation of peritoneum specify the substantial threat of peritonitis development.

For the *subhepatic location* of appendix the pain is characteristic in right hypochondrium. During palpation painfulness and tension of muscles can be marked.

Left-side appendicitis appears infrequently and, as a rule, in case of the reverse placing of all organs, however it can occur at a mobile blind gut. In this situation all signs which characterize acute appendicitis will be exposed not on the right, as usually, but on the left.



Among complications of acute appendicitis most value have appendiceal infiltrates and abscesses.

[Appendiceal infiltrate](#) is the conglomerate of organs and tissue not densely accrete round the inflamed vermiform appendix. It develops, certainly, on 3–5th day from the beginning of disease. Acute pain in the stomach calms down thus, the general condition of a patient gets better. Dense, not mobile, painful, with unclear contours, formation is palpated in the right iliac area. There are different sizes of infiltrate, sometimes it occupies all right iliac area. The stomach round infiltrate during palpation is soft and unpainful.

At reverse development of infiltrate (when resorption comes) the general condition of a patient gets better, sleep and appetite recommence, activity grows, the temperature of body and indexes of blood is normalized. Pain in the right iliac area calms down, infiltrate diminishes in size. In this phase of infiltrate physiotherapeutic procedure is appointed, warmth on the iliac area.

In two months after resorption of infiltrate appendectomy is conducted.

At abscessing of infiltrate the condition of a patient gets worse, the symptoms of acute appendicitis become more expressed, the temperature of body, which in most cases gains hectic character, rises, the fever appears. Next to that, pain in the right iliac area increases. Painful formation is felt there. In the blood test high leukocytosis is present with the acutely expressed change of leukocyte formula to the left.

Local abscesses of abdominal cavity, mainly, develops as a result of the atypical placing of appendix or suppuration. More frequent from other there are pelvic abscesses. Thus a patient is disturbed by pain beneath the umbilicus, there are dysuric disorders, diarrhea and tenesmus. The temperature of body rises to 38,0–39,0°C, and rectal — to considerably higher numbers. In the blood test leukocytosis, change of formula of blood is fixed to the left.

During the rectal examination the weakened sphincter of anus is found. The front wall of rectum at first is only painful, and then its overhanging is observed as dense painful infiltrate. [Slide](#).

A *subdiaphragmatic abscess* develops at the high placing of appendix. The pain in the lower parts of thorax and in an upper quarter of umbilicus often to the right, that increases at deep inhalation except for the signs of intoxication, is characteristic of it. A patient, generally, occupies semisitting position. Swelling in an epigastric area is observed in heavy cases, smoothing and painful intercostal intervals. The umbilicus often during palpation is soft, although tension in the area of right hypochondrium is possible. Painfulness at pressure on bottom (9–11) ribs is the early and permanent symptom of subdiaphragmatic abscess (the Krukov's symptom).

Roentgenologically the right half of diaphragm can fall behind from left one while breathing, and there is a present reactive exudate in the right pleura cavity. A gas bubble is considered the roentgenologic sign of subdiaphragmatic abscess with the horizontal level of liquid, which is placed under the diaphragm.

[Interloop abscesses](#) are not frequent complications of acute appendicitis. As well as all abscesses of abdominal cavity, they pass the period of infiltrate and abscess formation with the recreation of the proper clinic.

The poured festering peritonitis develops as a result of the timely unoperated appendicitis. Diagnostics of this pathology does not cause difficulties.

Pylephlebitis is a complication of both appendicitis and after-operative period of appendectomy.

The reason of this pathology is acute retrocecal appendicitis. At its development the thrombophlebitis process from the veins of appendix, passes to the veins of bowels mesentery, and then on to the portal vein. Patients complain at the expressed general weakness, pain in right hypochondrium, high hectic temperature of body, fever and strong sweating. Patients are adynamic, with expressed subicteritiousness of the scleras. During palpation painfulness is observed in the right half of umbilicus often and the symptoms of irritation of peritoneum are not acutely expressed.

In case with rapid passing of disease the icterus appears, the liver is increased, kidney-hepatic insufficiency makes progress, and patients die in 7-10 days from the beginning of disease. At gradual subacute development of pathology the liver and spleen is increased in size, and after the septic state of organism ascites arises.

Diagnostic program

1. Anamnesis information.
2. Information of objective examination.
3. General analysis of blood and urine.
4. Vaginal examination for women.
5. Rectal examination for men.

Differential diagnostics

Acute appendicitis is differentiated with the diseases which are accompanied by pain in the umbilical area.

Food toxicoinfection. Complaints for pain in the epigastric area of the intermittent character, nausea, vomiting and liquid emptying are the first signs of disease. The state of patients progressively gets worse from the beginning. Next to that, it is succeeded to expose that a patient used meal of poor quality. However, here patients do not have phase passing, which is characteristic of acute appendicitis, and clear localization of pain. Defining the symptoms of irritation of peritoneum is not succeeded, the peristalsis of intestine is, as a rule, increased.

Acute pancreatitis. In anamnesis in patients with this pathology there is a gallstone disease, violation of diet and use of alcohol. Their condition from the beginning of a disease is heavy. Pain is considerably more intensive, than during appendicitis, and is concentrated in the upper half of umbilical area. Vomiting is frequent and does not bring to the recovery of patients.

Perforative peptic and duodenum ulcer. Diagnostic difficulties during this pathology arise up only on occasion. They can be in patients with the covered perforation, when portion of gastric juice flows out in an abdominal cavity and stays too long in the right iliac area, or in case of atypical perforations. Taking it into account, it is needed to remember, that the pain in the perforative ulcer is considerably more intensive in epigastric, instead of in the right iliac area. On the survey roentgenogram of organs of abdominal cavity under the right cupula of diaphragms free gases can be found.

The apoplexy of ovary more frequent is with young women and, as a rule, on 10-14 day after menstruation. Pain appears suddenly and irradiate in the thigh and perineum. At the beginning of disease there can be a collapse. However, the general condition of patients suffers insignificantly. When not enough blood was passed in the abdominal cavity, all signs of pathology of abdominal cavity organs calm down after some time. Signs, which are characteristic of acute anemia, appear at considerable hemorrhage. Abdomen more frequent is soft and painful down, (positive Kulenkampff's symptom: acute pain during palpation of stomach and absent tension of muscles of the front abdominal wall).

During paracentesis of back fornix the blood which does not convolve is got.

Extra-uterine pregnancy. A necessity to differentiate acute appendicitis with the interrupted extra-uterine pregnancy arises, when during the examination the patient complains at the pain only down in the stomach, more to the right. Taking it into account, it is needed to remember, that at extra-uterine pregnancy a few days before there can be intermittent pain in the lower part of the abdomen, sometimes excretions of "coffee" colour appear from vagina. In anamnesis often there are the present gynaecological diseases, abortions and pathological passing of pregnancy. For the clinical picture of such patient inherent sudden appearance of intensive pain in lower part of the abdomen. Often there is a brief loss of consciousness. During palpation considerable painfulness is localized lower, than at appendicitis, the abdomen is soft, the positive Kulenkampff's symptom is determined. Violations of menstrual cycle testify for pregnancy, characteristic changes are in milk glands, vagina and uterus. During the vaginal examination it is sometimes possible to palpate increased tube of uterus. The temperature of body more frequently is normal. If hemorrhage is small, the changes in the blood test are not present. The convincing proof of the broken extra-uterine pregnancy is the dark colour of blood, taken at puncture of back fornix of vagina.

Acute cholecystitis. The high placing of vermiform appendix in the right half of abdomen during its inflammation can cause the clinic somewhat similar to acute cholecystitis. But unlike appendicitis, in patients with cholecystitis the pain is more intensive, has cramp-like character, is localized in right hypochondrium and irradiate in the right shoulder and shoulder-blade. Also the epigastric phase is absent. The attack of pain can arise after the reception of spicy food and, is accompanied by nausea and frequent vomiting by bile. In anamnesis patients often have information about a gallstone disease. During examination intensive painfulness is observed in right hypochondrium, increased gall-bladder and positive symptoms Murphy's and Ortner's.

Right-side kidney colic. For this disease termina at the level of kidney and in lumbus is inherent, hematuria and dysuric signs which can take place at the irritation of ureter by the inflamed appendix. Intensity of pain in kidney colic is one of the basic differences from acute appendicitis. Pain at first appears in lumbus and irradiate downward after passing of ureter in genital organs and front surface of the thigh. In diagnostics urogram survey is important, and if necessary — chromocystoscopy. Absence of function of right kidney to some extent allows to eliminate the diagnosis of acute appendicitis.

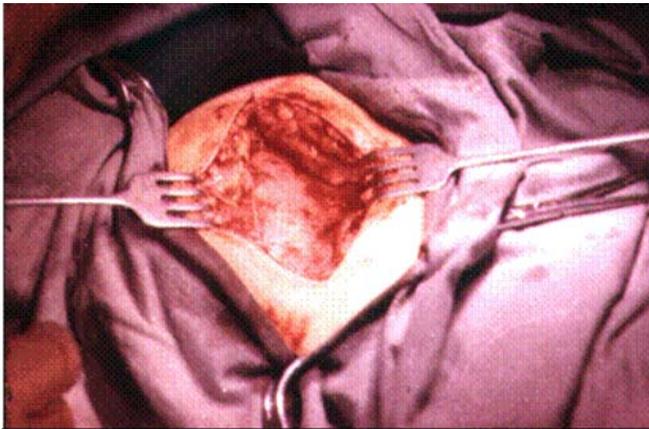
Tactics and choice of treatment method

As experience of surgeons of the whole world testifies, in acute appendicitis timely operation is the unique effective

method of treatment.

Access for appendectomy must provide implementation of operation. [McBurney's incision](#) is typical.

When during operation the appendix without the special difficulties can be shown out [in a wound](#), antegrade *appendectomy* is executed. On clamps its mesentery is cut off and ligated. Near the basis the appendix is [ligated and cut](#). Stump is processed by solution of antiseptic and peritonized by a purse-string suture .



McBurney's incision



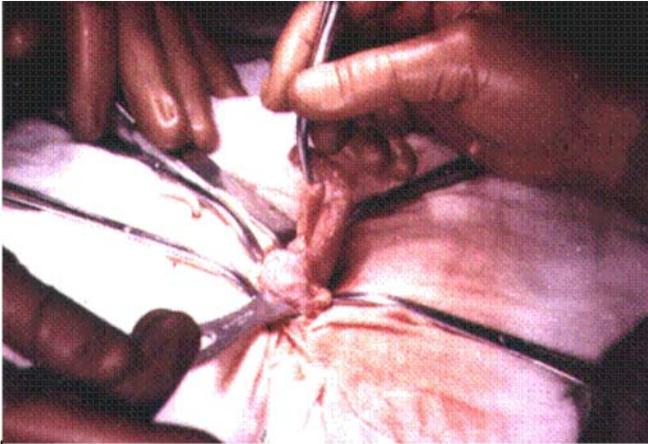
Dissecting of aponeurosis



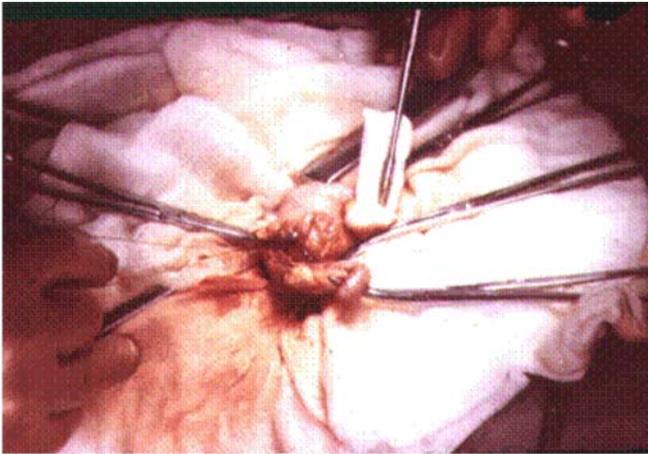
**Deviation of internal oblique and transverse
muscles**



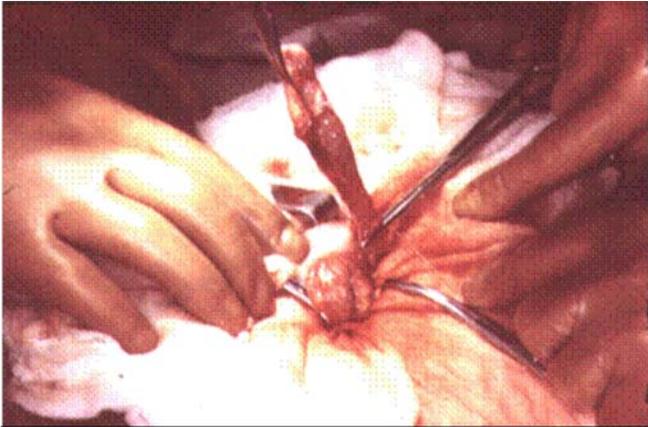
Dissection of the peritoneum



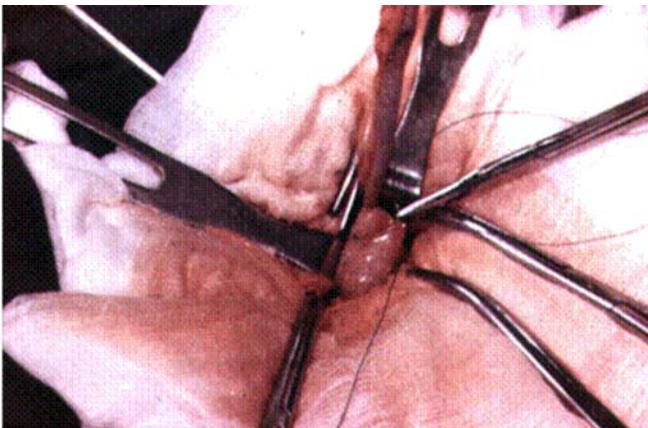
Drawing out the cecum (appendix) into the wound



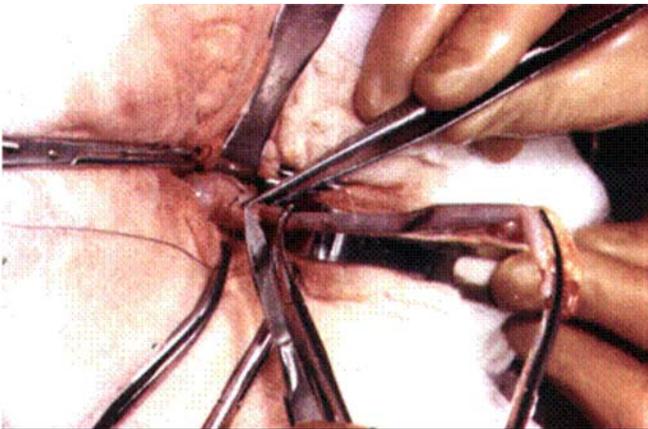
Ligation and cutting off the appendicular mesenterium



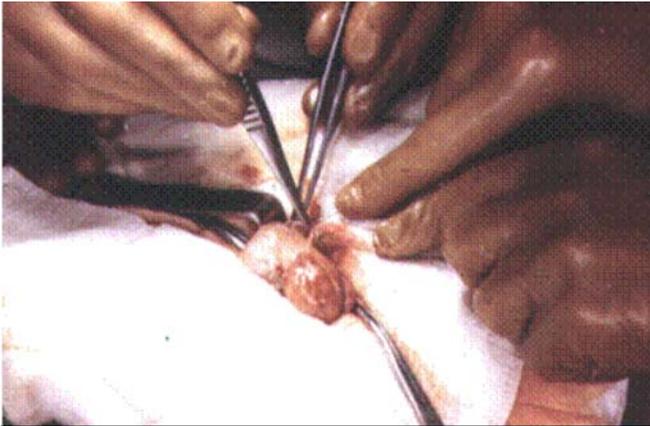
Ligation of appendix near the base



Purse-string suture on the base



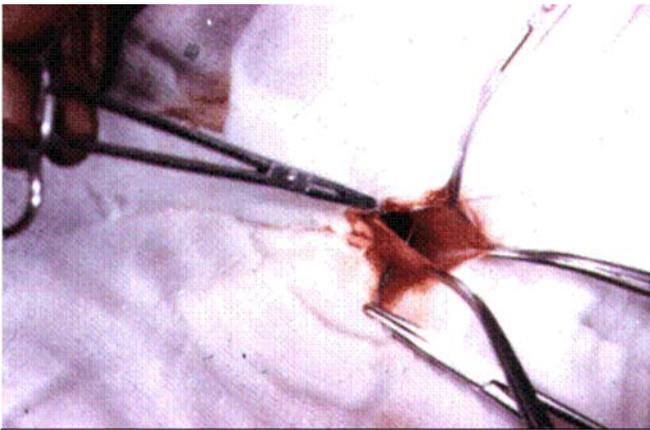
Cutting off the appendix



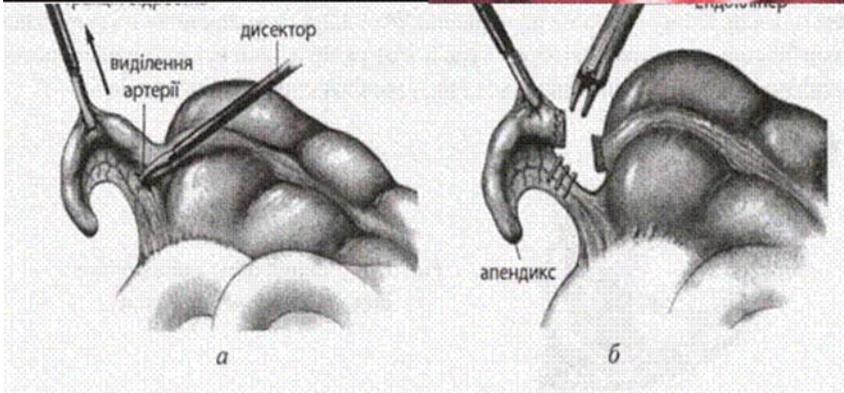
Plunging the appendicular stump



Z-like peritonealization of the appendicular stump

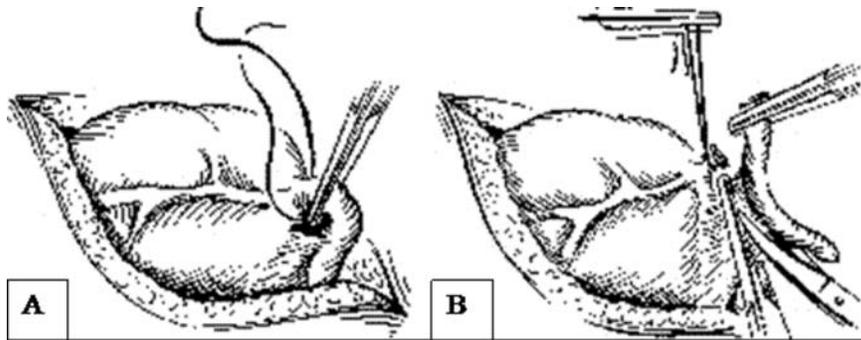


Suturing of peritoneum



Laparoscopic appenectomy

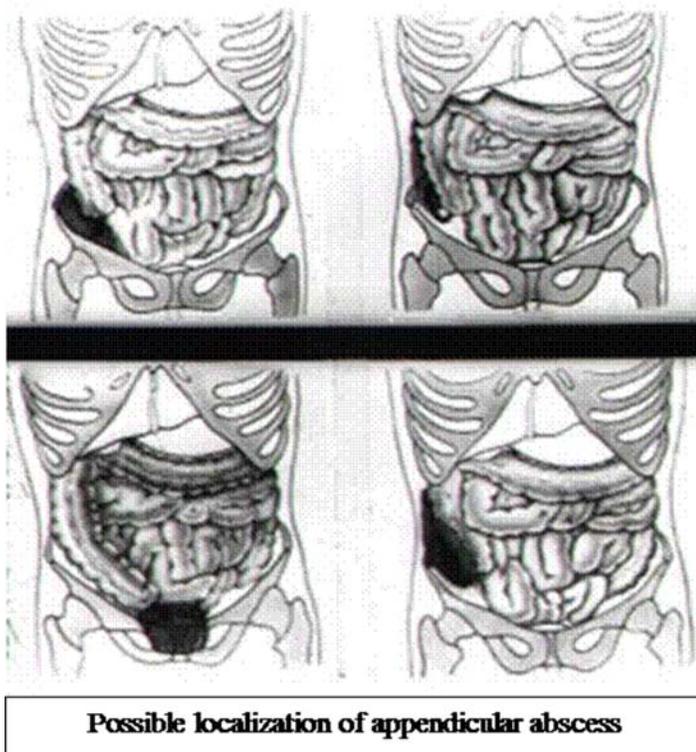
If only the basis of appendix is taken in a wound, and an apex is fixed in an abdominal cavity, more rationally retrograde appendectomy is performed. Thus the appendix near basis is cut between two ligatures. Stump is processed by antiseptic and peritonized. According to it the appendix is removed in the direction from basis to the apex.

**A****B****Retrograde appendectomy****A – ligation of appendicular base****B – mobilization of appendix**

According to indication operation is concluded by draining of abdominal cavity (destructive appendicitis, exudate in an abdominal cavity, capillary hemorrhage from the bed). In recent years the laparoscopy methods of appendectomy are successfully performed.

In patients with appendiceal infiltrate it is necessary to perform conservative-temporizing tactic. Taking it into account, bed rest is appointed, protective diet, cold on the area of infiltrate, antibiotic therapy. According to resorption of infiltrate, in two months, planned appendectomy is executed.

Treatment of appendiceal abscess must be only operative. Opening and drainage of abscess, from [retroperitoneal access](#), is performed. To delete here the appendix is not necessary, and because of danger of bleeding, peritonitis and intestinal fistula — even dangerously.



[video1](#)

[video2](#)

[video3](#)

Acute pancreatitis

The basis of disease of pancreas is degenerative-inflammatory processes which are considered to be acute pancreatitis tissue by its own enzymes. In the structure of acute pathology of organs of abdominal cavity this disease takes the appendicitis and cholecystitis. Women suffer from acute pancreatitis 3–3,5 times more frequently than men.

Anatomy(Fig.1;Fig.2; Fig.3.)

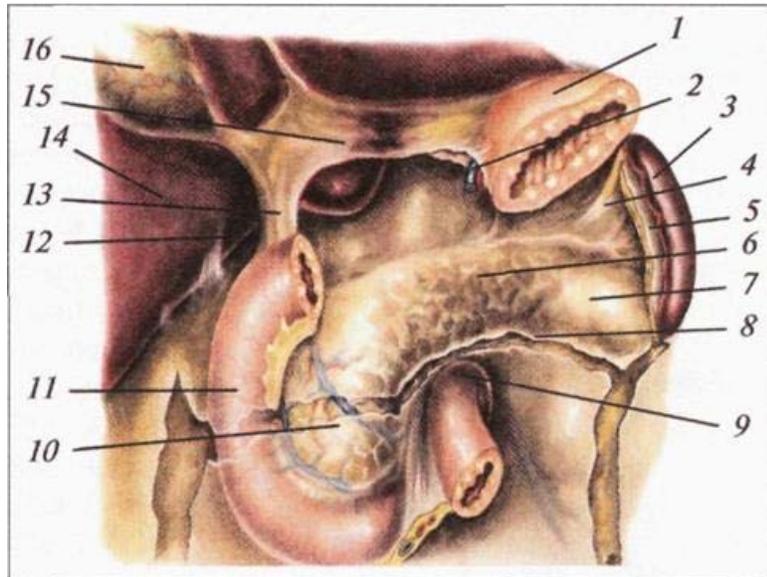


Fig.1. Anatomic and topographic features of the pancreas:

1 — ventriculus; 2 — a. et v. gastrica sinistra; 3— lien; 4— lig. phrenicolienale; 5 — lig. gastrolienal; 6 — c. cauda pancreatis; 8— proection of attaching of the mesocolon transversum; 9 — flexura duodenojejunalis; 10 — duodenum; 11 — foramen epiploicum; 12 — lig. hepatoduodenale; 13 — hepar; 14 — lig. hepatogastrolienale; 15 — lig. hepatocolic; 16 — fellea

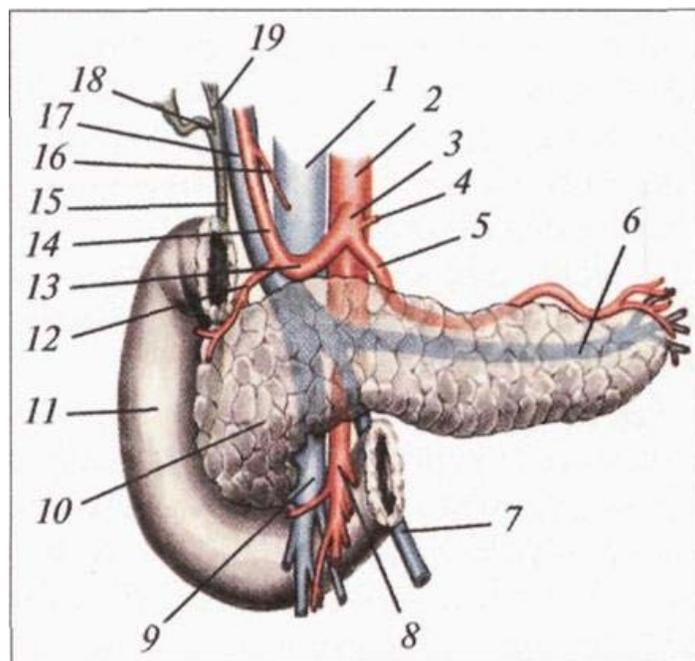


Fig.2. Topography of the pancreas:

1 — v. cava inferior; 2 — aorta abdominalis; 3 — truncus coeliacus; 4—art., gastrica sinistra; 5—art. lienalis; 6— v. lienalis; 7— v. mesenterica inferior; 8—art. mesenterica superior; 9 — v. mesenterica superior; 10— caput pancreatis; 11 — duodenum; 12— art. gastroduodenalis; 13 — art. hepatica communis; 14 — art. hepatica propria; 15 — ductus choledochus; 16 — art. gastrica dextra; 17 — v. portae; 18 — ductus cysticus; 19 — ductus hepaticus communis

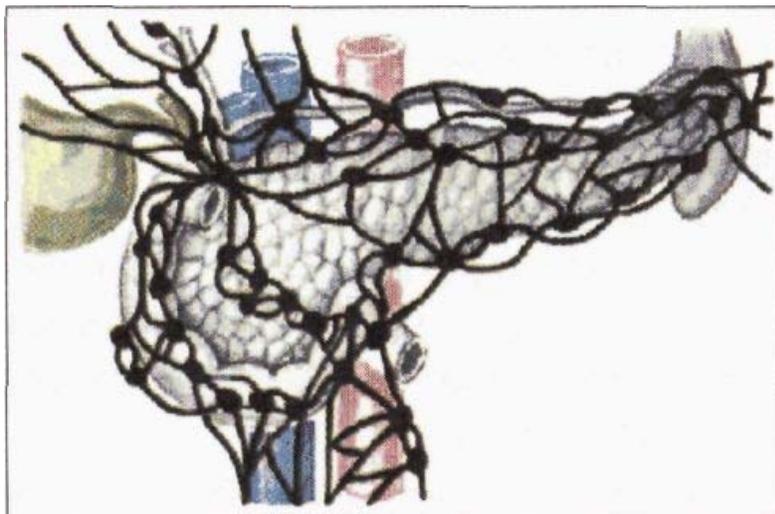


Fig.3. Lymphatic drainage of the pancreas

Etiology and pathogenesis

Acute pancreatitis is a polyetiology disease. Its secondary forms, which arise on the background of pathologies of bile-excreting system and duodenum are closely associated with anatomic and functionally with pancreas, and are met in clinical practice.

Among the “starting” factors of origin of cholelithiasis disease (biliary pancreatitis) abuse by an alcohol and food overloads (fat and irritating products), traumas of pancreas, operating-room in particular, and also separate infectious

diseases (parotitis, mononucleosis) are most frequent, especially infection of bilious ways. However, in 10–20 % of patients the reason of acute pancreatitis remains unknown (cryptogenic form).

In the basis of such damages of pancreas and enzymic toxemia lies mainly activating of pancreatic, and then the tissue enzymes (trypsin, lipase, amylase). Often the combination of the broken outflow of pancreatic secret and promoted secretion takes place, which provokes intraductal hypertension.

Among explanations of primary mechanisms of activating of pancreatic enzymes the most value belongs to: a) theory of “general duct” with reflux of bile in the ducts of pancreas; b) blockade of outflow of pancreatic juice with development of intraductal hypertension and penetration of secret in interstitial tissue; in) violation of blood flow of pancreas (vasculitis, thrombophlebitis and embolisms, cardiac insufficiency and others like that); g) toxic and allergic damages of gland. The role of alcohol in such situations can be dual: stimulation of secretion of pancreas and direct damaging action on its tissue.

Pathomorphology

The process of acute inflammation of pancreas consistently passes the stages of edema, pancreatonecrosis and festering pancreatitis. In the stage of edema there is pancreas of hyperemic, increased in volume, with the shallow hearths of necrosis or, as it is in swingeing majority of cases, without them.

Pancreatonecrosis can pass with fatty or hemorrhagic character. In the first case, as a rule, pancreas is increased, dense, cut whity-yellow hearths are selected to necrosis. Increase of crimson-black pancreas with darkly-brown infiltrate on a cut is characteristic for hemorrhagic pancreatonecrosis.

Dystrophy of parenchyma is exposed microscopically, up to necrosis, hemorrhages, thromboses of vessels and signs of inflammatory infiltration.

Classification

(V All-russian convention of surgeons, 1978)

I. Clinico-anatomy forms:

1. Arching form.
2. Fatty pancreatonecrosis.
3. Hemorrhagic pancreatonecrosis.

II. Prevalence of necrosis:

1. Local (focus) damage of gland.
2. Subtotal damage of gland.
3. Total damage of gland.

III. Ran across: abortive, progressive.

IV. Periods of disease:

1. Period of hemodynamic violations and pancreatogenic shock.
2. Period of functional insufficiency of parenchymatous organs.
3. Period of degenerative and festering complications.

Symptoms and clinical passing

The disease begins suddenly, after the surplus reception of rich spicy food and use of alcohol. Pain, vomiting and phenomena of dynamic intestinal obstruction are considered the most characteristic signs of acute pancreatitis.

A stomach-ache is permanent and so strong, that can result in shock, localized in an epigastric area and left hypochondrium. Some patients feel pain in right hypochondrium with irradiation in the back, loin or breastbone. (Fig.4)

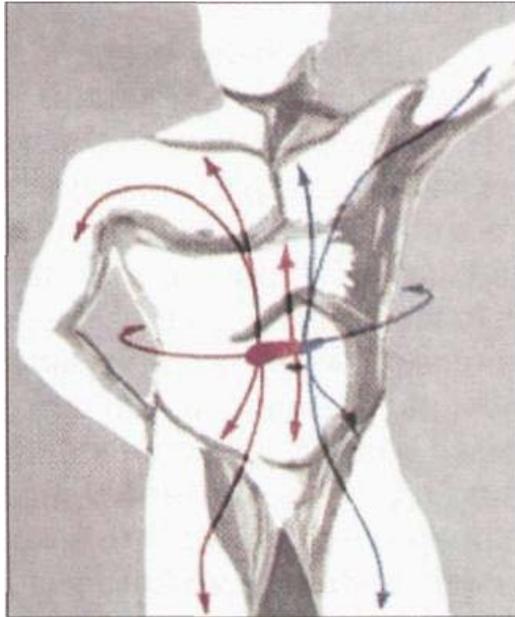


Fig.4.Scheme of pain irradiation at acute pancreatitis

In a short period of time after appearance of pain there is a repeated strong vomiting, that does not facilitate the state of patient.

In general vomiting is considered a frequent and characteristic symptom. It is repeated or continuous and never brings facilitation. Vomit masses contain bile, as admixture, and at the difficult form of acute pancreatitis remind “coffee-grounds”.

Nausea, hiccup, belch and dryness in a mouth are attributed as less characteristic symptoms of this pathology.

During the examination the skin is pale, often subicterus. Some patients have cyanosis with a “marble picture” as a result of violation of microcirculation. Later the component of respiratory insufficiency can join it. At progressive general condition the patient quickly gets worse to passing of acute pancreatitis, intoxication grows. The skin takes shelter with sticky sweat.

The temperature of body of patients at the beginning of disease can be normal. It rises at resorption of products of autolysis tissue and development of inflammatory process in bilious ways.

The pulse in most cases is at first slow, then becomes frequent, notably passing ahead the increase of temperature of body.

Arterial pressure goes down.

The tongue in the first hour of disease is moist, assessed by white and grey raid. At vomiting by bile the raid has yellow or greenish tint.

The abdominal is blown away, peristaltic noises are loosened. The signs of paresis of stomach and intestine demonstrate early. They need to be included in the pathological process of mesentery root of bowel. At palpation painfulness in an epigastric area and in right, and sometimes and in left, hypochondrium is marked. However, in spite of great pain in stomach, it remains soft for a long time. A little later there is moderate tension or resistance of muscles of front abdominal wall.

Poor local symptoms during heavy intoxication are characteristic for the early period of acute pancreatitis. Later there are symptoms of irritation of peritoneum, and at percussion dulling is marked in lateral parts of abdominal as a result of accumulation of liquid, and also the sign of aseptic phlegmon of retroperitoneal cellulose as slurred or edema of lumbar area is seen. For diagnostics of acute pancreatitis there is the row of characteristic symptoms which have different clinical value.

The Mondor's symptom is violet spots on face and trunk.

The Lagermph's symptom is acute cyanosis of person.

The Halsted's Symptom is cyanosis of abdominal skin.

The Gray's symptom is cyanosis of lateral walls of abdomen.

The Kullen's symptom is the yellow colouring of skin near a belly-button.

The Korte's symptom is painful resistance as a lumbar bar in a epigastric area on 6–7 cm higher belly-button.

The Voskresynskyy's symptom is absence of pulsation of abdominal aorta in an epigastric area.

The Mayo-Robson's symptom is feeling of pain at pressure by fingers in the left costal-vertebral corner.

The Rozdolsky's symptom — painfulness at percussion above pancreas.

The Blumberg's symptom — in patients with acute pancreatitis more frequently is low-grade. Such feature of this sign of irritation of peritoneum needs to be explained by character of localization of pathological process, mainly in retroperitoneal spacious.

In clinical passing of pancreatonecrosis it is possible to select three periods (V.S. Saveljev, 1978).

The I period (hemodynamic violations and pancreatogenic shock) lasts during 2–3 days. Violation of central hemodynamics, diminishment of volume of circulatory blood and disorders of microcirculation, which at first arise as a result of angiospasm, are considered the most characteristic signs, and later as a result of joining of the intravascular rolling up and laying of elements of blood.

The II period (insufficiency of parenchymatous organs) lasts from 3rd to the 7th day of disease. Violation of functions of basic organs and systems, sign of cardio-vascular, hepatic and kidney insufficiency and growth of violations of breathing are thus observed. In this period there is possible damaging of the central nervous system, which is erected mainly to disorders of psyche, appearances of delirium and commas which in the eventual result are the main reasons of patients' death.

The III period (postnecrosis dystrophic and festering complications) comes in 1–2 weeks after the beginning of disease. During it, on the background of progress of necrosis processes in pancreas, the regenerative changes develop, there are parapancreatic infiltrate and cysts, cystic fibrosis of pancreas. Aseptic retroperitoneal phlegmon which strengthens intoxication can also develop. There is festering pancreatitis at joining of infection. During this period such complications, as erosive bleeding, internal or external fistula, retroperitoneal phlegmon, can develop in patients. (Fig.5)



Fig.5. Scheme of pyo-inflammatory diffusion at acute pancreatitis

- 1- subhepatic space(6%)
- 2- right lateral flanc(23%)
- 3- subphrenic space(36 %)
- 4- left lateral flanc(40%)
- 5- retroperitoneal space(67%)

From laboratory information leucocytosis which at the necrosis and hemorrhagic forms of pancreatitis sometimes arrives at $25-30 \times 10^9/l$, lymphopenia, change of leukocytic formula to the left and the increased ESR are characteristic. Growth of activity of amylase of blood and urine is very often marked, and is the important sign of pancreatitis. For estimation of the state of other organs maintenance of general albumen and its factions, glucose of blood, bilirubin, urea, electrolytes, acid-base equilibrium (ABE), and also the state of blood coagulation are determined. It is necessary to mark that the exposure of hypocalcemia is considered a bad predictive sign of heavy passing of acute pancreatitis.

Ultrasonic examination of gall-bladder and pancreas often specifies the increase of their sizes, bulge of walls and presence or absence of concrement of gall-bladder and general bilious duct.

Computer tomography enables to describe in details the changes in pancreas and surrounding organs.

At *sciagraphy survey* of organs of abdominal cavity gives a possibility to expose the unfolded “horseshoe” of duodenum, pneumatization, expansion of transverse colon (*the Gobia's symptom*). On the 1st stage of diagnostics in the plan of differential diagnosis of acute destructive pancreatitis with other diseases of abdominal cavity, diagnostics of distribution of destructive damaging of different parts of pancreas and estimation of distribution of parapancreatitis is possible only by the method of computer tomography which depending on clinico-laboratory signs and weight of passing is needed to apply in a different period, and sometimes a few times in dynamics with interval of 4–5 days.

Laparoscopy and laparocentesis are often used for a doubtful diagnosis or necessity of taking away the exudation of abdominal cavity for biochemical or bacteriological examination.

Retrograde endoscopic cholangiopancreatography is used in case of mechanical icterus and suspicion of choledocholithiasis. The last methods are invasive and can if it is necessary transform from diagnostic to manipulation treatments: laparoscopic draining of abdominal cavity at pancreatogenic peritonitis and endoscopic papillotomy at choledocholithiasis and biliary pancreatitis.

Variants of clinical passing and complications

Clinical passing of disease can be abortive, slowly or quickly progressive. At *abortive passing* the process is limited to acute edema of pancreas with convalescence in 7–10 days.

Rapid progress is characteristic for pancreatonecrosis. In patients expressed toxemia, impregnation by exudation of retroperitoneal cellulose and development of fermentative hemorrhagic peritonitis can be seen. Strengthening of stomachache, continuous vomiting, proof paresis of intestine, positive symptoms of irritation of peritoneum and growth of hemodynamic violations are the clinical signs of necrosis of pancreas.

There is a formation of parapancreatic infiltrate at *slow progress*.

Among early complications of acute pancreatitis shock, peritonitis and acute cardiac, pulmonary, hepatic and kidney insufficiency can be distinguished.

Before later complications it is needed to deliver the abscesses of pancreas, subdiaphragmatic, interintestinal abscesses, pyogenic abscess omentum bag, phlegmons of retroperitoneal space and erosive bleeding.

In future formations of pseudocysts, fistula of pancreas, intestinal fistula and development of saccharine diabetes are possible.

Diagnosis program

1. Anamnesis and physical methods of inspection.
2. General analysis of blood and urine.
3. Biochemical blood test (amylase, bilirubin, sugar).
4. Analysis of urine on diastase.
5. Sonography.
6. Computer tomography.
7. Cholecystocholangiography.
8. Endoscopic retrograde cholangiopancreatography.
9. Laparoscopy.
10. Laparocentesis.

Differential diagnostics

Acute pancreatitis needs to be differentiated with the row of acute diseases of organs of abdominal cavity.

Acute mechanical intestinal obstruction. In patients with this pathology pain is of the alternated character and is accompanied by nausea, vomiting, delay of gases and emptying. It is possible to see the Klotz bowls on the sciagram survey of organs of abdominal cavity.

Acute cholecystitis runs with characteristic localization of pain and muscular defense, with presence of increased, painful gall-bladder or infiltrate in right hypochondrium. Often acute (especially lately) pancreatitis develops on the background of gallstone disease (biliary pancreatitis).

Thrombosis or embolism of mesenteric vessels. Both for pancreatitis and for the thrombosis of mesenteric vessels great pain at soft abdomen (absence of defense muscles of front abdominal wall), that precedes to development of peritonitis, is inherent. Yet from the beginning the disease gains heavy character of passing. In anamnesis in such

patients a heart disease or heart attack of myocardium rheumatic is met. As a result of gangrene of intestine, the symptoms of peritonitis appear very quickly and intoxication grows. The fragments of mucus shell are found in flushing waters of intestine at the detailed examination, which have the appearance of "meat flushing".

A perforated gastric and duodenum ulcer is distinguished by the presence of dagger pain, defense of abdominal wall, ulcerous anamnesis.

Tactics and choice of treatment method

The conservative method is considered the basic one for treatment of acute pancreatitis, but in connection with that unsuccessful conservative treatment of patients with acute pancreatitis can often put a question about the necessity of operation, therefore patients must be in permanent surgical establishment. Thus acute pancreatitis with heavy passing is necessary to be treated under the conditions of separation of intensive therapy.

Before conservative treatment hunger, bed rest, fight against pain and enzymic toxemia, conducting of acid-base state, prophylaxis of festering infection and acute ulcers of digestive duct are to be entered.

Patient's stomach is washed by cold soda solution and a cold on an epigastric area and left hypochondrium is used. Medicinal therapy is prescribed also: spasmolytics (papaverine, platyphyllin, no-shparum, baralgine, atropine); inhibitor of protease (contrical, trasilol, gordox, antagosan); cytostatic agent (5-fluorouracil, florafur). Positive action of inhibitor of protease is marked only in the first days of disease which are subject to conditioned application of large doses. Antibiotics of wide spectrum of action: a) tienam, which most effective in the prophylaxis of festering pancreatitis, as is selected by pancreatic juice; b) cephalosporins (kefzol, cefazoline); c) cefamizine (mefoxine).

Disintoxication therapy is conducted also (5 % but 10 % solutions of glucose, hemodes, reopolyhlukine, polyhlukine, plasma of blood, only from 3 to 5 liters on days, in accordance with a necessity).

For the improvement of rheological properties of blood heparine is prescribed (5 000 ODES every 4 hours).

If patients have the expressed pain syndrome and phenomena of general intoxication during all pain period plus 48 hours (by Bakulev), hunger is used. Such mode lasts on the average of 2–4 days. The parenteral feed of albuminous hydrolyzate is thus conducted, by the mixtures of amino acid and fatty emulsion. Alkaline water of to 1–2 l. and albuminous-carbohydrate diet are also appointed. Infusion therapy is complemented by plasma, by albumen, hemodes, reopolyhlukine. The improvements of microcirculation in pancreas are achieved due to introduction of reopolyhlukine, komplamine, trental and heparin 5000 ODES 6 times per days under the control the indexes of the coagulation system of blood. Anticholinergic drug (sulfate of atropine, methacin, platyphyllin), H₂-histamin blocker (cimetidine, ranisan, ranitidine, famotidine, omeprazol) are also applied. For the removal of pain: 1) sulfate of the atropine 0,1 % — 1 ml + promedol 2 % — 1 ml + papaverine 2 % — 2 ml + analgin 50 % — 2 ml; 2) isotonic solution of chloride of sodium — 500 ml + baralgine — 5 ml + diphenhydramine hydrochloride 1 % — 1 ml + papaverine 2 % — 2 ml + magnesium the sulfate 25 % — 5 ml + ascorbic acid — 5 ml + lipoic acid 0,5 % — 2 ml + novocaine 0,5 % — 10 ml. are used. From the first days by a nasogastral probe the permanent aspiration of gastric maintenance is conducted also. The Motility function of gastro-intestinal highway gets better at application of cerucal or primperane. With the same purpose forced diuresis (maninil, furosemide, aminophylline) is used on the background of intravenous introduction of plenty of liquid.

At uneffective conservative treatment of patients with acute pancreatitis of middle weight and heavy form it is expedient to apply surgical treatment.

Surgical treatment is carried out for patients with biliary pancreatitis (for a day long from the beginning of disease) in combination with the destructive forms of cholecystitis, at complications of acute pancreatitis by peritonitis, abscess of omentum bag or phlegmon of retroperitoneal cellulose.

Overhead-middle laparotomy, which allows to estimate the state of pancreas, bilious ways and other organs of abdominal cavity, is the best access in this situation. In case of destructive pancreatitis the possible use of lumbar laparotomy from left to right hypochondrium through a mesogastric area is useful.

Cholecystectomy is executed at calculous cholecystitis, phlegmonous inflammation of walls of gall-bladder and biliary pancreatitis. If there are more than 0,9 cm at expansion of choledochus, presence of concrement, ointment-like bile in it, increase of concentration of bilirubin in the whey of blood over 21 mmol/L, choledocholithotomy is complemented by external draining of choledochus. Information of lithiasis of general bilious duct is absent, cholecystectomy in patients with acute pancreatitis is complemented by external draining of choledochus, better by Pikovskyy method (through stump of cystic duct).

Transduodenal sphincteroplasty (Fig.6) is shown at fixed concrement of large duodenal papilla, if they are diagnosed intraoperative, and also in the cases of papillotomy with extraction of concrement when there is no possibility to execute endoscopic operation.

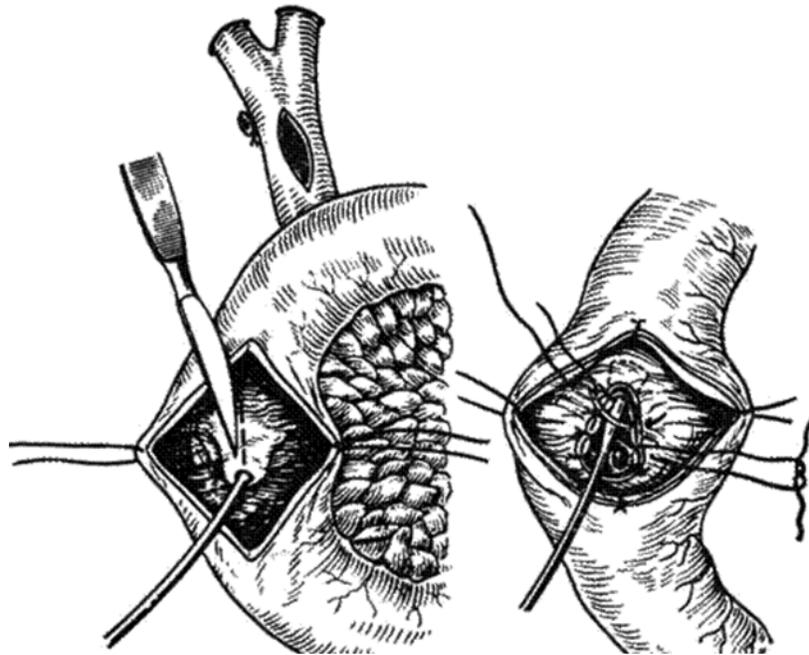


Fig.6. Transduodenal papillotomy with sphincteroplasty

Omentopancreatopexy. After laparotomy and cutting of gastro-colon and gastro-pancreatic ligament mobile part of large omentum through opening in gastro-colon ligament is conducted and fixed by separate stitches to the peritoneum along the overhead and lower edges of pancreas. Such operation needs to be considered rational at the expressed edema of pancreas and presence of necrosis in it.

Abdominisation of pancreas. A cellulose round pancreas (along the lower and overhead edges of body and tail) is infiltrated by solution of novocaine, after it parietal peritoneum is cut. Under the body and tail glands free end of omentum is conducted and is bundled by a gland. This operation is able to warn the hit of enzymes and products of disintegration in retroperitoneal space.

Sequestrectomy is deleting of necrosis part of gland within the limits of nonviable tissue. Operation is executed in a dull way.

Necrectomy (deleting of necrosis part of gland within the limits of healthy tissue) is executed by an acute way: tissue of gland is cut on verge of necrosis and bleeding vessels are carefully banded.

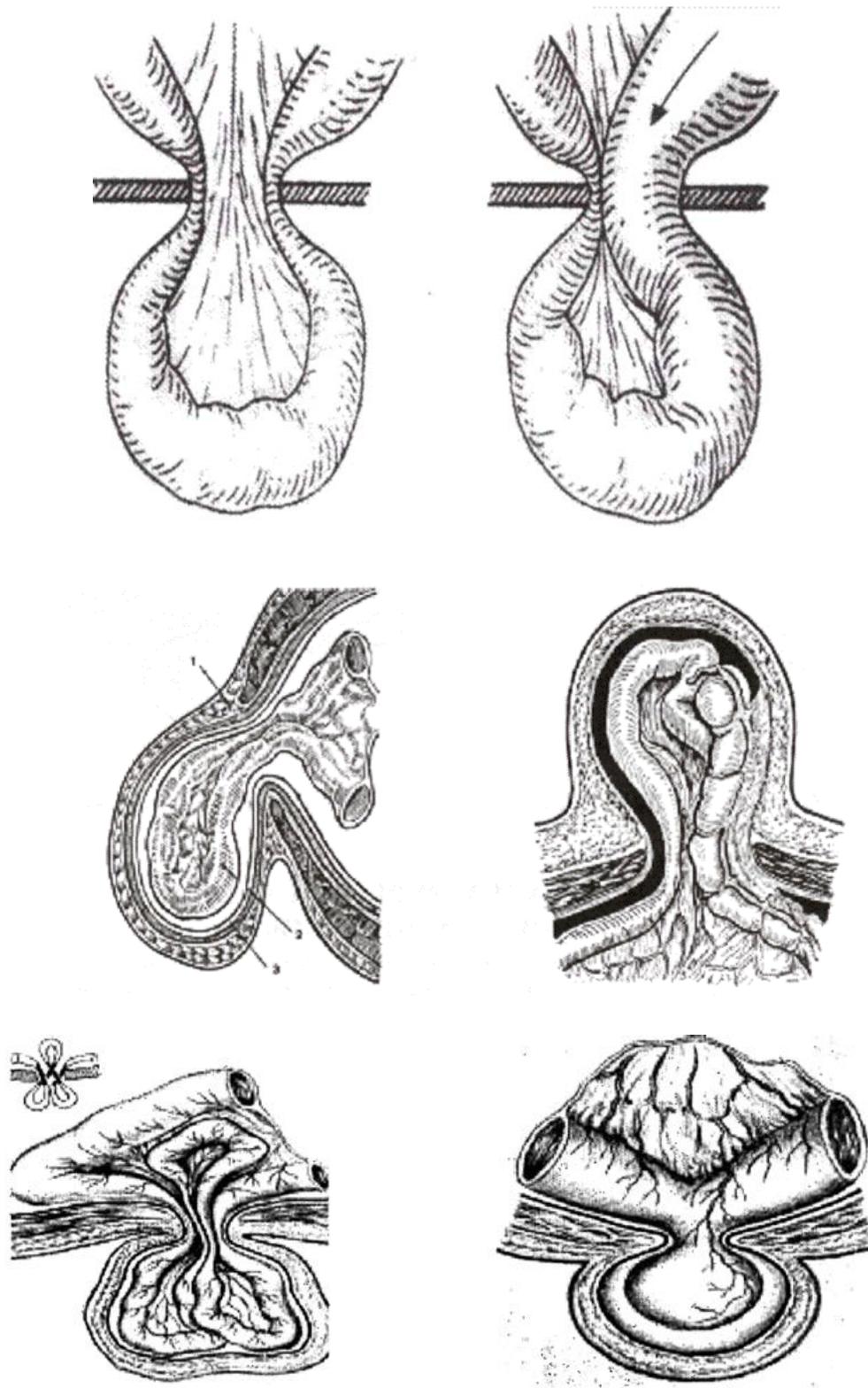
The resection of pancreas is deleting the part of organ with its transversal cutting within the limits of the unchanged (ad oculus) tissue of gland. The resections of tail and body of pancreas are distinguished.

Pancreatectomy is a complete deleting of pancreas. Operative treatment is applied infrequently. After the resection of pancreas adequate draining of its bed is very responsible.

The prognosis of disease depends on character of morphological changes of parapancreatic to the cellulose in pancreas. The more difficult destructive changes, the worst the prognosis.

INCARCERATED HERNIA

Incarcerated hernia is sudden pressing of hernia contents in a hernia orifice. Incarceration is the most frequent and most dangerous complication of hernia diseases.



Pic. Types of incarcerations

Etiology and pathogenesis

Depending on mechanism, the elastic and fecal incarceration is distinguished. At the *elastic incarceration*, after increasing intraabdominal pressure, one or a few organs relocated from an abdominal cavity to the hernia sack, where it is compressed with following ischemia and necrosis in the area of hernia gate. At the fecal incarceration in the intestinal loop which is in a hernia sack, plenty of excrement passed quickly. Proximal part of loop is

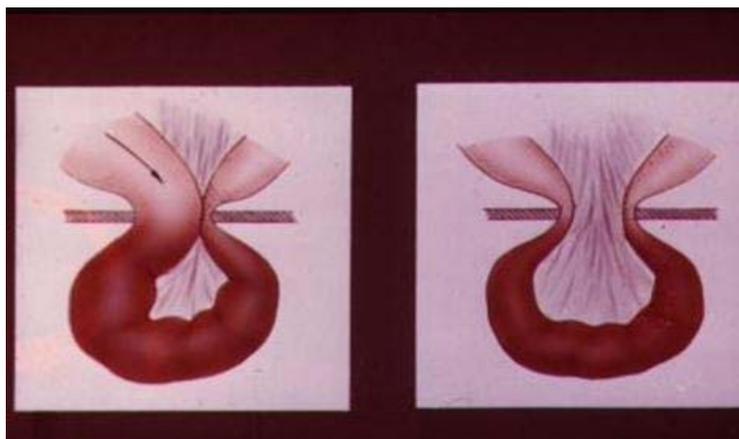
overfilled, and distal is compressed in a hernia gate. So, arose its strangulation, as well as at the elastic incarceration.

Most often the loop of bowel is incarcerated. Thus three parts are distinguished in it: proximal, distal loop, central part. The heaviest pathological changes during incarceration takes place in a strangulated furrow in the central part of the incarcerated bowel.

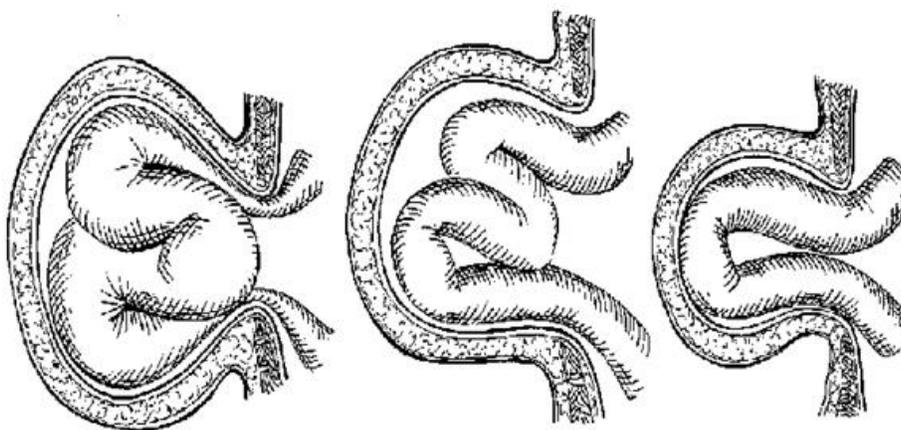
Pathomorphology

At incarcerated hernia an important role has all internal rings: inguinal, umbilical, “weak places” in a diaphragm, orifice of the omental bursa, numeral and “variant” folds of peritoneum.

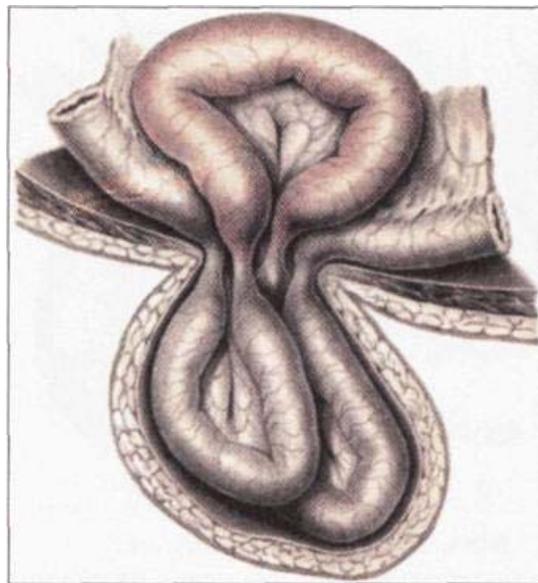
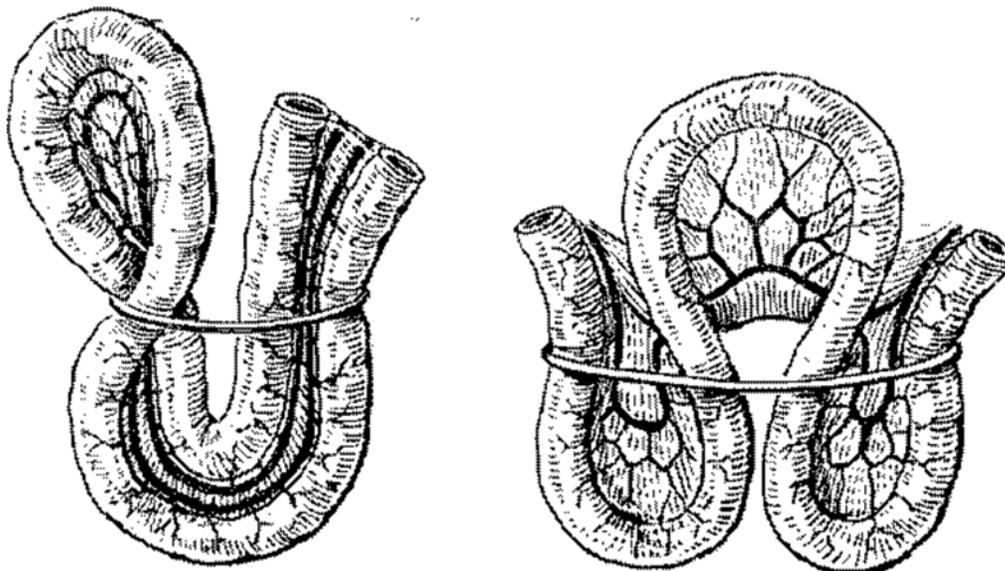
In the place of compressing of the bowels and mesentery, as a rule, it is possible to find a strangulation furrow. If circulation of blood changes, the wall of bowel cyanotic, with hemorrhages and necrosis of a different size. The loop of bowel which is located proximally the places of strangulation are extended, and distal loop mainly without changes.



Pic. Mechanizm of the incarceration.



Pic. Scheme of the incarceration.



Pic. Retrograde incarceration.

Classification of the incarcerated hernia

The incarcerated hernia is divided into the complete and incomplete. The other types of incarceration is partial (the Richter's hernia) and retrograde. The incarcerated hernia can be without the destructive changes of hernia contents and with the phlegmon of hernia sack.

Clinical management

The clinic of the incarcerated hernia depends on pulling in organ, character and duration of jamming. The clinical signs of the incarcerated hernia can be divided into three groups: 1) local changes; 2) common signs; 3) complication. From the most characteristic signs of local changes the most common is sharp pain, irreducible hernia, tension of hernia sack that and negative symptom of the "cough push".

Pain sometimes is so intensive that causes pain shock. In the case of intestinal obstruction a pain is attack-like. In case of occurring of peritonitis pain changes the character and becomes permanent.

It is necessary to mean that tensions of hernia sack and incarceration of the hernia, as signs of jamming, lose it value, if hernia was irreducible.

From other side, the isolation of hernia sack from an abdominal region during jamming is the reason of the *negative symptom of the "cough push"*.

The common signs at the incarcerated hernia has phase character. *Nausea* and *vomits* during first hours of disease has reflex reason, and on 2nd and 3rd days has toxic reason, that is consequence of antiperistaltic and reflux of intestinal contents to the stomach.

The *temperature of body* at first time is normal, and than rises, but usually low grade fever.

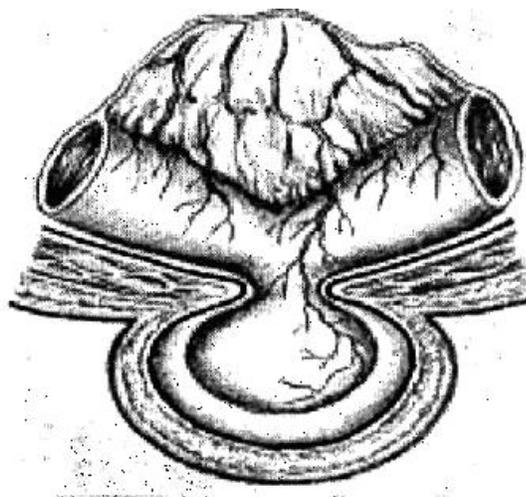
The clinic of acute intestinal obstruction and peritonitis develops at the protracted jamming of intestine. The phlegmon of hernia sack can develop in the area of the hernia swelling.

Clinical variants and complications

There are different forms of incarceration of internal organs, and accordingly — different clinical variants.

Retrograde incarceration. In such cases a hernia sack contains no less than two loops of intestine. But these loops are damaged less, than loop which is in an abdominal cavity. At this variant of jamming peritonitis arose quicker. So, surgeon during operation must always remember about the necessity of careful revision of the incarcerated loops of bowel.

Parietal incarceration (the Richter's hernia). Unlike retrograde, which has wide hernia gate, a similar pathology arises in case of narrow hernia gate. In a hernia sack in such patients located part of bowel wall, opposite it mesentery edge.



Thus, as a rule, patency of bowel is not broken. Such variant of jamming is dangerous, because there are no evident clinical signs or some of them are quite absent and intestinal patency almost is always present. Necrosis of bowel wall comes quickly and in 2-3 days the perforation with subsequent development of peritonitis begins after jamming.

The Littre's hernia. Jamming of Meckel's diverticulum can come at oblique inguinal hernia. Clinical signs of this pathology reminds the parietal incarceration. Sometimes is possible to palpate dense, short, thick tension bar in a hernia sack.

Incarceration at sliding hernia. It is observed at patients with inguinal hernia. At sliding hernia of colon, as a rule, there is the fecal incarceration. A bowel is the external wall of hernia sack in such cases. About it is necessary to remember during opening of hernia sack. Jammings of urinary bladder meet enough rarely, mainly at older-men at oblique sliding hernia of inguinal channel. It is necessary to ask before the operation, whether a patient had disorders of urination before jamming. Frequent urges, or, opposite, the reflex delay of urination is arose at the beginning of jamming already, and in urine expose macro- or microhematuria. If during operation at opening of hernia sack it medial wall has dense, doughy consistency, it is an urinary bladder.



At the incarcerated hernia the contents of hernia sack can be also omentum, appendages of colon, internal female genital organs. Sometimes combination of the incarcerated inguinal hernia with different pathological changes of testicle and deferent duct can take place.

Rough manual reduction of the incarcerated hernia can bring to pseudoreduction. Then the local signs of the incarcerated hernia disappear, and jamming of organs and its consequences is kept. There are five variants of the pseudoreduction: 1) at multicompartiment hernia sacks there is the possible moving of strangulated organs from one chamber in other, that located more deep or in a preperitoneal adipose tissue; 2) separation and reduction of hernia sack together with it content in an abdominal cavity or in a preperitoneal adipose tissue; 3) abruption of the neck from other part of hernia sack and reduction it together with content in an abdominal cavity or in a preperitoneal adipose tissue; 4) abruption of the neck from a hernia sack and from a parietal peritoneum with reduction of the incarcerated organs in an abdominal cavity; 5) break of the incarcerated bowel at the rough reduction of hernia.

Untimely operative at the incarcerated hernia, usually, is complicated by the gangrene of bowel, peritonitis or phlegmon of hernia sack. Such complications considerably worsen clinical status of patient and require other surgical tactic.

Diagnosis program

1. Anamnesis examination.
2. Physical examination.
3. Blood analysis and urine analysis.
4. Digital investigation of the rectum.
5. Survey X-Ray of abdominal cavity organs.

Differential diagnostics

As experience shows, the incarcerated hernia we should differentiate with irreducible, which as a rule, is not tense, positive symptom of the "cough push", painful on palpation. A patient complained for long duration of the disease. The incarcerated hernia needs to be differentiated with coprostasis. In such patients disorder of bowel loop patency, that is in a hernia sack, creates accumulation of excrement. Coprostasis mostly found at fecal hernia in older people, that suffer from intractable constipation. Clinically it develops gradually and slowly. The hernia swelling almost not painfully, some tense, a positive symptom of the "cough push". Beginning of coprostasis is unconnected with physical tension. Application of cleansing siphon enema washed of excrement and liquidated coprostasis.

Unreal jamming of hernia. In clinical practice there are often such situation, when during the acute surgical diseases of organs of abdominal cavity free external abdominal hernia becomes irreducible, painfully and tense, and looks like incarcerated. This is the unreal jamming of hernia, which can be observed at the acute surgical diseases of organs of abdominal cavity, ascites. During examination of such patients it is necessary to remember, that at the unreal jamming abdominal pain, vomiting, worsening of the general condition and signs of the intestine obstruction come earlier, than changes in a hernia sack.

In addition, during the operation in patients with incarcerated hernia, it is needed to make sure, whether there

is a strangulation furrow, or organ, that is in a hernia sack, fixed in a hernia gate. When these signs are absent, it is possible to consider that jamming is unreal.

The incarcerated femoral hernia must be differentiated with inguinal lymphadenitis, by varicose expansion of large hypodermic vein, varicose knot and their thrombophlebitis, tumor and abscess.

From such pathology without surgical procedure it is possible to differentiate only varicose expansion of veins (varicose knot), for which the positive Valsalv test — at horizontal position of patient with the leg heaved up a knot is empty.

The incarcerated inguinal hernia needs to be differentiated also with hydrocele and orchiepididymitis, cyst of deferent duct, cyst of round ligamentum of uterus, bartholinitis. Patients, who have with such diseases, a process usually does not spread higher external ring of inguinal channel. Also, absence of testicle in scrotum can be cryptorchism sign.

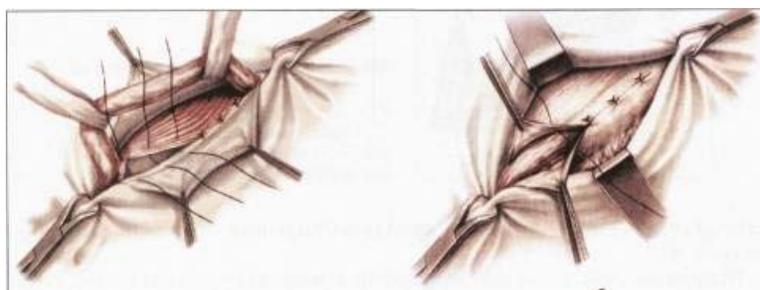
The common clinical signs of the internal incarcerated hernia is abdominal pain and symptoms of the intestinal obstruction. A final diagnosis is set during the operation.

Tactics and choice of treatment method

The incarcerated hernia, regardless of time of its origin, localization and age of patient, must be operated on. However, if a patient is hospitalized already with the expressed symptoms of intestinal obstruction, than should be preoperative treatment. Such conservative therapy must be brief (1–1,5 hours), but always actively directed for correction of violations of metabolism and prophylaxis of possible pulmonary and cardiovascular complications. It is necessary also to conduct evacuation of the gastric contents and other preparatory procedures.

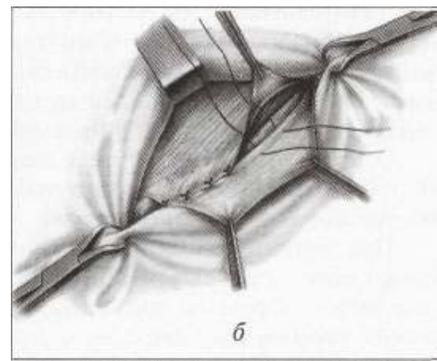
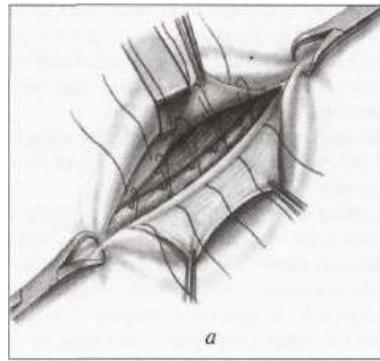
Patient with reduced hernia must be hospitalized and observed during 1–2 days. If a abdominal pain is contained or is growing, the signs of peritonitis and intoxication appear, than performed urgent laparotomy and necessary operation. If the symptoms of “acute” abdomen are not present, a patient examined and prepared for elective operation. On oblique inguinal hernias, we should strengthen anterior wall of the inguinal channel. On direct inguinal hernias, we should strengthen posterior wall of the inguinal channel. On recurrence hernias - we should strengthen anterior and posterior wall of the inguinal channel.

Bassini repair. After extraction of the hernia sac, we are taking spermatic duct on holders. Between the borders of transverse muscle, internal oblique muscle, transverse fascia and inguinal ligament interrupted sutures placed. Except that, couples sutures placed between border of abdominal rectus muscle sheath and pubic bone periosteum.



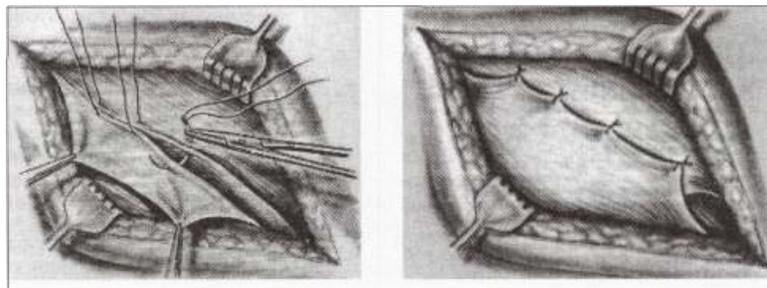
In such way, inguinal space closed and posterior wall strengthened. Spermatic duct placed on the new-formed posterior wall of the inguinal channel. Over the spermatic duct aponeurosis restored by interrupted sutures.

Girard in such kind of the operations propose to attach the edges of the internal oblique muscle and transversal muscle to the inguinal ligament over the spermatic duct. The aponeurosis of the external oblique muscle sutured by second layer of the suture. Excess of the aponeurosis is fixed to the muscle in the form of duplication.



Spasokukotskyy proposed to catch the edges of the internal oblique muscle and transversal muscle with aponeurosis of the external oblique muscles by single-layer interrupted suture.

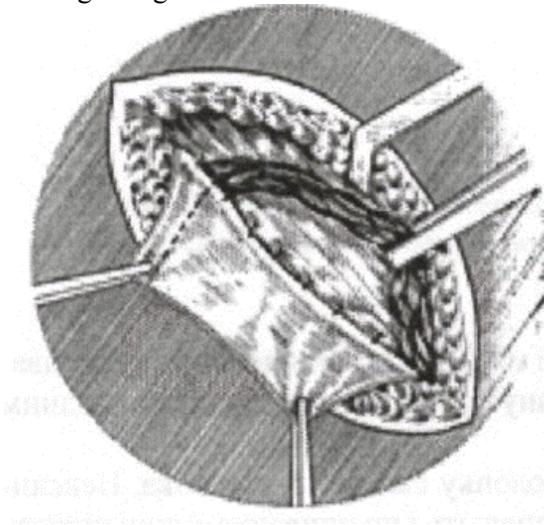
Martynov proposed the fixation to the Poupart's ligament only internal edge of the external oblique muscle aponeurosis without muscles. External edge of the aponeurosis sutured over internal in the form of duplication.



Kimbarovskyy, based on the principles of joining similar tissues, proposed special suture: Sutures placed on 1 cm from the edge of the external oblique abdominal muscle aponeurosis, grasped the part of the internal oblique and transversal muscle. After that, aponeurosis is sutured one more time from behind to the front and attached to the Poupart's ligament.

Kukudganov proposed to restore back wall of inguinal interval. Sutures are placed between the Couper's ligamentum, vagina of direct abdominal muscle and aponeurosis of the transversal muscle.

Postempyskyy proposed the deaf closing of inguinal interval with the lateral moving of spermatic duct.

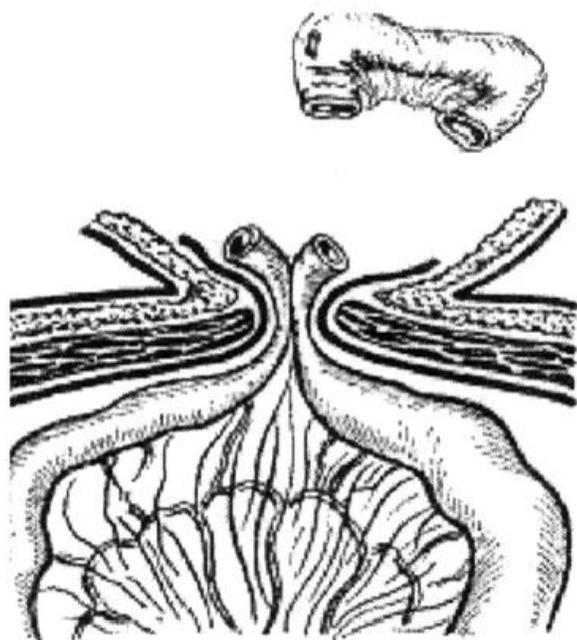


The plastic narrowing of internal inguinal ring of to 0,8 cm is the important moment of this modification. On occasion, when internal and external inguinal rings are in one plane, a spermatic duct is displaced inlateral direction by transversal incision of the oblique and transversal muscles. Then edge of the vagina of direct muscle and aponeurosis of the internal and transversal muscles is fixed to the Couper's ligament.

Operation at the incarcerated hernia is executed under the general anesthesia. A hernia sack is selected from surrounding tissue, cut it in the area of bottom and remove hernia water, defining its character and sending to

bacterial inoculation. Retaining the damaged organs, a strangulated ring is cut. It is necessary to remember, that at the incarcerated femoral hernia ring cut up and some medially, because a femoral vein passes from a lateral side.

If a bowel is contents of hernia sack, we must estimate its viability. Remembering about possibility of the retrograde jamming, special attention must be paid to the state of strangulation furrow. About viability of the bowels testify: 1) renewal of its normal color; 2) presence or renewal of peristalsis; 3) renewal of pulsation of vessels of mesentery and bowel. If there are the certain doubting, a bowel is dipped on a holder in an abdominal cavity and in 15–20 minutes it is examined repeatedly. If one of the resulted signs of viability is absent even, it is necessary to conduct the resection of bowel. The resection is executed, receded from the strangulation furrow on a proximal loop 30–40 cm and distal — 15–20 cm. Anastomosis between proximal and distal loops it is better to impose “end-to-end”. The plastic of hernia gate are conducted depending on indications after one of the surgical methods.



When the necrosis elements of omentum or fatty pendants of colon are contents of hernia sack, they must be removed within the limits of healthy tissue.

There can be necrosis of wall of colon or urinary bladder at sliding hernia. In such cases it is needed to be limited to the minimum surgical procedure: to dip a necrosis area by sutures inside the bowel or use it for forming of colostomy or epicystostomy. These are the best to conclude operation.

In similar situations at the incarcerated parietal hernia in most patients it is possible to be limited to peritonization of displaced area of wall of bowel. If after the peritonization there is the threat of narrowing of bowel or necrosis goes outside of the strangulation furrow, it is needed to conduct the resection of bowel.

Because of insufficient blood flow of Meckel's diverticulum and, permanent threat of it necrosis, at patients with Littre's hernia it resection must be performed.

At the phlegmon of hernia sack operation is begun with herniotomy. If the incarcerated organ is damaged by necrosis, and in a hernia sack present pus, than there is a necessity for surgeon to perform laparotomy. After that incarcerated organ resected within the limits of healthy tissue (in the generally accepted limits — 40 cm of proximal loop and 20 cm distal) and impose anastomosis. An abdominal cavity is sewn up. Incarcerated loops of bowel, together with it blind ends which located in an abdominal cavity, removed through a hernia sack, a peritoneum is sutured, the hernia sack is drained, the plastic of hernia gate are not performed. Skin is sewn up by widely spaced sutures.

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HEMORRHOIDS. RECTAL FISSURES. PARAPROCTITIS. RECTAL FISTULAS. EPITHELIAL PARACOCCIDIOIDAL CANALS

EMBRYOLOGY AND ANATOMY

Embryology

The embryonic gastrointestinal tract begins developing during the fourth week of gestation. The primitive gut is derived from the endoderm and divided into three segments: *foregut*, *midgut*, and *hindgut*. Both *midgut* and *hindgut* contribute to the colon, rectum, and anus.

The *midgut* develops into the small intestine, ascending colon, and proximal transverse colon, and receives blood supply from the superior mesenteric artery. During the sixth week of gestation, the midgut herniates out of the abdominal cavity, and then rotates 270 degrees counterclockwise around the superior mesenteric artery to return to its final position inside the abdominal cavity during the tenth week of gestation.

The *hindgut* develops into the distal transverse colon, descending colon, rectum, and proximal anus, all of which receive their blood supply from the inferior mesenteric artery. During the sixth week of gestation, the distal-most end of the hindgut, the *cloaca*, is divided by the urorectal septum into the urogenital sinus and the rectum.

The distal anal canal is derived from ectoderm and receives its blood supply from the internal pudendal artery. The dentate line divides the endodermal hindgut from the ectodermal distal anal canal.

Anatomy

The large intestine extends from the ileocecal valve to the anus. It is divided anatomically and functionally into the *colon*, *rectum*, and *anal canal*. The wall of the colon and rectum comprise five distinct layers: mucosa, submucosa, inner circular muscle, outer longitudinal muscle, and serosa. In the colon, the outer longitudinal muscle is separated into three *teniae coli*, which converge proximally at the appendix and distally at the rectum, where the outer longitudinal muscle layer is circumferential. In the distal rectum, the inner smooth-muscle layer coalesces to form the internal anal sphincter. The intraperitoneal colon and proximal one third of the rectum are covered by serosa; the mid and lower rectum lack serosa.

Colon Landmarks

The colon begins at the junction of the terminal ileum and cecum and extends 3 to 5 feet to the

rectum. The rectosigmoid junction is found at approximately the level of the sacral promontory and is arbitrarily described as the point at which the three *teniae coli* coalesce to form the outer longitudinal smooth muscle layer of the rectum. The *cecum* is the widest diameter portion of the colon (normally 7.5 to 8.5 cm) and has the thinnest muscular wall. As a result, the cecum is most vulnerable to perforation and least vulnerable to obstruction. The ascending colon is usually fixed to the retroperitoneum. The hepatic flexure marks the transition to the transverse colon. The intraperitoneal transverse colon is relatively mobile, but is tethered by the gastrocolic ligament and colonic mesentery. The greater omentum is attached to the anterior/superior edge of the transverse colon. These attachments explain the characteristic triangular appearance of the transverse colon observed during colonoscopy. The splenic flexure marks the transition from the transverse colon to the descending colon. The attachments between the splenic flexure and the spleen (the lienocolic ligament) can be short and dense, making mobilization of this flexure during colectomy challenging. The descending colon is relatively fixed to the retroperitoneum. The sigmoid colon is the narrowest part of the large intestine and is extremely mobile. Although the sigmoid colon is usually located in the left lower quadrant, redundancy and mobility can result in a portion of the sigmoid colon residing in the right lower quadrant. This mobility explains why volvulus is most common in the sigmoid colon and why diseases affecting the sigmoid colon, such as diverticulitis, may occasionally present as right-sided abdominal pain. The narrow caliber of the sigmoid colon makes this segment of the large intestine the most vulnerable to obstruction.

Colon Vascular Supply

The arterial supply to the colon is highly variable (Fig. 28-1). In general, the *superior mesenteric artery* branches into the *ileocolic artery* (absent in up to 20% of people), which supplies blood flow to the terminal ileum and proximal ascending colon, the *right colic artery*, which supplies the ascending colon, and the *middle colic artery*, which supplies the transverse colon. The *inferior mesenteric artery* branches into the *left colic artery*, which supplies the descending colon, several *sigmoidal branches*, which supply the sigmoid colon, and the *superior rectal artery*, which supplies the proximal rectum. The terminal branches of each artery form anastomoses with the terminal branches of the adjacent artery and communicate via the *marginal artery of Drummond*. This arcade is complete in only 15 to 20% of people.

Colon Lymphatic Drainage

The lymphatic drainage of the colon originates in a network of lymphatics in the muscularis mucosa. Lymphatic vessels and lymph nodes follow the regional arteries. Lymph nodes are found on the bowel wall (epicolic), along the inner margin of the bowel adjacent to the arterial arcades (paracolic), around the named mesenteric vessels (intermediate), and at the origin of the superior and inferior mesenteric arteries (main). The *sentinel lymph nodes* are the first one to four lymph nodes to drain a specific segment of the colon, and are thought to be the first site of metastasis in colon cancer. The utility of sentinel lymph node dissection and analysis in colon cancer remains controversial.

Colon Nerve Supply

The colon is innervated by both *sympathetic* (inhibitory) and *parasympathetic* (stimulatory) nerves, which parallel the course of the arteries. Sympathetic nerves arise from T6-T12 and L1-L3. The parasympathetic innervation to the right and transverse colon is from the vagus nerve; the parasympathetic nerves to the left colon arise from sacral nerves S2-S4 to form the *nervi erigentes*.

Anorectal Landmarks

The rectum is approximately 12 to 15 cm in length. Three distinct submucosal folds, the *valves of Houston*, extend into the rectal lumen. Posteriorly, the *preseccal fascia* separates the rectum from the presacral venous plexus and the pelvic nerves. At S4, the *rectosacral fascia (Waldeyer's fascia)* extends forward and downward and attaches to the fascia propria at the anorectal junction. Anteriorly, *Denonvilliers' fascia* separates the rectum from the prostate and seminal vesicles in men and from the vagina in women. The *lateral ligaments* support the lower rectum. The surgical anal canal measures 2 to 4 cm in length and is generally longer in men than in women. It begins at the anorectal junction and terminates at the anal verge. The *dentate* or *pectinate line* marks the transition point between columnar rectal mucosa and squamous anoderm. The 1 to 2 cm of mucosa just proximal to the dentate line shares histologic characteristics of columnar, cuboidal, and squamous epithelium and is referred to as the *anal transition zone*. The dentate line is surrounded by longitudinal mucosal folds, known as the *columns of Morgagni*, into which the anal crypts empty. These crypts are the source of cryptoglandular abscesses

In the distal rectum, the inner smooth muscle is thickened and comprises the *internal anal sphincter* that is surrounded by the *subcutaneous, superficial, and deep external sphincter*. The *deep external anal sphincter* is an extension of the *puborectalis muscle*. The *puborectalis, iliococcygeus, and pubococcygeus muscles* form the *levator ani muscle* of the pelvic floor.

Anorectal Vascular Supply

The *superior rectal artery* arises from the terminal branch of the inferior mesenteric artery and supplies the upper rectum. The *middle rectal artery* arises from the internal iliac; the presence and size of these arteries are highly variable. The *inferior rectal artery* arises from the internal pudendal artery, which is a branch of the internal iliac artery. A rich network of collaterals connects the terminal arterioles of each of these arteries, thus making the rectum relatively resistant to ischemia

The venous drainage of the rectum parallels the arterial supply. The *superior rectal vein* drains into the portal system via the inferior mesenteric vein. The *middle rectal vein* drains into the internal iliac vein. The *inferior rectal vein* drains into the internal pudendal vein, and subsequently into the internal iliac vein. A submucosal plexus deep to the columns of Morgagni forms the *hemorrhoidal plexus* and drains into all three veins.

Anorectal Lymphatic Drainage

Lymphatic drainage of the rectum parallels the vascular supply. Lymphatic channels in the upper and middle rectum drain superiorly into the inferior mesenteric lymph nodes. Lymphatic channels in the lower rectum drain both superiorly into the inferior mesenteric lymph nodes and laterally into the internal iliac lymph nodes. The anal canal has a more complex pattern of lymphatic drainage. Proximal to the dentate line, lymph drains into both the inferior mesenteric lymph nodes and the internal iliac lymph nodes. Distal to the dentate line, lymph primarily drains into the inguinal lymph nodes, but can also drain into the inferior mesenteric lymph nodes and internal iliac lymph nodes.

Anorectal Nerve Supply

Both sympathetic and parasympathetic nerves innervate the anorectum. Sympathetic nerve fibers are derived from L1-L3 and join the preaortic plexus. The preaortic nerve fibers then extend below the aorta to form the *hypogastric plexus*, which subsequently joins the parasympathetic fibers to form the pelvic plexus. Parasympathetic nerve fibers are known as the *nervi erigentes* and originate from S2-S4. These fibers join the sympathetic fibers to form the pelvic plexus. Sympathetic and parasympathetic fibers then supply the anorectum and adjacent urogenital organs.

The internal anal sphincter is innervated by sympathetic and parasympathetic nerve fibers; both types of fibers inhibit sphincter contraction. The external anal sphincter and puborectalis muscles are innervated by the *inferior rectal branch* of the *internal pudendal nerve*. The levator ani receives innervation from both the *internal pudendal nerve* and direct branches of S3 to S5. Sensory innervation to the anal canal is provided by the *inferior rectal branch* of the *pudendal nerve*. While the rectum is relatively insensate, the anal canal below the dentate line is sensate.

Congenital Anomalies

Perturbation of the embryologic development of the midgut and hindgut may result in anatomic abnormalities of the colon, rectum, and anus. Failure of the midgut to rotate and return to the abdominal cavity during the tenth week of gestation results in varying degrees of intestinal malrotation and colonic nonfixation. Failure of canalization of the primitive gut can result in colonic duplication. Incomplete descent of the urogenital septum may result in imperforate anus and associated fistulas to the genitourinary tract. Many infants with congenital anomalies of the hindgut have associated abnormalities in the genitourinary tract.

Normal Physiology

Fluid and Electrolyte Exchanges

Water, Sodium, Potassium, Chloride, Bicarbonate, and Ammonia

The colon is a major site for water absorption and electrolyte exchange. Approximately 90% of the water contained in ileal fluid is absorbed in the colon (1000 to 2000 mL/d), and up to 5000 mL of fluid can be absorbed daily. Sodium is absorbed actively via a Na-K ATPase. The colon can absorb up to 400 mEq of sodium per day. Water accompanies the transported sodium and is absorbed passively along an osmotic gradient. Potassium is actively secreted into the colonic lumen and absorbed by passive diffusion. Chloride is absorbed actively via a chloride–bicarbonate exchange.

Bacterial degradation of protein and urea produces ammonia. Ammonia is subsequently absorbed and transported to the liver. Absorption of ammonia depends in part upon intraluminal pH. A decrease in colonic bacteria (e.g., broad spectrum antibiotic usage) and/or a decrease in intraluminal pH (e.g., lactulose administration) will decrease ammonia absorption.

Short-Chain Fatty Acids

Short-chain fatty acids (acetate, butyrate, and propionate) are produced by bacterial fermentation of dietary carbohydrates. Short-chain fatty acids are an important source of energy for the colonic mucosa, and metabolism by colonocytes provides energy for processes such as active transport of sodium. Lack of a dietary source for production of short-chain fatty acids, or diversion of the fecal stream by an ileostomy or colostomy, may result in mucosal atrophy and "diversion colitis."

Colonic Microflora and Intestinal Gas

Approximately 30% of fecal dry weight is composed of bacteria (10^{11} to 10^{12} bacteria/g of feces). Anaerobes are the predominant class of microorganism, and *Bacteroides* species are the most common (10^{11} to 10^{12} organisms/mL). *Escherichia coli* are the most numerous aerobes (10^8 to 10^{10} organisms/mL). Endogenous microflora are crucial for the breakdown of carbohydrates and proteins in the colon and participate in the metabolism of bilirubin, bile acids, estrogen, and cholesterol. Colonic bacteria also are necessary for production of vitamin K. Endogenous bacteria also are thought to suppress the emergence of pathogenic microorganisms, such as *Clostridium difficile*. However, the high bacterial load of the large intestine may contribute to sepsis in critically ill patients and may contribute to intra-abdominal sepsis, abscess, and wound infection following colectomy.

Intestinal gas arises from swallowed air, diffusion from the blood, and intraluminal production. Nitrogen, oxygen, carbon dioxide, hydrogen, and methane are the major components of intestinal gas. Nitrogen and oxygen are largely derived from swallowed air. Carbon dioxide is produced by the reaction of bicarbonate and hydrogen ions, and by the digestion of triglycerides to fatty acids. Hydrogen and methane are produced by colonic bacteria. The production of methane is highly variable. The gastrointestinal tract usually contains between 100 and 200 mL of gas and 400 to 1200 mL per day are released as flatus, depending upon the type of food ingested.

Motility, Defecation, and Continence

Motility

Unlike the small intestine, the large intestine does not demonstrate cyclic motor activity characteristic of the migratory motor complex. Instead, the colon displays intermittent contractions of either low or high amplitude. Low-amplitude, short-duration contractions occur in bursts and appear to move the colonic contents both antegrade and retrograde. It is thought that these bursts of motor activity delay colonic transit and thus increase the time available for absorption of water and exchange of electrolytes. High-amplitude contractions occur in a more coordinated fashion and create "mass movements." Bursts of "rectal motor complexes" also have been described. In general, cholinergic activation increases colonic motility.

Defecation

Defecation is a complex, coordinated mechanism involving colonic mass movement, increased intra-abdominal and rectal pressure, and relaxation of the pelvic floor. Distention of the rectum causes a reflex relaxation of the internal anal sphincter (the rectoanal inhibitory reflex) that allows the contents to make contact with the anal canal. This "sampling reflex" allows the sensory epithelium to distinguish solid stool from liquid stool and gas. If defecation does not occur, the rectum relaxes and the urge to defecate passes (the *accommodation response*). Defecation proceeds by coordination of increasing intra-abdominal pressure via the Valsalva maneuver, increased rectal contraction, relaxation of the puborectalis muscle, and opening of the anal canal.

Continence

The maintenance of fecal continence is at least as complex as the mechanism of defecation. Continence requires adequate rectal wall compliance to accommodate the fecal bolus, appropriate neurogenic control of the pelvic floor and sphincter mechanism, and functional internal and external sphincter muscles. At rest, the puborectalis muscle creates a "sling" around the distal rectum, forming a relatively acute angle that distributes intraabdominal forces onto the pelvic floor. With defecation, this angle straightens, allowing downward force to be applied along the axis of the rectum and anal canal. The internal and external sphincters are tonically active at rest. The internal sphincter is responsible for most of the resting, involuntary sphincter tone (resting pressure). The external sphincter is responsible for most of the voluntary sphincter tone (squeeze pressure). Branches of the pudendal nerve innervate both the internal and external sphincter. Finally, the hemorrhoidal cushions may contribute to continence by mechanically blocking the anal canal. Thus, impaired continence may result from poor rectal compliance, injury to the internal and/or external sphincter or puborectalis, or nerve damage or neuropathy.

Clinical Evaluation

Clinical Assessment

A complete history and physical examination is the starting point for evaluating any patient with suspected disease of the colon and rectum. Special attention should be paid to the patient's past medical and surgical history to detect underlying conditions that might contribute to a gastrointestinal problem. If patients have had prior intestinal surgery, it is essential that one understand the resultant gastrointestinal anatomy. In addition, family history of colorectal disease, especially inflammatory bowel disease, polyps, and colorectal cancer, is crucial. Medication use must be detailed as many drugs cause gastrointestinal symptoms. Before recommending operative intervention, the adequacy of medical treatment must be ascertained. In addition to examining the abdomen, visual inspection of the anus and perineum and careful digital rectal exam are essential.

Endoscopy

Anoscopy

The anoscope is a useful instrument for examination of the anal canal. Anoscopes are made in a variety of sizes and measure approximately 8 cm in length. A larger anoscope provides better exposure for anal procedures such as rubber band ligation or sclerotherapy of hemorrhoids. The anoscope, with obturator in place, should be adequately lubricated and gently inserted into the anal canal. The obturator is withdrawn, inspection of the visualized anal canal is done, and the anoscope should then be withdrawn. It is rotated 90 degrees and reinserted to allow visualization of all four quadrants of the canal. If the patient complains of severe perianal pain and cannot tolerate a digital rectal examination, anoscopy should not be attempted without anesthesia.

Proctoscopy

The rigid proctoscope is useful for examination of the rectum and distal sigmoid colon and is occasionally used therapeutically. The standard proctoscope is 25 cm in length and available in various diameters. Most often, a 15- or 19-mm diameter proctoscope is used for diagnostic examinations. The large (25-mm diameter) proctoscope is useful for procedures such as polypectomy, electrocoagulation, or detorsion of a sigmoid volvulus. A smaller "pediatric" proctoscope (11-mm diameter) is better tolerated by patients with anal stricture. Suction is necessary for an adequate proctoscopic examination.

Flexible Sigmoidoscopy and Colonoscopy

Video or fiberoptic flexible sigmoidoscopy and colonoscopy provide excellent visualization of the colon and rectum. Sigmoidoscopes measure 60 cm in length. Full depth of insertion may allow visualization as high as the splenic flexure, although the mobility and redundancy of the sigmoid colon often limit the extent of the examination. Partial preparation with enemas is usually adequate for sigmoidoscopy and most patients can tolerate this procedure without

sedation. Colonoscopes measure 100 to 160 cm in length and are capable of examining the entire colon and terminal ileum. A complete oral bowel preparation is usually necessary for colonoscopy and the duration and discomfort of the procedure usually require conscious sedation. Both sigmoidoscopy and colonoscopy can be used diagnostically and therapeutically. Electrocautery should generally not be used in the absence of a complete bowel preparation because of the risk of explosion of intestinal methane or hydrogen gases. Diagnostic colonoscopes possess a single channel through which instruments such as snares, biopsy forceps, or electrocautery can be passed; this channel also provides suction and irrigation capability. Therapeutic colonoscopes possess two channels to allow simultaneous suction/irrigation and the use of snares, biopsy forceps, or electrocautery.

Imaging

Plain X-Rays and Contrast Studies

Despite advanced radiologic techniques, plain x-rays and contrast studies continue to play an important role in the evaluation of patients with suspected colon and rectal diseases. Plain x-rays of the abdomen (supine, upright, and diaphragmatic views) are useful for detecting free intra-abdominal air, bowel gas patterns suggestive of small or large bowel obstruction, and volvulus. Contrast studies are useful for evaluating obstructive symptoms, delineating fistulous tracts, and diagnosing small perforations or anastomotic leaks. While Gastrografin cannot provide the mucosal detail provided by barium, this water-soluble contrast agent is recommended if perforation or leak is suspected. Double-contrast barium enema has been reported to be 70 to 90% sensitive for the detection of mass lesions greater than 1 cm in diameter. 1 Detection of small lesions can be extremely difficult, especially in a patient with extensive diverticulosis. For this reason, a colonoscopy is preferred for evaluating nonobstructing mass lesions in the colon. Double-contrast barium enema has been used as a back-up examination if colonoscopy is incomplete.

Computed Tomography

Computed tomography (CT) is commonly employed in the evaluation of patients with abdominal complaints. Its utility is primarily in the detection of extraluminal disease, such as intra-abdominal abscesses and pericolic inflammation, and in staging colorectal carcinoma, because of its sensitivity in detection of hepatic metastases. 2 Extravasation of oral or rectal contrast may also confirm the diagnosis of perforation or anastomotic leak. Nonspecific findings such as bowel wall thickening or mesenteric stranding may suggest inflammatory bowel disease, enteritis/colitis, or ischemia. A standard CT scan is relatively insensitive for the detection of intraluminal lesions.

Virtual Colonoscopy

Virtual colonoscopy is a new radiologic technique that is designed to overcome some of the limitations of traditional CT scanning. This technology uses helical CT and three-dimensional

reconstruction to detect intraluminal colonic lesions. Oral bowel preparation, oral and rectal contrast, and colon insufflation are used to maximize sensitivity. Early evaluation of virtual colonoscopy suggests that accuracy may approach that of colonoscopy for detection of lesions 1 cm in diameter or greater.

Magnetic Resonance Imaging

The main use of magnetic resonance imaging (MRI) in colorectal disorders is in evaluation of pelvic lesions. MRI is more sensitive than CT for detecting bony involvement or pelvic sidewall extension of rectal tumors. MRI also can be helpful in the detection and delineation of complex fistulas in ano. The use of an endorectal coil may increase sensitivity.

Positron Emission Tomography

Positron emission tomography (PET) is used for imaging tissues with high levels of anaerobic glycolysis, such as malignant tumors. 18 F-fluorodeoxyglucose (FDG) is injected as a tracer; metabolism of this molecule then results in positron emission. PET has been used as an adjunct to CT in the staging of colorectal cancer and may prove useful in discriminating recurrent cancer from fibrosis. At present, the efficacy and utility of PET in the detection of recurrent and/or metastatic colorectal cancer remains unproven.

Angiography

Angiography is occasionally used for the detection of bleeding within the colon or small bowel. To visualize hemorrhage angiographically, bleeding must be relatively brisk (approximately 0.5 to 1.0 mL per minute). If extravasation of contrast is identified, infusion of vasopressin or angiographic embolization can be therapeutic.

Endorectal and Endoanal Ultrasound

Endorectal ultrasound is primarily used to evaluate the depth of invasion of neoplastic lesions in the rectum. The normal rectal wall appears as a five-layer structure (Fig. 28-6). Ultrasound can reliably differentiate most benign polyps from invasive tumors based upon the integrity of the submucosal layer. Ultrasound can also differentiate superficial T1-T2 from deeper T3-T4 tumors. Overall, the accuracy of ultrasound in detecting depth of mural invasion ranges between 81 and 94%.³ This modality also can detect enlarged perirectal lymph nodes, which may suggest nodal metastases; accuracy of detection of pathologically positive lymph nodes is 58 to 83%. Ultrasound may also prove useful for early detection of local recurrence after surgery.

Endoanal ultrasound is used to evaluate the layers of the anal canal. Internal anal sphincter, external anal sphincter, and puborectalis muscle can be differentiated. Endoanal ultrasound is particularly useful for detecting sphincter defects and for outlining complex anal fistulas.

Physiologic and Pelvic Floor Investigations

Anorectal physiologic testing uses a variety of techniques to investigate the function of the pelvic floor. These techniques are useful in the evaluation of patients with incontinence, constipation, rectal prolapse, obstructed defecation, and other disorders of the pelvic floor.

Manometry

Anorectal manometry is performed by placing a pressure-sensitive catheter in the lower rectum. The catheter is then withdrawn through the anal canal and pressures recorded. A balloon attached to the tip of the catheter also can be used to test anorectal sensation. The *resting pressure* in the anal canal reflects the function of the internal anal sphincter (normal: 40 to 80 mm Hg), while the *squeeze pressure*, defined as the maximum voluntary contraction pressure minus the resting pressure, reflects function of the external anal sphincter (normal: 40 to 80 mm Hg above resting pressure). The *high-pressure zone* estimates the length of the anal canal (normal: 2.0 to 4.0 cm). The *rectoanal inhibitory reflex* can be detected by inflating a balloon in the distal rectum; absence of this reflex is characteristic of Hirschsprung's disease.

Neurophysiology

Neurophysiologic testing assesses function of the pudendal nerves and recruitment of puborectalis muscle fibers. Pudendal nerve terminal motor latency measures the speed of transmission of a nerve impulse through the distal pudendal nerve fibers (normal: 1.8 to 2.2 msec); prolonged latency suggests the presence of neuropathy. EMG recruitment assesses the contraction and relaxation of the puborectalis muscle during attempted defecation. Normally, recruitment increases when a patient is instructed to "squeeze," and decreases when a patient is instructed to "push." Inappropriate recruitment is an indication of paradoxical contraction (nonrelaxation of the puborectalis). Needle EMG has been used to map both the pudendal nerves and the anatomy of the internal and external sphincters. However, this examination is painful and poorly tolerated by most patients. Needle EMG has largely been replaced by pudendal nerve motor-latency testing to assess pudendal nerve function and endoanal ultrasound to map the sphincters.

Rectal Evacuation Studies

Rectal evacuation studies include the balloon expulsion test and video defecography. Balloon expulsion assesses a patient's ability to expel an intrarectal balloon. Video defecography provides a more detailed assessment of defecation. In this test, barium paste is placed in the rectum and defecation is then recorded fluoroscopically. Defecography is used to differentiate nonrelaxation of the puborectalis, obstructed defecation, increased perineal descent, rectal prolapse and intussusception, rectocele, and enterocele. The addition of vaginal contrast and intraperitoneal contrast is useful in delineating complex disorders of the pelvic floor.

Laboratory Studies

Fecal Occult Blood Testing

Fecal occult blood testing (FOBT) is used as a screening test for colonic neoplasms in asymptomatic, average-risk individuals. The efficacy of this test is based upon serial testing because the majority of colorectal malignancies will bleed intermittently. FOBT has been a nonspecific test for peroxidase contained in hemoglobin; consequently, occult bleeding from any gastrointestinal source will produce a positive result. Similarly, many foods (red meat, some fruits and vegetables, and vitamin C) will produce a false-positive result. Patients were counseled to eat a restricted diet for 2 to 3 days prior to the test. Increased specificity is now possible by using immunochemical FOBT. These tests rely on monoclonal or polyclonal antibodies to react with the intact globin portion of human hemoglobin. Because globin does not survive in the upper gastrointestinal tract, the immunochemical tests are more specific for identifying occult bleeding from the colon or rectum. Dietary restrictions are not necessary. Any positive FOBT mandates further investigation, usually by colonoscopy.

Stool Studies

Stool studies are often helpful in evaluating the etiology of diarrhea. Wet-mount examination reveals the presence of fecal leukocytes, which may suggest colonic inflammation or the presence of an invasive organism such as invasive *E. coli* or *Shigella*. Stool cultures can detect pathogenic bacteria, ova, and parasites. *C. difficile* colitis is diagnosed by detecting bacterial toxin in the stool. 4 Steatorrhea may be diagnosed by adding Sudan red stain to a stool sample.

Serum Tests

Specific laboratory tests that should be performed will be dictated by the clinical scenario. Preoperative studies generally include a complete blood count and electrolyte panel. The addition of coagulation studies, liver function tests, and blood typing/cross-matching depends upon the patient's medical condition and the proposed surgical procedure.

Tumor Markers

Carcinoembryonic antigen (CEA) may be elevated in 60 to 90% of patients with colorectal cancer. Despite this, CEA is not an effective screening tool for this malignancy. Many practitioners follow serial CEA levels after curative-intent surgery in order to detect early recurrence of colorectal cancer. However, this tumor marker is nonspecific, and no survival benefit has yet been proven. Other biochemical markers (ornithine decarboxylase, urokinase) have been proposed, but none has yet proven sensitive or specific for detection, staging, or predicting prognosis of colorectal carcinoma. 5

Genetic Testing

Although familial colorectal cancer syndromes, such as familial adenomatous polyposis (FAP) and hereditary nonpolyposis colon cancer (HNPCC) are rare, information about the specific genetic abnormalities underlying these disorders has led to significant interest in the role of genetic testing for colorectal cancer. 6 Tests for mutations in the adenomatous polyposis coli

(APC) gene responsible for FAP, and in mismatch repair genes responsible for HNPCC, are commercially available and extremely accurate in families with known mutations. Although many of these mutations are also present in sporadic colorectal cancer, the accuracy of genetic testing in average-risk individuals is considerably lower and these tests are not recommended for screening. Because of the potential psychosocial implications of genetic testing, it is strongly recommended that professional genetic counselors be involved in the care of any patient considering these tests.

Evaluation of Common Symptoms

Pain

Abdominal Pain

Abdominal pain is a nonspecific symptom with a myriad of causes. Abdominal pain related to the colon and rectum can result from obstruction (either inflammatory or neoplastic), inflammation, perforation, or ischemia. Plain x-rays and judicious use of contrast studies and/or a CT scan can often confirm the diagnosis. Gentle retrograde contrast studies (barium or Gastrografin enema) may be useful in delineating the degree of colonic obstruction. Sigmoidoscopy and/or colonoscopy performed by an experienced endoscopist can assist in the diagnosis of ischemic colitis, infectious colitis, and inflammatory bowel disease. However, if perforation is suspected, colonoscopy and/or sigmoidoscopy are generally contraindicated. Evaluation and treatment of abdominal pain from a colorectal source should follow the usual surgical principles of a thorough history and physical examination, appropriate diagnostic tests, resuscitation, and appropriately timed surgical intervention.

Pelvic Pain

Pelvic pain can originate from the distal colon and rectum or from adjacent urogenital structures. Tenesmus may result from proctitis or from a rectal or retrorectal mass. Cyclical pain associated with menses, especially when accompanied by rectal bleeding, suggests a diagnosis of endometriosis. Pelvic inflammatory disease also can produce significant abdominal and pelvic pain. The extension of a peridiverticular abscess or periappendiceal abscess into the pelvis may also cause pain. CT scan and/or MRI may be useful in differentiating these diseases. Proctoscopy (if tolerated) also can be helpful. Occasionally, laparoscopy will yield a diagnosis.

Anorectal Pain

Anorectal pain is most often secondary to an anal fissure or perirectal abscess and/or fistula. Physical examination can usually differentiate these conditions. Other, less common causes of anorectal pain include anal canal neoplasms, perianal skin infection, and dermatologic conditions. Proctalgia fugax results from levator spasm and may present without any other anorectal findings. Physical exam is critical in evaluating patients with anorectal pain. If a

patient is too tender to examine in the office, an examination under anesthesia is necessary. MRI may be helpful in select cases where the etiology of pain is elusive.

Lower Gastrointestinal Bleeding

The first goal in evaluating and treating a patient with gastrointestinal hemorrhage is adequate resuscitation. The principles of ensuring a patent airway, supporting ventilation, and optimizing hemodynamic parameters apply and coagulopathy and/or thrombocytopenia should be corrected. The second goal is to identify the source of hemorrhage. Because the most common source of gastrointestinal hemorrhage is esophageal, gastric, or duodenal, nasogastric aspiration should always be performed; return of bile suggests that the source of bleeding is distal to the ligament of Treitz. If aspiration reveals blood or nonbile secretions, or if symptoms suggest an upper intestinal source, esophagogastroduodenoscopy is performed. Anoscopy and/or limited proctoscopy can identify hemorrhoidal bleeding. A technetium-99 (^{99m}Tc)-tagged red blood cell (RBC) scan is extremely sensitive and is able to detect as little as 0.1 mL/h of bleeding; however, localization is imprecise. If the ^{99m}Tc -tagged RBC scan is positive, angiography can then be employed to localize bleeding. Infusion of vasopressin or angioembolization may be therapeutic. Alternatively, a catheter can be left in the bleeding vessel to allow localization at the time of laparotomy. If the patient is hemodynamically stable, a rapid bowel preparation (over 4 to 6 hours) can be performed to allow colonoscopy. Colonoscopy may identify the cause of the bleeding, and cautery or injection of epinephrine into the bleeding site may be used to control hemorrhage. Colectomy may be required if bleeding persists despite these interventions. Intraoperative colonoscopy and/or enteroscopy may assist in localizing bleeding. If colectomy is required, a segmental resection is preferred if the bleeding source can be localized. "Blind" subtotal colectomy may very rarely be required in a patient who is hemodynamically unstable with ongoing colonic hemorrhage of an unknown source. In this setting, it is crucial to irrigate the rectum and examine the mucosa by proctoscopy to ensure that the source of bleeding is not distal to the resection margin

Occult blood loss from the gastrointestinal tract may manifest as iron-deficiency anemia or may be detected with fecal occult blood testing. Because colon neoplasms bleed intermittently and rarely present with rapid hemorrhage, the presence of occult fecal blood should always prompt a colonoscopy. Unexplained iron-deficiency anemia is also an indication for colonoscopy.

Hematochezia is commonly caused by hemorrhoids or fissure. Sharp, knife-like pain and bright-red rectal bleeding with bowel movements suggest the diagnosis of fissure. Painless, bright-red rectal bleeding with bowel movements is often secondary to a friable internal hemorrhoid that is easily detected by anoscopy. In the absence of a painful, obvious fissure, any patient with rectal bleeding should undergo a careful digital rectal examination, anoscopy, and proctosigmoidoscopy. Failure to diagnose a source in the distal anorectum should prompt colonoscopy.

Constipation and Obstructed Defecation

Constipation is an extremely common complaint, affecting more than 4 million people in the United States. Despite the prevalence of this problem, there is lack of agreement about an appropriate definition of constipation. Patients may describe infrequent bowel movements, hard stools, or excessive straining. A careful history of these symptoms often clarifies the nature of the problem.

Constipation has a myriad of causes. Underlying metabolic, pharmacologic, endocrine, psychologic, and neurologic causes often contribute to the problem. A stricture or mass lesion should be excluded by colonoscopy or barium enema. After these causes have been excluded, evaluation focuses upon differentiating *slow-transit constipation* from *outlet obstruction*. Transit studies, in which radiopaque markers are swallowed and then followed radiographically, are useful for diagnosing slow-transit constipation. Anorectal manometry and electromyography can detect nonrelaxation of the puborectalis, which contributes to outlet obstruction. The absence of an anorectal inhibitory reflex suggests Hirschsprung's disease and may prompt a rectal mucosal biopsy. Defecography can identify rectal prolapse, intussusception, rectocele, or enterocele.

Medical management is the mainstay of therapy for constipation and includes fiber, increased fluid intake, and laxatives. Outlet obstruction from nonrelaxation of the puborectalis often responds to biofeedback. 7 Surgery to correct rectocele and rectal prolapse has a variable effect on symptoms of constipation, but can be successful in selected patients. Subtotal colectomy is considered only for patients with severe slow-transit constipation (colonic inertia) refractory to maximal medical interventions. While this operation almost always increases bowel movement frequency, complaints of diarrhea, incontinence, and abdominal pain are not infrequent, and patients should be carefully selected. 8

Diarrhea and Irritable Bowel Syndrome

Diarrhea is also a common complaint and is usually a self-limited symptom of infectious gastroenteritis. If diarrhea is chronic or is accompanied by bleeding or abdominal pain, further investigation is warranted. Bloody diarrhea and pain are characteristic of colitis; etiology can be an infection (invasive *E. coli*, *Shigella*, *Salmonella*, *Campylobacter*, *Entamoeba histolytica*, or *C. difficile*), inflammatory bowel disease (ulcerative colitis or Crohn's colitis), or ischemia. Stool wet-mount and culture can often diagnose infection. Sigmoidoscopy or colonoscopy can be helpful in diagnosing inflammatory bowel disease or ischemia. However, if the patient has abdominal tenderness, particularly with peritoneal signs, or any other evidence of perforation, endoscopy is contraindicated.

Chronic diarrhea may present a more difficult diagnostic dilemma. Chronic ulcerative colitis, Crohn's colitis, infection, malabsorption, and short gut syndrome can cause chronic diarrhea. Rarely, carcinoid syndrome and islet cell tumors (vasoactive intestinal peptide-secreting tumor [VIPoma], somatostatinoma, gastrinoma) present with this symptom. Large villous lesions may cause secretory diarrhea. Collagenous colitis can cause diarrhea without any obvious mucosal

abnormality. Along with stool cultures, tests for malabsorption, and metabolic investigations, colonoscopy can be invaluable in differentiating these causes. Biopsies should be taken even if the colonic mucosa appears grossly normal.

Irritable bowel syndrome is a particularly troubling constellation of symptoms consisting of crampy abdominal pain, bloating, constipation, and urgent diarrhea. Work-up reveals no underlying anatomic or physiologic abnormality. Once other disorders have been excluded, dietary restrictions and avoidance of caffeine, alcohol, and tobacco may help to alleviate symptoms. Antispasmodics and bulking agents may be helpful.

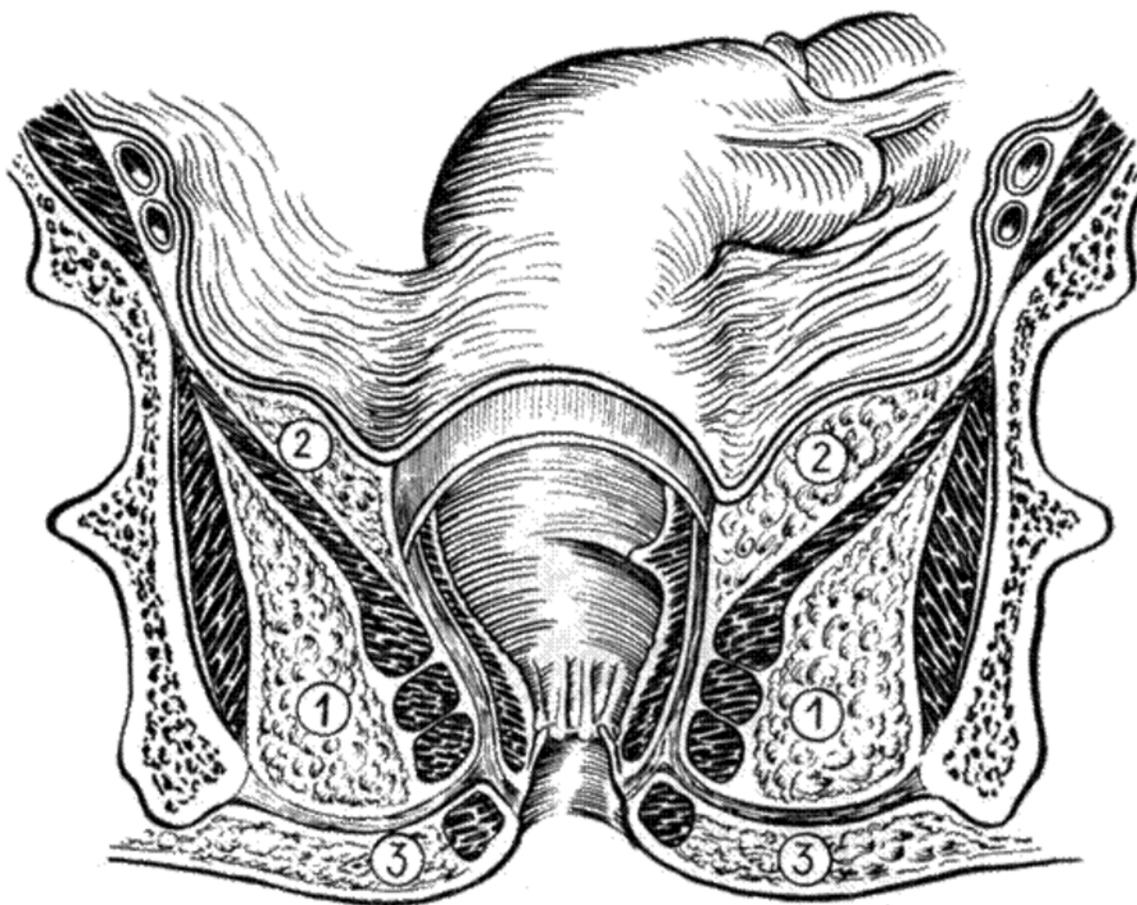
Incontinence

The incidence of fecal incontinence has been estimated to occur in 10 to 13 individuals per 1000 people older than age 65 years. Incontinence ranges in severity from occasional leakage of gas and liquid stool to daily loss of solid stool. The underlying cause of incontinence is often multifactorial and diarrhea is often contributory. In general, causes of incontinence can be classified as *neurogenic* or *anatomic*. Neurogenic causes include diseases of the central nervous system and spinal cord along with pudendal nerve injury. Anatomic causes include congenital abnormalities, procidentia, overflow incontinence secondary to impaction or neoplasm, and trauma. The most common traumatic cause of incontinence is injury to the anal sphincter during vaginal delivery. Other causes include anorectal surgery, impalement, and pelvic fracture.

After a thorough medical evaluation to detect underlying conditions that might contribute to incontinence, evaluation focuses on assessment of the anal sphincter and pudendal nerves. Pudendal nerve terminal motor latency testing may detect neuropathy. Anal manometry can detect low resting and squeeze pressures. Defecography can detect rectal prolapse. Endoanal ultrasound is invaluable in diagnosing sphincter defects.

HEMORRHOIDS

Hemorrhoids from Greek mean bleeding. Nowadays hemorrhoids are volume increase or dilation of cavernous bodies in rectum.



Ishioanal space (1), perirectal space (2), perianal space (3).

Etiology and Pathogenesis

Appearance of hemorrhoids is connected with such factors as functional insufficiency of connective tissue, increase of venous pressure at constipation, hard physical work, low-active life style, pregnancy, alcoholism, spicy food. Mechanism of hemorrhoids' development is connected with disorder of blood outflow by venules from cavernous bodies and their hyperplasia in distal part of rectum. Cavernous bodies are usually situated in basal part of anal columns (Morgagni's). Presence of direct arteriovenous anastomoses conditions in hemorrhoids bleeding of arterial character.

Pathomorphology

External hemorrhoids have their origin from veins of lower hemorrhoidal plexus, internal – from upper. External hemorrhoids are soft, of bluish color, they have not smooth surface and are filled with blood. Sometimes they are dense and filled with thrombs. Sometimes external hemorrhoids are prolapsed internal nodes. They have long leg deeply in anal canal.

Histologically they observe wall atrophy, anomalies of development, often – signs of thrombophlebitis.

Hemorrhoids are cushions of submucosal tissue containing venules, arterioles, and smooth-muscle fibers that are located in the anal canal (see Fig. 28-4). Three hemorrhoidal cushions are found in the left lateral, right anterior, and right posterior positions. Hemorrhoids are thought to function as part of the continence mechanism and aid in complete closure of the anal canal at rest. Because hemorrhoids are a normal part of anorectal anatomy, treatment is only indicated if they become symptomatic. Excessive straining, increased abdominal pressure, and hard stools increase venous engorgement of the hemorrhoidal plexus and cause prolapse of hemorrhoidal tissue. Outlet bleeding, thrombosis, and symptomatic hemorrhoidal prolapse may result.

External hemorrhoids are located distal to the dentate line and are covered with anoderm. Because the anoderm is richly innervated, thrombosis of an external hemorrhoid may cause significant pain. It is for this reason that external hemorrhoids should not be ligated or excised without adequate local anesthetic. A skin tag is redundant fibrotic skin at the anal verge, often persisting as the residual of a thrombosed external hemorrhoid. Skin tags are often confused with symptomatic hemorrhoids. External hemorrhoids and skin tags may cause itching and difficulty with hygiene if they are large. Treatment of external hemorrhoids and skin tags are only indicated for symptomatic relief.

Internal hemorrhoids are located proximal to the dentate line and covered by insensate anorectal mucosa. Internal hemorrhoids may prolapse or bleed, but rarely become painful unless they develop thrombosis and necrosis (usually related to severe prolapse, incarceration, and/or strangulation). Internal hemorrhoids are graded according to the extent of prolapse. First-degree hemorrhoids bulge into the anal canal and may prolapse beyond the dentate line on straining. Second-degree hemorrhoids prolapse through the anus but reduce spontaneously. Third-degree hemorrhoids prolapse through the anal canal and require manual reduction. Fourth-degree hemorrhoids prolapse but cannot be reduced and are at risk for strangulation.

Combined internal and external hemorrhoids straddle the dentate line and have characteristics of both internal and external hemorrhoids. Hemorrhoidectomy is often required for large, symptomatic, combined hemorrhoids. Postpartum hemorrhoids result from straining during labor, which results in edema, thrombosis, and/or strangulation. Hemorrhoidectomy is often the treatment of choice, especially if the patient has had chronic hemorrhoidal symptoms. Portal hypertension was long thought to increase the risk of hemorrhoidal bleeding because of the anastomoses between the portal venous system (middle and upper hemorrhoidal plexuses) and the systemic venous system (inferior rectal plexuses). It is now understood that hemorrhoidal disease is no more common in patients with portal hypertension than in the normal population. Rectal varices, however, may occur and may cause hemorrhage in these patients. In general, rectal varices are best treated by lowering portal venous pressure. Rarely, suture ligation may be necessary if massive bleeding persists. Surgical hemorrhoidectomy should be avoided in these patients because of the risk of massive, difficult-to-control variceal bleeding.

Classification

Hemorrhoids by etiological signs are divided onto innate and acquired, by localization – internal (submucosal), external and mixed (combined).

By clinical course hemorrhoids are: acute and chronic, not complicated and complicated (thrombosis, strangulation of hemorrhoids). There also define primary and secondary hemorrhoids (at liver cirrhosis, diseases of circulatory system, tumors).

Symptoms and clinical course

Early signs of hemorrhoids are rush feeling in anal region that appears due to skin maceration by mucous excretions from rectum. This sign is increased in diet violations that may be a consequence of constipation or diarrhea.

There define three degrees of hemorrhoids. At I degree nodes prolapse from anus during defecation, but they replace independently and are painful during palpation. At II degree there is a need to replace nodes. There is edema in perianal region and pain. At III degree nodes prolapse at very low physical loading, edema and pain are severe. The speciality of chronic hemorrhoids is that there are conditions for appearance of mucosal fissures, polyps and paraproctitis.

Variants of clinical course and complications

Initial part of hemorrhoids is characterized by gradual beginning and low reflected clinical signs – presence of hemorrhoidal nodes. Duration of this period may be different – from several months to years. Chronic course of hemorrhoids is characterized by periodical acute conditions and remissions. Difference of clinical course of hemorrhoids is only in complications.

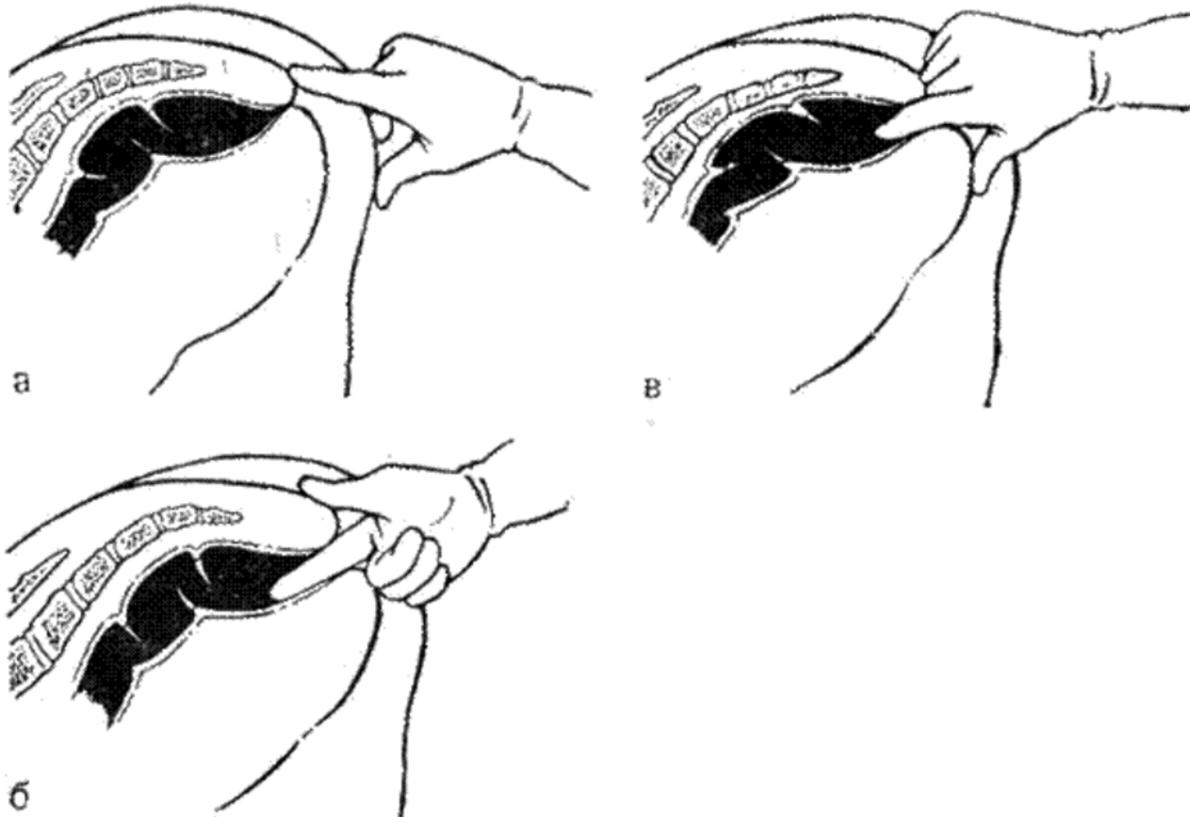
Bleeding from hemorrhoids appears mostly during or after defecation and may be as profuse as moderate. Blood is bright red (arterial).

Acute thrombosis of hemorrhoidal nodes is mostly seen in III degree of hemorrhoids. Thrombosis is a complication of as external as internal nodes. At this there appear edema of bluish color, severe pain. Progress of disease course sometimes may be conditioned by thrombosis of inferior vena cava.

Such complication as prolapse and strangulation in anal sphincter of internal hemorrhoidal nodes appear not so often. Nodes swell, become bluish, sometimes there occur necrosis that spreads on external nodes. Edema of anal region spreads to 10 cm. Body temperature increases to 39 degrees centigrade, defecation and diuresis are disordered, patients cannot sleep due to severe pain, they lose appetite, general status incredibly worsens.

Diagnostic program

1. Anamnesis and physical data.
2. Examination of anal region.
3. Finger investigation of rectum.
4. Examination of rectum by rectal mirror.
5. Rectoromanoscopy.
6. General analysis of blood and urine.
7. Coagulogram.
8. Sedimentation reactions (Reaction of Wassermann).



Digital examination of the rectum:

- a) examination of the perianal region; b) examination of the anus; c) examination of the ampula.**



Digital examination of the rectum

Differential diagnostics

Anal fissure is characterized by severe pain during of after defecation, spasm of sphincter and small bleeding during defecation.

The cancer of rectum at the beginning of disease, usually, is painless, a blood in the first portions of excrement appears. At the examination of rectum hard formation or ulcers with a dense bottom is observed. Histological examination of biopsy specifies a diagnosis.

Single or multiple polyps of rectum may be combined with polyposis of colon. Revealed symptoms are disorder of defecation and blood in feces. Examination of rectum lets us verify the diagnosis.

Incomplete prolapse or *prolapse of rectal mucosa* differs from hemorrhoids of III degree by combination with low reflected incontinention.

Tactics and choice of treating method

Conservative treatment is provided in patients with not complicated hemorrhoids (I degree of disease). Treatment must be complex and include diet therapy, remedies and physioprocedures.

For treatment of such patients they use massage through Diet therapy means excluding from ration spicy and salty food. rectum, recommend carrying of bandages. They also prescribe enemas (micro enemas with warm water, oils, antiseptics), warm as sitting bath, perineal shower, warming compresses.

In acute period they use bandages with cold water, cooling bandages with plumbum water or furacilinum.

Medicamentous therapy includes prescription of anti bleeding remedies and analgetics, antiseptics, anti-inflammatory remedies (orally, intravenously and locally as rectal suppositoria).

They also use physiotherapeutic methods (UHF, darsonvalization), treating physical training for strengthening of abdominal muscles and diaphragm, pelvis, spa treatment (H₂S baths, mud and radon sanatoria).

Indications for surgical treatment are frequent bleedings from hemorrhoidal nodes that are accompanied with anemia, big nodes that worsen defecationm inflammation, prolapse and strangulation of nodes.

There are known more than 30 methods of hemorrhoids' extraction. The main moments of operation are divulsion of sphincter, extraction and ligation of hemorrhoidal nodes situated in zones on 3, 7, and 11 hours at position of patient on back. Hemorrhoidal nodes are cut from external area to internal, leg is ligated by silk ligature and extracted. The most wide spread method is by Milligan-Morgan – extraction of nodes with renewal of mucosa in anus (Pic. 4.1.1).

In patients with complication of secondary hemorrhoids by bleeding that may not be treated by conservative therapy as a rule there is provided only ligation of bleeding areas.

Medical Therapy

Bleeding from first- and second-degree hemorrhoids often improves with the addition of dietary fiber, stool softeners, increased fluid intake, and avoidance of straining. Associated pruritus may often improve with improved hygiene. Many over-the-counter topical medications are desiccants and are relatively ineffective for treating hemorrhoidal symptoms.

Rubber Band Ligation

Persistent bleeding from first-, second-, and selected third-degree hemorrhoids may be treated by rubber band ligation.

Mucosa located 1 to 2 cm proximal to the dentate line is grasped and pulled into a rubber band applicator. After firing the ligator, the rubber band strangulates the underlying tissue, causing scarring and preventing further bleeding or prolapse (Fig. 28-30). In general, only one or two quadrants are banded per visit. Severe pain will occur if the rubber band is placed at or distal to the dentate line where sensory nerves are located. Other complications of rubber band ligation include urinary retention, infection, and

bleeding. Urinary retention occurs in approximately 1% of patients and is more likely if the ligation has inadvertently included a portion of the internal sphincter. Necrotizing infection is an uncommon, but life-threatening complication. Severe pain, fever, and urinary retention are early signs of infection and should prompt immediate evaluation of the patient usually with an exam under anesthesia. Treatment includes débridement of necrotic tissue, drainage of associated abscesses, and broad-spectrum antibiotics. Bleeding may occur approximately 7 to 10 days after rubber band ligation, at the time when the ligated pedicle necroses and sloughs. Bleeding is usually self-limited, but persistent hemorrhage may require exam under anesthesia and suture ligation of the pedicle.

Infrared Photocoagulation

Infrared photocoagulation is an effective office treatment for small first- and second-degree hemorrhoids. The instrument is applied to the apex of each hemorrhoid to coagulate the underlying plexus. All three quadrants may be treated during the same visit. Larger hemorrhoids and hemorrhoids with a significant amount of prolapse are not effectively treated with this technique.

Sclerotherapy

The injection of bleeding internal hemorrhoids with sclerosing agents is another effective office technique for treatment of first-, second-, and some third-degree hemorrhoids. One to 3 mL of a sclerosing solution (5-phenol in olive oil, sodium morrhuate, or quinine urea) are injected into the submucosa of each hemorrhoid. Few complications are associated with sclerotherapy, but infection and fibrosis have been reported.

Excision of Thrombosed External Hemorrhoids

Acutely thrombosed external hemorrhoids generally cause intense pain and a palpable perianal mass during the first 24 to 72 hours after thrombosis. The thrombosis can be effectively treated with an elliptical excision performed in the office under local anesthesia. Because the clot is usually loculated, simple incision and drainage is rarely effective. After 72 hours, the clot begins to resorb, and the pain resolves spontaneously. Excision is unnecessary, but sitz baths and analgesics are often helpful.

Operative Hemorrhoidectomy

A number of surgical procedures have been described for elective resection of symptomatic hemorrhoids. All are based on decreasing blood flow to the hemorrhoidal plexuses and excising redundant anoderm and mucosa.

Closed Submucosal Hemorrhoidectomy

The Parks or Ferguson hemorrhoidectomy involves resection of hemorrhoidal tissue and closure of the wounds with absorbable suture. The procedure may be performed in the prone or lithotomy position under local, regional, or general anesthesia. The anal canal is examined and an anal speculum inserted. The hemorrhoid cushions and associated redundant mucosa are identified and excised using an elliptical incision starting just distal to the anal verge and extending proximally to the anorectal ring. It is crucial to identify the fibers of the internal sphincter and carefully brush these away from the dissection in order to avoid injury to the sphincter. The apex of the hemorrhoidal plexus is then ligated and the hemorrhoid excised. The wound is then closed with a running absorbable suture. All three hemorrhoidal cushions may be removed using this technique; however, care should be taken to avoid resecting a large area of perianal skin in order to avoid postoperative anal stenosis.

Open Hemorrhoidectomy

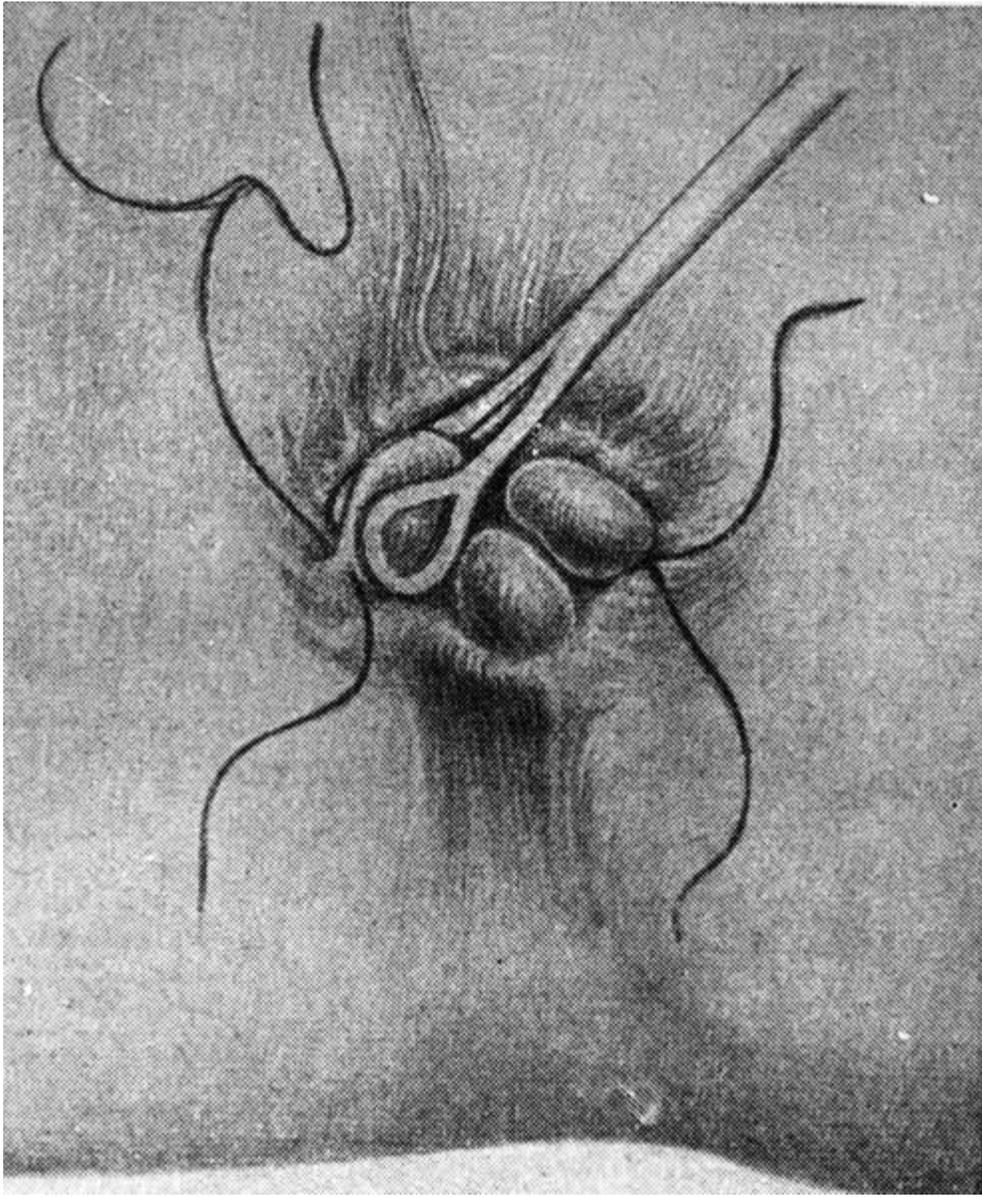
This technique, often called the Milligan and Morgan hemorrhoidectomy, follows the same principles of excision described above, but the wounds are left open and allowed to heal by secondary intention.

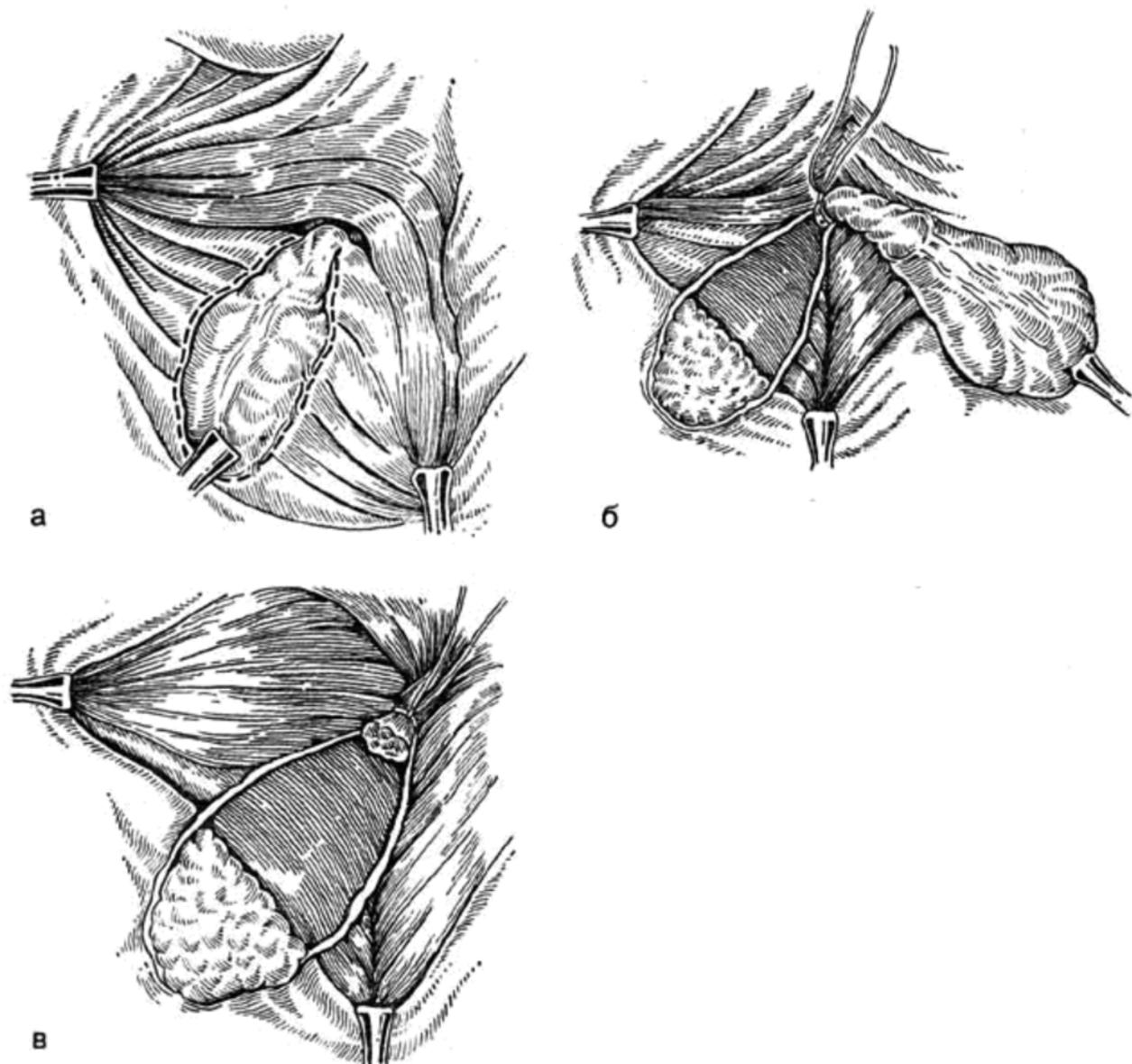
Whitehead's Hemorrhoidectomy

Whitehead's hemorrhoidectomy involves circumferential excision of the hemorrhoidal cushions just proximal to the dentate line. After excision, the rectal mucosa is then advanced and sutured to the dentate line. While some surgeons still use the Whitehead hemorrhoidectomy technique, most have abandoned this approach because of the risk of ectropion (Whitehead's deformity).

Stapled Hemorrhoidectomy

Stapled hemorrhoidectomy has been proposed as an alternative surgical approach. 81–83 Unlike excisional hemorrhoidectomy, stapled hemorrhoidectomy does not aim to excise redundant hemorrhoidal tissue. Instead, stapled hemorrhoidectomy removes a short circumferential segment of rectal mucosa proximal to the dentate line using a circular stapler. This effectively ligates the venules feeding the hemorrhoidal plexus and fixes redundant mucosa higher in the anal canal. Critics suggest that this technique is only appropriate for patients with large, bleeding, internal hemorrhoids, and is ineffective in management of external or combined hemorrhoids. Although stapled hemorrhoidectomy has not been widely accepted at this time, it remains a promising new technique.





Milligan-Morgan operation

After operation hemorrhoidectomy may appear early (bleeding from the wound) and later (stricture of anal canal) complications. With the aim of prevention of bleeding at hemorrhoidectomy leg is ligated with silk ligation, and the wound – by node ligations to bottom. For prevention of stricture of anal canal there makes sense to extract nodes not more than in 4 places. During this between extracted nodes there must stay not injured mucosa. When indicated, if after operation surgeon revealed the stricture of entrance into anal canal, hemorrhoidectomy is finished by dosed sphincterectomy.

During appearance of stricture of anal canal by posterior commissure scar in extracted, there is provided dosed sphincterectomy, and then mucosa is connected with perianal skin with further fixation by separate ligations.

Complications of Hemorrhoidectomy

Postoperative pain following excisional hemorrhoidectomy requires analgesia usually with oral narcotics. Nonsteroidal anti-inflammatory drugs, muscle relaxants, topical analgesics, and comfort measures, including sitz baths, are often useful as well. Several studies show that stapled hemorrhoidectomy is associated with a significant decrease in postoperative pain. Other complications are similar to those seen with excisional hemorrhoidectomy. Urinary retention is a common complication following hemorrhoidectomy and occurs in 10 to 50% of patients. The risk of urinary retention can be minimized by limiting intraoperative and perioperative intravenous fluids, and by providing adequate analgesia. Pain can also lead to fecal impaction. Risk of impaction may be decreased by preoperative enemas or a limited mechanical bowel preparation, liberal use of laxatives postoperatively, and adequate pain control. While a small amount of bleeding, especially with bowel movements, is to be expected, massive hemorrhage can occur after hemorrhoidectomy. Bleeding may occur in the immediate postoperative period (often in the recovery room) as a result of inadequate ligation of the vascular pedicle. This type of hemorrhage mandates an urgent return to the operating room where suture ligation of the bleeding vessel will often solve the problem. Bleeding may also occur 7 to 10 days after hemorrhoidectomy when the necrotic mucosa overlying the vascular pedicle sloughs. While some of these patients may be safely observed, others will require an exam under anesthesia to ligate the bleeding vessel or to oversee the wounds if no specific site of bleeding is identified. Infection is uncommon after hemorrhoidectomy; however, necrotizing soft-tissue infection can occur with devastating consequences. Severe pain, fever, and urinary retention may be early signs of infection. If infection is suspected, an emergent examination under anesthesia, drainage of abscess, and/or débridement of all necrotic tissue are required.

Long-term sequelae of hemorrhoidectomy include incontinence, anal stenosis, and ectropion (Whitehead's deformity). Many patients experience transient incontinence to flatus, but these symptoms are usually short-lived and few patients have permanent fecal incontinence. Anal stenosis may result from scarring after extensive resection of perianal skin. Ectropion may occur after a Whitehead's hemorrhoidectomy. This complication is usually the result of suturing the rectal mucosa too far distally in the anal canal and can be avoided by ensuring that the mucosa is sutured at or just above the dentate line.

RECTAL FISSURES

Rectal fissures are linear or triangle shaped defects of anal mucosa. This disease takes 3rd place after hemorrhoids and paraproctitis by frequency.

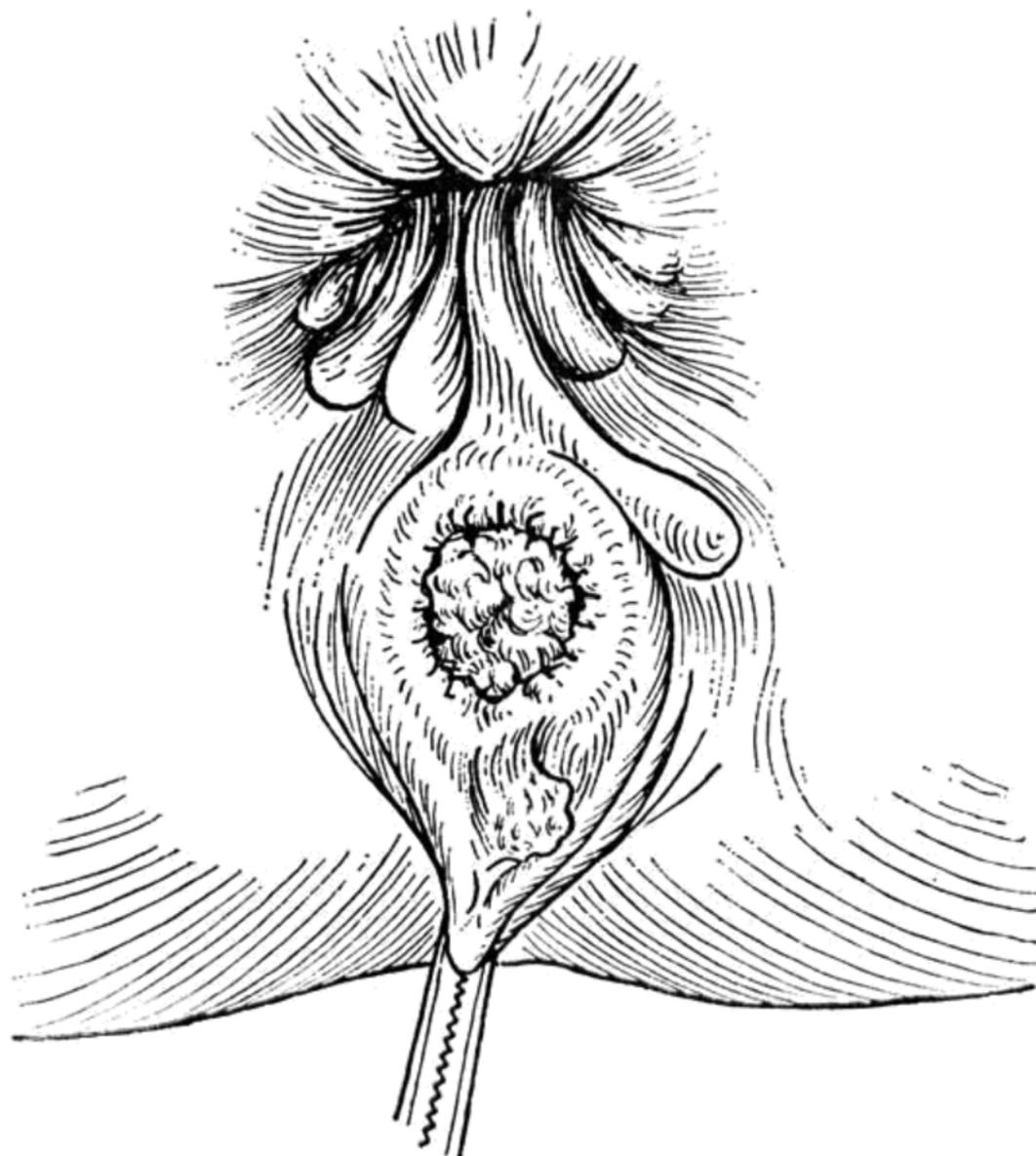
Etiology and pathogenesis

The most wide spread theories of appearance of fissures are mechanic and infectious. Due to the first, appearance of fissures is conditioned by injury of anal mucosa by dense feces in combination with constipation and diarrhea. Diseases that promote formation of fissures are proctosigmoiditis, enterocolitis and hemorrhoids. Often fissures accompany gastritis, gastric and duodenal ulcers.

By infectious theory fissures appear at inflammation of anal glands (criptitis) that lead to tissue fibrosis and decrease of their elasticity. Appearance of disease also may be promoted by syphilis, tuberculosis, homosexuality.

Classification

Rectal fissures by clinical course are divided onto acute and chronic. They may be complicated (paraproctitis, malignization, bleeding, pectenosis) and combined with other diseases of anal canal (hemorrhoids, criptitis, polypus).



Rectal fissures

Symptoms and clinical course

Such disease is seen mostly in women of middle age. Fissures are mostly situated in area of posterior commissure on 6 hours by clock dial in patient's position laying on back. More rarely fissures are situated on anterior and lateral walls. On posterior wall of anal canal conditions of bleeding are worse, that's why there is more danger of mucosal injury during defecation, it is connected with pressure of feces during their motion onto posterior and anterior commissures. Fissure mostly has longitudinal direction and hides between skin folds in anal area. In chronic course near external edge of fissure there appear skin fold with undermined edges, so called terminal tuberculum. On internal edge of fissure there is tuberculum of smaller size. In rare cases there are seen two fissures at the same time.

Clinical picture of anal fissure includes *triad of symptoms*: pain during or after

defecation, spasm of sphincter, and low bleeding during defecation.

Pain in patients with rectal fissures has burning character connected with moving of volumatic dense feces through anal canal. It depends also on degree of reflection of sphincter spasm, irradiates into perineum, genitalia, urinary bladder, back, and conditions disuric signs in males and dysmenorrhea in females.

Sphincter spasm during the onset of the disease may be small. In chronic course it may lead to proliferation of connective tissue.

Blood in feces appears during or after defecation, it has bright red color and is excreted mostly in small quantity.

After examination of patient with such pathology you should ask him to exert himself in relaxed sphincter, it is like defecation. In such conditions anus lowers and deep areas of skin, terminal fold and mucosa become visible. It lets us examine external area of fissure. In incredible spasm of sphincter patient is moved onto table in position on side with legs near abdomen or in "knee-elbow" position, with the help of napkins they carefully move tissues of anus toward different sides. During this in that region of anal ring where is predicted localization of fissure they examine deep skin folds. In careful investigation there may be found fissure without severe pain for patient.

Variants of clinical course and complications

Acute fissure is characterized by acute onset, presence in anal area of linear longitudinal wound with soft bottom and length 1-2 cm, width 0.3-0.5 cm, depth to 0.3 cm. Disease may have duration to two months. As a rule, acute fissures don't cause complications.

Chronic fissures may disturb patients from two months to 1 year and more. In prolonged existence fissure transforms into chronic ulcer with dense sclerozing bottom. In patients with chronic fissures there may appear complications.

Bleeding at rectal fissures is mostly low. There is observed excretion of bright red blood during or after defecation. In extraordinary cases even low but frequent bleeding from rectal fissures leads to revealed anemia.

Pectenosis is revealed in chronic fissures. At this pathology sphincter muscles are replaced by connective tissue and the patient gets coprostasis. Any examination of rectum is impossible because of narrowing of rigid anal ring.

At paraproctitis as a complication of fissure the entrance for infection is crypt through which inflammatory process is spreaded. The patient suffers from pain of pulsing character in area of rectum and perineum, fever. During palpation of perianal region there appear severe pain. Punction lets reveal purulent focus.

Malignization is characteric for prolonged fissure. In such cases pain decreases, there is observed ulcer with dense bottom with grey covering. In first portions of feces there appears blood with bad smell. Excisional biopsia with further histological investigation helps to find out the malignization of fissure.

Diagnostic program

1. Anamnesis and physical data.
2. Examination of canal by stretching of anal tissues.
3. Finger examination of rectum.
4. investigation of rectum by rectal mirror.
5. Rectoromanoscopia (contraindicated in pectenosis).
6. General analysis of blood and urine.
7. Coagulogram.
8. Sedimentation reactions (reaction of Wassermann).

Differential diagnostics

Chronic fissures of rectum should be often differentiated with other diseases that may have the same local signs.

Proctalgia. Pain is localized in the area of rectum. Consider, that the reason of proctalgia is pathology of the higher nervous system (neuroses, hysteria). At objective examination the visible organic changes are not exposed.

The anal form of non specific ulcerous colitis is characterized by the superficial damage of rectum – hyperemia and edema of mucus, formation of shallow ulcers and erosions. As a rule, non specific ulcerous colitis with all its signs begins from the distal part of rectum.

Kron's disease of rectum begins from submucosal layer, ulcers do not spread much, they mostly are like fissures that penetrate onto all depth of intestinal wall, have longitudinal and transversal directions, and may cause formation of fistules and abscesses. At histological investigation during Kron's disease there is revealed granuloma.

Cancer of rectum. Presence in anal canal of formation with not smooth edges and additions of blood in feces gives a possibility to suspect malignization of fissure. Morphological investigation of bioptates with presence of atypical cells proves malignant process.

Tactics and choice of treating method

Acute fissures are treated conservatively.

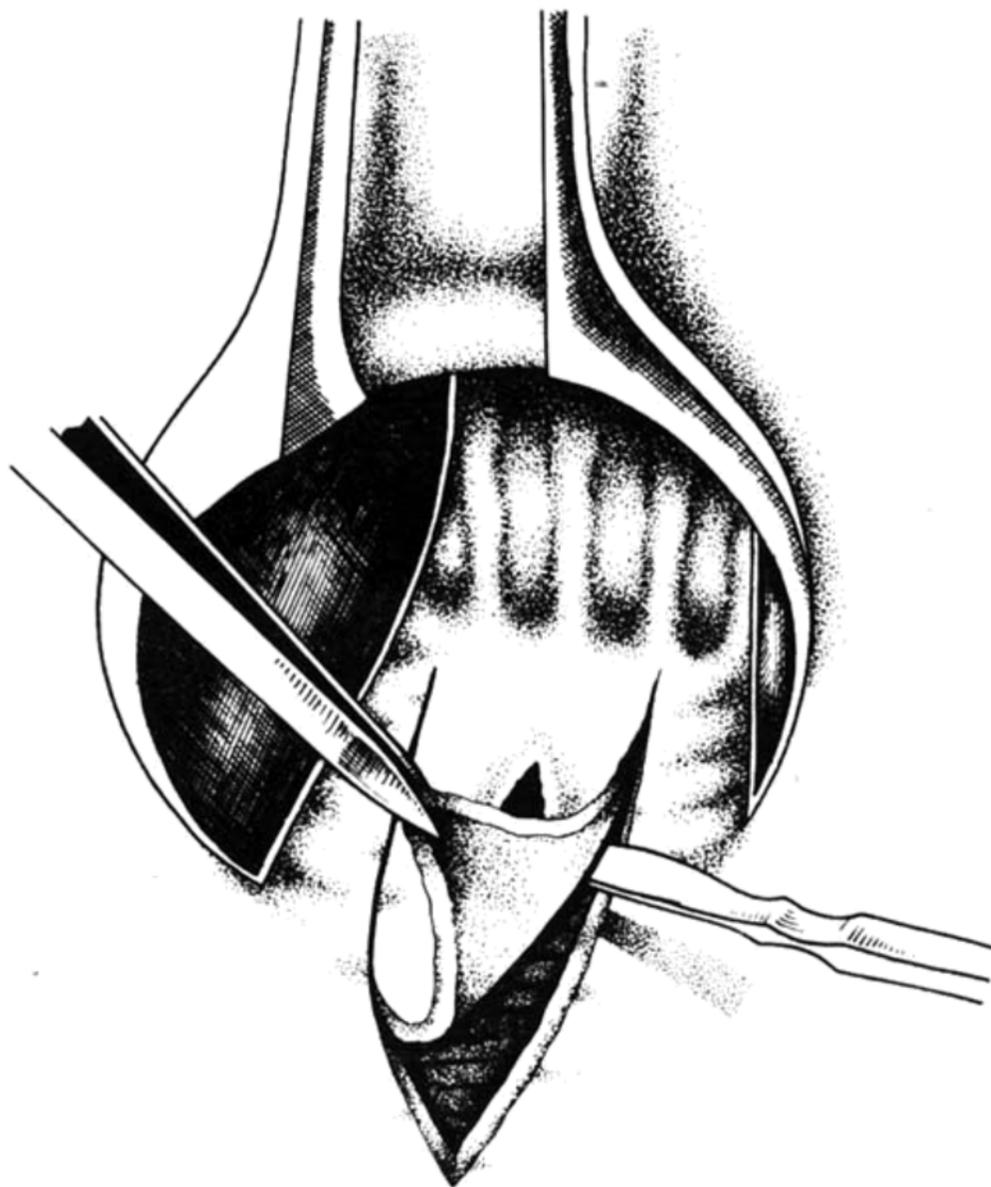
Treatment includes diet therapy, prescription of remedies and physiotherapeutic procedures.

Diet therapy means excluding from ration spicy and salty food, introduction of oral-oil substances that regulate feces.

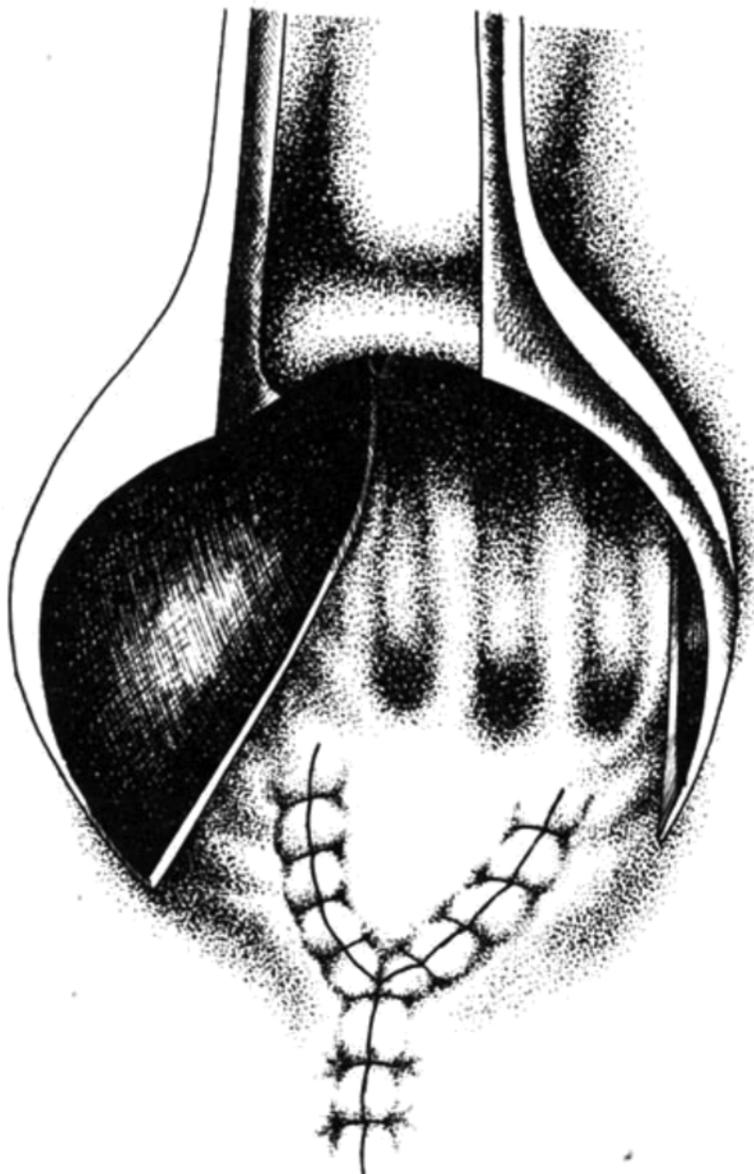
They use warm procedures (baths, hot water bottles), physiotherapeutic measures (darsonvalization, UHF, diathermia).

Medicamentous therapy means prescription of anti-spasmodic (spasmalginum, no-spa, spasmolytinum) and analgetics (promedolum, baralginum).

Rectally they use micro enemas (oily, antiseptic), rectal suppositories (with belladonna, anaesthesinum and their standard forms – proctosedylum, G preparation). Under the fissure by injection they introduce hydrocortizonum with novocainum.



Transverse cut of the mucosa above the upper merge of the Rectal fissures



End view of the operation

Indications for surgical treatment are chronic ulcer complicated by pectenosis, fistule, bleeding, and also non effective conservative treatment. From radical methods we recommend cutting of fissure in longitudinal direction including all the fissure into deleted part. Operation is added by dosed sphincterectomy. Defect of mucosa is closed in transversal direction by ligation of rectal mucosa to perianal skin.

POLYPS OF COLON AND RECTUM

Polyps are non malignant tumors on legs that grow from mucosa.

Etiology and pathogenesis

Reasons of polyp's appearance are disorders in embryonal development , inflammatory processes of mucosa and also viral infection.

Pathomorphology

Polypus of inflammatory origin differ from adenomatous ones by their incorrect form and size variety. They are soft, filled with blood, often with ulceration and hematomas. Such polypus usually combine with other signs of inflammation in intestine.

Adenomatous polypus are often part of syndromes. Thus, combination with non malignant tumors of bones, skin and soft tissues is characteristic for Gardner's syndrome, combination with focal melanosis of mucosa – for Peitz-Eggers' syndrome, combination with brain tumors – for Turco's syndrome.

Classification

By etiology:

- Innate (hereditary, family)
- Acquired (at inflammatory processes)

Separately they define also children (juvenile, innate and acquired).

By process' spreading:

- Single
- Multiple
- Total (injury of all intestines).

By external appearance and microscopic structure:

- True (glandular, fleesy, from multi layer epithelium)
- False (hypertrophic at ulceric colitis, fibrose).

Symptoms and clinical course

Men get this disease in 2-3 times more than women. Single polypus of colon and rectum have usually latent course and are often revealed accidentally.

Multiple polypus may lead to diarrhea, bleeding, changes in morphological and biochemical blood composition. At this children do not develop properly. At distal localization in some patients polypus prolapse and strangulate in anal ring causing pain. Low situated polypus irritate rectum and cause tenesmas, sometimes they cause prolapse of intestinal wall.

Among disease's symptoms first place belongs to disorders of defecation and blood in feces with further anemia, hypoproteinemia, decrease of workability. Fleesy polypus leads to disorders of water-salt and protein metabolism, because mucus' excretion at defecation may achieve 1.5 liters,

At multiple polyposis they define triad of symptoms:

1. Pigment spots (on the face, lips, mucosal membranes of cheeks, fingers, and other areas of covering epithelium).
2. Polyposis of digestive tract.
3. Hereditary character of disease.

Finger examination of rectum and observation with rectal mirror give a possibility to investigate its lower part. During this procedures you can reveal polypus of different size situated on jucosa of rectum and also polypus prolapsed with invaginate of sygmoid colon.

Irigography reveals single and multiple defects of filling (Pic. 4.1.13), so called symptom of “shooted aim” that may be seen in different parts of colon. At presence of invaginate as complication of polypus there is charactering defect of filling.

With the help of rectoromanoscope you can examine rectum and sigmoid colon on average height 30 cm, and fibrocolonoscope is to examine all parts of colon. This investigation method gives a possibility to reveal exact place of polypus’ localization, its appearance including leg width, to provide excisional byopsia and also electrocoagulation.

Variants of clinical course and complications

Polypus of small size at the onset have latent course. With their growth during moving of feces may occur disorder of their completeness with further possible complications. More often is bleeding that in most cases occur defecation. Depending on the height of polypus’ localization in intestine blood may be dark red to bright red color. Blood loss may be from small, light to severe, with signs of small and incredible anemia.

In most patients polypus have a tendense to malignization. Provided biopsia in pre operation period with morphological investigation reveals malignant transformation of tissues and proves this diagnosis not in all cases. It depends on that in what part polypus becomes malignant. Final diagnosis may be put after the operation during histological investigation of all polypus.

Polypus of big size situated in rectum usually strangulate. During this strangulation takes place on the level of anal ring and is accompanied with severe pain. Not reducible polypus may necrotize.

Polypus are situated in caecum, colon and sigmoid colon. In the most motile areas they may lead to intestinal invagination. In this case there appear spastic pain, blood excretion with feces. During abdominal palpation they reveal painful infiltrate. Invagination may prolapse into rectal lumen.

Diagnostic program

1. Anamnesis and physical data.
2. Finger investigation of rectum.
3. Investigation by rectal mirror.

4. Rectoromanoscopy.
5. Irigography.
6. Fibrocolonoscopy.
7. General analysis of blood and urine.
8. Coagulogram

Differential diagnosis

Polypus of colon and rectum should be differentiated with malignant and non epithelial tumors, non specific ulcer colitis and rectal fissures.

Cancer of right part of colon has a course with incredible anemia (toxicoanemic form) due to absorption of tumor's toxic products, their action on hemopoetic organs.

Cancer of left part of colon is usually accompanied with signs of obturative intestinal impassability. Additional examination (finger rectal examination, irigography, endoscopic methods) give a possibility to prove the diagnosis.

At chronic course of non specific ulcer colitis there is injured mucosa of intestine, and little islands of not injured mucosa between multiple big ulcers look like polypus, so called pseudopolyposis. Detailed anamnesis, specialities of clinical course of the disease, irigography and colonoscopy exclude presence of true polypus.

Not epithelial tumors (leiomyoma, lipoma) are situated under intestinal mucosa and at their small size do not cause any symptoms. With their growth mucosa is injured, there occurs bleeding with its signs. X-ray and endoscopic methods of investigation, and also histological investigation prove the final diagnosis.

Chronic fissure of rectum with not smooth edges and terminal tuberculum often simulates true polypus. Removal of this formation both with fissure and provided histological investigation help in proving of diagnosis.

Tactics and choice of treating method

Conservative method of treatment is rarely used. For this is used solution of green greater celandine for its introduction in enemas (3-4 grams of green mass for 1 kg of patient weight, on the average 50 g for adults). A celandine is ground down on a meat grinder, conduct in hot water in the ratio 1:10. A medical cycle includes 15-30 enemas. Per treatments course is conducted three cycles at intervals in one month. The low located polypuses from a stratified epithelium, pseudopolypuses and malignant polypuses are not treated by celandine.

Because of polypuses of small intestine and rectums are inclined to malignization, the basic method of its treatment is surgical .

Methods of operative treatment at polypus are divided into two groups:

1. Local operations (endoscopic electrocoagulation, polypus's removal). Indication for electrocoagulation is presence of single polypus on narrow leg, rarely – multiple polypus. Depending on the localization polypus on wide leg are removed through rectum or by laparotomy with further colotomy.
2. Radical operations as resection of separate segments of colon, right-side, left-side, subtotal colectomy or proctectomy are provided at multiple polypus.

PARAPROCTITIS

Acute paraproctitis is acute inflammation of pararectal cellular tissue. They take near 30% of all diseases of rectum.

Etiology and pathogenesis

In most cases paraproctitis is caused by polymicrobial flora. During inoculation of purulent content there are usually revealed staphylococci, E. coli, Gram positive and Gram negative rods. Causative agents of tuberculosis, actinomycosis, syphilis are rare causative agents of paraproctitis. Clostridial infection causes occurrence of gas gangrene of pelvic cellular tissue. In etiology of paraproctitis the great role belongs to penetration of infection into pararectal cellular tissue, status of organism's immunity, presence of additional diseases (diabetes mellitus). Infection may penetrate through anal glands, injured rectal mucosa, and also by hematogenic and lymphogenic way from neighbour organs injured by inflammatory process. Every anal crypt collects openings from 6-8 anal glands. Thus, anal crypt is opened gate for infection. After that follow swelling and obturation of duct connecting anal gland with anal crypt. Due to that there forms purulent cyst that opens and infection gets into perianal and perirectal spaces.

Pathomorphology

Morphologically there is defined purulent inflammation of crypts with further spreading to perirectal, ischiorectal and pelvic cellular tissue. Purulent inflammation usually is as phlegmone or (rarely) abscess.

Classification

1. By etiology – usual, anaerobic (gangrenous-putrefactive, ascendent anaerobic lymphangitis, anaerobic sepsis), specific, traumatic paraproctitis.
2. By localization – submucosal, subcutaneous, oschiorectal, pelviorectal, retrorectal paraproctitis

Separately there is defined secondary paraproctitis at which inflammatory process

spreads to pararectal cellular tissue from prostate gland or female genitalia.

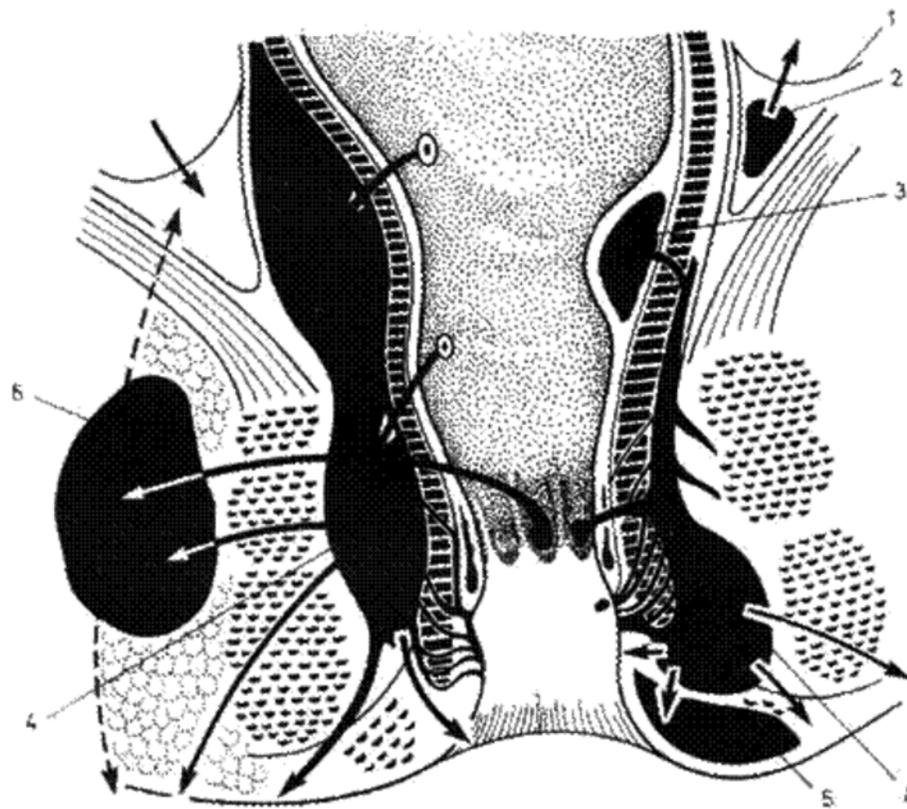
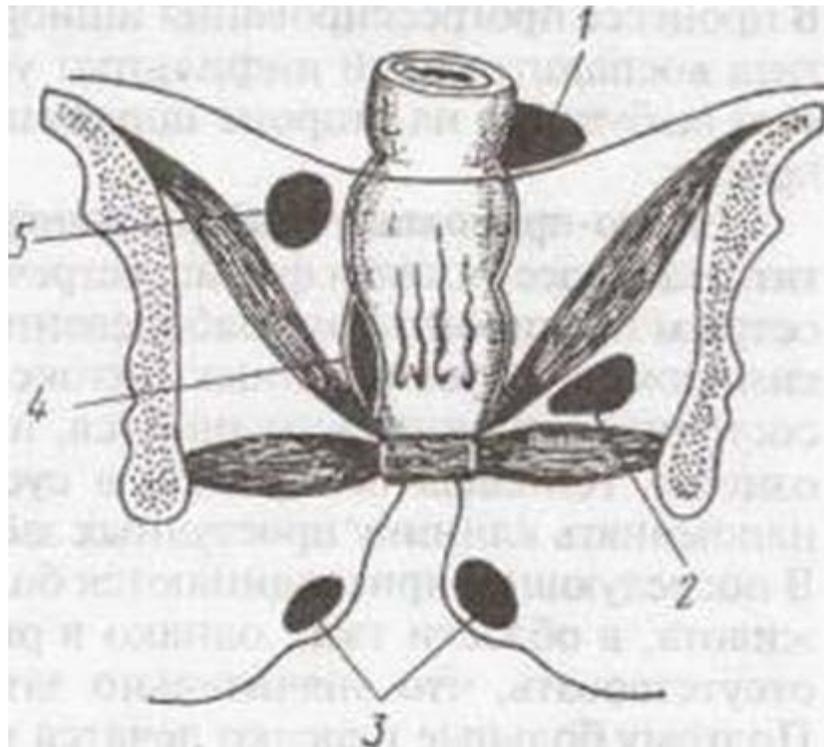
Symptoms and clinical course

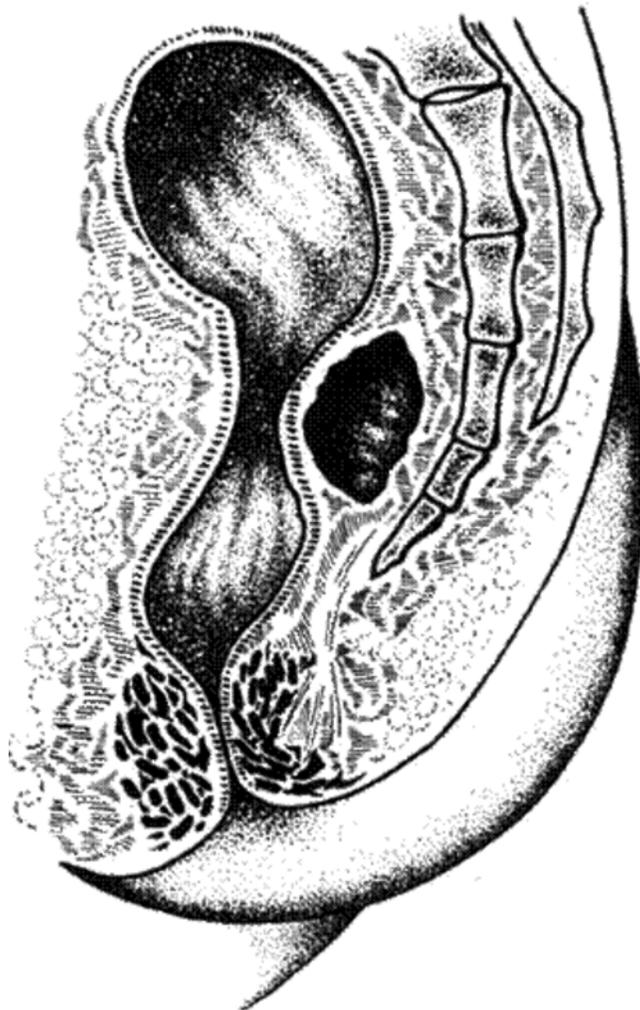
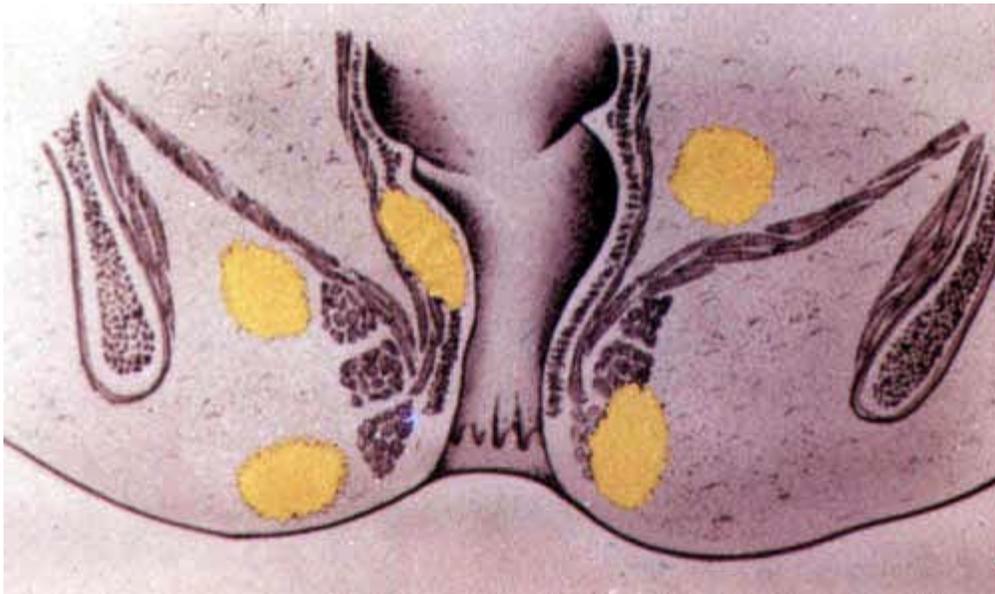
Paraproctitis has as local as general symptoms. The most often are pain in anal region of rectum, swelling, hyperemia, fluctuation, constipation, sometimes - disuria, increase of body temperature, loss of appetite and workability.

During general blood analysis there are leukocytosis with left disposition of leukocyte formula, SES increase. If in-time operative treatment shouldn't be provided, period of disease may increase to 10 days and more. After that there comes independent opening of abscess into rectum (chronic paraproctitis), formation of recurrent paraproctitis or reconvalescense.

Variants of clinical course and complications

Subcutaneous, submucoal and ischiorectal paraproctitis ar characterized mostly by typical course, there usually are no difficulties in putting of diagnosis. Pelviorectal paraproctitis as the most severe form is not revealed exactly. At first pain in inflammated area is not felt, disease starts from headache, fever, increase of body temperature. Sometimes on this stage of disease there is put such diagnosis as influenza. After that appears pain in lower part of pelvis that irradiates into uterus, urinary bladder causing disorders of urination. Patients get treatment in urologist, gynecologist and therapist for a long time. If inflammatory infiltrate of pelviorectal cellular tissue transforms into abscess, disease becomes acute. External signs are revealed at spreading of purulent process onto ischiorectal and subcutaneous cellular tissue. It lasts in limits of one place. At the same time, processes may spread onto other pelvis part and form horseshoe shaped paraproctitis. Retrorectal paraproctitis as a kind of pelviorectal from the very beginning is accompanied with pain. During this pain is concentrated in rectum, coccygeal area, it is increased in sitting position of patient and during defecation. At this localization of abscess there may be two-side horseshoe shaped injury. Paraproctitis with clostridial infection is characterized from the beginning with sever intoxicaton, high temperature.





Palpatory in this regions there is felt crepitation. Acute paraproctitis may be complicated by fistulas, phlegmones, lymphangitis, sepsis. At clostridial infection not in time and incomplete treatment may lead to death.

Diagnostic program

1. Anamnesis and physical data.
2. Examination of anal area and anal canal.
3. Finger examination.
4. Investigation by rectal mirror.
5. Rectoromanoscopy.
6. X-ray examination of ischial areas in lateral position.
7. Bacteriogram of purulent content.
8. General analysis of blood and urine.
9. Biochemical analysis of blood.
10. Coagulogram.
11. Sedimental reactions (reaction of Wassermann).

Differential diagnosis

Paraproctitis is differentiated with hemorrhoids, purulent dermoid cysts, suppuration of epithelial paracoccygeal canals, cancer of rectum, tumors and inflammatory diseases of sacral bone.

Acute hemorrhoids are accompanied with severe pain, swelling of perianal area, formation of thrombs in nodes, nodes' necrosis.

Suppurated presacral dermoid cyst has no connection with rectum, and paraproctitis usually does. Pararectal abscess is always connected with rectum in area of anal crypts. If suppurated cyst empties into intestine, then fistula's direction is beyond the linea dentate.

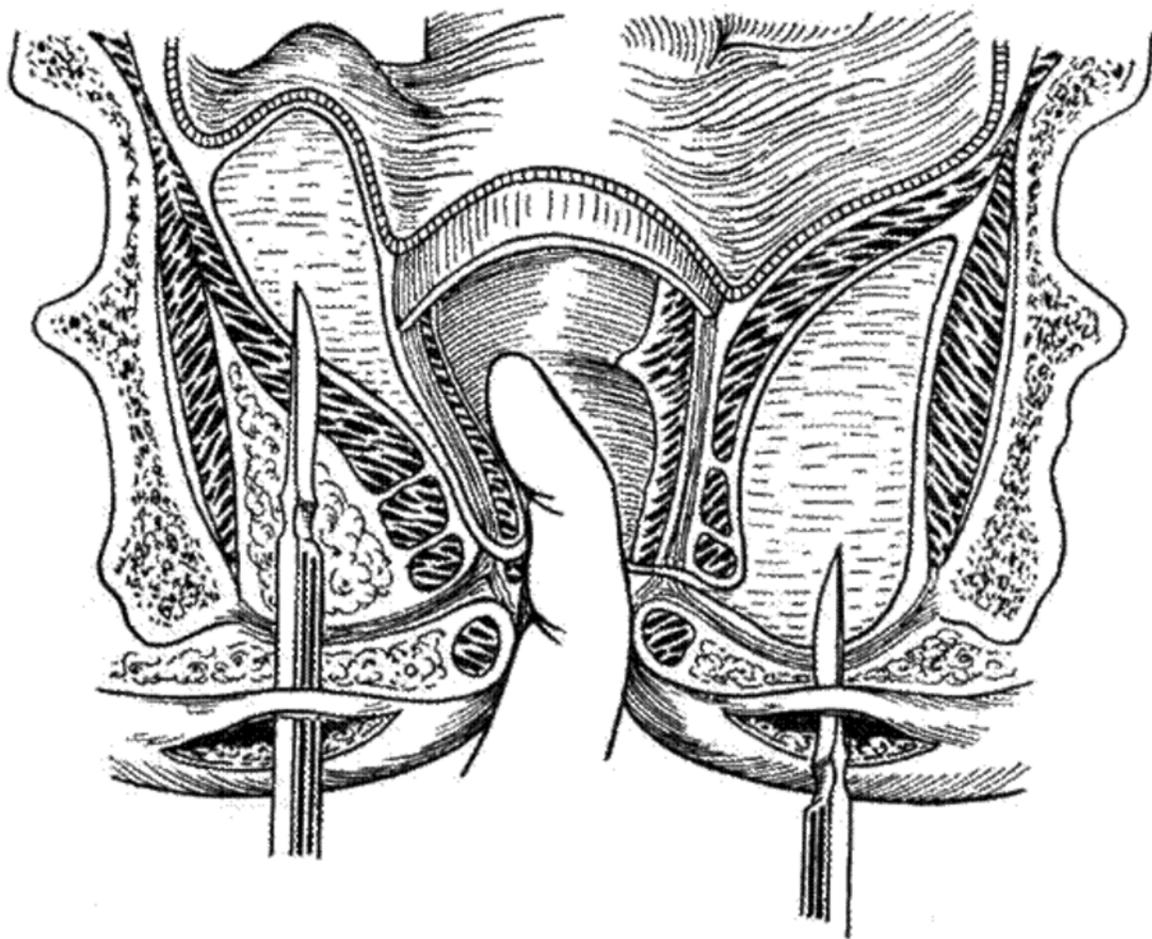
Suppurated epithelial pericoccygeal canals are characterized by presence of point openings on coccygeal level, fistula's direction is beyond rectum.

Cancer of rectum at the onset of the disease is usually accompanied with no pain. During examination of rectum there is revealed dense formation. Data of punctional biopsia help to prove the diagnosis.

Diseases of sacral bone as tumoral as inflammatory of the basis of osteomyelitis with injury of bone tissue structure are revealed by X-ray examination.

Tactics and choice of treating method

Method of choice for treatment of acute paraproctitis is surgical. But on early stages of pelviorectal paraproctitis with deep infiltration of tissues surrounding rectum, without signs of softening there is indicated conservative treatment (warming compress on area of perineum with 20% spiritus aethylicus, antibiotics of wide spectrum of stion, lumbal novocainum blockade, strict bed regime, exclusion of cellulose from food, usage of cleaning enemas). Surgical treatment means early operation by opening of abscess by semilunar incision with dreanging and liquidation of its inner opening (removal of crypt) through which abscess' cavity is connected with rectum.



Opening ischioanal paraprocitis.

At paraprocitis there may be observed transsphincter and extrasphincter directions of fistulas, at pelviorectal and retrorectal – extrasphincter. Important thing is choice of way of big abscess' opening: through skin or mucosa from rectum. With this aim before opening of abscess by thick needle you should provide puncture of infiltrated area and if exudates is got you should provide the incision. If pus of ischioanal area is spreaded on subcutaneous cellular tissue and there are changes in shape of buttock, fluctuation, question of surgical operation has no doubt. At retrorectal paraprocitis there makes sense to open the abscess from rectal lumen. If in this case there functions inner fistule, it usually brings no troubles for patients (Pic. 4.1.7).

At signs of anaerobic infection they provide wide multiple incisions, necrectomia, wound cavity is washed by solutions of oxidants (permanent washing), you should use antibiotics of wide action spectrum, polyvalent anti-gangrenous serum (250-300,000 units 1 time per 2-3 days), desintoxicative therapy, oxygen barotherapy (10 procedures at pressure 2 atmospheres), introduction of fresh blood, albumine, plasma, hemodes, rheopolyglucinum. At diabetes mellitus they provide insulin therapy and introduction of metrogilum.

After the operation there may appear early complications: bleeding from wound

bottom, especially at anaerobic paraproctitis, and late – insufficiency of distal part of rectum, recidivum of paraproctitis, formation of fistules. If bleeding occurs, they provide tamponade of these areas, and if it does not stop – bleeding places are ligated. At insufficiency of sphincter there should be provided one more operation: renewal of sphincter completeness by Π -like ligations.

RECTAL FISTULAS (CHRONIC PARAPROCTITIS)

Rectal fistulas are tubular purulent canals in cellular tissue surrounding rectum and anus.



Etiology and pathogenesis

Rectal fistulas occur mostly on the basis of acute paraproctitis. Reasons of chronic fistulas are:

- opening of purulent paraproctitis without cutting of crypt;
- shortening of external anal sphincter at which fistula's canal is pressed and

excretion of its content stops;

- decreased resistance to infection and low tissue regeneration;
- epithelization of coccygeal canals.

Classification

I. By etiology and pathogenesis:

1. Innate.
2. Acquired (traumatic, inflammatory, tumoral).

II. By infection character:

1. Vulgaric.
2. Anaerobic.
3. Specific (tuberculous, syphilitic, actinomycotic etc.)

III. By anatomical signs:

1. Depending on connection with intestinal lumen (complete, incomplete, internal, external).
2. By correlation to external sphincter (intrasphincter, extrasphincter, transsphincter).
3. Depending on primary localization of inflammatory process (subcutaneous, submucosal, ischiorectal, pelviorectal).
4. Depending on localization of external and internal fistula's canals (cutaneous, marginal, on crypt level).
5. By fistula's shape (simple – direct, complex – curve and containing cavities).

There define 4 degrees of extrasphincter fistulas:

I degree – scar and inflammatory changes are absent.

II degree – scar process around inner fistula's opening without inflammatory changes in pararectal cellular tissue.

III degree – purulent cavities or infiltrates in pararectal cellular tissue without scars around inner opening.

IV degree – incredible infiltrates or purulent cavities in pararectal cellular tissue and big scar process around inner opening.

Symptoms and clinical course

Self feeling and general status of patient with chronic course of paraproctitis in most cases is satisfying. At long time existing inflammatory focus workability decreases, increased irritability appears, sleep becomes worse. Depending on activity of inflammatory process, character of excretions from fistula changes. After abscess opening pain decreases and may be back when process becomes acute. In most patients with rectal fistulas there are observed signs of proctosigmoiditis and chronic recurrent paraproctitis.

When process becomes acute pain appears and temperature increases. There is formed purulent focus with formation of new fistula's canals.

In most patients fistula situated near anal ring goes inside the sphincter. Fistula situated 4-5 cm from anus and deeper is situated mostly outside the sphincter and may be of IV degree.

Variants of clinical course and complications

Patients with fistulas feel rush and heaviness in areas of rectum and anus. Long existing fistulas are accompanied with scar changes of anal ring (pectenosis) that difficult defecation. After fistula's closing (between acute periods) patients have no troubles and feel healthy. In some patients there stay painful infiltrates where inflammatory process may renew. The most dangerous and rare complication of chronic paraproctitis is malignant transformation of rectal fistulas.

Diagnostic program

1. Anamnesis and physical data.
2. Examination of anal area and anal canal.
3. Finger examination of rectum.
4. Investigation by rectal mirror.
5. Rectoromanoscopy.
6. Bacteriogram of purulent content.
7. General analysis of blood and urine.
8. Biochemical analysis of blood.
9. Sedimental reactions (reaction of Wassermann).
10. Contrast fistulography.
11. Introduction of catheter into fistula's canal.

Differential diagnosis

Chronic paraproctitis are differentiated with suppuration of epithelial paracoccygeal canals, diseases of sacral bone, fissures of rectum and suppurated presacral dermoid cysts.

Suppurated epithelial paracoccygeal canals are primary openings of fistulas in area of intergluteal folds, and fistula's canals are situated behind the dental line and are not connected with rectum.

Diseases of sacral bone on the basis of osteomyelitis are recognized by X-ray method. At this there are revealed disorders of bone structure.

Fissures of rectal mucosa are accompanied with severe pain, bleeding and spasms of sphincter. Presence of wound mostly on posterior commissure proves the diagnosis.

Suppurated presacral dermoid cyst has no connection with rectum. Even if it empties into rectum, fistula's canal is always situated beyond the linea dentata.

Tactics and choice of treating method

At conservative treatment of chronic paraproctitis (fistulas) they use sitting baths and warming compresses with 20% spiritus aethylicus. They also prescribe physiotherapeutic procedures (ultraviolet radiation, local darsonvalization, electrophoresis with 1% solution of potassium iodide, 1% novocainum solution), lavage of fistula with diluted solutions of antiseptics.

Operative treatment is indicated if fistula is present for a long time or closes for some time and then opens again after acute inflammatory period. In patients with intrasphincter fistulas there is used operation by Gabriel. Its basis is that fistula is cut from inner to external opening. Skin that covers the fistula is cut as triangle. Its peak includes internal opening, and basis is situated outside (Pic. 4.1.8).

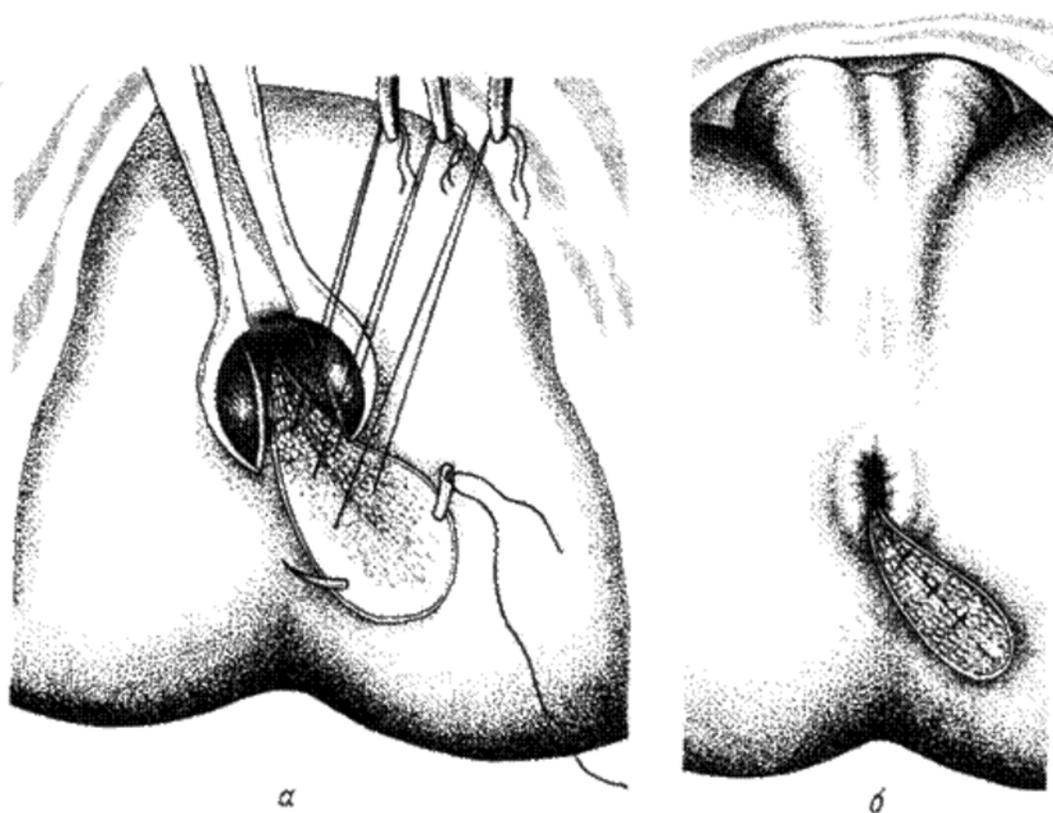
In case of fistula going through inner parts of sphincter (transsphincter fistula) during canal removal there are injured fibers of sphincter. For renewal of cut sphincter there are used node of II-like ligations. At extrasphincter fistulas there are used such operations:

Ryzhykh-I – they remove fistula's canal in perineal wound to rectal wall and cut in basis. Stump of fistula's canal is emptied with Folkmann's spoon, cleaned with iodine solution and ligated with two or three layers of catgut ligations. During this ligated stump of fistula's canal is covered by surrounding tissues. Operation is finished with dosed sphincterotomy of inner fibers. This method is used when inner fistula's opening is localized in posterior crypt.

Ryzhykh-II (second variant) is used when inner fistula's opening is localized in anterior crypt or on lateral wall. Removal of fistula in perineal wound is provided the same as in the first variant. Further, upon the inner fistula's opening there is separated piece of mucosa (width 1-1.5 cm and length to 4 cm). Inner fistula's opening is ligated with node catgut ligations. With this aim threads are knotted after removal of rectal mirror. Then there should be put several ligations between separated and partially cut pieces of mucosa and distal part of the wound. At the end of operation there is provided posterior dosed sphincterotomy.

By Blinnitchev, opening in rectal mucosa is closed by catgut ligations in two stairs. There is separated mucosa upon the ligated opening, it should be fixed including submucosal layer to muscular membrane and ligated by silk ligations to perianal skin. Sometimes mobilized piece is moved downwards. Mobilized mucosal-muscular piece is ligated in such a way that needle should go near the edge of terminal fold to the basis of mobilized piece. After that by separate silk ligations they fix piece's edge to perianal skin.

Ligation method (by Hippocrates). On the wall of anal canal there should be cut inner fistula's opening and perianal skin together with narrow stripe of mucosa. Into this layer there are put ligations No.6 and sphincter fibers are tightened. Thus, thread stretches with 1-1.5 cm width intestinal wall and sphincter fibers. Ligation is tightened again till complete cutting of tissues and sphincter with cellular tissue (Pic. 4.1.9).



At extrasphincter fistulas of I degree there is used operation of Ryzhykh of Blinnitchev, or removal of fistula with ligation of sphincter. At fistulas of II degree fistulas are removed with ligation of sphincter, at III degree – operation of Blinnitchev or ligation method. At fistulas of IV degree there is used ligation method.

EPITHELIAL PARACOCCHIDIOIDAL CANALS

The reasons of origin of paracocchidioidal ducts are: innate defect of development, remain of spinal chord, violation of reduction of coccygeal vertebrae with stretching of tail ligament, theory of moving cells. In the mechanism of occurrence of canal inflammation there play role as trauma as infection penetration. In most cases beginning of inflammatory process in epithelial canals occurs at the same time with period of sexual development (hormonal-endocrine rebuilding of organism).



Classification

There define such stages of course of epithelial paracoccygeal canals (by Yu.V. Dooltsev and L.V. Ryvkin, 1988):

- I. Epithelial paracoccygeal canals without any clinical findings.
- II. Acute inflammation of epithelial paracoccygeal canals:
 - (a) infiltrative stage;
 - (b) abscess formation.
- III. Chronic inflammation of epithelial paracoccygeal canals:
 - (a) infiltrative stage;
 - (b) recurrent abscess;
 - (c) purulent fistula.
- IV. Remission of inflammation of epithelial paracoccygeal canals.

Symptoms and clinical course

Epithelial paracoccygeal canals may have no clinical findings, that's why they are often revealed accidentally during medical examination. In such cases I distal part of intergluteal fold near coccygeal apex upon the edge of anal region there are seen one or several point openings where sometimes hair grows. Diameter of those openings is from 1 to 3 mm, they are situated by middle line one by one. During pressing on the skin upper from epithelial opening you can see excretion of drops of non transparent or purulent fluid. Self feeling and general status of patients are usually not changed. But when there appears also inflammation in coccygeal region there occurs abscess formation. Then patients feel pain, body temperature increases. Investigation of general blood analysis reveals leukocytosis with formula moving to the left, increase of SES.

Variants of clinical course and complications

At non complicated epithelial paracoccygeal canals patients suffer from dull pain, feeling of pressing in coccygeal region, especially during long time walking. Increased sweating in intergluteal region is accompanied with rush, sometimes there appears painful infiltrate that disappears independently.

Acute stage of the disease is characterized by changes of general and local character. Body temperature, as a rule, increases to 39-40 degrees centigrade and is accompanied with chilling. Local symptoms are severe pain in coccygeal region, sometimes with irradiation into rectum. At the same time there appear swelling, infiltration of intergluteal region, skin upon the place of inflammation becomes bluish-violet. Further, abscess is formed. During independent opening of abscess there goes purulent content of dark color with bad smell. Sometimes abscess from coccygeal region spreads to pararectal and even pelviorectal spaces, but as a rule it does not open into rectal lumen.

Diagnostic program

1. Anamnesis and physical data.
2. Examination of coccygeal and anal regions.
3. Finger examination of rectum.
4. Investigation by rectal mirror.
5. Rectoromanoscopy.
6. Bacteriogram of purulent content.
7. General analysis of blood and urine.
8. Biochemical analysis of blood.
9. Sedimental reactions (reaction of Wassermann).
10. Contrast fistulography.
11. X-ray examination of pelvis bones.

Differential diagnosis

Presacral cyst teratoma both with epithelial paracoccygeal canal at the initial stage and period of complications' absence are differentiated easily, because when teratoma is present, finger examination of rectum gives a possibility to reveal tumor-like formation in presacral region. Complication of cyst teratoma with fistulas is differentiated with suppurated paracoccygeal canal with incredible difficulties. Provided contrast fistulography of fistula's canals at cyst teratoma gives a possibility to define its localization in presacral region, and epithelial canal may often be branched and ends blindly in soft tissues behind the coccygeum.

At paraproctitis epithelial opening in intergluteal fold is usually absent, and fistula's

canal in most cases is connected with rectal lumen and is revealed during finger examination or investigation by rectal mirror. At pressing on pararectal region from fistula's canals there appears purulent content. Preliminarily introduced tampon is stained by methylene blue at the moment of its getting into external opening of fistula.

Osteomyelitis of pterygoideal bone and coccygeum is often connected with injury of this region. On observing X-ray grams there is revealed characteristic picture of injured bone (foci of osteoporosis and sequesters). Fistulography helps in proving of diagnosis. Two independent diseases – epithelial paracoccygela canals and osteomyelitis of sacral bone and coccygeum – at the same time may be combined in one patient extremely rarely.

Tactics and choice of treating method

In treatment of epithelial paracoccygela canals surgical method is preferred. Only in stage of infiltrate with the aim of stopping of further inflammation development they provide novocainum blockade (100 ml of 0.25% solution with antibiotics). Every day during 5-6 days there should be applied compresses with Vishnevskiy's ointment, physiotherapeutic methods are used (UHF, diathermia, ultraviolet rays).

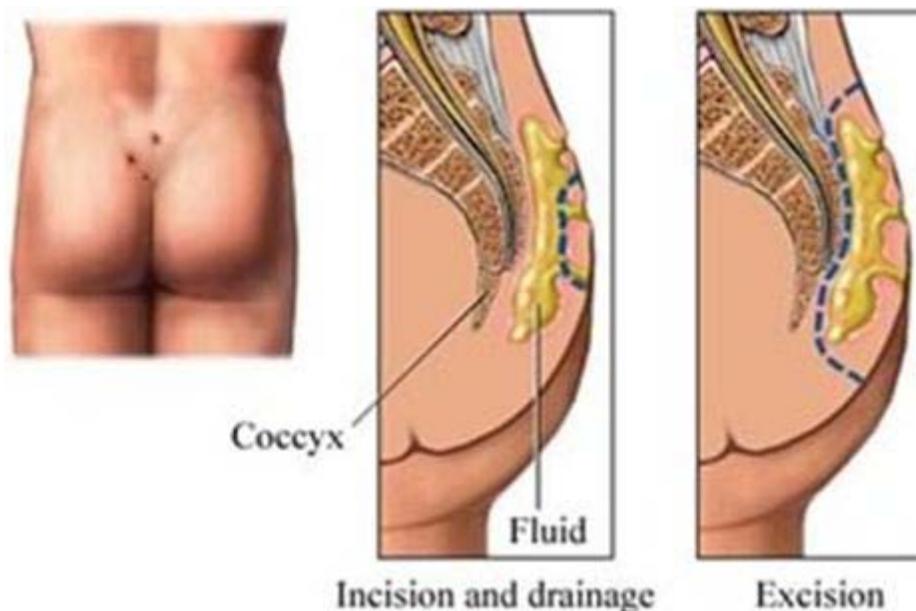
Important meaning in preparation to operation belongs to sanation of purulent fistula's canals. After inoculation of excretion from canals, determination of microflora and sensibility to antibiotics they provide pre operational sanation by some remedies.

In case of abscess formation of paracoccygeal canals abscess is opened in urgent order. During this they use two main positions of patient on operation table: on the abdomen and on the side. When patient lays on back (by Depage) feet end of the table is moved downwards on 45 degrees. Angle corresponds to projection of coxal articulations. Legs are moved to the sides and feet are situated on the support. Such position in most cases is used during operations under local anesthesia. General anaesthesia is used in position on the side. Patient lays on right side with legs bended in coxal articulations. Left leg is situated on the right, during this left thigh is near abdomen. After anesthesia by two semilunar incisions along the middle line surgeon opens the abscess. If it spreads laterally from intergluteal fold it should be opened and removed too. In average 10% of patients where abscess included epithelial canal and got proteolytic action of pus, after not palliative operation of abcess opening there was no recurrent and reconvalescence became. Thus in 90% cases during abscess opening there stay walls of epithelial canal and its branches, there is formed secondary fistula, but after lowering of inflammatory process there is needed one more radical surgical operation. It includes removal of epithelial canal, its branches together with infiltrated surrounding tissues, purulent formations, with this aim they introduce methylene blue preliminary. Further, depending on presence or absence of infiltrate and abscesses, quantity of fistulas, radical operation is finished by one of methods: complete closing of wound, opened wound tamponade, ligation of wound edges to the bottom. When epithelial canals are revealed accidentally or in small symptoms of inflammation, absence of infiltrates, abscesses wound is closed completely including its bottom (Pic. 4.1.10).

In rare cases when after opening and removal of abscesses there stays big wound surface, wound should be treated by opened method. For sanitation of such wounds, except usual remedies, they use laser and ultrasound.

At abscesses and secondary fistulas in most cases there should be provided radical operation: wound edges are to be ligated to its bottom. During removal of lateral fistula's canals because of absence of fossa in this region there are applied rare situational ligations onto wound edges not including the bottom.

In surgical practice in treatment epithelial paracoccygeal canals there should be taken into account anatomical variants of structure of sacral-coccygeal-gluteal region. In most patients there may be high position of buttocks, deep intergluteal fold, acute angle between buttocks and middle line of sacrococcygeal region, close position of primary epithelial opening from anus.



After incision of suppurated paracoccygeal canal before ligation of wound edges to its bottom there appears need to remove subcutaneous cellular tissue through all length from both wound sides. During this narrow skin pieces easily and closely situate during their ligation to wound bottom. The last in this case is healed with narrow scar.

In patients with middle and low localization of buttocks with more dull angle of position, less depth of intergluteal fold, higher position of epithelial paracoccygeal canal after its removal wound edges are ligated to the bottom in such a way that ligation includes skin, cellular skin, bottom and go outside through cellular tissue on the opposite side 1-1.5 cm higher than wound bottom.

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POLYPOSIS. DIVERTICULOSIS. UNSPECIFIC ULCEROUS COLITIS. CROHN'S DISEASE.

POLYPOSIS

Polyps are non malignant tumors on legs that grow from mucosa.

Etiology and pathogenesis

Reasons of polyp's appearance are disorders in embryonal development , inflammatory processes of mucosa and also viral infection.

Pathomorphology

Polypus of inflammatory origin differ from adenomatous ones by their incorrect form and size variety. They are soft, filled with blood, often with ulceration and hematomas. Such polypus usually combine with other signs of inflammation in intestine.

Adenomatous polypus are often part of syndromes. Thus, combination with non malignant tumors of bones, skin and soft tissues is characteristic for Gardner's syndrome, combination with focal melanosis of mucosa – for Peitz-Eggers' syndrome, combination with brain tumors – for Turco's syndrome.

Classification

By etiology:

- Innate (hereditary, family)
- Acquired (at inflammatory processes)

Separately they define also children (juvenile, innate and acquired).

By process' spreading:

- Single
- Multiple
- Total (injury of all intestines).

By external appearance and microscopic structure:

- True (glandular, fleesy, from multi layer epithelium)
- False (hypertrophic at ulceric colitis, fibrose).

Symptoms and clinical course

Men get this disease in 2-3 times more than women. Single polypus of colon and rectum have usually latent course and are often revealed accidentally.

Multiple polypus may lead to diarrhea, bleeding, changes in morphological and biochemical blood composition. At this children do not develop properly. At distal localization in some patients polypus prolapse and strangulate in anal ring causing pain. Low situated polypus irritate rectum and cause tenesmas, sometimes they cause prolapse of intestinal wall.

Among disease's symptoms first place belongs to disorders of defecation and blood in feces with further anemia, hypoproteinemia, decrease of workability. Fleesy polypus leads to disorders of water-salt and protein metabolism, because mucus' excretion at defecation may achieve 1.5 liters,

At multiple polyposis they define triad of symptoms:

1. Pigment spots (on the face, lips, mucosal membranes of cheeks, fingers, and other areas of covering epithelium).
2. Polyposis of digestive tract.
3. Hereditary character of disease.

Finger examination of rectum and observation with rectal mirror give a possibility to investigate its lower part. During this procedures you can reveal polypus of different size situated on jucosa of rectum and also polypus prolapsed with invaginate of sygmoid colon.

Irigography reveals single and multiple defects of filling, so called symptom of "shooted aim" that may be seen in different parts of colon. At presence of invaginate as complication of polypus there is charactering defect of filling.

With the help of rectoromanoscope you can examine rectum and sigmoid colon on average height 30 cm, and

fibrocolonoscope is to examine all parts of colon. This investigation method gives a possibility to reveal exact place of polypus' localization, its appearance including leg width, to provide excisional biopsy and also electrocoagulation.

Variants of clinical course and complications

Polypus of small size at the onset have latent course. With their growth during moving of feces may occur disorder of their completeness with further possible complications. More often is bleeding that in most cases occur defecation. Depending on the height of polypus' localization in intestine blood may be dark red to bright red color. Blood loss may be from small, light to severe, with signs of small and incredible anemia.

In most patients polypus have a tendency to malignization. Provided biopsy in pre operation period with morphological investigation reveals malignant transformation of tissues and proves this diagnosis not in all cases. It depends on that in what part polypus becomes malignant. Final diagnosis may be put after the operation during histological investigation of all polypus.

Polypus of big size situated in rectum usually strangulate. During this strangulation takes place on the level of anal ring and is accompanied with severe pain. Not reducible polypus may necrotize.

Polypus are situated in caecum, colon and sigmoid colon. In the most motile areas they may lead to intestinal invagination. In this case there appear spastic pain, blood excretion with feces. During abdominal palpation they reveal painful infiltrate. Invagination may prolapse into rectal lumen.

Diagnostic program

1. Anamnesis and physical data.
2. Finger investigation of rectum.
3. Investigation by rectal mirror.
4. Rectoromanoscopy.
5. Irigography.
6. Fibrocolonoscopy.
7. General analysis of blood and urine.
8. Coagulogram



Polyps. Endoscopic view.

Differential diagnosis

Polypus of colon and rectum should be differentiated with malignant and non epithelial tumors, non specific ulcer colitis and rectal fissures.

Cancer of right part of colon has a course with incredible anemia (toxicoanemic form) due to absorption of tumor's toxic products, their action on hemopoetic organs.

Cancer of left part of colon is usually accompanied with signs of obturative intestinal impassability. Additional examination (finger rectal examination, irigography, endoscopic methods) give a possibility to prove the diagnosis.

At chronic course of non specific ulcer colitis there is injured mucosa of intestine, and little islands of not injured mucosa between multiple big ulcers look like polypus, so called pseudopolyposis. Detailed anamnesis, specialities of clinical

course of the disease, irigography and colonoscopy exclude presence of true polypus.

Not epithelial tumors (leiomyoma, lipoma) are situated under intestinal mucosa and at their small size do not cause any symptoms. With their growth mucosa is injured, there occurs bleeding with its signs. X-ray and endoscopic methods of investigation, and also histological investigation prove the final diagnosis.

Chronic fissure of rectum with not smooth edges and terminal tuberculum often simulates true polypus. Removal of this formation both with fissure and provided histological investigation help in proving of diagnosis.

Tactics and choice of treating method

Conservative method of treatment is rarely used. For this is used solution of green greater celandine for its introduction in enemas (3-4 grams of green mass for 1 kg of patient weight, on the average 50 g for adults). A celandine is ground down on a meat grinder, conduct in hot water in the ratio 1:10. A medical cycle includes 15–30 enemas. Per treatments course is conducted three cycles at intervals in one month. The low located polypuses from a stratified epithelium, pseudopolypuses and malignant polypuses are not treated by celandine.

Because of polypuses of small intestine and rectums are inclined to malignization, the basic method of its treatment is surgical .

Methods of operative treatment at polypus are divided into two groups:

1. Local operations (endoscopic electrocoagulation, polypus's removal). Indication for electrocoagulation is presence of single polypus on narrow leg, rarely – multiple polypus. Depending on the localization polypus on wide leg are removed through rectum or by laparotomy with further colotomy.
2. Radical operations as resection of separate segments of colon, right-side, left-side, subtotal colectomy or colectomy are provided at multiple polypus. [video](#) [video2](#) [video3](#)

EMBRYOLOGY AND ANATOMY

Embryology

The embryonic gastrointestinal tract begins developing during the fourth week of gestation. The primitive gut is derived from the endoderm and divided into three segments: *foregut*, *midgut*, and *hindgut*. Both *midgut* and *hindgut* contribute to the colon, rectum, and anus.

The *midgut* develops into the small intestine, ascending colon, and proximal transverse colon, and receives blood supply from the superior mesenteric artery. During the sixth week of gestation, the midgut herniates out of the abdominal cavity, and then rotates 270 degrees counterclockwise around the superior mesenteric artery to return to its final position inside the abdominal cavity during the tenth week of gestation.

The *hindgut* develops into the distal transverse colon, descending colon, rectum, and proximal anus, all of which receive their blood supply from the inferior mesenteric artery. During the sixth week of gestation, the distal-most end of the hindgut, the *cloaca*, is divided by the urorectal septum into the urogenital sinus and the rectum.

The distal anal canal is derived from ectoderm and receives its blood supply from the internal pudendal artery. The dentate line divides the endodermal hindgut from the ectodermal distal anal canal.

Anatomy

The large intestine extends from the ileocecal valve to the anus. It is divided anatomically and functionally into the *colon*, *rectum*, and *anal canal*. The wall of the colon and rectum comprise five distinct layers: mucosa, submucosa, inner circular muscle, outer longitudinal muscle, and serosa. In the colon, the outer longitudinal muscle is separated into three *teniae coli*, which converge proximally at the appendix and distally at the rectum, where the outer longitudinal muscle layer is circumferential. In the distal rectum, the inner smooth-muscle layer coalesces to form the internal anal sphincter. The intraperitoneal colon and proximal one third of the rectum are covered by serosa; the mid and lower rectum lack serosa.

Colon Landmarks

The colon begins at the junction of the terminal ileum and cecum and extends 3 to 5 feet to the rectum. The rectosigmoid junction is found at approximately the level of the sacral promontory and is arbitrarily described as the point at which the three *teniae coli* coalesce to form the outer longitudinal smooth muscle layer of the rectum. The *cecum* is the widest diameter portion of the colon (normally 7.5 to 8.5 cm) and has the thinnest muscular wall. As a result, the cecum is most vulnerable to perforation and least

vulnerable to obstruction. The ascending colon is usually fixed to the retroperitoneum. The hepatic flexure marks the transition to the transverse colon. The intraperitoneal transverse colon is relatively mobile, but is tethered by the gastrocolic ligament and colonic mesentery. The greater omentum is attached to the anterior/superior edge of the transverse colon. These attachments explain the characteristic triangular appearance of the transverse colon observed during colonoscopy. The splenic flexure marks the transition from the transverse colon to the descending colon. The attachments between the splenic flexure and the spleen (the lienocolic ligament) can be short and dense, making mobilization of this flexure during colectomy challenging. The descending colon is relatively fixed to the retroperitoneum. The sigmoid colon is the narrowest part of the large intestine and is extremely mobile. Although the sigmoid colon is usually located in the left lower quadrant, redundancy and mobility can result in a portion of the sigmoid colon residing in the right lower quadrant. This mobility explains why volvulus is most common in the sigmoid colon and why diseases affecting the sigmoid colon, such as diverticulitis, may occasionally present as right-sided abdominal pain. The narrow caliber of the sigmoid colon makes this segment of the large intestine the most vulnerable to obstruction.

Colon Vascular Supply

The arterial supply to the colon is highly variable (Fig. 28-1). In general, the *superior mesenteric artery* branches into the *ileocolic artery* (absent in up to 20% of people), which supplies blood flow to the terminal ileum and proximal ascending colon, the *right colic artery*, which supplies the ascending colon, and the *middle colic artery*, which supplies the transverse colon. The *inferior mesenteric artery* branches into the *left colic artery*, which supplies the descending colon, several *sigmoidal branches*, which supply the sigmoid colon, and the *superior rectal artery*, which supplies the proximal rectum. The terminal branches of each artery form anastomoses with the terminal branches of the adjacent artery and communicate via the *marginal artery of Drummond*. This arcade is complete in only 15 to 20% of people.

Colon Lymphatic Drainage

The lymphatic drainage of the colon originates in a network of lymphatics in the muscularis mucosa. Lymphatic vessels and lymph nodes follow the regional arteries. Lymph nodes are found on the bowel wall (epicolic), along the inner margin of the bowel adjacent to the arterial arcades (paracolic), around the named mesenteric vessels (intermediate), and at the origin of the superior and inferior mesenteric arteries (main). The *sentinel lymph nodes* are the first one to four lymph nodes to drain a specific segment of the colon, and are thought to be the first site of metastasis in colon cancer. The utility of sentinel lymph node dissection and analysis in colon cancer remains controversial.

Colon Nerve Supply

The colon is innervated by both *sympathetic* (inhibitory) and *parasympathetic* (stimulatory) nerves, which parallel the course of the arteries. Sympathetic nerves arise from T6-T12 and L1-L3. The parasympathetic innervation to the right and transverse colon is from the vagus nerve; the parasympathetic nerves to the left colon arise from sacral nerves S2-S4 to form the *nervi erigentes*.

Anorectal Landmarks

The rectum is approximately 12 to 15 cm in length. Three distinct submucosal folds, the *valves of Houston*, extend into the rectal lumen. Posteriorly, the *preseacral fascia* separates the rectum from the presacral venous plexus and the pelvic nerves. At S4, the *rectosacral fascia (Waldeyer's fascia)* extends forward and downward and attaches to the fascia propria at the anorectal junction. Anteriorly, *Denonvilliers' fascia* separates the rectum from the prostate and seminal vesicles in men and from the vagina in women. The *lateral ligaments* support the lower rectum. The surgical anal canal measures 2 to 4 cm in length and is generally longer in men than in women. It begins at the anorectal junction and terminates at the anal verge. The *dentate* or *pectinate line* marks the transition point between columnar rectal mucosa and squamous anoderm. The 1 to 2 cm of mucosa just proximal to the dentate line shares histologic characteristics of columnar, cuboidal, and squamous epithelium and is referred to as the *anal transition zone*. The dentate line is surrounded by longitudinal mucosal folds, known as the *columns of Morgagni*, into which the anal crypts empty. These crypts are the source of cryptoglandular abscesses

In the distal rectum, the inner smooth muscle is thickened and comprises the *internal anal sphincter* that is surrounded by the *subcutaneous, superficial, and deep external sphincter*. The *deep external anal sphincter* is an extension of the *puborectalis muscle*. The *puborectalis, iliococcygeus, and pubococcygeus muscles* form the *levator ani muscle* of the pelvic floor.

Anorectal Vascular Supply

The *superior rectal artery* arises from the terminal branch of the inferior mesenteric artery and supplies the upper rectum. The *middle rectal artery* arises from the internal iliac; the presence and size of these arteries are highly variable. The *inferior rectal artery* arises from the internal pudendal artery, which is a branch of the internal iliac artery. A rich network of collaterals connects the terminal arterioles of each of these arteries, thus making the rectum relatively resistant to ischemia

The venous drainage of the rectum parallels the arterial supply. The *superior rectal vein* drains into the portal system via the inferior mesenteric vein. The *middle rectal vein* drains into the internal iliac vein. The *inferior rectal vein* drains into the internal pudendal vein, and subsequently into the internal iliac vein. A submucosal plexus deep to the columns of Morgagni forms the *hemorrhoidal plexus* and drains into all three veins.

Anorectal Lymphatic Drainage

Lymphatic drainage of the rectum parallels the vascular supply. Lymphatic channels in the upper and middle rectum drain superiorly into the inferior mesenteric lymph nodes. Lymphatic channels in the lower rectum drain both superiorly into the inferior mesenteric lymph nodes and laterally into the internal iliac lymph nodes. The anal canal has a more complex pattern of lymphatic drainage. Proximal to the dentate line, lymph drains into both the inferior mesenteric lymph nodes and the internal iliac lymph nodes. Distal to the dentate line, lymph primarily drains into the inguinal lymph nodes, but can also drain into the inferior mesenteric lymph nodes and internal iliac lymph nodes.

Anorectal Nerve Supply

Both sympathetic and parasympathetic nerves innervate the anorectum. Sympathetic nerve fibers are derived from L1-L3 and join the preaortic plexus. The preaortic nerve fibers then extend below the aorta to form the *hypogastric plexus*, which subsequently joins the parasympathetic fibers to form the pelvic plexus. Parasympathetic nerve fibers are known as the *nervi erigentes* and originate from S2-S4. These fibers join the sympathetic fibers to form the pelvic plexus. Sympathetic and parasympathetic fibers then supply the anorectum and adjacent urogenital organs.

The internal anal sphincter is innervated by sympathetic and parasympathetic nerve fibers; both types of fibers inhibit sphincter contraction. The external anal sphincter and puborectalis muscles are innervated by the *inferior rectal branch* of the *internal pudendal nerve*. The levator ani receives innervation from both the *internal pudendal nerve* and direct branches of S3 to S5. Sensory innervation to the anal canal is provided by the *inferior rectal branch* of the *pudendal nerve*. While the rectum is relatively insensate, the anal canal below the dentate line is sensate.

Congenital Anomalies

Perturbation of the embryologic development of the midgut and hindgut may result in anatomic abnormalities of the colon, rectum, and anus. Failure of the midgut to rotate and return to the abdominal cavity during the tenth week of gestation results in varying degrees of intestinal malrotation and colonic nonfixation. Failure of canalization of the primitive gut can result in colonic duplication. Incomplete descent of the urogenital septum may result in imperforate anus and associated fistulas to the genitourinary tract. Many infants with congenital anomalies of the hindgut have associated abnormalities in the genitourinary tract.

Normal Physiology

Fluid and Electrolyte Exchanges

Water, Sodium, Potassium, Chloride, Bicarbonate, and Ammonia

The colon is a major site for water absorption and electrolyte exchange. Approximately 90% of the water contained in ileal fluid is absorbed in the colon (1000 to 2000 mL/d), and up to 5000 mL of fluid can be absorbed daily. Sodium is absorbed actively via a Na-K ATPase. The colon can absorb up to 400 mEq of sodium per day. Water accompanies the transported sodium and is absorbed passively along an osmotic gradient. Potassium is actively secreted into the colonic lumen and absorbed by passive diffusion. Chloride is absorbed actively via a chloride-bicarbonate exchange.

Bacterial degradation of protein and urea produces ammonia. Ammonia is subsequently absorbed and transported to the liver. Absorption of ammonia depends in part upon intraluminal pH. A decrease in colonic bacteria (e.g., broad spectrum antibiotic usage) and/or a decrease in intraluminal pH (e.g., lactulose administration) will decrease ammonia absorption.

Short-Chain Fatty Acids

Short-chain fatty acids (acetate, butyrate, and propionate) are produced by bacterial fermentation of dietary carbohydrates. Short-chain fatty acids are an important source of energy for the colonic mucosa, and metabolism by colonocytes provides energy for processes such as active transport of sodium. Lack of a dietary source for production of short-chain fatty acids, or diversion of the fecal stream by an ileostomy or colostomy, may result in mucosal atrophy and "diversion colitis."

Colonic Microflora and Intestinal Gas

Approximately 30% of fecal dry weight is composed of bacteria (10^{11} to 10^{12} bacteria/g of feces). Anaerobes are the predominant

class of microorganism, and *Bacteroides* species are the most common (10^{11} to 10^{12} organisms/mL). *Escherichia coli* are the most numerous aerobes (10^8 to 10^{10} organisms/mL). Endogenous microflora are crucial for the breakdown of carbohydrates and proteins in the colon and participate in the metabolism of bilirubin, bile acids, estrogen, and cholesterol. Colonic bacteria also are necessary for production of vitamin K. Endogenous bacteria also are thought to suppress the emergence of pathogenic microorganisms, such as *Clostridium difficile*. However, the high bacterial load of the large intestine may contribute to sepsis in critically ill patients and may contribute to intra-abdominal sepsis, abscess, and wound infection following colectomy.

Intestinal gas arises from swallowed air, diffusion from the blood, and intraluminal production. Nitrogen, oxygen, carbon dioxide, hydrogen, and methane are the major components of intestinal gas. Nitrogen and oxygen are largely derived from swallowed air. Carbon dioxide is produced by the reaction of bicarbonate and hydrogen ions, and by the digestion of triglycerides to fatty acids. Hydrogen and methane are produced by colonic bacteria. The production of methane is highly variable. The gastrointestinal tract usually contains between 100 and 200 mL of gas and 400 to 1200 mL per day are released as flatus, depending upon the type of food ingested.

Motility, Defecation, and Continence

Motility

Unlike the small intestine, the large intestine does not demonstrate cyclic motor activity characteristic of the migratory motor complex. Instead, the colon displays intermittent contractions of either low or high amplitude. Low-amplitude, short-duration contractions occur in bursts and appear to move the colonic contents both antegrade and retrograde. It is thought that these bursts of motor activity delay colonic transit and thus increase the time available for absorption of water and exchange of electrolytes. High-amplitude contractions occur in a more coordinated fashion and create "mass movements." Bursts of "rectal motor complexes" also have been described. In general, cholinergic activation increases colonic motility.

Defecation

Defecation is a complex, coordinated mechanism involving colonic mass movement, increased intra-abdominal and rectal pressure, and relaxation of the pelvic floor. Distention of the rectum causes a reflex relaxation of the internal anal sphincter (the rectoanal inhibitory reflex) that allows the contents to make contact with the anal canal. This "sampling reflex" allows the sensory epithelium to distinguish solid stool from liquid stool and gas. If defecation does not occur, the rectum relaxes and the urge to defecate passes (the *accommodation response*). Defecation proceeds by coordination of increasing intra-abdominal pressure via the Valsalva maneuver, increased rectal contraction, relaxation of the puborectalis muscle, and opening of the anal canal.

Continence

The maintenance of fecal continence is at least as complex as the mechanism of defecation. Continence requires adequate rectal wall compliance to accommodate the fecal bolus, appropriate neurogenic control of the pelvic floor and sphincter mechanism, and functional internal and external sphincter muscles. At rest, the puborectalis muscle creates a "sling" around the distal rectum, forming a relatively acute angle that distributes intraabdominal forces onto the pelvic floor. With defecation, this angle straightens, allowing downward force to be applied along the axis of the rectum and anal canal. The internal and external sphincters are tonically active at rest. The internal sphincter is responsible for most of the resting, involuntary sphincter tone (resting pressure). The external sphincter is responsible for most of the voluntary sphincter tone (squeeze pressure). Branches of the pudendal nerve innervate both the internal and external sphincter. Finally, the hemorrhoidal cushions may contribute to continence by mechanically blocking the anal canal. Thus, impaired continence may result from poor rectal compliance, injury to the internal and/or external sphincter or puborectalis, or nerve damage or neuropathy.

Clinical Evaluation

Clinical Assessment

A complete history and physical examination is the starting point for evaluating any patient with suspected disease of the colon and rectum. Special attention should be paid to the patient's past medical and surgical history to detect underlying conditions that might contribute to a gastrointestinal problem. If patients have had prior intestinal surgery, it is essential that one understand the resultant gastrointestinal anatomy. In addition, family history of colorectal disease, especially inflammatory bowel disease, polyps, and colorectal cancer, is crucial. Medication use must be detailed as many drugs cause gastrointestinal symptoms. Before recommending operative intervention, the adequacy of medical treatment must be ascertained. In addition to examining the abdomen, visual inspection of the anus and perineum and careful digital rectal exam are essential.

Endoscopy

Anoscopy

The anoscope is a useful instrument for examination of the anal canal. Anoscopes are made in a variety of sizes and measure approximately 8 cm in length. A larger anoscope provides better exposure for anal procedures such as rubber band ligation or sclerotherapy of hemorrhoids. The anoscope, with obturator in place, should be adequately lubricated and gently inserted into the anal canal. The obturator is withdrawn, inspection of the visualized anal canal is done, and the anoscope should then be withdrawn. It is rotated 90 degrees and reinserted to allow visualization of all four quadrants of the canal. If the patient complains of severe perianal pain and cannot tolerate a digital rectal examination, anoscopy should not be attempted without anesthesia.

Proctoscopy

The rigid proctoscope is useful for examination of the rectum and distal sigmoid colon and is occasionally used therapeutically. The standard proctoscope is 25 cm in length and available in various diameters. Most often, a 15- or 19-mm diameter proctoscope is used for diagnostic examinations. The large (25-mm diameter) proctoscope is useful for procedures such as polypectomy, electrocoagulation, or detorsion of a sigmoid volvulus. A smaller "pediatric" proctoscope (11-mm diameter) is better tolerated by patients with anal stricture. Suction is necessary for an adequate proctoscopic examination.

Flexible Sigmoidoscopy and Colonoscopy

Video or fiberoptic flexible sigmoidoscopy and colonoscopy provide excellent visualization of the colon and rectum. Sigmoidoscopes measure 60 cm in length. Full depth of insertion may allow visualization as high as the splenic flexure, although the mobility and redundancy of the sigmoid colon often limit the extent of the examination. Partial preparation with enemas is usually adequate for sigmoidoscopy and most patients can tolerate this procedure without sedation. Colonoscopes measure 100 to 160 cm in length and are capable of examining the entire colon and terminal ileum. A complete oral bowel preparation is usually necessary for colonoscopy and the duration and discomfort of the procedure usually require conscious sedation. Both sigmoidoscopy and colonoscopy can be used diagnostically and therapeutically. Electrocautery should generally not be used in the absence of a complete bowel preparation because of the risk of explosion of intestinal methane or hydrogen gases. Diagnostic colonoscopes possess a single channel through which instruments such as snares, biopsy forceps, or electrocautery can be passed; this channel also provides suction and irrigation capability. Therapeutic colonoscopes possess two channels to allow simultaneous suction/irrigation and the use of snares, biopsy forceps, or electrocautery.

Imaging

Plain X-Rays and Contrast Studies

Despite advanced radiologic techniques, plain x-rays and contrast studies continue to play an important role in the evaluation of patients with suspected colon and rectal diseases. Plain x-rays of the abdomen (supine, upright, and diaphragmatic views) are useful for detecting free intra-abdominal air, bowel gas patterns suggestive of small or large bowel obstruction, and volvulus. Contrast studies are useful for evaluating obstructive symptoms, delineating fistulous tracts, and diagnosing small perforations or anastomotic leaks. While Gastrografin cannot provide the mucosal detail provided by barium, this water-soluble contrast agent is recommended if perforation or leak is suspected. Double-contrast barium enema has been reported to be 70 to 90% sensitive for the detection of mass lesions greater than 1 cm in diameter. 1 Detection of small lesions can be extremely difficult, especially in a patient with extensive diverticulosis. For this reason, a colonoscopy is preferred for evaluating nonobstructing mass lesions in the colon. Double-contrast barium enema has been used as a back-up examination if colonoscopy is incomplete.

Computed Tomography

Computed tomography (CT) is commonly employed in the evaluation of patients with abdominal complaints. Its utility is primarily in the detection of extraluminal disease, such as intra-abdominal abscesses and pericolic inflammation, and in staging colorectal carcinoma, because of its sensitivity in detection of hepatic metastases. 2 Extravasation of oral or rectal contrast may also confirm the diagnosis of perforation or anastomotic leak. Nonspecific findings such as bowel wall thickening or mesenteric stranding may suggest inflammatory bowel disease, enteritis/colitis, or ischemia. A standard CT scan is relatively insensitive for the detection of intraluminal lesions.

Virtual Colonoscopy

Virtual colonoscopy is a new radiologic technique that is designed to overcome some of the limitations of traditional CT scanning. This technology uses helical CT and three-dimensional reconstruction to detect intraluminal colonic lesions. Oral bowel preparation, oral and rectal contrast, and colon insufflation are used to maximize sensitivity. Early evaluation of virtual colonoscopy suggests that accuracy may approach that of colonoscopy for detection of lesions 1 cm in diameter or greater.

Magnetic Resonance Imaging

The main use of magnetic resonance imaging (MRI) in colorectal disorders is in evaluation of pelvic lesions. MRI is more sensitive than CT for detecting bony involvement or pelvic sidewall extension of rectal tumors. MRI also can be helpful in the detection and delineation of complex fistulas in ano. The use of an endorectal coil may increase sensitivity.

Positron Emission Tomography

Positron emission tomography (PET) is used for imaging tissues with high levels of anaerobic glycolysis, such as malignant tumors. ¹⁸F-fluorodeoxyglucose (FDG) is injected as a tracer; metabolism of this molecule then results in positron emission. PET has been used as an adjunct to CT in the staging of colorectal cancer and may prove useful in discriminating recurrent cancer from fibrosis. At present, the efficacy and utility of PET in the detection of recurrent and/or metastatic colorectal cancer remains unproven.

Angiography

Angiography is occasionally used for the detection of bleeding within the colon or small bowel. To visualize hemorrhage angiographically, bleeding must be relatively brisk (approximately 0.5 to 1.0 mL per minute). If extravasation of contrast is identified, infusion of vasopressin or angiographic embolization can be therapeutic.

Endorectal and Endoanal Ultrasound

Endorectal ultrasound is primarily used to evaluate the depth of invasion of neoplastic lesions in the rectum. The normal rectal wall appears as a five-layer structure (Fig. 28-6). Ultrasound can reliably differentiate most benign polyps from invasive tumors based upon the integrity of the submucosal layer. Ultrasound can also differentiate superficial T1-T2 from deeper T3-T4 tumors. Overall, the accuracy of ultrasound in detecting depth of mural invasion ranges between 81 and 94%.³ This modality also can detect enlarged perirectal lymph nodes, which may suggest nodal metastases; accuracy of detection of pathologically positive lymph nodes is 58 to 83%. Ultrasound may also prove useful for early detection of local recurrence after surgery.

Endoanal ultrasound is used to evaluate the layers of the anal canal. Internal anal sphincter, external anal sphincter, and puborectalis muscle can be differentiated. Endoanal ultrasound is particularly useful for detecting sphincter defects and for outlining complex anal fistulas.

Physiologic and Pelvic Floor Investigations

Anorectal physiologic testing uses a variety of techniques to investigate the function of the pelvic floor. These techniques are useful in the evaluation of patients with incontinence, constipation, rectal prolapse, obstructed defecation, and other disorders of the pelvic floor.

Manometry

Anorectal manometry is performed by placing a pressure-sensitive catheter in the lower rectum. The catheter is then withdrawn through the anal canal and pressures recorded. A balloon attached to the tip of the catheter also can be used to test anorectal sensation. The *resting pressure* in the anal canal reflects the function of the internal anal sphincter (normal: 40 to 80 mm Hg), while the *squeeze pressure*, defined as the maximum voluntary contraction pressure minus the resting pressure, reflects function of the external anal sphincter (normal: 40 to 80 mm Hg above resting pressure). The *high-pressure zone* estimates the length of the anal canal (normal: 2.0 to 4.0 cm). The *rectoanal inhibitory reflex* can be detected by inflating a balloon in the distal rectum; absence of this reflex is characteristic of Hirschsprung's disease.

Neurophysiology

Neurophysiologic testing assesses function of the pudendal nerves and recruitment of puborectalis muscle fibers. Pudendal nerve terminal motor latency measures the speed of transmission of a nerve impulse through the distal pudendal nerve fibers (normal: 1.8 to 2.2 msec); prolonged latency suggests the presence of neuropathy. EMG recruitment assesses the contraction and relaxation of the puborectalis muscle during attempted defecation. Normally, recruitment increases when a patient is instructed to "squeeze," and decreases when a patient is instructed to "push." Inappropriate recruitment is an indication of paradoxical contraction (nonrelaxation of the puborectalis). Needle EMG has been used to map both the pudendal nerves and the anatomy of the internal and external sphincters. However, this examination is painful and poorly tolerated by most patients. Needle EMG has largely been replaced by pudendal nerve motor-latency testing to assess pudendal nerve function and endoanal ultrasound to map the sphincters.

Rectal Evacuation Studies

Rectal evacuation studies include the balloon expulsion test and video defecography. Balloon expulsion assesses a patient's ability to expel an intrarectal balloon. Video defecography provides a more detailed assessment of defecation. In this test, barium paste is placed in the rectum and defecation is then recorded fluoroscopically. Defecography is used to differentiate nonrelaxation of the puborectalis, obstructed defecation, increased perineal descent, rectal prolapse and intussusception, rectocele, and enterocele. The addition of vaginal contrast and intraperitoneal contrast is useful in delineating complex disorders of the pelvic floor.

Laboratory Studies

Fecal Occult Blood Testing

Fecal occult blood testing (FOBT) is used as a screening test for colonic neoplasms in asymptomatic, average-risk individuals. The efficacy of this test is based upon serial testing because the majority of colorectal malignancies will bleed intermittently. FOBT has been a nonspecific test for peroxidase contained in hemoglobin; consequently, occult bleeding from any gastrointestinal source will produce a positive result. Similarly, many foods (red meat, some fruits and vegetables, and vitamin C) will produce a false-positive result. Patients were counseled to eat a restricted diet for 2 to 3 days prior to the test. Increased specificity is now possible by using immunochemical FOBT. These tests rely on monoclonal or polyclonal antibodies to react with the intact globin portion of human hemoglobin. Because globin does not survive in the upper gastrointestinal tract, the immunochemical tests are more specific for identifying occult bleeding from the colon or rectum. Dietary restrictions are not necessary. Any positive FOBT mandates further investigation, usually by colonoscopy.

Stool Studies

Stool studies are often helpful in evaluating the etiology of diarrhea. Wet-mount examination reveals the presence of fecal leukocytes, which may suggest colonic inflammation or the presence of an invasive organism such as invasive *E. coli* or *Shigella*. Stool cultures can detect pathogenic bacteria, ova, and parasites. *C. difficile* colitis is diagnosed by detecting bacterial toxin in the stool. 4 Steatorrhea may be diagnosed by adding Sudan red stain to a stool sample.

Serum Tests

Specific laboratory tests that should be performed will be dictated by the clinical scenario. Preoperative studies generally include a complete blood count and electrolyte panel. The addition of coagulation studies, liver function tests, and blood typing/cross-matching depends upon the patient's medical condition and the proposed surgical procedure.

Tumor Markers

Carcinoembryonic antigen (CEA) may be elevated in 60 to 90% of patients with colorectal cancer. Despite this, CEA is not an effective screening tool for this malignancy. Many practitioners follow serial CEA levels after curative-intent surgery in order to detect early recurrence of colorectal cancer. However, this tumor marker is nonspecific, and no survival benefit has yet been proven. Other biochemical markers (ornithine decarboxylase, urokinase) have been proposed, but none has yet proven sensitive or specific for detection, staging, or predicting prognosis of colorectal carcinoma. 5

Genetic Testing

Although familial colorectal cancer syndromes, such as familial adenomatous polyposis (FAP) and hereditary nonpolyposis colon cancer (HNPCC) are rare, information about the specific genetic abnormalities underlying these disorders has led to significant interest in the role of genetic testing for colorectal cancer. 6 Tests for mutations in the adenomatous polyposis coli (APC) gene responsible for FAP, and in mismatch repair genes responsible for HNPCC, are commercially available and extremely accurate in families with known mutations. Although many of these mutations are also present in sporadic colorectal cancer, the accuracy of genetic testing in average-risk individuals is considerably lower and these tests are not recommended for screening. Because of the potential psychosocial implications of genetic testing, it is strongly recommended that professional genetic counselors be involved in the care of any patient considering these tests.

Evaluation of Common Symptoms

Pain

Abdominal Pain

Abdominal pain is a nonspecific symptom with a myriad of causes. Abdominal pain related to the colon and rectum can result from obstruction (either inflammatory or neoplastic), inflammation, perforation, or ischemia. Plain x-rays and judicious use of contrast studies and/or a CT scan can often confirm the diagnosis. Gentle retrograde contrast studies (barium or Gastrografin enema) may be useful in delineating the degree of colonic obstruction. Sigmoidoscopy and/or colonoscopy performed by an experienced

endoscopist can assist in the diagnosis of ischemic colitis, infectious colitis, and inflammatory bowel disease. However, if perforation is suspected, colonoscopy and/or sigmoidoscopy are generally contraindicated. Evaluation and treatment of abdominal pain from a colorectal source should follow the usual surgical principles of a thorough history and physical examination, appropriate diagnostic tests, resuscitation, and appropriately timed surgical intervention.

Pelvic Pain

Pelvic pain can originate from the distal colon and rectum or from adjacent urogenital structures. Tenesmus may result from proctitis or from a rectal or retrorectal mass. Cyclical pain associated with menses, especially when accompanied by rectal bleeding, suggests a diagnosis of endometriosis. Pelvic inflammatory disease also can produce significant abdominal and pelvic pain. The extension of a peridiverticular abscess or periappendiceal abscess into the pelvis may also cause pain. CT scan and/or MRI may be useful in differentiating these diseases. Proctoscopy (if tolerated) also can be helpful. Occasionally, laparoscopy will yield a diagnosis.

Anorectal Pain

Anorectal pain is most often secondary to an anal fissure or perirectal abscess and/or fistula. Physical examination can usually differentiate these conditions. Other, less common causes of anorectal pain include anal canal neoplasms, perianal skin infection, and dermatologic conditions. Proctalgia fugax results from levator spasm and may present without any other anorectal findings. Physical exam is critical in evaluating patients with anorectal pain. If a patient is too tender to examine in the office, an examination under anesthesia is necessary. MRI may be helpful in select cases where the etiology of pain is elusive.

Lower Gastrointestinal Bleeding

The first goal in evaluating and treating a patient with gastrointestinal hemorrhage is adequate resuscitation. The principles of ensuring a patent airway, supporting ventilation, and optimizing hemodynamic parameters apply and coagulopathy and/or thrombocytopenia should be corrected. The second goal is to identify the source of hemorrhage. Because the most common source of gastrointestinal hemorrhage is esophageal, gastric, or duodenal, nasogastric aspiration should always be performed; return of bile suggests that the source of bleeding is distal to the ligament of Treitz. If aspiration reveals blood or nonbile secretions, or if symptoms suggest an upper intestinal source, esophagogastroduodenoscopy is performed. Anoscopy and/or limited proctoscopy can identify hemorrhoidal bleeding. A technetium-99 (^{99m}Tc)-tagged red blood cell (RBC) scan is extremely sensitive and is able to detect as little as 0.1 mL/h of bleeding; however, localization is imprecise. If the ^{99m}Tc -tagged RBC scan is positive, angiography can then be employed to localize bleeding. Infusion of vasopressin or angioembolization may be therapeutic. Alternatively, a catheter can be left in the bleeding vessel to allow localization at the time of laparotomy. If the patient is hemodynamically stable, a rapid bowel preparation (over 4 to 6 hours) can be performed to allow colonoscopy. Colonoscopy may identify the cause of the bleeding, and cautery or injection of epinephrine into the bleeding site may be used to control hemorrhage. Colectomy may be required if bleeding persists despite these interventions. Intraoperative colonoscopy and/or enteroscopy may assist in localizing bleeding. If colectomy is required, a segmental resection is preferred if the bleeding source can be localized. "Blind" subtotal colectomy may very rarely be required in a patient who is hemodynamically unstable with ongoing colonic hemorrhage of an unknown source. In this setting, it is crucial to irrigate the rectum and examine the mucosa by proctoscopy to ensure that the source of bleeding is not distal to the resection margin.

Occult blood loss from the gastrointestinal tract may manifest as iron-deficiency anemia or may be detected with fecal occult blood testing. Because colon neoplasms bleed intermittently and rarely present with rapid hemorrhage, the presence of occult fecal blood should always prompt a colonoscopy. Unexplained iron-deficiency anemia is also an indication for colonoscopy.

Hematochezia is commonly caused by hemorrhoids or fissure. Sharp, knife-like pain and bright-red rectal bleeding with bowel movements suggest the diagnosis of fissure. Painless, bright-red rectal bleeding with bowel movements is often secondary to a friable internal hemorrhoid that is easily detected by anoscopy. In the absence of a painful, obvious fissure, any patient with rectal bleeding should undergo a careful digital rectal examination, anoscopy, and proctosigmoidoscopy. Failure to diagnose a source in the distal anorectum should prompt colonoscopy.

Constipation and Obstructed Defecation

Constipation is an extremely common complaint, affecting more than 4 million people in the United States. Despite the prevalence of this problem, there is lack of agreement about an appropriate definition of constipation. Patients may describe infrequent bowel movements, hard stools, or excessive straining. A careful history of these symptoms often clarifies the nature of the problem.

Constipation has a myriad of causes. Underlying metabolic, pharmacologic, endocrine, psychologic, and neurologic causes often contribute to the problem. A stricture or mass lesion should be excluded by colonoscopy or barium enema. After these causes have

been excluded, evaluation focuses upon differentiating *slow-transit constipation* from *outlet obstruction*. Transit studies, in which radiopaque markers are swallowed and then followed radiographically, are useful for diagnosing slow-transit constipation. Anorectal manometry and electromyography can detect nonrelaxation of the puborectalis, which contributes to outlet obstruction. The absence of an anorectal inhibitory reflex suggests Hirschsprung's disease and may prompt a rectal mucosal biopsy. Defecography can identify rectal prolapse, intussusception, rectocele, or enterocele.

Medical management is the mainstay of therapy for constipation and includes fiber, increased fluid intake, and laxatives. Outlet obstruction from nonrelaxation of the puborectalis often responds to biofeedback. 7 Surgery to correct rectocele and rectal prolapse has a variable effect on symptoms of constipation, but can be successful in selected patients. Subtotal colectomy is considered only for patients with severe slow-transit constipation (colonic inertia) refractory to maximal medical interventions. While this operation almost always increases bowel movement frequency, complaints of diarrhea, incontinence, and abdominal pain are not infrequent, and patients should be carefully selected. 8

Diarrhea and Irritable Bowel Syndrome

Diarrhea is also a common complaint and is usually a self-limited symptom of infectious gastroenteritis. If diarrhea is chronic or is accompanied by bleeding or abdominal pain, further investigation is warranted. Bloody diarrhea and pain are characteristic of colitis; etiology can be an infection (invasive *E. coli*, *Shigella*, *Salmonella*, *Campylobacter*, *Entamoeba histolytica*, or *C. difficile*), inflammatory bowel disease (ulcerative colitis or Crohn's colitis), or ischemia. Stool wet-mount and culture can often diagnose infection. Sigmoidoscopy or colonoscopy can be helpful in diagnosing inflammatory bowel disease or ischemia. However, if the patient has abdominal tenderness, particularly with peritoneal signs, or any other evidence of perforation, endoscopy is contraindicated.

Chronic diarrhea may present a more difficult diagnostic dilemma. Chronic ulcerative colitis, Crohn's colitis, infection, malabsorption, and short gut syndrome can cause chronic diarrhea. Rarely, carcinoid syndrome and islet cell tumors (vasoactive intestinal peptide-secreting tumor [VIPoma], somatostatinoma, gastrinoma) present with this symptom. Large villous lesions may cause secretory diarrhea. Collagenous colitis can cause diarrhea without any obvious mucosal abnormality. Along with stool cultures, tests for malabsorption, and metabolic investigations, colonoscopy can be invaluable in differentiating these causes. Biopsies should be taken even if the colonic mucosa appears grossly normal.

Irritable bowel syndrome is a particularly troubling constellation of symptoms consisting of crampy abdominal pain, bloating, constipation, and urgent diarrhea. Work-up reveals no underlying anatomic or physiologic abnormality. Once other disorders have been excluded, dietary restrictions and avoidance of caffeine, alcohol, and tobacco may help to alleviate symptoms. Antispasmodics and bulking agents may be helpful.

Incontinence

The incidence of fecal incontinence has been estimated to occur in 10 to 13 individuals per 1000 people older than age 65 years. Incontinence ranges in severity from occasional leakage of gas and liquid stool to daily loss of solid stool. The underlying cause of incontinence is often multifactorial and diarrhea is often contributory. In general, causes of incontinence can be classified as *neurogenic* or *anatomic*. Neurogenic causes include diseases of the central nervous system and spinal cord along with pudendal nerve injury. Anatomic causes include congenital abnormalities, procidentia, overflow incontinence secondary to impaction or neoplasm, and trauma. The most common traumatic cause of incontinence is injury to the anal sphincter during vaginal delivery. Other causes include anorectal surgery, impalement, and pelvic fracture.

After a thorough medical evaluation to detect underlying conditions that might contribute to incontinence, evaluation focuses on assessment of the anal sphincter and pudendal nerves. Pudendal nerve terminal motor latency testing may detect neuropathy. Anal manometry can detect low resting and squeeze pressures. Defecography can detect rectal prolapse. Endoanal ultrasound is invaluable in diagnosing sphincter defects.

DIVERTICULAR DISEASE OF THE COLON

Diverticula are saclike protrusions of the colonic wall, varying in size from a few millimeters to several centimeters. True diverticula contain all layers of the colon wall and are believed to be congenital. They are very uncommon in the colon. False or *pseudodiverticula* represent herniations of the mucosa and submucosa through the circular muscle of the bowel wall. Unless otherwise stated in the text, the term *diverticula* refers to the predominant lesion, namely, colonic pseudodiverticula. The term *diverticulosis* simply indicates the presence of multiple diverticula of the colon.

PREVALENCE, PATHOGENESIS, AND PATHOLOGIC ANATOMY

The prevalence of diverticular disease in the general population ranges between 35% and 50%, as estimated by several large autopsy and radiographic series.¹³ Prevalence directly correlates with age, estimated to be less than 5% at age 40, increasing to 30% by age 60, and as high as 65% by age 85.²⁵ Males and females appear to be affected equally. Geographically, diverticular disease is much more common in the United States and Western Europe than in other less industrialized regions such as Africa, South America, and Asia. Although diet is thought by many to contribute significantly to the development of diverticular disease, the complete etiology is likely to involve other, as yet unrecognized, factors. For example, diverticular disease in Asian populations is localized predominantly to the right colon, in distinct contrast to the left-sided predilection observed in Western civilizations. Such variations in the anatomic distribution of diverticula among civilizations might suggest that factors other than diet alone exert a substantial influence on the character of this disease worldwide.

Mechanical Factors. Clinical studies within the past 30 years have implicated low fiber diets as a prominent etiologic factor in the development of diverticular disease.^{24, 25} Diets lacking vegetable fiber are presumed to predispose to the development of diverticula by altering colonic motility. Colonic motility is a complex process serving to transport feces distally while also permitting storage, thereby facilitating fluid and electrolyte absorption. Colonic motility is modulated by myogenic, hormonal, and neural influences. There is evidence that patients with diverticular disease manifest exaggerated contractile responses to feeding and hormonal stimuli. Resting pressures are usually normal, however. These abnormal muscular contractions are believed to cause colonic smooth muscle hypertrophy, a characteristic of diverticular disease.

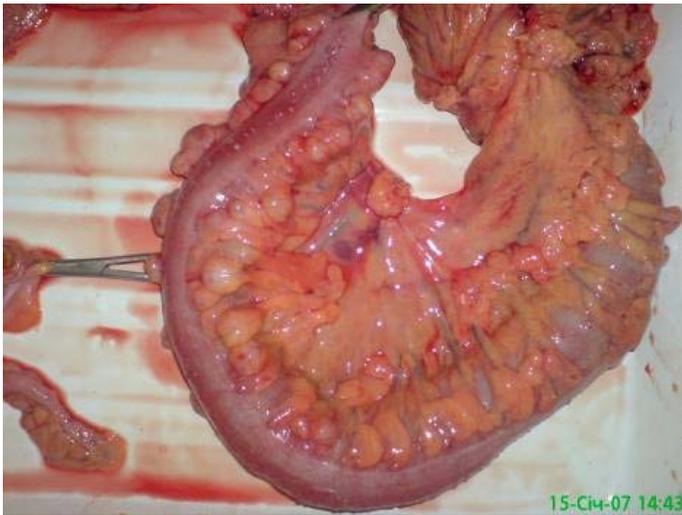
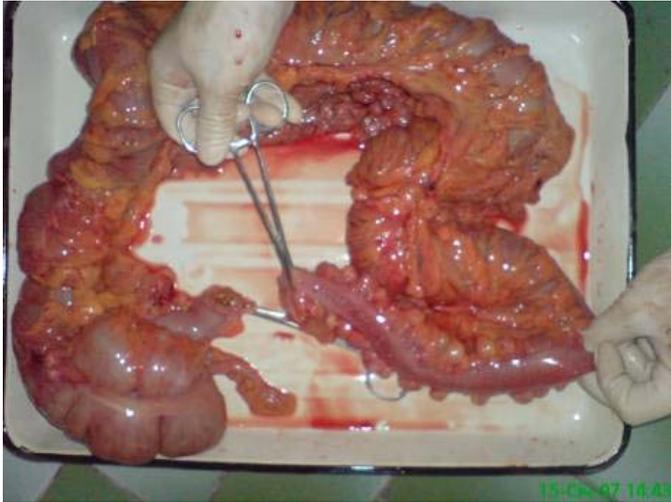
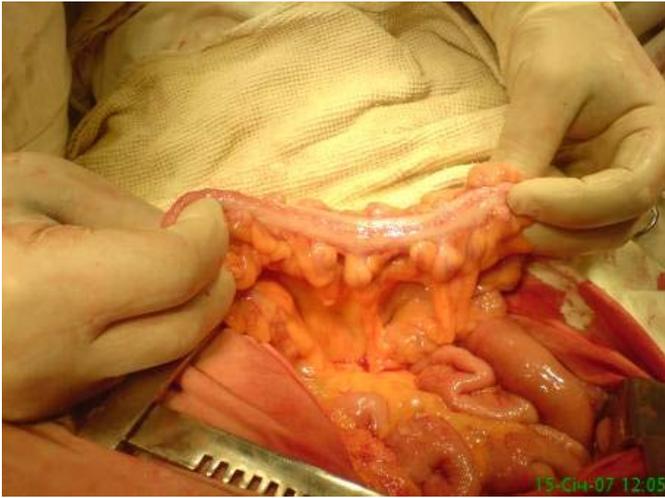
The possible role of dietary fiber in the development of diverticula is best explained by effects on colonic diameter and stool consistency. It has been postulated that colonic segments with bulky fecal contents and large luminal diameters are less likely to exhibit exaggerated segmentation. Low-fiber diets are associated with a narrowed colon filled with small, hardened feces; segmentation is enhanced, and high luminal pressures tend to develop. Although this concept has been widely disseminated, definitive evidence for a causal relationship between low dietary fiber and the development of diverticular disease does not exist.^{31, 39} Nonetheless, high-residue diets are in widespread use in the management of diverticular disease. Whether such therapy has a significant influence on the natural history of diverticular disease is unclear.

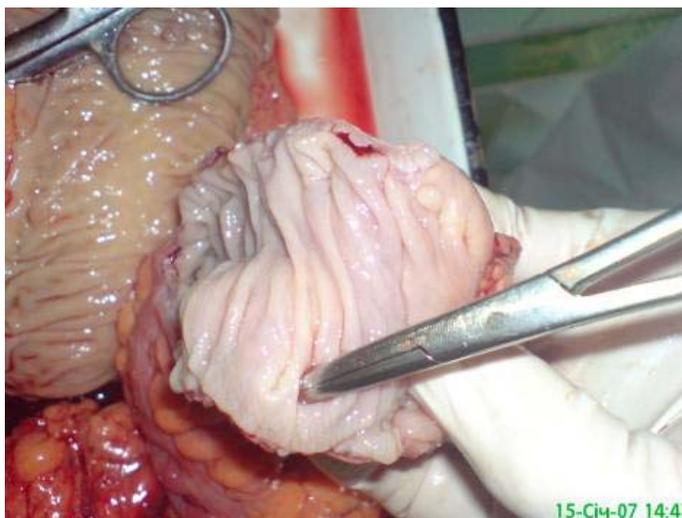
Anatomic Features. Diverticula tend to develop at specific points in the circumference of the colon. This localization is determined, in part, by the anatomic relationship between the colonic musculature and its nutrient blood supply. Diverticula form at so-called weak points where the nutrient blood vessels (*vasa recta*) penetrate the circular muscle layer en route to the mucosa. These *perforating* vessels tend to penetrate the colonic wall along the mesenteric border of the two antimesenteric taeniae. The gaps in the circular muscle layer where the *vasa recta* penetrate constitute points of potential weakness through which the mucosa and submucosa can herniate, forming diverticula. Diverticula, therefore, are usually located between the single mesenteric taenia and either of the two antimesenteric taeniae. Less commonly, diverticula form in the area between the antimesenteric taeniae. Although also consisting of mucosal herniation through the muscular layers of the colonic wall, these diverticula tend to be less prominent. In many instances the mucosal herniation does not quite extend to the serosa, causing these to be referred to as intramural diverticula.

The distribution of diverticula throughout the colon also tends to follow a pattern, but with considerable individual variation. The overwhelming majority of diverticula occur in the descending and sigmoid colon. It is estimated that 90% to 95% of patients with diverticulosis will have involvement of the sigmoid colon. Approximately 65% of patients will have disease limited to the sigmoid colon alone. Conversely, only a small number of patients (2% to 10%) will have disease confined to the right colon. Solitary diverticula occur most commonly in the cecum.

NATURAL HISTORY

After the first episode of diverticulitis, approximately one third of patients will sustain a second attack, usually within 3 to 5 years. Another 30% to 40% will suffer from intermittent symptoms of discomfort and crampy abdominal pain, without requiring hospitalization. The remainder can be expected to remain symptom free. The prognosis is worse after a second attack, with only 10% of patients remaining symptom free. The morbidity and the mortality from recurrent attacks are also higher than that associated with an initial episode. Complications such as abscess formation or fistulization develop in approximately 20% of patients after a single attack of diverticulitis, while the complication rate approaches 60% in patients who have had previous episodes.





Because diverticulosis is an acquired disease, the incidence of which clearly increases with age, it would seem logical that the number and size of diverticula would also increase with time. However, only 30% of patients demonstrate radiologic evidence of progression of their disease, either in the form of an increased number of diverticula or involvement of other segments of the colon. Progression of disease after resection of involved colon is also unusual, occurring in less than 10% to 15% of patients. [video](#) [video2](#) [video3](#) [video4](#)

CROHN'S DISEASE

Crohn's disease is a chronic, nonspecific inflammatory disease of the gastrointestinal tract of unknown etiology. It involves mainly the ileum and large intestine, most often producing symptoms of obstruction or localized perforation with fistula. Both medical and surgical treatments are palliative. Nonetheless, operative excision provides effective symptomatic relief and produces reasonable long-term benefit.

Epidemiology

Crohn's disease is a chronic, idiopathic inflammatory disease with a propensity to affect the distal ileum, although any part of the alimentary tract can be involved. Recent estimates of the incidence of Crohn's disease in the United States have ranged from 3.6 to 8.8 per 100,000. ³⁴ A dramatic increase in incidence in the United States was observed to occur from the mid-1950s through the early 1970s. Incidence rates have been stable since the 1980s. Substantial regional variations in incidence have been observed, with the highest incidences reported to exist in northern latitudes. The incidence of Crohn's disease varies among ethnic groups within the same geographic region. For example, members of the East European Ashkenazi Jewish population are at two- to fourfold higher risk of developing Crohn's disease than are members of other populations living in the same location.

Most studies suggest that Crohn's disease is slightly more prevalent in females than in males. The median age at which patients are diagnosed with Crohn's disease is approximately 30 years; however, age of diagnosis can range from early childhood through the entire life span.

Both genetic and environmental factors appear to influence the risk for developing Crohn's disease. The relative risk among first-degree relatives of patients with Crohn's disease is 14 to 15 times higher than that of the general population. Approximately 1 of 5 patients with Crohn's disease will report having at least one affected relative. The concordance rate among monozygotic twins is as high as 67%; however, Crohn's disease is not associated with simple mendelian inheritance patterns. Although there is a tendency within families for either ulcerative colitis or Crohn's disease to be present exclusively, mixed kindreds also occur, suggesting the presence of some shared genetic traits as a basis for both diseases.

Higher socioeconomic status is associated with an increased risk of Crohn's disease. Most studies have found breastfeeding to be protective against the development of Crohn's disease. Crohn's disease is more prevalent among smokers. Furthermore, smoking is associated with the increased risk for both the need for surgery and the risk of relapse after surgery for Crohn's disease.

Crohn's disease is characterized by sustained inflammation. Whether this inflammation represents an appropriate response to a yet unrecognized pathogen or an inappropriate response to a normally innocuous stimulus is unknown. Various

hypotheses on the roles of environmental and genetic factors in the pathogenesis of Crohn's disease have been proposed.

Many infectious agents have been suggested to be the causative organism of Crohn's disease. Candidate organisms have included Chlamydia, *Listeria monocytogenes*, *Pseudomonas* species, reovirus, *Mycobacterium paratuberculosis*, and many others. There is no conclusive evidence that any of these organisms is the causative agent.

Studies using animal models suggest that in a genetically susceptible host, nonpathogenic, commensal enteric flora are sufficient to induce a chronic inflammatory response resembling that associated with Crohn's disease. In these models, the sustained intestinal inflammation is the result of either abnormal epithelial barrier function or immune dysregulation. Poor barrier function is hypothesized to permit inappropriate exposure of lamina propria lymphocytes to antigenic stimuli derived from the intestinal lumen. In addition, a variety of defects in immune regulatory mechanisms, e.g., overresponsiveness of mucosal T cells to enteric flora-derived antigens, can lead to defective immune tolerance and sustained inflammation.

Specific genetic defects associated with Crohn's disease in human patients are beginning to be defined. For example, the presence of a locus on chromosome 16 (the so-called IBD1 locus) has been linked to Crohn's disease. The IBD1 locus has been identified as the NOD2 gene. 35,36 Persons with allelic variants on both chromosomes have a 40-fold relative risk of Crohn's disease when compared to those without variant NOD2 genes. The relevance of this gene to the pathogenesis of Crohn's disease is biologically plausible, because the protein product of the NOD2 gene mediates the innate immune response to microbial pathogens.

Although the pathologic hallmark of Crohn's disease is focal, transmural inflammation of the intestine, a spectrum of pathologic lesions can be present. The earliest lesion characteristic of Crohn's disease is the aphthous ulcer. These superficial ulcers are up to 3 mm in diameter and are surrounded by a halo of erythema. In the small intestine, aphthous ulcers typically arise over lymphoid aggregates. Granulomas are highly characteristic of Crohn's disease and are reported to be present in up to 70% of intestinal specimens obtained during surgical resection. These granulomas are noncaseating and can be found in both areas of active disease and apparently normal intestine, in any layer of the bowel wall, and in mesenteric lymph nodes.

As disease progresses, aphthae coalesce into larger, stellate-shaped ulcers. Linear or serpiginous ulcers may form when multiple ulcers fuse in a direction parallel to the longitudinal axis of the intestine. With transverse coalescence of ulcers, a cobblestone appearance of the mucosa may arise.

With advanced disease, inflammation can be transmural. Serosal involvement results in adhesion of the inflamed bowel to other loops of bowel or other adjacent organs. Transmural inflammation also can result in fibrosis, with stricture formation, intra-abdominal abscesses, fistulas, and, rarely, free perforation. Inflammation in Crohn's disease can affect discontinuous portions of intestine: so-called "skip lesions" that are separated by intervening normal-appearing intestine.

A feature of Crohn's disease that is grossly evident and helpful in identifying affected segments of intestine during surgery is the presence of fat wrapping (Fig. 27-17). This finding is virtually pathognomonic of Crohn's disease. It is the encroachment of mesenteric fat onto the serosal surface of the bowel. The presence of fat wrapping correlates well with the presence of underlying acute and chronic inflammation.

ETIOLOGY

No specific etiology of the disease has been identified. There are two main schools of investigation: the microbiologic and the immunologic. Microbiologists have long sought a specific micro-organism that might be the cause of the disease; however, none has yet been identified. Recent reports of the isolation of *Mycobacterium paratuberculosis* from segments of bowel affected with Crohn's disease excited interest, but this organism as a specific etiology for the disease has yet to be proved. Also, no virus has been identified as an etiologic agent.

An immunologic origin of the disease has also been sought. No doubt an immunologic response to the condition does exist. Some have postulated that a childhood sensitization to milk impairs mucosal integrity and allows bacteria or bacteriologic products to enter the body. A cellular and humoral immune response to these products then ensues. The ileocolic epithelium, in particular, may be the target of a necrotizing immune response, with ensuing ulceration, tissue destruction, and the clinical appearance of the disease. Although an immunologic response certainly plays a role in the pathogenesis of the condition, its role as an etiologic agent is still unclear.

Other data suggest that environmental factors have an etiologic role in the disease. The disease is more common among persons living in temperate climates than among those living in tropical climates. Smoking may exert a stimulating effect on the disease; many patients with Crohn's disease are heavy smokers. Spouses of persons with Crohn's disease have a higher incidence of the disease than persons in the general population. Although these data suggest that environmental factors have a role, no specific environmental factor has been identified.

SYMPTOMS

The most common symptoms of Crohn's disease are those from the intestinal lesions, with abdominal pain, especially of a cramping nature, topping the list. Diarrhea is frequent. The stools may contain blood, although they often do not. Patients experience abdominal distention or flatulence and sometimes nausea and vomiting. Eating becomes difficult, because it induces symptoms. Patients therefore decrease their food intake and lose weight. Should fistulas develop, the pain and discharge of intestinal content at the site of the fistulas to the skin or in the perianal area produce localized symptoms in these areas. Systemic responses include fever and malaise; localized pain and discomfort are related to the sites of extraintestinal involvement in the skin, eyes, and joints.

The course of the disease is one of exacerbations and remissions, but as the lesions mature and complications develop, the symptoms continue unabated and the disease becomes relentlessly progressive. About 70% of patients eventually come to operation, in spite of spontaneous remissions and medical or dietary therapy.

DIAGNOSIS

Diagnosis is based on the history, physical findings, and appropriate laboratory tests. The physical findings include the palpation of the thickened bowel wall or adjacent inflammatory response or abscesses in the abdomen. Hyperactive bowel tones are heard using auscultation, and peristaltic rushes in the small intestine may even be seen through a thin abdominal wall. Abdominal distention occurs. Fistulas are apparent, and probes and catheters can be passed through the cutaneous openings and into the lumen of the bowel through the tracts. On inspection, the perianal skin appears bluish, and perianal fissures, abscesses, and fistulas can be identified.

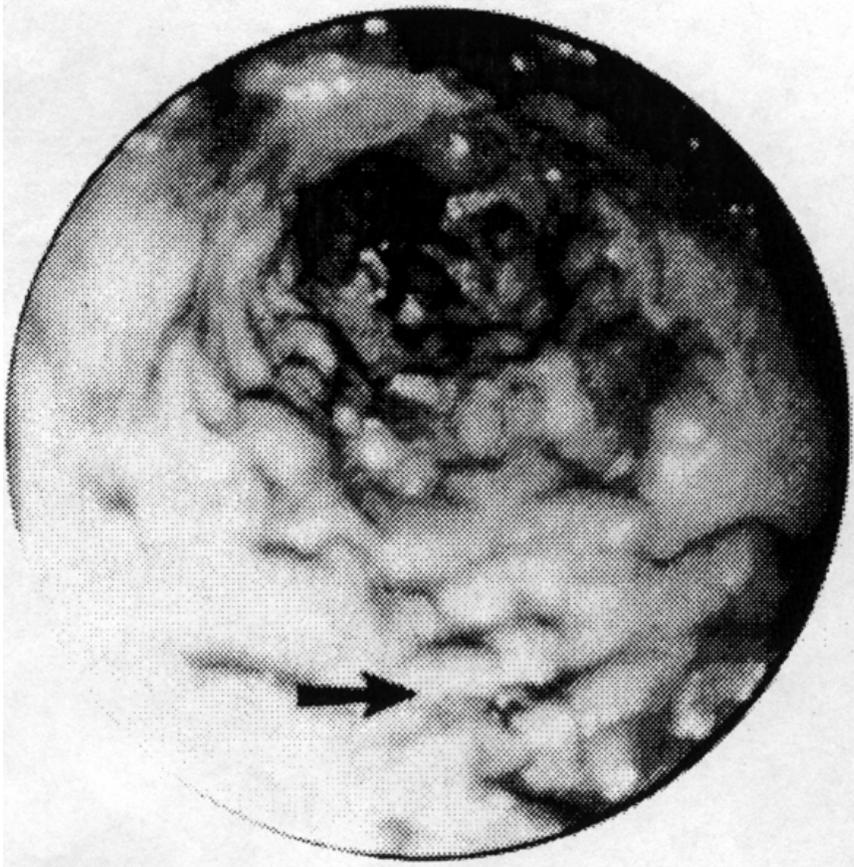
Proctoscopy often reveals the characteristic rectal aphthous ulcer with surrounding normal-appearing mucosa. With progressive and extensive involvement, the ulcerations involve more and more of the lumen of the bowel, with diminishing isolated segments of normal mucosa remaining. Anoscopy can show perianal abscesses, perianal fistulas, and even rectovaginal fistulas. Colonoscopy delineates the extent of the lesions in the large intestine. Sometimes the colonoscope can be passed through the colon and into the ileum to identify the ileal lesions of the disease. The hallmarks of Crohn's disease are the discontinuous and asymmetrical nature of the endoscopic findings. Biopsies taken during endoscopy show chronic inflammation and sometimes granulomas.

Roentgenographic examination of the gastrointestinal tract using BaSO₄. Proximal dilatation of the bowel accompanies obstructing lesions. Long lengths of narrowed terminal ileum may reduce the caliber of the lumen to the size of a string. Areas of dilatation may alternate with areas of constriction. The cobblestone appearance of the mucosa may be apparent, as may the rake ulcers. Fissures, fistulas, and perienteric abscesses may be found. Computed axial tomography may help delineate thickened bowel, perienteric abscesses, and perforations. In toxic megacolon, the transverse colon is greatly dilated and the bowel wall is thickened. A mass accompanying a narrowed or ulcerated area suggests cancer. Free air in the abdomen is present with free perforation.

The differential diagnosis includes both specific and nonspecific causes of intestinal inflammation. Specific microbiologic diseases that may be confused with Crohn's disease include bacterial inflammations such as those caused by salmonella and shigella, typhoid fever, intestinal tuberculosis, and protozoan infections such as amebiasis. Appropriate cultures and biopsies reveal the causative organisms in these conditions and rule them out. In regard to nonspecific intestinal inflammation, chronic ulcerative colitis can usually be differentiated from Crohn's disease. Although ulcerative colitis involves the mucosa of the large intestine, it does not extend deep into the wall of the bowel, as does Crohn's disease. Ulcerative colitis nearly always involves the rectum most severely, with lessening inflammation from the rectum to the ileocolic area. In contrast, Crohn's disease may be worse on the right side of the colon than on the left side, sometimes sparing the rectum. Ulcerative colitis also shows continuous involvement from rectum to proximal segments, whereas Crohn's disease shows segmental lesions. Although nonspecific, so-called backwash ileitis may be present in ulcerative colitis, ileal and small intestinal involvement suggests Crohn's disease. Bleeding is a more common symptom in ulcerative colitis and is less common in Crohn's disease. Perianal involvement and rectovaginal fistulas are unusual in ulcerative colitis but are more common in Crohn's disease. In most instances, the two diseases can be clearly separated, but a subgroup of 5% to 10% of all patients with chronic nonspecific inflammatory bowel disease cannot be clearly classified as having ulcerative colitis or Crohn's disease. These patients are usually given a diagnosis of "indeterminate" colitis. The true diagnosis often becomes apparent as the patients are followed through the years.



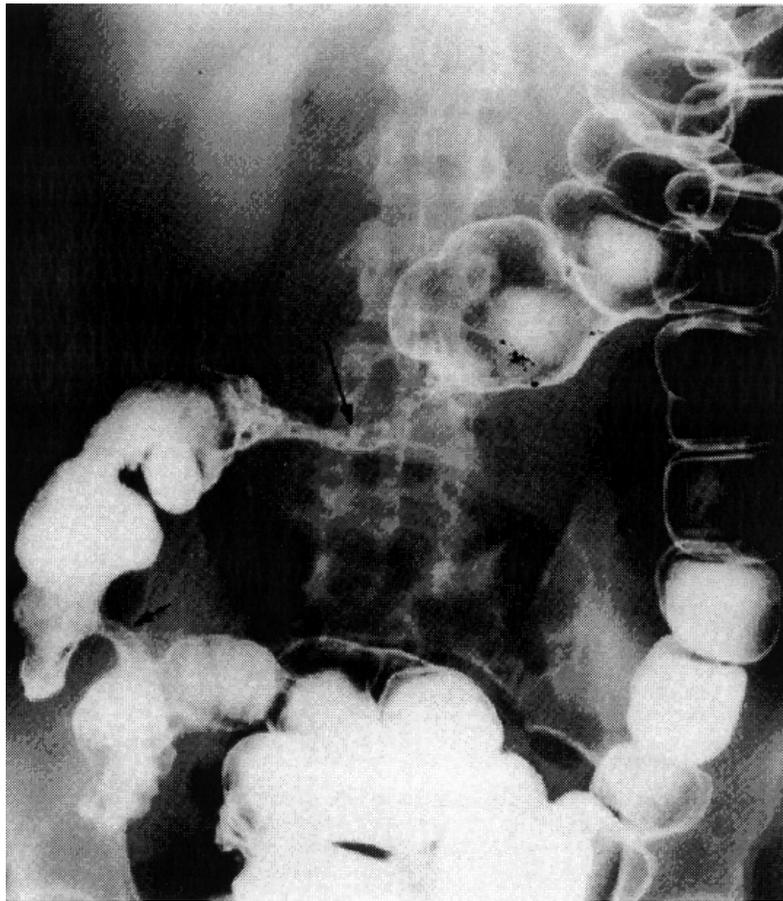
Endoscopic view



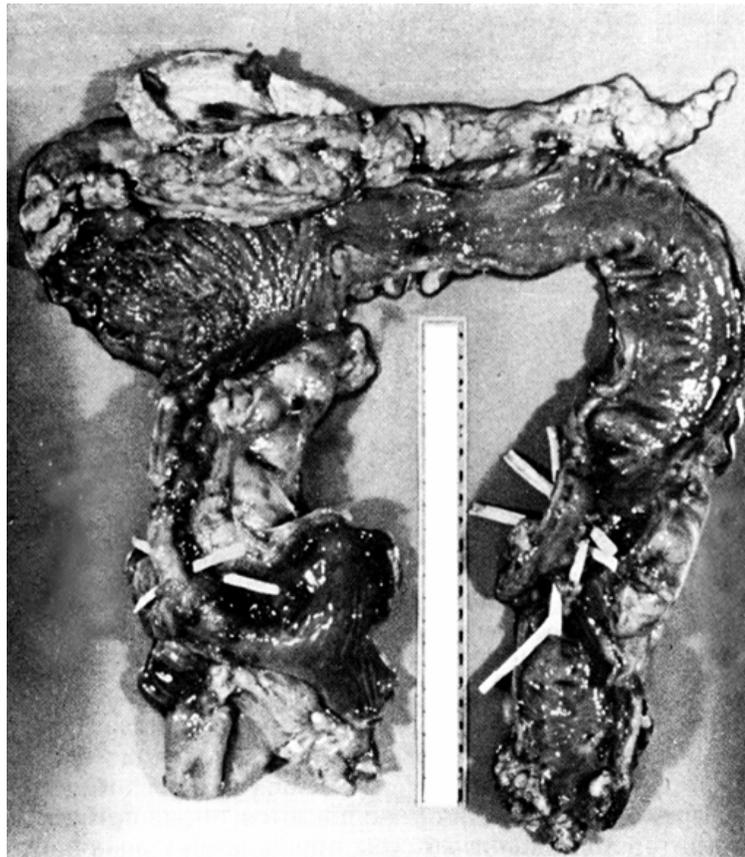
Crohn's disease, symptom of "roadway". Endoscopy examination



Crohn's disease. Hyperemia and deep cracks of mucus membrane of colon, ulcers, symptom of "roadway"



Stricture ascending part of the colon. Crohn's disease. Irrigogram.



Crohn's disease. Multiple fistulas.

Acute distal ileitis may be a manifestation of early Crohn's disease, but it also may be unrelated, such as when it is caused by a bacteriologic agent such as *Campylobacter* or *Yersinia*. Patients usually present in a manner similar to patients with acute appendicitis. They have a sudden onset of right lower quadrant pain, nausea, vomiting, and fever, with tenderness over the area of involvement. The diagnosis is made at operation by identifying an acutely inflamed segment of terminal ileum. No biopsy or resection should be done. The condition almost always subsides spontaneously, and the segment should not be excised. If the cecum is not involved, the appendix should be removed to prevent subsequent bouts of right lower quadrant inflammation from being confused with appendicitis.

THERAPY

Medical and Dietary Therapy

Medical therapy consists of sulfasalazine, 5-aminosalicylic acid, corticosteroids, antibiotics such as metronidazole and ampicillin, and immunosuppressive agents such as azathioprine and cyclosporine. Because no specific etiology has been identified for Crohn's disease, the treatments are also nonspecific. They suppress inflammation and improve symptoms but are not curative.

Manipulations of the diet ordinarily have little effect on the progress of Crohn's disease. However, complete abstinence of oral intake with total parenteral nutrition may lead to temporary remission of symptoms in some patients with Crohn's disease. Sometimes enteroenteric or enterocutaneous fistulas close. Few long-term benefits of total parenteral nutrition have been achieved, however. When oral intake is resumed, the patients usually have recurring difficulty, and the symptoms of the disease return.

Pharmacologic agents used to treat Crohn's disease include antibiotics, aminosalicylates, corticosteroids, and immunomodulators. Antibiotics have an adjunctive role in the treatment of infectious complications associated with Crohn's disease. They are also used to treat patients with perianal disease, enterocutaneous fistulas, and active colonic disease.

Most studies have shown sulfasalazine, the parent compound of all aminosalicylates used to treat Crohn's disease, to be superior to placebo in inducing disease remission. Its efficacy in the maintenance of remission is less clear. Aminosalicylates are associated with minimal toxicity and are available in a variety of formulations that allow for their delivery to specific regions of the alimentary tract.

Orally administered glucocorticoids are used to treat patients with mildly to moderately severe disease that does not respond to aminosalicylates. Patients with severe active disease usually require intravenous administration of glucocorticoids. Although glucocorticoids are effective in inducing remission, they are ineffective in preventing relapse and their adverse side-effect profile makes long-term use hazardous. Therefore, they should be tapered once remission is achieved. Some patients are unable to undergo glucocorticoid tapering without suffering recurrence of symptoms. Such patients are said to have glucocorticoid dependence and are candidates for therapy with glucocorticoid-sparing immune modulators.

The thiopurine antimetabolites azathioprine and 6-mercaptopurine have demonstrated efficacy in inducing remission, in maintaining remission, and in allowing for glucocorticoid tapering in glucocorticoid-dependent patients. There is also some evidence that they decrease the risk of relapse after intestinal resection for Crohn's disease. These agents are relatively safe, but can induce bone marrow suppression and promote infectious complications. For patients who do not respond to the thiopurines, methotrexate is an alternative. There is little role for cyclosporine in Crohn's disease; its efficacy/toxicity profile in this disease is poor.

Infliximab is a chimeric monoclonal anti-tumor-necrosis-factor antibody that has efficacy in inducing remission and in promoting closure of enterocutaneous fistulas. 37 Infliximab is generally well tolerated but should not be used in patients with ongoing septic processes, such as undrained intra-abdominal abscesses.

Surgical Therapy

Indications for Operation. Patients with Crohn's disease are usually operated on because an intestinal complication of Crohn's disease mandates the operation. All in all, about 70% of patients with Crohn's disease will come to operation. The most common complications leading to operation are recurrent intestinal obstruction, intestinal perforation with fistula formation and abscess, or a gastrointestinal bleeding.² Obstruction is usually partial and is seldom complete. With nasogastric suction and intravenous nutrition, the distended bowel usually decompresses, and bowel movements resume. Failure to achieve complete resolution or recurrence of obstructive symptoms with the resumption of oral feedings usually leads to operation. Perforation with fistula formation and resultant abdominal mass usually causes continuing pain, fever, malaise, and weight loss until operation can be accomplished. Perianal complications, such as abscess and fistula, commonly lead to operation. Bleeding, a less frequent cause of operation, is usually not massive, but it may be persistent and contribute to chronic anemia until the offending lesion or lesions can be resected. Patients with small intestinal Crohn's disease usually require operation for obstruction or perforation, whereas those with large intestinal Crohn's are usually operated on for chronic debility and failure to respond to medical therapy.

Severe systemic symptoms, intractable medical therapy, and weight loss, especially with growth failure in children, can also lead to operation. Prepubertal and early pubertal patients experiencing growth failure from Crohn's can be expected to sustain *catch-up* and accelerated growth after resection.¹⁶ Toxic megacolon and cancer of the small or large intestine are less common intestinal complications requiring operation. Extraintestinal complications in and of themselves seldom require intestinal operation, but they often contribute to the decision to operate. Most of the extraintestinal complications, with the exception of ankylosing spondylitis and the hepatic complications, subside with the excision of intestine grossly involved with Crohn's disease.

Preoperative Preparation. The nutritional status of the patient is optimized before operation. This sometimes, but not often, requires parenteral caloric supplementation. Anemia is treated by blood transfusion. For patients currently on or recently receiving corticoid therapy, additional steroids— usually 100 mg. of hydrocortisone intravenously every 8 hours—are given to ensure an adequate supply during the operative stress. The bowels are cleansed with laxatives and enemas the day prior to operation. Alternatively, 4 liters of an electrolyte solution (GoLYTELY) can be given by mouth the night before operation.³⁶ Diet is restricted to clear liquids the day before operation.

The growth of enteric bacteria in patients having the laxative-enema preparation is suppressed by giving oral neomycin 0.5 gm. every 4 hours, and tetracycline, erythromycin, or metronidazole 250 mg. every 4 hours, for 18 hours prior to operation. When the GoLYTELY preparation is used, 2 gm. neomycin and 2 gm. metronidazole are given by mouth 12 hours and 8 hours before operation. With both preparations, cefazolin 0.5 gm. is given intravenously just prior to operation and is continued every 8 hours for two more doses.

General Principles of Operation. Because Crohn's disease involves nearly the entire gastrointestinal tract in most patients, total excision of the disease is not possible. Thus, surgical treatment is directed at the most severe areas of involvement, including those that account for the complications of obstruction, bleeding, or perforation.

The two main operative approaches are to excise the lesions or to bypass them. Currently, most surgeons advise excision rather than bypass. Bypass allows the diseased intestine to remain in place, where it can cause continuing symptoms, require treatment, and perhaps even develop malignancy. The risk of cancer in bypassed small and large intestine with Crohn's disease is greater than the risk in healthy bowel. Excision is done with 3-cm. "disease-free" margins on both sides of the area of involvement. The disease-free margins are established by gross inspection. Most surgeons do not use microscopic confirmation of healthy borders. Although the authors have found that when borders are free of microscopic involvement there are fewer recurrences over the long term than when the border is involved,³⁷ others have not found a higher recurrence rate when histologic findings of

Crohn's are present in the margins. Certainly, demanding microscopic borders that are free of disease may lead to excessively large resections and result in the short bowel syndrome. Patients with this syndrome do not have enough intestine remaining to digest and absorb their food properly.

After resection and anastomosis of the index segment (or segments) of intestine that has led to the operation, fistulas from the index segment to adjacent organs, such as the stomach, colon, duodenum, bladder, or vagina, can usually be closed by suture of the entrance of the fistula into the adjacent segment. Resection of the adjacent segment is seldom required, unless it is primarily involved with gross Crohn's disease.

UNSPECIFIC ULCEROUS COLITIS

Ulcerative colitis, a diffuse inflammatory disease of the mucosal lining of the colon and rectum, is characterized by bloody diarrhea that exacerbates and abates without apparent cause. It is difficult to realize that a disease so devastating remains without an identified etiology or specific medical therapy. Total removal of the affected organs—the colon and rectum—provides a complete cure, but at a sacrifice, since patients so treated must learn to live with an external abdominal stoma (an ileostomy) for the remainder of their lives. Since the disease has its peak onset in early and middle adulthood, this represents a long time span for most patients. Fortunately, new surgical alternatives have eliminated the need for a permanent ileostomy without sacrificing definitive treatment of the disease.

ETIOLOGY

The etiology of ulcerative colitis remains unknown despite intensive work by many investigators. The examination of bacterial and viral agents continues to be an area of great activity. Whether the infectious agents are more likely to be triggers of disease or perpetuators of disease is of great controversy. To be a trigger, an infectious agent would have to act by initiation or reactivation. Agents could initiate an autoimmune response by altering antigens, affecting molecular immunity, or increasing immune responsiveness. The microbial agent might also trigger the pathologic response by increasing mucosal permeability or stimulating epithelial injury or localized ischemia. The microbial agent could reactivate the inflammatory process directly, by secondary infection, or by the release of toxins. Evidence for microbial agents as triggers in inflammatory bowel disease is only indirect.

Psychologic factors have long been thought to have a critical role in exacerbations of the disease. It is now clear that patients with ulcerative colitis have no unusual predisposing factors when compared with matched controls. Moreover, colectomy is usually followed by a marked improvement in pre-existing morbid psychologic states such as depression or social estrangement. Psychosomatic factors most likely only facilitate the colonic mucosal reaction to another as yet unidentified causative agent.

Another area of great interest has been that of cytokines and immunoregulatory molecules involved in the control of the immune response.⁶¹ The production of interferon during inflammation could have a significant role in the differentiation of mature memory and effector cells within the intestine. Specific activities of interleukins that are potentially relevant to inflammatory bowel disease have been identified. Most important of these may be interleukin-1 (IL-1), which activates T and B lymphocytes as well as macrophages and neutrophils. IL-1 stimulates production of eicosanoids, cytokines, growth factors, and destructive enzymes; increases adhesion of neutrophils and monocytes to endothelial cells; induces acute-phase response as well as fever, anorexia, and sleep; and stimulates collagen production and thus fibrosis. IL-1 has been shown to be elevated in ulcerative colitis as well as in experimental models of colitis. The increase in IL-1 levels seems to correlate with severity of disease. Alterations in IL-2, IL-6, IL-8, and interferon-gamma have been identified in tissues from patients with ulcerative colitis. The production of interferon during inflammation could play a significant role in the differentiation of mature memory and effector cells within the intestine. Tumor necrosis factor may also be particularly important in the activation of mesenchymal cells but has not been fully evaluated in ulcerative colitis. Thus, it appears that cytokines are integrally involved in the pathogenesis of inflammatory bowel disease with both immunoregulatory and proinflammatory properties.

PATHOLOGY

Ulcerative colitis is, for the most part, a disease confined to the mucosal and submucosal layers of the colonic wall, progressing from mucosal edema and lipemia to vascular congestion, superficial ulcers, increased cellular infiltration of the lamina propria, and cyst abscesses beginning in the rectum and advancing proximally to involve the entire colon. In 10% of patients, the terminal ileum may show mild inflammation and dilation, a process that has been called *backwash ileitis*. On gross inspection, the colonic mucosa demonstrates healed granular superficial ulcers superimposed on a friable and thickened mucosa with increased vascularity. Patients may also demonstrate superficial fissures and small and regular pseudopolyps. This is in contradistinction to the transmural inflammatory changes found in Crohn's disease of the colon, in which all layers may

be involved in a granulomatous inflammatory process. The pathologic changes observed in ulcerative colitis, however, are nonspecific and can be seen in shigellosis, amebiasis, and gonorrheal colitis.

CLINICAL MANIFESTATIONS

The initial presentation of ulcerative colitis can take many forms. Bloody diarrhea is the most common early symptom. Occasionally, extraintestinal manifestations, including arthritis, iritis, hepatic dysfunction, and skin lesions, may be paramount. The disease presents as a chronic, relatively low-grade illness in most patients. In a small number of patients (15%), it has an acute and catastrophic fulminating course. Such patients present with frequent bloody bowel movements (up to 30 per day), high fever, and abdominal pain. The disease therefore has a wide spectrum of clinical manifestations, ranging from a mild diarrheal illness to an overwhelming life-threatening event of short duration that demands immediate medical attention.

Onset of the disease occurs in patients less than 15 years of age in approximately 15% of cases, and presentation in patients over 40 years of age is not uncommon. The incidence of ulcerative colitis is 3.5 to 6.5 per 100,000 population, and the prevalence is 60 per 100,000. A slight female predominance has been reported.

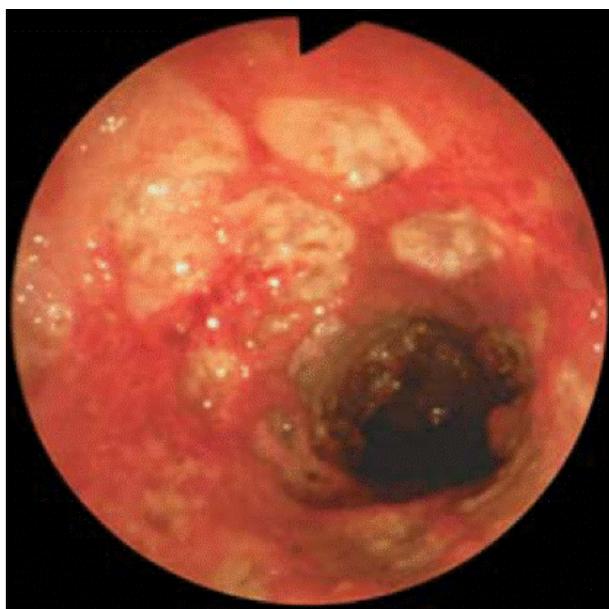
Physical findings are directly related to the duration and presentation of the disease. Weight loss and pallor are usually present. In the active phase, the abdomen, in the region of the colon, is usually tender to palpation. There may be signs of an acute abdomen accompanied by fever and decreased bowel sounds. This is especially true during acute attacks or in the fulminating form of the disease. Abdominal distention is unusual, except in patients who have toxic megacolon, in which case the patient is usually febrile and has signs of an acute abdomen. The perianal area may be excoriated from the numerous wipings associated with bowel movements. There may be evidence of perianal inflammation in the form of a fissure, abscess, or fistula in ano, although the last is more common in Crohn's disease. Rectal examination is almost always painful and, in the presence of perianal inflammation, should be done with gentle care. Examination of the integument, tongue, joints, and eyes is important, since the presence of disease in these areas suggests ulcerative colitis as a likely cause of the diarrheal illness.

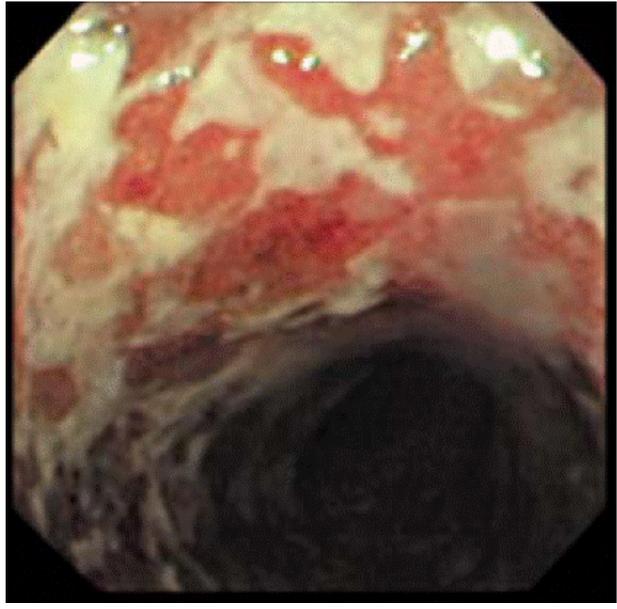


The contracted appearance of this colon, as viewed by barium roentgenogram, is typical of advanced ulcerative colitis in its chronic phase.

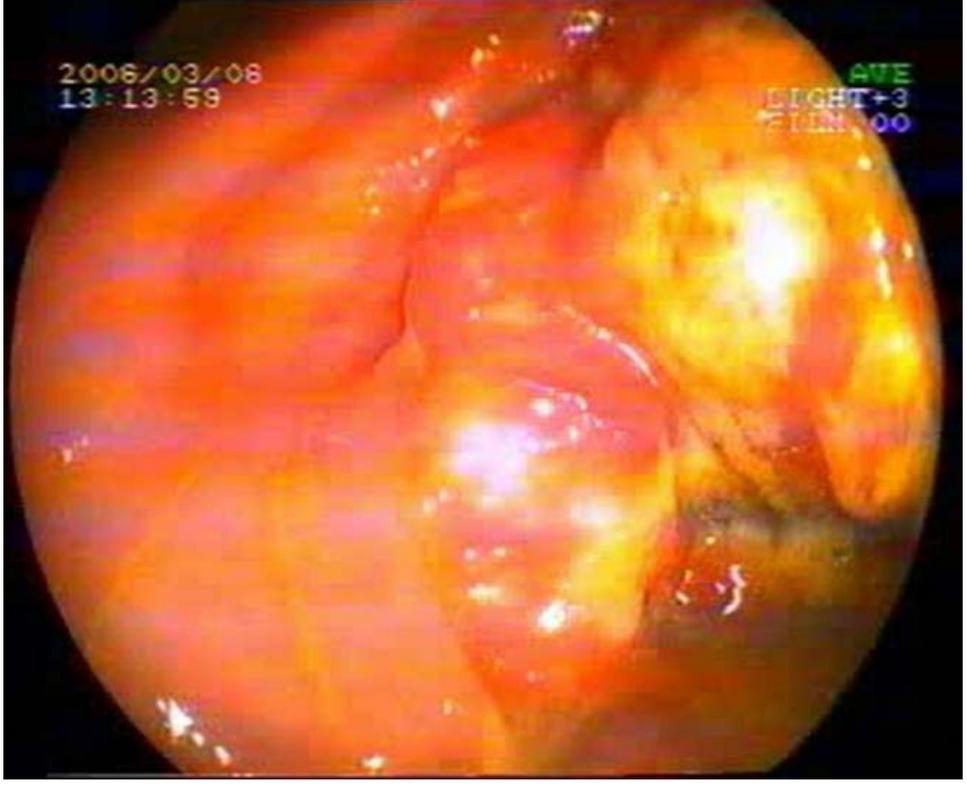
Proctosigmoidoscopy is a helpful and specific diagnostic aid, since ulcerative colitis involves the distal colon and rectum in 90% to 95% of cases. In fact, the mucosa of both the rectum and the sigmoid colon is usually erythematous and granular and bleeds easily when touched by the endoscope or rubbed with a cotton swab. Normal colonic vascular markings may be absent, or the mucosa may be hyperemic; in the disease-bearing mucosa, superficial (less than 2 mm.) mucosal alterations are seen. The intercolonic haustra are thick and blunted. Cobblestoning and deep linear ulceration, which are common endoscopic findings in Crohn's disease, are unusual in ulcerative colitis. In advanced disease, ulcers may be present, surrounded by hyperplastic areas of granulation tissue and edematous mucosa, which may assume a polypoid appearance (pseudopolyps). Mucosal bridging is also commonly found. In chronic advanced disease, the lumen of the rectosigmoid may be remarkably contracted. The use of flexible sigmoidoscopy has improved diagnostic accuracy and patient acceptance. Colonoscopic examination is of value in determining the extent and activity of the disease. Unless a distinct granuloma is identified, endoscopic biopsies are of little value in differentiating ulcerative colitis from Crohn's colitis.

Although recent studies suggest that previous reports may have overestimated the risk of cancer in the adult population with ulcerative colitis, patients with this disease still appear to be confronted with at least a 10% to 20% likelihood of developing carcinoma within 20 years of the diagnosis of ulcerative colitis. Adenocarcinoma in association with ulcerative colitis is multicentric in 15% of patients. In addition, the cancers tend to be flatter and perhaps more infiltrating. These tumors are more evenly distributed throughout the colon, with approximately 50% being found proximal to the splenic flexure. Carcinoma in association with ulcerative colitis is more difficult to diagnose by history and physical examination, stool guaiac testing, and radiographic studies. The likelihood of carcinoma in patients with ulcerative colitis appears to relate to both the extent of colonic involvement and the duration of disease. Although it is generally accepted that patients with extensive total ulcerative colitis are at increased risk of developing carcinoma, the question of what constitutes extensive colitis is still not fully resolved. In addition, the assessment is variable if judged radiographically or colonoscopically. The evidence that patients with left-sided ulcerative colitis, by any criteria, are at increased risk when compared with the general population—which carries a 4% to 6% likelihood of developing colorectal carcinoma, with three fourths of these cancers occurring on the left side—is far from overwhelming. The likelihood of cancer may be related to duration of activity and age of onset, although this has not been clearly established. Although it was held for some time that the carcinoma associated with ulcerative colitis was more aggressive than that in the general population, recent studies have demonstrated that the natural evolution of the cancer is likely the same in both groups.





Unspecific ulcerative colitis



Unspecific ulcerative colitis. Malignization



Unspecific ulcerative colitis. Pseuopoliposis

Rectal biopsies have also been advocated to assess the presence or absence of dysplasia. Morson and Pang⁷¹ advocate a surveillance program of rectal biopsy to assess the point at which a patient becomes at high risk for colonic cancer. When dysplasia of the rectal mucosa is identified, colectomy has been advocated. Other investigators in this field have found the test less useful, with false-negative results of 20% to 40% and false-positive results of 30% to 40%.⁹⁰ Colonoscopy may improve the accuracy of surveillance; however, random biopsies have a very low yield because of the immense sampling problem. Between 20 and 25 equally spaced biopsies are required on a 10-cm. length of colon to reasonably detect a patch of dysplasia 2 cm. in diameter. Moreover, the endoscopic appearances of both dysplasia and carcinoma in ulcerative colitis remain nearly undocumented. The biopsy of target lesions, that is, any lesion that cannot be reasonably accepted as part of the chronic disease state, is recommended. In addition, the end-point of surveillance remains controversial. Many gastroenterologists recommend colectomy only in the presence of high-grade dysplasia, a dysplasia-associated mass lesion, or a frank carcinoma. Unfortunately, the presence of dysplasia, whether low-grade or high-grade, can give rise directly to an invasive carcinoma, and all large centers have had patients under surveillance who developed and died of colorectal carcinoma. Some evidence suggests that even low-grade dysplasia unassociated with severe inflammation, if it is unequivocal, should prompt colectomy. To date, no prospective study has clearly demonstrated that surveillance lowers the mortality from colorectal cancer in association with ulcerative colitis, although a large review from St. Marks suggested an overall 5-year survival of 87% in those patients who underwent surveillance and 55% in those who did not.

A plain abdominal film may reveal a variant of the disease called *toxic megacolon*, in which there may be free air within the peritoneal cavity from perforation of the colon. A more common sign is a remarkable dilation of the transverse colon.

Barium enema examination, usually with air contrast, can be performed safely in most patients and is extremely helpful in identifying the extent and severity of the disease. Barium roentgenographic signs include loss of haustral markings and irregularities of the colon wall, which represent small ulcerations. These are well demonstrated in picture, which contrasts the appearance of the left side of the splenic flexure and that of the right. As the disease progresses, pseudopolyps become a prominent roentgenographic sign. In advanced disease, the colon assumes the appearance of a rigid contracted tube. The

barium roentgenogram, although useful, should be avoided in the presence of toxic megacolon, since it may exacerbate the colitis. When diarrhea is not present, a liquid diet for 3 days prior to examination is recommended. Barium roentgenogram should be omitted when the clinical signs of toxic megacolon are present. Upper gastrointestinal contrast studies are also indicated in most patients to exclude Crohn's disease.

The aforementioned clinical manifestations and simple diagnostic tests usually help identify the presence of ulcerative colitis. It is necessary, however, to obtain stool smears and cultures to exclude colitis due to viruses, *Chlamydia*, bacterial pathogens, and parasites. Particularly important and difficult to exclude are pseudomembranous colitis, the proctocolitis seen increasingly in homosexual males, and traveler's diarrhea. Cello and Meyer¹² provided a useful schema for distinguishing ulcerative colitis from granulomatous colitis. Note, however, the low frequency of discriminating clinical characteristics, except for associated small bowel disease or skip areas within the colon, when the etiology is Crohn's disease.

The strong association of cancer of the colon with ulcerative colitis bears further emphasis.¹¹⁵ For example, 40% of patients with total colonic involvement may die of cancer if they survive their disease and the colon is left in place.⁶² Three percent of children with ulcerative colitis have cancer of the colon at 10 years; 20% develop cancer during each ensuing decade.²⁰ With the availability of far more acceptable surgical alternatives to proctocolectomy and ileostomy, it is hoped that patients will obtain definitive treatment for the disease well before they enter the phase of accelerating cancer risk. These data support close medical management for such patients and surgical intervention, on this basis alone, when chronicity is well established.

The extracolonic manifestations of ulcerative colitis can be categorized as the colitis group, the pathophysiologic group, and the miscellaneous group of disorders. The colitis group of extracolonic manifestations generally parallels the activity of the underlying bowel disease, being present and most active when the colitis is active and usually subsiding when the colitis goes into remission induced by medical therapy, by surgical intervention, or spontaneously. It appears most likely that these extracolonic disorders represent antigen-antibody immune complex disorders. Ocular manifestations are common in ulcerative colitis and include conjunctivitis, iritis, and choroiditis. These are closely related to disease activity and respond to steroid therapy. More severe and rare eye diseases, including ulcerative panophthalmitis, are more difficult to treat, even with high-dose steroid suppression. Articular disorders, including peripheral joint disease, arthralgias, swelling, pain, and redness with migratory involvement, usually parallel the intensity of the colitis and respond to medical or surgical treatment. The joints of the lower extremities are most frequently involved. Fortunately, permanent deformity of these joints is very uncommon. A certain percentage of patients go on to develop clear evidence of rheumatoid arthritis even after colectomy. Ankylosing spondylitis and sacroiliitis, in contrast, can cause permanent fixation of the spine and need to be treated aggressively. Bone involvement specific to the axial skeleton is less closely related to the severity of the inflammatory state of the colon and, in fact, may precede frank evidence of ulcerative colitis. Patients with ulcerative colitis frequently experience dermatologic disorders, including erythema nodosum and pyoderma gangrenosum. Although these difficult problems resolve after colectomy in most patients, in others, they may precede the colonic disease or may not become manifest until after proctocolectomy has been performed.

Pathophysiologic disorders are more often seen in Crohn's disease than in ulcerative colitis, since in ulcerative colitis, the normal physiology of the terminal ileum is not disturbed. Liver disease is common in patients with both ulcerative colitis and Crohn's disease. Nonspecific inflammation and fatty metamorphosis manifested by mild increases in the serum transaminase values are common in ulcerative colitis. Pruritus and elevation of the alkaline phosphatase are commonly associated with the pericholangitis that occasionally accompanies ulcerative colitis. The most dreaded complication, sclerosing cholangitis, presents with pruritus, alkaline phosphatase elevation, right upper quadrant pain and tenderness, and jaundice. The diagnosis is most often made by endoscopic retrograde cholangiopancreatography or transhepatic cholangiography. It has been estimated that 50% of patients who present with sclerosing cholangitis already have or will develop frank ulcerative colitis. Controversy surrounds the treatment of this disorder. Whereas some patients respond to colectomy, many others show progression of their disease even after colon resection. Surgical drainage, internal stent placement, antibiotics, and ultimately liver transplantation have all been reported to be of value in the treatment of symptomatic sclerosing cholangitis. Cholangiocarcinomas have also been reported in patients with ulcerative colitis, usually after many years of sclerosing cholangitis.

MEDICAL MANAGEMENT

The outcome of an acute episode of ulcerative colitis relates to the severity of the disease as manifested by systemic symptoms. Duration of the disease and extent of involvement of the colon do not appear to be determinants of survival if ulcerative proctitis is excluded from consideration. Those who present with advanced signs of acute illness require hospitalization and supportive, as well as specific, therapy for associated metabolic and hematologic derangements. Because of the massive fluid and electrolyte loss per rectum, such patients often present with metabolic acidosis, contracted extracellular volume, and prerenal azotemia. The serum potassium level is usually low because of excessive loss in stool and urine. Intravenous administration of balanced salt solutions in amounts sufficient to replace these losses is an initial step in management. Patients with long-standing disease may have lost considerable protein and probably are in a depleted nutritional

state. The precise role of specialized nutritional support in ulcerative colitis and, in particular, of total parenteral nutrition is unclear. Despite early enthusiasm, total parenteral nutrition does not appear to have a specialized therapeutic role in this disease. Total parenteral nutrition improves the overall nutritional state of patients with ulcerative colitis and may reverse growth retardation in children, but it certainly does not replace conventional medical treatment or prevent or delay colectomy. In fact, in patients with severe acute colitis, it may be impossible to attain a positive nitrogen balance while the colon is still in place.

Corticosteroids and immunosuppressive agents have both been demonstrated to be effective in the management of ulcerative colitis. Both agents, however, are capable of producing significant side effects. In general, corticosteroids have been more readily accepted by the medical community as therapeutic agents and remain the mainstay of therapy in acute attacks. Between 40 and 60 mg. of prednisone in a single daily dose is effective in inducing remission.¹¹⁰ Rectal steroids have been shown to be effective in left colon disease or proctitis and may have therapeutic efficacy in universal colitis as well, perhaps because approximately 30% of the steroid given rectally is absorbed into the systemic circulation. In an attempt to avoid systemic effects of steroid enemas, tixocortol pivalate was synthesized by adding a thiol ester group at position 21 on the hydrocortisone molecule. In trials, this agent has been useful for treating patients with left-sided colitis and has resulted in a reduction in systemic steroid side effects. The controversy over intravenous steroids versus intravenous adrenocorticotropic hormone (ACTH) has now been resolved by a randomized trial that revealed a similar response to equipotent doses of either hormone.⁴⁹ A recent study suggests that ACTH may be more effective in patients not previously treated with corticosteroids, whereas corticosteroids appear to be preferable for patients already receiving steroid therapy.⁶⁸ A steroid-induced remission is not more likely to exacerbate than a spontaneous remission, and an ACTH-induced remission is not more likely to exacerbate than a corticosteroid-induced remission. The usual recommended doses are 300 mg. of hydrocortisone or 40 units of ACTH per day. Occasionally, massive doses of steroids (over 1 gm. per day) are required. The usual response is rapid, and acute signs of inflammation subside within a few days. The optimal duration of intravenous steroid therapy is 5 to 7 days, although this may be extended in patients supported nutritionally with total parenteral nutrition. Proctoscopic examination is useful in following response to therapy. There is still controversy as to whether maintenance steroid thereby reduces recurrence of the disease. Although maintenance steroids may be useful in controlling symptoms of patients with continuing activity, maintenance therapy with low-dose corticosteroids for patients with inactive disease has not been demonstrated to prevent relapse.⁵⁹ Patients must be monitored carefully for the long-term adverse sequelae of corticosteroid use, including hypertension, hyperglycemia, cataracts, osteoporosis, and osteomalacia.

Sulfasalazine has enjoyed widespread use in the chronic phases of ulcerative colitis. Its mode of action is unknown. Sulfasalazine may exert this prophylactic effect by inhibiting mucosal prostaglandin synthesis,⁴⁹ although not all studies have supported this mechanism.⁸¹ Whatever the mechanism of action, sulfasalazine appears to be associated with fewer exacerbations as assessed by controlled randomized trials.^{21, 108} The drug appears to be of lesser value in severe ulcerative colitis. Sulfasalazine is metabolized by bacteria to 5-aminosalicylic acid (5-ASA) and sulfapyridine. Dose-related side effects of sulfasalazine include nausea, vomiting, headache, and abdominal discomfort. Reversible hypospermia and infertility are observed in males. Hypersensitivity effects include fever, skin rash, agranulocytosis, and hemolytic anemia. Studies have indicated that the sulfapyridine produced by bacterial degradation of sulfasalazine is responsible for the majority of the side effects, whereas the 5-ASA component appears to be the effective moiety of the drug. Five-aminosalicylic acid is now available in this country for clinical use. In all studies to date, these compounds have been shown to be as efficacious as sulfasalazine in treating acute ulcerative colitis as well as in preventing relapse.

A third approach has been the use of immunosuppressive agents. Rosenberg and colleagues⁸⁸ concluded, based on a well-controlled study, that azathioprine allows reduction of the use of steroids in chronic cases but does not, in itself, control exacerbation of the disease. In a more recent controlled trial, however, Kirk and Lennard-Jones⁵⁴ demonstrated that clinical improvement may occur in about 25% of patients treated with a dose of azathioprine at 2 to 2.5 mg. per kg. Uncontrolled trials have demonstrated a favorable response to 6-mercaptopurine (6-MP) in 64% to 70% of patients with refractory ulcerative colitis.⁸¹ Because these drugs do not produce a clinical response for several months, they have no role in the treatment of acute flares of ulcerative colitis. Cyclosporine (CS), which has a more rapid onset of action, has been advocated for the treatment of severe, refractory acute ulcerative colitis. Both uncontrolled trials and one controlled study suggest that high-dose CS is efficacious for severe ulcerative colitis. There is, however, significant theoretical risk of irreversible CS-associated nephropathy following treatment with high-dose CS. Severe infectious complications may also occur.⁹² A trial of 6-MP or CS may be warranted when steroids and sulfasalazine have failed, when the disease is confined to the left side of the colon or rectum, when the patient is compliant, and when there is no absolute indication for immediate surgical therapy.⁸⁰ However, before prescribing these immunosuppressive agents, one must be fully familiar with the dosing, monitoring, toxicity, and possible induction of lymphoma or other malignancies associated with these drugs.

Although widely prescribed for both ulcerative colitis and Crohn's disease, metronidazole and other antibiotics have no proven value in the treatment of inflammatory bowel disease.

The major therapeutic problem between acute episodes is control of diarrhea and maintenance of nutrition. Diet therapy is

no longer recommended, and patients are encouraged to eat a substantial diet of their choice. Milk products are to be avoided only if they cause problems such as increasing diarrhea or cramps (as they may in about half of patients with ulcerative colitis). The reason for this is not clear but relates to something specific in cow's milk rather than to the lactase deficiency that exists in many patients with ulcerative colitis. Opiates such as codeine or paregoric should be avoided. Nocturnal diarrhea can be controlled by anticholinergics or diphenoxylate with atropine. The synthetic peripheral-acting opioid loperamide may be more effective than diphenoxylate in this situation and avoids the atropine side effect associated with this drug.⁷⁴ Stool bulk formers, such as psyllium, are also helpful. Finally, the importance of rest and peace of mind cannot be overemphasized. Patients are advised to remain at rest during episodes of exacerbation.

INDICATIONS FOR SURGICAL TREATMENT

Since total removal of the colon and rectum (proctocolectomy) cures ulcerative colitis, one might reasonably ask why all patients with established chronicity are not so treated. The incidence of surgical intervention appears to be related to the availability of skilled and knowledgeable gastrointestinal surgeons and enlightened physicians. For example, the clinic at Leeds offers surgical care to approximately half of its patient population,³⁸ whereas in another center the reported operative rate is below 10%.³⁵ There are several well-identified complications that require urgent operation for survival.³⁶ These include massive, unrelenting hemorrhage; toxic megacolon with impending or frank perforation; fulminating acute ulcerative colitis that is unresponsive to steroid therapy; obstruction from stricture; and suspicion or demonstration of colonic cancer. Surgical therapy is also recommended in children who fail to mature at an acceptable rate. The largest number of colectomies for ulcerative colitis are performed for less dramatic indications, as the disease enters an intractable chronic phase and becomes both a physical and a social burden to the patient.

Acute perforation occurs infrequently, with the incidence directly related to both the severity of the initial episode and the extent of the disease in the bowel. Although the overall incidence of perforation during a first attack is less than 4%, if it is severe, the incidence rises to 9.7%. If the total colon is involved, the perforation rate is 14.6%, and if the attack is both severe and involves the total colon, it increases to 19.2%.³⁷

Obstruction caused by benign stricture formation occurs in 11% of patients, 34% of these occurring in the rectum.¹⁹ They usually follow submucosal fibrosis and occasionally mucosal hyperplasia. Although they do not usually cause acute obstruction, the lesions must be differentiated from carcinoma by biopsy or excision, and particular attention should be given to excluding Crohn's disease. Strictures caused by carcinoma are less common than those caused by benign disease and are more prone to perforate.

Massive hemorrhage secondary to ulcerative colitis is rare, occurring in less than 1% of patients.³⁷ Prompt surgical intervention is indicated after hemodynamic stabilization. More than 50% of patients with acute colonic bleeding have toxic megacolon, so one should be suspicious of the coexistence of the two complications. Uncontrollable hemorrhage from the entire colorectal mucosa may be the one clear indication for emergency proctocolectomy. If possible, the rectum should be spared for later mucosal proctectomy with ileoanal anastomosis.

Acute toxic megacolon can occur in both ulcerative colitis and Crohn's disease. Its incidence is between 6% and 13% in patients with ulcerative colitis.³⁰ Patients usually present clinically with the onset of abdominal pain and severe diarrhea (greater than 10 stools per day), followed by abdominal distention and generalized tenderness. Once megacolon and toxicity develop, fever, leukocytosis, tachycardia, pallor, lethargy, and shock ensue. It is important to note that any of these manifestations can be masked by chronic steroid use and the generally poor nutritional condition of the patient. An abdominal radiograph usually shows dilation of the transverse and occasionally the sigmoid colon that is greater than 5 cm. and averages 9.2 cm. Thickening and nodularity of the bowel wall due to mucosal inflammation are also noted. Caprilli and colleagues¹⁰ reported abnormal gaseous distention of the small bowel in association with toxic megacolon; thus, this finding may be a useful predictor of its development in patients with severe colitis.

The morbidity and mortality for acute toxic megacolon remain high. Soyer and Aldrete¹⁰³ reported a series of 12 patients in which the incidence of postoperative sepsis was 50%, wound infection 58%, abscess of fistula 33%, and delayed wound healing 25%. Postoperative mortality ranges from 11% to 16%; for the subset of patients with perforation, mortality is 27% to 44%.^{42, 47} These data support the use of combined aggressive medical and surgical treatment of this disease.

Initial treatment for toxic megacolon includes intravenous fluid and electrolyte resuscitation, nasogastric suction, broad-spectrum antibiotics to include anaerobic and aerobic gram-negative coverage, and total parenteral nutrition to improve nutritional status. Proctoscopy may be helpful in determining the etiology of the attack, as may culture of the stool. Although the efficacy of steroids is still in question,⁶⁷ most patients presenting with toxic megacolon are already on steroid therapy and thus need stress doses of corticosteroids to prevent adrenal crisis. Most clinicians think that steroids help reduce the inflammation and may *cool down* an acute toxic episode in up to 50% of patients, although long-term remissions are not achieved.⁴⁰ Moreover, the short-term use of corticosteroids does not appear to increase surgical morbidity. Long-term use of larger doses, however, does increase the incidence of wound and septic complications. The authors agree with Fazio³⁰ that,

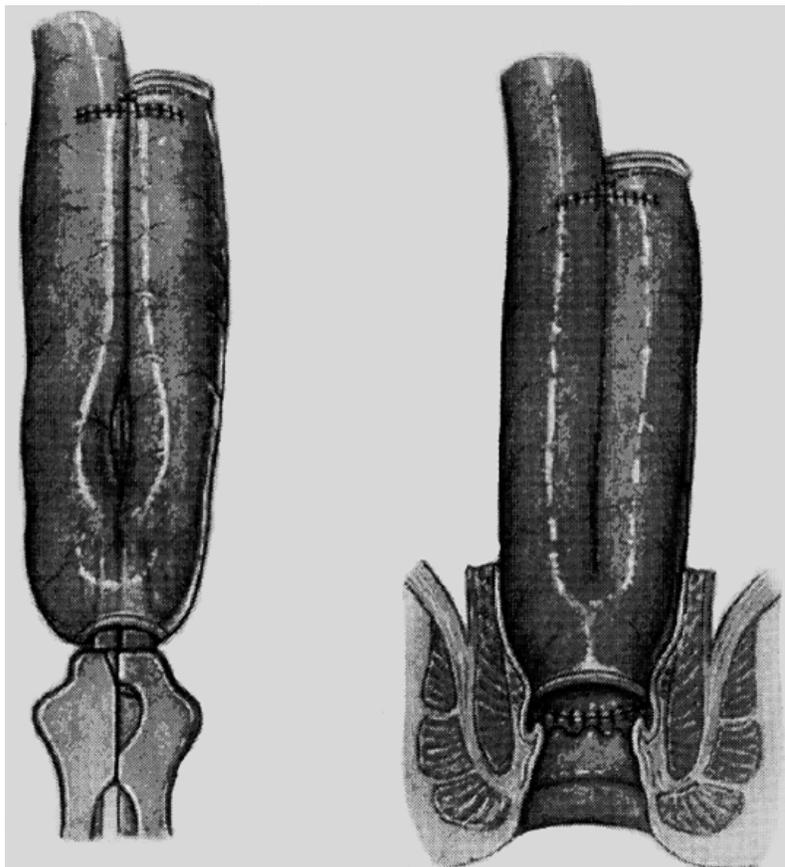
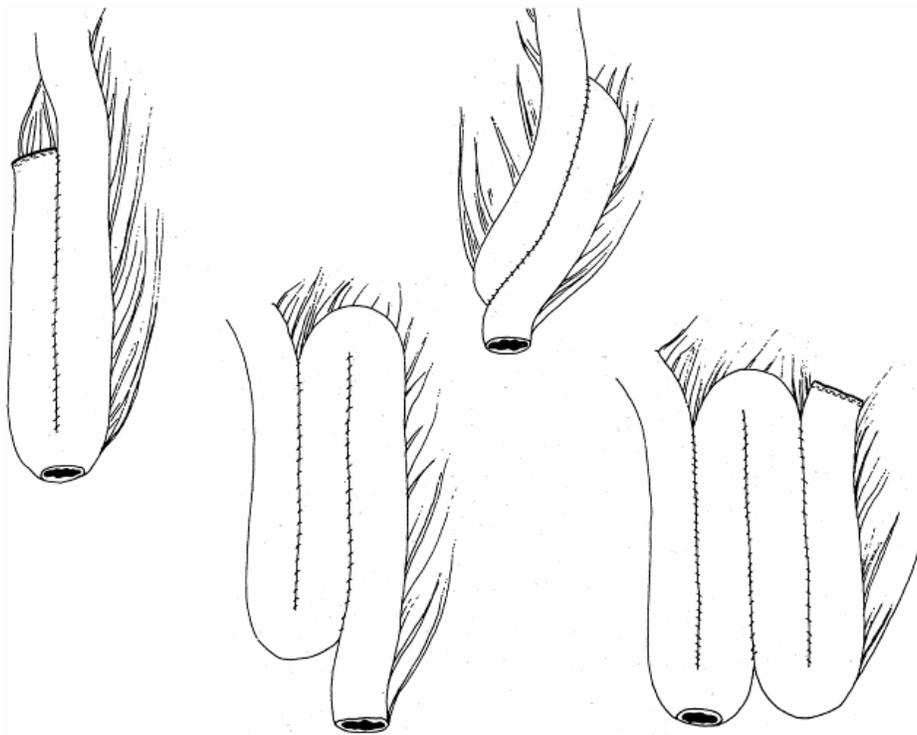
provided the patient is stable, initial medical trial is warranted in order to make the operation elective rather than urgent. If no clear response is obtained within 24 to 48 hours, surgical therapy is warranted. Larger doses of steroids after initial medical failure probably will not benefit the patient and, as noted, may be deleterious. During medical therapy, serial blood counts, serum electrolyte levels, and abdominal roentgenograms should be closely monitored.

In the presence of acute toxic megacolon caused by ulcerative colitis, surgical therapy can be associated with a high operative morbidity and mortality. Block and colleagues⁶ noted an overall mortality following emergency operation of 8.7%; 6.1% after total abdominal colectomy, and 14.7% after proctocolectomy. This suggests that more conservative surgical intervention is appropriate in the acute setting. Also, with the recent popularity of anal sphincter-sparing procedures, when operating for acute ulcerative colitis, one should weigh the possibility of subsequent surgical correction for continence. Specifically, leaving the rectum intact allows its use for subsequent surgical mucosal proctectomy and ileoanal anastomosis. When urgent colectomy is required, total abdominal colectomy, Brooke ileostomy, and Hartmann's pouch are appropriate.³⁹ 97 Although ileostomy alone for acute complications has been abandoned, it has been used in the recent past with good success by Turnbull and co-workers,¹¹¹ in combination with skin-level transverse and sigmoid colostomies, for toxic megacolon. This is a relatively simple procedure that spares such desperately ill patients a major operative intervention until their acute illness has subsided. Because the procedure involves only decompression of the colon and does not remove the acutely inflamed tissue, most surgeons prefer colon resection.

SURGICAL MANAGEMENT

Total proctocolectomy with permanent Brooke ileostomy offers definitive treatment for ulcerative colitis by eliminating diseased mucosa and the risk of malignant transformation. Nevertheless, it remains controversial and is poorly accepted by patients and their physicians. Patients with a permanent ileostomy are incontinent of gas and stool and must wear a collecting bag day and night. As many as 40% to 50% of patients with Brooke ileostomies have appliance-related problems, and the psychologic and social implications, particularly for young patients, are tremendous.⁷⁸ 89 Therefore, the search has continued for adequate alternatives to proctocolectomy and ileostomy.

Until recently, single-stage total proctocolectomy was the procedure of choice when complications of the disease were treated electively. This procedure is performed through a midline incision. The rectum is excised from the abdomen after mobilization and circumferential incision from the perineum. When cancer is not suspected, excision is performed rapidly, with division of the mesentery close to the bowel wall. This principle is especially important in the pelvic colon and rectum, where injury to the sacral parasympathetic nerves may lead to bladder and sexual dysfunction. Endorectal mucosal resection, as described later, appears to offer the best way to avoid such serious complications.³⁴ After standard proctocolectomy, management of the perineal wound is a problem, since chronic infection and poor healing may cause a lingering sinus tract between the buttocks. The authors' preference is to apply active closed drainage to this area for 3 to 5 days following operation. Gauze packing of the perineum should be reserved for pelvic hemorrhage that cannot otherwise be controlled. Perineal wound problems may be reduced by performing an intersphincteric proctectomy, which entails dissecting between the internal and external anal sphincter when removing the rectum, thus preserving the levator ani and external anal sphincter muscles. These muscles can then be included in the closure of the perineum. Using this technique, complete healing of the perineum approaches 95% at 6 months.



The most important modification of the operation was the creation of an ileal pouch or reservoir proximal to the ileoanal anastomosis.

The most frequent late complication in patients undergoing ileoanal anastomosis is ileal pouch dysfunction or pouchitis, which has been reported to occur in 10% to 50% of patients undergoing this procedure for ulcerative colitis. Pouchitis is an incompletely defined and poorly understood clinical syndrome consisting of increased stool frequency, watery stools, cramping, urgency, nocturnal leakage of stool, arthralgias, malaise, and fever. The syndrome is similar to that found in patients with Kock continent

ileostomy pouches. The etiology of this condition is unknown; speculations have included early Crohn's disease, bacterial overgrowth or bacterial dysbiosis, either primary or secondary malabsorption, stasis, ischemia, and nutritional or immune deficiencies.

Mortality for elective surgical therapy is in the range of 0% to 2%; for emergency operation, it is about 4% to 5%; and for toxic megacolon, it rises to 17%.⁵ These are remarkable statistics when one considers the debilitating nature of the disease and the fact that many patients have had long-term steroid therapy. The major complication in all reported series is sepsis, either in the wound or in the intra-abdominal cavity. There is little evidence that the development of more potent and specific antibiotics has significantly reduced the incidence of this complication; attention to the details of operative management continues to be the best way to ensure a smooth postoperative course. The most common late complication of resectional therapy with ileostomy or ileoanal anastomosis is intestinal obstruction, which occurs in about 10% of patients. Other bothersome but nonlethal complications following proctocolectomy include delay in perineal closure (25%), sexual dysfunction (5% to 10%), and renal stones (10%). Ileostomy dysfunction as a consequence of stenosis has been reduced to 2% by the Brooke-Turnbull ileostomy. Additional uncommon complications include prolapse, herniation, and ulceration of the stoma, which is usually a sign of the development of Crohn's disease within the ileal stoma. Whether the outcome following surgical therapy for Crohn's disease of the colon is as favorable as for ulcerative colitis continues to be a source of controversy.

The formation of social groups (ileostomy clubs) has provided an important mechanism for the education of patients by those who have already mastered the technique of ileostomy management. Some hospitals have enterostomal therapists. These professionals are highly skilled in dealing with the physical and emotional problems of stomal management. In institutions that perform a large number of ileal pouch operations, specialized patient-oriented support groups are essential.

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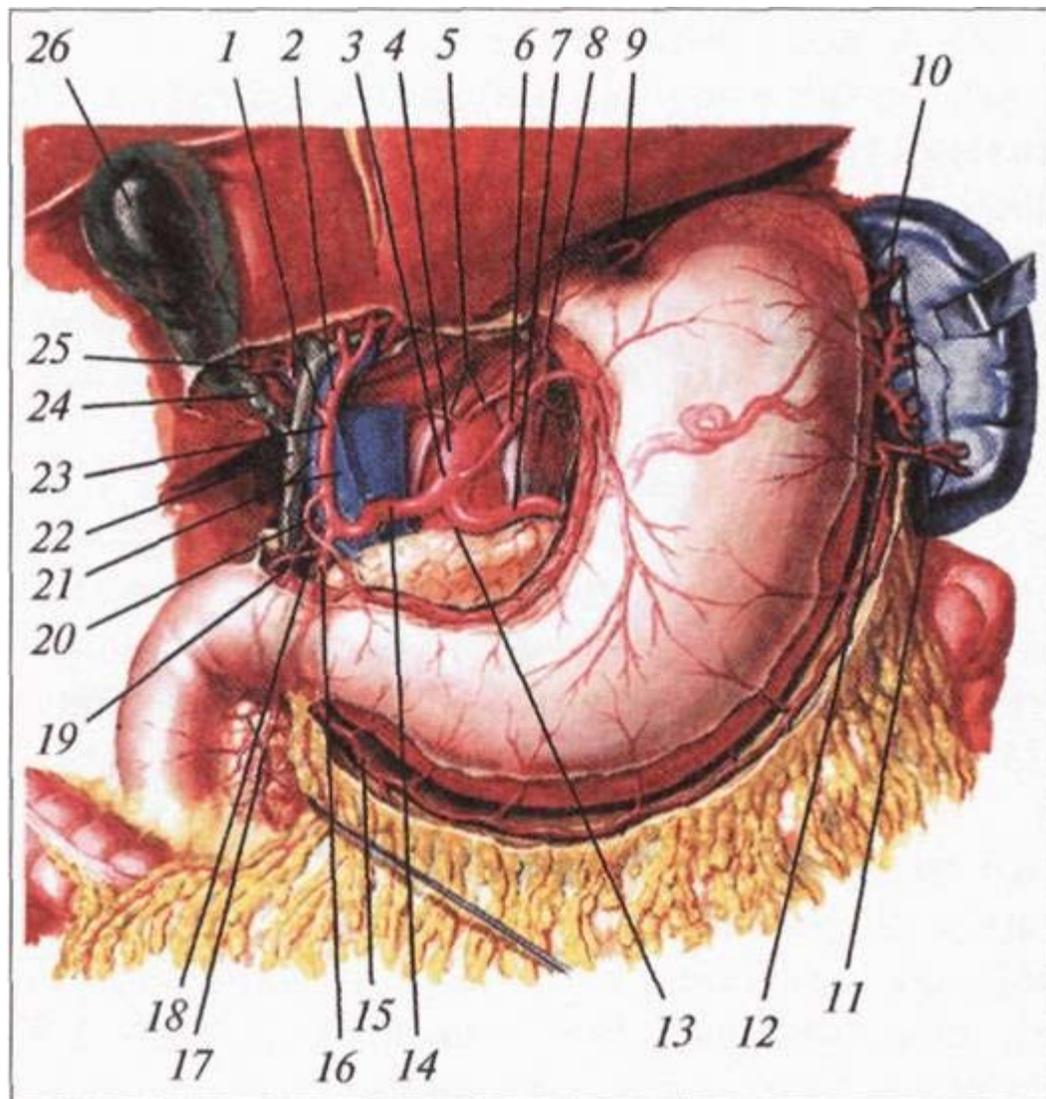
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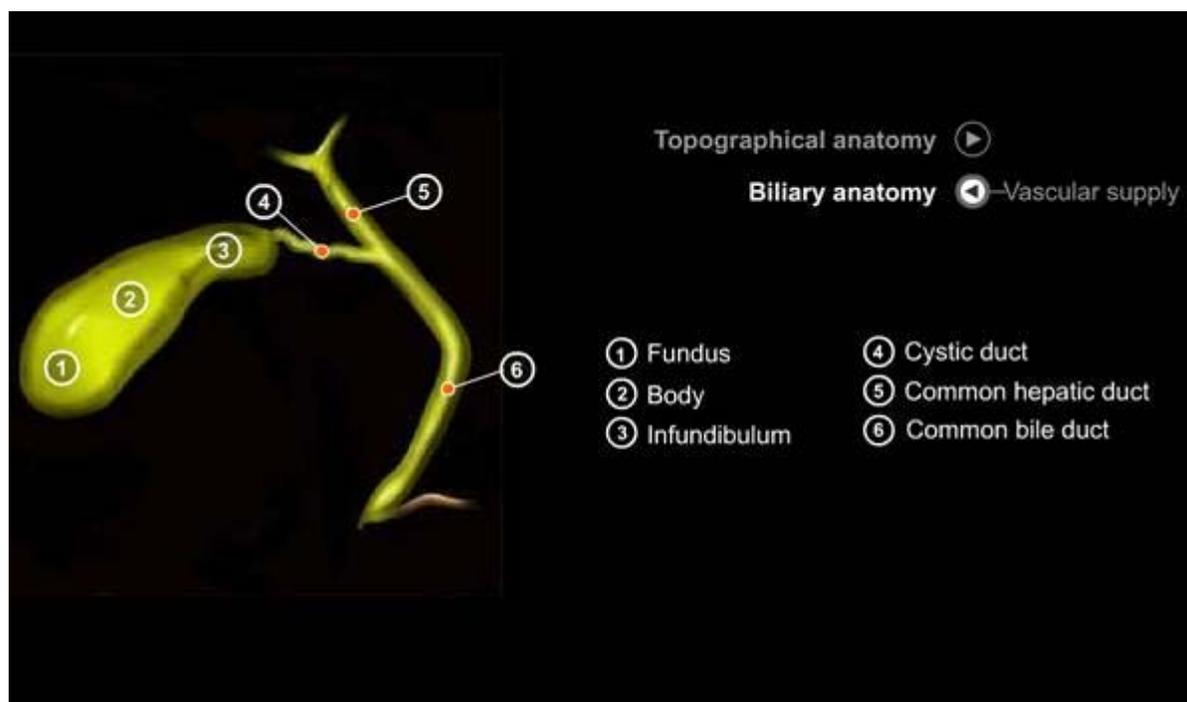
JAUNDICE. CAUSES. DIFFERENTIAL DIAGNOSTIC. MODERN APPROACHES IN TREATMENT. HEPATIC FAILURE IN SURGICAL DISEASES, PREVENTION METHODS AND TREATMENT.



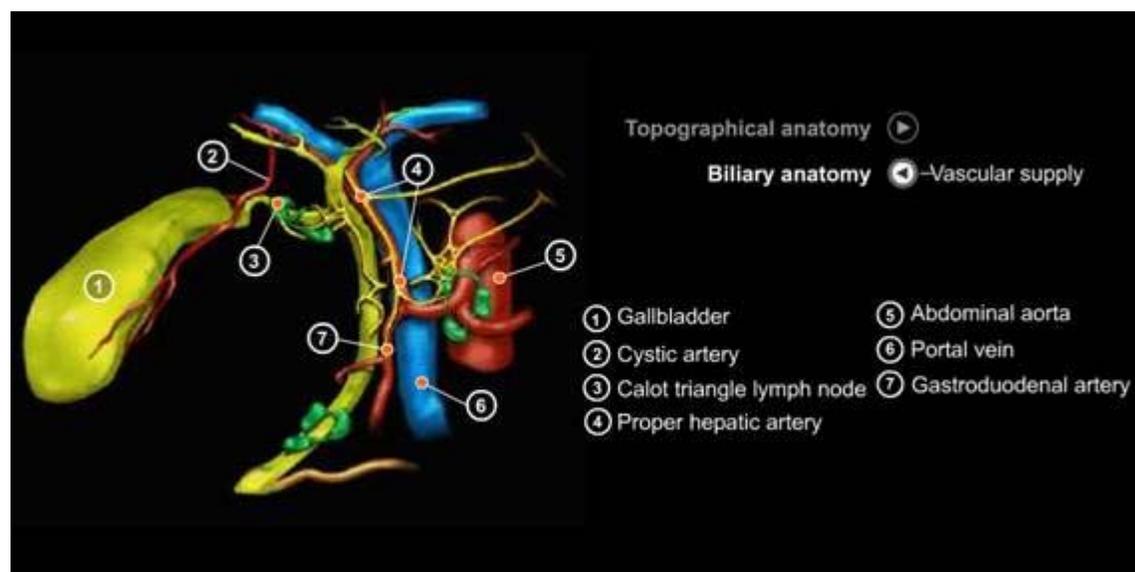
Anatomy of the hepatoduodenal region:

1-A. gastrica sinistra; 2-A. splenica (lienalis); 3 — Rami oesophageales (r. A. gastrica sinistra); 4 — A. phrenica inferior (dextra et sinistra); 5 — Aa. gastricae breves; 6 - A. splenica (lienalis) (Endastre); 7 - A. gastroomentalis (epiploica); 8-A. pancreatica dorsalis; 9-A. hepatica communis; 10 — A. gastroomentalis (-epiploica) dextra; 11 — A. gastroduodenalis; 12 — A. pancreaticoduodenalis; 13-A, pan-

creaticoduodenal superior posterior; 14 —A. supraduodenalis; 15—A. gastrica dextra; 16 — V. portae; 17 — Ductus choledochus; 18 — A. hepatica propria; 19 — Ductus cysticus; 20 — A. cystica; 21 — Ramus dexter; 22— Ramus sinister; 23 — Truncus coeliacus; 24 — Aorta abdominalis; 25 — A. phrenica inferior (dextra et sinistra); 26 — Vesica fellea



Biliary Anatomy

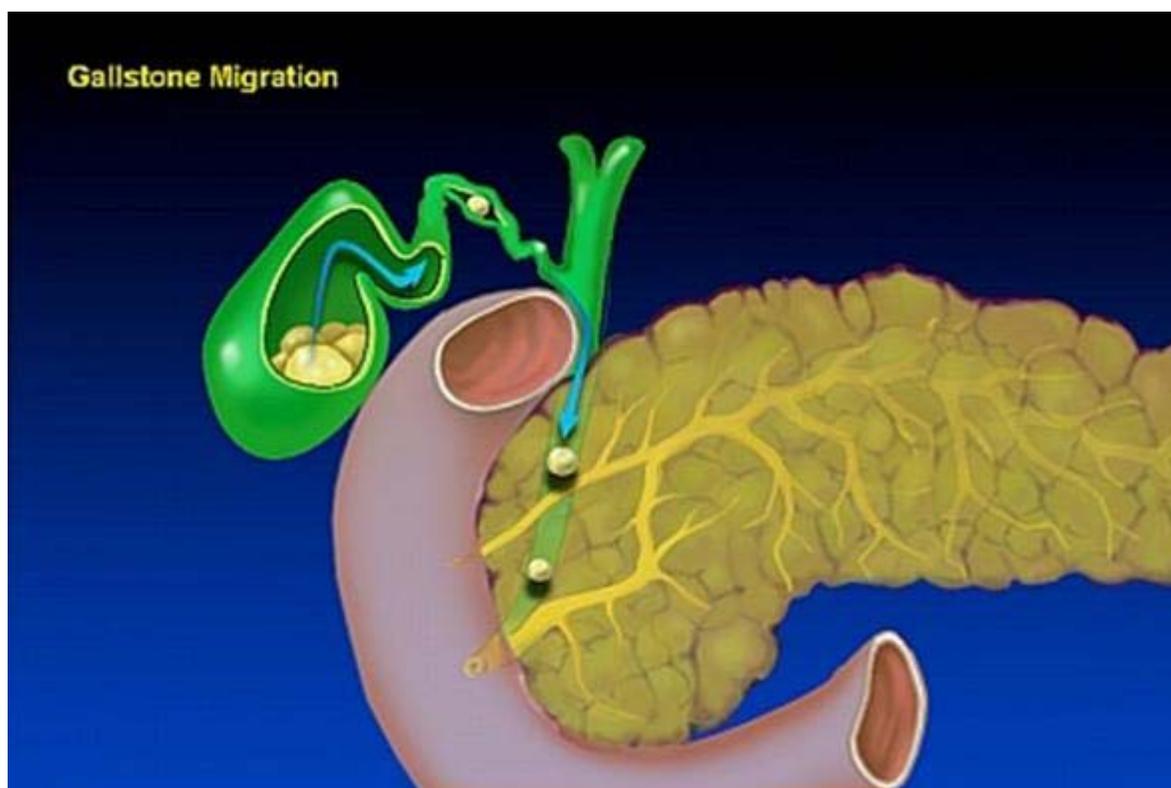


Anatomy: vascular supply

video

OBTURATIVE ICTERUS

A obturative icterus is the type of icterus the reason of which is violation of patency of bilious ways as a result of their obstruction from within or external compression, or cicatrix narrowing.



Obstructive jaundice, also called post-hepatic jaundice, is caused by an interruption to the drainage of bile in the biliary system. The most common causes are gallstones in the common bile duct, and pancreatic cancer in the head of the pancreas. Also, a group of parasites known as "liver flukes" can live in the common bile duct, causing obstructive jaundice. Other causes include strictures of the common bile duct, biliary atresia, cholangiocarcinoma, pancreatitis and pancreatic pseudocysts. A rare cause of obstructive jaundice is Mirizzi's syndrome.

In complete obstruction of the bile duct, no urobilinogen is found in the urine, since bilirubin has no access to the intestine and it is in the intestine that bilirubin gets converted to urobilinogen to be later released into the general circulation. In this case,

presence of bilirubin (conjugated) in the urine without urine-urobilinogen suggests obstructive jaundice, either intra-hepatic or post-hepatic.

The presence of pale stools and dark urine suggests an obstructive or post-hepatic cause as normal feces get their color from bile pigments. However, although pale stools and dark urine are a feature of biliary obstruction, they can occur in many intra-hepatic illnesses and are therefore not a reliable clinical feature to distinguish obstruction from hepatic causes of jaundice.

Patients also can present with elevated serum cholesterol, and often complain of severe itching or "pruritus" because of the deposition of bile salts.

No single test can differentiate between various classifications of jaundice. A combination of liver function tests is essential to arrive at a diagnosis.

Etiology and pathogenesis

Obstruction of external bilious ducts can arise for many reasons: as a result of inflammatory processes of surrounding organs (pancreas, paracholedochus lymphatic knots), damage of walls of ducts during operations. However, in most cases it is a gallstone disease, choledocholithiasis and related to them scar changes of large papilla of duodenum. The second place among the reasons of obturation icterus is taken by tumours of which the cancer of head of pancreas and large papilla of duodenum is most widespread.

Disregarding the fact that each of the adopted diseases has special clinical passing, obturation of bilious ducts causes the changes which have general character. At complete blockade of external bilious ducts and growth of pressure higher than 300 mm of waters. an item or 2,94 kPa (after a norm — not higher 150 mm or 1,47 kPa) in them, excretions of bile in bilious capillaries are halted. It is conditioned by the fact that the secretory mechanism of hepatic cells (hepatocyte) can not overcome such resistance. Thus bile through the blasted particles and lymphatic and vein vessels of liver gets into blood, causing the syndrome of mechanical icterus.

Classification

(by O.O. Shalimov, 1993)

Obturation icteruses are divided into:

I. According to the level of barrier:

- 1) obstruction of distal parts of general bilious duct;
- 2) obstruction of supraduodenal part of general bilious duct;
- 3) obstruction of initial part of general hepatic duct and fork of hepatic ducts.

II. According to the etiologic factor:

1) conditioned by obturation by bilious concrement, strange bodies, grume of blood during hemobilia, parasite, iatrogenic influence during operation;

2) obstruction at the diseases of wall of bilious ways — innate anomalies (hypoplasia, cysts and atresia), inflammatory diseases (obstructing papillitis and cholangitis), scar strictures (posttraumatic and inflammatory), bilious ways tumours of high quality;

3) obstruction caused by the out-of-ducts diseases, that pull them in the process (tubular stenosis of general bilious duct of pancreatic genesis, ulcerous disease of duodenum, paracholedocheal lymphadenitis, peritoneal commissures).

Except that, *according to the duration the disease is distinguished:*

- 1) acute obturative icterus, that to 10 days last;
- 2) protracted, that proceeds from 10 to 30 days;
- 3) chronic, that more than a month lasts.

Symptoms and clinical passing

The clinical picture of obturative icterus is founded, first of all, on the symptoms caused by violation of outflow of bile.

A *pain syndrome* is a characteristic accompaniment of gallstone disease and choledocholithiasis, that run with the attacks of hepatic colic. However, pain syndrome at these pathologies can often be not acutely expressed or quite absent. Pain is often observed at stricture of bilious ducts, but it is not quite typical of patients with the cancer of bile ducts.

An *icterus* is an important sign of obstruction of bilious ways, speed of origin and intensity of which depend on whether well-kept or broken passage of bile in intestine is. For choledocholithiasis with “valve” character of concretions transitional passing of icterus is inherent, and for cancer it is more proof and progressive.

The itch of body is a frequent accompaniment of icterus, that arises up as a result of action of bilious acids. It is thus needed to remember, that under the conditions of damage by tumour the itch which proceeds for a long time appears at first, only later there appears icterus. During examination the yellowness of sclera, mucus shells and skin are observed. At the same time patients specify urines and discolorations of excrement darkening (“argil”). The increase of temperature of body testifies the development of cholangitis, metastasis of tumors in liver is rarer.

In emaciated patients in right hypochondrium it is sometimes possible to see formation, that moves during breathing, probably it is a gall-bladder. If it is elastic, unpainful and it is accompanied by icterus (*the Courvoisier's symptom*), a patient then suffers from cancer of head of pancreas or distal parts of general bilious ducts.

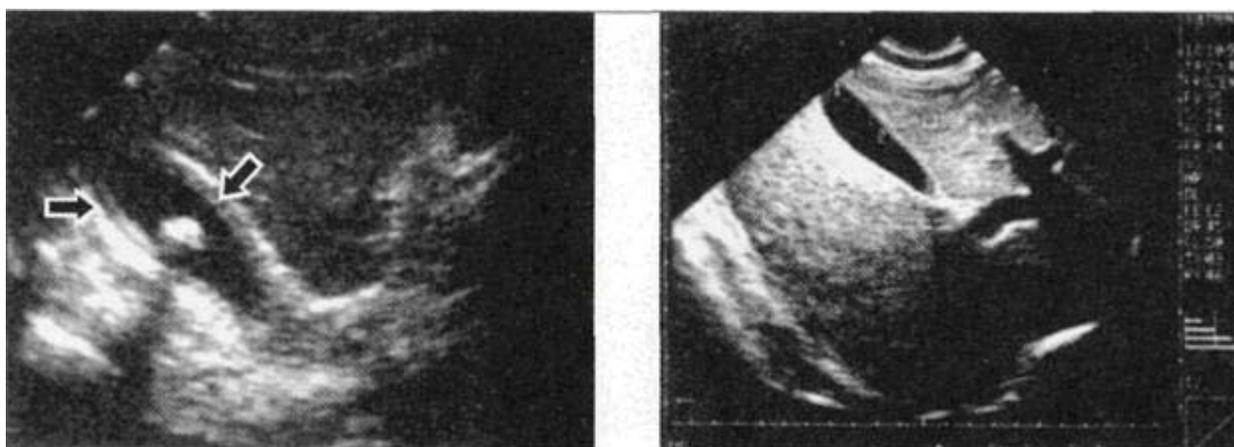
However, it is necessary to warn, that determination of character of icterus only on the basis of clinical signs to a great extent carries only conditional character. It must be connected to the fact that at parenchymatous hepatitis expressed cholestasis often is observed, and at obturation icterus damage of hepatic cells is characteristic. Therefore for establishment of diagnosis of obturative icterus, except for clinical information, special methods of examination are necessary.

Laboratory information. For obturative icterus a cholestatic syndrome with high bilirubinemia mainly due to direct fraction of bilirubin and bilirubinuria is characteristic,

by absence of urobilin in urine and stercobilin in excrement, by high activity of alkaline phosphatase at the insignificantly promoted transaminase activity and negative thymol test.

At growth of hyperbilirubinemia this intercommunication changes in the side of increase of direct bilirubin. In general analysis of blood unsteady changes are which depend on the degree of intoxication or occult bleeding (in cancer patients).

Sonography examination allows to define the sizes of liver, gall-bladder, state of internal and external hepatic ducts, presence and degree of expansion or narrowing, presence or absence of concrement and new formation in hepatic parenchyma.



Sonography examination

Duodenography in the conditions of artificial low blood pressure apply for the exposure of pathology of organs of pancreatoduodenal area.

Retrograde cholangiopancreatography enables to examine stomach, duodenum by endoscope, to conduct biopsy, to extract bile and pancreatic juice for examination, to get the roentgenologic image of ducts: external and internal hepatic ducts and duct of pancreas, and in a number of cases at presence of concrement to conduct endoscopic papillotomy and extraction of them through papillotomic access.



Retrograde cholangiopancreatography

Percutaneous transhepatic cholangioduodenography is used for the exposure of pathology of bilious ways. It to certain extent allows to expose both character and localization of obturation in the area of hepatoduodenal area.

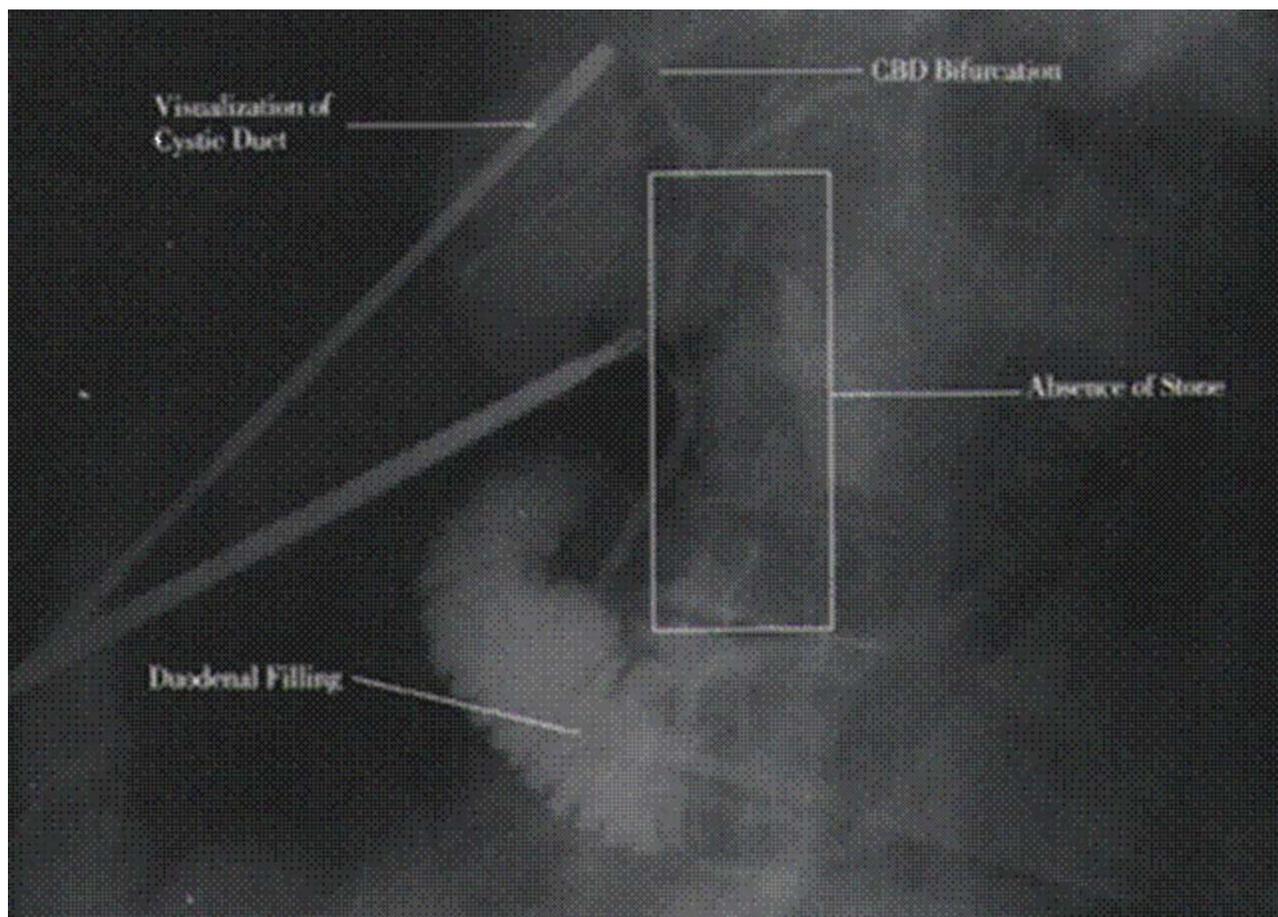
Laparoscopy is a diagnostic remedy that enables to define the sizes of liver, its colouring, character of surface, presence of metastases, size and degree of tension of gall-bladder. Under the control laparoscope it is possible to execute puncture of gall-bladder and conduct cholecystocholangiography and cholecystostomy.

The scanning of liver creates terms in which it is possible to expose the primary and metastatic tumours of liver or other pathology of organ.

Intraoperative cholangiography

Intraoperative cholangiography is contrasting of bilious ducts by introduction iodine preparations (bilignost, cardiost, verigraphine in the concentration 30–33 %) in them through stump of cystic duct of soluble. Cholangiography enables to define the width of ducts, presence or absence of concrement in them, and also cone-shaped narrowing of terminal part of choledochus characteristic of stenosis.





Cholangiomanometry

Cholangiomanometry is a method, that allows to expose the degree of bilious hypertension in ducts by the water manometer of the Valdmann device. Normal pressure is within the limits of 80–120 mm of waters. item (0,78–1,17 kPa), and the higher testifies to bilious hypertension.

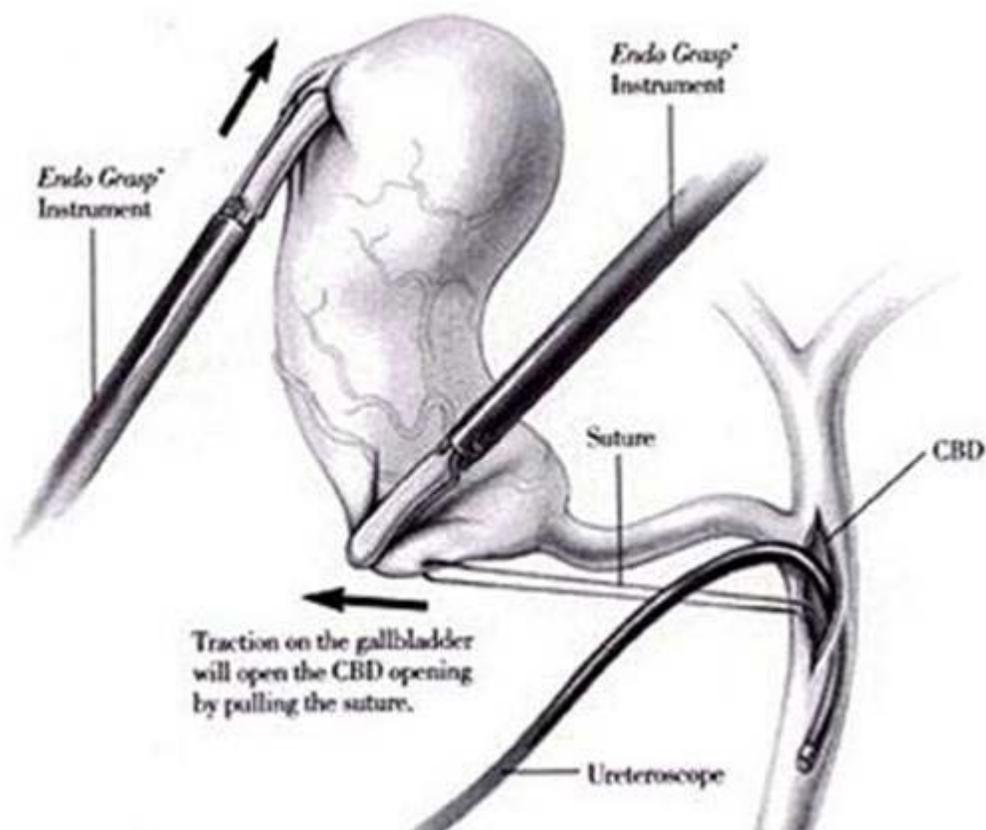
Debitomanometry

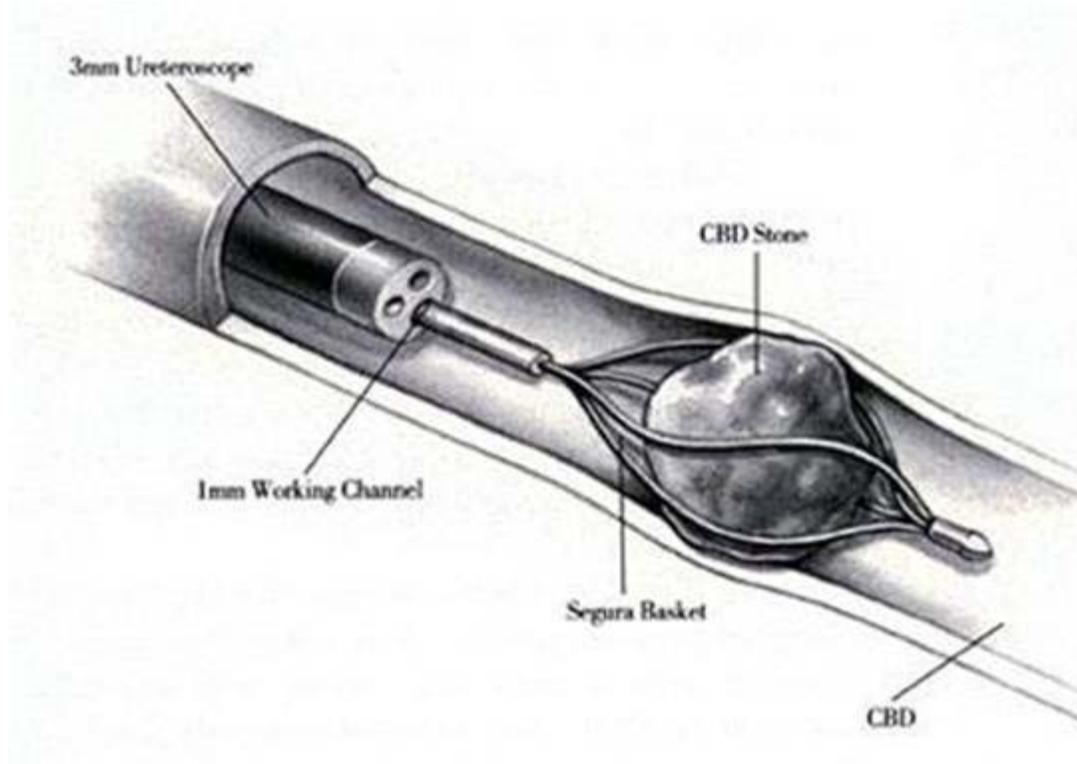
Debitomanometry is a method of determination of amount of liquid at perfusion through the Vater's papilla under permanent pressure for time unit (1 min). In patients with normal passing of bilious ducts the size to the debit of liquid at pressure 150 mmwater.col. (1,47 kPa) makes from 5 to 8 ml/min. In similar terms, at their obturation,

this debit diminishes, and at insufficiency of sphincteric device — is increased. However, much cholangio- and debitomanometry at operations in patients with acute cholecystitis are applied rarely. These methods of examination are used for chronic cholecystitis mainly.

Choledochoscopy

Choledochoscopy is a method of endoscopy examination of bilious ducts by choledochoscope during choledochostomy.



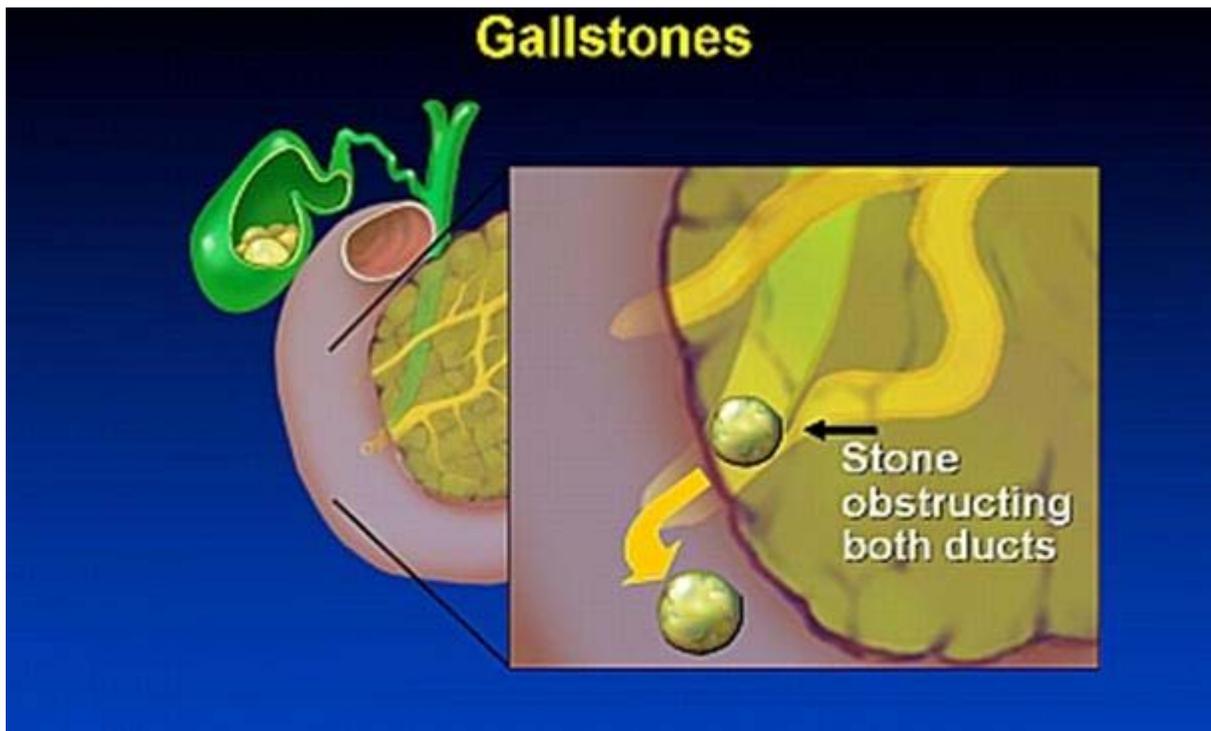


The Choledochoscopy. Retrieving the CBD Stones

Variants of clinical passing and complications

Clinical passing of icterus almost always depends on the reasons of obturation of bilious duct. In patients with tumours an icterus gradually makes progress and results in complete and permanent obturation, together with that, at presence of concrement in bilious ducts intensity of icterus can vary. Its temporal, transitional character takes place at choledocholithiasis, acute cholecystitis or pancreatitis.

On this background in the case of joining of infection cholangitis, abscess formation in liver and sepsis quickly develops. In other case there can be the bleeding (more frequent gastroduodenal) or hepatic-kidney insufficiency.



In some patients internal bilious fistula, which clinically is proof cholangitis, appears as a result of inflammatory and necrosis processes. On the sciagram survey of organs of abdominal cavity in such cases it is possible to see air in the hepatic ducts, the so called “aerocholia”.

Diagnosis program

1. Anamnesis and physical methods of examination.
2. General analysis of blood and urine.
3. Analysis of urine on diastasis.
4. Biochemical blood test (bilirubin, urea, albumin-globulin coefficient, blood on an australian antigen, amylase, alanine aminotransferase, asparaginase, alkaline phosphatase).
5. Coagulogram.
6. Sonography.

7. Endoscopy.
8. Retrograde cholangiopancreatography.
9. Laparoscopy with biopsy.
10. Percutaneous transhepatic cholangioduodenography.
11. Computer tomography.

CLASSIFYING THE BILIARY STONE PATIENT

Asymptomatic Cholelithiasis	Incidental Finding on Sonogram
Acute Cholecystitis	Cholelithiasis on Sonogram, clinical Cholecystitis diagnosis or Positive Pipida Scan
Symptomatic Cholelithiasis	Positive Sonogram, normal Liver Function Tests
Cholelithiasis with Suspected Choledocholithiasis	Abnormal Liver Function Tests (Serum Transaminases elevation or Bilirubin >3.0, gallstone pancreatitis)
Cholelithiasis with Choledocholithiasis	CBD Stone on Sonogram, MR Cholangiography or Jaundice
Cholelithiasis with Resolving Gallstone Pancreatitis	Pancreatitis on Sonogram, CT or MER Cholangiography or clinically, Documented High Serum Amylase and Lipase - WITH - Decreasing Serum Pancreatic Enzymes after initial attack

IDENTIFYING PATIENTS WITH CHOLEDOCHOLITHIASIS

MANAGEMENT PROTOCOLS FOR UNCOMPLICATED BILIARY STONE DISEASES	PROPOSED MANAGEMENT
Asymptomatic Cholelithiasis	No Surgical Intervention
Asymptomatic Cholelithiasis in Diabetic Patients	No Surgical Intervention
Symptomatic Cholelithiasis or Acute Cholecystitis	LapChole
Symptomatic Cholelithiasis with Suspected Choledocholithiasis	LapChole with Insertion of Cystic Duct Cannula with Cholangiography, if Choledocholithiasis, postop ERCP
Symptomatic Cholelithiasis with Choledocholithiasis	LapChole with Insertion of Cystic Duct Cannula with Cholangiography, if Choledocholithiasis, postop ERCP
Cholelithiasis with Resolving Pancreatitis	LapChole with Insertion of Cystic Duct Cannula with Cholangiography, if Choledocholithiasis, postop ERCP
Cholelithiasis with Unresolved Pancreatitis	After acute phase subsides, MRI Cholangiography or ERC, if Choledocholithiasis ERCP followed by LapChole

Asymptomatic Gallbladder Polyps	No Surgical Intervention
Symptomatic Gallbladder Polyps	LapChole
Severe, Gangrenous Cholecystitis with Subhepatic Phlegmon	LapChole, if not safely feasible, Anterior-subtotal LapChole
Post Cholecystectomy (Lap or open) Suspected Choledocholithiasis	MR Cholangiogram or ERC
Post Cholecystectomy (Lap or open) Choledocholithiasis	ERCP, if failure Laparoscopic Common Bile Duct Exploration.

Differential diagnostics

The main task of differential diagnostics of icterus is a determination of surgical or unsurgical nature. In fact the decision of this question enables to take the amount of doubtful diagnostic laparotomy to the minimum.

Taking it into account, it is always needed to remember, that among the diseases which can show up as icterus, viral hepatitis take a considerable place, mostly its cholestatic form, new formation of hepatopancreatoduodenal area and gallstone disease.

Viral hepatitis is one of many clinical forms of viral infection the characteristic feature of which is proof and protracted cholestasis. The disease carries the protracted character. For the pre-icteric period of hepatitis the inherent clinical triad is the itch of skin, fever, arthralgia. Approximately in half of patients it is possible to palpate a spleen

and moderately increased liver. Laboratory the increase of activity of alanine aminotransferase and aspartate aminotransferase and positive thymol test are determined. Also the reaction on bilious pigments, bilious acids and urobilin in urine is positive.

Difficult from the point of view of verification of diagnosis it is needed to count combination of such diseases, as viral hepatitis in patients with cholelithiasis or obturation by tumour in patients who suffer from alcoholism.

Taking into account information of previous methods, only successive application of sonography, endoscopic retrograde pancreatocholangiography or percutaneous transhepatic cholangioduodenography and laparoscopy, allows maximally exactly to set diagnosis. Thus on the first stage it is needed to use the non-invasive methods of diagnostics (sonography), on the second useful are invasive methods of the direct contrasting of bilious ways (retrograde endoscopic cholangiopancreatography, percutaneous transhepatic cholangiopancreatography) which in the case of necessity can transform from diagnostic into medical (endoscopic papillosphincterotomy at stenotic papillitis and choledocholithiasis, dilatation and endoprosthesis replacement of bilious ways at cicatrix stricture and tumours of bilious ducts).

Table of diagnostic tests

Function test	Pre-hepatic Jaundice	Hepatic Jaundice	Post-hepatic Jaundice
Total bilirubin	Normal / Increased	Increased	
Conjugated bilirubin	Normal	Increased	Increased
Unconjugated bilirubin	Normal / Increased	Increased	Normal
Urobilinogen	Normal / Increased	Increased	Decreased / Negative

Urine Color	Normal	Dark (urobilinogen + conjugated bilirubin)	Dark (conjugated bilirubin)
Stool Color	Normal	Normal/Pale	Pale
Alkaline phosphatase levels	Normal	Increased	
Alanine transferase and Aspartate transferase levels		Increased	
Conjugated Bilirubin in Urine	Not Present	Present	
Splenomegaly	Present	Present	Absent

Tactics and choice of treatment method

Final diagnosis, that maximally represents the character of obturative icterus and volume of the most operative treatment, as a rule, is set only during the intraoperative revision. At determination of medical tactics and choice of method of surgical treatment of such icterus it is needed also to objectively estimate weight of general condition of patients. For this purpose it is necessary to take into account the character of icterus, stage of hepatic insufficiency keeping in mind the duration and intensity of cholestasis, presence and character of cholangitis, weight and expression of accompanying pathology, eyelids of patients.

Medical measures in preoperative period must be directed on correction of violations of homeostasis, hemocoagulation (aminocapronic acid, vicasol, 10 % solution of chlorous calcium, one-group fresh-frozen plasma, inhibitor of protease), improvement

of microcirculation in liver (10 % solution of glucose with insulin, reopolyhucine, hepatoprotectors), deoxidation organism (neohemodes, enterosorbent), biliary decompression (percutaneous transhepatic cholangiopancreatocholangiostomy or cholecystostomy), antibacterial therapy at the phenomena of cholangitis taking into account the character of sown microflora and its sensitiveness to the antibiotics and vitamins.

In case of the gallstone disease complicated by choledocholithiasis and mechanical icterus, the volume of surgical interference must include: cholecystectomy, choledocholithotomy and external or internal draining of general bilious duct. At presence of the special apparatus in case of choledocholithiasis complicated by mechanical icterus, two-stage tactics of treatment is the method of choice — endoscopic papillosphincterotomy with subsequent extraction of concrement and their lithotripsy on the first stage and cholecystectomy — on the second. Endoscopic papillosphincterotomy is the method of choice at treatment of remaining (after cholecystectomy) choledocholithiasis.

For declining patients with heavy accompanying pathology combination of extracorporal shock-wave lithotripsy with endoscopic sanitation of hepaticocholedochus is an effective method. For some of them at the high risk of operative treatment and small concrement of general bilious duct (by the diameter of to 10 mm) endoscopic papillotomy can be effective.

At malignant new formations of bile-excreting ways with a obturative icterus, depending on distribution of tumour process, radical or palliative operative treatment are executed.

For patients with the tumours of head of pancreas, large papilla of duodenum and terminal part of choledochus, that is exposed to radical operation, a pancreatoduodenal resection is performed, thus in the case of high icterus on the first stage of treatment bilious decompression of the hepatobiliary system is performed (percutaneous hepaticocholangiostomy, forming of biliary-enteric anastomosis). A pancreatoduodenal resection is executed on the second stage, in 30–35 days after imposition of bile-excreting anastomosis and liquidation of icterus.

Palliative bile-excreting operations at the neglected tumours are mainly directed on liquidation of obstruction of bilious ducts. In such patients, as a rule, roundabout biliary-enteric anastomosis is imposed: cholecystoentero- or hepaticojejunostomy. If through technical difficulties and bad general condition of patients it is impossible to execute this, external draining of bilious ways is applied.

[video](#)

LAPAROSCOPIC MANAGEMENT OF BILIARY STONE DISEASE

The management of biliary stones diseases has dramatically changed with the advent of the Laparoscopic Cholecystectomy. It has now become a true outpatient laparoscopic procedure with negligible morbidity.

In the past few years, our surgical team has designed and revised numerous management protocols for various clinical settings effectively achieving impressive improvements in our surgical performance for the treatment of biliary stone diseases. This chapter will describe these management protocols and our latest technical updates.

The original Laparoscopic Cholecystectomy technique has undergone a vast maturation process over the past decade. Various technical steps has been modified and adapted to improve surgical performance and clinical outcome. As a result, nowadays, most surgeons in the Western World can safely perform a Laparoscopic Cholecystectomy with a minimal conversion rate.

ROUTINE INTRA-OPERATIVE CHOLANGIOGRAPHY

Routine operative cholangiography is recommended by most laparoscopic authors in the United States. However, recent reports demonstrate it does not significantly decrease the rate of common bile duct injury in cases where the anatomy is well-identified. Our recommendation is that routine intraoperative cholangiography should be performed by inexperienced laparoscopic surgeons and in cases where the

anatomy is not well-defined.

IDENTIFYING PATIENTS WITH CHOLEDOCHOLITHIASIS

In order to achieve the level of Maximum Surgical Performance with this procedure, patients at high risk of presenting with Common Bile Duct Stones need to be identified pre-operatively. The simplest methods to initially identify these patients are:

- 1) History and Physical Examination,
- 2) Liver Function Studies,
- 3) Sonographic Findings.

Patients with a recent history of gallstone pancreatitis, jaundice, or presenting with such symptoms are at a high risk of having common bile duct pathology; the same is valid for patients with altered liver function studies. The most accurate studies are the Serum Transaminases (SGOT, SGPT). Elevations of these enzymes over 20% of their normal values are significant. But patients with severe, acute cholecystitis can occasionally generate such elevations. Also, extreme elevations of these two enzymes could represent hepatocytes necrosis as seen in hepatitis. The bilirubin level may also be elevated in certain patients with acute cholecystitis, but elevations above 2.5 or 3.0 mg/dl could identify a patient with choledocholithiasis. Finally, we find the enzymes LDH and GGTP to have no real specific value in this clinical setting.

It is interesting that in spite of our intensive efforts to identify Common Bile Duct pathology preoperatively, missed Common Bile Duct Stones are found in 1.92% of all patients. Of these patients 76% will require additional surgical intervention (ERCP).

ROUTINE INTRA-OPERATIVE CHOLANGIOGRAPHY

This technology is being used with increasing frequency in our surgical service to

identify patients with choledocholithiasis. A GE Magnetic Resonance machine was used for all studies. To date the specificity and accuracy of these studies in our services is 98.2% for common bile duct stones over 1 mm in size.

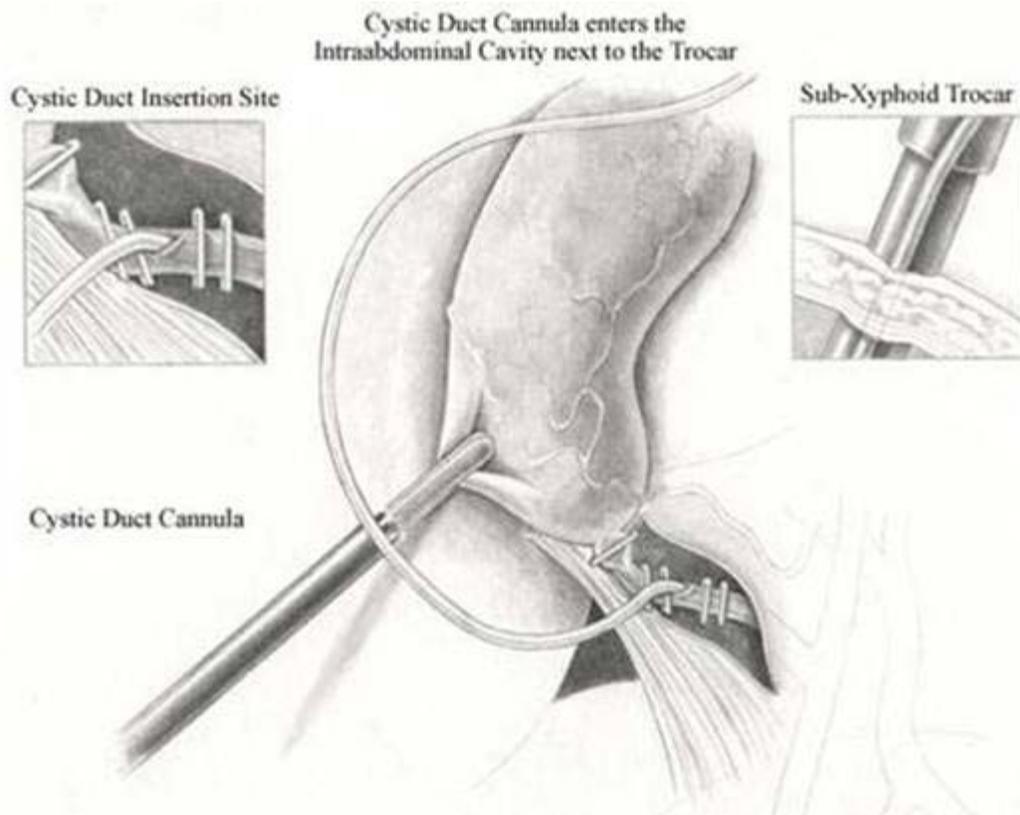
ROUTINE INTRA-OPERATIVE CHOLANGIO-SONOGRAPHY

Intra-operative cholangio-sonography is being used in many medical centers to rule out common bile duct stone. Although this modality was used on numerous occasions, we found it too time consuming to be used on a routine basis.

Methods of external draining

External draining of bilious ducts can be executed by such methods:

- 1) by Pickovskyy – polyethylene catheter which is entered through stump of cystic duct;
- 2) by Kehr — by T-shaped latex drainage;
- 3) by Vyshnevsky — drainage to the gate of liver;
- 4) by Holsted.



Indication to external draining: 1) after diagnostic choledochostomy; 2) after choledocholytotomy; 3) at accompanying cholangitis, pancreatitis.

For internal draining of bilious ducts are applied by transduodenal sphincteroplasty or choledochoduodenostomy. At acute cholecystitis these operations are executed under absolute indications. Such is the structure and tired out concrement of large duodenal papilla, multiple choledocholithiasis, presence of putty in ducts or their expansion.

The best method of renewal of normal outflow of bile at stenosis and jammed concrement of large duodenal papilla is considered its transduodenal dissection. This method of internal draining allows to liquidate the reason of impassability of ducts and store the physiology passage of bile in intestine.

At forming choledochoduodenoanastomosis most distribution acquired methods by Jurash, Flerken, Finsterer and Kirschner's methods. The principle difference of them consists in correlations of direction of cut of choledochus and duodenum (longitudinal, slanting, transversal). The width of anastomosis must be not less than 2,5–3 cm. It is thus

needed to remember, that imposition of choledochoduodenoanastomosis in the conditions of presence of inflammatory process in abdominal cavity always needs to be connected with external draining of choledochus by Pikovskyy (double draining).

[Biliary-enteric anastomosis: Interrupted technique \(Video 2\)](#)

[Biliary-enteric anastomosis: Running technique \(Video 3\)](#)

Portal hypertension syndrome

Portal hypertension refers to abnormally high pressure in the hepatic portal vein. It is defined as a portal pressure of 12 mm Hg or more (compared with the normal 5-10 mm Hg).

See related articles dealing with ascites, cirrhosis, hepatic encephalopathy, hepatorenal syndrome, liver failure and oesophageal varices, listed below under 'Complications'.

Etiology

Causes can be divided into: prehepatic, hepatic and posthepatic. Cirrhosis is a common cause.

Causes of portal hypertension

Prehepatic - blockage of the portal vein before the liver

Congenital atresia or stenosis.

Portal-vein thrombosis (idiopathic, umbilical and portal sepsis, malignancy, hypercoagulable states, pancreatitis).

Splenic vein thrombosis.

Extrinsic compression, eg tumours.

Hepatic

Cirrhosis.

Chronic hepatitis.

Schistosomiasis.

Myeloproliferative diseases.

Idiopathic portal hypertension.

Granulomata, eg sarcoid.

Nodular (nodular regenerative hyperplasia, partial nodular transformation).

Toxins (arsenic, vinyl chloride).

Fibropolycystic disease (including congenital hepatic fibrosis).

Posthepatic - blockage of hepatic veins or venules

Budd-Chiari syndrome (hepatic vein obstruction).

Constrictive pericarditis.

Right heart failure.

Veno-occlusive disease of the smaller hepatic veins/venules (due to ingestion of pyrrolizidine alkaloids; antileukaemic drugs, radiation).

Sclerosing hyaline necrosis.

Other causes

Increased hepatic blood flow:

Increased splenic blood flow, eg massive splenomegaly.

Hepatoportal arteriovenous fistula.

Idiopathic (a diagnosis of exclusion).

Left-sided (sinistral) portal hypertension

Rare. It is confined to the left side of the portal system.

It may present as bleeding from gastric varices.

Usually it is due to pathology involving the splenic vein or the pancreas.

Pathophysiology

Portal hypertension develops due to:

Increased vascular resistance in the portal venous system - from various mechanical causes (above), and also as an active process in which liver damage activates stellate cells and myofibroblasts, contributing to the abnormal blood flow patterns.

Increased blood flow in the portal veins - from splanchnic arteriolar vasodilatation, caused by an excessive release of endogenous vasodilators.

The raised portal pressure opens up venous collaterals, connecting the portal and systemic venous systems. These occur in various sites:

Gastro-oesophageal junction - producing varices which are superficial and easily bleed.

Anterior abdominal wall:

Via the umbilical vein - visible as caput medusae radiating from the umbilicus.

May also occur where adhesions exist between abdominal viscera and the parietal peritoneum, or at sites of stomas or previous surgery.

Anorectal junction - rarely cause bleeding.

Veins from retroperitoneal viscera - communicate with systemic veins on the posterior abdominal wall.

Other patterns of blood flow:

If individual tributaries of the portal vein are thrombosed, this causes local venous hypertension. With splenic vein block, oesophageal and gastric varices may result.

In Budd-Chiari syndrome (hepatic vein occlusion), collaterals open up within the liver; blood tends to be diverted through the caudate lobe whose short hepatic veins drain directly into the inferior vena cava.

Portosystemic venous anastomoses can cause encephalopathy, possibly due to

various 'toxins' bypassing the liver's 'detoxification' process.

Circulatory disturbances:

Portal hypertension and cirrhosis produce a hyperdynamic circulation, with splanchnic vasodilatation, increased cardiac output, arterial hypotension, and hypervolaemia.

There is salt and water retention, ascites and hyponatraemia.

Presentation

History

For causes of liver disease:

History of jaundice.

Alcohol consumption.

Blood transfusion, especially abroad; lifestyles that predispose to hepatitis B or hepatitis C.

Family history, eg Wilson's disease or hereditary haemochromatosis.

For complications of portal hypertension:

Haematemesis or melaena - suggest bleeding varices.

Lethargy, irritability and changes in sleep pattern - suggest encephalopathy.

Increased abdominal girth, weight gain - suggest ascites.

Abdominal pain and fever - suggest spontaneous bacterial peritonitis.

Examination

Signs of portal hypertension:

Dilated veins in the anterior abdominal wall and caput medusae (tortuous collaterals around the umbilicus). A venous hum, loudest during inspiration, is sometimes heard over large upper abdominal collaterals.

Splenomegaly.

Ascites.

Signs of liver disease:

Jaundice, spider naevi, palmar erythema.

Confusion, liver flap and fetor hepaticus are signs of encephalopathy.

Signs of hyperdynamic circulation: bounding pulse, low blood pressure, warm peripheries.

Enlarged or small liver.

Gynaecomastia and testicular atrophy.

Investigations

Blood tests:

LFTs, U&Es, glucose, FBC, clotting screen.

Investigations for liver disease if the cause not known, e.g ferritin (for haemochromatosis), hepatitis serology, autoantibodies, alpha-1-antitrypsin (for alpha-1-antitrypsin deficiency), ceruloplasmin (for Wilson's disease).

Scans:

Abdominal ultrasound - for liver and spleen size, ascites, portal blood flow and thrombosis of the portal or splenic veins.

Doppler ultrasound - can show direction of flow in blood vessels.

CT scan, especially spiral CT, may show portal vasculature - can be useful if ultrasound was inconclusive.

MRI scan - gives similar information to CT.

Elasticity measurement (FibroScan®) - a new technique based on the velocity of an elastic wave via an intercostally placed transmitter. Results correlate with liver stiffness and so with fibrosis.

Endoscopy - for oesophageal varices - essential for those with suspected portal hypertension. Varices indicate portal hypertension, but their absence does not exclude it.

Portal hypertension measurement:

Portal pressure is indirectly measured in clinical practice by the hepatic venous pressure gradient (HVPG).

Normal HVPG values are <5 mm Hg. HVPG >10 mm Hg predicts the development of oesophageal varices.

However, HVPG is moderately invasive and its clinical role is uncertain.

Liver biopsy - if indicated, may help diagnose the underlying cause.

Vascular imaging:

The site of the portal venous block can be demonstrated by examining the venous phase of a coeliac or superior mesenteric arteriogram, by splenic portography following injection of dye into the splenic pulp, or by retrograde portography via a hepatic vein.

Hepatic venography is helpful when hepatic vein block or idiopathic portal hypertension is suspected.

Management

The portal hypertension itself is difficult to treat effectively, except by:

Treating the underlying cause, where possible.

Liver transplantation, if indicated and feasible.

Portal venous pressure can be reduced by:

Betablockers \pm nitrates.

Shunt procedures - create an anastomosis between the portal and hepatic veins (below).

Complications

Complications of portal hypertension \pm cirrhosis are:

Bleeding from oesophageal or gastric varices - the most common complication of portal hypertension.

Ascites and its complications:

Spontaneous bacterial peritonitis.

Hepatorenal syndrome (a complication of cirrhosis with ascites).

Hepatic hydrothorax.

Pulmonary complications:

Portopulmonary hypertension (pulmonary arterial hypertension complicating portal hypertension in patients with liver disease).

Hepatopulmonary syndrome (a triad of hepatic dysfunction, hypoxemia and extreme vasodilation in the form of intrapulmonary vascular dilations).

Liver failure.

Hepatic encephalopathy.

Cirrhotic cardiomyopathy.

For specific management, see separate articles Ascites, Cirrhosis, Hepatic Encephalopathy, Hepatorenal Syndrome, Liver Failure and Oesophageal Varices.

The following gives an overview of treatments used on the portal vascular system:

Drug treatments

Betablockers:

Nonselective betablockers reduce portal pressure in many patients.

They reduce rates of bleeding and rebleeding in patients with oesophageal varices.

They may also protect against spontaneous bacterial peritonitis (perhaps through increasing intestinal transit).

Carvedilol (a nonselective betablocker with anti-alpha1-adrenergic effects) showed promising results in a recent study, and may have a role in preventing variceal bleeds.

Nitrates:

Added to betablocker therapy, they contribute to reducing portal pressure and

may reduce rates of variceal rebleeding.

Vasoactive drugs:

Terlipressin and octreotide are used to assist the control of acute variceal bleeding.

Endoscopic procedures

Endoscopy - to detect and monitor oesophageal varices.

Endoscopic vein ligation - to prevent bleeding of oesophageal varices.

For gastric varices with acute bleeding, endoscopic variceal obturation with tissue adhesives (eg cyanoacrylate) is effective - but there are recognised complications (mucosal ulceration and thromboembolism).

Transjugular intrahepatic portosystemic shunt (TIPS)

This is a radiological procedure, connecting the portal and hepatic veins using a stent. The purpose of a TIPS is to decompress the portal venous system, to prevent rebleeding from varices or to reduce the formation of ascites.

However, there are potential complications - hepatic encephalopathy and deteriorating liver function. The stent may stenose; it requires follow-up and may require repeat procedures. There are various contra-indications, detailed in recent guidelines.

TIPS is an established treatment option for:

Ascites - for patients requiring repeated and frequent paracentesis.

Oesophageal variceal bleeding refractory to medical treatment (acute bleeding or secondary prevention).

Bleeding from non-oesophageal varices, eg gastric varices.

TIPS may also have a role in treating:

Hepatorenal syndrome.

Hepatic hydrothorax.

Hepatopulmonary syndrome.

Budd-Chiari syndrome.

Surgical procedures

Surgical portosystemic shunts:

These require major surgery and an experienced surgeon. They are less likely to stenose than TIPS and can be used where TIPS is not feasible.

Shunts may be total, partial or selective.

Devascularisation procedures:

Include gastro-oesophageal devascularisation, splenectomy and oesophageal transection.

Generally used where other therapies are unsuitable.

Management of rectal varices

These are common in patients with portal hypertension but don't usually bleed. They are located at the anorectal junction.

If they bleed, suggested treatment is similar to that used for upper gastrointestinal varices - using drugs to reduce portal pressure, endoscopic banding and TIPS if bleeding persists.

Prognosis

This depends on the prognosis of the underlying disease, and on the outcome of any complications such as variceal bleeding.

Other scoring systems have been developed, including:

Model for End Stage Liver Disease (MELD).

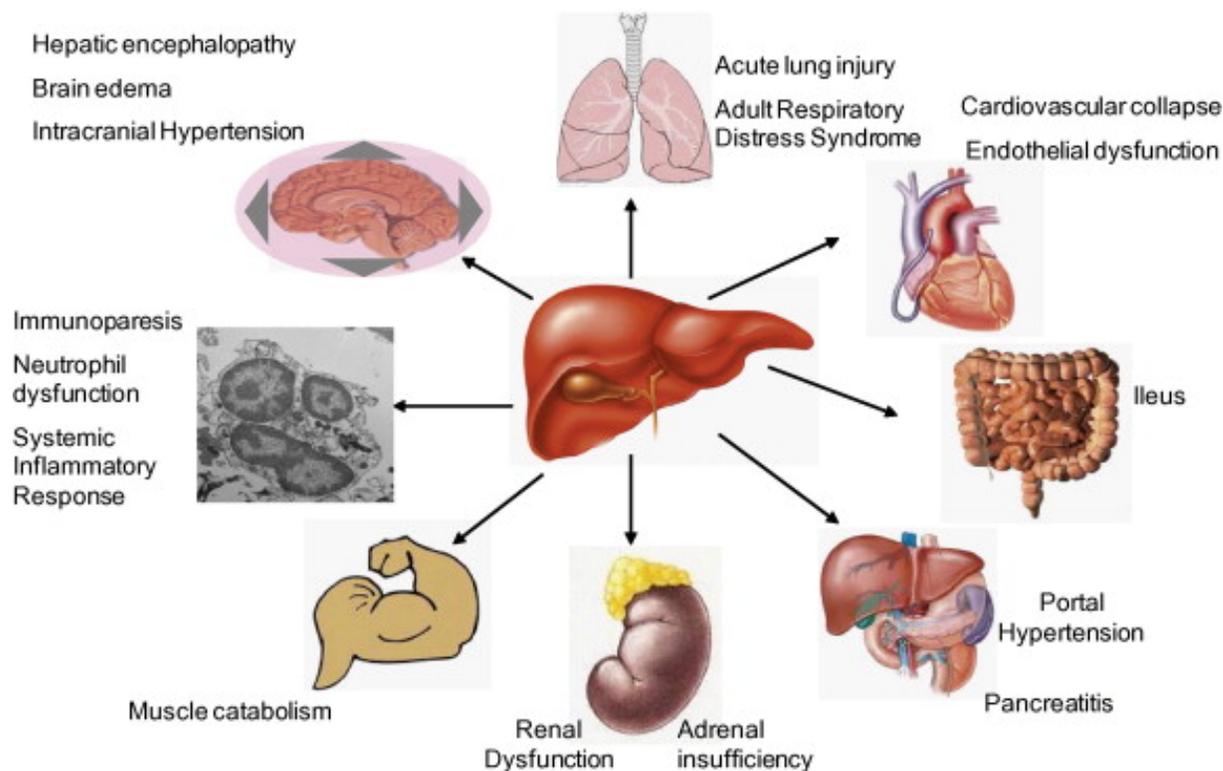
Additions to MELD such as MELD-Na (incorporates serum sodium) and MELD-ICG (incorporates indocyanine green clearance).

Variceal haemorrhage, especially from oesophageal varices, is the most common complication associated with portal hypertension. Almost 90% of patients with cirrhosis

develop varices, but only 30% of varices bleed. The first episode of variceal haemorrhage is estimated to carry a mortality rate of 30-50%.

ACUTE HEPATIC FAILURE

Systemic Manifestations of Acute Liver Failure



Acute liver failure (ALF) is a rare condition in which rapid deterioration of liver function results in altered mentation and coagulopathy in previously normal individuals.

U.S. estimates are placed at approximately 2,000 cases per year.⁴ The most prominent causes include drug-induced liver injury, viral hepatitis, autoimmune liver disease and shock or hypoperfusion; many cases (20%) have no discernible cause. Acute liver failure often affects young persons and carries a high morbidity and mortality.

Prior to transplantation, most series suggested less than 15% survival. Currently, overall short-term survival with transplantation is greater than 65%. Because of its rarity, ALF has been difficult to study in depth and very few controlled therapy trials have been

performed. As a result, standards of intensive care for this condition have not been established.

The most widely accepted definition of ALF includes evidence of coagulation abnormality, usually an INR 1.5, and any degree of mental alteration (encephalopathy) in a patient without preexisting cirrhosis and with an illness of 26 weeks duration. Patients with Wilson disease, vertically-acquired HBV, or autoimmune hepatitis may be included in spite of the possibility of cirrhosis if their disease has only been recognized for 26 weeks. A number of other terms have been used including fulminant hepatic failure and fulminant hepatitis or necrosis. Acute liver failure is a better overall term that should encompass all durations up to 26 weeks. Terms used signifying length of illness such as hyperacute (7 days), acute (7-21 days) and subacute (21 days and 26 weeks) are not particularly helpful since they do not have prognostic significance distinct from the cause of the illness. For example, hyperacute cases may have a better prognosis but this is because most are due to acetaminophen toxicity.

All patients with clinical or laboratory evidence of moderate to severe acute hepatitis should have immediate measurement of prothrombin time and careful evaluation for subtle alterations in mentation. If the prothrombin time is prolonged by 4-6 seconds or more (INR 1.5) and there is any evidence of altered sensorium, the diagnosis of ALF is established and hospital admission is mandatory. Since the condition may progress rapidly, with changes in consciousness occurring hour-by-hour, early transfer to the intensive care unit (ICU) is preferred once the diagnosis of ALF is made. History taking should include careful review of possible exposures to viral infection and drugs or other toxins. If severe encephalopathy is present, the history may be provided entirely by the family or may be unavailable. In this setting, limited information is available, particularly regarding possible toxin/drug ingestions. Physical examination must include careful assessment and documentation of mental status and a search for stigmata of chronic liver disease. Jaundice is often but not always seen at presentation.

Right upper quadrant tenderness is variably present. Inability to palpate the liver or even to percuss a significant area of dullness over the liver can be indicative of decreased liver volume due to massive hepatocyte loss.

An enlarged liver may be seen early in viral hepatitis or with malignant infiltration, congestive heart failure, or acute Budd-Chiari syndrome. History or signs of cirrhosis should be absent as such features suggest underlying chronic liver disease, which may have different management implications. Furthermore, the prognostic criteria mentioned below are not applicable to patients with acute-on-chronic liver disease.

Initial laboratory examination must be extensive in order to evaluate both the etiology and severity of ALF. In addition to coagulation parameters, early testing should include routine chemistries (especially glucose as hypoglycemia may be present and require correction), arterial blood gas measurements, complete blood counts, blood typing, acetaminophen level and screens for other drugs and toxins, viral hepatitis serologies (most prominently A and B), tests for Wilson disease, autoantibodies (anti-nuclear and anti-smooth muscle antibodies) and a pregnancy test in females. Plasma ammonia, preferably arterial,^{7,8} may also be helpful. A liver biopsy, most often done via the transjugular route because of coagulopathy, may be indicated when certain conditions such as autoimmune hepatitis, metastatic liver disease, lymphoma, or herpes simplex hepatitis are suspected. As the evaluation continues, several important decisions must be made: whether to admit the patient to an ICU, whether to transfer the patient to a transplant facility, and (if already at a transplant center) whether and when to place the

patient on the list for transplantation. For patients in a non-transplant center, the possibility of rapid progression of ALF makes early consultation with a transplant facility critical. Specific prognostic indicators may point toward the need for transplantation. For patients with acetaminophen-related ALF in particular, an arterial pH of 7.3 should prompt immediate consideration for transfer to a transplant center and placement on a transplant list.

Patients with altered mentation should generally be admitted to an ICU. Planning for transfer to a transplant center should begin in patients with grade I or II encephalopathy because they may worsen rapidly.

Early transfer is important as the risks involved with patient transport may increase or even preclude transfer once stage III or IV encephalopathy develops. Evaluation for transplantation should begin as early as possible. In these critically ill

patients with potential for rapid deterioration it is necessary to make treatment plans promptly. Social and financial considerations are unavoidably tied to the overall clinical assessment where transplantation is contemplated. It is important to inform the patient's family or other next of kin of the potentially poor prognosis and to include them in the decision-making process.

Table. Initial Laboratory Analysis

Prothrombin time/INR
Chemistries
sodium, potassium, chloride, bicarbonate, calcium, magnesium, phosphate
glucose
AST, ALT, alkaline phosphatase, GGT, total bilirubin, albumin
creatinine, blood urea nitrogen
Arterial blood gas
Arterial lactate
Complete blood count
Blood type and screen
Acetaminophen level
Toxicology screen
Viral hepatitis serologies
anti-HAV IgM, HBSAg, anti-HBc IgM, anti-HEV§, anti-HCV*
Ceruloplasmin Level#
Pregnancy test (females)
Ammonia (arterial if possible)
Autoimmune markers

ANA, ASMA, Immunoglobulin levels
HIV status‡
Amylase and lipase

**Done to recognize potential underlying infection.*

DETERMINING ETIOLOGIES AND SPECIFIC THERAPIES

Etiology of ALF provides one of the best indicators of prognosis, and also dictates specific management options.

LIVER BIOPSY

The characteristic finding on liver biopsy is a heavy deposit of hemosiderin granules in hepatocytes and bile duct cells. Fibrosis may range from minimal to well-established cirrhosis. At times, hereditary hemochromatosis is difficult to distinguish from cirrhosis with secondary iron overload. A preponderance of parenchymal iron relative to the amount of scar tissue and the presence of iron in the bile duct cells characterize hereditary hemochromatosis. Iron overload secondary to underlying cirrhosis is usually associated with advanced cirrhosis, relatively less stainable iron, and absence of iron in the bile ducts. When excess hepatic iron derives from an exogenous source, such as a series of massive transfusions for chronic hemolytic anemia, iron is prominent in the Kupffer cells. When the morphologic features of the liver biopsy do not clearly distinguish between hereditary hemochromatosis and secondary iron overload, quantitative analysis of the hepatic iron content may prove helpful. Patients with hemochromatosis typically have quantitative hepatic iron values ranging from 200 to 800 $\mu\text{mol/g}$ dry weight (normal, $< 35 \mu\text{mol/g}$), whereas patients with alcoholic siderosis have hepatic iron content ranging from 40 to 100 $\mu\text{mol/g}$. Another useful diagnostic test for hemochromatosis is calculation of the hepatic iron index; this value is usually

greater than 2 in homozygotes and less than 2 in patients with alcoholic siderosis. Finally, tissue obtained from the heart, pancreas, or skin of patients with hemochromatosis shows heavy infiltration of stainable iron.

ACETAMINOPHEN HEPATOTOXICITY

Acetaminophen hepatotoxicity is suggested by historic evidence for excessive ingestion either as an intended suicidal overdose or the inadvertent use of supra-therapeutic quantities of pain medications. Acetaminophen is a dose-related toxin; most ingestions leading to ALF exceed 10 gm/day. However, severe liver injury can occur rarely when doses as low as 3-4 gm/day are taken. Very high aminotransferases may be seen; serum levels exceeding 3,500 IU/L are highly correlated with acetaminophen poisoning and should prompt consideration of this etiology even when historic evidence is lacking. Because acetaminophen is the leading cause of ALF (at least in the

United States and Europe) and there is an available antidote, acetaminophen levels should be drawn in all patients presenting with ALF. Low or absent acetaminophen levels do not rule out acetaminophen poisoning since the time of ingestion may be remote or unknown, especially when overdose may have been unintentional and/or occurred over several days. If acetaminophen ingestion is known or suspected to have occurred within a few hours of presentation, activated charcoal may be useful for gastrointestinal decontamination. While it is most effective if given within one hour of ingestion, it may be of benefit as long as 3 to 4 hours after ingestion. Administration of activated charcoal (standard dose 1g/kg orally, in a slurry) just prior to administration of N-acetylcysteine does not reduce the effect of N-acetylcysteine. N-acetylcysteine (NAC), the antidote for acetaminophen poisoning, has been shown to be effective and safe for this purpose in numerous controlled trials. The standard acetaminophen toxicity nomogram¹⁹ may aid in determining the likelihood of serious liver damage, but cannot be used to exclude possible toxicity due to multiple doses over time, or altered metabolism in the alcoholic or fasting patient. Given these considerations, administration of NAC is recommended in any case of ALF in which acetaminophen overdose is a suspected or possible

cause. NAC should be given as early as possible, but may still be of value 48 hours or more after ingestion. 21 NAC may be given orally (140 mg/kg by mouth or nasogastric tube diluted to 5% solution, followed by 70 mg/kg by mouth q 4 h 17 doses) and has few side effects (occasional nausea, vomiting, rare urticaria or bronchospasm). In patients with ALF oral administration may often be precluded (for instance, by active gastrointestinal bleeding or worsening mental status), and NAC may be administered intravenously (loading dose is 150 mg/kg in 5% dextrose over 15 minutes; maintenance dose is 50 mg/kg given over 4 hours followed by 100 mg/kg administered over 16 hours). Allergic reactions may be successfully treated with discontinuation, antihistamines and epinephrine for bronchospasm.

WILSON DISEASE

Wilson disease is an uncommon cause of ALF (2%-3% of cases in the US ALFSG). Early identification is critical because the fulminant presentation of Wilson disease is considered to be uniformly fatal without transplantation.

The disease typically occurs in young patients, accompanied by the abrupt onset of hemolytic anemia with serum bilirubin levels 20 mg/dL. Due to the presence of hemolysis, the indirect-reacting bilirubin is often markedly elevated along with the total bilirubin. Kayser-Fleischer rings are present in about 50% of patients presenting with ALF due to Wilson disease.⁴⁰ Serum ceruloplasmin is typically low, but may be normal in up to 15% of cases and is often reduced in other forms of ALF; high serum and urinary copper levels as well as hepatic copper measurement may confirm the diagnosis. Very low serum alkaline phosphatase or uric acid levels are hints to suggest Wilson disease in the absence of other indicators. A high bilirubin (mg/dL) to alkaline phosphatase (IU/L) ratio (2.0) is a reliable albeit indirect indicator of Wilson disease in this setting.^{40,41} Renal function is often impaired as the released copper can cause renal tubular damage. Treatment to acutely lower serum copper and to limit further hemolysis should include albumin dialysis, continuous hemofiltration, plasmapheresis or plasma exchange. Initiation of treatment with penicillamine is not recommended in ALF as there

is a risk of hypersensitivity to this agent; acute lowering of the copper is more effectively accomplished using direct plasma copper reduction techniques, especially when renal function is impaired.⁴⁰ Although such copper lowering measures should be considered, recovery is infrequent without transplantation. Wilson disease is one of the special circumstances in which patients may already have evidence of cirrhosis and still be considered to have a diagnosis of ALF when rapid deterioration occurs. Please refer to the AASLD Practice Guideline on Wilson Disease for more detailed information regarding the diagnosis and management of patients with this condition.

BUDD-CHIARI SYNDROME

The Budd-Chiari syndrome (acute hepatic vein thrombosis) can also present as ALF. Abdominal pain, ascites and striking hepatomegaly are often present. The diagnosis should be confirmed with hepatic imaging studies (computed tomography, doppler ultrasonography, venography, magnetic resonance venography). In the presence of significant liver failure, transplantation may be required as opposed to venous decompression. As malignancy-associated hypercoagulability is one of the causes of Budd-Chiari syndrome, it is important to rule out underlying cancer prior to transplantation of these patients.

THERAPY: GENERAL CONSIDERATIONS

While patients with ALF represent a heterogeneous group, they have consistent clinical features, and share the common disease process of acute hepatocyte loss and its sequelae. Despite decades of research, however, no agent or therapy that is beneficial to all patients with ALF has been found. Systemic corticosteroids are ineffective in this condition.

Because most patients with ALF tend to develop some degree of circulatory dysfunction, agents that may improve hemodynamics have been of particular interest.

While prostacyclin and other prostaglandins have appeared promising in some reports, others have not supported their efficacy in ALF. NAC may improve systemic circulation parameters in patients with ALF, but this was not observed in all studies. NAC has been shown to improve liver blood flow and function in patients with septic shock. Use of NAC in all forms of ALF cannot be justified based on current evidence. A large, multi-center, randomized, double-blind controlled trial of intravenous NAC versus placebo for non-acetaminophen ALF is currently under way. Because there is no proven therapy for ALF in general, management consists of intensive care support once treatments for specific etiologies have been initiated. While some patients with evidence of acute liver injury but without significant coagulopathy or encephalopathy may be monitored on a medicine ward, any patient with altered mental status warrants admission to an ICU as the condition may deteriorate quickly. Careful attention must be paid to fluid management, hemodynamics and metabolic parameters as well as surveillance for and treatment of infection.

Maintenance of nutrition and prompt recognition and resuscitation of gastrointestinal bleeding are crucial as well. Coagulation parameters, complete blood counts, metabolic panels (including glucose) and arterial blood gas should be checked frequently. Serum aminotransferases and bilirubin are generally measured daily to follow the course of the condition, however changes in aminotransferase levels correlate poorly with prognosis.

Specific Issues. See Table.

Table. Intensive Care of Acute Liver Failure
<i>Cerebral Edema/Intracranial Hypertension</i>
Grade I/II Encephalopathy
Consider transfer to liver transplant facility and listing for transplantation
Brain CT: rule out other causes of decreased mental status; little utility to
identify cerebral edema
Avoid stimulation, avoid sedation if possible

Antibiotics: surveillance and treatment of infection required; prophylaxis possibly helpful
Lactulose: possibly helpful
Grade III/IV Encephalopathy
Continue management strategies listed above
Intubate trachea (may require sedation)
Elevate head of bed
Consider placement of ICP monitoring device
Immediate treatment of seizures required; prophylaxis of unclear value
Mannitol: use for severe elevation of ICP or first clinical signs of herniation
Hyperventilation: effects short-lived; may use for impending herniation
<i>Infection</i>
Surveillance for and prompt antimicrobial treatment of infection required
Antibiotic prophylaxis possibly helpful but not proven
<i>Coagulopathy</i>
Vitamin K: give at least one dose
FFP: give only for invasive procedures or active bleeding
Platelets: give for platelet counts $< 10,000/mm^3$ or invasive procedures
Recombinant activated factor VII: possibly effective for invasive procedures
Prophylaxis for stress ulceration: give H2 blocker or PPI
<i>Hemodynamics/Renal Failure</i>
Pulmonary artery catheterization
Volume replacement

Pressor support (dopamine, epinephrine, norepinephrine) as needed to
maintain adequate mean arterial pressure
Avoid nephrotoxic agents
Continuous modes of hemodialysis if needed
NAC, prostacyclin: effectiveness unknown
Vasopressin: not helpful in ALF; potentially harmful.
<i>Metabolic Concerns</i>
Follow closely: glucose, potassium, magnesium, phosphate
Consider nutrition: enteral feedings if possible or total parenteral nutrition

CENTRAL NERVOUS SYSTEM

Cerebral edema and intracranial hypertension (ICH) have long been recognized as the most serious complications of ALF. Uncal herniation may result and is uniformly fatal. Cerebral edema may also contribute to ischemic and hypoxic brain injury, which may result in long-term neurological deficits in survivors. The pathogenic mechanisms leading to the development of cerebral edema and ICH in ALF are not entirely understood. It is likely that multiple factors are involved, including osmotic disturbances in the brain and heightened cerebral blood flow due to loss of cerebrovascular autoregulation. Inflammation and/or infection, as well as factors yet unidentified may also contribute to the phenomenon. Several measures have been proposed and used with varying success to tackle the problem of cerebral edema and the associated ICH in patients with ALF. Some interventions are supported by more evidence than others; no uniform protocol has been established.

Prevention/Management of Elevated Intracranial Pressure (ICP). The occurrence of cerebral edema and ICH in ALF is related to severity of encephalopathy (Table). Cerebral edema is seldom observed in patients with

grade I-II encephalopathy. The risk of edema increases to 25% to 35% with progression to grade III, and 65% to 75% or more in patients reaching grade IV coma. A stepwise approach to management is therefore advised.

Grades I-II. Depending on the overall clinical picture, patients with only grade I encephalopathy may sometimes be safely managed on a medicine ward with skilled nursing in a quiet environment to minimize agitation, although management in an ICU is preferable. Frequent mental status checks should be performed with transfer to an ICU if level of consciousness declines. With progression to grade II encephalopathy, an ICU setting is indicated. Head imaging with computerized tomography (CT) is used to exclude other causes of decline in mental status such as intracranial hemorrhage. Sedation is to be avoided if possible; unmanageable agitation may be treated with short-acting benzodiazepines in small doses.

GRADES III-IV.

As patients progress to grade III or IV encephalopathy it is advisable to intubate the trachea for airway protection. Choice of sedation in this instance will vary according to clinician preference: propofol is often used because it may reduce cerebral blood flow; however, its effectiveness in this regard has not been shown in controlled studies. Small doses of propofol may be adequate, given its long half-life in patients with hepatic failure.

Patients in advanced stages of encephalopathy require close follow-up. Monitoring and management of hemodynamic and renal parameters as well as glucose, electrolytes and acid/base status becomes critical, and frequent neurological evaluation for signs of elevated intracranial pressure should be conducted. Patients should be positioned with head elevated at 30 degrees.⁸⁰ Efforts should be made to avoid patient stimulation. Maneuvers that cause straining or Valsalva-like movements in particular may increase ICP; it may be advisable to use endotracheal lidocaine prior to endotracheal suctioning.

Table 5. Grades of Encephalopathy
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CLASSIFICATION according to

Child - Turcotte and Pugh

<i>Parameter</i>	Class A Compensation (5-6 points)	Class B Subcompensation (7-9 points)	Class C Decompensation (10 and >points)
Bilirubin, mcmol/l	<35	35-55	>55
Albumine, g/l	>35	30-35	<30
Prothrombin time	60-80%	40-59%	<40%
Ascites	absent	treatable	untreatable
Encephalopathy	absent	1-2 st.	3-4 st.
<i>Points:</i>	<i>1</i>	<i>2</i>	<i>3</i>

Seizures.

Seizures, which may be seen as a manifestation of the process that leads to hepatic coma and ICH, should be controlled with phenytoin. Use of any sedative is discouraged in light of its effects on the evaluation of mental status. Only minimal doses of benzodiazepines should be used given their delayed clearance by the failing liver. Seizure activity may acutely elevate ICP⁸¹ and may also cause cerebral hypoxia and thus contribute to cerebraledema. Some experts have advocated prophylactic use of phenytoin, especially as seizure activity may be inapparent.

A small randomized controlled trial of prophylactic phenytoin in ALF showed no difference in overall survival, but a striking diminution in cerebral edema at autopsy in the treated group. A recent clinical trial did not show beneficial effects on the prevention of seizures, brain edema or survival. Further studies may clarify the value of this treatment, but it cannot be recommended as a prophylactic measure at this time.

COMPLICATION

Infection.

All patients with ALF are at risk for acquisition of bacterial or fungal infection or sepsis, which may preclude transplantation or complicate the post-operative course. Prophylactic antimicrobial therapy reduces the incidence of infection in certain groups of patients with ALF, but no actual survival benefit has been shown, making it difficult to recommend antibiotic prophylaxis uniformly. Although often given, poorly absorbable antibiotics for selective bowel decontamination have not been shown to impact survival either. Deterioration of mental status in hospital, particularly in patients with acetaminophen toxicity, may represent the onset of infection. If antibiotics are not given prophylactically, surveillance for infection (including chest radiography and periodic cultures of sputum, urine and blood for fungal and bacterial organisms) should be undertaken, while maintaining a low threshold for starting appropriate anti-bacterial or anti-fungal therapy. There are no controlled trials available to confirm whether the use of prophylactic antimicrobials decreases the likelihood of progression of encephalopathy and/or development of cerebral edema in ALF. Recent studies have suggested an association between infection and/or the systemic inflammatory response syndrome (SIRS) and progression to deeper stages of encephalopathy. Given that prophylactic antibiotics have been shown to reduce the risk of infection, that later stages of encephalopathy are associated with increased incidence of cerebral edema, and that fever may worsen ICH, it is possible that antibiotic and antifungal prophylaxis may decrease the risk of cerebral edema and ICH. This hypothesis is yet to be proven, however.

SPONTANEOUS BACTERIAL PERITONITIS

Spontaneous bacterial peritonitis (SBP) develops in 10% to 25% of cirrhotic patients followed prospectively for at least a year. The cirrhosis is usually advanced and

active, as manifested by hepatic encephalopathy, esophageal varices, and jaundice. The incidence of SBP is substantially higher in patients with ascitic fluid protein levels below 1.0 g/dl and serum bilirubin levels above 2.5 mg/dl. These findings could explain the increased risk of ascitic fluid infection, because the antibacterial activity of the ascitic fluid, as measured by opsonic activity, is proportional to the level of ascitic fluid protein. The exact pathogenesis is unknown. Presumably, hematogenous seeding of the ascitic fluid, which functions as an ideal bacterial culture medium, serves as a major route of infection. Cirrhosis undoubtedly facilitates the process by allowing enteric organisms to enter the bloodstream via the portosystemic collaterals, thus bypassing the major reticuloendothelial system in the liver.

DIAGNOSIS

Clinical features The typical attack of SBP is heralded by fever, peripheral leukocytosis, abdominal pain, hypoactive or absent bowel sounds, and rebound tenderness. Most patients do not demonstrate all these symptoms, and some have none. Hence, ascitic fluid should be analyzed whenever the condition of a patient with cirrhosis suddenly deteriorates.

Laboratory tests.

The ascitic fluid is often turbid because of leukocytosis and bacterial growth. Leukocyte cell counts greater than 1,000/mm³ consisting of more than 85% granulocytes are common. Almost all patients have ascitic fluid cell counts greater than 300/mm³; more than half of these are polymorphonuclear cells. However, not all patients with ascitic fluid leukocytosis have SBP. In practice, it is wise to treat patients with antibiotics when the clinical picture is suggestive and the ascitic fluid contains more than 500 white blood cells/mm³. The ultimate criterion for infection is demonstration of organisms either by Gram stain of the fluid (one fourth of cases) or by culture. To maximize detection of the responsible infectious organisms, 5 ml of ascitic fluid should be injected at bedside into both aerobic and anaerobic blood culture bottles. Two thirds of the causative organisms are enteric; *Escherichia coli* and *Klebsiella* species are the

most common agents. *Pneumococcus* and *Streptococcus* organisms are responsible for as many as 20% of cases. In nearly half of cases, blood cultures are positive for the same organism found in the ascitic fluid.

TREATMENT

Cefotaxime administered intravenously at a dosage of 2 g every 6 hours is an appropriate initial treatment for an episode of SBP; in patients with renal insufficiency, the dosage is adjusted downward. On this regimen, 75% to 80% of treated episodes resolve. A few episodes resolve after a change in the antibiotic regimen. However, 20% to 25% of patients die before the infection resolves. Therapy is equally effective whether given for 5 days or 10 days. Patients should be monitored closely and ascitic fluid checked at least once (e.g., after 48 hours) to ensure that the infection is being effectively treated. Despite optimal therapy, 40% to 60% of patients with SBP die. Half of the deaths are a direct result of the peritonitis; the other half succumb to other complications of their severe liver disease. Long-term oral therapy with norfloxacin (400 mg/day) or ciprofloxacin (750 mg once a week) reduces the incidence of recurrent spontaneous bacterial peritonitis attributable to aerobic gram-negative bacteria and should be considered in patients at high risk for recurrence.

COAGULOPATHY.

Clotting abnormalities are uniform in patients with ALF as previously discussed, leaving patients at increased risk for bleeding complications. While synthesis of coagulation factors is decreased, consumption of clotting factors and platelets also may occur, so that platelet levels are often 100,000/mm.³ In the absence of bleeding it is not necessary to correct clotting abnormalities with fresh frozen plasma (FFP). An exception is when an invasive procedure is planned and perhaps in the setting of profound coagulopathy (e.g., INR 7). In addition to the risks associated with transfusion of blood

products, use of plasma supplementation limits the value of coagulation parameters as a means of following the progress of ALF patients and can also lead to volume overload which may exacerbate ICH. Vitamin K is routinely given in a dose of 5-10 mg subcutaneously, regardless of whether poor nutritional status appears to be contributing to the coagulopathy. Experts differ regarding prophylactic use of platelets in thrombocytopenic patients or use of FFP for evidence of severe coagulopathy. Platelet transfusions are not generally used until a low threshold value is observed. In the absence of bleeding, it is safe to use a threshold platelet count of 10,000/mm³, although some experts recommend more conservative levels of 15-20,000/mm³ especially in patients with infection or sepsis.¹²¹ Experience in other conditions of thrombocytopenia suggests that values 10,000/mm³ are generally well tolerated.¹²² When invasive procedures must be performed, platelet counts of 50-70,000/mm³ are usually considered adequate.¹²¹ Patients who develop significant bleeding with platelet levels below approximately 50,000/mm³ should generally be transfused with platelets provided no contraindication exists.

Likewise, bleeding in the setting of a prolonged prothrombin time (INR 1.5) warrants administration of FFP. Recombinant activated factor VII (rFVIIa) may be used in treating coagulopathy in patients with liver disease.

A recent small nonrandomized trial of fifteen patients with ALF found that administration of rFVIIa in combination with FFP produced more effective temporary correction of coagulopathy and thus might be useful in facilitating performance of invasive procedures in these patients particularly in the setting of renal insufficiency in which volume overload is a concern.⁹⁶ This agent will require further study and analysis of cost-benefit ratio (current cost for one dose is approximately \$4,000) before it can be broadly recommended, however.

GASTROINTESTINAL BLEEDING.

Gastrointestinal (GI) bleeding is a recognized complication of ALF. A large prospective multi-center cohort study found that mechanical ventilation for more than 48

hours and coagulopathy were the only significant risk factors for bleeding in critically ill patients of all types. Additional risk factors for bleeding reported in smaller studies have included hepatic and renal failure, sepsis, shock and others. Patients with acute liver failure are thus at high risk for gastrointestinal hemorrhage. Histamine 2 receptor (H2) blocking agents such as ranitidine have long been used in the prophylaxis of GI bleeding in critically ill patients; their efficacy has been supported in several trials. Sucralfate has also been found to be effective in many studies, and there have been smaller randomized trials and a meta-analysis which suggested that sucralfate may be as effective in preventing gastrointestinal bleeding and might be associated with lower risk of nosocomial pneumonia than H2 blockers which lower gastric pH. More recently, however, a much larger (1,200 patients), well-designed trial comparing ranitidine to sucralfate in mechanically-ventilated patients found that ranitidine but not sucralfate decreased the risk of clinically significant bleeding; the incidence of pneumonia was similar for the two groups. Limited studies of proton pump inhibitors (PPIs) as bleeding prophylaxis have demonstrated their effectiveness in maintaining elevated intragastric pH. Two trials found no significant bleeding in PPI-treated patients on mechanical ventilation, but study size may have precluded detection of significant bleeding. H2 blockers have been proven to be effective and PPIs are almost certainly effective as well. PPIs may provide superior protection but this remains to be proven. Sucralfate may be acceptable as second-line treatment.

TREATMENT OF RECURRENT VARICEAL BLEEDING

TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNT

The placement of a transjugular intrahepatic portosystemic shunt (TIPS) is rapidly becoming an accepted technique for the treatment of bleeding esophageal varices refractory to endoscopic therapy. This procedure creates a shunt but avoids the complications of major surgery. The initial enthusiasm surrounding the introduction of TIPS has been tempered by recognition of the complications of encephalopathy, which

develops in 10% to 30% of patients and is refractory to medical therapy in approximately 5%, and shunt stenosis or occlusion, which develops in 30% to 50% of patients at 12 months. It seems reasonable to restrict this form of treatment to centers with experienced staff and to patients who are poor surgical candidates, are refractory to endoscopic therapy, or have bleeding from gastric rather than esophageal varices. A number of trials have compared TIPS with endoscopic therapy (either sclerotherapy or banding) after initial control of hemorrhage in patients with Child class A or B cirrhosis; several tentative conclusions can be drawn from these studies. Mortality associated with TIPS is not significantly different from that associated with endoscopic treatment. TIPS is superior to endoscopic therapy in the prevention of variceal rebleeding (19% versus 47%). TIPS may be particularly attractive for patients in whom compliance with follow-up endoscopy is in doubt. However, one must accept the increased risk of hepatic encephalopathy after TIPS (34%, versus 18% after endoscopic therapy). TIPS is less attractive for patients with advanced chronic liver disease and Child class C cirrhosis with poor synthetic function. The survival of patients after TIPS can be predicted by the Mayo Clinic endstage liver disease score, which includes the following four variables: serum bilirubin, serum creatinine, INR for prothrombin time, and cause of the underlying liver disease. The long-term utility of TIPS must also be evaluated in context of shunt stenosis or occlusion, which is a management problem after TIPS. Thus, for esophageal variceal bleeding, TIPS cannot be recommended as the first-choice treatment for prevention of variceal rebleeding.

SURGICAL PORTOSYSTEMIC SHUNT

Recurrent or continued bleeding may indicate a need for a surgical portosystemic shunt. This major operation carries a mortality of approximately 40% when performed on an emergency basis. If bleeding can be stopped and shunt surgery performed electively, mortality declines substantially. Although portosystemic shunting procedures do not appear to prolong survival, they do prevent subsequent bleeding. The major problem after surgery is intractable hepatic encephalopathy and hepatic failure. The

preferred shunt procedure is the one with which the surgeon is most experienced. A distal splenorenal shunt with concomitant gastroesophageal devascularization selectively decompresses esophageal varices while maintaining mesenteric blood flow to the liver. In most but not all studies, use of the distal splenorenal shunt reduced the incidence of severe encephalopathy as a late complication after surgery, compared with conventional shunts. The procedure is technically difficult; time will reveal if it possesses any long-term advantages.

PROPHYLACTIC TREATMENT FOR VARICEAL BLEEDING

Because the first episode of variceal bleeding can result in significant morbidity and mortality, there has been considerable interest in the prophylactic treatment of esophageal varices in persons who have never bled. Prophylactic portosystemic shunts decrease rebleeding but do not enhance survival. Prophylactic sclerotherapy has been studied in several centers with mixed results. In the largest study, which was restricted to alcoholic patients, this approach proved harmful. The experience with the beta-adrenergic antagonists propranolol and nadolol has been somewhat more encouraging because the drugs appear both to prevent the first episode of bleeding and to reduce mortality associated with bleeding in patients who have moderate or large esophageal varices.⁵¹ If a patient known to have large varices is well motivated and tolerates the medication, beta-adrenergic antagonists may be considered. Isosorbide-5-mononitrate, a long-acting nitrate, may also help prevent the first variceal hemorrhage.

HEMODYNAMICS/RENAL FAILURE

Hemodynamic derangements consistent with multiple organ failure occur in ALF; the underlying mechanisms are complex and incompletely understood. Management of hemodynamic balance becomes increasingly important and difficult in the face of elevated

ICP and/or compromised renal function. Preservation of renal function is imperative in this setting. In many ways patients with ALF resemble physiologically the patient with cirrhosis and hepatorenal syndrome. Intravascular volume deficits may be present on admission due to decreased oral intake resulting from altered mental status, transudation of fluid into the extravascular space, and possibly GI blood loss.

Most patients will require fluid resuscitation initially. Low systemic vascular resistance results in low blood pressures even in the fluid-resuscitated patient, and placement of a pulmonary artery catheter may aid in assessing volume status and guiding further management. Fluid replacement with colloid (such as albumin) is preferred rather than crystalloid (such as saline); all solutions should contain dextrose to maintain euglycemia.

While adequate fluid replacement and treatment of potential infection and sepsis may help to correct hypotension, inotropic or pressor support may be required in order to maintain mean arterial pressures of at least 50-60 mmHg. There has been debate over which agents are best used to support blood pressure in ALF and whether they are useful at all. Alpha-adrenergic agents such as epinephrine and norepinephrine have been thought to potentially worsen peripheral oxygen delivery. On the other hand, dopamine has actually been associated with increased systemic delivery of oxygen. In any case, the hypotension and vasodilatation associated with ALF will generally respond to these agents, and they should be used if needed to maintain perfusion of vital organs. Agents that promote vasoconstriction are generally avoided unless significant systemic hypotension is present, and therefore should not be used in the setting of decreased intracranial perfusion with normal systemic blood pressure.

Acute renal failure is a frequent complication in patients with ALF and may be due to dehydration, hepatorenal syndrome or acute tubular necrosis. The frequency of renal failure may be even greater with acetaminophen overdose or other toxins, where direct renal toxicity is seen. Although few patients die of renal failure alone, it often contributes to mortality and may portend a poorer prognosis. Every effort should be made to protect renal function by maintaining adequate hemodynamics, avoiding nephrotoxic agents such as aminoglycosides and non-steroidal anti-inflammatory drugs, and by the prompt identification and treatment of infection.

When dialysis is needed, continuous rather than intermittent modes of renal replacement therapy (e.g., continuous venovenous hemodialysis [CVVHD]) should be used, as they have been shown in randomized trials to result in improved stability in cardiovascular and intracranial parameters compared with intermittent modes of hemodialysis. Intravenous contrast agents are associated with nephrotoxicity in the setting of compromised hepatic function, and should be used with caution. If contrast must be administered, pretreatment with NAC may be of value, although this remains controversial. The potential utility of prostaglandins and NAC in improving hemodynamics and renal function was discussed previously; neither has sufficient evidence to be recommended as part of the management of hemodynamic derangements in ALF at this time, although NAC may have other benefits as discussed above. Evidence that terlipressin or vasopressin may be useful in patients with cirrhosis and hepatorenal syndrome has raised the question of whether this agent might benefit patients with ALF as well. A recent small study of terlipressin in patients with ALF found that even in very small doses, the drug was associated with increased cerebral blood flow and ICH. Such results indicate that at this time the risks associated with vasopressin use appear to outweigh its benefits in patients with ALF.

The observation that hemodynamic status as well as ICH tends to improve after removal of the native liver during transplantation for ALF led to a recommendation of hepatectomy as a “last resort” means of improving severe circulatory dysfunction in these patients. This option is based on uncontrolled studies and case reports, where successful outcomes have occasionally been reported even with patients who remained anhepatic for more than 48 hours. Despite these reports, hepatectomy to control hemodynamics cannot be recommended.

ASCITES

Occasionally, ascites presents as a right-sided pleural effusion. Portal hypertension, decreased serum albumin with consequent loss of oncotic force within the vascular and interstitial spaces, and renal retention of sodium and water contribute to

ascites formation. Although infectious, pancreatic, or neoplastic causes of ascites are infrequent, they should not be overlooked, because therapy and prognosis differ for each condition. To exclude such possible causes, a small amount of ascitic fluid should be removed from the abdominal cavity using a narrow-gauge needle. The gross appearance of the fluid may suggest an unusual etiology. For instance, cloudy fluid implies an infection; bloody fluid, a tumor; and milky fluid, lymphatic obstruction. Routine laboratory studies of the fluid should include white cell and differential cell counts, protein and albumin determinations, and culture. In ascites caused by cirrhosis, the serum-ascites albumin gradient is greater than 1.1, the total protein is less than 2.5 g/dl, the total white cell count is less than 300/mm³, the proportion of granulocytes is less than 30%, and cultures are negative. Approximately 5% of patients with ascites attributable to cirrhosis have ascitic fluid that has a total protein concentration greater than 2.5 g/dl.

PARACENTESIS

Large-volume paracentesis has become popular in the treatment of patients with ascites. In one study, paracentesis of 4 to 6 L/day was accomplished safely and resulted in shorter hospital stays and fewer complications than conventional diuretic therapy. Patients usually welcome paracentesis because it relieves considerable discomfort. It also provides the physician with the ascitic fluid necessary for diagnostic purposes. Subsequent work has shown that the administration of 6 to 8 g/L of intravenous albumin after 5 L or more prevents renal insufficiency and hyponatremia induced by paracentesis. In about 5% of patients, ascites do not respond to the usual dosages of conventional diuretic medication, or diuresis is achieved only at the expense of renal function. In these patients, insertion of the LeVeen peritoneovenous shunt has been considered. The shunt routes ascitic fluid subcutaneously from the peritoneal cavity to the internal jugular vein via a one-way valve. Compared with medical therapy alone, the peritoneovenous shunt results in speedier resolution of the ascites. However, placement of the shunt does not alter survival. Because most patients continue to require diuretics, although at lower doses, the shunt may produce benefit by increasing renal

blood flow. Serious complications of the shunt include bacterial infection of the peritoneum, disseminated intravascular coagulation, and rupture of esophageal varices. Because of these complications, the peritoneovenous shunt is seldom used.

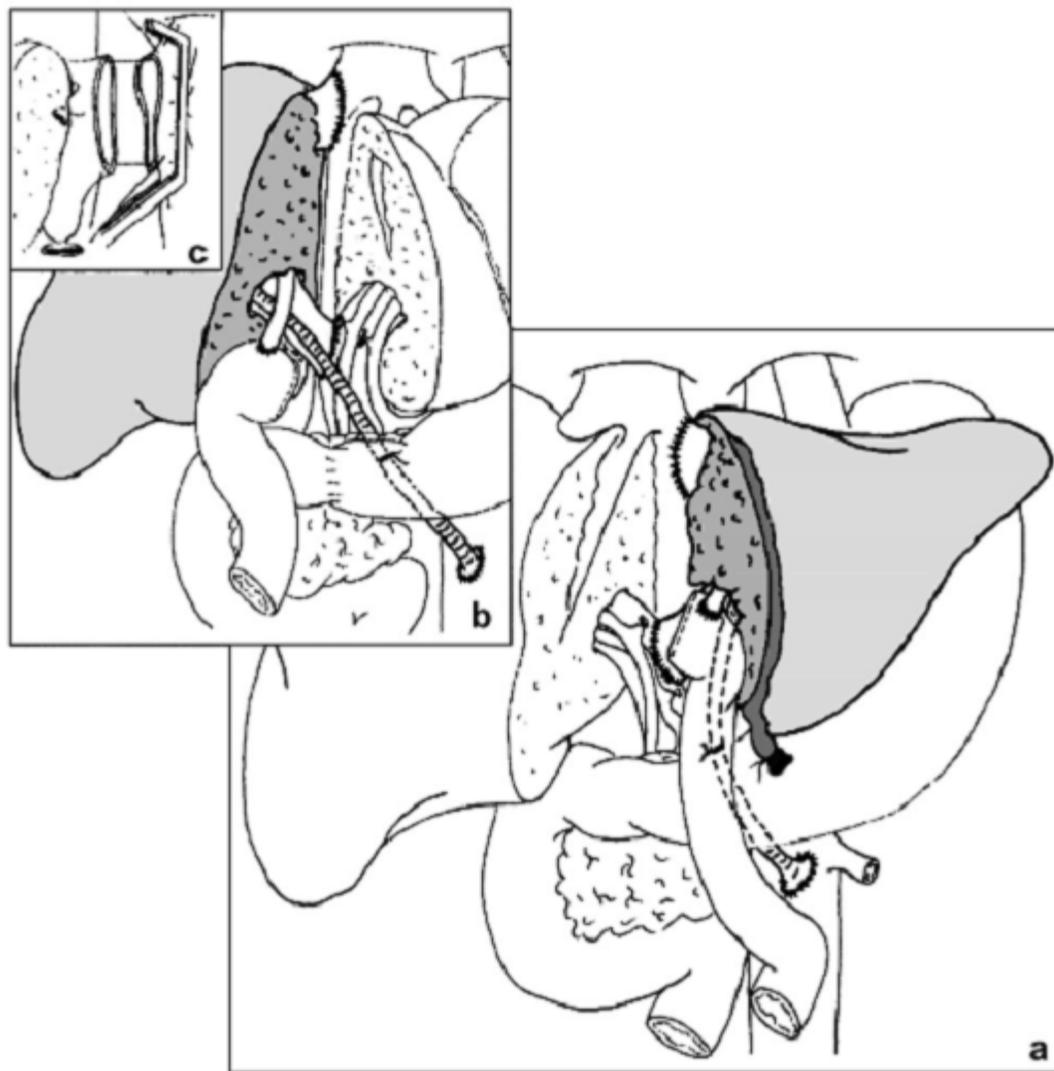
TREATMENT OF REFRACTORY ASCITES

Refractory ascites can also be effectively managed by placement of a TIPS. The majority of patients still require diuretic therapy, albeit at reduced dosages. The value of TIPS compared with repeated large-volume paracentesis for the management of refractory ascites awaits further study. However, TIPS is effective in the treatment of hepatic hydrothorax, which often accompanies refractory ascites.

TRANSPLANTATION AND PROGNOSIS

TRANSPLANTATION

Orthotopic liver transplantation remains the only definitive therapy for patients who are unable to achieve regeneration of sufficient hepatocyte mass to sustain life. As mentioned previously, the advent of transplantation has coincided with improvement in overall survival rates from as low as 15% in the pre-transplant era to 60% presently. Advances in critical care and changing trends toward more benign etiologies such as acetaminophen (having a better overall outcome) have likely helped. Spontaneous survival rates are now around 40%,⁵ compared to 15% in the pre-transplant era. Post-transplant survival rates for ALF have been reported to be as high as 80% to 90%,^{5,93} but accurate long-term outcome data are not yet available. In the largest U.S. study, only 29% of patients received a liver graft, while 10% of the overall group (1/4 of patients listed for transplantation) died on the waiting list.⁵ Other series have reported death rates of those listed for transplant as high as 40%,^{150,151} despite the fact that ALF remains the one condition for which the most urgent (UNOS status 1) listing is



LIVER SUPPORT SYSTEMS

A support device to replace the acutely failing liver seems a reasonable but elusive goal. The ideal replacement for the failing liver would detoxify, metabolize and synthesize; in short, perform all the liver's many functions. A variety of systems have been tested to date, with no certain evidence of efficacy. Sorbent systems embody only detoxification and no hepatocyte replacement. Such systems, employing charcoal or other adherent particles in a capsule or column device placed in an extracorporeal circuit, may show loss of platelets and worsening of coagulation parameters across the device. Transient improvement of hepatic encephalopathy may be observed but no improvement in hepatic function or long-term benefit has been shown. Hepatocytes, whether of human

or other mammalian origin, have been used in cartridges in extracorporeal circuits, either with or without sorbent columns.

Few controlled trials have been published, and some preliminary reports suggest no benefit to outcome, with or without transplantation. One recent multicenter trial did report improved short-term survival for a subgroup of patients with ALF who were treated with a porcine hepatocyte-based bioartificial liver, but corroboration of these results by further studies will likely be required before the true utility of this device can be established. All such trials are difficult to perform and to control properly due to the rarity of well-characterized patients, the heterogeneity of etiologies, varying levels of disease severity and varying access to transplantation. A recent meta-analysis, considering all forms of devices together, demonstrated no efficacy for bio-artificial liver devices for the treatment of ALF. A variety of other strategies have been employed including exchange transfusion, charcoal hemoperfusion, extracorporeal liver perfusions, and intra-portal hepatocyte infusions. To date, none can be recommended, and their use remains experimental. Efforts to improve hepatocyte regeneration have likewise been futile thus far.¹⁶⁰ When heterotopic or partial replacement transplantations have been performed it appears that the native liver can recover in some but not all situations, but this may require weeks or months to occur, underscoring the real challenge to liver replacement devices, that is, that liver assist devices might well be required for long periods of time.

PROGNOSIS

Table. Potentially Helpful Indicators* of Poor (Transplantfree)
Prognosis in Patients With ALF
Etiology
Idiosyncratic drug injury
Acute hepatitis B (and other non-hepatitis A viral infections)
Autoimmune hepatitis

Mushroom poisoning
Wilson disease
Budd-Chiari syndrome
Indeterminate cause
Coma grade on admission
III
IV
King's College Criteria:
Acetaminophen-induced ALF:
Arterial pH ≥ 7.3 (following adequate volume resuscitation) irrespective of
coma grade OR
PT ≥ 100 seconds (INR ≥ 6.5) \geq serum creatinine ≥ 300 μ mol/L (3.4 mg/
dL) in patients in grade III/IV coma
Non-acetaminophen-induced ALF:
PT ≥ 100 seconds irrespective of coma grade OR
Any three of the following, irrespective of coma grade:
– Drug toxicity, indeterminate cause of ALF
– Age ≥ 10 years or ≥ 40 years \ddagger
– Jaundice to coma interval ≥ 7 days \ddagger
– PT ≥ 50 seconds (INR ≥ 3.5)
– Serum bilirubin ≥ 300 μ mol/L (17.5 mg/dL)

Given limited organ availability, lack of good alternatives to transplantation, and potential complications of lifelong immunosuppression, accurate prognosis in ALF is a paramount goal. Prognostic scoring systems, although derived from data on relatively

large numbers of patients, still fail to achieve success, given the wide variety of etiologies that lead to this end stage syndrome. The traditional King's College Hospital criteria have been the most commonly utilized and most frequently tested of the numerous proposed prognostic criteria for ALF. Several studies evaluating these criteria have shown positive predictive values ranging from just below 70% to nearly 100% and negative predictive values ranging from 25% to 94%. Overall, such prognostic scores have proven to have acceptable specificity but low sensitivity to determine outcome. Criteria based on decreased levels of factor V in patients with encephalopathy predicted death in acute viral hepatitis cases with a positive predictive value of 82% and a negative predictive value of 98%, but subsequent studies in both acetaminophen and nonacetaminophen ALF have shown these criteria to be less accurate than King's College Hospital criteria in predicting outcome.

In a recent meta-analysis, Bailey et al. compared various prognostic criteria in patients with ALF due to acetaminophen, including King's College Hospital criteria, various combinations of elevated serum creatinine, encephalopathy, and prothrombin time elevations (both single and serial measurements), decreased factor V levels, the Acute Physiology and Chronic Health Evaluation (APACHE) II scores and Gc globulin (vitamin D binding protein, a liver-derived component of the actin-scavenging system [17]). The analysis found that King's College Hospital criteria and pH 7.30 alone were both fairly specific in predicting a poor outcome. While the King's College Hospital criteria were more sensitive than pH alone (69% versus 57% sensitivity), use of both criteria was still likely to miss many patients who would ultimately require transplantation. The authors also found that an APACHE II score of 15 on admission had a specificity of 92% (comparable to King's College Hospital criteria) with a much better sensitivity of 81%, but this measure was only examined in one limited study. Other factors such as age and the length of time between onset of illness and onset of encephalopathy have previously been proposed as important prognostic indicators in ALF, these parameters did not affect outcome in the largest U.S. multi-center study of ALF to date. Patients presenting in grade III or IV encephalopathy were less likely than those patients presenting in grade I or II encephalopathy to survive without receiving a liver graft. The most significant predictor of outcome in this study was etiology of ALF, as patients with ALF due to

acetaminophen, hepatitis A, shock liver, or pregnancy-related disease showed 50% transplant free survival, while all other etiologies showed 25% transplant-free survival. Other prognostic criteria have been proposed including severity of SIRS, Alpha fetoprotein (AFP) levels, ratios of factor VIII and factor V, liver histology, CT scanning of the liver, cytokine levels, serum phosphate levels, and adrenal insufficiency. Evaluations of these criteria have had varied results; while some appear promising, more research is needed to determine their reliability. The Model for Endstage Liver Disease (MELD) score, now widely used to predict mortality among patients with chronic liver disease who are under consideration for liver transplantation, cannot currently be recommended as applicable to ALF, a different condition from cirrhosis.

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Prepared ass. Romaniuk T.

POSTCHOLECYSTECTOMY SYNDROME

The name a “postcholecystectomy syndrome” (PCES) is understood as a complex of pathological processes and states, that arose after cholecystectomy and are clinically connected with cholecystitis and conducted earlier operative treatment. It is needed to mention, that the term “postcholecystectomy syndrome” (PCES) is a collapsible concept and as a nosology form of disease can serve as a valuable diagnosis. Postoperative violations do not always mean the failure of cholecystectomy, because they can arise as a result of other different diseases which are masked under cholecystitis.

Classification

(by O.O.Shalimov, 1988)

PCES is distinguished:

1. PCES, related to the diseases of gall-bladder and changes in bilious ducts which were uncorrected at cholecystectomy (51 %):

- a) stenotic papillitis;
- b) stenoses of bilious ducts;
- c) tubular stenosis of choledochus on soil of chronic pancreatitis;
- d) residual choledocholithiasis;
- e) mechanical violations of patency of duodenum (chronic duodenojejunal obstruction and arteriomesenteric obstruction);
- f) cysts of bilious ducts;
- g) parasite diseases of bilious ducts.

2. PCES, conditioned by the changes which arose as a result of operative treatment concerning cholecystitis (19 %):

- a) iatrogenic damage of bilious ducts;
- b) scar stricture and deformation of bilious ducts with violation of outflow of bile;
- c) deformation of duodenum with violation of evacuation of its maintenance;
- d) remaining gall-bladder;
- e) strange bodies of bilious ways (filaments, needles, prosthetic appliances, fragments of drainages);
- f) reflux after transduodenal sphincteroplasty or choledochoduodenostomia;
- g) neurinoma of the cut nerves.

3. Disease of hepatopancreatoduodenal area, connected with protracted cholecystitis (17 %):

- a) chronic cholangiohepatitis;
- b) chronic pancreatitis;
- c) pericholedocheal lymphadenitis;
- d) intrahepatic and parahepatic abscesses;
- e) cholangiogenic sepsis;
- f) tumour of liver, bilious ducts and pancreas.

4. Organic and functional diseases of other organs and systems, not connected with the disease of gall-bladder and its deleting (7 %):

- a) hernia of the esophagus opening of diaphragm;
- b) ulcerous disease of stomach and duodenum;
- c) chronic gastroduodenitis, colitis;
- d) tumours of stomach and intestine;
- e) nephroptosis;
- f) kidney-stone disease and chronic pyelonephritis;
- g) solar plexitis;
- h) diencephalic syndrome;
- i) asthenovegetative syndrome, psychopathy, hysteria;
- j) Abdominal ischemic syndrome;
- k) deforming spondylarthrosis.

5. Nervously-kinetic violations of biliary ducts and duodenum (6 %):

- a) dyskinesia of biliary ducts and large papilla of duodenum (LDP);
- b) blood pressure LDP low;
- c) LDP hypertension;
- d) hypokinesia of duodenum;
- e) reflux-gastritis.

Symptoms and clinical passing

Clinical symptoms of postcholecystectomy syndrome, as a rule, show up in four basic complex symptoms: by a pain syndrome, mechanical icterus, cholangitis, internal and external biliary fistula.

Most patients continue to be disturbed by the attacks of “hepatic colic” or dull aching pain appears in right hypochondrium or in an epigastric area.

A leading role in the PCES (35–40 %) development belongs to “forgotten concretum”. There is a repeated formation of stone in rare cases.

An icterus which shows up by discoloration of skin, sclera, sky, by the positive reaction on biliary pigments, diminishment or absence of urobilin in urine and stercobilin in an excrement, by the increase of maintenance of bilirubin and alkaline phosphatase in a blood, is the important sign of PCES. As a rule, such patients are disturbed by the itch of skin and general weakness. The increase of temperature of body and fever are the important sign of cholangitis, which arise after the pain attack of “hepatic colic”. Such attacks, as a rule, are accompanied by the changes in blood (leucocytosis, appearance of young and stab neutrophil) test and functional violations of liver.

During intravenous cholangiography on sciagrams dilatation ducts can be seen and their emptying from the contrasting matter is slow. Retrograde pancreatocholangiography (Pic. 3.4.32) can set a level and character of obstruction of ducts (stone, tumour, indurative pancreatitis and others like that). In the PCES diagnostics ultrasonic examination occupies

important place by which it is possible to verify the sizes of bilious ways and expose concrement (Pic. 3.4.33).

The presence of external bilious fistula or drainpipe in biliary passings enables to contrast both intrahepatic and external bilious ducts (Pic. 3.4.34). External fistula are localized in the area of postoperative scar. Thus bile and pancreatic juice cause maceration of skin, rashes and infiltrate are formed. External fistula periodically can be closed and opened, accompanied by icterus and cholangitis, presence of gases in bilious ways and contrasting mass during the roentgenologic inspection of patient.

Variants of clinical passing

PCES engulfs different types of pathology, which differ by both the reasons of origin and clinical signs. At the PCES development conditioned by the uncorrected changes at cholecystectomy, a leading role belongs to concrement forgotten in a general bilious duct during cholecystectomy (35–40 %).

The real relapse of cholelithiasis is met seldom, only in 5–7 % and, as a rule, in people with the protracted anamnesis of gallstone disease, after choledocholithotomy and previous choledocholithiasis. Both the first and the second variants of pathology (forgotten stone or recurrent cholelithiasis), can cause pain of a different character in the area of liver, icterus and cholangitis. Distinguishing the real relapse of choledocholithiasis from unreal is difficult, therefore acquired concrement cholegraphy, sonography, retrograde pancreatocholangiography have the decisive value in diagnostics.

Scar stricture of large duodenal papilla (LDP) is met in 30 % cases. They are divided into primary and secondary. Under the concept of “scar of LDP stricture” the damage of distal part of general bilious duct by length from 5 to 10 mm is understood, the most widespread reason of which can be a gallstone disease. This complication usually shows up the attacks of hepatic colic or permanent aching pain in right hypochondrium. At intravenous cholegraphy contrasting of all parts of bilious ways is marked in such patients, expressed ectasia and delay of selection of contrast. Retrograde pancreatocholangiography has the decisive value in diagnostics of this complication.

It is needed to mark that except for the organic stenoses LDP, occasionally there are undiagnosed before operation functional, transitional forms of defeats of bilious ways which can be accompanied by high blood pressure or low blood pressure of sphincter. Operating trauma of nervous branches, formation of connections and scars always negatively influence the LDP function. Violation of function of sphincter is accompanied by pain and dyspepsia disorders.

Scar strictures of distal part of choledochus on soil of chronic pancreatitis (tubular stenosis) are considered to be a difficult type of pathology of bilious ways (15–16 %). The first signs of this disease, as a rule, arise during 2–3 months after operation and show up by

permanent dull pain in right hypochondrium, dyspepsia disorders, cholangitis and mechanical icterus. Valuable information in diagnostics of tubular stenosis can be got from retrograde pancreatocholangiography and percutaneous transhepatic cholangioduodenography.

The frequent reason of origin of pain after cholecystectomy is an inflammatory process in regional lymphatic knots. The so called pericholedocheal lymphadenitis can squeeze a general bilious duct, cause violation of function of the Oddi sphincter and predetermine development of pancreatitis.

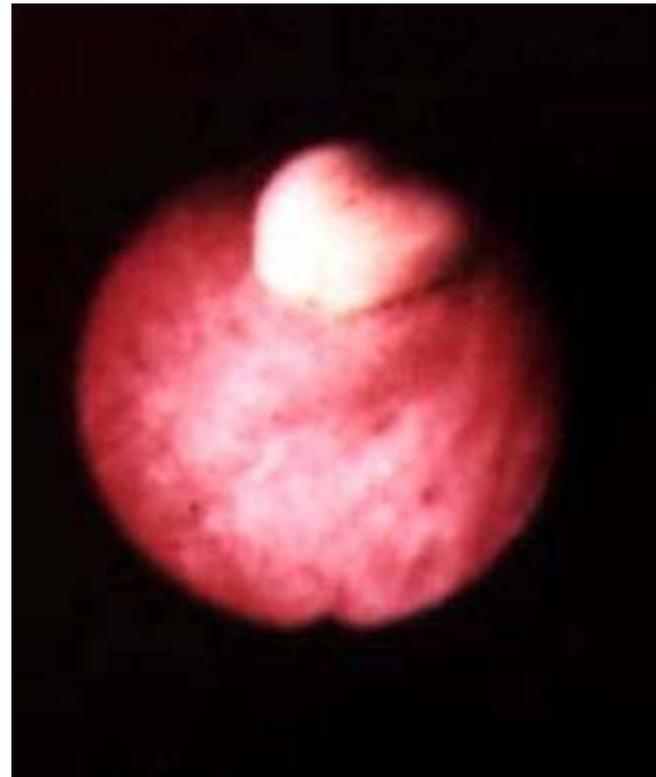
Pancreatitis, which is connected with cholecystectomy, can be met in 30–40 % patients with PCES. Similar pancreatitis arises up as a result of the undiagnosed stenosis of the Oddi sphincter and distal part of Wirsung's duct during operation on bilious ways. It results in violation of outflow of bile and pancreatic maintenance and provokes acutening of existing or origin of acute pancreatitis.

In 20–25 % cases the reason of PCES is duodenostasis undiagnosed before operation. Nausea, bitter taste in the mouth, periodic vomiting and loss of weight are the basic signs of this disease. Facilitation, as a rule, comes, when patients occupy knee-elbow position or lie down on the right side. Duodenomanometry is the basic method of diagnostics of duodenostasis (CDS) (normal pressure in a duodenum is 100–120 mm of col. or 1,17 kPa). During roentgenologic examination (tube relaxation duodenogram) violation of duodenal patency is exposed. The last can be conditioned by high duodenojejunal corner, arteriomesenteric compression, cicatricial periduodenitis, lacks of development of duodenum and others. Uncorrected during cholecystectomy such changes of duodenum can be the reason of unsatisfactory results of operative treatment of patients with cholecystitis.

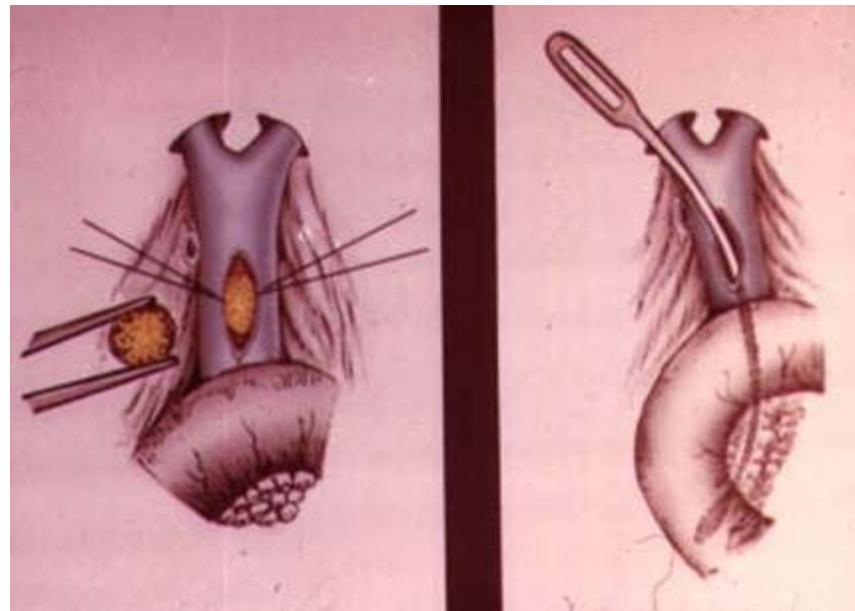
The organic and functional diseases of other organs and systems (hernia of diaphragm, ulcerous disease of stomach, duodenum and others like that), which are not connected with the disease of gall-bladder and its deleting, always require their preoperative diagnostics and previous treatment.

Diagnosis program

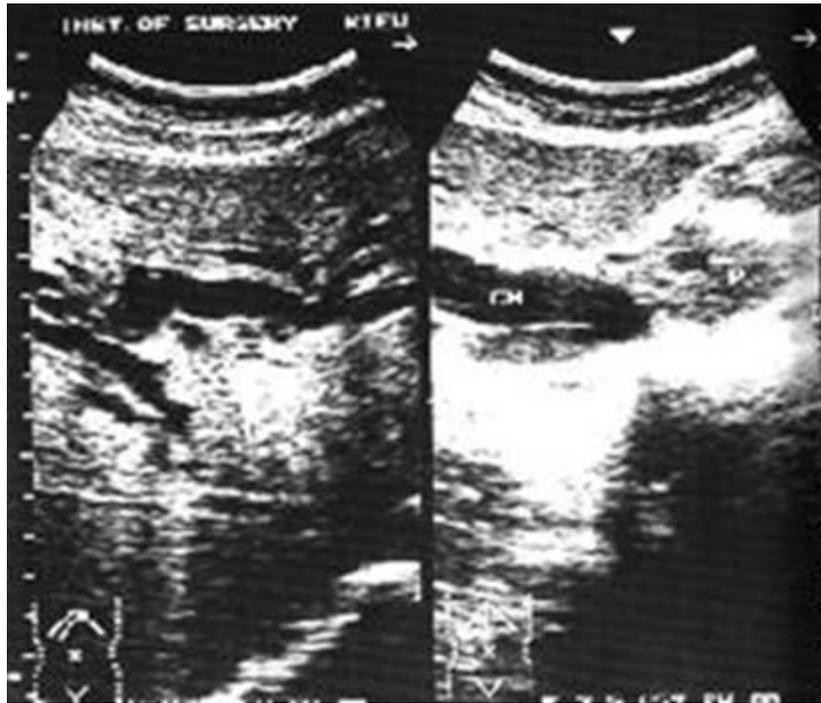
1. Anamnesis and physical methods of inspection.
2. Laboratory inspections (general analysis of blood, urines, biochemical blood test: bilirubin, cholesterol, creatinine, urea, amylase and others like that).
3. Duodenal intubation, duodenomanometry.
4. Roentgenoscopy and roentgenography of the stomach and duodenum, bilious ways.
5. Intravenous infusion cholegraphy.
6. Retrograde pancreatocholangiography.
7. Percutaneous transhepatic cholangioduodenography.
8. Computer tomography.
9. Scanning of livers.



Choledohoscopia. Choledochitis.



Supraduodenal choledochotomy



Sonography. Mechanical jaundice.



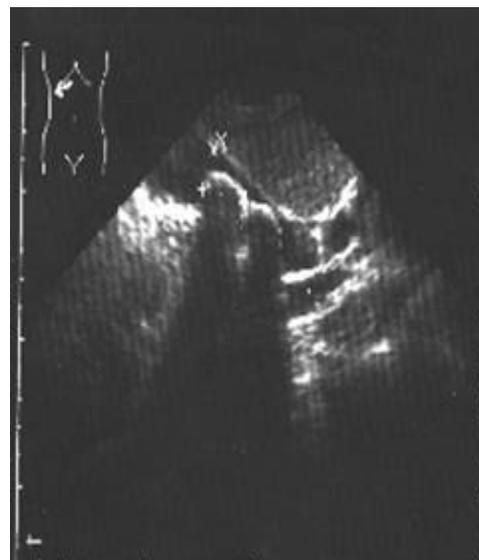
Choledocholithiasis. Retrograde cholangiopancreatography.



Choledocholithiasis on the terminal part of the choledochus. Transcutaneous transhepatic cholangiography.



Choledocholithiasis, ERCP



Sonography. Dilatation of the common bile duct.



Choledocholithiasis, fistulocholangiography.



Retrograde cholangiopancreatography. Choledocholithiasis.

Differential diagnostics

Chronic pancreatitis. Everyday medical practice testifies that in patients with the diseases of biliary ways the symptoms of damage of pancreas appear very often. Actually biliary pancreatitis is the most widespread form of chronic pancreatitis. For this disease belting pain and positive Mayo-Robson's symptom are characteristic. Thus, pain can be of “hepatic colics” character, accompanied by icterus or cholangitis. By palpation patients feel painfulness in the overhead half of abdomen.

At acutening of chronic pancreatitis there is growth of diastase activity in urines and amylase in blood. Ultrasonic examination, computer tomography have the important value in diagnostics of chronic pancreatitis.

Ulcerous disease of stomach and duodenum. Considerable difficulties can arise during conducting of the differential diagnostics of PCES with ulcerous disease, especially at its complications by penetration.

For diagnostics of this pathology character of pain, its intensity, time of origin and periodicity of appearance have the important value. In all doubtful cases it is necessary to conduct the x-Ray examination of both biliary ways and gastro-intestinal tract. Final confirmation of diagnosis can be got by endoscopy.

Tactics and choice of treatment method

Patients with a postcholecystectomy syndrome must be hospitalized in permanent surgical establishments, where after the inspection it is needed to define the character of treatment (conservative or operative).

Conservative treatment is conducted for:

- 1) patients with dyskinesia of bilious ways, with different diseases of gastro-intestinal tract, diabetes and other pathology of therapeutic type;
- 2) patients who are indicated to endoscopic papillosphincterotomy or surgical interference, but they renounce its conducting;
- 3) patients who need operative treatment, however, impossible it is to execute it because of difficult accompanying pathology.

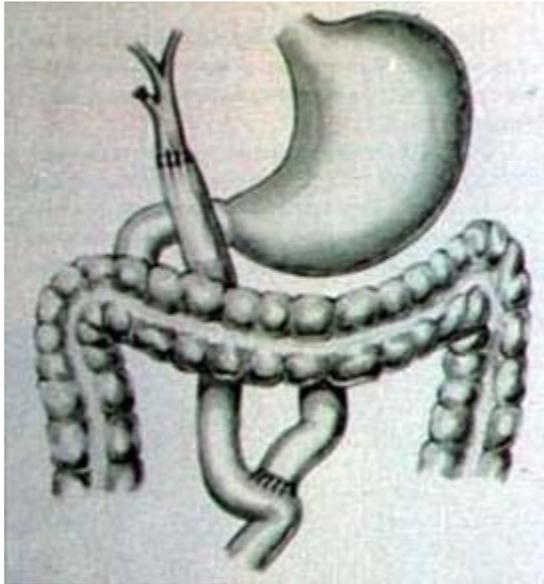
Conservative treatment is provided for:

- 1) removal of pain syndrome;
- 2) improvement of outflow of bile;
- 3) fight against an infection;
- 4) correction of exchange violations;
- 5) treatment of accompanying pathology.

Actually, taking into account the exposed reasons of PCES tactics of treatment can be chosen effectively. At residual choledocholithiasis, endoscopic papillosphincterotomy the stenotic papillitis method of choice with extraction of concrement must be chosen.

A diet is appointed to maximally spare the biliary system and gastro-intestinal tract, to foresee diminishment of calorie content, considerable limitation of fats and products which contain surplus of cholesterol.

From medicinal devices which normalize the function of sphincters of bilious ducts and duodenum (reglan, cerukal, sulperid, motillium, liquiriton, aprogen and others) preparations are used, they adsorb unconjugated bilious acids (fosfalugel, cholestyramin, bilignin), diminish inflammation of mucus shell (de-nol, vikair, venter) and repress activity of pathological microbe flora (furazolidon, biseptol, erytromicine and others).



Choledohejunostomy by Roux.

If the process spreads on liver or pancreas, correction is brought in medicinal therapy. The uneffectiveness of the conducted conservative therapy is an indication to the repeated operative treatment on bile-excreting ways.

Indication to operative treatment

1. Tumours of duodenal papilla, scar stricture of biliary duct, large stump of cystic duct and tubular stenosis of choledochus.
2. Mechanical icterus, internal and external fistula, expressed with the frequent relapses of cholangitis.

In argumentation of method of operative treatment of postcholecystectomy syndrome the intraoperative revision of biliary ways is of important value. In this plan of expansion of general biliary duct more than 10 mm is considered as a sign of biliary hypertension.

Considerable methods of the PCES diagnostics are also intraoperative cholangiography, choledochoscopy, cholangiomanometry and debitometry.

The choice of method of surgical treatment of postcholecystectomy syndrome, first of all, depends on character of pathology. Basic of them at postcholecystectomy syndrome are: 1) choledochoduodenostomy (by Finsterer, Flerken, Jurash, Kirschner); 2) choledohejunostomy (on the eliminated loop for Roux and after the Brawn method with choke by O.O. Shalimov); 3) transduodenal papillosphincteroplasty and wirsungoplasty.

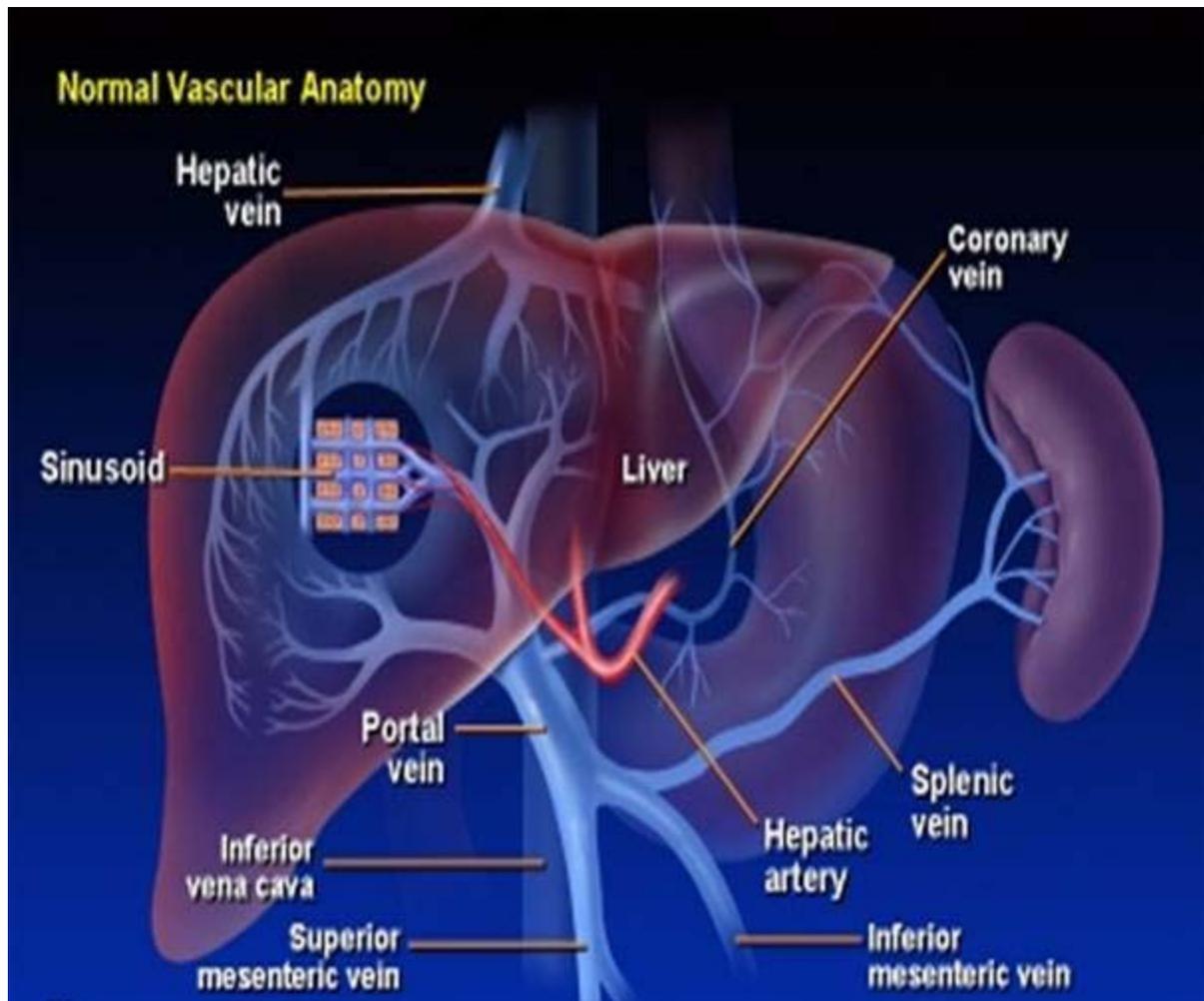
[Biliary-enteric anastomosis: Interrupted technique \(Video 1\)](#)

[Biliary-enteric anastomosis: Running technique \(Video 2\)](#)

SYNDROME OF PORTAL HYPERTENSION

LIVER TRANSPLANTATION CHRONIC PANCREATITIS

Syndrome of portal hypertension is a complex of diseases, which predetermine the origin of violations of outflow of blood from the system of portal vein. The increase of pressure of blood in the pool of portal vein, which is accompanied by specific symptoms and complications, is the basic display of pathology.



Classification

According to the causal factors the syndromes of portal hypertension are divided into three kinds:

1. Intrahepatic, that arises at different forms of cirrhosis of liver.
2. The suprahepatic arises at the thrombosis of hepatic veins (the Badda-Kiari syndrome).
3. Pre-hepatic is thrombosis of basic trunk of portal vein.

Stages of development of portal hypertension.

1. Preclinical.

2. Expressed clinical signs.
3. Complications.

Cirrhosis of liver. Intrahepatic portal hypertension

The cirrhosis of liver is a chronic progressive disease, the characteristic signs of which are the defeats of parenchymatous and interstitial tissue of organ, necrosis and dystrophy of hepatic cells, with the subsequent node regeneration as diffuse excrescence of connecting tissue. All this finally results in a different degree of insufficiency of functions of liver and origin of syndrome of portal hypertension.

[video](#)

Etiology and pathogenesis

Acute and chronic hepatitis, alcoholism, tuberculosis, malaria, syphilis, brucellosis, helminthiasis, lacks of heart, gallstone disease, exchange violations are considered the reasons of pathology (hemochromatosis, hepatolenticular degeneration, albuminous starvation and vitamin insufficiency (B6, B12, E), collagenosis (rheumatoid arthritis, lupus erythematosus), thireotoxicosis, toxic factors (industrial poisons, medicines).

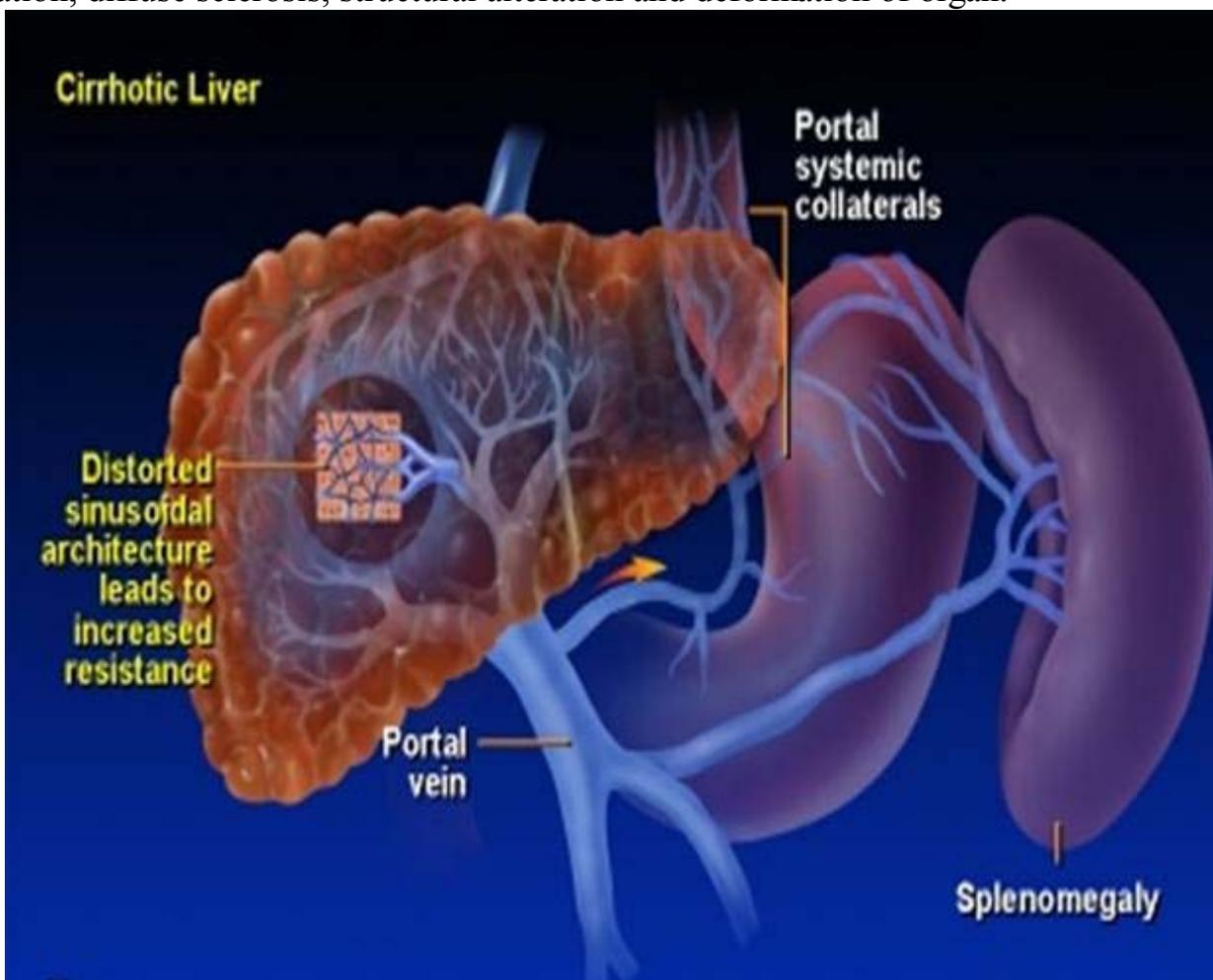
As a result of the influence of harmful factors there is a destruction of hepatocytes (cytolysis) and subsequent excrescence of connecting tissue. Thus there are violations of transhepatic bloodflow, which is the principal reason of portal hypertension. The increase of pressure in the system of portal vein results in expansion of natural portocaval anastomosis: 1) between the veins of cardial part of stomach and esophagus (porto-esophageal way); 2) between paraumbilical veins and umbilical (portoabdominal way); 3) between superior, middle and inferior rectal veins (portorectal way). Of less importance are other groups of anastomosis: portoileocecal, lumbal, rectal, pulmonary.

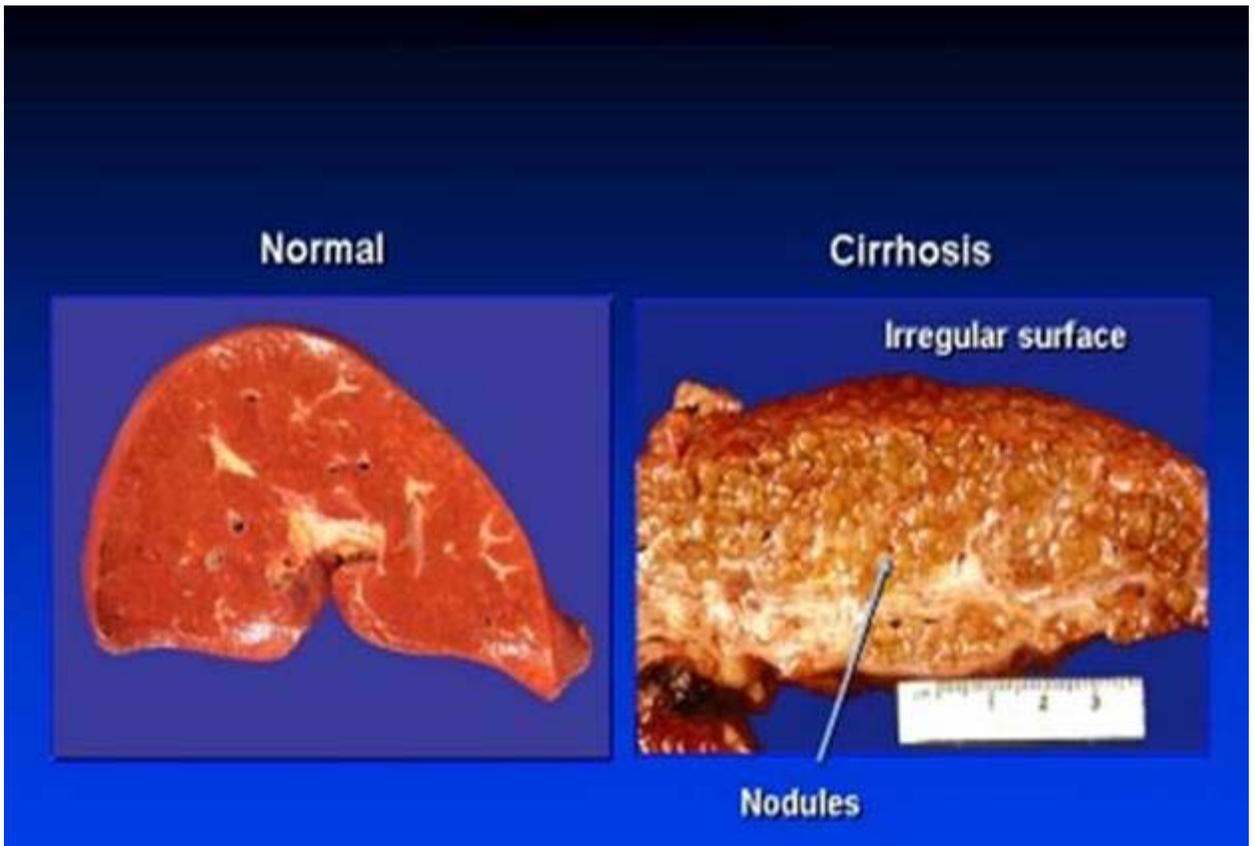
Pathomorphology

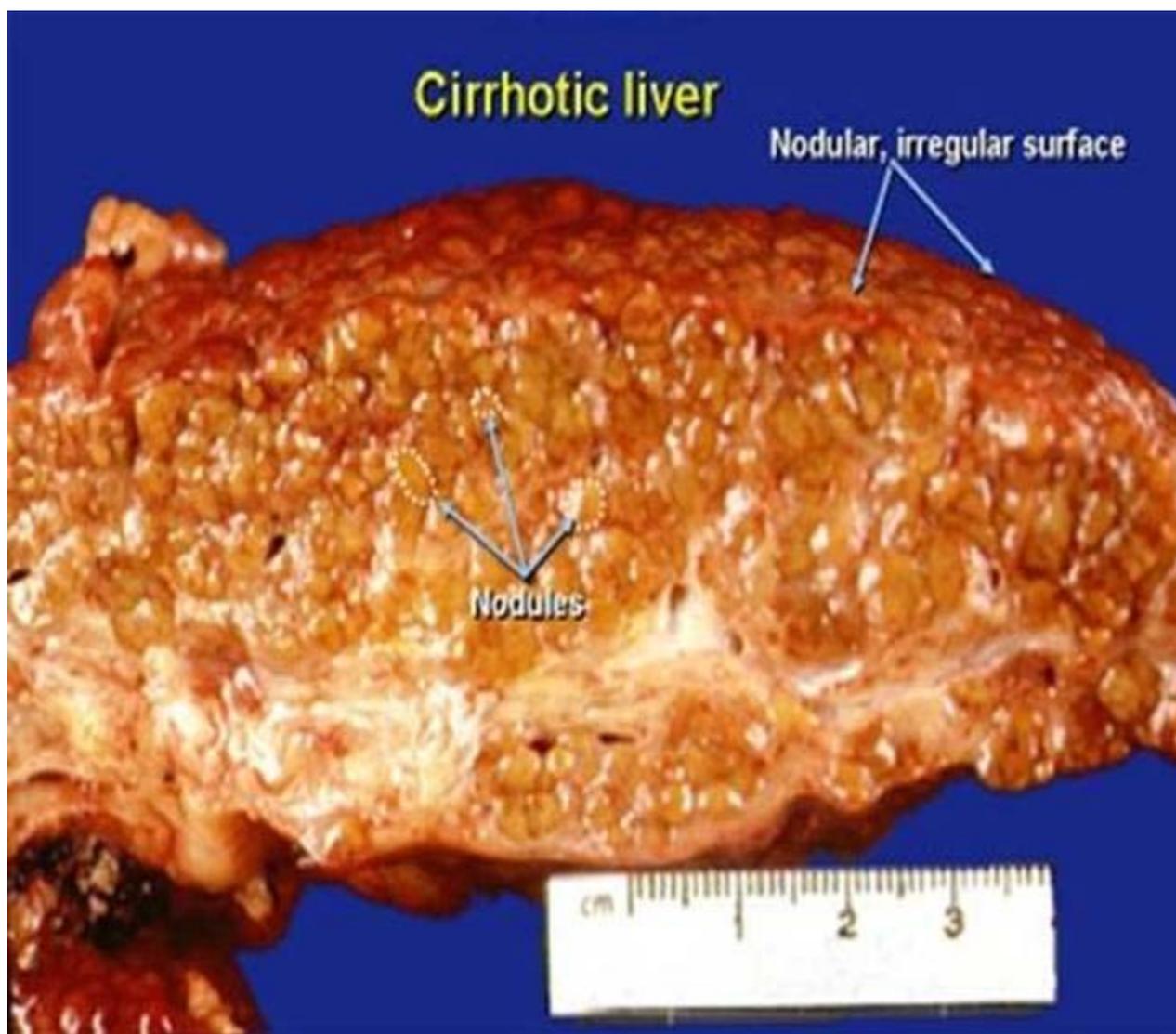
The liver at cirrhosis is dense macroscopically, knotted, diminished in size, rarely is increased. Depending on the size and character of knots, the macroscopic forms of cirrhosis are distinguished: small-nodular, large-nodular, mixed.

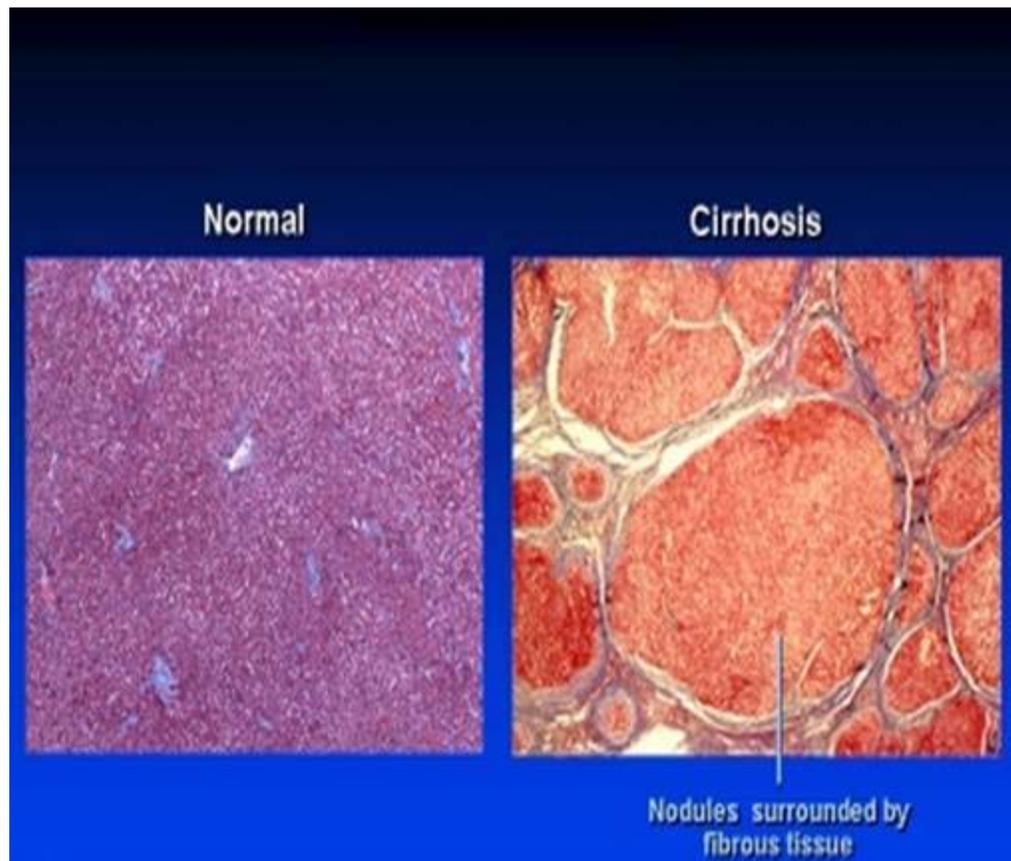
Postnecrosis cirrhosis — large-nodular or mixed; portal — small-nodular; initially bilious — small-nodular or with a smooth surface; second time bilious — small-nodular. At the first two types of cirrhosis the surface of cut is of red colour, at bilious — grey-green.

The histological signs of cirrhosis are: dystrophy and necrosis of hepatocytes, disfigured regeneration, diffuse sclerosis, structural alteration and deformation of organ.









Classification

According to Havana classification (1956), cirrrosises are divided into:

- 1) portal;
- 2) bilious;
- 3) postnecrosis;
- 4) mixed.

According to the stages of development:

- 1) initial;
- 2) formed cirrhosis;
- 3) dystrophic.

According to the degree of weight:

- 1) easy;
- 2) middle;
- 3) heavy.

According to passing:

- 1) progressive;
- 2) stable;
- 3) regressing.

Symptoms and clinical passing

Pain syndrome. It is characterized by permanent aching, sometimes intensive, pain in right hypochondrium with an irradiation in the right shoulder-blade and back.

To the dyspepsia syndrome is characterized by loss of appetite, nausea, vomiting and weight loss.

Syndrome of cholestasis. The colour of skin of patients with this type of pathology can be from insignificantly yellow to earthily-grey. The presence of icterus specifies the activity of inflammatory process in the liver.

In cases of asthenovegetative syndrome, as a result of progress of encephalopathy, the inversion of sleep comes (somnia in the day-time and insomnia at night). General weakness, rapid fatigueability, irritates and headache are characteristic.

Syndrome of endocrine violations. Patients with this syndrome are characterized by the gynecomastia and decline of potency for men, for women — disorders of menstrual cycle.

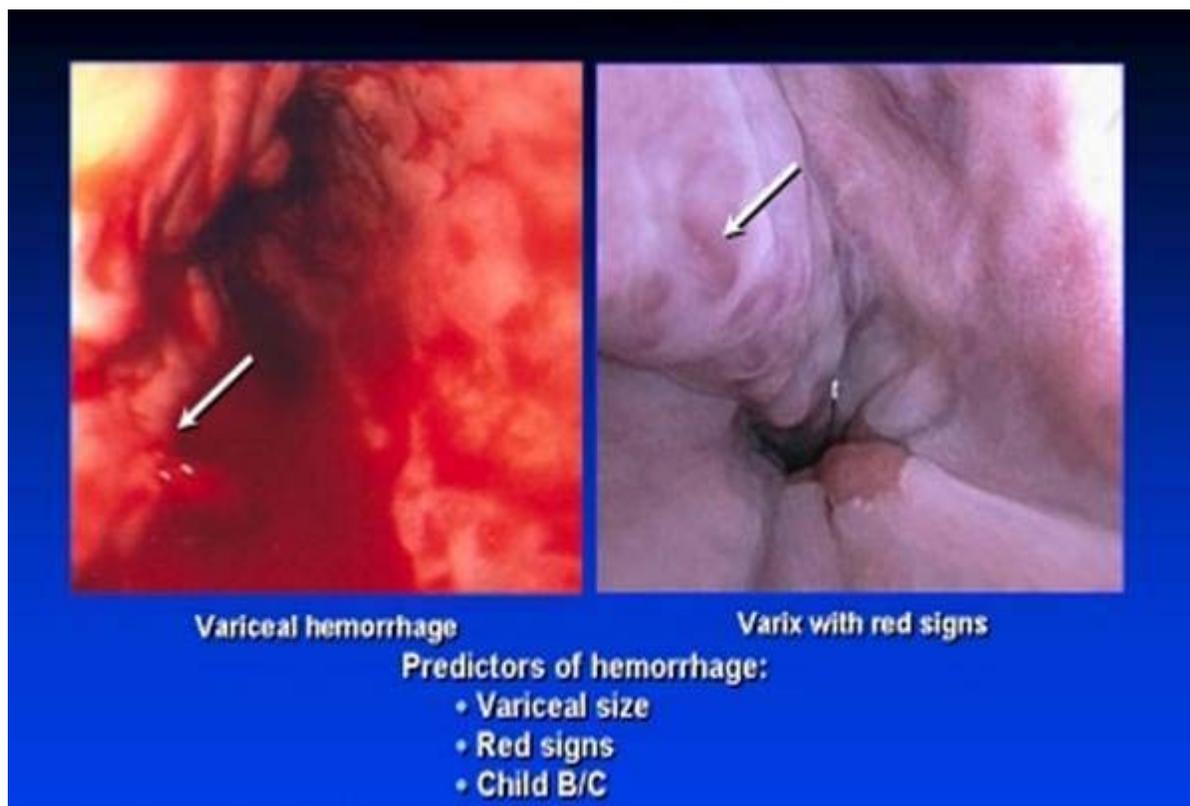
Syndrome of portal hypertension. Expansion of veins of oesophagus stomach, haemorrhoid veins, veins of front abdominal wall, splenomegalia, hypersplenism (anaemia, leucopenia, thrombocytopenia) and ascites are considered basic signs.

Objective data: at the observation patients display the icteric of skin and mucus, vascular “asterisks” on face, neck, brushes, breasts, “hepatic hands” (rose marble of skin), increased in sizes, as a result of ascites, stomach (“frog stomach”), varicose expansion of veins of abdominal wall (“head of jelly-fish”).

During palpation of abdomen the liver is increased, dense, with acute edge and fine-grained surface. At the atrophy cirrhosis it is impossible to palpate it. Spleen is increased, ascites is marked.

Laboratory information. Anaemia, leucopenia, thrombocytopenia, hypoproteinemia, relative hyperglobulinemia and hypoglobulinemia are characteristic signs of cirrhosis. The increase of level of alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, positive reaction of Takata-Ara; change of coagulate ribbon to the right (the Veltmann test), hypoprothrombinemia and increase of immunoglobulin A, C are observed also.

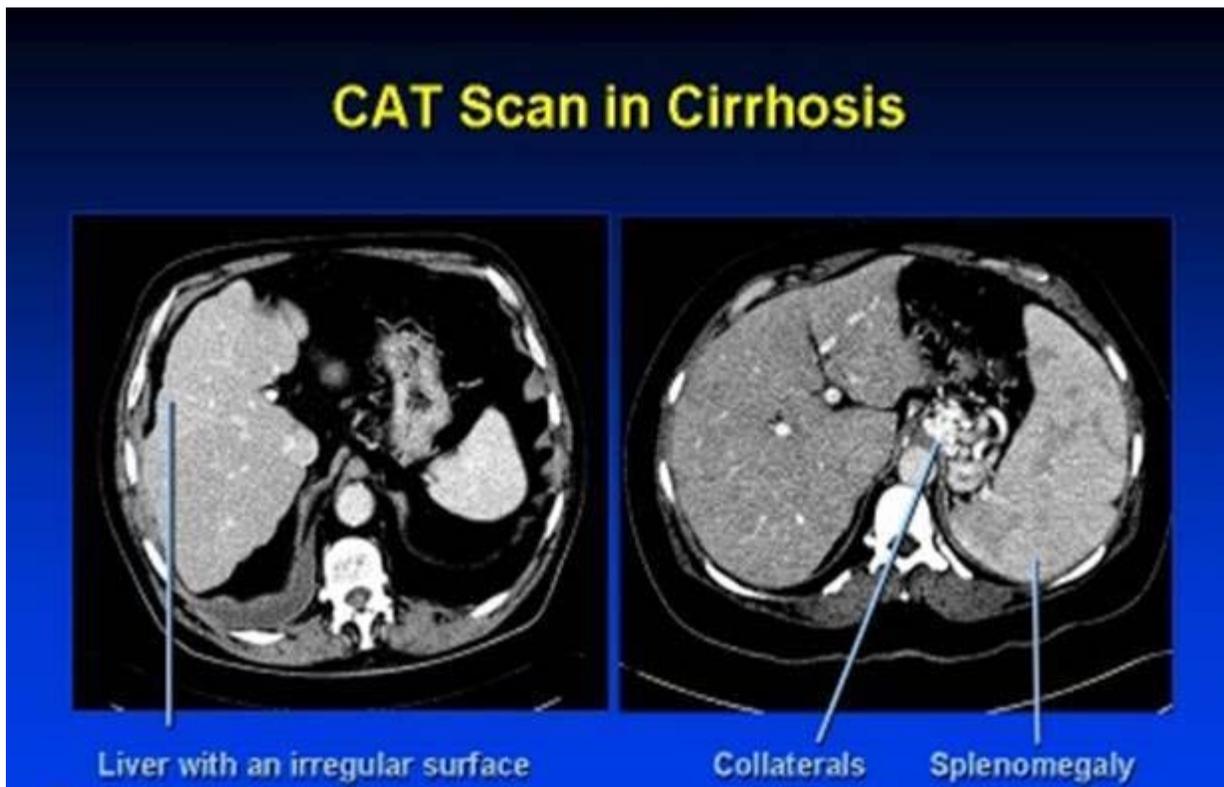
By endoscopy it is possible to expose varicose expansion of veins of oesophagus and cardial part of stomach.



Under roentgenoscopy, roentgenography of oesophagus and stomach the characteristic picture of varicose expansion of veins of oesophagus is observed.

Sonography enables to estimate sizes, structure of liver and spleens, to visualise a portal vein and its influxes, and also to expose ascites.

Computer tomography more precisely than sonography, allows to investigate the changes in the liver and neighbouring organs.



Hepatoscintigraphy gives information about position, sizes, degree of accumulation to nuclide in the liver and spleen.

Intravenous radiohepatography (albumen marked I131) can characterize arterial and portal bloodflow in liver and spleen.

Reohepatography enables to find out the state of bloodflow of liver.

Splenoportography visualises the vessels of the portal system, exposing thrombosis or considerable expansion of portal vein and its influxes, poverty of hepatic vascular picture (cirrhosis of liver). During this manipulation portal pressure which in such patients usually is higher 250 mm (2,45 kPa) of waters is measured.

By *celiacography and mesentericography* expansion and sinuosity of splenic artery and poverty of hepatic vascular picture are exposed. Expansion of splenic and gate veins is observed in the vein phase.

Application of laparoscopy can give valuable information about the state of liver, spleens and presence of ascites. Clinical passing of cirrhosis of liver depends on etiologic factors, activity of process, degree of functional insufficiency of liver, presence of complications and accompanying diseases.

Variants of clinical passing and complication

At *slow progressive passing* the symptoms of disease periodically either increase or become less expressed. Characteristic are the damage of parenchyma of liver and protracted

passing.

Together with that, quick progressive passing is a transitional form from subacute atrophy of liver to the cirrhosis. Death of patients at such passing of pathology, as a rule, comes as a result of hepatic insufficiency during a few months from the beginning of disease.

Most characteristic for the alcoholic cirrhosis of liver after the absolute exception of reception alcohol and adjusting of valuable nutrition is *regressive passing*.

Protracted periods of remission which come on the early stages of cirrhosis after adequate conservative treatment are inherent to *stable* character of passing.

Complications: bleeding from the varicose extended veins of esophagus and stomach, haemorrhoidal veins, choleic bleeding, thrombosis of portal vein, hepatogenic gastric ulcers, hepatic insufficiency, hepatic comma, transition of cirrhosis in cancer and encephalopathy.

Complication of portal hypertension by bleeding from the varicose extended veins of esophagus carries the instant danger for life of patient, if portal pressure grows over 250–300 mm of waters (2,45–2,94 kPa). Lethality of patients after the first bleeding makes 25 %, after the second — about 50 % and after the third — over 75 %. Such complications can arise both during complete rest and after the physical loading. Bleeding, as a rule, is profuse, and is accompanied by vomiting by “coffee-grounds” and grume of fresh blood. Thus patients often have collapse with the acute falling of arterial pressure and eclipse of consciousness. The frequent liquid emptying of black can also take place (melena).

Diagnosis program

1. Anamnesis and physical examination.
2. General analysis of blood and urine.
3. Biochemical blood test (albuminous factions, bilirubin, glucose, AlAT, ASAT, alkaline phosphatase, cholesterol, reaction of Takata-ara, the Veltmann test; electrophoresis albumens).
4. Coagulogram.
5. Endoscopy.
6. Contrasting sciagraphy of oesophagus, stomach.
7. Sonography.
8. Scanning of livers.
9. Splenoportography.
10. Laparoscopy.

Differential diagnostics

Insufficiency of circulation of blood after the right ventricle type can be accompanied by an increased painfulness of liver. It can be the reason of such symptomatic complex as rheumatism, atherosclerotic cardiosclerosis, innate lacks of heart and chronic pulmonary heart. However, for this pathology edemata on lower extremities which arises long before development of ascites is characteristic. It is needed to remember, that ascites well responds to

treatment by cardiac glycoside and diuretic. And yet, except for anasarca, for insufficiency of circulation of blood the shortness of breath is characteristic, acrocyanosis and the stagnant phenomena in lungs, changes of ECG. The icterus in such patients is very rarely, and the functional tests of liver change insignificantly.

Pericarditic pseudocirrhosis. This pathology develops at squeezing pericarditis. Except for development of ascites, that can be observed, for this pathology symptoms of constructional pericarditis are characteristic also: increase of vein pressure over 200 mm of waters. item (1,96 kPa), paradoxical pulse (disappearance during inhalation) and decline of amplitude of pulsation of heart on roentgenokymography. Roentgenologicly, laying of salts of calcium in a pericardium, “little” and “quiet” heart are found in patients. For this disease characteristic are the typical changes at ECG and phonocardiography.

Tactics and choice of treatment method

Conservative treatment. Patients with non-active compensated or minimum activity by cirrhosis, as a rule, do not need special medicinal treatment. Mainly the shown base therapy (principle of maximal medicinal and functional spare of liver, providing of the optimum dietary mode and employment, dynamic clinical supervision with correction of the mode and setting of vitamins, enzymes and other preparations) is applicable here. In relation to patients with subcompensated non-active or minimum activity by cirrhosis of liver, they are expedient to apply the medicinal course of treatment: daily intravenous in drops inflowing of Neohemodesis (200 ml) or solution of glucose (5 % — 200–300 ml), use of Esenciale (2 capsules three times on a day) or Legalon (Karsil) (2–3 drops three times on a day). 2 courses of the noted treatment are conducted for a year. At hypoalbuminemia it is rationally to connect anabolic steroid — Retabolil (50 mg 1 time per two weeks intramuscularly, course — 4–5 injections) also, transfusion 2–3 times per a week of solution of the albumen 10–20 % (10–20 g) or plasma (100–200 ml).

Patients with the active phase of cirrhosis, need to be hospitalised when they have expressed portal hypertension and presence of ascites, bed rest, limitation in day's feed of salt to 5 g and to conduct correction of water-electrolyte exchange are appointed to them.

It is possible also to recommend potassium-sparing diuretic: Veroshpiron (0,025 g 3–4 times per a day) or Triamteren (0,3 g 2 times per a day), Uregid or Oxodoline (0,05–0,1 g 1 time per 2–3 days).

At presence of ascites the concentrated plasma (125–150 ml 1 time per a week, course — 4–5 inflowing) and 20 % solution of albumen (100 ml 1 time per a week, course — 8–10 inflowing) is applied.

Abdominal paracentesis and deleting of liquid is conducted in case of proof ascites, that does not respond to adequate treatment, and also at hepatorenal syndrome or threat of the oesophagus bleeding.

At hepatic comma:

1. Through subclavian catheter the inwardly-vein introduction 5 % solution of glucose (1000–1500 ml on days) with cocarboxylase (300 mg) is put, by the vitamin B12 (500 mcg), by

lipoic acid (180 mg) — 20–30 drops for a minute.

2. Prednisolone (150 mg intravenously, by stream and then in each 4 hours for 90 mg).
3. Arginine (25 g intravenously, in drop, and then in that dose in every 8 hours), 20–25 g Ornacetil intravenously for the day long.
4. Kanamicine (0,5 g) or Polymixine of M (150 000 ODES) or ampicillin (1,0 g) — each 4 hours through a stomach-pump.
5. Washing of intestine is 1 time per days.
6. Exchange blood transfusions — daily, during 7–10 days.
7. Introduction of oxygen through a nasal catheter (2–4 л/min).
8. At development of metabolic acidosis — 200–600 ml 4 % solution of hydrogen carbonate of sodium.
9. In case of expressed metabolic alkalosis there are preparations to potassium (to 10 g on days).

All adopted measures are continued to taking the patient from comma.

It is thus needed to mark that from data of practice conservative treatment of patients with the cirrhosis of liver reverse development of cirrhosis or prevent progress of disease can not be provided. Without regard to it, medicinal therapy, however, can improve the general condition of patient.

However, in all patients with the disease of liver it is needed to look for possibility of transplantation of this organ, and by an indication it is needed to count appearance of hepatargia.

Surgical treatment. Operative treatments are divided into two groups:

- 1) radical, that can liquidate portal stagnation;
- 2) palliative, which are able only to level the separate signs of this pathology.

Contra-indication to operation: 1) decline of albumens of blood to the level of 2,5 % and below; 2) falling of albumin-globulin coefficient less than 0,7; 3) diminishment of prothrombin index below 30 %; 4) presence of symptoms which specify active destructively-inflammatory process in liver (hectic temperature of body, leukocytosis, acute increase of level of transaminase and globulin faction and positive reaction on a C- reactive albumen).

Radical operations

Among them most widespread are:

1. Direct portacaval shunt: a) latero-lateral; b) termino-lateral. (Fig. 1) Bleeding is considered an indication for application of this operative treatment from the varicose veins of esophagus (in anamnesis) on soil of intrahepatic block.

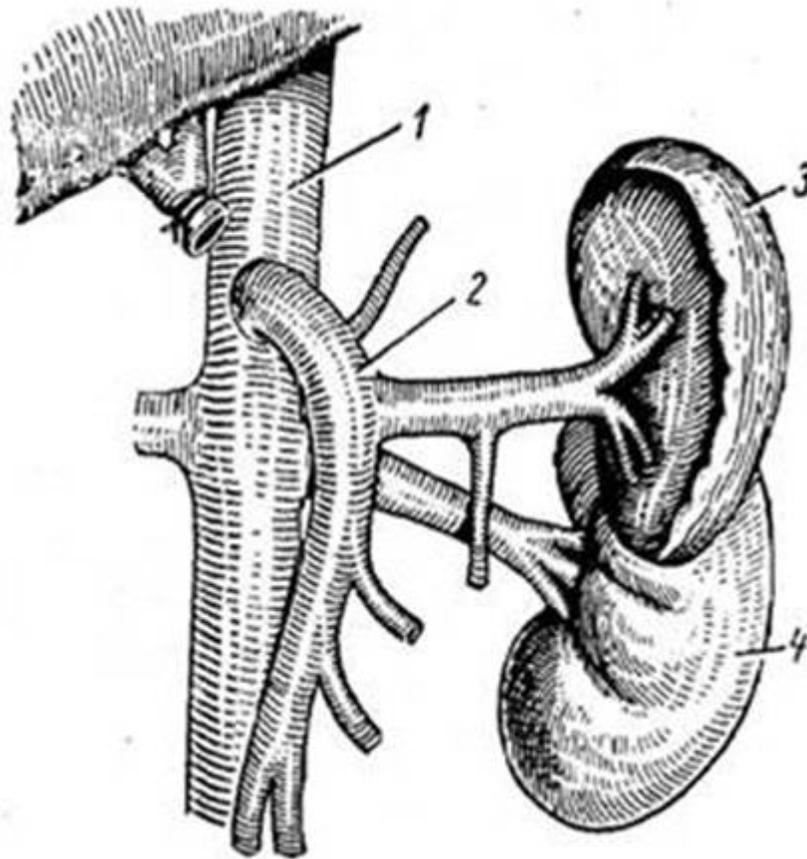


Fig. 1. Direct termino-lateral portocaval anastomosis

1-inferior vena cava

2-portal vein

3- spleen

4- left kidney

2. Splenorenal anastomosis: a) termino-lateral;(Fig.2) b) termino-terminal; c) H-shaped (by autovein or alloprosthesis). An indication is bleeding from the varicose veins of esophagus (in anamnesis) on soil of intrahepatic and suprahepatic blocks.

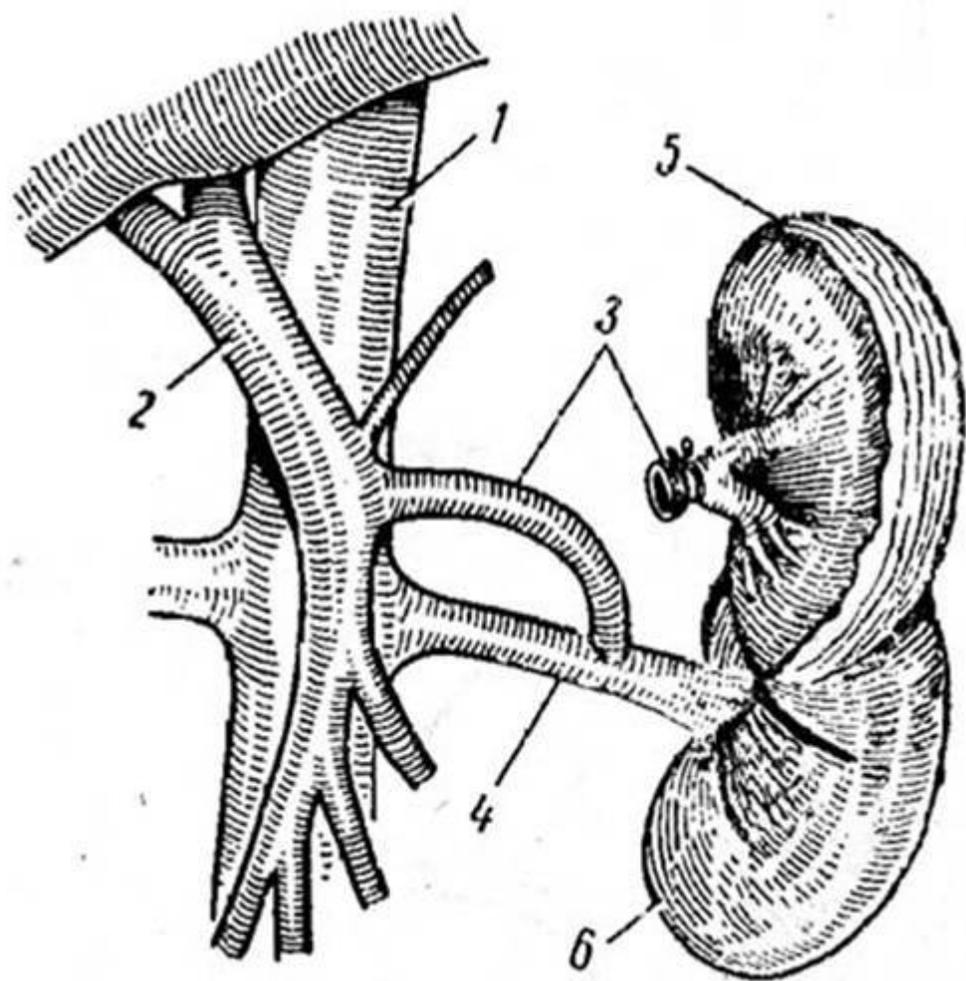


Fig.2 Termino-lateral splenorenal anastomosis

- 1-inferior vena cava
- 2-portal vein
- 3- splenic vein
- 4- left kidney vein
- 5- spleen
- 6- left kidney

3. Mesentericocaval anastomosis.(Fig.3) An indication is proof ascites at the relatively ungrave condition of patient.

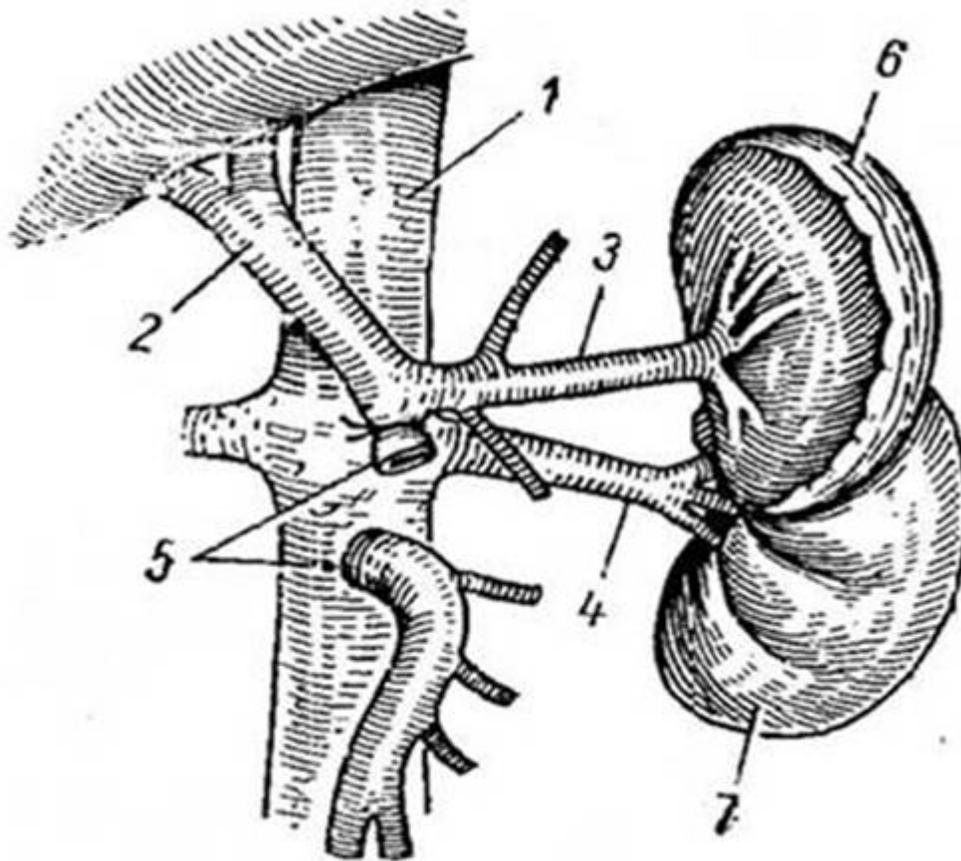


Fig.3 Mesentericocaval anastomosis

1-inferior vena cava

2-portal vein

3- splenic vein

4- left kidney vein

5-superior mesenteric vein

6- spleen

7- left kidney

4. Anastomosis of shallow veins is ineffective and is not presently used.

Palliative operations

Among them the widespread are used:

1. Operations directed on stimulation of regeneration of liver: a) partial (regional) resection of liver; b) electro-coagulation of surface of liver; c) cryogenic and laser destructions of surface of liver; d) periarterial neurectomy of general hepatic artery; e) bandaging of splenic artery and splenectomy; f) endovascular embolization of splenic artery.

2. Organoanastomosis:

a) omentopexy;

b) omentodiaphragmopexy;

c) omentohepatopexy.

3. Operations at ascites:

- a) laparocentesis; [VIDEO](#)
- b) taking of ascites liquid in preperitoneal, retroperitoneal and hypodermic cellulose, in urethra;
- c) lymph-venous anastomosis (between a pectoral lymphatic duct and internal or external jugular vein);
- d) peritoneovenous shunting by the synthetic tube with a valvular device, taken in an abdominal wall;
- e) extraperitonization of the livers (formation of vascular communications between a liver and diaphragm).

Tactics at bleeding from the varicose veins of esophagus

Treatment of patients at bleeding from the varicose veins of esophagus needs to be begun with the tamponade internal surface of esophagus and cardial part of stomach by the special double-balloon Sengstaken-Blakemore tube. Some other conservative measures directed on the stop of bleeding without the use of this probe are considered ineffective and tactically wrong.

The Sengstaken-Blakemore tube has three ducts, two of which are connected with rubber bulbs, one — with the cavity of stomach. (Fig.4)

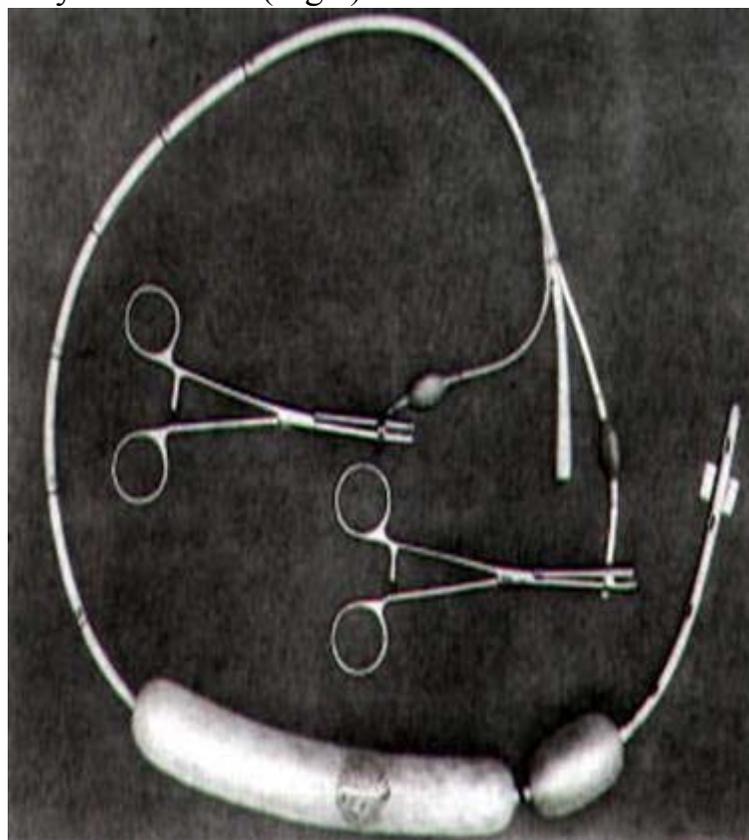


Fig.4 Sengstaken-Blakemore tube

Before application of probe by introduction of air the volume of bulbs is measured. Probe through a nose and in a distal (gastric) bulb is inserted, the necessary amount of air is forced (about 150,0 ml). After this by drawing out of it a round (distal) gastric bulb is pinned outside against cardia. Farther prolonged esophagus bulb is inflated to appearance at the patients feeling of arching (volume about 120,0 ml) and it is obturated. Then to the proximal end of

probe through the block, the load is suspended weighing about 1 kg. It warns reverse advancement of probe in stomach and by this provides stability of compression of the varicose extended veins. The control after hemostasis is carried out through the third, connected with stomach, duct of probe. By such method it can be succeeded to attain to hemostasis in 80–90 % cases. The probe in such position is held 2–3 days. After this decompression of repeated bleeding can come almost in half of patients. Therefore deleting the probe is not needed. Taking this into account, it is expedient to carry out decompression of bulbs in the light intervals of days and inflate bulbs at night, when the control after the possible bleeding is complicated.

Conservative treatment is reasonable: 1) at the easy degree of loss of blood and I degree of hepatic insufficiency (basic biochemical indexes either are not changed or with insignificant deviations from a norm; ascites and encephalopathy are absent);

2) at the III degree of hepatic insufficiency, progressive ascites and encephalopathy, regardless of degree of loss of blood.

Conservative therapy of bleeding from the varicose veins of esophagus must engulf the whole volume of medical measures, as at similar pathology of ulcerous genesis (hemostatic therapy, antacid, H₂- blocker histamine receptors).

For the decline of portal pressure pituitrin is entered. The endoscopic methods of stop of bleeding are applied also (imposition of clips on veins, sclerosis therapy — 76 % ethyl alcohol, Varicocide, 66 % solution of glucose, endovascular occlusion of veins, laser coagulation of veins). It is needed to count setting of preparations for stimulation of regeneration of liver (esenciale, lif-52 and others like that), application of disintoxication therapy.

Surgical treatment is considered applicable at bleeding of middle and heavy degrees with the I and the II degrees of hepatic insufficiency (general bilirubin not more large 50 $\mu\text{mol/L}$, general albumen not more small 60 g/L , prothrombin index not more low 60 %, present transient ascites) in the cases when the valuable conservative treatment directed on the stop of bleeding is not effective during 24–48 hours.

Types of operations

I. Operations directed to disconnection of gastroesophageal vein way.

1. The Taner's operation:

a) transversal cutting and sewing together of stomach in subcardial part;

b) cutting with the next sewing together of esophagus in supradiaphragmatic segment.

2. Operation by O.O. Shalimov is cutting and sewing together of esophagus by the device for circular anastomosis.

3. Sewing of cardioesophageal transition circular by U-shaped stitches after V.M.

Korotkyy.

4. Operation developed by M.D. Paciora, is sewing and bandaging by separate ligatures of bleeding veins of cardial part of stomach and esophagus from the side of mucus shell.

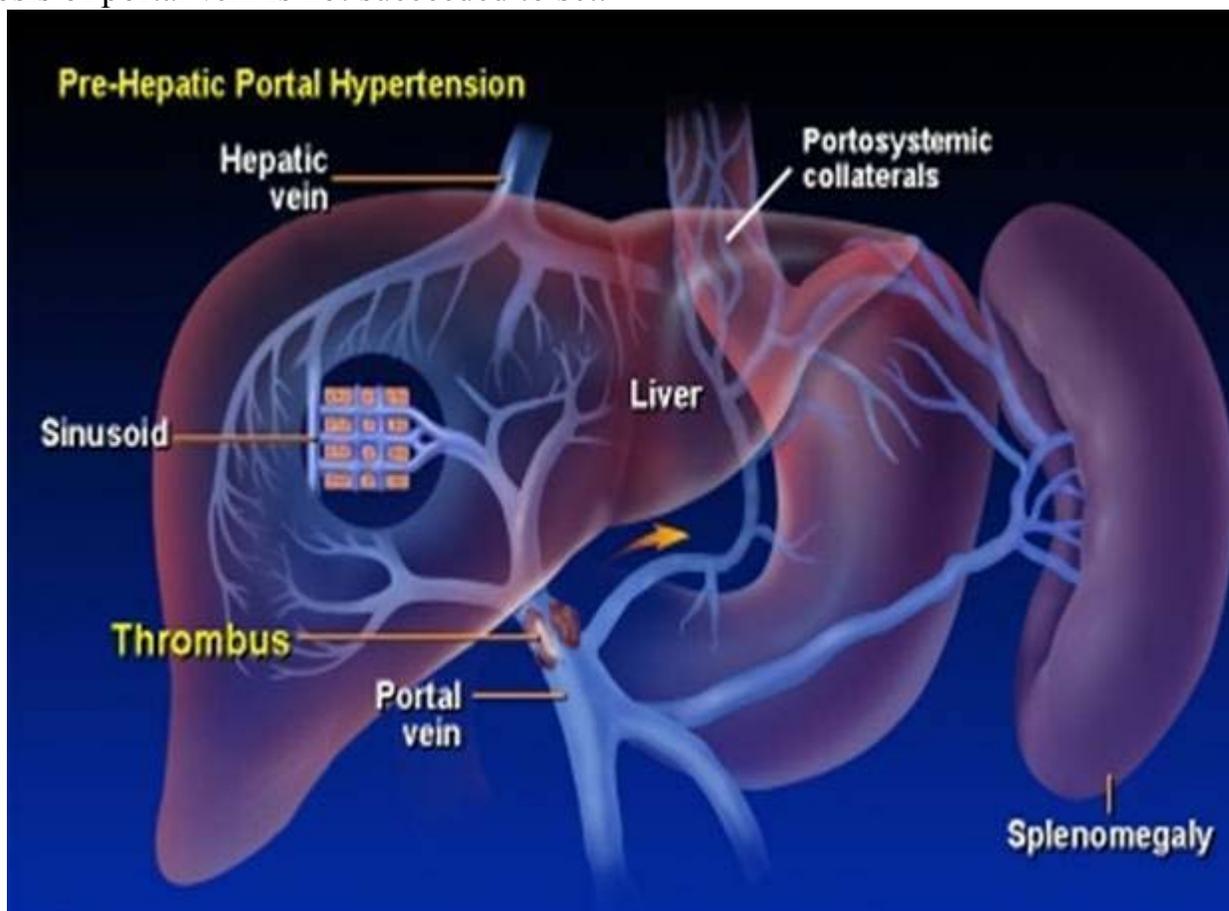
5. Resection of distal part of esophagus and proximal part of stomach.

II. Operations which reduce portal pressure: portocaval anastomosis (direct and selective), splenectomy.

Thrombosis of portal vein. Subhepatic portal hypertension

Etiology and pathogenesis

In children the basic etiologic factor which predetermines the thrombosis of portal vein, is infectious aggression. Inflammatory processes of abdominal cavity (acute appendicitis, cholecystitis traumas), the getting wet belly-button of new-born are those principal reasons which predetermine the origin of pylephlebitis and thrombopylephlebitis. In adults the most frequent reasons of thrombosis of portal vein are the cirrhosis of liver and hepatoma. Similar pathology can develop after splenectomy or operations on bilious ways. The reason of thrombosis can be the cardiovascular anomalies of development, in particular, innate defect of portal vein. However, it is needed to mark that in the half of patients reason of origin of thrombosis of portal vein is not succeeded to set.



Symptoms and clinical passing

In patients with portal hypertension, that arose on soil of thrombosis of portal vein, acute symptoms, as a rule, are absent. However, it is possible to observe the dyspeptic phenomena in them: nausea, bad appetite, flatulence. Moderate pain is felt in right hypochondrium, that is multiplied after acceptance of meal.

At this pathology appearance of early ascites which mainly carries temporal, transitional character is characteristic, sometimes it takes out chronic shape. On more late stages hypersplenism can develop and bleeding arise from the varicose extended veins of esophagus and cardia.

The general condition of patients here can remain satisfactory, the relapses of bleeding from the varicose extended veins of esophagus and cardia are seldom. If they arise, they are carried by patients relatively well. It can account for favourable passing of portal hypertension by development of collateral circulation of blood. Together with that, productively unfavourable passing of disease is observed on the background development of chronic ascites and portocaval encephalopathy.

The diagnosis is usually confirmed by sonography and splenoportography. Valuable information about such patients can be given by computer tomography on the background rapid introduction of contrasting matter to the portal system (computerized axial tomography).

Tactics and choice of treatment method

Effective preparations of conservative treatment of portal hypertension caused by the thrombosis of portal vein for modern medicine so far are unknown. However, as bleeding from the varicose extended veins of esophagus and cardia seldom are profuse and it is comparatively well added to conservative therapy, it is expedient to apply, even on an early stage, unoperative methods of treatment. Moreover, possibilities of surgical treatment at this pathology are limited. In fact in this situation technically adequate portocaval decompression is difficult to execute in connection with the thrombosis of basic branches of the portal system. However, in default of effect from conservative therapy in patients with complication by much bleeding, dissociative operations are to be applied to the type of Taner's or sewing of vessels on verge of esophagus and cardial part of stomach. Together with that, it is needed to mark that this operative treatment gives only a temporal effect.

Thrombosis of hepatic veins (the Budd-Chiari disease).

Suprahepatic portal hypertension

Etiology and pathogenesis

In most patients etiology of disease is unknown. Application of oral contraceptives, and also presence of intrahepatic and anhepatic tumours, is instrumental in the origin of pathology. The process can be accompanied by the thrombosis of lower hollow vein (the Budd-Chiari syndrome). This syndrome can arise also at thrombosis or stenosis of lower hollow vein at the level of hepatic veins. Portal hypertension, usually, develops early, and the cirrhosis of liver is formed after some time.

Symptoms and clinical passing

For the clinic of disease, as a rule, stormy passing is peculiar. Thus patients feel great pain in stomach, liver is increased, painful. Increasing ascites is often observed. As experience shows, development and expression of symptoms of disease depend on the depth of damage by the thrombosis process of hepatic veins and joining of thrombosis of lower hollow vein.

The disease is also accompanied by development of polycythemia, which is the second and conditioned, from one side, by concentration of blood, and from other — investigation of increased products of erythropoietin, that is selected by a regenerating liver.

At endoscopy the varicose extended veins of esophagus and cardial part of stomach are exposed.

In some patients it is possible to observe acute passing of the Budd-Chiari syndrome, that usually has bad prognosis and is often closed lethally. Portal hypertension in these patients, as a rule, develops early and has the expressed character, sometimes arriving at maximal numbers (390–400 mm of waters). Bleeding which arise on this background carries difficult profuse character.

Tactics and choice of treatment method

Conservative therapy in such cases is ineffective. At acute passing of disease lethal end can be warned only by forming of direct or selective portocaval anastomosis. The protracted (chronic) passing of disease is also accompanied by early and expressed portal hypertension, that can be complicated by bleeding from the varicose extended veins of esophagus. In such cases, as well as at acute forms, although not so quickly, the operative treatment directed on decompression of the portal system is possibly conducted.

LIVER TRANSPLANTATION

History

Canine liver grafts were shown to function after transplantation to the pelvis by Welch in 1955. Orthotopic liver transplantation in dogs was attempted by Cannon in 1956 and performed successfully by Moore in 1959. The first attempt at liver allotransplantation in man was made

by Starzl at the University of Colorado on March 1, 1963. The 3-year-old recipient with extrahepatic biliary atresia died of hemorrhage on the day of transplantation. Ensuing attempts in Denver, Boston, and Paris were unsuccessful until 1967, when the first extended survival of a human liver allograft recipient was achieved by Starzl. The addition of cyclosporine immunosuppression by Calne in 1978 and then combination therapy with cyclosporine and prednisone by Starzl in 1980, as well as better liver preservation and surgical techniques, improved the prospects for clinical liver transplantation

INDICATIONS FOR TRANSPLANTATION

The specific indications for liver transplantation have become more standardized owing to the better knowledge of the natural history of a number of liver diseases and concomitant improvement in short- and long-term results of orthotopic liver transplantation (OLT). As with most aspects of this topic, however, significant controversy remains about several disease states leading to end-stage liver failure.

Cholestatic Liver Diseases. Cholestatic diseases, including primary biliary cirrhosis (PBC), secondary biliary cirrhosis, and primary sclerosing cholangitis (PSC), are the diseases most successfully treated by liver transplantation. Operative survival is greater than 90%, and 5-year survival is approximately 80%. This rate is due mostly to the slow onset of illness, which allows for preoperative preparation of the recipient and appropriate timing of the procedure, as well as absence of recurrence of the disease in the graft. As a result of this success, cholestatic disease is one of the most frequent indications for transplantation, accounting for 22% of all transplants performed in the United States. Clearly, the timing of operation is critical, and a substantial literature has addressed the rate of disease progression.

The indications for transplantation in patients with PSC are based on refractory symptoms resulting from recurrent cholangitis or impaired synthetic function. The risk of cholangiocarcinoma, approximately 15% in these patients, is an additional relative indication for OLT. A current trend toward earlier intervention with liver transplantation in PSC is underway, with retrospective comparisons of transplant versus nontransplant therapy weighing heavily in favor of the transplant approach. Clearly, however, some patients, particularly those with predominantly extrahepatic biliary strictures do well with conventional bypass or endoscopic procedures. Thus, timing remains a matter of clinical judgment, with most patients benefiting from allotransplantation.

Several models of survival have been established to predict outcome in patients with PBC. All prognostic scores are based in part on the rise in serum bilirubin. In general, transplantation is recommended when the bilirubin level exceeds 15 mg./100 ml., although earlier transplantation for severe associated symptoms such as pruritus or fatigue, refractory ascites, or variceal bleeding is appropriate. Although close monitoring of asymptomatic patients is reasonable, it is important not to delay intervention until the clinical status of the patient deteriorates, because OLT is the only definitive treatment.

Alcoholic Liver Disease. Nineteen percent of all liver transplants performed in the United States have been for alcoholic cirrhosis. Although the procedure was originally avoided

because of fears of high postoperative recidivism and a tendency toward medical noncompliance, the national trend has been to transplant in this setting more frequently, and in 1993 it was the most common indication reported by the United Network for Organ Sharing (UNOS) registry. Experience has demonstrated that graft and patient survival are not significantly different from those of other favorable indications and that disease recurrence (return to heavy alcohol use) is approximately 12%. Given these results, combined with the extraordinary prevalence of alcohol-related liver disease compared with other transplantable diseases (36,000 deaths per year), transplantation for alcoholic cirrhosis could easily exhaust the already scarce supply of donor organs. Selection is clearly needed to exclude comorbidities, alcohol-associated organ failure such as cardiomyopathy, and patients with continued dependence on alcohol. Even so, the need far outpaces the supply. Considerable philosophical debate addresses the use of scarce resources for individuals with self-inflicted disease. At this point, the decision to transplant should be based on a thorough preoperative medical evaluation supplemented by involvement in alcoholic rehabilitation and abstinence for at least 6 months. Patients presenting *too sick to drink* must be evaluated individually with an estimate of recidivism made to optimize long-term results. Palliative procedures are appropriate when possible. The recent development of the transjugular intrahepatic portosystemic shunt (TIPS) procedure has allowed many of these patients to be stabilized without extensive surgical intervention, for more thorough evaluation in a nonemergent setting.

Hepatitis C and Cryptogenic Cirrhosis. Another rapidly increasing indication for OLT is hepatitis caused by the hepatitis C virus (HCV). This is frequently discussed in tandem with cryptogenic cirrhosis. Transplantation is pursued for symptomatic infection only. Since the discovery of reliable methods for detecting HCV, this pathogen has been identified as the etiologic agent for liver failure in an increasing number of recipients. In 1996, it was associated with more than 25% of transplants in most centers. Cryptogenic disease accounted for an additional 12%. Excellent short-term results have been achieved with a 3-year survival of 70% (63% nonretransplant survival). These results have been tempered, however, by reinfection at almost 90%, with active recurrent hepatitis at 50%. Recurrent hepatitis has led to few deaths, although a fourth of the reinfected patients have converted to a chronic state. More deaths from recurrent disease can be expected in this group, and longer follow-up is required to determine whether results will remain acceptable. Disease recurrence has also been seen in cryptogenic cases, again suggesting a viral infection. Several treatments have been studied for HCV infection, with the most promising being parenteral therapy with interferon alfa.

Metabolic Diseases. Several metabolic diseases have been successfully cured with OLT, including alpha₁-antitrypsin deficiency, Wilson's disease, hemochromatosis, Crigler-Najjar syndrome, tyrosinemia, primary hyperoxaluria, and familial homozygous hypercholesterolemia. Survival is uniformly excellent. The recipient hepatectomy is usually simple, and the preoperative state of the patient is stable. Timing of transplantation should be such that secondary effects of the disease are not extensive.

Fulminant Hepatic Failure. In the absence of pre-existing liver disease, rapid loss of hepatic function culminating in jaundice and coma is termed *fulminant hepatic failure* (FHF). The etiologic agent can vary. The time course relates inversely to the prognosis, with those

progressing to coma in less than 2 weeks having a 36% survival and those progressing in 2 to 8 weeks having a 7% survival. Despite the dramatic rate of progression of disease, these patients have very acceptable results from OLT (about 60% 5-year survival). Predictably, survival after OLT improves with improved health of the patient at the time of transplant. Survival for all etiologies of FHF has doubled in the past decade, primarily due to great strides in supportive intensive care. Unlike chronic viral diseases, recurrence of disease for viral causes of FHF is unusual.

Care of patients with FHF is complex, and OLT should be considered early. Thus, prompt transfer to a liver transplantation center is critical. In patients with rapid deterioration, supportive care (e.g., hyperventilation, diuresis) should be supplemented with monitoring of intracranial pressure (ICP) for best results. Subdural ICP monitors have obviated the need for intraventricular devices and have thus reduced the bleeding risks of this approach. Perfusion pressures (mean arterial pressure minus ICP) of less than 40 mm. Hg, especially when combined with pupillary fixation, suggest that irreversible brain injury has occurred, and OLT should be avoided. Patient decompensation can be rapid and unpredictable. In the absence of a suitable donor, several aggressive maneuvers can successfully delay the onset of brain death from intracranial pressure. These include hepatectomy with end-to-side portacaval shunt, xenogeneic *ex vivo* perfusion, and bioartificial hepatic support (see later). Donor criteria may be liberalized to achieve timely hepatic replacement, and ABO incompatibility is acceptable.

Chronic Hepatitis B. Transplantation for chronic hepatitis B virus (HBV) cirrhosis remains a controversial topic. The discouraging reinfection rate of over 80% of recipients associated with a high rate of clinical hepatitis recurrence (60% at 1 year) and high related mortality (30% at 1 year and 52% at 5 years) raises questions about the procedure. Although generally contraindicated, transplantation may be appropriate in certain settings. Recent intense investigation in this area has identified several factors affecting reinfection. In general, the state of viral replication at the time of transplantation is critical in establishing reinfection. Identification of active viral replication (serum HBV DNA and HBeAg detection) at the time of transplantation clearly worsens the prognosis. Immunoprophylaxis with anti-HBV antibodies improves outcome for replication-negative patients, but long-term results for patients so treated remain to be seen. In general, OLT in the presence of HBV infection should be reserved for patients enrolled in specific viral prophylaxis protocols.

Intrahepatic Malignancy. Predictably, transplantation for primary and metastatic cancer has been associated with a high recurrence of tumor and a poor 5-year survival. Thus, there is little indication for OLT in a patient with known malignancy. Liver transplantation is no longer an accepted therapy for hepatic metastasis except perhaps in some patients with rare neuroendocrine tumors. Today, liver transplantation is usually limited to patients with nonresectable hepatoma fulfilling the following criteria: asymptomatic hepatoma (i.e., not associated with recent weight loss, ascites, or constitutional symptoms); tumor less than 5 cm. in diameter; and fewer than or equal to three intrahepatic tumors. If feasible, liver resection should always be considered first. Extensive evaluation is required to exclude the presence of extrahepatic spread. Transplantation for cholangiocarcinoma is even more controversial and should probably be performed only under the guidance of specific protocols. The role of

adjuvant chemotherapy or chemoembolization in patients undergoing transplantation for cancer remains unknown.

Pediatric Indications. The most common indication for liver transplantation in children is biliary atresia. This diagnosis accounts for 55% of the pediatric recipients in the UNOS registry. Although the creation of a portoenterostomy (Kasai procedure) remains the standard initial treatment, long-term survival without eventual hepatic failure is uncommon. Five-year survival remains under 50%. For this reason, many have proposed that early intervention with OLT be considered. The results of liver transplantation in infants have been somewhat better, with 5-year patient survival of 64%. Thus, a reasonable course of action appears to be neonatal nontransplant surgical intervention, with transplantation reserved for those children developing hepatic insufficiency despite a Kasai procedure. The interim growth improves the donor pool substantially and decreases the technical difficulties inherent in the management of small children. Five-year survival improves to 74% at 3 to 5 years and 79% from 5 to 15 years. However, multiple reoperations and revisions after an initial portoenterostomy should be avoided because they rarely provide long-term disease-free survival and substantially hinder efforts to transplant the patient. The remaining usual indications for pediatric transplantation also occur in the adult population, with 13% performed for metabolic diseases (see above) and 10% associated with fulminant hepatic failure secondary to hepatitis of various etiologies.

SPECIFIC PATIENT SELECTION AND PREOPERATIVE CONSIDERATIONS

Patient Selection. The success of liver transplantation is closely related to the rational selection of patients most likely to benefit from the procedure. In fact, the single most important prognostic factor affecting survival is the medical condition of the recipient at the time of OLT. As with the recipients of other organs, patients should be without additional end-organ failure (other than that clearly related to hepatic insufficiency) and should be candidates for a major operative intervention. In general, dependence on alcohol or other harmful substances should be resolved for at least 6 months. Extrahepatic malignancy, sepsis, and diffuse mesenteric venous thrombosis represent absolute contraindications. Isolated portal vein thrombosis is a relative contraindication. The patient's liver function should be such that complications of dysfunction are emerging, with the predicted life span of the patient managed medically less than 2 years. With the improvements in survival after allotransplantation realized in the past decade, it is also appropriate to consider patients with metabolic diseases or moderately advanced liver disease with significant alterations in quality of life such as extreme fatigue, refractory pruritus, or encephalopathy.

Preoperative Preparation. Once advanced liver disease is identified, efforts to proceed with transplantation should be initiated, because the preoperative health of the recipient greatly affects the chance for success. Early evaluation by a multidisciplinary team schooled in the specific requirements for liver transplantation is critical. In addition to surgical evaluation, the selection of appropriate candidates for transplantation should be based on input from medical colleagues, including a hepatologist and infectious disease specialist. Evaluation by a social worker and psychiatrist to establish the ability of the patient and family to manage themselves postoperatively is important. Immediate efforts should optimize the candidate's nutritional

status and overall medical condition. Finally, education of the patient must be initiated early to ensure that complications of medical noncompliance are avoided postoperatively.

For all liver recipients, thorough preoperative evaluation to determine the antibody titer directed against hepatitis A, B, and C as well as cytomegalovirus, human immunodeficiency virus, Epstein-Barr virus, and herpes simplex is mandatory. In addition, viral antigen detection is required to identify active infection. The role of transplantation for primary viral hepatitis was discussed previously. Active infection with cytomegalovirus or herpesvirus requires clinical resolution before transplantation, but viral carriers can receive transplants given appropriate antiviral prophylaxis. Human immunodeficiency virus infection contraindicates OLT. In addition to the recipient's status, the donor's status for these viruses should be ascertained before implantation, again to allow for antiviral prophylaxis when indicated.

Several alternative therapies should be considered in the candidate for transplantation, not necessarily to obviate the need for transplantation but to improve the medical condition before transplantation. Patients with good synthetic function (normal bilirubin, normal coagulation, normal albumin), such as an individual with Child's A cirrhosis and recurrent variceal bleeding, may benefit from nontransplant surgical palliation such as a Warren shunt. The TIPS procedure is also a useful palliative step for refractory complications of portal hypertension. Stabilization of disease with these interventions gives more time to evaluate patients and optimize preoperative conditions. It allows for a more thorough evaluation of a patient's ability to comply with the posttransplant medical regimen, particularly alcoholic patients who present abstinent because they are *too sick to drink*. This compliance alone may stabilize the disease in an alcoholic patient with Child's A cirrhosis and avoid the need for transplantation. Reduction in the degree of portal hypertension may also minimize intraoperative bleeding during the recipient hepatectomy. Problems with these palliative procedures are frequent and require surveillance with particular attention to portal vein patency. Identified candidates should be treated at a center with transplant expertise to facilitate palliation that does not jeopardize future transplantation. Alternative treatment should not delay transplantation.

SELECTION OF DONORS

Immediate function of a transplanted liver is imperative. Unlike kidney, pancreas, or, to some extent, heart transplantation, no artificial means is readily available to support an anhepatic patient in the event of graft failure. Without a rapid restoration of synthetic function, death from bleeding or cerebral edema generally ensues within 72 hours. The single most important factor determining the early function of a liver allograft is the viability of the donor liver. Although this seems obvious, determining the state of the liver in a heart-beating cadaver remains imprecise, so careful attention to the conditions of the donor's death, the morphology and function of the organ before harvest, and the specifics of the extirpation is critical. Failure of a graft to function at all after a technically successful transplantation is known as primary nonfunction (PNF). The only treatment for PNF is retransplantation within 24 to 72 hours. Factors contributing to the development of PNF include parenchymal insufficiency unrecognized at the time of harvest, graft injury during the harvest or cadaver resuscitation,

preservation injury or prolonged cold ischemia, prolonged rewarming time, and reperfusion injury after implantation.

Several factors have been investigated to aid in the prediction of PNF. The most widely noted is the estimated parenchymal fat content. Donor liver biopsy specimens that show a 40% or greater parenchymal replacement by fat have a higher chance of PNF, and in some settings this is a reasonable indicator of the adequacy of a donor organ. This must, however, be balanced against the severity of the recipient's disease and the urgency of the planned operation. Other factors used to evaluate the donor's status include the age of the donor, the level of inotropic support, the mechanism of the donor's death, the level of hepatic or intra-abdominal trauma, the presence of hypernatremia, and the biochemical studies of liver function. No single parameter has been established absolutely governing the acceptance of a donor for organ harvest. Rather, combinations of risk factors are generally responsible for discarding a potential donor liver. In case of doubt about the quality of an organ, the personal inspection of the liver by an experienced transplant surgeon is often critical for decision about the use of an organ.

Donor use has been improved with the introduction of UW preservation solution as mentioned earlier. Until 1987 the outer limit for cold storage of the liver using EuroCollins solution was about 8 hours. Use of UW solution has extended the time to about 24 hours, allowing for better allocation of the organ and preparation of the recipient. The specifics of this and other preservation solutions are detailed in Part VII of this chapter.

The considerations of ABO typing and other immunologic concerns are discussed later. An additional issue is size compatibility. Smaller organs are easily adapted to a large recipient, but the converse is not true. One advance has been the use of reduced-size allografts, particularly in children. Usually the left lateral lobe (segments 2 and 3) or left lobe (segments 2, 3, and 4) is used, allowing up to 1/10 weight mismatch. This has been the most important factor in the success of transplantation in small children. This technique is sometimes used in adults when an emergency transplantation is required. Finally, male recipients of female organs have 10% worse survival than other sex-match combinations, according to the 1994 UNOS registry.

OPERATION ON THE DONOR

Harvest of the donor organ should be performed by an experienced surgeon, with particular care taken to optimize the preharvest resuscitation of the heart-beating cadaver. Because immediate hepatic function after transplantation is required, there is no room for error. Errors in resuscitation include injudicious use of vasopressors, prolonged acidosis, and hypoperfusion from hypovolemia. Visual inspection and palpation of the liver with knowledge of the potential recipient's status and size aid in the assessment of the appropriateness of a donor liver.

The liver is generally procured through a midline incision from jugular notch to pubis, including median sternotomy. Harvest is coordinated with the harvest teams for other organs. Complete mobilization of the liver is required, including division of both triangular ligaments and the falciform ligament. The hepatogastric ligament is divided. Particular attention is paid to the vascular supply, including preservation of aberrant hepatic arteries (20% aberrant right from

the superior mesenteric artery, 15% aberrant left from the left gastric artery). Unlike the relative impunity associated with arterial ligation in nontransplanted livers, failure to complete the arterial revascularization due to an unrecognized arterial supply is poorly tolerated after cold storage. PNF, late biliary stricture, or intrahepatic abscess may result. Additional care must be taken when simultaneous pancreas harvest is performed to avoid hepatic artery injury or portal vein transection. The suprahepatic vena cava should be preserved for the hepatic graft, and care should be taken to avoid caval injury during cardiac retrieval. The gallbladder is removed either *in situ* or after explantation.

Once mobilization is completed, perfusion with UW solution is initiated through the distal aorta or common iliac arteries with ligation of the supraceliac aorta. The inferior mesenteric vein or splenic vein is used for portal perfusion. When pancreatic harvest is performed, some surgeons prefer to use the portal vein for cold perfusion. UW solution is also flushed into the biliary tree. Topical slush is rapidly applied. Hepatic extirpation is performed after cardiopulmonary retrieval and before harvest of the pancreas and kidney. The iliac artery and vein should be harvested in the event that vascular reconstruction is required.

IMMEDIATE PREOPERATIVE MANAGEMENT

Patients awaiting liver transplantation are prioritized on a national waiting list based on severity of disease as defined by the UNOS. Status 4 patients are at home and functioning normally, status 3 patients can be at home but require continuous medical care. Status 2 patients are continuously hospitalized in an acute care unit for at least 5 days or are in the intensive care unit. Status 1 patients are in the intensive care unit because of acute or chronic liver failure with a life expectancy without a liver transplant of less than 7 days. Status 7 is reserved for patients taken from the active list for a temporary medical contraindication such as sepsis. These patients can accrue points for time on the list despite not being able to accept an organ. Strict adherence to guidelines for proper categorization is required to allow for ethical allocation of organs. As soon as a donor is identified, the organ is paired with a potential recipient, who is called to the hospital for preoperative evaluation before organ harvest.

Appropriate perioperative management of a transplant recipient begins with a thorough preoperative physical examination (including rectal and dental examinations) to rule out the possibility of ongoing infection or malignancy. Routine screening includes a complete blood cell count, electrolyte and metabolic profile, urinalysis, and chest film. Blood is crossmatched. Intravenous lines, including arterial lines and pulmonary artery catheters, should be placed with strict attention to aseptic technique because cutaneous contamination with bacteria or fungi can cause serious postoperative complications in the transplant patient. Selective decontamination of the gut and mechanical preparation of the bowel are advocated at many centers, as is a shower with an antimicrobial soap.

For patients in the intensive care unit, supportive measures are continued as needed. Of specific concern is the potential for neurologic recovery in patients with advanced encephalopathy and cerebral edema. A recent advance has been the use of intracranial pressure monitoring in the perioperative period. This was once shunned as a prohibitive risk for cerebral

bleeding in the coagulopathic fulminant hepatic failure patient, but routine assessment is now possible through minimally invasive techniques. Small-gauge catheter pressure monitors can be placed into the subdural space without the need for parenchymal puncture, thus decreasing the risk of the intervention substantially. Greatly elevated intracerebral pressures, especially in association with signs of transtentorial herniation in a Grade 5 coma patient, suggest an irreversible lesion that is unlikely to resolve after transplantation.

OPERATION ON THE RECIPIENT

Few surgical procedures require the fastidious attention to technical detail required in liver transplantation. Technical errors are translated directly into infectious complications or marginal biliary function. Thus, transplantation should be performed only by surgeons proficient in the procedure. In addition, the operative environment should include experienced nursing and ancillary support.

Intraoperative management by a knowledgeable anesthesiologist with experience in liver transplantation is critical for a successful technical result. The procedure presents the challenge of maintaining homeostasis of temperature, circulation (including oxygen-carrying capacity and coagulation competence), gluconeogenesis, and electrolyte concentration while establishing adequate anesthesia and paralysis with agents not requiring hepatic function for degradation. Intraoperative ICP monitoring is appropriate for patients with severe encephalopathy. In procedures using a venovenous bypass, a perfusionist is required, and procedures performed without bypass require adequate maintenance of preload during caval occlusion and correction of metabolic abnormalities after release of the congested portal circulation. After the initial function of the allograft, the most important factor predictive of technical success is the stability of the patient intraoperatively and his or her delivery to the intensive care unit normothermic with adequate circulatory competence.

Successful engraftment of the organ begins with a controlled recipient hepatectomy. This can be a formidable task in patients with severe portal hypertension and extensive collateral formation or in those with multiple operative interventions. In general, extirpation follows the basic surgical guidelines of establishing proximal and distal vascular control combined with lysis of all ligamentous attachments. Specific technical concerns include retaining maximal length on all vessels. Mobilization of the common bile duct depends on the planned biliary reconstruction (choledochocholedochostomy versus choledochojejunostomy). Care to avoid injury to the right adrenal vein during caval dissection is important. If venovenous bypass is planned, cannulation of the left axillary, femoral, and portal veins is performed.

The decision to place the recipient on bypass is routine at a number of centers; and liberal use of this technique, originally developed by Shaw and colleagues, has clearly led to improved operative mortality. Bypass avoids mesenteric congestion and minimizes the release of lactate and other by-products of hypoperfusion into the portal circulation. In addition, it improves venous return to the heart during implantation and thus improves hemodynamic stability during the period of caval occlusion. Bypass also diverts the portal flow during difficult recipient hepatectomies to minimize blood loss, particularly during dissection of the retrohepatic cava and bare spot. Despite these benefits, it is now clear that many patients tolerate OLT without

the additional manipulation required by bypass. It has thus become policy at many centers to employ this technique selectively after an intraoperative trial of portal vein and vena caval occlusion.

The implantation procedure begins with the suprahepatic vena caval anastomosis followed by the infrahepatic caval anastomosis. Alternatively, the donor vena cava can be anastomosed side to side with the recipient vena cava if it is left *in situ* during the recipient hepatectomy (piggyback technique). The operation then proceeds to the portal anastomosis. After all venous connections, the liver is reperfused with the suprahepatic vena cava temporarily occluded and the infrahepatic vena cava vented to allow washout of the hyperkalemic and adenosine-rich UW solution. The hepatic artery anastomosis is the final vascular step in the procedure. Some groups perform simultaneous arterial and venous reperfusion.

The biliary reconstruction remains an additional area of debate. Options include choledochocholedochostomy with or without externalized T-tube stents. This has the advantage of easy access for subsequent biliary manipulations or evaluation of bile, as well as preservation of the sphincter mechanism. Unfortunately, the anastomosis is particularly sensitive to ischemic injury of the common duct, and complications of leak when the T tube is removed remain vexing. The incidence of technical complications related to this method ranges from 12% to 50% in published series, with the cumulative average being 25%. These problems are obviated for the most part by use of a Roux-en-Y choledochojejunostomy at the expense of convenient biliary access. Leak or stricture is still observed in 4% to 30% of cases (mean 14%). This method is frequently used in pediatric transplants. One report of 300 transplants performed with a side-to-side choledochocholedochostomy technique has reported a remarkable 2.2% technical complication rate. This technique awaits confirmation in other centers.

POSTOPERATIVE MANAGEMENT

Management in the postoperative intensive care unit is similar to that after any major procedure. Ventilatory support and volume replacement are standard. Isolation is not required beyond standard universal precautions. No sedation is given until extubation. For unclear reasons, postoperative pain is usually mild, and any major discomfort should alert one for possible complications. Close monitoring of serologic liver enzymes is critical because increasing enzymes or a failure of enzyme values to correct rapidly suggests PNF or technical complications such as hepatic artery thrombosis. Liberal use of the Doppler ultrasonography and rapid return to the operating room are mandatory in these situations because early detection is the only factor separating a return to normal liver function from complete graft necrosis and patient death.

Use of drains and antibiotics is no different in this operation from that in any other major abdominal procedure. Closed-suction drains should be used and removed early after the threat of postoperative hemorrhage is over. Brief antibiotic prophylaxis is appropriate with an agent with adequate skin and biliary organism coverage. Prolonged use of prophylactic antibiotics is contraindicated. A protocol for decontaminating the small bowel can be used but is usually discontinued within a few weeks of transplantation. At Duke University Medical Center,

decontamination (colistin, 100 mg./10 ml.; gentamicin, 80 mg./10 ml.; nystatin, 2 Ч 10⁶/10 ml.) is discontinued at the time of normal enteral feeding, often within the first postoperative week).

If a T tube is used in the biliary reconstruction, a T-tube cholangiogram is obtained within a week with internalization contingent on a normal result. Patients are discharged when they are familiar with their medications. Patients living more than 2 hours from the transplant center generally stay in the vicinity for an additional 2 to 4 weeks. Close monitoring of hepatic function and medical compliance is continued twice a week for 4 weeks and weekly for an additional 4 weeks. After this outpatient evaluation, patients are returned to their referring community for chronic follow-up. It is important to establish open lines of communication between the community physician and the transplant center to ensure prompt recognition and referral of postoperative complications.

IMMUNOLOGIC MANAGEMENT

The liver must be considered separately from other solid organs with regard to immunologic management. Many well-established concepts of donor-host interaction after kidney or heart transplantation do not apply after hepatic transplantation, and failure to recognize the unique properties of this situation can lead to detrimental perioperative treatment.

HLA Typing. HLA matching is not feasible before liver transplantation. However, because new techniques have been developed that allow donor HLA data to be provided in a timely manner, the future use of these data is pertinent. Although matching donors and recipients with regard to HLA type clearly improves outcome after kidney, heart, and pancreas transplantation, no such correlation exists with liver transplantation. Indeed, matching may, in fact, reduce overall survival. The reasons for this lie in the dualistic nature of HLA in the pathophysiology of liver disease. T-cell-mediated rejection of the organ is mechanistically the same as with other organs, so rejection is reduced with improved HLA compatibility. However, the physiologic role of HLA is to present viral peptides to T cells to initiate destruction of virally infected cells. Thus, HLA compatibility potentiates the inflammation during viral reinfection after transplantation for viral hepatitis and increases the chance for clinical recurrence of the original disease. Similarly, T-cell-mediated autoimmune diseases (e.g., PBC) are etiologically based on T-cell recognition of HLA presented peptides. Therefore, recurrence of autoimmune diseases may be potentiated as well. Further knowledge regarding specific disease states worsened by certain HLA matches may be useful in selective typing in the future.

Crossmatch and ABO Matching. The lymphocytotoxic crossmatch is not used prospectively before liver transplantation. Again, temporal concerns are most pressing; but in this case, the value of a positive crossmatch in predicting subsequent poor outcome from hyperacute rejection is minimal. Indeed, hyperacute rejection is rarely seen even in the face of documented preformed antibodies and ABO incompatibility. The reasons for this remain controversial because hyperacute rejection can be readily produced experimentally. Although preformed antibodies reduce long-term graft survival somewhat, early results appear to be minimally affected. Grafts unmatched for ABO antigens can cause antibody-mediated graft-versus-host disease with mild hemolytic anemia and fever occurring between postoperative days

5 and 12. This is the result of intrahepatic B cells that secrete antibody directed against the recipient ABO antigens. Although it is usually self-limited, increased immunosuppression may be required. Alternatively, some groups suggest a decrease in immunosuppression to allow for a limited rejection of the offending B cells. It is preferred that rules for ABO compatibility be followed for elective transplantation. In the emergent setting, however, ABO-incompatible grafts can be used with acceptable results. The 5-year survival for grafts that are ABO incompatible is approximately 15% lower than that for grafts without ABO discrepancy. This not only reflects some immune preference but also the patient population that receives ABO mismatched grafts, that is, emergent transplants for FHF and PNF.

Acute Rejection. As with other allografts, T-cell-mediated destruction of the liver is inevitable without immunosuppressive therapy. The primary targets for T-cell recognition are HLA antigens on the biliary epithelium and vascular endothelium. The characteristics of this rejection, termed *acute rejection*, are similar to those of kidney or heart in that it develops in most of the cases during the first 6 months after transplantation, usually within the first 4 postoperative weeks. More than half of patients develop at least one episode of rejection. Symptoms are nonspecific, often including mild intermittent fever and general malaise with alteration in liver tests. The diagnosis should be confirmed by liver biopsy. Most episodes are readily reversible (90%), given prompt recognition and initiation of antirejection therapy. This contrasts sharply to chronic rejection, discussed later.

Monitoring for acute rejection is a continuous process. Needle-core biopsy of the allograft is the best diagnostic test. Histologically it appears as a predominantly T-cell and monocyte infiltrate in the portal tracts, with subendothelial (endotheliitis) and biliary epithelial aggregates. Eosinophils and polymorphonuclear leukocytes are present to a lesser degree but are more prevalent in hepatic rejection than in infiltrate seen in other organs. Although protocol biopsies are performed in some centers, most groups rely on monitoring of the liver function studies and/or the serum beta₂-microglobulin value, with biopsy used to clarify detected abnormalities. Because a primary target in acute rejection is biliary epithelium, it often presents initially as a cholestatic process with rapid increases in the alkaline phosphatase and bilirubin values. However, changes in hepatic biochemical parameters are nonspecific and can also indicate technical or infectious complications where alterations in the immunosuppressive regimen would be ineffective or even detrimental. Thus, liberal use of biopsy and Doppler ultrasound evaluation of hepatic blood flow and bile duct integrity is critical. Once acute rejection is diagnosed, rescue immunosuppression is initiated as described later.

Chronic Rejection. The development of liver allograft dysfunction over a period of months to years is termed *chronic rejection* and, like other allografts, is controversial and multifactorial in its etiology, and is usually not reversible. Histologically it appears as a paucity of bile duct epithelium without conspicuous lymphocytic infiltration and has thus been described as the “vanishing bile duct syndrome”. Additionally, an obliterative vasculopathy can occur with parenchymal fibrosis. The time course, histology, and refractory nature of chronic rejection suggest that direct cell-mediated destruction is not a primary mechanism. It is likely that the cumulative effects of mild subclinical immune recognition by several limbs of the immune

system, and the resulting exposure to soluble factors including fibrogenic cytokines, eventually take their toll on the fragile epithelium. Chronic rejection often requires retransplantation.

Immunosuppressive Pharmacology. Manipulation of the immune system is required to avoid graft loss from rejection. Identifying a safe, effective, and minimally immunosuppressive regimen requires a careful balance aimed at reducing infectious and neoplastic complications without a resultant increase in allograft rejection and/or dysfunction. Thus, rational, selective use of several immunosuppressive agents is required to manage successfully the broad array of patients who are transplanted. Three general classifications of immunosuppression are used: (1) induction therapy, a relatively intense initial conditioning of the newly transplanted recipient; (2) maintenance therapy, drugs given at minimal doses required to maintain graft function; and (3) rescue therapy, heightened immunosuppression given to reverse an episode of acute rejection. Most transplant centers have specific protocols for each situation.

Several issues specific to liver transplantation should be mentioned. Of major importance is that the liver appears to have less immunostimulatory antigenicity than other organs. The specifics of this perceived resistance remain somewhat controversial but may relate to several peculiarities of hepatic physiology. First, the portal circulation is exposed regularly to enteric pathogens and, more importantly, to absorbed peptides, which have potential antigenicity. A generalized perihepatic anergy has been postulated as protective in this setting to prevent vascular thrombosis and inappropriate hepatic inflammation. Clearly the reticuloendothelial system of the liver—the Kupffer cells—is important in establishing appropriate versus inappropriate presentation of portal antigen. Most of the Kupffer cells of the liver are replaced with those of recipient HLA type within a few weeks after transplantation. In addition, the Kupffer cells of the donor have been described as migrating to other recipient tissues and establishing a state of chimerism. Whether this chimerism creates specific tolerance or is the result of it remains to be seen. From a practical standpoint, maintenance immunosuppression after liver transplantation can be comparatively low, and many patients have been weaned to little or no immunosuppression for years. Also of importance in this regard, the liver can tolerate brief periods on immunosuppression withdrawal if it becomes necessary to combat a potentially lethal infection.

A recent issue of particular importance in liver transplant immunosuppressive pharmacology has been the development of the drug tacrolimus (FK 506). This agent has been suggested as having particular efficacy in the setting of liver transplantation, with truly remarkable results in early, uncontrolled trials performed at the University of Pittsburgh.⁴⁸ Tacrolimus has a mechanism of action similar to that of cyclosporin A and it has been used as a replacement for this drug. A possible benefit is the potential steroid-sparing effect of tacrolimus, exceeding that of cyclosporine. Single-drug maintenance therapy is being actively investigated. Multicenter, randomized, prospective trials comparing cyclosporine to tacrolimus are underway, with the initial 1-year follow-up demonstrating a significant reduction in rejection episodes and a lower need for rescue therapy with OKT3 in the tacrolimus group. No graft or patient survival advantage has been demonstrated. The side effect profile for tacrolimus differs from that of cyclosporine in that the cosmetic complications of hirsutism, acne, and gingival hyperplasia are not present but the potential for neurologic abnormalities, diabetes mellitus, and renal

impairment appears to be more prevalent. An important consideration is that the combination of cyclosporine and tacrolimus drastically increases the incidence and severity of side effects. Thus, therapy must be limited to one or the other, and limiting the use of either in the early postoperative setting may benefit renal function. Clearly, both cyclosporine and tacrolimus have proven efficacy for maintenance immunosuppression after liver transplantation. The specific selection of either remains to be established by randomized investigation. As a rescue agent during established acute rejection, neither tacrolimus nor cyclosporine is particularly efficacious compared with glucocorticosteroids or antilymphocyte antibody preparations.

Another practical issue unique to liver transplant immunosuppression relates to the absorption of cyclosporine and, to a lesser degree, tacrolimus. Drug uptake depends on the availability of enteric bile salts. Thus, with decreased production of bile in the initial posttransplant period or during a rejection episode, or biliary diversion through a T tube, cyclosporine should be administered intravenously to maintain adequate plasma levels. A new cyclosporine formulation (Neoral), which is bile salt independent for absorption, is now available.

Antiviral Immunity and Immunosuppression. The importance of viral infection in liver transplant patients cannot be overstated. *De novo* infection or latent virus reactivation of pathogens, including cytomegalovirus, herpes simplex virus, and Epstein-Barr virus, and reinfection of hepatotropic viruses, including hepatitis B and C, remain serious sources of posttransplant morbidity and mortality and are directly related to the intensity of the immunosuppressive regimen employed. In particular, the use of antilymphocyte antibody preparations, particularly OKT3, have been implicated in increasing the likelihood of viral infection and associated complications such as lymphoproliferative disorders. In response to this association, most transplant centers have incorporated antiviral prophylaxis into their immunosuppressive protocols, with the specific agents, duration of therapy, and dosages governed by the viral status of the recipient and the donor organ. The relationship between the pathophysiology of acute T-cell-mediated rejection and the physiologic function of T cells in viral immunity is underscored in the transplant patient on T-cell-directed immunosuppression.

OUTCOMES

Clearly, liver transplantation is the most significant advancement in the treatment of end-stage liver disease this century. Diseases treated by OLT are by definition terminal with few exceptions, and as such are lethal without hepatic replacement. Survival of a patient with no other hope for survival is the most obvious positive outcome. Operative survival now exceeds 90% for first grafts. Retransplant-free survival has improved steadily in the past 10 years and is now 73% at 1 year for all transplants reported to the UNOS registry. The 5-year survival reflecting transplantation before ganciclovir became available is approximately 60%, and improvements of 2% to 3% per year have been made each year since 1987. Predicted actuarial survival for transplants performed in 1995 is over 75%.

Survival and retransplant rates have been the principal, if not the only, measures of outcome in the transplant literature until recently. The technical issues of survival in the first decade of widespread transplantation have overshadowed other outcome measures to the point of their exclusion from the literature. Critical evaluation of costly health care interventions has become

a fundamental priority in our society. Outcomes can be defined either from a physician's, patient's, payer's, or society's perspective. The medical perspective is in the traditional purview of the clinician and clinician investigator, who use objective and quantifiable medical parameters, whereas the patient's perspective involves a more subjective assessment of quality of life. Today, survival has become the norm after OLT, and thus the next decade should clearly define outcome with regard to each of these perspectives.

Medical Perspective. Despite the recent advances in all aspects of liver transplantation, the procedure remains one with considerable morbidity. Most patients have some complications that deviate from an ideal recovery, and all patients accept the trade of their liver disease for the *disease* of immunosuppression. These negative outcomes are generally remedied by prompt recognition of problems and aggressive corrective intervention. It is, therefore, critical that the potential obstacles arising postoperatively are thoroughly understood. The authors have recently categorized negative outcomes under three headings: negative sequel, complications, and failure to cure.

A *negative sequel* is an adverse effect inherent to the transplant procedure. Transplantation in general carries with it the risk of lifelong immunosuppression. Patients must adapt their lifestyle to ensure that medications are taken as prescribed and that the immune system and organ function are monitored when necessary. The risk of opportunistic infection and malignancy that accompanies chronic immunosuppression persists for life. These factors, however, are less intrusive for liver transplant patients than for recipients of other solid organs because of their reduced need for immunosuppression, especially after the first transplant year.

Failure to cure refers to pre-existing conditions that remain unchanged or recur after the transplant procedure. The likelihood of cure reflects the primary disease. Metabolic and cholestatic diseases are generally resolved, as are the physiologic disorders of alcoholic cirrhosis. Unfortunately, viral infections remain generally uncured by liver replacement. As mentioned earlier, clinically significant recurrence of hepatitis B and hepatitis C may limit long-term cure. The cure for these illnesses remains in more potent antiviral therapeutics. Practically, however, viral reinfection that creates a clinically insignificant carrier state is viewed as a cure by the patient. Obviously, no extrahepatic malignancy can be cured by OLT, and the potential for cure in patients with intrahepatic malignancy is solely related to the presence or absence of metastatic disease at the time of recipient hepatectomy.

A *complication* is any other negative outcome that does not fit clearly into the definition of negative sequel or failure to cure. Complications of some kind occur in almost all patients, but the significance of these setbacks varies greatly. For example, acute rejection must be considered a complication of sorts, but most episodes are treated without significant alteration of a patient's comfort or residual disability. Conversely, PNF at best leads to retransplantation and at worst to death. The lack of uniform reporting of complications makes interpretation of the results of OLT difficult. The authors have presented a classification of complications stratified by severity. With the heightened requirement for outcomes-based research in the current economic environment, standardized evaluation of transplant programs with regard to complication rates will likely increase.

Several common complications, such as acute and chronic rejection, have been discussed.

Some complications deserve particular attention because of their seriousness and requirement for prompt intervention. PNF presents as a complete lack of synthetic function from the time of reperfusion. The patient develops encephalopathy, increased intracranial pressure, coagulopathy, hyperbilirubinemia, and hypertransaminasemia. Aggressive supportive therapy and prompt retransplantation are required within 72 hours. Total transplant hepatectomy with portacaval shunt can improve the hemodynamic and metabolic status of the patient for 24 to 36 hours if a suitable liver is not found immediately.

Hepatic artery thrombosis remains a complication, especially in the pediatric population. This presents as a rapid rise in serum transaminase levels. The transplanted liver does not tolerate loss of arterial flow, and failure to restore flow produces graft loss. Hepatic artery stricture or stenosis generally presents as a lesser degree of metabolic change later in the postoperative course. An alternative presentation of dearterialization is bile leak resulting from hepatic duct necrosis. An additional vascular complication that is less frequent but equally devastating is early portal vein thrombosis. Given the rapid and serious but reversible nature of these vascular complications, any suspect change in hepatic function requires immediate evaluation of the hepatic vasculature by Doppler ultrasonography, followed by either re-exploration or a confirmatory arteriogram.

Biliary complications, which occur in 15% to 30% of patients, suggest vascular compromise. Both leaks and strictures can occur regardless of the method of reconstruction. Percutaneous or endoscopic management is generally considered an acceptable first alternative, but reoperation should not be avoided for appropriate lesions at the expense of hepatic function or cholangitis.

Patient Perspective. Today, increasing emphasis is given to patient-oriented subjective outcomes such as quality of life and well-being. Subjective outcomes have inherent limitations, but they are arguably more relevant to individual patients. Well-being represents a composite of several different aspects, including mental, physical, and social criteria.

Due to the initial formidable technical issues of OLT, analyses of quality of life have been almost absent from the literature. With improved results these past few years, researchers in numerous centers are evaluating the effects of OLT on quality of life. Successful transplantation allows a return to an active lifestyle free from the metabolic and hematologic complications of hepatic failure or portal hypertension. Preliminary studies have already shown that self-image, functioning ability, and perception of health status are significantly improved after OLT. Currently, about 60% of patients undergoing OLT return to work within the first year,⁴ a figure that continues to increase long term.

Payer's Perspective. Once therapy has been shown to be effective, it is necessary to determine whether the treatment is cost-effective in order to compare it with other competing technologies. Analysis of cost-effectiveness is difficult to apply to liver transplantation because there is no alternative therapy with which the results of the procedure can be compared. Thus, the cost issue becomes one of establishing a monetary value for one's life. There is no doubt that OLT is expensive. The total first-year expenses for a new liver average \$200,000, with an additional \$10,000 to \$20,000 per year required thereafter. Cost for patients in the intensive care unit at the time of transplant are 3 to 5 times higher than those called in from home for the

procedure. With current health care reform underway, these amounts are falling somewhat. In addition, consolidation of transplant centers in the United States is likely to improve efficiency and reduce costs. As new immunosuppressive agents are introduced, competition is also likely to drive the cost down. Nonetheless, the extent to which physicians should go to return patients to health and to work is a matter of great societal debate. As with all other aspects of liver transplantation, rational selection of patients is the most important factor affecting cost.

EMERGING TECHNIQUES

Split and Reduced-Size Transplantation. The remarkable ability of the liver to regenerate and support the metabolic needs of an individual despite major resection has allowed for correction of most conditions with partial hepatic transplantation.¹⁰ This is useful for major size mismatches, especially in children, and can also help address the growing donor organ shortage. Anatomic division of the lobes of the liver with preservation of hepatic venous and arterial, portal, and biliary branches has become increasingly successful but remains technically formidable. The anatomic boundaries are based on the segmental anatomic system of Couinaud and Bismuth.¹⁷ Left lateral segments 2 and 3 or left lobe grafts (segments 2, 3, and 4) can be placed in recipients who are substantially smaller than the donor. Because of the anterior to posterior dimensions of the right lobe (segments 5 through 8), placement of this graft requires a recipient similar in size to the donor.

Several major centers have initiated protocols for split and reduced-size transplantation with excellent results. Predictably, biliary complications are increased in this procedure, but in children the problem of hepatic artery thrombosis is improved, owing to the comparatively large vasculature present in the graft when an adult liver is reduced for use in a child. Patient survival is as good as or better than full-size grafts in children. Use of reduced-size grafts has decreased the mortality of pediatric recipients on the waiting list to approximately 3%.

Living-Related Transplantation. Arising from the success of reduced-size grafting, living-related transplantation has been initiated at selected centers. This involves a reduced-size graft usually derived from a donor left lobe (segments 2 and 3 or 2, 3, and 4). Technically, this has been quite successful and has the benefits of reduced ischemic time, better HLA match, and better timing of transplantation, producing excellent graft survival. Because most of the transplants are performed for congenital anomalies, the negative aspects of HLA typing have not been problematic. The most pressing concern with this procedure is ethics. Although hepatic resection is generally safe, the mortality is not zero. One donor perioperative death has been reported, and many argue that with the success of reduced-size cadaveric allografts, a procedure that places a healthy parent at risk is not necessary. The concept of informed consent is difficult to establish because most parents disregard personal safety when the life of their child is at risk. One consensus hearing on the matter has approved this procedure, with the caveat that only centers with established success in reduced cadaveric grafts, pediatric transplantation, and adult hepatic surgery be involved.

Heterotopic Liver Transplantation. Placement of an allograft in an anatomically altered site has the advantages of avoiding the recipient hepatectomy (often the most morbid portion of the procedure) and preserving the orthotopic position for future use in the event of graft

failure. Metabolic abnormalities are correctable by this approach. Obviously, however, disorders leading to portal hypertension are not amenable to this mode of therapy.

ALTERNATIVE THERAPIES

The development of artificial support devices has revolutionized perioperative management in all areas of transplantation except hepatic. Renal dialysis, ventricular assist and intra-aortic counterpulsation devices, total parenteral nutrition, and insulin have all helped optimize the condition of solid organ recipients to some degree, making emergent transplantation unusual for any organ other than the liver. The importance of preoperative condition is clear from the survival statistics presented in this chapter. Thus, great effort has been directed toward finding adequate hepatic replacement.

Xenogeneic Support. Xenotransplantation, the use of organs from other species, has many theoretical advantages. A renewable supply of organs subject to genetic manipulation available on an elective basis would greatly alter the course of patients with liver failure. In addition, the hepatotropic viruses responsible for most hepatitis are generally specific for human hepatocytes, so the specter of reinfection would be abolished. Unfortunately, the immune barriers to transspecies transplantation remain formidable. Organs from discordant species, those phylogenetically distant animals to which preformed natural antibodies exist, are hyperacutely rejected. Organs from these animals also produce plasma proteins that are similar but not identical to their human homologues, thus raising the possibility of antigenic proteins subject to immune clearance. Concordant species, namely primates, are rejected in a more conventionally acute manner, but antibody-mediated rejection occurs. Although the immune barrier is less daunting, primates are slow-breeding animals that could quickly become extinct if widespread use were initiated. Of additional concern is the potential for introducing new viruses from primates to man.

Several efforts in xenogeneic organ use have been made in the past 5 years. Baboon livers were used to treat two patients with hepatitis B at the University of Pittsburgh. Both livers functioned well enough to carry out the major physiologic functions of the liver. Interpretation of the immune implications of these procedures, however, is difficult because one patient was infected with the human immunodeficiency virus with an inverted T4:T8 ratio pretransplant and the other received a concurrent baboon bone marrow transplant. An orthotopic pig liver transplant performed at Cedars Sinai Hospital in Los Angeles was hyperacutely rejected (L. Makowka, personal communication, 1994).

Ex vivo perfusion with porcine livers has been successfully employed as a bridge to orthotopic allotransplantation by the authors' group. Biochemical improvement in all measured parameters, including reversal of cerebral edema and reduction of coma, has been demonstrated by the authors' group and others. This approach has the advantage of being reversible without requiring a surgical procedure. It is, however, logistically difficult and temporary.

Bioartificial Liver. The ability of porcine hepatocytes to perform many of the functions of human hepatocytes has been exploited by several investigators by development of an *ex vivo* apparatus for hepatic support consisting of porcine hepatocytes attached to a hollow-fiber dialysis cassette. Early clinical trials have shown promise in reducing cerebral edema as well as

mild improvement in biochemical parameters. Additional investigation in this field will determine whether the volume of the liver can be reproduced in cellular form and whether temporary support will allow recovery of hepatic function, thus avoiding transplantation for some patients with viral or toxin-induced acute liver failure.

SUMMARY

Liver transplantation has evolved in the past decade from an experimental procedure to an accepted, effective therapy for end-stage diseases of the liver. Extended survival of over 75% in appropriately selected patients is now commonplace with return to an excellent quality of life. Continued improvements in perioperative management and operative technique are being realized. The most important predictors of success are the state of the patient at the time of transplantation and the disease being transplanted. Early intervention once end-stage disease is diagnosed is preferable. A critical shortage of suitable donor organs remains the single most important barrier to transplantation

CHRONIC PANCREATITIS

Chronic pancreatitis is a progressive inflammation of pancreas with the periodic acutening and remission, related to the process of autolysis, that shows up by pain, by violation of exocrine and endocrine functions of gland with the eventual result of fibrosis of organ and high risk of malignization.

Etiology and pathogenesis

A gallstone disease is considered the most frequent reason of chronic pancreatitis. Pathogenesis of cholangiogenic pancreatitis acted in pancreatic ducts (theory of general duct) is in part of difficulty of outflow of pancreatic secret and reflux of infected bile or maintenance of duodenum. Dyskinesia, spasms and stenosis of the Vater's papilla of duodenum are instrumental in reflux. Bile or duodenal maintenance, that gets Wirsung's duct, activates the enzymes of pancreas and is instrumental in the origin of its inflammation. Development of pancreatitis potentiates infection. The last can penetrate pancreas not only due to reflux but also in a hematogenic or lymphogenic way.

Thus, chronic pancreatitis develops as a result of functional violations of pancreas, which with the flow of time pass to organic. The reasons of such violations are the attack of acute pancreatitis suffered the in past, alcoholism, traumas of gland, pathology of its vessels, gastroduodenal ulcers, gastritis or duodenostasis.

Pathomorphology

The morphological changes in pancreas at chronic pancreatitis are mainly taken to development of passionately-degenerative processes and atrophy of parenchyma. Connecting tissue in such cases develops both in the particles of gland and between them. In one case the process has diffuse character, in the other it is limited. Thus pancreas becomes dense as a result of excrescence of connecting tissue. It can be multiplied, taking shape of chronic hypertrophy pancreatitis. Atrophy of gland comes in other cases, besides, not evenly in different parts.

The inflammatory edema of parenchyma is exposed in case of acutening of process. Hemorrhages, fatty necrosis and pseudocysts are exposed on the surface of cut.

Classification

(by O.O. Shalimov)

1. Chronic fibrous pancreatitis without violation of patency of main pancreatic duct.
2. Chronic fibrous pancreatitis with violation of patency of main pancreatic duct, dilatated

ducts of pancreas and hypertension of pancreatic juice.

3. Chronic fibrous-degenerative pancreatitis.

Taking into account clinical passing of chronic pancreatitis, classification of O.O. Shelagurov (1970) can have some changes. Such forms are selected:

1. Chronic recurrent pancreatitis.
2. Chronic pain pancreatitis.
3. Chronic painless (latent) pancreatitis.
4. Chronic pseudo tumor-like pancreatitis.
5. Chronic cholecystocholangiopancreatitis (cholangiogenic pancreatitis).
6. Chronic indurative pancreatitis.

Symptoms and clinical passing

As passing of disease has cyclic character with the periodic changes of remission and acuteening, the clinic of chronic pancreatitis depends on the phase of development of inflammatory process. Violation of excretory and incretory functions of pancreas influences polymorphic of symptoms which remission is especially determining in the phase.

Pain, dyspepsia phenomena and progressive loss of weight of body are the basic signs of chronic pancreatitis. Besides, pain, is permanent, changes only its intensity, mainly in epigastric region, sometimes on the left, burning, squeezing or prickly, comes forward the unique symptom of disease, complaints about it precede other symptoms. In some patients the pain feelings increase in lying position. Therefore patients occupy forced sitting position. Intensity of pain can change throughout a day. Patients explain it by acceptance of rich, fried food, boiled eggs, coffee. The last is the principal reason of acuteening of process with acute pain syndrome.

It is needed to mark, that occasionally passing of chronic pancreatitis can take hidden, smooth shapes, with the moderately expressed pain syndrome or pain, that has atypical character, for example, stenocardia. In such patients the symptoms related to violations of exogenous function of pancreas come forward. They complain about absence of appetite, nausea, belch, sometimes vomiting and diarrhea with putrid smell. Thirst, general weakness and progressive loss of weight is observed also.

At palpation of abdomen pain does not arise, or it is quite insignificant. It is sometimes succeeded to palpate horizontally placed pancreas as dense, moderately painful tension bar. The transmission of pulsation of aorta at palpation in a epigastric area count characteristic for pathology.

During intervals between the attacks the feeling of patients remains satisfactory.

Development of saccharine diabetes is the basic sign of endocrine insufficiency, hypoglycemia is rarer. The feature of this form of saccharine diabetes consists in the fact that it shows up in a few years after the beginning of disease, runs easier and often carries latent character. There can be hypoglycemia at the insufficient products of glucagon.

The syndrome of biliary hypertension with development of mechanical icterus and

cholangitis determining it can develop in some patients. The reasons for such cholestasis more frequently are tubular stenosis of choledochus, choledocholithiasis or stenotic papillitis. There is duodenal obstruction in some cases.

Important information about it can be given by the laboratory and instrumental methods of examination.

Examination of excretory function of pancreas is based on establishment of level of amylase in the whey of blood and urine. In acutening period of chronic pancreatitis this level of amylase rises, the numbers of tripsin and lipase grow.

Coprologic examination. Macroscopic picture of excrement gets greyish color, in large masses — with unpleasant smell. Steatorrhea (increase of amount of neutral fat) and creatorrhea are characteristic for it (a plenty of muscular fibres).

Examination of incretory function of pancreas includes: 1) determination of sugar in blood and urine (characteristic is hyperglycemia and glycosuria);

2) radioimmunoassay of hormones (insulin, C-peptide and glucagon).

Sciagraphy survey of organs of abdominal cavity in two projections enables to expose existent concrement in ducts and calcificat in parenchyma of pancreas.

Relaxation duodenography. Thus the development of “horseshoe” of duodenum and change of relief of its mucus can be seen.

Cholecystocholangiography with the purpose of diagnostics of gallstone disease and second damaging of bilious ways is conducted.

Ultrasonic examination (sonography) is one of the basic methods of diagnostics. With the help of symptoms of chronic pancreatitis it is possible to expose inequality of contours of gland, increase of closeness of its parenchyma, increase or diminishment of sizes of organ, expansion of pancreatic duct and wirsungolithiasis or presence of concrement of parenchyma. Thus it is necessary to inspect gall-bladder, liver and bile-excreting ways for diagnostics of gallstone disease and choledocholithiasis.

Scintigraphy of pancreas. On early stages strengthening of scintigraphic picture is observed, on later ones — defects of accumulation to radionuclide (symptom of “sieve” or “bee honeycomb”).

Computer tomography allows to expose the increase or diminishment of sizes of gland, presence of calcificats, concrement, inequality of contours of organ, focuses or diffuse changes of its structure .

Endoscopic retrograde cholangiopancreatography (ERCPG). Expansion of pancreatic duct its deformation, wirsungolithiasis is marked.

It is expedient to apply laparoscopy in the phase of acutening of chronic pancreatitis at development of fatty and hemorrhagic pancreatonecrosis (“stearin name-plates”, exudation).

The puncture biopsy of pancreas under sonography control can have an important value for differential diagnostics of pancreatitis and cancer.

Percutaneous transhepatic cholangioduodenography and -stomy. This method is used both for differential diagnostics of pseudo tumor-like form of chronic pancreatitis and cancer of pancreas and with the purpose of preoperative preparation at presence of icterus. During it there is a possibility to expose expansion of intra- and out-of-hepatic ducts, localization and

slowness of their stricture.

Variants of clinical passing and complications

Chronic recurrent pancreatitis. The changes of periods of acuteening and remission are characteristic for it. The first period shows up by the attacks of pain of different frequency and duration, and during remission patients feel satisfactory.

Chronic pain pancreatitis. Intensive pain in the overhead half of abdomen with an irradiation in loin and region of heart is inherent for this form. Also belting pain often appears.

Chronic painless (latent) pancreatitis. In patients with this form of pathology for a long time the pain is either absent in general or arises after the reception of spicy food rich and can be insignificantly expressed. Violation of excretory or incretory function of pancreas come forward on the first plan.

Chronic pseudo tumor-like pancreatitis. Dull pain in the projection of head of pancreas, dyspepsia disorders and syndrome of biliary hypertension are clinical its signs.

Chronic cholangiogenic pancreatitis. Both clinic of chronic cholecystitis and cholelithiasis and clinic of pancreatitis are characteristic for this form.

Chronic indurative pancreatitis. In patients with this diseases symptoms of excretory and incretory insufficiency of pancreas are present. The low indexes of amylase in blood and urine are characteristic. At the expressed sclerosis of head of pancreas the with including process of general bilious duct, development of mechanical icterus is possible.

Among complications of chronic pancreatitis, fatty dystrophy and cirrhosis of liver, stricture of terminal part of general bilious duct, ulcers of duodenum, thrombosis of splenic vein, saccharine diabetes, pseudocysts of pancreas, exudation pleurisy and pericarditis and heart attack of myocardium are observed.

Diagnosis program

1. Anamnesis and physical methods of inspection.
2. General analysis of blood.
3. Biochemical blood test (amylase, bilirubin, glucose).
4. Analysis of urine on diastase.
5. Coprograma.
6. Sonography.
7. Relaxation duodenogram.
8. Cholecystocholangiography.
9. Retrograde cholangiopancreatography.
10. Computer tomography.

Differential diagnostics

Disease of gall-bladder and biliary ways (gallstone disease, dyskinesia of biliary ways). For these diseases pain in right hypochondrium is inherent, that irradiates in right shoulder-blade and shoulder. At chronic pancreatitis pain is localized in epigastric area, left hypochondrium, often is of belting character. One of the basic additional methods of inspection for confirmation of diseases of gall-bladder and ducts is sonography.

Ulcerous disease of stomach and duodenum. Pain at ulcerous disease is seasonal (relapses more frequent in spring and autumn), unites with heartburn and has tendency to diminishment after vomiting. In patients with chronic pancreatitis pain arises after faults in a diet, often is of belting character. Frequent vomiting is determining, that does not bring facilitation to the patient. Also violations of excretory and incretory functions of pancreas can take place.

Abdominal ischemic syndrome. Patients with this pathology complain about pain, that arises at once after the reception of meal, somewhat diminishes after application of spasmolytics. For the disease considerable weight loss and waiver of meal in connection with dread of pain attack can be characteristic. The basic method of examination, with a necessity for differential diagnostics, celiacography is useful, which enables to expose occlusion of abdominal trunk or its compression. During conducting of differential diagnostics with two last nosologies it is necessary to state a possibility of origin of secondary pancreatitis.

Cancer of pancreas. Mechanical icterus and presence of Courvoisier's symptom are considered the clearest and most important displays of cancer of head of pancreas, and carcinoma of body and tail is a proof pain syndrome. For the cancer the damage of pancreas, rapid progress of symptomatology are characteristic, and for chronic pancreatitis the protracted passing with proper clinical symptomatology and changes which can be exposed by the laboratory, roentgenologic and instrumental methods of examination are characteristic. The most informative among methods of diagnostics of cancer of pancreas are sonography (echo-producing formations in parenchyma of pancreas), computer tomography (tumor knots) and puncture biopsy of gland with the histological examination (reliable diagnostics of cancer).

Heart attack of myocardium. In anamnesis of patients with the heart attack of myocardium it is possible to expose pain behind breastbone, that arises at the physical activity and emotional stress, it is irradiated in left shoulder-blade and left shoulder, unrelated with the reception of meal and disappears as a result of action of coronarolytics. The typical changes of ECG confirm the diagnosis of heart attack of myocardium. In addition, no violations of external and incretions of pancreas are characteristic. The roentgenologic and instrumental methods of examination can help in differential diagnostics.

Tactics and choice of treatment method

Treatment is conducted in the phase of acutening of chronic pancreatitis, as well as at its acute form. In the first days the bed rest and medical starvation is prescribed without limitation of alkaline drink (mineral water). The fight against pain syndrome includes application of

anaesthetic preparations and spasmolytics (promedol, analgin, baralgin, papaverine, no-shparum, platyphyllin). Preparation action is directed on the decline of pancreatic secretion (atropine, methacin, sandostatine, dalargin, stilamine, somatostatine) or on oppression of gastric secretion: H₂-blockers (hystodil, cimetidine, hastrocepin, ranisan, tagamet and others like that), antiacides (almagel, gastropan). Appoint, next to it, and antihistaminic preparations (diphenhydramine hydrochloride, suprastine, fenkarol, tavegil). Antienzymic therapy is also important: a) inhibitor of protease (contrical, trasilol, hordox, antagosan), the dose of which must depend on the level of hyperenzymeemia; b) cytostatic agent (ftorafur, 5-fluorouracil); c) chemical inhibitor of tripsin (aminocapronic acid, pentoxil). For the improvement of microcirculation at this pathology heparin, reopolyhlucline and reohluman are applied. The ponderable value is achieved by disintoxication therapy (hemodes, hluconeodes, enterodes). With the purpose of parenteral feed 5–10–40 % glucose with insulin, plasma, albumen, alvesyn, polyamin and lipofundine are used. Normalization of agile function of organs of digestion is achieved by settings of cerucal and reglan. In complex treatment it is necessary to include vitamins (C, B1, B6, B12) and anabolic hormones (retabolil, nerobol).

At calming down of the inflammatory phenomena a diet № 5 is prescribed in pancreas and conduct correction of excretory insufficiency of pancreas (festal, pansinorm, pancceosymin, digestal and others like that). With the purpose of stimulation of function of pancreas it is possible to apply secretin.

Correction of endocrine insufficiency of pancreas. At development of the secondary saccharine diabetes of easy degree a diet is recommended with limitation of carbohydrates, bukarban, maninil and other peroral preparations, at middle and heavy degrees — insulinotherapy.

Physical therapy procedures. Except medical treatment it is possible to apply inductothermy, microwave therapy (high frequency) and electro-stimulation of duodenum. For spa treatment visiting of Morshyn, Husjatyn, Shidnytsja is recommend.

Surgical methods of treatment of chronic pancreatitis

Indication to operation and its volume depend on the form of pancreatitis. Acutening of chronic cholangiogenic pancreatitis at presence of gallstone disease must be examined as indication to operation in first 24 hours since disease's beginning. Operative treatment is done in case of:

- 1) calcinosis pancreas with the expressed pain syndrome;
- 2) violation of patency of duct of pancreas;
- 3) presence of cyst or fistula of resistance to conservative therapy during 2–4 months;
- 4) mechanical icterus on soil of tubular stenosis of distal part of general bilious duct;
- 5) compression and thrombosis of portal vein;
- 6) gallstone disease complicated by chronic pancreatitis;
- 7) ulcerous disease of stomach and duodenum complicated by secondary pancreatitis;

8) duodenostasis, complicated by chronic pancreatitis;

9) impossibility of exception to operation tumors or violations of arterial circulation of blood of pancreas.

Cholecystectomy at presence of calculous cholecystitis and secondary pancreatitis, acute destructive cholecystitis or hydropsy of gall-bladder.

Choledocholithotomy is executed for patients with cholangiolithiasis: a) with the deaf stitch of general biliary duct (use rarely); b) with its external draining for taking of infected bile (cholangitis), decline of biliary hypertension (at the edema of head of pancreas); c) with internal draining (at tubular stenosis of distal part of general biliary duct, acute expansion of choledochus with the complete loss of elasticity of its wall (execute one of variants of choledochoduodenostomy).

Papillosphincterotomy: a) execute transduodenal with papillosphincteroplasty; b) endoscopic is recommended at the isolated or connected with choledocholithiasis stenosis of large duodenal papilla, fixed concrement of large papilla of duodenum.

Wirsungoplasty is scission of plastic arts of narrow part or distal part of main pancreatic duct (apply at patients with stricture of proximal part of duct by a slowness no more than 2 cm). Lately at the isolated stenosis of bee-entrance of main pancreatic duct endoscopic wirsungotomy is executed.

Pancreatojejunostomy:

a) longitudinal (it is executed at considerable expansion of pancreatic duct)(Fig.5);

b) caudal (by Duval) with the resection of distal part of pancreas.

Resection of pancreas: a) distal or caudal;

b) distal subtotal;

c) pancreatoduodenal (PDR);

e) total duodenopancreatectomy heads or bodies of gland (execute in case of fibrous-degenerative pancreatitis).

Occlusion of ducts of pancreas by polymeric connections (cianocrylat, prolamine, neopren and others like that) results in atrophy of exocrine parenchyma, but keeps to the islet of tissue.

Operations on the nervous system are used in case of the pain forms of chronic indurative pancreatitis, resistant to conservative therapy, in default of rough morphological changes of parenchyma, stroma of gland and deformation of main pancreatic duct: a) left-side splanchnicectomy; b) bilateral pectoral splanchnicectomy and sympathectomy; c) postganglionic neurotomy of pancreas. [video](#)

Cysts of pancreas

Cyst of pancreas is a cavity, filled by liquid (pancreatic juice, exudation, pus), intimately soldered with head, body or tail of organ, is limited by capsule, which has epithelium on internal surface.

Pseudocyst (unreal cyst) is a cavity in pancreas which appears as a result of its destruction,

limited by capsule, that does not have epithelium on internal surface.

Etiology and pathogenesis

The reasons of pseudocysts are destructive pancreatitis, traumas of pancreas, oklusion of Wirsung's duct by parasite, concrement, tumors, innate anomalies of development.

To the real cysts belong: innate (dysontogenetic) cysts which are anomalic in development; acquired retention cysts which develop as a result of difficult outflow of pancreatic juice, cystadenoma and cystadenocarcinoma (by mechanism the origins belong more frequently to proliferative, sometimes — degenerative cysts).

The mechanism of development of pseudocysts consists in the focus necrosis of gland, difficult normal outflow of its secret, there is a destruction of walls of pancreatic ducts with overrun of pancreatic juice gland that causes reactive inflammation of peritoneum of surrounding organs which form the walls of pseudocyst.

Pathomorphology

Morphologically the cysts of pancreas are divided into: pseudocysts retention to the duct are innate, single and multiple.

Pseudocysts are fresh and old. The internal surface of fresh pseudocyst is rough, granulating, grey-red. The table of contents is alkaline, grey or with a brown tint. In an old pseudocyst the wall is smooth and shiny, pale-grey. The table of contents is lighter. Epithelium pseudocysts is absent. More frequently they are met in body and tail of gland and are not connected with ducts.

Retention cysts connected with an obturated duct. The cavity has smooth, grey-white surface, maintenance is transparent, watery or mucous-like. Innate cysts are mainly multiple and shallow. A simple retention cyst differ from those that are always connected with the anomalies of development of ducts and are unite with polycystosis buds and liver.

Rarely there are echinococcus cysts, which have a clear chitinous shell, liquid in cavity and daughter's blisters. They are localized in the area of head of pancreas.

Classification

(by A.N. Bakulev and V.V. Vinogradov, 1952)

I. Innate cysts of pancreas:

1. Dermoid cysts.
2. Teratoid cysts.

3. Innate adenomas.
4. Fibrocystic degeneration.
5. Polycystic degeneration.

II. Inflammatory cysts:

1. Pseudocysts.
2. Retention cysts.

III. Traumatic cysts:

1. As a result of direct damage of gland.
2. As a result of indirect damage of gland.

IV. Parasite cysts:

1. Echinococcosis glands.
2. Cysticercosis glands.

V. Neoplasty cysts:

1. Cyst-adenoma.
2. Cyst-adenocarcinoma.
3. Cavernous hemangioma.
4. Cystic epithelioma.

Pathomorphologyo cysts are divided on:

1. The true cyst.
2. Pseudocysts.

According to clinical passing pseudocysts are divided into acute, subacute and chronic.
According to weight of passing — into simple (uncomplicated) and complicated.

Symptoms and clinical passing

In patients with the cystic damaging of pancreas there can be pain of different character and intensity (dull, permanent, cramp-like and belting). It is localized more frequently in right hypochondrium, epigastric area (cyst of head and body of gland), left hypochondrium (cyst of tail of pancreas). Pain is irradiated in the back, left shoulder-blade, shoulder and spine.

Dyspepsia violations are characteristic. Nausea, vomiting and belch are observed.

The syndrome of functional insufficiency of pancreas shows up by disorders of exocrine and endocrine insufficiency and depends on the degree of damage of organ. The unsteady emptying, replacement of diarrhea of constipation, steatorrhea and creatorrhea, development of the second diabetes are marked.

Compression syndrome. Arises as a result of compression of neighbouring organs. Clinically the compression of organs of gastro-intestinal highway shows up by complete or partial obstruction of general bilious duct (mechanical icterus), vein (portal hypertension) gate, splenic vein (splenomegaly).

During the examination patients with large cysts are marked by asymmetry of abdomen in epigastric and mesogastric areas. At palpation of abdomen tumular formation of elastic consistency with an even, immobile surface is found.

Sonography examination shows echo-free formation with a clear capsule, determines localization and sizes of cyst.

Contrasting roentgenologic examination of stomach and duodenum with the sulfate of barium at the cyst of head of pancreas exposes moving of pyloric part of stomach upwards and breeding of „horseshoe” duodenum (at relaxation duodenography in the conditions of low artificial blood pressure). If a cyst is localized in the area of body of gland, displacement of stomach is marked forward and upwards or downward, rapprochement of its walls, moving of duodenal transition and loops of thin bowel downward and to the right; at lateral projection the distance between stomach and spine is increased. The cyst localized in the area of tail of gland, displaces the stomach forward and upwards, to the left or to the right.

Cholecystocholangiography exposes calculous cholecystitis and cholelithiasis.

Retrograde pancreatocholangiography exposes the changed and deformed, infrequently extended pancreatic duct, occasionally there can be filling of cavity of cyst by the contrasting matter.

Computer tomography shows accumulation of liquid limited by the capsule of different closeness and thickness.

Laboratory examinations exposes hyperamylasemia, steatorrhea and creatorrhea, sometimes — hyperglycemia and glycosuria.

Clinical passing of cysts of pancreas depends on their kind, localization, size, stage of forming and complications.

Four stages of forming of pseudocyst are distinguished (P.G. Karaguljan, 1972).

I stage (1–1,5 months last) — in the center of inflammatory process the cavity of disintegration, which takes surrounding tissue, appears in an omentum bag.

The II stage (2–3 months) is characterized by the beginning of forming of capsule of pseudocyst. Cyst is magnificent, unformed, acute inflammatory phenomena calms down.

The III stage (3–12 months) is completion of forming of capsule of pseudocyst. Last accretes with surrounding organs.

The IV stage (begins an in year from the origin of cyst) is a separated cyst. The cyst is mobile, easily selected from connections with surrounding organs.

Retention cysts arise at closing of lumen of pancreatic duct (concrement, sclerosis). The internal surface of cyst is covered with epithelium. Pain syndrome, violation of exocrine function of gland are characteristic.

Traumatic cysts belong to the pseudocysts with similar passing and clinic, as well as inflammatory pseudocysts.

Parasite cysts (to echinococcus, cysticercotic) are met as casuistry. In such patients Kaconi test and serological Weinberg's reaction are positive.

The variants of clinical passing of the real and unreal cysts depend on their complications.

Perforation in free abdominal cavity. Clinic of the poured peritonitis is characteristic. Tormina, positive symptoms of irritation of peritoneum, possible shock state as a result of irritation of peritoneum by pancreatic juice arise.

Perforation in stomach, duodenum, small, rarer in large intestine is accompanied by diminishment of cyst in sizes or complete disappearance, sometimes diarrhea appears.

Suppuration of maintenance of cyst is accompanied by pain which becomes more intensive, temperature rises, leucocytosis grows.

The erosive bleeding appears suddenly and is accompanied by the symptoms of internal bleeding (expressed general weakness, dizziness). The pallor of skin and mucus shells, sticky death-damp, tachycardia and anemia are observed.

Mechanical icterus arises as a result of compression of cyst on the terminal part of choledochus. The icterus of skin and mucus shells, acholic excrement, dark urine, hyperbilirubinemia, increase of the ALT and AsT level are exposed.

Portal hypertension develops as a result of compression of portal vein. Ascites, varicose expansion of veins of esophagus and stomach, moderate icterus are diagnosed.

Reactive exudation pleurisy more frequently arises in left pleura cavity, where roentgenologic exudation is diagnosed with high maintenance of amylase.

At malignization the walls of cyst specific symptoms are absent, a diagnosis is set during operation (surgical biopsy of cyst wall).

Diagnosis program

1. Anamnesis.
2. Biochemical blood test (amylase, sugar, bilirubin).
3. Analysis of urine on diastase.
4. Coprograma.
5. Sonography.
6. Contrasting sciagraphy of stomach and duodenum (relaxation duodenography).
7. Retrograde pancreatocholangiography.
8. Computer tomography.

Differential diagnostics

The cysts of pancreas are differentiated with the tumors of abdominal cavity and of retroperitoneal space.

Cancer of pancreas. For the cancer tumor of pancreas syndrome of “small signs” (discomfort in epigastric area, loss of appetite, general weakness), permanent dull pain, unrelated with the reception and composition of meal, icterus (cancer of head of gland), Courvoisier's symptom (increased, unpainfully gall-bladder) are characteristic. Inconstant pain at cysts of pancreas is more frequently related to faults in a diet; in anamnesis destructive pancreatitis, traumas of gland are carried. Sonography examination, retrograde pancreatocholangiography and computer tomography help in establishment of diagnosis.

Tumors of retroperitoneal space are passed asymptomatic, clinic shows up by a considerable compression on neighbouring organs. Nausea, vomit, chronic intestinal obstruction, dysuric disorders arise. Clinic of cysts of pancreas, on the opposite, are expressed on early stages. Pain, dyspepsia syndromes, syndrome of exocrine and endocrine insufficiency of pancreas are characteristic. Pain is related to the reception of meal and alcohol.

Aneurism of abdominal aorta. Dull, indefinite pain in abdomen which is unrelated with the reception of meal, pulsation and pulsating formation in abdomen are characteristic, auscultatory is systolic murmur. Aortography allows to confirm a diagnosis.

The cyst of mesentery of thin bowel has painless passing, at palpation it is mobile, easily changes position in abdomen. The cysts of pancreas are practically immobile, pain, anamnesis and laboratory information are characteristic.

The cyst of liver has protracted asymptomatic passing. Pain appears at infection of cyst. For this pathology symptoms which take place at the cysts of pancreas are not typical (pain related to the reception of rich food, alcohol, hyperamylasemia). Topic diagnostics is carried out at ultrasonic examination, scintigraphy, computer tomography.

Differential diagnostics and clinical variants

The basis of disease of pancreas is degenerative-inflammatory processes which are considered to be acute pancreatitis, the so called autolysis tissue by its own enzymes. In the structure of acute pathology of organs of abdominal cavity this disease takes the third place after acute appendicitis and cholecystitis. Women suffer from acute pancreatitis 3–3,5 times more frequently than men.

Acute pancreatitis is a polyetiology disease. Its secondary forms, which arise on the background of pathologies of bile-excreting system and duodenum are closely associated with anatomic and functionally with pancreas, and are met in clinical practice.

Among the “starting” factors of origin of cholelithiasis disease (biliary pancreatitis) abuse by an alcohol and food overloads (fat and irritating products), traumas of pancreas, operating-room in particular, and also separate infectious diseases (parotitis, mononucleosis) are most frequent, especially infection of bilious ways. However, in 10–20 % of patients the reason of acute pancreatitis remains unknown (cryptogenic form).

In the basis of such damages of pancreas and enzymic toxemia lies mainly activating of pancreatic, and then the tissue enzymes (trypsin, lipase, amylase). Often the combination of the broken outflow of pancreatic secret and promoted secretion takes place, which provokes intraductal hypertension.

Among explanations of primary mechanisms of activating of pancreatic enzymes the most value belongs to: a) theory of “general duct” with reflux of bile in the ducts of pancreas; b) blockade of outflow of pancreatic juice with development of intraductal hypertension and penetration of secret in interstitial tissue; in) violation of blood flow of pancreas (vasculitis, thrombophlebitis and embolisms, cardiac insufficiency and others like that); g) toxic and allergic damages of gland. The role of alcohol in such situations can be dual: stimulation of secretion of pancreas and direct damaging action on its tissue.

The process of acute inflammation of pancreas consistently passes the stages of edema, pancreatonecrosis and festering pancreatitis. In the stage of edema there is pancreas of hyperemic, increased in volume, with the shallow hearths of necrosis or, as it is in swingeing majority of cases, without them.

Pancreatonecrosis can pass with fatty or hemorrhagic character. In the first case, as a rule, pancreas is increased, dense, cut whity-yellow hearths are selected to necrosis. Increase of

crimson-black pancreas with darkly-brown infiltrate on a cut is characteristic for hemorrhagic pancreatonecrosis.

Dystrophy of parenchyma is exposed microscopically, up to necrosis, hemorrhages, thromboses of vessels and signs of inflammatory infiltration.

The disease begins suddenly, after the surplus reception of rich spicy food and use of alcohol. Pain, vomiting and phenomena of dynamic intestinal obstruction are considered the most characteristic signs of acute pancreatitis.

A stomach-ache is permanent and so strong, that can result in shock, localized in an epigastric area and left hypochondrium. Some patients feel pain in right hypochondrium with irradiation in the back, loin or breastbone.

In a short period of time after appearance of pain there is a repeated strong vomiting, that does not facilitate the state of patient.

In general vomiting is considered a frequent and characteristic symptom. It is repeated or continuous and never brings facilitation. Vomit masses contain bile, as admixture, and at the difficult form of acute pancreatitis remind "coffee-grounds".

Nausea, hiccup, belch and dryness in a mouth are attributed as less characteristic symptoms of this pathology.

During the examination the skin is pale, often subicterus. Some patients have cyanosis with a "marble picture" as a result of violation of microcirculation. Later the component of respiratory insufficiency can join it. At progressive general condition the patient quickly gets worse to passing of acute pancreatitis, intoxication grows. The skin takes shelter with sticky sweat.

The temperature of body of patients at the beginning of disease can be normal. It rises at resorption of products of autolysis tissue and development of inflammatory process in bilious ways.

The pulse in most cases is at first slow, then becomes frequent, notably passing ahead the increase of temperature of body.

Arterial pressure goes down.

The tongue in the first hour of disease is moist, assessed by white and grey raid. At vomiting by bile the raid has yellow or greenish tint.

The abdominal is blown away, peristaltic noises are loosened. The signs of paresis of stomach and intestine demonstrate early. They need to be included in the pathological process of mesentery root of bowel. At palpation painfulness in an epigastric area and in right, and sometimes and in left, hypochondrium is marked. However, in spite of great pain in stomach, it remains soft for a long time. A little later there is moderate tension or resistance of muscles of front abdominal wall.

Poor local symptoms during heavy intoxication are characteristic for the early period of acute pancreatitis. Later there are symptoms of irritation of peritoneum, and at percussion dulling is marked in lateral parts of abdominal as a result of accumulation of liquid, and also the sign of aseptic phlegmon of retroperitoneal cellulose as slurred or edema of lumbar area is seen. For diagnostics of acute pancreatitis there is the row of characteristic symptoms which have different clinical value.

The Mondor's symptom is violet spots on face and trunk.

The Lagermph's symptom is acute cyanosis of person.

The Halsted's Symptom is cyanosis of abdominal skin.

The Gray's symptom is cyanosis of lateral walls of abdomen.

The Kullen's symptom is the yellow colouring of skin near a belly-button.

The Korte's symptom is painful resistance as a lumbar bar in an epigastric area on 6–7 cm higher belly-button.

The Voskresynskyy's symptom is absence of pulsation of abdominal aorta in an epigastric area.

The Mayo-Robson's symptom is feeling of pain at pressure by fingers in the left costal-vertebral corner.

The Rozdolskyy's symptom — painfulness at percussion above pancreas.

The Blumberg's symptom — in patients with acute pancreatitis more frequently is low-grade. Such feature of this sign of irritation of peritoneum needs to be explained by character of localization of pathological process, mainly in retroperitoneal spaces.

In clinical passing of pancreatonecrosis it is possible to select three periods (V.S. Saveljev, 1978).

The I period (hemodynamic violations and pancreatogenic shock) lasts during 2–3 days. Violation of central hemodynamics, diminishment of volume of circulatory blood and disorders of microcirculation, which at first arise as a result of angiospasm, are considered the most characteristic signs, and later as a result of joining of the intravascular rolling up and laying of elements of blood.

The II period (insufficiency of parenchymatous organs) lasts from 3rd to the 7th day of disease. Violation of functions of basic organs and systems, sign of cardio-vascular, hepatic and kidney insufficiency and growth of violations of breathing are thus observed. In this period there is possible damaging of the central nervous system, which is erected mainly to disorders of psyche, appearances of delirium and comas which in the eventual result are the main reasons of patients' death.

The III period (postnecrosis dystrophic and festering complications) comes in 1–2 weeks after the beginning of disease. During it, on the background of progress of necrosis processes in pancreas, the regenerative changes develop, there are parapancreatic infiltrate and cysts, cystic fibrosis of pancreas. Aseptic retroperitoneal phlegmon which strengthens intoxication can also develop. There is festering pancreatitis at joining of infection. During this period such complications, as erosive bleeding, internal or external fistula, retroperitoneal phlegmon, can develop in patients.

From laboratory information leucocytosis which at the necrosis and hemorrhagic forms of pancreatitis sometimes arrives at $25-30 \times 10^9$, lymphopenia, change of leukocytic formula to the left and the increased ESR are characteristic. Growth of activity of amylase of blood and urine is very often marked, and is the important sign of pancreatitis. For estimation of the state of other organs maintenance of general albumen and its fractions, glucose of blood, bilirubin, urea, electrolytes, acid-base equilibrium (ABE), and also the state of blood coagulation are determined. It is necessary to mark that the exposure of hypocalcemia is considered a bad

predictive sign of heavy passing of acute pancreatitis.

Ultrasonic examination (Pic. 3.5.1) of gall-bladder and pancreas often specifies the increase of their sizes, bulge of walls and presence or absence of concrement of gall-bladder and general bilious duct.

Computer tomography enables to describe in details the changes in pancreas and surrounding organs.

At sciagraphy survey of organs of abdominal cavity gives a possibility to expose the unfolded "horseshoe" of duodenum, pneumatization, expansion of transverse colon (the Gobia's symptom). On the 1st stage of diagnostics in the plan of differential diagnosis of acute destructive pancreatitis with other diseases of abdominal cavity, diagnostics of distribution of destructive damaging of different parts of pancreas and estimation of distribution of parapancreatitis is possible only by the method of computer tomography which depending on clinico-laboratory signs and weight of passing is needed to apply in a different period, and sometimes a few times in dynamics with interval of 4–5 days.

Laparoscopy and laparocentesis are often used for a doubtful diagnosis or necessity of taking away the exudation of abdominal cavity for biochemical or bacteriological examination.

Retrograde endoscopic cholangiopancreatography is used in case of mechanical icterus and suspicion of choledocholithiasis. The last methods are invasive and can if it is necessary transform from diagnostic to manipulation treatments: laparoscopic draining of abdominal cavity at pancreatogenic peritonitis and endoscopic papillotomy at choledocholithiasis and biliary pancreatitis.

Clinical passing of disease can be abortive, slowly or quickly progressive. At abortive passing the process is limited to acute edema of pancreas with convalescence in 7–10 days.

Rapid progress is characteristic for pancreatonecrosis. In patients expressed toxemia, impregnation by exudation of retroperitoneal cellulose and development of fermentative hemorrhagic peritonitis can be seen. Strengthening of stomachache, continuous vomiting, proof paresis of intestine, positive symptoms of irritation of peritoneum and growth of hemodynamic violations are the clinical signs of necrosis of pancreas.

There is a formation of parapancreatic infiltrate at slow progress.

Among early complications of acute pancreatitis shock, peritonitis and acute cardiac, pulmonary, hepatic and kidney insufficiency can be distinguished.

Before later complications it is needed to deliver the abscesses of pancreas, subdiaphragmatic, interintestinal abscesses, pyogenic abscess omentum bag, phlegmons of retroperitoneal space and erosive bleeding.

In future formations of pseudocysts, fistula of pancreas, intestinal fistula and development of saccharine diabetes are possible.

Acute pancreatitis needs to be differentiated with the row of acute diseases of organs of abdominal cavity.

Acute mechanical intestinal obstruction. In patients with this pathology pain is of the alternated character and is accompanied by nausea, vomiting, delay of gases and emptying. It is possible to see the Kloyber bowls on the sciagram survey of organs of abdominal cavity.

Acute cholecystitis runs with characteristic localization of pain and muscular defense,

with presence of increased, painful gall-bladder or infiltrate in right hypochondrium. Often acute (especially lately) pancreatitis develops on the background of gallstone disease (biliary pancreatitis).

Thrombosis or embolism of mesenteric vessels. Both for pancreatitis and for the thrombosis of mesenteric vessels great pain at soft abdomen (absence of defense muscles of front abdominal wall), that precedes to development of peritonitis, is inherent. Yet from the beginning the disease gains heavy character of passing. In anamnesis in such patients a heart disease or heart attack of myocardium rheumatic is met. As a result of gangrene of intestine, the symptoms of peritonitis appear very quickly and intoxication grows. The fragments of mucus shell are found in flushing waters of intestine at the detailed examination, which have the appearance of "meat flushing".

A perforated gastric and duodenum ulcer is distinguished by the presence of dagger pain, defense of abdominal wall, ulcerous anamnesis.

The conservative method is considered the basic one for treatment of acute pancreatitis, but in connection with that unsuccessful conservative treatment of patients with acute pancreatitis can often put a question about the necessity of operation, therefore patients must be in permanent surgical establishment. Thus acute pancreatitis with heavy passing is necessary to be treated under the conditions of separation of intensive therapy.

Before conservative treatment hunger, bed rest, fight against pain and enzymic toxemia, conducting of acid-base state, prophylaxis of festering infection and acute ulcers of digestive duct are to be entered .

Patient's stomach is washed by cold soda solution and a cold on an epigastric area and left hypochondrium is used. Medicinal therapy is prescribed also: spasmolytics (papaverine, platyphyllin, no-shparum, baralgin, atropine); inhibitor of protease (contrical, trasilol, gordox, antagosan); cytostatic agent (5-fluorouracil, florafur). Positive action of inhibitor of protease is marked only in the first days of disease which are subject to conditioned application of large doses. Antibiotics of wide spectrum of action: a) tienam, which most effective in the prophylaxis of festering pancreatitis, as is selected by pancreatic juice; b) cephalosporins (kefzol, cefazoline); c) cefamizine (mefoxine).

Disintoxication therapy is conducted also (5 % but 10 % solutions of glucose, hemodes, reopolyhluquine, polyhluquine, plasma of blood, only from 3 to 5 liters on days, in accordance with a necessity).

For the improvement of rheological properties of blood heparine is prescribed (5 000 ODES every 4 hours).

If patients have the expressed pain syndrome and phenomena of general intoxication during all pain period plus 48 hours (by Bakulev), hunger is used. Such mode lasts on the average of 2–4 days. The parenteral feed of albuminous hydrolyzate is thus conducted, by the mixtures of amino acid and fatty emulsion. Alkaline water of to 1–2 l. and albuminous-carbohydrate diet are also appointed. Infusion therapy is complemented by plasma, by albumen, hemodes, reopolyhluquine. The improvements of microcirculation in pancreas are achieved due to introduction of reopolyhluquine, komplamine, trental and heparin 5000 ODES 6 times per days under the control the indexes of the coagulation system of blood. Anticholinergic drug

(sulfate of atropine, methacin, platyphyllin), H₂-histamin blocker (cimetidine, ranisan, ranitidine, famotidine, omeprazol) are also applied. For the removal of pain: 1) sulfate of the atropine 0,1 % — 1 ml + promedol 2 % — 1 ml + papaverine 2 % — 2 ml + analgin 50 % — 2 ml; 2) isotonic solution of chloride of sodium — 500 ml + baralgin — 5 ml + diphenhydramine hydrochloride 1 % — 1 ml + papaverine 2 % — 2 ml + magnesium the sulfate 25 % — 5 ml + ascorbic acid — 5 ml + lipoic acid 0,5 % — 2 ml + novocaine 0,5 % — 10 ml. are used. From the first days by a nasogastral probe the permanent aspiration of gastric maintenance is conducted also. The Motility function of gastro-intestinal highway gets better at application of cerucal or primperane. With the same purpose forced diuresis (maninil, furosemide, aminophylline) is used on the background of intravenous introduction of plenty of liquid.

At uneffective conservative treatment of patients with acute pancreatitis of middle weight and heavy form it is expedient to apply surgical treatment.

Surgical treatment is carried out for patients with biliary pancreatitis (for a day long from the beginning of disease) in combination with the destructive forms of cholecystitis, at complications of acute pancreatitis by peritonitis, abscess of omentum bag or phlegmon of retroperitoneal cellulose.

Overhead-middle laparotomy, which allows to estimate the state of pancreas, bilious ways and other organs of abdominal cavity, is the best access in this situation. In case of destructive pancreatitis the possible use of lumbar laparotomy from left to right hypochondrium through a mesogastric area is useful.

Cholecystectomy is executed at calculous cholecystitis, phlegmonous inflammation of walls of gall-bladder and biliary pancreatitis. If there are more than 0,9 cm at expansion of choledochus, presence of concrement, ointment-like bile in it, increase of concentration of bilirubin in the whey of blood over 21 mmol/L, choledocholithotomy is complemented by external draining of choledochus. Information of lithiasis of general bilious duct is absent, cholecystectomy in patients with acute pancreatitis is complemented by external draining of choledochus, better by Pikovskyy method (through stump of cystic duct).

Transduodenal sphincteroplasty is shown at fixed concrement of large duodenal papilla (Pic. 3.5.2), if they are diagnosed intraoperative, and also in the cases of papillotomy with extraction of concrement when there is no possibility to execute endoscopic operation .

Omentopancreatopexy. After laparotomy and cutting of gastro-colon and gastro-pancreatic ligament mobile part of large omentum through opening in gastro-colon ligament is conducted and fixed by separate stitches to the peritoneum along the overhead and lower edges of pancreas. Such operation needs to be considered rational at the expressed edema of pancreas and presence of necrosis in it.

Abdominisation of pancreas. A cellulose round pancreas (along the lower and overhead edges of body and tail) is infiltrated by solution of novocaine, after it parietal peritoneum is cut. Under the body and tail glands free end of omentum is conducted and is bundled by a gland. This operation is able to warn the hit of enzymes and products of disintegration in retroperitoneal space.

Sequestrectomy is deleting of necrosis part of gland within the limits of nonviable tissue.

Operation is executed in a dull way.

Necrectomy (deleting of necrosis part of gland within the limits of healthy tissue) is executed by an acute way: tissue of gland is cut on verge of necrosis and bleeding vessels are carefully bandaged.

The resection of pancreas is deleting the part of organ with its transversal cutting within the limits of the unchanged (ad oculus) tissue of gland. The resections of tail and body of pancreas are distinguished (Pic. 3.5.3).

Pancreatectomy is a complete deleting of pancreas. Operative treatment is applied infrequently. After the resection of pancreas adequate draining of its bed is very responsible.

The prognosis of disease depends on character of morphological changes of parapancreatic to the cellulose in pancreas. The more difficult destructive changes, the worst the prognosis.

Chronic pancreatitis is a progressive inflammation of pancreas with the periodic acutening and remission, related to the process of autolysis, that shows up by pain, by violation of exocrine and endocrine functions of gland with the eventual result of fibrosis of organ and high risk of malignization.

A gallstone disease is considered the most frequent reason of chronic pancreatitis. Pathogenesis of cholangiogenic pancreatitis acted in pancreatic ducts (theory of general duct) is in part of difficulty of outflow of pancreatic secret and reflux of infected bile or maintenance of duodenum. Dyskinesia, spasms and stenosis of the Vater's papilla of duodenum are instrumental in reflux. Bile or duodenal maintenance, that gets Wirsung's duct, activates the enzymes of pancreas and is instrumental in the origin of its inflammation. Development of pancreatitis potentiates infection. The last can penetrate pancreas not only due to reflux but also in a hematogenic or lymphogenic way.

Thus, chronic pancreatitis develops as a result of functional violations of pancreas, which with the flow of time pass to organic. The reasons of such violations are the attack of acute pancreatitis suffered the in past, alcoholism, traumas of gland, pathology of its vessels, gastroduodenal ulcers, gastritis or duodenostasis.

The morphological changes in pancreas at chronic pancreatitis are mainly taken to development of passionately-degenerative processes and atrophy of parenchyma. Connecting tissue in such cases develops both in the particles of gland and between them. In one case the process has diffuse character, in the other it is limited. Thus pancreas becomes dense as a result of excrescence of connecting tissue. It can be multiplied, taking shape of chronic hypertrophy pancreatitis. Atrophy of gland comes in other cases, besides, not evenly in different parts.

The inflammatory edema of parenchyma is exposed in case of acutening of process. Hemorrhages, fatty necrosis and pseudocysts are exposed on the surface of cut.

As passing of disease has cyclic character with the periodic changes of remission and acutening, the clinic of chronic pancreatitis depends on the phase of development of inflammatory process. Violation of excretory and incretory functions of pancreas influences polymorphic of symptoms which remission is especially determining in the phase.

Pain, dyspepsia phenomena and progressive loss of weight of body are the basic signs of chronic pancreatitis. Besides, pain, is permanent, changes only its intensity, mainly in epigastric region, sometimes on the left, burning, squeezing or prickly, comes forward the

unique symptom of disease, complaints about it precede other symptoms. In some patients the pain feelings increase in lying position. Therefore patients occupy forced sitting position. Intensity of pain can change throughout a day. Patients explain it by acceptance of rich, fried food, boiled eggs, coffee. The last is the principal reason of acutening of process with acute pain syndrome.

It is needed to mark, that occasionally passing of chronic pancreatitis can take hidden, smooth shapes, with the moderately expressed pain syndrome or pain, that has atypical character, for example, stenocardia. In such patients the symptoms related to violations of exogenous function of pancreas come forward. They complain about absence of appetite, nausea, belch, sometimes vomiting and diarrhea with putrid smell. Thirst, general weakness and progressive loss of weight is observed also.

At palpation of abdomen pain does not arise, or it is quite insignificant. It is sometimes succeeded to palpate horizontally placed pancreas as dense, moderately painful tension bar. The transmission of pulsation of aorta at palpation in a epigastric area count characteristic for pathology.

During intervals between the attacks the feeling of patients remains satisfactory.

Development of saccharine diabetes is the basic sign of endocrine insufficiency, hypoglycemia is rarer. The feature of this form of saccharine diabetes consists in the fact that it shows up in a few years after the beginning of disease, runs easier and often carries latent character. There can be hypoglycemia at the insufficient products of glucagon.

The syndrome of biliary hypertension with development of mechanical icterus and cholangitis determining it can develop in some patients. The reasons for such cholestasis more frequently are tubular stenosis of choledochus, choledocholithiasis or stenotic papillitis. There is duodenal obstruction in some cases.

Important information about it can be given by the laboratory and instrumental methods of examination.

Examination of excretory function of pancreas is based on establishment of level of amylase in the whey of blood and urine. In acutening period of chronic pancreatitis this level of amylase rises, the numbers of tripsin and lipase grow.

Coprologic examination. Macroscopic picture of excrement gets greyish color, in large masses — with unpleasant smell. Steatorrhea (increase of amount of neutral fat) and creatorrhea are characteristic for it (a plenty of muscular fibres).

Examination of incretory function of pancreas includes: 1) determination of sugar in blood and urine (characteristic is hyperglycemia and glycosuria); 2) radioimmunoassay of hormones (insulin, C-peptide and glucagon).

Sciagraphy survey of organs of abdominal cavity in two projections enables to expose existent concrement in ducts and calcificat in parenchyma of pancreas.

Relaxation duodenography. Thus the development of “horseshoe” of duodenum and change of relief of its mucus can be seen (Pic. 3.5.4).

Cholecystocholangiography with the purpose of diagnostics of gallstone disease and second damaging of bilious ways is conducted.

Ultrasonic examination (sonography) is one of the basic methods of diagnostics. With the

help of symptoms of chronic pancreatitis it is possible to expose inequality of contours of gland, increase of closeness of its parenchyma, increase or diminishment of sizes of organ, expansion of pancreatic duct and wirsungolithiasis or presence of concrement of parenchyma. Thus it is necessary to inspect gall-bladder, liver and bile-excreting ways for diagnostics of gallstone disease and choledocholithiasis (Pic. 3.5.5).

Scintigraphy of pancreas. On early stages strengthening of scintigraphic picture is observed, on later ones — defects of accumulation to radionuclide (symptom of “sieve” or “bee honeycomb”).

Computer tomography allows to expose the increase or diminishment of sizes of gland, presence of calcificats, concrement, inequality of contours of organ, focuses or diffuse changes of its structure (Pic. 3.5.6, Pic. 3.5.7).

Endoscopic retrograde cholangiopancreatography (ERCPG). Expansion of pancreatic duct its deformation, wirsungolithiasis is marked, (Pic. 3.5.8).

It is expedient to apply laparoscopy in the phase of acutening of chronic pancreatitis at development of fatty and hemorrhagic pancreatonecrosis (“stearin name-plates”, exudation).

The puncture biopsy of pancreas under sonography control can have an important value for differential diagnostics of pancreatitis and cancer.

Percutaneous transhepatic cholangioduodenography and -stomy. This method is used both for differential diagnostics of pseudo tumor-like form of chronic pancreatitis and cancer of pancreas and with the purpose of preoperative preparation at presence of icterus. During it there is a possibility to expose expansion of intra- and out-of-hepatic ducts, localization and slowness of their stricture.

Chronic recurrent pancreatitis. The changes of periods of acutening and remission are characteristic for it. The first period shows up by the attacks of pain of different frequency and duration, and during remission patients feel satisfactory.

Chronic pain pancreatitis. Intensive pain in the overhead half of abdomen with an irradiation in loin and region of heart is inherent for this form. Also belting pain often appears.

Chronic painless (latent) pancreatitis. In patients with this form of pathology for a long time the pain is either absent in general or arises after the reception of spicy food rich and can be insignificantly expressed. Violation of excretory or incretory function of pancreas come forward on the first plan.

Chronic pseudo tumor-like pancreatitis. Dull pain in the projection of head of pancreas, dyspepsia disorders and syndrome of biliary hypertension are clinical its signs.

Chronic cholangiogenic pancreatitis. Both clinic of chronic cholecystitis and cholelithiasis and clinic of pancreatitis are characteristic for this form.

Chronic indurative pancreatitis. In patients with this diseases symptoms of excretory and incretory insufficiency of pancreas are present. The low indexes of amylase in blood and urine are characteristic. At the expressed sclerosis of head of pancreas the with including process of general bilious duct, development of mechanical icterus is possible.

Among complications of chronic pancreatitis, fatty dystrophy and cirrhosis of liver, stricture of terminal part of general bilious duct, ulcers of duodenum, thrombosis of splenic vein, saccharine diabetes, pseudocysts of pancreas, exudation pleurisy and pericarditis and

heart attack of myocardium are observed.

Disease of gall-bladder and bilious ways (gallstone disease, dyskinesia of bilious ways). For these diseases pain in right hypochondrium is inherent, that irradiates in right shoulder-blade and shoulder. At chronic pancreatitis pain is localized in epigastric area, left hypochondrium, often is of belting character. One of the basic additional methods of inspection for confirmation of diseases of gall-bladder and ducts is sonography.

Ulcerous disease of stomach and duodenum. Pain at ulcerous disease is seasonal (relapses more frequent in spring and autumn), unites with heartburn and has tendency to diminishment after vomiting. In patients with chronic pancreatitis pain arises after faults in a diet, often is of belting character. Frequent vomiting is determining, that does not bring facilitation to the patient. Also violations of excretory and incretory functions of pancreas can take place.

Abdominal ischemic syndrome. Patients with this pathology complain about pain, that arises at once after the reception of meal, somewhat diminishes after application of spasmolytics. For the disease considerable weight loss and waiver of meal in connection with dread of pain attack can be characteristic. The basic method of examination, with a necessity for differential diagnostics, celiacography is useful, which enables to expose occlusion of abdominal trunk or its compression. During conducting of differential diagnostics with two last nosologies it is necessary to state a possibility of origin of secondary pancreatitis.

Cancer of pancreas. Mechanical icterus and presence of Courvoisier's symptom are considered the clearest and most important displays of cancer of head of pancreas, and carcinoma of body and tail is a proof pain syndrome. For the cancer the damage of pancreas, rapid progress of symptomatology are characteristic, and for chronic pancreatitis the protracted passing with proper clinical symptomatology and changes which can be exposed by the laboratory, roentgenologic and instrumental methods of examination are characteristic. The most informative among methods of diagnostics of cancer of pancreas are sonography (echo-producing formations in parenchyma of pancreas), computer tomography (tumor knots) and puncture biopsy of gland with the histological examination (reliable diagnostics of cancer).

Heart attack of myocardium. In anamnesis of patients with the heart attack of myocardium it is possible to expose pain behind breastbone, that arises at the physical activity and emotional stress, it is irradiated in left shoulder-blade and left shoulder, unrelated with the reception of meal and disappears as a result of action of coronarolytics. The typical changes of ECG confirm the diagnosis of heart attack of myocardium. In addition, no violations of external and incretions of pancreas are characteristic. The roentgenologic and instrumental methods of examination can help in differential diagnostics.

Treatment is conducted in the phase of acutening of chronic pancreatitis, as well as at its acute form. In the first days the bed rest and medical starvation is prescribed without limitation of alkaline drink (mineral water). The fight against pain syndrome includes application of anaesthetic preparations and spasmolytics (promedol, analgin, baralgin, papaverine, no-shparum, platyphyllin). Preparation action is directed on the decline of pancreatic secretion (atropine, methacin, sandostatine, dalargin, stilamine, somatostatine) or on oppression of gastric secretion: H2-blockers (hystodil, cimetidine, hastrocepin, ranisan, tagamet and others like that), antiacides (almagel, gastropan). Appoint, next to it, and antihistaminic preparations

(diphenhydramine hydrochloride, suprastine, fenkarol, tavegil). Antienzymic therapy is also important: a) inhibitor of protease (contrical, trasilol, hordox, antagosan), the dose of which must depend on the level of hyperenzymeemia; b) cytostatic agent (ftorafur, 5-fluorouracil); c) chemical inhibitor of tripsin (aminocapronic acid, pentoxil). For the improvement of microcirculation at this pathology heparin, reopolyhlucline and reohluman are applied. The ponderable value is achieved by disintoxication therapy (hemodes, hluconeodes, enterodes). With the purpose of parenteral feed 5–10–40 % glucose with insulin, plasma, albumen, alvesyn, polyamin and lipofundine are used. Normalization of agile function of organs of digestion is achieved by settings of cerucal and reglan. In complex treatment it is necessary to include vitamins (C, B1, B6, B12) and anabolic hormones (retabolil, nerobol).

At calming down of the inflammatory phenomena a diet № 5 is prescribed in pancreas and conduct correction of excretory insufficiency of pancreas (festal, pansinorm, pancceosymin, digestal and others like that). With the purpose of stimulation of function of pancreas it is possible to apply secretin.

Correction of endocrine insufficiency of pancreas. At development of the secondary saccharine diabetes of easy degree a diet is recommended with limitation of carbohydrates, bukarban, maninil and other peroral preparations, at middle and heavy degrees — insulinotherapy.

Physical therapy procedures. Except medical treatment it is possible to apply inductothermy, microwave therapy (high frequency) and electro-stimulation of duodenum. For spa treatment visiting of Morshyn, Husjatyn, Shidnytsja is recommend.

Indication to operation and its volume depend on the form of pancreatitis. Acutening of chronic cholangiogenic pancreatitis at presence of gallstone disease must be examined as indication to operation in first 24 hours since disease's beginning. Operative treatment is done in case of:

- 1) calcinosis pancreas with the expressed pain syndrome;
- 2) violation of patency of duct of pancreas;
- 3) presence of cyst or fistula of resistance to conservative therapy during 2–4 months;
- 4) mechanical icterus on soil of tubular stenosis of distal part of general bilious duct;
- 5) compression and thrombosis of portal vein;
- 6) gallstone disease complicated by chronic pancreatitis;
- 7) ulcerous disease of stomach and duodenum complicated by secondary pancreatitis;
- 8) duodenostasis, complicated by chronic pancreatitis;
- 9) impossibility of exception to operation tumors or violations of arterial circulation of blood of pancreas.

Cholecystectomy at presence of calculous cholecystitis and secondary pancreatitis, acute destructive cholecystitis or hydropsy of gall-bladder.

Choledocholithotomy is executed for patients with cholangiolithiasis: a) with the deaf stitch of general bilious duct (use rarely); b) with its external draining for taking of infected bile (cholangitis), decline of biliary hypertension (at the edema of head of pancreas); c) with internal draining (at tubular stenosis of distal part of general bilious duct, acute expansion of choledochus with the complete loss of elasticity of its wall (execute one of variants of

cholecholedochoduodenostomy).

Papillosphincterotomy: a) execute transduodenal with papillosphincteroplasty; b) endoscopic is recommended at the isolated or connected with choledocholithiasis stenosis of large duodenal papilla, fixed concrement of large papilla of duodenum.

Wirsungoplasty is scission of plastic arts of narrow part or distal part of main pancreatic duct (apply at patients with stricture of proximal part of duct by a slowness no more than 2 cm). Lately at the isolated stenosis of bee-entrance of main pancreatic duct endoscopic wirsungotomy is executed.

Pancreatojejunostomy: a) longitudinal (it is executed at considerable expansion of pancreatic duct);) caudal (by Duval) with the resection of distal part of pancreas (Pic. 3.5.9).

Resection of pancreas: a) distal or caudal; b) distal subtotal; c) pancreatoduodenal (PDR); e) total duodenopancreatectomy heads or bodies of gland (execute in case of fibrous-degenerative pancreatitis).

Oklusion of ducts of pancreas by polymeric connections (cianocrylat, prolamine, neopren and others like that) results in atrophy of exocrine parenchyma, but keeps to the islet of tissue.

Operations on the nervous system are used in case of the pain forms of chronic indurative pancreatitis, resistant to conservative therapy, in default of rough morphological changes of parenchyma, stroma of gland and deformation of main pancreatic duct: a) left-side splanchnicectomy; b) bilateral pectoral splanchnicectomy and sympathectomy; c) postganglionic neurotomy of pancreas.

Cyst of pancreas is a cavity, filled by liquid (pancreatic juice, exudation, pus), intimately soldered with head, body or tail of organ, is limited by capsule, which has epithelium on internal surface.

Pseudocyst (unreal cyst) is a cavity in pancreas which appears as a result of its destruction, limited by capsule, that does not have epithelium on internal surface.

The reasons of pseudocysts are destructive pancreatitis, traumas of pancreas, oklusion of Wirsung's duct by parasite, concrement, tumors, innate anomalies of development.

To the real cysts belong: innate (dysontogenetic) cysts which are anomalic in development; acquired retention cysts which develop as a result of difficult outflow of pancreatic juice, cystadenoma and cystadenocarcinoma (by mechanism the origins belong more frequently to proliferative, sometimes — degenerative cysts).

The mechanism of development of pseudocysts consists in the focus necrosis of gland, difficult normal outflow of its secret, there is a destruction of walls of pancreatic ducts with overrun of pancreatic juice gland that causes reactive inflammation of peritoneum of surrounding organs which form the walls of pseudocyst.

Morphologically the cysts of pancreas are divided into: pseudocysts retention to the duct are innate, single and multiple.

Pseudocysts are fresh and old. The internal surface of fresh pseudocyst is rough, granulating, grey-red. The table of contents is alkaline, grey or with a brown tint. In an old pseudocyst the wall is smooth and shiny, pale-grey. The table of contents is lighter. Epithelium pseudocysts is absent. More frequently they are met in body and tail of gland and are not connected with ducts.

Retention cysts connected with an obturated duct. The cavity has smooth, grey-white surface, maintenance is transparent, watery or mucous-like. Innate cysts are mainly multiple and shallow. A simple retention cyst differ from those that are always connected with the anomalies of development of ducts and are unite with polycystosis buds and liver.

Rarely there are echinococcus cysts, which have a clear chitinous shell, liquid in cavity and daughter's blisters. They are localized in the area of head of pancreas.

According to clinical passing pseudocysts are divided into acute, subacute and chronic.

According to weight of passing — into simple (uncomplicated) and complicated.

In patients with the cystic damaging of pancreas there can be pain of different character and intensity (dull, permanent, cramp-like and belting). It is localized more frequently in right hypochondrium, epigastric area (cyst of head and body of gland), left hypochondrium (cyst of tail of pancreas). Pain is irradiated in the back, left shoulder-blade, shoulder and spine.

Dyspepsia violations are characteristic. Nausea, vomiting and belch are observed.

The syndrome of functional insufficiency of pancreas shows up by disorders of exocrine and endocrine insufficiency and depends on the degree of damage of organ. The unsteady emptying, replacement of diarrhea of constipation, steatorrhea and creatorrhea, development of the second diabetes are marked.

Compression syndrome. Arises as a result of compression of neighbouring organs. Clinically the compression of organs of gastro-intestinal highway shows up by complete or partial obstruction of general bilious duct (mechanical icterus), vein (portal hypertension) gate, splenic vein (splenomegaly).

During the examination patients with large cysts are marked by asymmetry of abdomen in epigastric and mesogastric areas. At palpation of abdomen tumular formation of elastic consistency with an even, immobile surface is found.

Sonography examination shows echo-free formation with a clear capsule, determines localization and sizes of cyst (Pic. 3.5.10).

Contrasting roentgenologic examination of stomach and duodenum with the sulfate of barium at the cyst of head of pancreas exposes moving of pyloric part of stomach upwards and breeding of „horseshoe” duodenum (at relaxation duodenography in the conditions of low artificial blood pressure). If a cyst is localized in the area of body of gland, displacement of stomach is marked forward and upwards or downward, rapprochement of its walls, moving of duodenal transition and loops of thin bowel downward and to the right; at lateral projection the distance between stomach and spine is increased. The cyst localized in the area of tail of gland, displaces the stomach forward and upwards, to the left or to the right (Pic. 3.5.11).

Cholecystocholangiography exposes calculous cholecystitis and cholelithiasis.

Retrograde pancreatocholangiography exposes the changed and deformed, infrequently extended pancreatic duct, occasionally there can be filling of cavity of cyst by the contrasting matter.

Computer tomography shows accumulation of liquid limited by the capsule of different closeness and thickness (Pic. 3.5.12).

Laboratory examinations exposes hyperamylasemia, steatorrhea and creatorrhea, sometimes — hyperglycemia and glycosuria.

Clinical passing of cysts of pancreas depends on their kind, localization, size, stage of forming and complications.

Four stages of forming of pseudocyst are distinguished (P.G. Karaguljan, 1972).

I stage (1–1,5 months last) — in the center of inflammatory process the cavity of disintegration, which takes surrounding tissue, appears in an omentum bag.

The II stage (2–3 months) is characterized by the beginning of forming of capsule of pseudocyst. Cyst is magnificent, unformed, acute inflammatory phenomena calms down.

The III stage (3–12 months) is completion of forming of capsule of pseudocyst. Last accretes with surrounding organs.

The IV stage (begins an in year from the origin of cyst) is a separated cyst. The cyst is mobile, easily selected from connections with surrounding organs.

Retention cysts arise at closing of lumen of pancreatic duct (concrement, sclerosis). The internal surface of cyst is covered with epithelium. Pain syndrome, violation of exocrine function of gland are characteristic.

Traumatic cysts belong to the pseudocysts with similar passing and clinic, as well as inflammatory pseudocysts.

Parasite cysts (to echinococcus, cysticercotic) are met as casuistry. In such patients Kaconi test and serological Weinberg's reaction are positive.

The variants of clinical passing of the real and unreal cysts depend on their complications.

Perforation in free abdominal cavity. Clinic of the poured peritonitis is characteristic. Tormina, positive symptoms of irritation of peritoneum, possible shock state as a result of irritation of peritoneum by pancreatic juice arise.

Perforation in stomach, duodenum, small, rarer in large intestine is accompanied by diminishment of cyst in sizes or complete disappearance, sometimes diarrhea appears.

Suppuration of maintenance of cyst is accompanied by pain which becomes more intensive, temperature rises, leucocytosis grows.

The erosive bleeding appears suddenly and is accompanied by the symptoms of internal bleeding (expressed general weakness, dizziness). The pallor of skin and mucus shells, sticky death-damp, tachycardia and anemia are observed.

Mechanical icterus arises as a result of compression of cyst on the terminal part of choledochus. The icterus of skin and mucus shells, acholic excrement, dark urine, hyperbilirubinemia, increase of the ALT and AsT level are exposed.

Portal hypertension develops as a result of compression of portal vein. Ascites, varicose expansion of veins of esophagus and stomach, moderate icterus are diagnosed.

Reactive exudation pleurisy more frequently arises in left pleura cavity, where roentgenologic exudation is diagnosed with high maintenance of amylase.

At malignization the walls of cyst specific symptoms are absent, a diagnosis is set during operation (surgical biopsy of cyst wall).

The cysts of pancreas are differentiated with the tumors of abdominal cavity and of retroperitoneal space.

Cancer of pancreas. For the cancer tumor of pancreas syndrome of “small signs” (discomfort in epigastric area, loss of appetite, general weakness), permanent dull pain,

unrelated with the reception and composition of meal, icterus (cancer of head of gland), Courvoisier's symptom (increased, unpainfully gall-bladder) are characteristic. Inconstant pain at cysts of pancreas is more frequently related to faults in a diet; in anamnesis destructive pancreatitis, traumas of gland are carried. Sonography examination, retrograde pancreatocholangiography and computer tomography help in establishment of diagnosis.

Tumors of retroperitoneal space are passed asymptomatic, clinic shows up by a considerable compression on neighbouring organs. Nausea, vomit, chronic intestinal obstruction, dysuric disorders arise. Clinic of cysts of pancreas, on the opposite, are expressed on early stages. Pain, dyspepsia syndromes, syndrome of exocrine and endocrine insufficiency of pancreas are characteristic. Pain is related to the reception of meal and alcohol.

Aneurism of abdominal aorta. Dull, indefinite pain in abdomen which is unrelated with the reception of meal, pulsation and pulsating formation in abdomen are characteristic, auscultatory is systolic murmur. Aortography allows to confirm a diagnosis.

The cyst of mesentery of thin bowel has painless passing, at palpation it is mobile, easily changes position in abdomen. The cysts of pancreas are practically immobile, pain, anamnesis and laboratory information are characteristic.

The cyst of liver has protracted asymptomatic passing. Pain appears at infection of cyst. For this pathology symptoms which take place at the cysts of pancreas are not typical (pain related to the reception of rich food, alcohol, hyperamylasemia). Topic diagnostics is carried out at ultrasonic examination, scintigraphy, computer tomography.

Conservative treatment. Treatment of acute or chronic pancreatitis is conducted in accordance with principles. At the unfavorable dynamics of passing the diseases hunger with the permanent sucking of gastric maintenance, parenteral feed and intravenous introduction of liquids are appointed. Puncture of cysts is used through abdominal wall under sonography control with aspiration of maintenance.

Surgical treatment is the method of choice of treatment of cysts of pancreas. The choice of treatment method depends on the stage of forming of pancreas cysts.

On the I stage operation is not used, conservative treatment of pancreatitis is conducted. On the II stage it is used at suppuration of pseudocyst (external draining of cyst). On the III — internal draining of cyst is used. More frequently cystojejunostomy on the eliminated loop of thin bowel by Roux (Pic. 3.5.13), cystojejunostomy with entero-entero anastomosis by Brawn and closing of afferent loop by Shalimov. Cystogastrostomy (Pic. 3.5.14) are executed and cystoduodenostomy is now not applied because of possible complications (infection of cyst, erosive bleeding). Marsupialization (opening and sewing down of cyst to the parietal peritoneum and skin) is used infrequently (at suppuration of cyst is seriously patientsing with the septic state). On the IV stage external and internal draining of cyst and radical operations are applied: a) enucleation of cysts (executed very rarely); b) distal resection of pancreas with a cyst.

The cancer of pancreas is a malignant tumor of epithelium tissue. Its specific gravity among all malignant tumors makes 10 %. Greater part of patients with cancer of pancreas (to 80 %) is made by the persons of capable working age.

The origin of cancer of pancreas is related to character of nutrition: with the promoted

maintenance of albumens and fats in meal.

Shortage of vitamins, especially B and C, harmful habits (abuse of alcohol, smoking), presence of carcinogenic matters in food (nitrite, nitrates and others like that), tonsillectomy suffered in the past also belong to etiologic factors. The cancer tumor of pancreas can arise on the background of protracted period of chronic pancreatitis.

A cancer tumor is localized in the head. Rarer — in the area of body or tail, rarer there is a diffuse damage of pancreas.

A tumor has the appearance of a dense knot or conglomerate of knots of different sizes. It resembles epithelium of pancreatic ducts or epithelium of acinous tissue, sometimes — the Langerhans' islet.

Adenocarcinoma (50–55 %) is exposed microscopically, carcinoid (32–35 %), epidermoid cancer or skir is seldom met.

The cancer of pancreas gives metastases quickly enough which spread in lymphogenic way in parapancreatic lymphatic knots, and afterwards — in the gate of liver. The hematogenic metastases are often exposed in lungs, bones, buds and brain. Possible also remote metastases to the type of Virhov's, Shnitsler's, Krukenberg's.

The clinical signs and passing of cancer of pancreas are various. They depend both on localization of tumor in pancreas and the mutual relations of pathological process with surrounding organs or tissue.

Pain is a permanent symptom on which 60–90 % patients specify. It is conditioned by involvement in the process of nervous elements of pancreas and retroperitoneum space. The pain feelings at cancer of pancreas are unrelated with acceptance of meal, can be periodic with irradiation in the back. The insignificant loss of weight makes progress and for a short time becomes considerable enough. Such is clinic of cancer of body and tail of pancreas.

Icterus is characteristic of the cancer of head of pancreas, that arises as a result of obturation of general bilious duct and develops slowly but with steady growth. At palpation of abdomen Courvoisier's symptom is observed. Protracted, to 3–4 weeks, icterus results in piling up of products of disintegration of bile in blood and tissue, causing heavy intoxication of organism, violation of liver unction, buds and coagulation system of blood. Obturation of duct of pancreas causes dyspepsia disorders: belch, nausea, vomiting, diarrhea (“fat” emptying). Distributions of tumor on duodenum and narrowing of its lumen show up by the signs of stenosis of exit from the stomach of a different degree (feeling of plenitude in a epigastric area, periodic pain, belch and vomiting).

Bilirubinemia grows gradually, mainly due to direct bilirubin. The increase of activity of alkaline phosphatase and level of cholesterol are observed in blood. As at mechanical icterus a bile does not get the intestine, stercobilin in excrement is absent (acholic excrement). There is also no urobiliny in urine, although bilious pigments are present there (bilirubin). It is possible to expose steatorrhea and kreatorrhea in excrement as a result of obturation of pancreatic duct and exception of enzymes of pancreas from digestion.

With the help of radioimmunoassay it is sometimes succeeded to mark the increase of level of tumor markers in the whey of blood: cancer-embryo to the antigen, ferritin.

Sciagraphy of gastro-intestinal highway can expose the cancer heads of pancreas, the

unfolded “horseshoe” of duodenum, and in case of localization of tumor in the body of gland — displacement of back wall of stomach forward. At duodenoscopy rigidity of mucus shell of descending part of duodenum, narrowing of its lumen are determined, and sometimes there is germination of bowel by tumor.

Scanning is an informing method of examination with the use of ^{75}Se -methionine. Such examinations can expose the hearths of reduced accumulation of isotope or its absence in tissue of gland at the damage by tumor.

During laparoscopy the cancer of pancreas is visualized infrequently, however, dissemination of peritoneum and its metastatic hearths in liver are diagnosed without difficulties.

By ultrasonic (sonography) examination it is succeeded to expose the places of promoted closeness of tissue of gland, sign of mechanical icterus at localization of tumor in the head.

Most informing among all is computer tomography (Pic. 3.5.15). It is possible to define both the tumor of gland and its size and metastatic knots. The changes of main duct of pancreas as segmental stenosis or breaking are fixed on retrograde endoscopic pancreatography.

Clinical passing of cancer of pancreas in 70 % patients is marked by the background diseases and complications. This circumstance allows to select a few clinical forms of the cancer of pancreas before appearance of icterus: pancreatitic, diabetogenic, cholangitic and gastritis-like. The names specify the feature of clinical signs of different forms of disease.

Mechanical icterus is the heaviest complication of cancer of pancreas. With the increase of duration and growth of its intensity development of such dangerous complications, as hepatic or hepatic-kidney insufficiency, cholemic bleeding is possible.

Anamnesis has an important value for differential diagnostics. The presence of attacks of pain or intermittent icterus testifies its calculous origin. A pain syndrome at the cancer of pancreas does not have such acuteness and intensity, as at gallstone disease. Icterus in cancer patients, unlike cholelithiasis, develops gradually, incessantly grows and is of proof character.

Often substantial difficulties arise during conducting the differential diagnostics of obturative and infectious icterus. It is necessary to remember, that at viral hepatitis the level of transaminase and aldolase in the whey of blood rises by 2–3 times. At obturation icterus their level does not change substantially, and the increase of activity of alkaline phosphatase and instead of that the level of cholesterol is marked.

However, most operation difficulties are met during conducting the differential diagnosis of the cancer of pancreas and chronic indurative pancreatitis. In fact both processes during examination and palpation produce similar pictures. In such cases puncture of the densest area of pancreas is executed and cytologic examination is quickly conducted.

Treatment of cancer of pancreas is mainly surgical. The choice of method and volume of operation depends on localization of tumor, stage of process, age of patient and his general condition.

Taking it into account, as practice shows, radical operations in the moment of establishment of final diagnosis are successfully executed only in 15–20 % of patients. Pancreatoduodenal resection is the method of choice of operation in patients with the damage of head of pancreas. Operation foresees deleting one block of head of pancreas, distal part of

stomach, duodenum and distal part of general bilious duct. Four anastomosis are thus imposed: gastroenteroanastomosis, cholecysto-enteroanastomosis or choledochoenteroanastomosis, pancreatoentero-anastomosis and enteroenteroanastomosis. Sometimes this operation is executed in two stages. On the first one biliary-enteric anastomosis is formed for taking bile and improvement of function of liver, and the second stage is carried out in 3–4 weeks. However, more frequently symptomatic operations are to be executed: cholecysto-enteroanastomosis or choledochoduodenoanastomosis. They are able to liquidate icterus and prolong the life of patients for 5–9 months. In case of damage of body and tail of pancreas the distal subtotal resection of gland with spleen is radical.

Chemotherapy can give partial remission. With this purpose 5-fluorouracil, adriablastine, methotrexate are mainly used.

Some palliative effect also comes after gamma-ray teletherapy.

Tactics and choice of treatment method

Conservative treatment. Treatment of acute or chronic pancreatitis is conducted in accordance with principles. At the unfavorable dynamics of passing the diseases hunger with the permanent sucking of gastric maintenance, parenteral feed and intravenous introduction of liquids are appointed. Puncture of cysts is used through abdominal wall under sonography control with aspiration of maintenance.

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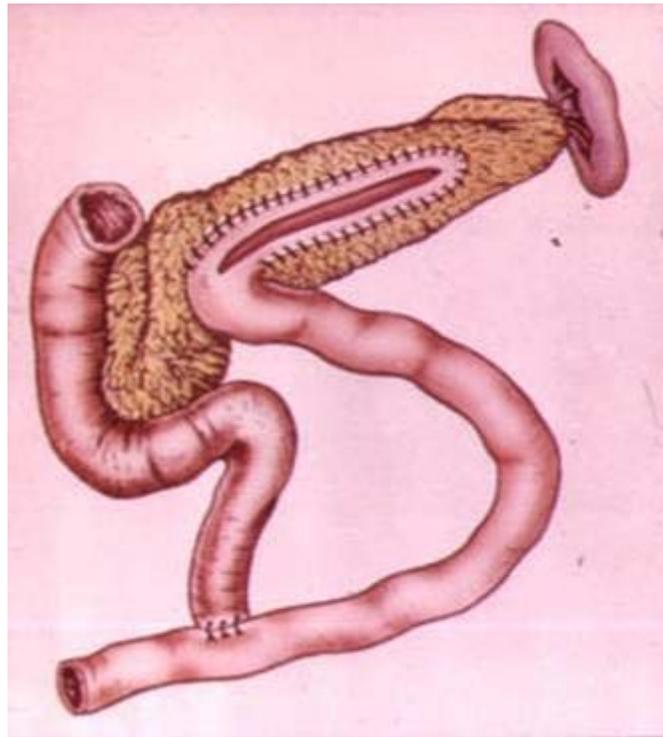


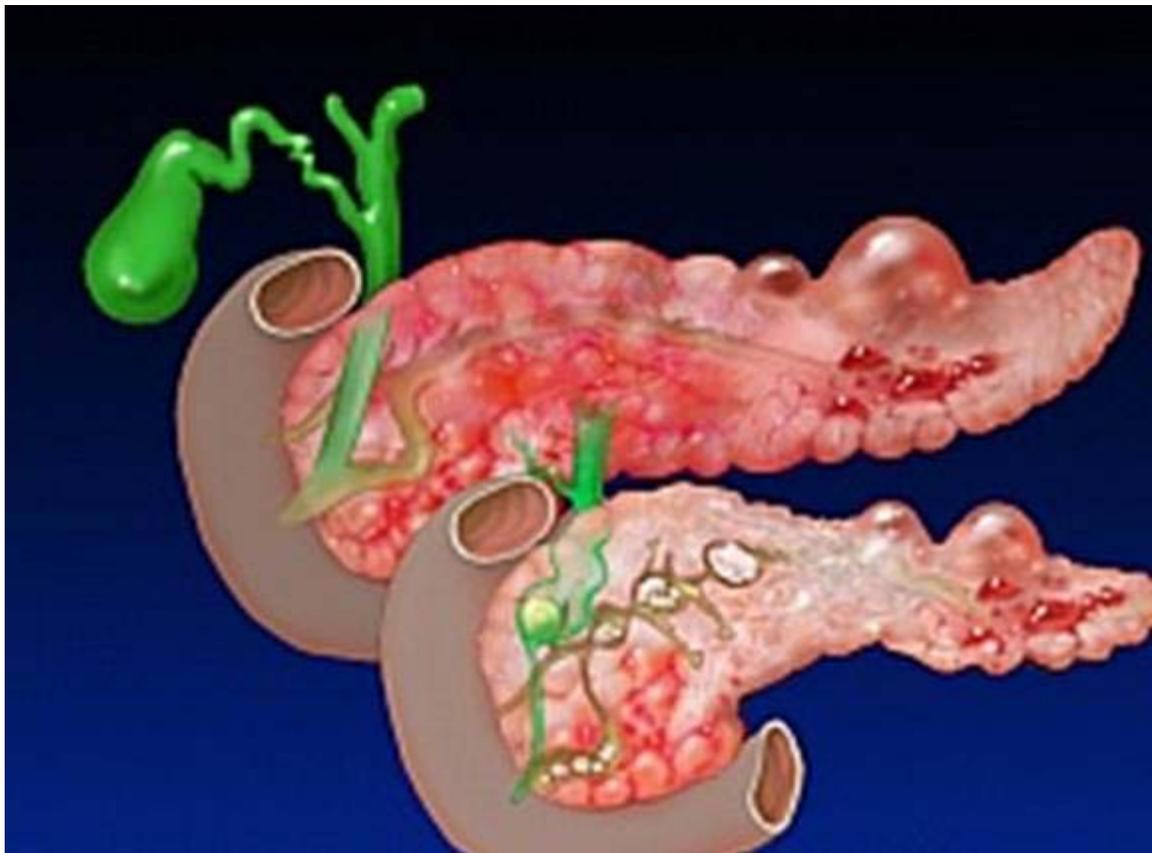
Fig.5

Cystogastrostomy are executed and cystoduodenostomy is now not applied because of possible complications (infection of cyst, erosive bleeding). Marsupialization (opening and sewing down of cyst to the parietal peritoneum and skin) is used infrequently (at suppuration of cyst is seriously patientsing with the septic state).

On the IV stage external and internal draining of cyst and radical operations are applied: a) enucleation of cysts (executed very rarely);
b) distal resection of pancreas with a cyst.

**ACUTE UNCOMPLICATED PANCREATITIS.
CHOLECYSTOPANCREATITIS. ACUTE COMPLICATED PANCREATITIS
(PANCREATITUOS PERITONITIS, PANCREONECROSIS, FALSE CYSTS
OF THE PANCREAS). TACTICS OF TREATMENT.**

ACUTE PANCREATITIS



Anatomy and function

The **pancreas** is an organ located in the abdomen. It plays an essential role in converting the food we eat into fuel for the body's cells. The pancreas has two main functions: an **exocrine** function that helps in digestion and an **endocrine** function that regulates blood sugar.

Basic Anatomy: The pancreas is located behind the stomach and is surrounded by other organs including the small intestine, liver, and spleen. It is about six inches long and is shaped like a flat pear. The wide part, called the head of the pancreas, is positioned

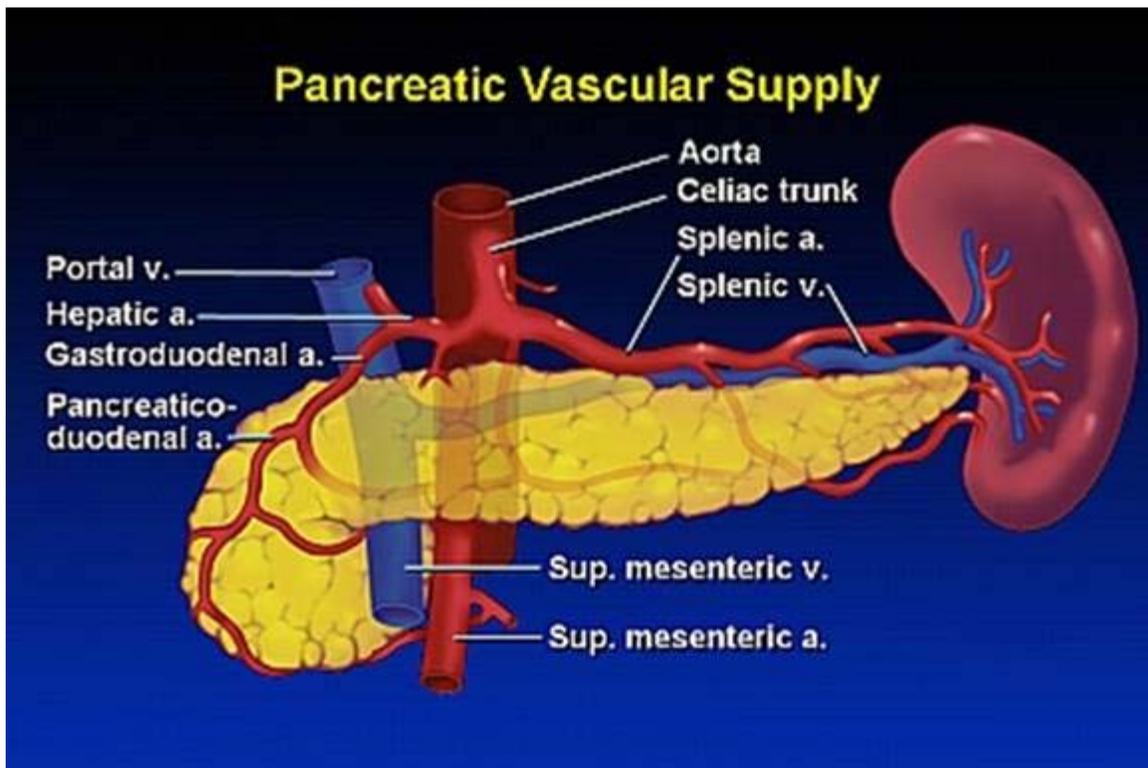
toward the center of the abdomen; the middle section is called the neck and the body of the pancreas; the thin end is called the tail and extends to the left side. Several major blood vessels surround the pancreas, the superior mesenteric artery, the superior mesenteric vein, the portal vein and the celiac axis, supplying blood to the pancreas and other abdominal organs.

Exocrine Function: The pancreas contains exocrine glands that produce **enzymes** important to digestion. When food enters the stomach, these pancreatic juices are released into a system of ducts that culminate in the main **pancreatic duct**. The pancreatic duct joins the **common bile duct** to form the **ampulla of Vater** which is located at the first portion of the small intestine, called the **duodenum**. The common bile duct originates in the liver and the **gallbladder** and produces another important digestive juice called **bile**. The pancreatic juices and bile that are released into the duodenum, help the body to digest fats, carbohydrates, and proteins.

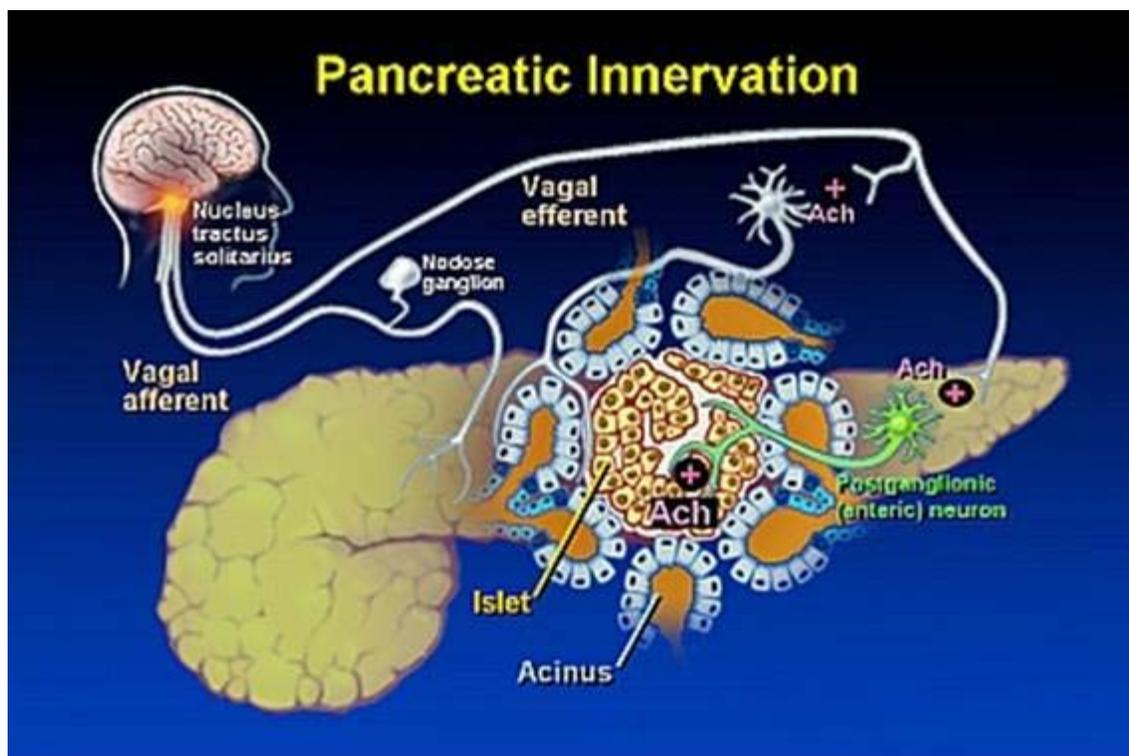
Endocrine Function: The endocrine component of the pancreas consists of islet cells that create and release important **hormones** directly into the bloodstream. Two of the main pancreatic hormones are **insulin**, which acts to lower blood sugar, and **glucagon**, which acts to raise blood sugar. Maintaining proper blood sugar levels is crucial to the functioning of key organs including the brain, liver, and kidneys.

Acute pancreatitis

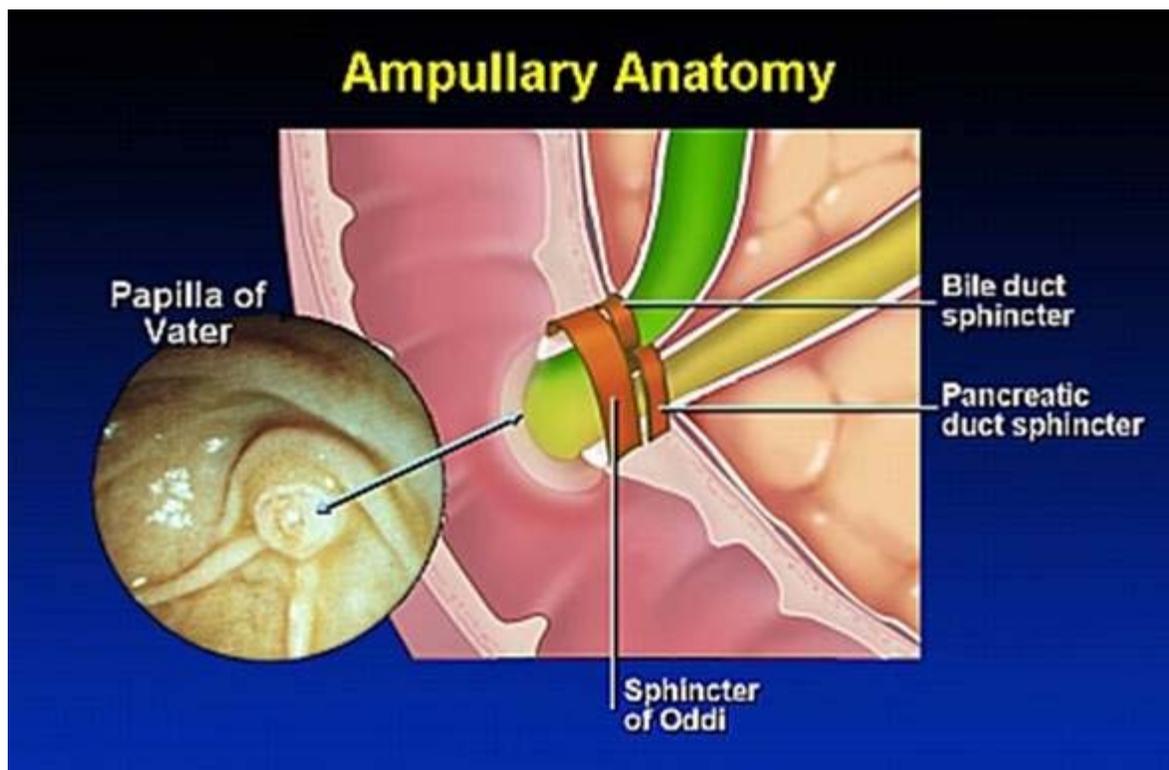
The basis of disease of pancreas is degenerative-inflammatory processes which are considered to be acute pancreatitis, the so called autolysis tissue by its own enzymes. In the structure of acute pathology of organs of abdominal cavity this disease takes the third place after acute appendicitis and cholecystitis. Women suffer from acute pancreatitis 3–3,5 times more frequently than men.



Pic. Pancreatic vascular supply.



Pic. Pancreatic Innervation

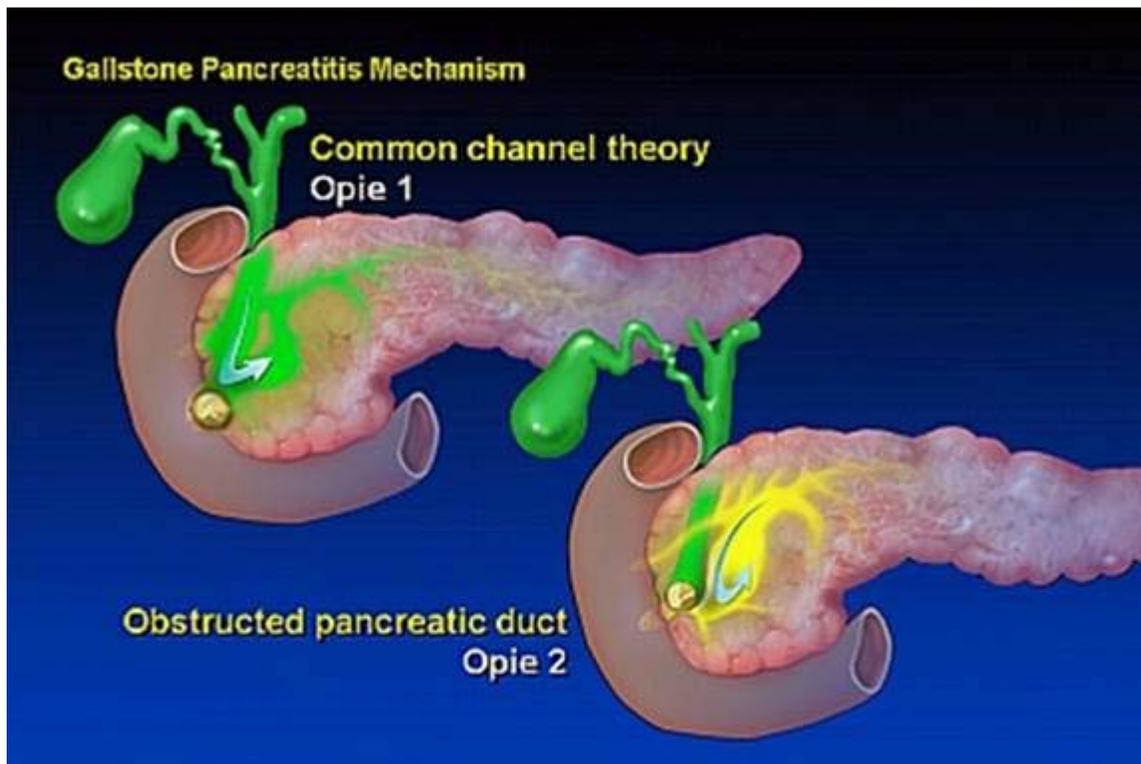


Pic. Ampullary Anatomy

Etiology and pathogenesis

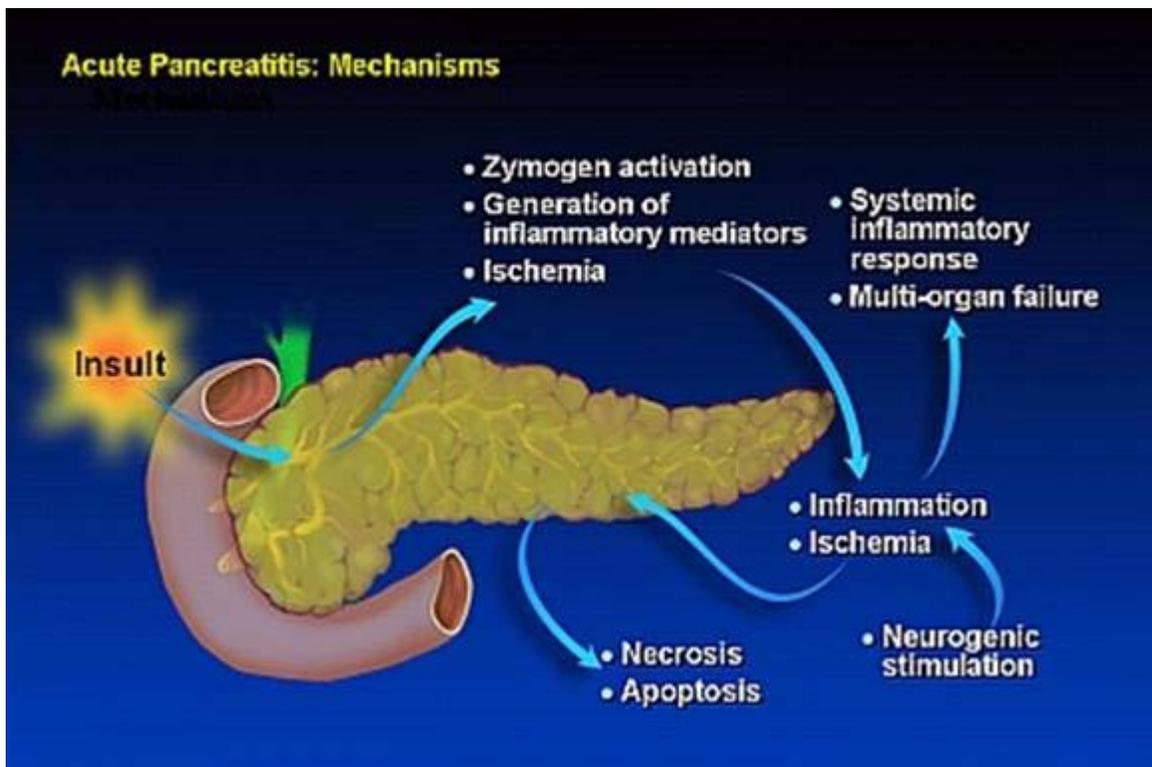
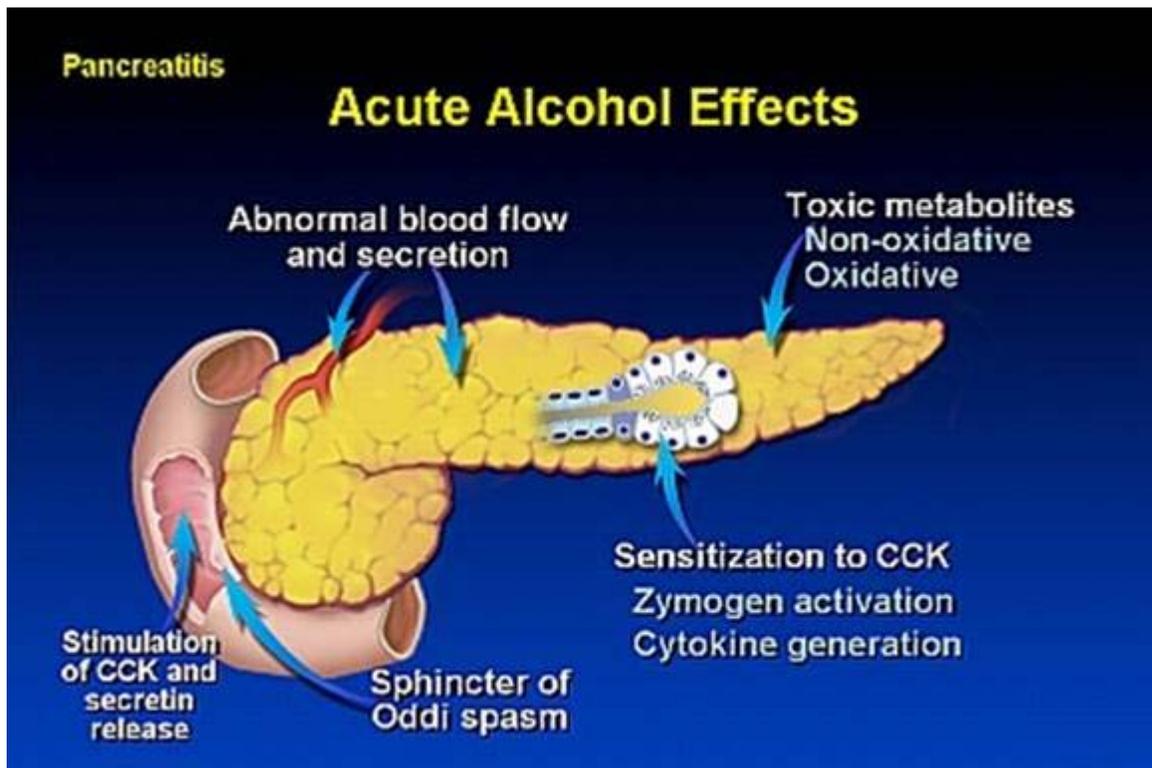
Acute pancreatitis is a polyetiology disease. Its secondary forms, which arise on the background of pathologies of bile-excreting system and duodenum are closely associated with anatomic and functionally with pancreas, and are met in clinical practice.

Among the “starting” factors of origin of cholelithiasis disease (biliary pancreatitis) abuse by an alcohol and food overloads (fat and irritating products), traumas of pancreas, operating-room in particular, and also separate infectious diseases (parotitis, mononucleosis) are most frequent, especially infection of bilious ways. However, in 10–20 % of patients the reason of acute pancreatitis remains unknown (cryptogenic form).



In the basis of such damages of pancreas and enzymic toxemia lies mainly activating of pancreatic, and then the tissue enzymes (trypsin, lipase, amylase). Often the combination of the broken outflow of pancreatic secret and promoted secretion takes place, which provokes intraductal hypertension.

Among explanations of primary mechanisms of activating of pancreatic enzymes the most value belongs to: a) theory of “general duct” with reflux of bile in the ducts of pancreas; b) blockade of outflow of pancreatic juice with development of intraductal hypertension and penetration of secret in interstitial tissue; in) violation of blood flow of pancreas (vasculitis, thrombophlebitis and embolisms, cardiac insufficiency and others like that); g) toxic and allergic damages of gland. The role of alcohol in such situations can be dual: stimulation of secretion of pancreas and direct damaging action on its tissue.



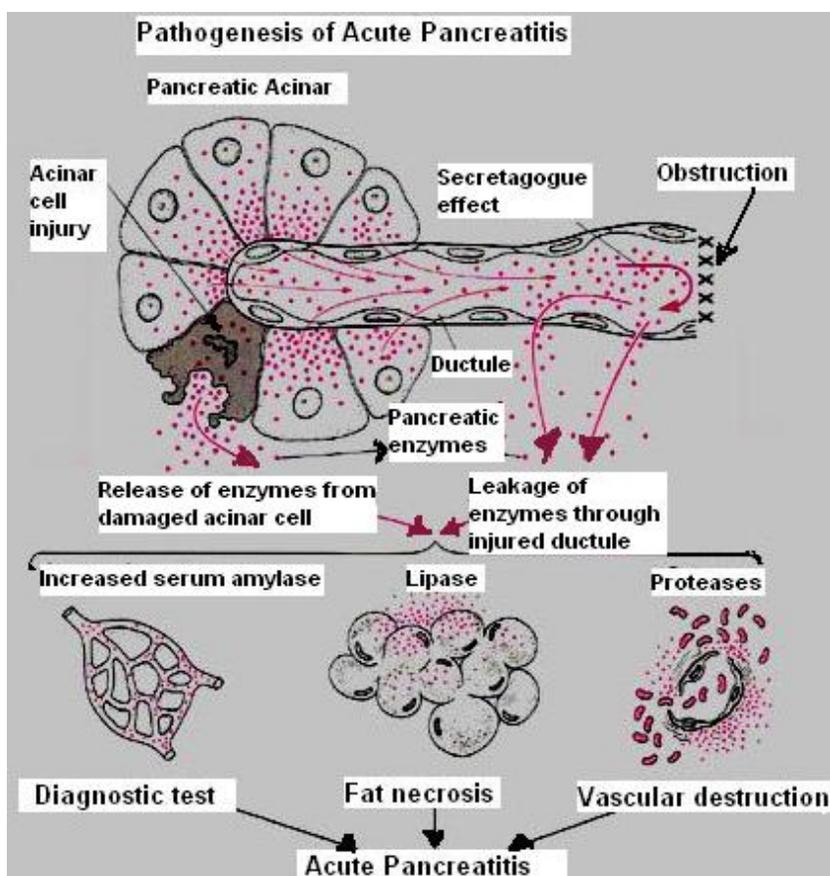
Acute pancreatitis: Mechanisms

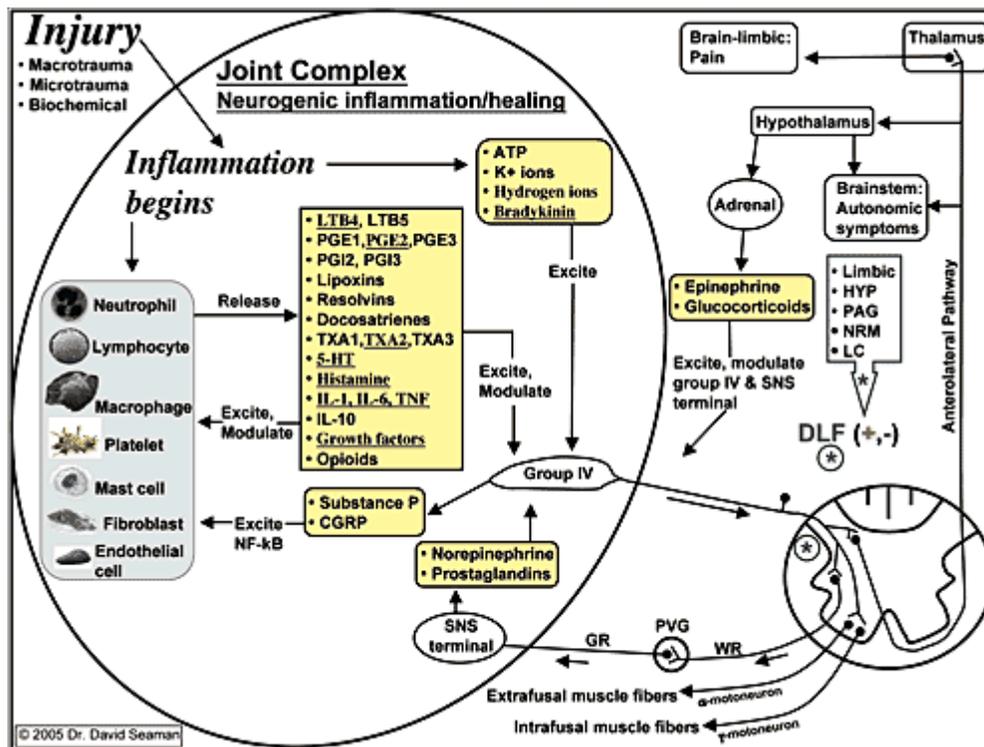
Pathomorphology

The process of acute inflammation of pancreas consistently passes the stages of edema, pancreatonecrosis and festering pancreatitis. In the stage of edema there is pancreas of hyperemic, increased in volume, with the shallow hearths of necrosis or, as it is in swingeing majority of cases, without them.

Pancreatonecrosis can pass with fatty or hemorrhagic character. In the first case, as a rule, pancreas is increased, dense, cut whity-yellow hearths are selected to necrosis. Increase of crimson-black pancreas with darkly-brown infiltrate on a cut is characteristic for hemorrhagic pancreatonecrosis.

Dystrophy of parenchyma is exposed microscopically, up to necrosis, hemorrhages, thromboses of vessels and signs of inflammatory infiltration.





Classification

(V All-russian convention of surgeons, 1978)

I. Clinico-anatomy forms:

1. Arching form.
2. Fatty pancreatonecrosis.
3. Hemorrhagic pancreatonecrosis.

II. Prevalence of necrosis:

1. Local (focus) damage of gland.
2. Subtotal damage of gland.
3. Total damage of gland.

III. Ran across: abortive, progressive.

IV. Periods of disease:

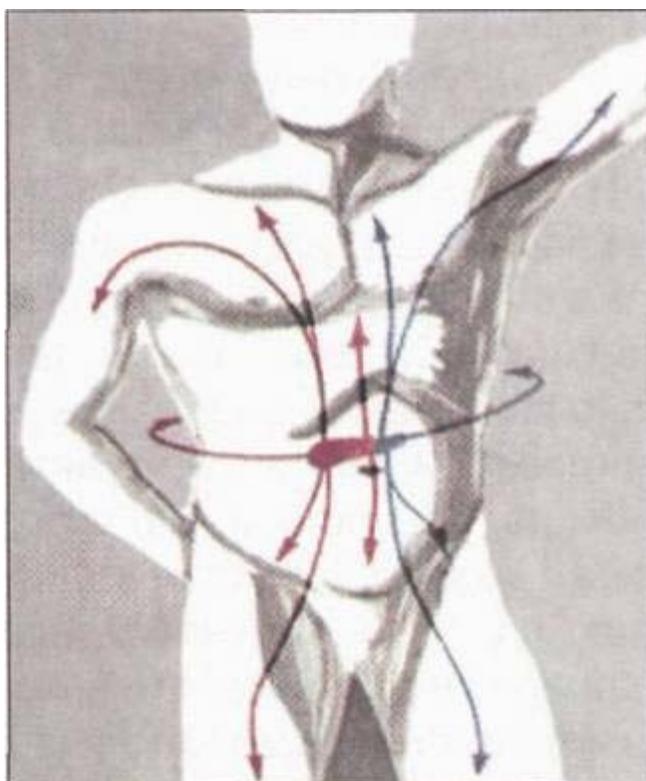
1. Period of hemodynamic violations and pancreatogenic shock.

2. Period of functional insufficiency of parenchymatous organs.
3. Period of degenerative and festering complications.

Symptoms and clinical passing

The disease begins suddenly, after the surplus reception of rich spicy food and use of alcohol. Pain, vomiting and phenomena of dynamic intestinal obstruction are considered the most characteristic signs of acute pancreatitis.

A stomach-ache is permanent and so strong, that can result in shock, localized in an epigastric area and left hypochondrium. Some patients feel pain in right hypochondrium with irradiation in the back, loin or breastbone.



Pain irradiation on acute pancreatitis

In a short period of time after appearance of pain there is a repeated strong vomiting, that does not facilitate the state of patient.

In general vomiting is considered a frequent and characteristic symptom. It is

repeated or continuous and never brings facilitation. Vomit masses contain bile, as admixture, and at the difficult form of acute pancreatitis remind “coffee-grounds”.

Nausea, hiccup, belch and dryness in a mouth are attributed as less characteristic symptoms of this pathology.

During the examination the skin is pale, often subicterus. Some patients have cyanosys with a “marble picture” as a result of violation of microcirculation. Later the component of respiratory insufficiency can join it. At progressive general condition the patient quickly gets worse to passing of acute pancreatitis, intoxication grows. The skin takes shelter with sticky sweat.

The temperature of body of patients at the beginning of disease can be normal. It rises at resorption of products of autolysis tissue and development of inflammatory process in bilious ways.

The pulse in most cases is at first slow, then becomes frequent, notably passing ahead the increase of temperature of body.

Arterial pressure goes down.

The tongue in the first hour of disease is moist, assessed by white and grey raid. At vomiting by bile the raid has yellow or greenish tint.

The abdominal is blown away, peristaltic noises are loosened. The signs of paresis of stomach and intestine demonstrate early. They need to be included in the pathological process of mesentery root of bowel. At palpation painfulness in an epigastric area and in right, and sometimes and in left, hypochondrium is marked. However, in spite of great pain in stomach, it remains soft for a long time. A little later there is moderate tension or resistance of muscles of front abdominal wall.

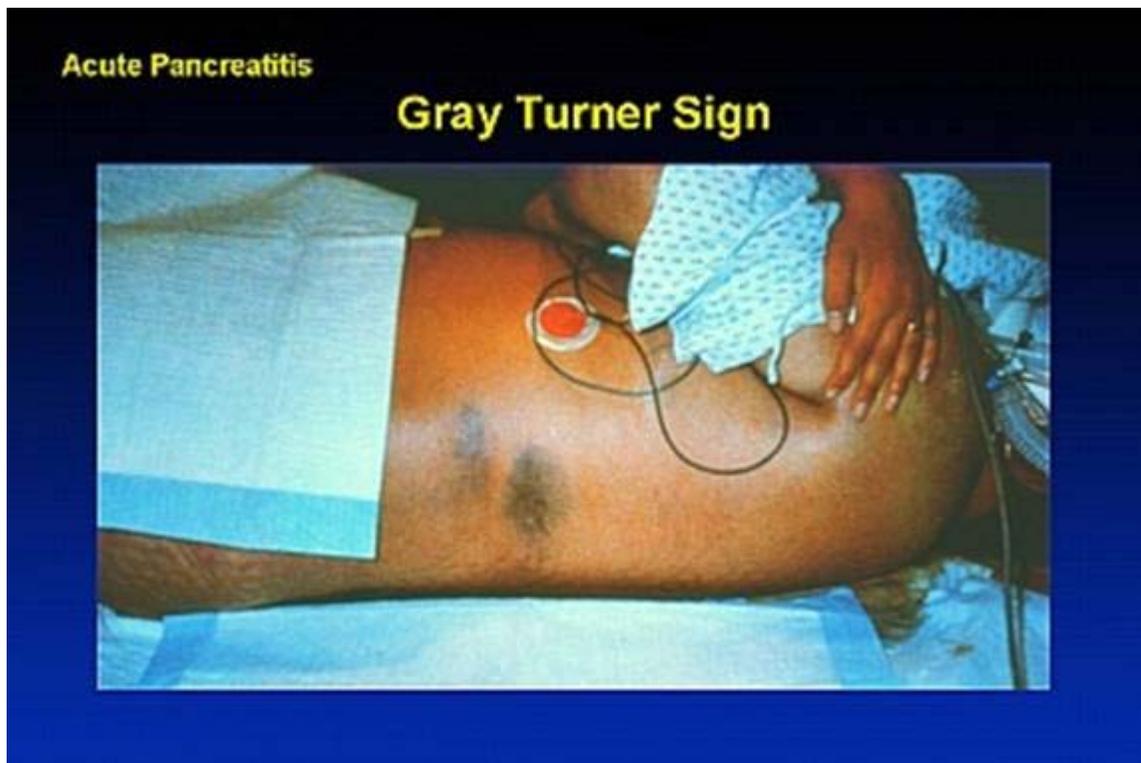
Poor local symptoms during heavy intoxication are characteristic for the early period of acute pancreatitis. Later there are symptoms of irritation of peritoneum, and at percussion dulling is marked in lateral parts of abdominal as a result of accumulation of liquid, and also the sign of aseptic phlegmon of retroperitoneal cellulose as slurred or edema of lumbar area is seen. For diagnostics of acute pancreatitis there is the row of characteristic symptoms which have different clinical value.

The Mondor's symptom is violet spots on face and trunk.

The Lagermph's symptom is acute cyanosis of person.

The Halsted's Symptom is cyanosis of abdominal skin.

The Gray's symptom is cyanosis of lateral walls of abdomen.



The Kullen's symptom is the yellow colouring of skin near a belly-button.

The Korte's symptom is painful resistance as a lumbar bar in a epigastric area on 6–7 cm higher belly-button.

The Voskresynskyy's symptom is absence of pulsation of abdominal aorta in an epigastric area.

The Mayo-Robson's symptom is feeling of pain at pressure by fingers in the left costal-vertebral corner.

The Rozdolskyy's symptom — painfulness at percussion above pancreas.

The Blumberg's symptom — in patients with acute pancreatitis more frequently is low-grade. Such feature of this sign of irritation of peritoneum needs to be explained by character of localization of pathological process, mainly in retroperitoneal spacious.

In clinical passing of pancreatonecrosis it is possible to select three periods (V.S.

Saveljev, 1978).

The I period (hemodynamic violations and pancreatogenic shock) lasts during 2–3 days. Violation of central hemodynamics, diminishment of volume of circulatory blood and disorders of microcirculation, which at first arise as a result of angiospasm, are considered the most characteristic signs, and later as a result of joining of the intravascular rolling up and laying of elements of blood.

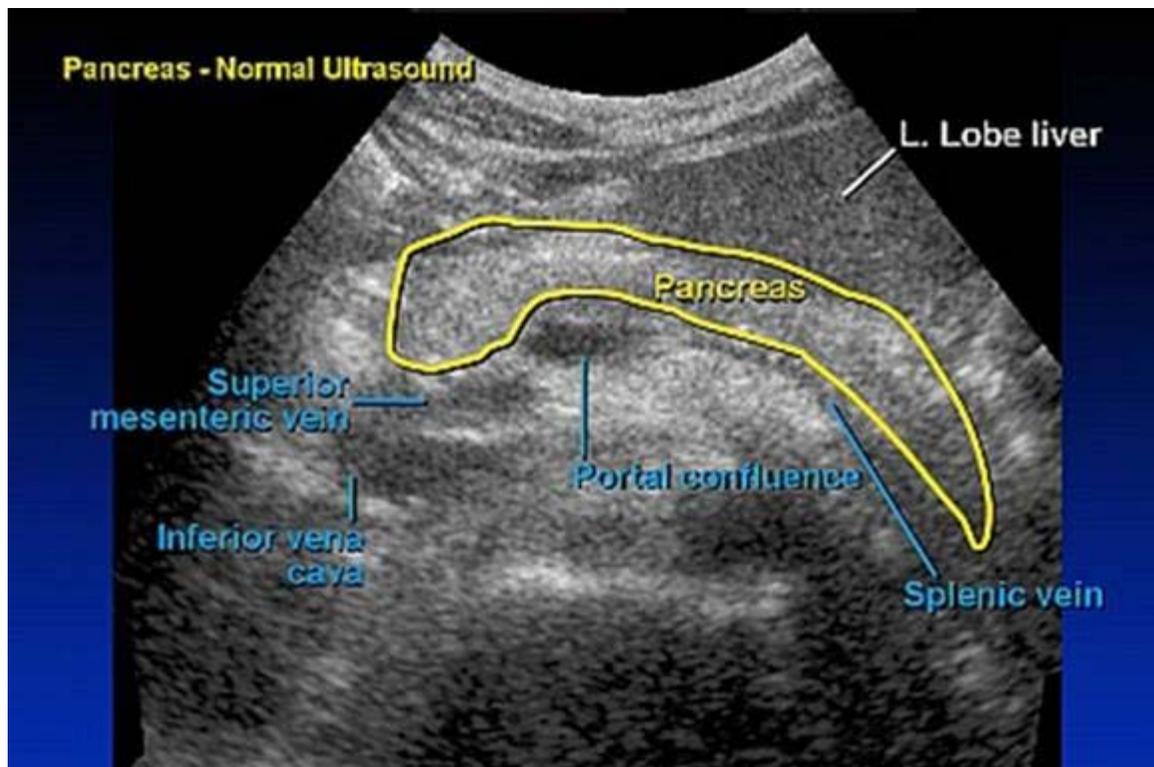
The II period (insufficiency of parenchymatous organs) lasts from 3rd to the 7th day of disease. Violation of functions of basic organs and systems, sign of cardio-vascular, hepatic and kidney insufficiency and growth of violations of breathing are thus observed. In this period there is possible damaging of the central nervous system, which is erected mainly to disorders of psyche, appearances of delirium and commas which in the eventual result are the main reasons of patients' death.

The III period (postnecrosis dystrophic and festering complications) comes in 1–2 weeks after the beginning of disease. During it, on the background of progress of necrosis processes in pancreas, the regenerative changes develop, there are parapancreatic infiltrate and cysts, cystic fibrosis of pancreas. Aseptic retroperitoneal phlegmon which strengthens intoxication can also develop. There is festering pancreatitis at joining of infection. During this period such complications, as erosive bleeding, internal or external fistula, retroperitoneal phlegmon, can develop in patients.

From laboratory information leucocytosis which at the necrosis and hemorrhagic forms of pancreatitis sometimes arrives at $25-30 \times 10^9$, lymphopenia, change of leukocytic formula to the left and the increased ESR are characteristic. Growth of activity of amylase of blood and urine is very often marked, and is the important sign of pancreatitis. For estimation of the state of other organs maintenance of general albumen and its factions, glucose of blood, bilirubin, urea, electrolytes, acid-base equilibrium (ABE), and also the state of blood coagulation are determined. It is necessary to mark that the exposure of hypocalcemia is considered a bad predictive sign of heavy passing of acute pancreatitis.

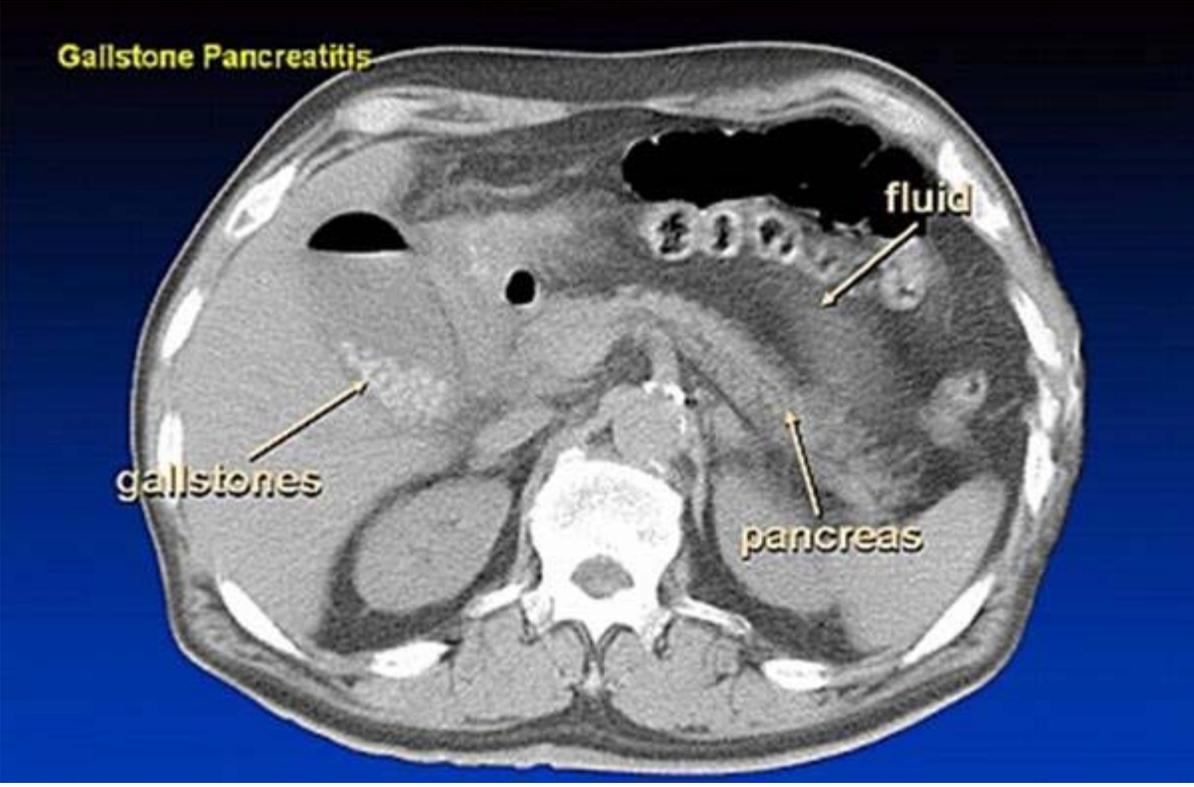
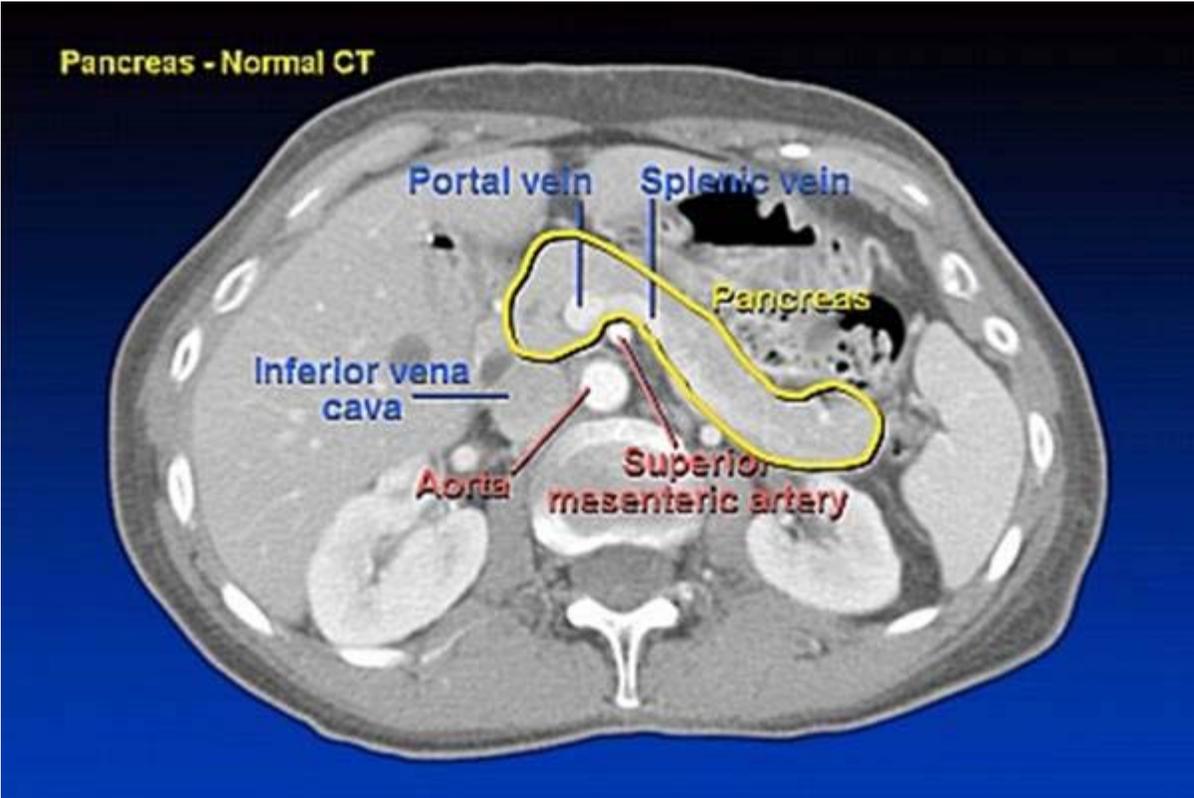
Ultrasonic examination of gall-bladder and pancreas often specifies the increase of their sizes, bulge of walls and presence or absence of concrement of gall-bladder and

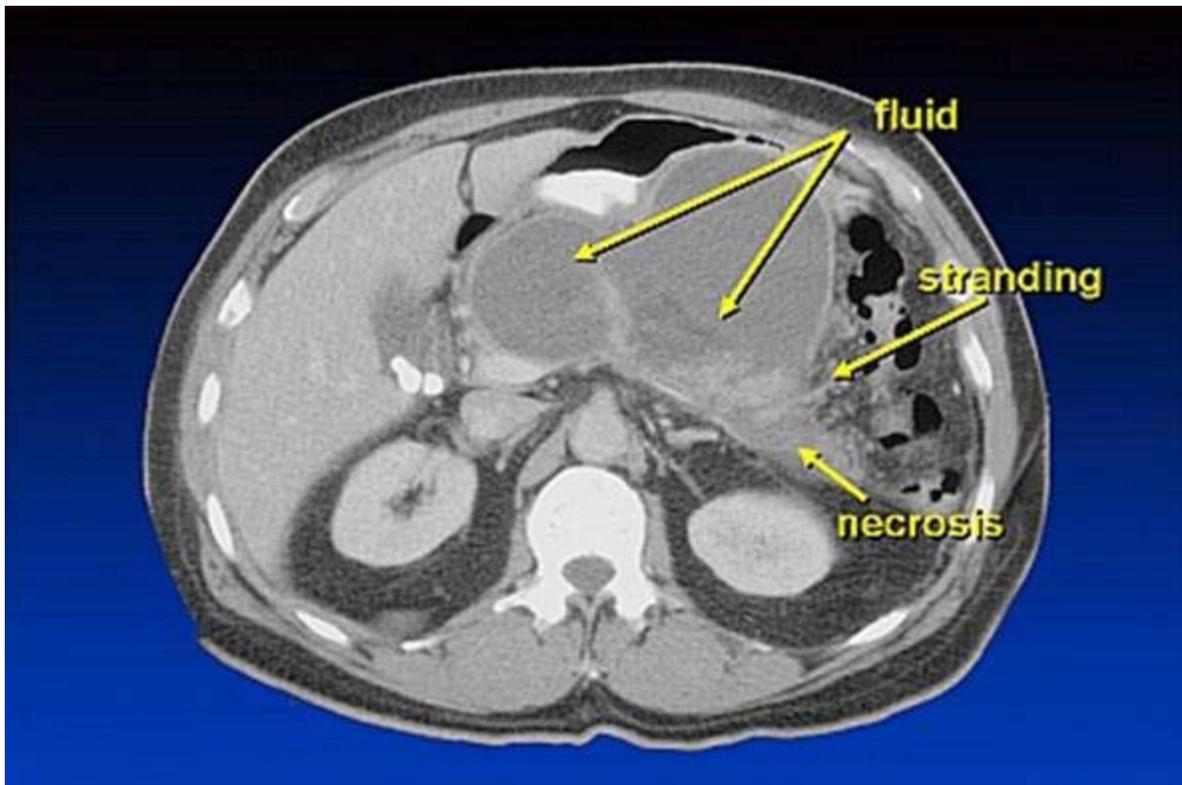
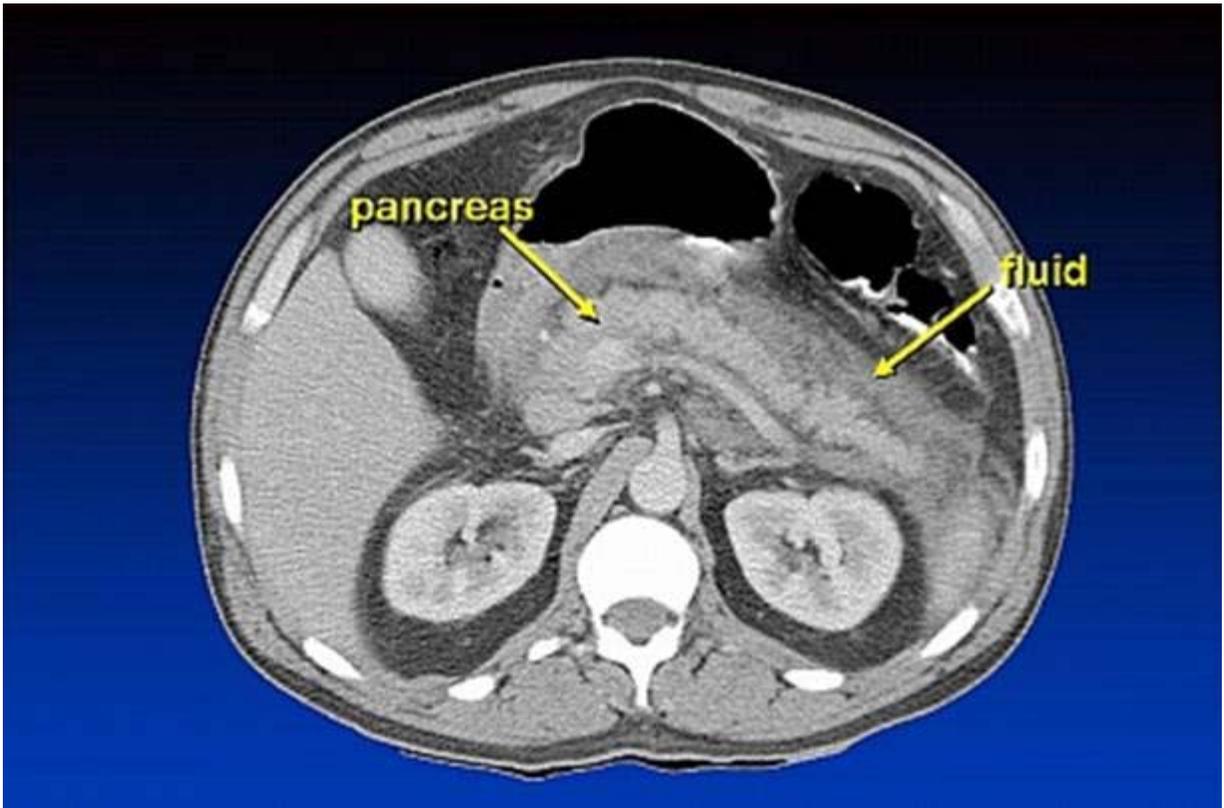
general bilious duct.



Pic. Sonography.

Computer tomography enables to describe in details the changes in pancreas and surrounding organs.



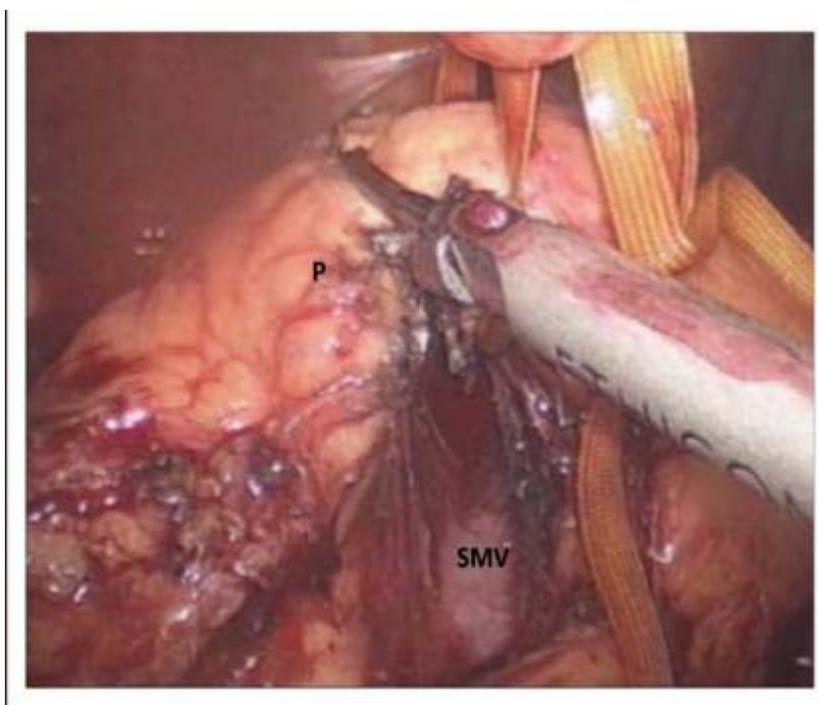


Pic. Computer tomography

At sciagraphy survey of organs of abdominal cavity gives a possibility to expose the

unfolded “horseshoe” of duodenum, pneumatization, expansion of transverse colon (*the Gobia's symptom*). On the 1st stage of diagnostics in the plan of differential diagnosis of acute destructive pancreatitis with other diseases of abdominal cavity, diagnostics of distribution of destructive damaging of different parts of pancreas and estimation of distribution of parapancreatitis is possible only by the method of computer tomography which depending on clinico-laboratory signs and weight of passing is needed to apply in a different period, and sometimes a few times in dynamics with interval of 4–5 days.

Laparoscopy and laparocentesis are often used for a doubtful diagnosis or necessity of taking away the exudation of abdominal cavity for biochemical or bacteriological examination.



Retrograde endoscopic cholangiopancreatography is used in case of mechanical icterus and suspicion of choledocholithiasis. The last methods are invasive and can if it is necessary transform from diagnostic to manipulation treatments: laparoscopic draining of abdominal cavity at pancreatogenic peritonitis and endoscopic papillotomy at choledocholithiasis and biliary pancreatitis.

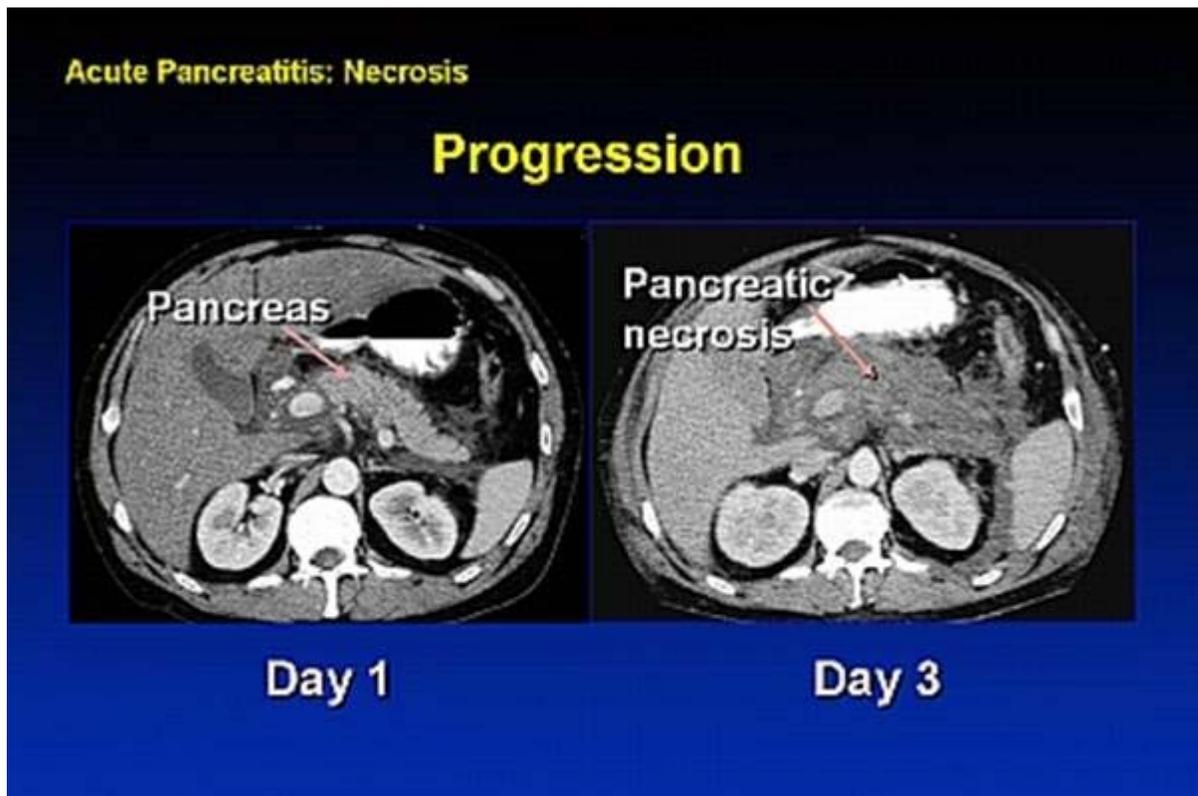


Variants of clinical passing and complications

Clinical passing of disease can be abortive, slowly or quickly progressive. At *abortive passing* the process is limited to acute edema of pancreas with convalescence in 7–10 days.

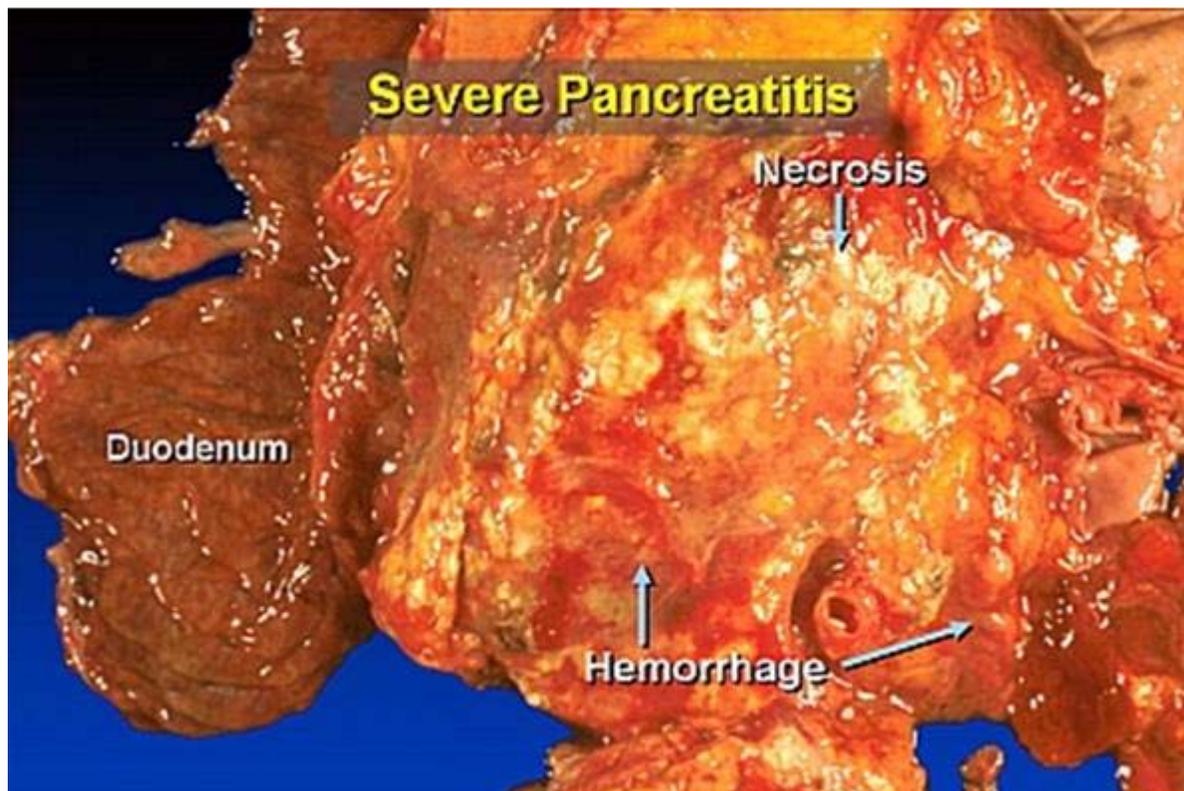
Rapid progress is characteristic for pancreatonecrosis. In patients expressed toxemia, impregnation by exudation of retroperitoneal cellulose and development of fermentative hemorrhagic peritonitis can be seen. Strengthening of stomachache, continuous vomiting, proof paresis of intestine, positive symptoms of irritation of peritoneum and growth of hemodynamic violations are the clinical signs of necrosis of pancreas.

There is a formation of parapancreatic infiltrate at *slow progress*.



Among early complications of acute pancreatitis shock, peritonitis and acute cardiac, pulmonary, hepatic and kidney insufficiency can be distinguished.

Before later complications it is needed to deliver the abscesses of pancreas, subdiaphragmatic, interintestinal abscesses, pyogenic abscess omentum bag, phlegmons of retroperitoneal space and erosive bleeding.



In future formations of pseudocysts, fistula of pancreas, intestinal fistula and development of saccharine diabetes are possible.

Diagnosis program

1. Anamnesis and physical methods of inspection.
2. General analysis of blood and urine.
3. Biochemical blood test (amylase, bilirubin, sugar).
4. Analysis of urine on diastase.
5. Sonography.
6. Computer tomography.
7. Cholecystocholangiography.
8. Endoscopic retrograde cholangiopancreatography.
9. Laparoscopy.
10. Laparocentesis.

Physical Examination:

The following physical examination findings may be noted, varying with the severity of the disease:

- Fever (76%) and tachycardia (65%) are common abnormal vital signs; hypotension may be noted
- Abdominal tenderness, muscular guarding (68%), and distention (65%) are observed in most patients; bowel sounds are often diminished or absent because of gastric and transverse colonic ileus; guarding tends to be more pronounced in the upper abdomen
- A minority of patients exhibit jaundice (28%)
- Some patients experience dyspnea (10%), which may be caused by irritation of the diaphragm (resulting from inflammation), pleural effusion, or a more serious condition, such as acute respiratory distress syndrome (ARDS); tachypnea may occur; lung auscultation may reveal basilar rales, especially in the left lung
- In severe cases, hemodynamic instability is evident (10%) and hematemesis or melena sometimes develops (5%); in addition, patients with severe acute pancreatitis are often pale, diaphoretic, and listless
- Occasionally, in the extremities, muscular spasm may be noted secondary to hypocalcemia

A few uncommon physical findings are associated with severe necrotizing pancreatitis:

- The Cullen sign is a bluish discoloration around the umbilicus resulting from hemoperitoneum
- The Grey-Turner sign is a reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting along tissue planes; more commonly, patients may have a ruddy erythema in the flanks secondary to extravasated pancreatic exudate
- Erythematous skin nodules may result from focal subcutaneous fat necrosis; these are usually not more than 1 cm in size and are typically located on extensor skin surfaces; in addition, polyarthrititis is occasionally seen

Rarely, abnormalities on fundoscopic examination may be seen in severe

pancreatitis. Termed Purtscher retinopathy, this ischemic injury to the retina appears to be caused by activation of complement and agglutination of blood cells within retinal vessels. It may cause temporary or permanent blindness.

Differential diagnostics

- Acute Mesenteric Ischemia
- Acute Respiratory Distress Syndrome
- Bacterial Pneumonia
- Cholangitis
- Cholecystitis
- Choledocholithiasis
- Cholelithiasis
- Chronic Pancreatitis
- Colon Adenocarcinoma
- Colonic Obstruction
- Community-Acquired Pneumonia
- Duodenal Ulcers
- Gastric Cancer
- Gastroenteritis in Emergency Medicine
- Irritable Bowel Syndrome
- Myocardial Infarction
- Pancreatic Cancer
- Pancreatic Pseudocysts
- Viral Hepatitis in Emergency Medicine

Acute pancreatitis needs to be differentiated with the row of acute diseases of organs of abdominal cavity.

Differential Diagnosis of Acute Pancreatitis		
Disease	Characteristics	Findings
Perforated viscus, especially peptic ulcer	Sudden onset of pain that increases over 30-60 min	Intraperitoneal air present
Acute cholecystitis and biliary colic	Epigastric or right upper quadrant pain that radiates to right shoulder or shoulder blade	Liver enzymes often elevated; ultrasonography may show thickened gallbladder, pericholecystic fluid
Intestinal obstruction	Constant colicky pain	Obstructive pattern can be seen on CT scan or abdominal series
Mesenteric vascular occlusion	Classic triad is postprandial abdominal pain, weight loss, and abdominal bruit	Discrepancy between symptoms (severe pain) and examination (benign abdominal examination)
Dissecting aortic aneurysm	Sudden onset; pain may radiate to the lower extremities	
Renal colic	Flank pain radiates to the genitals; dysuria may be present	Urinalysis with active sediment
Myocardial infarction	Upper abdominal or chest pain	Electrocardiography usually abnormal
Connective tissue disorders with vasculitis	Acute pancreatitis can be due to vasculitis	Other signs of vasculitis usually present (skin, joint, eye, and kidney involvement)
Appendicitis	Pain may start in epigastrium or periumbilical then migrate to right lower quadrant	Ultrasonography and and CT aid in diagnosis
Ectopic pregnancy	Sudden onset of pain; menstrual abnormalities often precede pain	Rapid drop in hematocrit and intraperitoneal pelvic fluid on imaging should raise suspicion
Pneumonia	Fever, malaise, and other respiratory symptoms (dyspnea, cough, sputum production, chest pain) usually present	Changes on physical examination of the chest and abnormalities on chest X-ray possibly due to ARDS or pleural effusion

AP = acute pancreatitis; ARDS = acute respiratory distress syndrome; CT = computed tomography; HCT = hematocrit.

Acute mechanical intestinal obstruction. In patients with this pathology pain is of the alternated character and is accompanied by nausea, vomiting, delay of gases and emptying. It is possible to see the Kloyber bowls on the sciagram survey of organs of abdominal cavity.

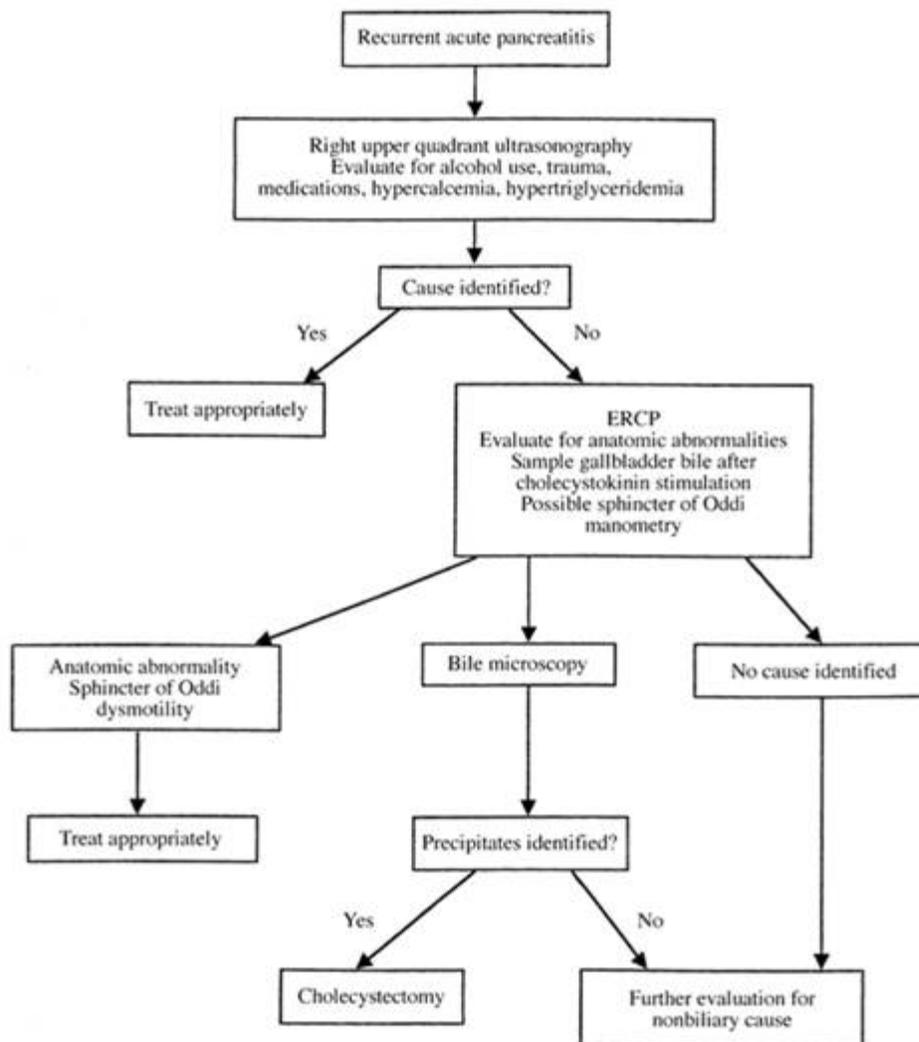
Acute cholecystitis runs with characteristic localization of pain and muscular defense, with presence of increased, painful gall-bladder or infiltrate in right hypochondrium. Often acute (especially lately) pancreatitis develops on the background of gallstone disease (biliary pancreatitis).

Thrombosis or embolism of mesenteric vessels. Both for pancreatitis and for the thrombosis of mesenteric vessels great pain at soft abdomen (absence of defense muscles of front abdominal wall), that precedes to development of peritonitis, is inherent. Yet from the beginning the disease gains heavy character of passing. In anamnesis in such patients a heart disease or heart attack of myocardium rheumatic is met. As a result of gangrene of intestine, the symptoms of peritonitis appear very quickly and intoxication grows. The fragments of mucus shell are found in flushing waters of intestine at the detailed examination, which have the appearance of "meat flushing".

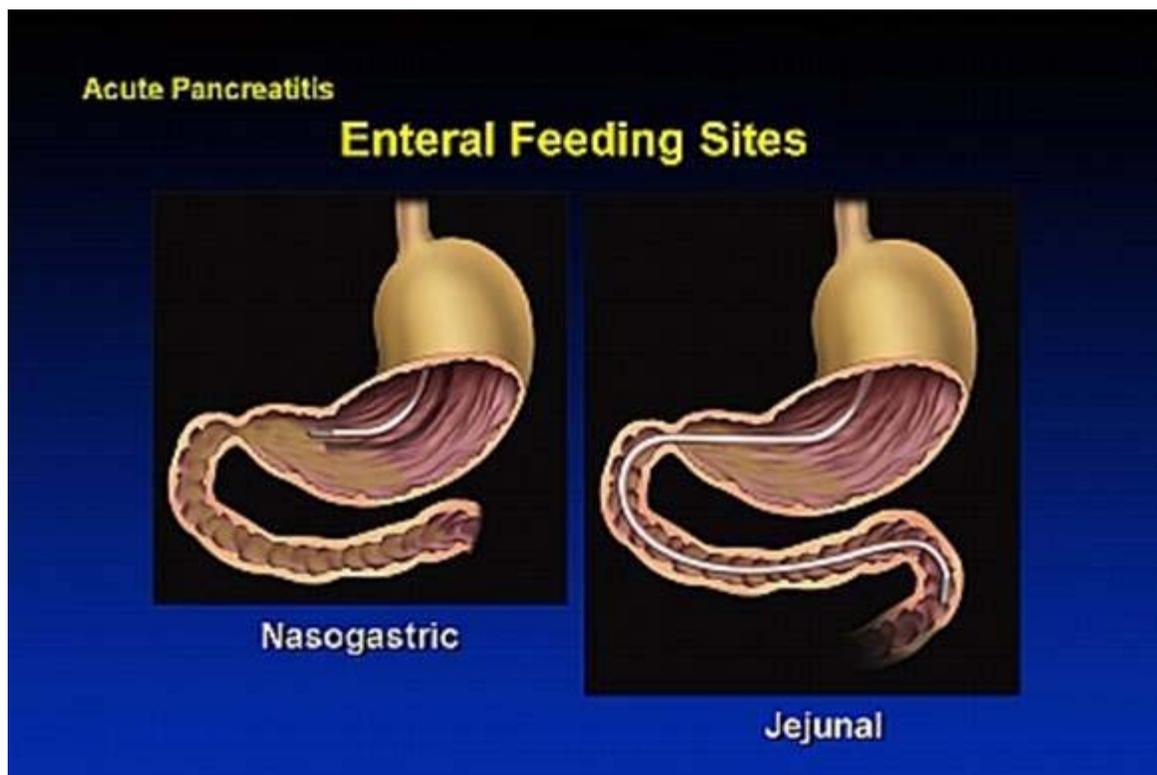
A perforated gastric and duodenum ulcer is distinguished by the presence of dagger pain, defense of abdominal wall, ulcerous anamnesis.

Tactics and choice of treatment method

The conservative method is considered the basic one for treatment of acute pancreatitis, but in connection with that unsuccessful conservative treatment of patients with acute pancreatitis can often put a question about the necessity of operation, therefore patients must be in permanent surgical establishment. Thus acute pancreatitis with heavy passing is necessary to be treated under the conditions of separation of intensive therapy.



Before conservative treatment hunger, bed rest, fight against pain and enzymic toxemia, conducting of acid-base state, prophylaxis of festering infection and acute ulcers of digestive duct are to be entered .



Patient's stomach is washed by cold soda solution and a cold on an epigastric area and left hypochondrium is used. Medicinal therapy is prescribed also: spasmolytics (papaverine, platyphyllin, no-shparum, baralgine, atropine); inhibitor of protease (contrical, trasilol, gordox, antagosan); cytostatic agent (5-fluorouracil, ftorafur). Positive action of inhibitor of protease is marked only in the first days of disease which are subject to conditioned application of large doses. Antibiotics of wide spectrum of action: a) tienam, which most effective in the prophylaxis of festering pancreatitis, as is selected by pancreatic juice; b) cephalosporins (kefzol, cefazoline); c) cefamizine (mefoxine).

Disintoxication therapy is conducted also (5 % but 10 % solutions of glucose, hemodes, reopolyhluquine, polyhluquine, plasma of blood, only from 3 to 5 liters on days, in accordance with a necessity).

For the improvement of rheological properties of blood heparine is prescribed (5 000 ODES every 4 hours).

If patients have the expressed pain syndrome and phenomena of general intoxication during all pain period plus 48 hours (by Bakulev), hunger is used. Such mode lasts on the average of 2–4 days. The parenteral feed of albuminous hydrolyzate is thus

conducted, by the mixtures of amino acid and fatty emulsion. Alkaline water of to 1–2 l. and albuminous-carbohydrate diet are also appointed. Infusion therapy is complemented by plasma, by albumen, hemodes, reopolyhluquine. The improvements of microcirculation in pancreas are achieved due to introduction of reopolyhluquine, komplamine, trental and heparin 5000 ODES 6 times per days under the control the indexes of the coagulation system of blood. Anticholinergic drug (sulfate of atropine, methacin, platyphyllin), H₂-histamin blocker (cimetidine, ranisan, ranitidine, famotidine, omeprazol) are also applied. For the removal of pain: 1) sulfate of the atropine 0,1 % — 1 ml + promedol 2 % — 1 ml + papaverine 2 % — 2 ml + analgin 50 % — 2 ml; 2) isotonic solution of chloride of sodium — 500 ml + baralgin — 5 ml + diphenhydramine hydrochloride 1 % — 1 ml + papaverine 2 % — 2 ml + magnesium the sulfate 25 % — 5 ml + ascorbic acid — 5 ml + lipoic acid 0,5 % — 2 ml + novocaine 0,5 % — 10 ml. are used. From the first days by a nasogastral probe the permanent aspiration of gastric maintenance is conducted also. The Motility function of gastro-intestinal highway gets better at application of cerucal or primperane. With the same purpose forced diuresis (maninil, furosemide, aminophylline) is used on the background of intravenous introduction of plenty of liquid.

At uneffective conservative treatment of patients with acute pancreatitis of middle weight and heavy form it is expedient to apply surgical treatment.

Surgical treatment is carried out for patients with biliary pancreatitis (for a day long from the beginning of disease) in combination with the destructive forms of cholecystitis, at complications of acute pancreatitis by peritonitis, abscess of omentum bag or phlegmon of retroperitoneal cellulose.

Overhead-middle laparotomy, which allows to estimate the state of pancreas, bilious ways and other organs of abdominal cavity, is the best access in this situation. In case of destructive pancreatitis the possible use of lumbar laparotomy from left to right hypochondrium through a mesogastric area is useful.

Cholecystectomy is executed at calculous cholecystitis, phlegmonous inflammation of walls of gall-bladder and biliary pancreatitis. If there are more than 0,9 cm at expansion of choledochus, presence of concrement, ointment-like bile in it, increase of

concentration of bilirubin in the whey of blood over 21 mmol/L, choledocholithotomy is complemented by external draining of choledochus. Information of lithiasis of general bilious duct is absent, cholecystectomy in patients with acute pancreatitis is complemented by external draining of choledochus, better by Pikovskyy method (through stump of cystic duct).

Transduodenal sphincteroplasty is shown at fixed concrement of large duodenal papilla, if they are diagnosed intraoperative, and also in the cases of papillotomy with extraction of concrement when there is no possibility to execute endoscopic operation .

Omentopancreatopexy. After laparotomy and cutting of gastro-colon and gastro-pancreatic ligament mobile part of large omentum through opening in gastro-colon ligament is conducted and fixed by separate stitches to the peritoneum along the overhead and lower edges of pancreas. Such operation needs to be considered rational at the expressed edema of pancreas and presence of necrosis in it.

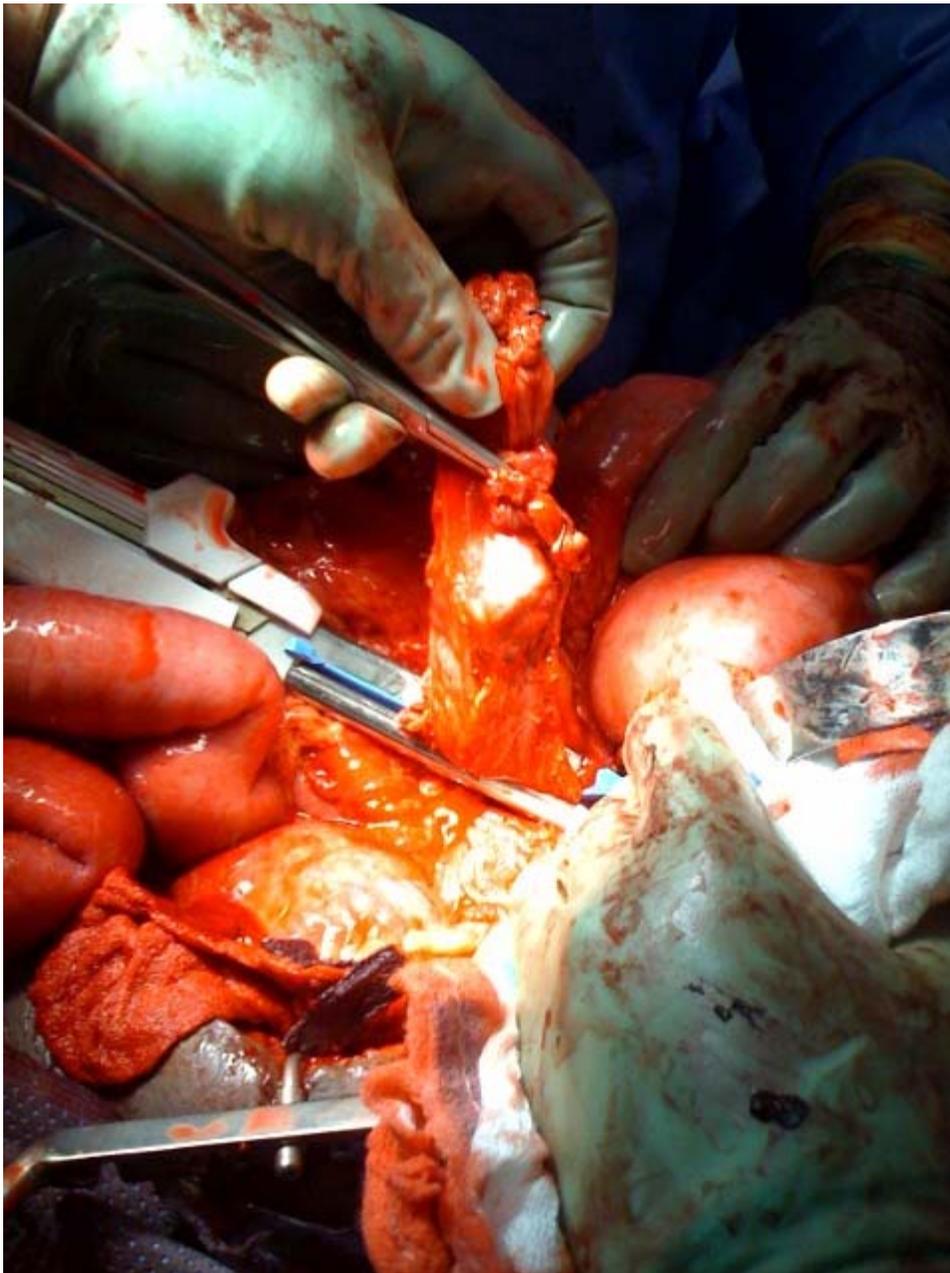
Abdominisation of pancreas. A cellulose round pancreas (along the lower and overhead edges of body and tail) is infiltrated by solution of novocaine, after it parietal peritoneum is cut. Under the body and tail glands free end of omentum is conducted and is bundled by a gland. This operation is able to warn the hit of enzymes and products of disintegration in retroperitoneal space.

Sequestrectomy is deleting of necrosis part of gland within the limits of nonviable tissue. Operation is executed in a dull way.

Necrectomy (deleting of necrosis part of gland within the limits of healthy tissue) is executed by an acute way: tissue of gland is cut on verge of necrosis and bleeding vessels are carefully bandaged.

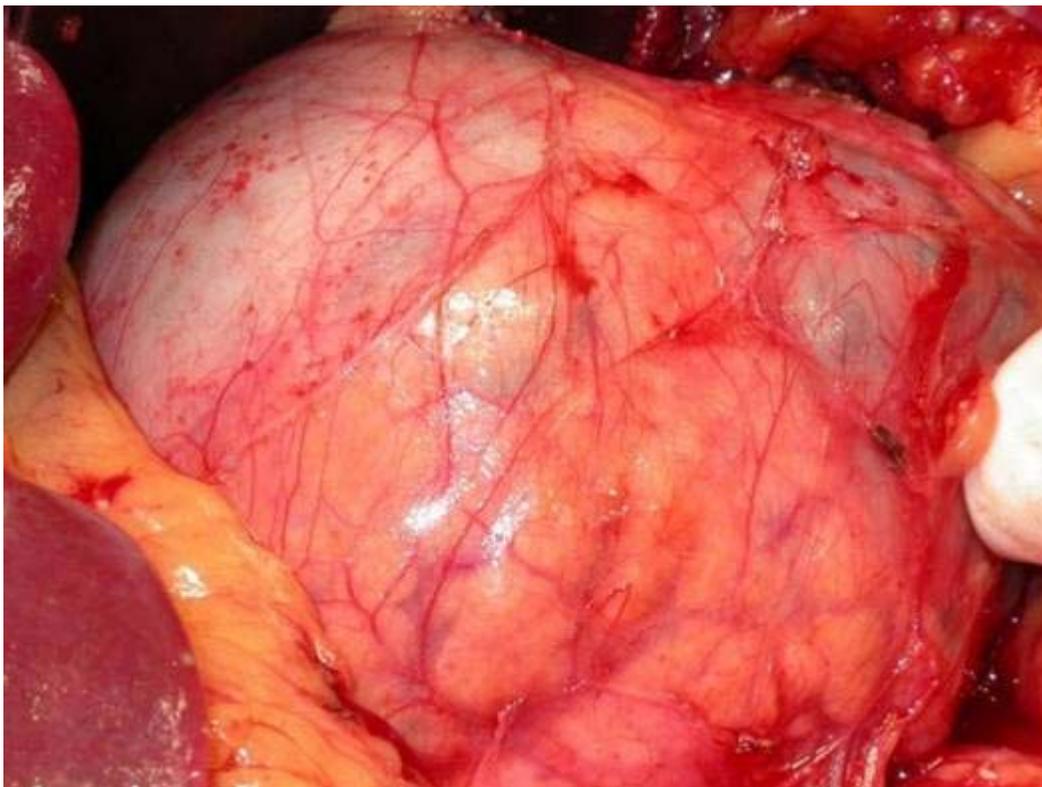
The resection of pancreas is deleting the part of organ with its transversal cutting within the limits of the unchanged (ad oculus) tissue of gland. The resections of tail and body of pancreas are distinguished.

Pancreatectomy is a complete deleting of pancreas. Operative treatment is applied infrequently. After the resection of pancreas adequate draining of its bed is very responsible.



The prognosis of disease depends on character of morphological changes of parapancreatic to the cellulose in pancreas. The more difficult destructive changes, the worst the prognosis.

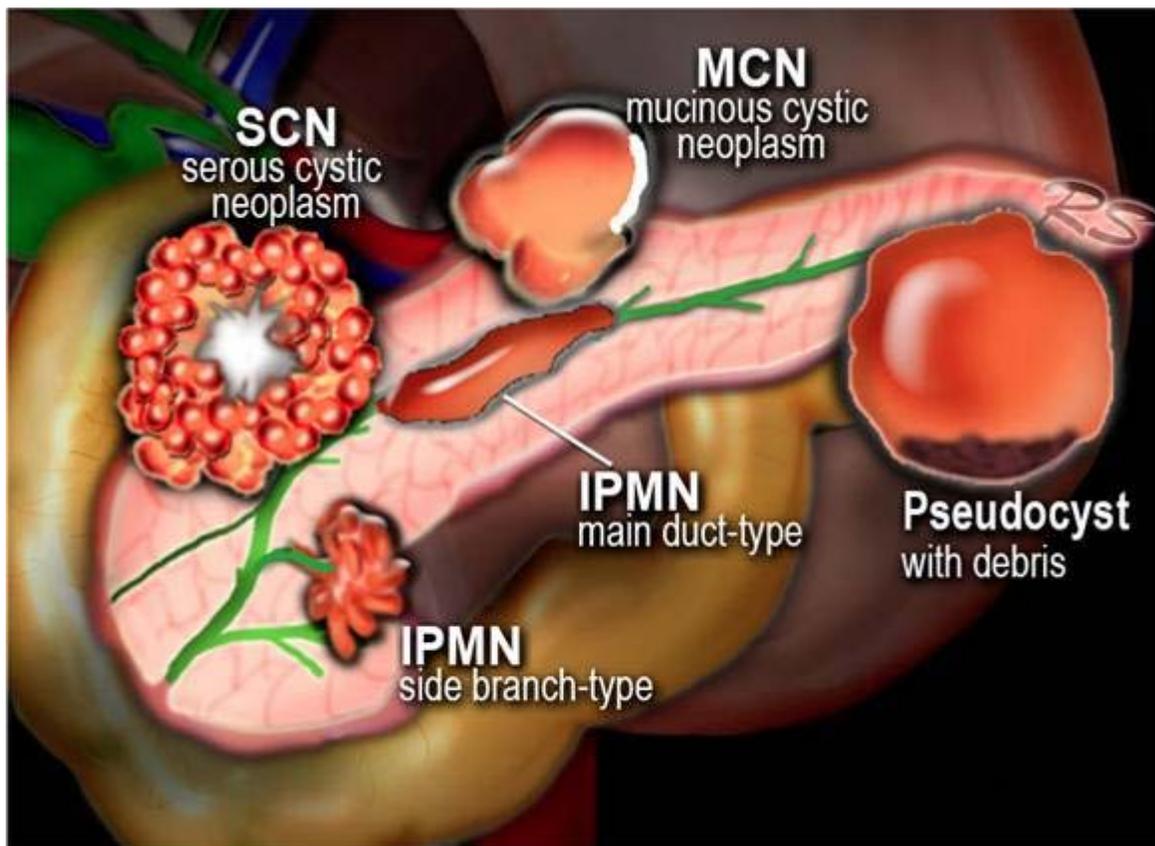
CYSTS OF PANCREAS



Cyst of pancreas is a cavity, filled by liquid (pancreatic juice, exudation, pus), intimately soldered with head, body or tail of organ, is limited by capsule, which has epithelium on internal surface.

Pseudocyst (unreal cyst) is a cavity in pancreas which appears as a result of its destruction, limited by capsule, that does not have epithelium on internal surface.

Etiology and pathogenesis



Acute or chronic pancreatitis or abdominal trauma causes pseudocysts. If no history of pancreatitis or trauma exists, the diagnosis must be carefully confirmed. The reasons of pseudocysts are destructive pancreatitis, traumas of pancreas, oklusion of Wirsung's duct by parasite, concrement, tumors, innate anomalies of development.

To the real cysts belong: innate (dysontogenetic) cysts which are anomalic in development; acquired retention cysts which develop as a result of difficult outflow of pancreatic juice, cystadenoma and cystadenocarcinoma (by mechanism the origins belong more frequently to proliferative, sometimes — degenerative cysts).

The mechanism of development of pseudocysts consists in the focus necrosis of gland, difficult normal outflow of its secret, there is a destruction of walls of pancreatic ducts with overrun of pancreatic juice gland that causes reactive inflammation of peritoneum of surrounding organs which form the walls of pseudocyst.

Pancreatic pseudocysts can be single or multiple. Multiple cysts are more frequently observed in patients with alcoholism, and they can be multiple in about 15% of cases. Size varies from 2-30 cm. About one third of pseudocysts manifest in the head of the gland, and two thirds appear in the tail. The fluid in pseudocysts has been well

characterized as clear or watery, or it can be xanthochromic. The fluid in pseudocysts usually contains very high amounts of amylase, lipase, and trypsin, though the amylase level may decrease over time.

The pathogenesis of pseudocysts seems to stem from disruptions of the pancreatic duct due to pancreatitis and extravasation of enzymatic material. Two thirds of patients with pseudocysts have demonstrable connections to the pancreatic duct. In the other third, an inflammatory reaction is supposed to have sealed the connection so that it is not demonstrable. The cause of pseudocysts parallels the cause of acute pancreatitis; 75-85% of cases are caused by alcohol or gallstone disease-related pancreatitis. In children, pseudocysts and trauma are frequently associated.

Pathomorphology

Morphologically the cysts of pancreas are divided into: pseudocysts retention to the duct are innate, single and multiple.



Pseudocysts are fresh and old. The internal surface of fresh pseudocyst is rough, granulating, grey-red. The table of contents is alkaline, grey or with a brown tint. In an old pseudocyst the wall is smooth and shiny, pale-grey. The table of contents is lighter. Epithelium pseudocysts is absent. More frequently they are met in body and tail of gland and are not connected with ducts.

Retention cysts connected with an obturated duct. The cavity has smooth, grey-white surface, maintenance is transparent, watery or mucous-like. Innate cysts are mainly multiple and shallow. A simple retention cyst differ from those that are always connected with the anomalies of development of ducts and are unite with polycystosis buds and liver.

Rarely there are echinococcus cysts, which have a clear chitinous shell, liquid in cavity and daughter's blisters. They are localized in the area of head of pancreas.

Classification

(by A.N. Bakulev and V.V. Vinogradov, 1952)

I. Innate cysts of pancreas:

1. Dermoid cysts.
2. Teratoid cysts.
3. Innate adenomas.
4. Fibrocystic degeneration.
5. Polycystic degeneration.

II. Inflammatory cysts:

1. Pseudocysts.
2. Retention cysts.

III. Traumatic cysts:

1. As a result of direct damage of gland.
2. As a result of indirect damage of gland.

IV. Parasite cysts:

1. Echinococcosis glands.
2. Cysticercosis glands.

V. Neoplasty cysts:

1. Cyst-adenoma.
2. Cyst-adenocarcinoma.
3. Cavernous hemangioma.
4. Cystic epithelioma.

Pathomorphologyo cysts are divided on:

1. The true cyst.
2. Pseudocysts.

According to clinical passing pseudocysts are divided into acute, subacute and chronic.

According to weight of passing — into simple (uncomplicated) and complicated.

Symptoms and clinical passing

In patients with the cystic damaging of pancreas there can be pain of different character and intensity (dull, permanent, cramp-like and belting). It is localized more frequently in right hypochondrium, epigastric area (cyst of head and body of gland), left hypochondrium (cyst of tail of pancreas). Pain is irradiated in the back, left shoulder-blade, shoulder and spine.

Dyspepsia violations are characteristic. Nausea, vomiting and belch are observed.

The syndrome of functional insufficiency of pancreas shows up by disorders of exocrine and endocrine insufficiency and depends on the degree of damage of organ. The unsteady emptying, replacement of diarrhea of constipation, steatorrhea and creatorrhea, development of the second diabetes are marked.

Compression syndrome. Arises as a result of compression of neighbouring organs. Clinically the compression of organs of gastro-intestinal highway shows up by complete or partial obstruction of general bilious duct (mechanical icterus), vein (portal

hypertension) gate, splenic vein (splenomegaly).

Physical:

- The sensitivity of physical examination findings is limited.
 - Patients very frequently have a tender abdomen.
 - Patients occasionally have a palpable mass in the abdomen.
- Peritoneal signs suggest rupture of the cyst or infection.
- Other possible findings include the following:
 - Fever
 - Scleral icterus
 - Pleural effusion

Diagnosis program

1. Anamnesis.
2. Biochemical blood test (amylase, sugar, bilirubin).
3. Analysis of urine on diastase.
4. Coprograma.
5. Sonography.
6. Contrasting sciagraphy of stomach and duodenum (relaxation duodenography).
7. Retrograde pancreatocholangiography.
8. Computer tomography.



X-ray examination. Shift the stomach to the left

During the examination patients with large cysts are marked by asymmetry of abdomen in epigastric and mesogastric areas. At palpation of abdomen tumular formation of elastic consistency with an even, immobile surface is found.

Abdominal ultrasound: Sonography examination shows echo-free formation with a clear capsule, determines localization and sizes of cyst. While cystic fluid collections in and around the pancreas may be visualized via ultrasound, the technique is limited by the operator's skill, the patient's habitus, and any overlying bowel gas. As such, ultrasound is not the study of choice to establish a diagnosis.



Sonography

Contrasting roentgenologic examination of stomach and duodenum with the sulfate of barium at the cyst of head of pancreas exposes moving of pyloric part of stomach upwards and breeding of „horseshoe” duodenum (at relaxation duodenography in the conditions of low artificial blood pressure). If a cyst is localized in the area of body of gland, displacement of stomach is marked forward and upwards or downward, rapprochement of its walls, moving of duodenal transition and loops of thin bowel downward and to the right; at lateral projection the distance between stomach and spine is increased. The cyst localized in the area of tail of gland, displaces the stomach forward and upwards, to the left or to the right.

Cholecystocholangiography exposes calculous cholecystitis and cholelithiasis.

Retrograde pancreatocholangiography exposes the changed and deformed, infrequently extended pancreatic duct, occasionally there can be filling of cavity of cyst

by the contrasting matter.

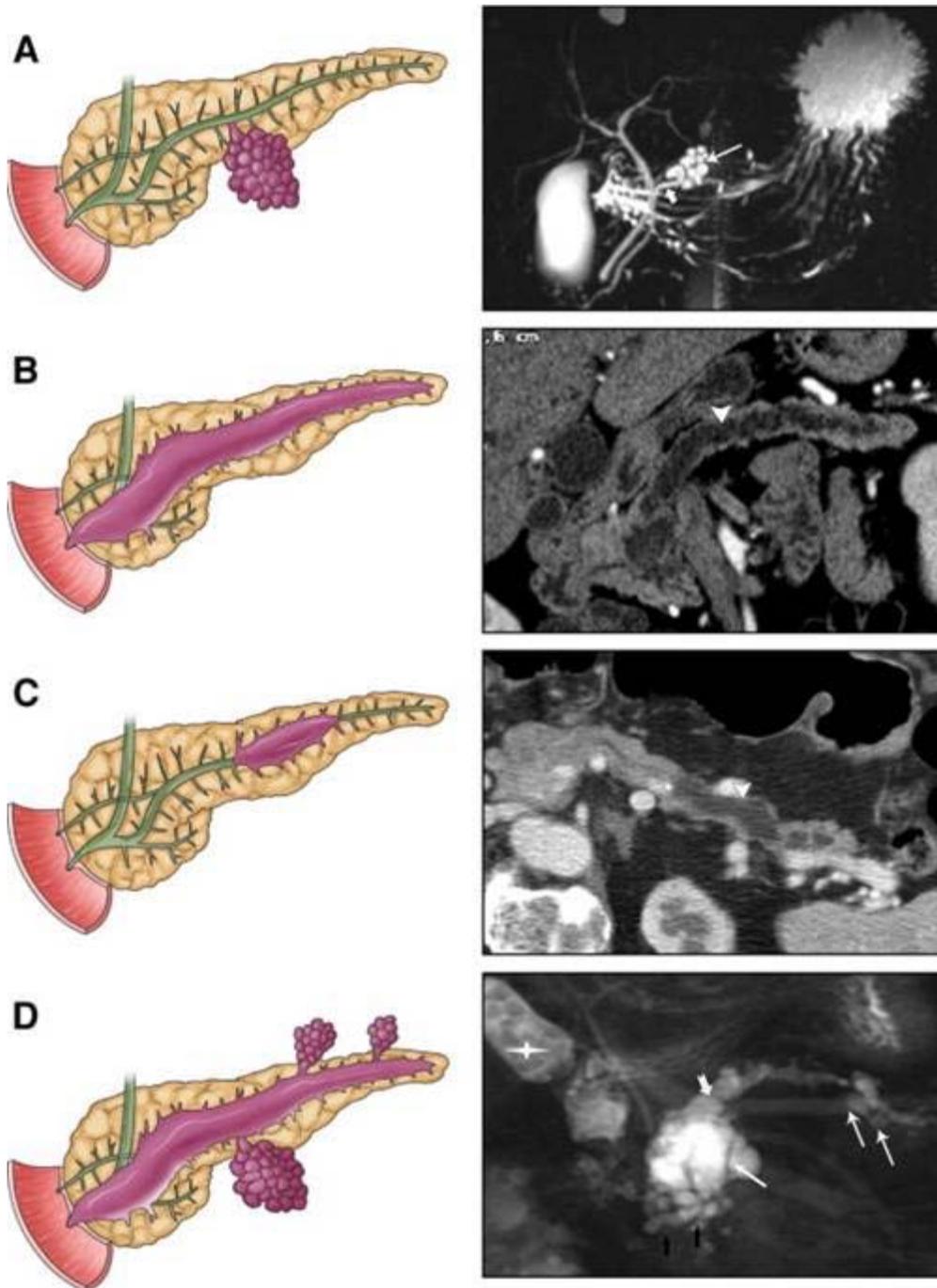
Abdominal CT scan: Computer tomography shows accumulation of liquid limited by the capsule of different closeness and thickness.



CT Scan. Cyst of the pancreas

- CT scan is the imaging criterion standard for pancreatic pseudocysts. It has a sensitivity of 90-100% and is not operator dependent.
- The usual finding on CT scan is a large cyst cavity in and around the pancreas.
- Multiple cysts may be present.
- The pancreas may appear irregular or have calcifications.
- Pseudoaneurysms of the splenic artery, bleeding into a pseudocyst, biliary and enteric obstruction, and other complications may be noted on CT scan.

- The CT scan provides a very good appreciation of the wall thickness of the pseudocyst, which is useful in planning therapy.



Endoscopic retrograde cholangiopancreatography:

- Endoscopic retrograde cholangiopancreatography (ERCP) is not necessary in diagnosing pseudocysts; however, it is useful in planning drainage strategy.
- A study by Neil et al investigated the use of ERCP and the treatment of pseudocysts and acute pancreatitis and reported that a change in management occurred 35% of the time after the ERCP findings in pseudocysts were evaluated. Therefore,

many authors recommend performing an ERCP before contemplated drainage procedures.



MRI:

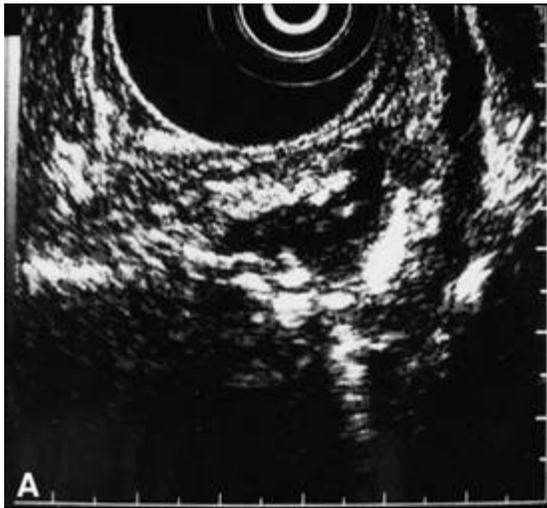
- MRI is not necessary to establish a diagnosis of pseudocysts; however, it is useful in detecting a solid component to the cyst and in differentiating between organized necrosis and a pseudocyst.

- A solid component makes catheter drainage difficult; therefore, in the setting of acute necrotizing pancreatitis with resultant pseudocyst, an MRI may be very important before a planned catheter drainage procedure.



Endoscopic ultrasound:

- Endoscopic ultrasound (EUS) is not necessary to establish a diagnosis but is very important in planning therapy, particularly if endoscopic drainage is contemplated.
- A gastric wall with a thickness greater than 1 cm next to the cyst tends to predict a poor outcome with endoscopic drainage.
- EUS may also be helpful in detecting small portal collaterals from otherwise undetected portal hypertension that may increase bleeding risks with transmural drainage.
- Transmural drainage may be performed only when the symptomatic pseudocyst is positioned next to the gut wall.



Laboratory examinations exposes hyperamylasemia, steatorrhea and creatorrhea, sometimes — hyperglycemia and glycosuria.

Laboratory Studies:

- Serum tests have limited use.
 - Amylase and lipase levels are often elevated but may be within reference ranges.
 - Bilirubin and liver function test (LFT) findings may be elevated if the biliary tree is involved.
- Analysis of the cyst fluid may help differentiate pseudocysts from tumors. Attempt to exclude tumors in any patient who does not have a clear history of pancreatitis.
 - Carcinoembryonic antigen (CEA) and carcinoembryonic antigen-125 (CEA-125) tumor marker levels are low in pseudocysts and elevated in tumors.
 - Fluid viscosity is low in pseudocysts and elevated in tumors.
 - Amylase levels are usually high in pseudocysts and low in tumors.
 - Cytology is occasionally helpful in diagnosing tumors, but a negative result does not exclude tumors.
 - A CEA level of greater than 400 ng/mL within the cyst fluid strongly suggests malignancy.

Clinical passing of cysts of pancreas depends on their kind, localization, size, stage of forming and complications.

Four stages of forming of pseudocyst are distinguished (P.G. Karaguljan, 1972).

I stage (1–1,5 months last) — in the center of inflammatory process the cavity of disintegration, which takes surrounding tissue, appears in an omentum bag.

The II stage (2–3 months) is characterized by the beginning of forming of capsule of pseudocyst. Cyst is magnificent, unformed, acute inflammatory phenomena calms down.

The III stage (3–12 months) is completion of forming of capsule of pseudocyst. Last accretes with surrounding organs.

The IV stage (begins an in year from the origin of cyst) is a separated cyst. The cyst is mobile, easily selected from connections with surrounding organs.

Retention cysts arise at closing of lumen of pancreatic duct (concrement, sclerosis). The internal surface of cyst is covered with epithelium. Pain syndrome, violation of exocrine function of gland are characteristic.

Traumatic cysts belong to the pseudocysts with similar passing and clinic, as well as inflammatory pseudocysts.

Parasite cysts (to echinococcus, cysticercotic) are met as casuistry. In such patients Kaconi test and serological Weinberg's reaction are positive.

The variants of clinical passing of the real and unreal cysts depend on their complications.

Perforation in free abdominal cavity. Clinic of the poured peritonitis is characteristic. Tormina, positive symptoms of irritation of peritoneum, possible shock state as a result of irritation of peritoneum by pancreatic juice arise.

Perforation in stomach, duodenum, small, rarer in large intestine is accompanied by diminishment of cyst in sizes or complete disappearance, sometimes diarrhea appears.

Suppuration of maintenance of cyst is accompanied by pain which becomes more intensive, temperature rises, leucocytosis grows.

The erosive bleeding appears suddenly and is accompanied by the symptoms of internal bleeding (expressed general weakness, dizziness). The pallor of skin and mucus shells, sticky death-damp, tachycardia and anemia are observed.

Mechanical icterus arises as a result of compression of cyst on the terminal part of choledochus. The icterus of skin and mucus shells, acholic excrement, dark urine, hyperbilirubinemia, increase of the ALT and AsT level are exposed.

Portal hypertension develops as a result of compression of portal vein. Ascites, varicose expansion of veins of esophagus and stomach, moderate icterus are diagnosed.

Reactive exudation pleurisy more frequently arises in left pleura cavity, where roentgenologic exudation is diagnosed with high maintenance of amylase.

At malignization the walls of cyst specific symptoms are absent, a diagnosis is set during operation (surgical biopsy of cyst wall).

Differential diagnostics

- Pancreatic Cancer
- Pancreatic Necrosis and Pancreatic Abscess
- Pancreatic Pseudoaneurysm
- Pancreatitis, Acute
- Pancreatitis, Chronic
- von Hippel-Lindau Disease

The cysts of pancreas are differentiated with the tumors of abdominal cavity and of retroperitoneal space.

Cancer of pancreas. For the cancer tumor of pancreas syndrome of “small signs” (discomfort in epigastric area, loss of appetite, general weakness), permanent dull pain, unrelated with the reception and composition of meal, icterus (cancer of head of gland), Courvoisier's symptom (increased, unpainfully gall-bladder) are characteristic. Inconstant pain at cysts of pancreas is more frequently related to faults in a diet; in anamnesis destructive pancreatitis, traumas of gland are carried. Sonography examination, retrograde pancreatocholangiography and computer tomography help in establishment of diagnosis.

Tumors of retroperitoneal space are passed asymptomatic, clinic shows up by a

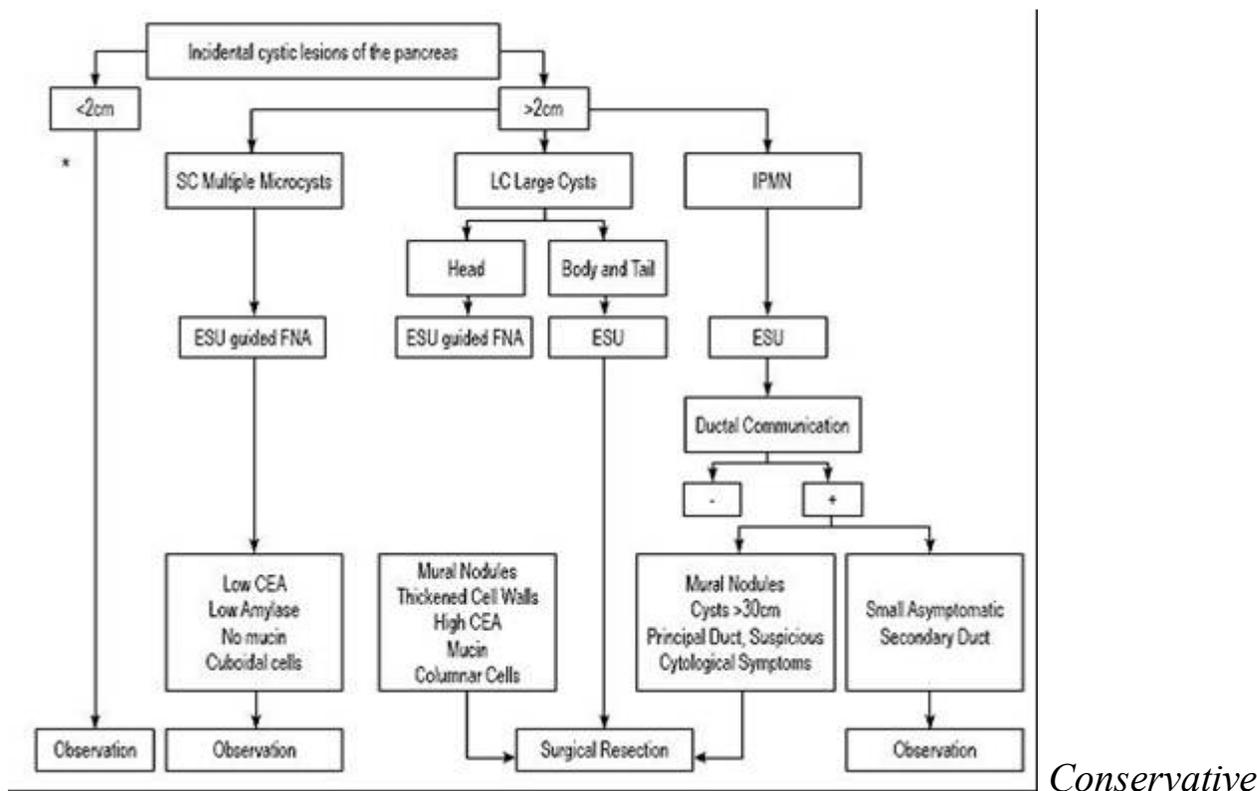
considerable compression on neighbouring organs. Nausea, vomit, chronic intestinal obstruction, dysuric disorders arise. Clinic of cysts of pancreas, on the opposite, are expressed on early stages. Pain, dyspepsia syndromes, syndrome of exocrine and endocrine insufficiency of pancreas are characteristic. Pain is related to the reception of meal and alcohol.

Aneurism of abdominal aorta. Dull, indefinite pain in abdomen which is unrelated with the reception of meal, pulsation and pulsating formation in abdomen are characteristic, auscultatory is systolic murmur. Aortography allows to confirm a diagnosis.

The cyst of mesentery of thin bowel has painless passing, at palpation it is mobile, easily changes position in abdomen. The cysts of pancreas are practically immobile, pain, anamnesis and laboratory information are characteristic.

The cyst of liver has protracted asymptomatic passing. Pain appears at infection of cyst. For this pathology symptoms which take place at the cysts of pancreas are not typical (pain related to the reception of rich food, alcohol, hyperamylasemia). Topic diagnostics is carried out at ultrasonic examination, scintigraphy, computer tomography.

Tactics and choice of treatment method



treatment. Treatment of acute or chronic pancreatitis is conducted in accordance with principles. At the unfavorable dynamics of passing the diseases hunger with the permanent sucking of gastric maintenance, parenteral feed and intravenous introduction of liquids are appointed. Puncture of cysts is used through abdominal wall under sonography control with aspiration of maintenance.

Peripancreatic fluid collections persisting for more than 4 weeks are referred to as acute pseudocysts. Pseudocysts lack an epithelial layer and thus are not considered true cysts. They also differ from true cysts in that they are usually filled with necrotic debris rather than fluid. Accordingly, pseudocysts may be better described by the term organized necrosis.

Surgical treatment is the method of choice of treatment of cysts of pancreas. The choice of treatment method depends on the stage of forming of pancreas cysts.

In selected patients with very large fluid collections, percutaneous aspiration of pancreatic pseudocysts is a reasonable approach. Even though treatment failures are common when the pseudocyst communicates with the pancreatic ductal system, percutaneous drainage serves as a temporizing measure that may later lead to successful endoscopic or surgical intervention. Often, an infected pseudocyst (which by definition is

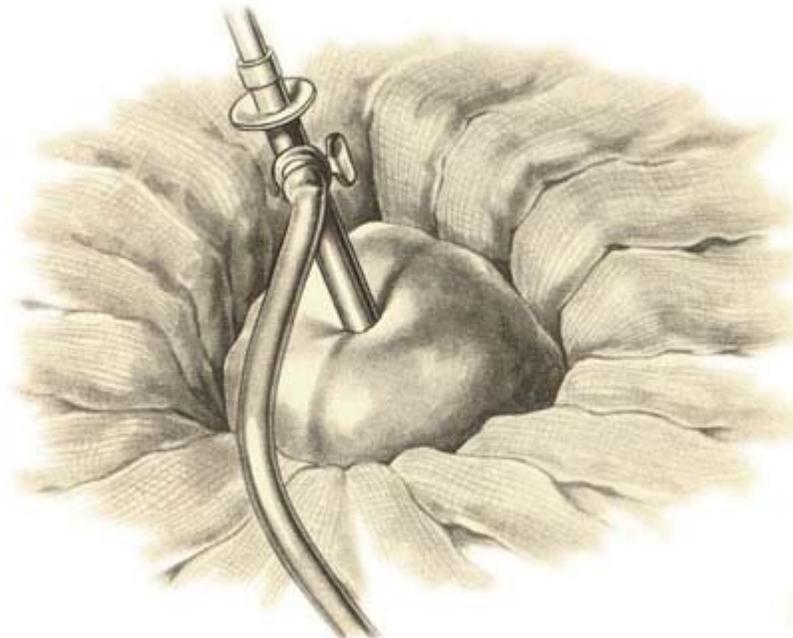
regarded as a pancreatic abscess) can be successfully managed by means of percutaneous drainage.

Pseudocysts may also be managed endoscopically with transpapillary or transmural techniques. Transpapillary drainage requires the main pancreatic duct to communicate with the pseudocyst cavity, ideally in the head or body of the gland. The proximal end of the stent (which should be smaller than the diameter of the pancreatic duct) is placed into the cavity. The technical success rate is 83%, the complication rate 12%. Generally, however, pancreatic stents are difficult to monitor and prone to obstruction and carry an increased risk of infection and ductal injury.

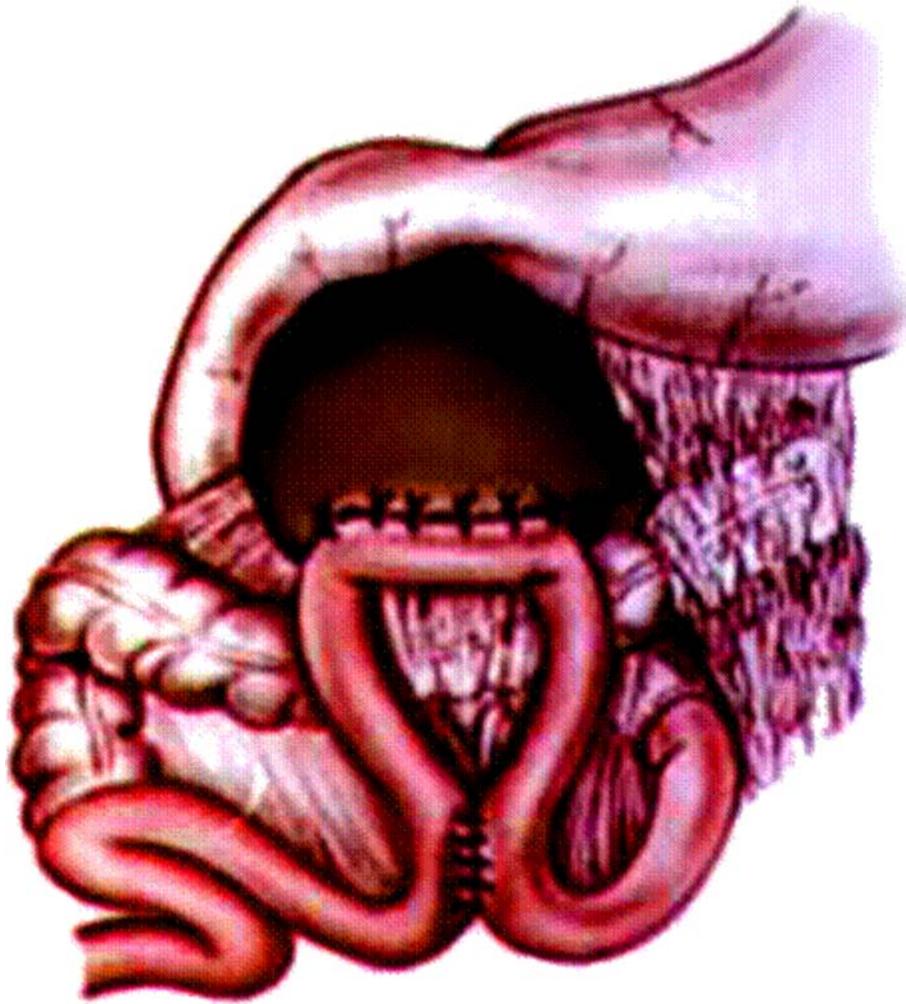
Some noncommunicating pseudocysts may be amenable to transmural enterocystostomy. Technical success requires a mature cyst that bulges into the foregut, and the distance from the lumen to the cyst cavity should be less than 1 cm. The success rate is 85%, the complication rate 17%. The transduodenal approach is associated with fewer complications and recurrences than the transgastric approach.

On the basis of prospective data from the 1970s, surgery was recommended for persistent large (> 7 cm) pancreatic pseudocysts because complications developed in 41% of patients, 13% of whom died. Internal pseudocyst-enteric anastomosis became the standard of care, with an operative mortality of 3-5%. This dogma was subsequently challenged by 2 retrospective studies in which patients with smaller (ie, < 5 cm) asymptomatic pseudocysts rarely ($< 10\%$) developed complications.

On the I stage operation is not used, conservative treatment of pancreatitis is conducted. On the II stage it is used at suppuration of pseudocyst (external draining of cyst). On the III — internal draining of cyst is used.



More frequently cystojejunostomy on the eliminated loop of thin bowel by Roux, cystojejunostomy with entero-entero anastomosis by Brawn and closing of afferent loop by Shalimov. Cystogastrostomy are executed and cystoduodenostomy is now not applied because of possible complications (infection of cyst, erosive bleeding). Marsupialization (opening and sewing down of cyst to the parietal peritoneum and skin) is used infrequently (at suppuration of cyst is seriously patientsing with the septic state). On the IV stage external and internal draining of cyst and radical operations are applied: a) enucleation of cysts (executed very rarely); b) distal resection of pancreas with a cyst.



Cystojejunostomy

Complications:

- Bleeding is the most feared complication and is caused by the erosion of the pseudocyst into a vessel.
 - Consider the possibility of bleeding in any patient who has a sudden increase in abdominal pain coupled with a drop in hematocrit level or a change in vital signs.
 - Therapy is emergent surgery or angiography with embolization of the bleeding vessel.
 - Do not perform a percutaneous or endoscopic drainage procedure under any circumstances in patients with suspected bleeding into a pseudocyst.
- Consider the possibility of infection of the pseudocyst in patients who develop

fever or an elevated WBC count. Treat infection with antibiotics and urgent drainage.

- GI obstruction, manifesting as nausea and vomiting, is an indication for drainage.
- The pseudocyst can also rupture.
 - A controlled rupture into an enteric organ occasionally causes GI bleeding.
 - On rare occasions, a profound rupture into the peritoneal cavity causes peritonitis and death.

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Prepared ass. Romaniuk T.

METHODICAL INSTRUCTIONS FOR 5th YEAR STUDENTS METHODOLOGICAL INSTRUCTION TO LESSON №9

PORTAL HYPERTENSION, CHRONIC PANCREATITIS

I. AIM. To be able to diagnose, to know symptoms of cirrhosis and portal hypertension, their complications depending on clinical type of the disease. Principles of conservative and surgical treatment. To be able to diagnose, to know symptoms of chronic pancreatitis, cysts and cancer of the pancreas, depending on clinical type of the disease. Principles of conservative and surgical treatment. To be able to diagnose, to know symptoms of euthyroid and toxic goiter, depending on clinical type of the disease, principles of conservative and surgical treatment.

II. Professional orientation of the student.

Hepatic cirrhosis progressing causes dangerous complications. Conservative treatment of these complications is not always effective. The knowledge of surgical treatment principles gives a possibility to correct patient's management, saving his life.

The consequences of chronic pancreatitis, pancreatic cancer are rather resistant to treatment. In-time diagnosing of these diseases and adequate surgical intervention leads to healing or significant improving of patient's state.

Diseases of thyroid gland set the first place in surgical endocrinology. Untimely and not proper choice of the method of treatment frequently causes irreversible consequences appearing, that cause patient's disablement or even are threatening to his life.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Theme №1. HEPATIC CIRRHOSIS AND PORTAL HYPERTENSION

1. Anatomical, morphological and functional peculiarities of liver (anatomy, topographical anatomy, anatomical pathology, histology, propaedeutical therapy).
2. Etiology, pathogenesis of hepatic cirrhosis and types of portal hypertension (pathological physiology,

propaedeutical therapy).

3. Estimation of laboratory and x-ray (transumbilical portography, splenoportography, celiacography) data, and also of radionuclide scanning, reography, echography, percutaneous transhepatic cholangioduodenography, laparoscopy, puncture biopsy.
4. Mechanism of hepatoprotectors, blood specimens, protamine sulfate solution action.

Theme №2. CHRONIC PANCREATITIS, CYSTS AND CANCER OF PANCREAS

1. Anatomical, morphological and functional peculiarities of pancreas (anatomy, anatomical pathology, histology).
2. Etiology, pathogenesis and classification of chronic pancreatitis, cysts and cancer of pancreas (pathological physiology, propaedeutical therapy).
3. Estimation of functional, x-ray, endoscopic findings (rhoetgenology).
4. Mechanism of pancreatic proteases inhibitors action.

Break 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Surgical peculiarities of liver blood supply.
2. Etiological factors of hepatic cirrhosis development.
3. Syndromes of hepatic cirrhosis
4. Clinical classification of hepatic cirrhosis (Hawaiian, 1958).
5. Clinical stages of cirrhoses.
6. Classification of portal hypertension.
7. Methods of examination of patients with portal hypertension.
8. Natural portocaval anastomoses (by Tonkov).
9. Complications of portocaval anastomoses.
10. Indications to surgical treatment of portal hypertension.
11. Palliative methods of portal hypertension management.
12. Radical methods of portal hypertension management.
13. Methods of surgical treatment of ascitis at hepatic cirrhosis.
14. Methods of portal system unloading.
15. Methods of treatment of bleeding from esophageal varicose veins.
16. Etiological factors of chronic pancreatitis.
17. Classification of chronic pancreatitis.
18. Clinical manifestations of chronic pancreatitis.
19. Non-invasive x-ray and instrumental methods of examination at chronic pancreatitis.
20. Invasive methods of examination at chronic pancreatitis.
21. Conservative treatment of chronic pancreatitis.
22. Operations on the pancreas and its ducts at chronic pancreatitis.

23. Operations at cholangiogenic pancreatitis.
24. Operations on vegetative nervous system at chronic pancreatitis.
25. Treatment of chronic pancreatitis, caused by the diseases of gastro-intestinal tract.
26. Classification of pancreatic cysts.
27. Clinical symptoms of pancreatic cysts.
28. X-ray signs of pancreatic cysts.
29. Surgical tactics, depending on the stage of pancreatic cysts formation.
30. Palliative operations at pancreatic cysts.
31. Radical operations at pancreatic cysts.
32. Groups of symptoms at pancreatic cancer.
33. Instrumental methods of pancreatic cancer diagnosing.
34. Classification of pancreas cancer (clinical and morphological ones).
35. Palliative and radical operations at cancer of pancreas.

The student has to be able to:

1. Reveal basic clinical signs of hepatic cirrhosis and portal hypertension.
2. Substantiate and formulate the clinical diagnosis.
3. Make differential diagnosis.
4. Substantiate pharmacotherapy, indications and contraindications to surgical treatment.
5. Reveal basic clinical signs of chronic pancreatitis, cysts and cancer of pancreas.
6. Substantiate and formulate the clinical diagnosis.
7. Make differential diagnosis.
8. Substantiate conservative treatment, indications for surgical treatment.
9. Reveal basic clinical signs of euthyreoid and toxic goiter.
10. Substantiate and formulate the clinical diagnosis.
11. Make differential diagnosis.
12. Substantiate conservative treatment, indications for surgical treatment.

Technical means and material provision of lectures (names and numbers of tables, compact discs, instruments quantity, subjectpatients, etc.) –
Multimedia projection of demonstration material (pictures, tables, videofilms) from compact disc “Clinical Surgery”, videofilms with operations.

Test evaluation and situational tasks 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. 51-year old patient, has alcohol abuse for a long time, suffers on hepatic cirrhosis. During recent months periodically noticed black stool. Once there was a bloody vomiting. The patient passed treatment courses in gastroenterological department, took syrepar, essentielle, albumin, aminocapronic acid infusions, vicasol, multivitamins. After course of treatment bleeding stopped. After repeated alcohol abuse bleeding has appeared again. Blood analysis data reveal insignificant anemia. Serum bilirubin is on normal level. Liver is insignificantly atrophied in size. Ascites is absent.

Management tactics?

2. 38-year old patient, had severe form of viral hepatitis in young age. During course of treatment hormonal therapy

was used. The patient keeps diet, has no alcohol abuse. During last year periodically notices dull pain in right hypochondrium. Liver is 2-3 sm under costal arch. Serum bilirubin periodically rises up to 28-32 mkmol/l.

Presumptive diagnosis?

Following tactics?

3. 54-years old patient, suffers on frequent attacks of chronic pancreatitis. On intravenous cholangiocholangiography 3 concrements in the distant part of choledochus were found. Whole blood bilirubin is 260 mkmol/l, the level of urine diastase is not increased. During an operation pancreas is dense. By means of supraduodenal choledochotomy only 1 stone was removed; the probe doesn't pass into duodenum.

Surgeon's tactics?

4. During an operation upon stomach cancer, no malignant process was found. The cyst of pancreas body, 15x15 cm in size, that is intimately adhered with back wall of the stomach, mesocolon and mesentery was found.

Surgeon's tactics?

5. 40-years old female patient, is ill with diffuse toxic goiter for 3 years. During all this time she has been undergoing the conservative treatment, in spite of what gradual increasing of thyroid gland up to the IV degree is observed. She mentions body weight loss and progressing of nerve-psycho disorders.

Tactics of treatment?

6. 50-years old patient with diagnosis "Diffuse toxic goiter of IV degree; thyreotoxicosis of severe degree". At the same time, he has chronic pneumonia with signs of lung insufficiency of II degree.

Surgeon's tactics?

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
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Additional –

1. Manual "Facultative Surgery" under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section "Diseases of the rectum and the colon",
2. Compact disc "Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
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The author: As. prof. A. Hospodarsky

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

ENDEMIC AND SPORADIC GOITER .

DIFFUSE GOITER WITH HYPERTHYROIDISM.

INFLAMMATORY DISEASES OF THYROID GLAND

DISEASES OF MAMMARY GLAND

Endemic goiter is the pathology of thyroid gland, which occurs in biogeochemical regions with iodine deficiency in environment (regional pathology).

Sporadic goiter is the disease of thyroid gland, which occurs in unendemic for goiter regions.

Anatomy(Fig.1;Fig.2;Fig.3.)

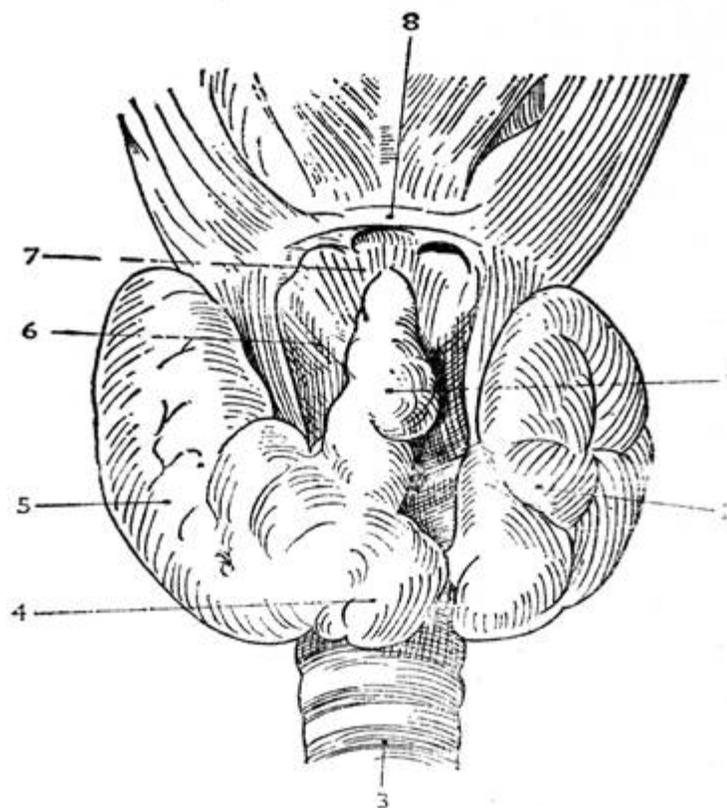


Fig.1. Anatomy of thyroid gland:

1 – lobus pyramidalis gl. thyreoideae; 2 - lobus sinister gl. thyreoideae; 3 – trachea; 4 – isthmus gl. thyreoideae; 5 - lobus dexter gl. thyreoideae; 6 – cartilago thyreoideae; 7 – membrana hyothyreoideae; 8 – os hyoides.

[Video](#)

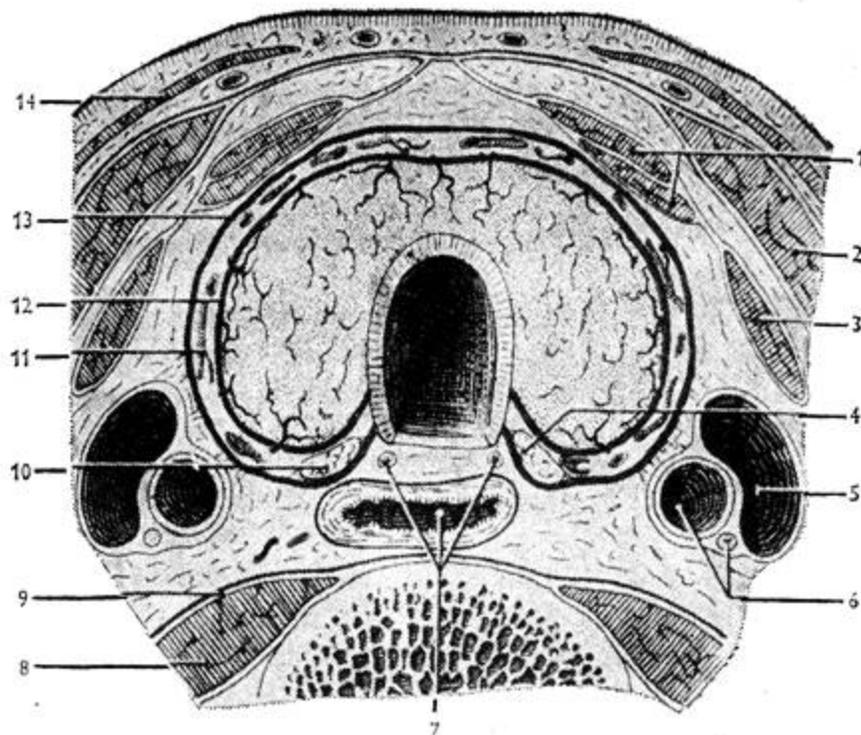


Fig.2. Transversal section of the neck on the thyroid gland level:

1 – mm. sternohyoideus et sternothyroideus; 2 – m. sternocleidomastoideus; 3 – m. omohyoideus; 4 - gl. parathyreoidea; 5 – v. jugularis int.; 6 – a. carotis communis i n. vagus; 7 – nn. recurrentes i стравохід; 8 – n. longus colli; 9 – fascia praevertebralis; 10 - gl. parathyreoidea; 11 – вени; 12 – capsula thyreoidea int.; 13 - capsula thyreoidea ext.; 14 – platysma (by Welti).



Fig.3. Topography of recurrens nerve and art. thyreoidea inferior:

1 – a. thyreoidea inf.; 2 - n. recurrens.

Etiology and pathogenesis

Except iodine deficiency, the goitrous endemia is contributed by excessive contents of calcium in environment, deficiency of bromine, poor sanitary and hygienic conditions. Decreased contents of cobalt, manganese, and zinc in environment also influence on expression of goitrous endemia. The lack of iodine in the organism blocks the synthesis of thyroid hormones. It results in hypersecretion of thyroidstimulating hormone of hypophysis, which in turn leads to hypertrophy and hyperplasia of thyroid epithelium that on initial stages can be compensated, but further transformed into the goiter.

The gravity of endemia is assessed by such indexes:

- 1) relationship between men and women with goiter (if it approaches to 1, the endemia is more severe);
- 2) predominance of the nodular form of goiter above its other forms;
- 3) presence of cretinism;
- 4) goiter in animals;
- 5) a number of persons with thyroid hyperplasia.

Sporadic goiter arises from relative iodine deficiency, which results from disturbance of iodine ingestion, liver dysfunction.

Pathology

Macroscopically the goiter is divided onto diffuse, nodular and mixed. According to histological structure distinguished parenchymatous and colloid ones. Sometimes occur cystic transformation of the gland and calcification. The enlarged nodes may result in atrophy of adjacent tissues and organs. So, the advanced forms of goiter can cause compression of trachea and its softening (tracheomalacia). The sings of hyperthyroid transformation of euthyroid goiter include: the transformation of thyroid epithelium from squamous cell into cubic and cylindrical, presence of papillomatous overgrowth, agglomeration of lymphocytes, liquid vacuolated colloid.

Classification

According to the form of enlargement of thyroid gland distinguished:

- 1) diffuse goiter;
- 2) nodular goiter;

3) mixed goiter.

For determining of the degree of goiter used such scale:

0 - the thyroid gland is not palpated;

I - the isthmus of the gland is noticeable during swallowing and could be palpated;

II - entire gland is noticeable during swallowing and could be palpated;

III - the enlargement of gland results in evident thickening of neck ("a thick neck");

IV – the gland considerably enlarged, and sharply deforms neck;

V - the enlargement reaches excessive size (goiter of major sizes).

According to the functional state of thyroid distinguished:

1) euthyroid goiter (normal function);

2) hyperthyroid goiter (excessive function);

3) hypothyroid (reduced function).

Diffuse enlargement of thyroid of I-II degrees without disturbances of function and nodular transformation referred to compensatory hyperplasia of the gland.

According to localization distinguished:

1) typical localization (anterior surface of neck);

2) retrosternal goiter;

3) ectopic goiter (goiter of the base of the tongue, intrathoracic goiter);

4) goiter of additional glands (aberrant goiter);

5) presternal goiter.

Symptomatology and clinical course

In the patients with **endemic euthyroid goiter** the clinical signs are basically caused by mechanical and reflex influence of enlarged thyroid gland on adjacent organs. Patients mainly complain of the presence of "tumour" and neck deformity. Sense of tightness in the neck, difficult breathing, swallowing, and also sudden attacks of cough (owing to compression of laryngeal nerves by goiter) trouble them. In case of great goiter (particularly retrosternal,) periodical dyspnea may develop (especially in the night), up to asphyxia, which is result of compression and inflection of trachea. Retrosternal goiter frequently accompanied with hoarseness, distended veins of face and neck.

The goiter with low thyroid function, as a rule, clinically manifests by general weakness, malaise, sleepiness, hypomnesia, chilliness, dry skin and edemas, particularly around eyes. Sometimes in such patients observed constipation.

The patients with **hyperthyroid goiter** complain of irritability, heartbeat, excessive sweating, and tremor of arms, sleeplessness, feeling of fever. Sometimes observed loss of weight, diarrhea. The thyroid hyperfunction in endemic goiter mainly slightly expressed, and not associated with exophthalmus.

Enlargement of thyroid gland in patients with endemic goiter mostly often has nodular or mixed character, and only in small number of the patients (mainly of younger age) observed diffuse enlargement of thyroid gland.

Nodular goiter is palpated as painless tumour with regular contours, smooth surface, not connected with adjacent tissues and displaced during swallowing. Such goiter is characterized by elastic or dense consistence. Long-term goiter leads to formation of fibrosis and calcification, it becomes solid, and tuberous.

The shape of **diffuse goiter** resembles the butterfly. It retains the contours, its surface is smooth, consistence – mostly elastic, sometimes soft or dense. Mixed goiters combine manifestations of the nodular and diffuse one, however tactically, the mixed goiters refer to nodular group.

The separate nodes or entire goiter can partially or completely be displaced behind breastbone. Palpation of such goiter requires the special devices. The examination is performed when the patient is supine with the bolster under scapulas. During the procedure the patient must force by himself or cough, that causes the emergence of the upper pole or entire goiter above breastbone.

Variants of clinical course and complications

Features of the clinical course of endemic and sporadic goiter caused by its form (nodular, diffuse, mixed), degree of thyroid enlargement, character of functional state (euthyroid, hypothyroid, hyperthyroid), location (typical, retrosternal, ectopic, aberrant goiter), constitutional features of the patient, duration of the disease and character of previous treatment.

Complication: inflammation of the goitrous thyroid gland (strumitis), hemorrhage in the tissue of goiter, asphyxia, malignancy.

The diagnostic program

1. Physical examination of the neck, palpation of thyroid gland(fig.4.).
2. Sonography, computer tomography.
3. Determining of thyroid function (serum hormones, serum iodine, and thyroid-iodine uptake) (Fig.5;Fig.6;Fig.7;Fig.8.).
4. According to indications: X-radiography of the neck (calcification, ossified foci) with barium swallow (compression of esophagus, trachea, their shift, and deformity).

5. Chest X-radiography, particularly of mediastinum, in two plains, pneumomediastinography (intrathoracic goiter).
6. Puncture biopsy(Fig.9.)



Fig.4. Palpation of thyroid gland(right lobe)



Fig.5. Thyroid gland scanning

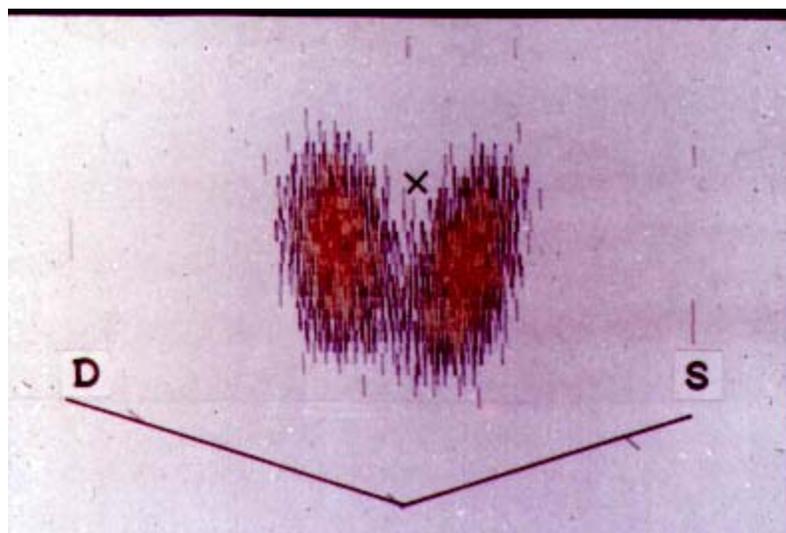


Fig.6. Scanogram of diffuse goiter

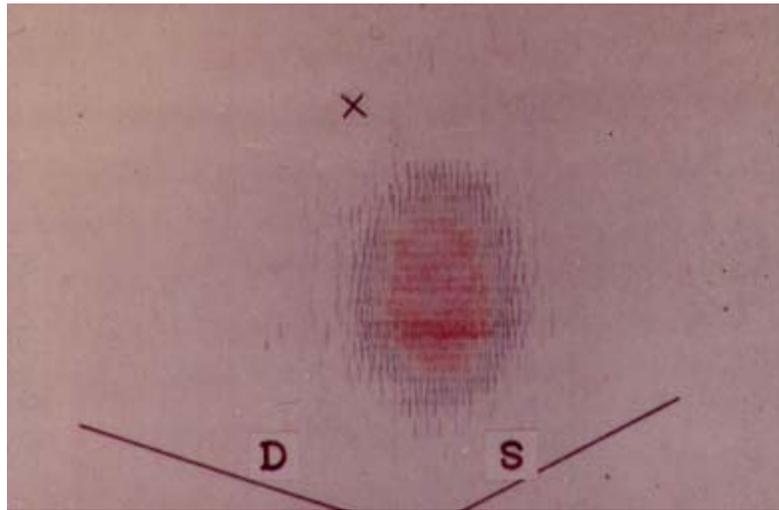


Fig.7. Scanogram of nodular goiter

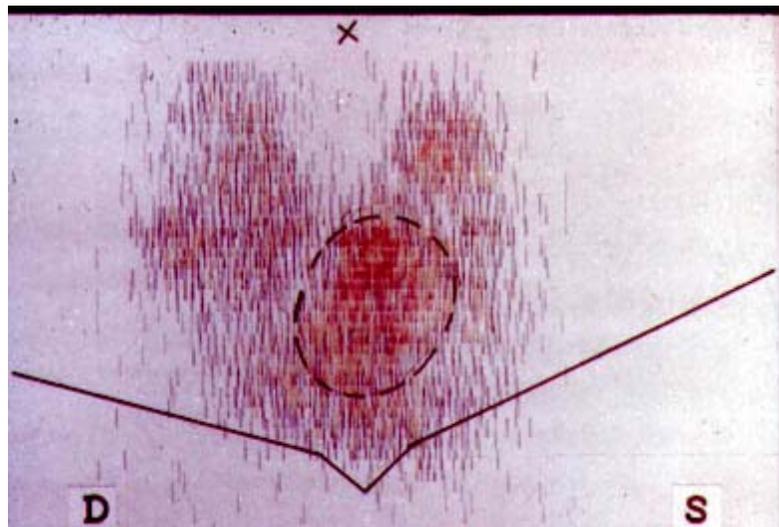


Fig.8. Scanogram of mixed goiter

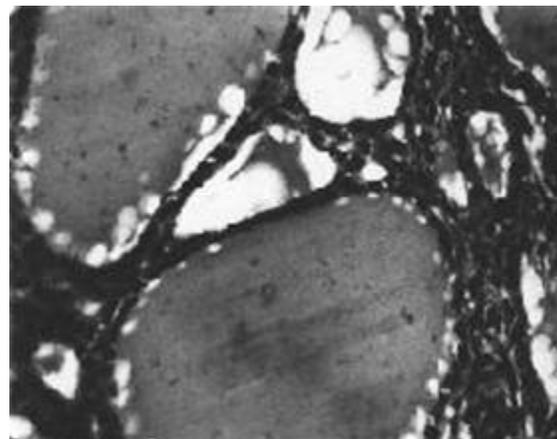


Fig.9. Endemic nontoxic goiter. Macro follicular

goiter with epithelium proliferation.

Differential diagnostics

Endemic and sporadic goiter requires the differential diagnostics with chronic autoimmune thyroiditis (Hashimoto's thyroiditis), Riedel's fibrous thyroiditis, neck cysts, lipomas and other tumours of the neck and mediastinum, malignant neoplasms of thyroid gland, metastases of tumours in cervical lymph nodes.

Hashimoto's thyroiditis is characterized by specific immunological indexes, diffuse density of thyroid, mosaic changes on sonogram and scintiscan, reducing of the goiter in response to prednisolone assay.

In case of **Riedel's goiter** the gland is tuberous, of woody consistency, quite often knitted with adjacent tissues (except skin).

The cysts, tumours of the neck, metastases in lymph nodes are well defined on sonograms, do not displace at swallowing and accumulate radioactive iodine.

The suspicion on **thyroid cancer** makes the necessity to carry out morphological verification by puncture biopsy with further cytological examination.

Tactics and choice of treatment

Endemic goiter is the subject for treatment in all its forms and all stages of the development. The choice of treatment depends on type of the goiter (diffuse, nodular, mixed), degree of enlargement of the thyroid (I-V) and character of complications of the goiter (inflammation, hemorrhage, asphyxia, and malignancy).

Conservative treatment includes the drugs of inorganic iodine, thyroidine and pure hormonal drugs (thyroxine, triiodothyronine). Thyroxine is the most effective one. The iodine drugs less effective and frequently are the cause of secondary hyperthyroidism. The medicament treatment is administered in diffuse thyroid enlargement without signs of compression of neck organs. Polynodular goiter (particularly in elder women) sometimes complicated by malignancy and consequently, even if the signs of compression of neck organs and hyperthyroidism are absent, also treated by conservative agents. The important argument of medicament treatment is their often recurrences after operation.

The **surgical approach** in endemic and sporadic goiter are determined by their spread and character of the lesion. There used the principle that all transformed into the goiter parenchyma should be removed, and healthy – preserved as much as possible.

The nodular and mixed form of the goiter, despite its function and size, is the subject for surgery. The hypothyroidism is not contraindication for operation, as the removal of the goiter results in functional normalization of unaltered, paranodular tissue. The operation, first of all, is indicated if present the signs of compression of neck organs, goiter of the major sizes, secondary hyperthyroidism and suspicion on malignancy. The goiter of additional thyroid glands (aberrant goiter) is the subject for obligatory surgical removal. The operation consists of removal of the aberrant gland with revision of the basic thyroid gland.

The intrathoracic goiter, which develops in retrosternal ectopy of thyroid gland, also requires obligatory

surgical removal(Fig.10.). The best access is the longitudinal sternotomy. The cervical goiter is possible to remove by means of cervical access without the special technical efforts.

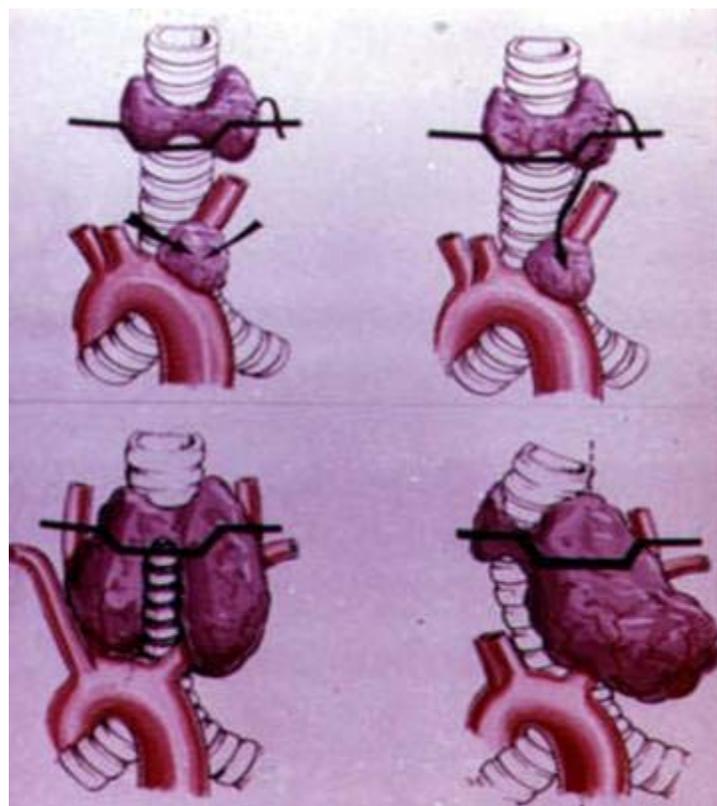


Fig.10. Scheme of goiter formation (retrosternal, intrathoracic)

In tongue ectopy if there are no severe disturbances of speech, swallow and breathe also possible observation and conservative treatment. Progressive growth of the goiter, presence of signs of compression, dysphagia, traumatic bleeding and suspicion on malignancy require the surgical treatment – removal of the goiter mostly through a lateral pharyngeal incision.

The acute disturbance of breathing (asphyxia) requires performance of urgent operation.

The optimal method of anesthesia is the endotracheal narcosis. This method prevents mechanical asphyxia during operation resulting from compression or inflection of trachea at the moment of mobilization and drawing out of the goiter. The method permits to perform careful revision of entire gland and neck spaces, first of all retrosternal, retrotracheal, retroesophageal, where there can be separate thyroid nodes. It is necessary to consider a local anesthesia, and also other methods of general anesthesia as reserve.

For removal of goiter used transversal incision by Kocher in the inferior part of the neck above the sternoclavicular junctions. Operation on thyroid gland must begin from careful revision and intraoperative diagnostics that permits to choose the adequate operative tactics. Trachea must be mobilized by means of isthmus dissection. It permits better orientation in topographic situation caused by the goiter, and to perform tracheostomy at occurrence of asphyxia. Further isthmus and pyramidal lobe must be removed as the most dangerous as for relapse of the goiter.

The volume of thyroid resection in endemic and sporadic goiter is determined individually. The resection is

performed subfascially that prevent the removal of parathyroid glands and trauma of laryngeal nerves. The node is necessary to eliminate together with paranodular tissue, as it is functionally failed. It is justified also by oncologic reasons. The operation of nodular enucleation is considered to be inadequate, thus never used. Meanwhile, in multinodular bilateral goiter, when practically entire thyroid gland is affected, it is necessary to eliminate separate nodes from healthy parenchyma, preserving its maximal amount, as the parenchyma is mainly disposed as lamina on their surface. This layer of parenchyma is necessary to dissect and draw aside by scissors out of node, having preserved its vascularization.(Fig.11-15.)

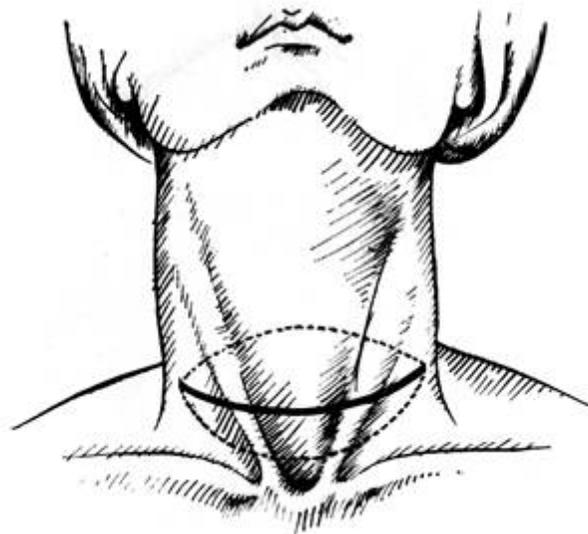


Fig.11. Operative incision

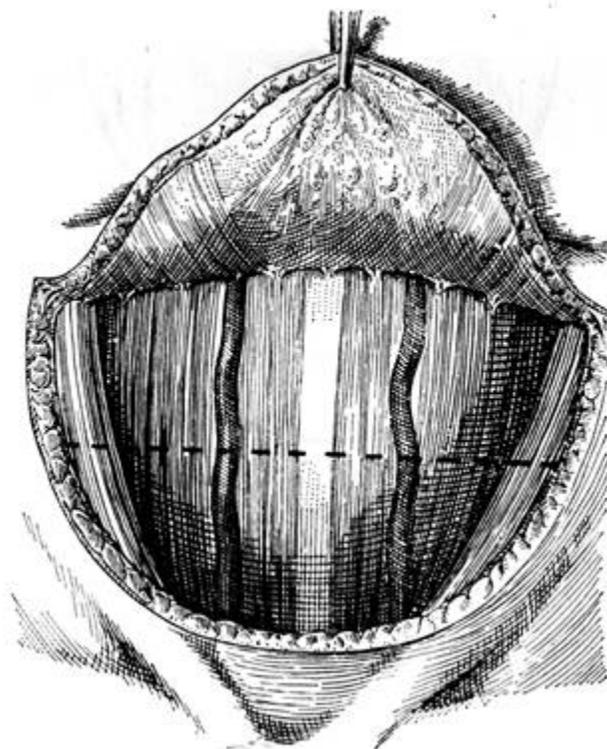


Fig.12. Preparation of upper layers of the skin

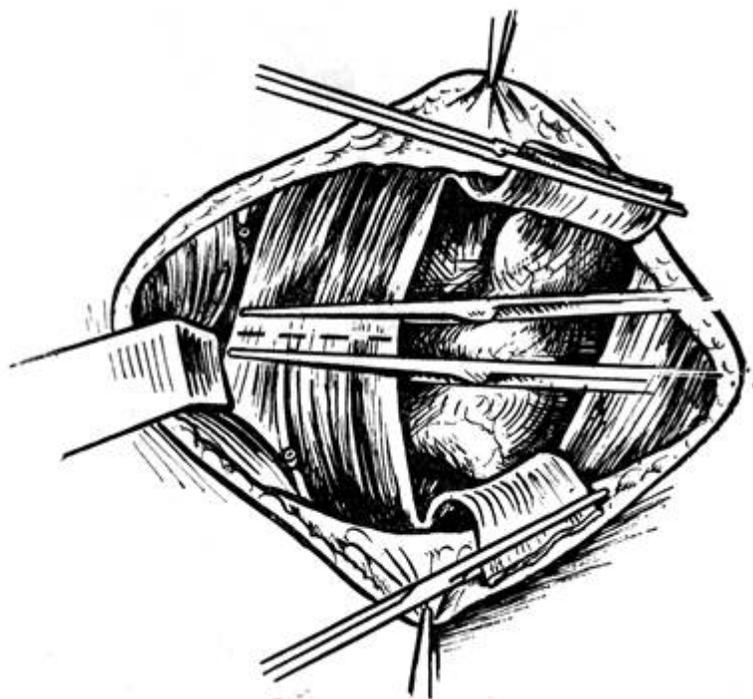


Fig.13. Transversal section of prethyroid muscles



Fig.14. Sutures imposition and closing with thyroid stump

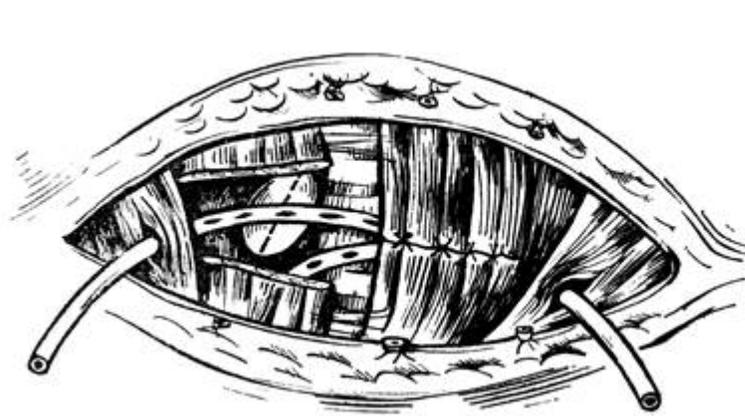


Fig.15.Sutures on prethyroid muscles
and drainage of operative wound

In endemic and sporadic goiter applied saving, extent and subtotal resection of the thyroid with obligatory indication of amount and site of leaving parenchyma.

For oncologic standpoint it is necessary in all cases to carry out intraoperative express cytology of the removed tissue.

For prophylaxis of goiter relapse after the operation necessary long-termed institution of thyroid hormones with the purpose to block thyroid stimulation by pituitary gland.

DIFFUSE GOITER WITH HYPERTHYROIDISM

Diffuse goiter with hyperthyroidism (Grave's or Basedow's disease, thyrotoxicosis, hyperthyroidism) is severe autoimmune and neuroendocrine disease resulting from excessive secretion of thyroid hormones by diffuse-enlarged thyroid gland with lesion of all organs and systems of the body.

The diffuse goiter with hyperthyroidism (thyrotoxicosis) mostly occurs at women. In 5 % of persons with hyperthyroidism develop ophthalmopathy and pretibial myxedema.

Etiology and pathogenesis

The scientific investigation and clinical examination testify that the diffuse goiter with hyperthyroidism is autoimmune disease. This disease is commonly results from infections, intoxication, craniocerebral injury, dysfunction of other endocrine glands, first of all genital, acute and chronic mental disorder, sunstroke. The disease develops under the influence of these factors directly on generically predisposed to thyrotoxicosis organism.

Pathology

The thyroid gland is 2-5 times enlarged, moderately dense, on incision pulpy, sanguineous, of grey-pink color. Histologically revealed a polymorphism of follicles. Follicular epithelium is cylindrical with papillomatous growths. The colloid is eosinophilic, contains plenty of resorptive vacuoles. In interstitial space – lot of lymphocytes, which form follicles. The severe form of thyrotoxicosis results in thyrotoxic heart, thyrotoxic liver cirrhosis, thyrotoxic ophthalmopathy, osteoporosis, and cachexia.

Classification

According to the clinical course distinguished mild, moderate and severe forms of the disease.

According to Milk's classification, there are four stages of hyperthyroidism.

I stage – neurotic, onset of thyrotoxicosis, slight enlargement of thyroid gland.

II stage – neurohormone, marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size.

III stage – visceropathic, is characterized by a thyrotoxic lesion of viscera.

IV stage – cachectic, is characterized by nonreversible dystrophy of organs and systems.

Symptomatology and clinical course

A diffuse toxic goiter affects practically all organs and systems and disturbs all types of metabolism. Except described in 1842 by Basedow classical triad (goiter, tachycardia and eye bulging), today is known about 70 signs, proper for thyrotoxicosis, which can be combined in three basic syndromes: hyperthyroidism, eye signs (ophthalmopathy) and lesion of skin (pretibial myxedema). By the may, the hyperthyroidism is the permanent phenomenon, and ophthalmopathy and pretibial myxedema occurs rather seldom (in 1-5 % of patients).

To initial sings of thyrotoxicosis can be regarded: general weakness, prompt fatigability, decreased work ability and muscular force, nervousness, irritability, sleeplessness, sweating and hyperemia of skin.

The basic signs of thyrotoxicosis are enlargement of thyroid gland (goiter), palpitation, exophthalmos, tremor and progressing loss of weight. (Fig. 16)



Fig.16. Thyrotoxicosis

The thyroid gland in the patients with thyrotoxicosis is diffuse enlarged and of moderate density. In some of them due to excessive blood supply it can pulsate. After long treatment by iodine the gland becomes dense and painless. Such long-term conservative treatment causes the development of sclerotic degenerative processes, sometimes with nodular transformation of the tissue, and the degree of thyroid enlargement frequently does not relate to the gravity of thyrotoxicosis.

Secretory activity of thyroid hyperplasia in the form of excessive releasing of its hormones (triiodothyronine and thyroxine) underlies the hyperthyroidism. The majority of effects of thyroid hyperfunction manifest through sympathetic nervous system: palpitation, tremor of fingers, tongue, and whole body (sign of "telegraphpole"), sweating. In the patients with thyrotoxicosis the protein, carbohydrate and lipid metabolism is elevated, which manifests by simultaneous excessive appetite and loss of weight.

The changes, which develop in organs of cardiovascular system and manifests by tachycardia, high systolic and low diastolic pressure, increase of pulse pressure and complete arrhythmia with the development of heart failure form a syndrome of thyrotoxic heart.

The excessive formation of heat owing to intensive metabolism, which results from the influence of thyroid hormones, leads to hyperthermal syndrome (feeling of fever, high body temperature). The signs of nervous dysfunction include irritability, anxiety, fear sensation, nervousness, sleeplessness, hyperactive tendon reflexes. The dysfunction of genitals manifests by oligo- or amenorrhea, and in the men by gynecomastia, which is the outcome of disturbed relation between estrogens and androgens. Thereafter libido and potency are reduced.

The thyrotoxicosis without treatment results in loss of weight, in advanced cases not only the subcutaneous fat disappears, but also a muscular tissue reduced, down to cachexia. Degenerative changes in muscles, and lesion of peripheral nervous system result in thyrotoxic myopathy.

In majority of patients develop characteristic eye signs. The predominant one is the exophthalmos. By the way, eye bulging, which occurs in 50 % of cases, frequently can be the initial manifestation of the disease, assigned by patient. Three types exophthalmos are distinguished: slight (14-17 mm), moderate (17-20 mm) and considerable (more than 20 mm). The exophthalmos in thyrotoxicosis is symmetric, the eye trophic and movements of does not disturbed. Except exophthalmos, there are lot of other eye signs observed in the patients with thyrotoxicosis.

- Graefe's sign – the upper lid lag when the patient looks downward;
- Stellwag's sign – infrequent winking;
- Mebius' sign – a weakness of convergence;
- Dalrymple's sign - wide palpebral fissure;
- Kocher's sign – retraction of the upper eyelid at prompt change of view.

The eye signs of diffuse toxic goiter are necessary to differentiate from ophthalmopathy (malignant exophthalmos), which observed approximately in 5 % of the patients with thyrotoxicosis. Such exophthalmos simultaneously associated with pain in the eyeballs, gritty sensation and eyewatering. Also detected lid edema, ocular injection. In considerable ophthalmopathy the eyeballs bulge from orbits, eyelids and conjunctiva are swollen, with signs of inflammation. It can result in keratitis with corneal ulceration, which finally can lead to blindness. The high orbital pressure caused by lymphoid infiltration, accumulation of fluid and edema of retroorbital tissues result in not only eye bulging – exophthalmoses, but also compression of optic nerve and loss of sight. It is necessary also to specify that the ophthalmopathy in thyrotoxicosis, as a rule, develops on the background of encephalopathy and has an autoimmune genesis. (Fig.17.)



Fig.17. Exophthalmoses

Pretibial myxedema arises on the anterior surface of lower legs. The skin becomes dense, thickened, of purple-red color, and hair follicles jut out of its surface.

Thyroid hypersecretion also negatively influences on the liver parenchyma. In severe cases it can result in toxic hepatitis, jaundice and further hepatargy. It is necessary to consider the toxic hepatitis in such patients unfavorable as for prognosis.

Under the direct cytotoxic influence of thyroid hormones on intestinal mucosa suppressed its enzymatic function that leads to intestinal hyperkinesis and osmotic diarrhea – thyrotoxic enteric syndrome. It is accompanied by gluco- and mineralocorticoid dysfunction of suprarenal gland, and leukopenia, granulocytosis and lymphocytosis in blood.

Variants of clinical course and complications

In clinical course of thyrotoxicosis distinguished the mild, moderate and severe forms.

The **mild form** of thyrotoxicosis is characterized by following signs: pulse 100 beat/min, loss of weight approximately 3-5 kg, slight sweating, eye signs absent or slightly expressed, normal arterial pressure, basal metabolism to +30 %, elevated thyroid-iodine uptake, and the maximum of iodine accumulation, which exceeds 30 % is detected after 24 hours.

The **moderate gravity** of the disease manifests by expressed symptomatology: loss of weight to 8-10 kg, tachycardia 101-120 beat/min, systolic pressure is elevated, and diastolic – decreased or normal. Frequently

observed exophthalmos, basal metabolism +31-50 %, thyroid-iodine uptake has been excessive since first hours.

The **severe form** of thyrotoxicosis is characterized by the sharply expressed symptomatology, which is caused by considerable visceral dysfunction. The pulse rate in such patients exceeds 120 per minute, and complete arrhythmia develops. Tremor and profuse sweating sharply expressed, pulse pressure considerably elevated circulatory failure and ophthalmopathy frequently observed. The loss of weight can overtop 10 kg, basal metabolism more than +50 %, the maximum of radioactive iodine accumulation detected in 4-6 hours after taking of isotope, and decreasing of accumulation curve exceed 24 hours.

According to the clinical course two forms of thyrotoxicosis distinguished: a) thyrotoxicosis with slow development; б) acute form of diffuse toxic goiter, which is characterized by an acute onset and prompt, sometimes within several hours, development. The acute thyrotoxicosis seldom occurs and in most cases ends lethally from thyrotoxic coma.

The clinics of acute thyrotoxicosis develop within some hours or days. Thus the thyroid gland is not enlarged, high temperature, vomiting, diarrhea, sharp loss of weight are observed.

The special forms of thyrotoxicosis include the thyrotoxicosis in childhood, in pregnant, in climacteric women, and people of the elderly age.

Among complications during the course of disease the most dangerous for the life is thyroid storm. It is observed in 0,02-0,05 % of the patients and develops, mainly, as the outcome of the lesion of provoking factor. Among them considered trauma (surgical intervention on thyroid gland or other organs), harsh palpation of the gland, mental trauma, emotional stress, infections, pregnancy, labors and radioiodine therapy.

The diagnostic program

1. Clinical examination, examination, palpation, auscultation of thyroid gland.
2. Detecting of basal metabolism, serum lipids, ECG.
3. Detecting of thyroid hormone concentration (common free thyroxine – T₄, common free triiodothyronine – T₃), serum iodine-binding globulin, serum thyroidstimulating hormone of hypophysis.
4. Determining of thyroidstimulating antibodies – immunoglobulins, antithyroid antibodies.
5. Sonography of thyroid gland.

Differential diagnostics

The manifestation of initial, vague and slightly expressed forms of thyrotoxicosis can resemble neuroses, rheumatic disease, tuberculosis, chroniosepsis, postcastrate syndrome, diencephalic lesions, and also malignant tumours. It particularly concerns those cases, when the enlargement of thyroid gland is slight or it is failed to detect.

All the mentioned diseases are characterized by palpitation, heart pain excessive sweating, subfebrile fever and loss of weight.

The acute development of thyrotoxicosis sometimes makes the necessity to rule out such acute infectious diseases, as dysentery, influenza or camp-fever.

The thyrotoxicosis with exophthalmos is necessary to differentiate with encephalitis, ophthalmopathy. Encephalitic exophthalmos is characterized by combination of looking upward paresis with diplopia, corneal ulceration, conjunctivitis, progressing, so-called malignant exophthalmos, which rather frequently can result in loss of eye. By the way, this type of exophthalmos is commonly unilateral.

Laboratory tests are important for differential diagnostics of thyrotoxicosis: detecting of thyroid hormones, serum protein-bounded iodine, thyroid-iodine uptake, biochemical, immunological investigations, sonography and scanning with the radioiodine or technetium.

In particularly severe cases it is advisable to apply trial antithyroid therapy.

Tactics and choice of treatment

The thyrotoxicosis revealed for the first time, and also its severe and moderate forms require institutional treatment. Three methods of treatment of thyrotoxicosis are commonly employed: a) antithyroid drugs; b) treatment by radioactive iodine; c) surgery.

The **antithyroid drug therapy** of the patients with thyrotoxicosis, first of all, should be directed to ameliorate hyperthyroidism. This is gained by the usage of iodine and thyrostatic agents, particularly mercasolil – synthetic antithyroid drug. In severe cases the treatment begins from 45-60 mg (9-12 tablets) per day, in the moderate form – from 30 mg (6 tablets), in mild – from 15 mg (3 tablets) per day. The maximal initial dose ordered within 2-4 weeks to gain expressed clinical relief of the disease (decrease of irritability, normalization of pulse rate, increase of weight). After that, if the state of the patient gradually improves, the dosage is reduced every 3-4 weeks by 1-2 tablets per day to supportive dose (1 or 1/2 tablets per day during 2-3 months). Commonly, the course of the treatment by mercasolil should be lasted for 1-1,5 years. Among complications, which can arise during the treatment, it is necessary to mention leukopenia, agranulocytosis and allergy.

In case of allergic response to mercasolil or development of complications used a reserve drug – lithium carbonate.

Such long conservative treatment of thyrotoxicosis is desirable in those patients, who gained euthyroidism in 1-3 months, that is the gradual reduce of goiter and eye signs. If during the treatment periodically exacerbation occurs, which manifests by thyroid enlargement, development of encephalopathy, activation of ophthalmopathy the surgery is indicated.

More recent studies showed that the **treatment by radioactive iodine** is a radical method of therapy of thyrotoxicosis. The radioactive iodine, which deposits in thyroid gland, irradiating its parenchyma, results in destruction of the active thyrocytes with their further replacement by connective tissue (bloodless thyroidectomy). The standard dosage is 0,1 mCi per gram of thyroid tissue, and it can be introduced at one time or partly.

Nevertheless such therapy has series of essential drawbacks. The lack of precise methods of determining the

weight of the gland results in miscalculations at selection of total dose of the isotope. It is also impossible to exclude the harmful influence of the isotope on the genetic kettle of the patient. Almost in 70 % of the patients the hypothyroidism develops after the treatment by radioactive iodine and there is a potential threat of the development of radioactive thyroid cancer. That's why the indication for application of this method rather restricted.

The treatment by radioactive iodine is commonly indicated for the patients with thyrotoxicosis after 40, with recurrent thyrotoxicosis, and after operations particularly, in combination with severe concomitant diseases and in case of refuse of surgery. It is not justified at young age, pregnancy and during lactation, thyrotoxic multinodular adenoma, expressed leukopenia, and kidney dysfunction or at severe acute thyrotoxicosis.

Sometimes introduction of radioactive iodine can cause the exacerbation of thyrotoxicosis, up to the development of thyroid storm. Thus, before administration of the radioactive iodine, particularly in patient with severe form of thyrotoxicosis in order to relieve thyrotoxicosis it is necessary to institute antithyroid drugs.

The **surgical method of treatment** is considered to be radical and the most effective. The operation almost always allows to liquidate the manifestations of hyperthyroidism together with its morphological base. The efficiency of this method in the specialized clinics reaches 95-97 %.

The indications for surgery include thyrotoxicosis of moderate gravity when the conservative treatment is inefficient during 2-3 months, severe forms of thyrotoxicosis, goiter of IV-V degree despite the gravity of thyrotoxicosis, and also nodular transformation of toxic goiter.

The surgical method is not recommended for the patients with thyrotoxicosis with severe concomitant diseases and dysfunction of vital systems.

The obligatory requirement of successful surgery of the patients with thyrotoxicosis is the careful **preoperative preparation**, which goal is the liquidation or decreasing of hyperthyroidism, that achievement of euthyroid state. Preoperative preparation should be complex, pathogenically proved and individual.

The appropriate place in preoperative period should possess psychological preparation. The patients stay in chambers together with patients recovering after operation. In severe form of thyrotoxicosis a strict bed regime is ordered. The diet should be high-caloric, rich with proteins, vitamins. The patient must take antithyroid drugs under the control of general blood analysis. To prevent leukopenia and agranulocytosis instituted leukopoetic agents. Besides antithyroid therapy, are advisable reserpin that characterized by hypotensive, sedative and antithyroid activity, beta-blockers and tranquilizers for decreasing stimulation of CNS.

In severe form of thyrotoxicosis, at presence of hypoproteinemia is advisable the intravenous infusion of protein substitute solutions (albumin, protein, plasma). With the purpose of detoxycation applied neohaemodes, neocompensan. For exhausted patients beside high-caloric diet applied parenteral infusion of glucose, intralipid, amino acids and vitamins, particularly of B-group. The patient with sings of heart failure simultaneously should take cardiac glycosides and other cardiac agents. One of the measures in preoperative preparation is the regulation of reduced function of suprarenal glands. Glycocorticoids (hydrocortisone etc.) administered in daily dosage of 25-50 mg 2-3 times per day during 3-4 days before the operation and 2-3 days after it. Preoperative preparation should also include regulation of hemostatic dysfunction (vicasol, aethamsylat, dicynon, inhibitors of proteases).

The preoperative preparation is considered to be sufficient, if the state of the patient is regarded to euthyroid or approximate to it. It is testified by normalization of pulse (90-80 per minute), increase of weight on 3-5 kg, liquidation of nervousness and irritability, disappearance of tremor, regulation of function of cardiovascular

system, liver, suprarenal glands, CNS and basal metabolism.

Anesthesia. The method of choice is endotracheal narcosis.

Operation. The most effective and rational surgery approach for thyrotoxicosis is the subtotal subfascial resection of the thyroid (O.V.Nickolayev, 1951) or thyroidectomy. The main difference of this procedure is the refuse of ligation of thyroid vessels before they enter the gland and subfascial resection of the gland. The goal of this technique is to gain bloodless and atraumatic procedure of operation, to prevent damage (removal) of parathyroid glands and laryngeal nerves. This procedure also favors the formation of a gland stumps in the site of parathyroid glands and passage of recurrent nerves. The volume of resection and, consequently, the size of the gland stamp must be based on the account of gravity of thyrotoxicosis, age of the patient, duration of the disease, previous treatment, morphology of the organ and immune state of the patient. (Fig.18)

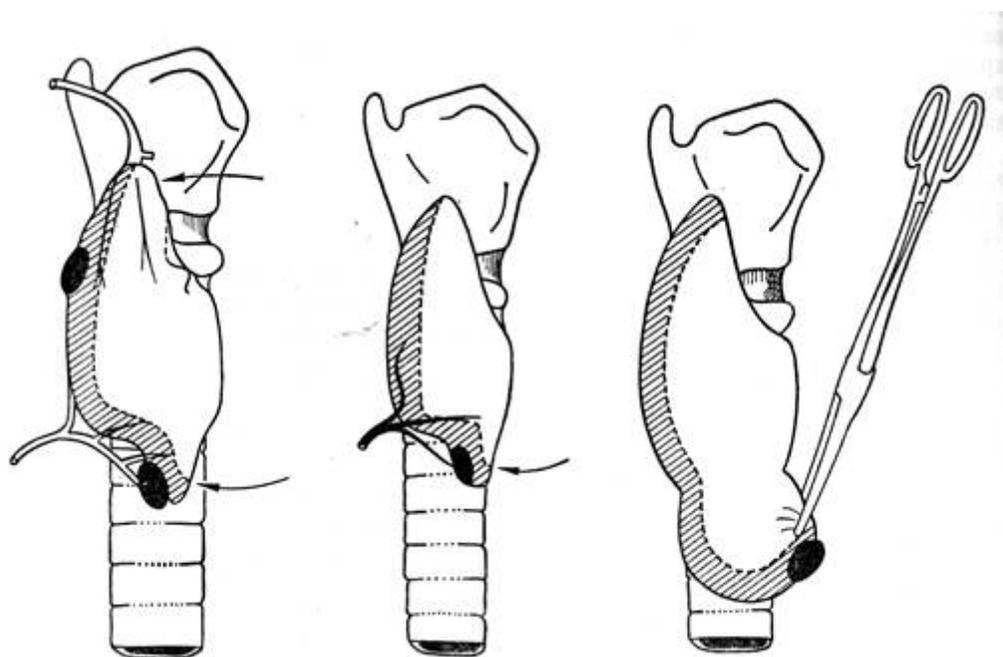


Fig.18. Subtotal resection of thyroid gland by Welti

It is generally accepted, that the more severe the form of thyrotoxicosis, more young the patient, more short duration of the disease, more intensive vascularization, more dark color of the gland, the smaller tissue remnant is necessary to leave (mainly less 6 g, on 1-3 g from each side). Morphologically in such patients revealed hyperfunctional type of histogram and autoimmune processes on initial stage of the development.

The elderly age of the patients with long conservative treatment, reduced blood supply of the gland, expressed plasmolymphatic infiltration of the tissue requires to leave greater thyroid remnant (6-10 g). Morphologically in such patients detected regenerate type of histogram.

Sometimes a long anamnesis and conservative treatment, in association with expressed sclerosis of the gland or its nodular or cystic transformation requires performing of thyroidectomy.

Postoperative period. The clinical course of early postoperative period in the patients undergone the operation mainly depends both on quality of preoperative preparation, and on technique of the surgical intervention. In some patients, particularly with severe form of thyrotoxicosis, during first days after the

operation it is possible to observe exacerbation of thyrotoxicosis – postoperative thyrotoxic response.

There are three degrees of postoperative thyrotoxic response: mild, moderate gravity and severe.

Characteristic signs of mild degree is the tachycardia up to 120 per minute, fever as high as 38°C, satisfactory state of the patient, tachypnea.

The moderate gravity of thyrotoxic response manifests by mild psychomotor excitement. They complain of general weakness, headache, fever sensation, rapid pulse to 120-140 per minute (rhythmic, tense), sometimes extrasystole. Temperature raises as high as 38,5-39°C. Characteristic the considerable sweating, tachypnea, superficial sleep.

Severe degree of thyrotoxic response is characterized by expressed psychomotor excitement. The patients are restless, frequently change positions in the bed, they complain of considerable sweating, permanent fever sensation and expressed tremor. Hyperemia of the face, pulsate vessels of the neck and cyanosis of lips are evident. The pulse rate usually exceeds 140 per minute, irregular and soft. The breathing is superficial. Body temperature is 39-40°C. The sleeplessness in such patients is almost impossible to liquidate by hypnotic and narcotics agents.

Complication of a postoperative period

1. **Thyroid storm** is the severe complication of postoperative period in the patients with thyrotoxicosis (thyrotoxic crisis, acute postoperative thyrotoxicosis). The crisis develops mainly on the second or third day after the operation. If is failed to liquidate it in a day after the onset, the patient can die.

The clinical development of such crisis is acute or fulminant. It manifests by excitement, up to psychosis and coma, motor disorders, tachycardia (pulse rate – 150-200 per minute), complete arrhythmia, fever as high as 40°C and more, hyperemia of the face, neck, limbs, cyanosis, extremely sweating, diarrhea.

Pathogenically thyroid storm is caused by excessive releasing of thyroid hormones. It can arise as the result of rough palpation of the thyroid, treatment by antithyroid drugs, radioactive iodine, infections and traumas.

The crisis requires an urgent and complex treatment. Infusion therapy includes transfusion of haemodes, solutions of glucose with vitamins, plasma, and albumin. Major doses of glucocorticoids, narcotics, neuroleptanalgesia are instituted. Also desirable administration of sedative antihistamine drugs, adrenergic blockers, cardiac glycosides, oxygenotherapy, hypothermia, particularly on regions of major vessels, medical narcosis, extracorporeal method of detoxycation.

The prophylaxis of thyroid storm suggests an adequate preoperative preparation in order to gain euthyroid state of the patient, and also atraumatic performance of surgical intervention.

2. **The damage of laryngeal nerves** is the severe complication of operations on thyroid gland. Thus the paralysis of laryngeal nerves can be unilateral or bilateral, temporary or permanent. The basic causes of the paralysis: cutting off the nerve, its crushing or ligating, distention or compression. It is also necessary to specify that the bilateral injury of inferior laryngeal nerves is particularly dangerous.

The prophylaxis of the damages of laryngeal nerves basically consists of careful technique of subfascial resection of thyroid gland. It is always necessary gently manipulate in the region of inferior poles and "dangerous zone". The hemostasis in order to obtain "dry" operative wound should be carried out only under

the visual control.

The development of asphyxia requires an urgent intubation of trachea, or tracheostomy.

3. **Asphyxia.** It is caused, except bilateral injury of inferior laryngeal nerves, by the damage of trachea, tracheomalacia, laryngeal edema or inflection of trachea.

The tracheal wall injuries must be sutured by atraumatic needle with further muscular plastics. Tracheomalacia requires supporting sutures on trachea or application of prosthetics from synthetic material. Sometimes it is necessary to perform temporary tracheostomy.

4. **Air embolism.** The cause of this infrequent complication is the entering of air in the neck veins owing to suctional activity of chest and negative venous pressure. The prophylaxis of such complications consists of clamping of veins before cutting.

5. **Parathyroid tetany** is a difficult postoperative complication, which is hardly to undergo rehabilitation. The basic cause parathyroid tetany is the removal of parathyroid glands together with thyroid tissue. Besides it can result from impaired blood supply of the glands after the operation. The tetany develops on the base of mineral metabolism disorders, first of all, extremely reduced serum calcium (less 2,5 mEq/l)

It manifests by acute attack of wide-spread or localized cramps of separate groups of muscles of the upper or lower limbs. The most dangerous in this plan is the development of laryngospasm or tonic contraction of diaphragm.

Early clinical manifestation of parathyroid tetany is the Chvostek's signs (percussion near mandible angle causes muscular contractions of the face), Trousseau (occurrence of paresthesias and the sign of "obstetrician's" hand" after applying of tourniquet on brachium.

The treatment of parathyroid tetany consists of prompt administration of calcium agents. The usage of parathormone in complex with vitamin D is usually beneficial. Simultaneously with conservative treatment also performed transplantation of bony tissue.

The careful technique of subfascial resection of thyroid gland prevents the damage of parathyroid glands (except cases of their thyroid ectopy).

6. **Bleeding.** The cause of intra- and postoperative bleedings is insufficient mechanical hemostasis.

The postoperative bleedings are observed within first hours after operation as the result of unreliable hemostasis or slipping of ligature from vessels. Bleedings are clinically characterized by the prompt enlargement of swelling in the region of neck, considerable sopping of bandage by blood. Meanwhile, the patients complain of feeling of tightness in the neck, fear, tachycardia, cyanosis and dyspnea. The treatment of this complication is only surgical. The goal consists of complete disclosure and revision of the wound, carrying out of careful hemostasis.

The prophylaxis of postoperative bleeding includes a complex of measures, the most important of which is a subfascial technique of thyroid resection, careful reliable hemostasis and anatomic operating. The special attention is necessary to pay on lateral thyroid veins (Kocher's veins), which are short and empty directly into interior jugular vein. If the vessels are sclerosed, fragile and easily broken, they should be tied at once after cutting, instead of leaving on clamps up to the end of gland removal.

The damage of larynx, esophagus, major vessels of neck, lymph duct and pleura very seldom occurs. The laryngeal defects are sewed up with further covering by muscles. The esophageal wound is sewed up tightly, and feeding of the patient during 7 days is carried out through the tube.

INFLAMMATORY DISEASES OF THYROID GLAND

PURULENT THYROIDITIS

The purulent thyroiditis is a suppurative septic lesion of thyroid parenchyma. There are also cases of purulent inflammation goitrous thyroid gland – acute purulent strumitis.

Etiology and pathogenesis

The purulent thyroiditis arises owing to invasion of the thyroid by bacterial infection which spreads by hematogenous or lymphogenous way. The infecting agent most often represented by pyogenic streptococcus or staphylococcus aureus.

Pathology

Morphologically according to the character of inflammation distinguished the plain and specific thyroiditis, according to the course – acute, subacute and chronic.

The acute thyroiditis mainly develops in one lobe. Histologically revealed formation of the necrotic foci, hemorrhages, leukocytic infiltration of stroma with admixture of lymphocytes and macrophages.

The subacute thyroiditis (de Kerven) is histologically represented by the developments granulomatous inflammation. The stroma is commonly infiltrated by lymphocytes, leukocytes and large cells, which remind the cells of foreign bodies.

The chronic thyroiditis can manifest in the form of Hashimoto's and Riedel's goiter or specific thyroiditis caused by tuberculosis, lues or actinomycosis.

Hashimoto's goiter is characterized by predominant lymphocytic infiltration with formation follicles (lymphocytic goiter). Riedel's goiter ("iron" goiter, fibrous thyroiditis) is represented by growth of fibrous tissue.

In specific thyroidites revealed specific granulomas.

Classification

Distinguished acute purulent thyroiditis as diseases, which arise in unaltered thyroid gland, and acute purulent strumitis – the lesion of the goitrous transformed thyroid gland.

Symptomatology and clinical course

The onset of the disease is usually acute. It manifests by spontaneous sharp pain in the region of the neck, which amplifies at movements, speech and swallowing, fever, chills, weakness, sweating and tachycardia. On examination it is possible to note local reddening and swelling. Palpation reveals tissue tension, thyroid enlargement, density, with fluctuation in the site of lesion. In blood observed neutrophil leukocytosis and increased erythrocyte sedimentation rate.

Variants of clinical course and complications

The clinical course of the disease is characterized by the signs of purulent septic pathology of the neck. The process spreads outside the thyroid. Late diagnostics and inappropriate treatment result in discharge of the abscess outside, development of neck phlegmon, mediastinitis and sepsis. Nevertheless, in general, the outcome is favorable, on the site of abscess replaced by fibrous tissue, and the function of gland tends to norm.

The diagnostic program

1. Clinical examination, palpation.
2. General blood analysis.
3. Sonography of thyroid gland.
4. Diagnostic puncture of thyroid gland.
5. Bacteriological investigation of exudate.

The purulent thyroidites must be differentiated from simple thyroiditis and strumitis. The stormy course and transformation of plane inflammation into purulent that detected clinically, and by means of diagnostic puncture (purulent exudate) distinguish acute thyroiditis from the other inflammatory processes in thyroid gland.

Tactics and choice of treatment

Diagnostic puncture is necessary in order to confirm the diagnosis. If present fluctuation and purulent exudate it is necessary to carry out surgical management (drainage of suppurative focus). Such patients require antibiotics, analgesics, antiinflammatory and sedative agents.

AUTOIMMUNE THYROIDITIS

Autoimmune (lymphomatous) thyroiditis is the disease described by Hashimoto in 1912. In most cases it occurs in women of age 40-50.

Etiology and pathogenesis

The basic etiologic factor in the development of autoimmune thyroiditis is the release and entering of thyroid antigens into the blood as the result of inflammatory processes and traumas combined with surgical operations on thyroid gland. It has been found the presence of antibodies to thyroglobulins, colloidal component of thyroid gland and microsomal fraction. However the presence of antithyroid antibodies not always results in the damage of the thyroid. The cytotoxic properties of these antibodies manifest only after their interaction with T-lymphocytes and HLA antigens.

Pathology

The histological sign of autoimmune thyroiditis is the diffuse or focal thyroid infiltration by lymphocytes and plasma cells, which results in destruction of follicles and their basal membranes. Further thyroid tissue is replaced by connecting that leads to the focal fibrosis, which resemble nodes.

Classification

Distinguished diffuse and focal, and also hypertrophic and atrophic form of autoimmune thyroiditis.

Symptomatology and clinical course

Hashimoto's thyroiditis is characterized by the slow growth of goiter, the thyroid density, and gradual hypothyroidism. Besides develops the symptomatology, resulting from compression of organs and tissues of the neck by goiter. The patients complain of enlargement of thyroid gland, sense of tightness in the neck, difficult swallowing and breathing, pain in the region of gland and general weakness.

Enlargement of the thyroid is symmetric, it, as a rule, of dense consistence, and on palpation detected its nodular character. During pressing on one of the lobe of thyroid gland the elevation of contralateral lobe is observed (the sign of "swing").

Variants of clinical course and complications

Autoimmune thyroiditis is characterized by the development of hypothyroidism. Nevertheless there are also atypical clinical forms of the disease: autoimmune thyroiditis with thyrotoxicosis (Hashitoxicosis) with gradual transferring into hypothyroidism, lesion of one lobe with clinical course according to the nodular type of euthyroid or hypothyroid goiter. The autoimmune thyroiditis can arise in thyroid stump after surgery for different forms of goiter. The combination of autoimmune thyroiditis with thyroid adenoma or cancer and its transferring into chronic rarely occur.

Autoimmune thyroiditis can be complicated with hypothyroidism, compression of neck organs, in some cases - malignancy.

The diagnostic program

1. Clinical examination of the patient (palpation of thyroid gland).
2. Detecting of thyroid hormone concentration and thyrotropin.
3. Sonography of thyroid gland.
4. Detecting of antibodies to different thyroid components.
5. Biopsy of thyroid gland.



Fig.19. Hashimoto's thyroiditis sonography picture

Differential diagnostics

It should be carried out with endemic and sporadic goiter, Riedel's fibrous goiter and thyroid cancer.

Symmetric enlargement of thyroid gland, its dense consistence, nodular character, presence of autoimmune diseases in family history, high antibody capacity to thyroglobulins and microsomal fraction, development of hypothyroidism, positive response as reducing of goiter at prednisolone assay (20 mg of prednisolone during 7-10 days) – all these distinguish autoimmune thyroiditis from endemic and sporadic goiter, Riedel's thyroiditis. It is usually impossible to differentiate autoimmune thyroiditis from thyroid cancer on the base of clinical, instrumental and laboratory findings. In this case exclusive value has the morphological investigation – biopsy of thyroid gland or express histological investigation during operation. Macroscopically the gland is of pale-pink- greyish color with yellowish tone (instead of red-brown in norm), with atrophic sheath and thin-walled veins.

Tactics and choice of treatment

There is no specific therapy of autoimmune thyroiditis for today. The phenomena of hypothyroidism require nominating of replaceable therapy by thyroid hormones (thyroidine, thyroxine). Glycocorticoids and antihistamine agents are used in subacute form of autoimmune thyroiditis.

Surgery is applied in case of compression of the neck organs and suspicion on malignant tumour of the thyroid. The volume of operation has been still controversial. Preserving operation (isthmusectomy in

combination with wedge-like resection of lateral lobes, resection of thyroid gland) expedient only on initial stages of the disease with maintained thyroid activity. Taking into account a reality of malignancy, often relapses of goiter after small resections, the role of thyroid remnants as the foci of autoimmune aggression, the thyroidectomy is performed. After such operation the patients till the end of life should take substitution therapy by thyroid hormones. It has been noticed, that after thyroidectomy, in comparison with thyroid resection, the patients considerably better response to substitution therapy by thyroid hormones.

RIEDEL'S THYROIDITIS

Invasive fibrous Riedel's thyroiditis (synonyms – Riedel's goiter, "woody" goiter) is the extremely rare pathology, which consist of 0,005 % of persons undergone the surgery for different thyroid lesions. The disease described by Riedel in 1894 and 1897, occurs mainly in males.

Etiology and pathogenesis

The etiology of the disease is still unknown. There is the hypothesis that the Riedel's thyroiditis is the similar to such diseases, as idiopathic fibrous mediastinitis, sclerosing cholangitis and retrobulbar fibrosis. It gives the suggestion that the fibrous lesions of different organs can be the manifestation of one disease. Some authors suggest its infectious origin, though there are no reliable findings.

Pathology

This disease is represented by the development of connective tissue in thyroid gland with further transformation into a thick-fiber fibrous tissue. Between its layers there are small foci of adenomatous parenchyma, mainly of a microfollicular structure. The fibrous connective tissue spreads outside the thyroid, penetrates into muscles of the neck, untimely adherents to esophagus and trachea, causing their constriction and deformity.

Symptomatology and clinical course

The patients complain of goiter, dysphagia, difficult respiration and changes of a voice quality (chestvoice) down to aphonia. The gland becomes of woody or iron consistence, with change of configuration. Frequently observed the signs of tracheal and esophageal compression.

The disease is characterized by severe "malignant" course with aggressive growth of a fibrous tissue, which

can go on even after thyroid resection and after repeated operations for goiter relapse.

The function of the gland commonly preserved, though occasionally the course of disease complicated with hypothyroidism.

The most common complication of the disease is compression of organs and tissues of the neck, which results in dysphagia, dyspnea, and vocal changes.

The diagnostic program

1. Clinical examination of the patient (palpation of thyroid gland).
2. Sonography of thyroid gland.
3. Scanning of thyroid gland.
4. Puncture biopsy of thyroid gland.
5. Morphological investigation of thyroid tissue during surgical management.

Differential diagnostics

Riedel's thyroiditis is necessary to differentiate with thyroid cancer. Such signs as nodular character, metastases in lymph nodes of neck and paralysis of recurrent nerves, are characteristic for cancer. Owing to high density of Riedel's thyroiditis a puncture biopsy of the thyroid is usually hardly performed. Thus, it is necessary to carry out intraoperative histological investigation.

Macroscopically the adhesion of neck muscles with thyroid capsule is observed. The tissue of the gland is grey, with pink foci, according to consistence resemble cartilage and homogeneous on incision.

Tactics and choice of treatment

The treatment of invasive fibrous Riedel's thyroiditis only surgical and consists of complete removal of affected thyroid tissue. The advantage should be given to thyroidectomy.

[video](#)

DISEASES OF MAMMARY GLAND

ANATOMY

Knowledge of the anatomy and embryology of the breast and the chest structures under it is required not only for the performance of surgical procedures but also in planning therapeutic radiation, predicting sites of locally recurrent disease, and assessing the adequacy of surgical procedures used in an increasing number of therapeutic trials. Embryologically, the human breast develops in the thickened portion of ectodermal tissue known as the *milk streak* coursing from the pubis to the axilla in early fetal life. Late in the first trimester, the milk streak atrophies, leaving only its pectoral portion, which continues to thicken and to form the nipple bud. The entire gland then forms as a dermally derived organ lying within the subcutaneous tissue in a manner similar to that of sweat gland development. The ductal system develops from the nipple bud by invasion and downgrowth of primitive ectodermal cells from the nipple surface. The mature breast parenchyma lies cushioned in fat between the layers of superficial pectoral fascia. Between the deep layer of the superficial fascia and the fascial investment of the pectoralis major muscle, the breast rests on a thin layer of loose areolar tissue, the retromammary space, containing lymphatics and small vessels. When a total mastectomy is performed, the correct plane is found under the pectoral fascia and includes the retromammary space as emphasized by earlier surgeons and anatomists.

Deep to the pectoralis major muscle, the pectoralis minor muscle is enclosed in the clavipectoral fascia that envelopes it and extends laterally to fuse with the axillary fascia. In a standard modified radical mastectomy, dissection along the lateral border of the pectoralis minor muscle divides the axillary fascia and exposes the contents of the axilla. The number of lymph nodes found in the axillary space of patients undergoing mastectomy varies depending on the extent of dissection and the diligence of methods used to identify these nodes. An upper limit is established by the work of Durkin and Haagensen using ethanol clearing. These investigators found an average of 50 nodes in 100 specimens obtained in the course of a Halsted-type radical mastectomy. The current approach to less radical procedures has reduced the number of nodes retrieved.

To standardize the extent of axillary dissection, the axillary space is arbitrarily divided into three levels. Level I nodes are those in the external mammary, scapular, axillary vein, and central axillary groups, which lie lateral to the lateral border of the pectoralis minor muscle. Level II nodes are those in the central axillary group, which lie under the pectoralis minor muscle. The level III nodes are difficult to visualize and remove unless the pectoralis minor muscle is sacrificed or divided and include those subclavicular nodes medial to the minor muscle. The apex of the axilla is defined by the costoclavicular ligament (Halsted's ligament), at which point the axillary vein passes into the thorax and becomes the subclavian vein. Lymph

nodes in the space between the pectoralis major and minor muscles are known as the interpectoral group, or Rotter's nodes, described by Grossman and Rotter. Unless this group is specifically exposed, they are not encompassed in surgical procedures that preserve the pectoral muscles.

The lymphatic drainage of the breast is rich, and appreciation of the major pathways allows one to predict the sites most commonly containing lymph-borne metastases. Lymphatic channels within the breast follow centrifugal pathways from the subareolar plexus along major lactiferous ducts and then along efferent veins to draining nodal beds. The major site of drainage is to central axillary nodes. The internal mammary and interpectoral nodes, although primary routes of lymph flow, are rarely the sites of nodal metastasis from breast cancer in the absence of simultaneous axillary disease. Secondly, the lymphatic spread of cancer is into the high axillary nodes in the subclavicular chain and henceforth into the supraclavicular fossa.

As the surgeon endeavors to remove the lymph nodes of the axilla, a keen knowledge of the nerve structures in the axilla is required to avoid their sacrifice. Coursing close to the chest wall on the medial side of the axilla is the long thoracic nerve, or the external respiratory nerve of Bell, which innervates the serratus anterior muscle. This muscle is important in fixation of the scapula to the chest wall during adduction of the shoulder and extension of the arm, and its denervation results in the winged scapula deformity. For this reason, the long thoracic nerve is carefully preserved during standard axillary dissection. The second major nerve trunk encountered during axillary dissection is the thoracodorsal nerve to the latissimus dorsi muscle at the lateral border of the axilla. This nerve arises from the posterior cord of the brachial plexus and enters the axillary space under the axillary vein, close to the entrance of the long thoracic nerve, and then crosses the axilla to the medial surface of the latissimus dorsi muscle. The thoracodorsal nerve is usually preserved during dissection of axillary nodes, unless its sacrifice is required for complete removal of tumor-containing nodes.

Innervation of the pectoralis major muscle has gained the attention of some who emphasize the advantage of protecting these nerves during modified radical mastectomy. Loss of innervation results in a flaccid and atrophic muscle and a diminished tissue covering over the chest wall after amputation of the breast. These investigators have named the pectoral nerves according to their actual position as encountered during axillary dissection. The lateral pectoral nerve has a variable course. In the majority of patients, the lateral pectoral nerve travels around the lateral margin of the pectoralis minor muscle and is in a vulnerable position during the division of the clavipectoral fascia and exposure of the axillary space. If possible, this branch can be saved without compromising the dissection.

The final nerves of interest to the surgeon are the large sensory intercostal brachial or brachial cutaneous nerves that span the axillary space and supply sensation to the undersurface of the upper arm and skin of the chest wall along the posterior margin of the axilla. Cutting these nerves, which is routinely done in removing the lymph node-containing tissues, causes cutaneous anesthesia in these areas. It is helpful to emphasize this to the patients before operation. Denervation of the areas supplied by these sensory nerves can cause chronic and uncomfortable pain syndromes in a small percentage of patients. Contemporary surgeons have

advocated preservation of the most superior brachial cutaneous nerve, which crosses the central axilla to supply cutaneous sensation to the posterior upper arm. This nerve can be preserved without compromising the axillary dissection in many patients.

Microscopic Anatomy of the Breast

The mature breast is composed of three principal tissue types: epithelium, fibrous stroma and supporting structures, and fat. The relative amounts tend to vary with age, but there is even greater variability among individual women. In youth, the predominant tissues are epithelium and stroma, replaced by fat in the breast of older women. For this reason, mammography in women younger than 30 years of age, whose breast tissue is dense with stroma and epithelium, produces images without much definition that are rarely useful clinically. In contrast, fat absorbs relatively little radiation and provides a contrasting background that favors detection of small density lesions in the older patient. Throughout the fat of the breast, coursing from the overlying skin to the underlying deep fascia, strands of dense connective tissue provide shape and hold the breast upward. These strands, devoid of epithelial elements, are called Cooper's ligaments. Because they are anchored into the skin, tethering of these ligaments by a small scirrhous carcinoma commonly produces a dimple or subtle deformity on the otherwise smooth surface of the breast.

The glandular apparatus of the breast is composed of a branching system of ducts, roughly organized in a radial pattern, which spread outward and downward from the nipple-areolar complex. These lactiferous ducts are so named because they carry the milk produced in the more distal lobular groupings. At the summit of the arborizing ductal system, the subareolar ducts widen to form the lactiferous sinuses, which then exit through 15 to 20 orifices on the nipple. These large ducts close to the nipple are lined with a low columnar or cuboidal epithelium that abruptly meets the squamous epithelium of the nipple surface, which invades the duct for a short distance. Awareness of this junction helps understand Paget's disease of the nipple, described later.

At the opposite end of the ductal system and after progressive generations of branching, the ducts end blindly in clusters of spaces that are called terminal ductules or acini. These are the milk-forming glands of the lactating breast and together with their small efferent ducts or ductules are known as the lobular units or lobules. The terminal ductules are invested in a specialized loose connective tissue that contains capillaries, lymphocytes, and other migratory mononuclear cells. This intralobular stroma is clearly distinguished from the denser and less cellular interlobular stroma and from the fat within the breast.

Under the luminal epithelium, the entire ductal system is surrounded by a specialized myoepithelial cell of ductal epithelial origin that has contractile properties and serves to propel secretion of milk toward the nipple. Outside the epithelial and myoepithelial layers, the ducts of the breast are surrounded by a continuous basement membrane containing laminin, type IV collagen, and proteoglycans. The basement membrane layer is extremely important in differentiating *in situ* from invasive breast cancer. Continuity of this layer around proliferations of ductal cells guarantees that progression to an invasive cancer has not yet occurred.

BREAST DEVELOPMENT AND PHYSIOLOGY

In many mammalian species, full breast development requires the stimulation of copulation or pregnancy. Humans do not require either of these two events to initiate and complete breast maturation. Appreciation of the stages of breast development is necessary to understand many benign and even malignant states that come to clinical attention. During adolescence, the breast is composed primarily of dense fibrous stroma and scattered ducts lined with epithelium. In the United States, puberty begins at about 12 years of age, during which time there is hormone-dependent maturation of the genital organs. In the breast, this process entails increased deposition of fat, formation of new ducts by branching and elongation, and the first appearance of lobular units. This process of growth entails cell division and is under the control of estrogen, progesterone, adrenal hormones, pituitary hormones, and trophic effects of insulin and thyroid hormone. There is evidence that local growth factor networks are also important, including epidermal growth factor, which can replace estrogen as a developmental mammogen. The exact timing of these events and the coordinated development of both breast buds may vary from the average in individual patients. The term *prepubertal gynecomastia* refers to the symmetrical enlargement and projection of the breast bud in a young girl before the average age of 12, unaccompanied by the other changes of puberty. This process, which may be unilateral, should not be confused with neoplastic growth and should not be subjected to biopsy.

The mature or *resting* breast contains fat, stroma, lactiferous ducts, and lobular units. During phases of the menstrual cycle or in response to exogenous hormones, the breast epithelium and lobular stroma undergo cyclic stimulation. It appears that the dominant process is hypertrophy and alteration of morphology rather than hyperplasia. In the late luteal (premenstrual) phase, there is accumulation of fluid and intralobular edema that appears to correspond to the clinical complaint of breast engorgement, which may be painful.

On physical examination, and even by mammography, this may lead to increased nodularity and even be mistaken for development of a dominant tumor. Accordingly, ill-defined masses in premenopausal women are correctly observed through the course of one or two menstrual cycles. Finally, any alteration in the periodicity of the menstrual cycle, such as anovulatory cycles, can cause accentuation of engorgement, pain, and nodularity.

[video](#)

With pregnancy, there is diminution of the fibrous stroma to accommodate the hyperplasia of the lobular units. This formation of many new acini or lobules is termed the *adenosis of pregnancy* and is influenced by high circulating levels of estrogen and progesterone and by levels of prolactin that steadily rise during gestation. After birth, there is sudden loss of the placental hormones and the continued high level of prolactin. This may be the principal trigger for lactation. The actual expulsion of milk is under hormonal control and is caused by the contraction of the myoepithelial cells that surround breast ducts and terminal ductules. There is no evidence for innervation of the myoepithelial cells; their contraction appears to be in response to the pituitary-derived peptide oxytocin. Stimulation of the nipple appears to be the

physiologic signal for continued pituitary secretion of prolactin and for the acute release of oxytocin.

When breast feeding ceases there is a fall in prolactin and no stimulus for release of oxytocin. The breast then returns to a resting state and to the cyclic changes induced when menstruation begins again. With the approach of menopause, phases of the menstrual cycle may not be as symmetrical and regular. This irregularity can induce functional nodularity and breast pain where there had been none in earlier years. Menopause is defined by a cessation in menstrual flow for a significant period of time (i.e., 6 months or more) and the variable appearance of constitutional systems such as diaphoresis, minor psychological disturbances, or even clinical depression. For the breast, menopause results in involution and a general decrease in the epithelial elements of the resting breast. These changes include increased fat deposition, diminished connective tissue, and the virtual disappearance of the lobular units. The persistence of lobules, hyperplasia of the ductal epithelium, and even cyst formation can all occur under the influence of exogenous ovarian hormones. Most commonly, hormones are administered to relieve the symptoms of menopause, to prevent demineralization of bone, or to slow the appearance of atherosclerosis. The surgeon evaluating patients at any age for breast disease should inquire about the menstrual history, establish the cessation of menses in postmenopausal women, and record the use of any exogenous hormones. It is important that the pathologist who is examining biopsy material also have this information.

Abnormal Physiology and Development

Gynecomastia. Hypertrophy of breast tissue in men is a common clinical entity for which there is frequently no identifiable cause. Haagensen distinguishes *pubertal hypertrophy*, occurring in young boys between the ages of 13 and 17 years, from *senescent hypertrophy*, which occurs in men older than 50. The enlargement in teenage boys is very common and is frequently bilateral but may be unilateral. Unless it is unilateral or painful, it passes unnoticed and regresses with adulthood. Pubertal hypertrophy is generally treated by reassurance and without operation. Surgical excision should be discussed only if the enlargement fails to regress and the breast is cosmetically unacceptable.

Hypertrophy in older men is also common and may regress spontaneously. It is frequently unilateral, although the contralateral breast may enlarge with passage of time. The discoid mass is smooth, firm, and symmetrically distributed beneath the areola. It may be tender, and patients occasionally complain of breast discomfort. A number of commonly used medications, such as digoxin, thiazides, estrogens, phenothiazines, and theophylline, may exacerbate senescent gynecomastia. In addition, gynecomastia may be a systemic manifestation of hepatic cirrhosis, renal failure, and malnutrition. There should be little confusion with carcinoma occurring in the male breast. Carcinoma is usually not tender, it is asymmetrically located either beneath or beside the areola, and may be fixed to the overlying dermis or to the deep fascia. As with pubertal hypertrophy, gynecomastia in older men is usually left untreated. A dominant mass suspected of carcinoma should be sampled or carefully observed.

Nipple Discharge. The appearance of a discharge from the nipple of a nonlactating woman

is frequently frightening to the patient and misunderstood by the physician. Nipple discharge is very common and rarely associated with an underlying carcinoma. It is important to establish whether the discharge comes from one breast or from both breasts, whether it comes from multiple duct orifices or from just one, and whether the discharge is grossly bloody or contains blood. A milky discharge from both breasts is termed *galactorrhea*. In the absence of lactation or history of recent lactation, galactorrhea may be associated with increased production of prolactin. Radioimmunoassay for serum prolactin is diagnostic. However, true galactorrhea is very rare and is diagnosed only when the discharge is milky (contains lactose, fat, and milk-specific proteins).

Unilateral non-milky discharge coming from one duct orifice is surgically significant and warrants special attention. However, the underlying cause is rarely a breast malignancy. In one review of 270 subareolar biopsy results of discharges coming from one identifiable duct, and without an associated breast mass, carcinoma was found in only 16 patients (5.9%). In each of these cases, the fluid was either grossly bloody or tested strongly positive for occult hemoglobin. In another series of 249 patients, including both multiple-duct and single-duct discharges, breast carcinoma was found in 10 (4%). In 8 of these patients, a mass lesion coexisted with the discharge. Among 67 patients with breast cancer presenting with nipple discharge and studied by Leis and colleagues, only 8 (12%) had no palpable mass and 7 (10%) had a negative mammogram. To conclude, nipple discharge that comes from a single duct and contains blood must be investigated further. However, in the absence of a palpable mass or a suspicious mammogram, this symptom is usually not associated with cancer.

The most common cause of spontaneous nipple discharge from a single duct is a solitary intraductal papilloma in one of the large subareolar ducts directly under the nipple. Fibrocystic change, or cystic mastopathy, typically produces multiple-duct discharge and is another commonly associated finding. Subareolar duct ectasia, producing inflammation and dilatation of large collecting ducts under the nipple, is a common finding in the aging breast and usually produces multiple-duct discharge. In summary, nipple discharge that is bilateral and comes from multiple ducts is usually not a surgical problem. Discharge from single ducts is not commonly associated with carcinoma in the absence of detectable blood or a palpable mass. Bloody discharge from a single duct requires surgical biopsy to establish diagnosis. Intraductal papilloma is found in the majority of cases. If an occult cancer is found, it invariably is an early intraductal lesion.

Breast Pain. Painful breast tissue is an exceedingly common symptom but is usually of functional origin and very rarely a symptom of breast cancer. Haagensen²⁴ carefully recorded the symptoms of women presenting with breast carcinoma and found pain as an unprompted symptom in only 5.4% of patients. Although not a symptom of cancer, breast pain is a common reason for patients to seek medical attention. Breast pain appears to be aggravated by abnormal menstrual cycles and may be seen in young women with menstrual irregularity, as a premenstrual symptom, or when exogenous ovarian hormones are administered during and after the menopause. In addition, fibrocystic change, in its severest forms, may cause disabling breast pain. Although many observers find painful cystic mastopathy is aggravated by excessive

intake of caffeine, nicotine, or commonly used antihistamines, other investigators disagree.

Fibrocystic Change (Cystic Mastopathy, Cystic Mastitis). Fibrocystic change, popularly referred to as *fibrocystic disease*, represents a spectrum of clinical and histologic findings and describes a loose association of cyst formation, breast nodularity, stromal proliferation, and epithelial hyperplasia. Fibrocystic change appears to represent an exaggerated response of breast stroma and epithelium to a variety of circulating and locally produced hormones and growth factors. Clinically, patients with fibrocystic change have dense, firm breast tissue with palpable lumps and frequently gross cysts. This condition is commonly painful and tender to touch. Histologically, the lesion recognized as fibrocystic complex contains macrocysts, microcysts, stromal fibrosis, adenosis, and a variable amount of epithelial metaplasia and hyperplasia. All these changes can occur alone or in combination and to a variable degree in the normal female breast. Autopsy studies have questioned whether any of these changes, except perhaps macrocysts, are abnormal. In fact, all of these lesions occur commonly in the breasts of elderly patients and appear to have no particular pathologic potential. It appears preferable to describe each of the lesions separately and comment about the extent and severity of the process. The term *fibrocystic disease* should be abandoned in the absence of any well-defined clinical and pathologic syndrome.

As discussed later, there is no consistent association between fibrocystic complex and breast cancer. It is well established that women who have undergone breast biopsy for any reason, regardless of the underlying pathology, have a slightly higher risk of developing subsequent breast cancer. Moreover, the incidence of finding fibrocystic disease in autopsied breasts from women dying of causes other than breast cancer exceeds the incidence of these same changes in cancer-containing breasts. For those patients with fibrocystic changes, higher risk appears to concentrate in those whose biopsy specimens show abnormal ductal and lobular hyperplasia and, to a lesser extent, cyst formation. Therefore, the fibrocystic complex appears to be an exaggerated or abnormal response to otherwise physiologic stimuli in most patients and represents a health risk only in certain subsets.

Galactocele. A galactocele is a milk-filled cyst that is round, well circumscribed, and easily movable within the breast. It usually occurs after the cessation of lactation or when feeding frequency has been curtailed significantly. Haagensen states that it may occur up to 6 to 10 months after breast feeding has stopped. The pathogenesis of galactocele is not known for certain, but it is thought that inspissated milk within a large lactiferous duct is responsible. The tumor is usually located in the central portion of the breast or under the nipple. Needle aspiration produces thick, creamy material that may be tinged dark-green or brown. Although it appears purulent, the fluid is sterile. The treatment is needle aspiration. Withdrawal of thick milky secretion confirms the diagnosis, operation is reserved for those cysts that cannot be aspirated or that become superinfected.

Absent or Accessory Breast Tissue. Absence of breast tissue (amastia) or absence of the nipple (athelia) are very rare anomalies. Unilateral rudimentary breast development is much more common, as is adolescent hypertrophy of one breast with more normal development of the other. In contrast, accessory breast tissue (polymastia) and accessory nipples (supernumerary

nipples) are both common. Supernumerary nipples are usually rudimentary and occur along the milk line from the axilla to the pubis in both males and females. They may be mistaken by the patient for a small mole. However, accessory nipples are removed only for cosmetic reason. True polythelia refers to more than one nipple serving a single breast and is very rare.

Accessory breast tissue is commonly located above the breast in the axilla. Rudimentary nipple development may be present, and lactation is possible with more complete development. Accessory breast tissue, which may present as an enlarging mass in the axilla during pregnancy, is treated by surgical removal if it is large, cosmetically deforming, or to prevent enlargement during future pregnancy.

Breast Imaging

The goal of any technique that seeks to image the breast is to extend the capability of physical examination to either detect smaller abnormalities or to provide more information about palpable abnormalities. Mammography is clearly the most sensitive and specific test that can be used to complement the physical examination of the breast. It is used either as a diagnostic modality that seeks to answer specific questions about the health of the breast or as a screening test that seeks to find any abnormality within the breast. A variety of other methods have been used to generate useful images of the breast. Of these, ultrasonography is the only one in common use today. Thermography, which images heat generated by the breast, was added to the Breast Cancer Detection Demonstration Project (BCDDP) to evaluate its usefulness as a screening tool. However, because of a low overall yield, thermography was dropped from the project. Computed tomography (CT) has been used by some investigators with success but can require contrast medium enhancement, has limited ability to resolve small abnormalities, and requires a larger exposure of radiation. CT appears to be the best way to image internal mammary nodes and to evaluate the chest and axilla after mastectomy. Magnetic resonance imaging (MRI) is a technique that requires expensive equipment. The long times required to construct a suitable image are acceptable for diagnostic applications but prohibit MRI as a screening tool. MRI may have a role in evaluating breasts that are difficult to image or after prosthetic implantation. Digital imaging is an evolving hybrid technology that has found application in chest radiography and may be used to store radiographic information in a digital format or to directly produce images. This technology is improving and may have application in mammography.

Diagnostic Mammography. Film-screen mammography has replaced xeromammography as the standard breast imaging technology. Xeromammography was developed by the Xerox corporation and produced a blue image viewed in ambient reflected light. Modern film-screen mammography uses a combination of an enhancing screen and a molybdenum anode tube that produces low kilovolt electron photons. The enhancing screen converts and amplifies a low-energy radiation beam into high-energy photons that, in turn, expose a standard x-ray film. This technique uses compression of the breast between plexiglass plates to lessen the thickness of the tissue through which the radiation must pass and to separate adjacent structures and improve resolution. The image, like standard x-rays, is viewed using transmitted light and is a

negative image. Film-screen mammography delivers an average glandular dose of radiation that is less than 100 mrad. (0.1 cGy. or 0.1 rad.). In comparison, the average dose to the center of the breast in patients undergoing barium swallow is more than 10-fold the dose of two-view mammography.

The mammographic features of malignancy can be broadly divided into density abnormalities (including masses, asymmetries, and architectural distortions) and microcalcifications. Each mammogram should also be assessed for the presence of abnormalities in the axillary nodes and for the presence of skin or nipple changes, such as thickening or retraction. These mammographic features can coexist in any one particular abnormality and may exist in the presence or absence of physical findings. In fact, integration of each of the radiographic features and the physical findings leads to a prediction of malignancy.

Nonpalpable Mammographic Abnormalities. Mammographic abnormalities that cannot be detected by physical examination are classified in three broad categories: (1) lesions consisting of microcalcifications only, (2) density lesions (masses, architectural distortions, and asymmetries), and (3) those with both calcifications and density abnormalities. The incidence of malignancy after biopsy depends on the characteristics of the radiographic finding. Lesions with both microcalcifications and a mass effect, spiculated masses, and linear branching calcifications carry the highest probability of being malignant. However, even well-defined densities can be malignant. To be certain, not every abnormality should undergo biopsy, and recommendations must be made by the surgeon in consultation with an experienced radiologist. For those patients not undergoing biopsy, interval mammograms must be done to ensure stability of the abnormality.

If a biopsy is performed, it is usually done after mammographic placement of a needle or hook wire. A newer alternative is automated stereotactic core-needle biopsy. This procedure requires a large and dedicated unit, which is expensive. The patient generally lies prone with the breast hanging through the table. A robotic arm and biopsy gun is positioned by computerized analysis of triangulated mammographic images. A small amount of local anesthesia is used at the point of core-needle entry into the breast. Firing the machine obtains a core biopsy through the abnormality. Many series containing hundreds of patients have shown good correlation between stereotactic biopsy and subsequent needle-placement open biopsy. For lesions with calcification, a specimen radiograph must be done to confirm the presence of the abnormality in the specimen. This is true for both stereotactic and open biopsy procedures. Cooperation is required between the radiologist, surgeon, and pathologist for the correct interpretation of biopsy material obtained by either technique. If stereotactic biopsy is available to the surgeon, judgment must be used to determine the optimal biopsy strategy. Because stereotactic biopsy does not remove the abnormality, a subsequent localization and open procedure will be required if a cancer is found and the patient wishes to attempt breast conservation. If the abnormality proves benign after stereotactic biopsy, follow-up mammography needs to be done if the lesion is indeterminate. The false-negative rate of stereotactic biopsy is low but finite.

Screening Mammography. The goals of screening mammography differ from those of diagnostic mammography. Screening studies seek to identify any abnormality, maximizing sensitivity and cost effectiveness. During the past three decades, there have been eight major randomized controlled trials that compared a screened population to women given usual care. Although screening intervals and mammographic techniques differed, each of these studies included mammography with or without clinical breast examination in the screened arms. In the United States, a single uncontrolled study was done to demonstrate the feasibility of a population-based screening program. The BCDDP screened 280,000 women annually in 29 centers throughout the United States. Each center offered free mammograms and a breast examination and taught breast self-examination. Participants in the BCDDP have been followed for 14 years, and survival data were published. Because this was not a controlled trial, comparison has been made to national statistics compiled by the Surveillance, Epidemiology, and End Results (SEER) program within the National Cancer Institute (NCI). Comparison of survival data 8 years after diagnosis of cancer in the BCDDP to corresponding data from the SEER program demonstrates improved survival in the screened population, which seems to be explained by a shift to earlier stage disease in the BCDDP.

BENIGN BREAST TUMORS AND RELATED DISEASES

Breast Cysts. Cysts within the breast are fluid-filled, epithelial-lined cavities that may vary in size from microscopic to large, palpable masses containing as much as 20 to 30 ml. of fluid. As discussed, cysts are generally discovered by physical examination and confirmed by ultrasound or needle aspiration. At least one woman in every 14 will develop a palpable cyst, and 50% of cysts are multiple or recurrent. Cysts occur as solitary abnormalities, called macrocysts or gross breast cysts, or as part of a generalized process of microscopic cyst formation. This latter disease process is frequently bilateral and the cystic transformation can be extensive. The pathogenesis of cystic formation is not well understood; however, cysts appear to arise from destruction and dilation of lobules and terminal ductules. Three-dimensional microscopic studies and extensive sectioning have shown that stricture and fibrosis at or near terminal branching of small ductules, combined with continued secretion by the distal lobule, result in expansion of a cavity containing fluid and lined by ductal epithelium.

Cysts are unquestionably influenced by ovarian hormones, a fact that explains their sudden appearance during the menstrual cycle, their rapid growth, and their spontaneous regression with completion of the menses. Most women with new cyst formation present after the age of 35 and rarely before the age of 25 years. The incidence of cyst development steadily increases until the age of menopause and sharply declines after menopause. Autopsy studies of women dying with clinically normal breasts generally confirm the age relationship of gross cyst development but do find that the breasts of older women can contain gross and microscopic cysts. New cyst formation detected clinically in older women commonly is explained by the use of exogenous hormone replacement.

When encountered during operation, cysts are frequently dark. These are often referred to as

blue dome cysts, and they reflect the dark cyst fluid contained within. Grossly, they are usually unilocular and lined by a smooth and glistening surface, although larger cystic structures may be trabeculated and multiloculated. Histologically, cysts are frequently lined by a flattened epithelium. However, the epithelial layer may display apocrine metaplasia or may have papillary features. Intracystic carcinoma is exceedingly rare. Rosemond was able to report only three examples in over 3000 cyst aspirations (0.1%), and other investigators confirm this exceedingly low incidence. Regarding the risk of developing cancer for women with cystic disease, no studies demonstrate an increased risk in women with small or microscopic cysts. For patients with large cysts, called gross cystic disease by Haagensen, there remains some controversy. Patey and Nurick found no increase in cancers subsequent to cyst aspiration. Of 810 cancers treated by Patey, only 10 had a previous history of gross cysts. Other recent reviews have emphasized that women with gross cysts have a risk of twofold to fourfold that of age-matched women without cysts. The studies of Page and associates and of Dupont and Page do not show a significant increase in cancers after long-term follow-up of over 2000 women who underwent biopsy of palpable cysts when compared with the slight increase borne by women who have had breast biopsy alone.

Fibroadenoma and Related Tumors. Fibroadenoma (adenofibroma) is a benign tumor composed of both stromal and epithelial elements in the breast. After carcinoma, fibroadenoma is the second most common solid tumor in the breast and is the most common tumor in women younger than age 30 years. The benign nature of this lesion was recognized in 1840 by Cooper, who referred to the lesions as “chronic mammary tumors.” Clinically, they present as firm, solitary tumors that may increase in size over several months of observation. They may be lobulated but will slip easily under the examining fingers. At operation, fibroadenomas appear to be well-encapsulated masses that may easily detach from the surrounding breast tissue. By history, fibroadenoma is favored over cyst in the adolescent or young adult; and on examination, these tumors are distinguished from cysts by the needle aspiration that yields no fluid. Mammography is of little help in distinguishing between cysts and fibroadenomas; however, ultrasound usually clearly shows the cavity of a cyst. The gross appearance and histopathology are distinctive of fibroadenoma. Grossly, the tumor appears well encapsulated, with smooth borders that may be lobulated. Histologically, a variable proportion of epithelial and stromal proliferation is present, and the stroma may be quite cellular or replaced by acellular swirls of collagen. In older patients, the lesions may contain deposits of calcium within dense fibrosis. The epithelium can display the entire spectrum of proliferative changes seen elsewhere in the breast. Although fibroadenomas are not considered to have a malignant potential, the epithelial elements appear to be at risk for neoplasia just as epithelium elsewhere in the breast. More than 100 invasive and noninvasive carcinomas have been reported in preexisting fibroadenomas since 1985. Most of these (50%) have been lobular carcinoma *in situ*, 35% were infiltrating carcinomas, and 15% were intraductal carcinoma. The risk of cancer in a newly discovered fibroadenoma found in the breast of a young woman is obviously exceedingly rare and is not an issue that influences treatment. A modest risk of subsequent carcinoma in women who have previously been treated for fibroadenoma has been reported, but the magnitude is about two times the general population. This is only slightly higher than the

reported excess risk for all women who have had a breast biopsy.

The treatment of fibroadenoma follows that for any unexplained solid mass within the breast. The great majority of patients in the United States are treated by excisional biopsy to remove the tumor and establish the diagnosis. It is worth recognizing that a different approach is taken by physicians in other countries. A typical fibroadenoma is frequently left untreated by European physicians when the tumor is encountered in the breast of a young women. If excision is recommended, the approach to a young women with a typical fibroadenoma on examination should be very different than the approach in older women with indeterminate masses. Cosmetic incisions around the areola with a modest amount of tunneling to remove the lesion are commonly used techniques and are proper for the treatment of fibroadenoma. Emphasis should be placed on removing a minimum amount of breast tissue adjacent to a typical fibroadenoma. If the gross appearance is that of a fibroadenoma, frozen section is superfluous, the patient can be immediately reassured, and final diagnosis can be established by inspecting permanent sections.

Juvenile Fibroadenoma, Giant Fibroadenoma. Clinicians treating breast masses should be aware of these two terms, which are sometimes confusing. *Giant fibroadenoma* is a descriptive term that applies to a fibroadenoma that attains an unusually large size, typically greater than 5 cm. Haagensen calls these lesions “massive adenofibromas in youth” to denote their common occurrence in adolescent women. *Juvenile fibroadenoma* refers to the occasional large fibroadenoma that occurs in adolescents and young adults and histologically is more cellular than the usual fibroadenoma. Both these lesions overlap, and both may display remarkably rapid growth within the breast. Although alarming to the patient and physician, prompt surgical removal is always curative. The term *benign cystosarcoma phyllodes* refers to a tumor that may be difficult to distinguish clearly from juvenile fibroadenoma. If the tumor has been completely removed, the diagnosis of benign cystosarcoma should reassure the surgeon and the patient that the risk of recurrence is low, particularly if the patient is an adolescent or a young adult. Malignant cystosarcoma phyllodes is a distinctive and aggressive tumor discussed later.

Hamartoma and Adenoma. Although probably not of the same histogenesis as fibroadenoma, these tumors are benign proliferations of variable amounts of epithelium and stromal supporting tissue. The hamartoma is a discrete nodule that contains closely packed lobules and prominent, ectatic extralobular ducts. By physical examination, mammography, and gross inspection, the hamartoma is indistinguishable from fibroadenoma. The nodule is entirely benign, and removal is curative. The mammary adenoma or tubular adenoma has been a more elusive entity to define. Page and Anderson describe this tumor as a cellular neoplasm of ductules packed closely together and forming a sheet of tiny glands without supporting stroma. During pregnancy and lactation, these tumors may increase in size and histologic examination shows secretory differentiation. Malignancy is not a feature of tubular adenoma or lactating adenoma, but biopsy is required to establish the diagnosis.

Breast Abscess and Infections. Breast abscess commonly occurs in the subareolar breast tissue and may be recurrent and difficult to treat. Although the exact cause is not known, subareolar duct ectasia and obstruction of major ducts may lead to proliferation of bacteria and subsequent abscess. Further destruction of the normal ductal openings leads to fistula formation and chronic recurrent abscess. Mammary duct ectasia, first named by Haagensen, is an inflammatory condition that causes distortion and dilation of the lactiferous sinuses under the nipple. It is a common entity and is frequently responsible for nipple inversion in older women. In understanding subareolar abscess and probably mastitis in general, it is useful to remember that the nipple and areolar complex contains secretory ducts that are exposed to the environment. Chronic inflammation, duct dilation, and obstruction may combine at the nipple to produce circumstances that favor bacterial invasion.

The treatment of acute abscess of periareolar tissue should be conservative if possible. Antibiotics with broad-spectrum coverage should be used initially. More severe infections may require hospitalization and intravenous antibiotics. A small incision with drainage is preferred if the process cannot be controlled by antibiotics alone. Needle aspiration may be attempted, but the abscess cavity is usually multiloculated. Recurrent infection is best treated by excision of the diseased subareolar ducts as described by Haagensen and others. However, recurrence is common and leads to chronic and recurring infection.

Mastitis describes a more generalized cellulitis of breast tissue that may involve a large area of the breast but may not form a true abscess. The etiology appears to be an ascending infection beginning in subareolar ducts and extending outward from the nipple. Occasionally, mastitis involves areas of cystic disease and may be sterile. Mastitis presents with erythema of the overlying skin, pain, and tenderness to palpation. There is induration of the skin and underlying breast parenchyma. Especially in young women, an apparent mastitis may develop that is dramatic in its presentation and responds poorly to antibiotics but resolves spontaneously. The etiology is unknown but may be related to menstrual cycle irregularity. More commonly, mastitis complicates lactation, possibly due to inspissation of milk, obstruction, and secondary infection. Local measures such as application of heat, ice packs, or use of a mechanical breast pump on the affected side have all been recommended. If conservative measures are not effective, administration of broad-spectrum antibiotics is usually indicated. In many situations, the differential diagnosis of acute mastitis includes inflammatory carcinoma. It is important to follow patients with mastitis and confirm that there has been a complete resolution of symptoms and signs. The erythema produced by an inflammatory carcinoma will not resolve with conservative measures and generally will worsen in a short period of follow-up.

Papilloma and Related Ductal Tumors. Solitary intraductal papillomas are true polyps of epithelial-lined breast ducts. Solitary papillomas are located under the areola in the majority of cases. In contrast, certain patients have multiple intraductal papillomas that Haagensen believes are more likely to be peripherally located and associated with an increased risk of cancer. Solitary papillomas may be located in peripheral ducts and can grow to large size, presenting as a breast mass. When papillomas attain a large size, they may appear to arise

within a cystic structure, probably representing a greatly expanded duct. In general, these lesions are less than 1 cm. but can grow to as large as 4 or 5 cm.

Tumors under the nipple and areolar complex often present with a bloody nipple discharge. Less frequently, they are discovered as a palpable mass under the areola or as a density lesion on the mammogram. Treatment is total excision through a circumareolar incision. The surgeon must keep in mind that one of the most difficult areas in differential diagnosis is between a papilloma and invasive papillary carcinoma. Because these lesions can infarct, scar, and even develop squamous metaplasia, they can appear bizarre and disordered. Most pathologists urge evaluation on permanent sections for the majority of papillary lesions before more extensive surgery is undertaken.

It is also important not to confuse the commonly used term *papillomatosis* with either solitary or multiple papillomas. Papillomatosis refers to epithelial hyperplasia that commonly occurs in younger women or is associated with fibrocystic change. This lesion is not composed of true papillomas. Hyperplastic epithelium in papillomatosis may fill individual ducts like a true polyp but has no stalk of fibrovascular tissue nor the frondlike growth. Solitary papillomas are entirely benign and do not predispose to development of cancer in the patients who have them. Page and Anderson state that the degree of subsequent risk for breast cancer in patients with either papillomatosis or with true papillomas, either solitary or multiple, relates to the degree of atypical epithelial proliferation associated with them.

MALIGNANT TUMORS OF THE BREAST

Epidemiology

The likelihood of developing breast cancer is highly dependent on both age and the interval over which an individual is at risk. Although the lifetime risk of developing breast cancer is estimated at 10%, more than one half of patients with breast cancer are older than 65. Furthermore, the 10% figure is based on a hypothetical interval that extends from birth to age 110. A more realistic view considers risk starting at a particular age and extends over a finite period of time. For instance, the chance that a woman age 35 will develop breast cancer during the next two decades of her life, until age 55, is only 2.5%. A woman who is 50 has close to a 5% chance of developing cancer before she turns 75 and a 65-year-old woman has a 5.5% chance of getting breast cancer before she turns 85 years of age. These figures apply to white women in the United States; the same idealized African-American woman has a lifetime risk of breast cancer that is 7% or 8%.

The odds of dying of breast cancer over the ideal lifetime of birth to 110 are about 3.6%. Although carcinoma of the lung has overtaken breast cancer as the leading cause of cancer-related death in American women, breast cancer remains far more common. Of 595,000 new cases of cancer among women in 1996, 186,000 occurred in the breast while 78,000 arose in the lung.

Several studies based on cancer registries in the SEER program of the NCI have reported an increasing attack rate for breast cancer in the United States. In Washington State there has been

an annual estimated increase of 2.5%. A greater increase was apparent among black and low-income women. In the Connecticut Tumor Registry the overall annual incidence rose from 53 cases per 100,000 women during the years 1935 to 1939 to an average annual rate of 86.4 per 100,000 for the years 1975 to 1979. The annual rate of increase during 1980 to 1985 averaged 3%; during the past 5 years, this rate of increase has declined to about 1% per year. Data from other studies and from other western countries confirm the increase in the incidence of breast cancer. Despite this increased incidence, the age-adjusted death rate from carcinoma of the breast was stable until 1979 and may have decreased in the 5-year period between 1979 and 1984. Although it is tempting to speculate that this is a real improvement attributable to early diagnosis or improved therapy, no direct proof can be cited.

Pathology of Breast Cancer

Modern classification of breast cancer attempts to recognize morphologic patterns that reflect both the histogenesis of the malignancy and its biologic behavior, or prognosis. As such, these classifications impose artificial divisions on diseases that are fundamentally poorly understood. As advances are made in our understanding of breast malignancies, the classifications presented today will be improved on by future generations of surgeons and pathologists. Malignancies of the breast are broadly divided into epithelial tumors of cells lining ducts and lobules and nonepithelial malignancies of the supporting stroma. A second important division of the epithelial tumors that recognizes their evolution is between noninvasive and invasive cancers. The noninvasive malignancies are proliferations of either ductal or lobular cells confined to the basement membrane. These are true carcinoma *in situ*. As in other organs, carcinoma *in situ* commonly coexists with invasive cancer. In the breast, this association is very frequent and argues for the progression of cancer through stages of noninvasive proliferation, disruption of the basement membrane, and invasion of the supporting stroma.

Most pathologists utilize the classification scheme proposed by the World Health Organization (WHO) and outlined in the fascicles of the Armed Forces Institute of Pathology. Historically, Foote and Stewart presented most of this material in 1945, including a recognition of noninvasive carcinoma and the differentiation between tumors originating in the breast lobule from those arising in the lactiferous ducts.

Ductal Carcinoma *in Situ* (DCIS), Intraductal Carcinoma. The concept of a purely noninvasive form of breast cancer and recognition of various subtypes have evolved slowly since the beginning of this century. DCIS was probably recognized first by surgeons who appreciated its favorable prognosis. Bloodgood, at the Johns Hopkins Hospital, impressed by the tumor of an early patient of Halsted, recognized a disease he termed “pure comedo tumor” that had an extremely favorable prognosis. Pathologically, breast ducts become swollen with proliferating malignant epithelium. In the solid or comedo type, the ducts can expand to visible proportions 1 or 2 mm. in diameter. The term *pure intraductal carcinoma* refers to the absence of detectable invasion of the basement membrane. Devoid of blood supply, the center of the lesion undergoes necrosis and the intraductal spaces fill with necrotic cellular debris. The

central detritus can undergo dystrophic calcification that is fine, focally clustered, and even linear and branching when seen on high-quality mammography. This process can locally produce a palpable mass if multiple ducts are involved.

Subtypes of DCIS are now well recognized and frequently reported pathologically. The solid or comedo type is most common and probably more virulent. Papillary or cribriform DCIS are characterized by papillary projections of tumor cells into the ductal lumen or by the presence of a branching, cribriform pattern filling ducts. These types are less likely to form palpable masses and uncommonly calcify to produce a mammographic abnormality. However, it is important to emphasize that these subtypes can coexist and that DCIS is best described by the pathologist in terms of its extent, multicentricity, and involvement of the surgical margin. Confusion arises in a number of ways. First, the uninitiated may confuse the term *infiltrating ductal carcinoma* with the term *intraductal carcinoma*; the former is invasive disease and the latter is noninvasive disease. Second, these two stages of tumorigenesis usually coexist, particularly when they are carefully searched for in pathologic specimens. Finally, as discussed later, the treatment and outcome for patients with intraductal disease may depend on variables such as multifocality, multicentricity, and extent of disease in a way more demanding for the pathologists and surgeons than in the past.

Lobular Carcinoma *in Situ* (LCIS) or Lobular Neoplasia. This disease of the breast lobules or acini was first clearly delineated by Foote and Stewart in 1941, who gave it the name *lobular carcinoma in situ*. Haagensen first used the term *lobular neoplasia* to emphasize its more benign course. Pathologically, it is a proliferation of small round epithelial cells within lumens of multiple breast acini. The resulting picture is multiple clusters of epithelial cells forming islands of neoplastic cells but maintaining a lobular architecture. Although the ducts expand with proliferating cells, they usually do not reach the large size seen with DCIS.

Unlike DCIS, LCIS never forms a palpable mass by itself and is therefore not recognized on physical examination. In addition, there are no mammographic findings in LCIS. It does not form a density and rarely calcifies, both of which are typical for DCIS. Therefore, this is a disease that is recognized incidentally after biopsy for another abnormality that is producing a clinical or mammographic finding. The treatment of this incidental pathologic entity remains controversial and is reviewed later.

Infiltrating Ductal Carcinoma. This is the most common malignant tumor in the breast recognized after biopsy. The term *ductal carcinoma* refers to its origin from ductal epithelium; *infiltrating* describes its growth pattern and distinguishes this lesion from noninvasive carcinoma. Some add the terms *not otherwise specified* (NOS) and *no special type* (NST) to emphasize that this disease is diagnosed after the other, more distinctive histologies have been eliminated. The tumor infiltrates into a variable amount of stroma as cords or islands of malignant epithelium. It may form primitive glandular forms, but not to the extent of a pure tubular carcinoma. As discussed, and as reflected in the WHO classification, many infiltrating carcinomas display an *in situ* component. This fact reflects its ductal origin and may be used to prove a mammary origin of the tumor. The stromal *reaction* may be intense and has led to the older term *scirrhous carcinoma* of the breast.

Clinically, most infiltrating ductal carcinomas present as a mass found on physical examination or as a density lesion on the mammogram. Microcalcifications seen mammographically are commonly found in the necrotic centers of the intraductal component but may be seen in the infiltrating component as well. The treatment of infiltrating carcinoma is discussed later, and the approach taken is generally the same regardless of the morphologic appearance. However, there is great biologic diversity among breast cancers; and after identification of an infiltrating ductal carcinoma, pathologists attempt to describe features that reflect the likely behavior of the tumor.

The evaluation of breast cancers should always specify the tumor size, the status of the surgical margin, and the content of estrogen and progesterone receptors. In addition, the nuclear and histologic grade is frequently reported and modern evaluation may include measurement of DNA content and estimation of the proliferating fraction, or S-phase. Vascular invasion, tumor necrosis, and the extent of the intraductal component are all used to make decisions about the primary or adjuvant treatment of patients with operable breast cancer. The dedicated breast surgeon must be acquainted with all of these parameters and their interpretation.

Invasive Lobular Carcinoma. This disease probably originates in the breast lobule. Invasive lobular carcinoma constitutes between 3% and 15% of all invasive breast cancers, depending on the series consulted. Histologically, the tumor is composed of small round cells that infiltrate surrounding stromal tissue in a peculiar *Indian file* fashion. Lobular carcinoma presents in an identical fashion as ordinary infiltrating ductal carcinoma and produces no distinguishing mammographic features. The treatment of lobular carcinoma is the same as for the more common ductal carcinomas and may carry a better prognosis. There may be a somewhat higher incidence of bilateral cancer or of second primary tumors in the contralateral breast. However, this is rarely used to justify prophylactic procedures in the contralateral breast in the absence of synchronous disease.

Less Common Forms of Ductal Carcinoma. These tumors, although heterogeneous, are all morphologic variants of common ductal carcinoma. In general, these less common variants have improved prognosis, reflecting their more differentiated phenotype. One exception to this rule is medullary carcinoma of the breast, which is pathologically characterized by bizarre and anaplastic tumor cells surrounded by a prominent lymphocytic infiltrate with a scant fibrous stroma. Although the epithelial component is undifferentiated, this phenotype appears to enjoy a small but significant survival advantage when compared with infiltrating ductal carcinoma NOS. Mucinous carcinoma, also called colloid carcinoma, is characterized by well-differentiated epithelial cells surrounded by a large accumulation of extracellular and extraluminal mucin that is secreted by the carcinoma cells. This histologic type enjoys a favorable prognosis in several published series. Although there are no definite clinical or mammographic signs of mucinous and medullary carcinoma, these tumor types are suggested by a well-circumscribed density with smooth borders that tend to be softer on physical examination. The final major histologic type that is distinctive and well differentiated is tubular carcinoma. This tumor is characterized by infiltrating tubular structures, lined by one

cell layer, and with an open central space. The tumor is characteristically small, is scirrhous, and has an excellent prognosis after treatment. The descriptions of these histologic variants refer to their predominant features. However, each may coexist with more undifferentiated infiltrating carcinoma of the usual type. In general, if the tumor is composed of definite infiltrating ductal carcinoma of poor differentiation, the final diagnosis reflects the poorest histologic pattern. Because tubular and mucinous variants are less likely to metastasize, some modern breast surgeons tailor their primary approach to these lesions by, for instance, omitting axillary node dissections for small and well-differentiated lesions.

Modern Surgical Procedures for Invasive Breast Cancer

The surgical treatment of breast cancer, for the most part, concerns the treatment of potentially curable cancer that is confined to the breast and regional lymph nodes. For early stages of breast cancer, surgical removal provides a reasonable chance for cure. Although the approach to operable breast cancer has changed dramatically over the past century, so, too, has the clinical presentation of breast tumors changed. In 1894, Halsted presented his first 50 patients treated by the “complete operation,” which became the radical mastectomy. Over the next 75 years, radical mastectomy was used to treat virtually every breast malignancy operated on for cure in the United States. Examination of Halsted's first cases found at least two thirds with locally advanced disease and 60% with clinically evident axillary nodal metastases. By comparison, a 1980 survey by the American College of Surgeons found that 85% of patients presented with Stage I or Stage II disease. The frequency of cases with positive axillary lymph nodes was 40%, and the average tumor presenting to physicians in the 1970s measured 2 cm. or less. In addition to these fundamental changes, realization that 90% of treatment failures will be systemic or visceral recurrences has led surgical oncologists to explore alternatives to radical mastectomy as an initial approach to operable breast cancer.

Surgical Procedures Past and Present

In 1982, the American College of Surgeons investigated surgical practice in cases of operable breast cancer and compared results with practice in earlier years. Clearly, a change in surgical practice occurred in the mid 1970s with an abrupt shift from radical mastectomy to modified radical mastectomy. Procedures that preserved the breast, as described later, were performed in only 7.2% of cases in this survey. Current estimates of conservative breast procedures range between 20% and 40%, and this procedure probably continues to increase in popularity. The following paragraphs describe procedures in widespread use now and in the past.

Radical and Extended Radical Mastectomy. In the radical mastectomy, the breast and underlying pectoralis muscles are sacrificed leaving a bare chest wall. Regional lymph nodes along the axillary vein up to the costoclavicular ligament (Halsted's ligament) are removed with the breast specimen. This procedure frequently requires a skin graft and uses incisions placed either vertically or obliquely. Prosthetic reconstruction is impossible unless muscle flaps are mobilized to cover the anterior chest defect. Cure of breast cancer can certainly be achieved by

the application of this procedure alone. Other studies document both the strengths and weaknesses of maximal local therapy represented by this procedure. The personal series of Haagensen reports results from treatment of 1036 patients; 727 patients with clinically negative nodes (Stage A, Columbia clinical staging) had a survival of 72.4% at 10 years. In contrast, only 42.3% of clinically node-positive patients (Stage B) survived at 10 years. These figures were confirmed by the National Surgical Adjuvant Breast and Bowel Project (NSABP) early trial of adjuvant thiotepa. By 10 years, 76% of patients with histologically positive nodes suffered recurrence of breast cancer and one fourth of patients with negative nodes failed surgical treatment. In contrast, *local failure* rates have been extremely low since introduction of the Halsted radical mastectomy. Published figures are generally between 5% and 7% and provide the standard against which newer procedures are judged.

The extended radical mastectomy is a standard radical mastectomy to which en bloc removal of internal mammary nodes is added. This procedure was popularized in the United States by Urban, who reported a 35.5% 10-year survival in patients undergoing extended radical mastectomy. Other studies have resulted in abandonment of the extended procedure. A large prospective trial and several uncontrolled series have failed to provide evidence of improved clinical outcome after extended radical mastectomy.

Modified Radical Mastectomy. Modified radical mastectomy refers to a procedure combining total mastectomy with removal of axillary lymph nodes in continuity with the mastectomy specimen. This is the most widely used procedure to treat operable breast cancer and is the alternative to breast-sparing procedures described later. Modified radical mastectomy leaves the pectoralis major muscle intact, providing a soft tissue covering over the chest wall and a normal-appearing junction of the shoulder with the anterior chest wall and avoiding the hollow defect inferior to the clavicle that accompanies the removal of the pectoralis muscle. The patient is left with intact musculature around the shoulder and a situation that is well suited to prosthetic reconstruction. Two forms of the procedure are in use by surgeons: the Patey procedure and modifications described by Scanlon and the procedure described by Auchincloss.

Patey, at the Middlesex Hospital in London, developed a procedure that preserves the pectoralis major muscle and sacrifices the underlying pectoralis minor muscle to remove levels I, II, and III lymph nodes in the axilla. A large number of Patey procedures performed by Handley, who wrote extensively about this procedure, were reviewed independently and reported by Donegan and associates. The survival of patients with negative axillary nodes was 82% at 10 years with a local recurrence rate of 5%. For patients with positive nodes, the survival was 48%, very similar to results with radical mastectomy. Thus, preservation of the pectoralis major muscle did not appear to produce inferior results. Scanlon modified the Patey procedure by dividing but not removing the pectoralis minor muscle, allowing removal of apical (level III) nodes and preservation of the lateral pectoral nerves to the major muscle.

The procedure described by Auchincloss differs from the Patey procedure by not removing or dividing the pectoralis minor muscle. This modification limits the complete removal of high axillary nodes but is justified by Auchincloss, who calculated that only 2% of patients will

potentially benefit by removal of the highest level nodes. It is probable that the Auchincloss mastectomy was the most popular procedure for breast cancer in the United States during the past decade.

Wide Local Excision and Primary Radiation Therapy. Excision of the primary tumor with preservation of the breast has been referred to by many names, including partial mastectomy, segmentectomy, tylectomy, or lumpectomy. *Wide local excision* seems to be the most descriptive term for the procedure, which removes the malignancy with a surrounding rim of grossly normal breast parenchyma. An even more aggressive local procedure designed to remove 1 to 2 cm. of adjacent breast and overlying skin is called *quadrantectomy*. In modern practice, these more limited surgical procedures are applied as part of a multidisciplinary approach to breast cancer and always include postoperative radiation therapy, giving at least 4500 cGy. to the whole breast and usually including a boost of radiation to the tumor bed. Axillary dissection is done through a separate incision in the majority of patients. Therefore, conservative breast surgery or breast preservation usually refers to wide local excision of the primary tumor, whole breast radiation, and a separate axillary dissection.

The Management of Noninvasive (*in Situ*) Carcinoma

Special attention to the problem of *in situ* carcinoma is justified by the increasing frequency of its recognition and the controversy surrounding the proper treatment of noninvasive cancer of the breast. Treatment decisions require appreciation of the various types and stages of *in situ* disease and demand that the surgical oncologist understand this sometimes complicated disease process. Moreover, its relationship to invasive cancer is a fascinating biologic question. Understanding these early proliferative states should give investigators a clue to the underlying cause of breast cancer. Finally, finding early malignancy is the goal of population screening. It is hoped that the high cure rate after treatment of noninvasive breast carcinoma will someday contribute to a decline in mortality from this malignancy.

Ductal Carcinoma in Situ (DCIS) or Intraductal Carcinoma

Before modern mammography, intraductal lesions presented as palpable tumors in 50% or more of patients. In the 1980s, more were recognized by the calcifications they produce, observable on mammograms, than by physical detection. The comedo or solid form fills small mammary ducts and is likely to undergo central necrosis. The central detritus within ducts undergoes dystrophic calcification, producing fine punctate and even linear calcification, which is seen first on mammography long before invasive disease develops into a palpable mass. Clearly, these are early lesions that can be approached in a different manner from that for usual invasive disease. Treatment recommendations for patients with intraductal carcinoma are based on consideration of several issues, including (1) occult invasive cancer coexisting with the *in situ* lesion, (2) multicentricity of intraductal carcinoma, (3) the occurrence of disease in the contralateral breast, and (4) the natural history after diagnosis by biopsy.

Axillary metastases are very rare in lesions that appear to be pure intraductal cancers. In the three studies just listed, a total of 316 intraductal carcinomas were studied. Axillary metastases

were present in only 5 of these; in one case a second primary invasive cancer in the ipsilateral breast was present. Although not all patients underwent axillary dissection, there were no nodal recurrences during the follow-up of these patients. Significantly, all patients with positive nodes had large tumors, palpable tumors, or microscopic invasion

Many authors who have written about intraductal carcinoma are influenced by the high incidence of multicentric, multifocal, and even bilateral disease. *Multifocal* is a term referring to disease within the vicinity or same quadrant as the dominant lesion. Multicentric refers to disease in distant sites or quadrants within the same breast. Bilateral implies the concurrent finding of disease in both breasts. The existence of multicentric disease has led many to favor mastectomy for the treatment of DCIS, and concerns about bilaterality have prompted the use of prophylactic procedures on the contralateral breast. The most widely reported figure for the incidence of multicentric disease within the ipsilateral breast is 33%, or one third of cases in which a biopsy discloses intraductal cancer as the predominant lesion. However, estimates vary depending on how extensively other quadrants of the breast are examined. For example, although a large review of NSABP material failed to find multicentric disease, only a single random section from remote quadrants was examined. In contrast, Schwartz reported an incidence of approximately 37% after examining four random sections from each remote quadrant and from under the areola. Other estimates range between the results of these two studies. The risk of multicentric disease appears to depend both on the histologic type of the intraductal tumor and on the size or extent of the primary cancer. In the study of Lagios and associates, 2 of 24 small tumors less than 2 cm. (8%) were associated with disease in other quadrants of the breast. In moderate size tumors, between 2 and 5 cm., 2 breasts of 16 examined (12.5%) had multicentric disease. In large tumors whose extent was greater than 5 cm., all 13 cases examined had disease in remote quadrants of the ipsilateral breast. The study of Patchefsky and colleagues examined multicentricity as a function of the histology of the primary tumor. In agreement with other authors, this series found that micropapillary pathology was associated with a high (80%) incidence of multicentric disease. An intermediate percentage (40%) of papillary and comedocarcinomas were associated with remote disease, and solid and cribriform types were lowest. Size was not noted in this study. Pathologic review of DCIS should include an estimate of size, a comment about pathologic margins, and assessment of multifocal disease within the surgical specimen. Histologic type should be noted and a statement made about the presence or absence of microscopic invasion.

Lobular Carcinoma in Situ

Lobular carcinoma *in situ* is a relatively uncommon disease that occurs predominantly in younger, premenopausal women. As noted earlier, this disease is rarely diagnosed before biopsy, does not form a palpable mass, and rarely calcifies. Haagensen has collected the largest series of patients, all of whom were identified by review of biopsy material. In this review, LCIS was found in 3.6% of more than 5000 biopsies done for benign disease. Haagensen prefers the term *lobular neoplasia* to emphasize that this pathologic entity predisposes to subsequent carcinoma after a long latency period. However, in a review of 297 patients with

LCIS (lobular neoplasia) treated by biopsy and careful observation, Haagensen determined the actuarial probability of developing carcinoma at the end of 35 years was 21.4%. Compared with the Connecticut Tumor Registry data, a risk ratio (observed to expected cases) of 7:1 was calculated. Significantly, 40% of the carcinomas that subsequently developed were purely *in situ* lesions and one half of all subsequent carcinomas occurred in the contralateral breast. Haagensen preferred a practice of close observation after a biopsy diagnosis of LCIS. Similar data have led others to express doubts about the need for mastectomy. These authors have recommended a conservative policy of close observation after a biopsy diagnosis of LCIS or lobular neoplasia.

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METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №7 "ABDOMINAL HERNIAS "

The aim. To master clinics, diagnostics, differential diagnostics of reducible and complicated abdominal hernias. To choose the treatment tactics, indications and contraindications for operative intervention, analgesia, type of plastics, principles of postoperative period course, expertise of the ability to work and the rehabilitation after operations for hernias.

Professional orientation of students. Abdominal hernias are common for 3-5% of population. Hernia limits the working ability of the hernia carrier and is a vitally dangerous illness due to eventual complications (incarceration, inflammation). The lethality at incarcerated hernias depend on many factors and therefore it ranges from 2 to 17% according to various authors.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened

Student's independent work program:

Theme №1. General herniology. Inguinal hernias.

1. Definitions: "Hernia", "Eventration", "Prolapse".
2. Classification of abdominal hernias. Hernias elements. Sliding hernias.
3. Causes of hernias.
4. General clinical signs of reducible hernias.
5. General principles of operative treatment of abdominal hernias.
6. Inguinal hernias. Classification. Clinical features.
7. Anatomic-topographic characteristics of inguinal area and channel.
8. Differential diagnosis of inguinal hernias.
9. Anatomic and physiological base of inguinal channel plastics in inguinal hernias treatment.
10. Peculiarities of operative treatment of congenital and sliding inguinal hernias.
11. Postoperative period after hernioplastics, prescriptions in the postoperative period.

Theme №2. Femoral, umbilical hernias, hernia of the linea alba, hernia of semilunar line (Spigelius').

1. Anatomic-topographic features of the femoral channel, linea alba, Spigelius' line and other "weak" sites of the abdominal wall.
2. Femoral hernias. Classification. Clinical features, differential diagnostics.

3. Principles of the operative treatment of femoral hernias.
4. Umbilical hernias. Classification. Clinical features, differential diagnostics.
5. Umbilical hernias. Operative treatment.
6. Hernias of the linea alba. Clinical features, differential diagnostics.
7. Hernias of the linea alba. Operative treatment principles.
8. Relapse and postoperative ventral hernias. Peculiarities of diagnostics, clinical course and curing tactics.
9. Usage of plastic materials of different origin in abdominal hernias treatment.

Theme №3. Hernial complications.

1. Classification of incarceration types.
2. Main clinical manifestation of hernial incarceration.
3. Peculiarities of diagnostics, differential diagnostics of incarceration depending on hernial localization.
4. General features of the operative treatment of hernial incarceration.
5. Peculiarities of the clinical course and curing tactics in retrograde hernial incarceration.
6. Parietal incarceration. Peculiarities of clinical course and curing tactics.
7. Peculiarities of clinical course and curing tactics in incarceration of sliding hernias.
8. Self reduction of incarcerated hernia. Curing tactics.
9. False hernia incarceration. Peculiarities of clinical course and curing tactics.
10. False reduction of incarcerated hernia. Peculiarities of clinical course and curing tactics.
11. Etiology of hernial irreducibility.
12. Clinical characteristics of the irreducible hernia.
13. Differential diagnostics of reducible and irreducible hernias.
14. Curing tactics in irreducible hernia.
15. Clinical characteristics and cause of hernial coprostasis.
16. Differential diagnostics of fecal and elastic hernial incarceration.
17. Curing tactics in hernial coprostasis.
18. Clinical characteristics and cause of inflamed hernia.
19. Differential diagnostics of inflamed and incarcerated hernias.
20. Differential diagnostics of inflamed and irreducible hernias.
21. Differential diagnostics of inflamed hernia and hernial coprostasis.
22. Curing tactics in inflamed hernia.
23. Hernia trauma. Peculiarities of diagnostics and curing tactics.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Anatomic and physiologic features of the anterior abdominal wall (weak sites of the abdominal wall).
2. Etiology, pathogenesis, classification of abdominal hernias and their complications.
3. Clinical characteristics of abdominal hernias of various localization.
4. Indications and contraindications for operative treatment of hernias, methods of hernial prophylaxis.
5. Typical variants of operative treatment of abdominal hernias of various localization.
6. Etiopathogenesis and classification of complications of abdominal hernias.
7. Clinics, diagnostics, differential diagnostics of the incarcerated, irreducible, inflamed, traumatic hernias, hernial

coprosthesis.

8. Indications and contraindications for conservative and operative treatment of complicated hernias.
9. Main peculiarities of operative treatment of complicated hernias.

The student has to be able to:

1. To take anamnesis of the patient's with suspicion for abdominal hernia.
2. To reveal main clinical signs of complicated and uncomplicated abdominal hernias.
3. To palpate the sites of inguinal and femoral rings, hernial protrusions.
4. To formulate the primary diagnosis.
5. To plan the examination of the patients and explain the examination data (general blood analysis, general urine analysis, X-ray examination of abdominal organs), to formulate the clinical diagnosis.
6. To define indications and contraindications for operative intervention, to choose the proper preoperative preparation, anesthesia, operative access and type of plastics according to the clinical form of the abdominal hernia.

Technical means and material provision of lectures (names and numbers of tables, compact discs, instruments quantity, subjectpatients, etc.) –

Multimedia projection of demonstration material (pictures, tables, videofilms) from compact disc “Facultative Surgery”, file “Hernias”.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. Which of the following statements regarding unusual hernias is incorrect?
 - A. An obturator hernia may produce nerve compression diagnosed by a positive Howship-Romberg sign.
 - B. Grynfeltt's hernia appears through the superior lumbar triangle, whereas Petit's hernia occurs through the inferior lumbar triangle.
 - C. Sciatic hernias usually present with a painful groin mass below the inguinal ligament.
 - D. Littre's hernia is defined by a Meckel's diverticulum presenting as the sole component of the hernia sac.
 - E. Richter's hernia involves the antimesenteric surface of the intestine within the hernia sac and may present with partial intestinal obstruction.

2. Staples may safely be placed during laparoscopic hernia repair in each of the following structures except:
 - A. Cooper's ligament.
 - B. Tissues superior to the lateral iliopubic tract.
 - C. The transversus abdominis aponeurotic arch.
 - D. Tissues inferior to the lateral iliopubic tract.
 - E. The iliopubic tract at its insertion onto Cooper's ligament.

3. The following Nyhus classification of hernias is correct except for:
 - A. Recurrent direct inguinal hernia—Type IVa.
 - B. Indirect inguinal hernia with a normal internal inguinal ring—Type I.
 - C. Femoral hernia—Type IIIc.
 - D. Direct inguinal hernia—Type IIIa.
 - E. Indirect inguinal hernia with destruction of the transversalis fascia of Hesselbach's triangle—Type II.

4. Which of the following statements about the causes of inguinal hernia is correct?
- A. Excessive hydroxyproline has been demonstrated in the aponeuroses of hernia patients.
 - B. Obliteration of the processus vaginalis is a contributing factor for the development of an indirect inguinal hernia.
 - C. Physical activity and athletics have been shown to have a protective effect toward the development of inguinal hernias.
 - D. Elevated levels of circulating serum elastolytic activity have been demonstrated in patients with direct herniation who smoke.
 - E. The majority of inguinal hernias are acquired.
5. The following statements about the repair of inguinal hernias are true except:
- A. The conjoined tendon is sutured to Cooper's ligament in the Bassini hernia repair.
 - B. The McVay repair is a suitable option for the repair of femoral hernias.
 - C. The Shouldice repair involves a multilayer, imbricated repair of the floor of the inguinal canal.
 - D. The Lichtenstein repair is accomplished by prosthetic mesh repair of the inguinal canal floor in a tension-free manner.
 - E. The laparoscopic transabdominal preperitoneal (TAPP) and totally extraperitoneal approach (TEPA) repairs are based on the preperitoneal repairs of Cheattle, Henry, Nyhus, and Stoppa.
6. What hernia is called sliding?
- A. Which is reducible
 - B. Which contains two or more organs
 - C. Which contains intraperitoneal organ
 - D. Which contains retroperitoneal organ
 - E. One of the wall of its hernial sac is represented by mesoperitoneal organ

The answers for the self-checking tests.

1 – C.

2 – D.

3 – E.

4 – D.

5 – A.

6 – E

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others "Hospital surgery". Ternopil: Ukrmendknyga, 1999.

3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%2007%20General%20herniology.%20Inguinal%20hernia.%20Femoral,%20umbilical%20hernias.htm

Additional –

1. Manual “Facultative Surgery” under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section “Diseases of the rectum and the colon”,
2. Compact disc “Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

The author: As. prof. A. Hospodarskyy

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №1 " ACUTE APPENDICITIS "

The aim. To master clinics, diagnostics, differential diagnostics of the acute simple, complicated and chronic appendicitis, peculiarities of acute appendicitis course in children, elderly people, pregnant women. To learn the treatment tactics, indications and contraindications for operative intervention, operative intervention, principles of postoperative period course, expertise of the working ability to and the reablement.

Professional orientation of students. Acute appendicitis is the most frequent urgent surgical illness. Its share makes up 60-70% of all urgent surgical illnesses. The lethality after appendectomy varies in the ranges of 0,1-0,15% and is determined also by diagnostic mistakes at untypical forms of the acute appendicitis, by diseases in children, elderly people and pregnant women

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened

Student's independent work program:

Theme №1. Acute noncomplicated appendicitis.

1. Clinical and pathanatomic classification of the acute appendicitis.
2. Clinical picture of the acute simple appendicitis
3. Clinical picture of the acute destructive appendicitis
4. Differential diagnosis of the acute appendicitis with the acute cholecystitis.
5. Differential diagnosis of the acute appendicitis with the acute pancreatitis.
6. Differential diagnosis of the acute appendicitis with the acute ileus.
7. Differential diagnosis of the acute appendicitis with the perforating ulcer of the stomach and the duodenum.
8. Differential diagnosis of the acute appendicitis with the extrauterine pregnancy.
9. Differential diagnosis of the acute appendicitis with the rightsided renal colic.
10. Examination programme of the patient with the acute appendicitis before the operation.
11. Choice of anesthesia, access and tpe of intervention.
12. Postoperative period after appendectomy, prescriptions in the postoperative period.
13. Expertise of the disablement and reablement of patients after the appendectomy.

Theme №2. Acute complicated appendicitis (infiltrate, abscess, peritonitis, pylephlebitis). Untypical forms of the acute appendicitis. Peculiarities of the acute appendicitis course in children, elderly people and pregnant

women.

1. Classification of the acute appendicitis complications.
2. Clinics, diagnostics and treatment of the appendicular infiltrate.
3. Clinics, diagnostics and treatment of the appendicular abscess.
4. Clinics, diagnostics and treatment of the appendicular peritonitis.
5. Clinical features of the retrocecal appendicitis.
6. Clinical features of the acute appendicitis at the pelvic appendix.
7. Clinical features of the acute appendicitis at the subhepatic appendix.
8. Clinical features of the acute appendicitis at the medial appendix.
9. Peculiarities of clinical course of the diagnosis of the acute appendicitis in children.
10. Peculiarities of clinical course of the diagnosis of the acute appendicitis in elderly people.
11. Peculiarities of clinical course of the diagnosis of the acute appendicitis in pregnant women.

Theme №3. Chronic appendicitis.

1. Classification of the chronic appendicitis.
2. Subjective clinical signs of the chronic appendicitis.
3. Objective clinical signs of the chronic appendicitis.
4. Differential diagnosis of the chronic appendicitis with chronic disease of the abdominal cavity organs:
 - a) ulcerative disease of stomach and duodenum,
 - b) chronic cholecystitis,
 - c) chronic pancreatitis,
 - d) Krohn's disease,
 - e) unspecific ulcerative colitis,
 - f) chronic diseases of female genital organs.
5. Differential diagnosis of the chronic appendicitis with the renal lithiasis.
6. Differential diagnosis of the chronic appendicitis with the lumbosacral radiculitis.
7. Treatment of patients with the chronic appendicitis.
8. Expertise of the disablement and reablement of patients

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.**Basic level of knowledge and skills:****The student must know:**

1. Anatomic and physiological features of the appendix.
2. Etiology, pathogenesis, classification of the acute and the chronic appendicitis and its complications.
3. Clinical characteristics of different forms of the acute and the chronic appendicitis.
4. Peculiarities the acute and the chronic appendicitis course in children.
5. Differential diagnostics of the acute and the chronic appendicitis.
6. Diagnostics peculiarities of the acute appendicitis complications (appendicular infiltrate, abscess, peritonitis, pylephlebitis, sepsis).
7. Major principles of treatment of the acute and the chronic appendicitis and their complications.
8. Principles of expertise of the disablement and reablement of patients who have undergone the appendectomy.

The student has to be able to:

1. To take the history of the patient with the suspect of the appendicitis.

2. To reveal main clinical signs and symptoms of different forms of the acute and the chronic appendicitis.
3. To argue and formulate the preliminary diagnosis.
4. To make a plan of the examination of the patient and explain the examination results (general blood test, general urine test, ultrasound diagnostics).
5. To define indications and contraindications for operative intervention, to choose properly the type anesthesia, operative access and tuype of intervention according to the clinical form of the appendicitis.

Technical means and material provision of lectures (names and numbers of tables, compact discs, instruments quantity, subjectpatients, etc.) –

Multimedia projection of demonstration material (pictures, tables, videofilms) from compact disc “Facultative Surgery”, file “Hernias”.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. Which of the following most often initiates the development of acute appendicitis?
 - A. A viral infection.
 - B. Acute gastroenteritis.
 - C. Obstruction of the appendiceal lumen.
 - D. A primary clostridial infection.

2. The diagnosis of acute appendicitis is most difficult to establish in:
 - A. Persons aged 60 and older.
 - B. Women aged 18 to 35.
 - C. Infants younger than 1 year.
 - D. Pregnant women.

3. Once a diagnosis of acute appendicitis has been made and appendectomy decided upon, which of the following is/are true?
 - A. Prophylactic antibiotics should be administered.
 - B. Prophylactic antibiotics are not necessary unless there is evidence of perforation.
 - C. If the appendix is not ruptured and not gangrenous, antibiotics may be discontinued after 24 hours.
 - D. Multiple antibiotics are in all cases preferable to a single agent.

4. Which of the following statements about pyogenic abscess of the liver are true?
 - A. The right lobe is more commonly involved than the left lobe.
 - B. Appendicitis with perforation and abscess is the most common underlying cause of hepatic abscess.
 - C. Mortality is largely determined by the underlying disease.
 - D. Mortality from hepatic abscess is currently greater than 40%.

5. Acute appendicitis is most commonly associated with which of the following signs?
 - A. Temperature above 104c F.
 - B. Frequent loose stools.

- C. Anorexia, abdominal pain, and right lower quadrant tenderness.
D. White blood cell count greater than 20,000 per cu. mm.
6. What manifestation is predominant for retroperitoneal appendicitis?
A. Clinic of acute abdomen
B. Dyspeptic syndrome
C. Clinic of retroperitoneal phlegmon
D. Clinic of acute intestinal obstruction
E. Clinic of acute pancreatitis
7. What manifestation is predominant for pelvic appendicitis?
A. Clinic of acute abdomen
B. Clinic of irritation of pelvic organs (dysuria, pulling rectal pain, tenesmi)
C. Clinic of retroperitoneal phlegmon
D. Clinic of acute intestinal obstruction
E. Clinic of acute pancreatitis

The answers for the self-checking tests.

- 1 – C.
2 – C.
3 – A.
4 – D.
5 – C.
6 – C.
7 – B.

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others "Hospital surgery". Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%2001%20Acute%20appendicitis.htm

Additional –

1. Manual "Facultative Surgery" under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section

“Diseases of the rectum and the colon”,

2. Compact disc “Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

The author: As. prof. A. Hospodarskyy

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №5 " ACUTE INTESTINAL OBSTRUCTION (AIO)"

The aim. To master clinics, diagnostics, differential diagnostics of various clinical forms of AIO. To learn the treatment tactics, indications and contraindications for operative intervention, operative intervention type at various clinical forms of AIO principles of postoperative period course, expertise of the working ability after operations for AIO.

Professional orientation of students. The acute intestinal obstruction makes up to 3,5% among the patients of surgical stationaries and up to 10% of patients with acute diseases of abdominal organs. The lethality at AIO according to different data, varies from 6 to 18-20%.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened

Student's independent work program:

Theme №1. Acute obstructive ileus.

1. Anatomic and physiologic data about the small and large intestines.
2. Definition of the concept and etiopathogenesis of the mechanical AIO.
3. Classification of AIO.
4. Reasons and types of the obstructive ileus.
5. Clinics, diagnostics, differential diagnostics of the obstructive ileus.
6. Differential diagnostics of the obstructive and strangulation intestinal obstruction
7. Verification methods of the obstructive ileus diagnosis.
8. Curing tactics choice at various clinical forms of the obstructive ileus.
9. Peculiarities of the preoperative preparation at various clinical forms of the obstructive ileus.
10. Principles of operative treatment, radical and palliative operations at the obstructive ileus.
11. Peculiarities of the postoperative period course, water-electrolytic and acid-alkaline balance correction after operations for the obstructive ileus.

Theme №2. Acute strangulate intestinal obstruction.

1. Classification of the strangulation intestinal obstruction.
2. Phases of clinical course of the strangulation intestinal obstruction.
3. Peculiarities of etiopathogenesis of the strangulation intestinal obstruction.

4. Clinics, diagnostics, differential diagnostics of various types of the strangulation intestinal obstruction.
5. Differential diagnosis of the strangulation intestinal obstruction.
6. Modern verification methods of diagnosis (ultrasonic examination, laparoscopy, roengenologic methods).
7. Curing tactics at the strangulation intestinal obstruction.
8. Choice of scope and method of operative interventions at various clinical types of the strangulation intestinal obstruction.
9. Peculiarities of the preoperative preparation and postoperative period course at the strangulation intestinal obstruction.
10. Principles of operative treatment of the strangulation intestinal obstruction.
11. Peculiarities of the postoperative period course.

Theme №3. Paralytic intestinal obstruction.

1. Concept definition, reasons of the paralytic intestinal obstruction.
2. Classification of the paralytic intestinal obstruction.
3. Clinics, diagnostics, differential diagnostics of various clinical forms of the paralytic intestinal obstruction.
4. Differential diagnosis of mechanical and dynamic paralytic intestinal obstruction.
5. Methods of diagnosis verification of the paralytic intestinal obstruction.
6. Curing tactics and method choice of paralytic intestinal obstruction depending on a clinical version.
7. Stimulation methods of motor-evacuative intestinal function.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Anatomic and physiologic features of various intestines sections.
2. Topography of abdominal organs.
3. Etiopathogenesis of the intestinal obstruction. Classification of AIO.
4. Phases of clinical course of the acute mechanical intestinal obstruction.
5. Reasons, clinics, diagnosis, differential diagnosis of the obstructive ileus.
6. Types, clinics, diagnostics, differential diagnostics of the strangulation intestinal obstruction.
7. Verification methods of diagnosis of the acute mechanical intestinal obstruction.
8. Peculiarities of water-electrolytic balance violations at AIO.
9. Curing tactics at AIO.
10. Choice of methods of conservative and operative treatment of patients with mechanical AIO.
11. Peculiarities of the preoperative preparation and principles of operative treatment of patients with mechanical AIO.
12. Correction principles of water-electrolytic and acid-alkaline balance violations and energetic balance in patients with AIO.
13. Expertise principles of the patients after operations for AIO.
14. Reasons, clinics, diagnosis, differential diagnosis of paralytic AIO.
15. Reasons, clinics, diagnosis, differential diagnosis of spastic AIO.

The student has to be able to:

- 1.To take the history of the patient with the suspect for AIO.
- 2.To reveal main clinical features and symptoms at various clinical forms of the obturative ileus.
- 3.To reveal main clinical features and symptoms at various clinical forms of the strangulation AIO.
- 4.To reveal main clinical features and symptoms at various clinical forms of the dynamic AIO.
- 5.To define free and limited liquid in the abdominal cavity, splash noise, characteristics of peristaltic violations.
- 6.To argue and explain the preliminary diagnosis.
- 7.To make a plan of patient examination and explain the examination results (general blood test, general urine test, roentgenologic examination of the alimentary tract, general view roentgenogram, barium passage, irriography, ultrasonic examination, biochemical blood test).
- 8.To define indications and contraindications for operative intervention, to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form of mechanical AIO.
- 9.To define principles of conservative treatment of the dynamic AIO.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.
(the instructor has tests & situational tasks)

1. History and physical examination permit the diagnosis of intestinal obstruction in most cases. Which of the following are important for the clinical diagnosis of small bowel obstruction?
 - A. Crampy abdominal pain.
 - B. Fever.
 - C. Vomiting.
 - D. Abdominal distention.
 - E. Leukocyte count above 12,000.
 - F. Abdominal tenderness.
2. Patients with established, complete, simple, distal small bowel obstruction usually have the following findings on plain and upright abdominal radiographs:
 - A. Distended small bowel identifiable by the valvulae conniventes.
 - B. Multiple air-fluid levels.
 - C. Modest amount of gas in the pelvis.
 - D. Peripheral, rather than central, distribution of gas.
 - E. Prominent haustral markings.
 - F. Free air.
3. Which of the following statement(s) about gallstone ileus is/are not true?
 - A. The condition is seen most frequently in women older than 70.
 - B. Concomitant with the bowel obstruction, air is seen in the biliary tree.
 - C. The usual fistula underlying the problem is between the gallbladder and the ileum.
 - D. When possible, relief of small bowel obstruction should be accompanied by definitive repair of the fistula since there is a significant incidence of recurrence if the fistula is left in place.
 - E. Ultrasound studies may be of help in identifying a gallstone as the obstructing agent.
4. Complete mechanical small bowel obstruction can cause dehydration by:

- A. Interfering with oral intake of water.
- B. Inducing vomiting.
- C. Decreasing intestinal absorption of water.
- D. Causing secretion of water into the intestinal lumen.
- E. Causing edema of the intestinal wall.

The answers for the self-checking tests.

1 – A,B,C,D,F.

2 – A,B.

3 – C.

4 – A,B,C,D,E.

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others "Hospital surgery". Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%2005%20Acute%20intestinal%20obstruction.htm
- 4.

Additional –

1. Manual "Facultative Surgery" under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section "Diseases of the rectum and the colon",
2. Compact disc "Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

The author: As. prof. A. Hospodarsky

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №2 " ACUTE CHOLECISTITIS, CHOLELITHIASIS "

The aim. To master clinics, diagnostics, differential diagnostics of various clinical forms of the cholelithiasis and pancreatitis. To learn to choose the treatment tactics, indications and contraindications for operative intervention, analgesia type, operative intervention type, principles of postoperative period course, expertise of the working ability after operations for biliary ducts and pancreas.

Professional orientation of students. Cholelithiasis and pancreatitis is the most frequent surgical illness of outer biliary ducts. The disease is constantly growing and is covering now up to 10-15% of the population; it is especially common for women after 40-50. Cholecystectomy still remains the main surgical treatment method. During the recent years endoscopic (laparoscopic) operative interventions on biliary ducts have become widely used in surgical practice.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Theme №1. Acute and chronic calculous cholecystitis.

1. Anatomic and physiologic data about extrahepatic biliary ducts.
2. Concept definition of etiopathogenesis of cholelithiasis.
3. Classification of the cholelithiasis.
4. Classification of the acute calculous cholecystitis.
5. Clinics, diagnostics, differential diagnostics of the acute simple calculous cholecystitis.
6. Clinics, diagnostics, differential diagnostics of the acute destructive calculous cholecystitis.
7. Clinics, diagnostics, differential diagnostics of the acute complicated calculous cholecystitis (perivesical infiltrate, abscess, gallbladder empyema, perforating cholecystitis, peritonitis, cholangitis, obstructive jaundice, cholepancreatitis).
8. Choice of treatment tactics at various clinical forms of the acute calculous cholecystitis.
9. Scope and methods of conservative and operative treatment of the acute calculous cholecystitis.
10. Peculiarities of the postoperative period course.
11. Classification of the chronic calculous cholelithiasis.
12. Clinical characteristics, diagnosis, differential diagnostics of the noncomplicated chronic calculous cholecystitis.
13. Clinical characteristics, diagnosis, differential diagnostics of the complicated chronic calculous cholecystitis (chronic empyema, gallbladder hydrops).

14. Modern methods of verification of chronic calculous cholecystitis diagnosis.
15. Choice of scope and methods of operative interventions at various clinical forms of chronic calculous cholecystitis.
16. Methods of intraoperative examination of biliary ducts
17. Peculiarities of the postoperative period course.
18. Disablement expertise after operations for the chronic calculous cholecystitis.

Theme №2. Obstructive jaundice.

1. Biochemical peculiarities of the bilirubin metabolism.
2. Jaundice classification.
3. Obstructive jaundice classification.
4. Differential diagnosis of the mechanical and parenchymatous jaundice.
5. Differential diagnosis of the mechanical and hemolytic jaundice.
6. Verification methods of obstructive jaundice reasons.
7. Peculiarities of the preoperative preparation of patients with obstructive jaundice.
8. Types of operative interventions which are used at various clinical versions of the obstructive jaundice.
9. Peculiarities of the postoperative period course after operations for obstructive jaundice.

Theme №3. Acute pancreatitis.

1. Anatomic and physiologic features of the pancreas.
2. Concept definition and pathogenesis of the acute pancreatitis.
3. Classification of the acute pancreatitis.
4. Clinics, diagnostics, differential diagnostics of the acute edematous pancreatitis.
5. Clinics, diagnostics, differential diagnostics of the acute hemorrhagic pancreatitis. 6. Cholecystopancreatitis. Clinics, diagnostics.
7. Curing tactics choice at various clinical forms of the acute pancreatitis.
8. Methods of conservative treatment of the acute pancreatitis.
9. Classification of the acute pancreatitis complications.
10. Clinical characteristics, diagnostics, differential diagnostics of the pancreatogenic fermentative peritonitis.
11. Clinical characteristics, diagnostics, differential diagnostics of the necrotic pancreatitis (pancreonecrosis).
12. Modern verification methods of diagnosis (ultrasonic examination, laparocentesis, laparoscopy).
13. Curing tactics at the acute complicated pancreatitis.
14. Choice of the scope and operative intervention method at various clinical forms of the acute complicated pancreatitis.
15. Clinical characteristics, diagnostics, differential diagnostics of the false cyst of the pancreas and its complications.
16. Choice of the scope and methods of conservative and operative treatment of patients with the false cyst of the pancreas.
17. Peculiarities of the postoperative period course.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Anatomic and physiologic data about the liver and the outer biliary ducts.
2. Modern theories and stages of biliary lithogenesis.
3. Definition and classification of various clinical forms of the cholelithiasis.
4. Modern pre-, intra- and postoperative examination methods of biliary ducts.
5. Clinical and biochemical characteristics of violations of hepatic functions at cholelithiasis.
6. Clinics, diagnostics, differential diagnostics of various clinical forms of the cholelithiasis (acute and chronic calculous cholecystitis, choledocholithiasis, biliodigestive fistula).
7. Indications and contraindications for conservative or operative treatment of various clinical versions of the cholelithiasis.
8. Peculiarities of the preoperative preparation.
9. Types of operative interventions at various clinical forms of the cholelithiasis.
10. Peculiarities of the postoperative period course.
11. Anatomic and physiologic features of the pancreas.
12. Modern theories of appearance of acute and chronic pancreatitis.
13. Acute pancreatitis classification.
14. Chronic pancreatitis classification.
15. Clinical and biochemical characteristics of secretory and excretory functions of the pancreas.
16. Clinics, diagnostics, differential diagnostics and curing tactics choice at various clinical forms of acute pancreatitis.
17. Clinics, diagnostics, differential diagnostics and curing tactics choice at various clinical forms of chronic pancreatitis.
18. Curing tactics at the acute pancreatitis.
19. Indications and contraindications for conservative and operative treatment of various clinical forms of acute pancreatitis.
20. Complex of conservative treatment of acute pancreatitis.
21. Types of operative interventions at various clinical forms of acute pancreatitis.
22. Complex of conservative treatment of chronic pancreatitis.
23. Types of operative interventions at various clinical forms of chronic pancreatitis.
24. Peculiarities of postoperative period course after operations at acute and chronic pancreatitis.

The student has to be able to:

1. To take the history of the patient with the suspect for the cholelithiasis.
2. To reveal main clinical features and symptoms at various clinical forms of the cholelithiasis.
3. To perform the palpation of the gallbladder.
4. To argue and formulate the preliminary diagnosis.
5. To make a plan of the examination of the patient and explain the examination results (general blood test, general urine test, duodenal intubation, roentgenologic examination of biliary ducts, ultrasonic examination, biochemical blood test).
6. To define indications and contraindications for operative intervention, to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form of the cholelithiasis.
7. To reveal main clinical features and symptoms at various clinical forms of acute pancreatitis.
8. To perform palpation of the pancreas.
9. To argue and formulate the preliminary diagnosis.
10. To make a plan of the examination of the patient and explain the examination results (general blood test, general urine test, duodenal intubation, roentgenologic examination of biliary ducts and pancreas, ultrasonic examination, biochemical blood test < KT).
11. To define curing tactics, indications and contraindications for operative intervention, to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form of acute and chronic pancreatitis.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. Which of the following statements about the diagnosis of acute calculous cholecystitis are true?
 - A. Pain is so frequent that its absence almost precludes the diagnosis.
 - B. Jaundice is present in a majority of patients.
 - C. Ultrasonography is the definitive diagnostic test.
 - D. Cholescintigraphy is the definitive diagnostic test.

2. Which statements about acute acalculous cholecystitis are correct?
 - A. The disease is often accompanied by or associated with other conditions.
 - B. The diagnosis is often difficult.
 - C. The mortality rate is higher than that for acute calculous cholecystitis.
 - D. The disease has been treated successfully by percutaneous cholecystostomy.

3. True statements about the surgical management of patients with acute calculous cholecystitis include:
 - A. Operation should be performed in all patients as soon as the diagnosis is made.
 - B. Antibiotic therapy should be initiated as soon as the diagnosis is made.
 - C. Dissection of the gallbladder is facilitated by decompression of the organ with the use of a trocar.
 - D. An operative cholangiogram should be done in every patient.

4. Which of the following are indications for cholecystectomy?
 - A. The presence of gallstones in a patient with intermittent episodes of right-side upper quadrant pain.
 - B. The presence of gallstones in an asymptomatic patient.
 - C. The presence of symptomatic gallstones in a patient with angina pectoris.
 - D. The presence of asymptomatic gallstones in a patient who has insulin-dependent diabetes.

5. Which of the following statements about laparoscopic cholecystectomy are correct?
 - A. The procedure is associated with less postoperative pain and earlier return to normal activity.
 - B. The incidence of bile duct injury is higher than for open cholecystectomy.
 - C. Laparoscopic cholecystectomy should be used in asymptomatic patients because it is safer than open cholecystectomy.
 - D. Pregnancy is a contraindication.

6. Which of the following statements about cholangitis are correct?
 - A. Charcot's triad is always present.
 - B. Associated biliary tract disease is always present.
 - C. Chills and fever are due to the presence of bacteria in the bile duct system.
 - D. The most common cause of cholangitis is choledocholithiasis.

7. The initial goal of therapy for acute toxic cholangitis is to:
 - A. Prevent cholangiovenous reflux by decompressing the duct system.
 - B. Remove the obstructing stone, if one is present.

- C. Alleviate jaundice and prevent permanent liver damage.
E. Prevent the development of gallstone pancreatitis.
8. Which of the following parameters is/are not included in the Ranson's prognostic signs useful in the early evaluation of a patient with acute pancreatitis?
A. Elevated blood glucose.
B. Leukocytosis.
C. Amylase value greater than 1000 U per dl.
D. Serum lactic dehydrogenase (LDH) greater than 350 IU per dl.
E. Alanine aminotransferase greater than 250 U per dl.
9. Standard supportive measures for patients with mild pancreatitis include the following:
A. Intravenous fluid and electrolyte therapy.
B. Withholding of analgesics to allow serial abdominal examinations.
C. Subcutaneous octreotide therapy.
D. Nasogastric decompression.
E. Prophylactic antibiotics.
10. Which of the following statements about chronic pancreatitis is/are correct?
A. Chronic pancreatitis is the inevitable result after repeated episodes of acute pancreatitis.
B. Patients with chronic pancreatitis commonly present with jaundice, pruritus, and fever.
C. Mesenteric angiography is useful in the evaluation of many patients with chronic pancreatitis.
D. Total pancreatectomy usually offers the best outcome in patients with chronic pancreatitis.
E. For patients with disabling chronic pancreatitis and a dilated pancreatic duct with associated stricture formation, a longitudinal pancreaticojejunostomy (Peustow procedure) is an appropriate surgical option.
11. Which of the following statements about pancreatic ascites is/are correct?
A. Patients typically present with painful ascites, reflecting the release of toxic pancreatic enzymes into the peritoneal cavity.
B. The standard evaluation of a patient with new-onset ascites includes abdominal paracentesis. In cases of pancreatic ascites, the peritoneal fluid contains high concentrations of both amylase and protein.
C. Pancreatic ascites can follow an episode of acute pancreatitis.
D. Patients with pancreatic ascites may fail to improve with nonoperative therapy and require surgical procedures. At abdominal exploration an acceptable approach to the pancreatic duct disruption involves suture ligation with omental patching.
12. Which of the following statements about adenocarcinoma of the pancreas is/are correct?
A. It is the fifth most common cause of cancer death in the U.S.
B. Most cases occur in the body and tail of the pancreas, making distal pancreatectomy the most commonly performed resectional therapy.
C. For cancers of the head of the pancreas resected by pancreaticoduodenectomy, prognosis appears to be independent of nodal status, margin status, or tumor diameter.
D. The most accurate screening test involves surveillance of stool for carbohydrate antigen (CA 19-9).

The answers for the self-checking tests.

1 – A,D.

2 – A,B,C,D.

3 – B,C.

4 – A.

5 – A,B.

6 – B,C,D.

7 – A.

8 – C,E.

9 – A.

10 – E.

11 – B.

12 – A.

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others “Hospital surgery”. Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%20202%20Acute%20cholecystitis.htm

Additional –

1. Manual “Facultative Surgery” under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section “Diseases of the rectum and the colon”,
2. Compact disc “Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

The author: As. prof. A. Hospodarskyy

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №12 " DISEASES OF THE RECTUM AND THE COLON "

The aim. To master clinics, diagnostics, differential diagnostics of diseases of rectum and colon: hemorrhoids, anal fissures, acute and chronic paraproctitis, Crohn's disease, nonspecific ulcerative colitis, rectal prolapse. To learn the treatment tactics, indications and contraindications for conservative and operative treatment, analgesia type, operative intervention type, principles of the pre- and postoperative periods, expertise of the working ability after operations on rectum and colon.

Professional orientation of students. Diseases of the rectum and the colon are very common for people regardless their sex, especially in mature and elderly age. Say, 10-20% of the population suffer with hemorrhoids, 0,5-6,1% of people of able to work age have rectal fistulas. Therefore, a doctor of any profile, especially a family one, must be accustomed with the peculiarities of curing-diagnostic tactics at those illnesses.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Theme №1. Hemorrhoids. Anal fissures.

1. Anatomic and physiologic data about the rectum.
2. Topography of cellular spaces of the pelvis.
3. Semiotics of the rectum diseases.
4. Hemorrhoids. Concept definition. Classification.
5. Clinical characteristics of the hemorrhoids.
6. Hemorrhoids complications (prolapse, thrombosis, strangulation, bleeding). Clinical characteristics.
7. Curing tactics at the hemorrhoids.
8. Methods of the conservative treatment of the hemorrhoids.
9. Indications and methods of the operative treatment of the hemorrhoids.
10. Peculiarities of the preoperative preparation and the postoperative period course in patients with the hemorrhoids.
11. Anal fissure. Concept definition, classification.
12. Clinical features of anal fissures depending on the clinical form.
13. Curing tactics and treatment of anal fissures depending on the clinical form.

Theme №2. Acute and chronic paraproctitis.

1. Acute paraproctitis. Concept definition, classification.
2. Clinics, diagnostics and differential diagnostics of the acute subcutaneous paraproctitis.
3. Clinics, diagnostics and differential diagnostics of the acute ischiorectal paraproctitis.
4. Clinics, diagnostics and differential diagnostics of the acute pelvirectal paraproctitis.
5. Curing tactics choice at the acute paraproctitis depending on the clinical form and the stage of the inflammatory process.
6. Chronic paraproctitis. Concept definition, classification.
7. Clinical signs, diagnostics of the chronic paraproctitis depending on the clinical form.
8. Fistulography – the main method of diagnosis verification: preparation, performance techniques, results assessment.
9. Features of the preoperative preparation and operative treatment of patients with the chronic paraproctitis.
10. Expertise of the disablement of patients after operations for acute and chronic paraproctitis.

Theme №3. Nonspecific ulcerative colitis (NUC).

1. Concept definition. Classification.
2. Clinical characteristics of the NUC depending on the clinical form and course stage.
3. Clinics, diagnostics and differential diagnosis of NUC complications.
4. Diagnosis verification methods (irrigography, colonoscopy, biopsy).
5. Curing tactics at NUC.
6. Principles of conservative and operative treatment of patients with NUC.
7. Clinics, diagnostics and differential diagnosis of various clinical forms of Crohn's disease.
8. Clinical characteristics of surgical complications of Crohn's disease.
9. Modern methods of diagnosis verification.
10. Curing tactics choice depending on the clinical form and complications of Crohn's disease.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.**Basic level of knowledge and skills:****The student must know:**

1. Anatomic and physiologic features of the rectum and the colon.
2. Topography of the rectum and the colon.
3. Classification of diseases of the rectum and the colon.
4. Hemorrhoids. Concept definition and classification.
5. Clinical characteristics, diagnostics and differential diagnostics of hemorrhoids depending on the clinical form and complications.
6. Curing tactics choice and treatment method of hemorrhoids depending on the clinical form and complications.
7. Methods of conservative and operative treatment of hemorrhoids.
8. Anal fissures. Classification.
9. Clinical characteristics of anal fissures depending on the clinical form.
10. Curing tactics and methods of conservative and operative treatment of anal fissures.
11. Acute paraproctitis. Concept definition and classification.
12. Clinics, diagnostics and differential diagnostics of the acute paraproctitis depending on the clinical form and stage of the inflammatory process.
13. Curing tactics and treatment methods of the acute paraproctitis depending on the clinical form and stage of the

inflammatory process.

14. Chronic paraproctitis. Concept definition and classification.

15. Clinical characteristics, diagnostics and differential diagnostics and diagnosis verification methods of chronic paraproctitis.

16. Curing tactics and treatment method of chronic paraproctitis depending on the clinical form.

17. Crohn's disease. Classification.

18. Clinical characteristics of Crohn's disease depending on the clinical form and course stage, complications.

19. Curing tactics and methods of conservative and operative treatment of Crohn's disease depending on the clinical form, course stage and complications.

20. Nonspecific ulcerative colitis (NUC). Concept definition and classification.

21. NUC clinics depending on the allocation, clinical course, complications.

22. Verification methods of NUC diagnosis.

23. Curing tactics and principles of conservative and operative treatment of NUC depending on the clinical form, course and complications.

24. Rectal prolapse. Concept definition and classification.

25. Clinics of the rectal prolapse depending on the course stage.

26. Curing tactics and treatment methods of the rectal prolapse depending on the course stage.

The student has to be able to:

1. To take the history of the patient with the rectum and the colon diseases.

2. To elicit major clinical signs and symptoms of diseases of the rectum and the colon.

3. To perform rectal examination of the rectum.

4. To make a plan of patient's examination and to explain the examination results (general blood test, general urine test, roentgenologic examinations (irrigoscopy, irrigography, fistulography), ultrasonic examination, biochemical blood test, anoscopy, colonoscopy, biopsy, rectoromanoscopy).

5. To define curing tactics, indications and contraindications for an operative intervention to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form of the disease, its course, inflammatory process stage, complications.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. Which of the following statements about hemorrhoids is/are not true?

A. Hemorrhoids are specialized "cushions" present in everyone that aid continence.

B. External hemorrhoids are covered by skin whereas internal hemorrhoids are covered by mucosa.

C. Pain is often associated with uncomplicated hemorrhoids.

D. Hemorrhoidectomy is reserved for third- and fourth-degree hemorrhoids.

2. Which answers are true? In contrast to ulcerative colitis, Crohn's disease of the colon:

A. Is not associated with increased risk of colon cancer.

B. Seldom presents with daily hematochezia.

C. Is usually segmental rather than continuous.

D. Has a lower incidence of perianal fistulas.

E. Never develops toxic megacolon.

3. Which of the following statements about the etiology of chronic ulcerative colitis are true?
- A. Ulcerative colitis is 50% less frequent in nonwhite than in white populations.
 - B. Psychosomatic factors play a major causative role in the development of ulcerative colitis.
 - C. Cytokines are integrally involved in the pathogenesis of ulcerative colitis.
 - D. Ulcerative colitis has been identified with a greater frequency in family members of patients with confirmed inflammatory bowel disease.
 - E. Ulcerative colitis is two to four times more common in Jewish than in non-Jewish populations.
4. Surgical alternatives for the treatment of ulcerative colitis include all of the following except:
- A. Colectomy with ileal pouch–anal anastomosis.
 - B. Left colectomy with colorectal anastomosis.
 - C. Proctocolectomy with Brooke ileostomy or continent ileostomy.
 - D. Subtotal colectomy with ileostomy and Hartmann closure of the rectum.
5. The initial management of toxic ulcerative colitis should include:
- A. Broad-spectrum antibiotics.
 - B. 6-Mercaptopurine.
 - C. Intravenous fluid and electrolyte resuscitation.
 - D. Opioid antidiarrheals.
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6. Which finding(s) suggest(s) the diagnosis of chronic ulcerative colitis as opposed to Crohn's colitis?
- A. Endoscopic evidence of backwash ileitis.
 - B. Granulomas on biopsy.
 - C. Anal fistula.
 - D. Rectal sparing.
 - E. Cobblestone appearance on barium enema.
7. Axial twisting of the right colon or cecal volvulus has been shown to be associated with each of the following except:
- A. A history of abdominal operation.
 - B. A mobile cecum.
 - C. An obstructing lesion in the transverse or left colon.
 - D. Inflammatory bowel disease.

The answers for the self-checking tests.

1 – C.

2 – B,C.

3 – A,C,D,E.

4 – B.

5 – A,C.

6 – A.

7 – D.

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others “Hospital surgery”. Ternopil: Ukrmendknyga, 1999.
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"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №12 " DISEASES OF THE RECTUM AND THE COLON "

The aim. To master clinics, diagnostics, differential diagnostics of diseases of rectum and colon: hemorrhoids, anal fissures, acute and chronic paraproctitis, Crohn's disease, nonspecific ulcerative colitis, rectal prolapse. To learn the treatment tactics, indications and contraindications for conservative and operative treatment, analgesia type, operative intervention type, principles of the pre- and postoperative periods, expertise of the working ability after operations on rectum and colon.

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TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

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(the instructor has tests & situational tasks)

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B. External hemorrhoids are covered by skin whereas internal hemorrhoids are covered by mucosa.

C. Pain is often associated with uncomplicated hemorrhoids.

D. Hemorrhoidectomy is reserved for third- and fourth-degree hemorrhoids.

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 - D. Subtotal colectomy with ileostomy and Hartmann closure of the rectum.
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 - B. 6-Mercaptopurine.
 - C. Intravenous fluid and electrolyte resuscitation.
 - D. Opioid antidiarrheals.
 - E. Colonoscopic decompression.
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 - C. Anal fistula.
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- A. A history of abdominal operation.
 - B. A mobile cecum.
 - C. An obstructing lesion in the transverse or left colon.
 - D. Inflammatory bowel disease.

The answers for the self-checking tests.

1 – C.

2 – B,C.

3 – A,C,D,E.

4 – B.

5 – A,C.

6 – A.

7 – D.

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others “Hospital surgery”. Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%2011%20HEMORRHOIDS.%20ANAL%20FISSURES.%20PARAPROCTITIS.htm

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The author: As. prof. A. Hospodarskyy

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №12 "DISEASES OF THE THYROID AND MAMMARY GLAND"

The aim. To master clinics, diagnostics, differential diagnostics of various clinical forms of goiter, thyroiditis. To learn the treatment tactics, indications and contraindications for operative intervention, operative intervention type at various clinical forms of goiter, principles of postoperative period course, expertise of the working ability after operations for endemic, sporadic, diffuse toxic goiters, thyroidites.

Professional orientation of students. Thyroid gland diseases (endemic, sporadic, diffuse, toxic goiters, thyroidites) occupy the leading place among surgical illnesses of endocrine glands. The actuality of the problem is connected with the endemic character of many geographical regions of Ukraine and also with the accident at Chernobyl Power Atomic Station.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Theme №1. Endemic, sporadic goiter.

1. Anatomic and physiologic data about the thyroid gland.
2. Topography of the neck.
3. Definition of concepts of endemic area, stages of endemic gravity, endemic and sporadic goiter.
4. Concept definition and classification of endemic, sporadic goiter.
5. Classification of the thyroid gland enlargement stages.
6. Characteristics of various clinical versions of the endemic and sporadic goiter.
7. Peculiarities of the clinical picture of endemic goiter depending on the availability of violations of thyroid gland function, on compressive signs, morphologic characteristics.
8. Differential diagnostics of the endemic and sporadic goiter.
9. Verification methods of endemic goiter diagnosis.
10. Curing tactics at the thyroid gland diseases.
11. Principles of treatment method choice of patients with endemic goiter, indications for operative treatment.
12. Methods of operative treatment of patients with the endemic goiter.
13. Postoperative period course in patients with endemic, sporadic goiter.

Theme №2. Diffuse toxic goiter (DTG).

1. Concept definition, etiopathogenesis of DTG.
2. Classification of DTG.
3. Clinics, diagnostics, differential diagnostics of DTG.
4. Verification methods of DTG diagnosis.
5. Treatment tactics and curing method choice at DTG, indications for operative treatment.
6. Peculiarities of preoperative preparation of patients with DTG depending on the gravity stage of the thyroiditis.
7. Principles of operative treatment of patients with DTG.
8. Peculiarities of postoperative period course in patients with DTG.
9. Early and late complications after operations for DTG (reasons, clinics, prophylaxis, treatment).
10. Thyrotoxic crisis –reasons, classification, clinics, prophylaxis and treatment.
11. Disablement expertise after the subtotal resection of the thyroid gland.

Theme №3. Strumites, thyroidites.

1. Concept definition. Classification.
2. Clinics, diagnostics, differential diagnostics of the acute purulent thyroiditis (strumitis).
3. Clinics, diagnostics, differential diagnostics of the chronic Riedel's thyroiditis.
4. Clinics, diagnostics, differential diagnostics of chronic Hashimoto's and De Kerven's thyroidites.
5. Curing tactics at acute and chronic thyroidites, strumites.
6. Principles of conservative and operative treatment of patients with acute and chronic thyroidites.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Anatomic and physiologic features of the thyroid gland.
2. Topography of the neck (thyroid gland, cellular spaces).
3. Definition of concepts "goiter", "strumitis", "thyroiditis",.

4. Classification of the thyroid gland diseases.
5. Etiopathogenesis, clinics, diagnostics, differential diagnostics of the diffuse toxic goiter.
6. Etiopathogenesis, clinics, diagnostics, differential diagnostics of the endemic goiter.
7. Diagnostics and tactics at the acute thyroiditis.
8. Diagnostics and tactics at the chronic thyroiditis (Riedel's, Hashimoto's, de Kerven's).
9. Treatment tactics at the thyroid gland diseases.
10. Peculiarities of the preoperative preparation of patients with the thyroid gland diseases.
11. Principles of operative treatment of patients with various clinical forms of the goiter.
12. Peculiarities of the postoperative period course in patients after the resection of the thyroid gland, thyroidectomy.
13. Expertise principles of the disablement of patients after operations on the thyroid gland.

The student has to be able to:

1. To take the history of the patient with the suspect for the thyroid gland diseases.
2. To palpate the thyroid gland.
3. To elicit major clinical signs and symptoms at various clinical forms of the endemic goiter.
4. To elicit major clinical signs and symptoms at various clinical forms of the diffuse goiter.
5. To elicit major clinical signs of the acute and chronic thyroiditis.
6. To argue and explain the preliminary diagnosis.
7. To make a plan of patient's examination and to explain the examination results (general blood test, general urine test, roentgenologic examinations of the neck (general view roentgenogram), ultrasonic examination, biochemical blood test (hepatic probes, electrolytes, thyroxin, TTG).
8. To define indications and contraindications for an operative intervention to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form of the goiter and the gravity stage of the thyrotoxicosis.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions. (the instructor has tests & situational tasks)

1. When progressive enlargement of a multinodular goiter causes symptomatic tracheal compression, the preferred management in otherwise good-risk patients is:
 - A. Iodine treatment.
 - B. Thyroid hormone treatment.
 - C. Surgical resection of the abnormal thyroid.
 - D. Radioactive iodine treatment.
2. The most precise diagnostic screening procedure for differentiating benign thyroid nodules from malignant ones is:
 - A. Thyroid ultrasonography.
 - B. Thyroid scintiscan.
 - C. Fine-needle-aspiration biopsy (FNAB).
 - D. Thyroid hormone suppression.
3. Therapy for Hashimoto's disease includes:
 - A. Radioiodine.
 - B. Antithyroid medications.
 - C. Subtotal thyroidectomy.
 - D. None of the above.
4. Which of the following statements is true about the synthesis of thyroid hormone and its physiology?
 - A. The iodine utilized in hormone synthesis is derived principally from dietary sources.
 - B. The role of thyroid-stimulating hormone (TSH) in thyroid physiology is limited to regulation of the release of thyroid hormone in plasma.
 - C. Enough thyroxine (T₄) is stored in the normal thyroid to provide a euthyroid state for 3 weeks despite absence of iodine intake.
 - D. The regulation of thyroid function involves pituitary, but not hypothalamic, input.
5. Correct statements about thyroid function tests include which of the following?
 - A. Contraceptive pills and pregnancy increase the amount of thyroxin-binding globulin (TBG), and, consequently, the total T₄ level.
 - B. Anticonvulsive medications and chronic debilitating illnesses decrease the amount of TBG and, consequently, the total T₄ level.
 - C. Intravenous pyleography can lower the rate of active iodine uptake by the thyroid.
 - D. A triiodothyronine (T₃) suppression test that demonstrates nonsuppressibility of thyroid function is compatible with the diagnosis of Graves' disease, toxic adenoma, or functioning carcinoma.
 - E. An increased serum cholesterol level in a hypothyroid patient indicates a thyroid cause.
6. Hyperthyroidism can be caused by all of the following except:
 - A. Graves' disease.
 - B. Plummer's disease.
 - C. Struma ovarii.
 - D. Hashimoto's disease.
 - E. Medullary carcinoma of the thyroid.
7. Arrange the following complications of thyroid surgery (bilateral subtotal thyroidectomy) in decreasing order of incidence in patients with Graves' disease.
 - A. Laryngeal nerve paralysis.
 - B. Hypoparathyroidism.
 - A. Hypothyroidism.
 - B. Recurrent hyperthyroidism.

The answers for the self-checking tests.

- 1 – C.
- 2 – C.
- 3 – D.
- 4 – A,C.
- 5 – A,B,C,D.
- 6 – E.
- 7 – A,B,C,D.

Information sources:**Main –**

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others "Hospital surgery". Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/iik/ptn/Surgery/4/Topic%2012%20Endemic,%20sporadic%20goiters.%20Diffuse%20toxic%20goiter.%20Thyrotoxicosis.%20Dishormonus%20diseases%20of%20the%20mammary%20gland.htm

Additional –

1. Manual "Facultative Surgery" under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section "Diseases of the rectum and the colon",
2. Compact disc "Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

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Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

EMPLOYMENT № 13 (Practical - 7 hours.)
NEW TECHNOLOGIES IN SURGERY. MODERN METHODS OF DIAGNOSTIC AND TREATMENT

- Themes: 1. Endoscopic surgery, prospects and possibilities of modern endoscopic technologies.
2. X-Ray surgery. Surgery under the control sonography.
3. Telesurgery.

Purpose: to learn the modern approaches to operative medical treatment with the use of endoscopic technologies. To know prospects, possibilities and to be able to ground testimonies to implementation of endoscopic operative interferences. Learn modern approaches to operative medical treatment with the use of sonography and X-Ray control. To know prospects, possibilities and to be able to ground a testimony to implementation of operative interferences under the control sonography and X-Ray control. To know diagnostic potential of modern medical telecommunication equipment. To familiarize with the basic systems vehicles, which is used in remote diagnostics of surgical pathology. Learn the principles of action, testimonies and contra-indications to application and diagnostic value of modern telemedical equipment.

Professional orientation of students: subsequent development of surgery largely will be determined by endoscopic technologies. Laparoscopic cholecystectomy is today named a «gold standard». Progress in endoscopic surgery is connected with the improvement of apparatus, optical systems and instruments, provide which implementation of these operations. Development of hardware necessary for endoscopic operative interferences takes place the parallel with the improvement of methods. Pre- and intraoperative diagnostics has the important value at the decision of testimonies, choice of methods and volume of endoscopic operative interferences.

X-Ray surgery is a new perspective direction of clinical medicine. Main point of it consists in conducting of diagnostic inspections and medical manipulations under the X-Ray control. Successfully made X-Ray endovascular interferences can only experienced specialists, who well know X-Ray anatomy of the vessels, is able thinly to conduct the diagnostic and medical stages of procedure, acquainted with possible complications and ways of their removal. Only high-quality preparation is conducted, clear determination of testimonies and contra-indications for every patient can justify the potential risk, connected with the use of this method. Modern possibilities of BONDS allow expressly to localize a pathological process and conduct medical manipulation, not damaging here surrounding fabrics.

Telemedicine provides for use telecommunication technologies for the grant of medicare. Last years there is a tendency to appearance of specialists in narrow industries of medicine. It led to certain centralization of medicare and often necessary specialist it is now possible to find gust in big town. Due to telemedicine a necessity in the physical presence of certain specialists diminishes considerably, and a patient can be consulted in the distance. Thus become possible access of more skilled consultation, that especially it is important at the using new dear diagnostic procedures (Computer tomography, densitometry, Dopplersonography and etc). Carefully using neat new technologies allows to accelerate the reduce the price of consultation from one or a few experts, in spite of physical distance from him, as by way of Internet it is possible to unite with a specialist even, who is on other continent.

Method of implementation of practical work.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.

2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

I. Theme №1 practical employments

Work 1. After the introductory word of teacher and control of basic level of knowledge and abilities of students (verbal questioning and writing control) it is necessary to made the following group settings:

- A) determinations and grounds of indications and contra-indications of endoscopic operative interferences;
- B) a student collects complaints, anamnesis of disease and lives, conducts the objective inspection, reveal, discover the basic clinical signs of disease of patient, estimates clinical-laboratory information of stationary card of patient, determines and grounds a indications and contra-indications for endoscopic operative interferences which are possible.

After it making individual settings, directed on setting of medical treatment to the patients after endoscopic operative interferences.

II. Theme №2 practical employments

Work 2. Making the following group of settings:

- A) determinations and grounds of testimonies and contra-indications for X-Ray endovascular manipulations;
- B) near the patient a facts student sick on the basis of clinical-laboratory determines and grounds a indication and contra-indication for operative interferences under the control sonography and X-Ray control. The inspection is conducted X-Ray controle, the sonography, a student interprets findings, determines their diagnostic value.

After it making individual settings, directed on setting of medical treatment to the patients after X-Ray endovascular operative interferences.

III Theme №3 practical employments

Work 3. Making following group settings:

- A) determination and ground of indication for the conducting of remote consultations: by phone, to the e-mail, video conference communication;
- B) a student collects complaints, anamnesis of disease and lives, conducts the objective inspection, reveal, discover the basic clinical signs of disease of patient, estimates clinical-laboratory information of stationary card of patient, are formulated by the request for remote consultation.

After it making individual settings: a student sets a diagnosis on the basis of digital facts about a patient, grounds and formulates the request for the additional methods of inspection and determines medical tactic.

After the sum of implementation of all settings estimation is proposed to every student.

Program of selftraining of students

Questions on wich a student must give answers:

I. Theme № 1 practical employments

1. Types of endoscopic surgery.
2. Advantages and lacks of endoscopic operations.
3. Endoscopic equipment and tool.
4. Technique of execution of laparoscopy.
5. Method of laparoscopic revision and its informing.

6. Laparoscopic anatomy of hepatopancreaticoduodenal area.
7. Intraoperative laparoscopic specific complications.
8. Specific and unspecific complications of early and late postoperative periods.
9. Indication and contra-indication for laparoscopic operations on bilious ways.
10. Method of laparoscopic cholecystectomy.
11. Invasive and noninvasive methods of laparoscopic intraoperative inspections of bilious ways.
12. Laparoscopic medical treatment of the complicated forms of cholecystitis (one- and two-stapes methods).
13. Laparoscopic methods of hepaticocholechocheal drenaging.
14. endoscopy, bronkhoscopy, kolonoscopy. Method of conducting, possibilities.

II. Theme № 2 practical employments

1. Types of X-Ray surgery and directions for using.
2. X-Ray endovascular surgery. Prospects. General principles, equipment.
3. X-Ray endovascular dilatation. General principles, mechanism dilatation.
4. X-Ray endovascular dilatation of coronal arteries. Testimony, method of conducting.
5. X-Ray endovascular dilatation of kidney arteries. Testimony, method of conducting.
6. X-Ray endovascular occlusion. General principles, mechanism of occlusion.
7. X-Ray endovascular occlusion at the pulmonary bleeding.
8. X-Ray endovascular occlusion at pathology of organs of abdominal region.
9. X-Ray endovascular occlusion at the pathological function of bud.
10. X-Ray endovascular prosthesis.
11. Raising of driver of rhythm under the control x-ray photography. Indication and method of implementation.
12. Endoscopic retrograde pancreatocholangiography. Indication and method of implementation.
13. Puncture under the control sonography. Indication and method of implementation.
14. Types of lithotripsy under the control sonography and X-Ray control. Indication and method of implementation.

III. Theme № 3 practical employments

1. Diagnostic, consultation and medical possibilities of telemedycine.
2. Types of telesurgery. Directions of application.
3. Telesurgery as newest link of medicine. Apparatus and rigging.
4. Telesurgical consultations as an effective method of the diagnostics urgent condition.
5. X-Ray endovascular in the distance as a stage of grant of the first aid is important.
6. Sonography in the distance as a stage of grant of the first aid is important.
7. Features of conducting of telesurgical operative interferences.
8. Telesurgical assisting at operative interferences.
9. Using of teleconsultations at operative interferences for the prophylaxis of intraoperative and postoperative complications, choice of tactic of medical treatment.
10. Using of digital records of hospital chart at the grant of stage medicare.
11. Features of formulation of query for remote teleconsultation.
12. Robotosurgery. Possibilities of application. Advantages and failings.

Break 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Seminar discussion of theoretical questions

The basic level of knowledges and abilities is checked up by the undoing of situation tasks to every theme, by answers for tests and structural questions.

(presence of complete sets of tests and situation tasks at a teacher)

A student must know:

1. Troubleshooting routines at preparation of patients to endoscopic interferences.
2. Sequence of physical and clinical inspections of patients.
3. Indications and contraindications for endoscopic interferences.
4. Variants of norm of endoscopic anatomy.
5. What apparatus and tool is used for the different types of laparoscopic operations and manipulations.
6. What equipment is used for the different types of telesurgery.
7. What equipment is used for X-Ray surgery.
8. Variants of the norm X-Ray anatomy.

A student must be able:

1. To prepare a patient to the inspections: endoscopy, colonoscopy, sonography, sciagraphies, tomography, angiography, cystoscopy, gysteroscopy, thoracoscopy, bronkhoscopy.
2. On the basis of clinico-laboratory findings to define and ground a indications and contraindications for endoscopic operative interferences.
3. To estimate sonography, sciagrams, tomogram, information endoscopy, bronkhoscopy and kolonoscopy.
4. To be able work with medical databases and electronic hospital charts.
5. To be able to formulate the request for remote teleconsultation.
6. To give remote teleconsultation from help of different communication facilities (e-mail, telephone message, vydeoteleconsultations).

Test evaluation and situational tasks 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. Patient To., 46 years, first days after laparoscopic operation concerning chronic calculous cholecystitis grumbles about a general weakness, nausea, multiple vomiting by a bile, pain and swelling of stomach. To the peristalsis is hearkened, gases do not depart. On a X-Ray film is free gas in an abdominal region.

Clinical diagnosis?

Medical tactic?

2. At a patient, 40 years, about of 5 month after carried laparoscopic cholecystectomy there recover the cramping hepatic colic, which was accompanied icteric of the skin covers, excrement of color of argil. What method of diagnostics will be most informing?

Answers:

- A. Peroral cholangiography
- B. Retrograde pankreatocholangiography
- C. Ultrasonography hepatobiliary area

Д. Radionuclide scanning

Е. Laparoscopy

3. A patient 78 years is hospitalized in the planned order with complaints about jaundice making progress, which appeared over 3 weeks to that ago, without a pain syndrome, general weakness, bad appetite. Objectively: temperature of body 36,8°C, pulse of 78/мин, a stomach is soft painless, symptoms of irritation of the peritoneum not exposed. From data of sonography tumular education in the area of gate of liver by sizes 4*6 см, intrahepatic bilious duct is extended. On the X-Ray sciagram of organs of pectoral cavity extended plural metastases in both 0,5 - 1 см easy by sizes. What operative medical treatment will be most optimum?

Answers:

A. Cholecystoenterostomy

B. Hepatoenteroanastomosis on a excluded loop for Ru

C. cholecystectomy, external drenagine of choledoch

Д. Choledochoduodenostomy

Е. Throughskin throughhepatic drenagine of intrahepatic bilious ducts under the control sonography

4. Patient of 53 years, entered a clinic concerning the gastro-intestinal bleeding of unclear etiology. During laparotomy there is a reveal big vascular tumour, which coverage on a duodenum, the Treyts copula and head of the pancreas. Deleting a tumour is not possible. During postoperative period it is conducted to the angiography gastroduodenal artery. On angiography was determined characteristic picture of vascular tumour.

What next medical tactic in relation to this patient?

A. Embolization systems of gastroduodenal artery.

B. Conservative hemostatic therapy.

C. Chemotherapy.

Д. Radial therapy.

Е. Embolization of abdominal trunk branches of aorta.

5. During telemedical vydeoteleconsultations you advise a domestic doctor, who is appealed to the patient in 62, during 10 years is ill gallstone illness. Three days to that ago he fell dull pain in right hypochondriac region, almost at once at a patient the turn of sclera and skin covers appeared yellow, at the same time of patient marked the complete lighting up of color of excrement the masses almost. Your recommendations?

А. Urgent hospitalization in surgical permanent establishment

B. Setting of diet № 5 for Pevznerom

C. Supervision after a patient in the conditions of polyclinic

Д. Conservative therapy in ambulatory terms

Е. Conducting of diagnostic measures for clarification of diagnosis

6. To your electronic address came a latter from a patient 50 years, in which she grumbles about pain in right hypochondriac region, nausea, arose up after the reception of rich food. After 6 hours of spasmolytics using she fell better. Similar to pain colic it was to 3 mounts that before. A patient did sonography - in a gall-bladder find out the concretion in diameter of 30 mm. What can be advised this patient?

A. To apply medicinal dissolution of stone

B. To offer conservative medical treatment

C. Ambulatory supervision following to new pain colic

Д. To apply extracorporal lithotripsy

Е. To offer cholecystectomy

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others "Hospital surgery". Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%2013.%20New%20technologies%20in%20surgery.%20Modern%20methods%20of%20diagnostic%20and%20treatment.htm

Additional –

1. Manual "Facultative Surgery" under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section "Diseases of the rectum and the colon",
2. Compact disc "Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

The author: As. prof. A. Hospodarskyy

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTION FOR 5th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION FOR LESSON №1 "PURULENT DISEASES OF LUNG AND PLEURA"

The aim: To be able to diagnose the acute and chronic abscess of lung depending on their localization and complications, to give urgent help to the patients with the acute abscess of lung complicated by the pulmonary bleeding, to diagnose and liquidate complications, indications for conservative and operative treatment, to know the methods of their operative treatment.

Professional orientation of students:

Over the past 40 years, the frequency of lung abscesses has declined in 10 times, whereas the mortality rate among patients decreased only on 5-10 % and consists of 4-7 %. In aspiration of fluid, which contains microflora, the mortality can reach 20 % and more, especially if the reaction of the liquid is acid. Most deaths in lung abscess associated with *Pseudomonas aeruginosa*, *Staphylococcus aureus* and *Klebsiella pneumoniae*. Late and inappropriate antibiotic therapy, inadequate drainage of the abscess, inadequate use of bracing treatment leads to the formation of chronic abscess and consequently its surgical treatment, with mortality rate more than 5 %.

Complications of suppurative processes of the lungs and pleura are difficult to treat and have a serious prognosis.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

To greet and name itself.

Friendly behaviour.

To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.

Correct and quiet conversation with a patient.

To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Work 1. A student collects complaints, anamnesis of disease and life of patient, performs the objective examination, exposes the basic clinical signs of acute abscess of lung, makes a troubleshooting routine, formulates a diagnosis. On the basis of complaints, anamnesis of disease and life, information of objective examination, laboratory and roentgenologic researches performs the differential diagnosis of patient with different variants of intrapleural complications.

A student must give a question on which to the answer.

Work 2. Students independently inspect sick purulent diseases of lung, meet with a medical document, confirm diagnosis and methods of treatment. After it the survey sciagrams of organs of thorax are analysed, differential diagnostics is performed with urgent thoracic pathology.

Work 3. Students independently inspect patients with different types of purulent diseases of lung, meet with the methods of diagnostics, by a medical document, confirm diagnosis and methods of treatment, the survey sciagrams of organs of thorax are analysed, CT, information of clinical methods of examination, differential diagnostics is performed with urgent thoracic pathology.

Individual Students Program.

1. Reasons of origin of acute abscess of lung.
2. Clinical forms of gangrene of lung.
3. Clinical symptoms of acute abscess of lung in a stage “before opening”.
4. Clinical symptoms of acute abscess of lung in a stage “after opening”.
5. Clinic of chronic abscess of lung.
6. Additional methods of examination of patients with the purulent diseases of lung.
7. Differential diagnostics of chronic abscess of lung and cancer.
8. Differential diagnostics of air cyst and chronic abscess of lung.
9. 11. Treatment of gangrenous abscess of lung.
10. Treatment of acute abscess of lung. Indication to operative treatment.
11. Tactic of treatment of the complicated acute abscess of lung.

Work 4. Students independently examine patients with the purulent diseases of pleura, meet with the methods of diagnostics, by a medical document, confirm diagnosis and methods of treatment, information of the survey sciagrams is analysed, clinical methods of examination, differential diagnostics is performed with urgent thoracic pathology.

Individual Students Program.

1. Acute empyema of pleura: Etiology, pathogeny, clinic.
2. Clinical signs of the limited empyema pleura: roentgenologic picture.
3. Clinic of limited piopneumothorax: roentgenologic signs.
4. Reasons of origin and clinic of total piopneumothorax.
5. Additional methods of examination at the chronic purulent processes of pleura.
6. Differential diagnostics of empyema pleura and piopneumothorax.
7. Clinic of chronic empyema.
8. Additional methods of examination at piopneumothorax.
9. Tactic of treatment of patients with piopneumothorax.
10. Indication to puncture of pleura cavity.
11. Technique of execution of pleura puncture.
12. Indication to drainage of pleura cavity.
13. Technique of execution of drainage of pleura cavity.
14. Features of conservative treatment of piopneumothorax.

Break 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

1. Reasons of origin of acute abscess of lung.
2. Clinical forms of gangrene of lung.
3. Clinical symptoms of acute abscess of lung in a stage “before opening”.
4. Clinical symptoms of acute abscess of lung in a stage “after opening”.
5. Clinic of chronic abscess of lung.
6. Additional methods of examination of patients with the purulent diseases of lung.
7. Differential diagnostics of chronic abscess of lung and cancer .
8. Differential diagnostics of air cyst and chronic abscess of lung.
9. Methods of sanation of bronchial tree.
10. Treatment of gangrenous abscess of lung.

11. Treatment of acute abscess of lung. Indication to operative treatment.
12. Tactic of treatment of the complicated acute abscess of lung.
13. Acute empyema of pleura: Etiology, pathogeny, clinic.
14. Clinical displays of the limited pleura empyema: roentgenologic picture.
15. Clinic of limited pyopneumothorax: roentgenologic signs.
16. Reasons of origin and clinic of total pyopneumothorax.
17. Additional methods of examination at the chronic purulent processes of pleura.
18. Differential diagnostics of empyema pleura and pyopneumothorax.
19. Clinic of chronic empyema.
20. Additional methods of examination at pyopneumothorax.
21. Tactic of treatment of patients with pyopneumothorax.
22. Indication to puncture of pleura cavity.
23. Technique of execution of pleura puncture .
24. Indication to drainage of pleura cavity.
25. Technique of execution of drainage of pleura cavity.
26. Features of conservative treatment of pyopneumothorax

Test evaluation and situational tasks 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.
(the instructor has tests & situational tasks)

The tests for self-checking of knowledge, skills.

1. What is the predominant factor which causes the lung abscess?
 - A. Increased cholesterol, dyslipoproteinemia
 - B. Pulmonary hypertension
 - C. Rheumatism, endocarditis
 - D. Myocardial infarction
 - E. Disturbances of bronchial permeability with the development of atelectasis

2. What is the cause of pyopneumothorax?
 - A. Bronchiectatic disease
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema

3. For the clinical manifestation of pyopneumothorax is typical:
 - A. Chest pain
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins

4. What acute complication is characteristic for lung abscess?

- A. Emphysema
 - B. Pulmonary bleeding
 - C. Rib fracture
 - D. Malignancy
 - E. Esophageal bleeding
5. What acute complication is characteristic for lung abscess?
- A. Rib fracture
 - B. Emphysema
 - C. Pyopneumothorax
 - D. Malignancy
 - E. Esophageal bleeding
6. What complication is characteristic for lung abscess?
- A. Esophageal bleeding
 - B. Rib fracture
 - C. Emphysema
 - D. Sepsis
 - E. Malignancy
7. What complication is characteristic for lung abscess?
- A. Esophageal bleeding
 - B. Rib fracture
 - C. Bronchogenic dissemination
 - D. Emphysema
 - E. Malignancy
8. In the patient of 35 years old during physical exertion have appeared a sharp pain in the left half of the chest. On examination: the patient is covered with cold sweat, difficult breathing on the left side. A tachycardia. By percussion: the bandbox sound from the left side. By auscultation: respiration on the right side is vesicular, on the left side is absent. The probable diagnosis?
- A. Spontaneous pneumothorax
 - B. Angina on exertion
 - C. Acute myocardial infarction
 - D. Left pleurisy
 - E. Pneumonia
9. The patient A., 37 years old, has entered with the complaints of cough with daily excretion of a purulent sputum to 150 ml, pain in the right half of the chest, increase of temperature to 38°C. Has been ill for 2 weeks. Before the day of entrance in clinic appeared expectoration of 300 ml of purulent sputum during cough. On examination: a shortening of percussion pulmonary sound and harsh breathing with coarse rales over the right scapula. What is the previous diagnosis?

- A. Acute bronchitis
- B. Acute abscess of lung
- C. Exacerbation of a chronic abscess
- D. Exacerbation of bronchiectatic disease
- E. Pleural empyema

10. The patient A., 42 years old entered the clinic. During two months was treated for acute abscess of the upper lobe of the right lung. The treatment included intramuscular injections of antibiotics. Improvement inappreciable as there stayed a cough with expectoration of a purulent fetid-odor sputum to 80-100 ml per day, high temperature. The diagnosis?

- A. Acute abscess of the right lung
- B. Tubercular cavern
- C. The cavity form of a lung cancer
- D. Chronic abscess of lung
- E. Purulent polycystosis

A student must know:

1. Reasons of origin of acute abscess of lung.
2. Clinical forms of gangrene of lung.
3. Clinical symptoms of acute abscess of lung in a stage “before opening”.
4. Clinical symptoms of acute abscess of lung in a stage “after opening”.
5. Clinic of chronic abscess of lung.
6. Additional methods of examination of patients with the purulent diseases of lung.
7. Differential diagnostics of chronic abscess of lung and cancer .
8. Differential diagnostics of air cyst and chronic abscess of lung.
9. Methods of sanation of bronchial tree.
10. Treatment of gangrenous abscess of lung.
11. Treatment of acute abscess of lung. Indication to operative treatment.
12. Tactic of treatment of the complicated acute abscess of lung.
13. Acute empyema of pleura: Etiology, pathogeny, clinic.
14. Clinical displays of the limited pleura empyema: roentgenologic picture.
15. Clinic of limited pyopneumothorax: roentgenologic signs.
16. Reasons of origin and clinic of total pyopneumothorax.
17. Additional methods of examination at the chronic purulent processes of pleura.
18. Differential diagnostics of empyema pleura and pyopneumothorax.
19. Clinic of chronic empyema.
20. Additional methods of examination at pyopneumothorax.
21. Tactic of treatment of patients with pyopneumothorax.
22. Indication to puncture of pleura cavity.
23. Technique of execution of pleura puncture .
24. Indication to drainage of pleura cavity.
25. Technique of execution of drainage of pleura cavity.
26. Features of conservative treatment of pyopneumothorax.

A student must be able:

1. To expose the basic clinical signs of the uncomplicated and complicated trauma of thorax, ground and formulate a clinical diagnosis, to perform a differential diagnosis, to ground surgical tactic of treatment.

2. To expose the basic variants of clinical display of acute and chronic abscess of lung, ground and formulate a diagnosis, to perform a differential diagnosis, to ground conservative and operative treatment.
3. To expose the basic clinical signs of acute empyema of pleura and pyopneumothorax, to formulate a clinical diagnosis, to perform a differential diagnosis, to ground the shows and methods of operative treatment.
4. To expose the basic clinical aspects of purulent processes of lung.
5. To ground and formulate a clinical diagnosis.
6. To perform a differential diagnosis.
7. To formulate the shows to surgical and conservative treatment.

The answers for the self-checking tests.

- 1 – E.
- 2 – A.
- 3 – A.
- 4 – B.
- 5 – C.
- 6 – D.
- 7 – C.
- 8 – A.
- 9 – B.
- 10 – D.

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Main

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The authors: Ass. Prof. A. Vayda

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

„Medical cure of patient with pancreatitis

ALGORITHM „ Examination of patient with pancreatitis”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination.To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, the error in the alcohol intake, development of the pain irradiation)
7. To examine the patient (palpation of the pancreas, determination of particular symptoms)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of the diagnosis

„Medical cure of patient with chronic cholecystitis, gallstone disease”

ALGORITHM „ Examination of patient with chronic cholecystitis”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination.To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, the error in the alcohol intake, development of the pain irradiation, presence in anamnesis of the similar symptoms)
7. To examine the patient (palpation, percussion of the abdomen , determination of particular symptoms)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„Medical cure of patient with the incarcerated hernia”

ALGORITHM „ Examination of patient with incarcerated hernia”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.

4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, period from the moment of strangulation, presence of clinic of intestinal obstruction)
7. To examine the patient (palpation of patient in horizontal and vertical positions, palpation of inguinal channel, scrotum, the sign of coughing push)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„Medical cure of patient with acute appendicitis”

ALGORITHM „ Examination of patient with acute appendicitis”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, irradiation of pain, presence of the dysuric phenomena)
7. To examine the patient (palpation, percussion of abdomen, determination of particular symptoms)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„Medical cure of patient with the acute urine delay”

ALGORITHM „ Examination of patient with the acute urine delay”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the period of the onset of complaints; frequency of urination, especially in a night time, presence of haematouria in anamnesis)
7. To examine the patient (examination, palpation and percussion of suprapubic area, determination of Pasternack's sign, digital rectal examination)

8. To detect the cause of acute urine delay

„To perform the catheterization of urinary bladder by a soft catheter”

ALGORITHM „To perform the catheterization of urinary bladder by a soft catheter”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. The position of the patient – horizontal, thereafter to prepare the instruments, necessary for performance of this manipulation
6. To wash up the external pudenda foramen of urethra by antiseptic solution
7. To perform catheterization of urinary bladder by a soft catheter with according to the rules of the introduction of the last.

"Medical cure of the patient with atherosclerosis of the lower extremities"

ALGORITHM "Examination of the patient with atherosclerosis of lower extremities"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention on the character, period of onset of complaints, presence of the intermittent claudication)
7. To examine the patient (Examination of skin covers, presence of destructive changes, presence of pulsation at different levels of the arterial system of lower extremities)
8. To detect the degree of arterial insufficiency of lower extremities)

"Medical cure of the patient with endarteriitis of the lower extremities"

ALGORITHM "Examination of the patient with endarteriitis of the lower extremities"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.

5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention on the character, period of the onset of complaints, presence of the intermittent claudication)
7. To examine the patient (examination of the skin, presence of destructive changes, presence of pulsation on different levels of the arterial system of the lower extremities)
8. To detect the degree of arterial insufficiency of the lower extremities

"Determination of pulsation of the lower extremities"

ALGORITHM "Determination of pulsation of the lower extremities"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To ask the patient to lie down and relax the muscles
6. Determination of pulsation on a. femoralis
7. Determination of pulsation on a. poplitea
8. Determination of pulsation on a. tibialis post
9. Determination of pulsation on a. dorsalis pedis

"Medical cure of the patient with thrombophlebitis of the lower extremities"

ALGORITHM "Examination of the patient with thrombophlebitis of the lower extremities"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (feeling of heaviness of the lower extremities, presence of acute pain in the lower extremity, the onset of the painful cord along the great saphenous vein, swelling of the ankles)
7. To examine the patient (examination of skin, presence of destructive changes, presence of the painful cord along the great saphenous vein, test on the patency of the deep veins)

8. To interpret the indexes of коагулограми

"Medical cure of the patient with varicosity of the lower extremities"

ALGORITHM "Examination of patient with varicosity of the lower extremities"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention on the onset of the complaints, presence: feeling of heaviness, painfulness in the region of varicosity, muscular cramps, swelling of the ankles, trophic ulcers)
7. To examine the patient (examination of skin, presence of the varicose veins, swelling of the ankles, presence of trophic ulcers, test on valvular incompetence of perforative and deep veins)
8. To detect the degree of venous insufficiency of the lower extremities.

"Determination of the valvular function of subcutaneous and deep veins of the lower extremities"

ALGORITHM "Determination of the valvular function of subcutaneous and deep veins of the lower extremities"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. Troyanov-Trendelenburg's test
6. Gakkenbruch's test
7. Pratt's test
8. Sheinice test
9. Talman's test

„Medical cure of the patient with the purulent destructive diseases of lungs"

ALGORITHM „Examination of the patient with the purulent destructive diseases of lungs"

1. 1. To greet and name itself.

2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, development of purulent destructive processes in pulmonary tissue, clinical course of the pathological process before to and after discharge into bronchial tube)
7. To examine the patient (physical examination, palpation, percussion, auscultation)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„Medical cure of the patient with pleurisy”

ALGORITHM „Examination of patient with pleurisy”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, development of pleurisy, clinical course of pathological process)
7. To examine the patient (physical examination, palpation, percussion, auscultation)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„Medical cure of the with the chest trauma”

ALGORITHM „ Examination of patient with the chest trauma”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, development of pleurisy, clinical course of pathological process)
7. To examine the patient (physical examination, palpation, percussion, auscultation)

8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

METHODICAL INSTRUCTIONS FOR 4th YEAR STUDENTS

METHODOLOGICAL INSTRUCTION TO LESSON №6 " PERITONITIS "

The aim. To master clinics, diagnostics, differential diagnostics of various clinical forms of peritonitis. To learn the treatment tactics, indications and contraindications for operative intervention, operative intervention type at various clinical forms of peritonitis, principles of postoperative period course.

Professional orientation of students. The course of acute surgical illnesses of peritoneal organs is complicated with peritonitis development in 20-30% of the cases. The lethality at the extensive forms of peritonitis depends on the skilled early diagnosis and in-time complex treatment.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Theme №1. Acute extensive purulent peritonitis.

1. Anatomic and physiologic data about of the peritoneum.
2. Topography of the peritoneum.
3. Concept definition and classification of the acute peritonitis.
4. Phases of clinical course of the acute extensive purulent peritonitis.
5. General clinical characteristics of the acute extensive purulent peritonitis.
6. Peculiarities of the clinical picture of acute extensive purulent peritonitis depending on reasons and phases of the clinical course.
7. Differential diagnosis of the acute extensive purulent peritonitis.
8. Violations of water-electrolytic and acid-alkaline balance at the acute extensive purulent peritonitis.
9. Verification methods of diagnosis of the acute extensive purulent peritonitis.
10. Curing tactics at peritonitis.
11. Peculiarities of the preoperative preparation at the extensive peritonitis.
12. Principles of operative treatment of patients with the acute extensive peritonitis depending on phases (stages) of the clinical course.
13. Postoperative period course in patients with the acute extensive peritonitis.

Theme №2. Encapsulated peritonitis. Specific clinical forms of peritonitis.

1. Concept definition. Encapsulated peritonitis.
2. Classification of encapsulated peritonitis (as per allocation, clinical course phase).
3. Subdiaphragmatic abscess. Classification.
4. Clinics, diagnostics, differential diagnostics and treatment of the subdiaphragmatic abscess.
5. Clinics, diagnostics, differential diagnostics of Douglas space abscess.
6. Clinics features, diagnostics, differential diagnostics of interintestinal abscesses.
7. Modern verification methods of diagnosis of the encapsulated peritonitis (general view roentgenogram, ultrasonic examination, laparoscopy, roentgenologic methods).
8. Curing tactics at the encapsulated peritonitis.
9. Choice of scope and method of conservative and operative treatment at various clinical forms of the encapsulated peritonitis.
10. Disablement expertise and patients reablement after the operations for encapsulated peritonitis.
11. Concept definition. Classification.
12. Clinics, diagnostics, differential diagnostics of the adhesive tuberculous peritonitis.
13. Clinics, diagnostics, differential diagnostics of the exsudative tuberculous peritonitis.
14. Clinics, diagnostics, differential diagnostics of the caseous tuberculous peritonitis.
15. Curing tactics choice at tuberculous peritonitis.
16. Principles of conservative and operative treatment of patients with various forms of tuberculous peritonitis.

Theme №3. Correction principles of water-electrolytic and acid-alkaline balance. Pre- and postoperative periods course in patients with acute extensive peritonitis.

1. Classification of dehydration and electrolytic violations.
2. Clinical characteristics of water-electrolytic violations at various course phases of the acute extensive peritonitis.
3. Classification of blood substitutes, hydroionic solutions which are used to correct the hydroionic violations.
4. Major principles of intravascular dehydration correction.
5. Principles of intravascular and mixed dehydration correction.
6. Concrete example of calculation of hyponatremia correction.
7. Concrete example of calculation of hypopotassiumemia correction.
8. General rules of infusion therapy.

BREAK 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.**Basic level of knowledge and skills:****The student must know:**

1. Anatomic and physiologic features of the peritoneum.
2. Topography of the peritoneum.
3. Definition of "the acute peritonitis" concept. Etiopathogenesis of the acute peritonitis.
4. Classification of the peritonitis.
5. Clinics, diagnostics, differential diagnostics of the acute extensive peritonitis depending on reasons and stages of clinical course.
6. Clinics, diagnostics, differential diagnostics of the encapsulated peritonitis (infiltrate, peritoneal abscess).
7. Diagnostics and curing tactics at the tuberculous peritonitis.

8. Peculiarities of clinical course and differential diagnosis of the false peritonitis (at diabetes mellitus, uremia, Schonlein-Henoch disease).
9. Peculiarities of the preoperative preparation in patients with the extensive peritonitis.
10. Principles of operative treatment of patients with various clinical versions of the peritonitis.
11. Peculiarities of the postoperative course in patients with the peritonitis.

The student has to be able to:

1. To take the history of the patient with the suspect for the peritonitis.
2. To reveal main clinical features and symptoms at various clinical forms of the extensive peritonitis.
3. To reveal main clinical features and symptoms at various clinical forms of the encapsulated peritonitis.
4. To reveal main clinical features and symptoms at various clinical forms of the tuberculous and false peritonitis.
5. To define free and limited liquid and gas in the abdominal cavity, splash noise, characteristics of peristaltic violations.
6. To argue and explain the preliminary diagnosis.
7. To make a plan of patient examination and explain the examination results (general blood test, general urine test, roentgenologic examination of the alimentary tract general view roentgenogram), ultrasonic examination, biochemical blood test, clinical, biochemical and bacteriologic examination of the peritoneal content).
8. To define curing tactics, indications and contraindications for operative intervention, to choose properly the preoperative preparation, anesthesia type, operative access and type of intervention according to the clinical form and course phase of peritonitis.
9. To define principles of conservative treatment of the peritonitis.

TEST EVALUATION AND SITUATIONAL TASKS 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.
(the instructor has tests & situational tasks)

1. The following statement about peritonitis are all true except:
 - A. Peritonitis is defined as inflammation of the peritoneum.
 - B. Most surgical peritonitis is secondary to bacterial contamination.
 - C. Primary peritonitis has no documented source of contamination and is more common in adults than in children and in men than in women.
 - D. Tuberculous peritonitis can present with or without ascites.
2. The most common indication for surgery secondary to acute diverticulitis is:
 - A. Abscess.
 - B. Colonic obstruction.
 - C. Colovesical fistula.
 - D. Free perforation.
 - E. Hemorrhage.
3. Which of the following statements most accurately describes the current therapy for pyogenic hepatic abscess?
 - A. Antibiotics alone are adequate for the treatment of most cases.
 - B. All patients require open surgical drainage for optimal management.
 - C. Optimal treatment involves treatment of not only the abscess but the underlying source as well.
 - D. Percutaneous drainage is more successful for multiple lesions than for solitary ones.

4. Which of the following statements characterize amebic abscess?
- A. Mortality is higher than that for similarly located pyogenic abscesses.
 - B. The diagnosis of amebic abscess may be based on serologic tests and resolution of symptoms.
 - C. In contrast to pyogenic abscess, the treatment of amebic abscess is primarily medical.
 - D. Patients with amebic abscess tend to be older than those with pyogenic abscess.

The answers for the self-checking tests.

1 – C.

2 – A.

3 – C.

4 – B,C.

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others "Hospital surgery". Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery/4/Topic%2006%20Acute%20peritonitis.htm

Additional –

1. Manual "Facultative Surgery" under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section "Diseases of the rectum and the colon",
2. Compact disc "Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

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Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

METHODICAL INSTRUCTIONS FOR 5th YEAR STUDENTS
METHODOLOGICAL INSTRUCTION TO LESSON №8

POSTRESECTION AND POSTVAGOTOMIC SYNDROMES. POSTCHOLECYSTECTOMIC SYNDROME

I. AIM. To be able to diagnose, to know symptoms of diseases of operated stomach, depending on clinical type of the disease. Principles of conservative and surgical treatment. To be able to diagnose, to know symptoms and principles of conservative and surgical treatment of of postcholecystectomic syndrome.

II. Professional orientation of the student.

Knowledge of symptoms, appearing after performed stomach resections and vagotomies, causes proper approach to choosing of operative intervention and the techniques of its performing.

Cholecystectomy is one of frequently performed operations in surgical clinics. Knowledge of symptoms, clinical signs and diagnosing of the diseases, that are grouped into the term "PCES", gives us a possibility to prevent its appearing in one cases, and to determine the expediency of conservative or surgical treatment in another ones.

Methodology of Practical Class 9.00-12.00.

Algorithm of communicative skills:

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. A patient is in vertical position persons to the doctor, neck and shoulders of patient is maximally weakened.

Student's independent work program:

Theme №1. POSTRESECTION AND POSTVAGOTOMIC SYNDROMES

1. Anatomical, morphological and functional peculiarities of stomach (anatomy, topographical anatomy, anatomical pathology, histology).
2. Etiology, pathogenesis and classification of diseases of operated stomach (pathological physiology, propaedeutical therapy, faculty surgery).
3. Estimation of x-ray, endoscopic, laboratory findings (rhoetgenology, functional diagnostics).
4. Mechanism of antiulcerogenic agents action, correction of electrolytes metabolism violation, use of digestive enzymes, hormones, vitamins, methods of operative intervention (pharmacology, faculty surgery).
5. Rontgenoscopy, estimation of x-ray reports, gastric juices test.

Theme №2. POSTCHOLECYSTECTOMIC SYNDROME

1. Anatomical, morphological and functional peculiarities of biliary tracts (anatomy, topographical anatomy, anatomical pathology).
2. Classification of postcholecystectomic syndrome (propaedeutical therapy)
3. Estimation of clinical and laboratory, x-ray, ultrasonographical, endoscopic, findings (rhoetgenology, infectious diseases, biochemistry).
4. Mechanism of antiinflammatory, spasmolytic, detoxicative, replacement agents action, methods of operative intervention on biliary tracts (pharmacology, topographical anatomy and operative surgery).

5. Constants of biochemical blood and urine tests.

Break 12.00-12.30

Seminar discussion of theoretical issues 12.30-14.00.

Basic level of knowledge and skills:

The student must know:

1. Surgical anatomy of stomach and duodenum.
2. Types of stomach resections at ulcerous disease.
3. Types of draining operations on stomach at ulcerous disease.
4. Choosing the method of surgical treatment of duodenum ulcer.
5. Types of vagotomies, their characteristics: indications, contraindications to their performing.
6. Classification of postresection syndromes.
7. Reasons of dumping syndrome appearing.
8. Theories of dumping syndrome appearing.
9. Clinical variants of dumping syndrome passing.
10. X-ray and clinical criteria of disease severeness degree estimation.
11. Conservative treatment of dumping syndrome.
12. Indications to surgical treatment of dumping syndrome. Types of operations.
13. Methods of stomach resection, that prevent dumping syndrome.
14. Afferent loop syndrome: its reasons, classification.
15. Afferent loop syndrome: clinical variants, diagnosis.
16. Peptic ulcer of gastroenteroanastomosis: its reasons, complications, diagnosis.
17. Peptic ulcer of gastroenteroanastomosis: types of surgical interventions.
18. Coloventricular fistula: clinical and X-ray diagnosis.
19. Coloventricular fistula: methods of surgical interventions.
20. Zollinger-Ellison syndrome: diagnosis and treatment.
21. Factors of stomach and duodenum ulcer relapsing after vagotomy.
22. Clinical variants of relapsing ulcer after vagotomy.
23. X-ray and endoscopic signs of stomach and duodenum ulcer relapsing.
24. Conservative methods of postvagotomic relapsing ulcers treatment.
25. Surgical treatment of postvagotomic relapsing ulcers.
26. Postvagotomic diarrhoea: reasons, classification by severeness degrees.
27. Treatment of postvagotomic diarrhoea.
28. Postvagotomic gastrostasis: its degrees, treatment.
29. Surgical anatomy of biliary tracts.
30. Instrumental methods of examination at PCES.
31. Classification of PCES by Shalimov.
32. Types of external draining of common bile duct.
33. Types of internal draining of common bile duct.
34. PCES, determined by changes, not corrected during cholecystectomy.
35. PCES, that has appeared as a result of cholecystectomy.
36. PCES: diseases of hepatobiliary system, that were not corrected by cholecystectomy.
37. PCES: organic and functional disorders of organs and systems, that are not connected with cholecystectomy.
38. PCES: Nerve-kinetic disorders of hepatic ducts and duodenum.
39. Cicatric biliary stricture. Types of operations.
40. Differential diagnosis of obstructive jaundice.

The student has to be able to:

1. Reveal basic clinical signs of diseases of operated stomach.
2. Substantiate and formulate the clinical diagnosis.
3. Make differential diagnosis.
4. Substantiate pharmacotherapy, indications for surgical treatment.
5. Reveal basic reasons and clinical signs of postcholecystectomic syndrome.
6. Substantiate and formulate the clinical diagnosis.
7. Make differential diagnosis.
8. Substantiate pharmacotherapy, indications for surgical treatment.

Technical means and material provision of lectures (names and numbers of tables, compact discs, instruments quantity, subjectpatients, etc.) –

Multimedia projection of demonstration material (pictures, tables, videofilms) from compact disc “Clinical Surgery”, videofilms with operations.

Test evaluation and situational tasks 14.00-15.00.

Initial level of knowledge and skills are checked by solving situational tasks for each topic, answers in test evaluations and constructive questions.

(the instructor has tests & situational tasks)

1. 40-years old patient, 3 years ago had an operation of stomach resection by Hoffmeister-Finsterer because of duodenum ulcer with petetration into the head of pancreas. Duting the last year notices everyday pain in right subcostal region, that are attended by bile vomiting up to 1 l in volume. After vomiting pain disappears. The patient several times have passed treatment courses in surgical department, but there was no improving of his state. The patient has lost 16 kg of weight. During x-ray examination the contrast gets into the afferent loop.

Your diagnosis?

Surgeon's tactics?

2. 45-year old patient, 3 years ago had an operation of 2/3 stomach resection by Hoffmeister-Finsterer because of stenosing ulcer of the antrum. Now the expressed dumping syndrome of severe degree with ptogressive weight loss is observed, despite repeated courses of in-patient and health resort treatment. Psychoneurological disorders are expressed slightly. There is no bile vomiting. At x-ray examination of gastric stump the accelerated evacuation is observed.

Your diagnosis?

Surgeon's tactics?

3. 48-years old patient, that has undergone an operation of cholecystectomy 3 years ago, has complaints on cramping pains, that were followed by raised temperature up to 38⁰C, scleral and mucous membranes icteritiousness. Whole bilirubin 128,0 mkmol/l, leukocites – 15,0x10⁹/l, stab neutrophils – 16 %.

What methods of examination should be used?

Your diagnosis?

Treatment tactics?

4. 35-year old female patient, a month ago had an operation of cholecystectomy. Now she complains on circumferential pain in abdomen, nausea, vomiting, icteritiousness of scleras and visible mucous membranes. Whole bilirubin is 60,0 mkmol/l. While palpation the patient feels pain in left mesogastral region. Symptoms of rebound stomach tenderness are absent.

Your diagnosis?

Surgeon's tactics?

Information sources:

Main –

1. Townsend CM, Beauchamp RD, Evers BM, Mattox KL, eds. Sabiston Textbook of Surgery. 19th ed. Philadelphia, Pa: Saunders Elsevier; 2012.
2. L.Ya.Kovalchuck, Yu.P.Spizhenko, V.F.Sayenko and others “Hospital surgery”. Ternopil: Ukrmendknyga, 1999.
3. Practical classes materials http://intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/Surgery

[/4/Topic%2008%20POSTRESECTION%20AND%20POSTVAGOTOMY%20SYNDROMES.%20CLASSIFICATION.%20CLINICS.htm](#)

Additional –

1. Manual “Facultative Surgery” under the edition of V.O.Shidlovsky – Ternopil: Ukrmedknyga, 2002, section “Diseases of the rectum and the colon”,
2. Compact disc “Facultative surgery.
3. Schwartz's Principles of Surgery, Ninth Edition. F. Brunicaudi. Philadelphia, Pa: Saunders Elsevier; 2009
4. Zollinger's Atlas of Surgical Operations, 9th Edition. Robert Zollinger Jr., Elsevier; 2010
5. Chen, Herbert. Illustrative Handbook of General Surgery. Berlin: Springer, 2010.

The author: As. prof. A. Hospodarskyy

Обговорено на засіданні кафедри
"11" січня 2013 р. протокол №9

„Medical cure of patient with acute cholecystitis”

ALGORITHM „ Examination of patient with acute cholecystitis”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, the error in the alcohol intake, development of the pain irradiation, presence in anamnesis of the similar symptoms)
7. To examine the patient (palpation, percussion of the abdomen , determination of particular symptoms)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

Practical skill "Determination of the symptoms of peritoneal irritation"

ALGORITHM for "Determination of the symptoms of peritoneal irritation"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
To ask a patient to lie down on the back and relax the muscles of front abdominal wall
6. The external examination of abdomen is performed, the doctor marks the
7. symmetry, presence of outpouching and participation of front abdominal wall in the act of breathing
8. The right arm of the doctor on the front abdominal wall (any area) slowly presses by 4 fingers of doctor (to estimate the degree of pain)
9. All hand is quickly taken off from abdominal wall to estimate the increase of pain degree (Schotkin – Blumberg's sign)
10. By the fingers tips of right arm the doctor performs dosed percussion of front abdominal wall (Rozdolsky's sign).

„Medical cure of patient with acute intestinal obstruction

ALGORITHM „To determine the symptoms of the acute intestinal obstruction”

1. To greet and name itself.

2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, defecation, swelling of the abdomen, development of the pain irradiation)
7. To examine the patient (palpation of the large and small intestine, auscultation of peristalsis, determination of particular symptoms)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of the diagnosis (X-Ray films)

Practical skill "To determine the symptoms of the perforative ulcer"

ALGORITHM for "To determine the symptoms of the perforative ulcer "

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints. Correct and quiet conversation with a patient. To ask a patient to lie down on the back and relax the muscles of front abdominal wall.
6. The external examination of abdomen is performed, the doctor marks the symmetry, presence of outpouching and participation of front abdominal wall in the act of breathing
7. Before the palpation epigastric region on a front abdominal wall is determined
8. Determination of wooden abdomen, ulcer anamnesis and knife-like pain (Mondore sign)
9. Confirming diagnosis by finding gas on X-Ray film which performed on vertical position of the patient.

„Medical cure of patient with acute appendicitis”

ALGORITHM „ Examination of patient with acute appendicitis”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the onset of the disease, irradiation of pain,

presence of the dysuric phenomena)

7. To examine the patient (palpation, percussion of abdomen, determination of particular symptoms)
8. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„Medical cure of patient with acute pancreatitis

ALGORITHM „To determine the symptoms of the acute pancreatitis”

9. To greet and name itself.
10. Friendly behaviour.
11. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
12. Correct and quiet conversation with a patient.
13. To get the agreement of patient for the performance of the examination. To take complaints
14. To take anamnesis (to pay attention to the onset of the disease, the error in the alcohol intake, development of the pain irradiation)
15. To examine the patient (palpation of the pancreas, determination of particular symptoms)
16. Interpretation of laboratory and instrumental methods of investigation, establishment of the diagnosis

Practical skill "To perform digital examination of rectum"

ALGORITHM for "To perform digital examination of rectum"

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
Examination of perianal area in knee-elbow position or in position of patient on a side with the flexed knees.
6. The index finger, dressed into the glove and smeared by solution of lubricant enters the rectum.
7. Estimated: tonus of anal sphincter, wall of the rectum, presence of compressions, excrescences, overhanging, pain syndrome, pathological maintenance.
8. After rectal examination the character of maintenance is estimated on a glove.

„Medical cure of patient with the incarcerated and non-incarcerated hernia”

ALGORITHM „ Curation of patient with incarcerated and non-incarcerated hernia”

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
1. To get the agreement of patient for the performance of the examination. To take complaints
2. To take anamnesis (to pay attention to the onset of the disease, period from the moment of strangulation, presence of clinic of intestinal obstruction)
3. To examine the patient (palpation of patient in horizontal and vertical positions, palpation of inguinal channel, scrotum, the sign of coughing push)
4. Interpretation of laboratory and instrumental methods of investigation, establishment of diagnosis

„To perform the catheterization of urinary bladder by a soft catheter”**ALGORITHM „To perform the catheterization of urinary bladder by a soft catheter”**

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. The position of the patient – horizontal, thereafter to prepare the instruments, necessary for performance of this manipulation
6. To wash up the external pudenda foramen of urethra by antiseptic solution
7. To perform catheterization of urinary bladder by a soft catheter with according to the rules of the introduction of the last.

„Medical cure of patient with the acute urine delay”**ALGORITHM „ Examination of patient with the acute urine delay”**

1. To greet and name itself.
2. Friendly behaviour.
3. To explain to the patient a purpose of the visit, duration of conversation and to get his agreement.
4. Correct and quiet conversation with a patient.
5. To get the agreement of patient for the performance of the examination. To take complaints
6. To take anamnesis (to pay attention to the period of the onset of complaints; frequency of urination, especially in a night time, presence of haematouria in

anamnesis)

7. To examine the patient (examination, palpation and percussion of suprapubic area, determination of Pasternacky's sign, digital rectal examination)
8. To detect the cause of acute urine delay

TOPIC №18. SYNDROME OF LIMB ISCHEMIA. CAUSES. CONSERVATIVE AND SURGICAL TREATMENT OF ARTERIAL OCCLUSIONS OF THE LOWER EXTREMITIES.

General Approach to the Vascular Patient

Because the vascular system involves every organ system in our body, the symptoms of vascular disease are as varied as those encountered in any medical specialty. Lack of adequate blood supply to target organs typically presents with pain; for example, calf pain with lower extremity (LE) claudication, postprandial abdominal pain from mesenteric ischemia, and arm pain with axillosubclavian arterial occlusion. In contrast, stroke and transient ischemic attack (TIA) are the presenting symptoms from middle cerebral embolization as a consequence of a stenosed internal carotid artery (ICA). The pain syndrome of arterial disease usually is divided clinically into acute and chronic types, with all shades of severity between the two extremes. Sudden onset of pain can indicate complete occlusion of a critical vessel, leading to more severe pain and critical ischemia in the target organ, resulting in lower limb gangrene or intestinal infarction. Chronic pain results from a slower, more progressive atherosclerotic occlusion, which can be totally or partially compensated by developing collateral vessels. Acute on chronic is another pain pattern in which a patient most likely has an underlying arterial stenosis that suddenly occludes; for example, the patient with a history of calf claudication who now presents with sudden, severe acute limb-threatening ischemia. The clinician should always try to understand and relate the clinical manifestations to the underlying pathologic process.

Vascular History

Appropriate history should be focused on the presenting symptoms related to the vascular system (Table 1). Of particular importance in the previous medical history is noting prior vascular interventions (endovascular or open surgical), and all vascular patients should have inquiry made about their prior cardiac history and current cardiac symptoms. Approximately 30% of vascular patients will be diabetic. A history of prior and current smoking status should be noted.

Table 1. Pertinent Elements in Vascular History

- History of stroke or transient ischemic attack
- History of coronary artery disease, including previous myocardial infarction and angina
- History of peripheral arterial disease
- History of diabetes
- History of hypertension
- History of tobacco use
- History of hyperlipidemia

The patient with carotid disease in most cases is completely asymptomatic, having been referred based on the finding of a cervical bruit or duplex finding of stenosis. Symptoms of carotid territory TIAs include transient monocular blindness (amaurosis), contralateral weakness or numbness, and dysphasia. Symptoms persisting longer than 24 hours constitute a stroke. In contrast, the patient with chronic mesenteric ischemia is likely to present with postprandial abdominal pain and weight loss. The patient fears eating because of the pain, avoids food, and loses weight. It is very unlikely that a patient with abdominal pain who has not lost weight has chronic mesenteric ischemia.

The patient with LE pain on ambulation has intermittent claudication that occurs in certain muscle groups; for example, calf pain upon exercise usually reflects superficial femoral artery (SFA) disease, while pain in the buttocks reflects iliac disease. In most cases, the pain manifests in one muscle group below the level of the affected artery, occurs only with exercise, and is relieved with rest only to recur at the same location, hence the term *window gazers disease*. Rest pain (a manifestation of severe underlying occlusive disease) is constant and occurs in the foot (not the muscle groups), typically at the metatarsophalangeal junction, and is relieved by dependency. Often, the patient is prompted to sleep with their foot hanging off one side of the bed to increase the hydrostatic pressure.

Vascular Physical Examination

Specific vascular examination should include abdominal aortic palpation, carotid artery examination, and pulse examination of the LE (femoral, popliteal, posterior tibial, and dorsalis pedis arteries). The abdomen should be palpated for an abdominal aortic aneurysm (AAA), detected as an expansile pulse above the level of the umbilicus. It also should be examined for the presence of bruits. Because the aorta typically divides at the level of the umbilicus, an aortic aneurysm is most frequently palpable in the epigastrium. In thin individuals, a normal aortic pulsation is palpable, while in obese patients even large aortic aneurysms may not be

detectable. Suspicion of a clinically enlarged aorta should lead to the performance of an ultrasound scan for a more accurate definition of aortic diameter.

The carotids should be auscultated for the presence of bruits, although there is a higher correlation with coronary artery disease (CAD) than underlying carotid stenosis. A bruit at the angle of the mandible is a significant finding, leading to follow-up duplex scanning. The differential diagnosis is a transmitted murmur from a sclerotic or stenotic aortic valve. The carotid is palpable deep to the sternocleidomastoid muscle in the neck. Palpation, however, should be gentle and rarely yields clinically useful information.

Upper extremity examination is necessary when an arteriovenous graft is to be inserted in patients who have symptoms of arm pain with exercise. Thoracic outlet syndrome can result in occlusion or aneurysm formation of the subclavian artery. Distal embolization is a manifestation of thoracic outlet syndrome; consequently, the fingers should be examined for signs of ischemia and ulceration. The axillary artery enters the limb below the middle of the clavicle, where it can be palpated in thin patients. It usually is easily palpable in the axilla and medial upper arm. The brachial artery is most easily located at the antecubital fossa immediately medial to the biceps tendon. The radial artery is palpable at the wrist anterior to the radius.

For LE vascular examination, the femoral pulse usually is palpable midway between the anterior superior iliac spine and the pubic tubercle. The popliteal artery is palpated in the popliteal fossa with the knee flexed to 45° and the foot supported on the examination table to relax the calf muscles. Palpation of the popliteal artery is a bimanual technique. Both thumbs are placed on the tibial tuberosity anteriorly and the fingers are placed into the popliteal fossa between the two heads of the gastrocnemius muscle. The popliteal artery is palpated by compressing it against the posterior aspect of the tibia just below the knee. The posterior tibial pulse is detected by palpation 2 cm posterior to the medial malleolus. The dorsalis pedis is detected 1 cm lateral to the hallucis longus extensor tendon, which dorsiflexes the great toe and is clearly visible on the dorsum of the foot. Pulses can be graded using either the traditional four-point scale or the basic two-point scale system (Table 2). The foot also should be carefully examined for pallor on elevation and rubor on dependency, as these findings are indicative of chronic ischemia. Note should also be made of nail changes and loss of hair. Ulceration and other findings specific to disease states are described in relevant sections below.

Table 2 Grading Scales for Peripheral Pulses

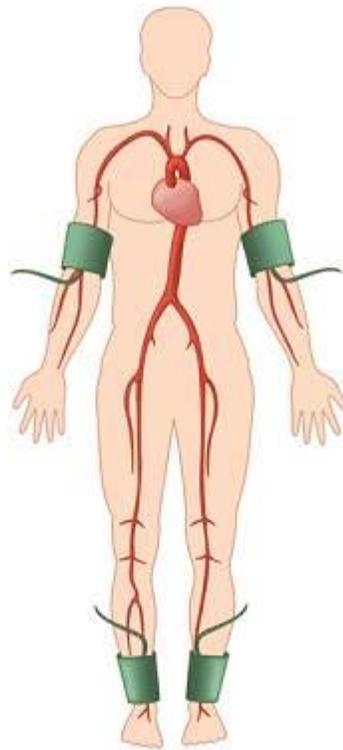
Traditional Scale		Basic Scale	
4+	Normal	2+	Normal
3+	Slightly reduced	1+	Diminished
2+	Markedly reduced	0	Absent
1+	Barely palpable	—	—
0	Absent	—	—

After reconstructive vascular surgery, the graft may be available for examination, depending on its type and course. The in situ LE graft runs in the subcutaneous fat and can be palpated along most of its length. A change in pulse quality, aneurysmal enlargement, or a new bruit should be carefully noted. Axillofemoral grafts, femoral-to-femoral grafts, and arteriovenous access grafts usually can be easily palpated as well.

Noninvasive Diagnostic Evaluation of the Vascular Patient

Ankle-Brachial Index

There is increasing interest in the use of the ankle-brachial index (ABI) to evaluate patients at risk for cardiovascular events. An ABI <0.9 correlates with increased risk of myocardial infarction and indicates significant, although perhaps asymptomatic, underlying peripheral vascular disease. The ABI is determined in the following ways. Blood pressure (BP) is measured in both upper extremities using the highest systolic BP as the denominator for the ABI. The ankle pressure is determined by placing a BP cuff above the ankle and measuring the return to flow of the posterior tibial and dorsalis pedis arteries using a pencil Doppler probe over each artery. The ratio of the systolic pressure in each vessel divided by the highest arm systolic pressure can be used to express the ABI in both the posterior tibial and dorsalis pedis arteries (Fig. 1). Normal is more than 1. Patients with claudication typically have an ABI in the 0.5 to 0.7 range, and those with rest pain are in the 0.3 to 0.5 range. Those with gangrene have an ABI of <0.3 . These ranges can vary depending on the degree of compressibility of the vessel. The test is less reliable in patients with heavily calcified vessels. Due to noncompressibility, some patients such as diabetics and those with end-stage renal disease may have an ABI of 1.40 or greater and require additional noninvasive diagnostic testing to evaluate for peripheral arterial disease (PAD). Alternative tests include toe-brachial pressures, pulse volume recordings, transcutaneous oxygen measurements, or vascular imaging (duplex ultrasound).

**Right ABI = ratio of**

Higher of the right ankle systolic pressures (posterior tibial or dorsalis pedis)

Higher arm systolic pressure (left or right arm)

Left ABI = ratio of

Higher of the left ankle systolic pressures (posterior tibial or dorsalis pedis)

Higher arm systolic pressure (left or right arm)

Fig. 1. Ankle-Brachial Index

Segmental Limb Pressures

By placing serial BP cuffs down the LE and then measuring the pressure with a Doppler probe as flow returns to the artery below the cuff, it is possible to determine segmental pressures down the leg. These data can then be used to infer the level of the occlusion. The systolic pressure at each level is expressed as a ratio, with the highest systolic pressure in the upper extremities as the denominator. Normal segmental pressures commonly show high thigh pressures 20 mmHg or greater in comparison to the brachial artery pressures. The low thigh pressure should be equivalent to brachial pressures. Subsequent pressures should fall by no more than 10 mmHg at each level. A pressure gradient of 20 mmHg between two subsequent levels is usually indicative of occlusive disease at that level. The most frequently used index is the ratio of the ankle pressure to the brachial pressure, the ABI. Normally the ABI is >1.0 , and a value <0.9 indicates some degree of arterial obstruction and has been shown to be correlated

with an increased risk of coronary heart disease.¹ Limitations of relying on segmental limb pressures include: (a) missing isolated moderate stenoses (usually iliac) that produce little or no pressure gradient at rest; (b) falsely elevated pressures in patients with diabetes and end-stage renal disease; and (c) the inability to differentiate between stenosis and occlusion.² Patients with diabetes and end-stage renal disease have calcified vessels that are difficult to compress, thus rendering this method inaccurate, due to recording of falsely elevated pressure readings. Noncompressible arteries yield ankle systolic pressures of 250 mmHg or greater and an ABI of >1.40 . In this situation, absolute toe and ankle pressures can be measured to gauge critical limb ischemia. Ankle pressures <50 mmHg or toe pressures <30 mmHg are indicative of critical limb ischemia. The toe pressure is normally 30 mmHg less than the ankle pressure, and a toe-brachial index of <0.70 is abnormal. False-positive results with the toe-brachial index are unusual. The main limitation of this technique is that it may be impossible to measure pressures in the first and second toes due to pre-existing ulceration.

Pulse Volume Recording

In patients with noncompressible vessels, segmental plethysmography can be used to determine underlying arterial occlusive disease. Cuffs placed at different levels on the leg detect changes in leg volume and produce a pulse volume recording (PVR) when connected to a plethysmograph (Fig. 2). To obtain accurate PVR waveforms the cuff is inflated to 60 to 65 mmHg to detect volume changes without causing arterial occlusion. Pulse volume tracings are suggestive of proximal disease if the upstroke of the pulse is not brisk, the peak of the wave tracing is rounded, and there is disappearance of the dicrotic notch.



A

Fig. 2 A Pulse volume recording is done by connecting blood pressure cuffs and plethysmograph to various levels of the leg

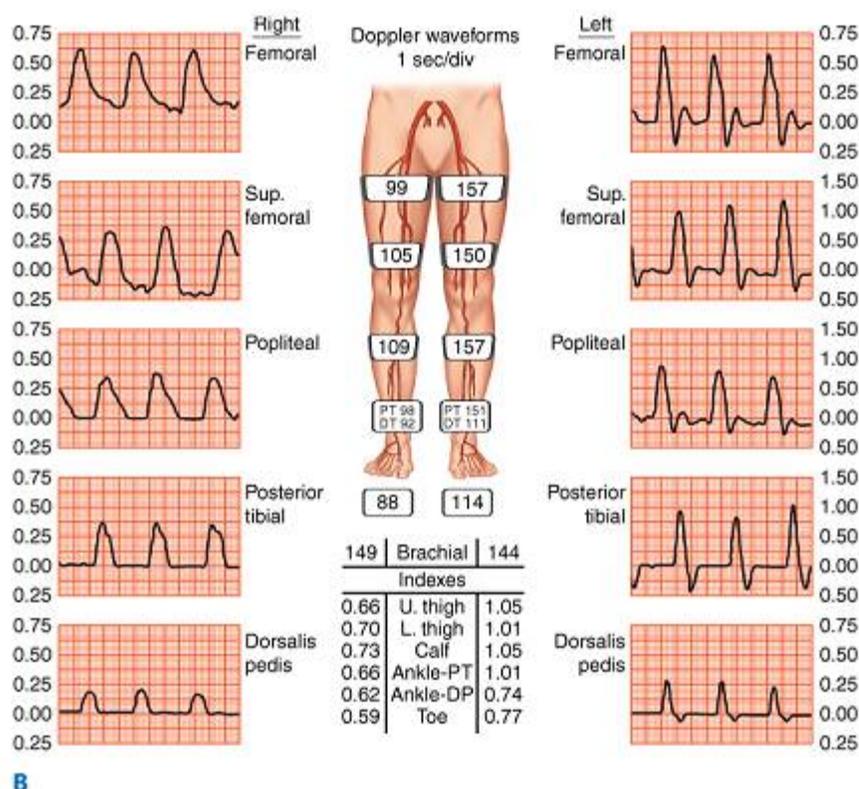


Fig. 2 B Typical report of peripheral vascular study with arterial segmental pressure measurement plus Doppler evaluation of the lower extremity.

Although isolated segmental limb pressures and PVR measurements are 85% accurate when compared with angiography in detecting and localizing significant atherosclerotic lesions, when used in combination, accuracy approaches 95%.³ For this reason, it is suggested that these two diagnostic modalities be used in combination when evaluating PAD.

Radiological Evaluation of the Vascular Patient

Ultrasound

Ultrasound examinations are relatively time consuming, require experienced technicians, and may not visualize all arterial segments. Doppler waveform analysis can suggest atherosclerotic occlusive disease if the waveforms in the insonated arteries are biphasic,

monophasic, or asymmetrical. B-mode ultrasonography provides black and white, real-time images. B-mode ultrasonography does not evaluate blood flow; thus, it cannot differentiate between fresh thrombus and flowing blood, which have the same echogenicity. Calcification in atherosclerotic plaques will cause acoustic shadowing. B-mode ultrasound probes cannot be sterilized. Use of the B-mode probe intraoperatively requires a sterile covering and gel to maintain an acoustic interface. Experience is needed to obtain and interpret images accurately. Duplex ultrasonography entails performance of B-mode imaging, spectral Doppler scanning, and color flow duplex scanning. The caveat to performance of duplex ultrasonography is meticulous technique by a certified vascular ultrasound technician, so that the appropriate 60° Doppler angle is maintained during insonation with the ultrasound probe. Alteration of this angle can markedly alter waveform appearance and subsequent interpretation of velocity measurements. Direct imaging of intra-abdominal vessels with duplex ultrasound is less reliable because of the difficulty in visualizing the vessels through overlying bowel. These disadvantages currently limit the applicability of duplex scanning in the evaluation of aortoiliac and infrapopliteal disease. In a recent study, duplex ultrasonography had lower sensitivity in the calculation of infrapopliteal vessel stenosis in comparison to conventional digital subtraction or computer tomography angiography.⁴ Few surgeons rely solely on duplex ultrasonography for preoperative planning in LE revascularizations. However, in the hands of experienced ultrasonographers, LE arteries can be assessed accurately by determining the significance of velocity criteria across the arterial stenosis. Duplex scanning is unable to evaluate recently implanted polytetrafluoroethylene (PTFE) and polyester (Dacron) grafts because they contain air, which prevents ultrasound penetration.

Computed Tomography Angiography

Computed tomography angiography (CTA) is a noninvasive, contrast-dependent method for imaging the arterial system. It depends on IV infusion of iodine-based contrast agents. The patient is advanced through a rotating gantry, which images serial transverse slices. The contrast-filled vessels can be extracted from the slices and rendered in three-dimensional format (Fig. 3). The extracted images can also be rotated and viewed from several different directions during postacquisition image processing. This technology has been advanced as a consequence of aortic endografting. CTA provides images for postprocessing that can be used to display the aneurysm in a format that demonstrates thrombus, calcium, lumen, and the outer wall, and allows "fitting" of a proposed endograft into the aneurysm (Fig. 4). CTA is increasingly being used to image the carotid bifurcation, and as computing power increases, the

speed of image acquisition and resolution will continue to increase. The major limitations of multidetector CTA are use of contrast and presence of artifacts caused by calcification and stents. CTA can overestimate the degree of instant stenosis, while heavy calcification can limit the diagnostic accuracy of the method by causing a "blooming artifact."⁵ The artifacts can be overcome with alteration in image acquisition technique. There are no randomized trials to document the superiority of multidetector CTA over traditional angiography, but there is emerging evidence to support the claim that multidetector CTA has sensitivity, specificity, and accuracy that rival invasive angiography.



Fig. 3 A. A multidetector computed tomographic angiogram with three-dimensional reconstruction of the iliofemoral arterial circulation in two patients with lower leg claudication. A 50-year-old man with an occluded right superficial femoral artery (*single long arrow*) with reconstituted superficial femoral artery at the level of mid thigh. Arterial calcifications (*short arrows*) in the bilateral distal superficial femoral arteries.



Fig. 3 B. A multidetector computed tomographic angiogram with three-dimensional reconstruction of the iliofemoral arterial circulation in two patients with lower leg claudication.



Fig. 4. Three-dimensional computed tomographic angiogram of an abdominal aortic aneurysm that displays various aneurysm components, including thrombus, aortic calcification, blood circulation, and aneurysm wall.

Magnetic Resonance Angiography

Magnetic resonance angiography (MRA) has the advantage of not requiring iodinated contrast agents to provide vessel opacification (Fig. 5). Gadolinium is used as a contrast agent for MRA studies, and as it is generally not nephrotoxic, it can be used in patients with elevated creatinine. MRA is contraindicated in patients with pacemakers, defibrillators, spinal cord stimulators, intracerebral shunts, cochlear implants, and cranial clips. Patients with claustrophobia may require sedation to be able to complete the test. The presence of metallic stents causes artifacts and signal dropout; however, these can be dealt with using alternations in image acquisition and processing. Nitinol stents produce minimal artifact.⁶ Compared to other modalities, MRA is relatively slow and expensive. However, due to its noninvasive nature and decreased nephrotoxicity, MRA is being used more frequently for imaging vasculature in various anatomic distributions.



Fig. 5. Magnetic resonance angiogram of aortic arch and carotid arteries. This study can provide a three-dimensional analysis of vascular structures such as aortic arch branches, as well as carotid and vertebral arteries

Diagnostic Angiography

Diagnostic angiography is considered the gold standard in vascular imaging. In many centers, its use is rapidly decreasing due to the development of noninvasive imaging modalities such as duplex arterial mapping, CTA, and MRA. Nevertheless, contrast angiography still remains in widespread use. The essential aspects of angiography are vascular access and catheter placement in the vascular bed that requires examination. The imaging system and the contrast agent are used to opacify the target vessel. Although in the past this function has largely been delegated to the interventional radiology service, an increasing number of surgeons are performing this procedure and following the diagnostic imaging with immediate surgical or endovascular intervention. There are several considerations when relying on angiography for imaging.

Approximately 70% of atherosclerotic plaques occur in an eccentric location within the blood vessel; therefore, images can be misleading when trying to evaluate stenoses because angiography is limited to a uniplanar "lumenogram." With increased use of intravascular stent deployment, it has been also noted that assessment of stent apposition and stent position in relation to surrounding branches may be inaccurate. Furthermore, angiography exposes the

patient to the risks of both ionizing radiation and intravascular contrast. Nevertheless, contrast angiography remains the most common invasive method of vascular investigation for both diagnostic and therapeutic intervention. The angiogram usually provides the final information needed to decide whether or not to proceed with operation or endovascular interventions.

Digital subtraction angiography (DSA) offers some advantages over conventional cut-film angiography, such as excellent visualization despite use of lower volumes of contrast media. In particular when multilevel occlusive lesions limit the amount of contrast reaching distal vessels, supplemental use of digital subtraction angiographic techniques may enhance visualization and definition of anatomy. Intra-arterial DSA uses a portable, axially rotatable imaging device that can obtain views from different angles. DSA also allows for real-time video replay (Fig. 6). An entire extremity can be filmed with DSA, using repeated injections of small amounts of contrast agent to obtain sequential angiographic images, the so-called *pulse-chase technique*.



Fig. 6. Digital subtraction angiography provides excellent visualization of intravascular circulation with intra-arterial contrast administration. As depicted in this digital subtraction

angiography study, multilevel lesions are demonstrated that include a focal left iliac artery stenosis (*large arrow*), right superficial femoral occlusion (*curved arrows*), left superficial femoral stenosis (*small arrow*), and multiple tibial artery stenoses (*arrowheads*).

Preoperative Cardiac Evaluation

The most important and controversial aspect of preoperative evaluation in patients with atherosclerotic disease requiring surgical intervention is the detection and subsequent management of associated CAD. Several studies have documented the existence of significant CAD in 40 to 50% or more of patients requiring peripheral vascular reconstructive procedures, 10 to 20% of whom may be relatively asymptomatic largely because of their inability to exercise. Myocardial infarction is responsible for the majority of both early and late postoperative deaths. Most available screening methods lack sensitivity and specificity to predict postoperative cardiac complications. There have been conflicting reports regarding the utility of preoperative dipyridamole-thallium nuclear imaging or dobutamine-echocardiography to stratify vascular patients in terms of perioperative cardiac morbidity and mortality. In nearly one half of patients, thallium imaging proves to be unnecessary because cardiac risk can be predicted by clinical information alone. Even with coronary angiography, it is difficult to relate anatomic findings to functional significance, and hence, surgical risk. There are no data confirming that percutaneous coronary interventions or surgical revascularization before vascular surgical procedures impacts mortality or incidence of myocardial infarctions. In fact, coronary angiography is associated with its own inherent risks, and patients undergoing coronary artery bypass grafting or coronary percutaneous transluminal angioplasty (PTA) before needed aortoiliac reconstructions are subjected to the risks and complications of both procedures.

The Coronary Artery Revascularization Prophylaxis trial showed that coronary revascularization in patients with peripheral vascular disease and significant CAD, who are considered high risk for perioperative complications, did not reduce overall mortality or perioperative myocardial infarction. Additionally, patients who underwent prophylactic coronary revascularization had significant delays before undergoing their vascular procedure and increased limb morbidity compared to patients who did not. Studies do support improvement in cardiovascular and overall prognosis with medical optimization of patients. Therefore, use of perioperative beta blockade, as well as use of antiplatelet medication, statins,

and angiotensin-converting enzyme (ACE) inhibitors is encouraged in vascular patients

AORTOILIAC OCCLUSIVE DISEASE

The distal abdominal aorta and the iliac arteries are common sites affected by atherosclerosis. The symptoms and natural history of the atherosclerotic process affecting the aortoiliac arterial segment are influenced by the disease distribution and extent. Atherosclerotic plaques may cause clinical symptoms by restricting blood flow due to luminal obstruction or by embolizing atherosclerotic debris to the LE circulation. If the aortoiliac plaques reach sufficient mass that impinge on the arterial lumen, obstruction of blood flow to lower extremities occurs. Various risk factors exist that can lead to the development of aortoiliac occlusive disease. Recognition of these factors and understanding this disease entity will enable physicians to prescribe the appropriate treatment strategy that may alleviate symptoms and improve quality of life.

Diagnostic Evaluation

On clinical examination, patients often have weakened femoral pulses and a reduced ABI. Verification of iliac occlusive disease is usually made by color duplex scanning that reveals either a peak systolic velocity ratio of 2.5 or greater at the site of stenosis and/or a monophasic waveform. Noninvasive tests such as pulse volume recording (PVR) of the LE with estimation of the thigh-brachial pressure index may be suggestive of aortoiliac disease. MRA and multidetector CTA are increasingly being used to determine the extent and type of obstruction. DSA offers the interventionalist the benefit of making a diagnosis and the option of performing an endovascular treatment in a single session. Angiography provides important information regarding distal arterial runoff vessels as well as the patency of the profunda femoris artery (PFA). Presence of pelvic and groin collaterals is important in providing crucial collateral flow in maintaining lower limb viability. It must be emphasized, however, that patients should be subjected to angiography only if their symptoms warrant surgical intervention.

Differential Diagnosis

Degenerative hip or spine disease, lumbar disc herniation, spinal stenosis, diabetic neuropathy, and other neuromuscular problems can produce symptoms that may be mistaken

for vascular claudication. Such cases can be distinguished from true claudication by the fact that the discomfort from neuromuscular problems often is relieved by sitting or lying down, as opposed to cessation of ambulation. In addition, complaints that are experienced upon standing suggest nonvascular causes. When confusion persists, the use of noninvasive vascular laboratory testing modalities, including treadmill exercise, can help establish the diagnosis.

Collateral Arterial Network

The principal collateral pathways in severe aortoiliac artery occlusive disease or chronic aortic occlusion that may provide blood flow distal to the aortoiliac lesion include: (a) the SMA to the distal IMA via its superior hemorrhoidal branch to the middle and inferior hemorrhoidals to the internal iliac artery (39%); (b) the lumbar arteries to the superior gluteal artery to the internal iliac system (37%); (c) the lumbar arteries to the lateral and deep circumflex arteries to the common femoral artery (CFA) (12%); and (d) Winslow's pathway from the subclavian to the superior epigastric artery to the inferior epigastric artery to the external iliac arteries at the groin (Fig. 7). In general, treatment indications for aortoiliac artery occlusive disease include disabling claudication, ischemic rest pain, nonhealing LE tissue wound, and LE microembolization that arise from aortoiliac lesions.



Fig. 7. Pertinent collateral pathways are developed in the event of chronic severe aortoiliac occlusive disease. As illustrated in this multidetector computed tomography angiography, these collaterals include epigastric arteries (*large white arrows*), an enlarged inferior mesenteric artery (*white arrowhead*), and enlarged lumbar arteries (*black arrows*).

Disease Classification

Based on the atherosclerotic disease pattern, aortoiliac occlusive disease can be classified into three various types (Fig. 8). Type I aortoiliac disease, which occurs in 5 to 10% of patients, is confined to the distal abdominal aorta and common iliac vessels (Fig. 9). Due to the localized nature of this type of aortic obstruction and formation of collateral blood flow around the occluded segment, limb-threatening symptoms are rare in the absence of more distal disease (Fig. 10). This type of aortoiliac occlusive disease occurs in a relatively younger group of patients (aged in their mid-50s), compared with patients who have more femoropopliteal disease. Patients with a type I disease pattern have a lower incidence of hypertension and diabetes, but a significant frequency of abnormal blood lipid levels, particularly type IV hyperlipoproteinemia. Symptoms typically consist of bilateral thigh or buttock claudication and fatigue. Men report diminished penile tumescence and may have complete loss of erectile function. These symptoms in the absence of femoral pulses constitute Leriche's syndrome. Rest pain is unusual with isolated aortoiliac disease unless distal disease coexists. Occasionally patients report a prolonged history of thigh and buttock claudication that recently has become more severe. It is likely that this group has underlying aortoiliac disease that has progressed to acute occlusion of the terminal aorta. Others may present with "trash foot" that represents microembolization into the distal vascular bed (Fig. 11).

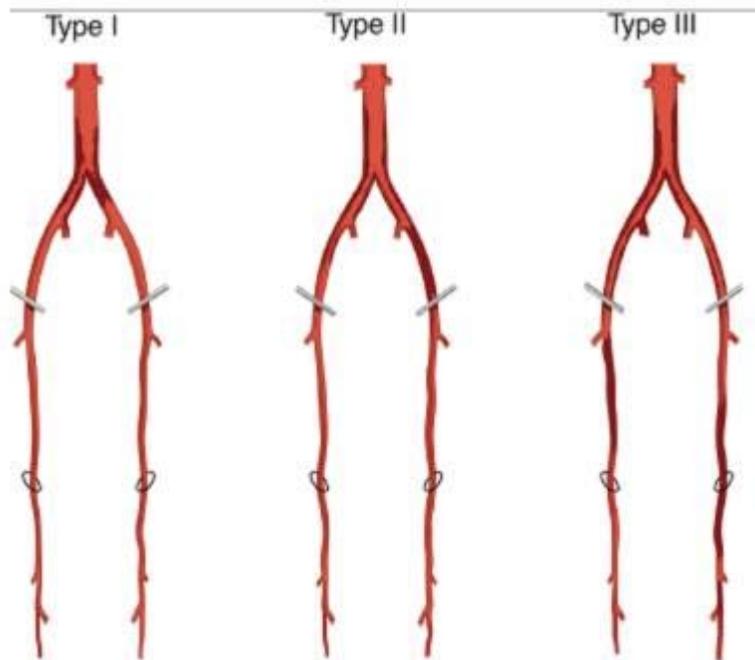


Fig. 8. Aortoiliac disease can be classified into three types. Type I represents focal disease affecting the distal aorta and proximal common iliac artery. Type II represents diffuse aortoiliac disease above the inguinal ligament. Type III represents multisegment occlusive disease involving aortoiliac and infrainguinal arterial vessels.



Fig. 9. Type I aortoiliac disease is confined to the distal abdominal aorta (*long arrow*) or proximal common iliac arteries. Due to the localized nature of this type of aortic obstruction and formation of collateral blood flow around the occluded segment (*short arrows*), limb-threatening symptoms are rare in the absence of more distal disease.



Fig. 10. Multidetector computed tomography angiography of the aortoiliac artery circulation in a 63-year-old man with buttock claudication. Three-dimensional image reconstruction showing intra-arterial calcification of the aorta (*large arrows*) and right common iliac artery (*small arrows*). This is consistent with a type I aortoiliac occlusive disease.



Fig. 11. Atherosclerotic disease involving the aortoiliac segment can result in microembolization of the lower leg circulation, resulting in trash foot or digital gangrene of toes.

Type II aortoiliac disease represents a more diffuse atherosclerotic progression that involves predominately the abdominal aorta with disease extension into the CIA. This disease pattern affects approximately 25% of patients with aortoiliac occlusive disease. Type III aortoiliac occlusive disease, which affects approximately 65% of patients with aortoiliac occlusive disease, is widespread disease that is seen above and below the inguinal ligament (Fig. 12). Patients with "multilevel" disease are older, more commonly male (with a male-to-female ratio of 6:1), and much more likely to have diabetes, hypertension, and associated atherosclerotic disease involving cerebral, coronary, and visceral arteries. Progression of the occlusive process is more likely in these patients than in those with localized aortoiliac disease. For these reasons, most patients with a type III pattern tend to present with symptoms of advanced ischemia and require revascularization for limb salvage rather than for claudication. These patients have a decreased 10-year life expectancy when compared to patients with localized aortoiliac disease.



Fig. 12. Type III aortoiliac occlusive disease is a multilevel disease pattern that affects the aortoiliac segment as well as infrainguinal femoropopliteal vessels. Most patients with this disease pattern tend to present with symptoms of advanced ischemia and require revascularization for limb salvage rather than for claudication.

The most commonly used classification system of iliac lesions has been set forth by the TASC II group with recommended treatment options. This lesion classification categorizes the extent of atherosclerosis and has suggested a therapeutic approach based on this classification (Table 3 and Fig. 13). According to this consensus document, endovascular therapy is the

treatment of choice for type A lesions, and surgery is the treatment of choice for type D lesions. Endovascular treatment is the preferred treatment for type B lesions, and surgery is the preferred treatment for good-risk patients with type C lesions. In comparison to the 2000 TASC II document, the commission has not only made allowances for treatment of more extensive lesions, but also took into account the continuing evolution of endovascular technology and the skills of individual interventionalists when stating that the patient's comorbidities, fully informed patient preference, and the local operator's long-term success rates must be considered when making treatment decisions for type B and type C lesions

Table 3. TASC II Classification of Aortoiliac Occlusive Lesions

Type A lesions
• Unilateral or bilateral stenoses of CIA
• Unilateral or bilateral single short (≤ 3 cm) stenosis of EIA
Type B lesions
• Short (≤ 3 cm) stenosis of infrarenal aorta
• Unilateral CIA occlusion
• Single or multiple stenosis totaling 3–10 cm involving the EIA not extending into the CFA
• Unilateral EIA occlusion not involving the origins of internal iliac or CFA
Type C lesions
• Bilateral CIA occlusions
• Bilateral EIA stenoses 3–10 cm long not extending into the CFA
• Unilateral EIA stenosis extending into the CFA
• Unilateral EIA occlusion that involves the origins of internal iliac and/or CFA
• Heavily calcified unilateral EIA occlusion with or without involvement of origins of internal iliac and/or CFA
Type D lesions
• Infrarenal aortoiliac occlusion
• Diffuse disease involving the aorta and both iliac arteries requiring treatment
• Diffuse multiple stenoses involving the unilateral CIA, EIA, and CFA
• Unilateral occlusions of both CIA and EIA
• Bilateral occlusions of EIA
• Iliac stenoses in patients with AAA requiring treatment and not amenable to endograft placement or other lesions requiring open aortic or iliac surgery

AAA = abdominal aortic aneurysm; CFA = common femoral artery; CIA = common iliac artery; EIA = external iliac artery; TASC II = Trans-Atlantic Inter-Society Consensus.

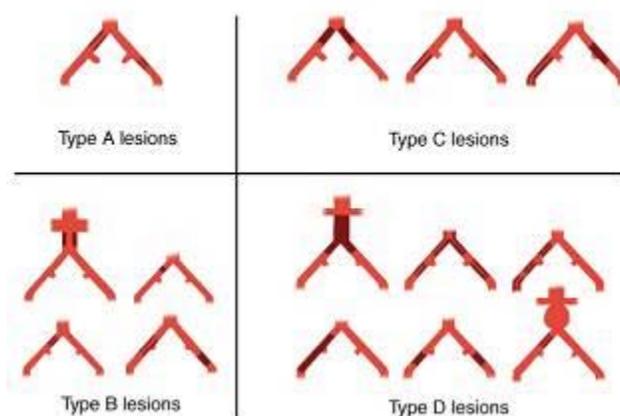


Fig. 13. Schematic depiction of Trans-Atlantic Inter-Society Consensus classification of aortoiliac occlusive lesions

General Treatment Considerations

There is no effective medical therapy for the management of aortoiliac disease, but control of risk factors may help slow progression of atherosclerosis. Patients should have hypertension, hyperlipidemia, and diabetes mellitus controlled. They should be advised to stop smoking. Most patients are empirically placed on antiplatelet therapy. A graduated exercise program may improve walking efficiency, endothelial function, and metabolic adaptations in skeletal muscle, but, there is usually minimal improvement in patients with aortoiliac disease who are treated with these measures. Failure to respond to exercise and/or drug therapy should prompt consideration for limb revascularization. Patients with buttock claudication and reduced or absent femoral pulses who fail to respond to exercise and drug therapy should be considered for revascularization because they are less likely than patients with more distal lesions to improve without concomitant surgical or endovascular intervention.

Surgical Reconstruction of Aortoiliac Occlusive Disease

Aortobifemoral Bypass

Surgical options for treatment of aortoiliac occlusive diseases consist of various configurations of aortobifemoral bypass (ABF) grafting, various types of extra-anatomic bypass grafts, and aortoiliac endarterectomy. The procedure performed is determined by several factors, including anatomic distribution of the disease, clinical condition of the patient, and personal preference of the surgeon.

In most cases, ABF is performed because patients usually have disease in both iliac systems. Although one side may be more severely affected than the other, progression does occur, and bilateral bypass does not complicate the procedure or add to the physiologic stress of the operation. ABF reliably relieves symptoms, has excellent long-term patency (approximately 70 to 75% at 10 years), and can be completed with a tolerable perioperative mortality (2 to 3%).

Technical Considerations for Aortobifemoral Bypass

Both femoral arteries are initially exposed to ensure that they are adequate for the distal

anastomoses. The abdomen is then opened in the midline, the small intestine is retracted to the right, and the posterior peritoneum overlying the aorta is incised. A retroperitoneal approach may be selected as an alternative in certain situations. This approach involves making a left flank incision and displacing the peritoneum and its contents to the right. Such an approach is contraindicated if the right renal artery is acutely occluded, because visualization from the left flank is very poor. Tunneling of a graft to the right femoral artery also is more difficult from a retroperitoneal approach, but can be achieved. The retroperitoneal approach has been reputed to be better tolerated than midline laparotomy for patients with multiple previous abdominal operations and with severe pulmonary disease. Further proposed advantages of the retroperitoneal approach include less GI disturbance, decreased third-space fluid losses, and ease with which the pararenal aorta can be accessed. There are randomized reports, however, that support and refute the superiority of this approach. A collagen-impregnated, knitted Dacron graft is used to perform the proximal aortic anastomosis, which can then be made in either an end-to-end or end-to-side fashion using 3-0 polypropylene suture. The proximal anastomosis should be made as close as possible to the renal arteries to decrease the incidence of restenosis from progression of the atherosclerotic occlusive process in the future.

An end-to-end proximal aortic anastomosis is necessary in those patients with an aortic aneurysm or complete aortic occlusion extending up to the renal arteries (Fig. 14). Although in theory, the end-to-end configuration allows for less turbulence and less chance of competitive flow with still patent host iliac vessels, there have not been consistent results to substantiate differences in patency between end-to-end and end-to-side grafts. Relative indications for an end-to-side proximal aortic anastomosis include the presence of large aberrant renal arteries, an unusually large IMA with poor back-bleeding, suggesting inadequate collateralization, and/or occlusive disease involving bilateral external iliac arteries. Under such circumstances, end-to-end bypass from the proximal aorta to the femoral level devascularizes the pelvic region because there is no antegrade or retrograde flow in the occluded external iliac arteries to supply the hypogastric arteries. As a result of the pelvic devascularization, there is an increased incidence of impotence, postoperative colon ischemia, buttock ischemia, and paraplegia secondary to spinal cord ischemia, despite the presence of excellent femoral and distal pulses.

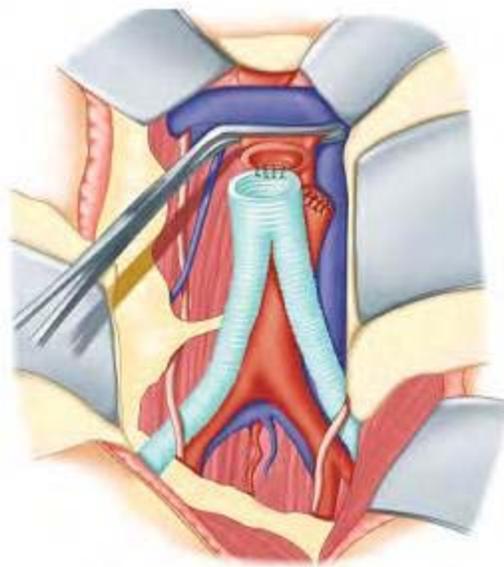


Fig. 14. In an end-to-end proximal aortic anastomosis, the aorta is divided in half. The proximal end of the aorta is anastomosed to the end of a prosthetic graft while the distal divided aortic stump is oversewn.

An end-to-side proximal aortic anastomosis can be associated with certain disadvantages, which include the potential for distal embolization when applying a partially occlusive aortic clamp (Fig. 15). Furthermore, the distal aorta often proceeds to total occlusion after an end-to-side anastomosis. There may also be a higher incidence of aortoenteric fistula following construction of end-to-side proximal anastomoses because the anterior projection makes subsequent tissue coverage and reperitonealization of the graft more difficult. The limbs of the graft are tunneled through the retroperitoneum to the groin, where an end-to-side anastomosis is fashioned between the graft and the bifurcation of the CFA using 5-0 polypropylene suture. Endarterectomy or patch angioplasty of the profunda femoris may be required concurrently. Once the anastomoses have been fashioned and the graft thoroughly flushed, the clamps are removed and the surgeon carefully controls the degree of aortic occlusion until full flow is re-established. During this period the patient must be carefully monitored for hypotension. Declamping hypotension is a complication of sudden restoration of aortic flow, particularly following prolonged occlusion. Once flow has been re-established, the peritoneum is carefully reapproximated over the prosthesis to prevent fistulization into the intestine.



Fig. 15. In an end-to-side aortic anastomosis, the end of a prosthetic graft is connected to the side of an aortic incision

Despite the presence of multilevel disease in most patients, a properly performed aortobifemoral operation can provide arterial inflow and alleviate claudication symptoms in 70 to 80% of patients; however, 10 to 15% of patients will require simultaneous outflow reconstruction to address distal ischemia and facilitate limb salvage. The advantage of concomitant distal revascularization is avoidance of reoperation in a scarred groin. As a rule, if the profunda femoris can accept a 4-mm probe and if a No. 3 Fogarty embolectomy catheter can be passed distally for 20 cm or more, the PFA will be sufficient for outflow and concomitant distal revascularization is not necessary.

Aortic Endarterectomy

Aortoiliac endarterectomy is rarely performed, as it is associated with greater blood loss, greater sexual dysfunction, and is more difficult to perform. Long-term patency is comparable with aortobifemoral grafting, and thus it remains a reasonable option in cases in which the risk of infection of a graft is excessive, because it involves no prosthetic tissue. Aortoiliac endarterectomy was useful when disease was localized to either the aorta or CIAs; however, at present, aortoiliac PTA, stents, and other catheter-based therapies have become first-line treatments in this scenario. Endarterectomy should not be performed if the aorta is aneurysmal because of continued aneurysmal degeneration of the endarterectomized segment. If there is total occlusion of the aorta to the level of the renal arteries, aortic transection several centimeters below the renal arteries with thrombectomy of the aortic cuff followed by graft insertion is easier and more expeditious when compared to endarterectomy. Involvement of the EIA makes aortic endarterectomy more difficult to complete because of decreased vessel

diameter, increased length, and exposure issues. The ability to establish an appropriate endarterectomy plane is compromised due to the muscular and inherently adherent nature of the media in this location. There is a higher incidence of early thrombosis and late failure with extended aortoiliofemoral endarterectomy when compared to bypass grafting as a result of recurrent stenosis.

Axillofemoral Bypass

An axillofemoral bypass is an extra-anatomic reconstruction that derives arterial inflow from the axillary artery to the femoral artery. This is a treatment option for those patients with medical comorbidities that prohibit an abdominal vascular reconstruction. It may be performed under local anesthesia and is used for limb salvage. Extra-anatomic bypasses have lower patency when compared to aortobifemoral, and therefore, are seldom recommended for claudication. Before performing this operation, the surgeon should check pulses and BP in both arms to ensure that there is no obvious disease affecting flow through the axillary system. Angiography of the axillosubclavian vasculature is not necessary, but can be helpful if performed at the time of aortography. The axillary artery is exposed below the clavicle, and a 6- to 8-mm externally reinforced PTFE graft is tunneled subcutaneously down the lateral chest wall and lateral abdomen to the groin. It is anastomosed to the ipsilateral distal at the CFA bifurcation into the superficial femoral and profunda femoris arteries. A femorofemoral crossover graft using a 6- to 8-mm externally reinforced PTFE graft is then used to revascularize the opposite extremity if necessary. Reported patency rates over 5 years vary from 30 to 80%. Paradoxically, although it is a less complex procedure than aortofemoral grafting, the mortality rate is higher (10%), reflecting the compromised medical status of these patients.

Iliofemoral Bypass

One option for patients with unilateral occlusion of the distal common iliac or external iliac arteries is iliofemoral grafting (Fig. 16). Long-term patency is comparable to aortounifemoral bypass and because the procedure can be performed using a retroperitoneal approach without clamping the aorta, the perioperative mortality is less.

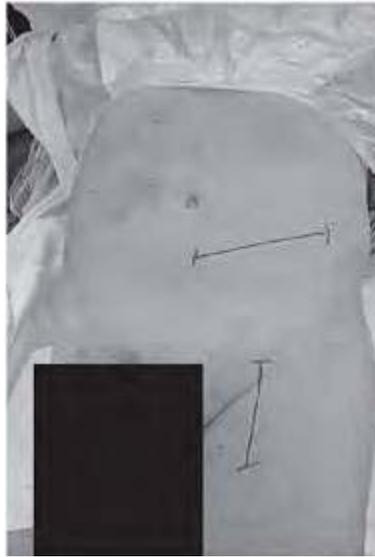


Fig. 16 A. Skin markings showing the incisions of an iliofemoral bypass



Fig. 16 B. A prosthetic bypass graft is used for an iliofemoral artery bypass in which the proximal anastomosis is connected to the common iliac artery (*long arrow*) while the distal anastomosis is connected to the common femoral artery (*short arrow*).

Femorofemoral Bypass

A femorofemoral bypass is another option for patients with unilateral stenosis or occlusion of the common or EIA who have rest pain, tissue loss, or intractable claudication. The primary (assisted) patency at 5 years is reported to be 60 to 70%, and, although this is inferior when compared to aortofemoral bypass, there are physiologic benefits, especially for

patients with multiple comorbidities, because it is not necessary to cross-clamp the aorta. There are no studies supporting the superiority of unsupported or externally supported PTFE over Dacron for choice of conduit. The fear of the recipient extremity stealing blood from the extremity ipsilateral to the donor limb is not realized unless the donor iliac artery and donor outflow arteries are diseased. Depending on the skills of the interventionalist/surgeon, many iliac lesions classified as TASC II B, C, or D can now be addressed using an endovascular approach, thus obviating the need to perform a femorofemoral bypass. Additionally, femorofemoral bypass can be used as an adjuvant procedure after iliac inflow has been optimized with endovascular methods.

Obturator Bypass

An obturator bypass is used to reconstruct arterial anatomy in patients with groin sepsis resulting from prior prosthetic grafting, intra-arterial drug abuse, groin neoplasm, or damage from prior groin irradiation. This bypass can originate from the CIA, EIA, or uninvolved limb of an ABF. A conduit of Dacron, PTFE, or autologous vein is tunneled through the anteromedial portion of the obturator membrane to the distal superficial femoral or popliteal artery. The obturator membrane must be divided sharply so as to avoid injury to adjacent structures, and care must be taken to identify the obturator artery and nerve that pass posterolaterally. After the bypass is completed and the wounds isolated, the infected area is entered, the involved arteries are débrided to healthy tissue, and vascularized muscle flaps are mobilized to cover the ligated ends. There have been varied results in terms of patency and limb salvage for obturator bypass. Some authors have reported 57% 5-year patency and 77% 5-year limb salvage rates, whereas others have shown a high rate of reinfection and low patency requiring reintervention.

Thoracofemoral Bypass

The indications for thoracofemoral bypass are (a) multiple prior surgeries with a failed infrarenal aortic reconstruction and (b) infected aortic prosthesis. This procedure is more physiologically demanding than other extra-anatomic reconstructions because the patient must not only tolerate clamping the descending thoracic aorta but also performance of a left thoracotomy. The graft is tunneled to the left CFA from the left thorax posterior to the left kidney in the anterior axillary line using a small incision in the periphery of the diaphragm and

an incision in the left inguinal ligament to gain access to the extraperitoneal space from below. The right limb is tunneled in the space of Retzius in an attempt to decrease kinking that is more likely to occur with subcutaneous, suprapubic tunneling. Thoracofemoral bypass has long-term patency comparable to aortofemoral bypass.

Complications of Surgical Aortoiliac Reconstruction

With current surgical techniques and conduits, early postoperative hemorrhage is unusual and occurs in 1 to 2% of cases. It is usually the result of technical oversight or coagulation abnormality. Acute limb ischemia (ALI) occurring after aortoiliac surgery may be the result of acute thrombosis or distal thromboembolism. The surgeon can prevent thromboembolic events by (a) avoiding excessive manipulation of the aorta, (b) ensuring adequate systemic heparinization, (c) judicious placement of vascular clamps, and (d) thorough flushing before restoring blood flow. Acute thrombosis of an aortofemoral graft limb in the early perioperative period occurs in 1 to 3% of patients.¹¹² Thrombectomy of the graft limb is performed through a transverse opening in the hood of the graft at the femoral anastomosis. With this approach, it is possible to inspect the interior of the anastomosis and pass embolectomy catheters distally to clear the superficial femoral and profunda arteries. Various complications may be encountered following aortoiliac or aortobifemoral reconstruction (Table 4).

Table 4. Perioperative Complications of Aortobifemoral Bypass Grafting

Medical complications
• Perioperative myocardial infarction
• Respiratory failure
• Ischemia-induced renal failure
• Bleeding from IV heparinization
• Stroke
Procedure-related complications
Early
• Declamping shock
• Graft thrombosis
• Retroperitoneal bleeding
• Groin hematoma
• Bowel ischemia/infarction
• Peripheral embolization
• Erectile dysfunction
• Lymphatic leak
• Chylous ascites
• Paraplegia
Late
• Graft infection
• Anastomotic pseudoaneurysm
• Aortoenteric fistula
• Aortourinary fistula
• Graft thrombosis

Intestinal ischemia following aortic reconstruction occurs in approximately 2% of cases; however, with colonoscopy, mucosal ischemia, which is a milder form, is seen more frequently. The surgeon can identify patients who require concomitant revascularization of the IMA, hypogastric arteries, or mesenteric arteries by examining the preoperative arteriogram for the presence of associated occlusive lesions in the celiac axis, the superior mesenteric arteries, or both. Likewise patients with patent and enlarged IMA or a history of prior colonic resections will benefit from IMA reimplantation.

In a comprehensive review of 747 patients who had aortoiliac operations for occlusive disease, secondary operations for late complications such as reocclusion, pseudoaneurysms, and infection were necessary in 21% of cases over a 22-year period. The most frequent late complication is graft thrombosis. Limb occlusion occurs in 5 to 10% of patients within 5 years of the index operation and in 15 to 30% of patients 10 years or more after the index operation. Anastomotic pseudoaneurysms occur in 1 to 5% of femoral anastomoses in patients with aortofemoral grafts. Predisposing factors to pseudoaneurysm formation include progression of degenerative changes within the host artery, excessive tension at the anastomosis, and infection. Due to the associated risks of thrombosis, distal embolization, infection, and rupture, anastomotic aneurysms should be repaired expeditiously.

Infection following aortoiliac reconstruction is a devastating complication that occurs in 1% of cases. Femoral anastomoses of aortofemoral reconstructions and axillofemoral bypasses are prone to infection. Use of prophylactic antibiotics and meticulous surgical technique are vital in preventing contamination of the graft at the time of implantation. If infection appears to be localized to a single groin, one may consider the treatment strategies of graft preservation, aggressive local wound débridement, antibiotic solution irrigation, and soft tissue coverage with rotational muscle flaps. This nonexcisional treatment approach may be useful in selective cases of localized femoral graft infection. Most patients with infected aortoiliofemoral reconstructions usually require graft excision and revascularization via remote uncontaminated routes or the use of in situ replacement to clear the infective process and maintain limb viability. Aortoenteric fistula and associated GI hemorrhage are devastating complications, with a 50% incidence of death or limb loss. The incidence of aortoenteric fistula formation appears to be higher after an end-to-side proximal anastomosis, because it is more difficult to cover the prosthesis with viable tissue and avoid contact with the GI tract with this configuration. Treatment of aortoenteric fistula requires resection of all prosthetic material, closure of the infrarenal abdominal aorta, repair of the GI tract, and revascularization by means of an extra-anatomic graft.

LOWER EXTREMITY ARTERIAL OCCLUSIVE DISEASE

The symptoms of LE occlusive disease are classified into two large categories: ALI and chronic limb ischemia (CLI). Ninety percent (90%) of acute ischemias are either thrombotic or embolic. Frequently, sudden onset of limb-threatening ischemia may be the result of acute exacerbation of the pre-existing atherosclerotic disease. Chronic ischemia is largely due to atherosclerotic changes of the LE that manifest from asymptomatic to limb-threatening gangrene. As the population ages, the prevalence of chronic occlusive disease of the LE is increasing and it significantly influences lifestyle, morbidity, and mortality. In addition, multiple comorbid conditions increase risks of surgical procedures. Endovascular interventions become an important alternative in treating LE occlusive disease. However, despite rapid evolving endovascular technology, LE endovascular intervention continues to be one of the most controversial areas of endovascular therapy.

Epidemiology

In a detailed review of the literature, McDaniel and Cronenwett concluded that claudication occurred in 1.8% of patients under 60 years of age, 3.7% of patients between 60 and 70 years of age, and 5.2% of patients over 70 years of age. Leng and his colleagues scanned 784 subjects using ultrasound in a random sample of men and women ages 56 to 77 years. Of the subjects that were scanned, 64% demonstrated atherosclerotic plaque. However, a large number of patients had occlusive disease without significant symptoms. In a study by Schroll and Munck, only 19% of the patients with peripheral vascular disease were symptomatic. Using ankle-brachial indices (ABIs), Stoffers and colleagues scanned 3171 individuals between the ages of 45 and 75 and identified 6.9 % of patients who had ABIs <0.95 , only 22% of whom had symptoms. In addition, they demonstrate that the concomitant cardiovascular and cerebrovascular diseases were three to four times higher among the group with asymptomatic peripheral vascular diseases than those without peripheral vascular disease. Furthermore, they confirm that 68% of all peripheral arterial obstructive diseases were unknown to the primary care physician and this group mainly represented less advanced cases of atherosclerosis. However, among patients with an ABI ratio <0.75 , 42% were unknown to the primary physicians.

Diagnostic Evaluation

The diagnosis of LE occlusive disease often is made based upon a focused history and physical examination, and confirmed by the imaging studies. A well-performed physical examination often reveals the site of lesions by detecting changes in pulses, temperature, and appearances. The bedside ABIs using BP cuff also aid in diagnosis. Various clinical signs and symptoms are useful to differentiate conditions of viable, threatened, and irreversible limb ischemia caused by arterial insufficiency.

Noninvasive studies are important in documenting the severity of occlusive disease objectively. Ultrasound Dopplers measuring ABIs and segmental pressures are widely used in North America and Europe. Normal ABI is >1.0 . In patients with claudication, ABIs decrease to 0.5 to 0.9 and to even lower levels in patients with rest pain or tissue loss. Segmental pressures are helpful in identifying the level of involvement. Decrease in segmental pressure between two segments indicates significant disease. Ultrasound duplex scans are used to identify the site of lesion by revealing flow disturbance and velocity changes. A meta-analysis of 71 studies by Koelemay and associates confirmed that duplex scanning is accurate for assessing arterial occlusive disease in patients suffering from claudication or critical ischemia,

with an accumulative sensitivity of 80% and specificity of over 95%. Adding an ultrasound contrast agent further increases sensitivity and specificity to ultrasound technology. Other noninvasive imaging technologies such as MRA and CTA are rapidly evolving and gaining popularity in the diagnosis of LE occlusive disease (Figs. 17 and 18).



Fig. 17. A high-resolution computed tomography angiography of a patient with normal right lower extremity arterial circulation. Distal occlusive disease is noted in the left tibial arteries (*arrow*).

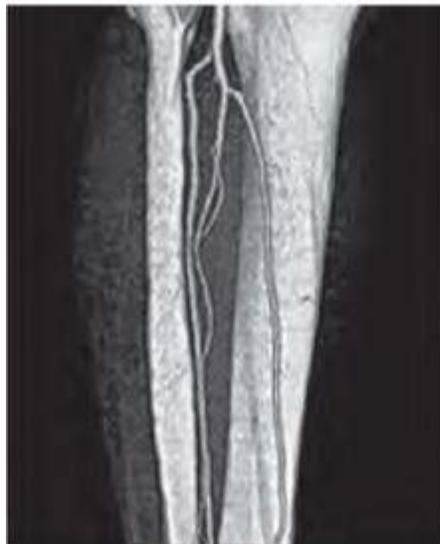


Fig 18 A. A multidetector computed tomographic angiography of a patient with an infrapopliteal arterial circulation



Fig 18 B. Pedal arterial circulation. The high spatial resolution and image quality of these images show three patent infrapopliteal runoff vessels and patent pedal vessels at the foot level

Contrast angiography remains the gold standard in imaging study. Using contrast angiography, interventionists can locate and size the anatomic significant lesions and measure the pressure gradient across the lesion, as well as plan for potential intervention. Angiography is, however, semi-invasive and should be confined to patients for whom surgical or percutaneous intervention is contemplated. Patients with borderline renal function may need to have alternate contrast agents such as gadolinium or carbon dioxide to avoid contrast-induced nephrotoxicity.

Differential Diagnosis

Arterial insufficiency frequently leads to muscle ischemic pain involving the LE muscularly, particularly during exercise. Intermittent claudication is pain affecting the calf, and, less commonly, the thigh and buttock, which is induced by exercise and relieved by rest. Symptom severity varies from mild to severe. Intermittent claudication occurs as a result of muscle ischemia during exercise caused by obstruction to arterial flow. For differential diagnosis of intermittent claudication, there are a variety of neurologic, musculoskeletal, and venous conditions that may produce symptoms of calf pain (Table 5). Additionally, various non-atherosclerotic conditions also can cause symptoms consistent with intermittent LE claudication (Table 6). Nocturnal calf muscle spasms or night cramps are not indicative of arterial disease. They are common but are difficult to diagnose with certainty. Foot ulceration is

not always the result of arterial insufficiency. Ischemic ulcers occur on the toes or lateral side of the foot and are painful. By comparison, venous ulcers, which also are common, occur above the medial malleolus. These venous stasis ulcers are typically surrounded by a peripheral area of darkened skin discoloration that is also known as *lipodermatosclerosis*. Neuropathic ulcers usually are found on weight-bearing surfaces, have thick calluses, and are pain free. Ulcers may be the result of more than one etiology. Rest pain must be distinguished from peripheral neuropathy, which is prevalent in diabetic patients. Patients with diabetic neuropathy tend to have decreased vibration and position sense and decreased reflexes. Spinal stenosis causes pain that is exacerbated with standing and back extension.

Table 5 Differential Diagnosis of Intermittent Claudication

Condition	Location of Pain or Discomfort	Characteristic Discomfort	Onset Relative to Exercise	Effect of Rest	Effect of Body Position	Other Characteristics
Intermittent claudication (calf)	Calf muscles	Cramping pain	After same degree of exercise	Quickly relieved	None	Reproducible
Chronic compartment syndrome	Calf muscles	Tight, bursting pain	After much exercise (e.g., jogging)	Subsides very slowly	Relief speeded by elevation	Typically heavy-muscled athletes
Venous claudication	Entire leg, but usually worse in thigh and groin	Tight, bursting pain	After walking	Subsides slowly	Relief speeded by elevation	History of iliofemoral deep venous thrombosis, signs of venous congestion, edema
Nerve root compression (e.g., herniated disk)	Radiates down leg, usually posteriorly	Sharp lancinating pain	Soon, if not immediately after onset	Not quickly relieved (also often present at rest)	Relief may be aided by adjusting back position	History of back problems
Symptomatic Baker's cyst	Behind knee, down calf	Swelling, soreness, tenderness	With exercise	Present at rest	None	Not intermittent
Intermittent claudication (hip, thigh, buttock)	Hip, thigh, buttocks	Aching discomfort, weakness	After same degree of exercise	Quickly relieved	None	Reproducible
Hip arthritis	Hip, thigh, buttocks	Aching discomfort	After variable degree of exercise	Not quickly relieved (and may be present at rest)	More comfortable sitting, weight taken off legs	Variable, may relate to activity level, weather changes
Spinal cord compression	Hip, thigh, buttocks (follows dermatome)	Weakness more than pain	After walking or standing for same length of time	Relieved by stopping only if position changed	Relief by lumbar spine flexion (sitting or stooping forward) pressure	Frequent history of back problems, provoked by increased intra-abdominal pressure
Intermittent claudication (foot)	Foot, arch	Severe deep pain and numbness	After same degree of exercise	Quickly relieved	None	Reproducible
Arthritic, inflammatory process	Foot, arch	Aching pain	After variable degree of exercise	Not quickly relieved (and may be present at rest)	May be relieved by not bearing weight	Variable, may relate to activity level

Table 6. Non-Atherosclerotic Causes of Intermittent Claudication

• Aortic coarctation
• Arterial fibrodysplasia
• Iliac syndrome of the cyclist
• Peripheral emboli
• Persistent sciatic artery
• Popliteal aneurysm
• Popliteal cyst
• Popliteal entrapment
• Primary vascular tumors
• Pseudoxanthoma elasticum
• Remote trauma or radiation injury
• Takayasu's disease
• Thromboangiitis obliterans

Lower Extremity Occlusive Disease Classification

LE occlusive disease may range from exhibiting no symptoms to limb-threatening gangrene. There are two major classifications based on the clinical presentations.

The Fontaine classification uses four stages: Fontaine I is the stage when patients are asymptomatic; Fontaine II is when they have mild (IIa) or severe (IIb) claudication; Fontaine III is when they have ischemic rest pain; and Fontaine IV is when patients suffer tissue loss such as ulceration or gangrene (Table 7).

Table 7 Classification of Peripheral Arterial Disease Based on the Fontaine and Rutherford Classifications

Fontaine Classification		Rutherford Classification		
Stage	Clinical	Grade	Category	Clinical
I	Asymptomatic	0	0	Asymptomatic
IIa	Mild claudication	I	1	Mild claudication
IIb	Moderate to severe claudication	I	2	Moderate claudication
			3	Severe claudication
III	Ischemic rest pain	II	4	Ischemic rest pain
IV	Ulceration or gangrene	III	5	Minor tissue loss
			6	Major tissue loss

The Rutherford classification has four grades (0–III) and seven categories (0–6). Asymptomatic patients are classified into category 0; claudicants are stratified into grade I and divided into three categories based on the severity of the symptoms; patients with rest pain belong to grade II and category 4; patients with tissue loss are classified into grade III and categories 5 and 6, based on the significance of the tissue loss. These clinical classifications help to establish uniform standards in evaluating and reporting the results of diagnostic

measurements and therapeutic interventions (see Table 7).

The most clinically useful classification of LE atherosclerotic disease should be based on morphologic characters of the lesions. The TASC II task force published a guideline separating LE arterial diseases into femoropopliteal and infrapopliteal lesions (Table 8). This guideline is particularly useful in determining intervention strategies based on the disease classifications. Based on the guideline, femoropopliteal lesions are divided into four types: A, B, C, and D. Type A lesions are single focal lesions <3 cm in length that did not involve the origins of the superficial femoral artery (SFA) or the distal popliteal artery; Type B lesions are single lesions 3 to 5 cm in length not involving the distal popliteal artery or multiple or heavily calcified lesions <3 cm in length; Type C lesions are multiple stenoses or occlusions >15 cm in length, or recurrent stenoses or occlusions that need treatment after two endovascular interventions. Type D lesions were those with complete occlusion of CFA, SFA, or popliteal artery.

Table 8. TASC II Classification of Femoral Popliteal Occlusive Lesions

Type A lesions
• Single stenosis ≤10 cm in length
• Single occlusion ≤5 cm in length
Type B lesions
• Multiple lesions (stenoses or occlusions), each ≤5 cm
• Single stenosis or occlusion ≤15 cm not involving the infra geniculate popliteal artery
• Single or multiple lesions in the absence of continuous tibial vessels to improve inflow for a distal bypass
• Heavily calcified occlusion ≤5 cm in length
• Single popliteal stenosis
Type C lesions
• Multiple stenoses or occlusions totaling >15 cm with or without heavy calcification
• Recurrent stenoses or occlusions that need treatment after two endovascular interventions
Type D lesions
• Chronic total occlusions of CFA or SFA (>20 cm, involving the popliteal artery)
• Chronic total occlusion of popliteal artery and proximal trifurcation vessels

CFA = common femoral artery; SFA = superficial femoral artery; TASC II = Trans-Atlantic Inter-Society Consensus.

In a similar fashion, infrapopliteal arterial diseases are classified into four types based on TASC II guideline (Fig. 19). Type A lesions are single lesions <1 cm in length not involving the trifurcation; Type B lesions are multiple lesions <1 cm in length or single lesions shorter than 1 cm involving the trifurcation; Type C lesions are those lesions extensively involving trifurcation or those that are 1- to 4-cm stenotic or 1- to 2-cm occlusive lesions; Type D lesions are occlusions longer than 2 cm or diffuse diseases.

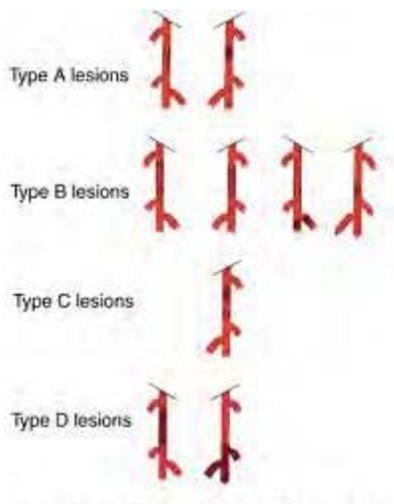


Fig. 19. Schematic depiction of Trans-Atlantic Inter-Society Consensus classification of femoral popliteal occlusive lesions

Clinical Manifestations of Chronic Limb Ischemia

The term *chronic limb ischemia* is reserved for patients with objectively proven arterial occlusive disease and symptoms lasting for more than 2 weeks. Symptoms include rest pain and tissue loss such as ulceration or gangrene (Table 9). The diagnosis should be corroborated with noninvasive diagnostic tests such as the ABI, toe pressures, and transcutaneous oxygen measurements. Ischemic rest pain most commonly occurs below an ankle pressure of 50 mmHg or a toe pressure <30 mmHg.² Ulcers are not always of an ischemic etiology (Table 10). In many instances, there are other etiologic factors (traumatic, venous, or neuropathic) that are contributory, but it is underlying peripheral arterial disease (PAD) that may be responsible for delayed or absent healing (Fig. 20). Healing of ulcers requires an inflammatory response and greater perfusion than is required to support intact skin and underlying tissues. As a result, the ankle and toe pressure levels needed for healing are higher than the pressures seen with ischemic rest pain. For patients with ulcers or gangrene, the presence of CLI is suggested by an ankle pressure <70 mmHg or a toe systolic pressure <50 mmHg.² It is important to understand that there is no definite consensus regarding the vascular hemodynamic parameters required to make the diagnosis of CLI.

Table 9. Clinical Categories of Chronic Limb Ischemia

Grade	Category	Clinical Description	Objective Criteria
0	0	Asymptomatic—no hemodynamically significant occlusive disease	Normal treadmill or reactive hyperemia test
	1	Mild claudication	Able to complete treadmill exercise ^a ; AP after exercise >50 mmHg but at least 20 mmHg lower than resting value
1	2	Moderate claudication	Between categories 1 and 3
	3	Severe claudication	Cannot complete standard treadmill exercise ^a and AP after exercise <50 mmHg
II ^b	4	Ischemic rest pain	Resting AP <40 mmHg, flat or barely pulsatile ankle or metatarsal PVR; TP <30 mmHg
III ^b	5	Minor tissue loss—nonhealing ulcer, focal gangrene with diffuse pedal ischemia	Resting AP <60 mmHg, ankle or metatarsal PVR flat or barely pulsatile; TP <40 mmHg
	6	Major tissue loss—extending above TM level, functional foot no longer salvageable	Same as category 5

Table 10. Symptoms and Signs of Neuropathic Ulcer versus Ischemic Ulcer

Neuropathic Ulcer	Ischemic Ulcer
Painless	Painful
Normal pulses	Absent pulses
Regular margins, typically punched-out appearance	Irregular margin
Often located on plantar surface of foot	Commonly located on toes, glabrous margins
Presence of calluses	Calluses absent or infrequent
Loss of sensation, reflexes, and vibration	Variable sensory findings
Increased in blood flow (atrioventricular shunting)	Decreased in blood flow
	Collapsed veins
Dilated veins	Cold foot
Dry, warm foot	No bony deformities
Bony deformities	Pale and cyanotic in appearance
Red or hyperemic in appearance	



Fig. 20 A A neuropathic ulcer is characterized by a punched-out appearance with loss of sensation in the surrounding skin. The foot may be warm to touch, and pulses may be present in the distal pedal arteries



Fig. 20 B An ischemic ulcer is characterized by a gangrenous skin change in the foot or toes. The foot is usually cold to touch with absent pedal pulses. The foot is painful to touch with decreased distal capillary refills.

One of the most common sites for occlusive disease is in the distal SFA as it passes deep through the adductor canal. It may be that the entrapment by the adductor hiatus prevents the compensatory dilation that occurs in atherosclerotic vessels. Stenoses, which develop here, progress to occlusion of the distal third of the SFA (Fig. 21). When distal SFA occlusion develops slowly, it may be totally asymptomatic because of development of collaterals from the proximal SFA or the profunda femoris artery (PFA) can bypass the occlusion and reconstitute the popliteal artery. Symptom development is a function of the extent of occlusion, adequacy of collaterals, and also the activity level of the patients.



Fig. 21. Computed tomography angiogram of a patient with an occluded left superficial femoral artery (*single long arrow*) with reconstituted superficial femoral artery at the level of midthigh. Diffuse arterial calcifications (*double small arrows*) are noted in the mid and distal left superficial femoral arteries.

Presenting symptoms of femoropopliteal occlusive disease are broadly classified into two types: limb-threatening and non-limb-threatening ischemia. Claudication is non-limb-threatening, while rest pain, ulceration, and gangrene are limb-threatening and warrant urgent intervention. Occlusive disease of the femoral artery may be isolated or occur in conjunction with multilevel disease that involves both the aortoiliac segment and the tibial vessels. Symptoms in patients with multilevel disease are more severe than in those with single-level disease. Pain from isolated SFA and popliteal occlusion typically manifests as calf claudication. Cramping pain develops in the calf on ambulation, occurs at a reproducible distance, and is relieved by rest. Activities such as climbing stairs or going uphill also exacerbate the pain. Many patients report worsening symptoms during cold weather. It is important to evaluate whether the symptoms are progressive or static. In >70% of patients, the disease is stable, particularly with risk factor modification.

Progression of the underlying atherosclerotic process is more likely to occur in patients with diabetes, those who continue to smoke, and those who fail to modify their atherosclerotic risk factors. In comparison, rest pain is constant, and usually occurs in the forefoot across the metatarsophalangeal joint. It is worse at night and requires placing the foot in a dependent position to improve symptoms. Patients may report that they either sleep in a chair or hang the

foot off the side of the bed. The pain is severe and relentless, even with narcotics. Ischemic ulceration most commonly involves the toes. Any toe can be affected. Occasionally, ulcers develop on the dorsum of the foot. Ulceration can occur in atypical positions in an ischemic foot from trauma such as friction from poorly fitting shoes. Injury to a foot with borderline ischemia can convert an otherwise stable situation into one that is limb-threatening. The initial development of gangrene commonly involves the digits. As with all vascular patients, it is important to evaluate their risk factors, intercurrent cardiac diseases, and any prior vascular interventions.

Treatment Considerations for Chronic Limb Ischemia

Patients with vascular diseases frequently have complicated medical comorbidities. Careful patient evaluation and selection should be performed for any peripheral arterial vascular procedure. The fundamental principle is to assess not only the surgical risk from the peripheral arterial system but also the global nature of the atherosclerotic process. Full cardiac evaluations are often necessary due to the high incidence of concomitant atherosclerotic coronary arteries disease, resulting in a high risk for ischemic events. Hertzner and associates reviewed coronary angiographies on 1000 patients undergoing elective vascular procedures and identified 25% of concomitant correctable CAD, including 21% in patients undergoing elective peripheral vascular intervention. Conte and associates analyzed their 20-year experience in a tertiary practice setting in 1642 open LE reconstructive surgeries and concluded that patients requiring LE reconstruction presented an increasingly complex medical and surgical challenge compared with the previous decade. With aging of the population, there are a growing number of vascular patients who have prohibitive medical comorbidities that are deemed high-risk for open surgical repair. Endovascular intervention provides an attractive alternative.

As for open surgical repair, the clinical indications for endovascular intervention of LE PADs include lifestyle-limiting claudication, ischemic rest pain, and tissue loss or gangrene. Importantly, endovascular procedures should be performed by a competent vascular interventionist who understands the vascular disease process and is familiar with a variety of endovascular techniques. In addition, certain lesions such as long segment occlusion, heavily calcified lesion, orifice lesion, or lesions that can be not be traversed by a guidewire may not be amendable to endovascular treatment or may be associated with poor outcomes. A proper selection of patients and techniques is critical in achieving a good long-term outcome.

Endovascular intervention for LE occlusive disease is continuously evolving. Success

and patency rates of endovascular intervention are closely related to the anatomic and morphologic characters of the treated lesions. The TASC II work group made recommendations on the intervention strategies of LE arterial diseases based on the morphologic characters. Based on the TASC II guidelines, endovascular treatment is recommended for type A lesions, open surgery is recommended for Type D lesions, and no recommendations were made for Types B and C lesions. However, with rapid advancement in endovascular technologies, there are increased numbers of lesions amenable to endovascular interventions.

There is less literature support on infrapopliteal endovascular intervention due to higher complication and lower success rates. The treatment is restricted to patients with limb-threatening ischemia who lack surgical alternatives. However, with further advancement of endovascular technology and the development of new devices, endovascular intervention will become an integral part of treatment (Table 11). By itself or combined with open technique, percutaneous intervention plays an important role in therapeutic options for LE occlusive disease. As described by the TASC II guidelines, four criteria should be measured to evaluate the clinical success of the treatment: improvement in walking distance, symptomatic improvement, quality of life, and overall graft patency. These criteria should all be carefully weighed and evaluated for each individual before endovascular therapy.

Table 11. Summary of Endovascular Treatment Strategies Using Device-Based Infrapopliteal Intervention

Intervention	Advantages	Disadvantages
Angioplasty	<ul style="list-style-type: none"> • Easy to use • Broad range of applications 	<ul style="list-style-type: none"> • Failure in long lesions, calcified lesions, and disease at multiple levels
Balloon-expandable stent	<ul style="list-style-type: none"> • Overcomes arterial recoil from angioplasty • Useful in treatment of flow-limiting dissection 	<ul style="list-style-type: none"> • Crushability can lead to restenosis • Poor distal runoff can result in stent thrombosis; limited data
Self-expanding stent	<ul style="list-style-type: none"> • Vessel conformability and wall apposition prevents kinking and crushing of stent 	<ul style="list-style-type: none"> • Limited sizes; limited data; multicenter trials under way
Bioabsorbable stent	<ul style="list-style-type: none"> • Overcomes arterial recoil from angioplasty • Absorbed long-term to prevent risk of stent thrombosis 	<ul style="list-style-type: none"> • Limited data; multicenter trials under way
Cryoplasty	<ul style="list-style-type: none"> • Reduces the risk of flow-limiting dissection, therefore reducing the need for stent implantation 	<ul style="list-style-type: none"> • Short-term results of a multicenter trial are promising; however, long-term data are limited
Cutting balloon	<ul style="list-style-type: none"> • Useful in anastomotic segments of bypass grafts and in-stent restenosis where "watermelon seeding" can prevent adequate expansion of plaque 	<ul style="list-style-type: none"> • Limited data
Mechanical atherectomy	<ul style="list-style-type: none"> • Allows for debulking of plaque without the need for stent implantation in most cases • Allows for removal of plaque for histologic analysis 	<ul style="list-style-type: none"> • Limited use in areas of heavy calcification • No large, randomized, prospective trial comparing this technique to angioplasty and stenting
Laser	<ul style="list-style-type: none"> • Useful in acute thrombotic and chronic total occlusions 	<ul style="list-style-type: none"> • Minimal data in infrapopliteal arteries • Need adjunctive treatment with angioplasty, stenting, or atherectomy

Endovascular Treatment

Technical Considerations

A sterile field is required in either an OR or an angiography suite with image capability. The most common and safest access site is the CFA via either a retrograde or antegrade approach. For diagnostic angiography, arterial access should be contralateral to the symptomatic sides. For therapeutic procedures, location of the lesion and the anatomic structures of the arterial tree determine the puncture site. To avoid puncturing the iliac artery or SFA, the femoral head is located under the fluoroscopy and used as the guide for the level of needle entry. In addition, there are several useful techniques in helping access a pulseless CFA, including puncturing guided by ultrasound, using a micropuncture kit, and targeting calcification in a calcified vessel. An antegrade approach may be challenging, particularly in obese patients. Meticulous technique is crucial in preventing complications, and a bony landmark can be used as guidance to ensure CFA puncture.

Traversing the lesion with a wire is the most critical part of the procedure. Typically, 0.035-in guidewires are used for femoropopliteal lesions and 0.014- or 0.018-in guidewires are used for infrapopliteal access. Hydrophilic-coated wires, such as Glide wires, are useful in navigating through tight stenosis or occlusion. An angled-tip wire with a torque device may be helpful in crossing an eccentric lesion, and a shaped selective catheter is frequently used in helping manipulate the wire across the lesion. The soft and floppy end of the wire is carefully advanced, crossing the lesion under fluoroscopy, and gentle force is applied while manipulating the wire. Once the lesion has been traversed, one needs to pay particular attention to the tip of the wire to ensure a secure wire access and avoid vessel wall perforation or dissection.

Once the access to the diseased vessel is secured and the wire has successfully traversed the lesion, several treatment modalities can be used either used alone or in conjunction with others, including angioplasty, stent or stent graft placement, and atherectomy. The available angioplasty techniques are balloon angioplasty, cryoplasty, subintimal angioplasty (SA), and cutting balloon. The most commonly used atherectomy techniques include percutaneous atherectomy catheter and laser atherectomy device.

Systemic anticoagulation should be maintained routinely during LE arterial interventions to minimize the risk of pericatheter thrombosis. Unfractionated heparin is the most commonly used agent, given on a weight-based formula. It is a common clinical practice to use a 80 to 100 mg/kg initial bolus for a therapeutic procedure to achieve the activated clotting time above 250

seconds on the catheter insertion and subsequently 1000 units for each additional hour of the procedure. Newer agents such as low molecular weight heparin, platelet IIb/IIIa inhibitor, direct thrombin inhibitor, or recombinant hirudin have been available and can be used either alone or in conjunction with heparin, particularly in patients sensitive to unfractionated heparin. After procedures, all patients are placed on antiplatelet therapy such as aspirin. Additional antiplatelet agents such as clopidogrel (Plavix) are given to selected patients with stent placement for at least 6 weeks after LE interventions, unless otherwise contraindicated.

Percutaneous Transluminal Balloon Angioplasty

After the lesion is crossed with a wire, an appropriate balloon angioplasty catheter is selected and tracked along the wire to traverse the lesion. The length of the selected catheter should be slightly longer than the lesion and the diameter should be equal to the adjacent normal vessel. The balloon tends to be approximately 10 to 20% oversized. The radiopaque markers of the balloon catheter are placed so that they will straddle the lesion. Then, the balloon is inflated with saline and contrast mixture to allow visualization of the insufflation process under the fluoroscopy (Fig. 22). The patient may experience mild pain, which is not uncommon. However, severe pain can be indicative of vessel rupture, dissection, or other complications. An angiography is crucial in confirming the intraluminal location of the catheter and absence of contrast extravasation. The inflation is continued until the waist of the atherosclerotic lesion disappears and the balloon is at full profile. Frequently, several inflations are required to achieve full profile of the balloon (Fig. 23). Occasionally, a lower profile balloon is needed to predilate the tight stenosis so that the selected balloon catheter can cross the lesion.

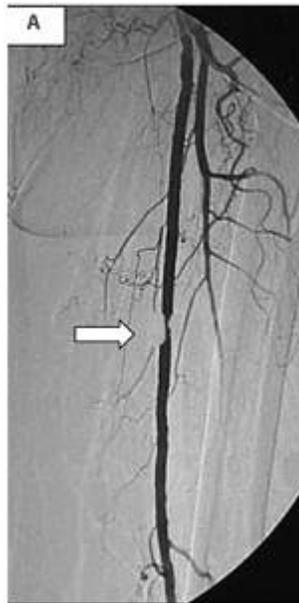


Fig. 22 A. Angiogram demonstrating a focal stenosis in the superficial femoral artery (*arrow*).



Fig. 22 B. This lesion was treated with a balloon angioplasty catheter that inflated a dilating balloon and expanded the flow lumen



Fig. 22 C. Completion angiogram demonstrating satisfactory radiographic result.



Fig. 23 A. Angiogram demonstrating a segmental occlusion in the distal superficial femoral artery (*single arrow*).



Fig. 23 B. This lesion was treated with cryoplasty, which lowered the balloon catheter temperature to a temporary freezing state during the balloon angioplasty procedure (*double arrows*)



Fig.23 C. Completion angiogram demonstrated satisfactory result with no evidence of vessel dissection.

Besides length and diameter, the operators need to be familiar with several balloon characters. Noncompliant and low-compliant balloons tend to be inflated to their preset diameter and offer greater dilating force at the site of stenosis. Low-compliant balloons are the

mainstay for peripheral intervention. A balloon with a low profile is used to minimize complication at the entry site and for crossing the tight lesions. Upon inflation, most balloons do not rewrap to their preinflation diameter and assume a larger profile. Furthermore, trackability, pushability, and crossability of the balloon should be considered when choosing a particular type. Lastly, shoulder length is an important characteristic when performing PTA to avoid injury to the adjacent arterial segments. After PTA, a completion angiogram is performed while the wire is still in place. Leaving the wire in place provides access for repeating the procedure if the result is unsatisfactory.

PTA is an established and effective therapy for select patients with LE occlusive diseases. Studies have shown that PTA of the femoropopliteal segment achieved over a 90% technical success rate and a 38 to 58% 5-year primary patency rate. However, efficacy of PTA is highly dependent upon anatomic selection and patient condition. PTA of lesions longer than 7 to 10 cm offer limited patency, while PTA of shorter lesions, such as those that are <3 cm, have fairly good results. Lofberg and associates performed 127 femoropopliteal PTA procedures and reported a primary 5-year success rate of 12% in limbs with an occlusion longer than 5 cm vs. 32% in limbs with an occlusion <5 cm in length.¹⁵¹ Occlusive lesions have much worse initial technical success rates than stenotic lesions. Concentric lesions respond better to PTA than eccentric lesions, and heavy calcifications have a negative impact on success rates. A meta-analysis by Hunink and associates showed that adjusted 5-year primary patencies after angioplasty of femoropopliteal lesions varied from 12 to 68%, the best results being for patients with claudication and stenotic lesions. Distal runoff is another powerful predictor of long-term success. Johnston analyzed 254 consecutive patients who underwent femoral and popliteal PTA and reported that 5-year patency rates of 53% for stenotic lesions and 36% for occlusive lesions in patients with good runoffs vs. a 5-year patency of 31% for stenotic lesions and 16% for occlusive lesions in patients with poor runoff. Literature reviews showed that 5-year patency rates varied from 27 to 67% based on the runoff statuses.

Due to limited success with infrapopliteal PTA, the indication for infrapopliteal artery PTA is stringent, reserved for limb salvage. Current patency rates from infrapopliteal PTA can be improved further by proper patient selection, ensuring straight-line flow to the foot in at least one tibial vessel, and close patient surveillance for early reintervention. Possible future advances including the use of drug-eluting stents (DES), cutting balloons, and atherectomy devices are being investigated to improve clinical outcomes following endovascular interventions on the tibial arteries. Varty and associates reported a 1-year limb salvage rate of

77% in patients with critical ischemia who underwent infrapopliteal PTA. In patients with favorable anatomies, a 2-year limb salvage rate after infrapopliteal artery PTA is expected to exceed 80 %.

Subintimal Angioplasty

The technique of SA was first described in 1987 when successful establishment of flow was made by accidental creation of a subintimal channel during treatment of a long popliteal artery occlusion. SA is recommended for chronic occlusion, long segments of lesion, and heavily calcified lesions. In addition, this technique is applicable for vessels with diffuse diseases and for vessels that had previously failed an intraluminal approach because it is difficult to negotiate the wire across the entire diseased segment without dissection.

The principle of this technique is to bypass the occlusion by deliberately creating a subintimal dissection plan commencing proximal to the lesion and continuing in the subintimal space before breaking back into the true lumen distal to the lesion. The occluded lumen is recanalized through the subintimal plan. SA can be performed through either ipsilateral antegrade or contralateral retrograde using the CFA approach. If selecting contralateral CFA puncture, a long guiding sheath is placed across the aortic bifurcation to provide access for the femoropopliteal and infrapopliteal vessels. The subintimal dissection is initiated at the origin of an occlusion by directing the tip of an angled guidewire, usually an angled hydrophilic wire such as Glidewire. A supporting catheter is used to guide the tip of the guidewire away from the important collaterals. When the wire is advanced, a loop is naturally formed at the tip of the guidewire. Once the subintimal plan is entered, the wire tends to move freely in the dissection space. Subintimal location of the wire and the catheter can be confirmed by injecting a small amount of diluted contrast. At this point, the wire and the catheter are then advanced along the subintimal plan until the occlusion segment is passed. A loss of resistance is often encountered as the guidewire re-enters the true lumen distal to the occlusion. Recanalization is confirmed by advancing the catheter over the guidewire beyond the point of re-entry and obtaining an angiogram. This is followed by a balloon angioplasty. To confirm the patency following balloon dilatation, a completion angiogram is performed before withdrawing the catheter and wire. If flow is impaired, repeat balloon dilatation may be necessary. Frequently, a stent is required to maintain a patent lumen and treat residual stenosis if more than a 30% luminal reduction is confirmed on completion angiogram.

Multiple studies have demonstrated the efficacy of SA. Bolia and his colleagues and

London and his colleagues reported their extensive experiences on SA for treating long segment occlusions of infrainguinal vessels. They achieved a technical success rate of over 80% for both femoropopliteal and tibial arteries. One-year patency rates varied from 53% for infrapopliteal vessels to 71% for femoropopliteal segments. Limb salvage rates reached over 80% at 12 months. They also reported that the factors influencing patency are smoking, number of runoff vessels, and occlusion length. Studies by other groups showed similar results. Treiman and colleagues treated 25 patients with 6- to 18-cm femoropopliteal occlusion and achieved a technical success rate of 92% and a 12-month primary patency rate of 92%, while Lipsitz and associates reported a technical success rate of 87% in treating 39 patients and achieved a 12-month cumulative patency rate of 74%. Additionally, Ingle and associates reported a technical success rate of 87% on 67 patients with femoropopliteal lesions and a 36-month limb salvage rate of 94%. As demonstrated herein, although technical success rates are similar in most series, the patency rates vary widely in different studies. Patient selection, anatomic character, and lesion locations may account for the wide range of outcomes.

Stent Placement

Although suggested by Dotter during the late 1960s, the use of an endoluminal stent was not pursued until the limitations of PTA were widely recognized. There are several situations where stent placement is appealing. The primary indication is the potential salvage of an unacceptable angioplasty result. Stent placement is typically used when residual stenosis after PTA is 30% or greater. An endoluminal stent is also used for dissection, perforation, and other PTA complications. Primary stent placement has become a viable alternative for treating ulcerative lesions that may potentially be the source for embolization. Primary stent is also used to treat occlusive lesions that have a tendency of reocclusion and distal embolization after PTA. In addition, an endoluminal stent is potentially beneficial for early restenosis post-PTA. DESs are currently under investigation in the United States and may be promising in decreasing restenotic rates.

Even though technical success rates are high, a published series on femoropopliteal artery stents show that patency rates are comparable to PTA alone with primary patency rates varying from 18 to 72% at 3 years. Gray and associates stented 58 limbs after suboptimal PTA for long SFA lesions and demonstrated a 1-year primary patency rate of 22%. However, Mewissen treated 137 limbs using self-expanding SMART nitinol stents in patients with TASC II A, B, and C femoropopliteal lesions and reported a 1-year primary patency rate of 76% and a

24-month primary patency rate of 60%. Appropriate patient selection and the anatomic characteristics of the lesions are crucial in the success of treatment outcomes. Additionally, stent characteristics may contribute to the patency rate.

Several clinical studies have demonstrated the significant improvements of the new generation of nitinol stents for the SFA lesions: the German Multicenter Experience, the Mewissen trial, the BLASTER Trial, and the SIROCCO trials.¹⁶¹ The German Multicenter Experience was a retrospective review of 111 SFA stenting procedures and found the 6-month patency rates for Smart stents and Wall stents were 82% and 37%, respectively. The BLASTER (Bilateral Lower Arterial Stenting Employing Reopro) Trial evaluated the feasibility of using nitinol stents with and without IV abciximab for the treatment of femoral artery disease. Preliminary results showed a 1-year clinical patency rate of 83%.

Furthermore, DESs, which proved effective at decreasing restenosis in coronary intervention, may offer another promising alternative in LE diseases. The drug, released over a period of time, interferes with smooth muscle cell proliferation, the main cellular element and source of extracellular-matrix-producing restenosis. The first DES clinical trial used Cordis Cypher SMART stents coated with sirolimus (SIROCCO trial). The SIROCCO results showed binary inlesion restenosis rates of 0% in the sirolimus-eluting group vs. 23.5% in the noneluting group at 6-month follow-up angiography.

Stent Graft

The concept of endobypass using stent graft in treating atherosclerotic SFA disease has been entertained. A stent graft is placed percutaneously across a long segment or multiple segments of lesions and is used to create a femoropopliteal bypass. Theoretically, endobypass has the potential of being as successful as surgical bypass graft by relining the vessel wall in its anatomical position without the negative impact of anastomosis. Stent grafts can be divided into two categories: unsupported and fully supported. The unsupported grafts consist of segments of bypass graft, such as PTFE, with an expandable stent at one or both ends. The unsupported grafts are flexible with a low profile, but prone to external compression. The supported stent grafts consist of a metallic skeleton covered with graft fabric. The presence of a dense metal skeleton promotes an extensive inflammatory response and increases the risk of thrombosis. There is no FDA-approved stent graft for peripheral intervention. However, Viabahn (WL Gore, Calif) is the most commonly used device in the United States, composed of an ultrathin PTFE graft externally supported by a self-expanding nitinol meshwork. The

Viabahn device has a specific delivery mechanism—pulling back the attached string—which results in a proximal-to-distal delivery of the endoprosthesis.

Although it is an intriguing concept, data on endobypass results are limited and the graft thrombosis rate is high. Additionally, covering major collateral vessels can potentially jeopardize the viability of the limb if stent graft occlusion occurs. Bauermeister treated 35 patients with Hemobahn and reported a 28.6% occlusion rate on an average 7-month follow-up. Kedora and colleagues recently conducted a prospective, randomized study comparing covered PTFE/nitinol self-expanding stent grafts with prosthetic above-the-knee femoropopliteal bypass. Fifty limbs were randomized into each group. Primary patency at 1-year was approximately 74% for both cohorts, with a mean follow-up of 18 months. The covered nitinol PTFE stent graft in the SFA had a 1-year patency rate comparable to surgical bypass, with a significantly shorter hospital stay (0.9 vs. 3.1 days).

Atherectomy

The basic principle of atherectomy is to remove the atheroma from obstructed arterial vessels. There are currently five atherectomy devices approved by the FDA: Simpson AtheroCath (DVI, Redwood City, Calif), Transluminal Extraction Catheter (Interventional Technologies, San Diego, Calif), Theratec recanalization arterial catheter (Trac-Wright), Auth Rotablator (Heart Technologies, Redmond, Wash), and SilverHawk system (FoxHollow Technologies, Redwood City, Calif). These devices either cut and remove or pulverize the atheroma plaques.

The Simpson AtheroCath has a directional cutting element that is exposed to one third of the circumference of the arterial wall. The atheroma protruding into the window is excised and pushed into the collection chamber. The Transluminal Extraction Catheter has an over-the-wire, nondirectional cutter mounted on the distal end of a torque tube. The excised atheroma is simultaneously removed by aspiration through the torque tube. The Theratec recanalization arterial catheter is a nondirectional, noncoaxial, atheroablative device. The rotating cam tip pulverizes the atheromatous lesion into minute particles. The Auth Rotablator is a nondirectional, coaxial, atheroablative device with a metal burr embedded with fine diamond chips. Lastly, the SilverHawk device, approved by the FDA for peripheral use in 2003, is a monorail catheter designed to overcome the drawback of direction atherectomy catheter, such as the Simpson AtheroCath. The working end consists of a hinged housing unit containing a carbide cutting blade. The blade is activated from the motor drive unit and the

catheter is then advanced through the length of the lesion. Once each pass is completed, the cutter then packs the tissue into the distal end of the nosecone to maximize collection capacity. The SilverHawk can then either be removed or torqued to treat a different quadrant in the same lesion or other lesions.

Despite the promising early technical and clinical success, the mid- and long-term results have been disappointing due to a high incidence of restenosis. However, a multicenter clinical registry of plaque atherectomy in patients with femoropopliteal occlusive disease showed potential clinical efficacy of this technology as the 6- and 12-month rates of survival free of target lesion revascularization were 90 and 80%, respectively. Importantly, nearly three fourths (73%) of patients treated with plaque excision modality did not require adjunctive endovascular therapy as infrainguinal stenting was necessary in only 6.3% of lesions. Results from the TALON registry support the role of plaque excision in selective patients with LE arterial disease.

Laser Atherectomy

Since laser atherectomy was reported in the 1960s, a variety of innovative approaches have been developed in an effort to overcome the limitation of laser angioplasty. Recent developments in excimer laser technology have led to increased optimism regarding the ability to safely deliver laser energy. Excimer laser atherectomy approved by the FDA for peripheral artery intervention uses precision laser energy control (shallow tissue penetration) and safer wavelengths (ultraviolet as opposed to the infrared spectra in older laser technology), which decreases perforation and thermal injury to the treated vessels.

A laser atherectomy catheter, with diameters varying from 0.9 mm to 2.5 mm, is tracked over the guidewire to the desired target. Once activated, the excimer laser uses ultraviolet energy to ablate the lesion and create a nonthrombogenic arterial lumen. This lumen is further dilated by an angioplasty balloon. Because the excimer laser can potentially reduce the rate of distal embolization by evaporating the lesion, it may be used as an adjunct tool for ostial lesions and lesions that can be traversed by a wire but not an angioplasty balloon catheter.

Several studies regarding the use of excimer laser atherectomy combined with balloon angioplasty on LE occlusive disease have shown promising clinical outcomes. Peripheral excimer laser angioplasty trials involved 318 patients with chronic SFA occlusion. They achieved a technical success rate of 83.2%, a 1-year primary patency rate of 33.6%, and an assisted primary patency rate of 65%.¹⁶⁸ Steinkamp and his colleagues treated 127 patients

with long-segment of popliteal artery occlusion using laser atherectomy followed by balloon angioplasty and reported a 3-year primary patency rate of 22%. The multicenter clinical trial evaluating the use of laser angioplasty for critical limb ischemia supports the efficacy of this treatment modality in selective patients as the 6-month primary patency rate and clinical improvement were 33 and 89%, respectively.

Complications of Endovascular Interventions

Angioplasty-Related Complications

Complications related to PTA vary widely, including dissection, rupture, embolization, pseudoaneurysms, restenosis, hematoma, and acute occlusion secondary to thrombosis, vasospasm, or intimal injury. Clark and associates analyzed the data from 205 patients in the SCVIR Transluminal Angioplasty and Revascularization registry and reported a complication rate of 7.3% for patients undergoing femoropopliteal angioplasty. Minor complications accounted for 75% of the cases, including distal emboli (41.7%), puncture site hematomas (41.7%), contained vessel rupture (8.3%), and vagal reactions (8.3%). In another study, Axisa and colleagues reported an overall rate of significant complications for patients undergoing PTA of the lower extremities as 4.2%, including retroperitoneal bleeding (0.2%), false aneurysm (0.2%), ALI (1.5%), and vessel perforation (1.7%).

Complications limiting the application of SA are parallel to those of PTA. A study investigating the use of SA in 65 patients with SFA occlusion found that complications developed in 15% of patients. These complications included significant stenosis (44%), SFA rupture (6%), distal embolization (3%), retroperitoneal hemorrhage (1.5%), and pseudoaneurysm (1.5%). Additional complications reported consist of perforation, thrombosis, dissection, and extensions beyond the planned re-entry site. Importantly, damage to significant collateral vessels may occur in 1 to 1.5% of patient who undergo SA. If a successful channel is not achieved in this situation, the patient may have a compromised distal circulation that necessitates distal bypass. Cryoplasty is a modified form of angioplasty, and long-term results on LE intervention are not yet available. Fava and associates treated 15 patients with femoropopliteal disease and had a 13% complication rate involving guidewire dissection and PTA-induced dissection of a tandem lesion remote to the cryoplasty zone.

Endoluminal Stent and Stent Graft–Related Complications

In addition to the aforementioned complications with angioplasty, endoluminal stents are associated with the risk of stent fracture and deformity. The adductor canal has nonlaminar flow dynamics, especially with walking. The forces exerted on the SFA include torsion, compression, extension, and flexion. These forces exert significant stress on the SFA and stents. In addition, the LE is subject to external trauma, which further increases the risk of stent deformity and fracture (Fig. 24). The SIROCCO study showed that stent fracture, although not associated with clinical symptoms, occurs in 18.2% of the procedures involving both the DES and control stent.

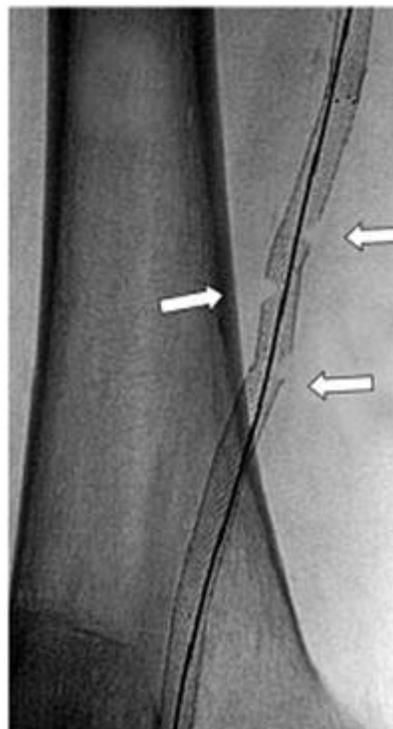


Fig. 24. Due to various geometric forces, including torsion, compression, extension, and flexion, which exert on the superficial femoral artery, stent fracture (*arrows*) is a known complication following superficial femoral artery stent placement.

Stent grafts may present an additional complication of covering important collaterals, which results in compromised distal circulation. A prospective study evaluating Hemobahn stent grafts in the treatment of femoropopliteal arterial occlusions demonstrated a 23% immediate complication rate, including distal embolization (7.7%), groin hematoma (13.5%), and arteriovenous fistula (1.9%).¹⁷⁵

Atherectomy-Related Complications

Overall complication rates associated with atherectomy range from 15.4 to 42.8% and include spasm, thrombosis, dissection, perforation, distal emboli, no reflow, and hematoma. Jahnke and associates conducted a prospective study evaluating high-speed, rotational atherectomy in 15 patients with infrapopliteal occlusive disease. They yielded a 94% technical success rate, which was complicated by vessel rupture (5%), distal embolization (5%), and arterial spasm (5%). Although the excimer laser atherectomy reduces embolic events by evaporating the lesion, embolization still remains a problematic complication. Studies show that distal embolic events occur in 3 to 4% of procedures, and perforation in 2.2 to 4.3% of cases. Other complications compromising laser atherectomy therapy include acute reocclusion, vasospasm, direct vessel injury, and dissection.

SURGICAL TREATMENT FOR CHRONIC LIMB ISCHEMIA DUE TO FEMOROPOPLITEAL DISEASE

Endarterectomy

Endarterectomy has a limited, albeit important, role in LE occlusive disease. It is most frequently used when there is disease in the CFA or involving the PFA. In this procedure, the surgeon opens the diseased segment longitudinally and develops a cleavage plane within the media that is developed proximally and distally. This permits the inner layer containing the atheroma to be excised. Great care must be taken at the distal end of the endarterectomy to ensure either a smooth transition or to tack down the distal endpoint to prevent the flow from elevating a potentially occlusive atheromatous flap. Currently, there is essentially no role for long open endarterectomy in the treatment of SFA stenoses or occlusions. The high incidence of restenosis is what limits use of endarterectomy in this location. Short-segment stenoses are more appropriately treated with balloon angioplasty. Endarterectomy using a catheter-based approach (e.g., Moll endarterectomy device) supplemented with stent grafting or stenting across the endpoint of the endarterectomy is currently being re-evaluated; however, no long-term data are available.

Bypass grafting

Bypass grafting remains the primary intervention for LE occlusive disease. The type of bypass and conduit are important variables to consider. Patients with occlusive disease limited

to the SFA, who have at least 4 cm (ideally 10 cm) of normal popliteal artery reconstituted above the knee joint and at least one continuous vessel to the foot, can be treated with an above-knee (AK) femoropopliteal bypass graft. Despite the fact that in this above-knee location, the differential patencies between prosthetic (PTFE) and vein graft are comparable; undoubtedly, it remains ideal to use a saphenous vein as the bypass conduit, if possible. Saving the vein for future coronary artery bypass or distal leg bypass grafting has been shown to be a flawed argument. One must also take into consideration that the consequences to the vascular outflow after a thrombosed prosthetic are worse than after a thrombosed vein graft.

When the disease extends to involve the popliteal artery or the tibial vessels, the surgeon must select an appropriate outflow vessel to perform a bypass. Suitable outflow vessels are defined as uninterrupted flow channels beyond the anastomosis into the foot. In order of descending preference, they are: AK popliteal artery, below-knee (BK) popliteal artery, posterior tibial artery, anterior tibial artery, and peroneal artery. In patients with diabetes, it is frequently the peroneal artery that is spared. Although the peroneal artery has no direct flow into the foot, collateralization to the posterior tibial and anterior tibial arteries makes it an appropriate outflow vessel. There is no objective evidence to preferentially select tibial over peroneal arteries if they are vessels of equal caliber and quality. The dorsalis pedis, which is the continuation of the anterior tibial in the foot, is frequently spared from atherosclerotic disease and can be used as a target for distal bypasses. Patency is affected by the length of the bypass (longer bypasses have reduced patency), quality of the recipient artery, extent of runoff to the foot, and quality of the conduit (saphenous vein/graft). Five-year assisted patency rates for infrapopliteal venous bypasses are 60%. Venous conduits also have been shown to be suitable for bypasses to plantar arteries. In this location, venous conduits have a 3-year limb salvage of 84% and a 3-year secondary patency of 74%. A meta-analysis suggests unsatisfactory results when PTFE-coated grafts are used to bypass to infrapopliteal arteries. In this location, prosthetic grafts have a 5-year primary patency rate of 30.5%. Additionally, due to distal embolization and compromise of outflow vessels, prosthetic graft occlusion may have more severe consequences than vein graft occlusion.

Two techniques are used for distal bypass grafting: reversed saphenous vein grafting and in situ saphenous vein grafting. There is no difference in outcomes (patency or limb salvage) between these techniques. In the former, the vein is excised in its entirety from the leg using open or endoscopic vein harvest, reversed to render the valves nonfunctional, and tunneled from the CFA inflow to the distal target vessels. End-to-side anastomoses are then created.

Several adjunctive techniques have been tried to improve the patency of bypass grafts to

tibial arteries. Creation of an arteriovenous fistula at the distal anastomosis is one option, but it has not been shown to improve patency. Another method involves creating various configurations of vein cuffs or patches at the distal anastomosis in an attempt to streamline the flow and to reduce the likelihood of neointimal hyperplasia. Results with this approach are more promising, especially when done to improve patency of a below-the-knee prosthetic; however, there are no definitive comparative trials that support the superiority of one configuration over another.

Amputation

Primary amputation is defined as an amputation that is performed without a prior attempt at surgical or endovascular revascularization. It is rarely necessary in patients who, as a result of neglect, present with class III ALI. Primary amputation may play a role in patients with critical limb ischemia who are deemed nonambulatory because of knee contractures, debilitating strokes, or dementia.

COMPLICATIONS OF SURGICAL RECONSTRUCTION

Vein Graft Stenoses

Fifteen percent of vein grafts will develop intrinsic stenoses within the first 18 months following implantation. Consequently, patients with a vein graft were entered into duplex surveillance protocols (scans every 3 months) to detect elevated (>300 cm/s) or abnormally low (<45 cm/s) graft velocities early. Stenoses greater than 50%, especially if associated with changes in ABI, should be repaired to prevent graft thrombosis. Repair usually entails patch angioplasty or short-segment venous interposition, but PTA/stenting is an option for short, focal lesions. Grafts with stenoses that are identified and repaired before thrombosis have assisted primary patency identical to primary patency, whereas a thrombosed autogenous bypass has limited longevity, resulting from ischemic injury to the vein wall. Secondary patency is markedly inferior to primary assisted patency. The recommendation for routine duplex ultrasound surveillance of autogenous infrainguinal bypasses was recently brought into question by a randomized, controlled trial that demonstrated no cost benefit or quality-of-life improvement after 18 months in patients with femoropopliteal venous bypasses. Many surgeons continue with programs of vein graft surveillance, as has been suggested in older

trials, awaiting further confirmation of the findings from the more recent study. When intervening on a failing infrainguinal bypass, the original indication for surgery is an important consideration. Limb salvage rates for occluded grafts are better if the indication for the original bypass was claudication rather than rest pain or tissue loss. An acutely occluded infrainguinal graft (≤ 30 postoperative days) has a 25% limb salvage rate.

Limb Swelling

Limb swelling is common following revascularization and usually returns to baseline within 2 to 3 months. The etiology is multifactorial, with lymphatic interruption, interstitial edema, and disruption of venous drainage all contributing. Limb swelling tends to worsen with repeat revascularization.

Wound Infection

Because the most common inflow vessel for distal bypass is the CFA, groin infection is common and occurs in 7% of cases. When an autogenous conduit such as a saphenous vein is used, most infection can be managed with local wound care because it involves the subcutaneous tissue or skin rather than infection of the actual vein. When a prosthetic graft has been used, management of graft infection is a major undertaking. Infection of a LE prosthetic bypass graft is associated with a significant amputation rate because of the tendency for graft thrombosis and anastomotic disruption. Prosthetic graft infections cannot be eradicated with antibiotics, and they mandate graft excision and complex revascularization using a vein, if available.

Choice of Conduit for Infrainguinal Bypass Grafting

Autogenous Vein

Autogenous vein is superior to prosthetic conduits for all infrainguinal bypasses, even in the AK position. This preference is applicable not only for the initial bypass but also for reoperative cases. For long bypasses, ipsilateral great saphenous vein (GSV), contralateral GSV, small saphenous vein, arm vein, and spliced vein are used, in decreasing order of preference. If only a short segment of vein is missing, the SFA can be endarterectomized and

the proximal anastomosis performed distally to decrease the length of the conduit and to avoid harvesting and splicing additional vein. When GSV is not available and a relatively short bypass is necessary, arm vein or small saphenous vein is effective. Small saphenous vein is of particular use when a posterior approach is used. If a longer bypass with vein is necessary, arm vein is preferable because it is less awkward to harvest. Another conduit alternative is to harvest the upper arm basilic, median cubital, and cephalic veins in continuity, while incising valves in the basilic segment and using the cephalic segment in reversed configuration to provide a relatively long, unspliced autogenous conduit.

Cryopreserved Grafts

Cryopreserved grafts are usually cadaveric arteries or veins that have been subjected to rate-controlled freezing with dimethyl sulfoxide and other cryopreservants. Cryopreserved vein grafts are more expensive than prosthetic grafts and are more prone to failure. The endothelial lining is lost as part of the freezing process, making these grafts prone to early thrombosis. Cryopreserved grafts also are prone to aneurysmal degeneration. Despite the fact that these grafts have not performed as well as prosthetic bypasses and autogenous vein in clinical practice, they can still play a role when revascularization is required following removal of infected prosthetic bypass grafts, especially when autogenous vein is unavailable to create a new bypass through clean tissue planes.

Human Umbilical Vein

Human umbilical vein (HUV) is less commonly used than PTFE, because it is thicker and more cumbersome to handle and because of concerns about aneurysmal degeneration. HUV allografts are stabilized with glutaraldehyde and do not have viable cells or antigenic reactivity. These grafts have poor handling characteristics and require extra care when suturing because of an outer Dacron mesh wrapping, which is used to decrease aneurysmal degeneration. Dardik and colleagues have reported favorable results after using HUV and an adjunctive distal arteriovenous fistula. One trial comparing HUV with PTFE and saphenous vein showed that HUV was better than PTFE but worse than saphenous vein in terms of 5-year patency in the AK location. In a systematic review, HUV appears to perform better than cryopreserved veins.

Prosthetic Conduits and Adjunctive Modifications

If vein is truly unavailable, PTFE or Dacron is the best option for AK bypass. The addition of rings to PTFE did not confer benefit in a single prospective, randomized clinical trial. For infrageniculate prosthetic bypasses, use of a vein patch, cuff, or other venous anastomotic modification can improve patency (52% patency at 2 years for PTFE with vein cuff vs. 29% for PTFE with no cuff) and also improve limb salvage (84% vs. 62%).

Although prosthetic grafts are readily available, easy to handle, and do not require extensive dissection to harvest, their propensity to undergo thrombosis and develop neointimal hyperplasia makes them a less favorable alternative when compared to vein. In a recent review of vein and prosthetic AK femoropopliteal bypasses, the 5-year primary patency rates were reported to be 74 and 39%, respectively. Outcomes were even worse for BK prosthetic bypasses. Unfortunately, the use of autologous venous conduits is not possible in as many as 30% of patients. The GSV may be unsuitable because of small size and poor quality or unavailable due to prior harvest.

Methods to improve prosthetic graft performance have consisted of altering the geometry at the distal anastomosis to get the benefit obtained with vein cuffs (Distaflo, Bard Peripheral Vascular, Tempe, Ariz) and covalently bonding agents onto the luminal surface with anticoagulant, anti-inflammatory and antiproliferative characteristics (Propaten, Gore, Flagstaff, Ariz). One randomized trial that compared precuffed PTFE and PTFE with a vein cuff enrolled 104 patients at 10 centers. Eighty-nine patients were randomized to 47 precuffed PTFE bypasses and 44 bypasses with a vein cuff. At 1 and 2 years, primary patency rates were 52 and 49% for the precuffed group and 62 and 44% for the vein cuffed group, respectively. At 1 year and 2 years, the limb salvage rate was 72 and 65% for the precuffed group and 75 and 62% in the vein cuffed group, respectively. Although numbers are small and follow-up short, the midterm analysis revealed that Distaflo precuffed grafts and PTFE grafts with vein cuff had similar results. The authors concluded that a precuffed graft was a reasonable alternative for infragenicular reconstruction in the absence of saphenous vein. Other authors have been less optimistic and question if there is any benefit derived from geometrically altering prosthetic conduits.

Another approach for improving outcomes when using prosthetic for bypass grafts involves bonding anticoagulants to the conduit. The Gore Propaten graft has heparin bonded onto the luminal surface of the PTFE graft using Carmeda BioActive Surface technology, which immobilizes the heparin molecule with a single covalent bond that does not alter its

anticoagulant properties. The heparin binding does not alter the microstructure and handling characteristics of the PTFE. A prospective, randomized trial by Devine and associates suggested that heparin-bonded Dacron or PTFE was superior to plain PTFE for AK popliteal bypasses. The 3-year primary patency for the heparin-bonded grafts was 55% compared with 42% for PTFE ($P < .044$). Both of these patency rates are inferior to GSV; however, if the improved results with heparin bonding continue to be substantiated, then heparin-bonded prosthetic grafts will become the preferred conduit for AK bypass in the absence of suitable vein. A recent review of available studies with this graft showed an 80% 1-year patency for BK bypasses.¹⁹⁵ Randomized, controlled clinical trials with more patients and longer follow-up are necessary to validate whether the PROPATEN vascular graft is superior to other prosthetics, and if, indeed, it is comparable to autogenous vein for BK interventions.

Clinical Results of Surgical and Endovascular Interventions for Femoropopliteal Occlusive Disease

Balloon angioplasty of the femoropopliteal vessels has not enjoyed the degree of success seen with iliac angioplasty. Patency in this region is dependent upon whether the patient presents with claudication vs. limb-threatening ischemia, the status of the distal runoff vessels, and lesion morphology. Initial technical success for femoropopliteal angioplasty is seen in 80 to 90% of cases, with failures to cross a lesion occurring in 7% of stenoses and 18% of occlusive lesions. Studies have shown that PTA of the femoropopliteal segment achieved greater than a 90% technical success rate and had a 59% primary patency rate at 5-years. PTA of lesions longer than 7 to 10 cm results in compromised patency, while PTA of shorter lesions (<3 cm) gives fairly good results. Lofberg and associates performed 127 femoropopliteal PTA procedures and reported a primary patency rate at 5-year follow-up of 12% in limbs with occlusion longer than 5 cm vs. 32% in limbs with occlusion <5 cm in length. Occlusive lesions have much worse initial technical success rates than stenotic lesions. Concentric lesions respond better to PTA than eccentric lesions, and heavy calcifications have a negative impact on success rates. Distal runoff is another powerful predictor of long-term success.

Johnston and associates analyzed 254 consecutive patients who underwent femoropopliteal PTA and reported a 5-year patency rate of 53% for stenotic lesions and 36% for occlusive lesions in patients with good runoff vs. 5-year patency of 31% for stenotic lesions and 16% for occlusive lesions in patients with poor runoff. A meta-analysis by Hunink and colleagues showed that adjusted 5-year primary patencies after angioplasty of femoropopliteal

lesions varied from 12 to 68%, the best results occurring in patients with claudication and stenotic lesions. Although the initial technical success is better for stenoses than occlusions, long-term patency rates for stenoses and short occlusions have been variable and there have been conflicting results regarding the efficacy of stent use. Early published series that examined efficacy of femoropopliteal artery stents showed patency rates that were comparable to stand-alone PTA with primary patency rates varying from 18 to 72% at 3 years. Patient selection and the anatomic character of the lesions may play important roles in the outcomes. Additionally, stent characteristics may contribute to the patency rate. Several clinical studies have demonstrated significant improvements in patency when the newer generations of nitinol stents are used to treat SFA lesions.

Mewissen treated 137 lower limbs in 122 patients with CLI, secondary to TASC II A ($n = 12$) or TASC II B or C ($n = 125$) lesions in the SFA. Patients were treated with Cordis SMART self-expanding nitinol stents. Binary restenosis ($>50\%$) was measured by standard duplex velocity criteria at various postintervention intervals. Primary stent patency, defined as absence of binary restenosis in this study, was calculated by life table methods from the time of intervention. The mean lesion length was 12.2 cm (range, 4 to 28 cm). The technical success was 98%. Mean follow-up was 302 days. The primary stent patency rates were 92%, 76%, 66%, and 60% at 6, 12, 18, and 24-months, respectively. Ferreira and associates treated 59 patients who had 74 femoropopliteal lesions (60% TASC II D) with Zilver nitinol self-expanding stents (COOK, Bloomington, Ind). Mean recanalization length was 19 cm (range 3 to 53 cm). Mean follow-up time was 2.4 years (range 3 days to 4.8 years). Kaplan-Meier estimates for primary patency rates were 90%, 78%, 74%, 69%, and 69% at 1, 2, 3, 4, and 4.8 years, respectively.

There is general agreement that for suboptimal PTA of an SFA lesion, stent placement is indicated, but a recent randomized trial by Schillinger and associates suggests that primary stenting results in lower restenosis rates than PTA and selective stenting. Restenosis rates at 2 years were 45.7% vs. 69.2% in favor of primary stenting compared with PTA and optional secondary stenting using an intention-to-treat analysis ($P = .031$). Consistently, stenting, both primary and selective, was superior to stand-alone PTA with respect to the occurrence of restenosis (49.2% vs. 74.3%; $P = .028$) by a treatment-received analysis.

Nitinol bare metal stents that are designed specifically for BK interventions are showing very encouraging results. Bosiers and colleagues reported their 12-month results using the commercially available non-drug-eluting Xpert (Abbott Vascular, Santa Clara, Calif) nitinol stent system in BK arterial interventions. They had a 12-month primary patency rate of 76.3%,

and a limb salvage rate of 95.9%. They followed patients to 12 months and performed angiography with quantitative vessel analysis on the 73% of patients available. Angiography revealed a binary restenosis rate (>50%) of only 20.5%, which is comparable to well-accepted coronary DES study outcomes. The authors attributed this optimal performance to the maintenance of flow dynamics because the stent was specifically designed for use in small vessels. Kickuth and colleagues also have obtained good results using the Xpert stent. After stent placement, the primary cumulative patency rate at 6 months for the study group of 35 patients was 82%. The sustained clinical improvement rate as evidenced by improved ABI was 80%, and freedom from major amputation was 100% at the 6-month follow-up. The rate of major complications was 17%.

Wolf and associates published a multicenter, prospective randomized trial comparing PTA with bypass in 263 men who had iliac, femoral, or popliteal artery obstruction. In 56 patients, cumulative 1-year primary patency after PTA was 43% and, after bypass surgery, was 82%, demonstrating that for long SFA stenoses or occlusions, surgery is better than PTA. Another recent randomized study (BASIL trial) of 452 patients with CLI demonstrated no difference in amputation-free survival at 6 months between surgery and PTA/stenting. The authors commented that surgery was somewhat more expensive and recommended that endovascular intervention should be used as first-line therapy, especially in medically unfit patients. They did conclude that at 2-year follow-up, healthy patients without medical comorbidities derived greater benefit from surgery because it was associated with decreased need for reintervention and had a decreased hazard ratio in terms of all-cause mortality. Using the 2000 TASC II definitions and a Markov state transition model decision analysis, Nolan and colleagues showed that PTA/stenting surpasses bypass efficacy for TASC II C lesions if PTA/stenting primary patency is greater than 32% at 5 years, patient age is >80 years, and/or GSV bypass operative mortality is greater than 6%.

MESENTERIC ARTERY DISEASE

Vascular occlusive disease of the mesenteric vessels is a relatively uncommon but potentially devastating condition that generally presents in patients more than 60 years of age, is three times more frequent in women, and has been recognized as an entity since 1936. The incidence of such a disease is low and represents 2% of the revascularization operations for atheromatous lesions. The most common cause of mesenteric ischemia is atherosclerotic vascular disease. Autopsy studies have demonstrated splanchnic atherosclerosis in 35 to 70%

of cases. Other etiologies exist and include FMD, panarteritis nodosa, arteritis, and celiac artery (CA) compression from a median arcuate ligament, but they are unusual and have an incidence of one in nine compared to that of atherosclerosis.

Chronic mesenteric ischemia is related to a lack of blood supply in the splanchnic region and is caused by disease in one or more visceral arteries: the celiac trunk, the SMA, and the IMA. Mesenteric ischemia is thought to occur when two of the three visceral vessels are affected with severe stenosis or occlusion; however, in as many as 9% of cases, only a single vessel is involved [superior mesenteric artery (SMA) in 5% and celiac trunk in 4% of cases].⁷³ This disease process may evolve in a chronic fashion, as in the case of progressive luminal obliteration due to atherosclerosis. On the other hand, mesenteric ischemia can occur suddenly, as in the case of thromboembolism. Despite recent progress in perioperative management and better understanding in pathophysiology, mesenteric ischemia is considered one of the most catastrophic vascular disorders, with mortality rates ranging from 50 to 75%. Delay in diagnosis and treatment are the main contributing factors in its high mortality. It is estimated that mesenteric ischemia accounts for one in every 1000 hospital admissions in this country. The prevalence is rising due in part to the increased awareness of this disease, the advanced age of the population, and the significant comorbidity of these elderly patients. Early recognition and prompt treatment before the onset of irreversible intestinal ischemia are essential to improve the outcome.

Anatomy and Pathophysiology

Mesenteric arterial circulation is remarkable for its rich collateral network. Three main mesenteric arteries provide the arterial perfusion to the GI system: the CA, SMA, and IMA. In general, the CA provides arterial circulation to the foregut (distal esophagus to duodenum), hepatobiliary system, and spleen; the SMA supplies the midgut (jejunum to midcolon); and the IMA supplies the hindgut (midcolon to rectum). The CA and SMA arise from the ventral surface of the infradiaphragmatic suprarenal abdominal aorta, while the IMA originates from the left lateral portion of the infrarenal aorta. These anatomic origins in relation to the aorta are important when a mesenteric angiogram is performed to determine the luminal patency. To fully visualize the origins of the CA and SMA, it is necessary to perform both an anteroposterior and a lateral projection of the aorta because most arterial occlusive lesions occur in the proximal segments of these mesenteric trunks.

Because of the abundant collateral flow between these mesenteric arteries, progressive

diminution of flow in one or even two of the main mesenteric trunks is usually tolerated, provided that uninvolved mesenteric branches can enlarge over time to provide sufficient compensatory collateral flow. In contrast, acute occlusion of a main mesenteric trunk may result in profound ischemia due to lack of sufficient collateral flow. Collateral network between the CA and the SMA exist primarily through the superior and inferior pancreaticoduodenal arteries. The IMA may provide collateral arterial flow to the SMA through the marginal artery of Drummond, the arc of Riolan, and other unnamed retroperitoneal collateral vessels termed *meandering mesenteric arteries* (Fig. 25). Lastly, collateral visceral vessels may provide important arterial flow to the IMA and the hindgut through the hypogastric arteries and the hemorrhoidal arterial network.



Fig. 25. An aortogram showing a prominent collateral vessel that is the arc of Riolan (*arrow*) in a patient with an inferior mesenteric artery occlusion. This vessel network provides collateral flow between the superior mesenteric artery and inferior mesenteric artery

Regulation of mesenteric blood flow is largely modulated by both hormonal and neural stimuli, which characteristically regulate systemic blood flow. In addition, the mesenteric

circulation responds to the GI contents. Hormonal regulation is mediated by splanchnic vasodilators such as nitric oxide, glucagon, and vasoactive intestinal peptide. Certain intrinsic vasoconstrictors such as vasopressin can diminish the mesenteric blood flow. On the other hand, neural regulation is provided by the extensive visceral autonomic innervation.

Clinical manifestation of mesenteric ischemia is predominantly postprandial abdominal pain, which signifies that the increased oxygen demand of digestion is not met by the GI collateral circulation. The postprandial pain frequently occurs in the midabdomen, suggesting that the diversion of blood flow from the SMA to supply the stomach impairs perfusion to the small bowel. This leads to transient anaerobic metabolism and acidosis. Persistent or profound mesenteric ischemia will lead to mucosal compromise with release of intracellular contents and by-products of anaerobic metabolism to the splanchnic and systemic circulation. Injured bowel mucosa allows unimpeded influx of toxic substances from the bowel lumen with systemic consequences. If full-thickness necrosis occurs in the bowel wall, intestinal perforation ensues, which will lead to peritonitis. Concomitant atherosclerotic disease in cardiac or systemic circulation frequently compounds the diagnostic and therapeutic complexity of mesenteric ischemia.

Types of Mesenteric Artery Occlusive Disease

There are three major mechanisms of visceral ischemia involving the mesenteric arteries, which include: (a) acute mesenteric ischemia, which can be either embolic or thrombotic in origin; (b) chronic mesenteric ischemia; and (c) nonocclusive mesenteric ischemia. Despite the variability of these syndromes, a common anatomic pathology is involved in these processes. The SMA is the most commonly involved vessel in acute mesenteric ischemia. Acute thrombosis occurs in patients with underlying mesenteric atherosclerosis, which typically involves the origin of the mesenteric arteries while sparing the collateral branches. In acute embolic mesenteric ischemia, the emboli typically originate from a cardiac source and frequently occur in patients with atrial fibrillation or following myocardial infarction (Figs. 26 and 27). Nonocclusive mesenteric ischemia is characterized by a low flow state in otherwise normal mesenteric arteries, and most frequently occurs in critically ill patients on vasopressors. Finally, chronic mesenteric ischemia is a functional consequence of a long-standing atherosclerotic process that typically involves at least two of the three main mesenteric vessels. The gradual development of the occlusive process allows the development of collateral vessels that prevent the manifestations of acute ischemia, but are not sufficient to meet the high

postprandial intestinal oxygen requirements, giving rise to the classical symptoms of postprandial abdominal pain and the resultant food fear.

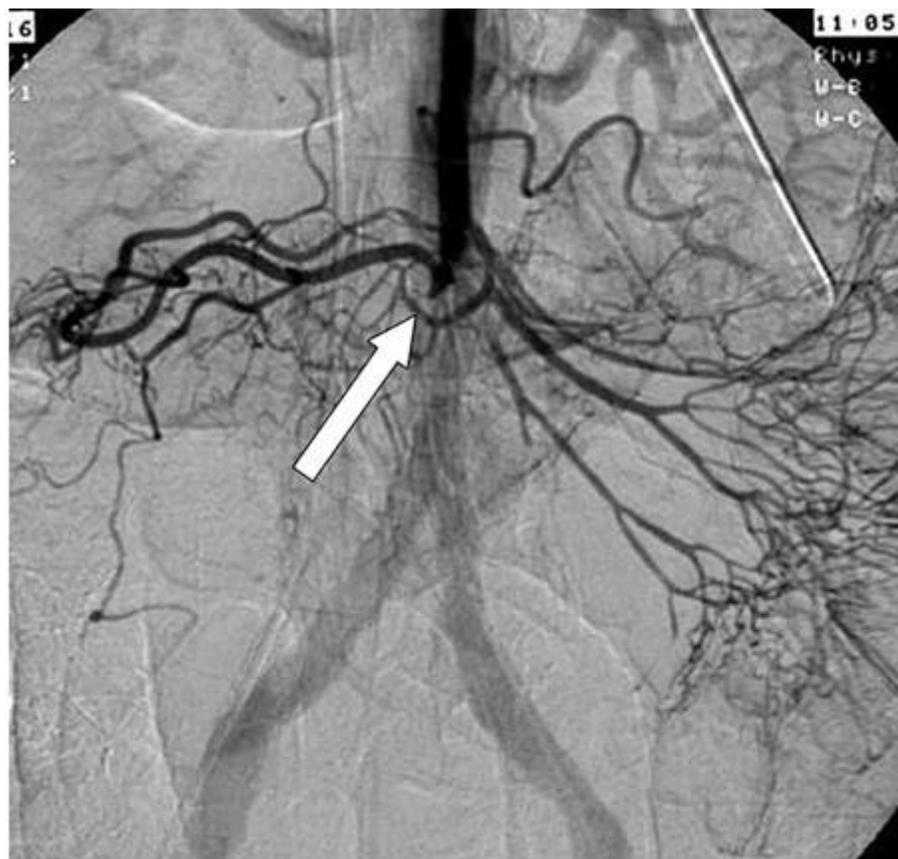


Fig.26. An anteroposterior view of a selective superior mesenteric artery angiogram showed an abrupt cutoff of the middle colic artery, which was caused by emboli (*arrow*) due to atrial fibrillation



Fig. 27. A lateral mesenteric angiogram showing an abrupt cutoff of the proximal superior mesenteric artery, which is consistent with superior mesenteric artery embolism (*arrow*).

Mesenteric arteriography also can play a therapeutic role. Once the diagnosis of nonocclusive mesenteric ischemia is made on the arteriogram, an infusion catheter can be placed at the SMA orifice and vasodilating agents such as papaverine can be administered intra-arterially. The papaverine infusion may be continued postoperatively to treat persistent vasospasm, a common occurrence following mesenteric reperfusion. Transcatheter thrombolytic therapy has little role in the management of thrombotic mesenteric occlusion. Although thrombolytic agents may transiently recannulate the occluded vessels, the underlying occlusive lesions require definitive treatment. Furthermore, thrombolytic therapy typically requires a prolonged period of time to restore perfusion, during which the intestinal viability will be difficult to assess.

A word of caution would be appropriate here regarding patients with typical history of chronic intestinal angina who present with an acute abdomen and classical findings of peritoneal irritation. Arteriography is the gold standard for the diagnosis of mesenteric occlusive disease; however, it can be a time-consuming diagnostic modality. In this group of patients, immediate exploration for assessment of intestinal viability and vascular reconstruction is the best choice.

Surgical Repair

Acute Embolic Mesenteric Ischemia

Initial management of patients with acute mesenteric ischemia includes fluid resuscitation and systemic anticoagulation with heparin to prevent further thrombus propagation. Significant metabolic acidosis not responding to fluid resuscitation should be corrected with sodium bicarbonate. A central venous catheter, peripheral arterial catheter, and a Foley catheter should be placed for hemodynamic status monitoring. Appropriate antibiotics are given before surgical exploration. The operative management of acute mesenteric ischemia is dictated by the cause of the occlusion. It is helpful to obtain a preoperative mesenteric arteriogram to confirm the diagnosis and to plan appropriate treatment options. However, the diagnosis of mesenteric ischemia frequently cannot be established before surgical exploration; and therefore, patients in a moribund condition with acute abdominal symptoms should undergo immediate surgical exploration, avoiding the delay required to perform an arteriogram.

The primary goal of surgical treatment in embolic mesenteric ischemia is to restore arterial perfusion with removal of the embolus from the vessel. The abdomen is explored through a midline incision, which often reveals variable degrees of intestinal ischemia from the midjejunum to the ascending or transverse colon. The transverse colon is lifted superiorly, and the small intestine is reflected toward the right upper quadrant. The SMA is approached at the root of the small bowel mesentery, usually as it emerges from beneath the pancreas to cross over the junction of the third and fourth portions of the duodenum. Alternatively, the SMA can be approached by incising the retroperitoneum lateral to the fourth portion of the duodenum, which is rotated medially to expose the SMA. Once the proximal SMA is identified and controlled with vascular clamps, a transverse arteriotomy is made to extract the embolus, using standard balloon embolectomy catheters. In the event the embolus has lodged more distally, exposure of the distal SMA may be obtained in the root of the small bowel mesentery by isolating individual jejunal and ileal branches to allow a more comprehensive thromboembolectomy. Following the restoration of SMA flow, an assessment of intestinal viability must be made, and nonviable bowel must be resected. Several methods have been described to evaluate the viability of the intestine, which include intraoperative IV fluorescein injection and inspection with a Wood's lamp, and Doppler assessment of antimesenteric intestinal arterial pulsations. A second-look procedure should be considered in many patients,

and is performed 24 to 48 hours following embolectomy. The goal of the procedure is reassessment of the extent of bowel viability, which may not be obvious immediately following the initial embolectomy. If nonviable intestine is evident in the second-look procedure, additional bowel resections should be performed at that time.

Acute Thrombotic Mesenteric Ischemia

Thrombotic mesenteric ischemia usually involves a severely atherosclerotic vessel, typically the proximal CA and SMA. Therefore, these patients require a reconstructive procedure to the SMA to bypass the proximal occlusive lesion and restore adequate mesenteric flow. The saphenous vein is the graft material of choice, and prosthetic materials should be avoided in patients with nonviable bowel, due to the risk of bacterial contamination if resection of necrotic intestine is performed. The bypass graft may originate from either the aorta or iliac artery. Advantages from using the supraceliac infradiaphragmatic aorta as opposed to the infrarenal aorta as the inflow vessel include a more smooth graft configuration with less chance of kinking, and the absence of atherosclerotic disease in the supraceliac aortic segment. Exposure of the supraceliac aorta is technically more challenging and time consuming than that of the iliac artery, which, unless calcified, is an appropriate inflow. Patency rates are similar regardless of inflow vessel choice.

Chronic Mesenteric Ischemia

The therapeutic goal in patients with chronic mesenteric ischemia is to revascularize mesenteric circulation and prevent the development of bowel infarction. Mesenteric occlusive disease can be treated successfully by either transaortic endarterectomy or mesenteric artery bypass. Transaortic endarterectomy is indicated for ostial lesions of patent CA and SMA. A left medial rotation is performed, and the aorta and the mesenteric branches are exposed. A lateral aortotomy is performed, encompassing both the CA and SMA orifices. The visceral arteries must be adequately mobilized so that the termination site of endarterectomy can be visualized. Otherwise, an intimal flap may develop, which can lead to early thrombosis or distal embolization.

For occlusive lesions located 1 to 2 cm distal to the mesenteric origin, mesenteric artery bypass should be performed. Multiple mesenteric arteries are typically involved in chronic mesenteric ischemia, and both the CA and SMA should be revascularized whenever possible.

In general, bypass grafting may be performed either antegrade from the supraceliac aorta or retrograde from either the infrarenal aorta or iliac artery. Both autogenous saphenous vein grafts and prosthetic grafts have been used with satisfactory and equivalent success. An antegrade bypass also can be performed using a small-caliber bifurcated graft from the supraceliac aorta to both the CA and SMA, which yields an excellent long-term result.

Celiac Artery Compression Syndrome

The decision to intervene in patients with CA compression syndrome should be based on both an appropriate symptom complex and the finding of CA compression in the absence of other findings to explain the symptoms. The treatment goal is to release the ligamentous structure that compresses the proximal CA and to correct any persistent stricture by bypass grafting. The patient should be cautioned that relief of the celiac compression cannot be guaranteed to relieve the symptoms. In a number of reports on endovascular management of chronic mesenteric ischemia, the presence of CA compression syndrome has been identified as a major factor of technical failure and recurrence. Therefore, angioplasty and stenting should not be undertaken if extrinsic compression of the CA by the median arcuate ligament is suspected based on preoperative imaging studies. Open surgical treatment should be performed instead.

Endovascular Treatment

Chronic Mesenteric Ischemia

Endovascular treatment of mesenteric artery stenosis or short segment occlusion by balloon dilatation or stent placement represents a less invasive therapeutic alternative to open surgical intervention, particularly in patients whose medical comorbidities place them in a high operative risk category. Endovascular therapy is also suited to patients with recurrent disease or anastomotic stenosis following previous open mesenteric revascularization. Prophylactic mesenteric revascularization is rarely performed in the asymptomatic patient undergoing an aortic procedure for other indications. However, the natural history of untreated chronic mesenteric ischemia may justify revascularization in some minimally symptomatic or asymptomatic patients if the operative risks are acceptable, because the first clinical presentation may be acute intestinal ischemia in as many as 50% of the patients, with a

mortality rate that ranges from 15 to 70%. This is particularly true when the SMA is involved. Mesenteric angioplasty and stenting is particularly suitable for this patient subgroup given its low morbidity and mortality. Because of the limited experience with stent use in mesenteric vessels, appropriate indications for primary stent placement have not been clearly defined. Guidelines generally include calcified ostial stenoses, high-grade eccentric stenoses, chronic occlusions, and significant residual stenosis greater than 30% or the presence of dissection after angioplasty. Restenosis after PTA is also an indication for stent placement.

Acute Mesenteric Ischemia

Catheter-directed thrombolytic therapy is a potentially useful treatment modality for acute mesenteric ischemia, which can be initiated with intra-arterial delivery of thrombolytic agent into the mesenteric thrombus at the time of diagnostic angiography. Various thrombolytic medications, including urokinase (Abbokinase, Abbott Laboratory, North Chicago, Ill) or recombinant tissue plasminogen activator (Activase, Genentech, South San Francisco, Calif), have been reported to be successful in a small series of case reports. Catheter-directed thrombolytic therapy has a higher probability of restoring mesenteric blood flow success when performed within 12 hours of symptom onset. Successful resolution of a mesenteric thrombus will facilitate the identification of the underlying mesenteric occlusive disease process. As a result, subsequent operative mesenteric revascularization or mesenteric balloon angioplasty and stenting may be performed electively to correct the mesenteric stenosis. There are two main drawbacks with regard to thrombolytic therapy in mesenteric ischemia. Percutaneous, catheter-directed thrombolysis (CDT) does not allow the possibility to inspect the potentially ischemic intestine following restoration of the mesenteric flow. Additionally, a prolonged period of time may be necessary to achieve successful CDT, due in part to serial angiographic surveillance to document thrombus resolution. An incomplete or unsuccessful thrombolysis may lead to delayed operative revascularization, which may further necessitate bowel resection for irreversible intestinal necrosis. Therefore, catheter-directed thrombolytic therapy for acute mesenteric ischemia should only be considered in selected patients under a closely scrutinized clinical protocol.

Nonocclusive Mesenteric Ischemia

The treatment of nonocclusive mesenteric ischemia is primarily pharmacologic with

selective mesenteric arterial catheterization followed by infusion of vasodilatory agents such as tolazoline or papaverine. Once the diagnosis is made on the mesenteric arteriography, intra-arterial papaverine is given at a dose of 30 to 60 mg/h. This must be coupled with the cessation of other vasoconstricting agents. Concomitant IV heparin should be administered to prevent thrombosis in the cannulated vessels. Treatment strategy thereafter is dependent on the patient's clinical response to the vasodilator therapy. If abdominal symptoms improve, mesenteric arteriography should be repeated to document the resolution of vasospasm. The patient's hemodynamic status must be carefully monitored during papaverine infusion, as significant hypotension can develop in the event that the infusion catheter migrates into the aorta, which can lead to systemic circulation of papaverine. Surgical exploration is indicated if the patient develops signs of continued bowel ischemia or infarction as evidenced by rebound tenderness or involuntary guarding. In these circumstances, papaverine infusion should be continued intraoperatively and postoperatively. The OR should be kept as warm as possible, and warm irrigation fluid and laparotomy pads should be used to prevent further intestinal vasoconstriction during exploration.

Techniques of Endovascular Interventions

To perform endovascular mesenteric revascularization, intraluminal access is performed via a femoral or brachial artery approach. Once an introducer sheath is placed in the femoral artery, an anteroposterior and lateral aortogram just below the level of the diaphragm is obtained with a pigtail catheter to identify the origin of the CA and SMA. Initial catheterization of the mesenteric artery can be performed using a variety of selective angled catheters, which include the RDC, Cobra-2, Simmons I (Boston Scientific/Meditech, Natick, Mass), or SOS Omni catheter (AngioDynamics, Queensbury, NY). Once the mesenteric artery is cannulated, systemic heparin (5000 IU) is administered IV. A selective mesenteric angiogram is then performed to identify the diseased segment, which is followed by the placement of a 0.035-in or less traumatic 0.014- to 0.018-in guidewire to cross the stenotic lesion. Once the guidewire is placed across the stenosis, the catheter is carefully advanced over the guidewire across the lesion. In the event that the mesenteric artery is severely angulated as it arises from the aorta, a second stiffer guidewire (Amplatz or Rosen Guidewire, Boston Scientific) may be exchanged through the catheter to facilitate the placement of a 6F guiding sheath (Pinnacle, Boston Scientific).

With the image intensifier angled in a lateral position to fully visualize the proximal

mesenteric segment, a balloon angioplasty is advanced over the guidewire through the guiding sheath and positioned across the stenosis. The balloon diameter should be chosen based on the vessel size of the adjacent normal mesenteric vessel. Once balloon angioplasty is completed, a postangioplasty angiogram is necessary to document the procedural result. Radiographic evidence of either residual stenosis or mesenteric artery dissection constitutes suboptimal angioplasty results that warrant mesenteric stent placement. Moreover, atherosclerotic involvement of the proximal mesenteric artery or vessel orifice should be treated with a balloon-expandable stent placement. These stents can be placed over a low profile 0.014- or 0.018-in guidewire system. It is preferable to deliver the balloon-mounted stent through a guiding sheath, which is positioned just proximal to the mesenteric orifice while the balloon-mounted stent is advanced across the stenosis. The stent is next deployed by expanding the angioplasty balloon to its designated inflation pressure. The balloon is then deflated and carefully withdrawn through the guiding sheath.

Completion angiogram is performed by hand injecting a small volume of contrast through the guiding sheath. It is critical to maintain the guidewire access until a satisfactory completion angiogram is obtained. If the completion angiogram reveals suboptimal radiographic results, such as residual stenosis or dissection, additional catheter-based intervention can be performed through the same guidewire. These interventions may include repeat balloon angioplasty for residual stenosis or additional stent placement for mesenteric artery dissection. During the procedure, intra-arterial infusion of papaverine or nitroglycerine can be used to decrease vasospasm. Administration of antiplatelet agents is also recommended, for at least 6 months or even indefinitely if other risk factors of cardiovascular disease are present.

Complications of Endovascular Treatment

Complications are not common and rarely become life threatening. These include access site thrombosis, hematomas, and infection. Dissection can occur during PTA and is managed with placement of a stent. Balloon-mounted stents are preferred over the self-expanding ones because of the higher radial force and the more precise placement. Distal embolization has also been reported but it never resulted in acute intestinal ischemia, likely due to the rich network of collaterals already developed.

TOPIC 9. ARTERIAL TROMBOSIS AND EMBOLISM

ACUTE UPPER EXTREMITY ISCHEMIA

Acute ischemia in the upper extremity constitutes 10–15% of all acute extremity ischemia. The etiology is emboli in 90% of the patients. The reason for this higher rate compared with the leg is that atherosclerosis is less common in arm arteries. Emboli have the same origins as in the lower extremity and usually end up obstructing the brachial artery. Sometimes plaques or an aneurysm in the subclavian or axillary arteries is the primary source of emboli. Embolization to the right arm is more common than to the left due to the vascular anatomy. For the 10% of patients with atherosclerosis and acute thrombosis as the main cause for their arm ischemia, the primary lesions are located in the brachiocephalic trunk or in the subclavian artery. Such pathologies are usually asymptomatic due to well-developed collaterals around the shoulder joint until thrombosis occurs, and they cause either micro- or macroembolization. Other less frequent causes of acute upper extremity ischemia are listed in Table 1

Table 1. Less common causes of acute upper extremity ischemia

Cause	Characteristics
Arteritis	Lesions in distal and proximal arteries
Buerger's disease	Digital ischemia in young heavy smokers
Coagulation disorders	Generalized or distal thrombosis
Raynaud's disease	Digital ischemia

Subclavian artery thrombosis is a condition in which the blood flow through the vessel is obstructed. The condition usually occurs secondary to some form of antecedent injury to the vessel, hypercoagulable state, or atherosclerotic changes. The condition is common in young athletic individuals who exert a significant amount of upper body activity. Sudden occlusion

from emboli followed by thrombosis of the artery is common in the population with signs of significant atherosclerotic disease.

The patient presenting with acute subclavian artery occlusion usually has a history of repetitive use and/or stress injury to the upper extremity on the affected side. A history of upper extremity claudication is common.

In situations in which the occlusion is secondary to atherosclerosis, acute thromboses of the artery are generally asymptomatic. In fact, in 9% of autopsy series, the left subclavian artery was either stenotic or occluded. If symptoms are present, upper extremity claudication on the affected side is most common. The patient may also present with dizziness, vertigo, imbalance, visual disturbances, or hemisensory dysfunction indicative of a subclavian steal syndrome. However, note that subclavian steal is observed on 2% of cerebral angiograms and causes no symptoms.

EMBOLISM

Embolism is considered the most common cause of acute arm ischemia (74%). The emboli are attributed to a variety of sources. Cardiac embolism is the most frequently reported cause of acute arm ischemia (58% to 93%) and atrial fibrillation is the usual etiology. Over the years the incidence of atrial fibrillation has remained fairly constant although the cause of fibrillation has changed from valvular heart disease as a result of rheumatic fever to ischemic heart disease and myocardial infarction. Rare causes include endocarditis, atrial myxoma, ventricular aneurysm, cardiac failure, and paradoxical embolism.

Non-cardiac embolism determines 1% to 32% of the acute arm emboli. Proximal upper limb stenosis caused by atherosclerotic plaque or external compression (cervical ribs) can result in thrombo-embolism or atheroembolism, which may cause large vessel occlusion or acute digital ischemia.

Other causes include atheroma in the aortic arch, primary subclavian aneurysm or aneurysm secondary to extrinsic compression from thoracic outlet syndrome, old fracture, and chronic trauma such as that from the use of crutches. Rarer sources are the proximal end of an occluded axillofemoral graft, arteritis, malignant emboli, and fibromuscular dysplasia. Despite a classic embolic presentation and operative findings, an embolic source may not be found in at least 12% of patients.

THROMBOSIS

Reports suggest that 5% of cases in population studies and 9% to 35% in surgical series are due to thrombosis. Jivegard et al. estimated that in patients who had embolectomy, the chance of thrombosis being the true cause was 5.5% in the arm. Most of the proximal arterial lesions that can cause emboli can also result in thrombosis, including atherosclerotic plaques, aneurysm, acute aortic dissection, and arteritis (Takayasu's disease).

Atherosclerosis in the upper extremity appears especially prominent in older men. The disease may be at the origin of the great vessels or distally in the axillary or brachial arteries. Aneurysms of the subclavian or axillary arteries may also result in upper extremity ischemia through two mechanisms. They may directly cause ischemic symptoms by thrombosis or by producing emboli that occlude the distal circulation (Raynaud's phenomenon).

Less common causes include arteritis from connective tissue disorders (scleroderma), radiation arteritis, hyperthrombotic conditions and thrombosis associated with malignancy or steroid use.

Clinical Presentation

Acute arm ischemia is usually apparent on the basis of the physical examination. The symptoms are often relatively discreet, especially early after onset. The explanation for this is the well developed collateral system circumventing the brachial artery around the elbow, which is the most common site for embolic obstruction. The “six Ps” – pain, pallor, paresthesia, paralysis, pulselessness, poikilothermia – are applicable also for acute arm ischemia, but coldness and color changes are more prominent than for the legs. Accordingly, the most common findings in the physical examination are a cold arm with diminished strength and disturbed hand and finger motor functions. Tingling and numbness are also frequent. The radial pulse is usually absent but is pounding in the upper arm proximal to the obstruction. Gangrene and rest pain appear only when the obstruction is distal to the elbow and affects both of the paired arteries in a finger or in the lower arm. Ischemic signs or symptoms suggesting acute digital artery occlusion in only one or two fingers, imply microembolization.

Diagnostics

Only the few patients with uncertain diagnosis, and those with a history and physical findings that indicates thrombosis, need additional work-up. Examples include patients with a history of chronic arm ischemia (arm fatigue, muscle atrophy, and microembolization) and

bruits over proximal arteries. Angiography should then be performed to reveal the site of the causing lesion. Duplex ultrasound is rarely needed to diagnose acute arm ischemia but may occasionally be helpful.

Management Before Treatment

Even though symptoms and examination findings may be so subtle that conservative treatment is tempting, surgical removal of the obstruction is almost always preferable. It has been suggested that in patients with a lower-arm blood pressure >60 mmHg embolectomy can be omitted, but such a strategy has not to our knowledge been evaluated systematically. In a patient series of nearly symptomless acute arm ischemia, which was left to resolve spontaneously or with anticoagulation as the only treatment, late symptoms developed in up to 45% of the cases. Surgical treatment is also fairly straightforward. It can be performed using local anesthesia and is associated with few complications. Very often an embolus is a manifestation of severe cardiac disease, so the patient's cardiopulmonary function should be assessed and optimized as soon as possible. Preoperative preparations include an electrocardiogram (ECG) and laboratory tests to guide anticoagulation treatment. Heparin treatment is started perioperatively and continued postoperatively in most patients.

Operation

Embolectomy

As mentioned previously, the most common site for embolic obstruction is the brachial artery.

The arm is placed on an arm table. We prefer to perform embolectomy using local anesthesia. Often a transverse incision placed over the palpable brachial pulse can be used. If proximal extension of the incision is required, this should be done in parallel with and dorsal to the dorsal aspect of the biceps muscle. It has to be kept in mind that 10–20% of patients may have a different brachial artery anatomy. The most common variation is a high bifurcation of the radial and ulnar arteries, and next in frequency is a doubled brachial artery.

An alternative location for embolectomy in the arm is to expose the brachial artery in the

bicipital groove. A longitudinal incision starting 10 cm above the elbow that is extended proximally is then used.

If it is hard to achieve a good inflow, a proximal lesion may cause the embolization or thrombosis. More complicated vascular procedures are then required to reestablish flow. The embolectomy attempt is then discontinued and the patient taken to the angiography suite for a complete examination. If practically feasible, an alternative is to obtain the angiogram in the operating room. Frequently, however, the preferred treatment is endovascular, and this is better done in the angiography suite. Occasionally the films will reveal a proximal obstruction that needs open repair. Examples of such are carotid-subclavian, subclavian-axillary, and axillary-brachial bypasses.

Embolectomy via the Brachial Artery

Brachial Artery in the Upper Arm

The incision is made along the posterior border of the biceps muscle; a length of 6–8 cm is usually enough.

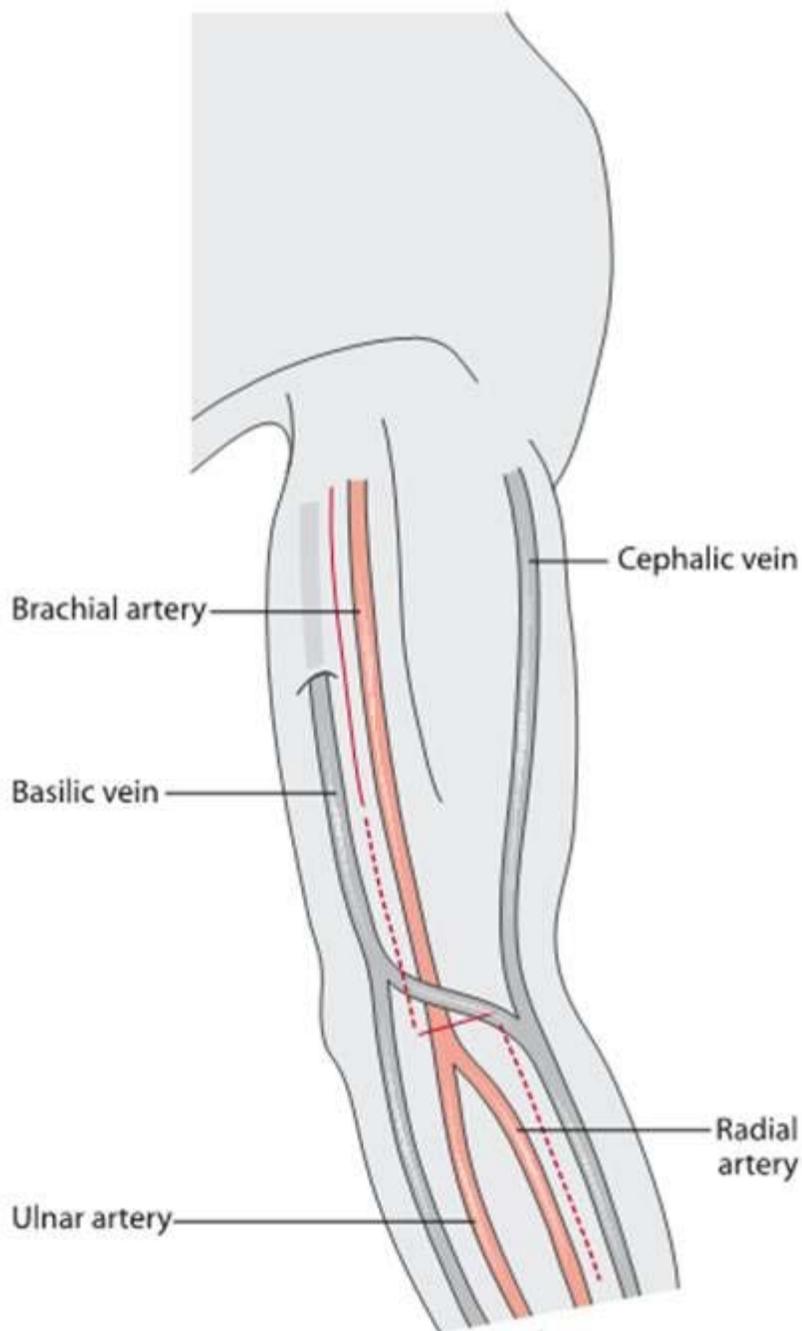


Fig. Transverse incision in the elbow for exposing the brachial artery and with possible elongations (dottedlines) when access to the ulnar and radial branches as well as to more proximal parts of the brachial artery is needed

The muscles are retracted medially and laterally, and the artery lies in the neurovascular bundle immediately below the muscles. The sheath is incised and the artery freed from the median nerve and the medial cutaneous nerve that surrounds it.

Brachial Artery at the Elbow

The incision is placed 2 cm below the elbow crease and should continue up on the medial side along the artery. If possible, veins transversing the wound should be preserved, but they can be divided if necessary for exposure. The medial insertion of the biceps tendon is divided entirely, and the artery lies immediately beneath it. By following the wound proximally, more of the artery can be exposed. If the origins of the radial and ulnar artery need to be assessed, the wound can be elongated distally on the ulnar side of the volar aspect of the arm. The median nerve lies close to the brachial artery, and it is important to avoid injuring it.

A transverse arteriotomy in the brachial artery is made as close as possible to the bifurcation of the ulnar and radial arteries. The embolectomy is performed in proximal and distal directions with #2 and #3 Fogarty catheters. Separate embolectomy in each branch should be done if technically simple. The Fogarty catheter otherwise slips down into the larger and straighter ulnar artery. The route of the catheter can be checked by palpation at the wrist level when the inflated balloon passes. On the other hand, restored flow in one of the arteries is usually enough for a result that is sufficient for adequate hand perfusion. The arteriotomy is closed with interrupted 6-0 sutures, and distal pulses and the perfusion in the hand are evaluated. If the result is inadequate – poor backflow after embolectomy, absence of pulse, a weak continuous-wave Doppler signal, and questionable hand perfusion – the arteriotomy should be reopened and intraoperative angiography performed.

Endovascular Treatment

Thrombolysis is as feasible for acute upper extremity ischemia as it is in the leg. The limited ischemia that often occurs after most embolic events because of the collateral network around the elbow also allows the time needed for planning and moving the patient to the angiosuite. The technique involves cannulation in the groin with a 7-French sheath. Long guide wires and catheters are required to reach the occluded site and makes identification of proximal lesions possible. A new arterial puncture in the brachial artery may be necessary for thrombolysis of distal occlusions. It can be argued that thrombolysis in spite of acceptable results, rarely is needed for treating this disease because open embolectomy can be performed under local anesthesia with good results and little surgical morbidity. The advantages with endovascular treatment are indeed limited. For patients in whom suspicion of thrombosis is

strong or when proximal lesions are likely, it should be attempted first. However, case series indicates that results of thrombolysis are inferior for forearm occlusions. In summary, thrombolysis is an alternative but has little to offer in reducing risk or improving outcome compared with embolectomy for most patients.

Management After Treatment

Patients usually regain full function of their hand immediately after the procedure, and postoperative regimens consist of anticoagulation and a search for the embolic source. The search for cardiac sources may advocate repeated ECGs, echocardiography, and duplex ultrasound of proximal arteries.

Results and Outcome

The number of salvaged arms after surgical intervention is very high, 90–95 %, and arm function is usually fully recovered. The remaining 5–10% represents patients with extensive thrombosis involving many vascular segments and most branches of the distal arteries. The postoperative mortality is around 10–40% in most patient series, reflecting that embolization often is a consequence of severe cardiac disease. Postoperative mortality is similar for thrombolysis to treat acute arm ischemia, while early technical success is slightly lower or similar. Less favorable results with thrombolysis are achieved when the distal arteries also are obstructed.

ACUTE LEG ISCHEMIA

Acute leg ischemia is associated with a great risk for amputation and death. The age of the patients is high, and to some extent acute leg ischemia can be considered an end-of-life disease. Patients' symptoms and the clinical signs of the afflicted leg vary. Sometimes grave ischemia immediately threatens limb viability, such as after a large embolization to a healthy vascular bed. Other times the symptoms are less dramatic, appearing as onset of rest pain in a patient with claudication. This is usually due to thrombosis of a previously stenosed artery.

Table 2. Incidence of acute leg ischemia

Country	Year	Surveyed population size	Population	Yearly incidence per 100,000 inhabitants
Sweden	1965-1983	1.5 million	All treated or amputated, >70 years old	125 (men) 150 (women)
USA	2000		All hospitalized	95
Sweden	1990-1994	2.0 million	All treated	60 (men) 77 (Women)
United Kingdom	1995	0.5 million	All diagnosed	14-16

It is the severity of ischemia that determines management and treatment. To minimize the risk for amputation or persistent dysfunction it is important to rapidly restore perfusion if an extremity is immediately threatened. When the leg shows signs of severe ischemia but is clearly viable, it is equally important to thoroughly evaluate and optimize the patient before any intervention is initiated. These basic management principles are generally applicable. Accordingly, we recommend “management by severity” rather than “management by etiology” (thrombosis versus embolus) but recognize that the latter can also be an effective strategy.

Embolism

Embolism is by far the most common cause of acute arm ischaemia, accounting for 74–100% of cases.

In the lower extremities, controversy exists regarding the ratio between arterial embolism and thrombosis, with different studies giving numbers ranging from 4:1 to 1:9.

The heart is invariably the most common origin of peripheral arterial emboli, and is responsible for 58–93% of cases. However, the pattern of the underlying heart disease has changed recently as the incidence of rheumatic valvular disease has decreased significantly

Cardiac Sources of Emboli

Nowadays, the most common sources of arterial emboli of cardiac origin are:

atrial fibrillation due to atherosclerotic heart disease, accounting for 32–75% of cases, followed by myocardial infarction with mural thrombi formation, which is responsible for 21–32% of peripheral emboli.

Less common cardiac sources of emboli are:

idiopathic dilated cardiomyopathy

prosthetic valves

rheumatic mitral valve disease

intracavitary cardiac tumours (mainly myxomas)

paradoxical embolization through an intracardiac defect, usually a patent foramen ovale

fungal or bacterial endocarditis.

Noncardiac Sources

Noncardiac sources of emboli are being identified with increasing frequency, at the expense of undetermined causes, the frequency of which has steadily decreased due to improvements in diagnostic methods. Noncardiac sources of emboli are nowadays found in 5–12% of patients, while in 9–12% the source of the emboli remains unknown.

Aneurysms are the most common noncardiac source of peripheral embolism, accounting for about 5% of distal emboli.

Ulcerated atherosclerotic plaques follow in order of frequency, carrying the risk of distal embolism from white thrombi adherent on their surface. Such emboli are usually sizeable, capable of obstructing major peripheral arteries.

A distinct variant of peripheral embolization due to an atherosclerotic plaque is atheroembolism, in which a portion of the plaque breaks off and undergoes embolization to peripheral arteries. Such emboli may evolve in three clinical forms:

1. The asymptomatic form, not diagnosed during the subject's lifetime and only recognized in autopsy studies.
2. A benign form such as blue toe syndrome or cutaneous livedo, with a spontaneous mild prognosis.
3. A diffuse multisystemic form with a very poor prognosis.

Cryptogenic Emboli

Despite complete diagnostic work-up, including complex investigations, the precise source of the emboli cannot be identified in 5–12% of cases. Such emboli are called cryptogenic and represent either the limited sensitivity of current diagnostic modalities or, in some cases, confusion with local thrombosis in situ.

Thrombosis

Thrombosis of an atherosclerotic artery or a vascular graft is another major cause of acute arterial occlusion of the extremities. As Virchow suggested in 1856, thrombus formation is the result of an interaction between an injured surface, stasis and the hypercoagulability of blood:

Arterial thrombosis most often develops at the points of severe stenosis. Since the course of atherosclerotic disease is chronic, a collateral network will have already developed and the clinical picture will be milder compared with arterial embolism. However, a thrombus can be formed in the absence of significant pre-existing stenosis, particularly when the surface of the plaque is ulcerated or after an intraplaque haemorrhage resulting in sudden arterial occlusion.

Low-flow conditions, such as congestive heart failure, hypovolaemia, hypotension of any cause or decreased blood flow due to a more proximal stenosis.

Hypercoagulable states, such as myeloproliferative disorders, hyperviscosity syndromes and coagulation disorders, may contribute to thrombosis of the diseased artery.

Other causes of arterial thrombosis include:

Arterial aneurysms, with the risk of thrombosis being higher the more peripherally the aneurysm is located.

Arterial by-pass graft thrombosis, which frequently induces acute limb-threatening ischaemia, as the graft has usually allowed collateral vessels to regress. Early graft failure, within the first postoperative month, is usually due to a technically suboptimal result, inappropriate indication or a transient episode of hypotension. Late graft failure, after 1 month, is secondary to intimal hyperplasia at the anastomotic sites or to progression of atherosclerotic disease.

Aortic dissection.

Fibromuscular dysplasia, occasionally involving the iliac arteries.

Cystic adventitial disease, usually affecting the popliteal artery and rarely the femoral.

Thromboangiitis obliterans, involving medium-sized muscular arteries.

Various arteritides, such as Takayasu's aortitis and giant cell arteritis.

Compartment syndrome.

Ehoracic outlet syndrome.

Popliteal entrapment syndrome.

Ergotism.

Magnitude of the Problem

It is difficult to find accurate incidence figures on acute leg ischemia. Data from some reports are given in Table 2. The numbers listed do not include conservatively treated patients or those whose legs were amputated as a primary procedure. The incidence increases with age and is seen with equal frequency in men and women. Regardless, the frequency indicates that it is a very common problem.

Pathogenesis

Acute leg ischemia is caused by a sudden deterioration of perfusion to the distal parts of the leg. While the abrupt inhibition of blood flow causes the ischemia, its consequences are variable because acute leg ischemia is multifactorial in origin. Hypercoagulable states, cardiac failure, and dehydration predispose the blood for thrombosis and make the tissue more vulnerable to decreased perfusion. Besides the fact that a healthy leg is more vulnerable than one accustomed to low perfusion, it is unknown what determines the viability of the tissue. The most important factor is probably the duration of ischemia. The type of tissue affected also influences viability. In the leg, the skin is more ischemia-tolerant than skeletal muscle.

Embolus and Thrombosis

The etiology of the occlusion is not what determines the management process. It is, however, of importance when choosing therapy. Embolus is usually best treated by embolectomy, whereas arterial thrombosis is preferably resolved by thrombolysis, percutaneous transluminal angioplasty (PTA), or a vascular reconstruction. The reason for this difference is that emboli often obstruct a relatively healthy vascular bed, whereas thrombosis occurs in an already diseased atherosclerotic artery. Consequently, emboli more often cause immediate threatening ischemia and require urgent restoration of blood flow. Thrombosis, on the other hand, occurs in a leg with previous arterial insufficiency with well-developed collaterals. In the latter case it is important not only to solve the acute thrombosis but also to get rid of the cause. It must be kept in mind that emboli can be lodged in atherosclerotic arteries as well, which then

makes embolectomy more difficult.

Table 3 summarizes typical findings in the medical history and physical examination that suggest thrombosis or embolism. Many risk factors, such as cardiac disease, are common for both embolization and thrombosis. Atrial fibrillation and a recent (less than 4 weeks) myocardial infarction with intramural thrombus are the two dominating sources for emboli (80–90%). Other possible origins are aneurysms and atherosclerotic plaques located proximal to the occluded vessel. The latter are often associated with microembolization (discussed later) but may also cause larger emboli.

Table 3. History and clinical findings differentiating the etiology of acute ischemia

Thrombosis	Embolism
Previous claudication	No previous symptoms of arterial insufficiency
No source of emboli	Obvious source of emboli (arterial fibrillation, myocardial infarction)
Long history (days to weeks)	Sudden onset (hours to days)
Less severe ischemia	Severe ischemia
Lack of pulses in the contralateral leg	Normal pulses in the contralateral leg
Positive signs of chronic ischemia	No signs of chronic ischemia

Plaque rupture, immobilization, and hypercoagulability are the main causes of acute thrombosis. Severe cardiac failure, dehydration, and bleeding are less common causes. Hypoperfusion due to such conditions can easily turn an extremity with longstanding slightly compromised perfusion into acute ischemia.

Location of embolic obstruction

Artery	%
Femoral	37–49
Iliac	20–27
Popliteal	12–16
Upper extremity:	15–18
Brachial	60
Axillary	25
Radial	8
Subclavian	7
Tibial	4–6
Aortic saddle	3–6

Clinical Presentation

Medical History

The typical patient with acute leg ischemia is old and has had a recent myocardial infarction. He or she describes a sudden onset of symptoms – a few hours of pain, coldness, loss of sensation, and poor mobility in the foot and calf. Accordingly, all signs of threatened leg viability are displayed. The event is most likely an embolization, and the patient needs urgent surgery. Unfortunately, such patients are unusual among those who are admitted for acute leg ischemia. The history is often variable, and sometimes it is difficult to decide even the time of onset of symptoms. It is important to obtain a detailed medical history to reveal any underlying conditions or lesions that may have caused the ischemia. Moreover, identifying and treating comorbidities may improve the outcome after surgery or thrombolysis.

Clinical Signs and Symptoms

The symptoms and signs of acute ischemia are often summarized as the “five Ps”: pain,

pallor, pulselessness, paresthesia, and paralysis. Besides being helpful for establishing diagnosis, careful evaluation of the five Ps is useful for assessing the severity of ischemia. Sometimes a sixth P's is used – poikilothermia, meaning a low skin temperature that does not vary with the environment.

Pain: For the typical patient, as the one described above, the pain is severe, continuous, and localized in the foot and toes. Its intensity is unrelated to the severity of ischemia. For instance, it is less pronounced when the ischemia is so severe that the nerve fibers transmitting the sensation of pain are damaged. Patients with diabetes often have neuropathy and a decreased sensation of pain.

Pallor: The ischemic leg is pale or white initially, but when ischemia aggravates the color turns to cyanotic blue. This cyanosis is caused by vessel dilatation and desaturation of hemoglobin in the skin and is induced by acidic metabolites in combination with stagnant blood flow. Consequently, cyanosis is a graver sign of ischemia than pallor.

Pulselessness: A palpable pulse in a peripheral artery means that the flow in the vessel is sufficient to give a pulse that is synchronous with vessel dilatation, which can be palpated with the fingers. In general, palpable pulses in the foot therefore exclude severe leg ischemia. When there is a fresh thrombus, pulses can be felt in spite of an occlusion, so this general principle must be applied with caution. Palpation of pulses can be used to identify the level of obstruction and is facilitated by comparing the presence of pulses at the same level in the contralateral leg.

When the examiner is not convinced that palpable pulses are present, distal blood pressures must be measured. It is prudent to always measure the ankle blood pressure. This is a simple way to verify ischemia and the measurement can be used to grade the severity and serve as a baseline for comparison with repeated examinations during the course of treatment. (This will be discussed further later.) The continuous-wave (CW) Doppler instrument does not give information about the magnitude of flow because it registers only flow velocities in the vessel. Therefore, an audible signal with a CW Doppler is not equivalent to a palpable pulse, and a severely ischemic leg can have audible Doppler signals.

Paresthesia: The thin nerve fibers conducting impulses from light touch are very sensitive to ischemia and are damaged soon after perfusion is interrupted. Pain fibers are less ischemia-sensitive. Accordingly, the most precise test of sensibility is to lightly touch the skin with the fingertips, alternating between the affected and the healthy leg. It is a common mistake to believe that the skin has been touched too gently when the patient actually has impaired

sensitivity. The examiner then may proceed to pinching and poking the skin with a needle. Such tests of pain fibers evaluate a much later stage of ischemic damage. The anatomic localization of impaired sensation is sometimes related to which nerves are involved. Frequently, however, it does not follow nerve distribution areas and is circumferential and most severe distally. Numbness and tingling are other symptoms of ischemic disturbance of nerve function.

Paralysis: Loss of motor function in the leg is initially caused by ischemic destruction of motor nerve fibers and at later stages the ischemia directly affects muscle tissue. When palpated, ischemic muscles are tender and have a spongy feeling. Accordingly, the entire leg can become paretic after proximal severe ischemia and misinterpreted as a consequence of stroke. Usually paralysis is more obscure, however, presenting as a decreased strength and mobility in the most distal parts of the leg where the ischemia is most severe. The most sensitive test of motor function is to ask the patient to try to move and spread the toes. This gives information about muscular function in the foot and calf. Bending the knee joint or lifting the whole leg is accomplished by large muscle groups in the thigh that remain intact for a long time after ischemic damage in the calf muscle and foot has become irreversible.

Evaluation of Severity of Ischemia

Classification

When a patient has been diagnosed to have acute leg ischemia, it is extremely important to evaluate its grade. Ischemic severity is the most important factor for selecting a management strategy, and it also affects treatment outcome. Classification according to severity must be done before the patient is moved to the floor or sent to the radiology department. We have found that the simple classification suggested by the Society for Vascular Surgery ad hoc committee (1997) is helpful for grading. It is displayed in Table 4.

Table 4. Categories of acute ischemia

		Sensibility	Motor function	Arterial Doppler signal	Venous Doppler signal
I	Viable	Normal	Normal	Audible (>30 mmHg)	Audible
IIa	Marginally threatened	Decreased or normal in the toes	Normal	Not audible	Audible
IIb	Immediately threatened	Decreased, not only in the toes	Mildly to moderately affected	Not audible	Audible
IV	Irreversibly damaged	Extensive anesthesia	Paralysis and rigor	Not audible	Not audible

Viable Leg

As indicated in Figure 2, a viable ischemic leg is not cyanotic, the toes can be moved voluntarily, and the ankle pressure is measurable. The rationale for choosing these parameters is that cyanosis and impaired motor function are of high prognostic value for outcome. The limit of 30mmHg for the ankle pressure is not important per se but is a practical limit useful to make sure that it is the arterial, and not a venous, pressure that has been measured. The dorsalis pedis, posterior tibial arteries, or branches from the peroneal artery can be insonated. The latter can be found just ventral to the lateral malleolus. If no audible signal is identified in any of these arteries or if there only is a weak signal that disappears immediately when the tourniquet is inflated, the ankle blood pressure should be recorded as zero. It is important to rely on the obtained results and not assume that there is a signal somewhere that is missed due to inexperience. Qualitative analysis of the Doppler signal is seldom useful when evaluating acute leg ischemia.

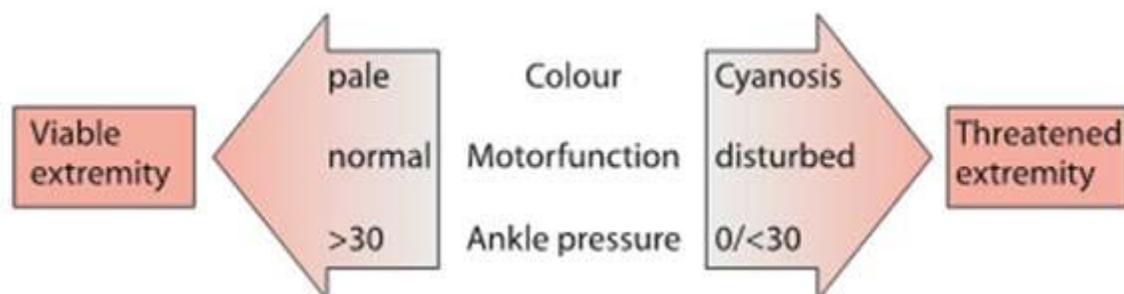


Fig. 2. Simplified algorithm to support the management of acute leg ischemia

Threatened Leg

As shown in Table 4, the threatened leg differs from the viable one in that the sensibility is impaired and there is no measurable ankle blood pressure. The threatened limb is further separated into marginally threatened and immediately threatened by the presence or absence of normal motor function. The threatened leg differs from the irreversibly damaged leg by the quality of the venous Doppler signal. In the irreversibly damaged leg, venous blood flow is stagnant and inaudible.

Management Strategy

A viable leg does not require immediate action and can be observed in the ward. A threatened leg needs urgent operation or thrombolysis. The latter is more time-consuming and recommended for the marginally threatened leg. The immediately threatened leg must be treated as soon as possible, usually with embolectomy or a vascular reconstruction. Irreversible ischemia is quite unusual but implies that the patient's leg cannot be saved. Figure 2 is intended to show a simplified algorithm to further support the management of acute leg ischemia.

Diagnostics

A well-conducted physical examination is enough to confirm the diagnosis of acute leg ischemia, determine the level of obstruction, and evaluate the severity of ischemia. When the leg is immediately threatened, further radiologic examinations or vascular laboratory tests should not under any circumstances delay surgical treatment. When the extremity is viable or marginally threatened, angiography should be performed. Duplex ultrasound is of limited value for evaluating acute leg ischemia and angiography is recommended for almost all patients in these two groups. If angiography is not available or if examination of the patient has verified that emboli is the cause and probably is best treated by embolectomy, angiography can be omitted. This situation is rare, however.

The arteriogram provides an anatomical map of the vascular bed and is very helpful in discriminating embolus and thrombosis. The former is essential for planning the surgical procedure, and the latter may be of importance for selecting the treatment strategy.

An arteriogram representing an embolus is shown in Fig. 3.

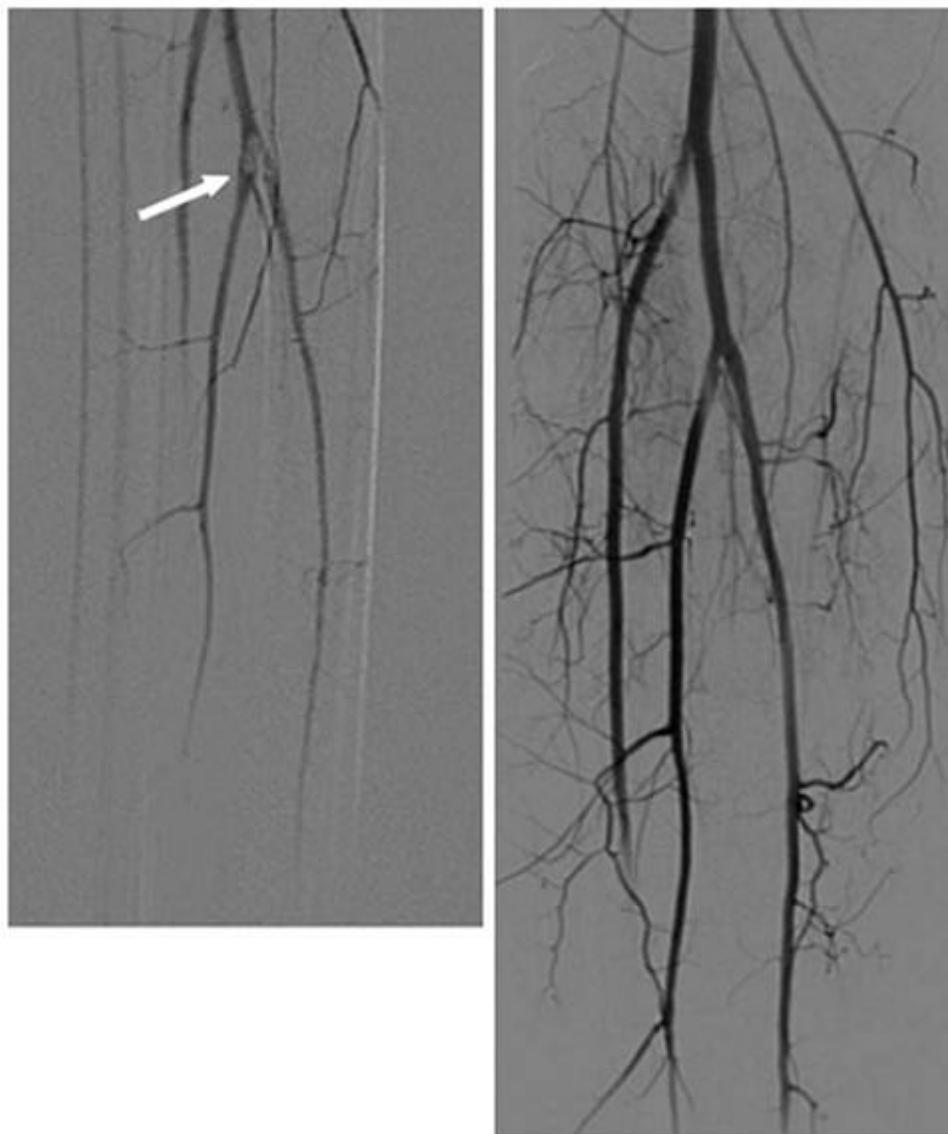


Fig. 3. Embolus lodged at the origins of the calf vessels (arrow).
Angiograms display ilms before and after thrombolysis

Angiographic signs of embolism are an abrupt, convex start of the occlusion and lack of collaterals. Thrombosis is likely when the arteriogram shows well-developed collaterals and

atherosclerotic changes in other vascular segments.

For most patients with viable and marginally threatened legs the diagnostic angiography is followed by therapeutic thrombolysis right away.

Angiography can be performed during daytime when qualified radiology staff is available. The patient should be optimized according to the recommendations given in the next section. Before angiography it is important to keep the patient well hydrated and to stop administration of metformin to reduce the risk of renal failure. Disturbances in coagulation parameters may interfere with arterial puncture and must also be checked before the investigation. The information is also important as baseline values in case of later thrombolysis.

The groin of the contralateral leg is the preferred puncture site for diagnostic angiography. A second antegrade puncture can be done in the ischemic extremity if thrombolysis is feasible.

Management and Treatment

Management Before Treatment

Viable Leg

If the leg is viable the patient is admitted for observation. A checklist of what needs to be done in the emergency department follows below:

1. Place an intravenous (IV) line.

2. Start infusion of fluids. Because dehydration is often a part of the pathogenic process, Ringer's acetate is usually preferred. Dextran is another option that also is beneficial for blood rheology.

3. Draw blood for hemoglobin and hematocrit, prothrombin time, partial thromboplastin time, complete blood count, creatinine, blood urea nitrogen, fibrinogen, and antithrombin. Consider the need to type and cross-match blood.

4. Order an electrocardiogram (ECG).

5. Administer analgesics according to pain intensity. Opiates are usually required (morphine 2.5–10mg IV).

6. Consider heparinization, especially if only Ringer's acetate is given. Heparin treatment should be postponed until after surgery if epidural anesthesia is likely.

Repeated assessments of the patient's clinical status are mandatory in the intensive care unit and when the patient has been moved to the ward. The time interval depends on the severity of ischemia and the medical history. This examination includes evaluating skin color, sensibility, and motor function as well as asking the patient about pain intensity.

Dextran is administered throughout the observation period. The risk for deterioration of heart failure due to dextran treatment is substantial and for patients at risk the volume load must be related to the treatment's expected possible benefits. For such patients it is wise to reduce the normal dose of 500ml in 12h to 250ml. Another option is to prolong the infusion time to 24h.

Heparin only or in combination with dextran is recommended when patients do have an embolic source or a coagulation disorder. There are two ways to administer heparin. The first is the standard method, consisting of a bolus dose of 5,000 units IV followed by infusion of heparin solution (100units/ml) with a drop counter. The dose at the start of infusion should be 500 units of heparin per kilogram of body weight per 24h. The dose is then adjusted according to activated partial thromboplastin time (APTT) values obtained every 4h. The APTT value should be 2–2.5 times the baseline value.

Low molecular weight heparin administered subcutaneously twice daily is the other option. A common dose is 10,000 units/day but it should be adjusted according to the patient's weight.

It is important to optimize cardiac and pulmonary function while monitoring the patient. Hypoxemia, anemia, arrhythmia, and hypotension worsen ischemia and should be abolished if possible. A cardiology consult is often needed.

The above-mentioned treatment regime of rehydration, anticoagulation, and optimization of cardiopulmonary function often improves the ischemic leg substantially. Frequently this is enough to sufficiently restore perfusion in the viable ischemic leg, and no other treatments are

needed. If no improvement occurs, angiography can be performed during the daytime, followed by thrombolysis, PTA, or vascular reconstruction.

Threatened Leg

If the leg is immediately threatened, the patient is prepared for operation right away. This includes the steps listed above for the viable leg, including contact with an anesthesiologist. When there is no cyanosis and motor function is normal – that is, the extremity is only marginally threatened – there is time for immediate angiography followed by thrombolysis or operation. An option is cautious monitoring and angiography as soon as possible.

Before starting the operation, the surgeon needs to consider the risk for having to perform a complete vascular reconstruction. It is probable that a bypass to the popliteal artery or a calf artery will be needed to restore circulation. If thrombosis is the likely cause and the obstruction is distal (a palpable pulse is felt in the groin but not distally), a bypass may also be required even when embolization is suspected.

Operation

Exposure of Different Vessel Segments in the Leg

Femoral Artery in the Groin, Fig. 4 A, B, C.

a A longitudinal skin incision starting 1–2 cm cranial to the inguinal skin fold and continued lateral to the artery is used to avoid the inguinal lymph nodes. A common mistake is to place the incision too far caudally, which usually means the dissection is taking place distal to the deep femoral.

b The dissection is continued sharply with the knife straight down to the fascia lateral to the lymph nodes and is then angulated 90° medially to reach the area over the artery. It should then be palpable. Lymph nodes should be avoided to minimize the risk for infection and development of seroma. The fascia is incised, and the anterior and lateral surfaces of the artery are approached.

c At this stage the anatomy is often unclear regarding the relation of branches to the

common femoral artery. Encircle the exposed artery with a vessel-loop, and gently lift the artery. Continue dissection until the bifurcation into superficial and deep femoral artery is identified. Its location varies from high up under the inguinal ligament up to 10 cm further down. At this stage, the surgeon must decide whether exposure and clamping of the common femoral are enough. This is usually the case for proximal control in trauma distally in the leg. In acute ischemia it is more common that the entire bifurcation needs to be exposed.

During the continued dissection, attention must be given to important branches that should be controlled and protected from iatrogenic injuries. These are, in particular, the circumflex iliac artery on the dorsal aspect of the common femoral artery and the deep femoral vein crossing over the anterior aspect of the deep femoral artery just after its bifurcation. To provide a safe and good exposure of the deep femoral to a level below its first bifurcation, this vein must be divided and suture-ligated. Partial division of the inguinal ligament is occasionally needed for satisfactory exposure.

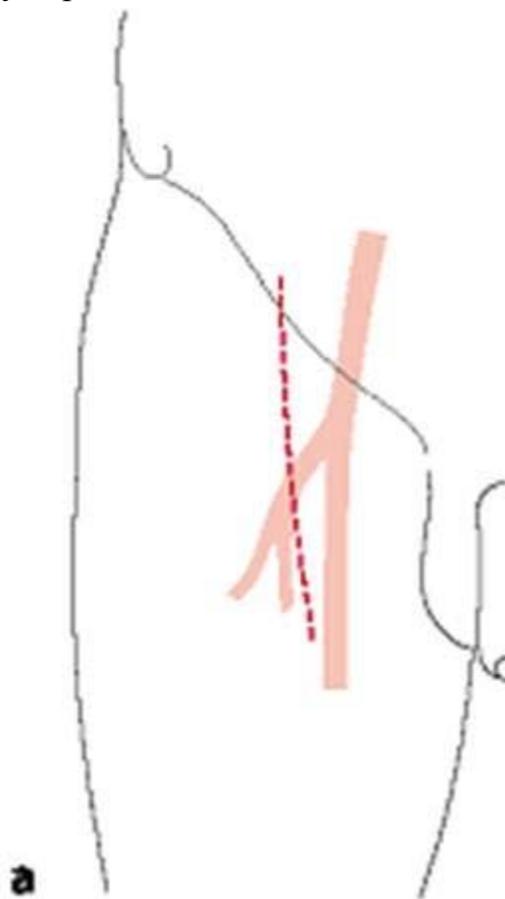


Fig. 4 A. Exposure of femoral artery in the groin

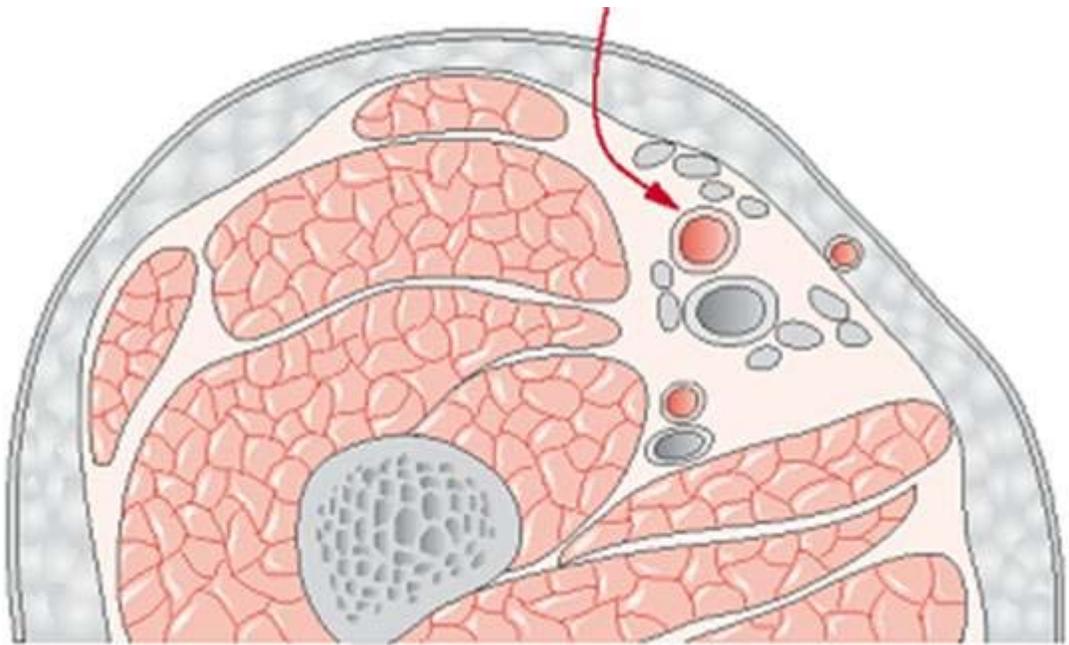


Fig. 4 B. Exposure of femoral artery in the groin

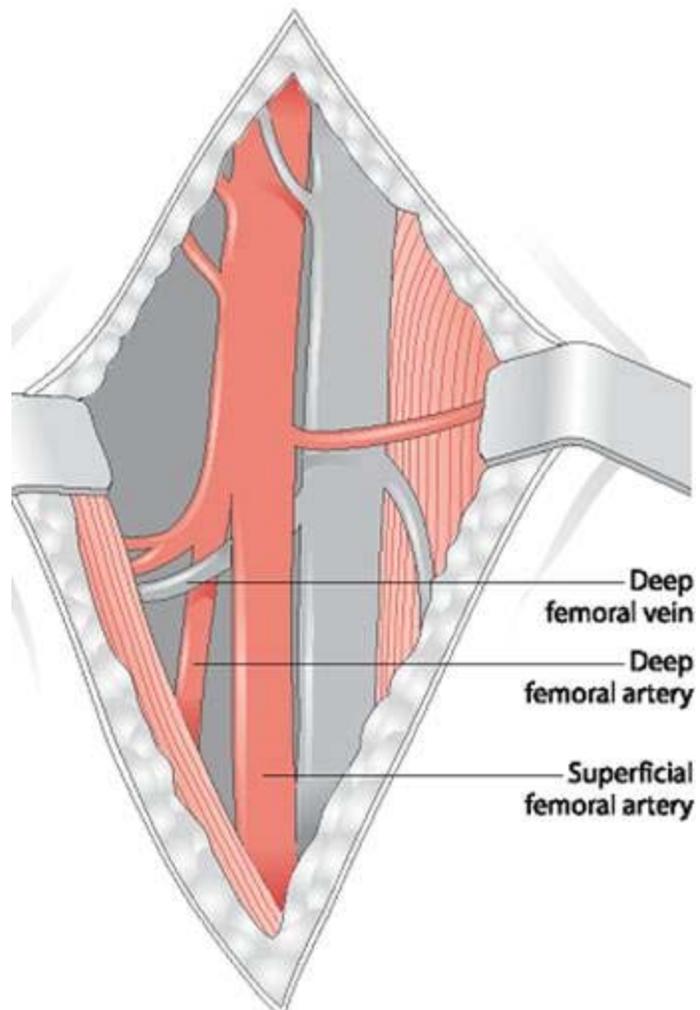


Fig. 4 C. Exposure of femoral artery in the groin

Superficial Femoral Artery, Fig. 5

A skin incision is made along the dorsal aspect of the sartorius muscle at a midhigh level. It is important to avoid injuries to the greater saphenous vein, which usually is located in the posterior flap of the incision. The incision can be elongated as needed. After the deep fascia is opened and the sartorius muscle is retracted anteriorly, the femoral artery is found and can be mobilized. Division of the adductor tendon is sometimes required for exposure.

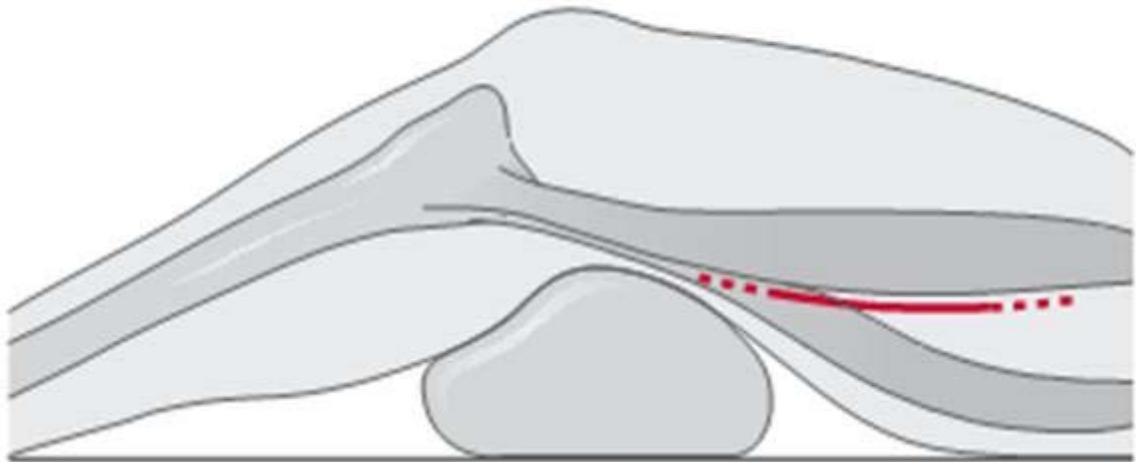


Fig. 5. Incision for exposure of the superficial femoral artery

Popliteal Artery Above the Knee, Fig. 6 A, B.

a The knee is supported on a sterile, draped pillow. The skin incision is started at the medial aspect of the femoral condyle and follows the anterior border of the sartorius muscle 10–15 cm in a proximal direction. Protect the greater saphenous vein and the saphenous nerve during dissection down to the fascia. After dividing the fascia longitudinally, continue the dissection in the groove between the sartorius and gracilis muscles, which leads to the fat in the popliteal fossa.

b The popliteal artery and adjacent veins and nerve are then, without further division of

muscles, easily found and separated in the anterior aspect of the fossa.

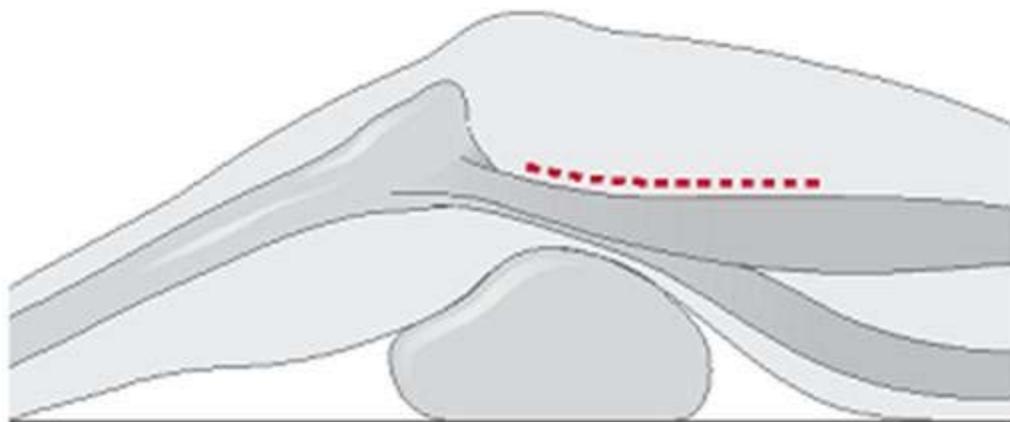


Fig. 6 A. Exposure of popliteal artery above the knee

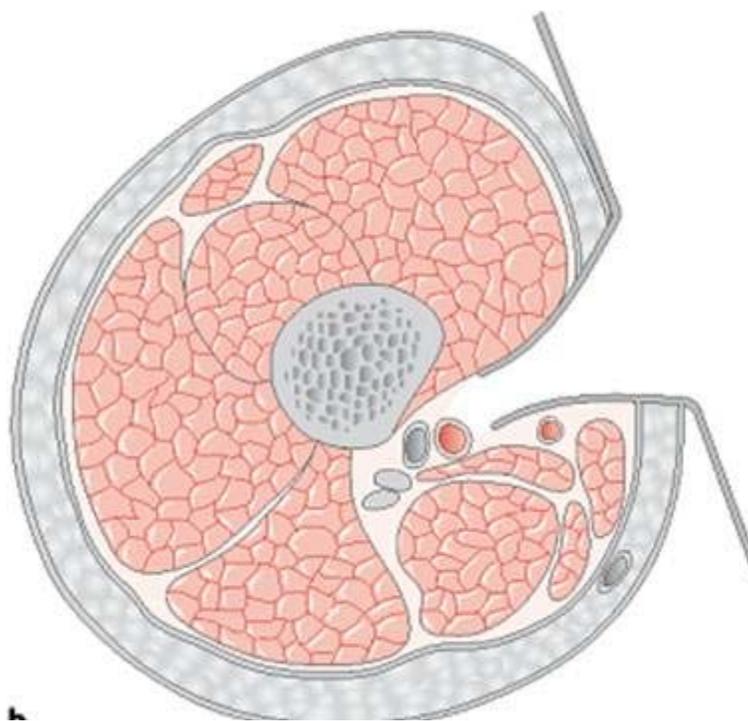


Fig. 6 B. Exposure of popliteal artery above the knee

Popliteal Artery Below the Knee, Fig. 7 A,B.

a A sterile pillow or pad is placed under the distal femur. The incision is placed 1 or 2 cm posterior to the medial border of the tibia, starting at the tibial tuberosity and extending 10–12 cm distally. Subcutaneous fat and fascia are sharply divided, with caution to the greater saphenous vein.

b The popliteal fossa is reached by retracting the gastrocnemius muscle dorsally. The deep fascia is divided and the artery usually easier to identify. Occasionally, pes anserinus must be divided for adequate exposure. The popliteal artery is often located just anterior to the nerve and in close contact with the popliteal vein and crossing branches from concomitant veins. If it is necessary to expose the more distal parts of the popliteal artery, the soleus muscle has to be divided and partly separated from the posterior border of the tibia.

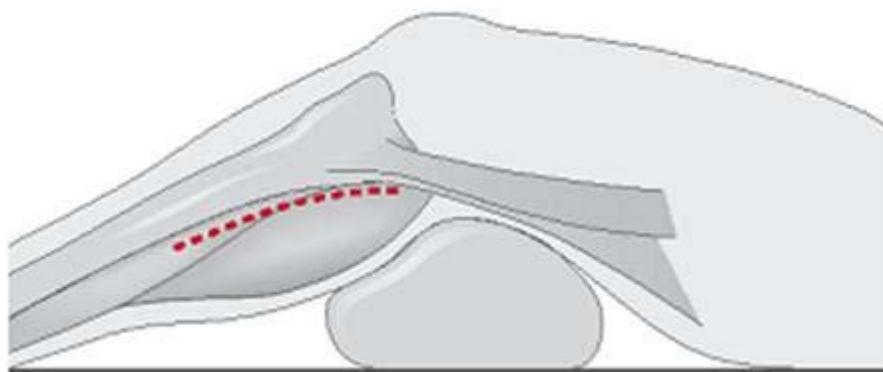


Fig. 7 A. Exposure of popliteal artery below the knee

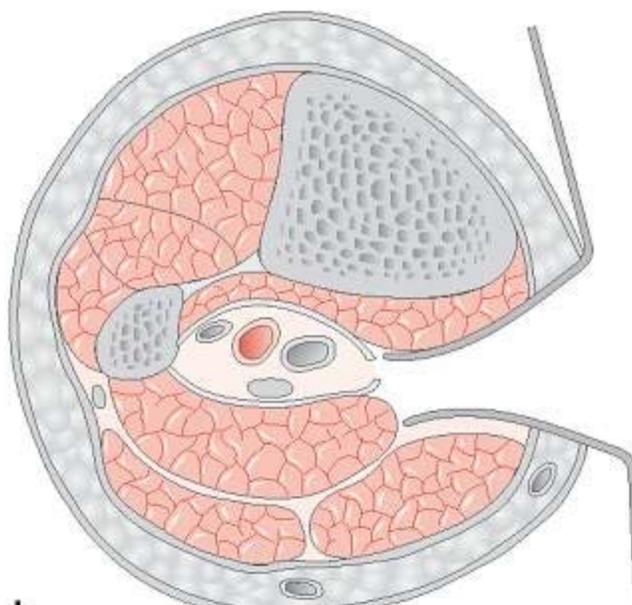


Fig. 7 B. Exposure of popliteal artery below the knee

Embolectomy

It is beyond the scope of this book to cover the technique for vascular reconstructions. But because embolectomy from the groin with balloon catheters (known as Fogarty catheters) is one of the most common emergency vascular operations in a general surgical clinic and may be performed by surgeons not so familiar with vascular surgery.

TECHNICAL TIPS

Embolectomy

Use an operating table that allows x-ray penetration. Local anesthesia is used if embolus is likely and the obstruction seems to be in the upper thigh or in pelvic vessels (no pulse in the groin). Make a longitudinal incision in the skin, and identify and expose the common, superficial, and deep femoral arteries. If the common femoral artery is soft-walled and free from arteriosclerosis – especially if a pounding pulse is felt proximal to the origin of the deep femoral artery – an embolus located in its bifurcation is likely. Make a short transverse arteriotomy including almost half the circumference. Place the arteriotomy only a few mm proximal to the origin of the profunda artery so it can be inspected and cannulated with ease. In most other cases, a longitudinal arteriotomy is preferable because it allows elongation and can be used as the site for the inflow anastomosis of a bypass. For proximal embolectomy, a #5 catheter is used.

Before the catheter is used the balloon should be checked by insufflation of a suitable volume of saline. Check the position of the lever of the syringe when the balloon is starting to fill, which gives a good idea of what is happening inside the artery. Wet the connection piece for the syringe to get a tight connection. It is smart to get external markers of the relationship between the catheter length and important anatomical structures; for example, the aortic bifurcation (located at the umbilicus level), the trifurcation level (located approximately 10 cm

below the knee joint), as well as the ankle level. The catheters have centimeter grading, which simplifies the orientation.

It is common for the embolus to already be protruding when the arteriotomy is done and a single pull with the catheter starting with the tip in the iliac artery is enough to ensure adequate inflow. This means that a strong pulse can be found above the arteriotomy, and a pulsatile heavy blood flow comes through the hole. For distal clot extraction, a #3 or #4 catheter is recommended. A slight bending of the catheter tip between the thumb and index finger might, in combination with rotation of the catheter, make it easier to pass down the different arterial branches (Fig. 8).

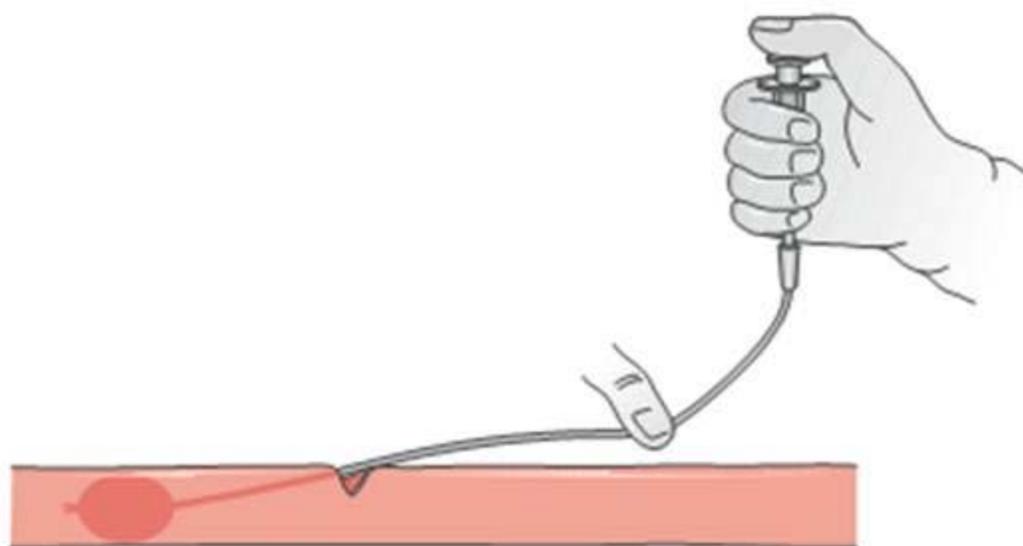


Fig. 8. Use of Fogarty catheter for embolectomy. Note that withdrawal is parallel to the artery

When the catheter is inserted into the artery and while the surgeon is working with it, hemostasis of the arteriotomy is achieved by a vessel-loop or by a thumb–index finger grip over the artery and the catheter. In a typical case, an embolus, including a possible secondary thrombus, can be passed relatively easily or with only slight resistance. If a major part of the catheter can be inserted the tip will be located in one of the calf arteries, most probably the posterior tibial artery or the peroneal artery. The balloon is insufflated simultaneously as the catheter is slowly withdrawn, which makes it easier to get a feeling for the dynamics and to not apply too much pressure against the vascular wall.

A feeling of “touch” is preferable, but a feeling of “pull” against the vascular wall

should be avoided.

To get the right feeling the same person needs to hold the catheter, pull it, and insufflate the balloon at the same time. To avoid damage in the arteriotomy, the direction of withdrawal should be parallel with the artery .

When the catheter is withdrawn it moves into larger segments of the artery and has to be successively insufflated until it reaches the arteriotomy. The reverse is, of course, valid when the embolectomy is done in a proximal direction. The thromboembolic masses can be suctioned or pulled out with forceps, and the arteriotomy should be inspected to be clean from remaining materials before the catheter is reinserted. The maneuver should be repeated until the catheter has been passed at least once without any exchange of thromboembolic materials and until there is an acceptable backflow from the distal vascular bed.

Depending on the degree of ischemia and collaterals, the backflow is, however, not always brisk.

If a catheter runs into early and hard resistance, this might be due to previously occluded segment that forced the catheter into a branch. It should then be withdrawn and reinserted, using great caution to avoid perforation. If the resistance cannot be passed and if acute ischemia is present, angiography should always be considered to examine the possibility of a vascular reconstruction.

Besides performing embolectomy in the superficial femoral, popliteal, and calf arteries, the deep femoral artery must be checked for an obstructing embolus or clot that needs to be extracted. Separate declamping of the superficial femoral and deep femoral arteries to check the backflow is the best way to do this. Remember the possibility that backflow from the distal vascular bed after embolectomy might emanate from collaterals located proximal to distally located clots. Back flow does not always assure that the peripheral vascular bed is free from further embolic masses. A basic rule is that every operation should be completed with intraoperative angiography to ensure good outflow and to rule out remaining emboli and secondary thrombus. To dissolve small amounts of remaining thrombus local infusion of 2–4 cc recombinant tissue plasminogen activator (rtPA) can be administered before the angiography catheter is pulled out.

Finally, the arteriotomy is closed. If necessary a patch of vein or synthetic material is used to avoid narrowing of the lumen. As mentioned before, the embolectomy procedure includes intraoperative angiography. If this examination indicates significant amounts of

emboli remaining in the embolectomized arteries or if the foot still appears as being inadequately perfused after the arteriotomy is closed, other measures need to be taken. If there are remaining emboli in the superficial femoral or popliteal arteries, another embolectomy attempt from the arteriotomy in the groin can be made. Clots, if seen in all the calf arteries, need to be removed through a second arteriotomy in the popliteal artery. This is done by a medial incision below the knee; note that local anesthesia is not sufficient for this. It is usually necessary to restore flow in two, or occasionally in only one, of the calf arteries.

Embolectomy at the popliteal level is the first treatment step when ischemia is limited to the distal calf and foot and when there is a palpable pulse in the groin or in the popliteal fossa.

Thrombosis

The preliminary diagnosis of embolus must be reconsidered if the exposed femoral artery in the groin is hard and calcified. In most situations, clot removal with Fogarty catheters will then fail. It is usually difficult or even impossible to pass the catheter distally, indicating the presence of stenoses or occlusions. Even if the embolectomy appears successful, early reocclusion is common. Such secondary thrombosis is usually more extensive and will aggravate the ischemia. Accordingly, angiography should be considered as the first step if the femoral artery is grossly arteriosclerotic and if it is hard to pass the catheter down to the calf level. It will confirm the etiology and reveal whether a bypass is required and feasible. Vascular reconstruction in acute leg ischemia is often rather difficult and experience in vascular surgery is required.

Intraoperative angiography

With the proximal clamp in position a 5 or 8 French baby-feeding catheter is inserted into the arteriotomy. The tip of the catheter is placed 5 cm into the superficial femoral artery and distal control around it is achieved with a vessel-loop. Contrast for intravascular use containing 140–300 mg iodine/ml is infused with a 20 cc syringe connected to a three-way valve. Heparinized Ringer's or saline (10 units/ml heparin) is flushed through the catheter before and after contrast injection to prevent thrombosis in the occluded vascular bed. If the patient is suspected to have renal failure, the amount of contrast used is kept at a minimum. Angled projections can be obtained without moving the C-arm by rotating the patient's foot.

The use of contrast in the Fogarty catheter balloon during fluoroscopy allows the calf vessel into which the catheter slides to be identified. The technique for intraoperative angiography is also a prerequisite for interoperative use of endovascular treatment options such as angioplasty (Fig. 9).

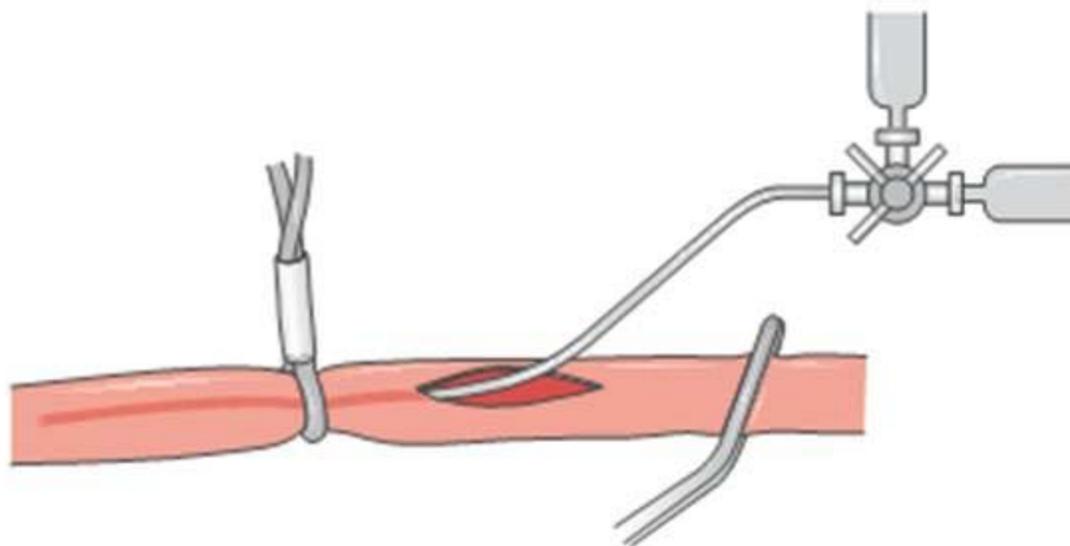


Fig. 9. Intraoperative angiography

Thrombolysis

Thrombolysis is performed in the angiosuite. A consultation with a specialist in coagulation disorders or a specialist in vascular medicine is sometimes needed to discuss possible problems related to coagulation before the procedure.

Treatment is usually directed toward resolving a fresh, thrombotic occlusion, but emboli and thrombi several weeks old can also be successfully lysed. The procedure starts with a diagnostic angiography via contralateral or antegrade ipsilateral arterial punctures. If thrombolytic treatment is decided the procedure continues right away, and the tip of a pulse-spray catheter is placed in the thrombus. The lytic agent is then forcefully injected directly into it to cause fragmentation. The primary choices for lytic agent are recombinant tissue plasminogen activator (rtPA) or urokinase. Because of the risk of allergic reactions, streptokinase should be avoided. Intermittent injections of 1 ml every 5–10 min to a total dose of 10–20 ml rtPA over 1–3 h is followed by angiographic control of the result. If the thrombus is completely lysed any underlying lesion is treated. If thrombus still remains, the rtPA infusion

is continued slowly over 6–12h with 1mg/h. If the initial thrombolysis fails, a variety of mechanical catheters can be used to try to further dissolve and aspirate the thrombus. Examples include the AngioJet and the Amplatz.

Because of the risk of bleeding and systemic complications, and also because the ischemic leg may deteriorate, careful monitoring during continued thrombolysis is necessary. This is best done in an intensive care or step-down unit. The patient should be kept supine in bed throughout the procedure. During this time the other measures suggested for optimizing coagulation and central circulation are continued. It is also necessary to check fibrinogen concentration to make sure the value does not decline to <1.0 mg/ml. Below this level surgical hemostasis is insufficient and the infusion should be stopped. Angiographic control of the result is performed afterwards, usually the following morning, and occasionally during the slow infusion to check the effect and allow repositioning of the catheter. The part of the thrombus surrounding the catheter is lysed first, which is why it often is beneficial to advance the catheter further into the thrombus after a few hours.

Finally, the lesion that caused the thrombosis is treated with angioplasty. To avoid unnecessary bleeding from the puncture site, the fibrinogen concentration is checked again before the sheath is withdrawn to ensure that the level exceeds 1.0 mg/ml.

Management After Treatment

Anticoagulation

Patients with embolic disease caused by cardiac arrhythmia or from other cardiac sources proven by ECG, medical history, or clinical signs should be anticoagulated postoperatively. Treatment regimens described previously are employed, followed by treatment with coumadin. Anticoagulation has no proven positive effect for the prognosis of the ischemic leg but is administered to reduce the risk of new emboli. The patient's abilities to comply with treatment and the risk for bleeding complications have to be weighed against the benefits. If the source of the emboli is not clear, it should be investigated. Findings of atrial fibrillation and heart thrombus can then be treated. If the ECG is normal, echocardiography is ordered to search for thrombus and valve deficiencies. If the left atrium is a likely embolic source, transesophageal echocardiography may be indicated.

When the etiology of leg ischemia is uncertain it is difficult to give general advice. There is no scientific evidence that long-term postoperative anticoagulation reduces the risk of

reocclusion or influences patient survival. Continued treatment with dextran or low molecular weight heparin is recommended at least during hospitalization.

If hypercoagulable states are suspected the patient needs to be worked up during the postoperative period to reduce the risk of reocclusion. Examples are patients with hyperhomocysteinemia, who may be treated with folates, and patients with antiphospholipid antibodies, who need coumadin and salicylic acid.

Reperfusion Syndrome

Patients treated for severe acute leg ischemia are at risk of developing reperfusion syndrome. This occurs when ischemic muscles are reperfused and metabolites from damaged and disintegrated muscle cells are spread systemically. A part of this process consists of leakage of myoglobin; it may be nephrotoxic and colors the urine red. The metabolites also affect central circulation and may cause arrhythmia and heart failure. The risk for reperfusion syndrome is higher when occlusions are proximal and the affected muscle mass is large. One example is saddle emboli located in the iliac bifurcation. The risk is also higher when the ischemia time is longer than 4–6 h.

The elevated mortality associated with severe acute leg ischemia may be due to reperfusion syndrome. Survival may therefore be improved by avoiding reperfusion and a lower mortality has been reported from hospitals where primary amputation is favored. It has also been suggested that thrombolysis saves lives by restoring perfusion gradually. For a threatened leg this is seldom an option because rapid restoration of perfusion is necessary to save it.

The best treatment for reperfusion syndrome is prevention by expeditious restoration of flow.

There are no clinically proven effective drugs but many have been successful in animal models, including heparin, mannitol, and prostaglandins.

Because heparin and mannitol also have other potential benefits and few side effects they are recommended during the postoperative period. Obviously, acidosis and hyperkalemia must be corrected, and the patient needs to be well hydrated and have good urine output. For patients with suspected reperfusion syn-

drome – urine acidosis and high serum myoglobin levels – alkalinization of the urine is often recommended in order to avoid renal failure despite weak support in the literature. If the urine is red, the urine pH <7.0, and serum myoglobin >10,000 mg/ml, 100 ml sodium bicarbonate is given IV. The dose is repeated until the pH is normalized.

Compartment Syndrome

The acute inflammation in the muscle after reestablishing perfusion leads to swelling and a risk for compartment syndrome. The available space for the muscles is limited in the leg and when the increased pressure in the compartments reduces capillary perfusion below the level necessary for tissue viability, nerve injury and muscle necrosis occur.

The essential clinical feature of compartment syndrome is pain – often very strong and “out of proportion,” which is accentuated by passive extension. The muscle is hard and tender when palpated. Unfortunately, nerves within the compartments are also affected, causing disturbance of sensibility and motor function. This makes diagnosis more difficult. Moreover, the patient is often not fully awake or disoriented, but early diagnosis is still important to save the muscle tissue. For that reason measurement of intracompartmental pressure is performed for diagnosis in some hospitals.

There are no precise limits that advocate fasciotomy, but 30mmHg has been proposed. The specificity for a correct diagnosis using this limit is high, but the sensitivity is much lower.

To notice signs of compartment syndrome after operation or thrombolysis for acute ischemia, frequent physical examinations are vital. Fasciotomy should be performed immediately following the procedure if any suspicion of compartment syndrome exists. Common advice is to always perform fasciotomy right after the vascular procedure when the ischemia is severe and has lasted over 4–6 h. To open all four compartments, we recommend using two long incisions, one placed laterally and one medially in the calf.

Results and Outcome

The outlook for patients with acute leg ischemia has generally been poor. The 30-day mortality when an embolus is the etiology varies between 10% and 40%. Survival is better when arterial thrombosis is the cause, around 90%. When considering the amputation rate after surgical treatment the figures are reversed – lower for embolic disease, at 10–30%, than for thrombosis, which of ten has an early amputation rate of around 40%.

A substantial number of the patients die or require amputation after 30 days. This is due to a combined effect of the patients’ advanced age and comorbidities. In studies not differentiating between etiologies, only 30–40% of the patients were alive 5 years after surgery,

and among those, 40–50% had had amputations.

Because the gradual release of ischemia is thought to reduce the risk for reperfusion syndrome and thereby the negative effects on the heart and kidneys mortality after thrombolysis is thought to be lower. It is difficult, however, to find data on thrombolytic therapy comparable to surgical results. A majority of patients will undergo surgery when thrombolysis is not technically possible, leaving a selected group to follow up. In the few randomized controlled trials that compare surgery and thrombolysis the short-term and long-term amputation rates are alike. Survival is also similar, but in one study it was lower after thrombolytic therapy at 1 year, 80%, compared with surgically treated patients, of whom only 60% were alive at that time.

Conditions Associated with Acute Leg Ischemia

Chronic Ischemia of the Lower Extremity

It is sometimes difficult to differentiate between acute leg ischemia, deterioration of chronic leg ischemia, and just severe end-stage chronic disease in general. Periods of pain escalation bring patients with chronic ischemia to the emergency department. Accentuated pain in these patients has a wide range of origins. Decreased foot perfusion can be due to dehydration or lowered systemic pressure as a consequence of heart failure or a change in medication. Ulcers are frequently painful, especially when complicated by infection or when dressings are changed. History and examination of vital functions and the leg usually disclose such conditions and can also sufficiently rule out acute leg ischemia that needs urgent treatment.

Patients with chronic ischemia benefit from careful planning of their treatment and should not – with few exceptions – be expeditiously treated. Elective therapy includes weighing risk factors against the outcome of the proposed treatment and all the work-up that is needed to get this information. (It is beyond this book's purpose to describe the management of chronic ischemia.) In the emergency department it is sensible to identify and directly treat the patients with true acute leg ischemia and schedule treatment of patients with chronic disease for later. Examples of findings in medical history and physical examination are listed in Table 5.

Table 5. Medical history and physical examination

findings suggesting chronic leg ischemia

History	Examination
Coronary artery disease and stroke	Lack of palpable pulses in both legs
Smoking	Ankle pressure 15–50 mmHg
Claudication, rest pain, and ischemic ulcers	Ulcers
Previous vascular surgery or amputation	Hyperemic foot skin while dependent
Lack of sudden onset of pain	

Acute Ischemia After Previous Vascular Reconstruction

A substantial number of patients have chronic leg ischemia and have undergone vascular reconstructions, so there is a high likelihood that emergency department physicians will have to take care of problems with postoperative acute leg ischemia in the operated leg. The clinical presentation of graft failure or occlusion is variable. An abrupt change in leg function and skin temperature accompanied by the onset of pain can occur any time after surgery, but especially within the first 6 months. Several years after the reconstruction it is slightly more common for progressive deterioration to occur and an eventual graft occlusion to pass unnoticed.

As discussed previously in this chapter the management principles are roughly the same as for primary acute leg ischemia. It is the status of the leg and the severity of ischemia that lead workup and management. Most patients will undergo angiography to establish diagnosis and to provide information about possibilities to restore blood flow. Thrombolysis is often the best treatment option because it exposes the underlying lesions that may have caused the occlusion. As for patients with acute ischemia, those with an immediately threatened leg after a reconstruction should be taken to the operating room and treated as fast as possible.

Blue Toe Syndrome

A toe that suddenly becomes cool, painful, and cyanotic, while pulses can be palpated in the foot, characterizes the classic presentation of blue toe syndrome. This has occasionally led to the assumption that the discoloration of the toe is not of vascular origin, and patients have been sent home without proper vascular assessment. Although coagulation disorders or vasculitis may contribute, such an assumption is dangerous. Atheroembolism is the main cause for blue toe syndrome and atheromatous plaques in the iliac or femoral arteries or thrombi in abdominal or popliteal aneurysms are the main sources. Blue toe syndrome can also present without palpable foot pulses. The presentation may then be less dramatic.

It is common that the patient does not notice the initial insult and wait to seek medical care until after several weeks. Ischemic ulceration at the tip of the toe may then be found in the examination. During the foot examination more signs of microembolization are usually found, including blue spots or patchy discoloration of the sole and heel. When both feet are affected it suggests an embolic source above the aortic bifurcation. The clinical examination should include assessing the aorta and all peripheral arteries, including pulses and auscultation for bruits. When pulses in the foot are not palpable, ankle blood pressure needs to be measured. In the search for aneurysms and stenoses patients need to be investigated with duplex ultrasound to verify examination findings. To prevent future embolization episodes lesions or aneurysms found should be treated as soon as possible.

Occasionally the pain is transient and the blue color will disappear within a few weeks. More common, however, is an extremely intense pain in the toe that is continuous and difficult to treat. Unfortunately, the pain often lasts several months until the toe is either amputated or healed.

The pain is best treated with oral opiates, and quite high doses are often required to ease the pain. A tricyclic antidepressant drug may be added to the regimen if analgesics are not enough.

While waiting for diagnostic studies and final treatment of the lesions, the patient is put on aspirin therapy. There is no scientific evidence for using other medications such as coumadin, steroids, or dipyridole. Still, if suspicion for a popliteal aneurysm is high we recommend anticoagulation with low molecular weight heparin until the aneurysm is corrected.

Popliteal Aneurysms

A common reason for acute leg ischemia is thrombosis of a popliteal aneurysm. Such

aneurysms are also one of the main sources for embolization to the digits in the foot and blue toe syndrome. Besides the clinical signs of acute ischemia discussed previously, a prominent wide popliteal pulse or a mass in the popliteal fossa is often palpated when popliteal aneurysm is the reason for the obstruction.

Popliteal aneurysms are frequent in men but rare in women. They are often bilateral – more than 50% – and associated with the presence of other aneurysms. For instance, 40% of patients with popliteal aneurysms also have an aneurysm in the abdominal aorta. Most popliteal aneurysms are identified during angiography performed as part of the management process for acute leg ischemia. When an aneurysm is suspected during angiography or examination, duplex ultrasound is performed to verify the finding and estimate the aneurysm's diameter.

If the severity of ischemia corresponds to the “immediately threatened” stage described earlier, the patient needs urgent surgery. The revascularization procedure is then often quite difficult. Exposure of the popliteal artery below the knee, including the origins of the calf arteries, should be followed by intraoperative angiography and an attempt to remove the thrombus. It is hoped that angiography can identify a spared calf artery distally. The calf arteries are sometimes slightly dilated in this patient group and can serve as a good distal landing site for a bypass excluding the aneurysm. Often, however, it is impossible to open up the distal vascular bed due to old embolic occlusions and the prognosis for the leg is poor. In such situations every possible alternative solution should be considered, including local thrombolysis, systemic prostaglandin infusion, and profundaplasty.

If the ischemia is less severe, thrombolysis may be considered following the angiography before surgical exclusion of the aneurysm. While thrombolysis previously has been considered questionable because of the risk for further fragmentation of thrombus within the popliteal aneurysm, this strategy may prove very favorable. Over the last few years several studies reporting restored calf vessels by thrombolysis have been published. This may lead to more successful bypasses and improved limb salvage. Once the bypass is accomplished good long-term results are probable. Interestingly, vein grafts used for bypasses in patients with popliteal aneurysms appear to be wider and stay patent longer than for other patient groups.

ACUTE INTESTINAL ISCHEMIA

Mesenteric artery thrombosis has the highest mortality rate of all causes of mesenteric ischemia. First described in the late 15th century, little progress was made in its treatment before the 20th century.

In 1901, a patient with a long history of postprandial pain was found to have an atherosclerotic plaque with overlying thrombus of the superior mesenteric artery (SMA). The physician concluded that if a patient could develop pain of the lower extremities secondary to atherosclerosis, it would stand to reason that a patient could present with postprandial pain due to narrowing of the mesenteric vessels. An example of complete occlusion is illustrated in the image below. The pathophysiologic mechanism by which ischemia produces pain remains poorly understood.

The arterial circulation to the gut has extensive collaterals and arcades providing multiple sources of blood inflow. This explains why vascular occlusion is well tolerated as evidenced by the relative lack of clinical intestinal ischemia despite the high prevalence of atherosclerotic disease of the aorta and visceral arteries. Certain collateral patterns are recognized, depending on which artery is blocked. When either the celiac or superior mesenteric artery (SMA) is compromised, the main collateral circulation is by the gastroduodenal and pancreaticoduodenal arteries. The main collateral channels between the SMA and inferior mesenteric artery (IMA) occur in the region of the splenic flexure between the middle and left colic arteries. In the presence of either SMA or IMA occlusion, the marginal artery of Drummond and the arch of Riolan (an ascending branch of the left colic artery anastomosing with branches of the SMA) enlarge significantly. In the presence of an IMA occlusion, another important collateral circulation is between the internal iliac artery and the left colic artery via the superior hemorrhoidal arteries.

The SMA is the critically important vessel in maintaining visceral perfusion, as demonstrated by increased blood flow after eating. This is not seen in the celiac artery. In chronic ischemia, all patients have SMA stenosis or occlusion, in addition to celiac artery and/or IMA involvement.

Etiology

OCCLUSIVE DISEASE

Emboli. The SMA is the most common site of embolic occlusion although the celiac artery can be affected. There is classically an underlying cardiac problem giving rise to the organized thrombus that embolizes. This is usually atrial fibrillation or less commonly a mural thrombus from an acute myocardial infarction. A history of previous embolic events is not uncommon. Other causes of emboli include iatrogenic intra-aortic manipulations, paradoxical emboli through a septal defect, atrial myxoma or primary aortic tumors.

The history is of constant severe epigastric or periumbilical pain of sudden onset. It is frequently followed by copious vomiting and explosive diarrhea.

Typically the patient has been previously well and asymptomatic. The abdominal signs are often lacking or nonspecific, with distension in association with absent or normal bowel sounds without any signs of peritonism. This combination of severe abdominal pain out of proportion to the clinical findings is typical. Peritonism or blood in the stool or vomitus indicates severe advanced intestinal ischemia with likely infarction and is generally a late clinical feature.

The presence of proximal SMA pulsation and the distribution of intestinal ischemia are intra-operative clues for an embolus. The occlusion in embolism is usually distal to the origin of the pancreaticoduodenal and middle colic branches, which allows some blood flow to the small intestine to be maintained. The stomach, duodenum, and proximal jejunum are normal with ischemia extending to the mid transverse colon.

Thrombosis.

Thrombosis of the superior mesenteric or celiac arteries is most often associated with a preexisting atherosclerotic lesion that already compromises flow. The most common preexisting pathology found in patients with acute mesenteric thrombosis is atherosclerosis.

Many patients present with histories consistent with chronic mesenteric ischemia. Wasting, postprandial pain, and phagophobia (fear of eating) are all common.

Typically, the atherosclerotic lesion gradually compromises flow to the gut, causing a progressive worsening of symptoms. During a period of low flow, the artery thromboses, and flow to the gut is compromised.

Unlike embolic events that occur in arterial branches and result in limited bowel

ischemia, thrombosis occurs at the vessel origin, resulting in extensive bowel involvement.

Superior mesenteric arterial thrombosis may occur as the result of progression of SMA stenosis that had not previously been diagnosed or treated. There is often a history of intestinal or food fear with severe weight loss, the hallmark of chronic intestinal ischemia in about 65% of patients. The typical patient is female and a heavy smoker, often with evidence of widespread arterial disease including previous myocardial infarction or claudication. As with embolic occlusion, the combination of severe abdominal pain out of proportion to the clinical findings is typical. The thrombosis of the SMA occurs at the origin of the artery.

In contrast to embolic disease, the proximal SMA pulse is absent and the distribution of intestinal ischemia is more extensive. Only the stomach, duodenum and distal colon are spared.

In the young patients, fibromuscular dysplasia can cause mesenteric arterial thrombosis with equally devastating results. Intravenous cocaine abuse is another increasing problem accounting for intestinal ischemia in the young patients. The extent of intestinal ischemia and infarction tends to be focal and less than that seen with atherosclerotic thrombosis. The mechanism of ischemia appears to be occlusive rather than due to vasospasm. Mesenteric ischemia should be considered in the differential diagnosis when evaluating a young patient with a history of cocaine abuse presenting with an acute abdomen.

Some prothrombotic states such as hyperhomocysteinemia or the 20210 A prothrombin gene mutation have resulted in primary arterial thrombosis.

Mesenteric venous thrombosis (MVT) is rare and accounts for 5% to 15% of all acute mesenteric ischemia. It is classified as primary (where no cause is recognized) or secondary. Secondary MVT may follow hypercoagulable states, portal venous stasis and hypertension, intra-abdominal infection and inflammation or malignancy, use of oral contraceptives and splenectomy. Long-term anticoagulation is required for MVT, because of the high recurrence rates. The clinical presentation is usually less acute than that of arterial occlusion.

Severe but vague abdominal pain that tends to be colicky and slowly progressive is usually present. Few abdominal signs are present except tenderness, distension and decreased bowel sounds. The pain is out of proportion to the physical findings. Fecal occult blood is present in the majority of patients.

There is a pyrexia of greater than 38 °C in 25% to 50% of patients, and 20% have a tachycardia. Leucocytosis ranges from 12000 to 29000.

Frank peritonitis is seen only when transmural infarction or perforation has occurred.

Surgical findings include blood-stained free peritoneal fluid at laparotomy. The affected bowel is cyanotic and edematous with a rubbery texture.

Mesenteric arterial pulsations are present but the veins contain fresh thrombus that extrude when the veins are cut. Infarction is most common in the mid small bowel.





FIG. A - Schematic representation of the collateral circulation of the

intestine. B - Angiographic appearance of arch of Riolan from superior mesenteric artery (stented at its origin). C - Angiographic appearance of marginal artery of Drummond. D - Initial angiogram demonstrates occluded IMA. The delayed film shows the colonic supply.

Diagnosis

Acute intestinal ischemia is a life-threatening surgical emergency, yet can be a difficult diagnosis to make, with delay contributing directly to infarction.

The majority of cases are diagnosed more than 12 hours after the onset of symptoms. Delayed diagnosis accounts for the majority of malpractice claims involving acute mesenteric ischemia in the United States. Diagnosis depends on a high index of suspicion. The main presenting feature is the combination of severe abdominal pain out of proportion to the clinical findings, as discussed above.

Serum levels of lactate and leucocytes are elevated in the majority (65% to 90%) of patients to greater than 50 U/L and 15000/mL, respectively.

Hyperamylasemia is seen in just under half the patients with acute mesenteric ischemia. Elevation of serum inorganic phosphate levels have been proposed as a marker of mesenteric ischemia, as it is extensively found in gut, but this only occurs in 15% to 33% of such patients.

However, in those patients who did have elevated phosphate levels, it predicted extensive injury and poor prognosis. The fibrinolytic marker D-dimer is elevated in thrombo-embolic occlusion of the SMA, although levels are also raised in other conditions of acute bowel ischemia such as strangulation or ruptured aortic aneurysm.

Animal studies have suggested intestinal fatty acid binding protein (I-FABP) as a serum marker reflecting bowel ischemia. Early human studies show promise, as patients with ischemic bowel disease demonstrate significantly higher I-FABP levels than either healthy subjects or patients with acute abdominal pain. Patients with mesenteric infarction had the highest serum I-FABP levels.

Plain radiographs of the abdomen may reveal nonspecific bowel dilatation or, in MVT, wall edema (thumbprinting); or gas in the bowel wall or portal vein. Unfortunately they are not

helpful in most cases.

Mesenteric angiography will confirm the diagnosis of arterial occlusion but at the cost of delay in treatment. If there are clear abdominal signs of peritonitism, urgent laparotomy without angiography is the best course of action. In the remainder of patients suspected of acute intestinal ischemia with-out abdominal signs, angiography is indicated with lateral views of the visceral aorta and its branches.

In acute SMA thrombosis, there is usually no visualization of the entire artery because of the ostial nature of the disease, although delayed views may show slow filling of the distal SMA. SMA embolization usually allows visualization of the proximal artery to just beyond the level of the middle colic artery.

Treatment

Nonsurgical

In all cases, the patient should be initially resuscitated, given broadspectrum intravenous antibiotics and fully heparinized. As yet, the twin goals of mesenteric revascularization and resection of nonviable bowel can only be achieved by surgical means.

Surgery is indicated in all patients with peritonitis. Angiography in patients without peritonitis may demonstrate NOMI or MVT. In NOMI, treatment is nonoperative and depends on optimizing cardiac output and treating underlying conditions such as sepsis. Intramesenteric arterial infusion of papaverine at a dose of 30 to 60 mg/lr may be beneficial.

Up to 65% of patients who have undergone cardiac surgery have had symptomatic improvement within hours when diagnosed early.

If MVT is diagnosed at angiography, intra-arterial thrombolytic therapy has been given successfully. Nonoperative management by full anticoagulation for acute MVT is feasible when the initial diagnosis is certain and when the bowel infarction has not led to transmural necrosis and bowel perforation. The morbidity, mortality, and survival rates are similar in cases of surgical and nonoperative management.

Other reported endovascular procedures for acute intestinal ischemia include fenestration and stent placement in aortic dissection, angioplasty and stenting in an acute occlusion in a patient with chronic mesenteric insufficiency, and angioplasty alone . This

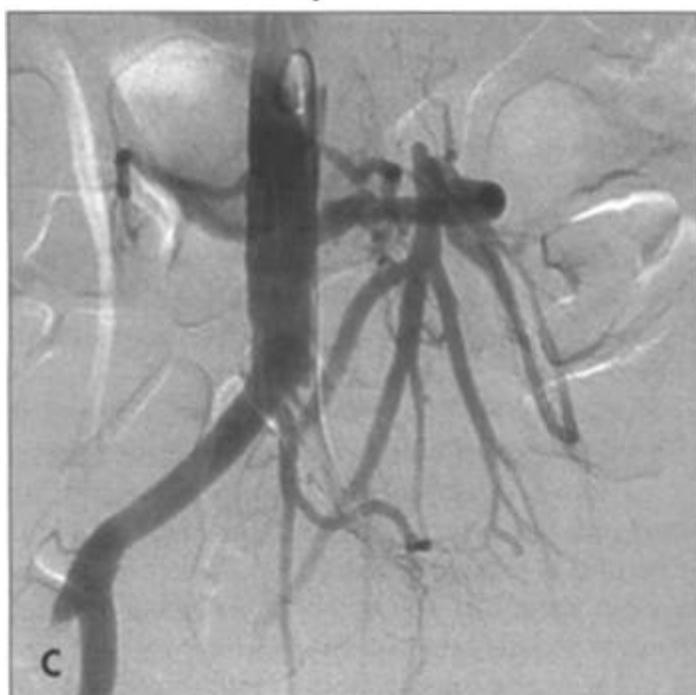
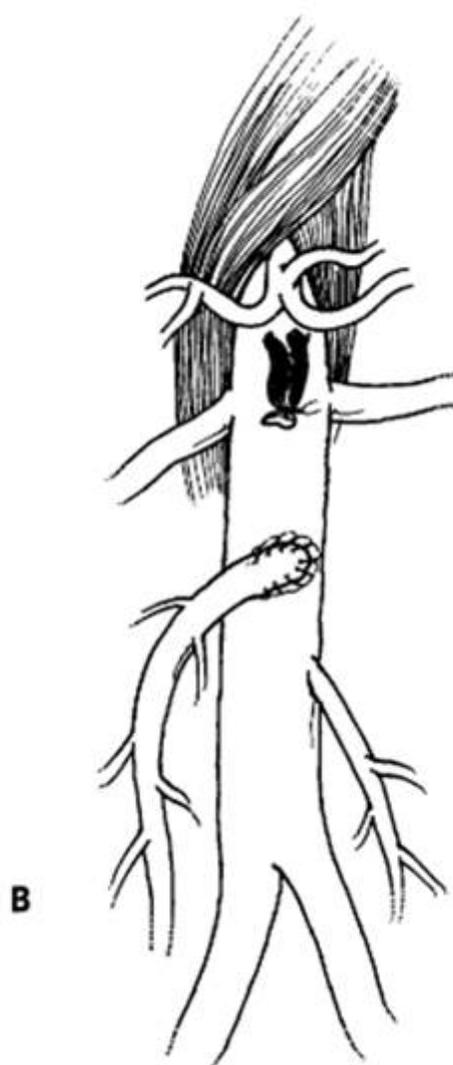
contrasts with an increasing use of angioplasty for chronic mesenteric ischemia.

SURGICAL

Laparotomy is indicated in patients with peritonitis after rapid resuscitation. The first step is to assess the degree and extent of bowel viability. Free, foul smelling peritoneal fluid is a sign of advanced necrosis even if perforation has not occurred. Ischemic bowel has a characteristic appearance with loss of its normal sheen. It is dull, gray in color and flabby in tone without any peristalsis. Infarcted bowel is purplish black in color, often friable and perforated. In many cases the bowel ischemia will be so extensive and advanced that no further surgical treatment is undertaken and palliative care given. Where there is hope of sufficient bowel viability, revascularization should be performed before any bowel resection is considered. After successful revascularization, previously precarious segments of intestine may recover and resection of clearly ischemic bowel can then take place.

SMA embolectomy. The proximal portion of the SMA is dissected free from the surrounding fat and lymphatic tissue just as it emerges from the pancreatic neck into the base of the mesentery. Approximately 3 to 4 centimeters of artery is cleared, with care taken not to damage the branches. Heparin (5000 units) is given intravenously. A transverse arteriotomy is made and a 3F or 4F embolectomy catheter is passed proximally and distally to clear the embolus and reestablish vigorous pulsatile flow. If proximal flow cannot be established, SMA thrombosis is likely and reconstructive surgery will be required.





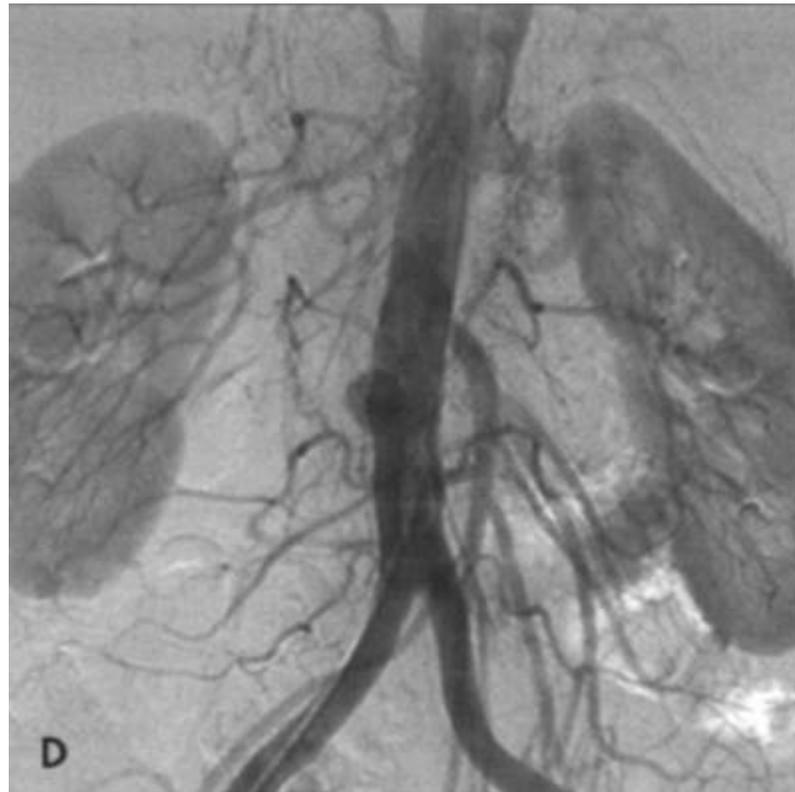


FIG. A - Schematic representation of revascularization of the SMA with: bypass taking care to avoid kinking and obstruction B - Or re-implantation of SMA into the aorta. C - Angiographic appearance of aorto-SMA bypass with vein graft. There is co-existing left common iliac occlusion. D - Angiographic appearance of re-implanted SMA into aorta, which has a small saccular aneurysm at the site of occluded vein graft (aortoceliac bypass).

TOPIC № 17. AORTIC ARCH SYNDROME. CAUSES OF AORTIC ARCH SYNDROME. CLINICAL CHARACTERISTICS. CLASSIFICATION.

Carotid Artery Disease

Atherosclerotic occlusive plaque is by far the most common pathology seen in the carotid artery bifurcation. Thirty to 60% of all ischemic strokes are related to atherosclerotic carotid bifurcation occlusive disease. In the following section, discussion will be focused on clinical presentation, diagnosis, and management, including medical therapy, surgical carotid endarterectomy, and stenting of atherosclerotic carotid occlusive disease. In the second part of the section, a brief review will be focused on other less common non-atherosclerotic diseases involving the extracranial carotid artery, including kink and coil, fibromuscular dysplasia (FMD), arterial dissection, aneurysm, radiation arteritis, Takayasu's arteritis, and carotid body tumor.

Epidemiology and Etiology of Carotid Occlusive Disease

Approximately 700,000 Americans suffer a new or recurrent stroke each year. Eighty-five percent of all strokes are ischemic and 15% are hemorrhagic. Hemorrhagic strokes are caused by head trauma or spontaneous disruption of intracerebral blood vessels. Ischemic strokes are due to hypoperfusion from arterial occlusion, or less commonly due to decreased flow resulting from proximal arterial stenosis and poor collateral network. Common causes of ischemic strokes are cardiogenic emboli (35%), carotid artery disease (30%), lacunar (10%), miscellaneous (10%), and idiopathic (15%). The term *cerebrovascular accident* (CVA) often is used interchangeably to refer to an ischemic stroke. A *transient ischemic attack* (TIA) is defined as a temporary focal cerebral or retinal hypoperfusion state that resolves spontaneously within 24 hours after its onset. However, the majority of TIAs resolve within minutes, and longer lasting neurologic deficits more likely represent a stroke. Recently, the term *brain attack* has been coined to refer to an acute stroke or TIA, denoting the condition as a medical emergency requiring immediate attention, similar to a heart attack.

Stroke due to carotid bifurcation occlusive disease usually is caused by atheroemboli (Fig. 1). The carotid bifurcation is an area of low-flow velocity and low-shear stress. As the blood circulates through the carotid bifurcation, there is separation of flow into the low-resistance ICA, and the high-resistance external carotid artery. Characteristically,

atherosclerotic plaque forms in the outer wall opposite to the flow divider (Fig. 2). Atherosclerotic plaque formation is complex, beginning with intimal injury, platelet deposition, smooth muscle cell proliferation, and fibroplasia, and leading to subsequent luminal narrowing. With increasing degree of stenosis in the ICA, flow becomes more turbulent, and the risk of atheroembolization escalates. The severity of stenosis is commonly divided into three categories according to the luminal diameter reduction: mild (less than 50%), moderate (50 to 69%), and severe (70 to 99%). Severe carotid stenosis is a strong predictor for stroke. In turn, a prior history of neurologic symptoms (TIA or stroke) is an important determinant for recurrent ipsilateral stroke. The risk factors for the development of carotid artery bifurcation disease are similar to those causing atherosclerotic occlusive disease in other vascular beds. Increasing age, male gender, hypertension, tobacco smoking, diabetes mellitus, homocysteinemia, and hyperlipidemia are well-known predisposing factors for the development of atherosclerotic occlusive disease.

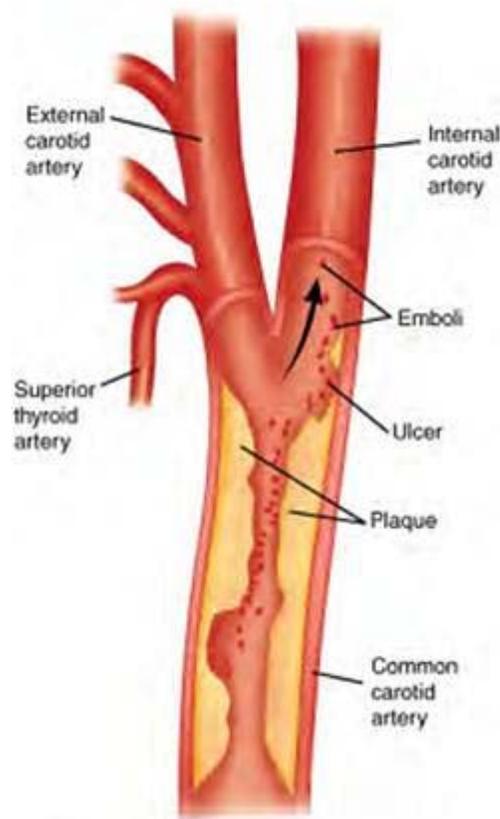


Fig. 1. Stroke due to carotid bifurcation occlusive disease is usually caused by atheroemboli arising from the internal carotid artery, which provides the majority of blood flow to the cerebral hemisphere

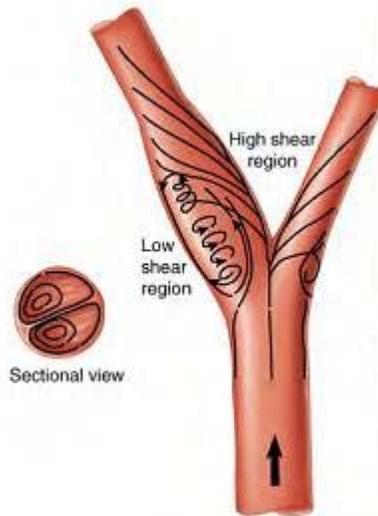


Fig.2 A. The carotid bifurcation is an area of low-flow velocity and low-shear stress. As the blood circulates through the carotid bifurcation, there is separation of flow into the low-resistance internal carotid artery and the high-resistance external carotid artery

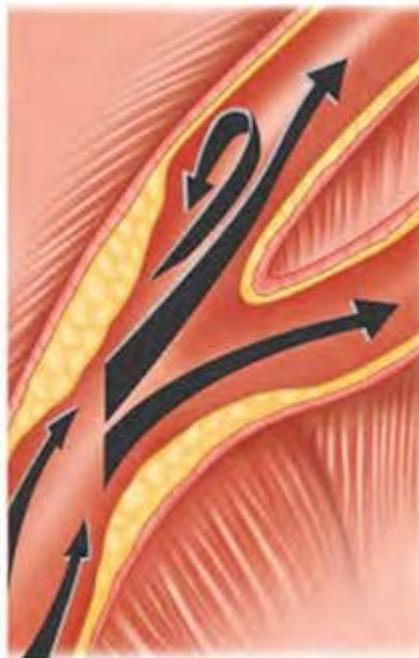


Fig. 2 B. The carotid atherosclerotic plaque typically forms in the outer wall opposite to the flow divider due in part to the effect of the low-shear stress region, which also creates a transient reversal of flow during cardiac cycle.

Clinical Manifestations of Cerebral Ischemia

TIA is a focal loss of neurologic function, lasting for <24 hours. *Crescendo TIAs* refer to a syndrome comprising repeated TIAs within a short period of time that is characterized by

complete neurologic recovery in between. At a minimum the term should probably be reserved either for those with daily events or multiple resolving attacks within 24 hours. Hemodynamic TIAs represent focal cerebral events that are aggravated by exercise or hemodynamic stress and typically occur after short bursts of physical activity, postprandially or after getting out of a hot bath. It is implied that these are due to severe extracranial disease and poor intracranial collateral recruitment. *Reversible ischemic neurologic deficits* refer to ischemic focal neurologic symptoms lasting longer than 24 hours but resolving within 3 weeks. When a neurologic deficit lasts longer than 3 weeks, it is considered a *completed stroke*. *Stroke in evolution* refers to progressive worsening of the neurologic deficit, either linearly over a 24-hour period, or interspersed with transient periods of stabilization and/or partial clinical improvement.

The patients who suffer CVAs typically present with three categories of symptoms, including ocular symptoms, sensory/motor deficit, and/or higher cortical dysfunction. The common ocular symptoms associated with extracranial carotid artery occlusive disease include amaurosis fugax and presence of Hollenhorst plaques. Amaurosis fugax, commonly referred to as *transient monocular blindness*, is a temporary loss of vision in one eye that patients typically describe as a window shutter coming down or gray shedding of the vision. This partial blindness usually lasts for a few minutes and then resolves. Most of these phenomena (>90%) are due to embolic occlusion of the main artery or the upper or lower divisions. Monocular blindness progressing over a 20-minute period suggests a migrainous etiology. Occasionally, the patient will recall no visual symptoms while the optician notes a yellowish plaque within the retinal vessels, which is also known as the *Hollenhorst plaque*. These are frequently derived from cholesterol embolization from the carotid bifurcation and warrant further investigation. Additionally, several ocular symptoms may be caused by microembolization from the extracranial carotid diseases, including monocular vision loss due to retinal artery or optic nerve ischemia, the ocular ischemia syndrome, and visual field deficits secondary to cortical infarction and ischemia of the optic tracts. Typical motor and/or sensory symptoms associated with CVAs are located in either an ipsilateral or contralateral neurologic deficit. Ischemic events tend to have an abrupt onset, with the severity of the insult being apparent from the onset and not usually associated with seizures or paraesthesia. In contrast, they represent loss or diminution of neurologic function. Furthermore, motor or sensory deficits can be unilateral or bilateral, with the upper and lower limbs being variably affected depending on the site of the cerebral lesion.

The combination of a motor and sensory deficit in the same body territory is suggestive

of a cortical thromboembolic event as opposed to lacunar lesions secondary to small vessel disease of the penetrating arterioles. However, a small proportion of the latter may present with a sensorimotor stroke secondary to small vessel occlusion within the posterior limb of the internal capsule. Pure sensory and pure motor strokes and those strokes where the weakness affects one limb only or does not involve the face are more typically seen with lacunar as opposed to cortical infarction. A number of higher cortical functions, including speech and language disturbances, can be affected by thromboembolic phenomena from the carotid artery with the most important clinical example for the dominant hemisphere being dysphasia or aphasia, and visuospatial neglect being an example of nondominant hemisphere injury.

Diagnostic Evaluation

Duplex ultrasonography is the most widely used screening tool to evaluate for atherosclerotic plaque and stenosis of the extracranial carotid artery. It is also commonly used to monitor patients serially for progression of disease, or after intervention (carotid endarterectomy or angioplasty). Duplex ultrasound of the carotid artery combines B-mode gray-scale imaging and Doppler waveform analysis. Characterization of the carotid plaque on gray-scale imaging provides useful information about its composition. However, there are currently no universal recommendations that can be made based solely on the sonographic appearance of the plaque. On the other hand, criteria have been developed and well refined for grading the degree of carotid stenosis based primarily on Doppler-derived velocity waveforms.

The external carotid artery has a high-resistance flow pattern with a sharp systolic peak and a small amount of flow in diastole. In contrast, a normal ICA will have a low-resistance flow pattern with a broad systolic peak and a large amount of flow during diastole. The flow pattern in the common carotid artery (CCA) resembles that in the ICA, as 80% of the flow is directed to the ICA, with waveforms that have broad systolic peaks and a moderate amount of flow during diastole. Conventionally, velocity measurements are recorded in the common, external carotid bulb, and the proximal, mid-, and distal portions of the ICA. Characteristically, the peak systolic velocity is increased at the site of the vessel stenosis. The end-diastolic velocity is increased with a greater degree of stenosis. In addition, stenosis of the ICA can lead to color shifts with color mosaics indicating a poststenotic turbulence. Dampening of the Doppler velocity waveforms are typically seen in areas distal to severe carotid stenosis where blood flow is reduced. It is well known that occlusion of the ipsilateral ICA can lead to a "falsely" elevated velocity on the contralateral side due to an increase in compensatory blood

flow. In the presence of a high-grade stenosis or occlusion of the ICA, the ipsilateral CCA displays high flow resistance waveforms, similar to that seen in the external carotid artery. If there is a significant stenosis in the proximal CCA, its waveforms may be dampened with low velocities.

The Doppler grading systems of carotid stenosis were initially established by comparison to angiographic findings of disease. Studies have shown variability in the measurements of the duplex properties by different laboratories, as well as heterogeneity in the patient population, study design, and techniques. One of the most commonly used classifications was established at the University of Washington School of Medicine in Seattle. Diameter reduction of 50 to 79% is defined by peak systolic velocity >125 cm/sec with extensive spectral broadening. For stenosis in the range of 80 to 99%, the peak systolic velocity is >125 cm/sec and peak diastolic velocity is >140 cm/sec. The ratio of internal carotid to common carotid artery (ICA/CCA) peak systolic velocity has also been part of various ultrasound diagnostic classifications. A ratio >4 is a great predictor of angiographic stenosis of 70 to 99%. A multispecialty consensus panel has developed a set of criteria for grading carotid stenosis by duplex examination (Table 1).

Table 1. Carotid Duplex Ultrasound Criteria for Grading Internal Carotid Artery Stenosis

Degree of Stenosis (%)	ICA PSV (cm/s)	ICA/CCA PSV Ratio	ICA EDV (cm/s)	Plaque Estimate (%)*
Normal	<125	<2.0	<40	None
<50	<125	<2.0	<40	<50
50–69	125–230	2.0–4.0	40–100	≥ 50
≥ 70 to less than near occlusion	>230	>4.0	>100	≥ 50
Near occlusion	High, low, or not detected	Variable	Variable	Visible
Total occlusion	Not detected	Not applicable	Not detected	Visible, no lumen

MRA is increasingly being used to evaluate for atherosclerotic carotid occlusive disease and intracranial circulation. MRA is noninvasive and does not require iodinated contrast agents. MRA uses phase contrast or time-of-flight, with either two-dimensional or three-dimensional data sets for greater accuracy. Three-dimensional, contrast-enhanced MRA allows data to be obtained in coronal and sagittal planes with improved image qualities due to shorter study time. In addition, the new MRA techniques allow for better reformation of images in various planes to allow better grading of stenosis. There have been numerous studies comparing the sensitivity and specificity of MRA imaging for carotid disease to duplex and selective contrast angiography. Magnetic resonance imaging (MRI) of the brain is essential in

the assessment of acute stroke patients. MRI with diffusion-weighted imaging can differentiate areas of acute ischemia, areas still at risk for ischemia (penumbra), and chronic cerebral ischemic changes. However, computed tomographic (CT) imaging remains the most expeditious test in the evaluation of acute stroke patients to rule out intracerebral hemorrhage. Recently, multidetector CTA has gained increasing popularity in the evaluation of carotid disease. This imaging modality can provide volume rendering, which allows rotation of the object with accurate anatomic structures from all angles (Fig. 3). The advantages of CTA over MRA include faster data acquisition time and better spatial resolution. However, grading of carotid stenosis by CTA requires further validation at the time of this writing before it can be widely applied.

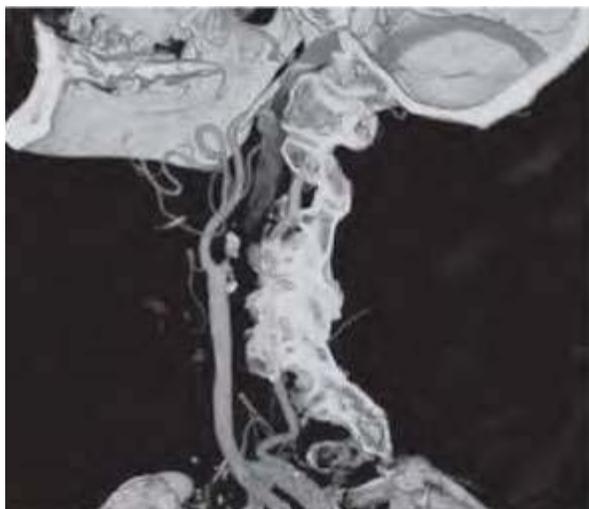


Fig. 3 A. Carotid computed tomography angiography is a valuable imaging modality that can provide a three-dimensional image reconstruction with high image resolution. A carotid artery occlusion is noted in the internal carotid artery.



Fig.3

B. The entire segment of extracranial carotid artery is visualized from the thoracic compartment to the base of skull.

Historically, DSA has been the gold standard test to evaluate the extra- and intracranial circulation (Fig. 4). This is an invasive procedure, typically performed via a transfemoral puncture, and involves selective imaging of the carotid and vertebral arteries using iodinated contrast. The risk of stroke during cerebral angiography is generally reported at approximately 1%, and is typically due to atheroembolization related to wire and catheter manipulation in the arch aorta or proximal branch vessels. Over the past decades, however, the incidence of neurologic complications following angiography has been reduced, due to the use of improved guidewires and catheters, better resolution digital imaging, and increased experience. Local access complications of angiography are infrequent and include development of hematoma, pseudoaneurysm, distal embolization, or acute vessel thrombosis. Currently, selective angiography is particularly used for patients with suspected intracranial disease and for patients in whom percutaneous revascularization is considered. The techniques of carotid angioplasty and stenting for carotid bifurcation occlusive disease are described in the "Techniques of Carotid Angioplasty and Stenting" section. Preoperative CTA or MRA is routinely utilized to get information about the aortic arch anatomy and presence of concomitant intracranial disease and collateral pathway in planning our strategy for carotid stenting or endarterectomy.



Fig. 4. A carotid angiogram reveals an ulcerated carotid plaque (*arrow*) in the proximal internal carotid artery, which also resulted in a high-grade, internal, carotid artery stenosis

Treatment of Carotid Occlusive Disease

Conventionally, patients with carotid bifurcation occlusive disease are divided into two broad categories: patients without prior history of ipsilateral stroke or TIA (asymptomatic) and those with prior or current ipsilateral neurologic symptoms (symptomatic). It is estimated that 15% of all strokes are preceded by a TIA. The 90-day risk of a stroke in a patient presenting with a TIA is 3 to 17%. According to the Cardiovascular Health Study, a longitudinal, population-based study of CAD and stroke in men and women, the prevalence of TIA in men was 2.7% between the ages of 65 to 69 and 3.6% for ages 75 to 79; the prevalence in women was 1.4% and 4.1%, respectively. There have been several studies reporting on the effectiveness of stroke prevention with medical treatment and carotid endarterectomy for symptomatic patients with moderate to severe carotid stenosis. Early and chronic aspirin therapy has been shown to reduce stroke recurrence rate in several large clinical trials.

Symptomatic Carotid Stenosis

Currently, most stroke neurologists prescribe both aspirin and clopidogrel for secondary stroke prevention in patients who had experienced a TIA or stroke. In patients with symptomatic carotid stenosis, the degree of stenosis appears to be the most important predictor in determining risk for an ipsilateral stroke. The risk of a recurrent ipsilateral stroke in patients with severe carotid stenosis approaches 40%. Two large multicenter randomized clinical trials, the European Carotid Surgery Trial and the North American Symptomatic Carotid Endarterectomy Trial, have both shown a significant risk reduction in stroke for patients with symptomatic high-grade stenosis (70 to 99%) undergoing carotid endarterectomy when compared to medical therapy alone. There has been much discussion regarding the different methodology used in the measurement of carotid stenosis and calculation of the life-table data between the two studies, which still led to similar results. Findings of these two landmark trials have also been reanalyzed in many subsequent publications. The main conclusions of the trials remain validated and widely acknowledged. Briefly, the North American Symptomatic Carotid

Endarterectomy Trial study showed that, for high-grade carotid stenosis, the cumulative risk of ipsilateral stroke was 26% in the medically treated group and 9% in the surgically treated group at 2 years. For patients with moderate carotid artery stenosis (50 to 69%), the benefit of carotid endarterectomy is less but still favorable when compared to medical treatment alone; the 5-year fatal or nonfatal ipsilateral stroke rate was 16% in the surgically treated group vs. 22% in the medically treated group. The risk of stroke was similar for the remaining group of symptomatic patients with less than 50% carotid stenosis, whether they had endarterectomy or medical treatment alone. The European Carotid Surgery Trial reported similar stroke risk reduction for patients with severe symptomatic carotid stenosis and no benefit in patients with mild stenosis, when carotid endarterectomy was performed vs. medical therapy.

The optimal timing of carotid intervention after acute stroke, however, remains debatable. Earlier studies showed an increased rate of postoperative stroke exacerbation and conversion of a bland to hemorrhagic infarction when carotid endarterectomy was carried out within 5 to 6 weeks after acute stroke. The dismal outcome reported in the early experience was likely related to poor patient selection. The rate of stroke recurrence is not insignificant during the interval period and may be reduced with early intervention for symptomatic carotid stenosis. Contemporary series have demonstrated acceptable low rates of perioperative complications in patients undergoing carotid endarterectomy within 4 weeks after acute stroke. In a recent retrospective series, carotid artery stenting, when performed early (<2 weeks) after the acute stroke, was associated with higher mortality than when delayed (>2 weeks).

Asymptomatic Carotid Stenosis

Whereas there is universal agreement that carotid revascularization (endarterectomy or stenting) is effective in secondary stroke prevention for patients with symptomatic moderate and severe carotid stenosis, the management of asymptomatic patients remains an important controversy to be resolved. Generally, the detection of carotid stenosis in asymptomatic patients is related to the presence of a cervical bruit or based on screening duplex ultrasound findings. In one of the earlier observational studies, the authors showed that the annual rate of occurrence of neurologic symptoms was 4% in a cohort of 167 patients with asymptomatic cervical bruits followed prospectively by serial carotid duplex scan. The mean annual rate of carotid stenosis progression to a greater than 50% stenosis was 8%. The presence of or progression to a greater than 80% stenosis correlated highly with either the development of a

total occlusion of the ICA or new symptoms. The major risk factors associated with disease progression were cigarette smoking, diabetes mellitus, and age. This study supported the contention that it is prudent to follow a conservative course in the management of asymptomatic patients presenting with a cervical bruit.

One of the first randomized clinical trials on the treatment of asymptomatic carotid artery stenosis was the Asymptomatic Carotid Atherosclerosis Study, which evaluated the benefits of medical management with antiplatelet therapy vs. carotid endarterectomy. Over a 5-year period, the risk of ipsilateral stroke in individuals with a carotid artery stenosis greater than 60% was 5.1% in the surgical arm. On the other hand, the risk of ipsilateral stroke in patients treated with medical management was 11%. Carotid endarterectomy produced a relative risk reduction of 53% over medical management alone. The results of a larger randomized trial from Europe, The Asymptomatic Carotid Surgery Trial, confirmed similar beneficial stroke risk reduction for patients with asymptomatic greater than 70% carotid stenosis undergoing endarterectomy compared to medical therapy. An important point derived from this latter trial was that even with improved medical therapy, including the addition of statin drugs and clopidogrel, medical therapy was still inferior to endarterectomy in the primary stroke prevention for patients with high-grade carotid artery stenosis. It is generally agreed that asymptomatic patients with severe carotid stenosis (80 to 99%) are at significantly increased risk for stroke and stand to benefit from either surgical or endovascular revascularization. However, revascularization for asymptomatic patients with a less severe degree of stenosis (60 to 79%) remains controversial.

Carotid Endarterectomy vs. Angioplasty and Stenting

Currently, the argument is no longer that medical therapy alone is inferior to surgical endarterectomy in stroke prevention for severe carotid stenosis. Rather, the debate now revolves around whether carotid angioplasty and stenting produces the same benefit that has been demonstrated by carotid endarterectomy. Since carotid artery stenting was approved by the FDA in 2004 for clinical application, this percutaneous procedure has become a treatment alternative in patients who are deemed "high-risk" for endarterectomy (Table 2). In contrast to many endovascular peripheral arterial interventions, percutaneous carotid stenting represents a much greater challenging procedure, because it requires complex, catheter-based skills using the 0.014-in guidewire system and distal protection device. Moreover, current carotid stent devices predominantly use the monorail guidewire system that requires more technical agility,

in contrast to the over-the-wire catheter system that is routinely used in peripheral interventions. This percutaneous intervention often requires balloon angioplasty and stent placement through a long carotid guiding sheath via a groin approach. Poor technical skills can result in devastating treatment complications such as stroke, which can occur due in part to plaque embolization during the balloon angioplasty and stenting of the carotid artery. Because of these various procedural components that require high technical proficiency, many early clinical investigations of carotid artery stenting, which included physicians with little or no carotid stenting experience, resulted in alarmingly poor clinical outcomes. A recent Cochrane review noted that, before 2006, a total of 1269 patients had been studied in five randomized controlled trials comparing percutaneous carotid intervention and surgical carotid reconstruction.³² Taken together, these trials revealed that carotid artery stenting had a greater procedural risk of stroke and death when compared to carotid endarterectomy (odds ratio 1.33; 95% CI 0.86–2.04). Additionally, greater incidence of carotid restenosis was noted in the stenting group than the endarterectomy cohorts. However, the constant improvement of endovascular devices, procedural techniques, and adjunctive pharmacologic therapy will likely improve the treatment success of percutaneous carotid intervention. Critical appraisals of these trials comparing the efficacy of carotid stenting vs. endarterectomy are available for review.³³ Several ongoing clinical trials will undoubtedly provide more insights on the efficacy of carotid stenting in the near future.

Table 2. Conditions Qualifying Patients as "High Surgical Risk" for Carotid Endarterectomy

Anatomical Factors	Physiological Factors
• High carotid bifurcation (above C2 vertebral body)	• Age ≥80 y
• Low common carotid artery (below clavicle)	• Left ventricular ejection fraction ≤30%
• Contralateral carotid occlusion	• New York Heart Association Class III/IV congestive heart failure
• Restenosis of ipsilateral prior carotid endarterectomy	• Unstable angina: Canadian Cardiovascular Society Class III/IV angina pectoris
• Previous neck irradiation	
• Prior radical neck dissection	• Recent myocardial infarction
• Contralateral laryngeal nerve palsy	• Clinically significant cardiac disease (congestive heart failure, abnormal stress test, or need for coronary revascularization)
• Presence of tracheostomy	
	• Severe chronic obstructive pulmonary disease
	• End-stage renal disease on dialysis

Surgical Techniques of Carotid Endarterectomy

Although carotid endarterectomy is one of the earliest vascular operations ever described and its techniques have been perfected in the last two decades, surgeons continue to debate

many aspects of this procedure. For instance, there is no universal agreement with regard to the best anesthetic of choice, the best intraoperative cerebral monitoring, whether to "routinely" shunt, open vs. eversion endarterectomy, and patch vs. primary closure. Various anesthetic options are available for a patient undergoing carotid endarterectomy including general, local, and regional anesthesia. Typically, the anesthesia of choice depends on the preference of the surgeon, anesthesiologist, and patient. However, depending on the anesthetic given, the surgeon must decide whether intraoperative cerebral monitoring is necessary or intra-arterial carotid shunting will be used. In general, if the patient is awake, then his or her abilities to respond to commands during carotid clamp period determine the adequacy of collateral flow to the ipsilateral hemisphere. On the other hand, intraoperative electroencephalogram or transcranial power Doppler (TCD) has been used to monitor for adequacy of cerebral perfusion during the clamp period for patients undergoing surgery under general anesthesia. Focal ipsilateral decreases in amplitudes and slowing of electroencephalogram waves are indicative of cerebral ischemia. Similarly, a decrease to less than 50% of baseline velocity in the ipsilateral middle cerebral artery is a sign of cerebral ischemia. For patients with poor collateral flow exhibiting signs of cerebral ischemia, intra-arterial carotid shunting with removal of the clamp will restore cerebral flow for the remaining part of the surgery. Stump pressures have been used to determine the need for intra-arterial carotid shunting. Some surgeons prefer to shunt all patients on a routine basis and do not use intraoperative cerebral monitoring.

The patient's neck is slightly hyperextended and turned to the contralateral side, with a roll placed between the shoulder blades. An oblique incision is made along the anterior border of the sternocleidomastoid muscle centered on top of the carotid bifurcation (Fig. 5). The platysma is divided completely. Typically, tributaries of the anterior jugular vein are ligated and divided. The dissection is carried medial to the sternocleidomastoid. The superior belly of the omohyoid muscle is usually encountered just anterior to the CCA. This muscle can be divided. The carotid fascia is incised and the CCA is exposed. The CCA is mobilized cephalad toward the bifurcation. The dissection of the carotid bifurcation can cause reactive bradycardia related to stimulation of the carotid body. This reflex can be blunted with injection of lidocaine 1% into the carotid body or reversed with administration of IV atropine. A useful landmark in the dissection of the carotid bifurcation is the common facial vein. This vein can be ligated and divided. Frequently, the twelfth cranial nerve (hypoglossal nerve) traverses the carotid bifurcation just behind the common facial vein. The external carotid artery is mobilized just enough to get a clamp across. Often, a branch of the external carotid artery crossing to the sternocleidomastoid can be divided to allow further cephalad mobilization of the ICA. For high

bifurcation, division of the posterior belly of the digastric muscle is helpful in establishing distal exposure of the ICA.



Fig 5. To perform carotid endarterectomy, the patient's neck is slightly hyperextended and turned to the contralateral side. An oblique incision is made along the anterior border of the sternocleidomastoid muscle centered on top of the carotid bifurcation.

IV heparin sulfate (1 mg/kg) is routinely administered just before carotid clamping. The ICA is clamped first using a soft, noncrushing vascular clamp to prevent distal embolization. The external and common carotid arteries are clamped subsequently. A longitudinal arteriotomy is made in the distal CCA and extended into the bulb and past the occlusive plaque into the normal part of the ICA. Endarterectomy is carried out to remove the occlusive plaque (Fig. 6). If necessary, a temporary shunt can be inserted from the CCA to the ICA to maintain continuous antegrade cerebral blood flow (Fig. 7). Typically, a plane is teased out from the vessel wall, and the entire plaque is elevated and removed. The distal transition line in the ICA where the plaque had been removed must be examined carefully and should be smooth. Tacking sutures are placed when an intimal flap remains in this transition to ensure no obstruction to flow (Fig. 8). The occlusive plaque is usually removed from the origin of the external carotid artery using the eversion technique. The endarterectomized surface is then irrigated and any debris removed. A patch (autogenous saphenous vein, synthetic such as polyester, PTFE, or biologic material) is sewn to close the arteriotomy (Fig. 9). Whether patch closure is necessary in all patients and which patch is the best remain controversial. However, most surgeons agree

that patch closure is indicated particularly for the small vessel (<7 mm). The eversion technique also has been advocated for removing the plaque from the ICA. In the eversion technique, the ICA is transected at the bulb, the edges of the divided vessel are everted, and the occluding plaque is "peeled" off the vessel wall. The purported advantages of the eversion technique are no need for patch closure and a clear visualization of the distal transition area. Reported series have not shown a clear superiority of one technique over the others. Surgeons will likely continue to use the technique of their choice. Just before completion of the anastomosis to close the arteriotomy, it is prudent to flush the vessels of any potential debris. When the arteriotomy is closed, flow is restored to the external carotid artery first and to the ICA second. IV protamine sulfate can be given to reverse the effect of heparin anticoagulation following carotid endarterectomy. The wound is closed in layers. After surgery, the patient's neurologic condition is assessed in the operating room (OR) before transfer to the recovery area.

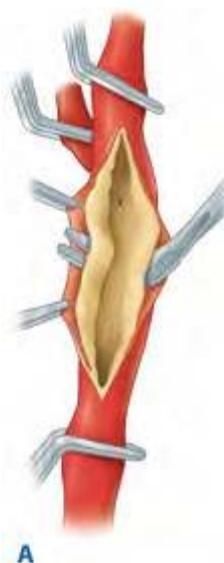


Fig. 6 A. During carotid endarterectomy, vascular clamps are applied in common carotid, external carotid, and internal carotid arteries. Carotid plaque is elevated from the carotid lumen

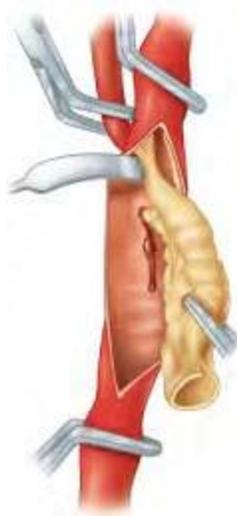


Fig. 6 B. Carotid plaque is removed and the arteriotomy is closed either primarily or with a patch angioplasty



Fig. 7. A temporary carotid shunt is inserted from the common carotid artery (*long arrow*) to the internal carotid artery (*short arrow*) during carotid endarterectomy to provide continuous antegrade cerebral blood flow.

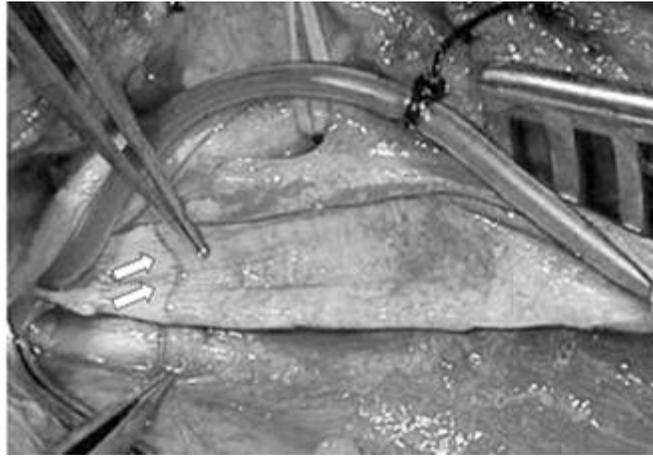


Fig. 8. The distal transition line (left side of the picture) in the internal carotid artery where the plaque had been removed must be examined carefully and should be smooth. Tacking sutures (*arrows*) are placed when an intimal flap remains in this transition to ensure no obstruction to flow.



Fig. 9 A. An autologous or synthetic patch can be used to close the carotid arteriotomy incision, which maintains the luminal patency



Fig 9 B. A completion closure of carotid endarterectomy incision using a synthetic patch.

Complications of Carotid Endarterectomy

Most patients tolerate carotid endarterectomy very well and typically are discharged home within 24 hours after surgery. Complications after endarterectomy are infrequent but can be potentially life threatening or disabling. Acute ipsilateral stroke is a dreaded complication following carotid endarterectomy. Cerebral ischemia can be due to either intraoperative or postoperative events. Embolizations from the occlusive plaque or prolonged cerebral ischemia are potential causes of intraoperative stroke. The most common cause of postoperative stroke is due to embolization. Less frequently, acute carotid artery occlusion can cause acute postoperative stroke. This is usually due to carotid artery thrombosis related to closure of the arteriotomy, an occluding intimal flap, or distal carotid dissection. When patients experience acute symptoms of neurologic ischemia after endarterectomy, immediate intervention may be indicated. Carotid duplex scan can be done expeditiously to assess patency of the extracranial ICA. Re-exploration is mandated for acute carotid artery occlusion. Cerebral angiography can be useful if intracranial revascularization is considered.

Local complications related to surgery include excessive bleeding and cranial nerve palsies. Postoperative hematoma in the neck after carotid endarterectomy can lead to devastating airway compromise. Any expanding hematoma should be evacuated and active bleeding stopped. Securing an airway is critical and can be extremely difficult in patients with large postoperative neck hematomas. The reported incidence of postoperative cranial nerve palsies after carotid endarterectomy varies from 1 to 30%. Well-recognized injuries involve the

marginal mandibular, vagus, hypoglossal, superior laryngeal, and recurrent laryngeal nerves. Often these are traction injuries but can also be due to severance of the respective nerves.

Techniques of Carotid Angioplasty and Stenting

Percutaneous carotid artery stenting has become an accepted alternative treatment in the management of patients with carotid bifurcation disease (Fig. 10). The perceived advantages of percutaneous carotid revascularization are related to the minimal invasiveness of the procedure compared to surgery. There are anatomical conditions based on angiographic evaluation in which carotid artery stenting should be avoided due to increased procedural-related risks (Table 3). In preparation for carotid stenting, the patient should be given oral clopidogrel 3 days before the intervention if the patient was not already taking the drug. The procedure is done in either the OR with angiographic capabilities or in a dedicated angiography room. The patient is placed in the supine position. The patient's BP and cardiac rhythm are closely monitored.



Fig. 10 A. Carotid angiogram demonstrated a high-grade stenosis of the left internal carotid artery



Fig. 10 B. Completion angiogram demonstrating a satisfactory result following a carotid stent placement.

Table 3. Unfavorable Carotid Angiographic Appearance in Which Carotid Stenting Should Be Avoided

• Extensive carotid calcification
• Polyloid or globular carotid lesions
• Severe tortuosity of the common carotid artery
• Long segment stenoses (>2 cm in length)
• Carotid artery occlusion
• Severe intraluminal thrombus (angiographic defects)
• Extensive middle cerebral artery atherosclerosis

To gain access to the carotid artery, a retrograde transfemoral approach is most commonly used as the access site for carotid intervention. Using the Seldinger technique, a diagnostic 5F or 6F sheath is inserted in the CFA. A diagnostic arch aortogram is obtained. The carotid artery to be treated is then selected using a 5F diagnostic catheter, and contrast is injected to show the carotid anatomy. It is important to assess the contralateral carotid artery, vertebrobasilar, and intracranial circulation if these are not known based on the preoperative, noninvasive studies. Once the decision is made to proceed with carotid artery stenting, with the tip of the diagnostic catheter still in the CCA, a 0.035-in, 260-cm long stiff glide wire is placed

in the ipsilateral external carotid artery. Anticoagulation with IV bivalirudin bolus (0.75mg/kg) followed by an infusion rate of 2.5 mg/kg per hour for the remainder of the procedure is routinely administered. Next, the diagnostic catheter is withdrawn and a 90-cm 6F guiding sheath is advanced into the CCA over the stiff glide wire. It is critical not to advance the sheath beyond the occlusive plaque in the carotid bulb. The stiff wire is then removed and preparation is made to deploy the distal embolic protection device (EPD). Several distal EPDs are available (Table 4). The EPD device is carefully deployed beyond the target lesion. With regard to the carotid stents, there are several stents that have received approval from the FDA and are commercially available for carotid revascularization (Table 5). All current carotid stents use the rapid-exchange monorail 0.014-in platform. The size selection is typically based on the size of CCA. Predilatation using a 4-mm balloon may be necessary to allow passage of the stent delivery catheter. Once the stent is deployed across the occlusive plaque, postdilatation is usually performed using a 5.5-mm or less balloon. It's noteworthy that balloon dilation of the carotid bulb may lead to immediate bradycardia due to stimulation of the glossopharyngeal nerve. The EPD is then retrieved and the procedure is completed with removal of the sheath from the femoral artery. The puncture site is closed using an available closure device or with manual compression. Throughout the procedure, the patient's neurologic function is closely monitored. The bivalirudin infusion is stopped, and the patient is kept on clopidogrel (75 mg daily) for at least 1 month and aspirin indefinitely.

Table 4. Commonly Used Embolic Protection Devices (EPDs)

Mechanism	Name of EPD	Pore Size (micrometers)
Distal balloon occlusion	PercuSurge Guard Wire, Export catheter (Medtronic)	N/A
Distal filter	Angioguard (Cordis)	100
	AccUNET (Abbott)	150
	EmboShield (Abbott)	140
	FilterWire (Boston Scientific)	110
	SpiderRx (EV3)	<100
Flow reversal ^a	Parodi Neuro Protection (Gore)	N/A

Table 5 Currently Approved Carotid Stents in the United States

Name of Stent	Manufacturer	Cell Design	Tapered Stent	Delivery System Size (F)
Acculink	Abbott	Open	Yes	6
Exact	Abbott	Closed	Yes	6
NexStent	Boston Scientific	Closed	Self-tapering	5
Protégé RX	EV3	Open	Yes	6
Precise RX	Cordis	Open	No	6
Exponent	Medtronic	Open	No	6

Complications of Carotid Stenting

Although there have been no randomized trials comparing carotid stenting with and without EPD, the availability of EPDs appears to have reduced the risk of distal embolization and stroke. The results of the various clinical trials and registries of carotid stenting have been reported and compared. It is well known that distal embolization as detected by TCD is much more frequent with carotid stenting, even with EPD, when compared to carotid endarterectomy. However, the clinical significance of the distal embolization detected by TCD is not clear, as most are asymptomatic. Acute carotid stent thrombosis is rare. The incidence of in-stent carotid restenosis is not well known but is estimated at 10 to 30%. Duplex surveillance shows elevated peak systolic velocities within the stent after carotid stenting can occur frequently. However, velocity criteria are being formulated to determine the severity of in-stent restenosis after carotid stenting by ultrasound duplex. It appears that systolic velocities exceeding 300 to 400 cm/s would represent greater than 70 to 80% restenosis. Bradycardia and hypotension occurs in up to 20% of patients undergoing carotid stenting. Systemic administration of atropine is usually effective in reversing the bradycardia. Other technical complications of carotid stenting are infrequent and include carotid artery dissection, and access site complications such as groin hematoma, femoral artery pseudoaneurysm, distal embolization, and acute femoral artery thrombosis.

NON-ATHEROSCLEROTIC DISEASE OF THE CAROTID ARTERY

Carotid Coil and Kink

A carotid coil consists of an excessive elongation of the ICA producing tortuosity of the vessel (Fig. 11). Embryologically, the carotid artery is derived from the third aortic arch and

dorsal aortic root, and is uncoiled as the heart and great vessels descend into the mediastinum. In children, carotid coils appear to be congenital in origin. In contrast, elongation and kinking of the carotid artery in adults is associated with the loss of elasticity and an abrupt angulation of the vessel. Kinking is more common in women than men. Cerebral ischemic symptoms caused by kinks of the carotid artery are similar to those from atherosclerotic carotid lesions, but are more likely due to cerebral hypoperfusion than embolic episodes. Classically, sudden head rotation, flexion, or extension can accentuate the kink and provoke ischemic symptoms. Most carotid kinks and coils are found incidentally on carotid duplex scan. However, interpretation of the Doppler frequency shifts and spectral analysis in tortuous carotid arteries can be difficult because of the uncertain angle of insonation. Cerebral angiography, with multiple views taken in neck flexion, extension, and rotation, is useful in the determination of the clinical significance of kinks and coils.

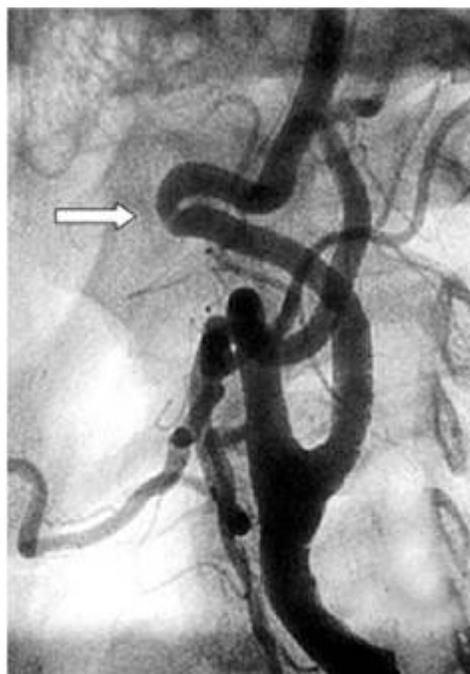


Fig. 11. Excessive elongation of the carotid artery can result in carotid kinking (*arrow*), which can compromise cerebral blood flow and lead to cerebral ischemia.

Fibromuscular Dysplasia

FMD usually involves medium-sized arteries that are long and have few branches (Fig. 12). Women in the fourth or fifth decade of life are more commonly affected than men. Hormonal effects on the vessel wall are thought to play a role in the pathogenesis of FMD.

FMD of the carotid artery is commonly bilateral, and in about 20% of patients, the vertebral artery also is involved. An intracranial saccular aneurysm of the carotid siphon or middle cerebral artery can be identified in up to 50% of the patients with FMD. Four histological types of FMD have been described in the literature. The most common type is medial fibroplasia, which may present as a focal stenosis or multiple lesions with intervening aneurysmal outpouchings. The disease involves the media with the smooth muscle being replaced by fibrous connective tissue. Commonly, mural dilations and microaneurysms can be seen with this type of FMD. Medial hyperplasia is a rare type of FMD, with the media demonstrating excessive amounts of smooth muscle. Intimal fibroplasia accounts for 5% of all cases and occurs equally in both sexes. The media and adventitia remain normal, and there is accumulation of subendothelial mesenchymal cells with a loose matrix of connective tissue causing a focal stenosis in adults. Finally, premedial dysplasia represents a type of FMD with elastic tissue accumulating between the media and adventitia. FMD also can involve the renal and the external iliac arteries. It is estimated that approximately 40% of patients with FMD present with a TIA due to embolization of platelet aggregates. DSA demonstrates the characteristic "string of beads" pattern, which represents alternating segments of stenosis and dilatation. The string of beads can also be shown noninvasively by CTA or MRA. FMD should be suspected when an increased velocity is detected across a stenotic segment without associated atherosclerotic changes on carotid duplex ultrasound. Antiplatelet medication is the generally accepted therapy for asymptomatic lesions. Endovascular treatment is recommended for patients with documented lateralizing symptoms. Surgical correction is rarely indicated.

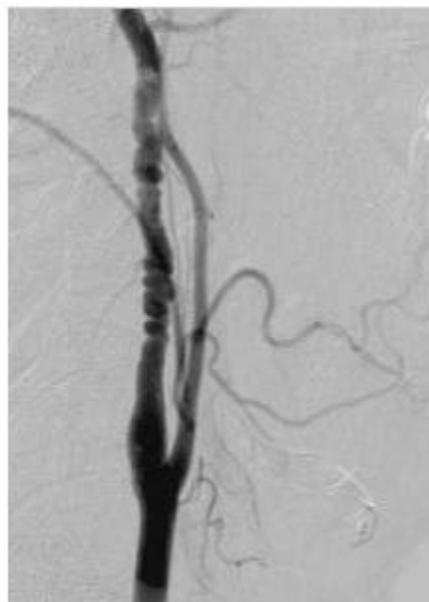


Fig. 12. A carotid fibromuscular dysplasia with typical characteristics of multiple stenosis with

intervening aneurysmal outpouching dilatations. The disease involves the media, with the smooth muscle being replaced by fibrous connective tissue

Carotid Artery Dissection

Dissection of the carotid artery accounts for approximately 20% of strokes in patients younger than 45 years of age. The etiology and pathogenesis of spontaneous carotid artery dissection remains incompletely understood. Arterial dissection involves hemorrhage within the media, which can extend into the subadventitial and subintimal layers. When the dissection extends into the subadventitial space, there is an increased risk of aneurysm formation. Subintimal dissections can lead to intramural clot or thrombosis. Traumatic dissection is typically a result of hyperextension of the neck during blunt trauma, neck manipulation, strangulation, or penetrating injuries to the neck. Even in supposedly spontaneous cases, a history of preceding unrecognized minor neck trauma is not uncommon. Connective disorders such as Ehlers-Danlos syndrome, Marfan syndrome, alpha1-antitrypsin deficiency, or FMD may predispose to carotid artery dissection. Iatrogenic dissections also can occur due to catheter manipulation or balloon angioplasty.

Typical clinical features of carotid artery dissection include unilateral neck pain, headache, and ipsilateral Horner's syndrome in up to 50% of patients, followed by manifestations of the cerebral or ocular ischemia and cranial nerve palsies. Neurologic deficits can result either because of hemodynamic failure (caused by luminal stenosis) or by an artery to artery thromboembolism. The ischemia may cause TIAs or infarctions, or both. Catheter angiography has been the method of choice to diagnose arterial dissections, but with the advent of duplex ultrasonography, MRI/MRA, and CTA, most dissections can now be diagnosed using noninvasive imaging modalities (Fig. 13). The dissection typically starts in the ICA distal to the bulb. Uncommonly, the dissection can start in the CCA, or is an extension of a more proximal aortic dissection. Medical therapy has been the accepted primary treatment of symptomatic carotid artery dissection. Anticoagulation (heparin and warfarin) and antiplatelet therapy have been commonly used, although there have not been any randomized studies to evaluate their effectiveness. The prognosis depends on the severity of neurologic deficit but is generally good in extracranial dissections. The recurrence rate is low. Therapeutic interventions have been reserved for recurrent TIAs or strokes, or failure of medical treatment. Endovascular options include intra-arterial stenting, coiling of associated pseudoaneurysms, or more recently, deployment of covered stents.

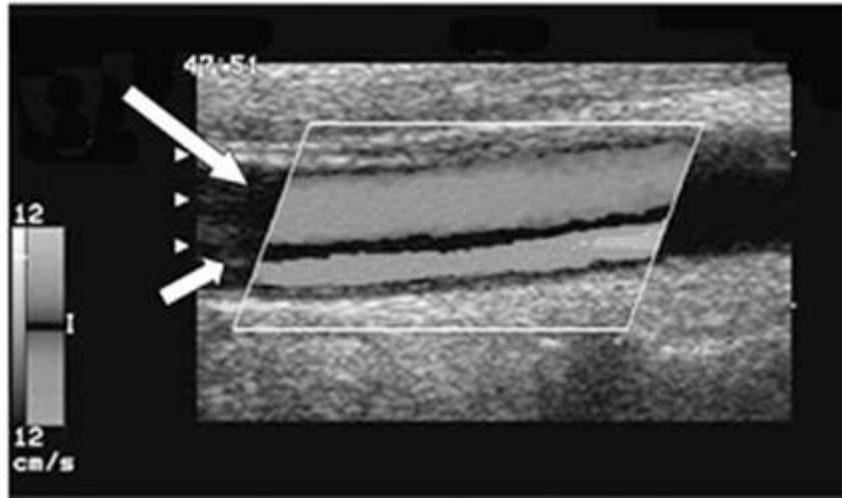


Fig. 13. Carotid ultrasound reveals a patient with a carotid artery dissection in which carotid flow is separated in the true flow lumen (*long arrow*) from the false lumen (*short arrow*).

Carotid Artery Aneurysms

Carotid artery aneurysms are rare, encountered in less than 1% of all carotid operations (Fig. 14). The true carotid artery aneurysm generally is due to atherosclerosis or medial degeneration. The carotid bulb is involved in most carotid aneurysms, and bilaterality is present in 12% of the patients. Patients typically present with a pulsatile neck mass. The available data suggest that, untreated, these aneurysms lead to neurologic symptoms from embolization. Thrombosis and rupture of the carotid aneurysm is rare. Pseudoaneurysms of the carotid artery can result from injury or infection. Mycotic aneurysms often involve syphilis in the past, but are now more commonly associated with peritonsillar abscesses caused by *Staphylococcus aureus* infection. FMD and spontaneous dissection of the carotid artery can lead to the formation of true aneurysms or pseudoaneurysms. Whereas conventional surgery has been the primary mode of treatment in the past, carotid aneurysms are currently being treated more commonly using endovascular approaches.



Fig. 14 A. An anteroposterior angiogram of the neck revealing a carotid artery aneurysm

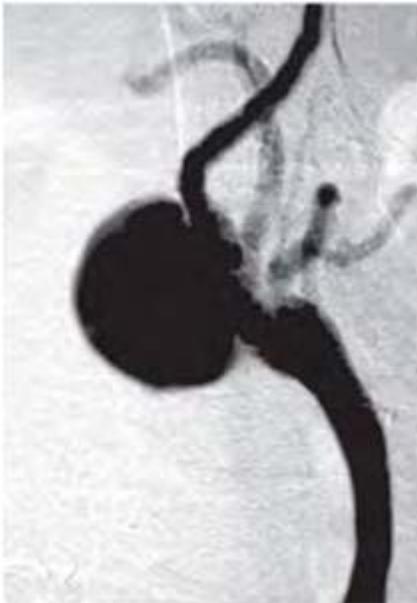


Fig. 14 B. A lateral projection of the carotid artery aneurysm



Fig. 14 C. Following endovascular placement, the carotid artery aneurysm is successfully excluded.

Carotid Body Tumor

The carotid body originates from the third branchial arch and from neuroectodermal-derived neural crest lineage. The normal carotid body is located in the adventitia or periadventitial tissue at the bifurcation of the CCA (Fig. 15). The gland is innervated by the glossopharyngeal nerve. Its blood supply is derived predominantly from the external carotid artery, but also can come from the vertebral artery. Carotid body tumor is a rare lesion of the neuroendocrine system. Other glands of neural crest origin are seen in the neck, parapharyngeal spaces, mediastinum, retroperitoneum, and adrenal medulla. Tumors involving these structures have been referred to as *paraganglioma*, *glomus tumor*, or *chemodectoma*. Approximately 5 to 7% of carotid body tumors are malignant. Although chronic hypoxemia has been invoked as a stimulus for hyperplasia of carotid body, approximately 35% of carotid body tumors are hereditary. The risk of malignancy is greatest in young patients with familial tumors.

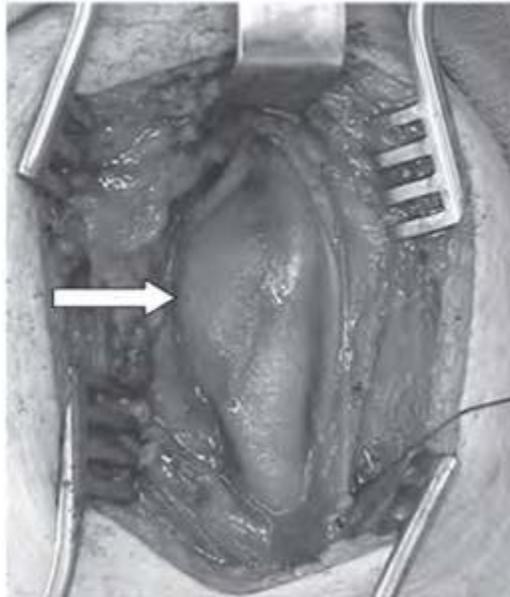


Fig. 15 A. A carotid body tumor (*arrow*) located adjacent to the carotid bulb



Fig. 15 B. Following periadventitial dissection, the carotid body tumor is removed

Symptoms related to the endocrine products of the carotid body tumor are rare. Patients usually present between the fifth and seventh decade of life with an asymptomatic lateral neck mass. The diagnosis of carotid body tumor requires confirmation on imaging studies. Carotid duplex scan can localize the tumor to the carotid bifurcation, but CT or MR imaging usually is required to further delineate the relationship of the tumor to the adjacent structures.

Classically, a carotid body tumor will widen the carotid bifurcation. The Shamblin classification describes the tumor extent: I. tumor is <5 cm and relatively free of vessel involvement; II. tumor is intimately involved but does not encase the vessel wall; and III. tumor is intramural and encases the carotid vessels and adjacent nerves. With good resolution CT and MR imaging, arteriography usually is not required. However, arteriography can provide an assessment of the vessel invasion and intracranial circulation, and allows for preoperative embolization of the feeder vessels, which has been reported to reduce intraoperative blood loss. Surgical resection is the recommended treatment for suspected carotid body tumor.

THORACIC AORTIC ANEURYSM

Introduction

Aneurysmal degeneration can occur anywhere in the human aorta. By definition, an aneurysm is a localized or diffuse dilation of an artery with a diameter at least 50% greater than the normal size of the artery.

A blood vessel has 3 layers: the intima (inner layer made of endothelial cells), media (contains muscular elastic fibers), and adventitia (outer connective tissue). Aneurysms are either true or false. The wall of a true aneurysm involves all 3 layers, and the aneurysm is contained inside the endothelium. The wall of a false or pseudoaneurysm only involves the outer layer and is contained by the adventitia. An aortic dissection is formed by an intimal tear and is contained by the media; hence, it has a true lumen and a false lumen.

Most aortic aneurysms (AA) occur in the abdominal aorta; these are termed abdominal aortic aneurysms (AAA). Although most abdominal aortic aneurysms are asymptomatic at the time of diagnosis, the most common complication remains life-threatening rupture with hemorrhage.

Aneurysmal degeneration that occurs in the thoracic aorta is termed a thoracic aneurysm (TA). Aneurysms that coexist in both segments of the aorta (thoracic and abdominal) are termed thoracoabdominal aneurysms (TAA). Thoracic aneurysms and thoracoabdominal aneurysms are also at risk for rupture. A recent population-based study suggests an increasing prevalence of thoracic aortic aneurysms. Thoracic aortic aneurysms are subdivided into 3 groups depending

on location: ascending aortic, aortic arch, and descending thoracic aneurysms or thoracoabdominal aneurysms. Aneurysms that involve the ascending aorta may extend as proximally as the aortic annulus and as distally as the innominate artery, whereas descending thoracic aneurysms begin beyond the left subclavian artery. Arch aneurysms are as the name implies.

Dissection is another condition that may affect the thoracic aorta. An intimal tear causes separation of the walls of the aorta. A false passage for blood develops between the layers of the aorta. This false lumen may extend into branches of the aorta in the chest or abdomen, causing malperfusion, ischemia, or occlusion with resultant complications. The dissection can also progress proximally, to involve the aortic sinus, aortic valve, and coronary arteries. Dissection can lead to aneurysmal change and early or late rupture. A chronic dissection is one that is diagnosed more than 2 weeks after the onset of symptoms. Dissection should not be termed dissecting aneurysm because it can occur with or without aneurysmal enlargement of the aorta.

The shape of an aortic aneurysm is either saccular or fusiform. A fusiform (or true) aneurysm has a uniform shape with a symmetrical dilatation that involves the entire circumference of the aortic wall. A saccular aneurysm is a localized outpouching of the aortic wall, and it is the shape of a pseudoaneurysm.

Treatment of abdominal aortic aneurysms, thoracoabdominal aneurysms, and thoracic aneurysms involves surgical repair in good-risk patients with aneurysms that have reached a size sufficient to warrant repair. Surgical repair may involve endovascular stent grafting (in suitable candidates) or traditional open surgical repair.

History of the Procedure

The development of treatment modalities for thoracic aneurysms followed successful treatment of abdominal aortic aneurysms. Estes' 1950 report revealed that the 3-y survival rate for patients with untreated abdominal aortic aneurysms was only 50%, with two thirds of deaths resulting from aneurysmal rupture. Since then, increased attempts were made to devise methods of durable repair.

Most of these initial successful repairs involved the use of preserved aortic allografts, thus triggering the establishment of numerous aortic allograft banks. Simultaneously, Gross and colleagues successfully used allografts to treat complex thoracic aortic coarctations, including those with aneurysmal involvement.

In 1951, Lam and Aram reported the resection of a descending thoracic aneurysm with allograft replacement.³ Ascending aortic replacement required the development of cardiopulmonary bypass and was first performed in 1956 by Cooley and DeBakey. They successfully replaced the ascending aorta with an aortic allograft. Successful replacement of the aortic arch, with its inherent risk of cerebral ischemia, was understandably more challenging and was not reported until 1957 by DeBakey et al.

Although the use of aortic allografts as aortic replacement was widely accepted in the early 1950s, the search for synthetic substitutes was well underway. Dacron was introduced by DeBakey. By 1955, Deterling and Bhonslay believed that Dacron was the best material for aortic substitution.⁶ Numerous types of intricately woven hemostatic grafts have since been developed and are now used much more extensively than their allograft counterparts. Such Dacron grafts are used to replace ascending, arch, thoracic, and thoracoabdominal aortic segments.

However, some patients required replacement of the aortic root, as well. Subsequently, combined operations that replaced the ascending aneurysm in conjunction with replacement of the aortic valve and reimplantation of the coronary arteries were performed by Bentall and De Bono in 1968, using a mechanical valve with a Dacron conduit. Ross, in 1962, and Barratt-Boyes, in 1964, successfully implanted the aortic homograft in the orthotopic position. In 1985, Sievers reported the use of stentless porcine aortic roots.

More recently, less invasive therapy for descending thoracic aortic aneurysm have been developed. Dake et al reported the first endovascular thoracic aortic repair in 1994.¹¹ In March 2005, the US Food and Drug Administration (FDA) approved the first thoracic aortic stent graft, the GORE TAG graft (W.L. Gore and Associates; Newark, Del).

Problem

Aneurysms are usually defined as a localized dilation of an arterial segment greater than 50% its normal diameter. Most aortic aneurysms occur in the infrarenal segment (95%). The average size for an infrarenal aorta is 2 cm; therefore, abdominal aortic aneurysms are usually defined by diameters greater than 3 cm.

The normal size for the thoracic and thoracoabdominal aorta is larger than that of the infrarenal aorta, and aneurysmal degeneration in these areas is defined accordingly. The average diameter of the mid-descending thoracic aorta is 26-28 mm, compared with 20-23 mm at the level of the celiac axis.

Frequency

Although findings from autopsy series vary widely, the prevalence of aortic aneurysms probably exceeds 3-4% in individuals older than 65 years.

Death from aneurysmal rupture is one of the 15 leading causes of death in most series. The estimated incidence of thoracic aortic aneurysms is 6 cases per 100,000 person-years. In addition, the overall prevalence of aortic aneurysms has increased significantly in the last 30 years. This is partly due to an increase in diagnosis based on the widespread use of imaging techniques. However, the prevalence of fatal and nonfatal rupture has also increased, suggesting a true increase in prevalence. Population-based studies suggest an incidence of acute aortic dissection of 3.5 per 100,000 persons; an incidence of thoracic aortic rupture of 3.5 per 100,000 persons; and an incidence of abdominal aortic rupture of 9 per 100,000 persons. An aging population probably plays a significant role.

Etiology

Aneurysmal degeneration occurs more commonly in the aging population. Aging results in changes in collagen and elastin, which lead to weakening of the aortic wall and aneurysmal dilation. According to the Laplace law, luminal dilation results in increased wall tension and the vicious cycle of progressive dilation and greater wall stress. Pathologic sequelae of the aging aorta include elastic fiber fragmentation and cystic medial necrosis. Arteriosclerotic (degenerative) disease is the most common cause of thoracic aneurysms.

A previous aortic dissection with a persistent false channel may produce aneurysmal dilation; such aneurysms are the second most common type. False aneurysms are more common in the descending aorta and arise from the extravasation of blood into a tenuous pocket contained by the aortic adventitia. Because of increasing wall stress, false aneurysms tend to enlarge over time.

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Authorities strongly agree that genetics play a role in the formation of aortic aneurysms. Of first-degree relatives of patients with aortic aneurysms, 15% have an aneurysm. This appears especially true in first-degree relatives of female patients with aortic aneurysms. Thus, inherited disorders of connective tissue appear to contribute to the formation of aortic aneurysms.

Pathophysiology

The occurrence and expansion of an aneurysm in a given segment of the arterial tree probably involves local hemodynamic factors and factors intrinsic to the arterial segment itself.

The medial layer of the aorta is responsible for much of its tensile strength and elasticity. Multiple structural proteins comprise the normal medial layer of the human aorta. Of these, collagen and elastin are probably the most important. The elastin content of the ascending aorta is high and diminishes progressively in the descending thoracic and abdominal aorta. The infrarenal aorta has a relative paucity of elastin fibers in relation to collagen and compared with the thoracic aorta, possibly accounting for the increased frequency of aneurysms in this area. In

addition, the activity and amount of specific enzymes is increased, which leads to the degradation of these structural proteins. Elastic fiber fragmentation and loss with degeneration of the media result in weakening of the aortic wall, loss of elasticity, and consequent dilation.

Hemodynamic factors probably play a role in the formation of aortic aneurysms. The human aorta is a relatively low-resistance circuit for circulating blood. The lower extremities have higher arterial resistance, and the repeated trauma of a reflected arterial wave on the distal aorta may injure a weakened aortic wall and contribute to aneurysmal degeneration. Systemic hypertension compounds the injury, accelerates the expansion of known aneurysms, and may contribute to their formation.

Hemodynamically, the coupling of aneurysmal dilation and increased wall stress is defined by the law of Laplace. Specifically, the law of Laplace states that the (arterial) wall tension is proportional to the pressure times the radius of the arterial conduit ($T = P \times R$). As diameter increases, wall tension increases, which contributes to increasing diameter. As tension increases, risk of rupture increases. Increased pressure (systemic hypertension) and increased aneurysm size aggravate wall tension and therefore increase the risk of rupture.

Aneurysm formation is probably the result of multiple factors affecting that arterial segment and its local environment.

Presentation

Most patients with aortic aneurysms are asymptomatic at the time of discovery. Thoracic aneurysms are usually found incidentally after chest radiographs or other imaging studies. Abdominal aortic aneurysms may be discovered incidentally during imaging studies or a routine physical examination as a pulsatile abdominal mass.

The most common complication of abdominal aortic aneurysms is rupture with life-threatening hemorrhage manifesting as pain and hypotension. The triad of abdominal pain, hypotension, and a pulsatile abdominal mass is diagnostic of a ruptured abdominal aortic aneurysm, and emergent operation is warranted without delay for imaging studies.

Patients with a variant of abdominal aortic aneurysm may present with fever and a

painful aneurysm with or without an obstructive uropathy. These patients may have an inflammatory aneurysm that can be treated with surgical repair.

Other presentations of abdominal aortic aneurysm include lower extremity ischemia, duodenal obstruction, ureteral obstruction, erosion into adjacent vertebral bodies, aortoenteric fistula (ie, GI bleed), or aortocaval fistula (caused by spontaneous rupture of aneurysm into the adjacent inferior vena cava [IVC]). Patients with aortocaval fistula present with abdominal pain, venous hypertension (ie, leg edema), hematuria, and high output cardiac failure.

Patients with thoracic aneurysms are often asymptomatic. Most patients are hypertensive but remain relatively asymptomatic until the aneurysm expands. Their most common presenting symptom is pain. Pain may be acute, implying impending rupture or dissection, or chronic, from compression or distension. The location of pain may indicate the area of aortic involvement, but this is not always the case. Ascending aortic aneurysms tend to cause anterior chest pain, while arch aneurysms more likely cause pain radiating to the neck. Descending thoracic aneurysms more likely cause back pain localized between the scapulae. When located at the level of the diaphragmatic hiatus, the pain occurs in the mid back and epigastric region.

Large ascending aortic aneurysms may cause superior vena cava obstruction manifesting as distended neck veins. Ascending aortic aneurysms also may develop aortic insufficiency, with widened pulse pressure or a diastolic murmur, and heart failure. Arch aneurysms may cause hoarseness, which results from stretching of the recurrent laryngeal nerves. Descending thoracic aneurysms and thoracoabdominal aneurysms may compress the trachea or bronchus and cause dyspnea, stridor, wheezing, or cough. Compression of the esophagus results in dysphagia. Erosion into surrounding structures may result in hemoptysis, hematemesis, or gastrointestinal bleeding. Erosion into the spine may cause back pain or instability. Spinal cord compression or thrombosis of spinal arteries may result in neurologic symptoms of paraparesis or paraplegia. Descending thoracic aneurysms may thrombose or embolize clot and atheromatous debris distally to visceral, renal, or lower extremities.

Patients who present with ecchymoses and petechiae may be particularly challenging because these signs probably indicate disseminated intravascular coagulation (DIC). The risk of significant perioperative bleeding is extremely high, and large amounts of blood and blood products must be available for resuscitative transfusion.

The most common complications of thoracic aortic aneurysms are acute rupture or dissection. Some patients present with tender or painful nonruptured aneurysms. Although debate continues, these patients are thought to be at increased risk for rupture and should undergo surgical repair on an emergent basis.

Marfan syndrome is a potentially lethal connective-tissue disease characterized by skeletal, heart valve, and ocular abnormalities. Individuals with this disease are at risk for aneurysmal degeneration, especially in the thoracic aorta. Marfan syndrome is an autosomal dominant genetic condition that results in abnormal fibrillin, a structural protein found in the human aorta. Patients with Marfan syndrome may develop annuloaortic ectasia of the sinuses of Valsalva, commonly associated with aortic valvular insufficiency and aneurysmal dilation of the ascending aorta.

Type IV Ehlers-Danlos syndrome results in a deficiency in the production of type III collagen, and individuals with this disease may develop aneurysms in any portion of the aorta. Imbalances in the synthesis and degradation of structural proteins of the aorta have also been discovered, which may be inherited or spontaneous mutations.

Atherosclerosis may play a role. Whether atherosclerosis contributes to the formation of an aneurysm or whether they occur concomitantly is not established. Other causes of aortic aneurysms are infection (ie, bacterial [mycotic or syphilitic]), arteritis (ie, giant cell, Takayasu, Kawasaki, Behçet), and trauma. Aortitis due to granulomatous disease is rare, but it can lead to the formation of aortic and, on occasion, pulmonary artery aneurysms. Aortitis caused by syphilis may cause destruction of the aortic media followed by aneurysmal dilation.

Traumatic dissection is a result of shearing from deceleration injury due to high speed motor vehicle accidents (MVA) or a fall from heights. The dissection occurs at a point of fixation, usually at the aortic isthmus (ie, at the ligamentum arteriosum, distal to the origin of the left subclavian artery), the ascending aorta, the aortic root, and the diaphragmatic hiatus.

The true etiology of aortic aneurysms is probably multifactorial, and the condition occurs in individuals with multiple risk factors. Risk factors include smoking, chronic obstructive pulmonary disease (COPD), hypertension, atherosclerosis, male gender, older age, high BMI, bicuspid or unicuspid aortic valves, genetic disorders, and family history. Aortic

aneurysms are more common in men than in women and are more common in persons with COPD than in those without lung disease.

Indications

Indications for surgery of thoracic aortic aneurysms are based on size or growth rate and symptoms. Because the risk of rupture is proportional to the diameter of the aneurysm, aneurysmal size is the criterion for elective surgical repair. Elefteriades published the natural history of thoracic aortic aneurysms and recommends elective repair of ascending aneurysms at 5.5 cm and descending aneurysms at 6.5 cm for patients without any familial disorders such as Marfan syndrome. These recommendations are based on the finding that the incidence of complications (rupture and dissection) exponentially increased when the size of the ascending aorta reached 6.0 cm (31% risk of complications) or when the size of the descending aorta reached 7.0 cm (43% risk).^{15,14} Patients with Marfan syndrome or familial aneurysms should undergo earlier repair, when the ascending aorta grows to 5.0 cm or the descending aorta grows to 6.0 cm.

In addition, relative aortic aneurysm size in relation to body surface area may be more important than absolute aortic size in predicting complications.¹⁶ Using the aortic size index (ASI) of aortic diameter (in cm) divided by body surface area (m²), patients are stratified into 3 groups: ASI <2.75 cm/m² are at low risk for rupture (4%/y), ASI 2.75-4.25 cm/m² are at moderate risk (8%/y), and ASI >4.25cm/m² are at high risk (20-25%/y).

Rapid expansion is also a surgical indication. Growth rates average 0.07 cm/y in the ascending aorta and 0.19 cm/y in the descending aorta.¹⁴ A growth rate of 1 cm/y or faster is an indication for elective surgical repair.

Symptomatic patients should undergo aneurysm resection regardless of size. Acutely symptomatic patients require emergent operation. Emergent operation is indicated in the setting of acute rupture. Rupture of the ascending aorta may occur into the pericardium, resulting in acute tamponade. Rupture of the descending thoracic aorta may cause a left hemothorax.

Patients with acute aortic dissection of the ascending aorta require emergent operation. They may present with rupture, tamponade, acute aortic insufficiency, myocardial infarction, or

end-organ ischemia. Acute dissection of the descending aorta does not require surgical intervention, unless complicated by rupture, malperfusion (eg, visceral, renal, neurologic, leg ischemia), progressive dissection, persistent recurrent pain, or failure of medical management.

Patients who undergo surgery for symptomatic aortic insufficiency or stenosis with an associated enlarged aneurysmal aorta should have concomitant aortic replacement if the aorta reaches 5 cm in diameter. Concomitant aortic replacement should be considered for patients with bicuspid aortic valves with an aorta >4.5 cm in diameter.

Summary of indications

Aortic size

Ascending aortic diameter ≥ 5.5 cm or twice the diameter of the normal contiguous aorta

Descending aortic diameter ≥ 6.5 cm

Subtract 0.5 cm from the cutoff measurement in the presence of Marfan syndrome, family history of aneurysm or connective tissue disorder, bicuspid aortic valve, aortic stenosis, dissection, patient undergoing another cardiac operation

Growth rate ≥ 1 cm/y

Symptomatic aneurysm

Traumatic aortic rupture

Acute type B aortic dissection with associated rupture, leak, distal ischemia

Pseudoaneurysm

Large saccular aneurysm

Mycotic aneurysm

Aortic coarctation

Bronchial compression by aneurysm

Aortobronchial or aorto-esophageal fistula

Relevant Anatomy

Ascending aortic aneurysms occur as proximally as the aortic annulus and as distally as the innominate artery. They may compress or erode into the sternum and ribs, causing pain or fistula. They also may compress the superior vena cava or airway. When symptomatic by rupture or dissection, they may involve the pericardium, aortic valve, or coronary arteries. They

may rupture into the pericardium, causing tamponade. They may dissect into the aortic valve, causing aortic insufficiency, or into the coronary arteries, causing myocardial infarction.

Aortic arch aneurysms involve the aorta where the innominate artery, left carotid, and left subclavian originate. They may compress the innominate vein or airway. They may stretch the left recurrent laryngeal nerve, causing hoarseness.

Descending thoracic aneurysms originate beyond the left subclavian artery and may extend into the abdomen. Thoracoabdominal aneurysms are stratified based on the Crawford classification. Type I involves the descending thoracic aorta from the left subclavian artery down to the abdominal aorta above the renal arteries. Type II extends from the left subclavian artery to the renal arteries and may continue distally to the aortic bifurcation. Type III begins at the mid-to-distal descending thoracic aorta and involves most of the abdominal aorta as far distal as the aortic bifurcation. Type IV extends from the upper abdominal aorta and all or none of the infrarenal aorta. Descending thoracic aneurysms and thoracoabdominal aneurysms may compress or erode into surrounding structures, including the trachea, bronchus, esophagus, vertebral body, and spinal column.

Contraindications

Aneurysm surgery has no strict contraindications. The relative contraindications are individualized, based on the patient's ability to undergo extensive surgery (ie, the risk-to-benefit ratio). Patients at higher risk for morbidity and mortality include elderly persons and individuals with end-stage renal disease, respiratory insufficiency, cirrhosis, or other comorbid conditions. For descending thoracic aneurysms, endovascular stent grafting is less invasive and is an ideal alternative (with appropriate anatomic considerations) to open repair for patients at high risk for complications of open repair. Stent grafts are also a reasonable alternative (with the appropriate anatomy) to open repair in patients who are not at high risk for complications. Patients must understand that life-long follow-up is required and that long-term durability is unknown.

Laboratory Studies

CBC count

Electrolyte evaluation and BUN/creatinine value: Determining renal function is important for stratifying morbidity.

Prothrombin time, international normalized ratio, and activated partial thromboplastin time

Blood type and crossmatch

Liver function tests and amylase lactate values: These tests are indicated for patients with acute dissection or risk of distal embolization.

Imaging Studies

Chest radiograph

In the case of ascending aortic aneurysms, chest x-rays may reveal a widened mediastinum, a shadow to the right of the cardiac silhouette, and convexity of the right superior mediastinum. Lateral films demonstrate loss of the retrosternal air space. However, the aneurysms may also be completely obscured by the heart, and the chest x-ray appear normal.

Plain chest radiographs may show a shadow anteriorly and slightly to the left for arch aneurysms and posteriorly and to the left for descending thoracic aneurysms. Aortic calcification may outline the borders of the aneurysm in the anterior, posterior, and lateral views in both the chest and abdomen.

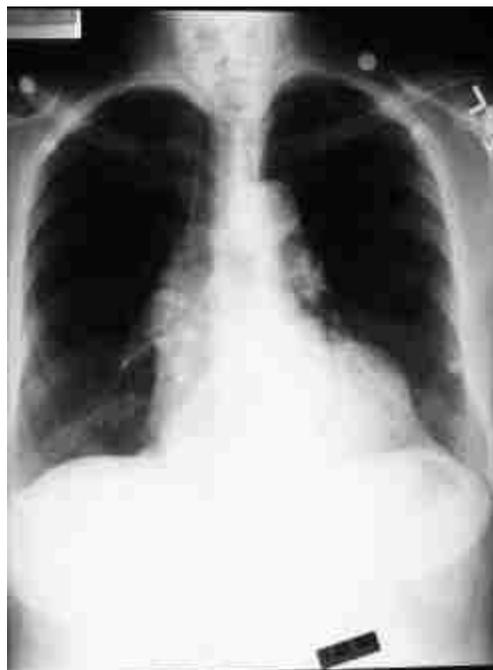


Fig. 16. Chest radiograph following aneurysm surgery

Echocardiography

Transthoracic echocardiography demonstrates the aortic valve and proximal aortic root. It may help detect aortic insufficiency and aneurysms of the sinus of Valsalva, but it is less sensitive and specific than transesophageal echocardiography.

Transesophageal echocardiography images show the aortic valve, ascending aorta, and descending thoracic aorta, but they are limited in the area of the distal ascending aorta, transverse aortic arch, and upper abdominal aorta. Transesophageal echocardiography can help accurately differentiate aneurysm and dissection, but the images must be obtained and interpreted by skilled personnel.

Ischemia may be evaluated using dipyridamole-thallium or dobutamine echocardiography scans.

Ultrasonography

Infrarenal abdominal aortic aneurysms may be visualized using ultrasonography, but these images do not help define the extent for thoracoabdominal aneurysms.

Carotid ultrasound may be needed for patients with carotid bruits, peripheral vascular disease, a history of transient ischemic attacks, or cerebrovascular accidents to evaluate for carotid disease.

Intraoperative intravascular ultrasound (IVUS) can also be used to provide additional anatomical information and guidance during placement of endovascular stents.

Intraoperative epiaortic ultrasound can be performed to scan the aorta for atherosclerotic disease or thrombus.

Aortography

Aortography images can delineate the aortic lumen, and they can help define the extent of the aneurysm, any branch vessel involvement, and the stenosis of branch vessels. It describes the takeoff of the coronary ostia.

For patients older than 40 years or those with a history suggestive of coronary artery disease, aortography helps evaluate coronary anatomy, ventricular function by ventriculography,

and aortic insufficiency. It does not help in defining the size of the aneurysm because the outer diameter is not measured, which may miss dissections.

Disadvantages include the use of nephrotoxic contrast and radiation. The risk of aortography includes embolization from laminated thrombus and carries a 1% stroke risk.

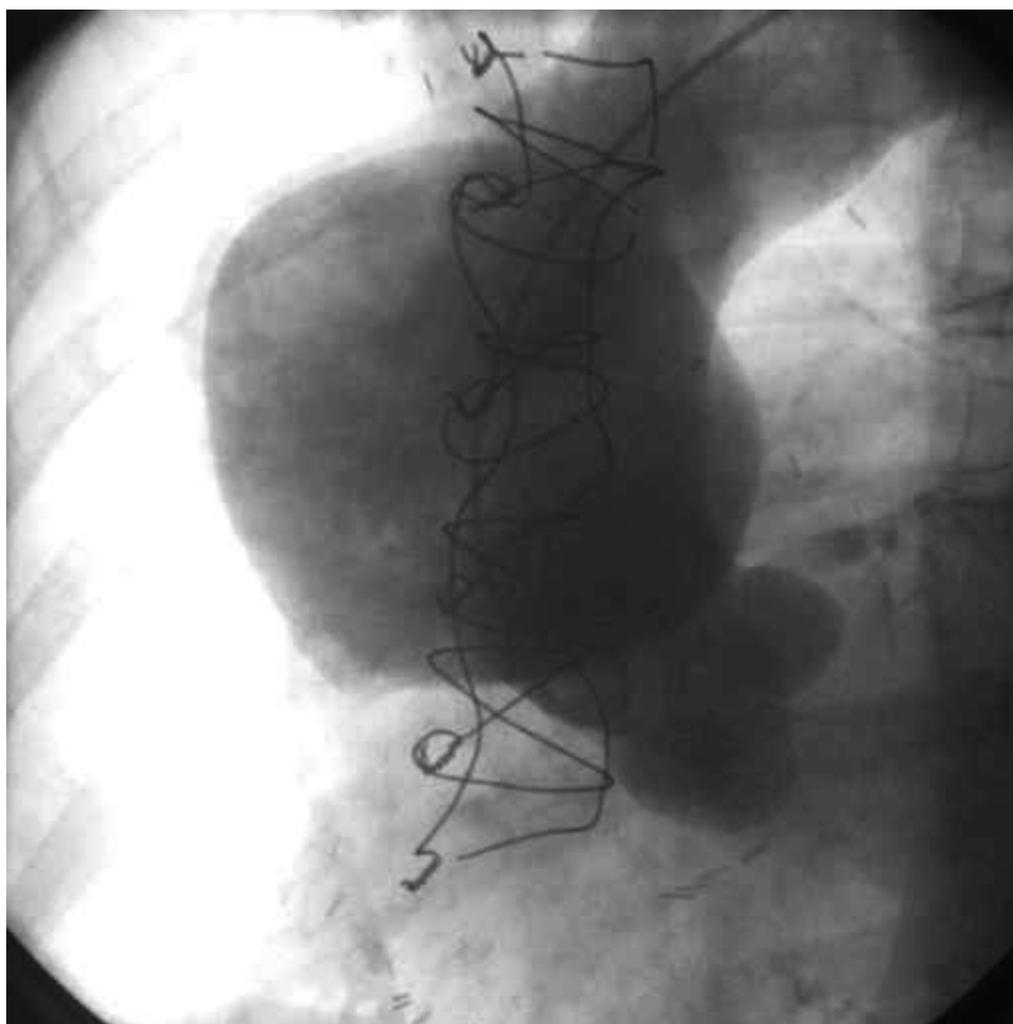


Fig.17. Ascending aortogram showing ascending aortic aneurysm
Computed tomography scan

CT scans with contrast have become the most widely used diagnostic tool. They rapidly and precisely evaluate the thoracic and abdominal aorta to determine the location and extent of the aneurysm and the relationship of the aneurysm to major branch vessels and surrounding structures. They can help accurately determine the size of the aneurysm and assesses dissection, mural thrombus, intramural hematoma, free rupture, and contained rupture with hematoma.

Sagittal, coronary, and axial images may be obtained with 3-dimensional reconstruction. Stent graft planning for endovascular descending thoracic aneurysm repairs requires fine-cut images from the neck through the pelvis to the level of the femoral heads. The takeoff of the arch vessels is critical to determine the adequacy of the proximal landing zone, as is assessing the patency of the vertebral arteries, if the left subclavian artery should be covered by the stent graft. Assessment of the common femoral artery access is essential to determine the feasibility of large-bore sheath access. A spiral CT scan with 1-mm cuts and 3-dimensional reconstruction with the ability to make centerline measurements is crucial to stent graft planning.

CT angiography may create multiplanar reconstructions and cines. This requires nephrotoxic contrast and radiation, but the procedure is noninvasive.

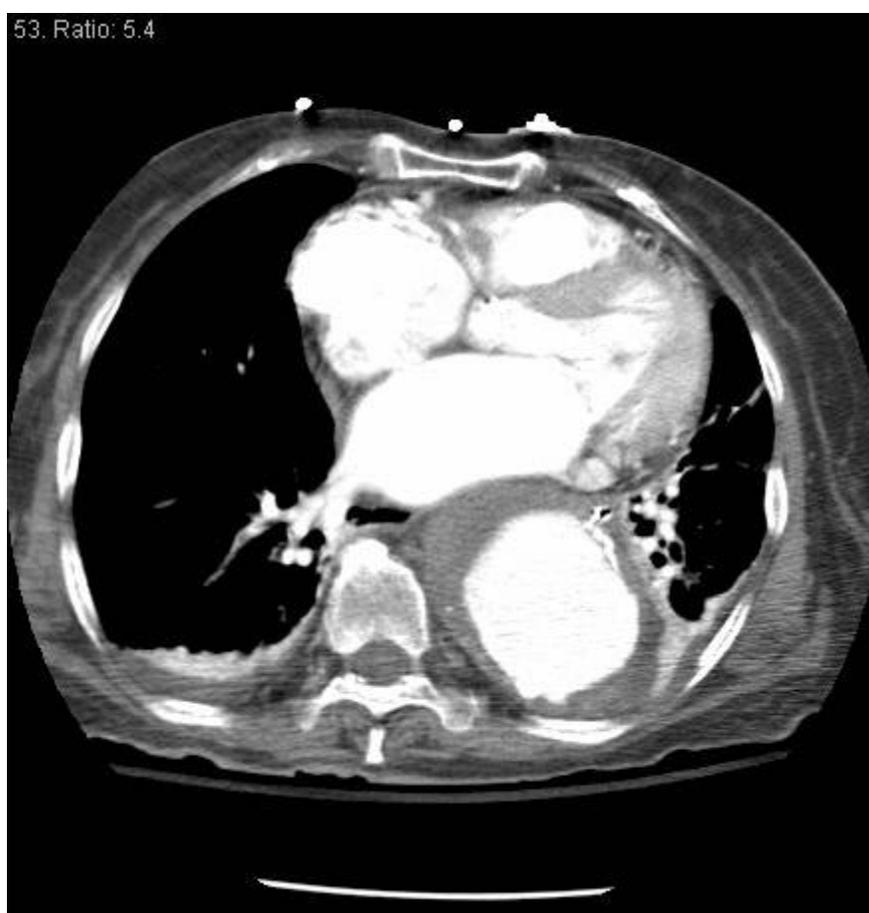


Fig.18. Descending thoracic aortic aneurysm with mural thrombus at the level of the left atrium

Magnetic resonance imaging

MRI and magnetic resonance angiography have the advantage of avoiding nephrotoxic

contrast and ionizing radiation compared with CT scans.

MRI and magnetic resonance angiography can also help accurately demonstrate the location, extent, and size of the aneurysm and its relationship to branch vessels and surrounding organs. These studies also precisely reveal aortic composition. However, they are more time consuming, less readily available, and more expensive than CT scans.

Other Tests

Electrocardiogram: Baseline ECG should be performed. Transthoracic echocardiograms noninvasively screen for valvular abnormalities and cardiac function.

Pulmonary function tests: Patients with a smoking history and COPD should be evaluated using pulmonary function tests with spirometry and room-air arterial blood gas determinations.

Diagnostic Procedures

Cardiac catheterization: Patients with a history of coronary artery disease or those older than 40 years should undergo cardiac catheterization.

Histologic Findings

Histologic findings may include elastic fiber fragmentation, loss of elastic fibers, loss of smooth muscle cells, cystic medial necrosis, intraluminal thrombus, and atherosclerotic plaque and ulceration.

Treatment

Medical Therapy

All aneurysms must be treated with risk-factor reduction. Systemic hypertension probably contributes to the formation of aneurysms and certainly contributes to expansion and rupture. This is especially true of thoracic aneurysms. Strict control of hypertension is implemented in all patients, regardless of aortic aneurysm size.

Tobacco use contributes to aneurysm formation, although the exact pathophysiology is not well understood. Cessation of smoking is recommended. Control of other risk factors for

peripheral arterial obstructive disease may be beneficial.

For acute aortic dissections, the first-line treatment of hypertension is with a short-acting beta-blocker (eg, esmolol). Beta blockade decreases the force of contraction, thus decreasing the dP/dt and shear force exerted on the dissection by minimizing the rate of rise of the aortic pressure. It also decreases the heart rate and the inotropic state of the myocardium, and reduces the likelihood of propagation of the dissection. A second-agent added is a vasodilator (eg, nitroprusside), which reduces the systolic blood pressure to, in turn, decrease the aortic wall stress and the possibility of rupture.

Surgical Therapy

Most aneurysm repairs involve aortic replacement with a Dacron tube graft. Dacron grafts allow ingrowth in the interstices to form a pseudoendothelial layer to minimize the risk of embolization. They may be knitted or woven. Knitted grafts are more porous and incorporate tissue well; however, they are prone to more bleeding. Woven grafts are more impervious and therefore are the most commonly used for aortic replacement. Grafts are typically impregnated with collagen to avoid preclotting the graft and to promote optimal healing.

Ascending aortic aneurysms

Surgical treatment of ascending aortic aneurysms depends on the extent of the aneurysm both proximally (eg, involvement of the aortic valve, annulus, sinuses of Valsalva, sinotubular junction, coronary orifices) and distally (eg, involvement to the level of the innominate artery). The choice of operation also depends on the underlying pathology of the disease, the patient's life expectancy, the desired anticoagulation status, and the surgeon's experience and preference.

Ascending aortic aneurysms with normal aortic valve leaflets, annulus, and sinuses of Valsalva are typically replaced with a simple supracoronary Dacron tube graft from the sinotubular junction to the origin of the innominate artery, with the patient under cardiopulmonary bypass.

If the aortic valve is diseased but the aortic sinuses and annulus are normal, the aortic valve is replaced separately and the ascending aortic aneurysm is replaced with a supracoronary

synthetic graft, leaving the coronary arteries intact (ie, Wheat procedure).

Sinus of Valsalva aneurysms with normal aortic valve leaflets and aortic insufficiency due to dilated sinuses may be repaired with a valve-sparing aortic root replacement. Two valve-sparing procedures have been developed: the remodeling method and the reimplantation method. The remodeling method involves resecting the aneurysmal sinus tissue while maintaining the tissue along the valve leaflets and scalloping the Dacron graft to form new sinuses to remodel the root. The reimplantation method involves reimplanting the scalloped native valve into the Dacron graft. Both require reimplantation of the coronary ostia into the Dacron graft.

Patients with an abnormal aortic valve and aortic root require aortic root replacement (ARR). In nonelderly patients who can undergo anticoagulation with reasonable safety, the aortic root may be replaced with a composite valve-graft consisting of a mechanical valve inserted into a Dacron graft coronary artery reimplantation (eg, classic or modified Bentall procedure, Cabrol procedure).

For elderly patients, young active patients who do not desire anticoagulation, women of childbearing age, and patients with contraindications to warfarin, the options include stentless porcine roots, aortic homografts, and pulmonary autografts (ie, Ross procedure). For elderly patients who cannot undergo a complex operation, another option is reduction aortoplasty (ie, wrapping of the ascending aorta with a prosthetic graft).

Patients with Marfan syndrome have abnormal aortas and cannot undergo tube graft replacement alone. They must have either a valve-sparing aortic root replacement or a complete aortic root replacement.

Aortic root replacement with a homograft is ideal in the setting of aortic root abscess from endocarditis.

Aortic arch aneurysms

Arch aneurysms pose a formidable technical challenge. Deep hypothermic circulatory arrest (DHCA) with or without antegrade or retrograde cerebral perfusion is usually used to

facilitate reanastomosis of the arch vessels. Aortic arch reconstruction techniques vary depending on the arch pathology.

In patients with proximal arch involvement extending from the ascending aorta, a hemiarch replacement may be performed. The ascending aorta is replaced with a Dacron graft beveled as a tongue along the undersurface of the arch. In patients whose conditions mandate replacement of the entire arch, the distal anastomosis is the Dacron graft to the descending thoracic aorta. The head vessels are reimplemented individually or as an island. Grafts have been developed with a trifurcated head-vessel attachment and with a fourth attachment for the cannula. In this case, the head vessels are attached individually to the trifurcated branches.

For patients in whom the arch replacement is part of a staged procedure, preceding the delayed repair of a concomitant descending thoracic aneurysm, an "elephant trunk" is used. That is, the Dacron graft used to reconstruct the transverse arch ends distally in an extended sleeve that is telescoped into the descending thoracic aorta, facilitating later replacement of the descending thoracic/abdominal aneurysm (2-stage procedure).

Descending thoracic aortic aneurysms and thoracoabdominal aneurysms

Descending thoracic aneurysms may be repaired with open surgery or, if appropriate, with endovascular stent grafting techniques. Stent graft repair of descending thoracic aortic aneurysms should be performed if the predicted operative risk is lower than that of an open repair. Patient age, comorbidities, symptoms, life expectancy, aortic diameter, characteristics and extent of the aneurysm, and landing zones, should also be taken into consideration.

Surgically, descending thoracic aneurysms may be repaired with or without the use of a bypass circuit from the left atrium to the femoral artery or femoral vein–femoral artery cardiopulmonary bypass, depending on the length of the anticipated ischemic cross-clamping and the experience of the surgeon. Discrete aneurysms with an anticipated clamp time of less than 30 minutes may be repaired without left heart or cardiopulmonary bypass (ie, "clamp and go" technique). More complex or larger aneurysms are probably safer to repair with the aid of either left heart, partial, or full cardiopulmonary bypass with hypothermic circulatory arrest. The use of left heart or cardiopulmonary bypass is favored to reduce hemodynamic instability and the risk of spinal cord paraplegia.

Descending thoracic aneurysms with the appropriate anatomy may now be repaired by endovascular stent grafts. The GORE TAG is an FDA-approved nitinol-based stent graft designed for descending thoracic aneurysm repair. An appropriate proximal neck of 2 cm prior to the aneurysm is required. Ideally, the proximal landing zone is beyond the left subclavian artery, though, in some circumstances, the stent may be placed proximal to the left subclavian artery. Distally, a sufficient landing zone of 2 cm prior to the celiac artery is required. The aortic inner neck diameters in the proximal and distal landing zones must fall within 23-37 mm. In addition, appropriately sized femoral and iliac arteries (typically >8 mm in diameter) that lack tortuosity and calcium are required for implantation.

The GORE TAG graft has been FDA-approved since March 2005. More recently, the Zenith TX2 endovascular graft (Cook Medical Inc.; Bloomington, Ind) was approved in March 2008, followed by the Talent Thoracic Stent Graft (Medtronic Inc.; Minneapolis, Minn) in June 2008.^{29,30} The Valiant Thoracic Stent Graft (Medtronic Inc.; Minneapolis, Minn) is approved for use outside the United States.

Thoracoabdominal aneurysms, comprising approximately 10% of thoracic aneurysms, may be repaired with the use of a partial bypass of the left atrium to the femoral artery. Crawford type I thoracoabdominal aneurysms involve Dacron graft replacement of the aorta from the left subclavian artery to the visceral and renal arteries as a beveled distal anastomosis, using sequential cross-clamping of the aorta. Crawford type II thoracoabdominal aneurysm repair requires a Dacron graft from the left subclavian to the aortic bifurcation with reattachment of the intercostal arteries, visceral arteries, and renal arteries. Crawford type III or IV thoracoabdominal aneurysm repairs, which begin lower along the thoracic aorta or upper abdominal aorta, may use either the partial bypass of the left atrial artery to the femoral artery or a modified atrio-visceral and/or renal bypass. Prevention of paraplegia is one of the principal concerns in the repair of descending and thoracoabdominal aneurysms.

Under investigational trials, Dr. Timothy Chuter at the University of California at San Francisco Medical Center and Dr. Roy Greenberg at the Cleveland Clinic have treated thoracoabdominal aneurysms using custom-built fenestrated and branched stent grafts. Such devices require precise anatomic tailoring of the grafts to the specific patient's anatomy for placement of the scallops (for visceral flow) or branches (for direct stenting into the visceral

vessels).

Preoperative Details

Ascending aortic aneurysm

Preoperative assessment of coronary artery disease is essential to determine the need for concomitant coronary artery bypass grafting. Transesophageal echocardiography is crucial preoperatively to examine the need for aortic valve replacement. Patients with aortic stenosis or aortic insufficiency in whom the valve leaflets are anatomically abnormal require replacement, whereas patients with aortic insufficiency and normal aortic valve leaflets may be candidates for valve-sparing procedures. Transesophageal echocardiography is valuable for accurate delineation of the aortic root at the sinuses of Valsalva and sinotubular junction.

Aortic arch aneurysm

The major morbidities from aortic arch aneurysm repair are neurologic, cardiac, and pulmonary in nature. All patients require preoperative assessment of cardiac function and evaluation for coronary artery disease. In the operating room, transesophageal echocardiography is used to monitor ventricular function and to assess for atherosclerosis of the aorta.

A major concern in arch surgery is neurologic injury, both transient neurologic dysfunction and permanent neurologic injury. Patients with a higher risk of stroke undergo preoperative noninvasive carotid ultrasound, and those with a history of stroke undergo a brain CT scan. In the operating room, steroids are often given at the onset of the procedure if hypothermic circulatory arrest is anticipated. Evidence suggests that steroids given preoperatively several hours before the operation may have benefit. Some institutions monitor electroencephalogram silence to assess for adequate duration and temperature of cerebral cooling for hypothermic circulatory arrest.

Descending thoracic aneurysms and thoracoabdominal aneurysms

A devastating complication of descending thoracic aneurysm and thoracoabdominal

aneurysm repair is spinal cord injury with paraparesis or paraplegia. Preoperatively, some groups perform spinal arteriograms to attempt to localize the artery of Adamkiewicz for reimplantation during surgery. Neurologic monitoring with somatosensory evoked potentials or motor evoked potentials is used by some to assess spinal cord ischemia and identify critical segmental arteries for spinal cord perfusion. Lastly, preoperative placement of catheters for cerebrospinal fluid drainage is performed to increase spinal cord perfusion pressure during aortic cross-clamping.

Spinal cord injury is less prevalent with endovascular stent grafting than with open repair but exists with both types of surgical treatment.^{24,25,27,28} For endovascular stent grafting, cerebrospinal fluid (CSF) drainage and avoidance of hypotension are the primary mechanisms used to prevent paraplegia. The use of CSF drainage is selective among most centers. For some discrete aneurysms, stent graft coverage may allow for preservation of spinal arteries. Others require coverage of the entire descending thoracic aorta. Indications for use of CSF drains include anticipated endograft coverage of T9-T12, coverage of the long segment of the thoracic aorta, compromised collateral pathways from prior infrarenal AAA repair, and symptomatic spinal ischemia.

Brain protection

Methods used for brain protection during deep hypothermic circulatory arrest (DHCA) include intraoperative EEG monitoring, evoked somatosensory potential monitoring, hypothermia (to temperatures <20°C), packing the patient's head in ice, Trendelenburg positioning (ie, head down), mannitol, CO₂ flooding, thiopental, steroids, and antegrade and retrograde cerebral perfusion.

Intraoperative Details

General monitoring and anesthesia

Venous access is obtained with 2 large-bore peripheral IVs and a central line. Filling pressures and cardiac output monitoring are performed with a pulmonary artery catheter. Continuous blood pressure monitoring is performed with a radial arterial line. Nasopharyngeal and bladder probes monitor systemic temperature. Intraoperative transesophageal

echocardiography is used to assess myocardial and valvular function.

Ascending aortic replacement

Cardiopulmonary bypass is established and the aorta is cross-clamped just below the innominate artery. The heart is arrested with cardioplegia. The aorta is transected at the sinotubular junction and sized for the appropriate Dacron tube graft. The tube graft is sutured to the proximal aorta with running 4-0 Prolene with a strip of felt. The tube graft is measured to length distally and sutured to the distal aorta using running 4-0 Prolene with a strip of felt.

Valve-sparing aortic root replacement

Once the aorta is transected at the sinotubular junction, the valve is inspected for normal anatomy. If sparing is feasible, the appropriate size tube graft is chosen to allow coaptation of the aortic valve leaflets without aortic insufficiency. In the remodeling technique, the tube graft is tailored to form aortic sinuses. The sinuses of Valsalva of the native aorta are removed, and the coronary ostia are mobilized. The neosinuses of the tube graft are sutured to the scalloped aortic valve with running 4-0 Prolene and a strip of felt.

In the reimplantation technique, Tycron sutures are placed along the subannular horizontal plane and passed through the tube graft. The scalloped aortic valve is placed within the tube graft, and the proximal suture line is secured. The scalloped aortic valve is positioned in the graft to achieve valve competence, and the subcoronary suture line along the scalloped valve is performed with running 4-0 Prolene. The valve is examined for competence within the graft. The coronary ostia are reimplanted in the graft. The graft is measured to length distally and sutured to the distal aorta.

In the reimplantation technique, Tycron sutures are placed along the subannular horizontal plane and passed through the tube graft. The scalloped aortic valve is placed within the tube graft, and the proximal suture line is secured. The scalloped aortic valve is positioned in the graft to achieve valve competence, and the subcoronary suture line along the scalloped valve is performed with running 4-0 Prolene. The valve is examined for competence within the graft. The coronary ostia are reimplanted in the graft. The graft is measured to length distally and sutured to the distal aorta.

Aortic root replacement

The aorta is transected, and the aortic valve is removed. The annulus is sized, and the appropriate valved conduit, stentless root, mechanical composite, or homograft is brought to the field. The coronary ostia are mobilized. Annular sutures are placed and are passed through the valve conduit. The proximal suture is thus secured. The coronary ostia are reimplanted. The distal suture line is performed for the mechanical valve composite, but an additional Dacron graft extension may be required for the stentless roots or homografts, depending on their length.

Modified Bentall procedure ("buttons"): The right and left coronary arteries are dissected as a button, which is then reimplanted into the Dacron composite graft as an aortic button.

Cabrol procedure: Rarely performed, this technique may be used when the aortic tear or dissection extends into the coronary ostia. It may also be used when adequate mobilization of the coronary ostia is not possible (i.e., from scarring in a reoperation), or when the coronary ostia are too low. The coronary buttons are dissected and anastomosed to a separate 6- or 8-mm Dacron interposition graft; this graft is then anastomosed into the Dacron composite graft. This technique results in a tension-free anastomosis of the coronary buttons and also allows for easier access for hemostasis. However, it is subject to twisting and kinking with resultant myocardial ischemia and, thus, is not as reproducible as the modified Bentall.

Open distal anastomosis

Deep hypothermic circulatory arrest with or without antegrade or retrograde cerebral perfusion is used. When cooled to 18°C (64.4°F), the pump is turned off and the arterial line is clamped. The patient is placed in the Trendelenburg position, and the aortic cross clamp is removed. The distal anastomosis is performed open with running 4-0 Prolene and a strip of felt. The distal anastomosis may be at the level of the innominate artery or, in the case of hemiarch replacement, along the undersurface of the arch to the level of the left subclavian artery. Once the anastomosis has been completed, the pump is restarted with blood flow antegrade into the new graft and open proximal tube graft to flush out air and debris. The graft is then clamped; the proximal aortic reconstruction is performed during rewarming.

Hypothermia decreases oxygen consumption. For each drop in temperature by 1°C, the oxygen consumption by the tissues is reduced by 10%.

Air (ie, nitrogen) is poorly soluble in blood. The risk of air embolism is reduced by flooding the surgical field with carbon dioxide. Carbon dioxide is denser than air and displaces air. It is rapidly soluble in blood and causes less risk of embolization. Any carbon dioxide absorbed in the blood is removed by increasing the sweep speed of cardiopulmonary bypass.

Aortic arch aneurysm repairs

Cannulation for arch repairs varies among groups. They include the femoral artery, right axillary artery, and ascending aorta. Hypothermic circulatory arrest is required for arch repairs, but the safe period of arrest to avoid neurologic injury is 30-45 minutes at 18°C (64.4°F), but some advocate a shorter period of 25 minutes. Antegrade cerebral perfusion to minimize neurologic injury is thus advocated. Others advocate cooling to 11-14°C (51.8-57.2°F).

Once the patient is cooled to the desired temperature, the circuit is turned off. For retrograde cerebral perfusion, flow is established through the superior vena cava as the arch reconstruction is performed. For antegrade cerebral perfusion, flow is continued through the axillary artery with the innominate artery clamped or individual perfusion catheters are placed into the innominate artery, left carotid artery, and left subclavian arteries. The arch reconstructions are also varied. They basically involve performing the distal anastomosis to the aorta beyond the left subclavian artery as an open distal procedure with or without an elephant trunk. The 3 head vessels may be reanastomosed individually or as an island. They may be reimplanted directly to the graft or anastomosed to a separate graft, which is then attached to arch graft.

Descending thoracic aneurysm and thoracoabdominal aneurysm repairs

Measures to reduce spinal cord injury include cerebrospinal fluid drainage, reimplantation of intercostal arteries, partial bypass, and mild hypothermia. A left thoracotomy or a thoracoabdominal incision is performed. The aorta is cross-clamped either just beyond the left subclavian or between the left carotid and left subclavian for Crawford types I and II. The cross clamp is placed more distally for Crawford types III and IV.

Atrial femoral bypass is established with a Bio-Medicus circuit, and the patient is cooled to 32-34°C (89.6-93.2°F). Distal cross-clamping is performed at T4-T7 to allow continued spinal cord, visceral, and renal perfusion. The proximal anastomosis is performed with running 4-0 Prolene and a strip of felt. When complete, the proximal clamp is released and reapplied more distally on the tube graft. The distal cross clamp is moved sequentially down, if feasible, to allow visceral and renal perfusion. The intercostal arteries may be reimplemented, if desired, or oversewn. If sequential cross-clamping is not feasible, direct catheters may be placed in the visceral and renal vessels to allow continuous perfusion.

If the distal aneurysm extends to the renals, then the distal anastomosis may be beveled to incorporate the visceral and renal vessels and distal aorta. If the distal aneurysm extends to the bifurcation, the visceral and renal vessels are reattached to the tube graft. The left renal artery typically requires a separate anastomosis, but the celiac, superior mesenteric, and right renal arteries are often incorporated as a single island. The patient is rewarmed, and the partial bypass is discontinued as the tube graft perfuses the intercostals and abdominal vessels. The distal anastomosis at the bifurcation is performed as an open distal procedure.

For appropriate descending thoracic aortic aneurysms, endovascular stent grafting is a good alternative. Depending on the size of the patient's femoral or iliac arteries and size of the stent graft required, femoral or iliac artery exposure is performed under general or local anesthesia plus sedation. A sheath is placed and a wire guided under fluoroscopy into the arch. When in proper position, the floppy wire is exchanged with a soft catheter and rewired to a stiffer wire for device placement. The sheath is exchanged for the appropriate device sheath. The contralateral groin is used for angiocatheter placement.

After angiography and determination of stent placement, the device is loaded and, under fluoroscopic guidance, is positioned and deployed. More than one stent may be used, with as much overlap as is feasible, for stability. The proximal and distal landing zones are ballooned to seal the endograft to the aorta. The overlapping stent-graft segments are also ballooned. Angiography is performed to check for endoleaks. Endoleaks may require additional stents.

Ross procedure (pulmonary autograft)

The aortic root and proximal ascending aorta are replaced with a pulmonary autograft.²³ The pulmonary valve is then replaced with a pulmonary homograft. Most commonly performed in children with congenital disease, the Ross operation may be used for active young adults with aneurysmal disease (excluding those with connective tissue disorders), women of childbearing age who desire pregnancy, or patients with contraindications to anticoagulation.

Postoperative Details

Patients who have undergone ascending aneurysm repairs are observed for signs of coronary ischemia, particularly if the coronary ostia were reimplanted, and for signs of aortic insufficiency when the aortic valve is repaired. Following the repair of arch aneurysms, particular attention must be given to neurological status, and patients who have had the elephant trunk repair must be observed for signs of paraplegia because the telescoped sleeve in the descending aorta may obstruct critical spinal vessels.

Paraplegia is the main concern in patients who have had repair of the descending and thoracoabdominal aorta. Cerebrospinal fluid drainage may be continued for up to 72 hours postoperatively if necessary, along with motor evoked potential monitoring. Paraplegia and paraparesis may be acute or delayed postoperatively. If paraparesis or paraplegia is delayed, increased mean arterial pressure with pressors and reinstatement of cerebrospinal fluid drainage may augment spinal cord perfusion to reverse this complication. Paraplegia due to occlusion of critical spinal arteries that were not reimplanted cannot be reversed by these maneuvers. Acute postoperative renal dysfunction may be due to extended periods of ischemic cross-clamping or to hypothermic circulatory arrest.

Patients undergoing endovascular stenting are often extubated early postoperatively with a decreased ICU length of stay.

Complications

Early morbidity and mortality are related to bleeding, neurologic injury (eg, stroke), cardiac failure, and pulmonary failure (eg, acute respiratory distress syndrome [ARDS]). Risk factors include emergent operation, older age, dissection, congestive heart failure (CHF), prolonged cardiopulmonary bypass time, arch replacement, previous cardiac surgery, need for

concomitant coronary revascularization, and reoperation for bleeding. Late mortality is usually related to cardiac disease or distal aortic disease.

Bleeding is a potential complication for all aneurysm repairs. It is minimized by the use of antifibrinolytics, felt strips, and factors, including fresh frozen plasma and platelets. For patients who undergo hypothermic circulatory arrest, the use of aprotinin is controversial, but most groups routinely use aminocaproic acid (Amicar). Coagulopathy and bleeding in severe cases may warrant the use of recombinant factor VII.

Aprotinin (Trasylol), an antifibrinolytic agent used to reduce operative blood loss in patients undergoing open heart surgery, is now only available via a limited-access protocol. Fergusson et al reported an increased risk for death compared with tranexamic acid or aminocaproic acid in high-risk cardiac surgery.³¹

Stroke is a major cause of morbidity and mortality and typically results from embolization of atherosclerotic debris or clot. Transesophageal echocardiography and epiaortic ultrasound may be beneficial in localizing appropriate areas to clamp. Patients undergoing arch repairs are at the highest risk of permanent and transient neurologic injury. Retrograde cerebral perfusion is beneficial for flushing out embolic debris, but it may be detrimental, with increased intracranial pressure and cerebral edema. Antegrade cerebral perfusion is beneficial for reducing neurologic injury during hypothermic circulatory arrest. Stroke incidence for open surgical repair versus endovascular repair of descending thoracic aneurysms is equivalent.

Myocardial infarction may occur with technical problems with coronary ostia implantation during root replacement for ascending aortic aneurysms and may require reoperation. Pulmonary dysfunction and renal dysfunction are other potentially morbid complications.

Paraparesis and paraplegia, either acute or delayed, are the most devastating complications of descending thoracic aneurysm and thoracoabdominal aneurysm repairs. Despite cerebrospinal drainage, reimplantation of intercostal arteries, evoked potential monitoring, mild hypothermia, and atrial femoral bypass, spinal cord injury still occurs. Endovascular stent grafting has not eliminated spinal cord paraplegia; the incidence varies widely, with an overall incidence of 2.7%.^{24,25,27,28}

Complications specific to endovascular stenting include endoleaks, stent fractures, stent graft migration or thrombosis, iliac artery rupture, retrograde dissection, microembolization, aorto-esophageal fistula, and complications at the site of delivery (eg, groin infection, lymphocele, seroma).

TOPIC 20. DISEASES OF THE VEINS. SYNDROME OF SUPERIOR VENA CAVA. SYNDROME OF INFERIOR VENA CAVA. CAUSES, DIAGNOSTIC, DIFFERENTIAL DIAGNOSTIC, TREATMENT TACTIC.

VARICOSE VEINS

Epidemiology of Chronic Peripheral Venous Disease

The term chronic venous disease, or more specifically of interest here, chronic peripheral venous disease (CPVD) has been used more generally to refer to either visible and/or functional abnormalities in the peripheral venous system. The most widely used classification of such abnormalities is the CEAP (Clinical, Etiological, Anatomic, Pathophysiologic), which includes both anatomic (superficial, deep, or perforating veins) and pathophysiologic (reflux, obstruction, both) categories.

The CEAP classification was created by an international committee of clinical experts, and reflects the clinical situation in patients typically referred to a vascular specialist for clinically significant venous disease. In contrast to the clinical situation, population studies of CPVD typically have focused on broader categories determined by visual inspection only. The three major categories of interest have been varicose veins (VV), chronic venous insufficiency (CVI), and venous ulcers. However, there has not been a standard definition of these categories. VV has been defined both including and excluding telangiectasias (spider veins), and at differing levels of visible disease severity. CVI typically has been defined by skin changes and/or edema in the distal leg. Venous ulcers, both active and healed, have been defined by visual inspection and subjective inference as to etiologic origin.

Two studies have now reported results on defined free-living populations with simultaneous assessment of both visible abnormalities and functional impairment by Duplex ultrasound.

The Duplex examination for the San Diego Population Study (SDPS) determined both obstruction and reflux, whereas the Edinburgh study determined only the latter. These studies have raised some questions regarding the validity of the assumptions based on earlier population studies and regarding the utility of the CEAP classification, at least as applied to largely healthy population samples. Specifically, the general concept that visible disease necessarily implied underlying functional disease, and vice versa, was true in the large majority of affected limbs, but not universally so.

Although these discrepancies occurred in a minority of cases, they were frequent enough to lead us to separately classify visible and functional CPVD in each limb evaluated in the SDPS. Specially, we classified each limb into four visible categories: normal, telangiectasias/spider veins (TSV), VV, and trophic changes (TCS), the latter category being one or more of hyperpigmentation, lipodermatosclerosis, or active or healed ulcer. The presence/absence of edema was not by itself a criterion for TCS. For functional disease, we determined the presence of obstruction and reflux separately for the superficial, perforating, and deep systems. The presence of either reflux or obstruction in superficial or deep veins was categorized as functional disease, and because of small numbers, abnormalities of the perforating veins were considered as deep disease. Three functional categories were defined: normal, superficial functional disease (SFD), and deep functional disease (DFD). Here, the term “functional” is essentially interchangeable with “anatomic.” Also, in this population study obstruction was uncommon, and virtually all legs with obstruction also had reflux, such that SFD and DFD essentially refer to reflux.

In addition to separately assessing edema, we asked about a history of superficial venous thrombosis (SVT) and deep venous thrombosis (DVT), with or without pulmonary embolism.

AGE AND CVPD

Using mutually exclusive categories for both visible and functional CVPD, we found a graded relationship with increasing age for VV, with those aged 70–79 years having nearly twice the prevalence of those aged 40–49 years. TSV also increased with age, but this difference was obscured by the mutually exclusive categories with increasing numbers of participants with TSV also having VV or TCS at older ages. TCS showed the most dramatic age-related increase, with the oldest age group having more than four times the prevalence of the youngest.

These findings for visible disease are consistent with most previous population studies, which generally have found a linear increase in TSV or VV with age (reviewed in Reference 4). Earlier studies typically defined CVI only by venous (assumed) ulcers, and reported exponential increases in CVI with age, findings similar to the dramatic age increase we reported for the broader TCS category.

For functional CVPD, SFD was more than twice as common and DFD was 64% more common in the oldest age group. SFD showed both a higher prevalence and a steeper age gradient than did DFD.

The only other population data on functional disease were from the Edinburgh study and were limited to reflux, and showed similar gradients with age.

Edema was strongly age-related as expected, but history of SVT and DVT were somewhat less so, perhaps reflecting selective recall bias in older participants.

Nonetheless, our data for DVT overall are quite similar to the lifetime prevalence in a large population-based study.

GENDER AND CPVD

For visible disease, we found nearly twice as much VV in women as in men, but TCS were 50% more common in men. These findings for VV are consistent with earlier studies, but earlier studies also have suggested a small excess of CVI in women, in contrast to our findings for the broader category of TCS. However, more concordant with our findings, the Edinburgh study reported that CVI was twice as common in men as women. For functional CPVD, only the Edinburgh study has comparable data, and only for reflux, and found a gender ratio for functional disease similar to the SDPS.

Edema was about 50% more common in men than women, consistent with a 50% greater history of DVT in men. The Edinburgh group reported more edema in women, but a discordance with CVI being more common in men.

ETHNICITY AND CPVD

The SDPS reported data for four ethnicities, nonHispanic White, Hispanic, African-American, and Asian. Non-Hispanic Whites showed the highest prevalence of CPVD, with only 14.3% with a normal examination. Non-Hispanic Whites had the highest rates of TSV, TCS, and DFD, and the second highest rates (after Hispanics) of VV and SFD. African-Americans and Asians had a somewhat lower prevalence of CPVD. Consistent with the visible and functional findings, Non-Hispanic Whites also had the highest rates of edema and DVT by history, and Hispanics the highest rate of SVT by history.

Several previous studies have suggested a higher prevalence in developed than developing countries, although these studies are not entirely consistent. The SDPS is the first population study to evaluate multiple ethnic groups who were residents of the same geographical area.

RISK FACTORS FOR CPVD

Age was positively consistently related to all levels of visible and functional disease in both sexes. In comparison with non Hispanic whites (NHW), African-American Asian had less TSV and VV in both sexes, less TCS in men, and less DFD in women. Our results thus confirm that older age and NHW ethnicity are risk factors for CPVD.

Family history of venous disease based on subject recall was a risk factor for all levels of visible and functional disease. Although this finding could be biased, it is consistent with many other studies, although not all.

Ankle motility was a risk factor for visible disease SFD in women and for TSV in men. It was protective for women with DFD and men with SFD. The association of increasing laxity in connective tissue with venous disease corroborated previous research.

The protective associations could reflect increased ankle motility leading to decreased venous pressure by increasing pumping action.

Lower limb injury was a risk factor in women for DFD. Coughlin et al., in a case-control study, found serious lower limb trauma to be a risk factor for CVI.

CVD-related factors, such as angina, PTCA, hypertension, and diastolic pressure were associated with less TSV, SFD, and DFD for men and women and less VV for men. Although some studies have found a relationship between atherosclerosis and venous disease, others have not.

The reason for any protective effect of cardiovascular disease and hypertension on CPVD is not readily apparent, although venous vasoconstriction and microthrombosis could conceivably be involved.

Hours spent walking or standing was positively associated with VV, TCS, and SFD in men and women. Fowkes et al. found that walking was a risk factor for women with venous insufficiency when age-adjusted, but less so when multiply adjusted. They found walking to be related to lessened risk of venous insufficiency in men. Our data indicate that standing was a strong risk factor for venous disease in women. This is concordant with a number of studies, and contrasts with some other studies.

Weight, height, waist, and BMI, defined as weight in kg divided by height squared in meters squared, were positively associated with TCS, and DFD in men and VV, TCS, and SFD in women. Weight, waist circumference, the waist/hip ratio, and body mass index are all measures of adiposity.

A number of studies have found an association of obesity with venous disease. Gourgou

et al. found a relationship in both men and women with VV. Our finding of increased waist circumference in men with TCS was consistent with findings that both obesity and male gender were associated with CVI and with the finding that weight was an independent risk factor for CVI in multivariate analysis. In contrast, Coughlin et al. and Fowkes et al. both found that obesity was not a factor in venous insufficiency among women. Fowkes et al. extended this finding to men as well. Other studies also have found no association between obesity and venous disease.

However, the Edinburgh group also found that for men and women combined, persons with greater severity of varices (i.e., more segments with reflux) had higher body mass indices than those with fewer segments involved. Additionally, Fowkes et al. found that varicosities in the superficial system, but not in the deep system, were related to body mass index in women. Exercise was associated with lower rates of TSV, TCS, and SFD in men. This is concordant with the finding of Gourgou et al. that physical activity is related to less VV.

During exercise the venomuscular pump is activated, which leads to a transient decrease in venous pressure, which should be protective for venous disease. This is consistent with our results in men.

HRT duration or parity was positively associated with all levels of visible and functional disease in women. Gourgou et al. found increasing VV prevalence with increasing numbers of births. Coughlin et al. found that multiparity was associated with varicose veins in pregnant women.

Some studies have found that the changes are effected with only one pregnancy. The increase of CPVD with HRT duration may indicate yet another underexamined systemic effect of HRT.

Our data indicate that age and family history were the strongest risk factors for CPVD, and neither is subject to intervention. Other significant findings on inherent factors included associations with connective tissue laxity and height. CVD-related factors were associated with lower rates of venous disease. Among volitional factors important findings were a relationship of CPVD with central adiposity, positional factors such as hours spent standing or sitting, exercise, and selected hormonal factors in women. In contrast with prior studies, we found no relationship with dietary fiber intake. In women but not men we confirmed the importance of a previous lower limb injury for DFD.

VENOUS ANATOMY, PHYSIOLOGY, AND PATHOPHYSIOLOGY

ANATOMY

The venous system in the lower extremities can be divided, for purposes of understanding, into three systems: the deep system, which parallels the tibia and femur; the superficial venous system, which resides in the superficial tissue compartment between the deep muscular fascia and the skin; and the perforating or connecting veins, which join the superficial to the deep systems. It is because these latter veins penetrate anatomic barriers, they are called perforating veins.

Although the superficial veins are the targets of most therapy, the principal return of blood flow from the lower extremities is through the deep veins. In the calf, these deep veins are paired and named for their accompanying arteries.

Therefore, the anterior tibial, posterior tibial, and peroneal arteries are accompanied by their paired veins, which are interconnected. These crural veins join and form the popliteal vein. Occasionally the popliteal veins as well as more proximal deep veins are also paired like the calf veins.

As the popliteal vein ascends, it becomes the femoral vein. Formerly, this was called the superficial femoral vein, but that term has been abandoned. Near the groin the femoral vein is joined by the deep femoral vein, and the two become the common femoral vein, which ascends to become the external iliac vein proximal to the inguinal ligament.

Ultrasound imaging has shown that the superficial compartment of the lower extremities consists of two compartments, one enclosing all the structures between the muscular fascia and the skin, and the other, within the superficial compartment enclosing the saphenous vein and bounded by the muscular fascia inferiorly and the superficial fascia superiorly, is termed the saphenous compartment (see Figure 1). The importance of this anatomic structure is underscored by its being targeted during percutaneous placement of endovenous catheters and the instillation of tumescent anesthesia.

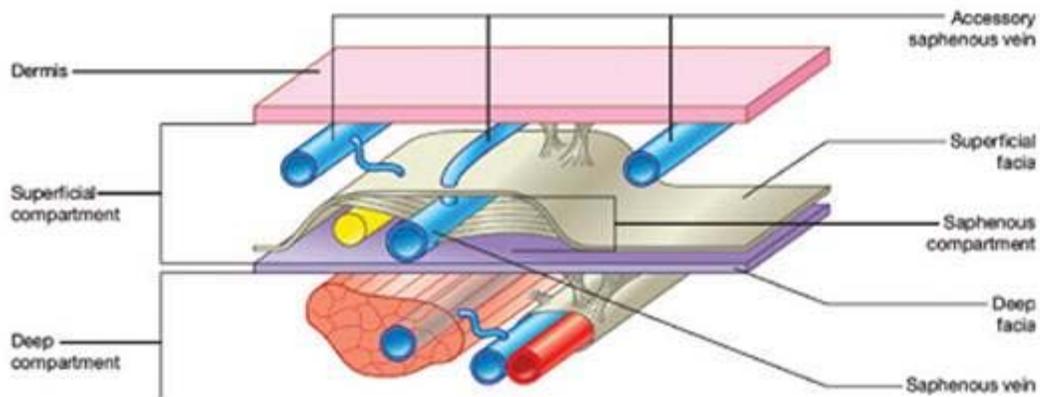


Fig. 1 This diagram of the Saphenous Compartment shows its relationships with the Superficial and Deep compartments as well as the Saphenous Vein (SV) and Nerve and their relationships to the Medial, Anterior, and Lateral Accessory Saphenous Veins (ASV).

The main superficial veins are the great saphenous vein and the small saphenous vein. These receive many interconnecting tributaries, and these tributaries may be referred to as communicating veins. They are correctly called tributaries rather than branches of the main superficial veins. The great saphenous vein has its origin on the dorsum of the foot.

It ascends anterior to the medial malleolus of the ankle and further on the anteromedial aspect of the tibia. At the knee, the great saphenous vein is found in the medial aspect of the popliteal space. It then ascends through the anteromedial thigh to join the common femoral vein, just below the inguinal ligament. Throughout its course, it lies within the saphenous compartment. The small saphenous vein originates laterally from the dorsal venous arch of the foot and travels subcutaneously behind the lateral malleolus at the ankle. As it ascends in the calf, it enters the deep fascia and ascends between the heads of the gastrocnemius muscle to join the popliteal vein behind the knee (see Figure 2). In fact, there are many variations of the small saphenous vein as it connects both to the popliteal vein and to cranial extensions of the saphenous vein, as well as connections to the posteromedial circumflex vein (vein of Giacomini).

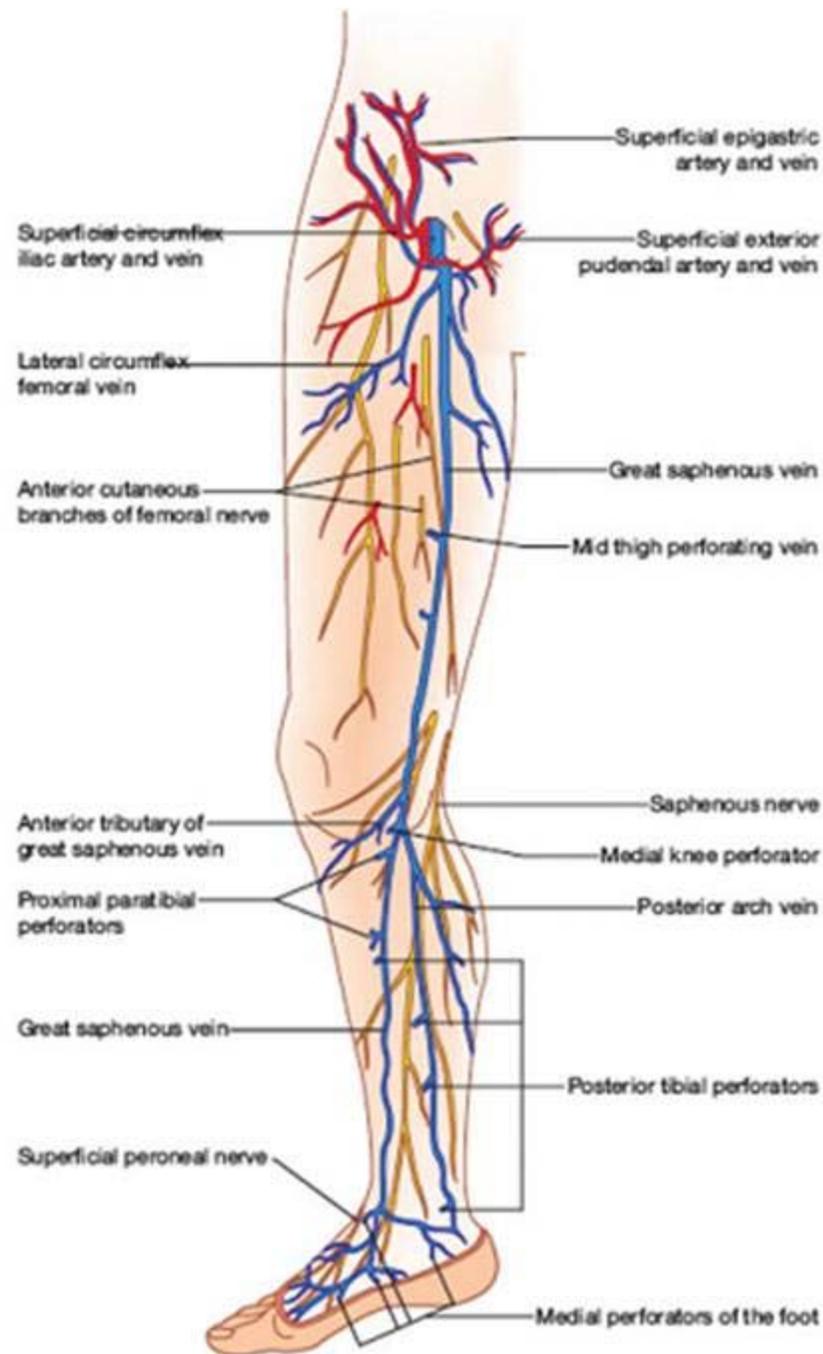


Fig.2. This diagrammatic representation of the Great Saphenous Vein emphasizes its relationship to perforating veins and the Posterior Arch Vein.

The third system of veins is called the perforating vein system. As indicated earlier, they connect the superficial and deep systems of veins. There is a fundamental fact, which confuses understanding of perforating veins. This relates to flow direction. Some perforating veins produce normal flow from the superficial to the deep circulation, others conduct abnormal

outflow from the deep circulation to the superficial circulation. This is termed perforating vein reflux. Any of these perforating veins may demonstrate bidirectional flow (see Table 1).

Table 1. Summary of Important Changes in Nomenclature of Lower Extremity Veins

Old terminology	New terminology
Femoral Vein	Common Femoral Vein
Superficial Femoral Vein	Femoral Vein
Sural Veins	Sural Veins
	Soleal Veins
	Gastrocnemius Veins (Medial and Lateral)
Hungarian Perforator	Mid Thigh Perforator
Cockett's Perforators	Paratibial Perforator
	Posterior Tibial Perforators
May's Perforator	
Gastrocnemius Point	Intergemellar Perforator

In the leg, the principal clinically important perforating veins are on the medial aspect of the ankle and leg, and are found anatomically at approximately 6 cm intervals from the base of the heel through the upper portion of the leg. They are therefore at roughly 6, 12, 18, and 24 cm from the floor (see Figure 3). These medial perforating veins may become targets for treatment of severe chronic venous insufficiency. Smaller perforating veins can be found along intermuscular septa and these allow direct drainage of blood from surface veins into the deep venous system.

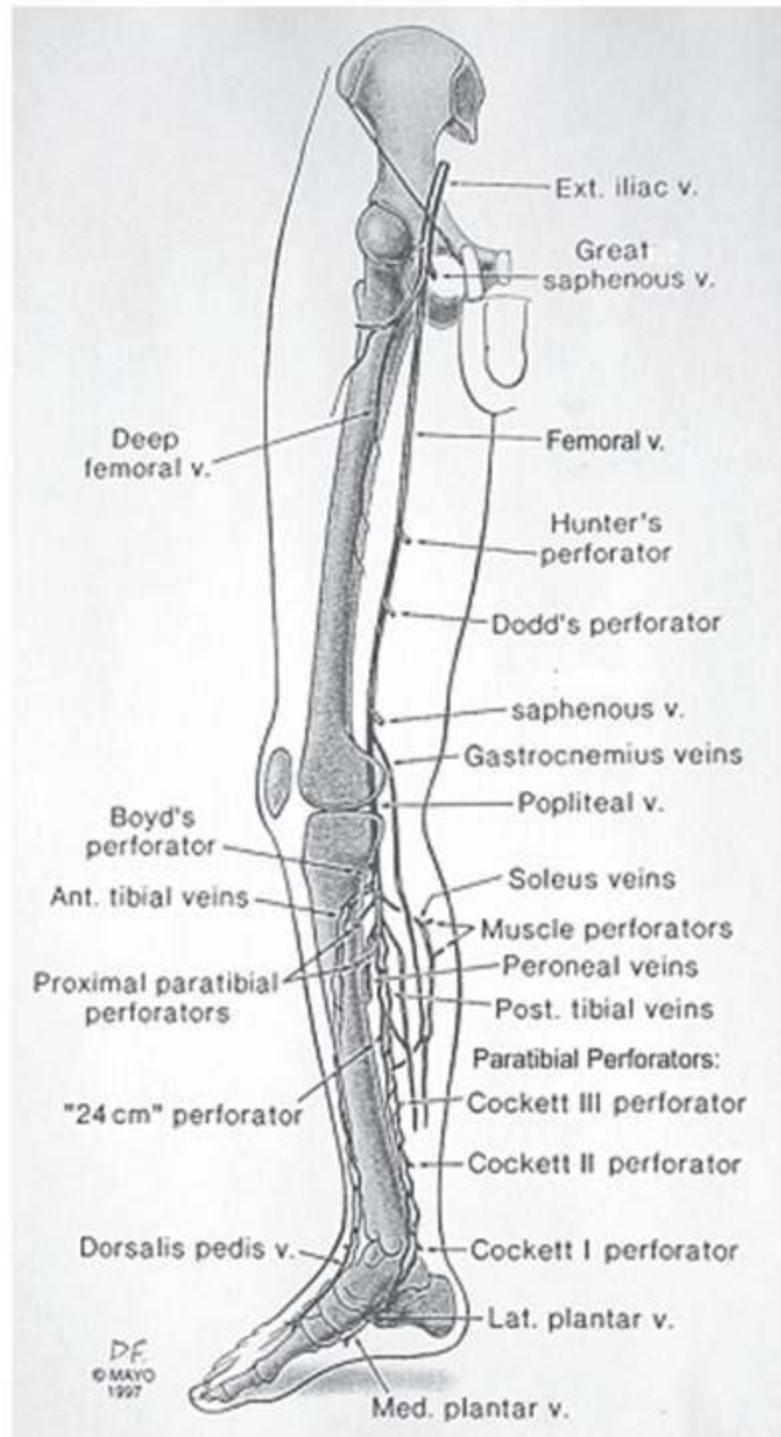


Fig.3. Deep connections of the main thigh and leg perforating veins are shown in this diagram of the deep veins of the lower extremity.

Conversely, when they are dysfunctional, they allow muscular compartment pressure to be transmitted directly to unsupported cutaneous and subcutaneous veins and venules.

VENOUS PHYSIOLOGY

It is estimated that 60 to 75% of the blood in the body is to be found in the veins. Of this total volume, about 80% is contained in the veins that are less than 200 μm in diameter. It is important to understand this reservoir function as it is related to the major components. The splanchnic venous circulation and the veins of the skin are richly supplied by the sympathetic nervous system fibers, but muscular veins have little or none of these. The veins in skeletal muscle, on the other hand, are responsive to catecholamines.

Although arterial pressures are generated by muscular contractions of the heart, pressures in the venous system largely are determined by gravity. In the horizontal position, pressures in the veins of the lower extremity are similar to the pressures in the abdomen, chest, and extended arm. However, with the assumption of the upright position, there are dramatic changes in venous pressure. The only point in which the pressure remains constant is the hydrostatic indifferent point just below the diaphragm. All pressures distal to this point are increased due to the weight of the blood column from the right atrium. When assuming the upright position, there is an accumulation of approximately 500 ml of blood in the lower extremities, largely due to reflux through the valveless vena cava and iliac veins. There is some loss of fluid into the tissues, and this is collected by the lymphatic system and returned to the venous system.

Venous valves play an important role in transporting blood from the lower extremities to the heart. In order for valve closure to occur, there must be a reversal of the normal transvalvular pressure gradient. A pressure and generated velocity flow exceeding 30 cm/second leads to valve closure. Direct observation of human venous valves has been made possible by specialized ultrasound techniques.

Venous flow is not in a steady state but is normally pulsatile, and venous valves undergo regular opening and closing cycles. Even when fully opened, the cross-sectional area between the leaflets is 35% smaller than that of the vein distal to the valve. Flow through the valve separates into a proximally directed jet and vortical flow into the sinus pocket proximal to the valve cusp. The vortical flow prevents stasis and ensures that all surfaces of the valve are exposed to shear stress. Valve closure develops when the vortical flow pressure exceeds the proximally directed jet flow.

The role of venous valves in an individual quietly standing is not well understood. Pressures in the superficial and deep veins are essentially the same during quiet standing, but as Arnoldi has found, the pressure in the deep veins is 1 mm higher, which would tend to keep the valves in the perforating veins closed.

Normally functioning perforating vein valves protect the skin and subcutaneous tissues from the effects of muscular contraction pressure. This muscular contraction pressure may exceed 100 to 130 mmHg.

Intuitively, the role of venous valves during muscular exercise is obvious, since their major purpose is to promote antegrade flow from superficial to deep. Volume and pressure changes in veins within the calf occur with muscular activity. In the resting position, with the foot flat on the floor, there is no flow. However, in the heel strike position, the venous plexus under the heel and plantar surface of the foot (Bejar's plexus) is emptied proximally. Blood flows from the foot and ankle into the deep veins of the calf. Then, calf contraction transports this blood into the deep veins of the thigh, and henceforth, blood flow proceeds to the pelvic veins, vena cava, and ultimately to the heart all due to the influence of lower extremity muscular contraction.

PATHOPHYSIOLOGY

Abnormal functioning of the veins of the lower extremities is recognized clinically as venous dysfunction or, more commonly, venous insufficiency. Cutaneous telangiectases and subcutaneous varicose veins usually are grouped together under the title Primary Venous Insufficiency, and limbs with skin changes of hyperpigmentation, edema, and healed or open venous ulceration are termed Chronic Venous Insufficiency (CVI).

Primary Venous Insufficiency

Explanations of venous pathophysiology as published in reviews, texts, and monographs are now for the most part out of date. The new science as we now know it is incorporated in the following summary.

A dysfunctional venous system follows injury to vein walls and venous valves. This injury is largely due to inflammation, an acquired phenomenon. Factors, which are not acquired, also enter into such injury. These include heredity, obesity, female gender, pregnancy, and a standing occupation in women. Vein wall injury allows the vein to elongate and dilate thus producing the visual manifestations of varicose veins. An increase in vein diameter is one cause of valve dysfunction that results in reflux. The effect of persistent reflux through axial veins is a chronic increase in distal venous pressure. This venous pressure increases as one proceeds from the inguinal ligament past the knee to the ankle.

Prolonged venous hypertension initiates a cascade of pathologic events. These manifest themselves clinically as lower extremity edema, pain, itching, skin discoloration, and ulceration.

The earliest signs of venous insufficiency often are elongated and dilated veins in the epidermis and dermis, called telangiectasias. Slightly deeper and under the skin are flat, blue-green veins of the reticular (network) system. These may become dilated and elongated as well (see Figure 4).

And finally, still deeper but still superficial to the superficial fascia are the varicose veins themselves. All of these abnormal veins and venules have one thing in common: they are elongated, tortuous, and have dysfunctional venous valves.

This implies a common cause, which is inflammation.

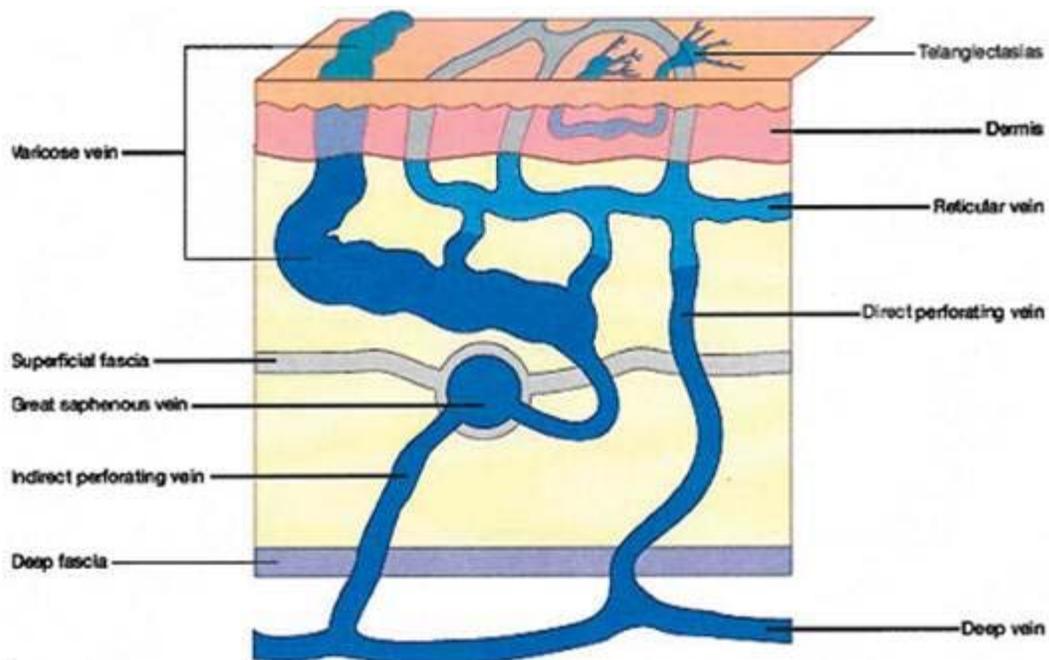


Fig. 4. This cross-sectional view of the subcutaneous venous circulation shows how venous hypertension is transmitted to the unsupported veins of the dermis and subcutaneous tissues from axial veins (GSV) and the deep veins of the muscular compartments.

Chronic Venous Insufficiency

Skin changes of hyperpigmentation, scarring from previous ulceration, and active ulcerations are grouped together under the term chronic venous insufficiency (CVI). Numerous theories have been postulated regarding the cause of chronic venous insufficiency and the cause of venous ulceration.

All the theories proposed in the past century have been disproved. An example is the theory of venous stasis, first proposed in a manuscript by John Homans of Harvard in 1916.

It was a treatise on diagnosis and management of patients with chronic venous insufficiency, and in it, Dr. Homans coined the term “post-phlebotic syndrome” to describe the skin changes of CVI. He stated that, “Over-stretching of the vein walls and destruction of the valves . . . interferes with the nutrition of the skin . . . there-fore, skin which is bathed under pressure with stagnant venous blood will form permanent open sores or ulcers.”

That statement, like many others that describe venous conditions and their treatments, is

steeped in dogma and is short of observational fact. The erroneous term stasis ulcer honors that misconception, as do the terms venous stasis disease and stasis dermatitis.

Alfred Blalock, who later initiated cardiac surgery, disproved the stasis theory by studying oxygen content from varicose veins and normal veins.

He pointed out that the oxygen content of the femoral vein in patients with severe chronic venous insufficiency was greater than the oxygen content of the contralateral nonaffected limb. Because oxygen content was higher, some investigators felt that arteriovenous fistulas caused venous stasis and varicose veins.

That explanation, though disproved, has some basis in fact since the entire thermal regulatory apparatus in limbs depends on the opening and closing of arteriovenous shunts. These shunts are important as they explain some terrible accidents that happen during sclerotherapy when sclerosant entering a vein is shunted into the arterial system and distributed in its normal territory.

Microsphere investigations have failed to show any shunting and the theory of arteriovenous communications has died despite the fact that these shunts actually exist and do open under the influence of venous hypertension.

Hypoxia and its part in causation of chronic venous insufficiency was investigated throughout the last 25 years of the twentieth century. English investigators thought that a fibrin cuff, observed histologically, blocked transport of oxygen and was responsible for skin changes of CVI at the ankles and distally.

That theory has been abandoned even though a true periarteriolar cuff is easily identified histologically.

The two elements that make up all the manifestations of lower extremity venous insufficiency are failure of the vein valves and vein walls and skin changes at the ankles, both of which are related to venous hypertension.

Failure of Vein Walls and Valves

Our work suggests that venous hypertension causes a shear stress dependent leukocyte-endothelial interaction, which has all the manifestations of chronic inflammation.

These are leukocyte rolling, firm adhesion to endothelium, and subsequent migration of the cells through the endothelial barrier into parenchyma of valves and vein walls.

There, macrophages elaborate matrix metalloproteases, which destroy elastin and possibly collagen as well. Vein walls become stretched and elongated. Vein valves become

perforated, torn, and even scarred to the point of near total absence. These changes are seen both macroscopically and angioscopically.

Similar changes have been produced in the experimental animal by constructing an arteriovenous fistula to mimic the venous hypertension of venous dysfunction in humans.

Skin Changes

The second manifestation of chronic venous insufficiency is expressed in the skin where leukocytes also are implicated in the observed changes. There is evidence that leukocyte activation in the skin, perhaps related to venous hypertension, plays a major role in the pathophysiology of CVI. Thomas, working with Dormandy, reported that 25% fewer white cells and platelets left the dependent foot of the patients with venous hypertension. When the foot was elevated there was a significant washout of white cells but not platelets, suggesting platelet consumption within the microcirculation of the dependent foot. They concluded that the decrease in white cell exodus was due to leukocyte trapping in the venous microcirculation secondary to venous hypertension. They further speculated that trapped leukocytes may become activated, resulting in release of toxic metabolites causing damage to the microcirculation and overlying skin. Apparently, the primary injury in the skin is extravasation of macromolecules and red blood cells into the dermal interstitium. Red blood cell degradation products and interstitial protein extravasations are potent chemoattractants and represent the initial chronic inflammatory signal responsible for leukocyte recruitment.

The important observations of Dormandy's group were historically the first to implicate abnormal leukocyte activity in the pathophysiology of CVI.

The importance of leukocytes in the development of dermal skin alterations was further emphasized by Coleridge Smith and his team. They obtained punch biopsies from patients with primary varicose veins, lipodermatosclerosis, and patients with lipodermatosclerosis and healed ulcers. They counted the median number of white blood cells per high power field in each group but there was no attempt to identify the types of leukocytes. In patients with primary varicose veins, lipodermatosclerosis, and healed ulceration there was a median of 6, 45, and 217 WBCs per mm², respectively. This demonstrated a correlation between clinical disease severity and the number of leukocytes in the dermis of patients with CVI.

The types of leukocytes involved in dermal venous stasis skin changes remain controversial. T-lymphocytes, macrophages, and mast cells have been observed on immunohistochemical and electron microscopic examinations.

The variation in types of leukocytes observed may reflect the types of patients investigated. The London group biopsied patients with erythematous and eczematous skin changes, whereas Pappas has evaluated predominantly older patients with dermal fibrosis. Patients with eczematous skin changes may have an autoimmune component to their CVI whereas patients with dermal fibrosis may have experienced pathologic alterations consistent with chronic inflammation and altered tissue remodeling. Skin biopsies have shown that in liposclerotic, eczematous skin macrophages and lymphocytes were predominant in such diseased skin. Infiltration of leukocytes into the extracellular space has been documented by observing the localization of these leukocytes around capillaries and post-capillary venules. Accompanying the leukocytes is a disorganized collagen deposition. Clearly, chronic venous insufficiency of the skin and its subcutaneous tissues is a disease of chronic inflammation, again dependent upon venous hypertension.

SYMPTOMS OF PRIMARY VENOUS INSUFFICIENCY

It is well known that the presence and severity of symptoms do not correlate with the size or severity of the varicose veins present. Symptoms usually attributable to varicose veins include feelings of heaviness, tiredness, aching, burning, throbbing, itching, and cramping in the legs (see Table 2). These symptoms are generally worse with prolonged sitting or standing and are improved with leg elevation or walking. A premenstrual exacerbation of symptoms is also common. Generally, patients find relief with the use of compression in the form of either support hose or an elastic bandage. Weight loss or the commencement of a regular program of lower extremity exercise may also lead to a diminution in the severity of varicose vein symptoms. Clearly, these symptoms are not specific, as they may also be indicative of a variety of rheumatologic or orthopedic problems. However, their relationship to lower extremity movement and compression is usually helpful in establishing a venous origin for the symptoms. Significant symptoms suggestive of venous disease should prompt further evaluation for valvular insufficiency and calf muscle pump dysfunction. If a venous etiology is suspected but all examinations are negative, repeat examination during a symptomatic period is warranted and often fruitful.

The recent development of an extremely painful area on the lower leg at the ankle

associated with an overlying area of erythema and warmth may be indicative of lipodermatosclerosis, which may be associated with insufficiency of an underlying perforator vein, and examination for this lesion should be performed. Lipodermatosclerosis may precede ulceration and has been shown to be improved by stiff compression and certain pharmacologic interventions. Patients with a history of iliofemoral thrombophlebitis who describe “bursting” pain with walking may be suffering from venous claudication. In these patients an evaluation for persistent hemodynamically significant obstruction, possibly treatable with angioplasty and stenting, may be in order.

Table 2. Symptoms of Varicose Veins and
Telangiectasias

Aching Heaviness (on standing, prolonged sitting)
Aching Pain (on standing, prolonged sitting)
Burning (venous neuropathy)
Itching (cutaneous inflammation)
Nocturnal Cramps (recumbent edema reduction)

PHYSICAL EXAMINATION

Using no special equipment, the practitioner can obtain a degree of information regarding overall venous out flow from the leg, the sites of valvular insufficiency, the presence of primary versus secondary varicose veins, and the presence of DVT. The screening physical examination consists of careful observation of the legs. Any patient with the following conditions should be examined more fully: large varicose veins; bulges in the thigh, calf, or the inguinal region representative of incompetent perforating veins (IPVs) or a saphena varix; signs of superficial venous hypertension such as an accumulation of telangiectasias in the ankle region (corona phlebectatica); or any of the findings suggestive of venous dermatitis (pigmentation, induration, eczema). This includes patients with obvious cutaneous signs of venous disease such as venous ulceration, *atrophie blanche*, or lipodermatosclerosis. An obvious but often forgotten point is the necessity of observing the entire leg and not confining the examination simply to the area that the patient feels is abnormal.

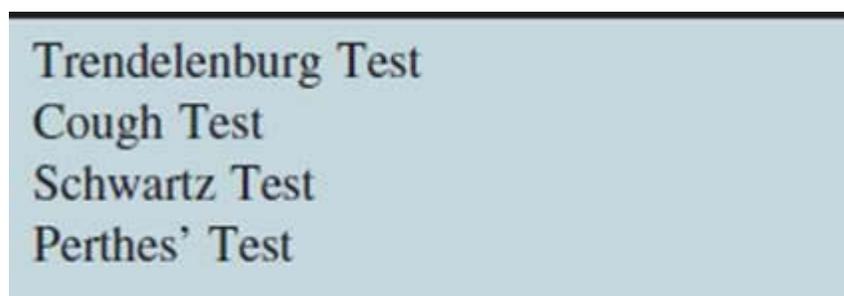
Finally, because the veins of the leg empty into the pelvic and abdominal veins, inspection of the abdomen is very important, since dilation of veins on the abdominal wall or

across the pubic region suggests an old iliofemoral thrombus. Dilated veins along the medial or posterior aspect of the proximal thigh or buttocks most often arise from varicosities involving the pudendal or other pelvic vessels, and these can be of ovarian reflux origin.

CLINICAL TESTING

Historically important tests of venous function have been part of the physical examination of venous insufficiency (see Table 3). These tests have been laid aside largely because of their lack of specificity and sensitivity. The continuouswave Doppler examination has replaced most of these tests, and confirmatory duplex testing has relegated them to an inferior role. However, the educated physician who treats venous insufficiency must have knowledge of these tests and their physiologic background, such as the Trendelenburg test or Brodie-Trendelenburg test.

Table 3. Tests of Historic Interest



Trendelenburg Test
Cough Test
Schwartz Test
Perthes' Test

Trendelenburg Test

A tourniquet may be placed around the patient's proximal thigh while the patient is standing. The patient then assumes the supine position with the affected leg elevated 45 degrees. The tourniquet is removed, and the time required for the leg veins to empty, which is indicative of the adequacy of venous drainage, is recorded. When compared with the contralateral leg, the method just described may demonstrate a degree of venous obstructive disease. Another approach is to elevate the leg while the patient is supine and to observe the height of the heel in relation to the level of the heart that is required for the prominent veins to collapse. Unfortunately, neither procedure is sufficiently sensitive nor accurate and does not differentiate acute from chronic obstruction, thus being of minimal assistance in current

medical practice.

Cough Test

One hand is placed gently over the GSV or SFJ, and the patient is asked to cough or perform a Valsalva maneuver. Simply palpating an impulse over the vein being examined may be indicative of insufficiency of the valve at the SFJ and below to the level of the palpating hand.

Percussion/Schwartz Test

One hand is placed over the SFJ or SPJ, and the other hand is used to tap very lightly on a distal segment of the GSV or SSV. The production of an impulse in this manner implies insufficiency of the valves in the segment between the two hands. Confirmation of the valvular insufficiency can be achieved by tapping proximally while palpating distally. This test can also be used to detect whether an enlarged tributary is in direct connection with the GSV or SSV by palpating over the main trunk and tapping lightly on the dilated tributary, or vice versa. The presence of a direct connection results in a palpable impulse being transmitted from the percussing to the palpating hand. As might be expected, these tests are far from infallible.

Perthes' Test

The Perthes' test has several uses, including distinguishing between venous valvular insufficiency in the deep, perforator, and superficial systems and screening for DVT. To localize the site of valvular disease, the physician places a tourniquet around the proximal thigh with the patient standing. When the patient walks, a decrease in the distension of varicose veins suggests a primary process without underlying deep venous disease because the calf muscle pump effectively removes blood from the leg and empties the varicose veins. Secondary varicose veins do not change caliber (if there is patency of the deep venous system) because of the inability to empty blood out of the veins as a result of impairment of the calf muscle pump. In the setting of a current DVT, they may increase in size. If there is significant chronic or acute obstructive disease in the iliofemoral segment, the patient may note pain (venous claudication) as a result of the obstruction to outflow through both the deep and superficial systems. The Perthes' test is now of more historical than actual clinical importance.

CLASSIFYING VENOUS DISEASE

The Swedish physician and scientist Carl von Linné published a classification of plants based on the number of stamina and pistils in 1735 in *Systema Naturae*. Today, classification of diseases is a basic instrument for uniform diagnosis and meaningful communication about the disease. In chronic venous disorders (CVD) reliance for too long has been placed on the clinical appearance of the superficial effects of CVD, such as spider veins, varicose veins, swelling, skin changes, and ulcerations, without requiring accurate objective testing of the venous system to substantiate the diagnosis. This practice has caused errors of diagnosis and has been largely responsible for the poor correlation of results between treatment methods. There have been several classifications in the past that have added to our understanding of CVD, but all lack the completeness and objectivity needed for scientific accuracy.

Clinical Classification

C0: No visible or palpable signs of venous disease

C1: Telangiectasias or reticular veins

C2: Varicose veins

C3: Edema

C4a: Pigmentation and/or eczema

C4b: Lipodermatosclerosis and/or atrophie blanche

C5: Healed venous ulcer

C6: Active venous ulcer

S: Symptoms including ache, pain, tightness, skin irritation, heaviness, muscle cramps, as well as other complaints attributable to venous dysfunction

A: Asymptomatic

Etiologic Classification

Ec: Congenital

Ep: Primary

Es: Secondary (postthrombotic)

En: No venous etiology identified

Anatomic Classification

As: Superficial veins

Ap: Perforator veins

Ad: Deep veins

An: No venous location identified

Pathophysiologic Classification

Basic CEAP:

Pr: Reflux

Po: Obstruction

Pr,o: Reflux and obstruction

Pn: No venous pathophysiology identifiable

Same as basic, with the addition that any of 18 named venous segments can be utilized as locators for venous pathology:

Superficial veins:

1. Telangiectasias/reticular veins
2. Great saphenous vein (GSV) above knee
3. GSV below knee
4. Small saphenous vein
5. Nonsaphenous veins

Deep veins:

6. Inferior vena cava
7. Common iliac vein
8. Internal iliac vein
9. External iliac vein
10. Pelvic: gonadal, broad ligament veins, other
11. Common femoral vein
12. Deep femoral vein
13. Femoral vein
14. Popliteal vein
15. Crural: anterior tibial, posterior tibial, peroneal veins (all paired)
16. Muscular: gastrocnemial, soleal veins, other

Perforating veins:

17. Thigh

18. Calf

TREATMENT OF VENOUS INSUFFICIENCY

The term venous insufficiency implies that normal functioning is deranged. Terms used to describe the various manifestations of venous insufficiency lend confusion to the general topic. Some of these terms, such as telangiectasias, thread veins, and spider veins are descriptive but imply different conditions. And it is in the chronic disorders, dominated by venous reflux through failed check valves causing hyperpigmentation, ulceration, and corona phlebectatica, where disorientation reigns. Some order can come from subscribing to a unifying theory of primary venous insufficiency and of a common theory of effects of an inflammatory cascade that clarify both situations.

The manifestations of simple primary venous insufficiency appear to be different from one another. However, reticular varicosities, telangiectasias, and major varicose veins are all elongated, dilated, and are tortuous. Investigations into valve damage and venous wall abnormalities eventually may lead to an understanding of the problem, and therefore, a solution by surgery or pharmacotherapy.

Scanning electron microscopy has shown varying degrees of thinning of the varicose venous wall. These areas of thinning coincide with areas of varicose dilation and replacement of smooth muscle by collagen, which is also a characteristic of varicose veins. Our approach to this has been to assume that both the venous valve and the venous wall are affected by the elements that cause varicose veins. We and others have observed that in limbs with varicose veins, an absence of the subterminal valve at the saphenofemoral junction is common. Further, perforation, splitting, and atrophy of saphenous venous valves have been seen both by angioscopy and by direct examination of surgical specimens.

Supporting the theory of weakness of the venous wall leading to valvular insufficiency is the observation that there is an increase in the vein wall space between the valve leaflets. This is the first and most commonly observed abnormality associated with valve reflux. Realizing these facts, our investigations have led us to explore the possible role of leukocyte infiltration

of venous valves and the venous wall as part of the cause of varicose veins. In our investigations of surgical specimens, leukocytes in great number have been observed in the venous valves, and wall and monoclonal antibody staining has revealed their precise identification as monocytes. Similar findings are present in the skin of patients with venous insufficiency.

SURGICAL TREATMENT

Removal of the Great Saphenous vein (GSV) from the circulation is one of two essential steps in treating lower limb varicose veins. Incompetent valves along the GSV allow blood to reflux down the vein and into its tributaries, transmitting high pressure into smaller tributaries, which become varicose as a result. Much emphasis has been placed on the correct technique of high sapheno-femoral ligation, in which meticulous attention is paid to identifying, ligating, and dividing all the tributaries of the GSV as they join the vein in the groin. It has always been a matter of surgical dogma that overlooking any of these allows continued reflux into the residual tributary and subsequent development of recurrent varicose veins.

A number of studies have confirmed that patients in whom the GSV is stripped tend to have fewer than those undergoing simple high ligation of the Sapheno-femoral junction (SFJ). Sarin et al. studied 89 limbs in 69 patients with LSV incompetence.

Legs were randomized to SFJ ligation with or without stripping, and evaluated by photoplethysmography (PPG), duplex scanning, clinical examination, and patient satisfaction. The follow-up period was 18 months. Significant differences in favor of the stripped group were found in all four parameters at final evaluation.

A similar study of 78 patients (110 limbs) was reported by Dwerryhouse et al. in 1999, with a longer follow-up period of five years. This demonstrated a significantly lower reoperation rate among patients undergoing GSV stripping (6%), as opposed to 20% in those undergoing high SFJ ligation alone.

Duplex scanning showed a much lower incidence of residual reflux in the remaining GSV when the proximal vein had been stripped to the knee than when it had not. However, the patient satisfaction rate was not significantly different between the two groups. Ninety percent of the stripped groups were satisfied as opposed to 87% in the nonstripped group ($p = ns$). A further study from Jones et al. came to similar conclusions.

One hundred patients (133 limbs) were randomized as before. After two years, 43% of those who had not had GSV stripping demonstrated recurrent varicose veins as opposed to

25% who had. There was a statistically significant difference.

NEOVASCULARIZATION

Of great importance was the fact that duplex scanning showed that neovascularization in the groin was the commonest cause of varicose recurrence. It was often seen in the ligation group that reflux through the neovascularization entered the residual saphenous vein and perpetuated the old varices while new ones developed. The authors concluded that by stripping the GSV, one was removing the run-off into which the new vessels could drain. Again, however, the satisfaction was broadly similar between the two groups: 91% in the stripped group and 87% in the unstripped.

All these authors concluded that stripping the long GSV gave better long-term results than simple high saphenous ligation. This appears to be true in terms of objective assessment of recurrence rates and in objective measurement of post-operative venous function but is not generally reflected in patient satisfaction rates, which tend to be similar whichever procedure is performed. This led Woodyer and Dormandy to reach a contrary conclusion—that stripping the LSV was a procedure based on surgical dogma, and one that did not confer subjective benefit to the patients so treated. This leads one to conclude that a better method of evaluation of treatment results should be developed.

NONSURGICAL TREATMENT

In recent years, endovenous ablation has been found to be safe and effective in eliminating the proximal portion of the GSV from the venous circulation, with even faster recovery and better cosmetic results than stripping. The two currently available methods used to achieve ablation of the GSV are the Closure© procedure using a radiofrequency (RF) catheter and generator (VNUS Medical Technologies, Inc., Sunnyvale, California), and the endovenous laser ablation (EVLT) procedure using a laser fiber and generator (various manufacturers). Both systems use electromagnetic energy to destroy the GSV in situ. One of the difficulties in evaluating reports of successful ablation of the Great Saphenous vein lies in the definition of success. Some, especially in the RF ablation reports, define success as “no reflux in any segment longer than 5 cm.” Some laser reports refer to success as “stable occlusion” or “reduction in reflux,” and Min has applied the much clearer standard of success as “no flow by color flow Doppler.” Those who report results have not used the life table method, which

takes into consideration drop outs and early and mid-term failures. Thus the reported favorable four and five year rates of elimination of reflux may be exaggerated.

The major difficulty with defining success as reduction or absence of reflux is that attempts to establish whether reflux is present in a portion of a previously closed GSV may be inaccurate. Also, most recurrent patency is seen in the proximal portion of the treated GSV. Therefore, distal compression of the closed portion of the GSV to identify reflux in a proximal segment is futile. Likewise, using the Valsalva maneuver is unreliable and lacks reproducibility. Finally, the importance of distinguishing a partially patent channel with flow, from one with reflux, is academic, since the valves are just as thoroughly destroyed as the rest of the vein wall.

CHEMICAL VENOUS CLOSURE

Some phlebologists have advocated liquid sclerotherapy of the saphenous vein, but the results of such treatment have been disappointing, and published long-term results are absent. Comparisons between liquid and foam sclerotherapy have been done and the results strongly favor foam.

Ultrasound-guided sclerotherapy (USGS) with foam must be considered as a completely new treatment of varicose veins. Although it needs proper training and some skill, it is simple, affordable, and extremely efficient.

Sclerosing agents produce a lesion of the venous wall, predominantly of the endothelium and, to a minor extent, of the media. The reaction that follows depends on the concentration of the agent and on the duration of the contact. If the venous diameter is greater than 3 mm, injections of liquid do not achieve this aim and dilution with blood quickly decreases their efficacy at short distances from the point of injection. Injections of foam have the advantage of a total filling of the vein, at least under 12 mm diameter. A further reduction in venous diameter can be obtained by leg elevation, compression with the hand, duplex probe or bandage, and venous spasm. In very large veins, foam will float over blood and induce a lesion of the upper venous wall, despite apparent correct filling of the vein observed on duplex, thus the importance of massaging and compression.

Making the foam is easy and quick. Based on the technique initially described by Tessari, it can be prepared with two 5 cc syringes and a three-way stopcock. Only detergent sclerosing agents can be used: Sotradecol and Polidocanol at any desired concentration from 0.25% to 3%. Microbubbles of foam sclerosing agents are hyperechogenic and represent an excellent contrast medium for ultrasound techniques. They appear as a shadow within the

lumen early, and like a hyperechogenic mass later with a acoustic shadow. Massaging the sclerosing agent to the desired part of the varicose network with the duplex probe or the hand is also very easily carried out. Progression from the varicose clusters to the GSV and then to the SFJ is always visible, provided a sufficient volume has been injected. Venous spasm usually is observed within minutes. The importance of the initial spasm has been emphasized in several studies and protocols.

Post-sclerotherapy compression is mandatory: on the varicose clusters for 48 hours, and then whole limb compression with 20–30 mmHg thigh-high medical elastic stockings. They must be worn during the daytime for at least 15 days. Patients must be examined both clinically and with duplex at 7 to 15 days.

The absolute risk of deep venous thrombosis is not confirmed. A few cases have been reported: most of them are gastrocnemius vein thrombosis, typically after telangiectasia and reticular vein sclerotherapy. Most frequent complications are visual disorders. These adverse reactions have been observed also with liquid sclerosing agents but their incidence is much higher with foam; they can be estimated at 0.5–1 per 100 foam sessions.

They are observed more frequently in patients suffering from migraine with visual aura. They usually reproduce this aura. The patho-physiology of this phenomenon has been questioned but has received no answer so far. The existence of a patent foramen ovale is the most likely explanation, as has been the liberation of toxic component associated with endothelial cell destruction (endothelin).

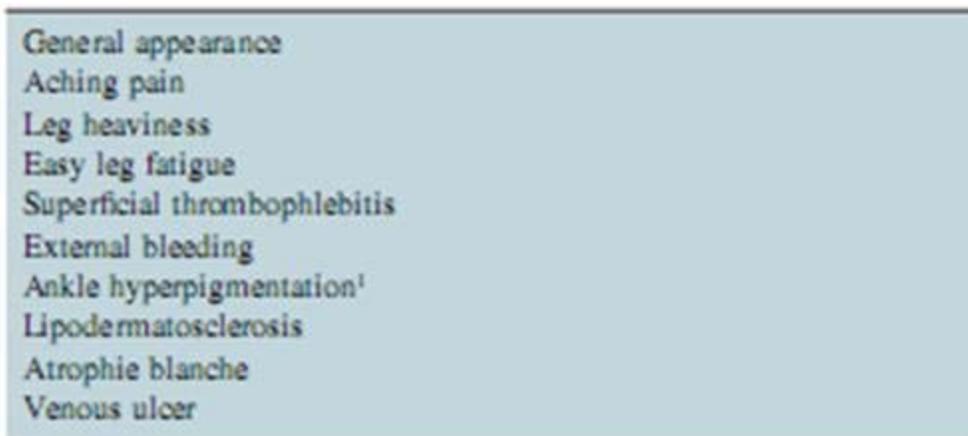
All published results demonstrate an immediate efficacy better than 80% in terms of immediate/primary venous occlusion. Repetition of injections in case of initial failure allows closure to approach 95% of efficacy with two to three sessions. Early and mid-term results demonstrate a recurrence rate of about 20%. The re-do injections remain as simple as primary injections and at least as efficient.

INVERSION STRIPPING OF THE SAPHENOUS VEIN

One of the cornerstones of surgery for varicose veins is removal of the Great Saphenous vein (GSV) from the circulation. This can be done using minimally invasive techniques described elsewhere in this volume, but specific indications for performing saphenous surgery remain. These are largely institutional and geographic but they justify the following exposition. Indications for intervention in primary venous insufficiency are listed in Table 4. Often, it is the appearance of telangiectatic blemishes or protuberant varicosities that stimulates

consultation. Ultimately, this may be the only indication for intervention.

Table 4. Varicose Veins: Indications for Intervention



General appearance
Aching pain
Leg heaviness
Easy leg fatigue
Superficial thrombophlebitis
External bleeding
Ankle hyperpigmentation ¹
Lipodermatosclerosis
Atrophie blanche
Venous ulcer

Characteristic symptoms include aching, pain, easy leg fatigue, and leg heaviness, all relieved by leg elevation, and worsened on the first day of a menstrual period. Other indications for intervention for venous varicosities include superficial thrombophlebitis in varicose clusters, external bleeding from high-pressure venous blebs, or advanced changes of chronic venous insufficiency such as severe ankle hyperpigmentation, subcutaneous lipodermatosclerosis, atrophie blanche, or frank ulceration. Symptoms are frequent throughout the CEAP Classes 1 through 6. Clinical Disability Scores parallel the clinical classification.

Objectives of treatment should be ablation of the hydrostatic forces of axial reflux and removal of the effects of hydrodynamic forces of perforator vein reflux. The latter can be accomplished by removal of the saphenous vein in the thigh and the varicose veins without specific perforating vein interruption. In France, the two most performed procedures recently were, respectively, high ligation + saphenous trunk stripping + tributary stab avulsion (71.9%) and high ligation + saphenous trunk stripping (17.3%). Isolated phlebectomy was done in 5.6%, high ligation + tributary stab avulsion + saphenous trunk preservation 2.8%, isolated high ligation 2.2%.

Ligation of the saphenous vein at the saphenofemoral junction has been practiced widely in the belief that this would control gravitational reflux while preserving the vein for subsequent arterial bypass. It is true that the saphenous vein is largely preserved after proximal ligation. Unfortunately, reflux continues and hydrodynamic forces are not controlled. Less reflux persists when the long saphenous vein has been stripped. There is a better functional outcome after stripping and fewer junctional recurrences. Randomized trials show efficacy of

stripping compared to simple proximal ligation.

Earlier comparisons of saphenous ligation versus stripping were flawed by today's standards. Subjective evaluation was the only means of measuring outcome for a time.

Duplex scanning came into use, verifying that stripping was superior to proximal ligation; this fact was supported by PPG. Despite those facts, it was acknowledged that the period of disability after stripping was greater than that after simple ligation. In attempts to decrease disability and improve efficacy, high tie was added to saphenous vein sclerotherapy, but foot volumetry showed that radical surgery, including stripping produced superior results.

Ultimately, attention became focused on saphenous nerve injury associated with ankle to groin stripping. It was concluded that nerve injury was reduced by groin to ankle stripping (see Figure 5). Preservation of calf veins by stripping to the knee was shown to reduce nerve injury and did not adversely affect early venous hemodynamic improvement.

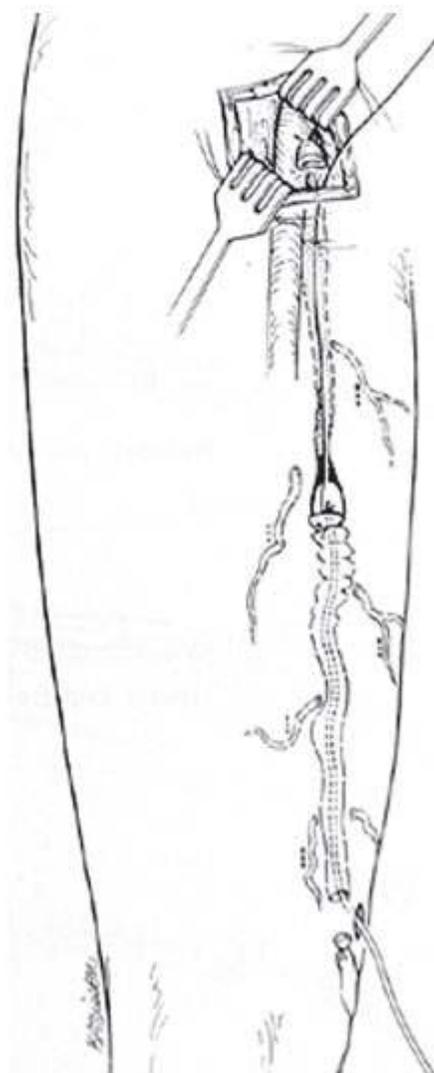


Fig. 5.

This fact is contrainuitive, and the subject deserves further study. Attempts to reduce nerve injury and simultaneously clean up varicose vein surgery led to use of the hemostatic tourniquet. In a study with level 1 evidence, it was shown that use of a hemostatic cuff tourniquet during varicose vein surgery reduces perioperative blood loss, operative time, and postoperative bruising without any obvious drawbacks.

Recurrent varicose veins after surgery are acknowledged to be a major problem for patients and society. Traditionally, it was thought that the most common reason for varicose recurrence was failure to perform an adequate saphenofemoral junction dissection (see Figure 6), or to correctly identify the saphenous vein for removal.

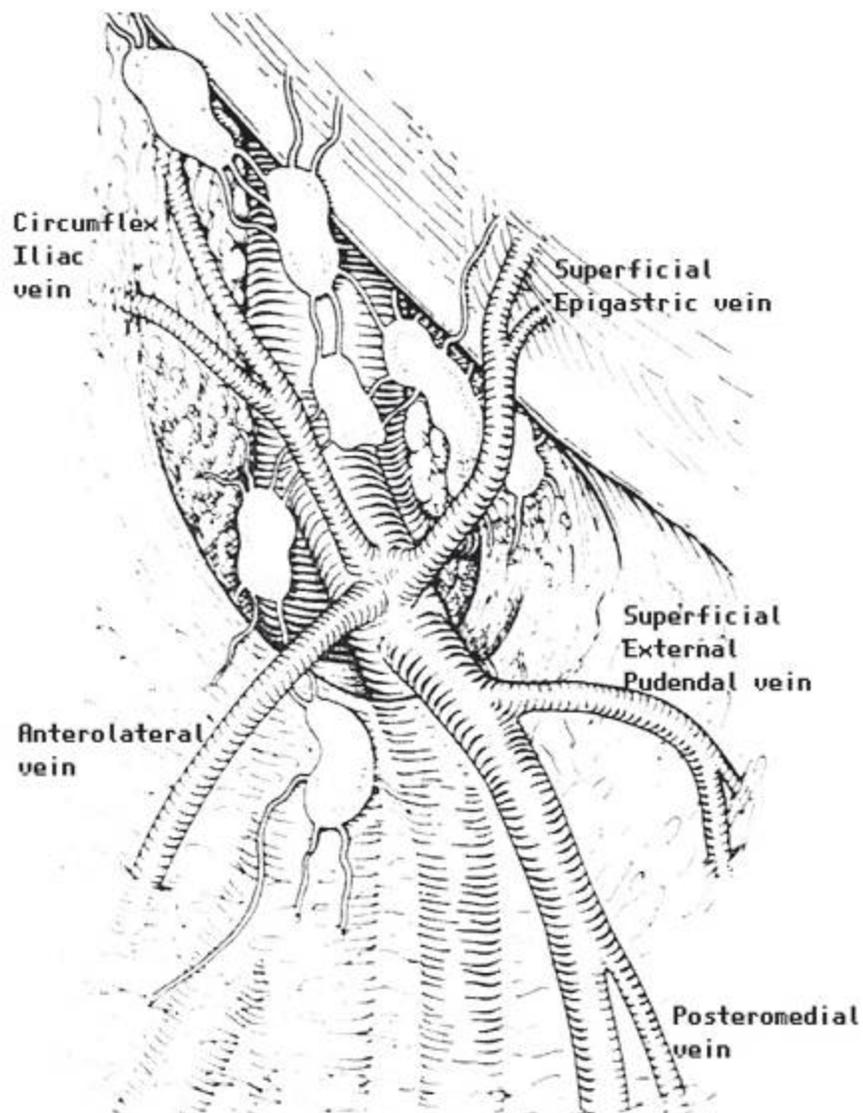


Fig.6. In the past, a proper groin dissection consisted of laying out each of the named saphenofemoral junction tributaries and dissecting them back beyond their primary tributaries. Now, this is acknowledged by most to be the strongest stimulus to neovascularization.

Duplex scans have clarified this situation and instead of technical error, some investigators are convinced that new vessel growth contributes to recurrent varicose veins. In particular, incomplete superficial surgery, at the saphenofemoral and saphenopopliteal junctions, is a less frequent cause of recurrent disease, and neovascular reconnection and persistent abnormal venous function are the major contributors to disease recurrence.

PREOPERATIVE PREPARATION

Over the years, much space has been given to clinical examination of the patient with varicose veins. Many clinical tests have been described. Most carry the names of now-dead surgeons who were interested in venous pathophysiology. This august history notwithstanding, the Trendelenburg test, the Schwartz test, the Perthes test, and the Mahorner and Ochsner modifications of the Trendelenburg test essentially are useless in preoperative evaluation of patients today.

The clinical evaluation can be improved by using hand-held Doppler devices. However, preoperative evaluation is best performed by means of duplex scanning and a focused physical examination. Our protocol for duplex mapping of incompetent superficial veins has been published. Although many cite cost considerations as a reason for omitting duplex evaluation, we believe that duplex scanning for venous insufficiency is in fact both simple and cost effective. Duplex mapping defines individual patient anatomy with considerable precision and provides valuable information that supplements the physician's clinical impression.

Three principal goals must be kept in mind in planning treatment of varicose veins: 1) the varicosities must be permanently removed and the underlying cause of venous hypertension treated; 2) the repair must be done in as cosmetic a fashion as possible; 3) complications must be minimized.

Current practice of treating the source of venous hypertension, the saphenous vein alone either by EVLT or VNUS technology, is inadequate. The patient's complaint, the varicose veins, must be addressed. This is as important as the physician's knowledge that the sources of venous hypertension must be addressed.

To speak of permanent removal of varicosities implies that all potential causes of recurrence have been considered and that surgery has been planned so as to address them. There are four principal causes of recurrence of varicose veins, of which three can be dealt with at the time of the primary operation.

One cause of recurrent varicosities is failure to perform the primary operation in a correct fashion. Common errors include missing a duplicated saphenous vein and mistaking an anterolateral or accessory saphenous vein for the greater saphenous vein. Such errors can be eliminated by careful and thorough groin dissection. Accordingly, failure to do a proper groin dissection has long been held to be a second principal cause of recurrent varicose veins. It is now known, however, that such dissection causes neovascularization in the groin, leading to recurrence of varicose veins. A third cause of recurrent varicosities is failure to remove the greater saphenous vein from the circulation. As mentioned earlier, reasons often cited for this failure is the desire to preserve the saphenous vein for subsequent use as an arterial bypass. It is clear, however, that the preserved saphenous vein continues to reflux and continues to elongate and dilate its tributaries. This produces more and larger varicosities. A fourth cause of recurrent varicosities is persistence of venous hypertension through nonsaphenous sources—chiefly, perforating veins with incompetent valves. Muscular contraction generates enormous pressures that are directed against valves in perforating veins. Venous hypertension induces a leukocyte endothelial reaction, which, in turn, incites an inflammatory response that ultimately destroys the venous valves and weakens the venous wall. The perforating veins most commonly associated with recurrent varicosities are the midthigh perforating vein, the distal thigh perforating vein, the proximal anteromedial calf perforating vein, and the lateral thigh perforating vein, which connects the profunda femoris vein to surface varicosities.

Finally, there is a fifth cause of recurrent varicosities, which is out of control of the operating surgeon—namely, the genetic tendency to form varicosities through development of localized or generalized vein wall weakness, localized blowouts of venous walls, or stretched, elongated, and floppy venous valves.

SAPHENOUS SURGERY

For varicose vein surgery to be successful, two tasks must be accomplished. The first is ablation of reflux from the deep to the superficial veins, including the saphenofemoral junction, the saphenopopliteal junction, and midthigh varices from the Hunterian perforating vein. Accomplishment of this task is guided by the careful preoperative duplex mapping of major

superficial venous reflux.

The second task is removal or destruction of all varicosities present at the time of the surgical intervention. Accomplishment of this task is guided by meticulous marking of all varicose vein clusters. A number of options are available for surgical treatment of varicose veins. Regardless of the specific approach taken, the general technical objectives are the same: 1) ablation of the hydrostatic forces of axial saphenous vein reflux (see Figure 7) and 2) removal of the hydrodynamic forces of perforator vein outflow.

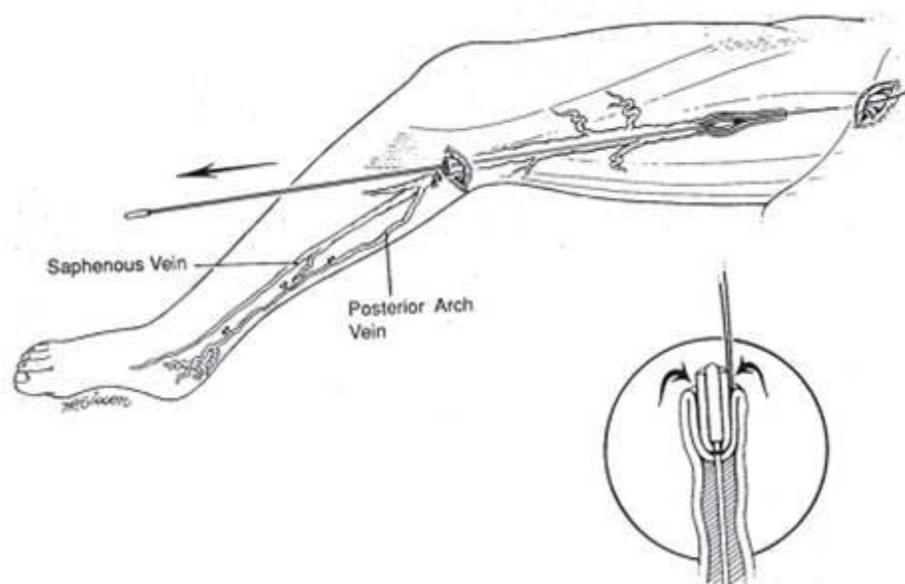


Fig. 7. Inversion stripping of the saphenous vein was an important step forward in minimizing soft tissue trauma while accomplishing the principal objective of ablating hydrostatic venous hypertension by removing saphenous reflux. Tearing of the vein during its removal flawed its performance.

Ankle-to-groin stripping of the saphenous vein has been a dominant treatment of varicose veins over the past 100 years. One argument against routine stripping of the leg (i.e., ankle-to-knee) portion of the saphenous vein is the risk of concomitant saphenous nerve injury. Another argument is that whereas the objective of saphenous vein removal is detachment of perforating veins emanating from the saphenous vein, which are seen in the thigh, the perforating veins in the leg are actually part of the posterior arch vein system rather than the saphenous vein system. This latter argument notwithstanding, preoperative ultrasonography frequently shows that the leg portion of the saphenous vein is in fact directly connected to

perforating veins. Therefore, removal of the saphenous vein from ankle to knee should be a consideration in every surgical case.

OPERATIVE TECHNIQUE

The surgical approach taken must be individually tailored to each patient and each limb. Groin-to-knee stripping of the saphenous vein should be considered in every patient requiring surgical intervention. In nearly all patients, this measure is supplemented by removal of the varicose vein clusters via stab avulsion or some form of sclerotherapy.

Preoperative marking, if correctly performed, will have documented the extent of varicose vein clusters and identified the clinical points where control of varices is required. Incisions can then be planned. As a rule, incisions in the groin and at the ankle should be transverse and should be placed within skin lines. In the groin, an oblique variation of the transverse incision may be appropriate. This incision should be placed high enough to permit identification of the saphenofemoral junction.

Generally, throughout the leg and the thigh, the best cosmetic results are obtained with vertical incisions. Transverse incisions are used only in the region of the knee, and oblique incisions are appropriate over the patella when the incisions are placed in skin lines.

A major cause of discomfort and occasional permanent skin pigmentation is subcutaneous extravasation of blood during and after saphenous vein stripping. Such extravasation can be minimized by applying a hemostatic tourniquet after Esmarch exsanguination of the limb. The pressure in the hemostatic tourniquet should be between 250 and 300 mm Hg, and the tourniquet should not be in place for longer than one hour. If a tourniquet is not used, the entire operation on one limb can be performed with the limb elevated 30° so that the major varicose clusters are higher than the heart. In addition, hemostatic packing can be placed into the saphenous vein tunnel.

The practice of identifying and carefully dividing each of the tributaries to the saphenofemoral junction has been dominant over the past 50 years. The rationale for this practice has been that it would be inadvisable to leave behind a network of interanastomosing inguinal tributaries. Accordingly, special efforts have been made to draw each of the saphenous tributaries into the groin incision so that when they are placed on traction, their primary and even secondary tributaries can be controlled. The importance of these efforts has been underscored by descriptions of residual inguinal networks as an important cause of varicose vein recurrence. Currently, however, this central practice of varicose vein surgery is under

challenge, on the grounds that groin dissection can lead to neovascularization and hence to recurrence of varicosities.

Preoperative duplex studies have already demonstrated incompetent valves in the saphenous system, and a disposable plastic stripper can be introduced from above downward; alternatively, a metal stripper can be employed. Both of these devices can be used to strip the saphenous vein from groin to knee via the inversion technique. This approach should reduce soft tissue trauma in the thigh.

In the groin, the stripper is inserted proximally into the upper end of the divided internal saphenous vein and passed down the main channel through incompetent valves until it can be felt lying distally approximately 1 cm medial to the medial border of the tibia at a point approximately 4 to 6 cm distal to the level of the tibial tubercle. The saphenous vein is anatomically constant in this location, just as it is in the groin and ankle. If the saphenous vein is removed from the groin to this level, both the midthigh perforating vein, which usually enters the saphenous vein, and the most distal incompetent perforating veins, which are in the distal third of the thigh, will be treated. A small incision is made over the palpable distal end of the stripper. The saphenous vein will subsequently be divided through this incision, and the stripper and the inverted vein will be delivered through it. In exposing the saphenous vein at knee level, the superficial fascia must be incised so as to enter the saphenous compartment. If the stripper passes unimpeded to the ankle, it can be exposed there with an exceedingly small skin incision placed in a carefully chosen skin line. Passage of the stripper from above downward to the ankle serves to confirm the absence of functioning valves, and stripping of the vein from above downward is unlikely to cause nerve damage. At the ankle, the vein should be carefully and cleanly dissected to free it from surrounding nerve fibers. If this is not done, saphenous nerve injury will result, and the patient will experience numbness of the foot below the ankle.

Stripping of the saphenous vein has been shown to produce profound distal venous hypertension. This occurs in virtually every operation, even when the limb is elevated. Therefore, after the stripper is placed, one should consider performing the stab avulsion portion of the procedure before the actual stripping maneuver.

Incisions to remove varicose clusters vary according to the size of the vein, the thickness of the vein wall, and the degree to which the vein is adhering to the perivenous tissues. In general, vertical incisions 1 to 3 mm in length are appropriate, except in areas where skin lines are obviously horizontal. Successive incisions are spaced as widely as possible. Varicosities are exteriorized by means of hooks or forceps. Particularly useful for this purpose are the specially

designed vein hooks known by the names Varady dissector, Mueller hook, and Oesch hook. These devices efficiently detach perforating veins from their tributary varicose clusters. Dissection of each perforating vein at the fascial level is not required, and in fact may be cosmetically undesirable. There is no need to ligate or clip the ends of each vein: the combination of leg elevation, trauma-induced venospasm, and direct pressure typically ensures adequate hemostasis. Once exteriorized, the varicosity is divided and avulsed for as long a length as possible. After avulsion, skin edges are approximated with tape or with a single absorbable monofilament suture.

Phlebectomy techniques for varicose clusters have been markedly refined by experienced workers in Europe.

Once the stab avulsion portion of the procedure is complete, the previously placed stripper is pulled distally to remove the saphenous vein. Although plastic disposable vein strippers and their metallic equivalents were designed to be used with various-sized olives to remove the saphenous vein, in fact, a more efficient technique is simply to tie the vein to the stripper below its tip so that the vessel can then be inverted into itself and removed distally. To decrease oozing into the tract created by stripping, a 5 cm roller gauze soaked in a 1% lidocaine-epinephrine solution is attached to the stripper by using the ligature fastening the saphenous vein to the device (see Figure 25.4). Thus, inversion stripping is accompanied by hemostatic packing. The hemostatic pack, which lies within the saphenous vein, can be pulled into the tract with minimum tissue trauma; when it is not inverted into the vein itself, it can act as an obturator to facilitate removal of the saphenous vein without tearing. As the vein is removed by inversion, the gauze is left in place for hemostasis while the remainder of the surgical procedure is being completed.

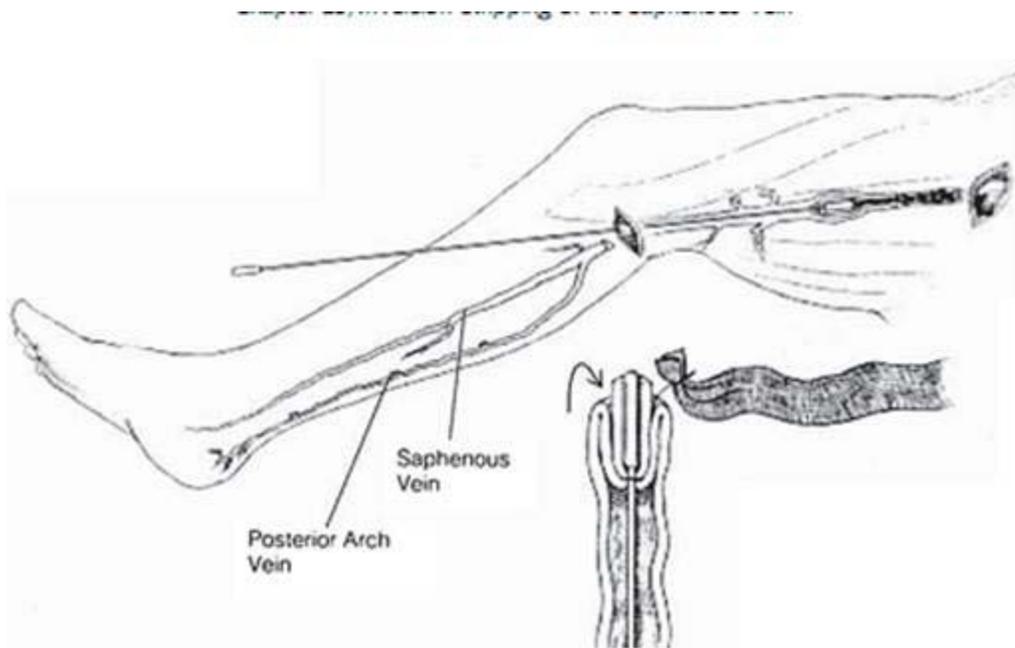


Fig. 8 . Adding a hemostatic pack to inversion stripping corrected the principal flaw in inversion stripping, the tearing of the saphenous vein. The pack acted as an obturator, which insured total vein removal. In most instances, the pack entered the vein as it was being removed, thus minimizing the soft tissue trauma.

POST-THROMBOTIC SYNDROME

Postthrombotic syndrome (PTS) is a frequent sequel to deep venous thrombosis (DVT). Awareness of this long-term debilitating complication is low among treating physicians whose main focus is the acute embolic complications of DVT. PTS may take years and even decades to fully evolve when the patient is no longer in the care of the original treating physician. Recurrent DVT that may occur years later after the initial event is a known risk factor for the development of PTS. Serial follow up of patients after onset of DVT has provided important new perspectives on many aspects of PTS. After a bout of DVT, only a third of the patients are asymptomatic long term; but the other two thirds have PTS, half of them severe. The direct and indirect costs of this disease that affects all adult age groups is estimated to be enormous, arousing the interest of public health planners.

CLINICAL FEATURES

Major symptoms are limb pain, swelling, and stasis skin changes including ulceration. Recurrent thrombophlebitis and recurrent cellulitis, the latter related to underlying tissue edema, are less well known and less frequent features. Symptoms are present in varying combinations and severity in individual patients. Pain is an important but variable component of the symptom complex. Limb swelling may be described by the patient as severe because it is painful even though only mild pitting is evident on examination. Some patients may not even be aware of limb edema evident to the examiner because it is pain free. Pain is absent in about 20% of patients. In about 10% of patients, pain may be the only symptom without other signs; a diagnosis of PTS may altogether be missed, because the limb looks normal. Severity of pain present may be exaggerated or understated by the patient due to individual variations in pain tolerance and other socioeconomic factors such as work situation; daily or frequent use of nonsteroidal or narcotic dependency for pain relief may not be readily disclosed unless specifically questioned. Other essential elements of history may not be readily forthcoming as well. For example, previous DVT or severe trauma to the limb may not be volunteered because the remote event years ago had been forgotten or not considered relevant to current complaints. Because of these variables, a detailed comprehensive history-taking with leading questions is essential for proper assessment; clinical features detected on examination should be recorded and graded for severity during initial and follow-up visits for proper assessment of outcome.

All components of relevant history and physical examination preloaded on a handheld device is a useful guide to those who see these patients only infrequently. The CEAP classification and Venous Severity Scoring endorsed by the vascular societies can serve as readily usable templates for this purpose. In our own system, we have made some additional enhancements that we find useful. Pain is measured on a visual analogue scale, a simple reliable measure of pain that can be used for outcome assessment as well. Limb swelling is variable throughout the day; limb measurement of swelling should be carried out at the same time of the day to be valid for follow-up assessment. Quality of life (QOL) measurements provide a view of outcome from the patient's perspective. The degree of disability and social constraint imposed by this disease can be surprising. Many QOL forms (e.g., CIVIQ) are brief enough for routine use.

DIFFERENTIAL DIAGNOSIS

A clinical diagnosis of chronic venous insufficiency is readily apparent from history and physical examination in most cases but other rarer causes with somewhat similar clinical features have to be borne in mind: Periarteritis nodosa, ruptured Baker's cyst, rheumatoid arthritis, gout, Marjolin's ulcer, arterio-venous malformations of the calf muscles, adverse drug reactions with limb pain and swelling, acanthoma nigricans, pyoderma, and numerous other dermatological and systemic conditions. As venous insufficiency is common, particularly among the elderly, mixed pathologies that aggravate venous symptoms do occur. Combined arterial/venous insufficiency is not uncommon in the elderly; attention to the arterial component first is usually recommended. Differentiating primary from PTS may not be easy and mixed presentations occur as the following discussion on pathology indicates. Differentiation cannot be made on clinical grounds alone as history and physical findings may be similar including the appearance and size of ulcers. About 30% of DVT are estimated to be silent. In others DVT following trauma or surgery is simply missed as symptoms are submerged by expected postoperative pain—a common occurrence following orthopedic procedures on the hip or knee or for treatment of fractures. Patients with deep valvular insufficiency whether primary or post-thrombotic not infrequently present with new onset of acute calf pain and increased swelling in the context of ongoing chronic symptoms. In some, new or recurrent thrombosis is found. In others, no new thrombus is found; the symptoms are presumably due to decompensation of the calf pump from minor injury, low grade cellulitis or other obscure insult that disturbs the equilibrium of the calf pump. A diagnosis of PTS vs primary venous insufficiency is academic from the surgical viewpoint as the approach is the same regardless.

But a diagnosis of PTS may have implications for long-term anticoagulation. In many cases, further investigations may provide helpful clarification.

PATHOLOGY

Our current view of PTS pathology is strongly influenced by the work of Strandness and colleagues. Before then, post-thrombotic clinical syndrome had been viewed as primarily related to the development of reflux. In a remarkable series of landmark papers, these authors showed that the dominant pathology was a combination of obstruction and reflux even though isolated obstruction and reflux occurred in some. The location and progression of post-thrombotic reflux followed by serial duplex were unexpected and intriguing. Reflux occurred not only in segments involved by thrombus but also in segments remote from them. Reflux occurred and progressed over time not only in deep venous segments distal to the thrombotic segment but also in segments proximal; in the distal segments, dilatation of the valve station due to cephalad obstruction was not found to be the cause of reflux. The fact that reflux occurs and progresses over time in superficial as well as deep valves proximal to the obstructed segment suggests a different (maybe cytokines), as yet poorly understood, mechanism.

Some patients present with femoral valve reflux and thrombosis in the distal femoral popliteal segment or even the calf. This clinical profile could be due to reflux stasis-induced distal thrombosis. Repair of the valve reflux can abate recurrent thrombosis. Similar type of clinical presentation also can result from evolution of de novo reflux above the thrombotic segment as described by Strandness and colleagues. Perivenous and mural fibrosis is a feature of these valves with constriction and foreshortening of the valve station (see Figure 9). The valve cusps themselves are redundant and reflexive apparently as a result of the fibrotic wall changes. The fibrotic valve station is somewhat smaller than the classic primary valve, but the cusps themselves appear normal but redundant and can be repaired like the primary valve using direct repair techniques. A plausible explanation for these features and perhaps for the remote reflux described by Strandness's group is that perivenous and mural fibrosis may extend beyond the thrombosed segment to involve adjacent segments of preserving valve cusps, but inducing secondary reflux from valve station restriction.

Valves may also escape destruction because the thrombus in the resident segment lyses, but not without inducing fibrotic changes as described.

'SECONDARY' VALVE REFLUX

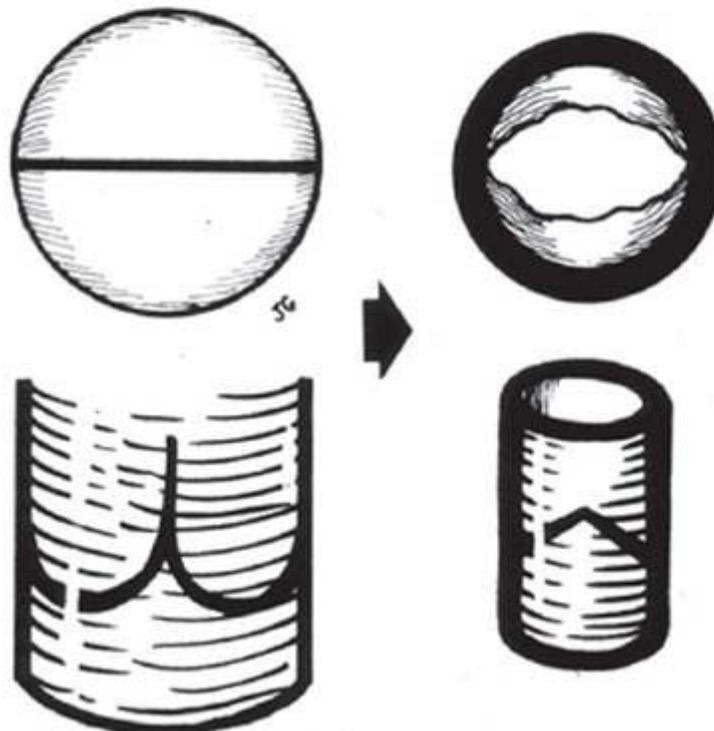


Fig. 9. A possible mechanism for the production of valve redundancy and reflux in post-thrombotic valve stations. Valve station fibrosis may lead to luminal constriction resulting in secondary valve leaflet redundancy and reflux. Foreshortening of the valve station may lead to widening of the commissural valve angle, contributing further to development of reflux.

INVESTIGATIONS

A comprehensive set of investigations are necessary for proper management of patients, particularly if invasive or other surgical intervention is contemplated. The aim is to clarify the pathology, identify the sites and nature of pathology, and grade its severity.

Duplex

Duplex is the initial and in many centers the only technique used. It has many deficiencies when used alone in assessment. As a qualitative tool, it can detect local reflux but cannot grade it nor can it adequately provide a measure of the overall severity of reflux in the

limb when multiple segments are involved. Valve closure time (VCT) has received much attention as a quantitative tool in this regard. Though it can identify reflux in a particular segment, VCT has poor correlation with the severity of reflux present. Trivial reflux may be associated with prolonged VCT and conversely high grade reflux may have only slightly prolonged VCT. Peak reflux velocity has a better correlation, but not to a degree that is clinically useable. At present relatively crude indices such as multisegment score (number of refluxive segments) or the presence of axial reflux are the best measures available. Iliac vein outflow obstruction, an important contributor to PTS, is frequently impervious to duplex.

Venography

Unlike duplex, ascending venography provides a more composite view of venous pathology below the inguinal ligament. Post-thrombotic changes, segmental occlusions, and collateral patterns are readily apparent. The profunda femoris vein is the major natural collateral pathway in femoral stenoses and occlusions. This has an embryologic basis as the profunda femoris is the early axial vein receding to the mature pattern later in embryologic development. A putative profunda-popliteal connection apparently exists as a high resistance embryologic residue; profunda collateral flow can be observed as early as a few hours after onset of acute DVT in venograms. In chronic femoral vein occlusions, the profunda enlarges to the same caliber as the normal femoral vein (see Figure 11). This pattern of complete axial transformation of the profunda femoris vein occurs in about 15% of postthrombotic limbs. Reflux may result from enlargement of the profunda valve station and may be severe with symptoms. Lesser degrees of profunda enlargement can be found in other cases where the femoral vein is not totally occluded but is stenotic.

Because the direction of collateral flow in the profunda is the same as natural flow direction in the vessel, it is very efficient. Once fully developed, the profunda fully compensates for the loss of femoral flow with few residual clinical symptoms from outflow obstruction. In iliac vein occlusions, collateral flow is mainly through tributaries of the iliac vein itself, requiring reversal of normal flow direction. Collateral flow seems to be less efficient and residual outflow obstruction is present in nearly half the cases with iliac occlusions.

These differential patterns of collateral development and function have clinical import. In patients with symptoms of outflow obstruction, iliac vein pathology is likely to be the culprit even if associated femoral vein occlusion is more readily seen on ascending venography. Ascending venography is inadequate for assessment of the iliac vein and the vena cava due to

contrast dilution; stenotic lesions may easily be missed.



Fig. 11. Axial transformation of profunda femoral vein through a large profunda-popliteal connection. The femoral vein is largely occluded with the distal end seen as a stump.

Transfemoral venography is the procedure of choice for pelvic venous assessment. Exercise femoral venous pressures can be concurrently measured, which can be helpful in grading severity of outflow obstruction. Descending venography can also be performed at the same time to define the architecture of femoral valves. Descending venography is no longer used for grading reflux due to lack of specificity.

A common pattern in severely post-thrombotic limbs is where the entire outflow appears to occur through the superficial veins with nonvisualization of deep veins giving the appearance of wiped-out deep system. This is invariably an artifact of technique. In most such cases a patent but post-thrombotic deep system with numerous collateral elements can be demonstrated on descending venography (see Figure 12). Presumably, there is a positive gradient across

superficial to deep venous connections in these cases that contrast flow preferentially is restricted to the superficial system. The collateral contribution of the superficial system in such cases is negligible. Since the deep system is patent, reconstructive procedures can be planned despite the spurious appearance on ascending venography.

Several authors beginning with Rokitanski have documented the development of a dense perivenous sheath in postthrombotic iliac veins. This prevents or retards the development of collaterals. Surgical attempts have been made to remove the sheath for improving flow. The venographic appearance in such cases is one of diffuse stenosis without collaterals. Iliac vein pathology is easily missed in such cases, especially with ascending venography. With transfemoral venography, the diffuse lesion can be quite evident or subtle requiring measurements of vein diameter, which is seldom practiced. Because of this and other factors cited earlier, the sensitivity of venography in iliac vein pathology is only in the order of 60%.



Fig. 12. Ascending venogram opacifies only superficial network (right). The deep system appears wiped out. This is often a technical artifact ample deep venous elements are demonstrated on descending

venography (left).

Intravascular Ultrasound (IVUS)

Intravascular ultrasound is superior to venography in the assessment of post-thrombotic iliac vein and the inferior vena cava. Perivenous and mural fibrosis, stenoses and trabeculae are readily seen. It is invaluable in iliac vein stent placement.

Lymphangiography

About 30% of patients with deep venous insufficiency have lymphographic abnormalities such as pooling and delayed or absent lymphatic transport. Most are thought to be secondary to venous pathology from lymphatic exhaustion or damage. Some may be reversible with correction of venous pathology. Lymphographic information has prognostic value in resolution of leg swelling and affected patients may be adequately forewarned before interventions.

Airplethysmography (APG)

Measurement of ejection fraction and residual volume have been suggested as indirect indices of outflow obstruction. In our own and others' experience, specificity and sensitivity have been inconsistent. VFI appears to be a useful measure of reflux.

Ambulatory Venous Pressure Measurement

Ambulatory venous pressure measurement provides a global index of venous function in the limb encompassing multiple components. Post-exercise pressure (% drop) has an inconsistent relationship to the severity of outflow obstruction presumably because of the variability of calf pump efficiency. The recovery time or venous filling time (VFT) has been useful in assessing severity of postthrombotic pathology and reflux. A postoperative VFT of >5 seconds bodes well for a good surgical outcome; a VFT of <5 seconds the opposite. The mean improvement in VFT after successful repairs with good clinical outcome is generally in the order of about 6 ± 4 (SD) seconds. After successful valve repair, postoperative VFT does not reach normal levels in many patients. VFT is influenced not only by reflux but a

multiplicity of other factors. Compliance of the conduit below the valve profoundly affects VFT even more than reflux at the valve. Failure to normalize or substantially improve VFT is probably related to the poor venous compliance in post-thrombotic extremities.

Measurement of Outflow Obstruction

Reduced or absent phasicity on duplex examination is often indicative of outflow obstruction at the iliac vein level, the information being qualitative. There are no reliable methods of functionally quantifying and grading outflow obstruction at the present time. Plethysmographic outflow fraction measurement such as with strain gauge technique and APG yield unacceptably high false positives due to compliance changes in the post-thrombotic calf; a reduced outflow fraction (<50%) results from subpar emptying of the venous pool from poor compliance as often as from outflow obstruction per se. And poor compliance may be present without obstruction. A reduced outflow fraction is indicative of post-thrombotic changes, not necessarily obstruction.

Pressure-based tests to detect and grade severity of obstruction such as arm/foot venous pressure differential with reactive hyperemia, exercise femoral venous pressures measurement, and intraoperative femoral vein pressure measurement with papavarine are positive only in about a third of cases. Assessment of outflow obstruction currently rests entirely on morphologic methodology (IVUS) restricted to the iliac vein segment.

TREATMENT

Compression Therapy

Compression therapy is the oldest and until recently the only therapeutic option available to treat PTS. It has been reported anecdotally to be ineffective in PTS but no systematic study has been undertaken. Compression therapy remains the initial approach in chronic venous disease including PTS. Some patients do fail compression therapy despite faithful compliance. Noncompliance, however, is the major cause of compression failure and recurrent symptoms. Noncompliance is high even in cold climates as documented in several community surveys. Longerm supervision or monitoring by health care workers has been advocated to improve compliance. However, noncompliance is high even under supervision. The reasons for noncompliance are many—tightness or fit (cutting off circulation), warm

weather, lack of efficacy, contact dermatitis, recurrent cost and inability to apply stockings due to frailty or arthritis are among the many reasons/excuses cited by patients. But the main underlying reason, often unstated, appears to be the restrictions and negatives of compression regimens in today's image-conscious world with expectations of an unrestricted lifestyle. Thus compression is a quality of life issue from the patient's viewpoint. Demands for compliance are unlikely to succeed after previous entreaties have failed and may not be appropriate when therapeutic alternatives have become available. Compression should be viewed not as an end itself, but complementary to the extent patients are willing to use them. Compression should be considered a failure regardless of the cause including noncompliance if symptom relief is not obtained after trial over a reasonable period of time, say three to six months depending on the clinical and socioeconomic situation of the patient. Worsening of symptoms or onset of complications such as recurrent infections during the trial period are also considered failures. Some patients are not candidates for compression therapy at all due to comorbidities (e.g., arthritis, frailty, or arterial compromise) or special work situations. Nonresponders should be offered alternatives, not life-long gunna boot regimens as was the case before by necessity, and continues to be so in many parts of the world due to a conservative philosophy of health care delivery.

Saphenous Vein Ablation

There has been traditional advice against saphenous ablation in the presence of deep venous obstruction (secondary varices) to preserve its collateral contribution. The collateral contribution of saphenous vein in the presence of deep venous obstruction is insignificant. Stripping of a refluxive saphenous vein in PTS cases can provide significant symptom benefit by eliminating the reflux component without jeopardizing the limb. Stripping can be easily combined with valve reconstruction in the femoral area. The newer minimally invasive techniques of saphenous ablation are suitable alternatives as well and are easily combined with iliac vein stent placement when indicated

Valvuloplasty

In PTS patients, direct femoral or popliteal valve repair can be performed if the basic valve architecture is preserved. Eriksson stressed the importance of profunda valve repair in post-thrombotic cases due to the frequent presence of collateral reflux. We prefer an external or

transmural technique without a venotomy for these cases as they are faster and hence multiple repairs (i.e., femoral and profunda) can be performed in a single sitting; and repairs can be carried out even in constricted or small valve stations. The internal technique is disadvantaged in comparison.

Trancommissural technique, which closes the wide valve angle present at the commissure and simultaneously tightens the lax valve cusps by transmural sutures that can be placed blindly in a reliable fashion. The first step in the procedure is to carry out an adventitial dissection to peel away the fibrous sheath surrounding the valve station. Valve attachment lines should become visible after the dissection. They should be defined in their entirety, which is necessary for placement of trancommissural sutures. Though the sutures are placed blindly, adherence to the technique as described in the original publication will result in technical success of >95%.

Absent or interrupted valve attachment lines invariably indicate cusp dissolution or damage beyond direct repair. In such cases one should proceed forthwith with axillary vein transfer without wasting time on performing a venotomy in a futile search for repairable valve cusps.

Axillary Vein Transfer

Axillary vein transfer is the mainstay of repair in PTS cases when direct valve repair is not feasible due to damage to the valve cusps. Seemingly a simple technique, it is in fact, quite demanding, requiring precise execution. Proctored learning is recommended to achieve consistently good results. The transferred valve should match the size of the native valve station being reconstructed. In most cases, the axillary vein is the preferred donor site to obtain a good size match. In a minority, the proximal brachial vein may also be suitable in size. We approach the axillary-brachial veins through a transverse incision in the armpit along the skin crease; exposure of 5 to 6 cm length of axillary vein segment will require ligation and division of three or more tributaries in the area. One or more valves will then come into view. We have not found preoperative duplex examination useful in locating a transferable valve. The valve with a good size match is chosen for transfer. A valve high up in the axilla at or near the first rib is consistently present and is the largest. This may require additional dissection for exposure. The chosen valve is then tested for competence by negative (emptying the infravalvular segment) and positive (squeezing the supra-avalvular segment) strip tests. About 40% of axillary valves

will fail the strip tests, in which case they should be repaired by the transcommissural technique preferably in situ or on the bench before transfer. A 4 cm vein segment housing the valve is excised and the ends of the remaining vein ligated. A lesser length will interfere with later anastomoses as the excised segment shrinks, risking the valve cusps being caught up in the suture lines. Reconstruction of the donor vein is not required; outflow obstructive symptoms in the donor limb are extremely rare. The transferable valve is dropped in cold balanced salt solution for a few minutes and then transferred to the recipient site. A 1 cm segment of the recipient vein is excised, which results in retraction of the cut ends leaving a longer gap. The axillary valve is transferred in proper orientation and the proximal anastomosis is performed first. Interrupted 6° monofilament permanent sutures should be used throughout. Continuous sutures, howsoever expertly applied, will result in postoperative suture line stenosis as the native and transferred vein segments dilate to their normal caliber, freed of intraoperative spasm. Once the upper anastomosis is completed, the valve should be retested for competence by the strip tests. Some axillary valve sinuses are shallow and are prone to de novo reflux with minor distortions of architecture that may occur during the transfer procedure. A transcommissural repair may then be required at this stage to achieve perfect competence. Before starting the distal anastomosis, the distal end of the recipient vein should be trimmed to match the length of the donor segment put on a mild stretch. A slack or overstretched donor segment will result in reflux. Proper rotational orientation of the transferred segment is crucial. There should be no hesitancy to take down and redo the distal suture line if imperfections or reflux is discovered after completion. Final positive and negative strip tests are performed to assure competence. The axillary vein has a thinner muscle layer than the native recipient vein because of the higher orthostatic pressures prevalent in the latter. This may result in gradual dilatation of the transferred axillary vein segment with onset of reflux. This problem encountered in early experience was addressed by placing a prosthetic sleeve around the transferred vein segment. Currently an 8 to 10 mm PTFE sleeve, 3 cm long, is split open and sutured back as a loose fitting sleeve around the transferred axillary valve with one or two anchoring sutures to the adventitia to prevent migration. Slipping an unopened sleeve over the lower end of the transferred segment before beginning the distal suture line may obscure rotational orientation of the transferred valve resulting in reflux after completion of the suture line and is to be avoided. The incision is closed with a closed drainage system. Penrose drainage is provided for the axillary incision. To avoid compression of the repair by fluid collection in a tight space, only the superficial fascia is closed with interrupted sutures and the deep fascia is left open.

In trabeculated post-thrombotic veins, modifications of the basic technique are

necessary. The trabeculae at the site of proximal and distal suture lines are excised (see Figure 13, 14, 15) to create a single lumen at the site for anastomoses.

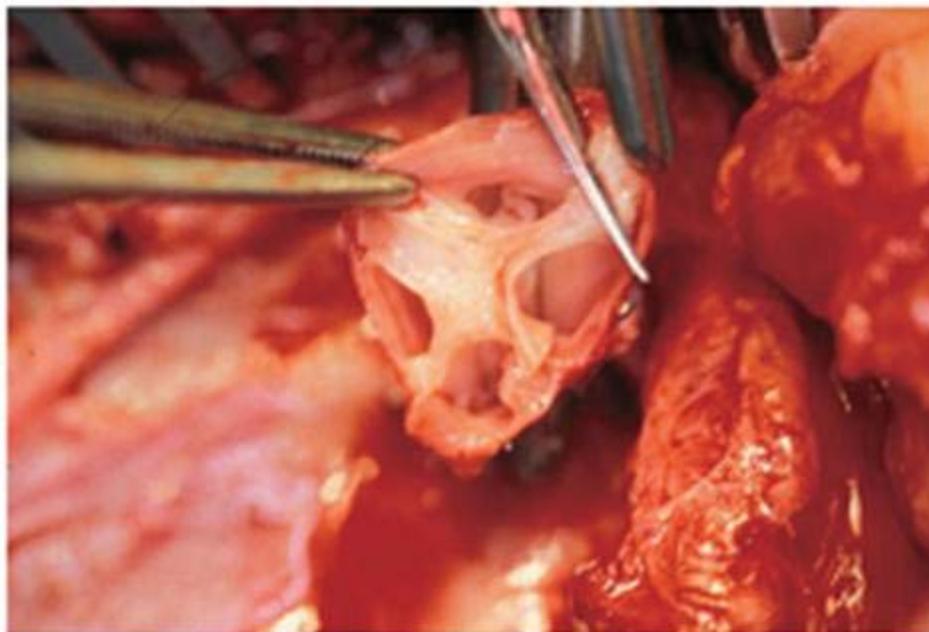


Fig. 13.

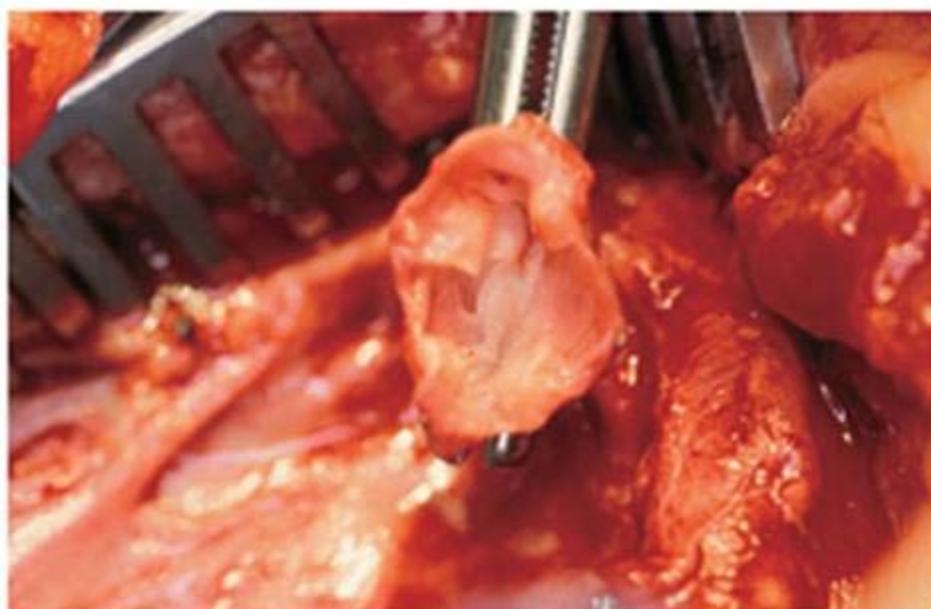


Fig. 14.

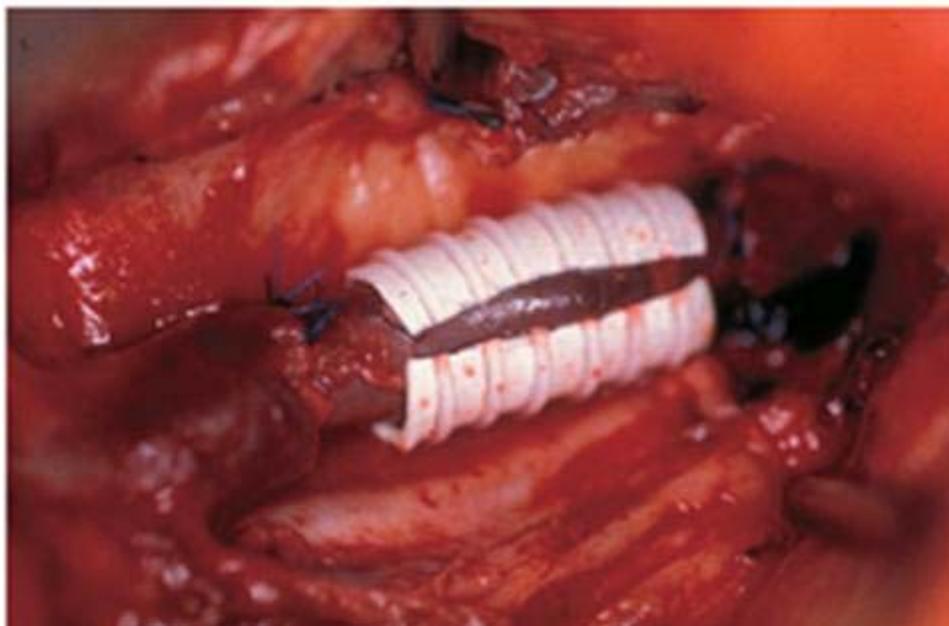


Fig. 15.

In a subset of PTS patients both the femoral and profunda femoral veins are severely post-thrombotic with destroyed valve structures. The femoral confluence can be repaired with individual axillary vein transfers or by en bloc transfer of basilic-brachial confluence provided valves are present and size match requirements are satisfied (see Figure 16).



Fig.16.

LYMPHEDEMA

Background

Lymphedema is an abnormal collection of protein-rich fluid in the interstitium due to a defect in the lymphatic drainage network. Lymphedema most commonly affects the extremities, but it can involve the face, genitalia, or trunk. Numerous causes, both primary and secondary in nature, have been identified for this condition.

The primary causes are due to abnormalities in the lymphatic system that are present at birth, although not always clinically evident until later in life. The 3 primary categories of lymphedema (due to genetic factors) are congenital lymphedema (Milroy disease), lymphedema praecox (Meige disease), and lymphedema tarda. Primary lymphedema can also be associated with various cutaneous syndromes.

Secondary lymphedema is due to an acquired obstruction or infiltration of the lymphatic system. Secondary lymphedema has a number of causes, which include malignancy, infection, obesity, trauma, congestive heart failure, portal hypertension, and therapeutic intervention. Despite the fact that the underlying etiologies of secondary lymphedema vary, clinical progression is similar and difficult to control.

Lymphedema is a progressive, deforming condition that is both physically and psychologically debilitating.

Angiosarcoma arising in an area of long-standing lymphedema is termed Stewart-Treves syndrome. Most cases of Stewart-Treves syndrome occur in the arm after surgery for breast cancer; however, sometimes angiosarcomas can arise in a chronically lymphedematous leg.

Lymphedema is a notoriously debilitating progressive condition with no known cure. The unfortunate patient faces a lifelong struggle of medical, and sometimes surgical, treatment fraught with potentially lethal complications.

The underlying problem is lymphatic dysfunction, resulting in an abnormal accumulation of interstitial fluid containing high molecular weight proteins. This condition underscores the tremendous importance of a normally functioning lymphatic system, which returns proteins, lipids, and accompanying water from the interstitium to the venous circulation near the subclavian vein–internal jugular vein junction, bilaterally. The normal and abnormal flow of

interstitial fluid through the lymphatic system are demonstrated below.

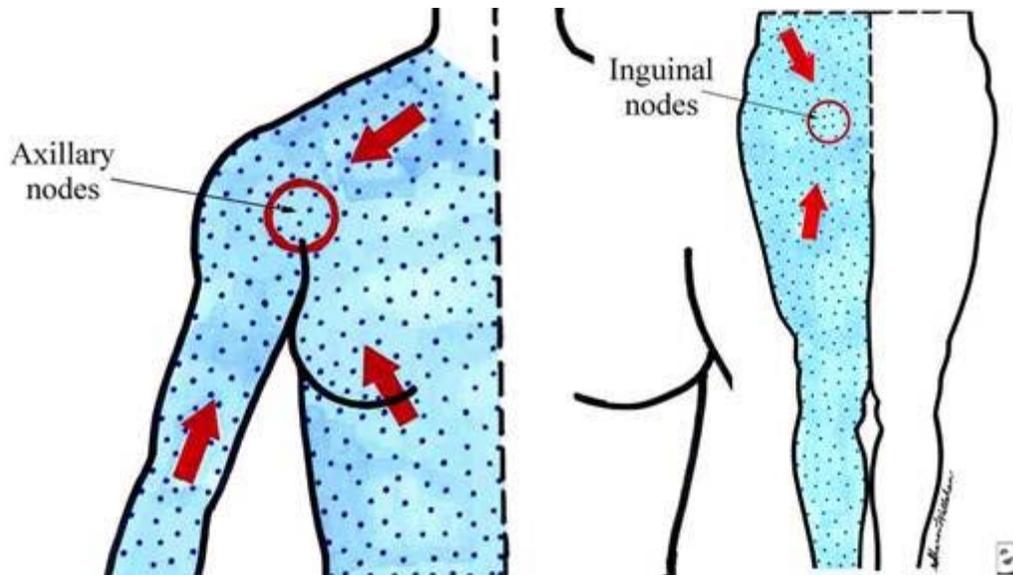


Fig. 17. The body quadrants of superficial lymph drainage

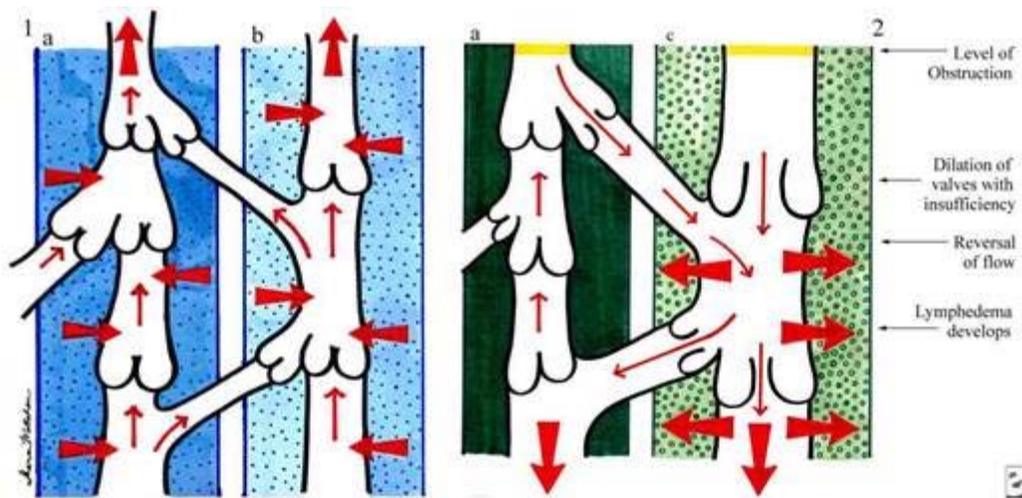


Fig. 18. (1) Normal lymphatic flow in (a) deep systems and (b) superficial systems. Note the small collateral vessels interconnecting the 2 systems. (2) Lymphedema develops from obstruction, dilation of valves, valvular insufficiency, and subsequent reversal of lymphatic flow.

Pathophysiology

The normal function of the lymphatics is to return proteins, lipids, and water from the interstitium to the intravascular space; 40-50% of serum proteins are transported by this route

each day. High hydrostatic pressures in arterial capillaries force proteinaceous fluid into the interstitium, resulting in increased interstitial oncotic pressure that draws in additional water.

Interstitial fluid normally contributes to the nourishment of tissues. About 90% of the fluid returns to the circulation via entry into venous capillaries. The remaining 10% is composed of high molecular weight proteins and their oncotically associated water, which are too large to readily pass through venous capillary walls. This leads to flow into the lymphatic capillaries where pressures are typically subatmospheric and can accommodate the large size of the proteins and their accompanying water. The proteins then travel as lymph through numerous filtering lymph nodes on their way to join the venous circulation.

In a diseased state, the lymphatic transport capacity is reduced. This causes the normal volume of interstitial fluid formation to exceed the rate of lymphatic return, resulting in the stagnation of high molecular weight proteins in the interstitium. It usually occurs after flow has been reduced by 80% or more. The result, as compared to other forms of edema that have much lower concentrations of protein, is high-protein edema, or lymphedema, with protein concentrations of 1.0-5.5 g/mL. This high oncotic pressure in the interstitium favors the accumulation of additional water.

Accumulation of interstitial fluid leads to massive dilatation of the remaining outflow tracts and valvular incompetence that causes reversal of flow from subcutaneous tissues into the dermal plexus. The lymphatic walls undergo fibrosis, and fibrinoid thrombi accumulate within the lumen, obliterating much of the remaining lymph channels. Spontaneous lymphovenous shunts may form. Lymph nodes harden and shrink, losing their normal architecture.

In the interstitium, protein and fluid accumulation initiates a marked inflammatory reaction. Macrophage activity is increased, resulting in destruction of elastic fibers and production of fibrosclerotic tissue. Fibroblasts migrate into the interstitium and deposit collagen. The result of this inflammatory reaction is a change from the initial pitting edema to the brawny nonpitting edema characteristic of lymphedema. Consequently, local immunologic surveillance is suppressed, and chronic infections, as well as malignant degeneration to lymphangiosarcoma, may occur.

The overlying skin becomes thickened and displays the typical *peau d'orange* (orange skin) appearance of congested dermal lymphatics. The epidermis forms thick scaly deposits of keratinized debris and may display a warty verrucosis. Cracks and furrows often develop and accommodate debris and bacteria, leading to lymphorrhea, the leakage of lymph onto the surface of the skin.

Frequency

United States

A common cause of lymphedema reported in the United States is surgical therapeutic intervention (postsurgical complication), most commonly following mastectomy with axillary dissection and radiation therapy for breast cancer. Although not reported as often as postmastectomy induced–lymphedema, obesity is one of the most common causes of lymphedema seen in practice today.

Among the primary causes of lymphedema, lymphedema praecox is the most commonly reported.

International

Worldwide, the most common cause of lymphedema is filariasis infection. More than 100 million people are affected in endemic areas worldwide

Mortality/Morbidity

The outcome for persons with lymphedema depends on its chronicity, the complications that result, and the underlying disease state that caused the lymphedema.

The development of angiosarcoma (ie, Stewart-Treves syndrome) in the setting of lymphedema is the most serious complication of secondary lymphedema. The mean survival rate, after treatment, is approximately 24 months. The 5-year survival rate is 10%.

Other complications that increase morbidity are the development of recurrent cellulitis, bacterial or fungal infections, and lymphangioadenitis.

Sex

Primary lymphedema is most common in females. Lymphedema praecox is the most common form and affects 1 in 100,000 girls and 1 in 400,000 boys.

Age

Secondary lymphedema can affect persons of any age group, and its onset is determined by the primary cause. Hereditary (primary) lymphedema can be divided into 3 groups based on the age of onset of clinical lymphedema, as follows.

- Milroy disease is the familial form of lymphedema that usually manifests from birth to age 1 year.
- Lymphedema praecox (ie, Meige disease) occurs from age 1-35 years. It most commonly occurs around menarche.
- Lymphedema tarda manifests after age 35 years

CLINICAL

History

Patients often report that chronic swelling of an extremity preceded lymphedema. Eighty percent of patients present with lower extremity involvement, although the upper extremities, face, genitalia, and trunk can also be involved. The history confirms involvement of a distal extremity initially, with proximal involvement following. Patients with lymphedema often report painless swelling and leg heaviness.

Fevers, chills, and generalized weakness may be reported. Patients may have a history of recurrent episodes of cellulitis, lymphangitis, fissuring, ulcerations, and/or verrucous changes. Patients have a higher prevalence of bacterial and fungal infections.

In primary lymphedema, patients have a congenital defect in the lymphatic system; therefore, the history of onset is more typical of the specific type.

Also more common is for primary lymphedema to be associated with other anomalies and genetic disorders, such as yellow nail syndrome, Turner syndrome, Noonan syndrome, xanthomatosis,² hemangiomas, neurofibromatosis type 1, distichiasis lymphedema,^{3,4} Klinefelter syndrome, congenital absence of nails, trisomy 21, trisomy 13, and trisomy 18.

A rare inherited disorder, distichiasis-lymphedema syndrome, is characterized by the presence of extra eyelashes (distichiasis) and swelling of the arms and legs (lymphedema). Swelling of the legs, especially below the knees, and eye irritation are common in people with

this disorder. Spinal cysts (epidural) with or without other abnormalities of the spinal column can accompany distichiasis lymphedema. Distichiasis-lymphedema syndrome is inherited as an autosomal dominant genetic trait due to a mutation of the FOX2 gene.

In secondary lymphedema, the associated history should be more evident, based on the primary etiology.

If due to filariasis, the history should include travel or habitation in an endemic area.

Other patients should have a clear history of a neoplasm obstructing the lymphatic system, recurrent episodes of lymphangitis and/or cellulitis, obesity, trauma, or lymphedema resulting after surgery and/or radiation therapy.

A recent history of varicose vein surgery also is reported.

In 2009, Lu et al noted 24 cases of localized lymphedema presenting as solitary large polyps, solid or papillomatous plaques, pedunculated edematous lesions, or tumors that imitated sarcoma. Lesions most commonly occurred on the vulva.

Physical

The earliest symptom of lymphedema is nontender pitting edema of the affected area, most commonly the distal extremities. The face, trunk, and genitalia also may be involved. Radial enlargement of the area occurs over time, which progresses to a nonpitting edema resulting from the development of fibrosis in the subcutaneous fat.

The distal extremities are involved initially, followed by proximal advancement.

Patients have erythema of the affected area and thickening of the skin, which appears as peau d'orange skin and woody edema.

With long-term involvement, ENV develops, which is an area of cobble-stoned, hyperkeratotic, papillomatous plaques most commonly seen on the shins. The plaques of ENV can be covered with a loosely adherent crust, can be weepy or oozing a clear or yellow fluid, and/or can have a foul-smelling odor. The changes of ENV have been described as cobblestone, pebbly, hyperkeratotic, papillomatous, and verrucous.

Fissuring, ulcerations, skin breakdown, and lymphorrhea can also be seen. Lymphorrhea involves the weeping or oozing of clear, yellow, or straw-colored fluids. Superinfection is common and can manifest as impetigo with yellow crusts.

Four cases of cutaneous verruciform xanthomas in association with lymphedema have been cited in the literature. More recent reports have suggested that verruciform xanthomas may be a rare reactive phenomenon found in persons with common cutaneous conditions. Because

verruciform xanthoma is considered by some authorities to be a reactive condition, the link between these 2 entities remains unclear at this time.

A positive Stemmer sign (inability to pinch the dorsal aspect of skin between the first and second toes) may be elicited upon examination.

Other associated physical findings specific for the cause of secondary lymphedema and genetic disorders involving lymphedema may be noted upon examination.

Causes

Both primary and secondary lymphedema can have many causes.

Primary lymphedema

Primary lymphedema is divided into 3 main types, which are distinguished by their age of onset. All are caused by a congenital abnormality in the lymphatic system, although these defects may not always be clinically evident until later in life. Additionally, primary lymphedema also can be associated with other cutaneous and genetic disorders not among the 3 main age-based categories.

Congenital lymphedema, also known as Milroy disease, is an autosomal dominant familial disorder of the lymphatic system that manifests at birth to age 1 year. It is often due to anaplastic lymphatic channels. The lower extremity edema is most commonly bilateral, pitting, and nonpainful. This condition may be linked to a mutation that inactivates VEGFR3. It has been associated with cellulitis, prominent veins, intestinal lymphangiectasias, upturned toenails, and hydrocele.

Lymphedema praecox, also known as Meige disease, is the most common form of primary lymphedema. Seventy percent of cases are unilateral, with lower extremity swelling being more common. This type of primary edema is most often due to hypoplastic lymphatic channels. This condition most often manifests clinically around menarche, suggesting that estrogen may play a role in its pathogenesis.

Lymphedema tarda manifests later in life, usually in persons older than 35 years. It is thought to be due to a defect in the lymphatic valves, resulting in incompetent valve function. Whether this defect is congenital or acquired is difficult to determine.

As mentioned, primary lymphedema is also seen in association with other cutaneous and

genetic disorders.

Lymphedema-distichiasis syndrome is a form of hereditary early- and late-onset lymphedema associated with distichiasis (double row of eyelashes). Affected persons usually manifest bilateral lower extremity lymphedema by age 8-30 years. Lymphatic vessels are usually larger in affected areas. It is a hereditary condition with an autosomal dominant pattern with variable penetrance. It reportedly is associated with a mutation in FOXC2 transcription factor.⁵ Other associated anomalies may include vertebral abnormalities, spinal arachnoid cysts, hemangiomas, cleft palate, ptosis, short stature, webbed neck, strabismus, thoracic duct abnormalities, and microphthalmia.

Primary lymphedema has also been associated with yellow nail syndrome. This entity may be associated with recurrent pleural effusions and bronchiectasis.

Other genetic syndromes and cutaneous conditions associated with primary lymphedema are Turner syndrome, Noonan syndrome, Klinefelter syndrome, neurofibromatosis type 1, hemangiomas, xanthomatosis, and congenital absence of nails. One reported case described lymphedema in association with CHARGE (coloboma, heart anomalies, choanal atresia, somatic and mental retardation, genitourinary anomalies, ear abnormalities) syndrome.

Secondary lymphedema

Secondary Lymphedema is caused by an acquired defect in the lymphatic system and is commonly associated with obesity, infection, neoplasm, trauma, or therapeutic modalities.

The most common cause of secondary lymphedema worldwide is filariasis. This is due to a mosquito-borne nematode infection with the parasite *Wucheria bancrofti*. It commonly occurs in developing countries around the world. This infection results in permanent lymphedema of the limb.

In the developed world, the most common cause of secondary lymphedema is malignancy and treatment.

- It can result from obstruction from metastatic cancer or primary lymphoma or can be secondary to radical lymph node dissection and excision. Although lymphatics are thought to regenerate after transection via surgery, when combined with radiotherapy to the area, the risk of lymphedema increases because of scarring and fibrosis of the tissue.

- The most commonly affected area is the axillary region after mastectomy and radical dissection for breast cancer. Lymphedema can also be seen after regional dissection of pelvic,

para-aortic, and neck lymph nodes.

- Other associated neoplastic diseases are Hodgkin lymphoma, metastatic prostate cancer, cervical cancer, breast cancer, and melanoma.

Morbid obesity frequently causes impairment of lymphatic return and commonly results in lymphedema, as shown in the image below.



Fig. 19. Morbidly obese patient with lymphedema.

- Lymphedema is also associated with trauma, varicose vein surgery, congestive heart failure, portal hypertension, and extrinsic pressure, as shown in the image below.



Fig. 20. Lymphedema in a patient with hypertension, diabetes, and impaired cardiac function

- Recurrent episodes of cellulitis or streptococcal lymphangitis have been linked to the development of lymphedema.

Laboratory Studies

Analysis of blood, urine, or tissue is not needed to make the diagnosis of lymphedema. Such tests, however, help to define the underlying causes of lower extremity edema when the etiology is unclear.

- Liver function, BUN/creatinine levels, and urinalysis results should be checked if a renal or hepatic etiology is suspected.

- Specific markers should be checked if a neoplasm is suspected.

- CBC count with differential should be checked if an infectious etiology is being considered.

Imaging Studies

Imaging is not necessary to make the diagnosis, but it can be used to confirm it, to assess the extent of involvement, and to determine therapeutic intervention.

- Lymphangiography is an invasive technique that can be used to evaluate the lymphatic system and its patency. Although it was once thought to be the first-line imaging modality for lymphedema, it is now rarely used because of the potential adverse effects.

- Lymphoscintigraphy is the new criterion standard to assess the lymphatic system. It allows for detailed visualization of the lymphatic channels with minimal risk. The anatomy and the obstructed areas of lymphatic flow can be assessed.

- Ultrasonography can be used to evaluate the lymphatic and venous systems. Volumetric and structural changes are identified within the lymphatic system. Venous abnormalities such as deep vein thrombosis can be excluded based on ultrasonography findings.

- MRI and CT scanning can also be used to evaluate lymphedema. These radiologic tests can be helpful in confirming the diagnosis and monitoring the effects of treatment. They are also recommended when malignancy is suspected.

Other Tests

A biopsy should be performed if the diagnosis is not clinically apparent, if areas of chronic lymphedema look suspicious, or if areas of chronic ulceration exist.

Histologic Findings

Histologic findings include hyperkeratosis with areas of parakeratosis, acanthosis, and diffuse dermal edema with dilated lymphatic spaces. In chronic lymphedema, marked fibrosis and scattered foci of inflammatory infiltrate can be seen.

Medical Care

The goal of therapy is to restore function, to reduce physical and psychologic suffering, and to prevent the development of infection.

- The first-line treatment is complex physical therapy. This therapy is aimed at improving lymphedema with manual lymphatic drainage, massage, and exercise. It advocates the use of compression stockings (at a minimum of 40 mm Hg), multilayer bandaging, or pneumatic pumps. Leg elevation is essential. Appropriate skin care and debridement is also stressed to prevent recurrent cellulitis or lymphangitis.
- In secondary lymphedema, the underlying etiology (ie, neoplasm, infection) should also be properly treated to relieve the lymphatic obstruction and to improve lymphedema.

- In cases of recurrent cellulitis or lymphangitis, long-term antibiotic therapy with agents such as penicillins or cephalosporins is sometimes used.
- Filariasis has been treated with diethylcarbamazine and albendazole.
- In cases associated with obesity, weight loss is strongly recommended.
- A few pharmacological therapies have been found to be effective in the treatment of lymphedema.
 - The benzopyrones (including coumarin and flavonoids) are a group of drugs that have been found to be successful in treating lymphedema when combined with complex physical therapy. They aid in decreasing excess edematous fluid, softening the limb, decreasing skin temperature, and decreasing the number of secondary infections. The benzopyrones successfully increase the number of macrophages, leading to proteolysis and protein reabsorption. Of note, however, is that hepatotoxicity has been associated with coumarin therapy.
 - Case reports have suggested effective treatment of chronic lymphedematous changes (eg, elephantiasis nostra verrucosa [ENV]) with oral and topical retinoids. These therapies are thought to help normalize keratinization and decrease inflammatory and fibrotic changes.
 - Topical emollients and keratolytics, such as ammonium lactate, urea, and salicylic acid, have been recommended to improve secondary epidermal changes.
 - Diuretics are not effective in treating lymphedema.

Surgical treatment is palliative, not curative, and it does not obviate the need for continued medical therapy. Moreover, it is rarely indicated as the primary treatment modality. Rather, reserve surgical treatment for those who do not improve with conservative measures or in cases where the extremity is so large that it impairs daily activities and prevents successful conservative management.

Medication

Retinoids

These therapies are thought to help normalize keratinization and decrease inflammatory and fibrotic changes.

Acitretin (Soriatane)

Metabolite of etretinate and related to both retinoic acid and retinol (vitamin A). Mechanism of action unknown; however, thought to exert therapeutic effect by modulating keratinocyte differentiation, keratinocyte hyperproliferation, and tissue infiltration by inflammatory cells.

Tazarotene, topical (Tazorac)

Topical gel 0.1%. Retinoid prodrug whose active metabolite modulates differentiation and proliferation of epithelial tissue; may also have anti-inflammatory and immunomodulatory properties. Ensure skin is dry before applying gel

Anthelmintics

Filaria can cause lymphedema by obstruction.

Albendazole (Albenza, Valbazen)

A benzimidazole carbamate drug that inhibits tubulin polymerization, resulting in degeneration of cytoplasmic microtubules. Decreases ATP production in worm, causing energy depletion, immobilization, and, finally, death. Converted in the liver to its primary metabolite, albendazole sulfoxide. Less than 1% of the primary metabolite excreted in urine. Plasma level is noted to rise significantly (as much as 5-fold) when ingested after high-fat meal. Experience with patients <6 y limited. To avoid inflammatory response in CNS, patient must also be started on anticonvulsants and high-dose glucocorticoids.

Emollients/keratolytics

Ammonium lactate lotion (AmLactin, AmLactin XL, Lac-Hydrin)

Alpha-hydroxy acid; normal constituent of tissues and blood. Believed to act as humectant when applied to skin. This may influence hydration of the stratum corneum. In addition, when applied to skin, may act to decrease corneocyte cohesion. The mechanism by which this is accomplished is not yet known. Used to decrease scaling and pruritus. Found in a variety of topical emollient lotions. Ammonium lactate 5% lotion is available over the counter, and lactic acid 12% cream and lotion are available by prescription.

Urea, topical (Carmol, Keralac, Ureacin)

Promotes hydration and removal of excess keratin in conditions of hyperkeratosis

Indications

Surgical treatment is palliative, not curative, and it does not obviate the need for continued medical therapy. Moreover, it is rarely indicated as the primary treatment modality. Rather, reserve surgical treatment for those who do not improve with conservative measures or in cases where the extremity is so large that it impairs daily activities and prevents successful conservative management.

Relevant Anatomy

Before embarking on the treatment of lymphedema, a thorough knowledge of the relevant anatomy is essential. Blind-ended lymphatic capillaries arise within the interstitial spaces of the dermal papillae. These unvalved superficial dermal lymphatics drain into interconnected subdermal channels, which parallel the superficial venous system. These subsequently drain into the deeper, epifascial system of valved trunks lined with smooth muscle cells and located just above the deep fascia of the extremity. This system is responsible for the drainage of lymph from the skin and subcutaneous tissues. Valves provide for unidirectional flow towards regional lymph nodes and eventually the venous circulation in the neck. Flow is achieved by variations of tissue pressure through skeletal muscle contractions, pulsatile blood flow, and contractions of the spiral smooth muscle fibers surrounding larger lymphatic channels.

A deeper valved subfascial system of lymphatics is responsible for the drainage of lymph from the fascia, muscles, joints, ligaments, periosteum, and bone. This subfascial system parallels the deep venous system of the extremity. The epifascial and subfascial systems normally function independently, although valved connections do exist in the popliteal, inguinal, antecubital, and axillary regions where lymph nodes form interconnected chains. These connections probably do not function under normal conditions; however, in lymphedema, some reversed flow through perforators from the epifascial to the subfascial system may occur as a mechanism of decompression of the epifascial system. In lymphedema, the derangement is almost always exclusive to the epifascial lymphatic system, with the

subfascial system being uninvolved. This is the basis for the surgical approaches to lymphedema, which focus on the epifascial system.

Contraindications

Contraindications to intermittent pneumatic pump compression therapy include congestive heart failure, deep vein thrombosis, and active infection.

Surgical Therapy

Surgical treatment is palliative, not curative, and it does not obviate the need for continued medical therapy. Moreover, it is rarely indicated as the primary treatment modality. Rather, reserve surgical treatment for those who do not improve with conservative measures or in cases where the extremity is so large that it impairs daily activities and prevents successful conservative management. The goals of surgical therapy are volume reduction to improve function, facilitation of conservative therapy, and prevention of complications. A myriad of surgical procedures have been advocated, reflecting a lack of clear superiority of one procedure over the others. In general, surgical procedures are classified as physiologic or excisional.

Physiologic procedures attempt to improve lymphatic drainage. Multiple techniques have been described, including omental transposition, buried dermal flaps, enteromesenteric bridging, lymphangioplasty, and microvascular lympholymphatic or lymphovenous anastomoses.⁸ None of these techniques has clearly documented favorable long-term results. Further evaluation is necessary. Moreover, many of these physiologic techniques also include an excisional component, making it difficult to distinguish between the 2 approaches.

Excisional techniques remove the affected tissues, thus reducing the lymphedema load. Some authors advocate suction-assisted removal of subcutaneous tissues, but this technique is difficult because of the extensive subcutaneous fibrosis that is present. Additionally, this approach does not reduce the skin envelope, and the lymphedema often rapidly recurs. Suction-assisted removal of subcutaneous tissue followed by excision of the excess skin envelope has no clear advantage over direct excisional techniques alone.

The Charles procedure is another quite radical excisional technique. This procedure involves the total excision of all skin and subcutaneous tissue from the affected extremity. The underlying fascia is then grafted, using the skin that has been excised. This technique is extreme and is reserved for only the most severe cases. Complications include ulceration,

hyperkeratosis, keloid formation, hyperpigmentation, weeping dermatitis, and severe cosmetic deformity.

Van der Walt et al developed a modified Charles procedure in which negative-pressure dressing was employed following debulking surgery, with skin grafting delayed for 5-7 days.⁹ In a report on 8 patients suffering from severe primary lymphedema who underwent the procedure, the authors reported that the patients experienced no major complications. Minor complications, including operative blood loss and, in 3 patients, the need for additional grafting, did occur.

Staged excision has become the option of choice for many authors. This procedure involves removing only a portion of skin and subcutaneous tissue, followed by primary closure. After approximately 3 months, the procedure is repeated on a different area of the extremity. This procedure is safe, reliable, and demonstrates the most consistent improvement with the lowest incidence of complications.

Preoperative Details

Prior to surgery, appropriate documentation is necessary to evaluate the outcome of treatment. This includes photographic documentation as well as extremity measurements. Ideally, these measurements are of limb volume by water displacement, although some rely on circumferential measurements alone. Obtain measurements and photographs at the same time of day each time, document affected extremities and contralateral extremities, and preferably conduct documentation in the morning after extremity elevation in bed overnight.

Institute strict elevation and pneumatic compression, if available, 24-72 hours prior to surgery. This allows maximum excision to be performed. The extremity must also be free of infection at the time of surgery, and a single dose of preoperative intravenous antibiotic is administered.

Intraoperative Details

After the establishment of appropriate anesthesia, the operative field is sterilized and draped according to surgeon preference.

A pneumatic tourniquet is placed at the root of the extremity and insufflated after the extremity has been exsanguinated.

A longitudinal incision is made along the entire extremity, and skin flaps, 1.0-1.5 cm

thick, are elevated.

Subcutaneous tissue is then excised, taking care not to injure peripheral sensory nerves.

Some authors also excise a strip of deep fascia, but this should not be performed around joints because it may cause instability.

Once the subcutaneous excision is complete, redundant skin is resected. Often, a strip that is 5-10 cm wide may be removed.

The wound is closed over suction drains.

Postoperative Details

Postoperatively, the extremity is immobilized in a splint and elevated while the patient is placed on strict bed rest.

Antibiotics may be continued until drain removal, according to surgeon preference.

Drains are typically removed at 5-7 days postoperatively, as dictated by a decrease in drain output.

Sutures are removed at 10-14 days and replaced by Steri-Strips.

Measure the patient for a new compression garment when the new dimensions of the extremity have stabilized.

After approximately 10 days, the patient may gradually begin dependency on the extremity with compression bandages or an elastic garment in place.

Follow-up

Once discharged from the hospital, the patient should be seen regularly in the outpatient clinic.

Patients must wear compression garments for 4-6 weeks continuously, and dependency on the involved extremity may be gradually increased at the discretion of the treating physician.

Once healed to physician satisfaction, the patient may return to a normal routine of elevation at night and compression garment therapy during the day.

Follow-up visits should include documentation of circumferential measurement or water displacement of the affected and contralateral extremities as well as photographic documentation.

When staging procedures, allow approximately 3 months between procedures to allow complete healing of the initial operative site.

Complications

Patients with chronic lymphedema for 10 years have a 10% risk of developing lymphangiosarcoma, the most dreaded complication of this disease. Patients with this tumor commonly present with a reddish purple discoloration or nodule that tends to form satellite lesions. It may be confused with Kaposi sarcoma or traumatic ecchymosis. This tumor is highly aggressive, requires radical amputation of the involved extremity, and has a very poor prognosis. The 5-year survival rate is less than 10%, and the average survival following diagnosis is 19 months. This malignant degeneration is most commonly observed in patients with postmastectomy lymphedema (Stewart-Treves syndrome), where incidence is estimated to be 0.5%.

Other complications of lymphedema include recurrent bouts of cellulitis and/or lymphangitis, deep venous thrombosis, severe functional impairment, cosmetic embarrassment, and necessary amputation. Complications following surgery are common and include partial wound separation, seroma, hematoma, skin necrosis, and exacerbation of foot or hand edema.

Outcome and Prognosis

At present, no cure for lymphedema exists. Surgery is palliative at best, and it may be a part of the lifelong therapy patients must endure to manage this disease.

Future and Controversies

A myriad of surgical procedures have been advocated, reflecting a lack of clear superiority of one procedure over the others. Multiple physiological and excisional techniques have been described. None of the physiological techniques has clearly documented favorable long-term results; further evaluation is necessary. Moreover, many of the physiologic techniques also include an excisional component, making it difficult to distinguish between the 2 approaches.

DEEP VEIN THROMBOSIS

Introduction of acute deep venous thrombosis

Deep venous thrombosis (DVT) most commonly involves the deep veins of the leg or arm, often resulting in potentially life-threatening emboli to the lungs or debilitating venous alular dysfunction and chronic leg swelling. Deep venous thrombosis (DVT) is also one of the most prevalent medical problems today, with an annual incidence of 117 cases per 100,000. Each year in the United States, more than 200,000 people develop venous thrombosis; of those, 50,000 cases are complicated by pulmonary embolism.¹ Early recognition and appropriate treatment of deep venous thrombosis (DVT) and its complications can save many lives.

Pathophysiology

Over a century ago, Rudolf Virchow described 3 factors that are critically important in the development of venous thrombosis: (1) venous stasis, (2) activation of blood coagulation, and (3) vein damage. Over time, refinements have been made in their description and importance to the development of venous thrombosis. The origin of venous thrombosis is frequently multifactorial, with components of the triad of variable importance in individual patients.

Studies have shown that low flow sites, such as the soleal sinuses, behind venous valve pockets, and at venous confluences, are at most risk for the development of venous thrombi. However, stasis alone is not enough to facilitate the development of venous thrombosis. Experimental ligation of rabbit jugular veins for periods of up to 60 minutes have failed to consistently cause venous thrombosis. Although, patients that are immobilized for long periods of time seem to be at high risk for the development of venous thrombosis, an additional stimulus is required to develop deep venous thrombosis (DVTs).

Mechanical injury to the vein wall appears to provide an added stimulus for venous thrombosis. Hip arthroplasty patients with the associated femoral vein manipulation represent a high-risk group that cannot be explained by just immobilization, with 57% of thrombi originating in the affected femoral vein rather than the usual site of stasis in the calf.⁶ Endothelial injury can convert the normally antithrombogenic endothelium to become prothrombotic by stimulating the production of tissue factor, von Willebrand factor, and fibronectin.

Genetic mutations within the blood's coagulation cascade represent those at highest risk for the development of venous thrombosis (See Table 1).

Table 1. Relative Risk for Venous Thrombosis

Thrombophilic Defect	Relative Risk
Antithrombin deficiency	8-10
Protein C deficiency	7-10
Protein S deficiency	8-10
Factor V Leiden	3-7
Hyperhomocysteinemia	2.5
Anticardiolipin antibodies	3.2

Primary deficiencies of coagulation inhibitors antithrombin, protein C, and protein S are associated with 5-10% of all thrombotic events. Resistance of procoagulant factors to an intact anticoagulation system has also recently been described with the recognition of factor V Leiden mutation, representing 10-65% of patients with deep venous thrombosis (DVT).⁸ In the setting of venous stasis, these factors are allowed to accumulate in thrombosis prone sites, where mechanical vessel injury has occurred, stimulating the endothelium to become prothrombotic.

Components of the Virchow triad are of variable importance in individual patients, but the end result is early thrombus interaction with the endothelium. This interaction stimulates local cytokine production and facilitates leukocyte adhesion to the endothelium, both of which promote venous thrombosis. Depending on the relative balance between activated coagulation and thrombolysis, thrombus propagation occurs.

Over time, thrombus organization begins with the infiltration of inflammatory cells into the clot. This results in a fibroelastic intimal thickening at the site of thrombus attachment in most patients and a fibrous synechia in up to 11%. In many patients, this interaction between vessel wall and thrombus leads to alular dysfunction and overall vein wall fibrosis. Histological examination of vein wall remodeling after venous thrombosis has demonstrated an imbalance in connective tissue matrix regulation and a loss of regulatory venous contractility that contributes to the development of chronic venous insufficiency.

Risk factors

Many factors have been identified as known risk factors for the development of venous thrombosis. The single most powerful risk marker remains a prior history of DVT with up to 25% of acute venous thrombosis occurring in such patients. Pathologically, remnants of previous thrombi are often seen within the specimens of new acute thrombi. However, recurrent thrombosis may actually be the result of primary hypercoagulable states. Abnormalities within the coagulation cascade are the direct result of discrete genetic mutations within the coagulation cascade. Deficiencies of protein C, protein S, or antithrombin III account for approximately 5-10% of all cases of deep venous thrombosis (DVT).

Age has been well studied as an independent risk factor for venous thrombosis development. Although a 30-fold increase in incidence is noted from age 30 to age 80, the effect appears to be multifactorial, with more thrombogenic risk factors occurring in the elderly than in those younger than 40 years. Venous stasis, as seen in immobilized patients and paralyzed limbs, also contributes to the development of venous thrombosis. Autopsy studies parallel the duration of bed rest to the incidence of venous thrombosis, with 15% of patients in those studies dying within 7 days of bedrest to greater than 80% in those dying after 12 weeks.² Within stroke patients, deep venous thrombosis (DVT) is found in 53% of paralyzed limbs, compared with only 7% on the nonaffected side.

Malignancy is noted in up to 30% of patients with venous thrombosis. The thrombogenic mechanisms involve abnormal coagulation, as evidenced by 90% of cancer patients having some abnormal coagulation factors.¹⁸ Chemotherapy may increase the risk of venous thrombosis by affecting the vascular endothelium, coagulation cascades, and tumor cell lysis. The incidence has been shown to increase in those patients undergoing longer courses of therapy for breast cancer, from 4.9% for 12 weeks of treatment to 8.8% for 36 weeks. Additionally, deep venous thrombosis (DVT) complicates 29% of surgical procedures done for malignancy.

Postoperative venous thrombosis varies depending on a multitude of patient factors, including the type of surgery undertaken. Without prophylaxis, general surgery operations typically have an incidence of deep venous thrombosis (DVT) around 20%, while orthopedic hip surgery can occur in up to 50% of patients. Based on radioactive labeled fibrinogen, about half of lower extremity thrombi develop intraoperatively. Perioperative immobilization,

coagulation abnormalities, and venous injury all contribute to the development of surgical venous thrombosis.

Other clinical settings commonly reported as risk factors have also been identified and are shown in Table 2, 3

Table 2. Risk Factors for Venous Thromboembolic Disease

Risk Factor	Odds Ratio
Hospitalization with recent surgery	21.72
Hospitalization without surgery	7.98
Trauma	12.69
Malignancy	6.53
Central venous catheter	5.55
Neurologic disease with paralysis	3.04

Table 3. Risk Factors for Venous Thromboembolism

Acquired	Inherited
Advanced age	Factor V Leiden
Hospitalization/immobilization	Prothrombin 20210A
Hormone replacement therapy and oral contraceptive use	Antithrombin deficiency
	Protein C deficiency
Pregnancy and puerperium	Protein S deficiency
Prior venous thromboembolism	Factor XI elevation
Malignancy	Dysfibrinogenemia
Major surgery	Mixed Etiology
Obesity	Homocysteinemia
Nephrotic syndrome	Factor VII, VIII, IX, XI elevation
Trauma or spinal cord injury	Hyperfibrinogenemia
Long-haul travel (>6 h)	Activated protein C resistance without factor V Leiden
Varicose veins	
Antiphospholipid antibody syndrome	
Myeloproliferative disease	
Polycythemia	

Clinical and diagnostic evaluation

The clinical diagnosis of deep venous thrombosis (DVT) is difficult and fraught with uncertainty. The classic signs and symptoms of deep venous thrombosis (DVT) are those associated with obstruction to venous drainage and include pain, tenderness, and unilateral leg swelling. Other associated nonspecific findings are warmth, erythema, a palpable cord, and pain upon passive dorsiflexion of the foot (Homan sign). However, even with patients with classic symptoms, up to 46% have negative venograms. Furthermore, up to 50% of those with image-documented venous thrombosis lack any specific symptom. Deep venous thrombosis (DVT) simply cannot be diagnosed or excluded based on clinical findings; thus, diagnostic tests must be performed whenever the diagnosis of deep venous thrombosis (DVT) is being considered.

When a patient has deep venous thrombosis (DVT), symptoms may be present or absent, unilateral or bilateral, or mild or severe. Thrombus that does not cause a net venous outflow obstruction is often asymptomatic. Thrombus that involves the iliac bifurcation, the pelvic veins, or the vena cava produces leg edema that is usually bilateral rather than unilateral. High partial obstruction often produces mild bilateral edema that is mistaken for the dependent edema of right-sided heart failure, fluid overload, or hepatic or renal insufficiency.

Severe venous congestion produces a clinical appearance that can be indistinguishable from the appearance of cellulitis. Patients with a warm, swollen, tender leg should be evaluated for both cellulitis and deep venous thrombosis (DVT) because patients with primary deep venous thrombosis (DVT) often develop a secondary cellulitis, while patients with primary cellulitis often develop a secondary deep venous thrombosis (DVT). Superficial thrombophlebitis, likewise, is often associated with a clinically inapparent underlying DVT.

If a patient is thought to have pulmonary embolism (PE) or has documented PE, the absence of tenderness, erythema, edema, or a palpable cord upon examination of the lower extremities does not rule out thrombophlebitis, nor does it imply a source other than a leg vein. More than two thirds of patients with proven PE lack any clinically evident phlebitis. Nearly one third of patients with proven PE have no identifiable source of deep venous thrombosis (DVT), despite a thorough investigation. Autopsy studies suggest that even when the source is

clinically inapparent, it lies undetected within the deep venous system of the lower extremity and pelvis in 90% of cases.

Vascular Lab and Radiologic Evaluation

Duplex Ultrasound

DUS is now the most commonly performed test for the detection of infrainguinal DVT, both above and below the knee, and has a sensitivity and specificity of >95% in symptomatic patients. DUS combines real-time B-mode ultrasound with pulsed Doppler capability. Color flow imaging is useful in more technically difficult examinations, such as in the evaluation of possible calf vein DVT. This combination offers the ability to noninvasively visualize the venous anatomy, detect occluded and partially occluded venous segments, and demonstrate physiologic flow characteristics using a mobile self-contained device.

In the supine patient, normal lower extremity venous flow is phasic (Fig. 1), decreasing with inspiration in response to increased intra-abdominal pressure with the descent of the diaphragm and then increasing with expiration. When the patient is upright, the decrease in intra-abdominal pressure with expiration cannot overcome the hydrostatic column of pressure existing between the right atrium and the calf. Muscular contractions of the calf, along with the one-way venous valves, are then required to promote venous return to the heart. Flow also can be increased by leg elevation or compression and decreased by sudden elevation of intra-abdominal pressure (Valsalva's maneuver). In a venous DUS examination performed with the patient supine, spontaneous flow, variation of flow with respiration, and response of flow to Valsalva's maneuver are all assessed. However, the primary method of detecting DVT with ultrasound is demonstration of the lack of compressibility of the vein with probe pressure on B-mode imaging. Normally, in transverse section, the vein walls should coapt with pressure. Lack of coaptation indicates thrombus.

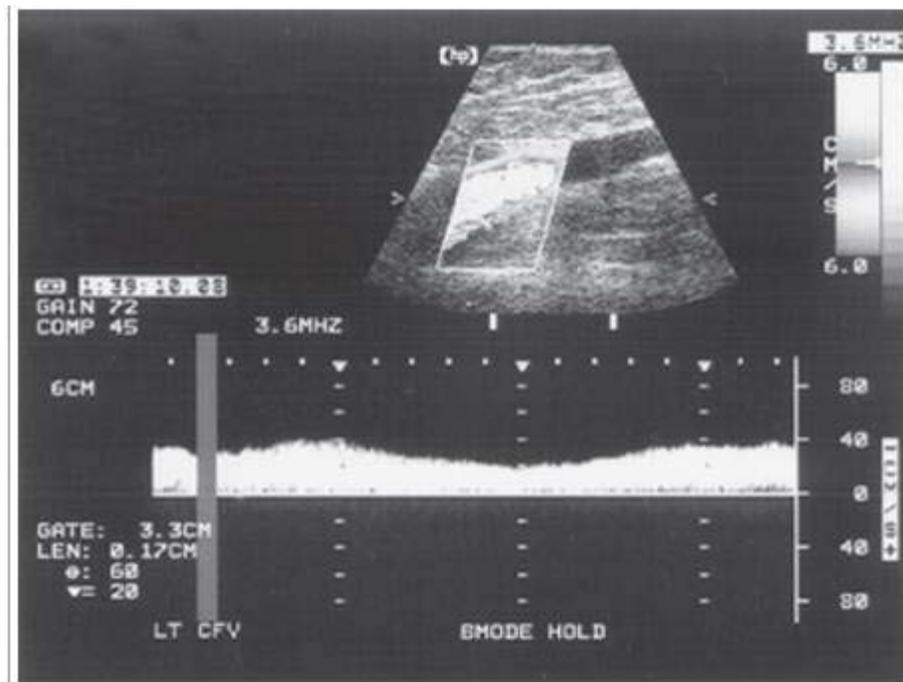


Fig. 1. Duplex ultrasound scan of a normal femoral vein with phasic flow signals.

The examination begins at the ankle and continues proximally to the groin. Each vein is visualized, and the flow signal is assessed with distal and proximal compression. Lower extremity DVT can be diagnosed by any of the following DUS findings: lack of spontaneous flow (Fig. 2), inability to compress the vein (Fig. 3), absence of color filling of the lumen by color flow DUS, loss of respiratory flow variation, and venous distention. Again, lack of venous compression on B-mode imaging is the primary diagnostic variable. Several studies comparing B-mode ultrasound to venography for the detection of femoropopliteal DVT in patients clinically suspected to have DVT report sensitivities of >91% and specificities of >97%. The ability of DUS to assess isolated calf vein DVT varies greatly, with sensitivities ranging from 50 to 93% and specificities approaching 100%.

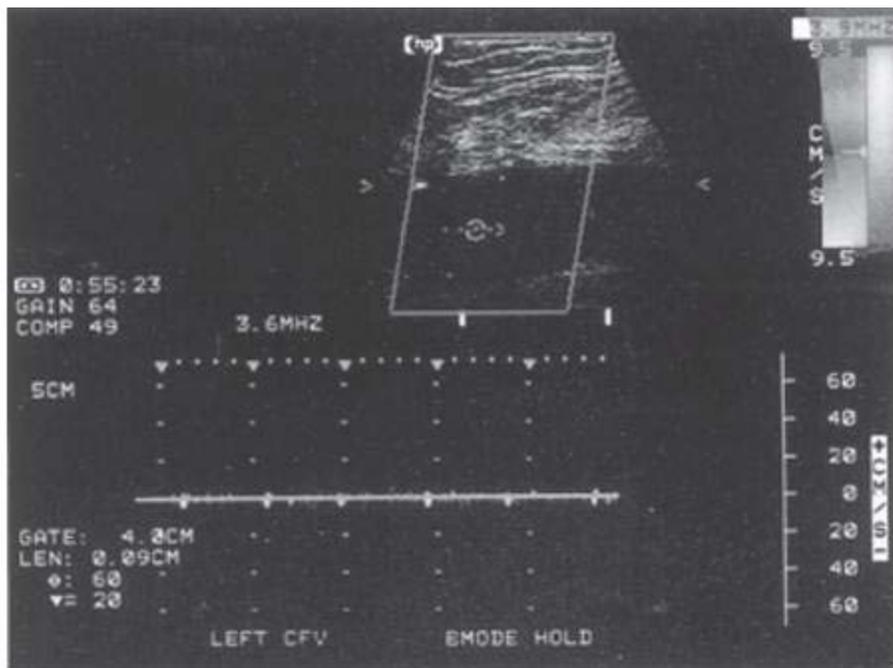


Fig. 2. Duplex ultrasound of a femoral vein containing thrombus demonstrating no flow within the femoral vein

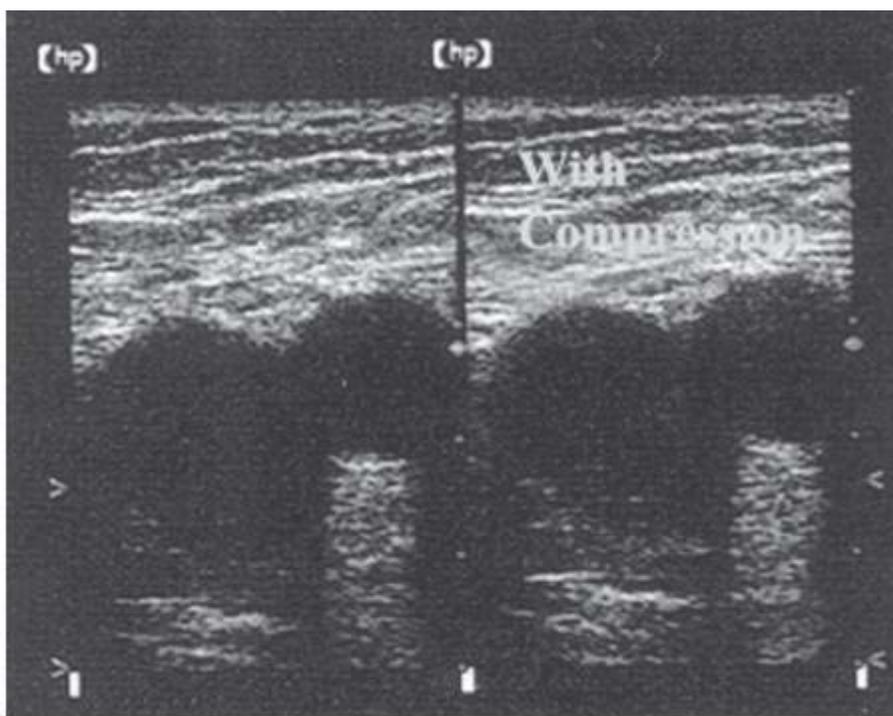


Fig. 3. B-mode ultrasound of the femoral vein in cross-section. The femoral vein does not collapse with external compression

Impedance Plethysmography

Impedance plethysmography (IPG) was the primary noninvasive method of diagnosing DVT before the widespread use of DUS but is infrequently used today. IPG is based on the principle that resistance to the flow of electricity between two electrodes, or electrical impedance, occurs as the volume of the extremity changes in response to blood flow. Two pairs of electrodes containing aluminum strips are placed circumferentially around the leg approximately 10 cm apart and a low-level current is delivered to the two outer electrodes. A pneumatic cuff is inflated over the thigh for venous outflow obstruction and then rapidly deflated. Changes in electrical resistance resulting from lower extremity blood volume changes are quantified. IPG is less accurate than DUS for the detection of proximal DVT, with an 83% sensitivity in symptomatic patients. It is a poor detector of calf vein DVT.

Iodine 125 Fibrinogen Uptake

Iodine 125 fibrinogen uptake (FUT) is a seldom used technique that involves IV administration of radioactive fibrinogen and monitoring for increased uptake in fibrin clots. An increase of 20% or more in one area of a limb indicates an area of thrombus.²³ FUT can detect DVT in the calf, but high background radiation from the pelvis and the urinary tract limits its ability to detect proximal DVT. It also cannot be used in an extremity that has recently undergone surgery or has active inflammation. In a prospective study, FUT had a sensitivity of 73% and specificity of 71% for identification of DVT in a group of symptomatic and asymptomatic patients. Currently, FUT is primarily a research tool of historic interest.

Venography

Venography is the most definitive test for the diagnosis of DVT in both symptomatic and asymptomatic patients. It is the gold standard to which other modalities are compared. This procedure involves placement of a small catheter in the dorsum of the foot and injection of a radiopaque contrast agent. Radiographs are obtained in at least two projections. A positive study result is failure to fill the deep system with passage of the contrast medium into the superficial system or demonstration of discrete filling defects (Fig. 4). A normal study result virtually excludes the presence of DVT. In a study of 160 patients with a normal venogram followed for 3 months, only two patients (1.3%) subsequently developed DVT and no patients

experienced symptoms of PE.

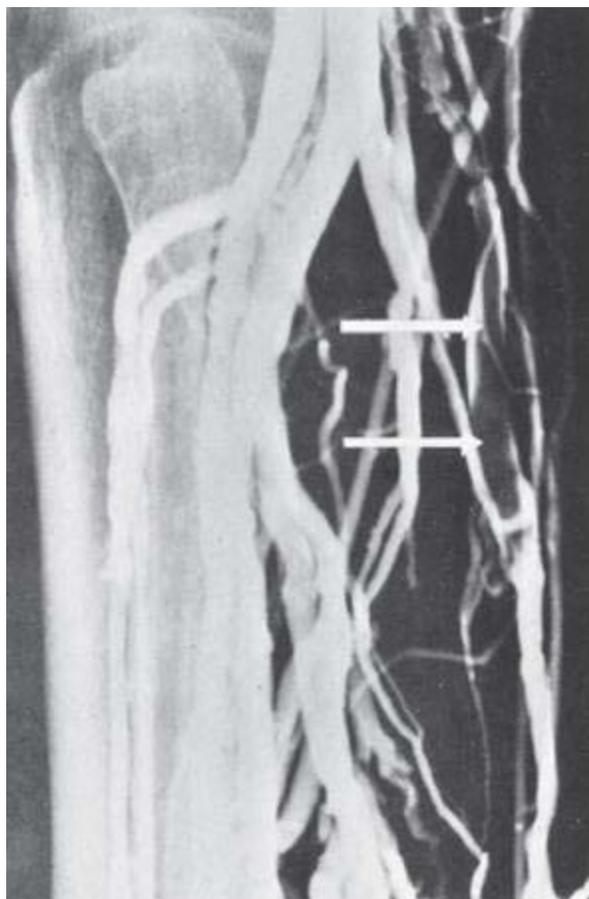


Fig. 4. Venogram showing a filling defect in the popliteal vein (*arrows*).

Venography is not routinely used for the evaluation of lower extremity DVT because of the associated complications discussed previously. Currently, venography is reserved for imaging before operative venous reconstruction and catheter-based therapy. It does, however, remain the procedure of choice in research studies evaluating methods of prophylaxis for DVT.

Laboratory analysis has also been used in aiding the diagnosis of venous thrombosis. D-dimers are degradation products of cross-linked fibrin by plasmin that are detected by diagnostic assays. Although highly sensitive, up to 97%, elevated levels are not specific with rates as low as 35%.²⁷ Many other clinical situations can result in elevated D-dimer levels, including infection, trauma, postoperative states, and malignancy.²⁸ Additional blood work should include coagulation studies to evaluate for a hypercoagulable state, if clinically indicated. A prolonged prothrombin time or activated partial thromboplastin time does not imply a lower risk of new thrombosis. Progression of deep vein thrombosis (DVT) and PE can occur despite full therapeutic anticoagulation in 13% of patients.

Treatment

Once the diagnosis of VTE has been made, antithrombotic therapy should be initiated promptly. If clinical suspicion for VTE is high, it may be prudent to start treatment while the diagnosis is being objectively confirmed. The theoretic goals of VTE treatment are the prevention of mortality and morbidity associated with PE and the prevention of the postphlebotic syndrome. However, the only proven benefit of anticoagulant treatment for DVT is the prevention of death from PE. Treatment regimens may include antithrombotic therapy, vena caval interruption, catheter-directed or systemic thrombolytic therapy, and operative thrombectomy.

Antithrombotic Therapy

Antithrombotic therapy may be initiated with IV or SC unfractionated heparin, SC low molecular weight heparin, or SC fondaparinux (a synthetic pentasaccharide). This initial therapy usually is continued for at least 5 days, while oral vitamin K antagonists are being simultaneously administered. The initial therapy typically is discontinued when the international normalized ratio (INR) is ≥ 2.0 for 24 hours.

Unfractionated heparin (UFH) binds to antithrombin via a specific 18-saccharide sequence, which increases its activity over 1000-fold. This antithrombin-heparin complex primarily inhibits factor IIa (thrombin) and factor Xa and, to a lesser degree, factors IXa, XIa, and XIIa. In addition, UFH also binds to tissue factor pathway inhibitor, which inhibits the conversion of factor X to Xa, and factor IX to IXa. Finally, UFH catalyzes the inhibition of thrombin by heparin cofactor II via a mechanism that is independent of antithrombin.

UFH therapy is most commonly administered with an initial IV bolus of 80 units/kg or 5000 units. Weight-based UFH dosages have been shown to be more effective than standard fixed boluses in rapidly achieving therapeutic levels. The initial bolus is followed by a continuous IV drip, initially at 18 units/kg per hour or 1300 units per hour. The half-life of IV UFH ranges from 45 to 90 minutes and is dose dependent. The level of antithrombotic therapy should be monitored every 6 hours using the activated partial thromboplastin time (aPTT), with the goal range of 1.5 to 2.5 times control values. This should correspond with plasma heparin anti-Xa activity levels of 0.3 to 0.7 IU/mL.

Initial anticoagulation with UFH may be administered SC, although this route is less commonly used. Adjusted-dose therapeutic SC UFH is initiated with 17,500 units, followed by

250 units/kg twice daily, and dosing is adjusted to an aPTT goal range similar to that for IV UFH. Fixed-dose unmonitored SC UFH is started with a bolus of 333 units/kg, followed by 250 units/kg twice daily.

Hemorrhage is the primary complication of UFH therapy. The rate of major hemorrhage (fatal, intracranial, retroperitoneal, or requiring transfusion of >2 units of packed red blood cells) is approximately 5% in hospitalized patients undergoing UFH therapy (1% in medical patients and 8% in surgical patients).²⁷ For patients with UFH-related bleeding complications, cessation of UFH is required, and anticoagulation may be reversed with protamine sulfate. Protamine sulfate binds to UFH and forms an inactive salt compound. Each milligram of protamine neutralizes 90 to 115 units of heparin, and the dosage should not exceed 50 mg IV over any 10-minute period. Side effects of protamine sulfate include hypotension, pulmonary edema, and anaphylaxis. Patients with prior exposure to protamine-containing insulin (NPH) and patients with allergy to fish may have an increased risk of hypersensitivity, although no direct relationship has been established. The protamine infusion should be terminated if any side effects occur.

In addition to hemorrhage, heparin also has unique complications. Heparin-induced thrombocytopenia (HIT) results from heparin-associated antiplatelet antibodies (HAAs) directed against platelet factor 4 complexed with heparin. HIT occurs in 1 to 5% of patients being treated with heparin. In patients with repeat heparin exposure (such as vascular surgery patients), the incidence of HAAs may be as high as 21%. HIT occurs most frequently in the second week of therapy and may lead to disastrous venous or arterial thrombotic complications. Therefore, platelet counts should be monitored periodically in patients receiving continuous heparin therapy. All forms of heparin should be stopped if there is a high clinical suspicion or confirmation of HIT [usually accompanied by an unexplained thrombocytopenia (<100,000/

L) or platelet count decrease of 30 to 50%]. Fortunately, direct thrombin inhibitors (recombinant hirudin, argatroban, bivalirudin) now are available as alternative antithrombotic agents (see later). Another complication of prolonged high-dose heparin therapy is osteopenia, which results from impairment of bone formation and enhancement of bone resorption by heparin.

Low molecular weight heparins (LMWHs) are derived from the depolymerization of porcine UFH. Like UFH, LMWHs bind to antithrombin via a specific pentasaccharide sequence to expose an active site for the neutralization of factor Xa. However, LMWHs lack the sufficient number of additional saccharide units (18 or more), which results in less

inactivation of thrombin (factor IIa). In comparison to UFH, LMWHs have increased bioavailability (>90% after SC injection), longer half-lives (approximately 4 to 6 hours), and more predictable elimination rates. Weight-based once- or twice-daily SC LMWH injections, for which no monitoring is needed, provide a distinct advantage over continuous IV infusions of UFH for treatment of VTE.

Most patients who receive therapeutic LMWH do not require monitoring. Patients who do require monitoring include those with significant renal insufficiency or failure, pediatric patients, obese patients of >120 kg, and patients who are pregnant. Monitoring may be performed using anti-Xa activity assays. However, the therapeutic anti-Xa goal range will depend on the type of LMWH and the frequency of dosing. Numerous LMWHs are commercially available. The various preparations differ in their anti-Xa and anti-IIa activities, and the treatment dosing for one LMWH cannot be extrapolated for use with another. The action of LMWHs may be partially reversed (approximately 60%) with protamine sulfate.

Numerous well-designed trials comparing SC LMWH with IV and SC UFH for the treatment of DVT have been critically evaluated in several meta-analyses. The more recent studies demonstrate a decrease in thrombotic complications, bleeding, and mortality with LMWHs. LMWHs also are associated with a decreased rate of HAAb formation and HIT (<2%) compared with UFH (at least in prophylactic doses). However, patients with established HIT should not subsequently receive LMWHs due to significant rates of cross reactivity. A major benefit of LMWHs is the ability to treat patients with VTE as outpatients. In a randomized study comparing IV UFH and the LMWH nadroparin calcium, there was no significant difference in recurrent thromboembolism (8.6% for UFH vs. 6.9% for LMWH) or major bleeding complications (2.0% for UFH vs. 0.5% for LMWH). There was a 67% reduction in mean days in the hospital for the LMWH group.

A patient with VTE should meet several criteria before receiving outpatient LMWH therapy. First, the patient should not require hospitalization for any associated conditions. The patient should not require monitoring of the LMWH therapy (which is necessary in patients with severe renal insufficiency, pediatric patients, obese patients, and pregnant patients). The patient should be hemodynamically stable with a low suspicion of PE and have a low bleeding risk. An established outpatient system to administer LMWH and warfarin, as well as to monitor for recurrent VTE and bleeding complications, should be present. In addition, the patient's symptoms of pain and edema should be controllable at home.

Fondaparinux currently is the only synthetic pentasaccharide that has been approved by the U.S. Food and Drug Administration (FDA) for the initial treatment of DVT and PE. Its

five-polysaccharide sequence binds and activates antithrombin, causing specific inhibition of factor Xa. In two large noninferiority trials, fondaparinux was compared with the LMWH enoxaparin for the initial treatment of DVT and with IV UFH for the initial treatment of PE. The rates of recurrent VTE ranged from 3.8 to 5%, with rates of major bleeding of 2 to 2.6%, for all treatment arms. The drug is administered SC once daily with a weight-based dosing protocol: 5 mg, 7.5 mg, or 10 mg for patients weighing <50 kg, 50 to 100 kg, or >100 kg, respectively. The half-life of fondaparinux is approximately 17 hours in patients with normal renal function. There are rare case reports of fondaparinux-induced thrombocytopenia.

Direct-thrombin inhibitors (DTIs) include recombinant hirudin, argatroban, and bivalirudin. These antithrombotic agents bind to thrombin, inhibiting the conversion of fibrinogen to fibrin as well as thrombin-induced platelet activation. These actions are independent of antithrombin. The direct thrombin inhibitors should be reserved for (a) patients in whom there is a high clinical suspicion or confirmation of HIT, and (b) patients who have a history of HIT or test positive for heparin-associated antibodies. In patients with established HIT, DTIs should be administered for at least 7 days, or until the platelet count normalizes. Warfarin may then be introduced slowly, overlapping therapy with a DTI for at least 5 days.⁴¹ Because bivalirudin is approved primarily for patients with or without HIT who undergo percutaneous coronary intervention, it is not discussed here in further detail.

Commercially available hirudin is manufactured using recombinant DNA technology. It is indicated for the prophylaxis and treatment of patients with HIT. In patients with normal renal function, recombinant hirudin is administered in an IV bolus dose of 0.4 mg/kg, followed by a continuous IV infusion of 0.15 mg/kg per hour. The half-life ranges from 30 to 60 minutes. The aPTT is monitored, starting approximately 4 hours after initiation of therapy, and dosage is adjusted to maintain an aPTT of 1.5 to 2.5 times the laboratory normal value. The less commonly used ecarin clotting time is an alternative method of monitoring. Because recombinant hirudin is eliminated via renal excretion, significant dosage adjustments are required in patients with renal insufficiency.

Argatroban is indicated for the prophylaxis and treatment of thrombosis in HIT. It also is approved for patients with, or at risk for, HIT who undergo percutaneous coronary intervention. Antithrombotic prophylaxis and therapy are initiated with a continuous IV infusion of 2 g/kg per minute, without the need for a bolus. The half-life ranges from 39 to 51 minutes, and the dosage is adjusted to maintain an aPTT of 1.5 to 3 times normal. Large initial boluses and higher rates of continuous infusion are reserved for patients with coronary artery thrombosis and myocardial infarction. In these patients, therapy is monitored using the activated clotting

time. Argatroban is metabolized by the liver, and the majority is excreted via the biliary tract. Significant dosage adjustments are needed in patients with hepatic impairment. There is no reversal agent for argatroban.

Vitamin K antagonists, which include warfarin and other coumarin derivatives, are the mainstay of long-term antithrombotic therapy in patients with VTE. Warfarin inhibits the γ -carboxylation of vitamin K–dependent procoagulants (factors II, VII, IX, X) and anticoagulants (proteins C and S), which results in the formation of less functional proteins. Warfarin usually requires several days to achieve its full effect, because normal circulating coagulation proteins must first undergo their normal degradation. Factors X and II have the longest half-lives, in the range of 36 and 72 hours, respectively. In addition, the steady-state concentration of warfarin is usually not reached for 4 to 5 days.

Warfarin therapy usually is monitored by measuring the INR, calculated using the following equation:

$$\text{INR} = \left(\frac{\text{patient prothrombin time}}{\text{laboratory normal prothrombin time}} \right)^{\text{ISI}}$$

where *ISI* is the international sensitivity index. The ISI describes the strength of the thromboplastin that is added to activate the extrinsic coagulation pathway. The therapeutic target INR range is usually 2.0 to 3.0, but the response to warfarin is variable and depends on liver function, diet, age, and concomitant medications. In patients receiving anticoagulation therapy without concomitant thrombolysis or venous thrombectomy, the vitamin K antagonist may be started on the same day as the initial parenteral anticoagulant, usually at doses ranging from 5 to 10 mg. Smaller initial doses may be needed in older and malnourished patients, in those with liver disease or congestive heart failure, and in those who have recently undergone major surgery.

The recommended duration of warfarin antithrombotic therapy is increasingly being stratified based on whether the DVT was provoked or unprovoked, whether it was the first or a recurrent episode, where the DVT is located, and whether malignancy is present. Current American College of Chest Physicians (ACCP) recommendations for duration of warfarin therapy are summarized in Table 24-3. In patients with proximal DVT, several randomized clinical trials have demonstrated that shorter-term antithrombotic therapy (4 to 6 weeks) is associated with a higher rate of recurrence than 3 to 6 months of anticoagulation. In these trials, most of the patients with transient risk factors had a low rate of recurrent VTE; most of

the recurrences were in patients with continuing risk factors. These studies support the ACCP recommendation that 3 months of anticoagulation is sufficient to prevent recurrent VTE in patients whose DVT occurred around the time of a transient risk factor (e.g., hospitalization, orthopedic or major general surgery).

Table 4. Summary of American College of Chest Physicians Recommendations Regarding Duration of Long-Term Antithrombotic Therapy for Deep Vein Thrombosis (DVT)

Clinical Subgroup	Antithrombotic Treatment Duration
First episode DVT/transient risk	VKA for 3 mo
First episode DVT/unprovoked	VKA for at least 3 mo
	Consider for long-term therapy if:
	• Proximal DVT
	• Minimal bleeding risk
	• Stable coagulation monitoring
Distal DVT/unprovoked	VKA for 3 mo
Second episode DVT/unprovoked	VKA long-term therapy
DVT and cancer	LMWH 3–6 mo
	Then VKA or LMWH indefinitely until cancer resolves

LMWH = low molecular weight heparin; VKA = vitamin K antagonist.

Source: Adapted with permission from Kearon C, Kahn SR, Agnelli G, et al:

Antithrombotic therapy for venous thromboembolic disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th edition). *Chest* 133:454S, 2008.

In contrast to patients with thrombosis related to transient risk factors, patients with idiopathic VTE are much more likely to develop recurrence (rates as high as 40% at 10 years). In this latter group of patients, numerous clinical trials have compared 3 to 6 months of anticoagulation therapy with extended-duration warfarin therapy, both at low intensity (INR of 1.5 to 2.0) and at conventional intensity (INR of 2.0 to 3.0). In patients with idiopathic DVT, extended-duration antithrombotic therapy is associated with a relative reduction in the rate of recurrent VTE by 75% to >90%. In addition, conventional-intensity warfarin reduces the risk even further compared with low-intensity warfarin (0.7 events per 100 person-years vs. 1.9 events per 100 person-years), but the rate of bleeding complications is no different.

In patients with VTE in association with a hypercoagulable condition, the optimal duration of anticoagulation therapy is influenced more by the clinical circumstances at the time of the VTE (idiopathic vs. secondary) than by the actual presence or absence of the more common thrombophilic conditions. In patients with VTE related to malignancy, increasing evidence suggests that longer-term therapy with LMWH (up to 6 months) is associated with a

lower VTE recurrence rate than treatment using conventional vitamin K antagonists.

The primary complication of warfarin therapy is hemorrhage, and the risk is related to the magnitude of INR prolongation. Depending on the INR and the presence of bleeding, warfarin anticoagulation may be reversed by (a) omitting or decreasing subsequent dosages, (b) administering oral or parenteral vitamin K, or (c) administering fresh-frozen plasma, prothrombin complex concentrate, or recombinant factor VIIa. Warfarin therapy rarely may be associated with the development of skin necrosis and limb gangrene. These conditions occur more commonly in women (4:1), and the most commonly affected areas are the breast, buttocks, and thighs. This complication, which usually occurs in the first days of therapy, is occasionally, but not exclusively, associated with protein C or S deficiency and malignancy. Patients who require continued anticoagulation may restart low-dose warfarin (2 mg) while receiving concomitant therapeutic heparin. The warfarin dosage is then gradually increased over a 1- to 2-week period.

Systemic and Catheter-Directed Thrombolysis

Patients with extensive proximal DVT may benefit from systemic thrombolysis or catheter-directed thrombolysis, which can potentially reduce acute symptoms more rapidly than anticoagulation alone. These techniques also may decrease the development of postthrombotic syndrome. Several thrombolysis preparations are available, including streptokinase, urokinase, alteplase (recombinant tissue plasminogen activator), reteplase, and tenecteplase. All these agents share the ability to convert plasminogen to plasmin, which leads to the degradation of fibrin. They differ with regard to their half-lives, their potential for inducing fibrinogenolysis (generalized lytic state), their potential for antigenicity, and their FDA-approved indications for use.

Streptokinase is purified from beta-hemolytic *Streptococcus* and is approved for the treatment of acute myocardial infarction, PE, DVT, arterial thromboembolism, and occluded central lines and arteriovenous shunts. It is not specific for fibrin-bound plasminogen, however, and its use is limited by its significant rates of antigenicity. Fevers and shivering occur in 1 to 4% of patients. Urokinase is derived from human neonatal kidney cells, grown in tissue culture. Currently, it is only approved for lysis of massive PE or PE associated with unstable hemodynamics. Alteplase, reteplase, and tenecteplase all are recombinant variants of tissue plasminogen activator. Alteplase is indicated for the treatment of acute myocardial infarction, acute ischemic stroke, and acute massive PE. However, it often is used for catheter-directed

thrombolysis of DVT. Reteplase and tenecteplase are indicated only for the treatment of acute myocardial infarction.

Systemic thrombolysis was evaluated in numerous older prospective and randomized clinical trials, and its efficacy was summarized in a recent Cochrane Review. In 12 studies involving over 700 patients, systemic thrombolysis was associated with significantly more clot lysis [relative risk (RR) 0.24 to 0.37] and significantly less postthrombotic syndrome (RR 0.66). However, venous function was not significantly improved. In addition, more bleeding complications did occur (RR 1.73), but the incidence appears to have decreased in later studies, probably due to improved patient selection.

In an effort to minimize bleeding complications and increase efficacy, catheter-directed thrombolytic techniques have been developed for the treatment of symptomatic DVT. With catheter-directed therapy, venous access may be achieved through percutaneous catheterization of the ipsilateral popliteal vein, retrograde catheterization through the contralateral femoral vein, or retrograde cannulation from the internal jugular vein. Multi-side-hole infusion catheters, with or without infusion wires, are used to deliver the lytic agent directly into the thrombus.

The efficacy of catheter-directed urokinase for the treatment of symptomatic lower extremity DVT has been reported in a large multicenter registry. Two hundred twenty-one patients with iliofemoral DVT and 79 patients with femoropopliteal DVT were treated with catheter-directed urokinase for a mean of 53 hours. Complete lysis was seen in 31% of the limbs, 50 to 99% lysis in 52% of the limbs, and <50% lysis in 17%. Overall, 1-year primary patency was 60%. Patency was higher in patients with iliofemoral DVT than in patients with femoropopliteal DVT (64% vs. 47%, $P < .01$). In addition, patients with acute symptoms (≤ 10 days) had a greater likelihood of complete lysis (34%) than patients with chronic symptoms (> 10 days; 19%). Major bleeding occurred in 11%, but neurologic involvement and mortality were rare (both 0.4%). Adjunctive stent placement to treat residual stenosis and/or short segment occlusion was required in 103 limbs.

One small randomized trial and numerous other retrospective studies have demonstrated similar rates of thrombolysis, with some also showing improved valve preservation and quality of life. Combining thrombolysis with percutaneous thrombus fragmentation and extraction has the added benefit of decreasing the infusion time, the hospital stay, and the overall cost of treatment. These studies, as well as the current ACCP guidelines, suggest that catheter-directed thrombolysis (with adjunctive angioplasty, venous stenting, and pharmacomechanical fragmentation and extraction) may be useful in selected patients with extensive iliofemoral

DVT. Patients should have a recent onset of symptoms (<14 days), good functional status, decent life expectancy, and low bleeding risk.

Inferior Vena Caval Filters

Since the introduction of the Kimray-Greenfield filter in the United States in 1973, numerous vena caval filters have been developed. Although the designs are variable, they all prevent pulmonary emboli, while allowing continuation of venous blood flow through the IVC. Early filters were placed surgically through the femoral vein. Currently, less invasive techniques allow percutaneous filter placement through a femoral vein, internal jugular vein, or small peripheral vein under fluoroscopic or ultrasound guidance. Complications associated with IVC filter placement include insertion site thrombosis, filter migration, erosion of the filter into the IVC wall, and IVC thrombosis. The rate of fatal complications is <0.12%.

Placement of an IVC filter is indicated for patients who develop recurrent DVT (significant propagation of the original thrombus or proximal DVT at a new site) or PE despite adequate anticoagulation therapy and for patients with pulmonary hypertension who experience recurrent PE. In patients who receive IVC filters for these indications, therapeutic anticoagulation should be continued. The duration of anticoagulation is determined by the underlying VTE and not by the presence of the IVC filter itself. Practically speaking, however, many patients who require an IVC filter for recurrent VTE are the same ones who would benefit most from indefinite anticoagulation. The other major indication for placement of an IVC filter is a contraindication to, or complication of, anticoagulation therapy in the presence of an acute proximal DVT. In patients who are not able to receive anticoagulants due to recent surgery or trauma, the clinician should continually reassess if antithrombotic agents may be started safely at a later date. Even some patients who develop anticoagulation-associated bleeding complications may be able to restart therapy at a lower intensity of anticoagulation later in the hospital course. As before, the clinical circumstances surrounding the VTE should determine the duration of anticoagulation.

Placement of permanent IVC filters has been evaluated as an adjunct to routine anticoagulation in patients with proximal DVT. In this study, routine IVC filter placement did not prolong early or late survival in patients with proximal DVT but did decrease the rate of PE (hazard ratio, 0.22; 95% confidence interval, 0.05 to 0.90). An increased rate of recurrent DVT was seen in patients with IVC filters (hazard ratio, 1.87; 95% confidence interval, 1.10 to 3.20). More controversial indications for IVC filter placement include prophylaxis against PE

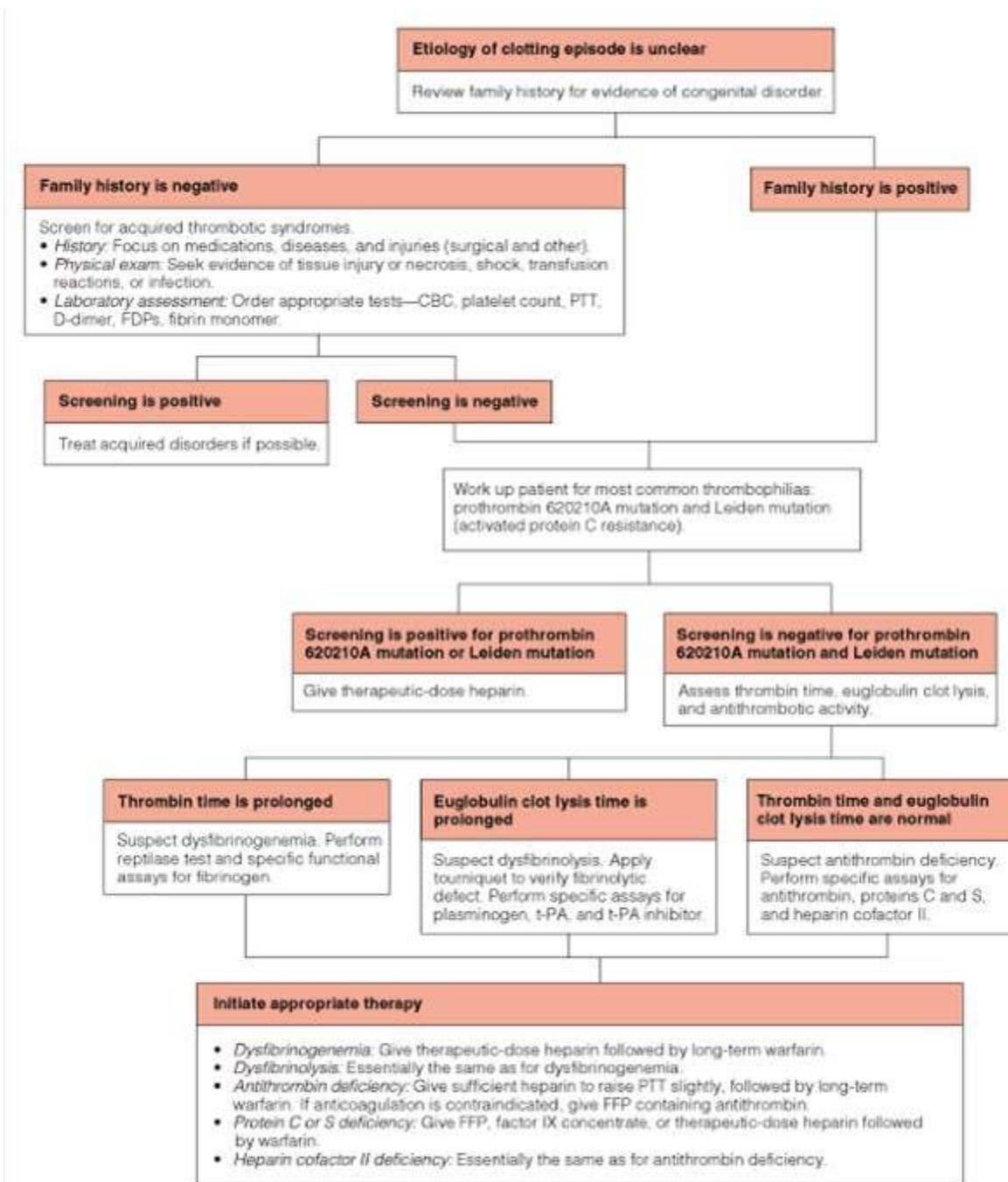
in patients receiving catheter-directed thrombolysis and in high-risk patients without established DVT or PE.

Hypercoagulability States

Certain patients seem to have a tendency to clot spontaneously. So-called hypercoagulability states were long thought to exist, but they were difficult to document except on clinical grounds. Currently, however, these clotting tendencies are better understood, thanks in large part to recognition of the role of antithrombins. If an antithrombin deficiency exists and clotting goes unchecked, activation of a clotting cascade could theoretically progress to clotting throughout the entire vasculature. Another important development was the recognition that deficiencies of certain natural clot-removing substances in the blood may lead to a clinical thrombotic tendency. Both types of deficiency can be either acquired or congenital.

Screening

When the etiology of a clotting episode is unclear, the family history should be reviewed for evidence of a congenital disorder. Even if the history is negative, the patient should be screened for both acquired and congenital disorders (table 6).



Acquired Clotting Conditions

Screening for acquired clotting conditions [see Table 7] is based on the history, physical examination, and laboratory assessment. The history should include medications, diseases, and surgical procedures or other injuries. Examination may disclose causes of hypercoagulability. Soft tissue injury, for example, is a potent activator of the coagulation system. If the injury is severe enough, it may be capable of causing a severe acquired coagulopathy. The problem is usually obvious, but on occasion, detailed study may be necessary to identify tissue damage or ischemic injury to bowel or extremities. Hypovolemia—especially hypovolemic

shock markedly reduces clotting time: blood from a patient in profound shock may clot instantaneously in the syringe as it is being drawn. The breakdown of red cells in a hemolytic transfusion reaction can cause clotting. Severe infection, especially from gram-negative organisms, is a potent activator of coagulation.

Table 7. Etiology of Acquired Hypercoagulability



Tissue and cellular damage
Shock
Trauma
Surgery
Tissue necrosis
Transfusion necrosis
Drugs
Estrogens
Drug reactions and interactions
Heparin platelet antibody
Warfarin
Disease
Blood dyscrasias
Cancer
Diabetes
Homocystinuria
Hyperlipidemia
Presence of lupuslike anticoagulant
Severe infection
Pregnancy

Of the acquired hypercoagulability syndromes, Trousseau syndrome is a particularly important condition for surgeons to recognize because it occurs in the surgical population (cancer patients) and must be treated with heparin (it is unresponsive to warfarin). It occurs when an adenocarcinoma secretes a protein recognized by the body as tissue factor, resulting in multiple episodes of venous thromboembolism over time (migratory thrombophlebitis). Simple depletion of vitamin K-dependent factors is ineffective. Patients should receive therapeutic-dose heparin indefinitely or until the cancer is brought into remission.

Laboratory screening may facilitate diagnosis. A complete blood count may document the presence of polycythemia or leukemia. Thrombocytopenia may be a manifestation of a hypercoagulable disorder, and thrombocytopenia after the administration of heparin raises the possibility of intravascular platelet aggregation. A prolonged aPTT is suggestive of lupuslike anticoagulant. Increased levels of D-dimers, fibrin degradation products (FDPs), or fibrin

monomers in the plasma may reflect low-grade intravascular coagulation.

Congenital Clotting Conditions

Congenital clotting tendencies can result from deficiencies in inhibitors of thrombosis (antithrombin, proteins C and S, and possibly heparin cofactor II), dysfibrinogenemias, or dysfibrinolysis [see Table 8]. Most congenital clotting defects are transmitted as an autosomal dominant trait. A negative family history does not preclude inherited thrombophilia, because the defects have a low penetrance, and fresh mutations may have occurred.

Table 8. Congenital Clotting Disorders

<p>Deficiencies in thrombosis inhibitors</p> <ul style="list-style-type: none">Antithrombin III deficiencyProteins C and S deficiencyActivated protein C resistance <p>Dysfibrinogenemias</p> <p>Dysfibrinolysis</p> <ul style="list-style-type: none">HypoplasminogenemiaImpaired release of t-PAHigh levels of t-PA inhibitorFactor XII deficiency
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INITIAL LABORATORY ASSESSMENT

Initial evaluation of a patient with an unexplained thrombotic episode should be directed at the most common causes of hypercoagulability. Acquired causes of clotting are more commonly seen by surgeons than congenital causes and therefore must be excluded first. If a clotting disorder is determined to be congenital, a laboratory workup should be undertaken. Several of the relevant assays "specifically, the functional assays" should be performed after the acute phase of the disorder has passed. If they are performed during the acute phase, levels of several antithrombotics (e.g., antithrombin and proteins C and S) will be misleadingly low not because deficiencies of these substances caused the underlying thrombotic process but because they were consumed in that process.

Specific Causes Of Thrombotic Tendency

The most common congenital causes of accelerated clotting are mutations of prothrombin (prothrombin G20210A mutation) and factor V (Leiden mutation, or activated protein C resistance). The prevalence of each of these ranges from 1% to 5% in the general population and may be much higher in specific ethnic subpopulations.¹ Each mutation may be identified conclusively by means of polymerase chain reaction (PCR) techniques. Detection of these mutations, unlike assays for antithrombin and proteins C and S, is not dependent on the patient's current inflammatory state. It must be remembered that the presence of one of these mutations, especially in the heterozygous form, does not imply that it is the sole cause of thrombosis. In many patients, a second precipitating factor must be present for the pathologic genetic thrombotic potential to be manifested.

Prothrombin G20210A Mutation

The prothrombin G20210A mutation is known to involve a single amino acid substitution in the prothrombin gene, but precisely how this increases the risk of venous thromboembolism is unclear. The one apparent manifestation of the mutation is a 15% to 40% increase in circulating prothrombin. Regardless of the mechanism at work, patients who are at least heterozygous for the trait are at two- to sixfold greater risk for venous thromboembolism than those without the mutation.

Resistance to Activated Protein C (Factor V Leiden)

Resistance of human clotting factors to inactivation by activated protein C is believed to be the most common inherited procoagulant disorder.¹¹⁴ Normally, activated factor V is degraded by activated protein C in the presence of membrane surface as part of normal regulation of thrombosis. Activated protein C resistance is caused by a single substitution mutation in the factor V gene, which is passed in an autosomal dominant fashion. The mutant factor V that results, termed factor V Leiden, is resistant to inactivation by activated protein C and thus has a greater ability to activate thrombin and accelerate clotting.

Two techniques are commonly used to diagnose this disorder. The first is a functional assay that compares a standard aPTT to one performed in the presence of exogenous activated protein C. If the latter aPTT does not exhibit significant prolongation, the patient is probably

resistant to activated protein C. The results of this assay must be interpreted with caution if the patient is still in the acute phase of the illness. The second technique, which is more reliable, involves direct detection of the mutation via PCR analysis of DNA.

Antithrombin Deficiency

Antithrombin (once termed antithrombin III) is a 65 kd protein that decelerates the coagulation system by inactivating activated factors, primarily factor Xa and thrombin but also factors XII, XI, and IX. Antithrombin therefore acts as a scavenger of activated clotting factors. Its activity is enhanced 100-fold by the presence of heparans on the endothelial surface and 1,000-fold by administration of exogenous heparin.

Congenital antithrombin deficiency occurs in approximately 0.01% to 0.05% of the general population and 2% to 4% of patients with venous thrombosis. The trait is passed on as an autosomal dominant trait, with the heterozygous genotype being incompatible with life. Antithrombin-deficient patients are at increased risk for thromboembolism when their antithrombin activity falls below 70% of normal.

Patients with congenital antithrombin deficiency frequently present after a stressful event. They usually have DVT but sometimes have PE. If anticoagulation is not contraindicated, the treatment of choice is heparin at a dosage sufficient to raise the aPTT to the desired level, followed by warfarin. If anticoagulation is contraindicated (as it is during the peripartum period), antithrombin concentrate should be given to raise the antithrombin activity to 80% to 120% of normal during the period when anticoagulants cannot be given.

Acquired antithrombin deficiency is a well-recognized entity. In most patients undergoing severe systemic stress, antithrombin levels fall below normal. Patients with classic risk factors for venous thromboembolism tend to have the lowest levels.

Protein C and Protein S Deficiency

Protein C is a 62 kd glycoprotein with a half-life of 6 hours. Because it is vitamin K dependent, a deficiency will develop in the absence of vitamin K. Acquired protein C deficiency is seen in liver disease, malignancy, infection, the postoperative state, and disseminated intravascular coagulation. Protein C deficiency occurs in approximately 4% to 5% of patients younger than 40 to 45 years who present with unexplained venous thrombosis. It is transmitted as an autosomal dominant trait, and the family history is usually positive for a

clotting tendency. Protein C levels range from 70% to 164% of normal in patients without a clotting tendency; levels below 70% of normal are associated with a thrombotic tendency. The most appropriate tests for screening are functional assays; there are cases of dysfunctional protein C deficiency in which protein C antigen levels are normal but protein C activity is low, and these would not be detected by the usual immunoassays.

Protein S is a vitamin K-dependent protein that acts as a cofactor for activated protein C by enhancing protein C-induced inactivation of activated factor V. The incidence of protein S deficiency is similar to that of protein C deficiency. It is transmitted as a dominant trait, and the family history is often positive for a thrombotic tendency.

Hyperhomocysteinemia

Although hyperhomocysteinemia is more commonly associated with cardiac disease and arterial thrombosis, it may also be associated with an increased incidence of venous thromboembolism. This association is not as strong as those already discussed. Accordingly, anticoagulation of asymptomatic patients with elevated homocysteine levels is not currently recommended.

Dysfibrinogenemia

More than 100 qualitative abnormalities of fibrinogen (dysfibrinogenemias) have been reported. Dysfibrinogenemias are inherited in an autosomal dominant manner, with most patients being heterozygous. Most patients with dysfibrinogenemia have either no clinical symptoms or symptoms of a bleeding disorder; a minority (about 11%) have clinical features of a recurrent thromboembolic disorder. Congenital dysfibrinogenemias associated with thrombosis account for about 1% of cases of unexplained venous thrombosis occurring in young people. The most commonly observed functional defect in such dysfibrinogenemias is abnormal fibrin monomer polymerization combined with resistance to fibrinolysis. Decreased binding of plasminogen and increased resistance to lysis by plasmin have been noted.

In addition to a prolonged TT, patients who have dysfibrinogenemia associated with thromboembolism may have a prolonged INR. The diagnosis is confirmed if the reptilase time is also prolonged. Measured with clotting techniques, fibrinogen levels may be slightly or moderately low; measured immunologically, levels may be normal or even increased.

Dysfibrinolysis

Fibrinolysis can be impaired by inherited deficiencies of plasminogen, defective release of t-PA from the vascular endothelium, and high plasma levels of regulatory proteins (e.g., t-PA inhibitors). In addition, factor XII (contact factor) deficiency may induce failure of fibrinolysis activation.

Inherited plasminogen deficiency is probably only rarely responsible for unexplained DVT in young patients. It is transmitted as an autosomal dominant trait. In heterozygous persons with a thrombotic tendency, plasminogen activity is about one half normal (3.9 to 8.4 $\mu\text{mol/ml}$). The euglobulin clot lysis time is prolonged. Functional assays should be carried out, and there should be full transformation of plasminogen into plasmin activators.

The important role of t-PA inhibitors I and II in the regulation of fibrinolysis is well defined. In normal plasma, t-PA inhibitor I is the primary inhibitor for both t-PA and urokinase. Release of t-PA inhibitor I by platelets results in locally increased concentrations where platelets accumulate. The ensuing local inhibition of fibrinolysis may help stabilize the hemostatic plug. t-PA inhibitor II is present in and secreted by monocytes and macrophages.

Factor XII deficiency is a rare cause of impaired fibrinolysis. Initial contact activation of factor XII not only results in activation of the clotting cascade and of the inflammatory response but also leads to plasmin generation. This intrinsic activation of fibrinolysis requires factor XII, prekallikrein, and high-molecular-weight kininogen. Patients with factor XII deficiencies can be identified by a prolonged aPTT in the absence of clinical bleeding.

Operative Venous Thrombectomy

In patients with acute iliofemoral DVT, surgical therapy is generally reserved for patients who worsen with anticoagulation therapy and those with phlegmasia cerulea dolens and impending venous gangrene. If the patient has phlegmasia cerulea dolens, a fasciotomy of the calf compartments is first performed. In iliofemoral DVT, a longitudinal venotomy is made in the common femoral vein and a venous balloon embolectomy catheter is passed through the thrombus into the IVC and pulled back several times until no further thrombus can be extracted. The distal thrombus in the leg is removed by manual pressure beginning in the foot. This is accomplished by application of a tight rubber elastic wrap beginning at the foot and extending to the thigh. If the thrombus in the femoral vein is old and cannot be extracted, the vein is ligated. For a thrombus that extends into the IVC, the IVC is exposed transperitoneally

and the IVC is controlled below the renal veins. The IVC is opened and the thrombus is removed by gentle massage. An intraoperative completion venogram is obtained to determine if any residual thrombus or stenosis is present. If a residual iliac vein stenosis is present, intraoperative angioplasty and stenting can be performed. In most cases, an arteriovenous fistula is then created by anastomosing the great saphenous vein (GSV) end to side with the superficial femoral artery in an effort to maintain patency of the thrombectomized iliofemoral venous segment. Heparin is administered postoperatively for several days. Warfarin anticoagulation is maintained for at least 6 months after thrombectomy. Complications of iliofemoral thrombectomy include PE in up to 20% of patients and death in <1% of patients.

One study followed 77 limbs for a mean of 8.5 years after thrombectomy for acute iliofemoral DVT. In limbs with successful thrombectomies, valvular competence in the thrombectomized venous segment was 80% at 5 years and 56% at 10 years. More than 90% of patients had minimal or no symptoms of postthrombotic syndrome. There were 12 (16%) early thrombectomy failures. Patients were required to wear compression stockings for at least 1 year after thrombectomy.

Survival rates for surgical pulmonary embolectomy have improved over the past 20 years with the addition of cardiopulmonary bypass. Emergency pulmonary embolectomy for acute PE is rarely indicated. Patients with preterminal massive PE (Fig. 5) for whom thrombolysis has failed or who have contraindications to thrombolytics may be candidates for this procedure. Open pulmonary artery embolectomy is performed through a posterolateral thoracotomy with direct visualization of the pulmonary arteries. Mortality rates range between 20 and 40%.



Fig. 5. Autopsy specimen showing a massive pulmonary embolism.

Percutaneous catheter-based techniques for removal of a PE involve mechanical thrombus fragmentation or embolectomy using suction devices. Mechanical clot fragmentation is followed by catheter-directed thrombolysis. Results of catheter-based fragmentation are based on small case series. In a study in which a fragmentation device was used in 10 patients with acute massive PE, fragmentation was successful in 7 patients with a mortality rate of 20%.⁶⁵ Transvenous catheter pulmonary suction embolectomy has also been performed for acute massive PE with a reported 76% successful extraction rate and a 30-day survival of 70%.⁶⁶

Prophylaxis

Patients who undergo major general surgical, gynecologic, urologic, and neurosurgical procedures without thromboprophylaxis have a significant incidence of perioperative DVT (15 to 40%). The incidence is even higher with major trauma (40 to 80%), hip and knee replacement surgery (40 to 60%), and spinal cord injury (60 to 80%). The goal of prophylaxis is to reduce the mortality and morbidity associated with VTE. The first manifestation of VTE may be a life-threatening PE (Fig. 6), and as indicated earlier, clinical evaluation to detect DVT before PE is unreliable.

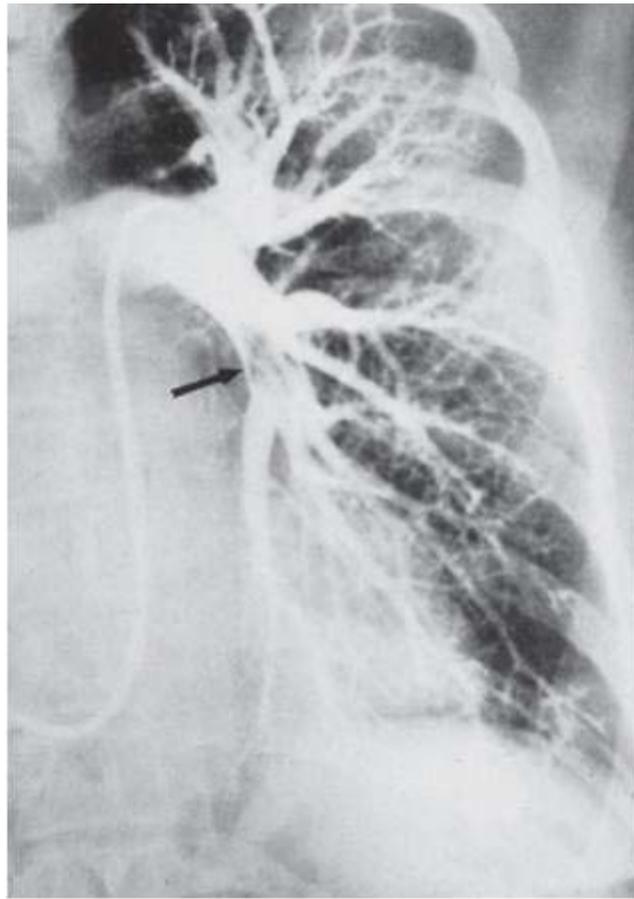


Fig. 6. Pulmonary angiogram showing a pulmonary embolism (*arrow*).

Effective methods of VTE prophylaxis involve the use of one or more pharmacologic or mechanical modalities. Currently available pharmacologic agents include low-dose UFH, LMWH, synthetic pentasaccharides, and vitamin K antagonists. Mechanical methods include intermittent pneumatic compression (IPC) and graduated compression stockings. Aspirin therapy alone is not adequate for DVT prophylaxis. These prophylaxis methods vary with regard to their efficacy, and the 2008 ACCP Clinical Practice Guidelines stratify their uses according to the patient's level of risk (Table 6).

Table 6. Thromboembolism Risk and Recommended Thromboprophylaxis in Surgical Patients

Level of Risk	Approximate DVT Risk without Thromboprophylaxis (%)	Suggested Thromboprophylaxis Options
Low risk	<10	No specific thromboprophylaxis
Minor surgery in mobile patients		Early and "aggressive" ambulation
Moderate risk	10-40	LMWH (at recommended doses), LDUH bid or tid, fondaparinux
Most general, open gynecologic, or urologic surgery		Mechanical thromboprophylaxis
Moderate VTE risk plus high bleeding risk		
High risk	40-80	LMWH (at recommended doses), fondaparinux, oral vitamin K antagonist (INR 2-3)
Hip or knee arthroplasty, hip fracture surgery		
Major trauma, spinal cord injury		Mechanical thromboprophylaxis
High VTE risk plus high bleeding risk		

Complications

Complications after venous thrombosis can vary from life threatening to chronically debilitating. Pulmonary embolism develops as venous thrombi break off from their location of origin and travel through the right heart and into the pulmonary artery, causing a ventilation perfusion defect and cardiac strain. PE occurs in approximately 10% of patients with acute deep venous thrombosis and can cause up to 10% of in hospital deaths.^{60,61} However, most patients (up to 75%) are asymptomatic. Traditionally, proximal venous thrombosis are thought to be at highest risk for causing pulmonary emboli; however, the single largest autopsy series ever performed to specifically to look for the source of fatal PE was performed by Havig in 1977, who found that one third of the fatal emboli arose directly from the calf veins.⁶

PHLEGMASIA ALBA AND CERULEA DOLENS

More than 600,000 cases of venous thromboembolism are estimated to occur each year in the United States. Pulmonary embolism (PE) complicates approximately 50% of cases of untreated proximal deep venous thrombosis (DVT) and contributes to 10-15% of all hospital deaths. Less frequent manifestations of venous thrombosis include phlegmasia alba dolens, phlegmasia cerulea dolens (PCD), and venous gangrene. These form a clinical spectrum of the same disorder. All 3 manifestations result from acute massive venous thrombosis and obstruction of the venous drainage of an extremity

History of the Procedure

In the 16th century, Fabricius Hildanus first described the clinical syndrome of what is currently called PCD. In 1938, Gregoire made an outstanding description of the condition and used the term PCD to differentiate ischemia-associated massive venous thrombosis from phlegmasia alba dolens, which describes fulminant venous thrombosis without ischemia.¹ The exact incidence of these disorders is not well reported.

In 1939, Leriche and Geissendorfer performed the first thrombectomy for cases of PCD.² Historically, surgical thrombectomy has been the procedure of choice for PCD refractory to medical therapy and in patients with established or impending gangrene.

Frequency

More than 600,000 cases of venous thromboembolism are estimated to occur annually in the United States. Phlegmasia alba dolens, PCD, and venous gangrene occur at any age but are more common during the fifth and sixth decades of life. Incidence is higher in females than in males.

Etiology

The main causative factor in phlegmasia is massive thrombosis and occlusion of major venous channels with significantly compromised venous outflow. Multiple triggering factors exist. Malignancy is the most common triggering factor and is present in approximately 20-40% of patients with PCD. Other associated risk factors include hypercoagulable syndrome, surgery, trauma, ulcerative colitis, gastroenteritis, heart failure, mitral valve stenosis, vena caval filter insertion, and May-Thurner syndrome (compression of the left iliac vein by the right iliac artery). Pregnancy has often been associated with phlegmasia alba dolens, especially during the third trimester when the uterus is large enough to compress the left common iliac vein against the pelvic rim (ie, milk leg syndrome). Finally, 10% of patients with phlegmasia have no apparent risk factors.

Pathophysiology

In phlegmasia alba dolens, the thrombosis involves only major deep venous channels of the extremity, therefore sparing collateral veins. The venous drainage is decreased but still

present; the lack of venous congestion differentiates this entity from PCD.

In PCD, the thrombosis extends to collateral veins, resulting in venous congestions with massive fluid sequestration and more significant edema. Without established gangrene, these phases are reversible if proper measures are taken.

Of PCD cases, 40-60% also have capillary involvement, which results in irreversible venous gangrene that involves the skin, subcutaneous tissue, or muscle. Under these conditions, the hydrostatic pressure in arterial and venous capillaries exceeds the oncotic pressure, causing fluid sequestration in the interstitium. Venous pressure may increase rapidly, as much as 16- to 17-fold within 6 hours. Fluid sequestration may reach 6-10 L in the affected extremity within days. Circulatory shock, which is present in about one third of patients, and arterial insufficiency may ensue.

The exact mechanism for the compromised arterial circulation is debatable but may involve shock, increased venous outflow resistance, and collapse of arterioles due to increased interstitial pressure. Vasospasm of the resistance vessels has also been hypothesized but has never been observed experimentally or radiographically.

Presentation

In the lower extremities, left-sided involvement is more common by a 3:1 or 4:1 ratio. Involvement of upper extremities occurs in less than 5% of patients with PCD. Manifestations may be gradual or fulminant. Of PCD cases, 50-60% are preceded by phlegmasia alba dolens, with symptoms of edema, pain, and blanching (alba) without cyanosis. The blanching, which previously was thought to be caused by arterial vasospasm, is caused by subcutaneous edema, without venous congestion.

Patients with PCD present with the clinical triad of edema, agonizing pain, and cyanosis. Massive fluid sequestration may lead to bleb and bullae formation. The pain is constant, usually starting at the femoral triangle and then progressing to the entire extremity. Cyanosis is the pathognomonic finding of PCD, progressing from distal to proximal areas.

When venous gangrene occurs, it has a similar distribution with the cyanosis. Arterial pulses may be present when the venous gangrene is superficial; however, gangrene that involves

the muscular compartment may result in increased compartment pressures and a pulse deficit. In addition, the pulses may be difficult to appreciate because of the significant edema. Various degrees of shock may be present because of significant fluid loss.

Indications

Historically, surgical thrombectomy has been the procedure of choice for phlegmasia cerulea dolens (PCD) refractory to medical therapy and for patients with established or impending gangrene. The standard treatment of phlegmasia and venous gangrene is evolving, but most clinicians attempt endovascular approaches to thrombolysis, if possible.

Relevant Anatomy

The main causative factor in phlegmasia is massive deep venous thrombosis (DVT) and occlusion of major venous channels with significantly compromised venous outflow. In phlegmasia alba dolens, the thrombosis involves only major deep venous channels of the extremity, therefore sparing collateral veins and preserving some venous outflow from the limb. In phlegmasia cerulea dolens (PCD), the thrombosis extends to collateral veins, with complete obstruction of venous outflow, resulting in massive venous congestion and fluid sequestration and more significant edema.

Contraindications

For phlegmasia alba dolens and mild nongangrenous forms of phlegmasia cerulea dolens (PCD), conservative medical treatment, rather than surgical thrombectomy, should be the initial course of therapy. Thrombolysis may be initiated if conservative management does not elicit a response and if the patient has no contraindication to lytic therapy.

Surgical thrombectomy cannot open the small venules that are affected in venous gangrene, and it does not prevent valvular incompetence or postphlebotic syndrome. For these reasons, thrombolysis seems to be an attractive alternative in the management of PCD and venous gangrene.

Imaging Studies

The diagnoses of phlegmasia alba dolens, phlegmasia cerulea dolens (PCD), and venous gangrene are established mainly via clinical criteria with the assistance of contrast venography and duplex ultrasonography.

Although venography is considered the criterion standard for diagnosis, technical difficulties may be encountered in as many as 20-25% of patients. Attempts to perform ascending venography when extensive deep system thrombosis is present may result in nonvisualization of the deep system and a nondiagnostic study result. In these cases, descending venography via the contralateral femoral vein or via the upper extremity veins may provide more information about the ilio caval system and proximal extent of the thrombus.

Recent improvements in ultrasonography have made this modality a more reliable and accurate way to assess for proximal deep venous thrombosis (DVT) with less morbidity. In addition, duplex imaging may be repeated as needed to monitor for thrombus propagation. Ultrasonography can also be performed at the bedside in patients who are critically ill or unstable. Ultrasonography is often used to guide the initial venipuncture for diagnostic venography and initiation of thrombolytic therapy.

Magnetic resonance venography (MRV) is an evolving modality of diagnostic imaging. Its principal advantage is its ability to easily reveal the proximal and distal extent of thrombus with a single study. Its principal disadvantage is the inability to image acutely ill patients with hemodynamic instability or motion artifacts due to pain.

Medical Therapy

The standard treatment of phlegmasia and venous gangrene is still evolving. The optimal therapeutic modality remains under debate. So far, the results of treatment have been moderately successful. For phlegmasia alba dolens and mild nongangrenous forms of phlegmasia cerulea dolens (PCD), conservative medical treatment, such as steep limb elevation, anticoagulation with intravenous administration of heparin, and fluid resuscitation, should be the initial course of therapy.

Initiate heparin administration with an intravenous bolus of 80-100 U/kg, followed by a continuous infusion of 15-18 U/kg/h. Frequently monitor the activated partial thromboplastin time (aPTT), with a goal range of 2-2.5 times the laboratory reference range. Frequently monitor platelet counts to allow the early detection of heparin-induced thrombocytopenia.

The purpose of rapid heparin anticoagulation is to decrease the risk of proximal clot propagation or thromboembolism. Heparin does not directly affect limb swelling. The best nonsurgical method to decrease edema is steep leg elevation.

Recent studies have demonstrated that low molecular weight heparins are safe and effective in the treatment of proximal deep venous thrombosis (DVT) and pulmonary embolism (PE); however, no good evidence supports the use of these newer agents in phlegmasia and venous gangrene.

If heparin-induced thrombocytopenia occurs, immediately discontinue the use of heparin and replace it with an alternative anticoagulant. Danaparoid and lepirudin are effective alternative agents; however, heparin-associated antibodies exhibit a 10-19% cross-reactivity with danaparoid. Thus, perform cross-reactivity testing before the initiation of danaparoid in patients with these antibodies. Lepirudin is a direct thrombin inhibitor that does not demonstrate any cross-reactivity. The recommended dosage of lepirudin in patients without renal failure is 0.4 mg/kg as an intravenous bolus followed by a continuous infusion of 0.15 mg/kg/h. Use aPTT to monitor therapy, with a goal range of 2-2.5 times the laboratory reference range.

Continue long-term anticoagulation with warfarin (or other coumarin derivatives) for at least 6 months. Life-long anticoagulation is recommended in patients with hypercoagulable states.

Patients should wear long-term prescription compression stockings with at least 30-40 mm Hg of graded pressure. Many physicians erroneously have the patient fitted for a prescription stocking while the limb is still severely edematous. Instead, the patient may use nonprescription stockings or an elastic bandage, in combination with elevation, to minimize edema prior to being fit for a prescription stocking.

Surgical Therapy

Surgical thrombectomy performed through a femoral venotomy allows instant decompression of the venous hypertension. An intraoperative Trendelenburg position may be

used to decrease the risk of PE. Transabdominal cavotomy and thrombectomy is an alternative approach that permits better control of the cava above the thrombus and, thus, provides protection against PE. Procedures that have been performed in an effort to decrease the rethrombosis rate include cross-pubic vein-to-vein reconstruction with polytetrafluoroethylene (PTFE) or the greater saphenous vein (GSV) or the creation of an arteriovenous fistula between the femoral artery and the GSV. These adjuvant procedures may be especially beneficial in cases that involve proximal iliofemoral vein constriction, damage, or external compression.

Concomitant administration of heparin and long-term anticoagulation are mandatory. Regardless, thrombectomy in patients with PCD is associated with a high rate of rethrombosis. Surgical thrombectomy cannot open the small venules that are affected in venous gangrene, and it does not prevent valvular incompetence or postphlebotic syndrome. The incidence of postphlebotic syndrome may be as high as 94% among survivors.

For the above reasons, thrombolysis seems to be an attractive alternative in the management of PCD and venous gangrene. In 1970, Paquet was the first to use thrombolysis for the treatment of PCD.⁴ Some authors propose catheter-directed thrombolysis directly into the vein with high doses of urokinase or tissue plasminogen activator (t-PA). Other authors support the method of intra-arterial low-dose thrombolysis via the common femoral artery, reasoning that the arterial route delivers the thrombolytic agent to the arterial capillaries and, subsequently, to the venules. The intra-arterial approach seems to be more effective in cases with venous gangrene. Systemic thrombolysis has also been used. Many authors have strongly recommended the insertion of a vena caval filter prior to initiation of thrombolytic therapy. Combine thrombolysis with heparin administration and long-term oral anticoagulation.

Fasciotomy alone or in conjunction with thrombectomy or thrombolysis reduces compartmental pressures; however, it significantly increases morbidity because of the prolonged wound healing and the risk of infection.

Finally, if all efforts fail and amputation is required, delay the procedure as long as possible. Take all precautions to reduce edema, allow venous channels to recanalize, and allow necrotic tissue to demarcate.

Preoperative Details

Patients who require emergent venous thrombectomy should have heparin continued throughout the perioperative period. Banked red blood cells should be available. The proximal extent of the thrombosis must be defined using a combination of venous ultrasonography for infrainguinal veins and retrograde venography of the iliac veins and inferior vena cava using a jugular or contralateral femoral approach. If the thrombosis extends into the iliac veins and vena cava, preparations should be made to control the cava via a right retroperitoneal incision. A high-quality fluoroscopy unit should be available to aid in catheter manipulation and completion venography.

Intraoperative Details

Operative exposure depends on the proximal and distal extent of the thrombus. The involved veins should be controlled proximally and distally prior to venotomy.

Iliac venous thrombectomy should be performed with large-bore thrombectomy balloon catheters (as large as 10F). Extension of the thrombus into the inferior vena cava may require proximal control of the cava. The anesthesiologist should apply positive airway pressure while the thrombus is being extracted from the ilio caval system. Digital subtraction venography should be performed to ascertain completeness of the thrombectomy.

Infrainguinal extraction of the thrombus is aided by the intraoperative placement of an Esmarch bandage from foot to thigh. Thrombectomy balloon catheters (3F and 4F) are passed through the femoral veins and, possibly, the posterior tibial veins as well.

Thrombolytic agents may be administered intraoperatively through the posterior tibial veins. t-PA is the most commonly used agent and may be administered intraoperatively.

After the thrombectomy is performed, an arteriovenous fistula should be constructed, connecting the proximal greater saphenous vein or one of its larger tributaries to the superficial femoral artery in an end-to-side fashion.

Completion venography should be performed to exclude the presence of residual thrombus or proximal venous stenosis. If one is present, balloon angioplasty with or without stent placement may be necessary.

When percutaneous endovascular therapy is performed as a single treatment modality, and many centers are now reporting this as a first-line therapy, the popliteal veins are usually

accessed with duplex ultrasonography as an aid. Prone positioning is rarely necessary. If extensive thrombus is present, access via the posterior tibial vein is usually successful. A 6-F sheath is usually adequate. An infusion wire is passed through the thrombus just to its proximal extent, often into the vena cava. Infusion is usually performed in the most proximal segment first, usually in the iliac veins.

A common protocol is to infuse tPA (1 mg/hr) through the infusion wire as well as through the sheath for 24 hours, then to change the sheath perfusion to lower dose heparin after 24 hours. The infusion is then performed in the superficial femoral and popliteal vein segments. Clinical improvement is often noted with clearing of the profunda venous segment. Performance of simultaneous percutaneous mechanical thrombectomy is controversial and may not give better results than postprocedure balloon dilation.

Postoperative Details

Intravenous heparin is administered throughout the postoperative period to prolong the aPTT (2-2.5 times the reference range for aPTT). This is continued until the patient is adequately anticoagulated with warfarin or one of the coumarin derivatives (international normalized ratio [INR] range of 2.0-3.0). The optimal duration of oral anticoagulation is not established.

A sequential compression device is also placed, or, at a minimum, an ace bandage is placed for control of edema. Once the edema is at its minimum, the patient may be fitted for a thigh-length compression stocking. Ambulation is encouraged, if the patient is able.

Complications

The incidence of postphlebotic syndrome may be as high as 94% among survivors. Pulmonary embolism is common, and prophylactic placement of an inferior vena cava filter is recommended in most cases. Thrombectomy in patients with phlegmasia cerulea dolens (PCD) is associated with a high rate of rethrombosis. Amputation and death are common.

Outcome and Prognosis

Despite all of the therapeutic modalities described above, phlegmasia cerulea dolens (PCD) and venous gangrene still remain life-threatening and limb-threatening conditions with

overall mortality rates of 20-40%. Pulmonary embolism (PE) is responsible for 30% of the deaths reported from PCD. Overall, amputation rates of 12-50% have been reported among survivors. The postphlebitic sequelae are apparent in 60-94% of survivors. Strict adherence to the use of long-term compression stockings helps to control chronic edema.

Future and Controversies

Phlegmasia alba dolens, phlegmasia cerulea dolens (PCD), and venous gangrene still remain a challenge to the vascular surgeon. Treatment modalities continue to evolve. Endovascular management may offer hope of successful and more effective management, with less morbidity, than traditional surgery. The role of mechanical thrombectomy, compared with thrombolysis, is unclear. Small numbers of patients and lack of randomized trials preclude clear recommendations.

SUPERIOR VENA CAVA SYNDROME

Introduction

Background

Superior vena cava syndrome (SVCS) is obstruction of blood flow through the superior vena cava (SVC). It is a medical emergency and most often manifests in patients with a malignant disease process within the thorax. A patient with superior vena cava syndrome (SVCS) requires immediate diagnostic evaluation and therapy.

William Hunter first described the syndrome in 1757 in a patient with syphilitic aortic aneurysm.¹ In 1954, Schechter reviewed 274 well-documented cases of superior vena cava syndrome (SVCS) reported in the literature; 40% of them were due to syphilitic aneurysms or tuberculous mediastinitis. In more recent times, these infections have gradually decreased as the primary cause of superior vena cava (SVC) obstruction. Lung cancer, particularly adenocarcinoma, is now the underlying process in approximately 70% of the patients with superior vena cava syndrome (SVCS). However, up to 40% of the causes are due to nonmalignant causes.

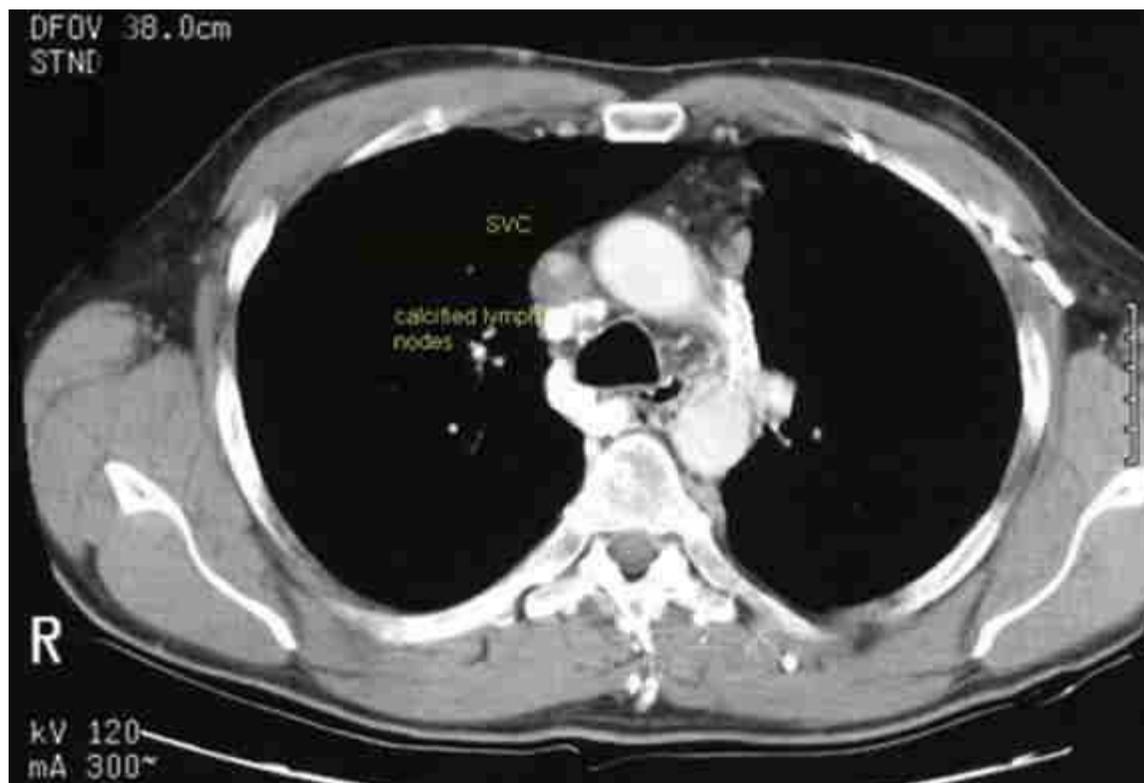


Fig. 1. Superior vena cava syndrome (case 1). The patient was a 35-year-old man with a 3-year history of progressive upper-extremity and fascial swelling. The patient had undergone treatment for histoplasmosis in the past. CT scan shows a narrowed superior vena cava with adjacent calcified lymph nodes and posterior soft tissue thickening

Pathophysiology

The superior vena cava (SVC) is the major drainage vessel for venous blood from the head, neck, upper extremities, and upper thorax. It is located in the middle mediastinum and is surrounded by relatively rigid structures such as the sternum, trachea, right bronchus, aorta, pulmonary artery, and the perihilar and paratracheal lymph nodes. It extends from the junction of the right and left innominate veins to the right atrium, a distance of 6-8 cm. It is a thin-walled, low-pressure, vascular structure. This wall is easily compressed as it traverses the right side of the mediastinum.⁷

Obstruction of the superior vena cava (SVC) may be caused by neoplastic invasion of the venous wall associated with intravascular thrombosis or, more simply, by extrinsic pressure of a tumor mass against the relatively fixed thin-walled superior vena cava (SVC). Postmortem examinations reveal that complete superior vena cava (SVC) obstruction is the result of

intravascular thrombosis in combination with extrinsic pressure. Incomplete superior vena cava (SVC) obstruction is more often secondary to extrinsic compression without thrombosis. Other causes include compression by intravascular arterial devices. The incidence is on the rise in line with the increased usage of endovascular devices.⁴

An obstructed superior vena cava (SVC) initiates collateral venous return to the heart from the upper half of the body through 4 principal pathways. The first and most important pathway is the azygous venous system, which includes the azygous vein, the hemiazygous vein, and the connecting intercostal veins. The second pathway is the internal mammary venous system plus tributaries and secondary communications to the superior and inferior epigastric veins. The long thoracic venous system, with its connections to the femoral veins and vertebral veins, provides the third and fourth collateral routes, respectively.

Despite these collateral pathways, venous pressure is almost always elevated in the upper compartment if obstruction of the superior vena cava (SVC) is present. Venous pressure as high as 200-500 cm H₂O has been recorded in patients with severe superior vena cava syndrome (SVCS).

Frequency

United States

Superior vena cava syndrome (SVCS) develops in 5-10% of patients with a right-sided malignant intrathoracic mass lesion. In 1969, Salsali and Clifton observed superior vena cava syndrome (SVCS) in 4.2% of 4960 patients with lung cancer; 80% of the tumors inducing superior vena cava syndrome (SVCS) were of the right lung. In 5 large series of small cell lung cancer, 9-19% of patients demonstrated superior vena cava syndrome (SVCS). In 1987, Armstrong and Perez found superior vena cava syndrome (SVCS) in 1.9% of 952 patients with lymphoma.

Mortality/Morbidity

Survival in patients with superior vena cava syndrome (SVCS) depends mainly on the course of the underlying disease. No mortality, per se, results directly from mild venous congestion.

In patients with benign superior vena cava syndrome (SVCS), life expectancy is unchanged.

If superior vena cava syndrome (SVCS) is secondary to a malignant process, patient survival correlates with the histology of the tumor. Patients with signs and symptoms of laryngeal and cerebral edema have the most life-threatening manifestations of this syndrome and are in danger of sudden death. Clinical observations show that approximately 10% of patients with a bronchogenic carcinoma and 45% of patients with lymphoma treated with irradiation live at least 30 months. In contrast, patients with untreated malignant superior vena cava syndrome (SVCS) survive only approximately 30 days.⁵

Race

The frequency of superior vena cava syndrome (SVCS) in different races depends largely on the frequency of lung cancer and lymphomas in these populations.

Sex

Malignant causes of superior vena cava syndrome (SVCS) are most commonly observed in males because of the high incidence of lung cancer in this population.

In contrast, no sex difference is observed in cases related to benign causes.

Age

Malignant causes of superior vena cava syndrome (SVCS) are predominantly observed in individuals aged 40-60 years.

Benign causes account for most of the cases diagnosed in individuals aged 30-40 years.

Obstruction of the superior vena cava (SVC) in the pediatric age group is rare and has a different etiologic spectrum.

Clinical

History

Early in the clinical course, partial superior vena cava (SVC) obstruction may be

asymptomatic, but more often, minor symptoms and signs are overlooked.

As the syndrome advances toward total superior vena cava (SVC) obstruction, the classic symptoms and signs become more obvious.

Dyspnea is the most common symptom and is observed in 63% of patients with superior vena cava syndrome (SVCS).^{7,10}

Other symptoms include facial swelling, head fullness, cough, arm swelling, chest pain, dysphagia, orthopnea, distorted vision, hoarseness, stridor, headache, nasal stuffiness, nausea, pleural effusions, and light-headedness.^{7,10,11}

Physical

The characteristic physical findings of superior vena cava syndrome (SVCS) include venous distension of the neck and chest wall, facial edema, upper extremity edema, mental changes, plethora, cyanosis, papilledema, stupor, and even coma.

Bending forward or lying down may aggravate the symptoms and signs.

Causes

More than 80% of cases of superior vena cava syndrome (SVCS) are caused by malignant mediastinal tumors.^{12,13,14}

Bronchogenic carcinomas account for 75-80% of all these cases, with most of these being small-cell carcinomas.³

Non-Hodgkin lymphoma (especially the large cell type) represents 10-15% of cases.

Causes of superior vena cava syndrome (SVCS) appear similar to the relative incidence of primary lung and mediastinal tumors.

Rare malignant diagnoses include Hodgkin disease, metastatic cancers,¹⁵ primary leiomyosarcomas of the mediastinal vessels, and plasmocytomas.^{16,17,18}

Nonmalignant conditions causing superior vena cava syndrome (SVCS) include mediastinal fibrosis; vascular diseases such as aortic aneurysm, vasculitis, and arteriovenous fistulas; infections such as histoplasmosis, tuberculosis, syphilis, and actinomycosis; benign mediastinal tumors such as teratoma, cystic hygroma, thymoma, and dermoid cyst; cardiac causes, such as pericarditis and atrial myxoma; and thrombosis related to the presence of central vein catheters. These account for approximately 22% of the causes of superior vena cava syndrome (SVCS).

Imaging Studies

Patients presenting with overt superior vena cava syndrome (SVCS) may be diagnosed by means of physical examination alone. However, subtle presentations require diagnostic imaging. Chest radiography may reveal a widened mediastinum or a mass in the right side of the chest. Only 16% of the patients studied by Parish and colleagues in 1981 had normal findings on chest radiography.

CT has the advantage of providing more accurate information on the location of the obstruction and may guide attempts at biopsy by mediastinoscopy, bronchoscopy, or percutaneous fine-needle aspiration.

It also provides information on other critical structures such as the bronchi and the vocal cords.

The additional information is necessary because the involvement of these structures requires prompt action for relief of pressure.

MRI has not been sufficiently investigated, but it appears promising.

It has several potential advantages over CT scanning, including the fact that it provides images in several planes of view and allows direct visualization of blood flow. Furthermore, MRI does not require iodinated contrast material. This is especially important when stenting is anticipated.

Disadvantages may include increased scanning time with attendant problems in patient compliance and increased cost.

Invasive contrast venography is the most conclusive diagnostic tool.

It precisely defines the etiology of obstruction.

It is especially important if surgical management is being considered for the obstructed vena cava.

Radionuclide technetium-99m venography is an alternative minimally invasive method of imaging the venous system. Although images obtained by this method are not as well defined as those achieved with contrast venography, they demonstrate potency and flow patterns.

Gallium single-proton emission CT scanning may be of value in select cases.

Procedures

Most patients with superior vena cava syndrome (SVCS) present before the primary diagnosis is established.

Controversy often arises in the treatment of a patient with superior vena cava syndrome (SVCS) in regard to the need for pathologic confirmation of malignancy before the start of therapy.

Treatment without an established diagnosis should be initiated only in patients with rapidly progressive symptoms or those in whom multiple attempts to obtain a tissue diagnosis have been unsuccessful.

Fortunately, relatively noninvasive measures establish the diagnosis in a high percentage of patients with superior vena cava syndrome (SVCS).

Sputum cytologic results are diagnostic in 68% of the cases, whereas biopsy of a palpable supraclavicular node is positive in 87%.

Bronchoscopy has a 60% success rate, while thoracotomy is 100% successful.

Open biopsy is rarely needed for diagnosis. Dosios et al showed that cervical mediastinoscopy and anterior mediastinoscopy are effective in establishing a histologic diagnosis.

Medical Care

The goals of superior vena cava syndrome (SVCS) management are to relieve symptoms and to attempt cure of the primary malignant process. Only a small percentage of patients with a rapid-onset superior vena cava (SVC) obstruction are at risk for life-threatening complications.

Patients with clinical superior vena cava syndrome (SVCS) often gain significant symptomatic improvement from conservative treatment measures, including elevation of the head of the bed and supplemental oxygen.

Emergency treatment is indicated when brain edema, decreased cardiac output, or upper airway edema is present. Corticosteroids and diuretics are often used to relieve laryngeal or cerebral edema, although documentation of their efficacy is questionable.

Radiotherapy has been advocated as a standard treatment for most patients with superior vena cava syndrome (SVCS). It is used as the initial treatment if a histologic diagnosis cannot

be established and the clinical status of the patient is deteriorating; however, recent reviews suggest that superior vena cava syndrome (SVCS) obstruction alone rarely represents an absolute emergency that requires treatment without a specific diagnosis.

The fractionation schedule of radiation usually includes 2-4 large initial fractions of 300-400 cGy, followed by conventional fractionation of 150-200 cGy daily, to a total dose of 3000-5000 cGy. The radiation dose depends on tumor size and radioresponsiveness. The radiation portal should include a 2-cm margin around the tumor.

During irradiation, patients improve clinically before objective signs of tumor shrinkage are evident on chest radiography. Radiation therapy palliates superior vena cava (SVC) obstruction in 70% of patients with lung carcinoma and in more than 95% with lymphoma.

In patients with superior vena cava syndrome (SVCS) secondary to non-small-cell carcinoma of the lung, radiotherapy is the primary treatment. The likelihood of patients benefiting from such therapy is high, but the overall prognosis of these patients is poor.

Chemotherapy may be preferable to radiation for patients with chemosensitive tumors.

In 1983, Maddox and associates reported on 56 patients with small-cell lung cancer who presented with superior vena cava syndrome (SVCS). Correction of superior vena cava syndrome (SVCS) was obtained in 9 (56%) of 16 patients treated with radiation therapy alone, in 23 (100%) of 23 given chemotherapy, and in 5 (83%) of 6 who received combined therapy.²⁷

The most extensive experience in superior vena cava syndrome (SVCS) management secondary to non-Hodgkin lymphoma is reported from the M.D. Anderson cancer center. Patients were treated with chemotherapy alone, chemotherapy combined with radiation therapy, or radiation therapy alone. All patients achieved complete relief of superior vena cava syndrome (SVCS) symptoms within 2 weeks of the institution of any type of treatment. No treatment modality appeared to be superior in achieving clinical improvement.²⁸

When superior vena cava syndrome (SVCS) is due to thrombus around a central venous catheter, patients may be treated with antifibrinolytics (eg, streptokinase, urokinase, recombinant tissue-type plasminogen activator) or anticoagulants (eg, heparin, oral anticoagulants). Removal of the catheter, if possible, is another option, and it should be combined with anticoagulation to avoid embolization.

In a 1988 report, Adelstein et al discuss prophylaxis against embolic events in the presence of a superior vena cava (SVC) obstruction in the management of 25 patients with malignant superior vena cava syndrome (SVCS).

Ten patients were retrospectively reviewed after having been diagnosed clinically without

venography and treated without anticoagulation. Five thromboembolic complications occurred, 2 of which proved fatal.

Fifteen patients were prospectively evaluated by means of angiography and then treated with anticoagulants. Angiographic evidence of intraluminal subclavian vein or superior vena cava (SVC) thrombosis was found in 5 of these patients, and no thromboembolic complications occurred.

Of the 20 patients who were ultimately given anticoagulation therapy, 2 had fatal intracranial hemorrhages.

The authors suggested the need for randomized prospective trials if the role of venography and anticoagulation in this syndrome is to be determined.

Surgical Care

Surgical bypass of the superior vena cava (SVC) may be a useful way to palliate symptoms in carefully selected patients.

Indications to proceed with such procedures are much less clear.

For the most part, these are patients with advanced intrathoracic disease amenable only to palliative therapy (ie, after failure of radiation therapy and chemotherapy).

Patients with benign disease appear to be the best candidates for bypass.

Superior vena cava (SVC) stenting can provide rapid symptomatic relief within few days in most patients with superior vena cava syndrome (SVCS).

Superior vena cava (SVC) stenting may provide relief of severe symptoms for patients while the histological diagnosis of the malignancy causing the obstruction is being actively pursued.

Stenting may also be indicated in patients in whom chemotherapy or radiation has failed.

Some literature recommends stenting as a first-line treatment to be performed early in the management of superior vena cava syndrome (SVCS).

Cases of excimer laser removal of pacemaker leads followed by venoplasty and stenting have been reported.

Medication

The goals of pharmacotherapy are to reduce morbidity and to prevent complications.

Corticosteroids

These agents reduce swelling in patients with cerebral or laryngeal edema.

Dexamethasone (Decadron, Dexasone)

Important therapeutic agent in a number of malignant diseases. Exerts biologic action predominately by binding to glucocorticoid receptor. For symptomatic management in tumor-associated edema.

Thrombolytics

The potential benefits of thrombolytics for the treatment of pulmonary embolism include fast dissolution of physiologically compromising pulmonary emboli, quickened recovery, prevention of recurrent thrombus formation, and rapid restoration of hemodynamic disturbances. For deep vein thrombosis, lysis of the thrombus can prevent pulmonary embolism and permanent pathologic changes, such as venous valvular dysfunction and postphlebitic syndrome.

Urokinase (Abbokinase)

Converts plasminogen to plasmin, which degrades fibrin clots, fibrinogen, and other plasma proteins.

Anticoagulants

In superior vena cava syndrome (SVCS), these agents are used mainly to prevent pulmonary embolism from superior vena cava (SVC) thrombus.

Heparin

Inhibits thrombosis by inactivating activated factor X and inhibiting conversion of prothrombin to thrombin.

Warfarin (Coumadin)

Inhibits synthesis of vitamin K–dependent coagulation factors (factors II, VII, IX, X).

Further Inpatient Care

Admit the patient to the hospital if symptoms of superior vena cava syndrome (SVCS) are moderate to severe and/or when a patient requires the administration of thrombolytic therapy or anticoagulation.

Further Outpatient Care

Instruct patients to use supportive measures, such as elevation of the head of the bed.

Carefully monitor the patient's symptoms and the adverse effects of the administered treatment. Patients should notify the physician immediately if any change in symptoms occurs.

Inpatient & Outpatient Medications

Oxygen supplementation may be provided if needed.

Antiemetics may be provided as needed to prevent nausea and vomiting.

For those patients started on steroids, taper steroids slowly, depending on the patient's condition.

Transfer

Transfer may be required for further diagnostic evaluation and surgical intervention.

Complications

Complications include laryngeal edema, cerebral edema, decreased cardiac output with hypotension, and pulmonary embolism (when an associated thrombus is present).

Prognosis

The survival of patients with superior vena cava syndrome (SVCS) depends mainly on

the course of the underlying disease.

Untreated patients and those not responding to treatment survive approximately 30 days.

TOPIC 12. THROMBOSIS OF MAJOR VEINS.

DEEP VENOUS THROMBOSIS

Introduction of acute deep venous thrombosis

Deep venous thrombosis (DVT) most commonly involves the deep veins of the leg or arm, often resulting in potentially life-threatening emboli to the lungs or debilitating venous alular dysfunction and chronic leg swelling. Deep venous thrombosis (DVT) is also one of the most prevalent medical problems today, with an annual incidence of 117 cases per 100,000. Each year in the United States, more than 200,000 people develop venous thrombosis; of those, 50,000 cases are complicated by pulmonary embolism.¹ Early recognition and appropriate treatment of deep venous thrombosis (DVT) and its complications can save many lives.

Pathophysiology

Over a century ago, Rudolf Virchow described 3 factors that are critically important in the development of venous thrombosis: (1) venous stasis, (2) activation of blood coagulation, and (3) vein damage. Over time, refinements have been made in their description and importance to the development of venous thrombosis. The origin of venous thrombosis is frequently multifactorial, with components of the triad of variable importance in individual patients.

Studies have shown that low flow sites, such as the soleal sinuses, behind venous valve pockets, and at venous confluences, are at most risk for the development of venous thrombi. However, stasis alone is not enough to facilitate the development of venous thrombosis. Experimental ligation of rabbit jugular veins for periods of up to 60 minutes have failed to consistently cause venous thrombosis. Although, patients that are immobilized for long periods of time seem to be at high risk for the development of venous thrombosis, an additional stimulus is required to develop deep venous thrombosis (DVTs).

Mechanical injury to the vein wall appears to provide an added stimulus for venous thrombosis. Hip arthroplasty patients with the associated femoral vein manipulation represent a high-risk group that cannot be explained by just immobilization, with 57% of thrombi originating in the affected femoral vein rather than the usual site of stasis in the calf.⁶

Endothelial injury can convert the normally antithrombogenic endothelium to become prothrombotic by stimulating the production of tissue factor, von Willebrand factor, and fibronectin.

Genetic mutations within the blood's coagulation cascade represent those at highest risk for the development of venous thrombosis (See Table 1).

Table 1. Relative Risk for Venous Thrombosis

Thrombophilic Defect	Relative Risk
Antithrombin deficiency	8-10
Protein C deficiency	7-10
Protein S deficiency	8-10
Factor V Leiden	3-7
Hyperhomocysteinemia	2.5
Anticardiolipin antibodies	3.2

Primary deficiencies of coagulation inhibitors antithrombin, protein C, and protein S are associated with 5-10% of all thrombotic events. Resistance of procoagulant factors to an intact anticoagulation system has also recently been described with the recognition of factor V Leiden mutation, representing 10-65% of patients with deep venous thrombosis (DVT).⁸ In the setting of venous stasis, these factors are allowed to accumulate in thrombosis prone sites, where mechanical vessel injury has occurred, stimulating the endothelium to become prothrombotic.

Components of the Virchow triad are of variable importance in individual patients, but the end result is early thrombus interaction with the endothelium. This interaction stimulates local cytokine production and facilitates leukocyte adhesion to the endothelium, both of which promote venous thrombosis. Depending on the relative balance between activated coagulation and thrombolysis, thrombus propagation occurs.

Over time, thrombus organization begins with the infiltration of inflammatory cells into the clot. This results in a fibroelastic intimal thickening at the site of thrombus attachment in most patients and a fibrous synechia in up to 11%. In many patients, this interaction between vessel wall and thrombus leads to alular dysfunction and overall vein wall fibrosis. Histological examination of vein wall remodeling after venous thrombosis has demonstrated an imbalance in connective tissue matrix regulation and a loss of regulatory venous contractility that contributes to the development of chronic venous insufficiency.

Risk factors

Many factors have been identified as known risk factors for the development of venous thrombosis. The single most powerful risk marker remains a prior history of DVT with up to 25% of acute venous thrombosis occurring in such patients. Pathologically, remnants of previous thrombi are often seen within the specimens of new acute thrombi. However, recurrent thrombosis may actually be the result of primary hypercoagulable states. Abnormalities within the coagulation cascade are the direct result of discrete genetic mutations within the coagulation cascade. Deficiencies of protein C, protein S, or antithrombin III account for approximately 5-10% of all cases of deep venous thrombosis (DVT).

Age has been well studied as an independent risk factor for venous thrombosis development. Although a 30-fold increase in incidence is noted from age 30 to age 80, the effect appears to be multifactorial, with more thrombogenic risk factors occurring in the elderly than in those younger than 40 years. Venous stasis, as seen in immobilized patients and paralyzed limbs, also contributes to the development of venous thrombosis. Autopsy studies parallel the duration of bed rest to the incidence of venous thrombosis, with 15% of patients in those studies dying within 7 days of bedrest to greater than 80% in those dying after 12 weeks.² Within stroke patients, deep venous thrombosis (DVT) is found in 53% of paralyzed limbs, compared with only 7% on the nonaffected side.

Malignancy is noted in up to 30% of patients with venous thrombosis. The thrombogenic mechanisms involve abnormal coagulation, as evidenced by 90% of cancer patients having some abnormal coagulation factors.¹⁸ Chemotherapy may increase the risk of venous thrombosis by affecting the vascular endothelium, coagulation cascades, and tumor cell lysis. The incidence has been shown to increase in those patients undergoing longer courses of

therapy for breast cancer, from 4.9% for 12 weeks of treatment to 8.8% for 36 weeks. Additionally, deep venous thrombosis (DVT) complicates 29% of surgical procedures done for malignancy.

Postoperative venous thrombosis varies depending on a multitude of patient factors, including the type of surgery undertaken. Without prophylaxis, general surgery operations typically have an incidence of deep venous thrombosis (DVT) around 20%, while orthopedic hip surgery can occur in up to 50% of patients. Based on radioactive labeled fibrinogen, about half of lower extremity thrombi develop intraoperatively. Perioperative immobilization, coagulation abnormalities, and venous injury all contribute to the development of surgical venous thrombosis.

Other clinical settings commonly reported as risk factors have also been identified and are shown in Table 2, 3

Table 2. Risk Factors for Venous Thromboembolic Disease

Risk Factor	Odds Ratio
Hospitalization with recent surgery	21.72
Hospitalization without surgery	7.98
Trauma	12.69
Malignancy	6.53
Central venous catheter	5.55
Neurologic disease with paralysis	3.04

Table 3. Risk Factors for Venous Thromboembolism

Acquired	Inherited
Advanced age	Factor V Leiden
Hospitalization/immobilization	Prothrombin 20210A
Hormone replacement therapy and oral contraceptive use	Antithrombin deficiency
	Protein C deficiency
Pregnancy and puerperium	Protein S deficiency
Prior venous thromboembolism	Factor XI elevation
Malignancy	Dysfibrinogenemia
Major surgery	Mixed Etiology
Obesity	Homocysteinemia
Nephrotic syndrome	Factor VII, VIII, IX, XI elevation
Trauma or spinal cord injury	Hyperfibrinogenemia
Long-haul travel (>6 h)	Activated protein C resistance without factor V Leiden
Varicose veins	
Antiphospholipid antibody syndrome	
Myeloproliferative disease	
Polycythemia	

Clinical and diagnostic evaluation

The clinical diagnosis of deep venous thrombosis (DVT) is difficult and fraught with uncertainty. The classic signs and symptoms of deep venous thrombosis (DVT) are those associated with obstruction to venous drainage and include pain, tenderness, and unilateral leg swelling. Other associated nonspecific findings are warmth, erythema, a palpable cord, and pain upon passive dorsiflexion of the foot (Homan sign). However, even with patients with classic symptoms, up to 46% have negative venograms. Furthermore, up to 50% of those with image-documented venous thrombosis lack any specific symptom. Deep venous thrombosis (DVT) simply cannot be diagnosed or excluded based on clinical findings; thus, diagnostic tests must be performed whenever the diagnosis of deep venous thrombosis (DVT) is being considered.

When a patient has deep venous thrombosis (DVT), symptoms may be present or absent, unilateral or bilateral, or mild or severe. Thrombus that does not cause a net venous outflow obstruction is often asymptomatic. Thrombus that involves the iliac bifurcation, the pelvic veins, or the vena cava produces leg edema that is usually bilateral rather than unilateral. High partial obstruction often produces mild bilateral edema that is mistaken for the dependent edema of right-sided heart failure, fluid overload, or hepatic or renal insufficiency.

Severe venous congestion produces a clinical appearance that can be indistinguishable from the appearance of cellulitis. Patients with a warm, swollen, tender leg should be evaluated

for both cellulitis and deep venous thrombosis (DVT) because patients with primary deep venous thrombosis (DVT) often develop a secondary cellulitis, while patients with primary cellulitis often develop a secondary deep venous thrombosis (DVT). Superficial thrombophlebitis, likewise, is often associated with a clinically inapparent underlying DVT.

If a patient is thought to have pulmonary embolism (PE) or has documented PE, the absence of tenderness, erythema, edema, or a palpable cord upon examination of the lower extremities does not rule out thrombophlebitis, nor does it imply a source other than a leg vein. More than two thirds of patients with proven PE lack any clinically evident phlebitis. Nearly one third of patients with proven PE have no identifiable source of deep venous thrombosis (DVT), despite a thorough investigation. Autopsy studies suggest that even when the source is clinically inapparent, it lies undetected within the deep venous system of the lower extremity and pelvis in 90% of cases.

Vascular Lab and Radiologic Evaluation

Duplex Ultrasound

DUS is now the most commonly performed test for the detection of infrainguinal DVT, both above and below the knee, and has a sensitivity and specificity of >95% in symptomatic patients. DUS combines real-time B-mode ultrasound with pulsed Doppler capability. Color flow imaging is useful in more technically difficult examinations, such as in the evaluation of possible calf vein DVT. This combination offers the ability to noninvasively visualize the venous anatomy, detect occluded and partially occluded venous segments, and demonstrate physiologic flow characteristics using a mobile self-contained device.

In the supine patient, normal lower extremity venous flow is phasic (Fig. 1), decreasing with inspiration in response to increased intra-abdominal pressure with the descent of the diaphragm and then increasing with expiration. When the patient is upright, the decrease in intra-abdominal pressure with expiration cannot overcome the hydrostatic column of pressure existing between the right atrium and the calf. Muscular contractions of the calf, along with the one-way venous valves, are then required to promote venous return to the heart. Flow also can be increased by leg elevation or compression and decreased by sudden elevation of intra-abdominal pressure (Valsalva's maneuver). In a venous DUS examination performed with the patient supine, spontaneous flow, variation of flow with respiration, and response of flow to

Valsalva's maneuver are all assessed. However, the primary method of detecting DVT with ultrasound is demonstration of the lack of compressibility of the vein with probe pressure on B-mode imaging. Normally, in transverse section, the vein walls should coapt with pressure. Lack of coaptation indicates thrombus.

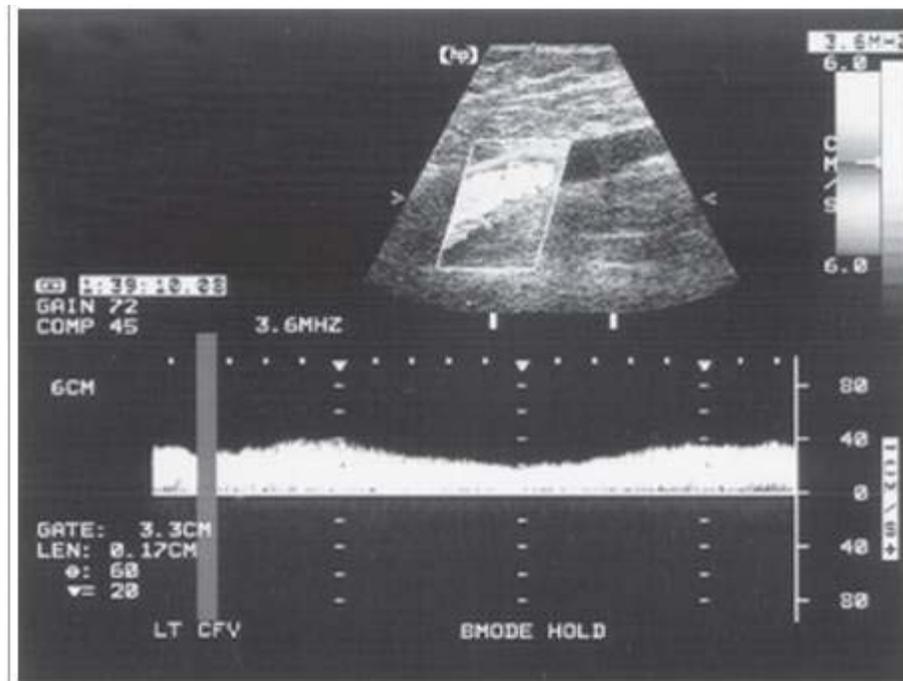


Fig. 1. Duplex ultrasound scan of a normal femoral vein with phasic flow signals.

The examination begins at the ankle and continues proximally to the groin. Each vein is visualized, and the flow signal is assessed with distal and proximal compression. Lower extremity DVT can be diagnosed by any of the following DUS findings: lack of spontaneous flow (Fig. 2), inability to compress the vein (Fig. 3), absence of color filling of the lumen by color flow DUS, loss of respiratory flow variation, and venous distention. Again, lack of venous compression on B-mode imaging is the primary diagnostic variable. Several studies comparing B-mode ultrasound to venography for the detection of femoropopliteal DVT in patients clinically suspected to have DVT report sensitivities of >91% and specificities of >97%. The ability of DUS to assess isolated calf vein DVT varies greatly, with sensitivities ranging from 50 to 93% and specificities approaching 100%.

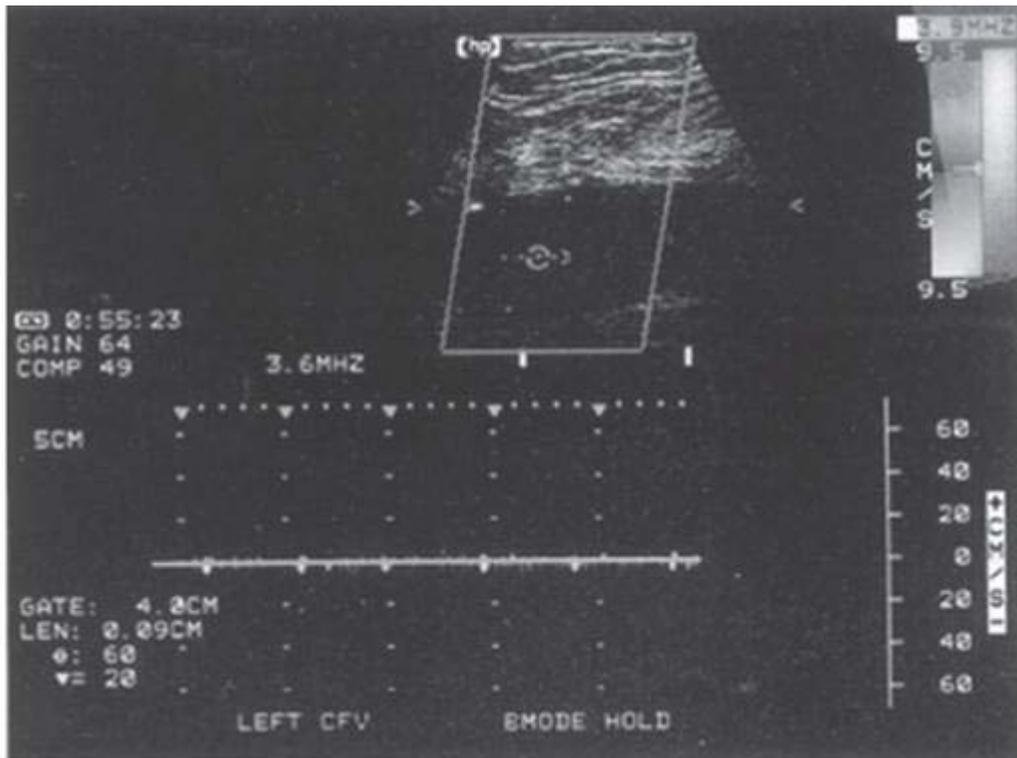


Fig. 2. Duplex ultrasound of a femoral vein containing thrombus demonstrating no flow within the femoral vein

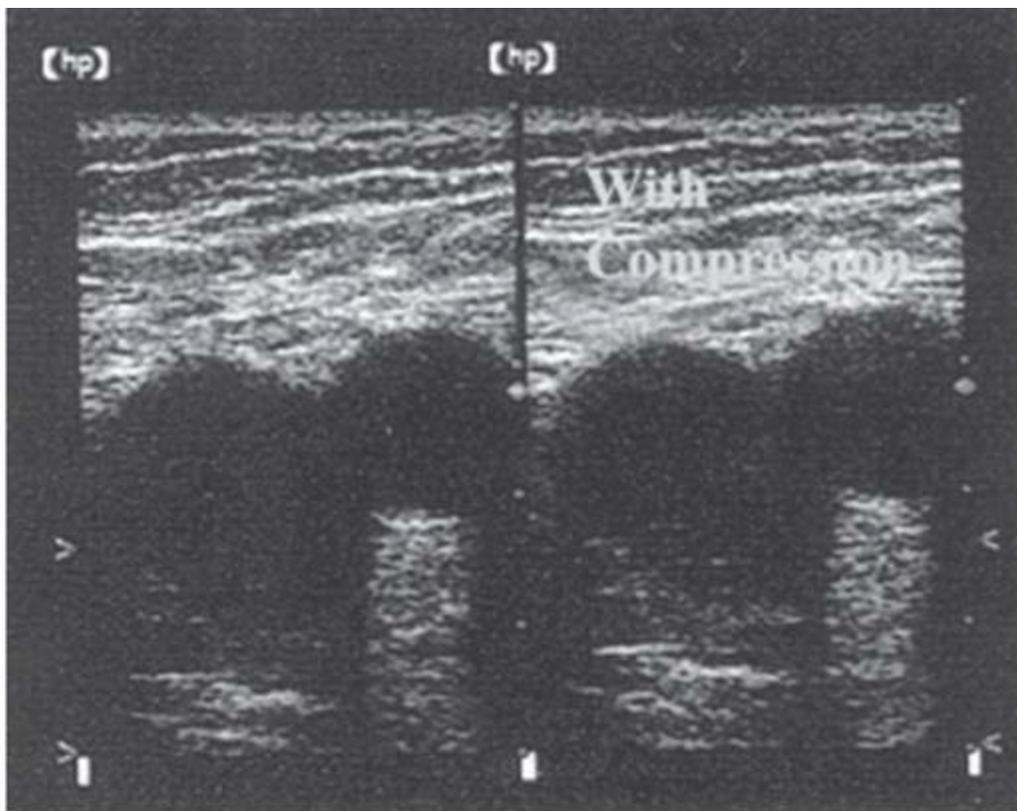


Fig. 3. B-mode ultrasound of the femoral vein in cross-section. The femoral vein does not compress.

not collapse with external compression

Impedance Plethysmography

Impedance plethysmography (IPG) was the primary noninvasive method of diagnosing DVT before the widespread use of DUS but is infrequently used today. IPG is based on the principle that resistance to the flow of electricity between two electrodes, or electrical impedance, occurs as the volume of the extremity changes in response to blood flow. Two pairs of electrodes containing aluminum strips are placed circumferentially around the leg approximately 10 cm apart and a low-level current is delivered to the two outer electrodes. A pneumatic cuff is inflated over the thigh for venous outflow obstruction and then rapidly deflated. Changes in electrical resistance resulting from lower extremity blood volume changes are quantified. IPG is less accurate than DUS for the detection of proximal DVT, with an 83% sensitivity in symptomatic patients. It is a poor detector of calf vein DVT.

Iodine 125 Fibrinogen Uptake

Iodine 125 fibrinogen uptake (FUT) is a seldom used technique that involves IV administration of radioactive fibrinogen and monitoring for increased uptake in fibrin clots. An increase of 20% or more in one area of a limb indicates an area of thrombus.²³ FUT can detect DVT in the calf, but high background radiation from the pelvis and the urinary tract limits its ability to detect proximal DVT. It also cannot be used in an extremity that has recently undergone surgery or has active inflammation. In a prospective study, FUT had a sensitivity of 73% and specificity of 71% for identification of DVT in a group of symptomatic and asymptomatic patients. Currently, FUT is primarily a research tool of historic interest.

Venography

Venography is the most definitive test for the diagnosis of DVT in both symptomatic and asymptomatic patients. It is the gold standard to which other modalities are compared. This procedure involves placement of a small catheter in the dorsum of the foot and injection of a radiopaque contrast agent. Radiographs are obtained in at least two projections. A positive study result is failure to fill the deep system with passage of the contrast medium into the

superficial system or demonstration of discrete filling defects (Fig. 4). A normal study result virtually excludes the presence of DVT. In a study of 160 patients with a normal venogram followed for 3 months, only two patients (1.3%) subsequently developed DVT and no patients experienced symptoms of PE.

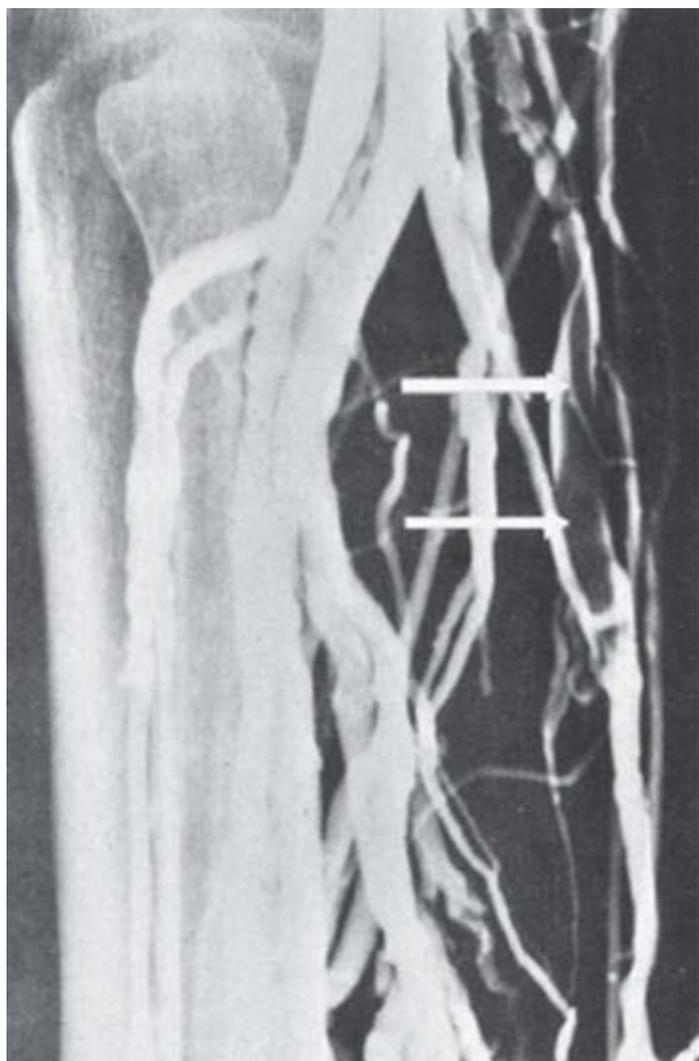


Fig. 4. Venogram showing a filling defect in the popliteal vein (*arrows*).

Venography is not routinely used for the evaluation of lower extremity DVT because of the associated complications discussed previously. Currently, venography is reserved for imaging before operative venous reconstruction and catheter-based therapy. It does, however, remain the procedure of choice in research studies evaluating methods of prophylaxis for DVT.

Laboratory analysis has also been used in aiding the diagnosis of venous thrombosis. D-dimers are degradation products of cross-linked fibrin by plasmin that are detected by diagnostic assays. Although highly sensitive, up to 97%, elevated levels are not specific with

rates as low as 35%.²⁷ Many other clinical situations can result in elevated D-dimer levels, including infection, trauma, postoperative states, and malignancy.²⁸ Additional blood work should include coagulation studies to evaluate for a hypercoagulable state, if clinically indicated. A prolonged prothrombin time or activated partial thromboplastin time does not imply a lower risk of new thrombosis. Progression of deep venous thrombosis (DVT) and PE can occur despite full therapeutic anticoagulation in 13% of patients.

Treatment

Once the diagnosis of VTE has been made, antithrombotic therapy should be initiated promptly. If clinical suspicion for VTE is high, it may be prudent to start treatment while the diagnosis is being objectively confirmed. The theoretic goals of VTE treatment are the prevention of mortality and morbidity associated with PE and the prevention of the postphlebotic syndrome. However, the only proven benefit of anticoagulant treatment for DVT is the prevention of death from PE. Treatment regimens may include antithrombotic therapy, vena caval interruption, catheter-directed or systemic thrombolytic therapy, and operative thrombectomy.

Antithrombotic Therapy

Antithrombotic therapy may be initiated with IV or SC unfractionated heparin, SC low molecular weight heparin, or SC fondaparinux (a synthetic pentasaccharide). This initial therapy usually is continued for at least 5 days, while oral vitamin K antagonists are being simultaneously administered. The initial therapy typically is discontinued when the international normalized ratio (INR) is ≥ 2.0 for 24 hours.

Unfractionated heparin (UFH) binds to antithrombin via a specific 18-saccharide sequence, which increases its activity over 1000-fold. This antithrombin-heparin complex primarily inhibits factor IIa (thrombin) and factor Xa and, to a lesser degree, factors IXa, XIa, and XIIa. In addition, UFH also binds to tissue factor pathway inhibitor, which inhibits the conversion of factor X to Xa, and factor IX to IXa. Finally, UFH catalyzes the inhibition of thrombin by heparin cofactor II via a mechanism that is independent of antithrombin.

UFH therapy is most commonly administered with an initial IV bolus of 80 units/kg or 5000 units. Weight-based UFH dosages have been shown to be more effective than standard fixed boluses in rapidly achieving therapeutic levels. The initial bolus is followed by a

continuous IV drip, initially at 18 units/kg per hour or 1300 units per hour. The half-life of IV UFH ranges from 45 to 90 minutes and is dose dependent. The level of antithrombotic therapy should be monitored every 6 hours using the activated partial thromboplastin time (aPTT), with the goal range of 1.5 to 2.5 times control values. This should correspond with plasma heparin anti-Xa activity levels of 0.3 to 0.7 IU/mL.

Initial anticoagulation with UFH may be administered SC, although this route is less commonly used. Adjusted-dose therapeutic SC UFH is initiated with 17,500 units, followed by 250 units/kg twice daily, and dosing is adjusted to an aPTT goal range similar to that for IV UFH. Fixed-dose unmonitored SC UFH is started with a bolus of 333 units/kg, followed by 250 units/kg twice daily.

Hemorrhage is the primary complication of UFH therapy. The rate of major hemorrhage (fatal, intracranial, retroperitoneal, or requiring transfusion of >2 units of packed red blood cells) is approximately 5% in hospitalized patients undergoing UFH therapy (1% in medical patients and 8% in surgical patients).²⁷ For patients with UFH-related bleeding complications, cessation of UFH is required, and anticoagulation may be reversed with protamine sulfate. Protamine sulfate binds to UFH and forms an inactive salt compound. Each milligram of protamine neutralizes 90 to 115 units of heparin, and the dosage should not exceed 50 mg IV over any 10-minute period. Side effects of protamine sulfate include hypotension, pulmonary edema, and anaphylaxis. Patients with prior exposure to protamine-containing insulin (NPH) and patients with allergy to fish may have an increased risk of hypersensitivity, although no direct relationship has been established. The protamine infusion should be terminated if any side effects occur.

In addition to hemorrhage, heparin also has unique complications. Heparin-induced thrombocytopenia (HIT) results from heparin-associated antiplatelet antibodies (HAAs) directed against platelet factor 4 complexed with heparin. HIT occurs in 1 to 5% of patients being treated with heparin. In patients with repeat heparin exposure (such as vascular surgery patients), the incidence of HAAb may be as high as 21%. HIT occurs most frequently in the second week of therapy and may lead to disastrous venous or arterial thrombotic complications. Therefore, platelet counts should be monitored periodically in patients receiving continuous heparin therapy. All forms of heparin should be stopped if there is a high clinical suspicion or confirmation of HIT [usually accompanied by an unexplained thrombocytopenia (<100,000/

L) or platelet count decrease of 30 to 50%]. Fortunately, direct thrombin inhibitors (recombinant hirudin, argatroban, bivalirudin) now are available as alternative antithrombotic

agents (see later). Another complication of prolonged high-dose heparin therapy is osteopenia, which results from impairment of bone formation and enhancement of bone resorption by heparin.

Low molecular weight heparins (LMWHs) are derived from the depolymerization of porcine UFH. Like UFH, LMWHs bind to antithrombin via a specific pentasaccharide sequence to expose an active site for the neutralization of factor Xa. However, LMWHs lack the sufficient number of additional saccharide units (18 or more), which results in less inactivation of thrombin (factor IIa). In comparison to UFH, LMWHs have increased bioavailability (>90% after SC injection), longer half-lives (approximately 4 to 6 hours), and more predictable elimination rates. Weight-based once- or twice-daily SC LMWH injections, for which no monitoring is needed, provide a distinct advantage over continuous IV infusions of UFH for treatment of VTE.

Most patients who receive therapeutic LMWH do not require monitoring. Patients who do require monitoring include those with significant renal insufficiency or failure, pediatric patients, obese patients of >120 kg, and patients who are pregnant. Monitoring may be performed using anti-Xa activity assays. However, the therapeutic anti-Xa goal range will depend on the type of LMWH and the frequency of dosing. Numerous LMWHs are commercially available. The various preparations differ in their anti-Xa and anti-IIa activities, and the treatment dosing for one LMWH cannot be extrapolated for use with another. The action of LMWHs may be partially reversed (approximately 60%) with protamine sulfate.

Numerous well-designed trials comparing SC LMWH with IV and SC UFH for the treatment of DVT have been critically evaluated in several meta-analyses. The more recent studies demonstrate a decrease in thrombotic complications, bleeding, and mortality with LMWHs. LMWHs also are associated with a decreased rate of HAAb formation and HIT (<2%) compared with UFH (at least in prophylactic doses). However, patients with established HIT should not subsequently receive LMWHs due to significant rates of cross reactivity. A major benefit of LMWHs is the ability to treat patients with VTE as outpatients. In a randomized study comparing IV UFH and the LMWH nadroparin calcium, there was no significant difference in recurrent thromboembolism (8.6% for UFH vs. 6.9% for LMWH) or major bleeding complications (2.0% for UFH vs. 0.5% for LMWH). There was a 67% reduction in mean days in the hospital for the LMWH group.

A patient with VTE should meet several criteria before receiving outpatient LMWH therapy. First, the patient should not require hospitalization for any associated conditions. The patient should not require monitoring of the LMWH therapy (which is necessary in patients

with severe renal insufficiency, pediatric patients, obese patients, and pregnant patients). The patient should be hemodynamically stable with a low suspicion of PE and have a low bleeding risk. An established outpatient system to administer LMWH and warfarin, as well as to monitor for recurrent VTE and bleeding complications, should be present. In addition, the patient's symptoms of pain and edema should be controllable at home.

Fondaparinux currently is the only synthetic pentasaccharide that has been approved by the U.S. Food and Drug Administration (FDA) for the initial treatment of DVT and PE. Its five-polysaccharide sequence binds and activates antithrombin, causing specific inhibition of factor Xa. In two large noninferiority trials, fondaparinux was compared with the LMWH enoxaparin for the initial treatment of DVT and with IV UFH for the initial treatment of PE. The rates of recurrent VTE ranged from 3.8 to 5%, with rates of major bleeding of 2 to 2.6%, for all treatment arms. The drug is administered SC once daily with a weight-based dosing protocol: 5 mg, 7.5 mg, or 10 mg for patients weighing <50 kg, 50 to 100 kg, or >100 kg, respectively. The half-life of fondaparinux is approximately 17 hours in patients with normal renal function. There are rare case reports of fondaparinux-induced thrombocytopenia.

Direct-thrombin inhibitors (DTIs) include recombinant hirudin, argatroban, and bivalirudin. These antithrombotic agents bind to thrombin, inhibiting the conversion of fibrinogen to fibrin as well as thrombin-induced platelet activation. These actions are independent of antithrombin. The direct thrombin inhibitors should be reserved for (a) patients in whom there is a high clinical suspicion or confirmation of HIT, and (b) patients who have a history of HIT or test positive for heparin-associated antibodies. In patients with established HIT, DTIs should be administered for at least 7 days, or until the platelet count normalizes. Warfarin may then be introduced slowly, overlapping therapy with a DTI for at least 5 days.⁴¹ Because bivalirudin is approved primarily for patients with or without HIT who undergo percutaneous coronary intervention, it is not discussed here in further detail.

Commercially available hirudin is manufactured using recombinant DNA technology. It is indicated for the prophylaxis and treatment of patients with HIT. In patients with normal renal function, recombinant hirudin is administered in an IV bolus dose of 0.4 mg/kg, followed by a continuous IV infusion of 0.15 mg/kg per hour. The half-life ranges from 30 to 60 minutes. The aPTT is monitored, starting approximately 4 hours after initiation of therapy, and dosage is adjusted to maintain an aPTT of 1.5 to 2.5 times the laboratory normal value. The less commonly used ecarin clotting time is an alternative method of monitoring. Because recombinant hirudin is eliminated via renal excretion, significant dosage adjustments are required in patients with renal insufficiency.

Argatroban is indicated for the prophylaxis and treatment of thrombosis in HIT. It also is approved for patients with, or at risk for, HIT who undergo percutaneous coronary intervention. Antithrombotic prophylaxis and therapy are initiated with a continuous IV infusion of 2 g/kg per minute, without the need for a bolus. The half-life ranges from 39 to 51 minutes, and the dosage is adjusted to maintain an aPTT of 1.5 to 3 times normal. Large initial boluses and higher rates of continuous infusion are reserved for patients with coronary artery thrombosis and myocardial infarction. In these patients, therapy is monitored using the activated clotting time. Argatroban is metabolized by the liver, and the majority is excreted via the biliary tract. Significant dosage adjustments are needed in patients with hepatic impairment. There is no reversal agent for argatroban.

Vitamin K antagonists, which include warfarin and other coumarin derivatives, are the mainstay of long-term antithrombotic therapy in patients with VTE. Warfarin inhibits the γ -carboxylation of vitamin K–dependent procoagulants (factors II, VII, IX, X) and anticoagulants (proteins C and S), which results in the formation of less functional proteins. Warfarin usually requires several days to achieve its full effect, because normal circulating coagulation proteins must first undergo their normal degradation. Factors X and II have the longest half-lives, in the range of 36 and 72 hours, respectively. In addition, the steady-state concentration of warfarin is usually not reached for 4 to 5 days.

Warfarin therapy usually is monitored by measuring the INR, calculated using the following equation:

$$\text{INR} = \left(\frac{\text{patient prothrombin time/laboratory normal prothrombin time}}{\text{ISI}} \right)^{\text{ISI}}$$

where *ISI* is the international sensitivity index. The ISI describes the strength of the thromboplastin that is added to activate the extrinsic coagulation pathway. The therapeutic target INR range is usually 2.0 to 3.0, but the response to warfarin is variable and depends on liver function, diet, age, and concomitant medications. In patients receiving anticoagulation therapy without concomitant thrombolysis or venous thrombectomy, the vitamin K antagonist may be started on the same day as the initial parenteral anticoagulant, usually at doses ranging from 5 to 10 mg. Smaller initial doses may be needed in older and malnourished patients, in those with liver disease or congestive heart failure, and in those who have recently undergone major surgery.

The recommended duration of warfarin antithrombotic therapy is increasingly being

stratified based on whether the DVT was provoked or unprovoked, whether it was the first or a recurrent episode, where the DVT is located, and whether malignancy is present. Current American College of Chest Physicians (ACCP) recommendations for duration of warfarin therapy are summarized in Table 24-3. In patients with proximal DVT, several randomized clinical trials have demonstrated that shorter-term antithrombotic therapy (4 to 6 weeks) is associated with a higher rate of recurrence than 3 to 6 months of anticoagulation. In these trials, most of the patients with transient risk factors had a low rate of recurrent VTE; most of the recurrences were in patients with continuing risk factors. These studies support the ACCP recommendation that 3 months of anticoagulation is sufficient to prevent recurrent VTE in patients whose DVT occurred around the time of a transient risk factor (e.g., hospitalization, orthopedic or major general surgery).

Table 4. Summary of American College of Chest Physicians Recommendations Regarding Duration of Long-Term Antithrombotic Therapy for Deep Vein Thrombosis (DVT)

Clinical Subgroup	Antithrombotic Treatment Duration
First episode DVT/transient risk	VKA for 3 mo
First episode DVT/unprovoked	VKA for at least 3 mo
	Consider for long-term therapy if:
	• Proximal DVT
	• Minimal bleeding risk
	• Stable coagulation monitoring
Distal DVT/unprovoked	VKA for 3 mo
Second episode DVT/unprovoked	VKA long-term therapy
DVT and cancer	LMWH 3–6 mo
	Then VKA or LMWH indefinitely until cancer resolves

LMWH = low molecular weight heparin; VKA = vitamin K antagonist.

Source: Adapted with permission from Kearon C, Kahn SR, Agnelli G, et al:

Antithrombotic therapy for venous thromboembolic disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th edition). *Chest* 133:454S, 2008.

In contrast to patients with thrombosis related to transient risk factors, patients with idiopathic VTE are much more likely to develop recurrence (rates as high as 40% at 10 years). In this latter group of patients, numerous clinical trials have compared 3 to 6 months of anticoagulation therapy with extended-duration warfarin therapy, both at low intensity (INR of 1.5 to 2.0) and at conventional intensity (INR of 2.0 to 3.0). In patients with idiopathic DVT, extended-duration antithrombotic therapy is associated with a relative reduction in the rate of recurrent VTE by 75% to >90%. In addition, conventional-intensity warfarin reduces the risk

even further compared with low-intensity warfarin (0.7 events per 100 person-years vs. 1.9 events per 100 person-years), but the rate of bleeding complications is no different.

In patients with VTE in association with a hypercoagulable condition, the optimal duration of anticoagulation therapy is influenced more by the clinical circumstances at the time of the VTE (idiopathic vs. secondary) than by the actual presence or absence of the more common thrombophilic conditions. In patients with VTE related to malignancy, increasing evidence suggests that longer-term therapy with LMWH (up to 6 months) is associated with a lower VTE recurrence rate than treatment using conventional vitamin K antagonists.

The primary complication of warfarin therapy is hemorrhage, and the risk is related to the magnitude of INR prolongation. Depending on the INR and the presence of bleeding, warfarin anticoagulation may be reversed by (a) omitting or decreasing subsequent dosages, (b) administering oral or parenteral vitamin K, or (c) administering fresh-frozen plasma, prothrombin complex concentrate, or recombinant factor VIIa. Warfarin therapy rarely may be associated with the development of skin necrosis and limb gangrene. These conditions occur more commonly in women (4:1), and the most commonly affected areas are the breast, buttocks, and thighs. This complication, which usually occurs in the first days of therapy, is occasionally, but not exclusively, associated with protein C or S deficiency and malignancy. Patients who require continued anticoagulation may restart low-dose warfarin (2 mg) while receiving concomitant therapeutic heparin. The warfarin dosage is then gradually increased over a 1- to 2-week period.

Systemic and Catheter-Directed Thrombolysis

Patients with extensive proximal DVT may benefit from systemic thrombolysis or catheter-directed thrombolysis, which can potentially reduce acute symptoms more rapidly than anticoagulation alone. These techniques also may decrease the development of postthrombotic syndrome. Several thrombolysis preparations are available, including streptokinase, urokinase, alteplase (recombinant tissue plasminogen activator), reteplase, and tenecteplase. All these agents share the ability to convert plasminogen to plasmin, which leads to the degradation of fibrin. They differ with regard to their half-lives, their potential for inducing fibrinogenolysis (generalized lytic state), their potential for antigenicity, and their FDA-approved indications for use.

Streptokinase is purified from beta-hemolytic *Streptococcus* and is approved for the treatment of acute myocardial infarction, PE, DVT, arterial thromboembolism, and occluded

central lines and arteriovenous shunts. It is not specific for fibrin-bound plasminogen, however, and its use is limited by its significant rates of antigenicity. Fevers and shivering occur in 1 to 4% of patients. Urokinase is derived from human neonatal kidney cells, grown in tissue culture. Currently, it is only approved for lysis of massive PE or PE associated with unstable hemodynamics. Alteplase, reteplase, and tenecteplase all are recombinant variants of tissue plasminogen activator. Alteplase is indicated for the treatment of acute myocardial infarction, acute ischemic stroke, and acute massive PE. However, it often is used for catheter-directed thrombolysis of DVT. Reteplase and tenecteplase are indicated only for the treatment of acute myocardial infarction.

Systemic thrombolysis was evaluated in numerous older prospective and randomized clinical trials, and its efficacy was summarized in a recent Cochrane Review. In 12 studies involving over 700 patients, systemic thrombolysis was associated with significantly more clot lysis [relative risk (RR) 0.24 to 0.37] and significantly less postthrombotic syndrome (RR 0.66). However, venous function was not significantly improved. In addition, more bleeding complications did occur (RR 1.73), but the incidence appears to have decreased in later studies, probably due to improved patient selection.

In an effort to minimize bleeding complications and increase efficacy, catheter-directed thrombolytic techniques have been developed for the treatment of symptomatic DVT. With catheter-directed therapy, venous access may be achieved through percutaneous catheterization of the ipsilateral popliteal vein, retrograde catheterization through the contralateral femoral vein, or retrograde cannulation from the internal jugular vein. Multi-side-hole infusion catheters, with or without infusion wires, are used to deliver the lytic agent directly into the thrombus.

The efficacy of catheter-directed urokinase for the treatment of symptomatic lower extremity DVT has been reported in a large multicenter registry. Two hundred twenty-one patients with iliofemoral DVT and 79 patients with femoropopliteal DVT were treated with catheter-directed urokinase for a mean of 53 hours. Complete lysis was seen in 31% of the limbs, 50 to 99% lysis in 52% of the limbs, and <50% lysis in 17%. Overall, 1-year primary patency was 60%. Patency was higher in patients with iliofemoral DVT than in patients with femoropopliteal DVT (64% vs. 47%, $P < .01$). In addition, patients with acute symptoms (≤ 10 days) had a greater likelihood of complete lysis (34%) than patients with chronic symptoms (> 10 days; 19%). Major bleeding occurred in 11%, but neurologic involvement and mortality were rare (both 0.4%). Adjunctive stent placement to treat residual stenosis and/or short

segment occlusion was required in 103 limbs.

One small randomized trial and numerous other retrospective studies have demonstrated similar rates of thrombolysis, with some also showing improved valve preservation and quality of life. Combining thrombolysis with percutaneous thrombus fragmentation and extraction has the added benefit of decreasing the infusion time, the hospital stay, and the overall cost of treatment. These studies, as well as the current ACCP guidelines, suggest that catheter-directed thrombolysis (with adjunctive angioplasty, venous stenting, and pharmacomechanical fragmentation and extraction) may be useful in selected patients with extensive iliofemoral DVT. Patients should have a recent onset of symptoms (<14 days), good functional status, decent life expectancy, and low bleeding risk.

Inferior Vena Caval Filters

Since the introduction of the Kimray-Greenfield filter in the United States in 1973, numerous vena caval filters have been developed. Although the designs are variable, they all prevent pulmonary emboli, while allowing continuation of venous blood flow through the IVC. Early filters were placed surgically through the femoral vein. Currently, less invasive techniques allow percutaneous filter placement through a femoral vein, internal jugular vein, or small peripheral vein under fluoroscopic or ultrasound guidance. Complications associated with IVC filter placement include insertion site thrombosis, filter migration, erosion of the filter into the IVC wall, and IVC thrombosis. The rate of fatal complications is <0.12%.

Placement of an IVC filter is indicated for patients who develop recurrent DVT (significant propagation of the original thrombus or proximal DVT at a new site) or PE despite adequate anticoagulation therapy and for patients with pulmonary hypertension who experience recurrent PE. In patients who receive IVC filters for these indications, therapeutic anticoagulation should be continued. The duration of anticoagulation is determined by the underlying VTE and not by the presence of the IVC filter itself. Practically speaking, however, many patients who require an IVC filter for recurrent VTE are the same ones who would benefit most from indefinite anticoagulation. The other major indication for placement of an IVC filter is a contraindication to, or complication of, anticoagulation therapy in the presence of an acute proximal DVT. In patients who are not able to receive anticoagulants due to recent surgery or trauma, the clinician should continually reassess if antithrombotic agents may be

started safely at a later date. Even some patients who develop anticoagulation-associated bleeding complications may be able to restart therapy at a lower intensity of anticoagulation later in the hospital course. As before, the clinical circumstances surrounding the VTE should determine the duration of anticoagulation.

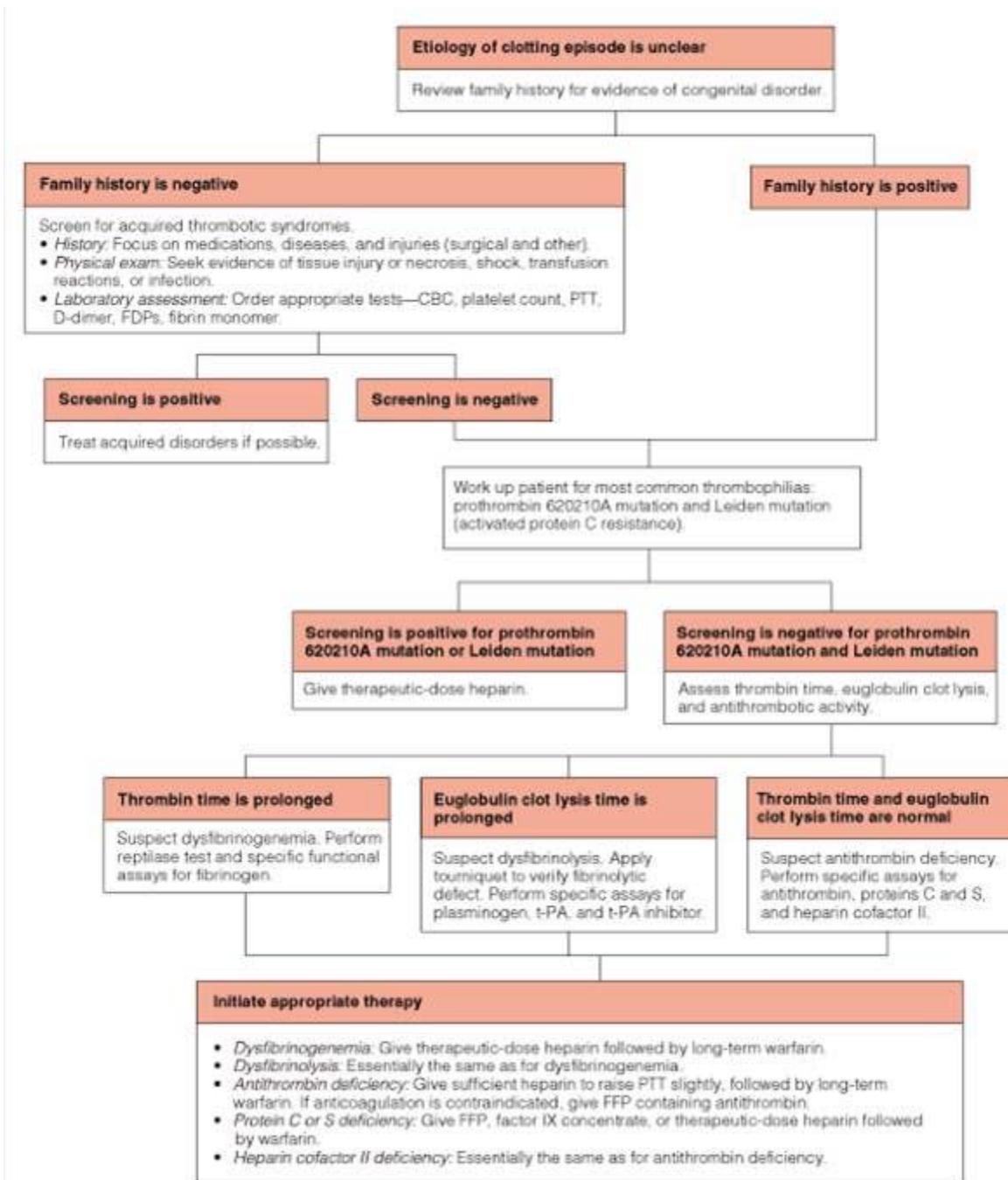
Placement of permanent IVC filters has been evaluated as an adjunct to routine anticoagulation in patients with proximal DVT. In this study, routine IVC filter placement did not prolong early or late survival in patients with proximal DVT but did decrease the rate of PE (hazard ratio, 0.22; 95% confidence interval, 0.05 to 0.90). An increased rate of recurrent DVT was seen in patients with IVC filters (hazard ratio, 1.87; 95% confidence interval, 1.10 to 3.20). More controversial indications for IVC filter placement include prophylaxis against PE in patients receiving catheter-directed thrombolysis and in high-risk patients without established DVT or PE.

Hypercoagulability States

Certain patients seem to have a tendency to clot spontaneously. So-called hypercoagulability states were long thought to exist, but they were difficult to document except on clinical grounds. Currently, however, these clotting tendencies are better understood, thanks in large part to recognition of the role of antithrombins. If an antithrombin deficiency exists and clotting goes unchecked, activation of a clotting cascade could theoretically progress to clotting throughout the entire vasculature. Another important development was the recognition that deficiencies of certain natural clot-removing substances in the blood may lead to a clinical thrombotic tendency. Both types of deficiency can be either acquired or congenital.

Screening

When the etiology of a clotting episode is unclear, the family history should be reviewed for evidence of a congenital disorder. Even if the history is negative, the patient should be screened for both acquired and congenital disorders (table 6).



Acquired Clotting Conditions

Screening for acquired clotting conditions [see Table 7] is based on the history, physical examination, and laboratory assessment. The history should include medications, diseases, and surgical procedures or other injuries. Examination may disclose causes of hypercoagulability. Soft tissue injury, for example, is a potent activator of the coagulation system. If the injury is severe enough, it may be capable of causing a severe acquired coagulopathy. The problem is usually obvious, but on occasion, detailed study may be necessary to identify tissue damage or ischemic injury to bowel or extremities. Hypovolemia—especially hypovolemic

shock markedly reduces clotting time: blood from a patient in profound shock may clot instantaneously in the syringe as it is being drawn. The breakdown of red cells in a hemolytic transfusion reaction can cause clotting. Severe infection, especially from gram-negative organisms, is a potent activator of coagulation.

Table 7. Etiology of Acquired Hypercoagulability

Tissue and cellular damage
Shock
Trauma
Surgery
Tissue necrosis
Transfusion necrosis
Drugs
Estrogens
Drug reactions and interactions
Heparin platelet antibody
Warfarin
Disease
Blood dyscrasias
Cancer
Diabetes
Homocystinuria
Hyperlipidemia
Presence of lupuslike anticoagulant
Severe infection
Pregnancy

Of the acquired hypercoagulability syndromes, Trousseau syndrome is a particularly important condition for surgeons to recognize because it occurs in the surgical population (cancer patients) and must be treated with heparin (it is unresponsive to warfarin). It occurs when an adenocarcinoma secretes a protein recognized by the body as tissue factor, resulting in multiple episodes of venous thromboembolism over time (migratory thrombophlebitis). Simple depletion of vitamin K-dependent factors is ineffective. Patients should receive therapeutic-dose heparin indefinitely or until the cancer is brought into remission.

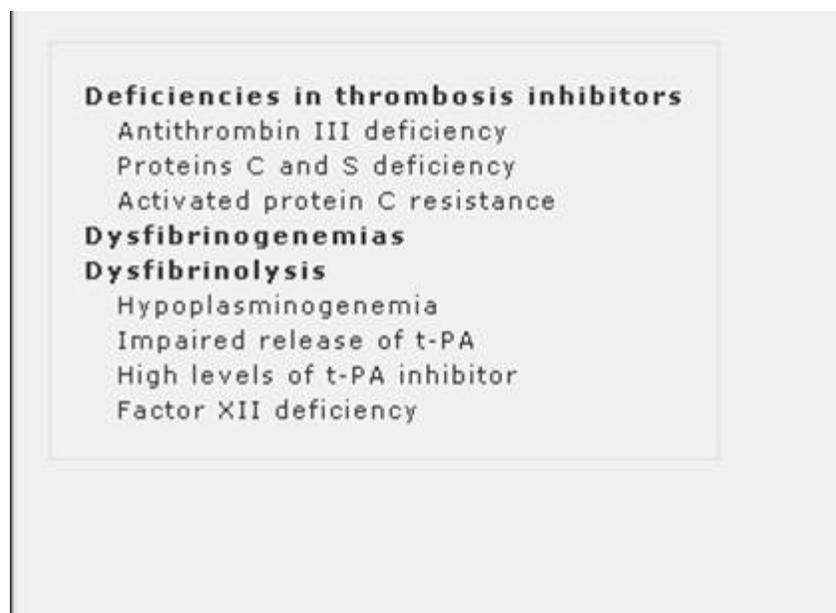
Laboratory screening may facilitate diagnosis. A complete blood count may document the presence of polycythemia or leukemia. Thrombocytopenia may be a manifestation of a hypercoagulable disorder, and thrombocytopenia after the administration of heparin raises the possibility of intravascular platelet aggregation. A prolonged aPTT is suggestive of lupuslike anticoagulant. Increased levels of D-dimers, fibrin degradation products (FDPs), or fibrin

monomers in the plasma may reflect low-grade intravascular coagulation.

Congenital Clotting Conditions

Congenital clotting tendencies can result from deficiencies in inhibitors of thrombosis (antithrombin, proteins C and S, and possibly heparin cofactor II), dysfibrinogenemias, or dysfibrinolysis [see Table 8]. Most congenital clotting defects are transmitted as an autosomal dominant trait. A negative family history does not preclude inherited thrombophilia, because the defects have a low penetrance, and fresh mutations may have occurred.

Table 8. Congenital Clotting Disorders



Deficiencies in thrombosis inhibitors
Antithrombin III deficiency
Proteins C and S deficiency
Activated protein C resistance
Dysfibrinogenemias
Dysfibrinolysis
Hypoplasminogenemia
Impaired release of t-PA
High levels of t-PA inhibitor
Factor XII deficiency

INITIAL LABORATORY ASSESSMENT

Initial evaluation of a patient with an unexplained thrombotic episode should be directed at the most common causes of hypercoagulability. Acquired causes of clotting are more commonly seen by surgeons than congenital causes and therefore must be excluded first. If a clotting disorder is determined to be congenital, a laboratory workup should be undertaken. Several of the relevant assays "specifically, the functional assays" should be performed after the acute phase of the disorder has passed. If they are performed during the acute phase, levels of several antithrombotics (e.g., antithrombin and proteins C and S) will be misleadingly low not because deficiencies of these substances caused the underlying thrombotic process but because they were consumed in that process.

Specific Causes Of Thrombotic Tendency

The most common congenital causes of accelerated clotting are mutations of prothrombin (prothrombin G20210A mutation) and factor V (Leiden mutation, or activated protein C resistance). The prevalence of each of these ranges from 1% to 5% in the general population and may be much higher in specific ethnic subpopulations.¹ Each mutation may be identified conclusively by means of polymerase chain reaction (PCR) techniques. Detection of these mutations, unlike assays for antithrombin and proteins C and S, is not dependent on the patient's current inflammatory state. It must be remembered that the presence of one of these mutations, especially in the heterozygous form, does not imply that it is the sole cause of thrombosis. In many patients, a second precipitating factor must be present for the pathologic genetic thrombotic potential to be manifested.

Prothrombin G20210A Mutation

The prothrombin G20210A mutation is known to involve a single amino acid substitution in the prothrombin gene, but precisely how this increases the risk of venous thromboembolism is unclear. The one apparent manifestation of the mutation is a 15% to 40% increase in circulating prothrombin. Regardless of the mechanism at work, patients who are at least heterozygous for the trait are at two- to sixfold greater risk for venous thromboembolism than those without the mutation.

Resistance to Activated Protein C (Factor V Leiden)

Resistance of human clotting factors to inactivation by activated protein C is believed to be the most common inherited procoagulant disorder.¹¹⁴ Normally, activated factor V is degraded by activated protein C in the presence of membrane surface as part of normal regulation of thrombosis. Activated protein C resistance is caused by a single substitution mutation in the factor V gene, which is passed in an autosomal dominant fashion. The mutant factor V that results, termed factor V Leiden, is resistant to inactivation by activated protein C and thus has a greater ability to activate thrombin and accelerate clotting.

Two techniques are commonly used to diagnose this disorder. The first is a functional assay that compares a standard aPTT to one performed in the presence of exogenous activated

protein C. If the latter aPTT does not exhibit significant prolongation, the patient is probably resistant to activated protein C. The results of this assay must be interpreted with caution if the patient is still in the acute phase of the illness. The second technique, which is more reliable, involves direct detection of the mutation via PCR analysis of DNA.

Antithrombin Deficiency

Antithrombin (once termed antithrombin III) is a 65 kd protein that decelerates the coagulation system by inactivating activated factors, primarily factor Xa and thrombin but also factors XII, XI, and IX. Antithrombin therefore acts as a scavenger of activated clotting factors. Its activity is enhanced 100-fold by the presence of heparans on the endothelial surface and 1,000-fold by administration of exogenous heparin.

Congenital antithrombin deficiency occurs in approximately 0.01% to 0.05% of the general population and 2% to 4% of patients with venous thrombosis. The trait is passed on as an autosomal dominant trait, with the heterozygous genotype being incompatible with life. Antithrombin-deficient patients are at increased risk for thromboembolism when their antithrombin activity falls below 70% of normal.

Patients with congenital antithrombin deficiency frequently present after a stressful event. They usually have DVT but sometimes have PE. If anticoagulation is not contraindicated, the treatment of choice is heparin at a dosage sufficient to raise the aPTT to the desired level, followed by warfarin. If anticoagulation is contraindicated (as it is during the peripartum period), antithrombin concentrate should be given to raise the antithrombin activity to 80% to 120% of normal during the period when anticoagulants cannot be given.

Acquired antithrombin deficiency is a well-recognized entity. In most patients undergoing severe systemic stress, antithrombin levels fall below normal. Patients with classic risk factors for venous thromboembolism tend to have the lowest levels.

Protein C and Protein S Deficiency

Protein C is a 62 kd glycoprotein with a half-life of 6 hours. Because it is vitamin K dependent, a deficiency will develop in the absence of vitamin K. Acquired protein C deficiency is seen in liver disease, malignancy, infection, the postoperative state, and disseminated intravascular coagulation. Protein C deficiency occurs in approximately 4% to 5% of patients younger than 40 to 45 years who present with unexplained venous thrombosis.

It is transmitted as an autosomal dominant trait, and the family history is usually positive for a clotting tendency. Protein C levels range from 70% to 164% of normal in patients without a clotting tendency; levels below 70% of normal are associated with a thrombotic tendency. The most appropriate tests for screening are functional assays; there are cases of dysfunctional protein C deficiency in which protein C antigen levels are normal but protein C activity is low, and these would not be detected by the usual immunoassays.

Protein S is a vitamin K-dependent protein that acts as a cofactor for activated protein C by enhancing protein C-induced inactivation of activated factor V. The incidence of protein S deficiency is similar to that of protein C deficiency. It is transmitted as a dominant trait, and the family history is often positive for a thrombotic tendency.

Hyperhomocysteinemia

Although hyperhomocysteinemia is more commonly associated with cardiac disease and arterial thrombosis, it may also be associated with an increased incidence of venous thromboembolism. This association is not as strong as those already discussed. Accordingly, anticoagulation of asymptomatic patients with elevated homocysteine levels is not currently recommended.

Dysfibrinogenemia

More than 100 qualitative abnormalities of fibrinogen (dysfibrinogenemias) have been reported. Dysfibrinogenemias are inherited in an autosomal dominant manner, with most patients being heterozygous. Most patients with dysfibrinogenemia have either no clinical symptoms or symptoms of a bleeding disorder; a minority (about 11%) have clinical features of a recurrent thromboembolic disorder. Congenital dysfibrinogenemias associated with thrombosis account for about 1% of cases of unexplained venous thrombosis occurring in young people. The most commonly observed functional defect in such dysfibrinogenemias is abnormal fibrin monomer polymerization combined with resistance to fibrinolysis. Decreased binding of plasminogen and increased resistance to lysis by plasmin have been noted.

In addition to a prolonged TT, patients who have dysfibrinogenemia associated with thromboembolism may have a prolonged INR. The diagnosis is confirmed if the reptilase time is also prolonged. Measured with clotting techniques, fibrinogen levels may be slightly or moderately low; measured immunologically, levels may be normal or even increased.

Dysfibrinolysis

Fibrinolysis can be impaired by inherited deficiencies of plasminogen, defective release of t-PA from the vascular endothelium, and high plasma levels of regulatory proteins (e.g., t-PA inhibitors). In addition, factor XII (contact factor) deficiency may induce failure of fibrinolysis activation.

Inherited plasminogen deficiency is probably only rarely responsible for unexplained DVT in young patients. It is transmitted as an autosomal dominant trait. In heterozygous persons with a thrombotic tendency, plasminogen activity is about one half normal (3.9 to 8.4 $\mu\text{mol/ml}$). The euglobulin clot lysis time is prolonged. Functional assays should be carried out, and there should be full transformation of plasminogen into plasmin activators.

The important role of t-PA inhibitors I and II in the regulation of fibrinolysis is well defined. In normal plasma, t-PA inhibitor I is the primary inhibitor for both t-PA and urokinase. Release of t-PA inhibitor I by platelets results in locally increased concentrations where platelets accumulate. The ensuing local inhibition of fibrinolysis may help stabilize the hemostatic plug. t-PA inhibitor II is present in and secreted by monocytes and macrophages.

Factor XII deficiency is a rare cause of impaired fibrinolysis. Initial contact activation of factor XII not only results in activation of the clotting cascade and of the inflammatory response but also leads to plasmin generation. This intrinsic activation of fibrinolysis requires factor XII, prekallikrein, and high-molecular-weight kininogen. Patients with factor XII deficiencies can be identified by a prolonged aPTT in the absence of clinical bleeding.

Operative Venous Thrombectomy

In patients with acute iliofemoral DVT, surgical therapy is generally reserved for patients who worsen with anticoagulation therapy and those with phlegmasia cerulea dolens and impending venous gangrene. If the patient has phlegmasia cerulea dolens, a fasciotomy of the calf compartments is first performed. In iliofemoral DVT, a longitudinal venotomy is made in the common femoral vein and a venous balloon embolectomy catheter is passed through the thrombus into the IVC and pulled back several times until no further thrombus can be extracted. The distal thrombus in the leg is removed by manual pressure beginning in the foot. This is accomplished by application of a tight rubber elastic wrap beginning at the foot and extending to the thigh. If the thrombus in the femoral vein is old and cannot be extracted, the

vein is ligated. For a thrombus that extends into the IVC, the IVC is exposed transperitoneally and the IVC is controlled below the renal veins. The IVC is opened and the thrombus is removed by gentle massage. An intraoperative completion venogram is obtained to determine if any residual thrombus or stenosis is present. If a residual iliac vein stenosis is present, intraoperative angioplasty and stenting can be performed. In most cases, an arteriovenous fistula is then created by anastomosing the great saphenous vein (GSV) end to side with the superficial femoral artery in an effort to maintain patency of the thrombectomized iliofemoral venous segment. Heparin is administered postoperatively for several days. Warfarin anticoagulation is maintained for at least 6 months after thrombectomy. Complications of iliofemoral thrombectomy include PE in up to 20% of patients and death in <1% of patients.

One study followed 77 limbs for a mean of 8.5 years after thrombectomy for acute iliofemoral DVT. In limbs with successful thrombectomies, valvular competence in the thrombectomized venous segment was 80% at 5 years and 56% at 10 years. More than 90% of patients had minimal or no symptoms of postthrombotic syndrome. There were 12 (16%) early thrombectomy failures. Patients were required to wear compression stockings for at least 1 year after thrombectomy.

Survival rates for surgical pulmonary embolectomy have improved over the past 20 years with the addition of cardiopulmonary bypass. Emergency pulmonary embolectomy for acute PE is rarely indicated. Patients with preterminal massive PE (Fig. 5) for whom thrombolysis has failed or who have contraindications to thrombolytics may be candidates for this procedure. Open pulmonary artery embolectomy is performed through a posterolateral thoracotomy with direct visualization of the pulmonary arteries. Mortality rates range between 20 and 40%.

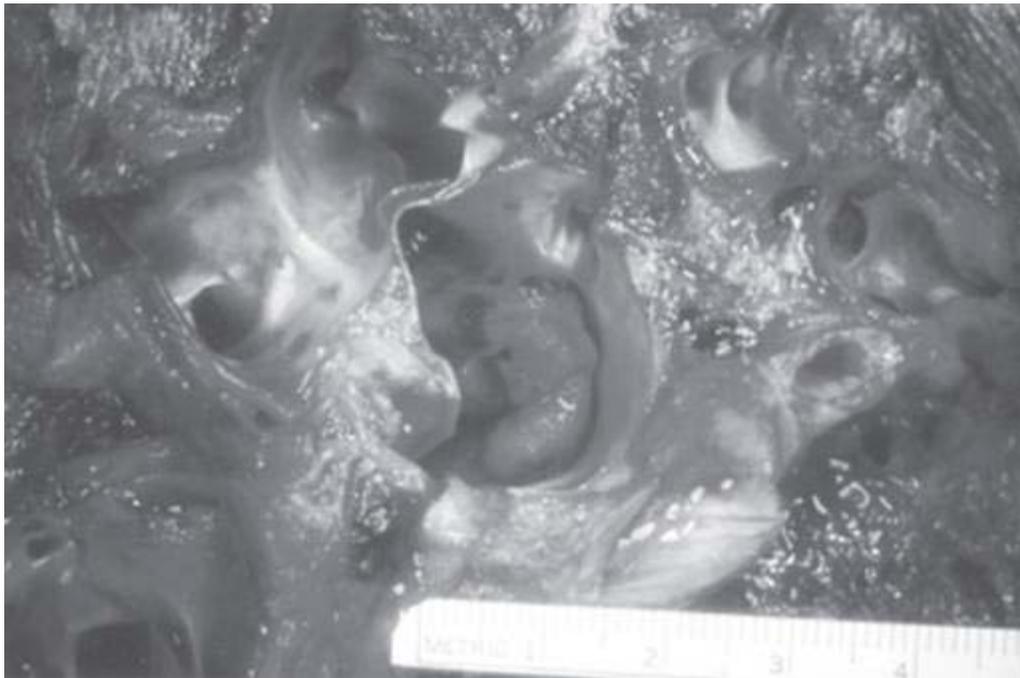


Fig. 5. Autopsy specimen showing a massive pulmonary embolism.

Percutaneous catheter-based techniques for removal of a PE involve mechanical thrombus fragmentation or embolectomy using suction devices. Mechanical clot fragmentation is followed by catheter-directed thrombolysis. Results of catheter-based fragmentation are based on small case series. In a study in which a fragmentation device was used in 10 patients with acute massive PE, fragmentation was successful in 7 patients with a mortality rate of 20%.⁶⁵ Transvenous catheter pulmonary suction embolectomy has also been performed for acute massive PE with a reported 76% successful extraction rate and a 30-day survival of 70%.⁶⁶

Prophylaxis

Patients who undergo major general surgical, gynecologic, urologic, and neurosurgical procedures without thromboprophylaxis have a significant incidence of perioperative DVT (15 to 40%). The incidence is even higher with major trauma (40 to 80%), hip and knee replacement surgery (40 to 60%), and spinal cord injury (60 to 80%). The goal of prophylaxis is to reduce the mortality and morbidity associated with VTE. The first manifestation of VTE may be a life-threatening PE (Fig. 6), and as indicated earlier, clinical evaluation to detect DVT before PE is unreliable.

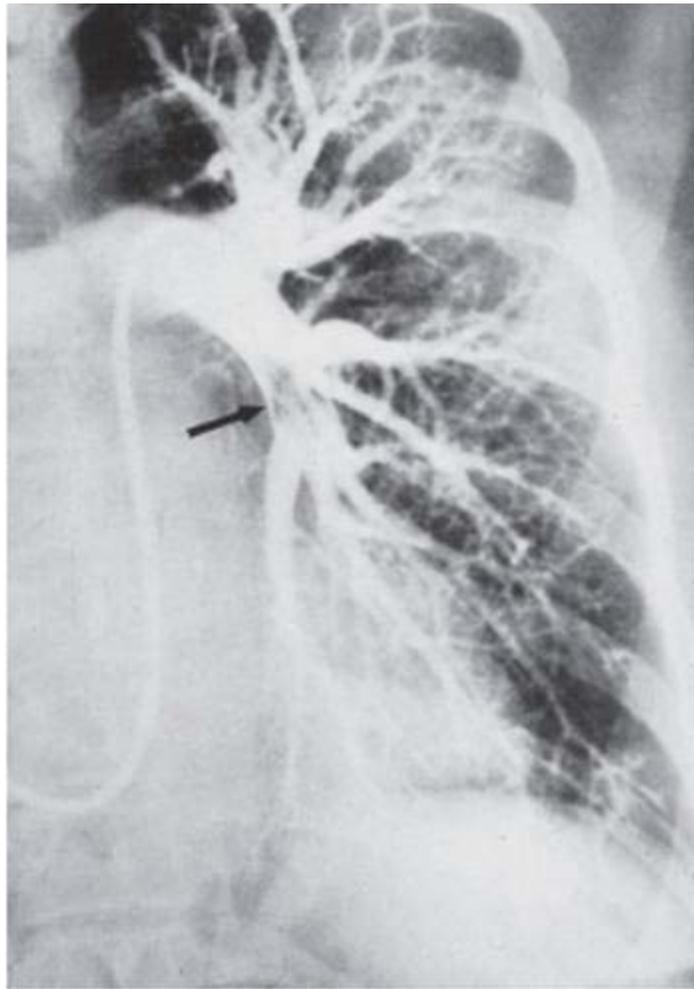


Fig. 6. Pulmonary angiogram showing a pulmonary embolism (*arrow*).

Effective methods of VTE prophylaxis involve the use of one or more pharmacologic or mechanical modalities. Currently available pharmacologic agents include low-dose UFH, LMWH, synthetic pentasaccharides, and vitamin K antagonists. Mechanical methods include intermittent pneumatic compression (IPC) and graduated compression stockings. Aspirin therapy alone is not adequate for DVT prophylaxis. These prophylaxis methods vary with regard to their efficacy, and the 2008 ACCP Clinical Practice Guidelines stratify their uses according to the patient's level of risk (Table 6).

Table 6. Thromboembolism Risk and Recommended Thromboprophylaxis in Surgical Patients

Level of Risk	Approximate DVT Risk without Thromboprophylaxis (%)	Suggested Thromboprophylaxis Options
Low risk	<10	No specific thromboprophylaxis
Minor surgery in mobile patients		Early and "aggressive" ambulation
Moderate risk	10-40	LMWH (at recommended doses), LDUH bid or tid, fondaparinux
Most general, open gynecologic, or urologic surgery		Mechanical thromboprophylaxis
Moderate VTE risk plus high bleeding risk		
High risk	40-80	LMWH (at recommended doses), fondaparinux, oral vitamin K antagonist (INR 2-3)
Hip or knee arthroplasty, hip fracture surgery		
Major trauma, spinal cord injury		Mechanical thromboprophylaxis
High VTE risk plus high bleeding risk		

Complications

Complications after venous thrombosis can vary from life threatening to chronically debilitating. Pulmonary embolism develops as venous thrombi break off from their location of origin and travel through the right heart and into the pulmonary artery, causing a ventilation perfusion defect and cardiac strain. PE occurs in approximately 10% of patients with acute deep venous thrombosis and can cause up to 10% of in hospital deaths.^{60,61} However, most patients (up to 75%) are asymptomatic. Traditionally, proximal venous thrombosis are thought to be at highest risk for causing pulmonary emboli; however, the single largest autopsy series ever performed to specifically to look for the source of fatal PE was performed by Havig in 1977, who found that one third of the fatal emboli arose directly from the calf veins.⁶

PHLEGMASIA ALBA AND CERULEA DOLENS

More than 600,000 cases of venous thromboembolism are estimated to occur each year in the United States. Pulmonary embolism (PE) complicates approximately 50% of cases of untreated proximal deep venous thrombosis (DVT) and contributes to 10-15% of all hospital deaths. Less frequent manifestations of venous thrombosis include phlegmasia alba dolens, phlegmasia cerulea dolens (PCD), and venous gangrene. These form a clinical spectrum of the same disorder. All 3 manifestations result from acute massive venous thrombosis and obstruction of the venous drainage of an extremity

History of the Procedure

In the 16th century, Fabricius Hildanus first described the clinical syndrome of what is currently called PCD. In 1938, Gregoire made an outstanding description of the condition and used the term PCD to differentiate ischemia-associated massive venous thrombosis from phlegmasia alba dolens, which describes fulminant venous thrombosis without ischemia.¹ The exact incidence of these disorders is not well reported.

In 1939, Leriche and Geissendorfer performed the first thrombectomy for cases of PCD.² Historically, surgical thrombectomy has been the procedure of choice for PCD refractory to medical therapy and in patients with established or impending gangrene.

Frequency

More than 600,000 cases of venous thromboembolism are estimated to occur annually in the United States. Phlegmasia alba dolens, PCD, and venous gangrene occur at any age but are more common during the fifth and sixth decades of life. Incidence is higher in females than in males.

Etiology

The main causative factor in phlegmasia is massive thrombosis and occlusion of major venous channels with significantly compromised venous outflow. Multiple triggering factors exist. Malignancy is the most common triggering factor and is present in approximately 20-40% of patients with PCD. Other associated risk factors include hypercoagulable syndrome, surgery, trauma, ulcerative colitis, gastroenteritis, heart failure, mitral valve stenosis, vena caval filter insertion, and May-Thurner syndrome (compression of the left iliac vein by the right iliac artery). Pregnancy has often been associated with phlegmasia alba dolens, especially during the third trimester when the uterus is large enough to compress the left common iliac vein against the pelvic rim (ie, milk leg syndrome). Finally, 10% of patients with phlegmasia have no apparent risk factors.

Pathophysiology

In phlegmasia alba dolens, the thrombosis involves only major deep venous channels of the extremity, therefore sparing collateral veins. The venous drainage is decreased but still

present; the lack of venous congestion differentiates this entity from PCD.

In PCD, the thrombosis extends to collateral veins, resulting in venous congestions with massive fluid sequestration and more significant edema. Without established gangrene, these phases are reversible if proper measures are taken.

Of PCD cases, 40-60% also have capillary involvement, which results in irreversible venous gangrene that involves the skin, subcutaneous tissue, or muscle. Under these conditions, the hydrostatic pressure in arterial and venous capillaries exceeds the oncotic pressure, causing fluid sequestration in the interstitium. Venous pressure may increase rapidly, as much as 16- to 17-fold within 6 hours. Fluid sequestration may reach 6-10 L in the affected extremity within days. Circulatory shock, which is present in about one third of patients, and arterial insufficiency may ensue.

The exact mechanism for the compromised arterial circulation is debatable but may involve shock, increased venous outflow resistance, and collapse of arterioles due to increased interstitial pressure. Vasospasm of the resistance vessels has also been hypothesized but has never been observed experimentally or radiographically.

Presentation

In the lower extremities, left-sided involvement is more common by a 3:1 or 4:1 ratio. Involvement of upper extremities occurs in less than 5% of patients with PCD. Manifestations may be gradual or fulminant. Of PCD cases, 50-60% are preceded by phlegmasia alba dolens, with symptoms of edema, pain, and blanching (alba) without cyanosis. The blanching, which previously was thought to be caused by arterial vasospasm, is caused by subcutaneous edema, without venous congestion.

Patients with PCD present with the clinical triad of edema, agonizing pain, and cyanosis. Massive fluid sequestration may lead to bleb and bullae formation. The pain is constant, usually starting at the femoral triangle and then progressing to the entire extremity. Cyanosis is the pathognomonic finding of PCD, progressing from distal to proximal areas.

When venous gangrene occurs, it has a similar distribution with the cyanosis. Arterial pulses may be present when the venous gangrene is superficial; however, gangrene that involves

the muscular compartment may result in increased compartment pressures and a pulse deficit. In addition, the pulses may be difficult to appreciate because of the significant edema. Various degrees of shock may be present because of significant fluid loss.

Indications

Historically, surgical thrombectomy has been the procedure of choice for phlegmasia cerulea dolens (PCD) refractory to medical therapy and for patients with established or impending gangrene. The standard treatment of phlegmasia and venous gangrene is evolving, but most clinicians attempt endovascular approaches to thrombolysis, if possible.

Relevant Anatomy

The main causative factor in phlegmasia is massive deep venous thrombosis (DVT) and occlusion of major venous channels with significantly compromised venous outflow. In phlegmasia alba dolens, the thrombosis involves only major deep venous channels of the extremity, therefore sparing collateral veins and preserving some venous outflow from the limb. In phlegmasia cerulea dolens (PCD), the thrombosis extends to collateral veins, with complete obstruction of venous outflow, resulting in massive venous congestion and fluid sequestration and more significant edema.

Contraindications

For phlegmasia alba dolens and mild nongangrenous forms of phlegmasia cerulea dolens (PCD), conservative medical treatment, rather than surgical thrombectomy, should be the initial course of therapy. Thrombolysis may be initiated if conservative management does not elicit a response and if the patient has no contraindication to lytic therapy.

Surgical thrombectomy cannot open the small venules that are affected in venous gangrene, and it does not prevent valvular incompetence or postphlebotic syndrome. For these reasons, thrombolysis seems to be an attractive alternative in the management of PCD and venous gangrene.

Imaging Studies

The diagnoses of phlegmasia alba dolens, phlegmasia cerulea dolens (PCD), and venous gangrene are established mainly via clinical criteria with the assistance of contrast venography and duplex ultrasonography.

Although venography is considered the criterion standard for diagnosis, technical difficulties may be encountered in as many as 20-25% of patients. Attempts to perform ascending venography when extensive deep system thrombosis is present may result in nonvisualization of the deep system and a nondiagnostic study result. In these cases, descending venography via the contralateral femoral vein or via the upper extremity veins may provide more information about the ilio caval system and proximal extent of the thrombus.

Recent improvements in ultrasonography have made this modality a more reliable and accurate way to assess for proximal deep venous thrombosis (DVT) with less morbidity. In addition, duplex imaging may be repeated as needed to monitor for thrombus propagation. Ultrasonography can also be performed at the bedside in patients who are critically ill or unstable. Ultrasonography is often used to guide the initial venipuncture for diagnostic venography and initiation of thrombolytic therapy.

Magnetic resonance venography (MRV) is an evolving modality of diagnostic imaging. Its principal advantage is its ability to easily reveal the proximal and distal extent of thrombus with a single study. Its principal disadvantage is the inability to image acutely ill patients with hemodynamic instability or motion artifacts due to pain.

Medical Therapy

The standard treatment of phlegmasia and venous gangrene is still evolving. The optimal therapeutic modality remains under debate. So far, the results of treatment have been moderately successful. For phlegmasia alba dolens and mild nongangrenous forms of phlegmasia cerulea dolens (PCD), conservative medical treatment, such as steep limb elevation, anticoagulation with intravenous administration of heparin, and fluid resuscitation, should be the initial course of therapy.

Initiate heparin administration with an intravenous bolus of 80-100 U/kg, followed by a continuous infusion of 15-18 U/kg/h. Frequently monitor the activated partial thromboplastin time (aPTT), with a goal range of 2-2.5 times the laboratory reference range. Frequently monitor platelet counts to allow the early detection of heparin-induced thrombocytopenia.

The purpose of rapid heparin anticoagulation is to decrease the risk of proximal clot propagation or thromboembolism. Heparin does not directly affect limb swelling. The best nonsurgical method to decrease edema is steep leg elevation.

Recent studies have demonstrated that low molecular weight heparins are safe and effective in the treatment of proximal deep venous thrombosis (DVT) and pulmonary embolism (PE); however, no good evidence supports the use of these newer agents in phlegmasia and venous gangrene.

If heparin-induced thrombocytopenia occurs, immediately discontinue the use of heparin and replace it with an alternative anticoagulant. Danaparoid and lepirudin are effective alternative agents; however, heparin-associated antibodies exhibit a 10-19% cross-reactivity with danaparoid. Thus, perform cross-reactivity testing before the initiation of danaparoid in patients with these antibodies. Lepirudin is a direct thrombin inhibitor that does not demonstrate any cross-reactivity. The recommended dosage of lepirudin in patients without renal failure is 0.4 mg/kg as an intravenous bolus followed by a continuous infusion of 0.15 mg/kg/h. Use aPTT to monitor therapy, with a goal range of 2-2.5 times the laboratory reference range.

Continue long-term anticoagulation with warfarin (or other coumarin derivatives) for at least 6 months. Life-long anticoagulation is recommended in patients with hypercoagulable states.

Patients should wear long-term prescription compression stockings with at least 30-40 mm Hg of graded pressure. Many physicians erroneously have the patient fitted for a prescription stocking while the limb is still severely edematous. Instead, the patient may use nonprescription stockings or an elastic bandage, in combination with elevation, to minimize edema prior to being fit for a prescription stocking.

Surgical Therapy

Surgical thrombectomy performed through a femoral venotomy allows instant decompression of the venous hypertension. An intraoperative Trendelenburg position may be

used to decrease the risk of PE. Transabdominal cavotomy and thrombectomy is an alternative approach that permits better control of the cava above the thrombus and, thus, provides protection against PE. Procedures that have been performed in an effort to decrease the rethrombosis rate include cross-pubic vein-to-vein reconstruction with polytetrafluoroethylene (PTFE) or the greater saphenous vein (GSV) or the creation of an arteriovenous fistula between the femoral artery and the GSV. These adjuvant procedures may be especially beneficial in cases that involve proximal iliofemoral vein constriction, damage, or external compression.

Concomitant administration of heparin and long-term anticoagulation are mandatory. Regardless, thrombectomy in patients with PCD is associated with a high rate of rethrombosis. Surgical thrombectomy cannot open the small venules that are affected in venous gangrene, and it does not prevent valvular incompetence or postphlebotic syndrome. The incidence of postphlebotic syndrome may be as high as 94% among survivors.

For the above reasons, thrombolysis seems to be an attractive alternative in the management of PCD and venous gangrene. In 1970, Paquet was the first to use thrombolysis for the treatment of PCD.⁴ Some authors propose catheter-directed thrombolysis directly into the vein with high doses of urokinase or tissue plasminogen activator (t-PA). Other authors support the method of intra-arterial low-dose thrombolysis via the common femoral artery, reasoning that the arterial route delivers the thrombolytic agent to the arterial capillaries and, subsequently, to the venules. The intra-arterial approach seems to be more effective in cases with venous gangrene. Systemic thrombolysis has also been used. Many authors have strongly recommended the insertion of a vena caval filter prior to initiation of thrombolytic therapy. Combine thrombolysis with heparin administration and long-term oral anticoagulation.

Fasciotomy alone or in conjunction with thrombectomy or thrombolysis reduces compartmental pressures; however, it significantly increases morbidity because of the prolonged wound healing and the risk of infection.

Finally, if all efforts fail and amputation is required, delay the procedure as long as possible. Take all precautions to reduce edema, allow venous channels to recanalize, and allow necrotic tissue to demarcate.

Preoperative Details

Patients who require emergent venous thrombectomy should have heparin continued throughout the perioperative period. Banked red blood cells should be available. The proximal extent of the thrombosis must be defined using a combination of venous ultrasonography for infrainguinal veins and retrograde venography of the iliac veins and inferior vena cava using a jugular or contralateral femoral approach. If the thrombosis extends into the iliac veins and vena cava, preparations should be made to control the cava via a right retroperitoneal incision. A high-quality fluoroscopy unit should be available to aid in catheter manipulation and completion venography.

Intraoperative Details

Operative exposure depends on the proximal and distal extent of the thrombus. The involved veins should be controlled proximally and distally prior to venotomy.

Iliac venous thrombectomy should be performed with large-bore thrombectomy balloon catheters (as large as 10F). Extension of the thrombus into the inferior vena cava may require proximal control of the cava. The anesthesiologist should apply positive airway pressure while the thrombus is being extracted from the ilio caval system. Digital subtraction venography should be performed to ascertain completeness of the thrombectomy.

Infrainguinal extraction of the thrombus is aided by the intraoperative placement of an Esmarch bandage from foot to thigh. Thrombectomy balloon catheters (3F and 4F) are passed through the femoral veins and, possibly, the posterior tibial veins as well.

Thrombolytic agents may be administered intraoperatively through the posterior tibial veins. t-PA is the most commonly used agent and may be administered intraoperatively.

After the thrombectomy is performed, an arteriovenous fistula should be constructed, connecting the proximal greater saphenous vein or one of its larger tributaries to the superficial femoral artery in an end-to-side fashion.

Completion venography should be performed to exclude the presence of residual thrombus or proximal venous stenosis. If one is present, balloon angioplasty with or without stent placement may be necessary.

When percutaneous endovascular therapy is performed as a single treatment modality, and many centers are now reporting this as a first-line therapy, the popliteal veins are usually

accessed with duplex ultrasonography as an aid. Prone positioning is rarely necessary. If extensive thrombus is present, access via the posterior tibial vein is usually successful. A 6-F sheath is usually adequate. An infusion wire is passed through the thrombus just to its proximal extent, often into the vena cava. Infusion is usually performed in the most proximal segment first, usually in the iliac veins.

A common protocol is to infuse tPA (1 mg/hr) through the infusion wire as well as through the sheath for 24 hours, then to change the sheath perfusion to lower dose heparin after 24 hours. The infusion is then performed in the superficial femoral and popliteal vein segments. Clinical improvement is often noted with clearing of the profunda venous segment. Performance of simultaneous percutaneous mechanical thrombectomy is controversial and may not give better results than postprocedure balloon dilation.

Postoperative Details

Intravenous heparin is administered throughout the postoperative period to prolong the aPTT (2-2.5 times the reference range for aPTT). This is continued until the patient is adequately anticoagulated with warfarin or one of the coumarin derivatives (international normalized ratio [INR] range of 2.0-3.0). The optimal duration of oral anticoagulation is not established.

A sequential compression device is also placed, or, at a minimum, an ace bandage is placed for control of edema. Once the edema is at its minimum, the patient may be fitted for a thigh-length compression stocking. Ambulation is encouraged, if the patient is able.

Complications

The incidence of postphlebotic syndrome may be as high as 94% among survivors. Pulmonary embolism is common, and prophylactic placement of an inferior vena cava filter is recommended in most cases. Thrombectomy in patients with phlegmasia cerulea dolens (PCD) is associated with a high rate of rethrombosis. Amputation and death are common.

Outcome and Prognosis

Despite all of the therapeutic modalities described above, phlegmasia cerulea dolens (PCD) and venous gangrene still remain life-threatening and limb-threatening conditions with overall mortality rates of 20-40%. Pulmonary embolism (PE) is responsible for 30% of the deaths reported from PCD. Overall, amputation rates of 12-50% have been reported among survivors. The postphlebitic sequelae are apparent in 60-94% of survivors. Strict adherence to the use of long-term compression stockings helps to control chronic edema.

Future and Controversies

Phlegmasia alba dolens, phlegmasia cerulea dolens (PCD), and venous gangrene still remain a challenge to the vascular surgeon. Treatment modalities continue to evolve. Endovascular management may offer hope of successful and more effective management, with less morbidity, than traditional surgery. The role of mechanical thrombectomy, compared with thrombolysis, is unclear. Small numbers of patients and lack of randomized trials preclude clear recommendations.

TOPIC № 13. PULMONARY EMBOLISM

Introduction

Background

Pulmonary embolism (PE) is as well recognized a thromboembolic disease, as are cerebral infarction and myocardial infarction in Western countries. However, the number of the patients with PE is still much lower in Japan than in the West. The Ministry of Health, Labor and Welfare in Japan reported in a patient survey that there were 1,220,000 patients with cerebral infarction, 82,000 with myocardial infarction, and only 4,000 with PE in 1999 . Vital statistics in the same year found 51,688 deaths from cerebral infarction, 48,806 from myocardial infarction, and only 1,738 from pulmonary embolism. In this chapter, we show the epidemiological results on PE in Japan, and consider the reasons for the difference in the incidence of PE between Japan and Western countries.

From the point of view of vital statistics, crude deaths from PE rapidly increased, and female deaths exceeded male deaths in the 1990s.

Age-specific mortality was higher in the elderly and, in recent years, lower in females than in males. The results of crude deaths reflect the fact that many more elderly people are female than male. The adjusted mortality in 1996 was 0.71 per 100,000 persons in males and 0.83 in females, which is comparable to the mortality of non-Blacks and non-Whites in the United States. The reduced mortality in the United States was achieved by widespread prevention of venous thromboembolism.

Pulmonary embolism (PE) is a relatively common cardiovascular emergency. By occluding the pulmonary arterial bed it may lead to acute life-threatening but potentially reversible right ventricular failure. PE is a difficult diagnosis that may be missed because of non-specific clinical presentation. However, early diagnosis is fundamental, since immediate treatment is highly effective. Depending on the clinical presentation, initial therapy is primarily aimed either at life-saving restoration of flow through occluded pulmonary arteries (PA) or at the prevention of potentially fatal early recurrences.

Both initial treatment and the long-term anticoagulation that is required for secondary prevention must be justified in each patient by the results of an appropriately validated diagnostic strategy.

Pulmonary embolism (PE) is a common and potentially lethal condition. Most patients who succumb to pulmonary embolism do so within the first few hours of the event. In patients who survive, recurrent embolism and death can be prevented with prompt diagnosis and therapy. Unfortunately, the diagnosis is often missed because patients with pulmonary embolism present with nonspecific signs and symptoms. If left untreated, approximately one third of patients who survive an initial pulmonary embolism die from a subsequent embolic episode.

The most important conceptual advance regarding pulmonary embolism over the last several decades has been the realization that pulmonary embolism is not a disease; rather, pulmonary embolism is a complication of venous thromboembolism, most commonly deep venous thrombosis (DVT). Virtually every physician who is involved in patient care (eg, internist, family physician, orthopedic surgeon, gynecologic surgeon, urologic surgeon, pulmonary subspecialist, cardiologist) encounters patients who are at risk for venous thromboembolism, and therefore at risk for pulmonary embolism.



Fig. 1. A large pulmonary artery thrombus in a hospitalized patient who died suddenly.



Fig. 2. Pulmonary embolism was identified as the cause of death in a patient who developed shortness of breath while hospitalized for hip joint surgery

Pathophysiology

The pathophysiology of pulmonary embolism encompasses several aspects, as described below.

Natural history of venous thrombosis

In the 19th century, Virchow identified a triad of factors that lead to venous thrombosis: venous stasis, injury to the intima, and enhanced coagulation properties of the blood. Thrombosis usually originates as a platelet nidus on valves in the veins of the lower extremities. Further growth occurs by accretion of platelets and fibrin and progression to red fibrin thrombus, which may either break off and embolize or result in total occlusion of the vein. The endogenous thrombolytic system leads to partial dissolution; then, the thrombus becomes organized and is incorporated into the venous wall.

Natural history of pulmonary embolism

Pulmonary emboli usually arise from the thrombi originating in the deep venous system of the lower extremities; however, rarely they may originate in the pelvic, renal, or upper extremity veins or the right heart chambers. After traveling to the lung, large thrombi can lodge at the bifurcation of the main pulmonary artery or the lobar branches and cause hemodynamic compromise. Smaller thrombi typically travel more distally, occluding smaller vessels in the lung periphery. These are more likely to produce pleuritic chest pain by initiating an inflammatory response adjacent to the parietal pleura. Most pulmonary emboli are multiple, and the lower lobes are involved more commonly than the upper lobes.

Respiratory consequences

Acute respiratory consequences of pulmonary embolism include increased alveolar dead space, pneumoconstriction, hypoxemia, and hyperventilation. Later, 2 additional consequences may occur: regional loss of surfactant and pulmonary infarction (see the image below). Arterial hypoxemia is a frequent but not universal finding in patients with acute embolism. The mechanisms of hypoxemia include ventilation-perfusion mismatch, intrapulmonary shunts, reduced cardiac output, and intracardiac shunt via a patent foramen ovale. Pulmonary infarction is an uncommon consequence because of the bronchial arterial collateral circulation.



Fig. 3. Lung infarction secondary to pulmonary embolism occurs rarely.

Hemodynamic consequences

Pulmonary embolism reduces the cross-sectional area of the pulmonary vascular bed, resulting in an increment in pulmonary vascular resistance, which, in turn, increases the right ventricular afterload. If the afterload is increased severely, right ventricular failure may ensue. In addition, the humoral and reflex mechanisms contribute to the pulmonary arterial constriction. Prior poor cardiopulmonary status of the patient is an important factor leading to hemodynamic collapse. Following the initiation of anticoagulant therapy, the resolution of emboli occurs rapidly during the first 2 weeks of therapy. Significant long-term nonresolution of emboli causing pulmonary hypertension or cardiopulmonary symptoms is uncommon.

Frequency

United States

The incidence of pulmonary embolism in the United States is estimated at 1 case per 1000 persons per year.¹ Studies from 2008 suggest that the increasing use of computed tomography (CT) for assessing patients with possible pulmonary embolism has led to an increase in the reported incidence of pulmonary embolism.^{2,3}

Pulmonary embolism is present in 60-80% of patients with DVT, even though more than half these patients are asymptomatic. Pulmonary embolism is the third most common cause of death in hospitalized patients, with at least 650,000 cases occurring annually. Autopsy studies have shown that approximately 60% of patients who died in the hospital had pulmonary embolism, and the diagnosis was missed in up to 70% of the cases. Prospective studies have demonstrated DVT in 10-13% of all medical patients placed on bed rest for 1 week, 29-33% of all patients in medical intensive care units, 20-26% of patients with pulmonary diseases who are given bed rest for 3 or more days, 27-33% of those admitted to a critical care unit after a myocardial infarction, and 48% of patients who are asymptomatic after a coronary artery bypass graft.

A population-based study covering the years 1966-1995 collated the cases of DVT or pulmonary embolism in women during pregnancy or postpartum. The relative risk was 4.29, and the overall incidence of venous thromboembolism (absolute risk) was 199.7 incidents per 100,000 woman-years. Among postpartum women, the annual incidence was 5 times higher than in pregnant women (511.2 vs 95.8 incidents per 100,000 women).

The incidence of DVT was 3 times higher than that of pulmonary embolism (151.8 vs 47.9 incidents per 100,000 women). Pulmonary embolism was relatively less common during pregnancy versus the postpartum period (10.6 vs 159.7 incidents per 100,000 women).⁴ A national review of severe obstetric complications from 1998-2005 found a significant increase in the rate of pulmonary embolism associated with the increasing rate of cesarean delivery.

International

The incidence of pulmonary embolism may differ substantially from country to country; observed variation is likely due to differences in the accuracy of diagnosis rather than in the actual incidence.

Mortality/Morbidity

From 1979-1998, the age-adjusted death rate for pulmonary embolism in the United States decreased from 191 deaths per million population to 94 deaths per million population.¹ Regional studies covering more recent years have found either a slight decrease or no change in mortality.^{2,3}

As a cause of sudden death, massive pulmonary embolism is second only to sudden cardiac death. Autopsy studies of patients who died unexpectedly in a hospital setting have shown approximately 80% of these patients died from massive pulmonary embolism.

Approximately 10% of patients who develop pulmonary embolism die within the first hour, and 30% die subsequently from recurrent embolism. Anticoagulant treatment decreases the mortality rate to less than 5%.

The diagnosis of pulmonary embolism is missed in approximately 400,000 patients in the United States per year; approximately 100,000 deaths could be prevented with proper diagnosis and treatment.

Race

The incidence of pulmonary embolism appears to be significantly higher in blacks than in whites.⁶ Mortality rates from pulmonary embolism for blacks have been 50% higher than those for whites, and those for whites have been 50% higher than those for people of other races (eg, Asians, Native Americans).¹

Sex

The risk of pulmonary embolism is increased in pregnancy and during the postpartum period.

Data are conflicting regarding male sex as a risk factor for pulmonary embolism; however, an analysis of national mortality data found that death rates from pulmonary embolism were 20-30% higher among men than among women.¹

Age

In hospitalized elderly patients, pulmonary embolism is commonly missed and often is the cause of death.

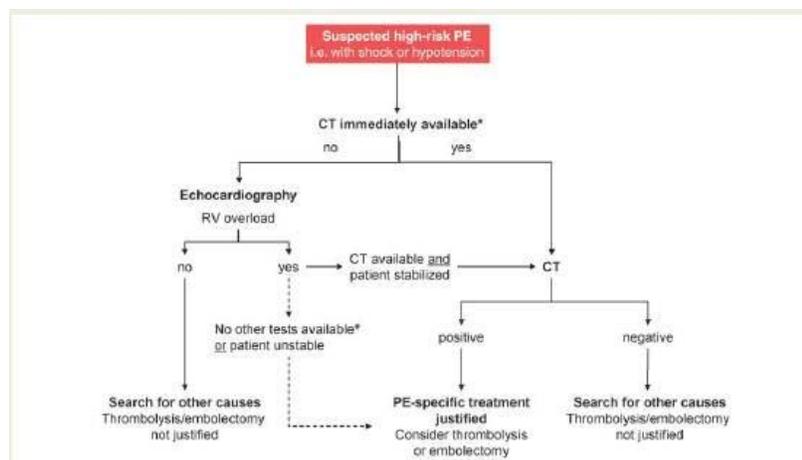
Clinical

Predisposing factors for venous thromboembolism

Predisposing factor	Patient-related	Setting-related
Strong predisposing factors (odds ratio >10)		
Fracture (hip or leg)		✓
Hip or knee replacement		✓
Major general surgery		✓
Major trauma		✓
Spinal cord injury		✓
Moderate predisposing factors (odds ratio 2–9)		
Arthroscopic knee surgery		✓
Central venous lines		✓
Chemotherapy		✓
Chronic heart or respiratory failure	✓	
Hormone replacement therapy	✓	
Malignancy	✓	
Oral contraceptive therapy	✓	
Paralytic stroke	✓	
Pregnancy/postpartum		✓
Previous VTE	✓	
Thrombophilia	✓	
Weak predisposing factors (odds ratio <2)		
Bed rest >3 days		✓
Immobility due to sitting (e.g. prolonged car or air travel)		✓
Increasing age	✓	
Laparoscopic surgery (e.g. cholecystectomy)		✓
Obesity	✓	
Pregnancy/antepartum	✓	
Varicose veins	✓	

Clinical prediction rules for PE: the Wells score and the revised Geneva score

Revised Geneva score ⁶⁴		Wells score ⁶⁵	
Variable	Points	Variable	Points
Predisposing factors		Predisposing factors	
Age >65 years	+1	Previous DVT or PE	+1.5
Previous DVT or PE	+3	Recent surgery or immobilization	+1.5
Surgery or fracture within 1 month	+2	Cancer	+1
Active malignancy	+2	Symptoms	
Symptoms		Haemoptysis	
Unilateral lower limb pain	+3		+1
Haemoptysis	+2	Clinical signs	
Clinical signs		Heart rate	
Heart rate		>100 beats/min	+1.5
75–94 beats/min	+3	Clinical signs of DVT	
≥ 95 beats/min	+5		+3
Pain on lower limb deep vein at palpation and unilateral oedema	+4	Clinical judgement	
Clinical probability		Alternative diagnosis less likely than PE	
	Total		+3
Low	0–3	Clinical probability (3 levels)	
Intermediate	4–10	Low	0–1
High	≥11	Intermediate	2–6
		High	≥7
		Clinical probability (2 levels)	
		PE unlikely	0–4
		PE likely	>4



Diagnostic algorithm for patients with suspected high-risk PE

History

The presentation of pulmonary embolism (PE) may vary from sudden catastrophic hemodynamic collapse to gradually progressive dyspnea. The diagnosis of pulmonary embolism should be sought actively in patients with respiratory symptoms unexplained by an alternate diagnosis. The symptoms of pulmonary embolism are nonspecific; therefore, a high index of suspicion is required, particularly when a patient has risk factors for the condition/

The presentation of patients with pulmonary embolism can be categorized into 4 classes based on the acuity and severity of pulmonary arterial occlusion. These categories are (1) massive pulmonary embolism, (2) acute pulmonary infarction, (3) acute embolism without infarction, and (4) multiple pulmonary emboli.

Massive pulmonary embolism

Large emboli compromise sufficient pulmonary circulation to produce circulatory collapse and shock.

The patient has hypotension; appears weak, pale, sweaty, and oliguric; and develops impaired mentation.

Acute pulmonary infarction

Approximately 10% of patients have peripheral occlusion of a pulmonary artery causing parenchymal infarction.

These patients present with acute onset of pleuritic chest pain, breathlessness, and hemoptysis.

Although the chest pain may be clinically indistinguishable from ischemic myocardial pain, normal electrocardiogram findings and no response to nitroglycerin rules it out.

Acute embolism without infarction: Patients have nonspecific symptoms of unexplained dyspnea and/or substernal discomfort.

Multiple pulmonary emboli

This group comprises 2 subsets of patients.

The first subset has repeated documented episodes of pulmonary emboli over years, eventually presenting with signs and symptoms of pulmonary hypertension and cor pulmonale.

The second subset has no previously documented pulmonary emboli but has widespread obstruction of the pulmonary circulation with clot. They present with gradually progressive dyspnea, intermittent exertional chest pain, and, eventually, features of pulmonary hypertension and cor pulmonale.

Most patients with pulmonary embolism have no obvious symptoms at presentation. In contrast, patients with symptomatic DVT commonly have pulmonary embolism confirmed on diagnostic studies in the absence of pulmonary symptoms.

The most common symptoms of pulmonary embolism in the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study were dyspnea (73%), pleuritic chest pain (66%), cough (37%), and hemoptysis (13%).⁷ However, patients with pulmonary embolism may present with atypical symptoms. In such cases, strong suspicion of pulmonary embolism based on the presence of risk factors can lead to consideration of pulmonary embolism in the differential diagnosis. These symptoms include the following:

Seizures

Syncope

Abdominal pain

Fever

Productive cough

Wheezing

Decreasing level of consciousness

New onset of atrial fibrillation

Flank pain

Delirium (in elderly patients)

Pleuritic chest pain without other symptoms or risk factors may be a presentation of pulmonary embolism.

Physical

Physical examination findings are quite variable in pulmonary embolism and, for convenience, may be grouped into 4 categories as follows:

Massive pulmonary embolism

These patients are in shock. They have systemic hypotension, poor perfusion of the extremities, tachycardia, and tachypnea.

Additionally, signs of pulmonary hypertension such as palpable impulse over the second left intercostal space, loud P2, right ventricular S3 gallop, and a systolic murmur louder on inspiration at left sternal border (tricuspid regurgitation) may be present.

Acute pulmonary infarction

These patients have decreased excursion of the involved hemithorax, palpable or audible pleural friction rub, and even localized tenderness.

Signs of pleural effusion, such as dullness to percussion and diminished breath sounds, may be present.

Acute embolism without infarction

These patients have nonspecific physical signs that may easily be secondary to another disease process.

Tachypnea and tachycardia frequently are detected, pleuritic pain sometimes may be present, crackles may be heard in the area of embolization, and local wheeze may be heard rarely.

Multiple pulmonary emboli or thrombi

Patients belonging to both the subsets in this category have physical signs of pulmonary hypertension and cor pulmonale.

Patients may have elevated jugular venous pressure, right ventricular heave, palpable impulse in the left second intercostal space, right ventricular S3 gallop, systolic murmur over the left sternal border that is louder during inspiration, hepatomegaly, ascites, and dependent pitting edema.

These findings are not specific for pulmonary embolism and require a high index of suspicion for pursuing appropriate diagnostic studies.

The most common physical signs in the PIOPED study were as follows:

Tachypnea (70%)

Rales (51%)

Tachycardia (30%)

Fourth heart sound (24%)

Accentuated pulmonic component of the second heart sound (23%)

Fever of less than 39°C may be present in 14% of patients; however, temperature higher than 39.5°C is not from pulmonary embolism. Finally, chest wall tenderness upon palpation, without a history of trauma, may be the sole physical finding in rare cases.

Causes

The causes for pulmonary embolism are multifactorial and are not readily apparent in many cases. The following causes have been described in the literature:

Venous stasis

Venous stasis leads to accumulation of platelets and thrombin in veins.

Increased viscosity may occur due to polycythemia and dehydration, immobility, raised venous pressure in cardiac failure, or compression of a vein by a tumor.

Hypercoagulable states

The complex and delicate balance between coagulation and anticoagulation is altered by many diseases, by obesity, after surgery, or by trauma.

Concomitant hypercoagulability may be present in disease states where prolonged venous stasis or injury to veins occurs.

Hypercoagulable states may be acquired or congenital. Factor V Leiden mutation causing resistance to activated protein C is the most common risk factor. Factor V Leiden mutation is present in up to 5% of the normal population and is the most common cause of familial thromboembolism.

Primary or acquired deficiencies in protein C, protein S, and antithrombin III are other risk factors. The deficiency of these natural anticoagulants is responsible for 10% of venous thrombosis in younger people

Immobilization

Immobilization leads to local venous stasis by accumulation of clotting factors and fibrin, resulting in thrombus formation.

The risk of pulmonary embolism increases with prolonged bed rest or immobilization of a limb in a cast.

Paralysis increases the risk.

Surgery and trauma

Both surgical and accidental trauma predispose patients to venous thromboembolism by activating clotting factors and causing immobility.

Fractures of the femur and tibia are associated with the highest risk, followed by pelvic, spinal, and other fractures.

Severe burns carry a high risk of DVT or pulmonary embolism.

A prospective study by Geerts and colleagues in 1994 indicated that major trauma was associated with a 58% incidence of DVT in the lower extremities and an 18% incidence in proximal veins.¹⁰

Pulmonary embolism may account for 15% of all postoperative deaths. Leg amputations and hip, pelvic, and spinal surgery are associated with the highest risk.

Pregnancy

The incidence of thromboembolic disease in pregnancy has been reported to range from 1 case in 200 deliveries to 1 case in 1400 deliveries.

Fatal events may occur rarely, 1-2 cases per 100,000 pregnancies.

The mechanism of DVT is venous stasis, decreasing fibrinolytic activity, and increased procoagulant factors.

Oral contraceptives and estrogen replacement

Estrogen-containing birth control pills have increased the occurrence of venous thromboembolism in healthy women.

The risk is proportional to the estrogen content and is increased in postmenopausal women on hormonal replacement therapy.

The relative risk is 3-fold, but the absolute risk is 20-30 cases per 100,000 persons per year.

Malignancy

Malignancy has been identified in 17% of patients with venous thromboembolism.

The neoplasms most commonly associated with pulmonary embolism, in descending order of frequency, are pancreatic carcinoma; bronchogenic carcinoma; and carcinomas of the genitourinary tract, colon, stomach, and breast.

In the PIOPED II study, immobilization (usually because of surgery) was the risk factor most commonly assessed in patients with pulmonary embolism; 94% of all patients with pulmonary embolism had 1 or more of the following risk factors¹¹ :

Immobilization

Travel of 4 hr or more in the past month

Surgery within the last 3 months

Malignancy, especially lung cancer

Current or past history of thrombophlebitis

Trauma to the lower extremities and pelvis during the past 3 months

Smoking

Central venous instrumentation within the past 3 months

Stroke, paresis, or paralysis

Prior pulmonary embolism

Heart failure

Chronic obstructive pulmonary disease

Other recognized risk factors include the following:

Obesity

Varicose veins

Inflammatory bowel disease

Laboratory Studies

Clinical signs and symptoms for pulmonary embolism (PE) are nonspecific; therefore, patients suspected of having pulmonary embolism—because of unexplained dyspnea, tachypnea, or chest pain or the presence of risk factors for pulmonary embolism—must undergo diagnostic tests until the diagnosis is ascertained or eliminated or an alternative diagnosis is confirmed. Further, routine laboratory findings are nonspecific and are not helpful in pulmonary embolism, although they may suggest another diagnosis.

Evidence-based literature supports the practice of determining the clinical probability of pulmonary embolism before

proceeding with testing. A clinical practice guideline from the American Academy of Family Physicians (AAFP) and the American College of Physicians (ACP) recommends that validated clinical prediction rules be used to estimate pretest probability of pulmonary embolism and to interpret test results. The guideline, Current diagnosis of venous thromboembolism in primary care, advocates use of the Wells prediction rule for this purpose, but notes that the Wells rule performs better in younger patients without comorbidities or a history of venous thromboembolism.

Table 1. Wells Prediction Rule for Diagnosing Pulmonary Embolism: Clinical Evaluation Table for Predicting Pretest Probability of Pulmonary Embolism

Clinical Characteristic	Score
Previous pulmonary embolism or deep vein thrombosis	+1.5
Heart rate greater than 100 beats per minute	+1.5
Recent surgery or immobilization (within the last 30 d)	+1.5
Clinical signs of deep vein thrombosis	+3
Alternative diagnosis less likely than pulmonary embolism	+3
Hemoptysis	+1
Cancer (treated within the last 6 mo)	+1

Note: Clinical probability of pulmonary embolism: low 0–1; intermediate 2–6; high 7 or more
 Reprinted from *Am J Med*, Vol 113, Chagnon I, Bounameaux H, Aujesky D, et al, Comparison of two clinical prediction rules and implicit assessment among patients with suspected pulmonary embolism, pp 269-75, Copyright 2002.

Another validated clinical prediction rule for use in the diagnosis of pulmonary embolism is the revised Geneva score. The performance of the revised Geneva score appears equivalent to that of the Wells score.

Table 2 The Revised Geneva Score

Risk Factors	Points
Age older than 65 y	1
Previous DVT or PE	3
Surgery (under general anesthesia) or fracture (of the lower limbs) within 1 mo	2
Active malignant condition (solid or hematologic, currently active or considered cured less than 1 y)	2
Symptoms	
Unilateral lower limb pain	3
Hemoptysis	2
Clinical Signs	
Heart rate 75–94 beats/min	3
Heart rate ≥ 95 beats/min	5
Pain on lower limb deep venous palpation and unilateral edema	4
Clinical Probability	
Low	0–3 total
Intermediate	4–10 total
High	≥ 11 total

*Adapted from Prediction of pulmonary embolism in the emergency department: the revised Geneva score. Le Gal G, Righini M, Roy PM, Sanchez O, Aujesky D, Bounameaux H, Perrier A. *Ann Intern Med.* 2006 Feb 7;144(3):165-71.

Simplified versions of the Wells score and the revised Geneva score have been developed. Initial studies support the validity of these scores, which assign 1 point to each of the criteria.

D-dimer testing

When clinical prediction rule results indicate that the patient has a low or moderate pretest probability of pulmonary embolism, D-dimer testing is the usual next step. Negative results on a high-sensitivity D-dimer test in a patient with a low pretest probability of pulmonary embolism indicate a low likelihood of venous thromboembolism and reliably exclude pulmonary embolism. A large prospective randomized trial found that in patients with a low probability of pulmonary embolism who had negative D-dimer results, forgoing additional diagnostic testing was not associated with an increased frequency of symptomatic venous thromboembolism during the subsequent 6 months.²⁰

D-dimer, a degradation product produced by plasmin-mediated proteases of cross-linked fibrin, is measured by a variety of assay types, including quantitative, semiquantitative, and qualitative rapid enzyme-linked immunosorbent assays (ELISAs); quantitative and semiquantitative latex; and whole-blood assays. A systematic review of prospective studies of high methodologic quality concluded that the ELISAs—especially the quantitative rapid ELISA—dominate the comparative ranking among the D-dimer assays for sensitivity and negative likelihood ratio. The quantitative rapid ELISA has a sensitivity of 0.95 and negative likelihood ratio of 0.13; the latter is similar to that for a normal to near-normal lung scan in patients with suspected pulmonary embolism.

D-dimer testing is most reliable for excluding pulmonary embolism in younger patients who have no associated comorbidity or history of venous thromboembolism and whose symptoms are of short duration.¹⁴ D-dimer testing is of questionable value in patients who are older than 80 years, are hospitalized, or have cancer and in pregnant women, because nonspecific elevation of D-dimer concentrations is common in such patients. D-dimer test should not be used when the clinical probability of pulmonary embolism is high, because the test has low negative predictive value in such cases.

Troponins

Serum troponin levels can be elevated in up to 50% of patients with a moderate-to-large pulmonary embolism, presumptively due to acute right ventricular myocardial stretch.

Although not currently recommended as part of the diagnostic workup, studies have shown that elevated troponin levels in the setting of pulmonary embolism correlate with increased mortality. Currently, further studies need to be performed to identify subsets of patients with pulmonary embolism who might benefit from this testing.

A meta-analysis by Jimenez et al suggests that in acute symptomatic pulmonary embolism (PE), elevated troponin levels do not distinguish between patients who are at high risk for death and those who are at low risk. Pooled results from studies including 1366 normotensive patients with acute symptomatic PE showed that elevated troponin levels were associated with a 4.26-fold increased odds of overall mortality (95% confidence interval [CI], 2.13-8.50; heterogeneity $\chi^2 = 12.64$; degrees of freedom = 8; $P = .125$). Summary receiver operating characteristic curve analysis showed a relationship between the sensitivity and specificity of troponin levels to predict overall mortality (Spearman rank correlation coefficient = 0.68; $P = .046$). Pooled likelihood ratios (LRs) were not extreme (negative LR, 0.59 [95% CI, 0.39-0.88]; positive LR, 2.26 [95% CI, 1.66-3.07]).

Brain natriuretic peptide

Although neither sensitive nor specific, patients with pulmonary embolism tend to have higher levels of brain natriuretic peptide (BNP). In one case-control study of 2213 hemodynamically stable patients with suspected acute pulmonary embolism, BNP testing had a sensitivity and specificity of only 60% and 62%, respectively.

Elevated levels of BNP or its precursor, N-terminal pro-brain natriuretic peptide (NT-proBNP), do correlate with an increased risk of subsequent complications and mortality in patients with acute pulmonary embolism. One meta-analysis revealed that patients with a BNP level greater than 100 pg/mL or an NT-proBNP level greater than 600 ng/L had an all-cause in-hospital mortality rate 6- and 16-fold higher than those below these cutoffs, respectively. In a second smaller observational study, serum BNP levels greater than 90 pg/mL were associated with a higher rate of complications, such as the need for cardiopulmonary resuscitation, need for mechanical ventilation, need for vasopressor therapy, and death.²⁸

BNP testing is not currently recommended as part of the standard evaluation of acute pulmonary embolism, and future studies may aid in defining its role in this setting.

Arterial blood gases

Arterial blood gas determinations characteristically reveal hypoxemia, hypocapnia, and respiratory alkalosis; however, the predictive value of hypoxemia is quite low.

Both the PaO₂ and the calculation of alveolar-arterial oxygen gradient contribute to the diagnosis in a general population thought to have pulmonary embolism.

Nonetheless, in high-risk settings such as patients in postoperative states in whom other respiratory conditions can be ruled out, a low PaO₂ in conjunction with dyspnea may have a strong positive predictive value.

Imaging Studies

Chest radiography

The American College of Radiology (ACR) recommends chest radiography as the most appropriate study for ruling out other causes of chest pain in patients with suspected pulmonary embolism.

Initially, the chest radiography findings are normal in most cases of pulmonary embolism. However, in later stages,

radiographic signs may include a Westermark sign (dilatation of pulmonary vessels and a sharp cutoff), atelectasis, a small pleural effusion, and an elevated diaphragm.

Although chest radiography findings may indicate an alternate diagnosis, this study alone is not sufficient to confirm the diagnosis of pulmonary embolism.

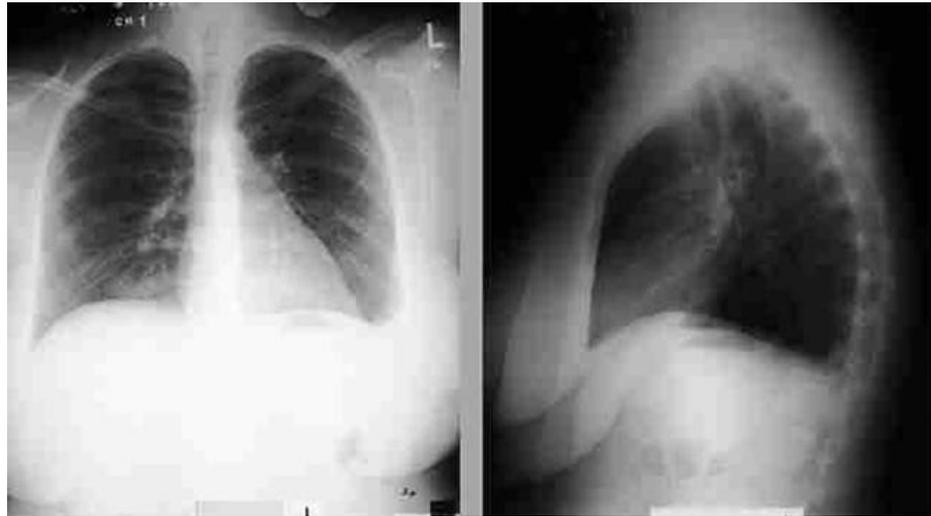


Fig. 4. Posteroanterior and lateral chest radiograph findings are normal, which is the usual finding in patients with pulmonary embolism

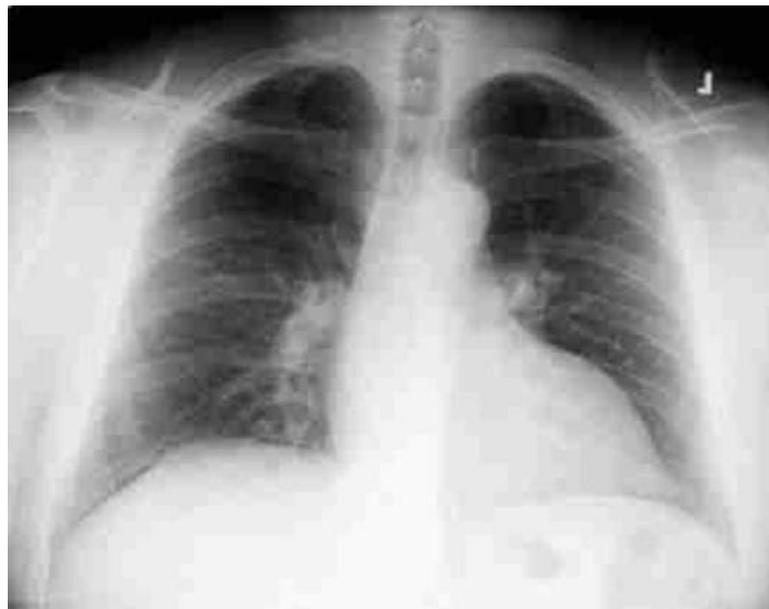


Fig. 5. A chest radiograph with normal findings in a 64-year-old woman who presented with worsening breathlessness.

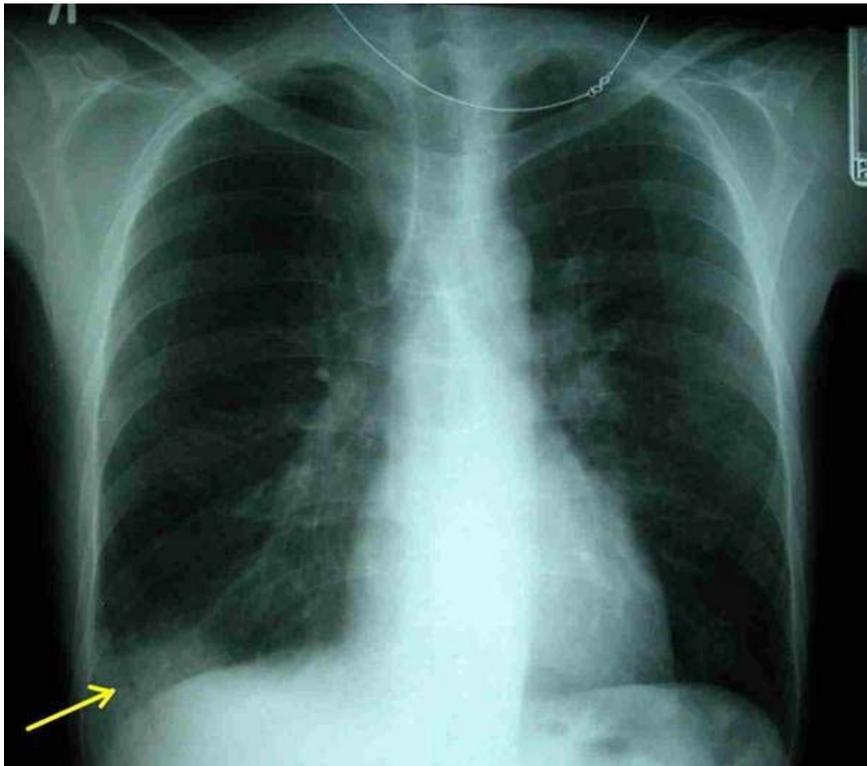


Fig.6. A posteroanterior chest radiograph showing a peripheral wedge-shaped infiltrate caused by pulmonary infarction secondary to pulmonary embolism. Hampton hump is a rare and nonspecific finding

Computed tomography

CT angiography (CTA) is the initial imaging modality of choice for stable patients with suspected pulmonary embolism. The ACR considers chest CTA the current standard of care for the detection of pulmonary embolism.²⁹

In patients with a negative CTA, the likelihood for subsequent thromboembolic events is extremely small.

The Christopher study, a prospective trial, used CT as part of a management algorithm for 3306 outpatients with suspected acute pulmonary embolism. Patients in whom the Wells rule indicated that pulmonary embolism was unlikely underwent D-dimer testing; if the result was normal, pulmonary embolism was considered excluded. All other patients underwent multidetector CT arteriography, and pulmonary embolism was considered present or excluded based on the results. Among patients with negative scan results who did not receive anticoagulation therapy, the 3-month incidence of venous thromboembolism was 1.3%; death, possibly from pulmonary embolism, occurred in 0.5%.³⁰

Similarly, a meta-analysis published in 2004 reviewed 23 studies reporting on 4657 patients with negative pulmonary CTA results for pulmonary embolism who did not receive anticoagulation. The rate of venous thromboembolism was 1.4% and the rate of fatal pulmonary embolism was 0.51% at 3 months. These results are similar to negative results on conventional pulmonary angiography. These investigators concluded that withholding anticoagulation after negative pulmonary CTA results appears to be safe.³¹

Spiral CT can visualize main, lobar, and segmental pulmonary emboli with a reported sensitivity of greater than 90%. Spiral CT scanning can help detect emboli as small as 2 mm that are affecting up to the seventh border division of the pulmonary artery. A further benefit of spiral CT scanning is that the results may suggest an alternative diagnosis in up to 57% of patients. A significant limitation of spiral CT scanning is that small subsegmental emboli may not be detected.

The technique is as follows:

Spiral CT examination is performed immediately after infusion of 150-200 mL of 30% contrast material.

Scanning is performed from the level of the aortic arch to approximately 2 cm below the level of the inferior pulmonary vein while the patient is holding his or her breath at full inspiration.

If the patient is not able to hold his or her breath for 20-30 seconds, scanning may be performed during gentle breathing.

Sensitivity and specificity of spiral CT scanning for pulmonary embolism are as follows:

The reported sensitivity is 53-100%.

The reported specificity is 78-96%.

The negative predictive value is 81-100%, and the positive predictive value is 60-100% for detecting emboli in segmental or larger arteries.

Positive findings on CT imaging include a central intravascular filling defect within the vessel lumen, eccentric tracking of contrast material around a filling defect, and complete vascular occlusion. Smooth filling defects making an obtuse angle with a vessel wall may represent chronic thrombi or recent recanalization. In the lung parenchyma, signs of pulmonary embolism include oligemia, pulmonary hemorrhage (ground-glass attenuation), and pulmonary infarction (peripheral wedge-shaped pleural-based opacification).

Pitfalls include the following:

Technically inadequate scans may result from patients' dyspnea and/or obliquely or horizontally oriented vessels within the right middle lobe and left lingula.

False filling defects may result from breathing artifact cardiac motion or unilateral extensive air space consolidation as a result of the significant decrease in blood flow through pulmonary arteries in these areas.

Combined spiral CT scanning for detection of pulmonary embolism and deep venous thrombosis (DVT)

A combined CT scan for PE/DVT enhances the utility of spiral CT scanning by further identifying emboli in the deep venous system of the lower extremities or the pelvic veins.

Good venous enhancement of the lower extremity veins occurs 2 minutes following lung CT scanning as 5-mm scans are performed at 5-cm intervals from the upper calves to the diaphragm.

Alternatively, 1-cm images are performed from the iliac bones to the tibial plateau. The additional radiation dose needs to be considered in the formulation of this protocol. With this technique, up to 4% of patients with negative results on CT scanning examination for pulmonary embolism have been identified to have DVT

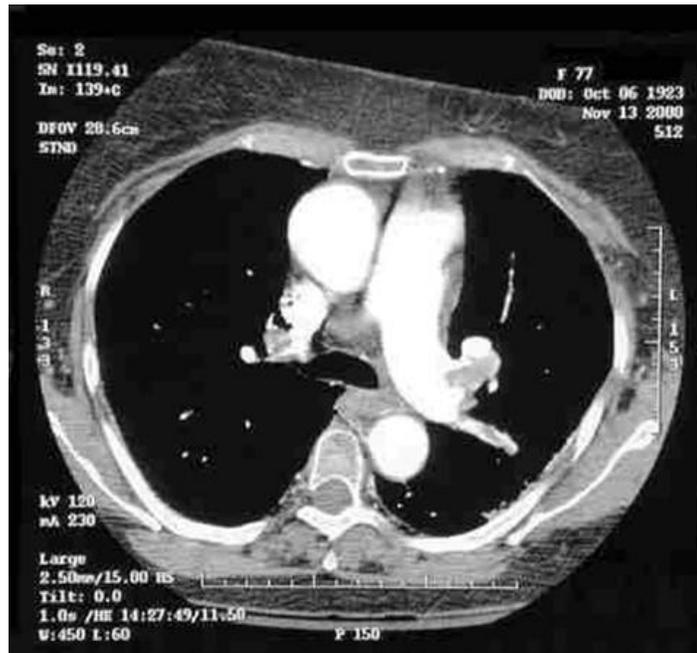


Fig. 7. A spiral CT scan shows thrombus in bilateral main pulmonary arteries.

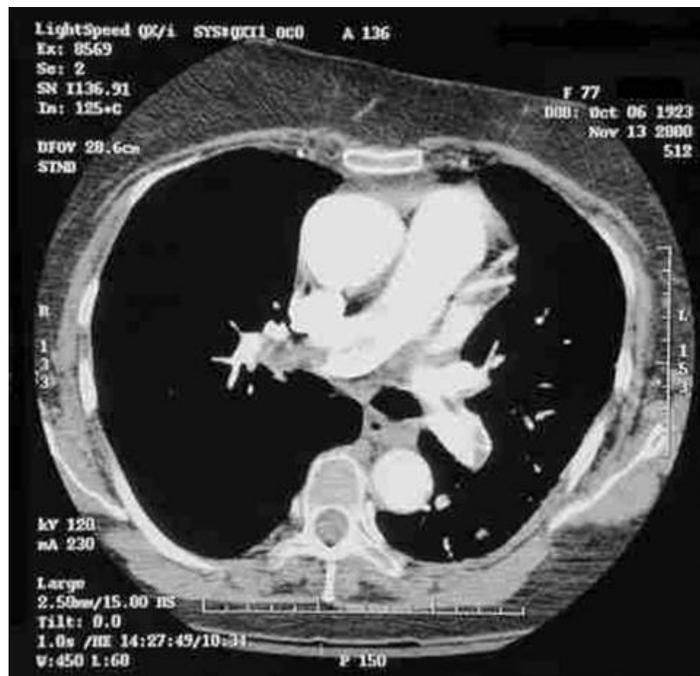


Fig. 8. A spiral CT scan shows thrombus in bilateral main pulmonary arteries

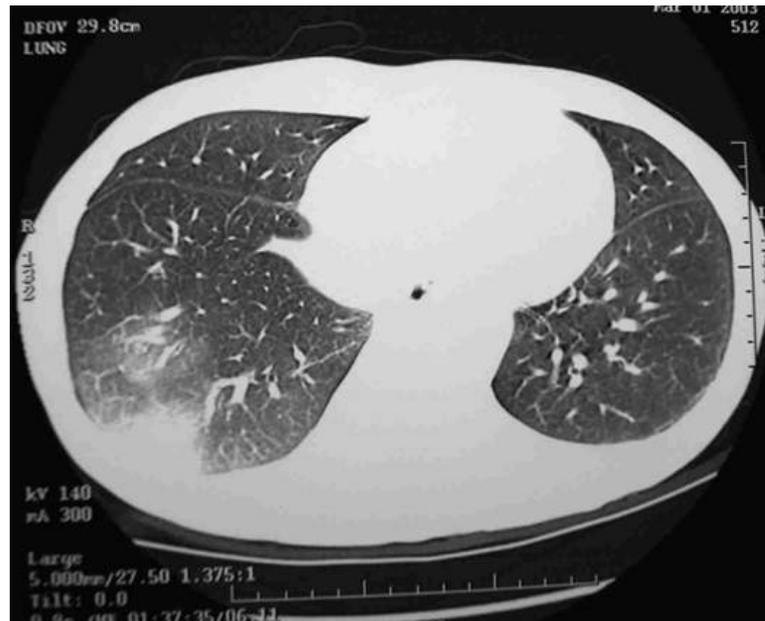


Fig. 9. CT scan

Ventilation-perfusion (V/Q) scanning of the lungs: This is an important diagnostic modality for establishing the diagnosis of pulmonary embolism. However, V/Q scanning should be used only when CT scanning is not available or if the patient has a contraindication to CT scanning or intravenous contrast material.

New criteria for V/Q scanning diagnosis of pulmonary embolism, from the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) II trial:

High probability criteria are as follows:

Two large (more than 75% of a segment) segmental perfusion defects without corresponding ventilation or chest radiographic abnormalities

One large segmental perfusion defect and 2 moderate (25-75% of a segment) segmental perfusion defects without corresponding ventilation or radiographic abnormalities

Four moderate segmental perfusion defects without corresponding ventilation or chest radiographic abnormalities

Intermediate probability criteria are as follows:

One moderate to fewer than 2 large segmental perfusion defects without corresponding ventilation or chest radiographic abnormalities

Corresponding V/Q defects and radiographic parenchymal opacity in lower lung zone

Single moderate matched V/Q defects with normal chest radiographic findings

Corresponding V/Q and chest radiography small pleural effusion

Difficult to categorize as normal, low, or high probability

Low probability criteria are as follows:

Multiple matched V/Q defects, regardless of size, with normal chest radiographic findings

Corresponding V/Q defects and radiographic parenchymal opacity in upper or middle lung zone

Corresponding V/Q defects and large pleural effusion

Any perfusion defects with substantially larger radiographic abnormality

Defects surrounded by normally perfused lung (stripe sign)

More than 3 small (less than 25% of a segment) segmental perfusion defects with normal chest radiographic findings

Nonsegmental perfusion defects (cardiomegaly, aortic impression, enlarged hila)

Very low criterion is 3 small (less than 25% of a segment) segmental perfusion defects with normal chest radiograph findings.

Normal finding is no perfusion defects and perfusion outlines the shape of the lung seen on a chest radiograph.

In the PIOPED II study, very low-probability V/Q scans in patients whose Wells score indicated low pretest probability of pulmonary embolism reliably excluded pulmonary embolism.

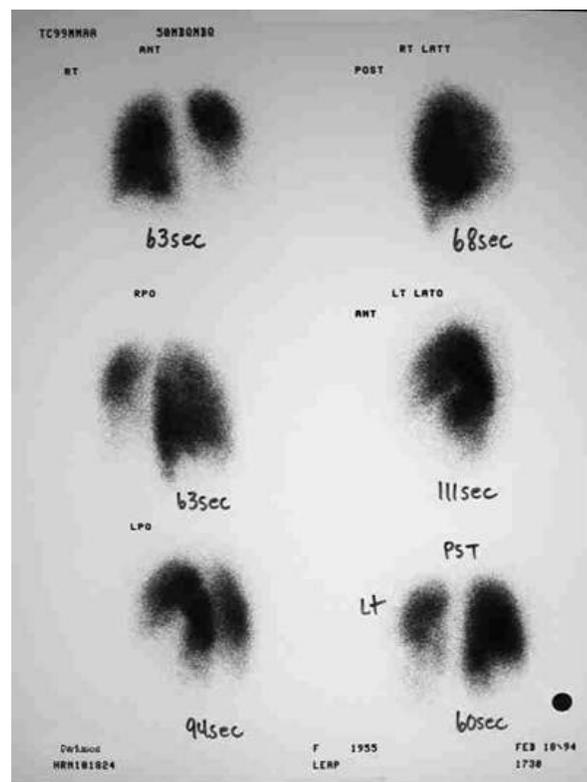


Fig. 10. High-probability perfusion lung scan shows segmental perfusion defects in the right upper lobe and subsegmental perfusion defects in right lower lobe, left upper lobe, and left lower lobe

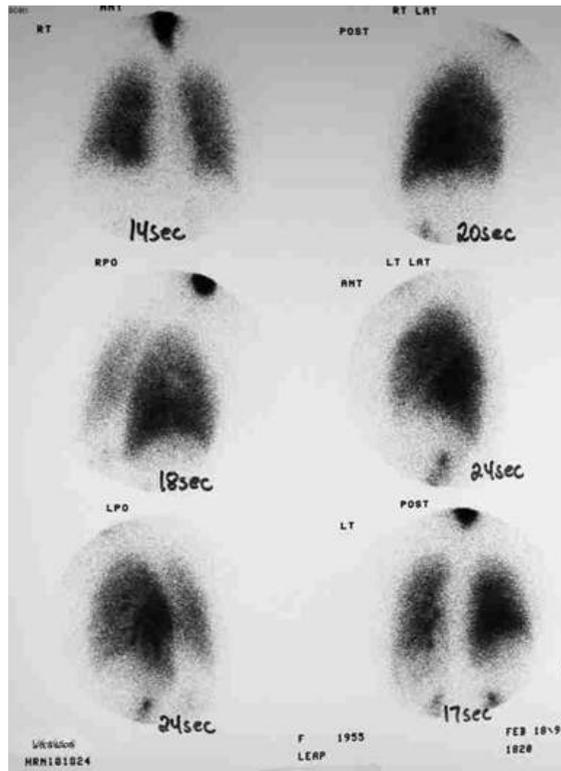


Fig. 11. A normal ventilation scan will make the above-noted defects in Image 5 a mismatch and, hence, a high-probability ventilation-perfusion scan

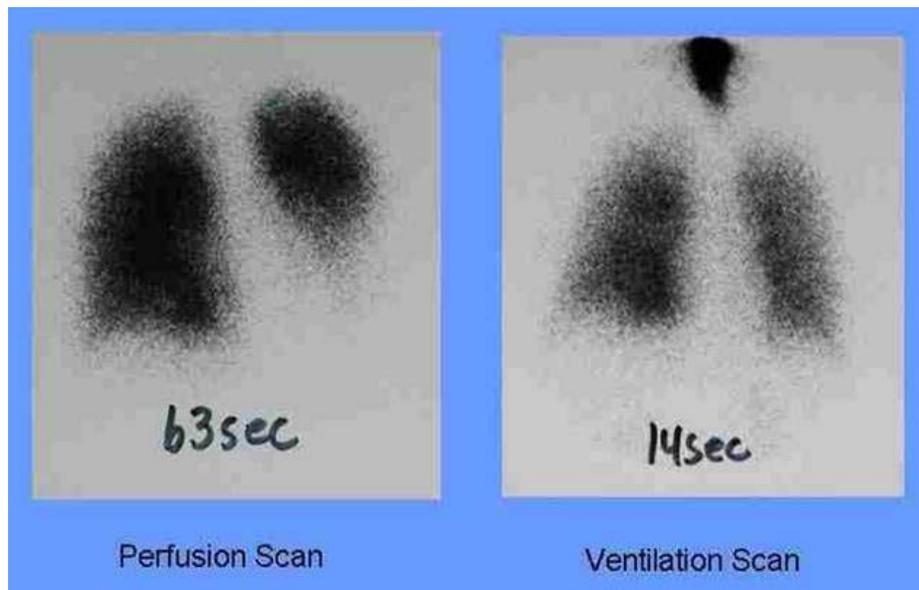


Fig. 12. Anterior views of perfusion and ventilation scans are shown here. A perfusion defect is present in the left lower lobe, but perfusion to this lobe is intact, making this a high-probability scan

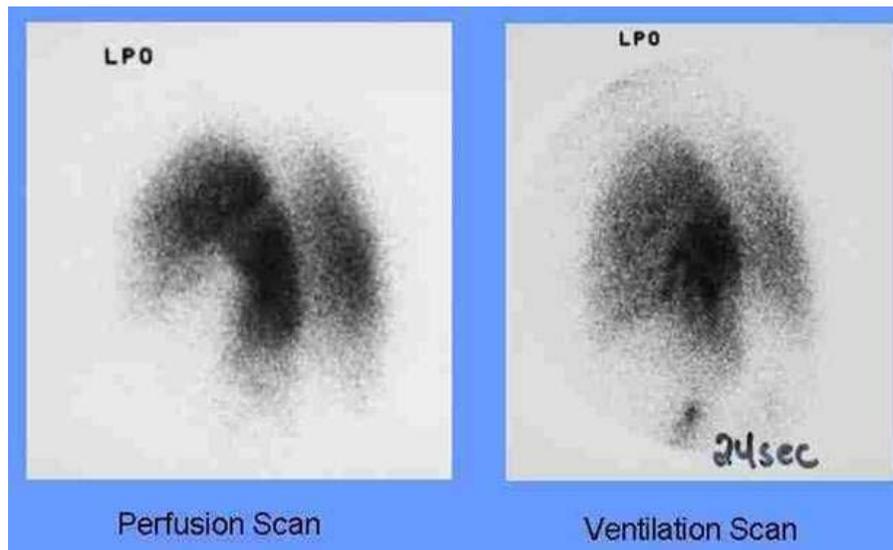


Fig. 13. A segmental ventilation perfusion mismatch evident in a left anterior oblique projection.

Noninvasive tests for lower extremity DVT

These may be helpful in the evaluation of patients who have nondiagnostic V/Q scan patterns of intermediate and low probability.

Color-flow Doppler imaging and compression ultrasonography have a high sensitivity (89-100%) and specificity (89-100%) for detection of proximal DVT in symptomatic patients. However, compression ultrasonography has a low sensitivity (38%) and a low positive predictive value (26%) in patients without symptoms of DVT. Patients with positive findings for DVT can be anticoagulated irrespective of their V/Q scan results; other patients must have more invasive investigations performed to definitively rule out pulmonary embolism.

Pulmonary angiography

Pulmonary angiography remains the criterion standard for the diagnosis of pulmonary embolism.

Following injection of iodinated contrast, anteroposterior, lateral, and oblique studies are performed on each lung.

Positive results consist of a filling defect or sharp cutoff of the affected artery. Nonocclusive emboli are described to have a tram-track appearance.

Abnormal findings on V/Q scans performed prior to angiography guide the operator to focus on abnormal areas.

Angiography generally is a safe procedure. The mortality rate for patients undergoing this procedure is less than 0.5%, and the morbidity rate is less than 5%.

Patients who have long-standing pulmonary arterial hypertension and right ventricular failure are considered high-risk patients.

Negative pulmonary angiogram findings, even if false negative, exclude clinically relevant pulmonary embolism.



Fig. 14. A pulmonary angiogram shows the abrupt termination of the ascending branch of the right upper-lobe artery, confirming the diagnosis of pulmonary embolism

Magnetic resonance imaging

With MRI, evidence of pulmonary emboli may be detected by using standard or gated spin-echo techniques.

Pulmonary emboli demonstrate increased signal intensity within the pulmonary artery. By obtaining a sequence of images, this signal that is originating from slow blood flow may be distinguished from pulmonary embolism. However, this remains a problem in pulmonary hypertension.

Magnetic resonance angiography is performed following intravenous administration of gadolinium. Gadolinium-based contrast agents (gadopentetate dimeglumine [Magnevist], gadobenate dimeglumine [MultiHance], gadodiamide [Omniscan], gadoversetamide [OptiMARK], gadoteridol [ProHance]) have been linked to the development of nephrogenic systemic fibrosis (NSF) or nephrogenic fibrosing dermopathy (NFD). The disease has occurred in patients with moderate to end-stage renal disease after being given a gadolinium-based contrast agent to enhance MRI or magnetic resonance angiography scans.

NSF/NFD is a debilitating and sometimes fatal disease. Characteristics include red or dark patches on the skin; burning, itching, swelling, hardening, and tightening of the skin; yellow spots on the whites of the eyes; joint stiffness with trouble moving or straightening the arms, hands, legs, or feet; pain deep in the hip bones or ribs; and muscle weakness.

MRI has a sensitivity of 85% and specificity of 96% for central, lobar, and segmental emboli; MRI is inadequate for the diagnosis of subsegmental emboli.

Echocardiography

This modality generally has limited accuracy in the diagnosis of pulmonary embolism.

Transesophageal echocardiography may identify central pulmonary embolism, and the sensitivity for central pulmonary embolism is reported to be 82%.

Overall sensitivity and specificity for central and peripheral pulmonary embolism is 59% and 77%.

Echocardiography may demonstrate right ventricular dysfunction in acute pulmonary embolism, predicting a higher mortality and possible benefit from thrombolytic therapy. Vanni et al reported that a right ventricular strain pattern is associated with a worse short-term outcome.

Differential Diagnoses

Acute Coronary Syndrome

Acute Respiratory Distress Syndrome

Anxiety Disorders

Aortic Stenosis

Atrial Fibrillation

Atrial Fibrillation, Diagnosis and Management

Cardiogenic Shock

Cardiomyopathy, Dilated

Cardiomyopathy, Restrictive

Chronic Obstructive Pulmonary Disease

Congestive Heart Failure and Pulmonary Edema

Cor Pulmonale

Emphysema

Extrinsic Allergic Alveolitis

Fat Embolism

Lung, Arteriovenous Malformation

Mitral Stenosis

Myocardial Infarction

Myocardial Ischemia

Pericarditis and Cardiac Tamponade

Pneumothorax

Pulmonary Edema, Noncardiogenic

Pulmonary Hypertension, Primary

Pulmonary Hypertension, Secondary

Sudden Cardiac Death

Sudden Cardiac Death

Superior Vena Cava Syndrome

Syncope

Medical Care

Immediate full anticoagulation is mandatory for all patients suspected to have deep vein thrombosis (DVT) or pulmonary embolism (PE). Diagnostic investigations should not delay empirical anticoagulant therapy. Current guidelines recommend starting unfractionated heparin (UFH), low molecular weight heparin (LMWH) or fondaparinux (all grade 1A) in addition to an oral anticoagulant (warfarin) at the time of diagnosis, and to discontinue UH, LMWH, or fondaparinux only after the international normalized ratio (INR) is 2.0 for at least 24 hours, but no sooner than 5 days after warfarin therapy has been started (grade 1C recommendation).³⁴ The recommended duration of UH, LMWH, and fondaparinux is based on evidence suggesting that the relatively long half-life of factor II, along with the short half-lives of protein C and protein S, may provoke a paradoxical hypercoagulable state if these agents are discontinued prematurely.

Thrombolytic therapy

Thrombolytic therapy should be considered for patients who are hemodynamically unstable, patients who have right-sided heart strain, and high-risk patients with underlying poor cardiopulmonary reserve.

Although most studies demonstrate superiority of thrombolytic therapy with respect to resolution of radiographic and hemodynamic abnormalities within the first 24 hours, this advantage disappears 7 days after treatment. Controlled clinical trials have not demonstrated benefit in terms of reduced mortality rates or earlier resolution of symptoms when currently compared with heparin.

Until randomized clinical trials demonstrate a clear morbidity or mortality benefit, the role of thrombolytic therapy in the management of acute pulmonary embolism remains controversial. The currently accepted indications for thrombolytic therapy include hemodynamic instability or right ventricular dysfunction demonstrated on echocardiography.

Goals of anticoagulation therapy

The efficacy of heparin therapy depends on achieving a critical therapeutic level of heparin within the first 24 hours of treatment. The critical therapeutic level of heparin is 1.5 times the baseline control value or the upper limit of normal range of the activated partial thromboplastin time (aPTT).

This level of anticoagulation is expected to correspond to a heparin blood level of 0.2-0.4 U/mL by the protamine sulfate titration assay and 0.3-0.6 by the antifactor X assay.

Each laboratory should establish the minimal therapeutic level for heparin, as measured by the aPTT, to coincide with a heparin blood level of at least 0.2 U/mL for each batch of thromboplastin reagent being used.

If intravenous UFH is chosen, an initial bolus of 80 U/kg or 5000 U followed by an infusion of 18 U/kg/h or 1300 U/h should be given, with the goal of rapidly achieving and maintaining the aPTT at levels that correspond to therapeutic heparin levels. Fixed-dose and monitored regimens of subcutaneous UFH are available and are acceptable alternatives.

Low molecular weight heparin

Current guidelines for patients with acute nonmassive pulmonary embolism recommend LMWH over UFH (grade 1A). In patients with massive pulmonary embolism, if concerns regarding subcutaneous absorption arise, severe renal failure exists, or if thrombolytic therapy is being considered, intravenous UFH is the recommended form of initial anticoagulation (grade 2C).³⁴

LMWHs have many advantages over UFH. These agents have a greater bioavailability, can be administered by subcutaneous injections, and have a longer duration of anticoagulant effect.

A fixed dose of LMWH can be used, and laboratory monitoring of aPTT is not necessary.

Trials comparing LMWH with UFH have shown that LMWH is at least as effective and as safe as UFH.

The studies have not pointed to any significant differences in recurrent thromboembolic events, major bleeding, or mortality between the 2 types of heparin.

LMWH can be administered safely in an outpatient setting. This has led to the development of programs in which clinically stable patients with pulmonary embolism are treated at home, at substantial cost savings.

Fondaparinux

Fondaparinux is a synthetic polysaccharide derived from the antithrombin binding region of heparin. Fondaparinux catalyzes factor Xa inactivation by antithrombin without inhibiting thrombin.

Fondaparinux has not been directly compared with subcutaneous UFH or LMWH, but one randomized open-label study of 2213 patients with symptomatic pulmonary embolism compared once daily subcutaneous fondaparinux with intravenous UFH. The 2 regimens were found to have similar rates of recurrent pulmonary embolism, bleeding, and death.³⁵

With the exception of patients presenting with massive pulmonary embolism (defined by hemodynamic compromise), LMWH or fondaparinux is recommended over intravenous UFH. This is because of a more predictable bioavailability, more rapid onset of full anticoagulant effect, and benefit of not typically needing to monitor anticoagulant effect.

However, in cases in which an anticoagulant with a shorter half-life is more desirable (ie, patients at particularly high risk of bleeding) or in patients with impaired renal function, intravenous UFH may be preferred (grade 2C).³⁴

Oral anticoagulant therapy

The anticoagulant effect of warfarin is mediated by the inhibition of vitamin K–dependent factors, which are II, VII, IX, and X. The peak effect does not occur until 36–72 hours after drug administration, and the dosage is difficult to titrate.

A prothrombin time ratio is expressed as an INR and is monitored to assess the adequacy of warfarin therapy. The recommended therapeutic range for venous thromboembolism is an INR of 2–3. This level of anticoagulation markedly reduces the risk of bleeding without the loss of effectiveness. Initially, INR measurements are performed on a daily basis; once the patient is stabilized on a specific dose of warfarin, the INR determinations may be performed every 1–2 weeks or at longer intervals.

Duration of anticoagulation

A patient with a first thromboembolic event occurring in the setting of reversible risk factors such as immobilization, surgery, or trauma, should receive warfarin therapy for at least 3 months. Among patients with idiopathic (or unprovoked) first events, 2 studies have compared 6 versus 3 months of anticoagulant therapy and no difference in the rate of recurrence was observed in either study.^{36,37} The current recommendation is anticoagulation for at least 3 months in these patients, and the need for extending the duration of anticoagulation should be reevaluated at that time.

Warfarin treatment for longer than 6 months is indicated in patients with recurrent venous thromboembolism or in those in whom a continuing risk factor for venous thromboembolism exists, including malignancy, immobilization, or morbid obesity.

Patients who have pulmonary embolism and preexisting irreversible risk factors, such as deficiency of antithrombin III, protein S and C, factor V Leiden mutation, or the presence of antiphospholipid antibodies, should be placed on long-term anticoagulation.

Compression stockings: For patients who have had a proximal DVT, elastic compression stockings with a pressure of 30-40 mm Hg at the ankle for 2 years following the diagnosis is recommended (grade 1A) to reduce the risk of postphlebotic syndrome.

Surgical Care

Indication of Surgical Removal of Thrombi

When acute pulmonary thromboembolism is diffuse, the bilateral main pulmonary arteries are rapidly occluded and most patients could die within hours after onset. Furthermore, many mortalities due to acute pulmonary thromboembolism are caused by circulatory collapse in the stage immediately after the onset and by early recurrence. Therefore, in cases of circulatory failure or shock the main objective of treatment is to recanalize the occluded pulmonary arteries as quickly as possible. Generally, the indications for pulmonary thrombectomy in this situation are (1) cases whose hemodynamics are extremely unstable and who do not respond to medication, (2) cases with angiogram or CT scan findings of obstructions over a wide area of the pulmonary arteries, (3) cases showing rapid progression of heart failure or respiratory failure, (4) cases in which thrombolytic therapy is contraindicated, and (5) cases with thrombi suspended from the right atrium to the right ventricle.

Many cases that fall into sudden shock before this disease is diagnosed cannot be operated on. Postoperative cases or long-term clinical patients who experience sudden dyspnea or who show signs of hypoxemia or right ventricular dilatation in their echocardiograms should be suspected of acute pulmonary thromboembolism, and percutaneous cardiopulmonary support (PCPS) should start immediately at the bedside. Furthermore, if there are no fatal cerebrovascular complications, and circulatory collapse due to the thromboembolism is identified, pulmonary artery thrombectomy should be carried out. Patients may include some who have thromboembolism (subacute pulmonary thromboembolism) of more than 2 weeks duration. Such cases are difficult to treat with conventional thrombectomy and should be diagnosed and their surgical indication determined with care.

Surgical Treatment of Acute Pulmonary Thromboembolism

Surgical treatment of this condition involves removal of the thromboemboli by transvenous thrombectomy or open thrombectomy.

Transvenous Thrombectomy (Catheter Intervention)

This is a method whereby the thromboemboli are removed by suction using a catheter inserted transvenously into the pulmonary artery. Catheters used in this method are the Greenfield catheter, guiding catheter for percutaneous transluminal coronary angioplasty (PTCA), and the HYDROLYSER embolectomy catheter. This method can be used for patients in whom thrombolytic therapy is contraindicated, and there are many reports, such as that of Greenfield et al. , of its efficacy. Still, because of the ever-present danger of circulatory collapse, transvenous embolectomy should be carried out under conditions where open embolectomy or PCPS is possible.

Open Thrombectomy

With extracorporeal circulation using an artificial heart-lung, namely under cardiopulmonary bypass, the pulmonary artery is opened and the thromboemboli are removed. If the patient has respiratory failure or poor hemodynamics preoperatively, extracorporeal circulation is quickly initiated using the femoral artery and vein. In cases of shock in the ward where hemodynamics cannot be maintained, the patient is transported to the operating room with PCPS. In cases of circulatory collapse with this disease, the speed of initiation of extracorporeal circulation is the key determinant of survival.

As for the surgical technique, extracorporeal circulation is initiated after median sternal incision, incisions are made bilaterally in the main pulmonary arteries, and open thrombectomy is carried out. Thrombi of acute pulmonary thromboembolism

cases, in contrast to the organized thrombi of chronic cases, are soft and rod shaped, and the removed thrombi are relatively new and red. Although it is preferable to remove the thrombi as peripherally as possible, remaining thrombi can be treated postoperatively by thrombolytic methods if the major central thrombi have been removed. If they include those of more than 2 weeks duration (subacute pulmonary thromboembolism) that are firmly attached to the pulmonary arterial wall, care must be taken not to injure the arterial wall during the thrombectomy. The surgery can take place with the heart beating, but cardiac arrest is recommended for cases with multiple small thrombi in the segmental arteries or cases with thrombi firmly attached to the arterial wall.

The current grade 1A recommendation is that patients with acute pulmonary embolism should not routinely receive vena cava filters in addition to anticoagulants.

Inferior vena cava (IVC) interruption by the insertion of an IVC filter (Greenfield filter) is only indicated in the following settings:

Patients with acute venous thromboembolism who have an absolute contraindication to anticoagulant therapy (eg, recent surgery, hemorrhagic stroke, significant active or recent bleeding)

Patients with massive pulmonary embolism who survived but in whom recurrent embolism invariably will be fatal

Patients who have objectively documented recurrent venous thromboembolism, adequate anticoagulant therapy notwithstanding

An ideal IVC filter should have the following characteristics³⁸ :

Easy and safe placement by percutaneous technique

Biocompatible and mechanically stable

Ability to trap emboli without causing occlusion of the vena cava

One large trial has shown that during the first 12 days after insertion of IVC filters, significantly fewer patients had recurrent pulmonary embolism. However, following a 2-year follow-up, no significant differences in survival rates existed between the 2 groups. Furthermore, significantly higher rates of recurrent DVT occurred among patients who received an IVC filter. Other complications of IVC filters include proximal migration of the filter into the right-sided heart chambers and perforation of the IVC.



Temporary inferior vena cava

Bleeding/hematoma at puncture site: 16 (27.5%)
 Thrombolysis for intrafilter thrombus: 7 (12.1%)
 Slight fever: 6 (9.7%)
 Retroperitoneal hematoma [percutaneous cardiopulmonary support (PCPS) site]: 1 (1.7%)
 IVC occlusion: 1 (1.7%)
 Filter migration: 1 (1.7%)
 Pneumothorax: 0 (0%)

Complications of temporary IVC filter

aPE may recur asymptotically, and aPE recurrence has no specific symptoms. Even if any symptoms appear and are recorded by echocardiography, blood flow scintigraphy, CT, etc., similar records of the patient immediately before the events must be available for comparison to confirm that they are indeed symptoms of recurrence. That would be impossible, and there are many obstacles to obtaining an accurate recurrence rate. We therefore evaluated symptoms that appeared anew during acute-stage management of aPE which, although not perfect, are highly reliable indicators of aPE recurrence.

The results suggested that tIVCF users present fewer new symptoms during use and are easier to control. The tIVCF user who had a recurrence and died had a giant ovarian cancer with the tip of the tumor extending to the epigastrium. The inferior vena cava was extruded directly below the diaphragm by the tumor, and the tIVCF had to be placed inside the flattened inferior vena cava. When the Neuhaus Protect, which is made of Teflon, is dilated inside a flattened inferior vena cava it is easily distorted, so its

ability to capture thrombi must have been compromised. On the other hand, we thought it was highly probable that a metallic tIVCF would injure the vascular wall and decided against it. Secondary prevention in cases like this is considered to be an important issue for the future.

The most frequent complication of tIVCF was bleeding/hematoma of the puncture site. Because the basic treatments for aPE are anticoagulant therapy and fibrinolytic therapy, and the shaft of the tIVCF is extracorporeal, caution is constantly required to prevent this complication. The best way to prevent it is to improve the attending clinician's technique and to puncture the vein at the first attempt. The complications that are generally cited in connection with tIVCF use are hemorrhagic complications caused by basic aPE therapy, filter displacement, venous thrombus, infections, air embolism, and filter breakage. Thrombus in the filter site is another complication that should be considered. When contrast radiography of the filter site at the time of removal revealed a thrombus occupying more than one-fourth of the lumen, we administered fibrinolytic treatment from the filter tip. The thrombus size was reduced in all cases, and the filter could be removed in a few days without any problems.

The major reason for using a pIVCF was poor ADL in the tIVCF users and APE recurrence in the nonusers. ADL is determined by the underlying disease condition and poor ADL is a major risk factor for DVT, so the use of pIVCF was inevitable in such cases. pIVCF use due to aPE recurrence could be avoided by using tIVCF, which is considered to be clinically effective in acute-stage control.

The basic principle of aPE prevention is extended ADL. Long-term bed rest slows the venous blood flow rate and encourages the formation of venous thrombi, so it should be avoided as much as possible. Although recurrence could easily occur when shifting from bed rest to expanded ADL, we believe that if unstable DVT entering the venous bloodstream are captured by the tIVCF, expanded ADL can proceed safely in the acute phase.

As retrievable filters that can be removed with certainty are developed in the future, we can look forward to fewer complications of bleeding and infections and the realization of aPE acute-stage management that is safer than ever before.

Activity

Activity is recommended as tolerated. Early ambulation is recommended over bed rest when feasible (grade 1A recommendation).

Medication

Immediate therapeutic anticoagulation is initiated for patients with suspected deep venous thrombosis (DVT) or pulmonary embolism (PE). Anticoagulation therapy with heparin reduces mortality rates from 30% to less than 10%. Thrombolytic therapy is recommended for 3 groups of patients: (1) those patients who are hemodynamically unstable, (2) those who have right-sided heart strain, and (3) those who have limited cardiopulmonary reserve.

Chronic anticoagulation is critical to prevent relapse of DVT or pulmonary embolism following initial heparinization. The optimum duration of anticoagulation has not been well studied and is controversial. The general consensus is that a significant reduction in recurrence is associated with 3-6 months of anticoagulation.

Thrombolytics

Thrombolysis is indicated for hemodynamically unstable patients with pulmonary embolism. Thrombolysis dramatically improves acute cor pulmonale. Thrombolytic therapy has replaced surgical embolectomy as the treatment for hemodynamically

unstable patients with massive pulmonary embolism.

Thrombolytic regimens currently in use for pulmonary embolism include 2 forms of recombinant tissue-plasminogen activators, alteplase (t-PA) and reteplase (r-PA), along with urokinase and streptokinase. The comparative clinical trials have shown that administration of a 1-h infusion of alteplase is more rapidly effective than urokinase or streptokinase over a 12-h period. The safety and efficacy of different thrombolytic agents is comparable. Streptokinase may cause anaphylaxis, hypotension, and other adverse reactions, leading to the cessation of therapy in many cases.

Wang et al conducted a prospective, randomized, multicenter trial in 118 patients with acute pulmonary thromboembolism (PTE) and either hemodynamic instability or massive pulmonary artery obstruction. 40 Patients were randomly assigned to receive either alteplase (rt-PA) at 50 mg IV infused over 2 h (n = 65) or the current FDA-approved dose of 100 mg IV infused over 2 h (n = 53). Efficacy was measured by improvements of right ventricular dysfunction (RVD) on echocardiograms, lung perfusion defects on ventilation perfusion lung scans, and pulmonary artery obstructions on CT angiograms. Adverse events, including death, bleeding, and PTE recurrence, were also evaluated.

Progressive improvements in RVDs, lung perfusion defects, and pulmonary artery obstructions were found to be similarly significant in both treatment groups. No difference in outcome was observed for patients with hemodynamic instability or massive pulmonary artery obstruction. Three (6%) patients in the 100-mg group and 1 (2%) in the 50-mg group died as the result of either PTE or bleeding. The 50-mg regimen resulted in less bleeding tendency than the 100 mg/2 h regimen (3% vs 10%), especially in patients with a body weight of less than 65 kg (14.8% vs 41.2%, $P = .049$). Fatal recurrent PTE did not occur in either group.

Rarely, empiric thrombolysis may be indicated in selected patients who are hemodynamically unstable, eg, the clinical likelihood of pulmonary embolism is overwhelming and the patient's condition is rapidly deteriorating (with the possibility of imminent death). In such patients, the possible risk of severe complications from thrombolysis should be carefully evaluated against the potential benefits.

Reteplase (Retavase)

Second-generation recombinant plasminogen activator that forms plasmin after facilitating cleavage of endogenous plasminogen. In clinical trials, shown to be comparable to t-PA in achieving TIMI, 2 or 3 patency, at 90 min. Given as a single bolus or as 2 boluses administered 30 min apart.

As a fibrinolytic agent, seems to work faster than its forerunner, t-PA, and may be more effective in patients with larger clot burdens. Also reported to be more effective than other agents in lysis of older clots. Two major differences help explain these improvements. Compared with t-PA, r-PA does not bind fibrin so tightly, allowing the drug to diffuse more freely through the clot. Another advantage seems to be that it does not compete with plasminogen for fibrin-binding sites, allowing plasminogen at the site of the clot to be transformed into clot-dissolving plasmin.

The FDA has not approved r-PA for use in patients with PE. Studies for PE have used the same dose approved by the FDA for coronary artery fibrinolysis.

Alteplase (Activase)

Used in management of AMI, acute ischemic stroke, and PE. Drug most often used to treat patients with PE in the ED. Usually given as a front-loaded infusion over 90-120 min. FDA-approved for this indication. Most ED personnel are familiar with its use because it is widely used for treatment of patients with AMI. An accelerated 90-min regimen is widely used, and most believe it is both safer and more effective than the approved 2-h infusion. Accelerated regimen dose is based on patient weight.

Heparin therapy should be instituted or reinstated near the end of or immediately following infusion, when the aPTT or

thrombin time returns to twice normal or less.

Urokinase (Abbokinase)

Direct plasminogen activator produced by human fetal kidney cells grown in culture. Acts on the endogenous fibrinolytic system and converts plasminogen to the enzyme plasmin, which, in turn, degrades fibrin clots, fibrinogen, and other plasma proteins. Advantage is that this agent is nonantigenic; however, more expensive than streptokinase and, thus, limits use. When used for localized fibrinolysis, given as local catheter-directed continuous infusion directly into area of thrombus with no loading dose. When used for PE, loading dose is necessary.

Streptokinase (Kabikinase, Streptase)

Acts with plasminogen to convert plasminogen to plasmin. Plasmin degrades fibrin clots, fibrinogen, and other plasma proteins. Increase in fibrinolytic activity that degrades fibrinogen levels for 24-36 h takes place with IV infusion of streptokinase. Highly antigenic. Highly likely that treatment will be interrupted due to allergic drug reactions.

Chills, fever, nausea, and skin rashes are frequent (up to 20%). Blood pressure and heart rate drop in approximately 10% of cases during or shortly after treatment.

Late complications may include purpura, respiratory distress syndrome, serum sickness, Guillain-Barré syndrome, vasculitis, and renal or hepatic dysfunction.

Anticoagulants

Heparin augments activity of the natural anticoagulant antithrombin III and prevents conversion of fibrinogen to fibrin. Full-dose LMWH or unfractionated IV heparin should be initiated at the first suspicion of DVT or pulmonary embolism. Heparin does not dissolve an existing clot, but it does prevent clot propagation and embolization. Recurrence or extension of DVT and pulmonary embolism may occur despite therapeutic anticoagulation with heparin.

With proper dosing, several LMWH products have been found to be safer and more effective than UFH for prophylaxis and treatment of patients with DVT and pulmonary embolism. Not necessary or useful to monitor aPTT while using LMWH. Drug is most active in tissue phase, and, as opposed to UFH, LMWH does not exert most of its effects on coagulation factor IIa.

Many different LMWH products are currently available. Because of the pharmacokinetic differences, dosing and interval of administration is highly product-specific. Presently, 3 LMWH products are available in the United States (enoxaparin, dalteparin, ardeparin). Enoxaparin is the only one that is approved by the FDA for treatment of patients with DVT. The FDA has approved all 3 for DVT prophylaxis at a lower dose. LMWH administered via subcutaneous route is preferred for commencing anticoagulation therapy. Maintenance therapy with warfarin usually is initiated simultaneously. The weight-adjusted heparin dosing regimens have proven to be efficacious for treatment of patients with DVT and pulmonary embolism and are endorsed by the experts.

Enoxaparin (Lovenox)

Enhances inhibition of factor Xa and thrombin by increasing antithrombin III activity. In addition, preferentially increases inhibition of factor Xa. First LMWH in United States. Only LMWH approved by FDA for treatment and prophylaxis of DVT and PE. Widely used in pregnancy, although clinical trials are not yet available to demonstrate that it is as safe as UFH.

Dalteparin (Fragmin)

LMWH with many similarities to enoxaparin but with a different dosing schedule. Approved for DVT prophylaxis in patients undergoing abdominal surgery. Except in overdoses, no utility exists in checking PT or aPTT because aPTT does not correlate with anticoagulant effect of fractionated LMWH.

Ardeparin (Normiflo)

LMWH recently released in United States for DVT prophylaxis in patients undergoing hip and knee surgery. Except in overdoses, no utility exists in checking PT or aPTT because the aPTT does not correlate with anticoagulant effect of fractionated LMWH.

Heparin (Hep-Lock, Liquaemin)

Augments activity of antithrombin III and prevents conversion of fibrinogen to fibrin. Does not actively lyse but is able to inhibit further thrombogenesis. Prevents reaccumulation of clot after spontaneous fibrinolysis. When UFH is used, the aPTT should not be checked until 6 h after the initial heparin bolus because an extremely high or low value during this time should not provoke any action.

Interferes with hepatic synthesis of vitamin K–dependent coagulation factors. Used for prophylaxis and treatment of venous thrombosis, PE, and thromboembolic disorders. Never administer to patients with thrombosis until after fully anticoagulated with heparin (first few days of warfarin therapy produce a hypercoagulable state). Failing to anticoagulate with heparin before starting warfarin causes clot extension and recurrent thromboembolism in approximately 40% of patients, compared with 8% of those who receive full-dose heparin before starting warfarin. Heparin should be continued for the first 5-7 d of oral warfarin therapy, regardless of the PT time, to allow time for depletion of procoagulant vitamin K–dependent proteins.

Tailor dose to maintain an INR in the range of 2.5-3.5. Risk of serious bleeding (including hemorrhagic stroke) is approximately constant when the INR is 2.5-4.5 but rises dramatically when the INR is >5. In the United Kingdom, higher INR target of 3-4 often is recommended.

Evidence suggests that 6 mo of anticoagulation reduces rate of recurrence to half of the recurrence rate observed when only 6 wk of anticoagulation is given. Long-term anticoagulation is indicated for patients with an irreversible underlying risk factor and recurrent DVT or recurrent PE.

Procoagulant vitamin K–dependent proteins are responsible for a transient hypercoagulable state when warfarin is first started and stopped. This is the phenomenon that occasionally causes warfarin-induced necrosis of large areas of skin or of distal appendages. Heparin is always used to protect against this hypercoagulability when warfarin is started; but, when warfarin is stopped, the problem resurfaces, causing an abrupt temporary rise in the rate of recurrent venous thromboembolism.

At least 186 different foods and drugs reportedly interact with warfarin. Clinically significant interactions have been verified for a total of 26 common drugs and foods, including 6 antibiotics and 5 cardiac drugs. Every effort should be made to keep the patient adequately anticoagulated at all times because procoagulant factors recover first when warfarin therapy is inadequate.

Patients who have difficulty maintaining adequate anticoagulation while taking warfarin may be asked to limit their intake of foods that contain vitamin K.

Foods that have moderate to high amounts of vitamin K include Brussels sprouts, kale, green tea, asparagus, avocado, broccoli, cabbage, cauliflower, collard greens, liver, soybean oil, soybeans, certain beans, mustard greens, peas (black-eyed peas, split peas, chick peas), turnip greens, parsley, green onions, spinach, and lettuce.

Fondaparinux sodium (Arixtra)

Synthetic anticoagulant that works by inhibiting factor Xa, a key component involved in blood clotting. Provides highly predictable response. Bioavailability is 100%. Has a rapid onset of action and a half-life of 14-16 h, allowing for sustained

antithrombotic activity over 24-h period. Does not affect prothrombin time or activated partial thromboplastin time, nor does it affect platelet function or aggregation.

Deterrence/Prevention

Heparin prophylaxis

The incidence of venous thrombosis, pulmonary embolism (PE), and death can be significantly reduced by embracing a prophylactic strategy in high-risk patients. Prevention of deep vein thrombosis (DVT) in the lower extremities inevitably reduces the frequency of pulmonary embolism; therefore, populations at risk must be identified, and safe and efficacious prophylactic modalities should be used. The risk groups identified in clinical practice and the prophylaxis recommended by the Sixth Consensus Conference on Antithrombotic Therapy are described in the Table.

Prophylaxis Against Venous Thromboembolism

Condition	Risk (% P)	General Surgery	Recommendations
Low risk	3	(1) Early ambulation	
Moderate risk	29	(1) Unfractionated heparin: 5000 U OC given 2 h preoperatively and q12h postoperatively (2) Dalteparin: 2500 U 1-2 hr before surgery, then once daily Enoxaparin: 2000 U before surgery, then once daily Nadroparin: 3100 U 2 hr before surgery, then once daily Tinzaparin: 3500 U 2 hr before surgery, then once daily	
High risk	39	(1) Unfractionated heparin: 5000 U OC given 2 h preoperatively and q12h postoperatively (2) Dalteparin: 5000 U 10-12 hr before surgery, then once daily Enoxaparin: 4000 U 10-12 hr before surgery, then once daily	
Very high risk	80	(1) Unfractionated heparin: 5000 U OC given 2 h preoperatively and q12h postoperatively; dalteparin: 2500 U given 2 h preoperatively and q6; plus, intermittent pneumatic compression applied intraoperatively (2) Dalteparin: 5000 U 10-12 hr before surgery, then once daily Enoxaparin: 4000 U 10-12 hr before surgery, then once daily (3) Perioperative warfarin: INR 2-3	
Orthopedic Surgery/Neurological Surgery/Trauma			
Total hip replacement	51	(1) Dalteparin: 5000 U 1-2 hr before surgery, then once daily Enoxaparin: 3000 U 10-12 hr before surgery, then once daily Nadroparin: 40 U/kg 2 hr before surgery, then once daily Tinzaparin: 50 U/kg 2 hr before surgery, then 75 U/kg once daily (2) Warfarin: Preoperatively and adjusted to INR of 2-3 postoperatively, continue up to 4 wk after surgery	
Total knee replacement	61	(1) Dalteparin: 5000 U 1-2 hr before surgery, then once daily Enoxaparin: 3000 U 10-12 hr before surgery, then once daily Nadroparin: 40 U/kg 2 hr before surgery, then once daily Tinzaparin: 50 U/kg 2 hr before surgery, then 75 U/kg once daily (2) Warfarin: Preoperatively and adjusted to INR of 2-3 postoperatively, continue up to 4 wk after surgery	
Hip fracture surgery	46	(1) Dalteparin: 5000 U 1-2 hr before surgery, then once daily Enoxaparin: 3000 U 10-12 hr before surgery, then once daily Nadroparin: 40 U/kg 2 hr before surgery, then once daily Tinzaparin: 50 U/kg 2 hr before surgery, then 75 U/kg once daily (2) Warfarin: Preoperatively and adjusted to INR of 2-3 postoperatively, continue up to 4 wk after surgery	
Neurosurgery	24	(1) Intermittent pneumatic compression (2) Unfractionated heparin: 5000 U OC q12h and intermittent pneumatic compression for high-risk patients	
Acute spinal cord injury with leg paralysis	40	(1) Unfractionated heparin: OC in doses adjusted to paralytic produce aPTT = 1.5 × control 6 h after dose (2) Enoxaparin: 3000 U twice daily (3) Warfarin: Adjusted to INR of 2-3 in rehabilitation phase (4) Intermittent pneumatic compression plus unfractionated heparin: 5000 U OC q12h	
Multiple trauma	53	(1) Intermittent pneumatic compression until further bleeding is unlikely; then, give (2) Enoxaparin: 30 mg OC q12h or (3) Warfarin: Adjusted to INR of 2-3	
Medical Conditions			
Acute myocardial infarction	24	Unfractionated heparin: 5000 U OC q12h unless therapeutic anticoagulation used	
Ischemic stroke with paralysis	42	Unfractionated heparin: 5000 U OC q12h	
Medical patients (cancer, bedrest, congestive heart failure, severe lung disease)	20	(1) Unfractionated heparin: 5000 U OC q12h (2) Dalteparin: 2500 U once daily Enoxaparin: 2000 U once daily	

Sequential compression devices

Compression stockings provide a compression of 30-40 mm Hg gradient and are a safe and effective therapy to prevent venous thromboembolism in patients who are at high risk when heparin therapy is not desirable or is contraindicated. These devices provide a gradient of compression that is highest at the toes and gradually decreases to the level of the thigh. This mechanism reduces the capacitative venous volume by approximately 70% and increases the measured velocity of blood flow by a factor of 5 or more in lower extremity veins.

A meta-analysis calculated a DVT risk ratio of 0.28 for gradient compression stockings (compared with no prophylaxis) in patients undergoing abdominal surgery, gynecologic surgery, or neurosurgery. Other studies have reported that gradient

compression stockings and low molecular weight heparin (LMWH) were the most effective modalities in reducing the incidence of DVT after hip surgery.

The universal white stockings, known as antiembolic stockings or Ted stockings, produce a maximum compression of only 18 mm Hg. Ted stockings rarely are fitted in such a way as to provide adequate gradient compression to the deep venous system. Therefore, Ted stockings have no proven efficacy in the prevention of DVT and pulmonary embolism.

Gradient compression pantyhose (30-40 mmHg) are available in pregnant sizes. They are recommended by many specialists for all women who are pregnant because they prevent DVT and reduce or prevent the development of varicose veins.

Although strict bed rest was recommended in the past for acute DVT to reduce the risk of pulmonary embolism, a study has shown no benefit from prescribing bed rest. Therefore, strict bed rest for 5 days is not justified if adequate therapy with LMWH and adequate compression is assured.

Complications

Sudden cardiac death

Obstructive shock

Pulseless electrical activity

Atrial or ventricular arrhythmias

Secondary pulmonary arterial hypertension

Cor pulmonale

Severe hypoxemia

Right-to-left intracardiac shunt

Lung infarction

Pleural effusion

Paradoxical embolism

Prognosis

The prognosis of patients with pulmonary embolism depends on 2 factors: (1) the underlying disease state and (2) appropriate diagnosis and treatment.

Most patients treated with anticoagulants do not develop long-term sequelae upon follow-up evaluation.

At 5 days of anticoagulant therapy, 36% of lung scan defects are resolved; at 2 weeks, 52% are resolved; at 3 months, 73% are resolved.

The mortality rate in patients with undiagnosed pulmonary embolism is 30%.

Elevated plasma levels of natriuretic peptides (brain natriuretic peptide and N-terminal pro-brain natriuretic peptide) have been associated with higher mortality in patients with pulmonary embolism.⁴¹ In one study, levels of N-terminal pro-brain natriuretic peptide greater than 500 ng/L was independently associated with central pulmonary embolism and was a possible predictor of death from pulmonary embolism.

In the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) (PIOPED) study, the 1-year mortality rate was 24%.⁷ The deaths occurred due to cardiac disease, recurrent pulmonary embolism, infection, and cancer.

The risk of recurrent pulmonary embolism is due to the recurrence of proximal venous thrombosis; approximately 17% of patients with recurrent pulmonary embolism were found to have proximal DVT.

In a small proportion of patients, pulmonary embolism does not resolve; hence, chronic thromboembolic pulmonary arterial hypertension results.

Medicolegal Pitfalls

Pulmonary embolism (PE) is an extremely common disorder. It presents with nonspecific clinical features and requires specialized investigations for confirmation of diagnosis. Therefore, many patients die from unrecognized pulmonary embolism. The other common pitfalls are as follows:

- Disregarding patient's complaints of unexplained dyspnea as anxiety or hyperventilation

- Blaming complaints of unexplained chest pain on musculoskeletal pain

- Failing to recognize, diagnose, and treat deep vein thrombosis (DVT)

- Failing to initiate an appropriate diagnostic workup in patients with symptoms consistent with pulmonary embolism

- Failing to initiate therapeutic anticoagulant therapy with heparin in patients suspected to have pulmonary embolism, before the V/Q scan or other investigations

The role of thrombolytic therapy in patients who are hemodynamically stable remains uncertain. No particular diagnostic strategy appears to be superior to another at present. More clinical studies are needed to evaluate the utility of new diagnostic approaches for pulmonary embolism. The availability of the diagnostic tests, the expertise of the radiologists, cost-effective analysis, and local traditions appear to be the considerations in the workup of a patient suspected to have pulmonary embolism.

Special Concerns

Pregnancy

The risk of venous thromboembolism is increased during pregnancy and the postpartum period. Pregnant women who are in a hypercoagulable state or have had previous venous thromboembolism should receive prophylactic anticoagulation during pregnancy.

Pulmonary embolism is the leading cause of death in pregnancy. Guidelines by the professional societies make this difficult diagnosis easier and reduce the risks of radiation to the fetus. If the patient has a low pretest probability for pulmonary embolism and a normal D-dimer test result, clinical exclusion from further investigations is recommended. When the suspicion is high, the patients should have bilateral leg Doppler assessment. If the results are positive, the patient should be treated for pulmonary embolism. If the results are negative, CT pulmonary angiography is the next step. To rule out contrast-induced hypothyroidism, all neonates exposed to the iodinated contrast in utero should have their serum thyrotropin level checked in the first week of life.

Pregnant patients diagnosed with DVT or pulmonary embolism are treated with unfractionated heparin or LMWH throughout their pregnancy. Warfarin is contraindicated because it crosses the placental barrier and can cause fetal malformations. Therefore, either subcutaneous unfractionated heparin or LMWH at full anticoagulation doses should be continued until delivery. Women experiencing a thromboembolic event during pregnancy should receive therapeutic treatment with unfractionated heparin or LMWH during pregnancy, with anticoagulation continuing for 4-6 weeks postpartum, and for a total of at least 6 months.

Heparin-induced thrombocytopenia

Heparin-induced thrombocytopenia (HIT) is a transient prothrombotic disorder initiated by heparin.

Main features are (1) thrombocytopenia resulting from immunoglobulin G-mediated platelet activation and (2) in vivo thrombin generation and increased risk of venous and arterial thrombosis.

The highest frequency of HIT, 5%, has been reported in post-orthopedic surgery patients receiving up to 2 weeks of unfractionated heparin. HIT occurred in approximately 0.5% of post-orthopedic surgery patients receiving LMWH for up to 2 weeks.

HIT may manifest clinically as extension of the thrombus or formation of new arterial thrombosis. HIT should be suspected whenever the patient's platelet count falls to less than 100,000/ μ L or less than 50% of the baseline value, generally after 5-15 days of heparin therapy. The definitive diagnosis is made by performing a platelet activation factor assay.

The treatment of patients who develop HIT is to stop all heparin products, including catheter flushes and heparin-coated catheters, and to initiate an alternative nonheparin anticoagulant, even when thrombosis is not clinically apparent. Preferred agents include direct thrombin inhibitors such as lepirudin or argatroban. Start warfarin while the patient receives an alternative nonheparin anticoagulant and only when the platelet count has recovered to at least 100,000/ μ L, preferably 150,000/ μ L.

Resistance to heparin

Few patients with venous thromboembolism require large doses of heparin for achieving an optimal activated partial thromboplastin time (aPTT). These patients have increased plasma concentrations of factor VIII and heparin-binding proteins. Increased factor VIII concentration causes a dissociation between aPTT and plasma heparin values. The aPTT is suboptimal, but patients have adequate heparin levels upon protamine titration. This commonly occurs in patients with a concomitant inflammatory disease.

Monitoring the antifactor Xa assay results in this situation is safe and effective and results in less escalation of the heparin dose when compared with monitoring with aPTT. Whenever a therapeutic level of aPTT cannot be achieved with large doses of unfractionated heparin administration, either determination of plasma heparin concentration or therapy with LMWH should be instituted.

Elderly individuals

Pulmonary embolism is increasingly prevalent among elderly patients, yet the diagnosis is missed more often in this population because respiratory symptoms often are dismissed as being chronic.

Even when the diagnosis is made, appropriate therapy frequently is inappropriately withheld because of bleeding concerns.

An appropriate diagnostic workup and therapeutic anticoagulation with a careful risk-to-benefit assessment is recommended in this patient population.

Future research

The advances over the past several decades have significantly improved diagnostic abilities and have refined the treatment of patients with pulmonary embolism. However, several areas need further research and properly conducted therapeutic trials. The role of LMWH and the optimal duration of anticoagulant therapy in different subgroups of patients with venous thromboembolism require further study.

Future studies should determine whether less intense warfarin therapy (international normalized ratio [INR] less than 2), which will result in less bleeding, is effective in preventing recurrences.

Whether drugs that inhibit the action of thrombin (eg, hirudin) are useful in treating patients with venous thromboembolic

disease needs to be determined by future trials.

TOPIC 10. VASCULAR INJURY

VASCULAR INJURIES IN THE ARM

Arteries and veins in the arms are the second most common location for vessel injuries in the body and constitute almost half of all peripheral vascular injuries. Much more often than in the legs they occur together with neurological and skeletal injuries. Although vascular injuries in the arms rarely lead to fatal or serious bleeding, ischemic consequences are common. The extensive collateral network around the elbow makes clinical signs variable and often minute. On the other hand, if the brachial artery is obstructed proximal to the origin of the deep brachial artery, the risk for amputation is substantial: up to 50% of such patients lose the arm if the vessel is not repaired. While the vascular injury per se often can be managed easily, it is the damaged nerves that cause the main functional disturbances in the long run.

Because arm vessel trauma is common and sometimes appear without signs and symptoms, missed injuries cause considerable morbidity in trauma patients. Awareness and optimal management may reduce this morbidity. The arteries supplying blood to the arms – the subclavian and axillary arteries – are located in the thorax or thoracic outlet, and if these vessels are traumatized, the consequences are often more serious.

Etiology and Pathophysiology

Injury mechanisms are the same in the arms and legs, and the brachial, radial, and ulnar arteries can be damaged by both penetrating and blunt trauma. Knives and gunshots usually cause penetrating injuries (most often to the brachial artery), but lacerations secondary to fractures occur regularly. Sharp fragments commonly penetrate vessel walls (Fig. 1).



Fig. 1. Angiography showing an occluded brachial artery severed by the sharp ends of a shaft fracture of the humerus

Blunt injuries occur in road traffic accidents because of fractures and joint dislocations. The most frequent orthopedic arm injuries associated with vessel damage are listed in Table 1.

Table 1. Most common sites for combined orthopedic and vascular injury

Orthopedic injury	Vascular injury
Fractured clavicle	Subclavian artery
Shoulder dislocation	Axillary artery
Supracondylar fracture of the humerus	Brachial artery
Elbow dislocation	Brachial artery

There are also types of trauma specific for arm vessel injuries. A large number of upper limb vascular trauma are caused by industrial and domestic accidents. Splintered glass as well as self-in-flicted wounds regularly damage vessels below the elbow. The popularity of using the brachial artery as a site for vascular access for endovascular procedures has caused an increase in iatrogenic catheter-related injuries to the brachial artery in proximity to the elbow. Pseudoaneurysms are often caused by radial artery punctures for arterial blood samples.

As in all traumatized vessels, transection or laceration may cause bleeding, thrombosis, or both. Transections, intimal tears, and contusions are more frequent after blunt trauma. Tissue in the distal parts of the arm is as susceptible to ischemia as in the legs, and the time limit of 6–8h before irreversible damage occurs is also valid for arm injuries. Concomitant nerve injuries, as mentioned, are the main cause of morbidity long term. Such injuries are equally common after penetrating and blunt trauma. In the literature, 35–60% of arterial injuries in the upper arm are associated with nerve injuries, and over 75% are associated with nerve, bone, or venous damage.

Clinical Presentation

Medical History

Patients with vascular injuries in the arms arrive at the emergency department after accidents, knife or shooting assaults, or car crashes causing multiple injuries. As with all injuries, it is important to interview the rescue personnel and accompanying persons about the type of injury and the type of bleeding. The exact time of the injury should be established to facilitate planning of repair. Because orthopedic injuries are associated with arterial damage, it

is also essential to ask whether joint dislocations or fractures were noted or reduced. Complaints of pain from areas around a joint indicate a possible luxation. Even more important is to ask for symptoms of nerve damage, including permanent or transient numbness and impaired motor function in any part of the arm.

Clinical Signs and Symptoms

Both the “hard” and the “soft” signs of vascular trauma occur after upper extremity vascular injuries. Examples, in descending frequency of occurrence, include diminished or absent radial pulse, motor deficit, sensory loss, hemorrhage, and expanding hematoma. It is common for a diminished radial pulse or an abnormal brachial blood pressure to be the only sign of vascular obstruction. Because pulse wave propagation through a thrombus is possible, a palpable radial pulse does not completely exclude arterial obstruction; therefore, a high suspicion of arterial injury is necessary even when a palpable pulse is found. As a guideline, a difference of more than 20 mmHg in blood pressure between the arms should make the examiner suspect a vascular injury. Inability to move the fingers, hands, and arms as well as disturbances in sensation are frequently associated with vascular injury. The sensory and motor functions must therefore be carefully examined and evaluated to disclose any nerve damage that should be repaired. A list of what this examination should cover is given in Table 2. In unconscious patients, this examination is the only way to reveal indications of arterial damage.

Diagnostics

Investigations beyond the physical examination of the patient should be done only in stable patients. Accordingly, most patients with distal arm injuries can undergo angiography or duplex scanning provided that these investigations do not delay treatment. Arteriography is indicated when arterial involvement is not obvious. For example, patients with trauma in the elbow region – blunt or penetrating – with clear ischemia and no radial pulse do not need arteriography before surgery.

If the patient has multiple injuries, shotgun injuries, or suspected proximal arterial involvement, arteriography is recommended to determine the exact site of injury. Arteriography should also be done when there are indistinct signs of ischemia and arterial injury is only suspected. Included in this indication for arteriography is the so-called proximity injury, referring to injury in patients without signs of distal ischemia but with trauma in close

proximity to a major artery. Of patients undergoing arteriography for this indication, 10–20% are reported to have arterial lesions. If the arteriogram reveals injury to the subclavian or axillary artery, endovascular treatment can proceed right away. Duplex scanning has replaced arteriography in some hospitals and is probably just as accurate in experienced hands. Intimal flaps and small areas of vascular wall thrombosis may be difficult to identify with duplex scanning under some circumstances, but such small lesions in the arm can, on the other hand, usually be treated without exploration.

Computed tomography (CT) angiography is an important modality for diagnosing proximal arterial injuries in particular. It is reported to be at least as accurate as arteriography in this area. The use of CT angiography is likely to increase in the near future because it is quicker than angiography, and most trauma centers have rapid access to good-quality CT.

Management Before Treatment

Severely Injured and Unstable Patients

Patients arriving to the emergency department with active serious bleeding after a single injury to an arm are rare. When this does occur, manual direct pressure over the wound can control the bleeding while general resuscitation measures according to Advanced Trauma Life Support principles are undertaken: oxygen, monitoring of vital signs, placement of intravenous (IV) lines, and infusion of fluids. It is important not to forget to administer analgesics (5–10mg of an opiate IV) and, when indicated, antibiotics and tetanus prophylaxis.

Multiply injured patients with signs of arm ischemia should be treated according to the hospital's general trauma management protocol, and the vascular injury is usually evaluated during the second survey. Serious ongoing bleeding has high priority, but arm ischemia should be managed after resuscitation and treatment of life-threatening injuries but before orthopedic repair in most circumstances. When the patient has stabilized, arteriography can be performed if indicated.

Less Severe Injuries

Most patients with arm injuries arrive in the emergency department in a stable condition without ongoing bleeding but with signs of hand ischemia. For these patients, careful examination of the arm including assessment of nerve function, is essential. Dislocated

fractures or luxations should be reduced under proper analgesia. After reduction, examination of vascular function should be repeated. If the radial pulse and distal perfusion return, the position should be stabilized and fixed. Repeated examinations during the following 4h are mandatory to ensure that the returned perfusion is persistent.

If the vessel injury is definite – an absent radial pulse and reduced hand perfusion – and the site of vascular injury is apparent, the patient can be transferred to the operating room without further diagnostic measures. Patients with findings indicating vascular injury at examination, and those with obvious arterial disruption but with arms so traumatized that the site of arterial injury cannot be determined, should undergo arteriography or duplex scanning. Expediency of repair is required for all locations of arterial injuries in the arm. The proposed time limits indicating a low risk for permanent tissue damage range from 4h for brachial artery injuries and up to 12h for forearm injuries. The risk limit for irreversible ischemia following forearm injuries is valid for patients with an incomplete palmar arch. The frequency of this anatomical variation is 20% in most Western populations.

Suspected injuries to the radial and ulnar artery should be treated according to the general principles discussed above. Even cases with normal perfusion in the hand but without a radial pulse should be explored and repaired if reasonably simple. When forearm arterial injury is unclear, the Allen test (Table 2) can be added to the examination procedure. A positive Allen test together with a history of trauma to an area in close proximity indicate that the radial or ulnar artery is indeed affected. The wound should then be explored and the traumatized artery inspected and mended. Patients with multiple severe injuries and high-risk patients should not be explored if perfusion to the hand is rendered sufficient. For those circumstances, repeat examinations every hour are mandatory to make sure that perfusion is adequate and stable.

Table 2. Allen test

1. Elevate the arm over the head
2. Occlude the radial and ulnar arteries at the wrist
4. Lower the arm
5. Release blood flow through the ulnar artery
6. Inspect and time the return of perfusion
7. Repeat, and release blood flow through the radial artery instead.
8. A return of perfusion >5 s is considered a positive Allen test, and the artery is suspected to be inadequate

Amputation

Some arms with vascular injuries are so extensively damaged that amputation is a treatment option. The decision of when to perform a primary amputation versus trying to repair vessels, nerves, tendons, and muscles is difficult. As a general principle, arms with multiple fractures, nerve disruption, ischemia-time longer than 6h, and extensive crush injuries involving muscle and skin will never regain function and should be amputated. Another principle is that when four out of the five components of the arm are injured – skin, bone, muscles, and vessels – but there is only minor nerve injury, an attempt to save the arm is reasonable. One must keep in mind, however, that the arm needs at least some protective sensation in order to be functional. Children have a greater chance of regaining a functional arm than adults do, and a generous attitude to surgical repair in children is recommended.

The mangled extremity severity score (MESS) is a grading system designed to aid the decision process for managing massive upper and lower extremity trauma. A score of 7 or more has been proposed as a cut-off value for indicating when amputation cannot be avoided and should be performed as the primary procedure. In some studies, a score 7 predicted an eventual amputation with 100% accuracy. The basis of the MESS scoring system is given in Table 3. As shown in Table 3.4, a crush injury is regarded as particularly unfavorable. The duration of ischemia is also a significant factor taken into account in the MESS system.

Table 3. MESS: Mangled Extremity Severity Score (BP blood pressure)

Types	Injury characteristics	Points
Low energy	Sab wounds, simple closed fractures, small-caliber gunshot wounds	1
Medium energy	Open fractures, multiple fractures, dislocations, small crush injuries	2
High energy	Shotgun blasts, high-velocity gunshot wounds	3
Massive crush	Logging, railroad accidents	4
No shock (BP normal)	BP stable at the site and at the hospital	1
Transient hypotension	BP unstable at the site but normalizes after fluid substitution	2
Prolonged hypotension	BP <90 mmHg	3
No distal ischemia	Distal pulses, no signs of ischemia	1
Mild ischemia	Absent or diminished pulses, no signs of ischemia	2 ^a
Moderate ischemia	No signals by continuous-wave Doppler, signs of distal ischemia	3 ^a
Severe ischemia	No pulse; cool, paralyzed limb; no capillary refill	4 ^a
<30 years old patient		1
>30 years old patient		2
>50 years old patient		3

^a Points are doubled if ischemia lasts longer than 6 h.

Operation

Preoperative Preparation

Hemodynamically stable patients are placed on their back with the arm abducted 90° on an arm surgery table. The forearm and hand should be in supination. Peripheral or central IV lines should not be inserted on the injured side. Any continuing bleeding is controlled manually directly over the wound. If the site of injury is the brachial artery or distal to it, a tourniquet can be used to achieve proximal control. It is then placed before draping and should be padded to avoid direct skin contact with the cuff. This minimizes the risk for skin problems during inflation. The arm is washed so the skin over the appropriate artery can be incised without difficulty. The draping should allow palpation of the radial pulse and inspection of finger pulp perfusion. One leg is also prepared in case vein harvest is needed.

The position of the arm is the same for more proximal injuries. Proximal control of high brachial and axillary artery trauma may involve exposure and skin incisions in the vicinity of the clavicle and the neck, so for proximal injuries the draping must also allow incisions at this level.

Proximal Control

For distal vessel injury, proximal control can be achieved by inflating the previously placed tourniquet to a pressure around 50 mmHg above systolic pressure. The cuff should be inflated with the arm elevated to minimize bleeding by venous congestion. After inflation, the wound is explored directly at the site of injury.

For more proximal injuries, control is achieved by exposing a normal vessel segment above the wounded area. The most common sites for proximal control in the arm are the axillary artery below the clavicle, and the brachial artery (which is what the artery is called distal to the teres major muscle) somewhere in the upper arm.

Exposure for Proximal Control of Arteries in the Arm

Axillary Artery Below the Clavicle

An 8-cm horizontal incision is made 3 cm below the clavicle (Fig. 2). The pectoralis major muscle fibers are split parallel to the skin incision. The pectoralis minor muscle is divided close to its insertion. The nerve crossing the pectoralis minor muscle can also be divided without subsequent morbidity. The axillary artery lies immediately below the fascia together with the vein inferiorly, and the lateral cord of the brachial plexus is located above the artery.

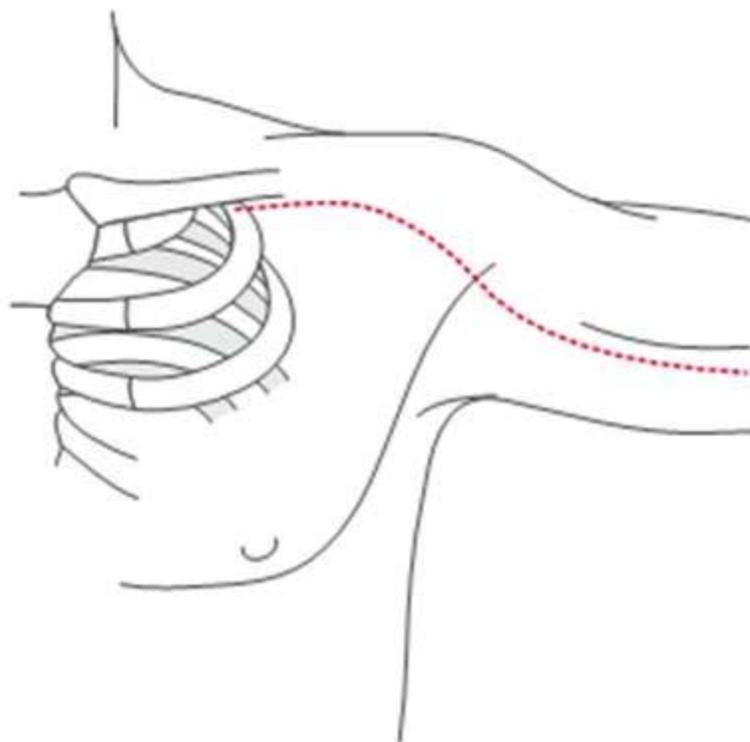


Fig. 2

Brachial Artery in the Upper Arm

The incision is made along the posterior border of the biceps muscle; a length of 6–8 cm is usually enough (Fig. 3.). The muscles are retracted medially and laterally, and the artery lies in the neurovascular bundle immediately below the muscles. The sheath is incised and the artery freed from the median nerve and the medial cutaneous nerve that surrounds it.

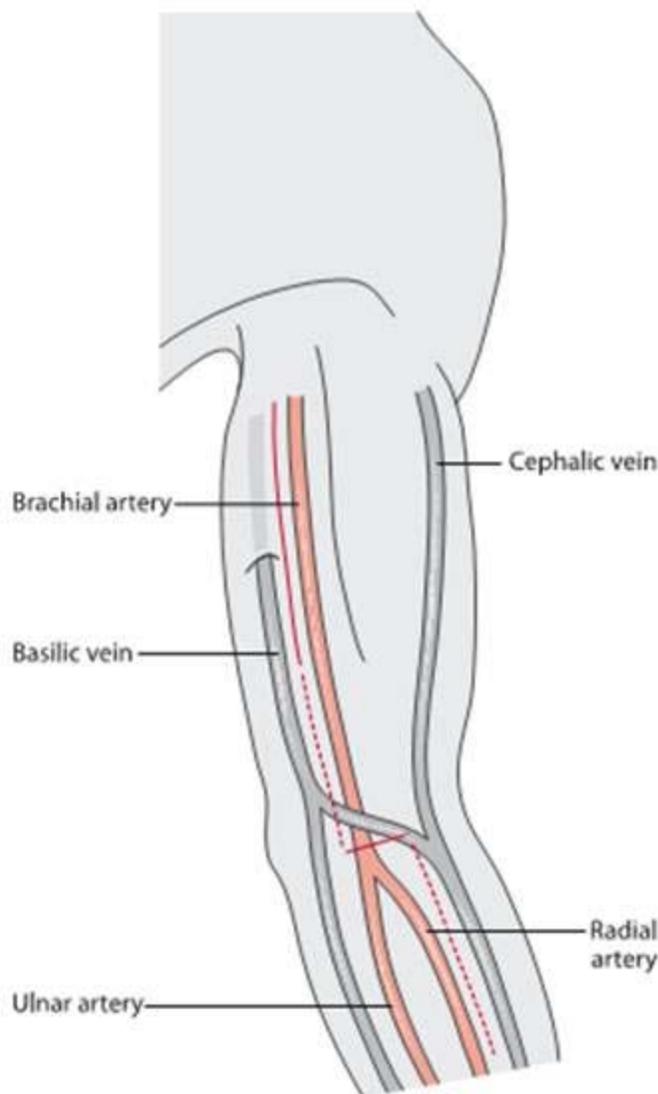


Fig. 3. Transverse incision in the elbow for exposing the brachial artery and with possible elongations (dottedlines) when access to the ulnar and radial ranches as well as to more proximal parts of the brachial artery is needed

Brachial Artery at the Elbow

The incision is placed 2 cm below the elbow crease and should continue up on the medial side along the artery. If possible, veins transversing the wound should be preserved, but they can be divided if necessary for exposure. The medial insertion of the biceps tendon is divided entirely, and the artery lies immediately beneath it. By following the wound proximally, more of the artery can be exposed (Fig. 3.). If the origins of the radial and ulnar artery need to be assessed, the wound can be elongated distally on the ulnar side of the volar aspect of the arm. The median nerve lies close to the brachial artery, and it is important to avoid injuring it.

Exploration and Repair

Distal control is achieved by exploring the wound. Sometimes this requires additional skin incisions. The most common site for vascular damage in the arm is the brachial artery at the elbow level. These injuries occurs, for example, because of supracondylar fractures in children and adults. In such cases, exposure and repair of the brachial artery through an incision in the elbow crease is appropriate. Hematomas should be evacuated to allow inspection of nerves and tendons.

For supracondylar fractures, the brachial artery, the median nerve, and the musculocutaneous nerves must sometimes be pulled out of the fracture site. Before the artery is clamped, the patient is given 50 units of heparin/kg body weight IV. Repair should also be preceded by testing inflow and backflow from the distal vascular bed by temporary tourniquet or clamp release. It is often also wise to pass a #2 Fogarty catheter distally to ensure that no clots have formed. Occasionally, inflow is questionable, and proximal obstruction must be ruled out. As a general principle, all vascular injuries in the arms should be repaired, except when revascularization may jeopardize the patient's life. Arterial ligation should be performed only when amputation is planned. Postoperative arm amputation rates are reported to be 43% if the axillary artery is ligated and 30% at the brachial artery level. Another exception is forearm injuries. When perfusion to the hand is rendered adequate – as assessed by pulse palpation and the Allen test – one of these two arteries can be ligated without morbidity. In a substantial number of patients with differing vessel anatomy, however, ligation of either the ulnar or radial artery may lead to hand amputation. If both arteries are damaged, the ulnar artery should be prioritized because it is usually responsible for the main part of the perfusion to the hand.

For most arterial injuries, vein interposition is necessary for repair. Veins are harvested from the same arm, from parts of the cephalic or basilic vein if the trauma is limited, or from the leg. The saphenous vein in the thigh is suitable for axillary and brachial artery repair, while distal ankle vein pieces can be used for interposition grafts to the radial and ulnar arteries. Before suturing the anastomoses, all damaged parts of the artery must be excised to reduce the risk of postoperative thrombosis. Rarely, primary suture with and without patching can be used to repair minor lacerations. Shunting of an arterial injury to permit osteosynthesis is rarely needed in the arm. Vascular interposition grafting can usually be done with an appropriate graft length before final orthopedic repair. Also, extremity shortening due to fractures is less of a problem in the arms (in contrast to the legs), and orthopedic treatment without osteosynthesis

is common especially in older patients. Nevertheless, for some arm injuries shunting is a practical technique that allows time for fracture fixation, thus avoiding the risks of redisplacement and repeated vessel injury. One example is injuries to the axillary or brachial artery caused by a proximal humeral fracture, where the fragment needs to be fixed in order to prevent such injuries. Another example is humeral shaft fracture, which needs to be rigidly fixed to abolish the instability that may otherwise endanger the vascular graft. Veins should also be repaired if reasonably simple. If the vein injury is caused by a single wound with limited tissue damage, concomitant veins to the distal brachial artery can be ligated. For more extensive injuries where the superficial large veins are likely to be ruined, it is wise to try to repair the deep veins. For very proximal injuries in the shoulder region, vein repair is important to avoid longterm problems with arm swelling. It is also important to cover the mended vessel segment with soft tissue to minimize the risk for infection that may involve the arteries.

Finishing the Operation

When the repaired artery or graft's function is doubtful and when the surgeon suspects distal clotting, intraoperative arteriography should be performed. After completion, all devitalized tissue should be excised and the wound cleaned. For penetrating wounds, damaged tendons and transected nerves should also be sutured. This is not worthwhile for most blunt injuries. Fasciotomy should also be considered before finishing the operation. As in the leg, long ischemia times and successful repair increase the risk of reperfusion and compartment syndrome, but the overall risk for compartment syndrome is reported to be less in the arm than in the leg. For a description of arm fasciotomy techniques, we recommend consulting orthopedic textbooks. After the wounds are dressed, a fractured arm is put into a plaster splint for stabilization.

Endovascular Treatment

In contrast to proximal arm vessel trauma, there are few instances in distal injuries when endovascular treatment is a feasible treatment option. Because the brachial artery and the forearm vessels are easy to expose with little morbidity, open repair during exploration of the wound is usually the best option. Possible exceptions to this are treatment of the late consequences of vascular trauma, such as arteriovenous fistulas and pseudoaneurysms. Especially in the shoulder region, including the axilla, primary endovascular treatment is often

the best treatment option. Another circumstance when endovascular treatment is favorable is bleeding from axillary artery branches – such as the circumflex humeral artery – due to penetrating trauma. Active bleeding from branches, but not from the main trunk, observed during arteriography is preferably treated by coiling. The bleeding branches are then selectively cannulated with a guidewire and coiled, using spring coils or injections of thrombin to occlude the bleeding artery.

Management After Treatment

Postoperative monitoring of hand perfusion and radial pulse is recommended at least every 30 min for the first 6 h. When deteriorated function of the repaired artery is suspected, duplex scanning can verify or exclude postoperative problems. Apparent occlusions should be treated by reoperation as soon as possible. Compartment syndrome in the lower arm may also evolve over time, and swelling, muscle tenderness, and rigidity must also be monitored during the initial days. For most patients, treatment with low molecular weight heparin is continued postoperatively. A common dose is 5,000 units subcutaneously twice daily.

Keeping the hand elevated as much as possible may reduce swelling of the hand and arm as well as problems with hematoma formation around the wound. Early mobilization of the fingers facilitate blood flow to the arm and should be encouraged.

VASCULAR INJURIES IN THE LEG

Vascular trauma to extremity vessels is caused by violent behavior or accidents. Because of the rise in the number of endovascular procedures, iatrogenic injuries have also become an increasing part of vascular trauma. Vascular injuries may cause life-threatening major bleeding, but distal ischemia is more common. Ischemia occurs after both blunt and penetrating trauma. The vascular injury is often one of many injuries in multiply traumatized patients that make the recognition of signs of vascular injury – which can be blurred by more apparent problems – and the diagnosis difficult. Table 9.1 lists common locations of combined orthopedic and vascular injury. Multiple injuries also bring problems regarding priority.

Table 4. Most common locations for combined orthopedic and vascular injury

Orthopedic injury	Vascular injury
Femoral shaft fracture	Superficial femoral artery
Knee dislocation	Popliteal artery
Fractured clavicle	Subclavian artery
Shoulder dislocation	Axillary artery
Supracondylar fracture of the humerus	Brachial artery
Elbow dislocation	

Data on the true incidence of vascular injuries to the legs is hard to gather. The incidence of vascular trauma varies among countries and also between rural and urban areas. It is usually higher where gunshot wounds are common. There is an equal share of blunt and penetrating injury in most studies from Europe, whereas penetrating injury is slightly more common in the United States. Approximately 75% of all vascular injuries are localized to the extremities and more than 50% to the legs. The true incidence of iatrogenic trauma is unknown.

Etiology and Pathophysiology

Penetrating Injury

Penetrating vascular injury is caused by stab and cutting injuries, gunshots, and fractures, the latter when sharp bone fragments penetrate the vascular wall. Gunshots cause major bleeding by direct artery trauma, while high-velocity bullets create a cavitation effect with massive soft tissue destruction and secondary arterial damage. In fact, after all types of penetrating trauma both bleeding and indirect blunt arterial injury with ischemia may occur. Bleeding is more often exsanguinating after sharp injury and partial vessel transection. Complete avulsion, especially when caused by blunt trauma, makes the vessel more prone to retraction, spasm, and thrombosis. This diminishes the risk for major bleeding. Iatrogenic injuries can be caused by catheterization and during surgical dissection.

Blunt Injury

Blunt vascular injuries are usually caused by motor vehicle and other accidents. The consequences are thrombosis and ischemia distal to the injured vessel. The media and the intimal layers of the vessel wall are easily separated, and subsequent dissection by the bloodstream between the layers may lead to lumen obstruction. Blunt injuries also induce thrombosis. This type of vessel injury is particularly common when the artery is hyperextended as in knee joint luxations and upper arm fractures. Contusion of the vessel may also cause bleeding in the vessel wall. Thrombosis and ischemia by this mechanism can occur several hours after the traumatic situation. Narrowing of the arterial lumen following blunt trauma is rarely caused by spasm and it can be disregarded as etiology.

Pathophysiology

The main pathophysiological issue after vascular injuries to the extremities is ischemia. The process is identical to what happens during acute leg ischemia due to embolization. Irreversible damage to the distal parts of the legs is not infrequent and the diagnosis is more difficult to determine than for other types of leg ischemia. The reason is the multiple manifestations of the trauma. It must be kept in mind that the time limit for acute leg ischemia – 4–6 h before permanent changes occur – is also valid for trauma.

A vascular injury missed during the initial examination may develop into a pseudoaneurysm or an arteriovenous fistula. A pseudoaneurysm is a hematoma with persistent blood flow within it that may enlarge over time and cause local symptoms and sometimes even rupture. When both an artery and an adjacent vein are injured simultaneously an arteriovenous fistula may develop. These can become quite large with time and even cause cardiac failure due to increased cardiac output.

Medical History

Most patients with major vascular injury present with any or several of the “hard signs” of vessel injury (Table 5) and the diagnosis is obvious. Penetrating injury patients who arrive in the emergency department without active hemorrhage are usually not in shock because the bleeding was controlled at the trauma scene. Shock in patients with penetrating injury usually means that the bleeding is ongoing. Still, information about the trauma mechanism is often needed to estimate the likelihood for vessel injury and to facilitate the management process.

Besides interviewing the patient, additional background information may be available from medical personnel and accompanying persons. The few minutes required to establish a picture of the trauma situation are usually worthwhile. For example, a history of a large amount of bright red pulsating bleeding after penetrating trauma suggests a severe arterial injury. Venous bleedings are often described as a steady flow of dark red blood. In high-impact accidents the risk for a severe vascular injury is increased.

Besides being helpful when assessing the risk for a major injury estimation of the blood loss is also important for later volume replacement. Knowledge of the exact time when the injury happened is helpful for determining the available time before irreversible damage occurs from ischemia. The duration of ischemia also influences the management priority in multitrauma patients, and the time elapsed affects the presentation of the ischemic symptoms. For example, an initial severe pain may vanish with time as a consequence of ischemic nerve damage. Even a major internal hemorrhage may be present without being clinically obvious after a very recent injury. Information about complicating diseases and medication is also helpful. For instance, betablockers may abolish the tachycardia in hypovolemia.

Table 5. Signs of vascular injury

Hard signs	Soft signs
Active hemorrhage	History of significant bleeding
Hematoma (large, pulsating, expanding)	Small hematoma
Distal ischemia ("six Ps")	Adjacent nerve injury
Bruit	Proximity of wound to vessel location
	Unexplained shock

Clinical Signs and Symptoms

The physical examination is performed after the primary and secondary surveys of a multitrauma patient and should focus on identifying major vessel injury. The examination

should be thorough, especially regarding signs of distal ischemia. It should include examination and auscultation of the injured area, palpation of pulses in both legs, and assessment of skin temperature, motor function, and sensibility. The presence of one or more of the classic hard signs of vascular injury listed in Table 5 suggests that a major vessel is damaged and that immediate repair is warranted. Findings of “soft” signs should bring the examiner’s attention to the fact that a major vessel may be injured but that the definite diagnosis requires additional work-up. As noted in Table 5, the hard sign of distal ischemia as suggested by the “six Ps” suggests vascular injury.

The principles of the vascular examination suggested for acute leg ischemia are also valid for vascular injuries, but certain details need to be emphasized. Vascular trauma in the legs usually strikes young persons, so it should be assumed that the patient had a normal vascular status before the injury. A palpable pulse does not exclude vascular injury; 25% of patients with arterial injuries that require surgical treatment have a palpable pulse initially. This is due to propagation of the pulse wave through soft thrombus. Pulses may be palpated initially in spite of an intimal flap or minor vessel wall narrowing and can later cause thrombosis and occlude the vessel. Ankle pressure measurements and calculation of the ankle brachial index (ABI) should therefore supplement palpation of pulses. If the ABI is ≤ 0.9 , arterial injuries should be suspected.

Findings in the physical examination of a patient in shock are particularly difficult to interpret. In several aspects findings of distal ischemia caused by vascular injury are similar to vasoconstriction of the skin vessels in the foot. Differences in pallor, the presence of pulses, and skin temperature between the injured and uninjured leg therefore should be interpreted as the possible presence of vascular injury. Ankle pressure measurements are also valuable during such circumstances.

It is important to remember to listen for bruits and thrills over the wounded area to reveal a possible arteriovenous fistula.

Diagnostics

Recommendations for management of suspected vascular injuries in the leg have evolved from mandatory exploration of all suspected injuries (a common practice during past wars), to routine angiography for most patients, to a more selective approach today. Regarding exploration and subsequent angiography, it was found that negative explorations and arteriograms were obtained in over 80% of the patients. The associated risk for complications

and morbidity after these invasive procedures is the rationale for a more selective approach. Rapid transportation, clinical examination, ankle pressure measurements, careful monitoring, and duplex examination leave angiography for some of the patients and urgent exploration for a few.

Angiography

Angiography is unnecessary when a vascular injury is obvious after the examination. The two most common indications for excluding vascular injury are (1) when there are no hard signs at the examination, and (2) when clinical findings are imprecise but the ABI is <0.9 .

Angiography is more often indicated after blunt trauma than penetrating. The reason for this is the more difficult clinical examination because of the more extensive soft tissue and nerve damage after blunt trauma. It may occasionally be helpful to perform angiography even when injury is evident in order to exactly locate the injured vessel. An option is to perform it intraoperatively. Contralateral puncture is important when the injury is close to the groin.

The purpose of the arteriography is to identify and locate lesions such as occlusions, narrowing, and intimal flaps. Contrast leakage outside the vessel can be visualized, and it also serves to provide a road map before surgery. It has, however, been argued that it is unnecessary to search for minimal lesions; some studies have shown that it is safe and effective to manage such lesions nonoperatively. On the other hand, angiography may be the first step in the final treatment of such small lesions by stenting. When the injury is caused by a shotgun blast, angiography should always be performed because multiple vascular injuries are common. It is then indicated regardless of the clinical signs and symptoms. The risk for complications after angiography is very low, but the risk of complications is higher when the punctured artery is small. Children therefore have a rather high rate of complications. A contributing factor is that their very vasoactive arteries are prone to temporary spasm. Overall, as described above, the risk for complications after angiography does not warrant avoiding it when indicated. Occasionally it is worthwhile to order venography. It may be indicated in patients not subjected to exploration because arterial injury was ruled out but in whom a major venous injury is suspected. As an example, 5–10% of all popliteal venous injuries are reported to occur without arterial damage.

Duplex Ultrasound

Despite the usefulness of duplex scanning in general for vascular diagnosis, it has not been universally accepted for diagnosis of vascular trauma despite the fact that it is noninvasive. It is operator dependent and vessels may be difficult to assess in multiply injured patients, legs with skeletal deformities, large hematomas, and through splints and dressings. In some hospitals with expertise in duplex assessment and round-the-clock access to skilled examiners, duplex has replaced angiography to a large extent. The indications proposed are then the same as for angiography. Duplex is also the method of choice for diagnosis of most of the late consequences of vascular injuries to the legs – arteriovenous fistulas, pseudoaneurysms, and hematomas.

Management Before Treatment

Severe Vessel Injury

Major external bleeding not adequately stopped when the patient arrives to the emergency department should immediately be controlled with digital pressure or bandages. No other measures to control bleeding are taken in the emergency department and attempts to clamp vessels are saved for the operating room.

The patient is surveyed according to the trauma principles used in the hospital. For most patients without obvious vascular injuries to the leg vessels, more careful vascular assessment takes place after the secondary survey. If the vascular injury is one of many in a multitrauma patient, general trauma principles for trauma care are applied. Treatment of the vascular problem is then initiated as soon as possible when the patient's condition allows it.

Patients with hard signs of vascular injury but without other problems should be transferred immediately to the operating room. Before transfer the following can be done:

1. Give the patient oxygen.
2. Initiate monitoring of vital signs (heart rate, blood pressure, respirations, SpO₂).
3. Place at least one large-bore intravenous (IV) line.
4. Start infusion of fluids. Dextran preceded by 20ml Promiten is advised especially if

the patient has distal ischemia.

5. Draw blood for hemoglobin and hematocrit, prothrombin time, partial thromboplastin time, complete blood count, creatinine, sodium, and potassium as well as a sample for blood type and cross-match.

6. Obtain informed consent.

7. Consider administering antibiotics and tetanus prophylaxis.

8. Consider administering analgesics (5–10 mg opiate IV).

Less Severe Injuries

ABI must be measured when vascular injury is suspected. Patients with soft signs of vascular injury and an ABI <0.9 usually need arteriography to rule out or verify vascular damage. This is performed as soon as possible. Before the patient is sent to the angiosuite other injuries need to be taken into account and the priority of management discussed. Ischemic legs should be given higher priority than, for example, skeletal and soft tissue injury, and temporary restoration of blood flow can be achieved by shunting.

Patients with an ABI >0.9 and a normal physical examination (little suspicion of vascular injury) can be monitored in the ward. Repeated examinations of the patient's clinical status are important and hourly assessment of pulses and ABI the first 4–6 h are warranted. If the ABI deteriorates to a value <0.9 or if pulses disappear, angiography should be carried out.

Angiography Findings

Operative treatment and restoration of blood flow are done as soon as possible if the angiography shows arterial occlusion in the femoral, popliteal, or at least two calf arteries in proximity to the traumatized area. It should be kept in mind that occlusion of the popliteal artery is detrimental for distal perfusion and is associated with a high risk for amputation due to a long ischemia time. Patients with popliteal occlusion should therefore be taken immediately to the operating room. Debate is ongoing whether one patent calf artery in an

injured leg is sufficient to allow nonoperative treatment. Some reports have found that as long as one of the tibial vessels is intact, there is no difference in limb loss or foot problems during follow-up between operative and nonoperative treatment. Our recommendation, however, is to try to restore perfusion if more than one of the calf arteries is obstructed. If combined with ischemic symptoms or signs of embolization, angiography findings of intimal flaps, minor narrowing of an artery, or minor pseudoaneurysm (<5 mm in diameter) should also be treated. Endovascular stenting is then a good alternative to operative treatment. Expectancy could be favorable for asymptomatic patients with normal ABI. Such occult arterial injuries appear to have an uneventful course and late occlusion is extremely rare. The occasional pseudoaneurysm that will enlarge with time appears to benefit from later repair.

Primary Amputation

In most circumstances, but not always, it is reasonable to repair injured vessels. For a few patients, however, primary amputation is a better option. This is often a difficult decision. Primary amputation is favorable for the patient if the leg is massacred or if the duration of ischemia is very long (>12 h) and appears to be irreversible in the clinical examination. Primary amputation may also be considered for certain patients: multitrauma patients, patients with severe comorbid disease, and those in whom the leg was already paralyzed at the time of injury. Extensive nerve damage, lack of soft tissue to cover the wound, and duration of ischemia >6 h support primary amputation for these patient groups.

There are scoring systems, such as the Mangled Extremity Severity Score (MESS), to aid in making the decision to amputate a leg or an arm. For example, a patient over 50 years old with persistent hypotension and a cool paralyzed distal leg after high-energy trauma should have the leg amputated according to MESS. It must be stressed, however, that repair of both venous and arterial injuries is superior for most patients.

Operation

Surgical treatment of vascular injuries in the leg usually proceeds in a particular order common for most patients. First the patient is scrubbed, anesthetized, and prepared for surgery. The next step is to achieve proximal control. Occasionally, control of the bleeding by manual compression with a gloved hand needs to be maintained throughout these first two steps. Proximal control is followed by measures to achieve distal control, often accomplished during

exploration of the wound. Finally, the vessels are repaired and the wound covered with soft tissue. When the patient has other injuries that motivate urgent treatment, or has fractures in the leg that need to be surgically repaired, this last step can be delayed while perfusion to the distal leg is maintained by a shunt temporarily bypassing the injured area.

Preoperative Preparation

The patient is placed on a surgical table that allows x-ray penetration. If not administered previously, infection prophylaxis treatment is started. The entire injured extremity is scrubbed with the foot draped in a transparent plastic bag. A very good marginal of the sterile field is essential because incisions need to be placed much more proximal than the wound to achieve proximal control. The contralateral leg should also be scrubbed and draped to allow harvest of veins for grafts. The venous system in the injured leg should be kept intact if possible. If a patient is in shock and the bleeding is difficult to control, it is recommended to delay inducing the anesthesia until just before the operation begins in order to avoid increased bleeding and an accentuated drop in systemic blood pressure due to loss of adrenergic activity.

Proximal Control

In patients with injuries proximal to the femoral vessels, control is achieved through an incision in the abdominal fossa. The common or external iliac artery can then be exposed retroperitoneally and secured. Proximal control for injuries in the thigh, proximal to the popliteal fossa, is usually obtained by exposing the common femoral artery and its branches in the groin. Popliteal vessel trauma can be controlled by exposing the distal superficial femoral artery or the proximal popliteal artery through a medial incision above the knee. Inflow control for calf vessel injuries is reached by exposing the popliteal artery below the knee.

If there is no ongoing bleeding when the artery is exposed a vessel-loop is applied. Clamping should be postponed until later. If bleeding is brisk and continuous, however, the clamp is placed right away. Clamping should be attempted even if the bleeding appears to be mainly venous in origin. Arterial clamping often diminishes such bleedings substantially.

An alternative way to achieve proximal control of distal femoral, popliteal, and calf vessels is to use a cuff. A padded cuff, the width in accordance with the leg circumference, is then wrapped around the leg well above the wounded area before scrubbing and draping. In the thigh a 20-cm wide cuff is often suitable. It is important to have at least 10 cm from the lower

edge of the cuff to the wound to allow prolongation of incisions if necessary. The cuff is inflated if bleeding starts during exploration. For distal injuries this often works well and may spare the patient one surgical wound.

Exposure of Different Vessel Segments in the Leg

Common or External Iliac Arteries, Fig. 4 A, B.

a A skin incision 5 cm above and parallel to the inguinal ligament is used. This incision allows exposure of all vascular segments from the external iliac up to the aortic bifurcation.



Fig. 4 A. Exposure of common or external iliac arteries.

b The muscles are split in the direction of the fibers. Dissection is totally retroperitoneal, with attention to the ureter crossing the vessels in this region. Be careful with the iliac vein, which is separated from the artery by only a thin tissue layer. Exposure of the proximal common iliac artery and the aortic bifurcation is facilitated by a table-fixed self-retaining retractor (i.e., Martin arm).

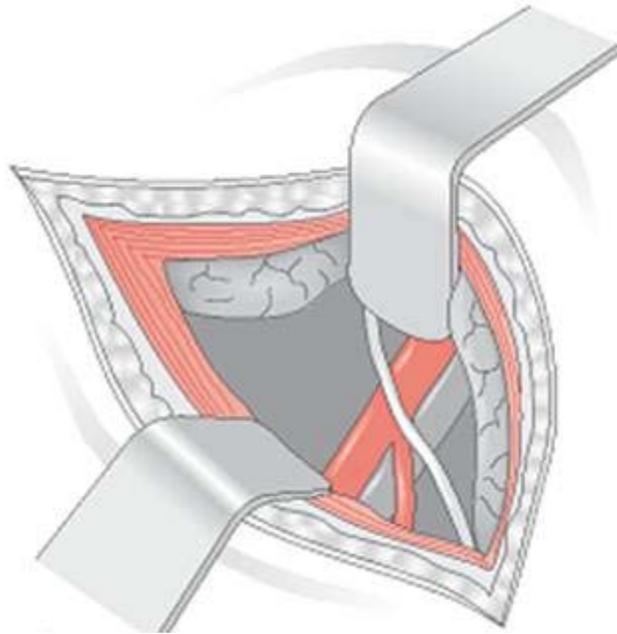


Fig. 4 B. Exposure of common or external iliac arteries.

Femoral Artery in the Groin, Fig. 5 A, B, C.

a A longitudinal skin incision starting 1–2 cm cranial to the inguinal skin fold and continued lateral to the artery is used to avoid the inguinal lymph nodes. A common mistake is to place the incision too far caudally, which usually means the dissection is taking place distal to the deep femoral.



Fig 5A. Exposure of femoral artery in the groin

b The dissection is continued sharply with the knife straight down to the fascia lateral to the lymph nodes and is then angulated 90° medially to reach the area over the artery. It should then be palpable. Lymph nodes should be avoided to minimize the risk for infection and development of seroma. The fascia is incised, and the anterior and lateral surfaces of the artery are approached.

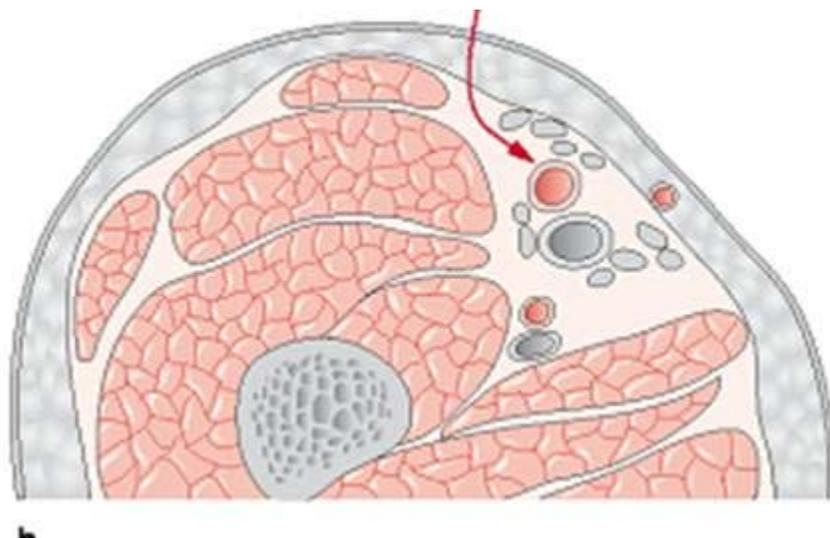


Fig. 5. Exposure of femoral artery in the groin

c At this stage the anatomy is often unclear regarding the relation of branches to the common femoral artery. Continue dissection until the bifurcation into superficial and deep femoral artery is identified. Its location varies from high up under the inguinal ligament up to 10 cm further down. At this stage, the surgeon must decide whether exposure and clamping of the common femoral are enough. This is usually the case for proximal control in trauma distally in the leg. In acute ischemia it is more common that the entire bifurcation needs to be exposed.

During the continued dissection, attention must be given to important branches that should be controlled and protected from iatrogenic injuries. These are, in particular, the circumflex iliac artery on the dorsal aspect of the common femoral artery and the deep femoral vein crossing over the anterior aspect of the deep femoral artery just after its bifurcation. To provide a safe and good exposure of the deep femoral to a level below its first bifurcation, this vein must be divided and suture-ligated. Partial division of the inguinal ligament is occasionally needed for satisfactory exposure.

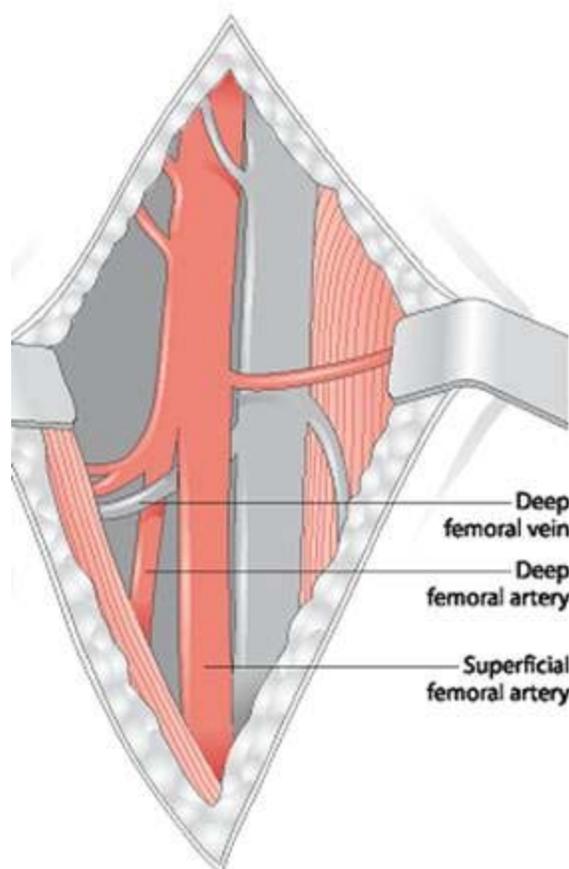


Fig. 5 C. Exposure of femoral artery in the groin

Superficial Femoral Artery, Fig. 6.

A skin incision is made along the dorsal aspect of the sartorius muscle at a midhigh level. It is important to avoid injuries to the greater saphenous vein, which usually is located in the posterior flap of the incision. The incision can be elongated as needed. After the deep fascia is opened and the sartorius muscle is retracted anteriorly, the femoral artery is found and can be mobilized. Division of the adductor tendon is sometimes required for exposure.

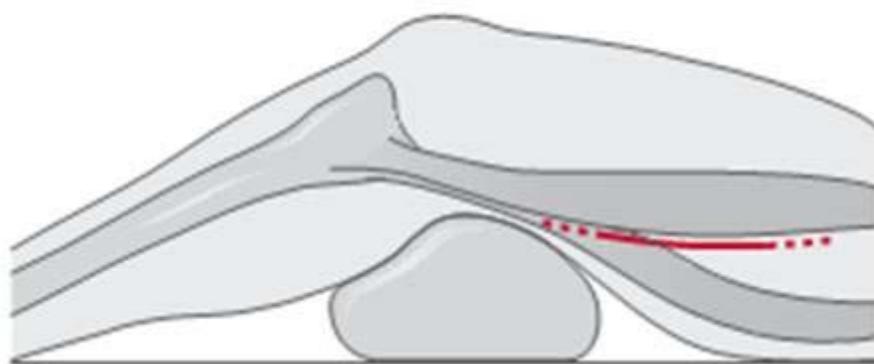


Fig. 6. Incision for exposure of the superficial femoral artery

Popliteal Artery Above the Knee, Fig. 7 A, B.

a The knee is supported on a sterile, draped pillow. The skin incision is started at the medial aspect of the femoral condyle and follows the anterior border of the sartorius muscle 10–15 cm in a proximal direction. Protect the greater saphenous vein and the saphenous nerve during dissection down to the fascia. After dividing the fascia longitudinally, continue the dissection in the groove between the sartorius and gracilis muscles, which leads to the fat in the popliteal fossa.

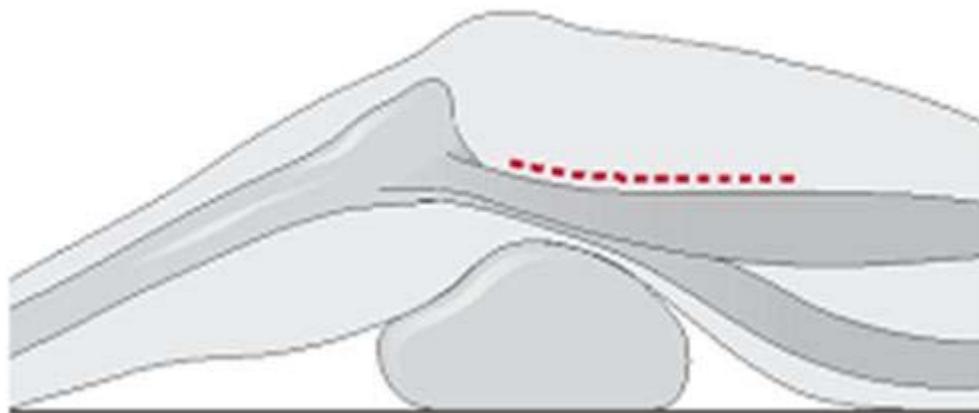


Fig. 7 . Exposure of popliteal artery above the knee

b The popliteal artery and adjacent veins and nerve are then, without further division of muscles, easily found and separated in the anterior aspect of the fossa.

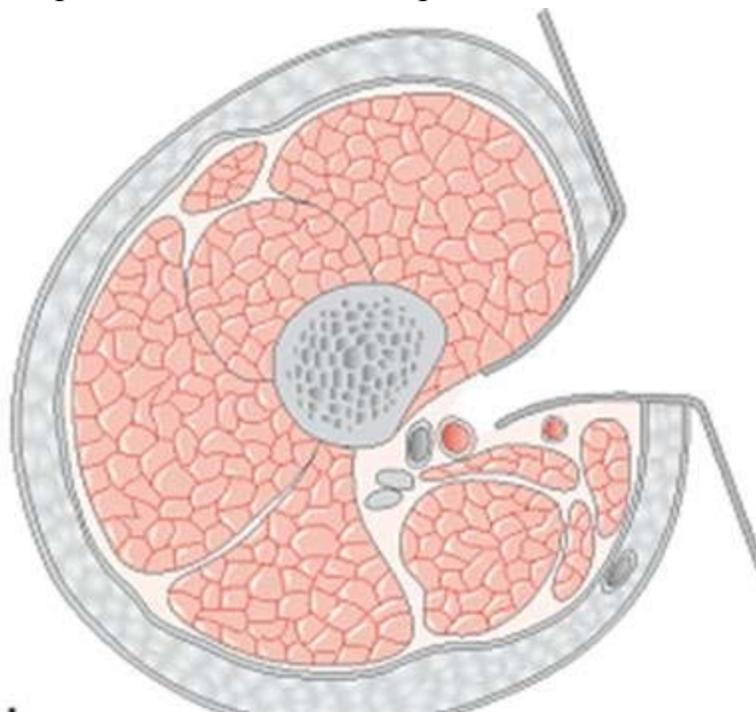


Fig. 7 B. Exposure of popliteal artery above the knee

Popliteal Artery Below the Knee, Fig. 8 A, B.

a A sterile pillow or pad is placed under the distal femur. The incision is placed 1 or 2 cm posterior to the medial border of the tibia, starting at the tibial tuberosity and extending

10–12 cm distally. Subcutaneous fat and fascia are sharply divided, with caution to the greater saphenous vein.

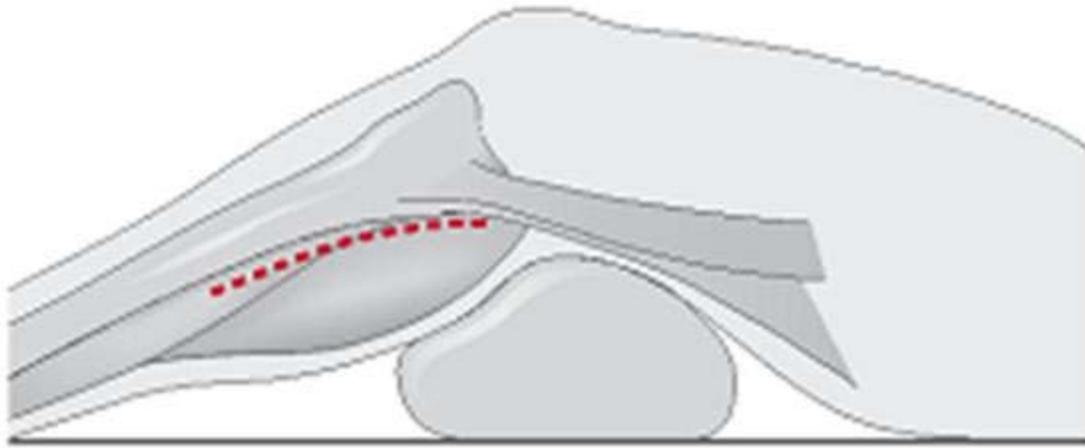


Fig. 8. A. Exposure of popliteal artery below the knee

b The popliteal fossa is reached by retracting the gastrocnemius muscle dorsally. The deep fascia is divided and the artery usually easier to identify. Occasionally, pes anserinus must be divided for adequate exposure. The popliteal artery is often located just anterior to the nerve and in close contact with the popliteal vein and crossing branches from concomitant veins. If it is necessary to expose the more distal parts of the popliteal artery, the soleus muscle has to be divided and partly separated from the posterior border of the tibia.

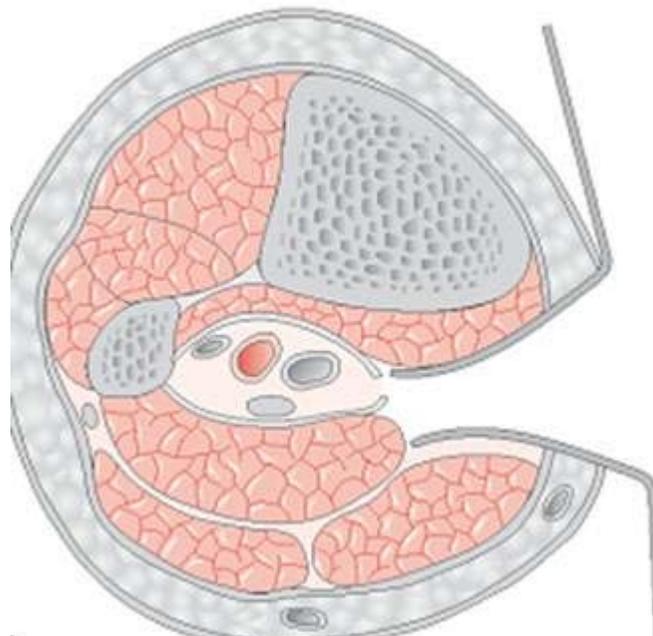


Fig. 8. B. Exposure of popliteal artery below the knee

Distal Control and Exploration

Distal control is achieved by distal elongation of the incision used to explore the site of injury. Through this incision careful dissection in intact tissue distal to the injured area usually reveals the injured artery. When it is identified and found to be not completely transected, a vessel-loop is positioned around it. If the artery is cut, a vascular clamp is applied on the stump. It is also possible to gain distal control during the exploration of the injured area, but dissection may be tricky because of hematoma, edema, and distorted anatomy. Usually the backbleeding from the distal artery is minimal and does not disturb visualization during dissection. Simultaneous venous bleeding that emerges from major veins must also be controlled. This can be done by balloon occlusion or clamps. The latter should be used with caution and closed as little as possible.

When control is obtained the wound is explored and the site of vessel injury identified. The best way is to follow the artery proximally from where the artery was exposed for distal control. Of course, any foreign materials encountered need to be removed. The injured artery should be explored in both directions until a normal arterial wall is reached. Several centimeters of free vessels are needed. Side branches are controlled using a double loop of suture with a hanging mosquito or by small vascular clamps. Thrombosed arteries usually have a hematoma in the vessel wall giving it a dark blue color. Such parts need to be cut out and the vessel edges trimmed before shunting or repair. For penetrating injuries all parts of the vascular wall that are lacerated must be excised to ensure that the intima will be enclosed in the suture line during repair. After this procedure the vessel can be shunted or repaired.

Finally, other parts of the wound are explored and all devitalized skin and muscle tissue is excised. Injured small adjacent veins should be ligated and all larger veins, such as the femoral veins and the popliteal vein, must be repaired.

Shunting

Insertion of a temporary shunt to restore distal perfusion is sensible if the vascular injury appears to require more extensive repair than a simple suture or an end-to-end anastomosis.

Shunting provides the time needed to either perform a vascular reconstruction, including harvesting of a vein graft from the uninjured leg, or to wait for help. Shunting can also be valuable when patients have other injuries that need attention or when leg fractures must be repositioned to give the appropriate vascular graft length. In patients with fractures shunting allows both repositioning and fixation without increasing the risk of ischemic damage.

A novel vascular reconstruction seldom tolerates the forces required for reposition of fractures. When perfusion is restored by a shunt the surgeon has plenty of time to carefully explore the wound and other injuries. When a shunt is used for distal perfusion while other procedures are performed, it is important to check its function at least every 30min.

The principles of vascular shunting are simple. Specially designed shunts can be used if available; examples are Pruitt–Inhara and Javid. Most have inflatable balloons in both ends for occlusion and side channels with stopcocks through which the function of the shunt can be tested (Figs. 9, 10). The side holes also enable infusion of a heparinized solution and contrast for fluoroscopy. The extra channels can be used to draw blood during the operation. It is not, however, necessary to use manufactured shunts. Any kind of sterile plastic or rubber tubing is sufficient. It is then important to use dimensions of the tube in accordance with the artery's inner diameter. The tube is cut into suitable lengths and the edges carefully trimmed with scalpel and scissors to avoid damage when inserted. The tube is positioned and secured with vessel loops that abolish the space between the artery and tube to manage bleeding. A loosely applied suture around the middle part of the shunt can be used to secure it. It is advantageous to simultaneously shunt concomitant vein injuries – at least the femoral and popliteal veins – to avoid swelling and to facilitate distal flow.

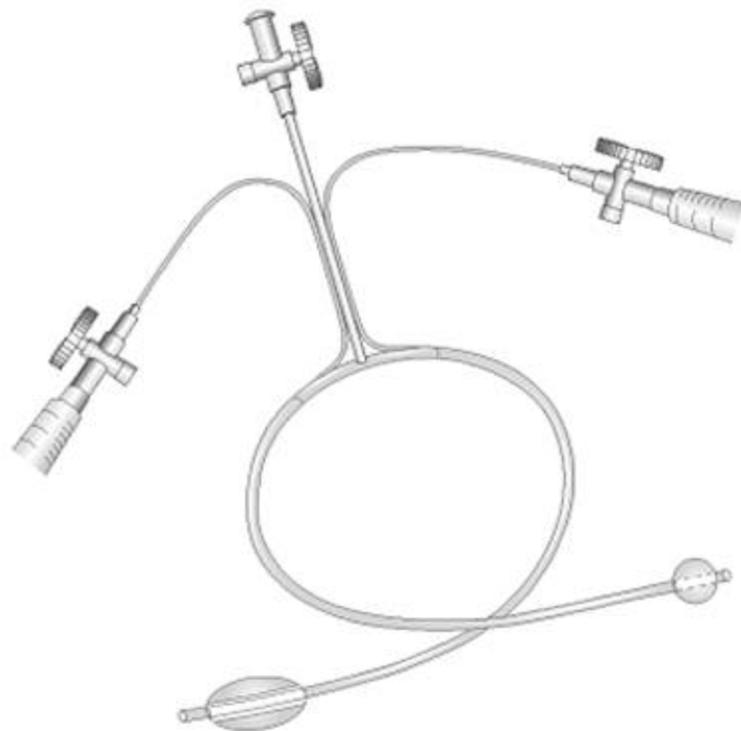


Fig. 9. A special catheter for shunting (Pruitt–Inahara shunt).

The shunt has a larger balloon in one end aimed for the proximal inflow vessel and a smaller one for placement in the outflow vessel. The occluding balloons are controlled by injecting saline through separate channels with stopcocks. The shunt can also be flushed through a third channel

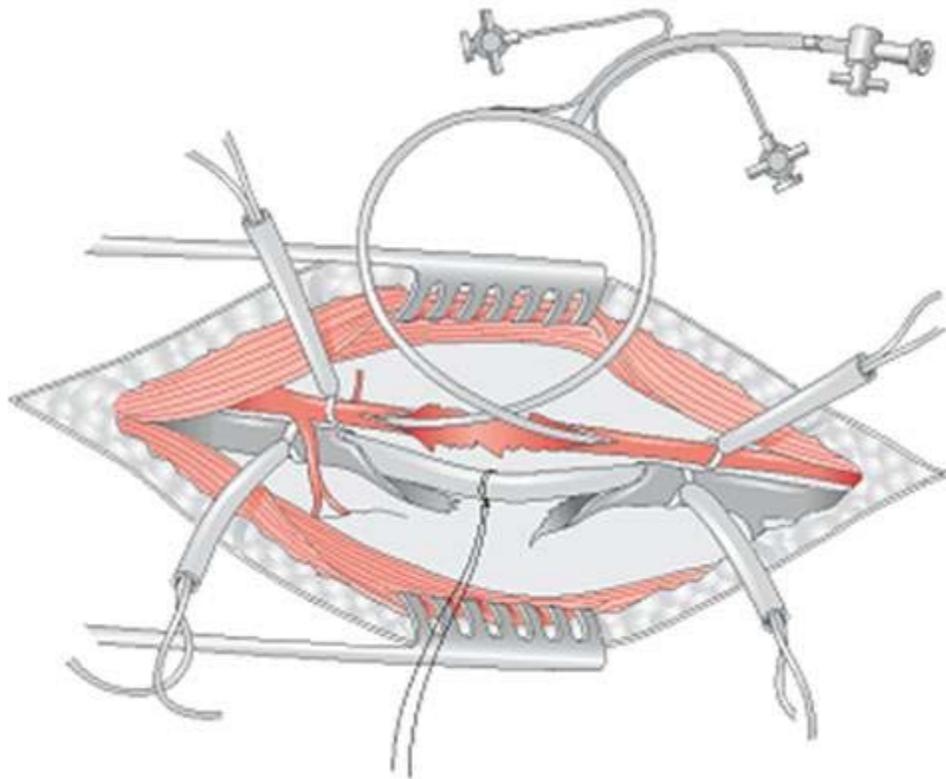


Fig. 10. Example of shunting in a severe artery and vein injury. The artery is shunted with a Pruitt–Inahara shunt and its balloons secured with vessel-loops. For shunting of the vein a piece of ordinary rubber tubing is used. It is also secured with vessel-loops and with a suture

Vessel Repair

While we advocate repairing both the artery and the vein, we do not favor reconstructing the injured vein before the artery. If both can be mended within a reasonable timeframe we recommend that the most difficult reconstruction is performed first. If, however, the artery is shunted it may be advantageous to start with the vein to achieve optimal outflow as soon as possible. Some vascular surgeons favor vein ligation as a general principle because of the potential risk for embolization from vein segments that thrombose after repair.

Arterial Injuries

In general, all injured arteries should be repaired. Sometimes, when necessary in order to save the patient's life or when interruption of an artery does not influence blood flow to the leg, the injured vessel may be ligated. The former is extremely uncommon, but the decision is

difficult when it arises. As an aid we have listed in Table 6 the amputation rates, as obtained from the literature, following ligation of vessels. Among proximal vessels, only branches from the deep femoral artery, but not the main branch, can be ligated without morbidity. Distally, we recommend repair of at least two calf arteries to be on the safe side, but it is possible to leave two interrupted, provided the remaining vessel is not the peroneal artery.

Before definitive repair the surgeon must be sure that the inflow and outflow vascular beds remain open and are free of clots. Liberal use of Fogarty catheters is therefore wise. If the backbleeding is questionable, intraoperative angiography should be performed to make sure the outflow tract is free of clots. Local heparinization, is always indicated. Systemic heparinization can be used for selected patients without other injuries considered to have a low risk for continued bleeding from the wound after debridement. If the foot's appearance or intraoperative angiography suggests microembolization, local thrombolysis can be tried.

The goal for repair is to permanently restore continuity of the artery without stenosis or tension. The type of injury determines the choice of technique. It varies from a couple of vascular sutures to reconstruction with a patch, interposition, or a bypass grafting. Lateral suture for repair of minor lesions, including patching.

A graft is usually needed, and only occasionally can an end-to-end anastomosis be performed without tension. It is always better to use an interposition graft or a bypass to avoid what is even perceived as insignificant tension because anastomotic rupture and graft necrosis may occur when leg swelling and movements pull the arterial ends further apart postoperatively. The major bleeding that can result from this may even be fatal.

Table 6. Amputation frequencies after ligation of different arteries

Vessel ligated	Amputation rate
Common iliac artery	54%
External iliac artery	47%
Common femoral artery	81%
Superficial femoral artery	55%
Popliteal artery	73%

An autologous vein is always the preferred graft material. Vein is more infection resistant than synthetic materials and is more flexible. It allows both elongation and vasodilatation to adjust variations in flow requirements. The great saphenous vein from the contralateral leg is the primary choice for a graft. It is a serious mistake to use the great saphenous vein from the traumatized leg if the deep vein is also injured or if injury is suspected. Interruption of the saphenous vein with obstructed concomitant deep veins will rapidly cause severe distal swelling of the leg and graft occlusion within days. The vein can be harvested at a level in the leg where the saphenous vein diameter fits the artery that needs to be repaired. A graft slightly larger than the artery should be obtained if possible. For common femoral artery lesions, two pieces of saphenous vein, both open longitudinally and then sutured together, might be required. An option is to use arm veins as graft material. If veins not are available expanded polytetrafluoroethylene (ePTFE) is the second choice. The main reasons not to use it are the slightly higher risks for postoperative graft occlusion and infection.

Venous injuries

Most venous injuries are exposed when the wound is explored. While vein ligation may lead to leg swelling, it rarely causes ischemia or amputation. On the other hand, the only benefit of vein ligation is rapid bleeding control and a reduced operating time. Major veins can therefore be ligated to save the life of an unstable patient. If possible, however, most veins should be repaired, especially the popliteal and the common femoral veins. Calf veins can be ligated without morbidity. In patients with combined injuries, both the vein and the artery should be repaired to enhance the function of the arterial reconstruction. Control of bleedings can be achieved by fingers or a “strawberry” or “peanut” to compress the vein proximal and distal to the injured site, or by using gentle vascular clamps. A continuous running suture, almost without traction in the suture line, is often sufficient to close a stab wound. When the vein is more extensively damaged, a patch or an interposition graft may be needed for repair. As with arteries, it is important to match the caliber of the interposition graft and the injured vein. Veins without blood are collapsed, so it is easy to underestimate their size. An autologous vein is preferred over synthetic materials.

Finishing the Operation

After the vascular reconstruction is finished the resulting improvement in distal

perfusion should be checked. This is indicated as well-perfused skin in the foot and palpable pulses. If there is any doubt about the result control angiography should be performed. Preferably, it is done from the proximal control site to enable visualization of all anastomoses. If problems are revealed the reconstruction must be redone and distal clots extracted as previously described. While vascular spasm is rare and should not be regarded as the main explanation for lack of distal blood flow, injection of 1–2 ml papaverine into the graft can also be tried.

After vascular repair all devitalized tissue and foreign material should be removed to reduce the risk of postoperative infection. Grafts and vessels should be covered with healthy tissue by loosely adapting it with interrupted absorbable sutures. For injuries with massive tissue loss it may be impossible to cover the graft. This increases the risk for postoperative anastomotic necrosis and rupture. Split or partial skin grafts or biological dressings can be used to prevent this. Occasionally it may be better to perform a bypass through healthy tissue, thus avoiding the traumatized area, to minimize graft infection and postoperative complications.

Endovascular Treatment

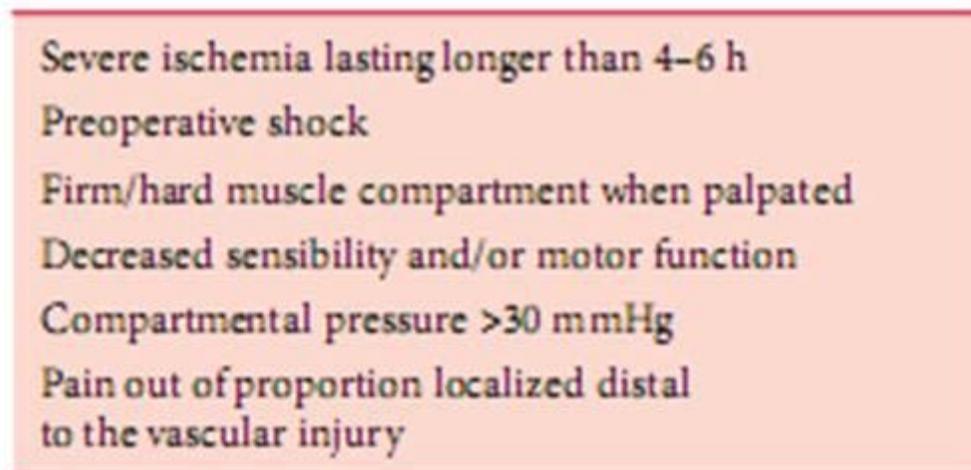
Endovascular treatment of vascular injuries to leg vessels is attractive because it provides a way to achieve proximal control, reduce ischemia time, and simplify complex procedures. There is not a lot of experience – at least not reported in the literature – with endovascular treatment. Some centers have reported successful semiselective treatment of pseudoaneurysms and arteriovenous fistulas in the groin. Small patient series have been published on embolization of bleeding branches to the deep femoral artery and stent graft control of small lacerations in the femoral arteries. We have only treated occasional patients this way so far. Endovascular treatment will probably be an option for many patients with soft signs of vascular injury, who will undergo angiography and then will be found to have a minor bleeding, a pseudoaneurysm, a fistula, or an intimal flap. The possibilities of considering endovascular repair for patients with hard signs of vascular injury to the legs will depend on logistics and the organization of a hospital's endovascular team. With around-the clock availability, patients with less severe distal ischemia may be subjected to angiography with possibilities for endovascular treatment in mind. Patients with penetrating injuries that are actively bleeding may undergo balloon occlusion of a proximal artery to accomplish control and then be transferred to the operating room for repair. In other hospitals where an endovascular team is

not available during weekends, angiography is skipped in some patients with soft signs, and they are treated with an open procedure and intraoperative angiography instead. But it is likely that endovascular treatment will be the treatment of choice for a significant proportion of patients in the near future.

Management After Treatment

As for most vascular procedures, the risk for bleeding and graft thrombosis of the reconstructed artery is higher during the first 24 h after the operation. Postoperative monitoring of limb perfusion, including inspection of foot skin and wounds and palpation of pulses, is necessary at least every 30 min for the first 6 h. If pulses are difficult to ascertain, ankle pressure should be measured. Loss of pulses or an abrupt drop in pressure indicate that reoperation may be required, even when the graft appears to be patent either clinically or on duplex scanning. As for acute leg ischemia caused by thrombosis, there is also a substantial risk for compartment syndrome. Particularly when the ischemia duration has been long, this risk is considerable, and it is important to examine calf muscles for signs of compartment syndrome. This assessment includes motor function, tenderness, and palpation of the muscle compartments in the calf. Findings that may suggest fasciotomy are listed in Table 7.

Table 7. Medical history and clinical findings suggesting that fasciotomy may be needed



Severe ischemia lasting longer than 4–6 h
Preoperative shock
Firm/hard muscle compartment when palpated
Decreased sensibility and/or motor function
Compartmental pressure >30 mmHg
Pain out of proportion localized distal to the vascular injury

For most patients administration of dextran or low molecular weight heparin is indicated to avoid postoperative thrombosis. This is especially important for patients with both venous and arterial reconstruction. Patients who have been subjected to venous ligation need extra

attention and measures against limb swelling. Antibiotic treatment started preoperatively or intraoperatively is usually continued after the operation. We use a combination of benzylpenicillin and isoxazolyl penicillin to cover streptococci, clostridia, and staphylococci.

Fasciotomy

When the compartment pressure is clearly increased or when there is a risk of developing increased pressure, fasciotomy of muscular compartments distal to a vascular injury should be performed in association with the vascular repair. Factors suggesting an increased pressure are listed in Table 7.

Fasciotomy of the Calf Compartments (Two Incisions)

Medial Incision

A 15–20-cm-long skin incision, starting slightly below the midpoint of the calf and downwards parallel to and 2–3 cm dorsally of the medial border of the tibia, is used to decompress the deep and superficial posterior muscle compartments (Fig. 11). It is important to avoid injury to the great saphenous vein. Sharp division of skin and subcutaneous fat reaches the fascia. The skin and subcutaneous fat is mobilized en bloc anteriorly and posteriorly to expose it enough to provide access to the compartments. The fascia is then opened in a proximal direction and distally down toward the malleolus under the soleus muscle. At this level, the soleus muscle has no attachments to the tibia, and the deep posterior compartment is more superficial and easier to access. Through the same skin incision, the fascia of the superficial posterior muscle compartment is cleaved 2–3 cm dorsally and parallel to the former. A long straight pair of scissors with blunt points is used to cut the fascia using a distinct continuous movement in the distal direction, down to a level of 5 cm above the malleoli and proximally along the entire fascia.

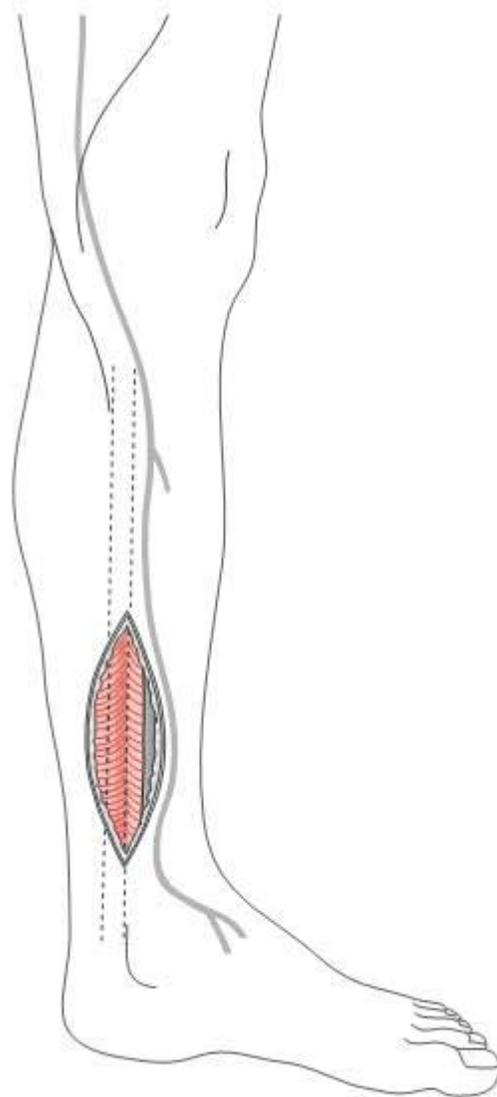


Fig. 11. Incision and exposure for fasciotomy of posterior compartments. The posteriodottedline indicates fasciotomy incision for the superficial compartment and the anteriordottedline for the deep compartment

Lateral Incision

The skin incision for fasciotomy of the lateral and anterior muscle compartments is oriented anterior to and in parallel with the fibula. The dorsal position of the lateral compartment, however, often requires more extensive mobilization of the subcutaneous fat to reach it. The superficial peroneal nerve is located anteriorly under the fascia and exits the compartment through the anterior aspect of the fascia distally in the calf. To preserve this nerve, direct the scissors dorsally when making the fasciotomy in both directions. (See Fig.

11.).

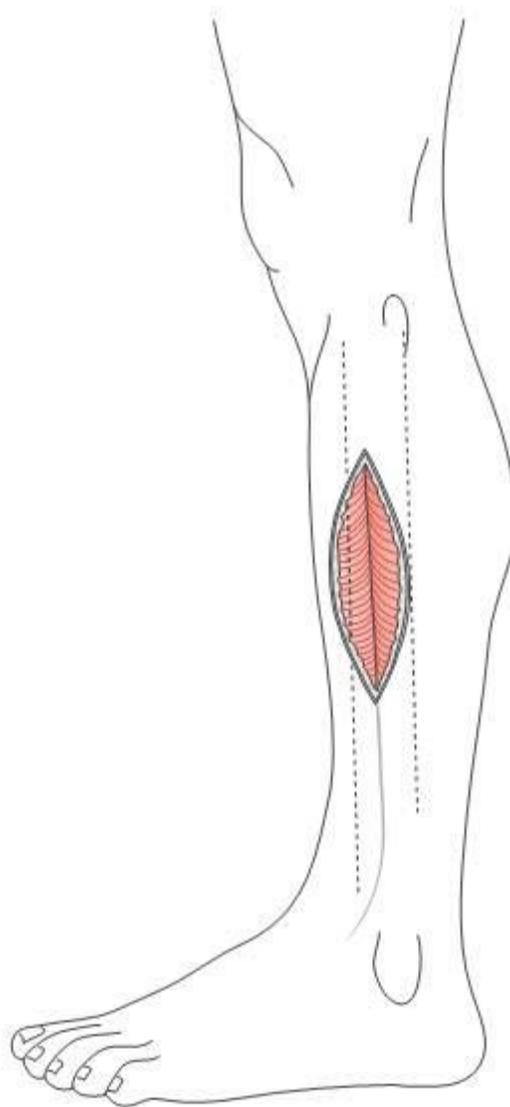


Fig. 11. Incision for fasciotomy of anterior and lateral compartments. Through the demonstrated skin incision, long incisions are made along the dottedlines in the fascia. Caution must be taken with the peroneal nerve

Unless the swelling is very extensive, 2-0 prolene intradermal suture is loosely placed to enable wound closure later (Fig. 12). It is important to leave long suture ends at both sides to allow wound edge separation the first postoperative days. The skin incisions are left open with moist dressings.

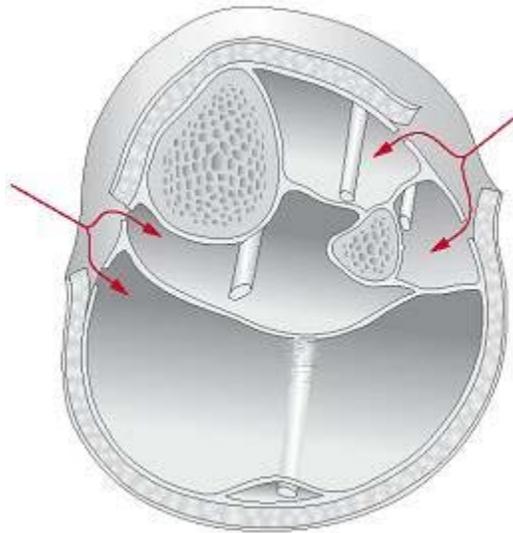


Fig. 12. Cross-section demonstrating the principles for decompression of all four muscle compartments in the calf through a medial and a lateral incision. The location of major nerve bundles in the different compartments is indicated

TRAUMATIC INJURY OF THE VENA CAVA AND ITS MAJOR BRANCHES

Trauma mechanisms

In general, the superior thoracic, intrapericardial and retrohepatic segments of the caval vein may sustain either a penetrating or a closed trauma. The VC is usually involved in single penetrating injuries.

Closed trauma

Closed injuries usually result from a severe deceleration trauma in the horizontal direction such as in traffic accidents, or in the vertical direction, like in a fall from high altitude. The resulting shear forces cause a partial or total avulsion of the venous segment, as can be observed at the atriocaval or hepatocaval junctions, or at the level of the azygos, renal, or superior mesenteric veins.

The common and internal iliac veins are damaged where they pass bony structures, especially after pelvic fractures.

In the literature, 10 cases of post-traumatic thrombosis of the IVC were found in 1999, occurring 3 days to 3 years after a high-energetic trauma. This thrombosis could have been secondary to organization of an initially localized thrombus, which allowed spontaneous hemostasis after an injury to the vessel wall.

As an anecdote, a laceration of the IVC was reported following a nonpenetrating trauma caused by the high-pressure water jet of an industrial cleaner.

Penetrating trauma

The most frequent penetrating traumas are those from gunshots rather than stabbing weapons. The statistical preponderance of dexterity among the attackers explains why the caval vein, in its right lateral position, is spared most of the time during frontal attacks on the left part of the body.

All kinds of trauma, varying between a localized puncture and injuries with substantial tissue loss, can occur. Bullet injuries are the most destructive. Except for the trauma to the caval vein, they are often responsible for other serious lesions to neighboring structures that induce a risk of sepsis, which in part influences the prognosis.

Iatrogenic trauma

This may concern vessel wall perforations due to the introduction of an endoluminal catheter or caval filter, of which the prognosis is usually good when untreated, or injuries occurring during surgery. Trauma occurring during thoracoscopy or laparoscopy may either pass unnoticed or be revealed secondarily, or may be diagnosed directly because of excessive blood loss during the procedure.

As this type of trauma is becoming more and more frequent due to the increasing use of these techniques, it forms the topic of another chapter of this book. Finally, direct iatrogenic damage to the left innominate vein or the intrapericardial caval vein can complicate certain cardiac re-interventions.

General intervention principles

Generally speaking, no additional investigations should interfere with the initial reanimation of a patient with an injured abdomen or thorax. Not until the condition has been

stabilized can a transthoracic cardiac or abdominal ultrasound exam be performed. When the clinical suspicion of a major vascular trauma has risen, a surgical exploration is justified.

In several North American series, patients in whom reanimation remained insufficient underwent an urgent clamping of the thoracic descending aorta through a left anterolateral thoracotomy.

Although meant to restore arterial pressure in these dying patients, this heroic maneuver yielded disappointing results. In 2001 Carr et al. Reported only 14 survivors out of 151 cases in the literature treated in this manner. In another series reporting on the outcome of 302 abdominal vascular injuries, thoracotomy was performed in 131 cases (43%), of which 43 times (14%) were in the shockroom and 88 times (29%) in the operating room.

The survival rates in these two groups were only 2% and 10%, respectively. In another recent series on 136 IVC injuries, one single patient survived out of 25 who underwent a thoracotomy for clamping. These results can be explained by the fact that the most severely injured patients would obviously have died already on the site of the accident, were it not for the modern means of transport and prehospital reanimation.

Local consequences of caval vein trauma

Half of the patients who suffered from a caval vein trauma, and particularly those with an arterial lesion, present with a severe hemorrhagic shock. As opposed to the arteries, the veins show a poor vasoconstrictor response and cannot generate an effective hemostasis through their own physiologic properties.

Because of the absence of valves, IVC injuries are not only characterized by bleeding from the iliac, but also by reflux from the atrial side. These veins, however, do have some features that can lead to a spontaneous hemostasis, at least temporarily.

The circulation, including the caval veins, is maintained at low pressure. In case of an IVC injury, the neighboring tissues can cause an effective plugging that limits the hemorrhage and sometimes leads to thrombus formation and healing of the vessel wall. The hemorrhage may thus be contained by the retroperitoneum, the pancreas, the duodenum or the posterior side of the liver. This phenomenon is particularly seen in simple lesions caused by stabbing weapons or low-velocity shot wounds, but is exceptional in case of a perforating trauma of the IVC or damage secondary to highvelocity fire weapons.

Common principles of major venous injuries

Patients who make it to the operating room should be treated by a surgical team of sufficient number.

In order to respond to a possible decompensation during anesthetic induction or surgical incision, they are usually anesthetized after preparation of the skin and operation field. In most cases the patient is positioned in the dorsal decubitus position, and the operation field runs down from the thoracic and abdominal areas to the knees. The unanimously acknowledged surgical access in case of an abdominal venous injury is a median laparotomy from xyphoid to pubic area, to which a median sternotomy is added. After the intestines are moved aside, the peritoneal cavity is emptied of a variable amount of blood by means of aspiration into a reservoir. A nonpulsating hematoma of black blood directly identifies a venous injury. Continuous aspiration of it carries the risk of exsanguination and should be prohibited. Preferably, gauzes should be packed into all abdominal quadrants, as it is difficult to localize the origin of the venous bleeding, which expands like a flat surface rather than a jet.

This hemostasis enables to optimize the hemodynamic situation by correcting the volume. A temporary clamping of the infrarenal aorta only, performed without difficulty and visibly, or even simple compression, can help to reach a stable situation.

In contrast with arterial injury, for which clamping of the inflow to obtain adequate hemostasis usually suffices, hemostasis of a venous injury requires checking of both the inflow and the outflow tract. Use of conventional clamps is dissuaded, especially when they have to be applied half blindly. Moreover, the posterior side of the IVC should be checked even more carefully to avoid additional damage.

Whenever possible, hemostasis should be achieved through direct digital compression, fixed gauzes, covered atraumatic clamps placed under visual control or by using tourniquets. Most of the traumatic lesions of the caval vein and its branches may then be treated by means of direct closure or simple ligation in certain localizations. The use of prosthetic material in the form of patches or tubular grafts is rarely necessary. Autologous venous jugular or saphenous grafts, sometimes composed and organized in order to obtain sufficient material for the reconstruction (helical graft), are preferred.

TRAUMA OF THE IVC

The IVC is more frequently subjected to trauma than the SVC. The abdominal vascular structure are mostly injured, mainly through a penetrating trauma, The problems of each

segment are described separately.

Infrarenal IVC. The infrarenal IVC is involved in 25% to 50% of the injuries to the IVC. Its exposure via a right mediovisceral approach is preferred over a median retroperitoneal approach, which offers the right access to the abdominal aorta and the left renal vein but less to the IVC.

The coloparietal exposure is pursued while retaining the duodenal area and the head and body of the pancreas to the left. This allows exposure of the right kidney and its ureter. After rotation of the viscera, the IVC can be exposed nicely from the junction of the iliac veins to its juxtahepatic segment.

A serious hemorrhage occurring during exposure of the IVC should be compressed by an assistant to allow the surgeon to continue the exposure. Subsequently, various procedures can be chosen for the hemostasis and reconstruction. The simplest one is to apply gauze over the lesion. However, maintaining sufficient compression during the whole duration of the venous repair is difficult. Total clamping of the IVC at a distance from the lesion by means of conventional clamps or tourniquets is risky due to the vulnerability of the vessel wall and the possibility of permanent reflux supplied by the collateral circulation from the azygos and lumbar veins.

A smaller vessel wall trauma can be treated by means of bipolar endoluminal clamping using occlusive balloon catheters (Fogarty), or urinary catheters (Foley). Lesions of the anterior or lateral sides of the IVC can best be handled using a large Satinsky clamp, which has the advantage that it partially clamps the lumen and thereby does not induce venous hypertension. Direct closure is the technique of choice to repair lesions of the infrarenal IVC. In urgent cases, a quick closure using thicker stitches (3/0 or 4/0) is preferable to trying to do a more esthetical but more time-consuming reconstruction using thinner stitches. This is underlined by the finding that there is no relation between the outcome of the reconstruction and the degree of residual stenosis. Rarely, a secondary enlargement of the sutured area is required. Lesions of the posterior side can be closed, either via direct exposure by rotating the IVC after resection of one or more lumbar veins, or via a transcaval approach after having enlarged the entry opening in the anterior side, which is to be repaired afterward. In case of major destruction of the vessel wall, particularly in combination with shock and other vascular lesions, ligation of the infrarenal IVC or ilio caval junction together with ligation of all lumbar veins is generally advocated.

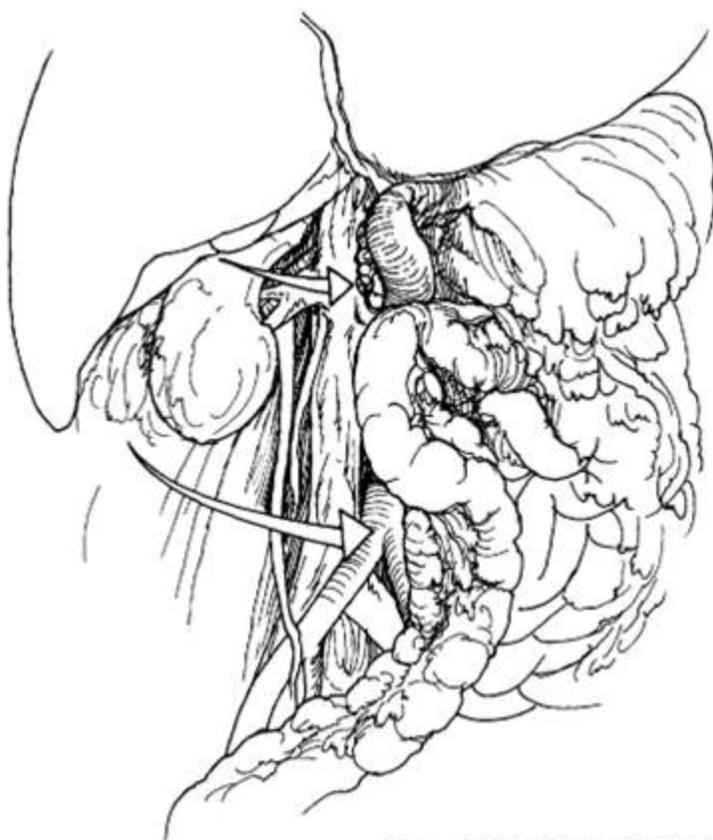


FIG. 1 Approach of the IVC, from its origin to the subhepatic segment, by right mediovisceral rotation after mobilizing the right colon, duodenum, and pancreas.

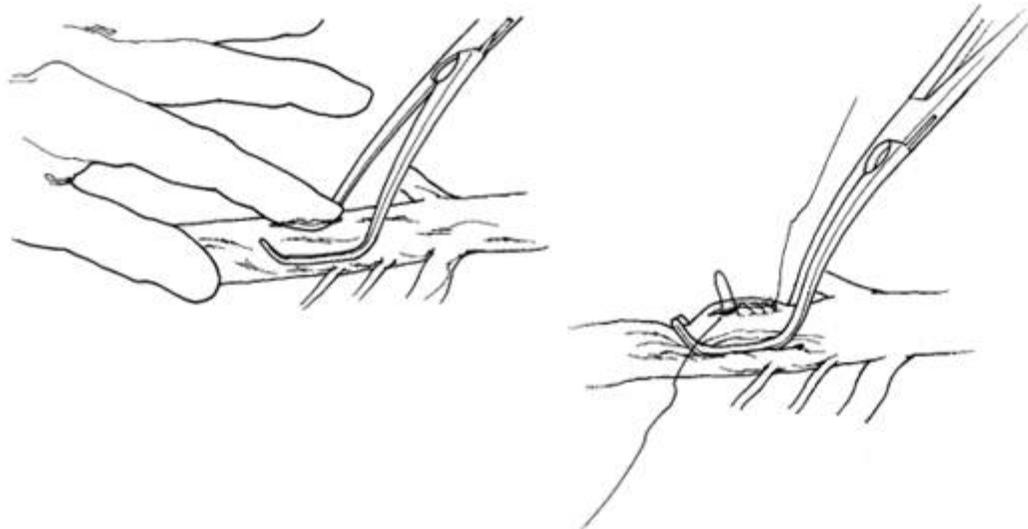


FIG. 2 Lateral clamping of the vena cava by means of a Satinsky clamp after digital control of the bleeding.



FIG. 3 Exposure and closure of a lesion in the posterior side of the IVC after transection of lumbar veins and axial rotation.

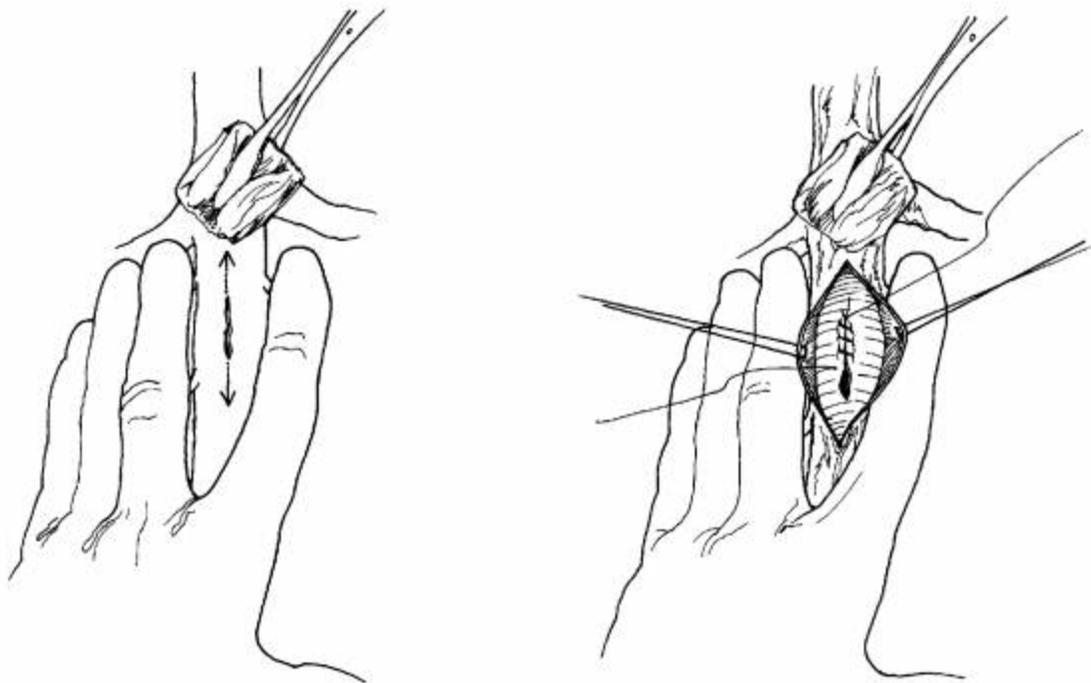


FIG. 4 Closure of a perforating lesion of the IVC. The posterior wall is sutured first via the interior of the vein after having enlarged the lesion in the front. This is closed secondarily.

Juxtarenal and juxtahepatic IVC.

The juxtarenal IVC extends two centimeters above and below the junction with the renal veins. It runs upward through a short juxtahepatic IVC, accessible via the lower border of the liver. Involvement of these segments comprises 20% to 50% of the traumatic lesions of the IVC.

Access to the Juxtarenal segment is obtained by a right mediovisceral rotation (see above). Exposure of the posterior side by means of an axial rotation requires either a right retrorenal release, mobilizing the kidney to the median line, or a complete resection between two clamps of the right renal vein, which is to be reconstructed after repositioning the IVC. The juxtahepatic segment can be approached similarly, by means of a complete right mediovisceral rotation, or more selectively by mobilizing only the duodenum and pancreas. The narrow relation between the anterior side of the IVC and the posterior side of the portal vein should be kept in mind, to avoid any damage to the latter during the dissection.

The clamping techniques do not differ much from those used for the infrarenal segment. To obtain a dry operation field at the Juxtarenal level requires the simultaneous clamping of both renal veins, sometimes including the renal arteries.

Control over the inflow and outflow of the Juxtarenal segment usually does not cause any problems. However, the outflow of the juxtahepatic IVC may be difficult because it is a short segment. Exerting anteroposterior pressure on the liver and thereby compressing the end of the juxtahepatic JVC leads to the same result. Reflux from the suprarenal vein, if any, is usually minimal. The reconstruction options for the Juxtarenal and juxtahepatic segments of the IVC can be superposed on those for the infrarenal segment in case of simple lesions. They basically consist of direct closure. Although a stenosis with a 75% caliber reduction due to this suture may be acceptable, we find it important to preserve a residual lumen of at least 30% of the initial diameter. Correction of remaining stenoses regarded excessive is the only indication for enlargement procedures by means of a venous patch rather than prosthetic material. Complete replacement of this caval segment using a venous or prosthetic graft is exceptional and restricted to cases with extensive vessel wall damage in patients whose hemodynamic condition and hemostasis allow for a complex reconstruction.

INJURIES OF THE MAJOR BRANCHES

Iliac veins.

Apart from the osseous trauma to the pelvis, iatrogenic lesions from laparoscopic procedures or maneuvers to obtain control at the common iliac artery are the most common causes of this type of injury. Access to the ilio caval junction and the distal ends of the iliac veins becomes difficult due to the bleeding that quickly fills the pelvic cavity. The best route is obtained via mobilization of the right colon, retaining it to the left of the aorta and the right common iliac artery, transected between two clamps. The ureter is the only critical structure in this region. Hemostasis is obtained by compressing the IVC with gauzes against the spine and the common iliac veins against the promontory. Hemostasis of iliac vein injuries extending into the internal iliac veins is more difficult, because of their rich collateral circulation. Direct closure is standard. A too extensive venous damage should be treated with ligation of an iliac vein or ilio caval junction. The large number of side branches between the iliac veins and the reno-azygolumbar network explains why these ligations are usually well tolerated.

Renal veins.

Injuries to the renal veins account for 8% to 12% of abdominal vascular injuries. They occur in 6% to 13% of the IVC injuries. These veins are injured by penetrating as well as by closed traumas. Iatrogenic trauma of the left retro-aortic renal vein during abdominal aortic surgery, should be avoided by careful analysis of the pre-operative computed tomography (CT) scan. Surgical exploration of retroperitoneal peri-renal hematomas is not justified in the case of a closed trauma, until an intravenous pyelography, a renal angiogram or a CT scan in a stable patient have shown a normal renal excretion.

In contrast, surgical exploration of peri-renal penetrating injuries is standard.

The right renal vein is accessed by means of mobilization of the duodenum and colon. The left renal vein is approached via a median longitudinal incision of the retroperitoneum. To reduce the bleeding it may be useful to control and clamp the renal arteries. A lateral closure, performed under digital control, either after clamping the injured vein or after lateral clamping of the IVC at its ostium, can most often be realized. If ligation of the right renal vein is necessary, one should choose between a nephrectomy and a renal autotransplantation if the hemodynamics allow for it. Ligation of the left renal vein is well tolerated as long as the capsular and genital veins are spared.

Azygos vein.

The azygos vein can be injured through penetrating and closed traumas. A massive hemothorax is common and indicates an approach via a right thoracotomy. The bleeding can be enhanced while spreading the ribs. There is a considerable risk of air embolization, which is why one should opt for an initial compression maneuver rather than excessive aspiration of the operation field. Final treatment consists of ligation of the azygos vein with lateral closure of the SVC when it is controlled by a Satinsky clamp.

Venous brachiocephalic trunk.

The right and left venous brachiocephalic trunks join to form the SVC. A lesion at this level may extend into the SVC.

In practice, the left trunk is the most affected because of its length and its tract, which crosses the median line and is exposed particularly during sternotomies for cardiac procedures. Ligation is well tolerated without any sequelae.

Postoperative measures

Whether the lesion of the IVC is repaired, ligated or simply compressed, certain measures must be taken to prevent thrombo-embolic complications resulting from stasis of blood in the lower part of the body. These comprise elevation of the legs, elastic compression and antithrombotic drugs. There is no consensus on the anticoagulation regimen.

Late sequelae, such as deep venous insufficiency, are rare regardless of the treatment chosen.

Follow-up and prognostic factors

In nearly 50% of cases, patients suffering from a traumatic lesion of the IVC die before any treatment is given. The results of 20 series in the literature, comprising 2032 operated patients, are presented in the Table, which shows that the mortality rate for all the different locations is 44% on average, ranging from 21% to 75%. No improvement is clear with time, despite a substantial progression in treatment options. Nevertheless it is plausible that the

improvement of collection and transport of the wounded has led to the operation of more and more diseased patients.

Death occurs mostly within 24 hours. In more than 90% of the cases this is due to massive bleeding, mainly during attempts to explore and repair the damage. During the early follow-up period, the bleeding induces an irreversible disseminated intravascular coagulopathy. Multi-organ failure or a sepsis, often related to intestinal lesions, are the other major causes of death after this period. The presence of shock on arrival, the existence of concomitant lesions, especially of the aorta, the closed character of the trauma, absent spontaneous hemostasis of the lesion and the retrohepatic or juxtadiaphragmatic localization are significantly associated with a poor outcome

Methodical instructions for students V years

(Vascular Surgery)

Lesson № 9 (Practical - 6 hours).

TOPIC: Arterial thrombosis and embolism.

Topics: 1. Arterial thrombosis

2. Arterial embolism.

3. Surgical treatment of arterial thrombosis and embolism

Purpose: To be able to diagnose, to know the clinic of acute vascular insufficiency of the lower extremities, depending on the causes, clinical features of disease, and to justify the conservative and surgical treatment

Professional oriented students:

Statistics show an increase in acute arterial embolism and thrombosis - severe complications, which will meet the doctors of all specialties. However, the problem of care for patients with this disease is still far from solved, as evidenced by the high mortality rate - from 20 to 35%, and the high frequency of limb amputation at the gangrene - almost 20% of patients.

Injuries are an important social and medical problem. In recent years, increased the number of serious injuries and connected, which often lead to severe bleeding and death

Methodology for practical work

The practical part of the session 9.00 - 12.00

Algorithm for communication of students with patients with any pathology, which is seen on the subject (communication skills):

1. Greet and identify himself.
2. The face should be welcoming smile - this allows you to establish a trust relationship with the patient.
3. A patient in a pleasant form should explain the purpose of the visit, the subject and the duration of the conversation and get his consent.
4. If a patient only comes to the hospital to conduct correctly, and quiet conversation with his relatives, which together with the doctor to inform them about the previous diagnosis, hospitalization, for some surveys, which are scheduled to perform in the future.
5. Before the physical examination survey methods to explain what the patient survey will be carried out, indicate some discomfort and discomfort that may feel the patient at the time of the survey, noting bound by the survey in the diagnosis of this disease and to obtain his consent.
6. If you need transportation to the place of examination (visual room, X-ray, ultrasound study) to explain its need for the patient.
7. Prepare for the survey (for data pathologies meant inspection, palpation limb aortoarteriography, ultrasound vascular feet, Doppler) - wash your hands with warm water, put on signets, prepare tools for the performance of other diagnostic manipulation.
8. To hold something or other planned medical examination or manipulation.
9. Together with the attending physician in the correct and easy to understand format patient to explain the results of either survey.
10. Involve family and patient to talk in simple terms to explain to them the results of the survey data and in the presence of previous surveys to compare their results, be sure to ascertaining whether they understand your explanations.
11. Required only in the presence of the attending physician to substantiate the feasibility of surgical intervention for the treatment of this pathology in charge of the patient.

12. Following surgical treatment only in the presence of the attending physician and with his consent should communicate the results of surgical intervention the patient and his relatives as well as the possibility of this or early or late postoperative complications.

13. Under the conditions of examination of patients in the postoperative period should be explained to the patient as true to perform hygiene procedures and the like.

14. In polite to get consent from the patient to participate in the ligation.

15. Together with your doctor to explain to the patient, and when the needs and the immediate family, those or other actions regarding manipulations that have been implemented or planned to run in the future as well as the tactics of the subsequent treatment.

16. Finish the conversation should always be the wish of the patient the most rapid recovery.

Work 1

1. Collection of complaints, history, examination of the patient.
2. Identification of clinical symptoms and instrumental.
3. Grouping symptoms syndromes.
4. Determination of the leading syndrome.
5. Interpretation of laboratory - instrumental data
6. Rationale preliminary diagnosis
7. Differential diagnosis.
8. Formulation of clinical diagnosis.
9. Appointment of differentiated treatment programs according to the clinical protocol

Work 2. Browsing the Internet, reading room with cathedral library topical literature.

Program self-students

1. Anatomical functional characteristics of arteries and veins of the lower extremities.

2. The etiology, pathogenesis and classification of acute ischemia of lower extremities.
3. Factors convolution and protivosvertochnoy systems.
4. The mechanism of thrombosis

Break - 12.00-12.30

Seminar discussion of theoretical issues - 12.30-14.00

Theme № 1. Arterial thrombosis

1. The causes of arterial thrombosis.
2. Clinical characteristics of arterial thrombosis.
3. Classification of arterial thrombosis.
4. Differential diagnosis of acute thrombosis of major arteries.
7. Indications for conservative treatment, drugs that are used.
8. Anticoagulants, phybrinolytic, thrombolytic drugs.
9. Control methods for convolution system, their characteristics.

Theme № 2 Arterial embolism

1. Causes of arterial embolism.
2. Clinical characteristics of arterial embolism.
3. Classification of arterial embolism
4. Differential diagnosis of arterial embolism
7. Indications for conservative treatment, drugs that are used.
8. Anticoagulants, fibrinolytic, thrombolytic drugs.
9. Control methods for the convolution system, their characteristics

Theme № 3 Surgical treatment of arterial thrombosis and embolism

1. Indications for surgical treatment of arterial thrombosis
2. Indications for surgical treatment of arterial embolism
3. Contraindications to surgical treatment of arterial thrombosis
4. Contraindications to surgical treatment of arterial embolism
5. Access to the main arteries

Break - 14.00-14.15

Hour self-study students - 14.15-15.00

- Tutorial test licensing examinations «Step 2»;
- Assessment of students who have not passed on the eve of the test control system «Moodle»;
- Letting students practical skills in the corresponding entry in matrikulyar book.

Sample test items and situational problems.

1. The patient admitted to hospital with a diagnosis: embolism the left popliteal artery . What is the most probable cause of embolism?
2. The patient admitted to hospital with suspected embolism the left popliteal artery. What is the symptom will testify in favor of embolism?
3. What phase hemocoagulation inhibits heparin:
 - A. Only the formation of thrombin
 - B. No right answer
 - C. Only the formation of fibrin

D. Only the formation of thromboplastin

E. It operates on the phase of thrombotic

4. Indirect anticoagulants include:

A. Preparation of 4-oksikumarina

B. Preparations of alkaloids

C. Fibrinolytic drugs

D. Pentoksiphylin

E. Thrombolytic drugs

5. The absolute contraindication of anticoagulants include:

A. Bleeding of any location, hemorrhagic diathesis

B. Aplastic anemia

C. Respiratory failure

D. Cardiovascular insufficiency

E. Lack of cerebral circulation

The student should be able to:

1. Septic endocarditis

2. Apply trombolitichiskie drugs and anticoagulants.

3. Appoint conservative therapy for acute lower limb ischemia.

4. Determine the ripple in the typical spots on the major arteries of the lower extremities.

5. Rate dopplerometry.

6. Apply methods to temporarily stop bleeding.

7. Appoint conservative therapy in acute blood loss.

The answers to tests and situational tasks:

1. Septic endocarditis
2. Severe pain in the limbs
3. A.
4. A.
5. A.

Information:

Basic:

1. Jack L. Cronenwett, Wayne Johnston Rutherford's Vascular Surgery. 7th Revised edition. - Elsevier -Health Sciences Division. – 2010.
2. Alun H. Davies and Colleen M. Brophy (Eds) Vascular Surgery. – Springer. - 2006.
3. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
4. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – 2Т.1.
5. Hospital surgery / Edited by L. Kovalchuk, V. Sayenko, G. Knysov, M. Nychytailo . – Ternopil: Ukrmedknyga, 2004.
6. [Materials for practical classes](#)

Additional:

1. К. Заринш, Б. Гевертс. Атлас по сосудистой хирургии. – Москва 2009. – Гэотар-Медиа

Guidelines made

ass. Kostiv S.Ya.

Обсуждено на заседании кафедры

" 11" января 2013 р. протокол № 9

Methodical instructions for students 5-year

Methodical instructions for lesson № 8

THEME: "Obliterating diseases of arteries of lower extremities.

Intestinal angina.

Practical - 6 hours

Topics: 1. Obliterating atherosclerosis of lower extremities

2. Occlusive disease of lower extremities

3. Abdominal angina

Aim: To be able to diagnose, to know the clinic chronic and acute ischemia of lower extremities, depending on the causes, clinical features of disease and to justify the conservative and surgical treatment

Professional oriented students:

Obliterating diseases of the extremities are the first place for the clinical importance and frequency and form a core group of organic arteriopathy, which are accessible to surgical treatment.

Statistics show an increase in acute arterial embolism and thrombosis - severe complications, which is necessary to meet the doctors of all specialties. However, the problem of care for patients with this disease is still far from solved, as evidenced by the high mortality

rate - from 20 to 35% and high frequency of limb amputation at the gangrene - almost 20% of patients.

Methodology for practical work

The practical part of the session 9.00 - 12.00

An algorithm for communication of students with patients, which is seen on the subject (communication skills):

Greet and identify himself.

The face should be welcoming smile - this allows you to establish a trust relationship with the patient.

A patient in a pleasant form should explain the purpose of the visit, the subject and the duration of the conversation and get his consent.

If a patient only comes to the hospital to conduct correctly, and quiet conversation with his relatives, which together with the doctor to inform them about the previous diagnosis, hospitalization, for some surveys, which are scheduled to perform in the future.

Before the physical examination survey methods to explain what the patient survey will be carried out, indicate some discomfort and discomfort that may feel the patient at the time of the survey, noting bound by the survey in the diagnosis of this disease and to obtain his consent.

If you need transportation to the place of examination (visual room, X-ray, ultrasound study) to explain its need for the patient.

Prepare for the survey (for data pathologies meant inspection, palpation limb aortoarteriografiya, ultrasound vascular legs, Doppler) - wash your hands with warm water, put on signets, prepare tools for the performance of other diagnostic manipulation.

To hold something or other planned medical examination or manipulation.

Together with the attending physician in the correct and easy to understand format patient to explain the results of either survey.

Involve family and patient to talk in simple terms to explain to them the results of the survey data, and in the presence of previous surveys to compare their results, be sure to

ascertaining whether they understand your explanations.

Must be only in the presence physician to substantiate the feasibility of surgical intervention for the treatment of this disease to supervise the patient.

Following surgical treatment only in the presence of the attending physician and with his consent should communicate the results of surgical intervention the patient and his relatives as well as the possibility of this or early or late postoperative complications.

Under the conditions of examination of patients in the postoperative period should be explained to the patient as true to perform hygiene procedures and the like.

In polite to get consent from the patient to participate in the ligation.

Together with your doctor to explain to the patient, and when the needs and next of kin of those or other actions regarding manipulations that have been implemented or planned to run in the future as well as the tactics of the subsequent treatment.

Finish the conversation should always be the wish of the patient the most rapid recovery.

Work 1

1. Collection of complaints, history, examination of the patient.
2. Identification of clinical symptoms and instrumental.
3. Grouping symptoms syndromes.
4. Determination of the leading syndrome.
5. Interpretation of laboratory - instrumental data
6. Rationale preliminary diagnosis
7. Differential diagnosis.
8. Formulation of clinical diagnosis.
9. Appointment of differentiated treatment programs according to the clinical protocol

Work 2. Browsing the Internet, reading room with cathedral library topical

literature.

Program self-students

1. The anatomic and functional features of arteries of lower extremities
2. The etiology, pathogenesis and classification of chronic lower limb ischemia
3. The etiology, pathogenesis and classification of acute lower limb ischemia.
4. The mechanism of action and dose antispasmodics, analgesics, anticoagulants, antiplatelet agents, angioprotectors and vitamin preparations/

Break - 12.00-12.30

Seminar discussion of theoretical issues - 12.30-14.00

Theme № 1 obliterating atherosclerosis of lower extremities

Etiology and pathogenesis of obliterative atherosclerosis of lower extremities.

Classification of chronic ischemia on Fontane.

Classification of chronic ischemia and the level of occlusion of arteries in obliterating atherosclerosis of A.A. Shalimov.

Modern theory and risk factors in the development of atherosclerosis.

Methods of the arterial system.

Complications of atherosclerotic lesions of lower limb arteries and methods for their prevention.

Definition, clinical characteristics and methods of surgical treatment of Leriche syndrome.

Indications and contraindications for conservative treatment of obliterative atherosclerosis of arteries of lower extremities.

Indications and contraindications for surgical treatment of obliterative atherosclerosis of arteries of the lower extremities.

Early postoperative complications of reconstructive operations on arteries of lower

extremities in obliterating atherosclerosis and methods for their prevention.

Rehabilitation of patients who had undergone reconstructive surgery on the aorta and major arteries.

Modern methods aortoarteriografii, complications of aortography and methods of prevention.

Methods to reduce the concentration of cholesterol and lipoproteins in blood plasma (pharmacological, instrumental).

Theme № 2 obliterating endarteriit lower extremities

Etiology and pathogenesis of obliterative endarteriitis lower extremities.

Classification of obliterative endarteriitis lower limbs Shabanov and fountains.

Features of clinical course of obliterative endarteriitis lower extremities.

Differential diagnosis of obliterative endarteriitis lower extremities with obliterative atherosclerosis.

Differential diagnosis of obliterative endarteriitis lower extremities with diabetic angiopathy, Raynaud's syndrome, nonspecific cryoglobulinemia.

Indications and contraindications for conservative treatment of obliterative endarteriitis lower extremities.

Principles of conservative treatment of obliterative endarteriitis lower extremities

Indications and contraindications for surgical treatment of obliterative endarteriitis lower extremities, methods of surgical treatment.

Rehabilitation of patients with obliterative endarteriitom lower extremities.

Theme № 3 abdominal angina

Causes of abdominal ischemic syndrome

Clinical characteristics of abdominal ischemic syndrome

Classification of abdominal ischemic syndrome

Differential diagnosis of abdominal ischemic syndrome.

Surgical tactics and methods of surgical treatment of abdominal ischemic syndrome.

Features of the postoperative period after surgery for abdominal ischemic syndrome.

Indications for conservative treatment, drugs that are used.

Break - 14.00-14.15

Hour self-study students - 14.15-15.00

- Tutorial test licensing examinations «Step 2»;
- Assessment of students who have not passed on the eve of the test control system «Moodle»;
- Letting students practical skills in the corresponding entry in matrikulyar book.

Sample test items and situational challenges.

1. Patient 64 years old, complained of rapid fatigue when walking, the feeling of coldness in the lower extremities, the appearance of pain in the lower leg muscles during walking a distance of 150-200 meters is ill about 12 years in the past 3 years noted the deterioration. When viewed from the skin of the lower extremities with a pale shade, cool at the feet, hyperkeratosis, poor body hair on the shins. Ripple in all arteries of both lower extremities (foot artery, popliteal, femoral) is absent. Diagnosis?

2. Sick 29 years old, was admitted with complaints of rapid fatigue, feeling cold in the lower extremities, the appearance of pain in the muscles of legs at a distance of 300 meters had felt unwell for 6 years and connects onset of supercooling. The general condition of the patient was satisfactory. Skin of the lower extremities at the level of the feet and lower third of the

tibia with a pale shade, cool to the touch, delicate, dry. Ripple in the arteries of the lower extremity is determined by the femoral artery at the knee - is weakened, the arteries of the foot - is absent. Ripple in the right thigh and popliteal arteries is satisfactory, at the foot arteries - is absent. Diagnosis?

3. The patient, 61 years old, was admitted to the vascular department with complaints of constant freezing toes, rapid fatigue and the appearance of pain in the muscles of legs, thighs, buttocks and sometimes when walking at a distance of 120-150 meters Sick for 6 years, the deterioration of their condition noted in last year. Above the abdominal aortic systolic murmur auscultated. On examination the skin of the lower extremities, with a pale shade, especially at the feet, dry and cool to the touch. Ripple in the peripheral arteries of the lower extremities is absent at all levels. Diagnosis?

4. What operations are used in obliterating atherosclerosis of the aorta and arteries?

- A. Saphenektomy;
- B. Embolectomy;
- C. Endarterectomy, bypass surgery, prosthetics.
- D. Operation Troyanov-Trendelenburg, Babcock;
- E. Operation Linton, Kukketa.

5. Please provide the most frequent complications of reconstructive operations in arteriosclerosis obliterans of the aorta and arteries of the lower extremities.

- A. Acute thrombophlebitis in the great saphenous vein;
- B. Acute thrombophlebitis in the small saphenous vein;
- C. Thrombosis of the inferior vena cava;
- D. Thrombosis segment reconstruction;

E. Chronic venous insufficiency.

6. Leriche syndrome develops:

A. At an atherosclerotic occlusion of the subclavian artery;

B. At an atherosclerotic occlusion of the popliteal artery;

C. At an atherosclerotic occlusion of the femoral artery;

D. At an atherosclerotic occlusion of terminal part of the abdominal aorta or common, external iliac arteries;

E. At an atherosclerotic occlusion of arteries shins.

7. What complications may arise during percutaneous catheterization of the aorta by Seldinger?

A. Bleeding, thrombosis, embolism.

B. Acute thrombophlebitis leg veins.

C. Relaxation of the diaphragm.

D. Thrombosis of the subclavian vein.

E. Endocarditis.

The student must know:

1. The etiology of atherosclerotic lesions and endarteriitis lower extremities.

2. Classification of chronic lower limb ischemia by Fontane.

3. Classification of atherosclerotic lesions at the level of occlusion (AA Shalimov).

4. Definition, clinical characteristics and treatment methods Leriche syndrome.

5. Indications and contraindications for surgical treatment of obliterative atherosclerosis endarteriitis and lower limb arteries.
6. Methods of the arterial system.
7. The differential diagnosis of obliterating atherosclerosis and obliterating endarteriitis arteries of the lower extremities.
8. Rehabilitation of patients who had undergone reconstructive surgery on the aorta and major arteries at the atherosclerotic lesions.
9. Methods to reduce the concentration of cholesterol and lipoproteins in blood plasma (pharmacological, instrumental).
10. The aetiology of abdominal ischemic syndrome.
11. Classification of abdominal ischemic syndrome ..
12. Clinical characteristics of abdominal ischemic syndrome.
13. The differential diagnosis of abdominal ischemic syndrome.
14. Surgical tactics in abdominal ischemic syndrome.
15. Complications of abdominal ischemic syndrome.
16. Features of the postoperative period after surgery for abdominal ischemic syndrome.

The student should be able to:

1. To assess the aorto-Arteriogram.
2. Apply thrombolytic drugs, and anticoagulants.
3. Appoint conservative therapy in obliterating atherosclerosis and occlusive disease of the lower extremities.
4. Determine the ripple in the typical spots on the major arteries of the lower extremities.

5. Rate this result is coagulation.

The answers to tests and situational tasks:

1 - obliterating atherosclerosis of lower extremities, Leriche, chronic arterial insufficiency II B degree.;

2 - obliterating endarteriitis lower extremities, chronic arterial insufficiency II A degree.

3 - obliterating atherosclerosis of lower limb arteries. Bilateral Leriche. chronic arterial insufficiency II degree.

4 - C

5 - D

6 - D

7 - A

Information:

Basic:

1. Jack L. Cronenwett, Wayne Johnston Rutherford's Vascular Surgery. 7th Revised edition. - Elsevier -Health Sciences Division. – 2010.
2. Alun H. Davies and Colleen M. Brophy (Eds) Vascular Surgery. – Springer. - 2006.
3. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
4. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – 2Т.1.
5. Hospital surgery / Edited by L. Kovalchuk, V. Sayenko, G. Knysov, M. Nychytailo . – Ternopil: Ukrmedknyga, 2004.
6. [Materials for practical classes](#)

Additional:

1. К. Заринш, Б. Гевертс. Атлас по сосудистой хирургии. – Москва 2009. – Гэотар-

Медиа

Guidelines made

ass. prof. Kostiv S. Ya.

Обсуждено на заседании кафедры

" 11" января 2013 р. протокол № 9

Methodical instruction for students 5-year

Methodical instruction for lesson № 11

TOPIC: Varicose veins of lower extremities. Postthrombotic syndrome.

Lymphedema.

Practical - 6 hours

Topics: 1. Varicose veins of lower extremities.

2. Postthrombotic syndrome.

3. Lymphedema.

Purpose: To be able to diagnose varicose disease according to stage of disease, clinical features, to substantiate the diagnosis, methods of conservative and surgical treatment. Teach diagnosis, differential diagnosis, research methods, the choice of treatment, choice of surgical method of reconstruction of venous thrombosis, postthrombotic syndrome, and lymphedema.

Professional oriented students:

Varicose disease is one of the most common surgical disease, which occurs in 12-17% of the population. It is more common in middle age, and its complication as venous ulcers, chronic dermatitis, thrombophlebitis, bleeding from varicose veins often leads to lower efficiency, as is sometimes the cause of disability.

Thrombosis of the venous system occur in 27,4-41,0% of all cases of pathology of the venous system. Thrombosis of the venous system is accompanied by a high level of complications, which lead to postthrombotic syndrome, and cause pulmonary embolism. Postthrombotic syndrome is diagnosed in 0,3-0,4% of cases as a result of delayed or full treatment of acute deep vein thrombosis or thrombosis of the inferior vena cava.

Correction of metastatic spread remains a pressing problem of modern surgery. In

connection with the development and improvement of special methods of research of the lymphatic system, the introduction of the practice of microsurgical techniques, new directions in the treatment of lymphedema - performing operations directly on the lymphatic system.

Methodology for practical work

The practical part of the session 9.00 - 12.00

Algorithm for communication of students with patients with any pathology, which is seen on the subject (communication skills):

1. Greet and identify himself.
2. The face should be welcoming smile - this allows you to establish a trust relationship with the patient.
3. A patient in a pleasant form should explain the purpose of the visit, the subject and the duration of the conversation and get his consent.
4. If a patient only comes to the hospital to conduct correctly, and quiet conversation with his relatives, which together with the doctor to inform them about the previous diagnosis, hospitalization, for some surveys, which are scheduled to perform in the future.
5. Before the physical examination survey methods to explain what the patient survey will be carried out, indicate some discomfort and discomfort that may feel the patient at the time of the survey, noting bound by the survey in the diagnosis of this disease and to obtain his consent.
6. If you need transportation to the place of examination (visual room, X-ray, ultrasound study) to explain its need for the patient.
7. Prepare for the survey (for data pathologies meant inspection, palpation limb aortoarteriografiya, ultrasound vascular feet, Doppler) - wash your hands with warm water, put on signets, prepare tools for the performance of other diagnostic manipulation.
8. To hold something or other planned medical examination or manipulation.
9. Together with the attending physician in the correct and easy to understand format patient to explain the results of either survey.
10. Involve family and patient to talk in simple terms to explain to them the results of

the survey data and in the presence of previous surveys to compare their results, be sure to ascertaining whether they understand your explanations.

11. Must be only in the presence physician to substantiate the feasibility of surgical intervention for the treatment of this disease to supervise the patient.

12. Following surgical treatment only in the presence of the attending physician and with his consent should communicate the results of surgical intervention the patient and his relatives as well as the possibility of this or early or late postoperative complications.

13. Under the conditions of examination of patients in the postoperative period should be explained to the patient as true to perform hygiene procedures and the like.

14. In polite to get consent from the patient to participate in the ligation.

15. Together with your doctor to explain to the patient, and when the needs and next of kin of those or other actions regarding manipulations that have been implemented or planned to run in the future as well as the tactics of the subsequent treatment.

16. Finish the conversation should always be the wish of the patient the most rapid recovery.

Work 1

1. Collection of complaints, history, examination of the patient.
2. Identification of clinical symptoms and instrumental.
3. Grouping symptoms syndromes.
4. Determination of the leading syndrome.
5. Interpretation of laboratory - instrumental data
6. Rationale preliminary diagnosis
7. Differential diagnosis.
8. Formulation of clinical diagnosis.
9. Appointment of differentiated treatment programs according to the clinical protocol

Work 2. Browsing the Internet, reading room with cathedral library topical

literature.

Break - 12.00-12.30

Seminar discussion of theoretical issues - 12.30-14.00

Theme № 1 Varicose veins of lower extremities.

1. Etiology and pathogenesis of varicose veins.
2. Clinical characteristics of varicose veins.
3. Classification of varicose veins.
4. Methods and indications for phlebography.
5. Functional Tests to determine the state of valve surface, communicative and deep veins of the lower extremities.
6. Indications and contraindications for surgical treatment of varicose veins of lower extremities.
7. Sequence of operations saphenectomy.
8. Causes of recurrence of varicose veins of the lower extremities after saphenectomy, methods of prevention.
9. Repeated operations in case of recurrent varicose veins of lower extremities.

Theme № 2 Postthrombotic syndrome

1. Causes of postthrombotic syndrome.
2. Classification of postthrombotic syndrome
3. Clinic postthrombotic syndrome
4. Diagnosis of postthrombotic syndrome
5. Differential diagnosis of postthrombotic syndrome
6. Kosevativnoe treatment postthrombotic syndrome

7. Surgical treatment of postthrombotic syndrome
8. Rehabilitation of patients with postthrombotic syndrome

Theme № 3 Lymphedema.

1. Etiology and pathogenesis of lymphedema.
2. Clinical characteristics of lymphedema.
3. Classification of lymphedema.
4. Diagnosis: lymphography.
5. Methods of conservative treatment.
6. Indications for surgical treatment.
7. Methods of surgical treatment for lymphedema.

Break - 14.00-14.15

Hour self-study students - 14.15-15.00

- Tutorial test licensing examinations «Step 2»;
- Assessment of students who have not passed on the eve of the test control system «Moodle»;
- Letting students practical skills in the corresponding entry in matrikulyar book.

Sample test items and situational challenges.

1. A patient admitted to hospital with a diagnosis: Varicose veins the left lower extremity. At the examination revealed only varicose saphenous veins of legs without edema. What stage of varicose veins is in a patient?
2. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb,

chronic venous insufficiency I degree. What kind of treatment the patient is the best?

3. How much blood are flows through the superficial veins?

- a. 10-15%
- b. 5%
- c. 30-40%
- d. 70-80%
- e. 90%

4. Where are falls the great saphenous vein?

- A. Femoral vein
- B. Calf veins
- C. Popliteal vein
- D. External iliac vein
- E. Vena cava inferior

5. Where are runs a small subcutaneous vein?

- A. Popliteal vein
- B. Vena cava inferior
- C. External iliac vein
- D. Thigh vein
- E. Calf veins

Source of knowledge and skills

The student must know:

1. The aetiology and pathogenesis of varicose disease.
2. Clinical characteristics of venous insufficiency.
3. Methods of examination of the venous system of lower limbs.
4. Functional Tests to determine the state of valve surface, communicative and deep

veins of the lower extremities.

5. Indications and contraindications for surgical treatment of varicose veins of lower extremities.

6. Sequence of operations saphenectomy.

7. Causes of recurrence of varicose veins of the lower extremities after saphenectomy, methods of prevention.

8. The etiology, pathogenesis, conditions postthrombotic syndrome.

9. Classification of postthrombotic syndrome.

10. Clinical manifestations of postthrombotic syndrome.

11. Indications and contraindications for surgical treatment of postthrombotic syndrome.

12. Indications and contraindications for surgical treatment in postthrombotic syndrome.

14. Clinical manifestations of postthrombotic syndrome.

15. Clinical manifestations of postthrombotic syndrome.

16. Anticoagulants, thrombolytic, fibrinolytic drugs.

17. Aetiology, pathogenesis lymphedema.

18. Classification of lymphedema.

19. Clinical manifestations of lymphedema of the lower extremities.

20. Methods of studying the lymphatic system.

21. Indications and methods for performing lymphography.

22. Indications and contraindications for surgical treatment of lymphedema.

23. Indications and methods of conservative treatment of lymphedema.

24. Differential diagnosis limfedematic limbs

The student should be able to:

1. Carry out functional tests to determine the status of valves surface, communicative and deep veins of the lower extremities.
2. Rate dopplerometry.
3. To evaluate clotting system.
4. To evaluate anticoagulant system.
5. Rate venogram.
6. Sample Rate Homans, Levenberg, Moses.
7. Rate limphogram.
8. Conduct a differential diagnosis lymphostasis with vascular pathology of the lower extremities.
9. Appoint conservative treatment lymphostasis.

The answers to tests and situational tasks:

- 1 - I
- 2 - Elastic compression
- 3 - A
- 4 - A
- 5 - A

Information:

Basic:

1. Jack L. Cronenwett, Wayne Johnston Rutherford's Vascular Surgery. 7th Revised edition. - Elsevier -Health Sciences Division. – 2010.
2. Alun H. Davies and Colleen M. Brophy (Eds) Vascular Surgery. – Springer. - 2006.
3. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
4. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – 2Т.1.
5. Hospital surgery / Edited by L. Kovalchuk, V. Sayenko, G. Knysov, M. Nychytailo . – Ternopil: Ukrmedknyga, 2004.
6. Materials for practical classes

Additional:

1. К. Заринш, Б. Гевертс. Атлас по сосудистой хирургии. – Москва 2009. – Гэотар-Медиа

Guidelines made

ass.pof. Kostiv S.Ya.

Обсуждено на заседании кафедры

" 11" января 2013 р. протокол № 9

Methodological instruction for students 5-year

Methodical instruction for lesson № 12

Topic: Thrombosis of main veins.

Practical - 6 hours

Topics: 1. Deep vein thrombosis in the inferior vena cava.

2. Deep vein thrombosis in the superior vena cava.

3. Thrombosis of superficial veins.

Purpose: To be able to diagnose, to know the clinic vein thrombosis, depending on the causes, clinical features of disease, and to justify the conservative and surgical treatment

Professional oriented students:

Venous thrombosis - an acute disease characterized by the formation of a blood clot in a vein with more or less pronounced inflammatory process and a violation of the blood flow. The presence of inflammatory component in the area of thrombosis determine the other name of this disease - thrombophlebitis.

This complication frequently encountered in the postoperative period, averaging 27-30%. The importance of prevention, early diagnosis and treatment phlebemphraxis due to the fact that 2-10% of patients developed venous thrombosis is threatening complications such as pulmonary embolism (PE), with 0,26% of the cases it becomes a cause of death. Pulmonary embolism and deep vein thrombosis as a state with common pathogenetic roots, unite the concept of venous thromboembolism (embolic complications). The number of patients with a feasibility study of the main disease is increasing every year.

Methodology for practical work

The practical part of the session 9.00 - 12.00

Algorithm for communication of students with patients with any pathology, which is seen on the subject (communication skills):

1. Greet and identify himself.
2. The face should be welcoming smile - this allows you to establish a trust relationship with the patient.
3. A patient in a pleasant form should explain the purpose of the visit, the subject and the duration of the conversation and get his consent.
4. If a patient only comes to the hospital to conduct correctly, and quiet conversation with his relatives, which together with the doctor to inform them about the previous diagnosis, hospitalization, for some surveys, which are scheduled to perform in the future.
5. Before the physical examination survey methods to explain what the patient survey will be carried out, indicate some discomfort and discomfort that may feel the patient at the time of the survey, noting bound by the survey in the diagnosis of this disease and to obtain his consent.
6. If you need transportation to the place of examination (visual room, X-ray, ultrasound study) to explain its need for the patient.
7. Prepare for the survey (for data pathologies meant inspection, palpation limb aortoarteriografiya, ultrasound vascular feet, Doppler) - wash your hands with warm water, put on signets, prepare tools for the performance of other diagnostic manipulation.
8. To hold something or other planned medical examination or manipulation.
9. Together with the attending physician in the correct and easy to understand format patient to explain the results of either survey.
10. Involve family and patient to talk in simple terms to explain to them the results of the survey data and in the presence of previous surveys to compare their results, be sure to ascertaining whether they understand your explanations.
11. Must be only in the presence physician to substantiate the feasibility of surgical intervention for the treatment of this disease to supervise the patient.

12. Following surgical treatment only in the presence of the attending physician and with his consent should communicate the results of surgical intervention the patient and his relatives as well as the possibility of this or early or late postoperative complications.

13. Under the conditions of examination of patients in the postoperative period should be explained to the patient as true to perform hygiene procedures and the like.

14. In polite to get consent from the patient to participate in the ligation.

15. Together with your doctor to explain to the patient, and when the needs and next of kin of those or other actions regarding manipulations that have been implemented or planned to run in the future as well as the tactics of the subsequent treatment.

16. Finish the conversation should always be the wish of the patient the most rapid recovery.

Work 1

1. Collection of complaints, history, examination of the patient.
2. Identification of clinical symptoms and instrumental.
3. Grouping symptoms syndromes.
4. Determination of the leading syndrome.
5. Interpretation of laboratory - instrumental data
6. Rationale preliminary diagnosis
7. Differential diagnosis.
8. Formulation of clinical diagnosis.
9. Appointment of differentiated treatment programs according to the clinical protocol

Work 2. Browsing the Internet, reading room with cathedral library topical literature.

Program self-students

1. Anatomical functional features of the veins of the upper and lower extremities.

2. The etiology, pathogenesis and classification of thrombosis.
3. Factors convolution and protivosvertochnoy systems.
4. The mechanism of thrombosis

Break - 12.00-12.30

Seminar discussion of theoretical issues - 12.30-14.00

Theme № 1 Deep vein thrombosis in the vena cava inferior.

1. Etiology of deep vein thrombosis in the vena cava inferior
2. Clinical characteristics of deep vein thrombosis in the vena cava inferior.
3. Classification of deep vein thrombosis in the vena cava inferior
4. Methods and indications for phlebography.
5. Diagnosis of deep vein thrombosis in the vena cava inferior
6. Indications to surgical treatment of deep vein thrombosis in the vena cava inferior
7. Contraindications to surgical treatment of deep vein thrombosis in the vena cava inferior.
8. Conservative treatment of deep vein thrombosis in the vena cava inferior.
9. Surgical treatment of deep vein thrombosis in the vena cava inferior

Theme № 2 Deep vein thrombosis in the vena cava superior.

1. Etiology of deep vein thrombosis in the vena cava superior
2. Clinical characteristics of deep vein thrombosis in the vena cava superior.
3. Classification of deep vein thrombosis in the vena cava superior
4. Methods and indications for phlebography.
5. Diagnosis of deep vein thrombosis in the vena cava superior

6. Indications to surgical treatment of deep vein thrombosis in the vena cava superior
7. Contraindications to surgical treatment of deep vein thrombosis in the vena cava superior.
8. Conservative treatment of deep vein thrombosis in the vena cava superior.
9. Surgical treatment of deep vein thrombosis in the vena cava superior

Theme № 3 Thrombosis of superficial veins.

1. The etiology of thrombosis of superficial veins
2. The clinical characteristics of thrombosis of superficial veins.
3. Classification of thrombosis of superficial veins
4. Methods and indications for ultrasound.
5. Diagnosis of thrombosis of superficial veins
6. Indications to surgical treatment of thrombosis of superficial veins
7. Contraindications to surgical treatment of superficial venous thrombosis.
8. Conservative treatment of superficial venous thrombosis.
9. Surgical treatment of thrombosis of superficial veins

Break - 14.00-14.15

Hour self-study students - 14.15-15.00

- Tutorial test licensing examinations «Step 2»;
- Assessment of students who have not passed on the eve of the test control system «Moodle»;
- Letting students practical skills in the corresponding entry in matrikulyar book.

Sample test items and situational challenges.

1. The patient complains on burning pain in the limbs and the presence of painful dense strand of progress varicose veins in the leg. What is the most likely diagnosis in a patient?

2. The patient complains of burning pain in the limbs and the presence of painful dense strand of progress varicose veins in the leg with the transition to the lower third of the thigh. What is the most likely diagnosis in a patient?

3. Edema of lower limbs characteristic:

- A. Thrombosis of the inferior vena cava
- B. Leriche
- C. Aortic aneurysm
- D. Paget's Syndrome-Schröter
- E. Thrombosis of superior vena cava

4. Violation of the sensitivity characteristic:

- A. Artery Embolism
- B. Varicose
- C. Deep vein thrombosis
- D. Subcutaneous thrombophlebitis
- E. Lymphedema

5. Paresis of the limbs is characteristic:

- A. A. Atherothrombosis
- B. B. Varicose
- C. C. DVT
- D. D. Subcutaneous thrombophlebitis
- E. E. Lymphedema

Source of knowledge and skills

The student must know:

1. The etiology, pathogenesis, conditions of thrombosis of the venous system.

- 2 Classification of thrombosis of the venous system.
3. Clinical manifestations of acute thrombophlebitis of subcutaneous veins of the lower extremities.
4. Indications and contraindications for surgical treatment of acute thrombophlebitis of subcutaneous veins of the lower extremities.
5. Methods of operating the elimination of acute thrombophlebitis of subcutaneous veins of the lower extremities.
6. Indications and contraindications for conservative treatment of acute thrombophlebitis of subcutaneous veins of the lower extremities.
7. Clinical manifestations of venous thrombosis shins.
8. Clinical manifestations of acute ilio-femoral segment.
9. Clinical manifestations of thrombosis of the inferior vena cava.
10. Acute thrombosis of the hepatic veins - syndrome Budd-Hiari.
11. Clinical manifestations of the syndrome Pedzhetta-Shrettera.
12. Clinical manifestations of blue flegmazi, treatment, prognosis.
13. Anticoagulants, thrombolytic, fibrinolytic drugs.

The student should be able to:

1. Carry out functional tests to determine the status of valves surface, kommunikantnyh and deep veins of the lower extremities.
2. Rate ultrasound.
3. To evaluate clotting system.
4. To evaluate anticoagulant system.
5. Rate venogram.

6. Appoint conservative treatment in Thrombosis

The answers to tests and situational tasks:

1 - Thrombophlebitis of subcutaneous veins.

2 - Ascending thrombophlebitis of subcutaneous veins

3 - A

4 – A

5 - A

Information:

Basic:

1. Jack L. Cronenwett, Wayne Johnston Rutherford's Vascular Surgery. 7th Revised edition. - Elsevier -Health Sciences Division. – 2010.
2. Alun H. Davies and Colleen M. Brophy (Eds) Vascular Surgery. – Springer. - 2006.
3. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
4. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
5. Hospital surgery / Edited by L. Kovalchuk, V. Sayenko, G. Knysov, M. Nychytailo . – Ternopil: Ukrmedknyga, 2004.
6. [Materials for practical classes](#)

Additional:

1. К. Заринш, Б. Гевертс. Атлас по сосудистой хирургии. – Москва 2009. – Гэотар-Медиа

Обсуждено на заседании кафедры

" 11" января 2013 р. протокол № 9

Methodical instruction for students V- years

(Vascular Surgery)

Lesson № 10 (PRACTICAL - 6 hours)

THEME: Injuries vessels.

Topics: 1. Trauma arteries

2. Trauma veins

3. Combined vascular injury

Purpose: To be able to diagnose, to know the clinic injuries limb vessels, depending on the causes, clinical features of disease, and to justify the conservative and surgical treatment

Professional oriented students:

Until recently, injury and damage to blood vessels were considered pathological, specific wartime. Nevertheless, many domestic and foreign authors have convincing evidence of a significant increase in the number of injuries of major blood vessels in time of peace, which in the overall structure of injury reach 2%.

The rapid development of industry and technology, agricultural mechanization, household contribute to the high level of injuries. This situation increases the importance of emergency angiosurgery in addressing these important social objectives, as a reduction of disability and reduce the time not disability, early medical and vocational rehabilitation of victims.

Further improvement in the quality of treatment of injuries of major vessels is dependent on improvements in diagnostic techniques, the introduction of new diagnostic and medical equipment, developing operational methods, the introduction of modern science, pharmacology, rehabilitation, rational organization of the Vascular Service.

In peacetime, the share of damage to major vessels of the upper extremities account for 32,3%, lower extremities - 58,8%.

Methodology for practical work

The practical part of the session 9.00 - 12.00

Algorithm for communication of students with patients with any pathology, which is seen on the subject (communication skills):

1. Greet and identify himself.
2. The face should be welcoming smile - this allows you to establish a trust relationship with the patient.
3. A patient in a pleasant form should explain the purpose of the visit, the subject and the duration of the conversation and get his consent.
4. If a patient only comes to the hospital to conduct correctly, and quiet conversation with his relatives, which together with the doctor to inform them about the previous diagnosis, hospitalization, for some surveys, which are scheduled to perform in the future.
5. Before the physical examination survey methods to explain what the patient survey will be carried out, indicate some discomfort and discomfort that may feel the patient at the time of the survey, noting bound by the survey in the diagnosis of this disease and to obtain his consent.
6. If you need transportation to the place of examination (visual room, X-ray, ultrasound study) to explain its need for the patient.
7. Prepare for the survey (for data pathologies meant inspection, palpation limb aortoarteriograffiya, ultrasound vascular leg doplerograffiya) - wash your hands with warm water, put on signets, prepare tools for the performance of other diagnostic manipulation.
8. To hold something or other planned medical examination or manipulation.
9. Together with the attending physician in the correct and easy to understand format patient to explain the results of either survey.
10. Involve family and patient to talk in simple terms to explain to them the results of the survey data and in the presence of previous surveys to compare their results, be sure to

ascertaining whether they understand your explanations.

11. Required only in the presence of the attending physician to substantiate the feasibility of surgical intervention for the treatment of this pathology in charge of the patient.

12. Following surgical treatment only in the presence of the attending physician and with his consent should communicate the results of surgical intervention the patient and his relatives as well as the possibility of this or early or late postoperative complications.

13. Under the conditions of examination of patients in the postoperative period should be explained to the patient as true to perform hygiene procedures and the like.

14. In polite to get consent from the patient to participate in the ligation.

15. Together with your doctor to explain to the patient, and when the needs and the immediate family, those or other actions regarding manipulations that have been implemented or planned to run in the future as well as the tactics of the subsequent treatment.

16. Finish the conversation should always be the wish of the patient the most rapid recovery.

Work 1

1. Collection of complaints, history, examination of the patient.
2. Identification of clinical symptoms and instrumental.
3. Grouping symptoms syndromes.
4. Determination of the leading syndrome.
5. Interpretation of laboratory - instrumental data
6. Rationale preliminary diagnosis
7. Differential diagnosis.
8. Formulation of clinical diagnosis.
9. Appointment of differentiated treatment programs according to the clinical protocol

Work 2. Browsing the Internet, reading room with cathedral library topical literature.

Program self-students

1. Anatomical functional characteristics of arteries and veins of the lower extremities
2. Anatomical functional characteristics of arteries and veins of upper extremities
3. Etiology of vascular injury
4. Pathogenesis of vascular injury
5. Classification of vascular injury
6. Clinic injuries arteries
7. The clinic combined injuries
8. Diagnosis of vascular injuries
7. Temporary stop bleeding
8. Final stop bleeding
9. Factors convolution and coagulation systems
10. Postoperative management of patients with vascular injury

Break - 12.00-12.30

Seminar discussion of theoretical issues - 12.30-14.00

Theme № 1 Trauma arteries

1. Causes of injury of the arteries.
2. Clinical characteristics of injuries of the arteries.
3. Classification of injuries of the arteries.
4. Differential diagnosis of arterial injury.
5. Surgical tactics in injuries of the arteries
6. Methods of operative treatment of injuries of the arteries.
7. Features of the postoperative period after surgery on the injured arteries.

8. Indications for conservative treatment, drugs that are used.
9. Anticoagulants, fibrinolytic thrombolytic drugs.
10. Control methods for convolution system, their characteristics.

Theme № 2 Trauma veins

1. Causes of injury veins.
2. Clinical characteristics of injured veins.
3. Classification of injuries veins.
4. Differential diagnosis of venous injury.
5. Surgical tactics in injuries veins
6. Methods of operative treatment of injured veins.
7. Features of the postoperative period after surgery on the injured veins.
8. Indications for conservative treatment, drugs that are used.
9. Anticoagulants, fibrinolytic thrombolytic drugs.
10. Control methods for convolution system, their characteristics.

Theme № 3 Combined vascular injury

1. Causes of combined injuries of the vessels.
2. Clinical characteristics of combined injuries of the vessels.
3. Classification of vascular injury.
4. Differential Diagnosis of combined injuries of the vessels.
5. Surgical tactics in combined injuries of vessels
6. Methods of surgical treatment of combined injuries of the vessels.

7. Features of the postoperative period after surgical intervention for combined injuries of the vessels.
8. Indications for conservative treatment, drugs that are used.
9. Anticoagulants, fibrinolytic thrombolytic drugs.
10. Control methods for convolution system, their characteristics.

Break - 14.00-14.15

Hour self-study students - 14.15-15.00

- Tutorial test licensing examinations «Step 2»;
- Assessment of students who have not passed on the eve of the test control system «Moodle»;
- Letting students practical skills in the corresponding entry in matrikulyar book.

The seminars discussion of theoretical issues

Sample test items and situational problems.

1. A patient admitted to hospital with a gunshot wound in the lower groin ligament. Injury what vessel should be suspected?
2. A patient admitted to hospital with a gunshot wound in the popliteal fossa. Injury what vessel should be suspected?
 - A. Popliteal artery
 - B. Common femoral artery
 - C. Common iliac
 - D. Aorta
 - E. Internal iliac artery
3. Where, anatomically, is the most dangerous part brachial artery ligation:
 - A. No right answer
 - B. Distal separation of the deep artery of arm
 - C. In the middle
 - D. In the lower third of
 - E. Near the ulnar artery
4. The vessel should be bind:

- A. Near the injury
 - B. Distal from the injury site
 - C. On the middle of damage
 - D. It does not matter
 - E. In place of separation of the artery
5. Complications the stop bleeding by ligation of the vessel:
- A. Rebleeding
 - B. Ischemic contracture of the limbs
 - C. Trophic ulcer
 - D. The complications do not develop
 - E. Paresis of the limbs

The student should be able to:

1. To assess the aorto-arteriogramu
2. Rate coagulogram.
3. Evaluate the results ultrasound research
4. Apply thrombolytic drugs and anticoagulants.
5. Appoint conservative therapy for vascular injury
6. Determine the ripple in the typical spots on the major arteries of the lower extremities.
7. Apply methods to temporarily stop bleeding.
8. Appoint conservative therapy in acute blood loss.

The answers to tests and situational tasks:

1- Common femoral vein

2 - Popliteal artery

3 – A

4 – A

5 - A

Sources of information:

Information:

Basic:

1. Jack L. Cronenwett, Wayne Johnston Rutherford's Vascular Surgery. 7th Revised edition. - Elsevier -Health Sciences Division. – 2010.
2. Alun H. Davies and Colleen M. Brophy (Eds) Vascular Surgery. – Springer. - 2006.
3. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
4. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – 2Т.1.
5. Hospital surgery / Edited by L. Kovalchuk, V. Sayenko, G. Knysov, M. Nychytailo . – Ternopil: Ukrmedknyga, 2004.
6. [Materials for practical classes](#)

Additional:

1. К. Заринш, Б. Гевертс. Атлас по сосудистой хирургии. – Москва 2009. – Гэотар-Медиа

Guidelines made

ass. prof. Kostiv S.Ya.

Обсуждено на заседании кафедры

" 11" января 2013 р. протокол № 9

METHODICAL INSTRUCTIONS FOR STUDENTS V YEARS

Lesson № 13

Topic: Pulmonary embolism.

Practical - 6 hours

Purpose: To be able to diagnose pulmonary embolism know the clinic, the clinical features of disease and to justify the conservative and surgical treatment

Professional oriented students:

VTE is a major cause of sudden death in people. It is difficult to 0,1-0,3% of surgical operations and is responsible for 20-40% of fatal cases. However, PE is not less frequently observed in medical patients, because of the prevalence of risk factors for this pathology.

Methodology for practical work

The practical part of the session 9.00 - 12.00

Algorithm for communication of students with patients with any pathology, which is seen on the subject (communication skills):

1. Greet and identify himself.
2. The face should be welcoming smile - this allows you to establish a trust relationship with the patient.
3. A patient in a pleasant form should explain the purpose of the visit, the subject and the duration of the conversation and get his consent.
4. If a patient only comes to the hospital to conduct correctly, and quiet conversation with his relatives, which together with the doctor to inform them about the previous diagnosis,

hospitalization, for some surveys, which are scheduled to perform in the future.

5. Before the physical examination survey methods to explain what the patient survey will be carried out, indicate some discomfort and discomfort that may feel the patient at the time of the survey, noting bound by the survey in the diagnosis of this disease and to obtain his consent.

6. If you need transportation to the place of examination (visual room, X-ray, ultrasound study) to explain its need for the patient.

7. Prepare for the survey (for data pathologies meant inspection, palpation limb aortoarteriograffiya, ultrasound vascular leg doplerograffiya) - wash your hands with warm water, put on signets, prepare tools for the performance of other diagnostic manipulation.

8. To hold something or other planned medical examination or manipulation.

9. Together with the attending physician in the correct and easy to understand format patient to explain the results of either survey.

10. Involve family and patient to talk in simple terms to explain to them the results of the survey data and in the presence of previous surveys to compare their results, be sure to ascertaining whether they understand your explanations.

11. Required only in the presence of the attending physician to substantiate the feasibility of surgical intervention for the treatment of this pathology in charge of the patient.

12. Following surgical treatment only in the presence of the attending physician and with his consent should communicate the results of surgical intervention the patient and his relatives as well as the possibility of this or early or late postoperative complications.

13. Under the conditions of examination of patients in the postoperative period should be explained to the patient as true to perform hygiene procedures and the like.

14. In polite to get consent from the patient to participate in the ligation.

15. Together with your doctor to explain to the patient, and when the needs and next of kin of those or other actions regarding manipulations that have been implemented or planned to run in the future as well as the tactics of the subsequent treatment.

16. Finish the conversation should always be the wish of the patient the most rapid

recovery.

Work 1

1. Collection of complaints, history, examination of the patient.
2. Identification of clinical symptoms and instrumental.
3. Grouping symptoms syndromes.
4. Determination of the leading syndrome.
5. Interpretation of laboratory - instrumental data
6. Rationale preliminary diagnosis
7. Differential diagnosis.
8. Formulation of clinical diagnosis.
9. Appointment of differentiated treatment programs according to the clinical protocol

Work 2. Browsing the Internet, reading room with cathedral library topical literature.

Program self-students

1. The anatomic and functional characteristics of veins of lower extremities and pelvis
2. Factors convolution and protivosvertochnoy systems.
3. The mechanism of thrombosis in the venous system.
4. The anatomic and functional features of pulmonary arteries
5. Principles of postoperative period

Break - 12.00-12.30

Seminar discussion of theoretical issues - 12.30-14.00

1. Terms of thrombosis in the venous system.
2. Indications for surgical and conservative treatment of venous thrombosis.

3. Principles of surgical and conservative treatment of venous thrombosis.
4. Classification of pulmonary embolism.
5. Peculiarities of hemodynamics of the right heart and pulmonary circulation.
6. Clinical manifestations of pulmonary embolism.
7. Clinical forms of pulmonary embolism (cardiac, pulmonary, cerebral).
8. ECG signs of pulmonary embolism.
9. Radiological signs of pulmonary embolism.
10. Contemporary pulmonary embolism diagnosis (determination of D-dimer, ultrasonography, angiopulmonografiya).
11. Differential diagnosis of pulmonary embolism with myocardial infarction.
12. Therapy of pulmonary embolism, the control of the convection of the blood system.
13. Indications and methods of surgical treatment of pulmonary embolism.
14. Indications and methods of endovascular interventions for pulmonary embolism.
15. Total prevention of pulmonary embolism.
16. Indications and contraindications to the use of vena filters.
17. Indications and methods of correction of pulmonary hypertension.

Break - 14.00-14.15

Hour self-study students - 14.15-15.00

- Tutorial test licensing examinations «Step 2»;
- Assessment of students who have not passed on the eve of the test control system «Moodle»;
- Letting students practical skills in the corresponding entry in matrikulyar book.

The workshops discuss theoretical issues

Sample test items and situational problems.

1. The patient admitted to the hospital with a diagnosis deep vein thrombosis left lower extremity. The patient suddenly began to worry shortness of breath, retrosternal pain. What complication can be suspected in a patient?
2. The patient admitted to the hospital with a diagnosis deep vein thrombosis left lower extremity. The patient suddenly began to worry shortness of breath, retrosternal pain. What method diagnosis should be used to refine the diagnosis?
3. What is the main danger the subcutaneous thrombophlebitis?
 - A. Pulmonary embolism
 - B. Venous insufficiency
 - C. Trophic ulcer
 - D. Gangrene fingers on the lower extremity
 - E. Paralysis
4. Palee skin is characteristic for:
 - A. Artery Embolism
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
5. Expressed cold extremities is characteristic for:
 - A. Atherothrombosis
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb

The student should be able to:

1. Rate coagulogram.
2. Rate ECG.
3. Apply thrombolytic drugs, and anticoagulants.
4. Appoint conservative therapy in the body.

The answers to tests and situational tasks:

- 1 - Pulmonary embolism
- 2 - ECG
- 3 - A.
- 4 – A
- 5 - A

Information:

Basic:

1. Jack L. Cronenwett, Wayne Johnston Rutherford's Vascular Surgery. 7th Revised edition. - Elsevier -Health Sciences Division. – 2010.
2. Alun H. Davies and Colleen M. Brophy (Eds) Vascular Surgery. – Springer. - 2006.
3. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – Т.1.
4. Э. Ашера Сосудистая хирургия по Хаймовичу. – Москва 2010. – Бином. – 2Т.1.
5. Hospital surgery / Edited by L. Kovalchuk, V. Sayenko, G. Knysov, M. Nychytailo . – Ternopil: Ukrmedknyga, 2004.
6. [Materials for practical classes](#)

Additional:

1. К. Заринш, Б. Гевертс. Атлас по сосудистой хирургии. – Москва 2009. – Гэотар-
Медиа

Guidelines made

ass. Kostiv S. Ya.

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" 11" января 2013 р. протокол № 9

MODULE 1. ABDOMINAL SURGERY

Text test questions

1. Acute appendicitis in the 1st phase is necessary to differentiate from:
 - A. * gastric ulcer
 - B. pancreatitis
 - C. cholecystitis
 - D. intestinal obstruction
 - E. strangulated hernia

2. After appendectomy the patient stands out of bed on:
 - A. * first day
 - B. second day
 - C. third day
 - D. fourth day
 - E. fifth day

3. After appendectomy to early postoperative complications belongs:
 - A. * peritonitis
 - B. intestinal fistula
 - C. ventral hernia
 - D. ligature fistula
 - E. colitis

4. After appendectomy to late postoperative complications belongs:
 - A. * intestinal fistula
 - B. appendicular infiltrate
 - C. pilephlebitis
 - D. peritonitis
 - E. colitis

5. Appendectomy after the treatment of appendicular infiltrate performed after:
 - A. * 2-4 months
 - B. 1-2 weeks
 - C. 3-4 weeks
 - D. 1-2 months
 - E. 3-5 days

6. Appendectomy, as a rule, is performed under such anaesthesia:
 - A. * intravenous anaesthesia
 - B. local anaesthesia
 - C. ether anaesthesia
 - D. conducting anaesthesia
 - E. endotracheal anaesthesia

7. Appendicular infiltrate appears after:
 - A. * 3-5 days
 - B. 1-2 days
 - C. 5-6 days
 - D. 7-8 days
 - E. 8-10 days

8. Appendicular infiltrate is treated:

- A. * conservative therapy, then surgery
 - B. only conservative therapy
 - C. puncture
 - D. drainage
 - E. only surgical treatment
9. Appendicular infiltrate is treated:
- A. * antibiotics, paranephral blockade, detoxication therapy
 - B. antiseptics, analgesia, antibiotics, anti-inflammatory therapy
 - C. antibiotics, diuretics, antispasmodic, anti-inflammatory therapy
 - D. analgesia, antibiotics, diuretics, anti-inflammatory therapy
 - E. anti-inflammatory drugs, paranephral blockade, detoxication therapy
10. Bartomier's sign is typical for:
- A. * acute appendicitis
 - B. acute cholecystitis
 - C. acute intestinal obstruction
 - D. food poisoning
 - E. acute pancreatitis
11. Black colour, fibrino-purulent fur, perforation are the signs of:
- A. * gangreno-perforative appendicitis
 - B. phlegmonous appendicitis
 - C. gangrenous appendicitis
 - D. catarrhal appendicitis
 - E. dystrophic appendicitis
12. Black colour, fibrino-purulent fur, pus in the lumen are the signs of:
- A. * gangrenous appendicitis
 - B. phlegmonous appendicitis
 - C. catarrhal appendicitis
 - D. gangreno-perforative appendicitis
 - E. dystrophic appendicitis
13. Blumberg's sign in Petit triangle is:
- A. * Gabay's sign
 - B. Rovsing's sign
 - C. Sitkovsky's sign
 - D. Obrastsow's sign
 - E. Kulenkampf's sign
14. Blumberg's sign is typical for:
- A. * acute appendicitis
 - B. acute thrombophlebitis
 - C. pneumothorax
 - D. food poisoning
 - E. bleeding ulcer
15. Causes of the appendicular infiltrate development:
- A. * late hospitalisation, misdiagnosed appendicitis
 - B. aggressive infection, impaired immunity
 - C. adhesions, increased immunity
 - D. peritonitis, abscessing
 - E. surgical trauma, infection

16. Characteristic changes in the general blood analysis in appendicitis:
- A. * neutrophil leucocytosis with deviation of the differential count to the left
 - B. neutrophil lymphocytosis with deviation of the differential count to the left
 - C. neutrophil eosinophilia with deviation of the differential count to the left
 - D. neutrophil leucocytosis with deviation of the differential count to the right
 - E. white cells neutrophilia with deviation of the differential count to the right
17. Chronic primary appendicitis - is the development of pathological changes in appendix after:
- A. * without the signs of acute appendicitis in anamnesis
 - B. acute appendicitis
 - C. appendicular infiltrate
 - D. appendicular abscess
 - E. pilephlebitis
18. Chronic residual appendicitis arises up after:
- A. * acute appendicitis
 - B. chronic appendicitis
 - C. colicks
 - D. recurrent appendicitis
 - E. primary chronic appendicitis
19. Chronic residual appendicitis arises up after:
- A. * appendicular infiltrate
 - B. chronic appendicitis
 - C. recurrent appendicitis
 - D. colicks
 - E. primary chronic appendicitis
20. Chronic residual appendicitis arises up after:
- A. * appendicular abscess
 - B. colicks
 - C. chronic appendicitis
 - D. recurrent appendicitis
 - E. primary chronic appendicitis
21. Conservative treatment of appendicular infiltrate is going on:
- A. * 1-2 weeks
 - B. 1 week
 - C. 3-4 weeks
 - D. 1-2 months
 - E. 2-4 months
22. Dunphy's sign is typical for:
- A. * acute appendicitis
 - B. acute cholecystitis
 - C. acute intestinal obstruction
 - D. food poisoning
 - E. acute pancreatitis
23. During appendectomy the most frequent complication is:
- A. * bleeding
 - B. infiltrate
 - C. leak of the sutures

- D. infecting
- E. peritonitis

24. Dyspeptic syndrome is characteristic for acute appendicitis in:

- A. * children
- B. females
- C. males
- D. pregnant
- E. elderly patients

25. Dyspeptic syndrome is characteristic for acute appendicitis in:

- A. * children
- B. females
- C. males
- D. pregnant
- E. elderly patients

26. For acute appendicitis is typical:

- A. * Kocher-Volkovitch's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

27. For acute appendicitis is typical:

- A. * Rovsing's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

28. For acute appendicitis is typical:

- A. * Sitkovsky's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

29. For acute appendicitis is typical:

- A. * Bartomier's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

30. For acute appendicitis is typical:

- A. * Dunphy's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

31. For acute appendicitis is typical:

- A. * Blumberg's sign

- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

32. For acute appendicitis is typical:

- A. * Voskresenky's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

33. For acute appendicitis is typical:

- A. * Rozdolsky's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

34. For acute appendicitis is typical:

- A. * Yaure-Rozanov's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

35. For acute appendicitis is typical:

- A. * Gabay's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

36. For acute appendicitis is typical:

- A. * Psoas sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

37. For acute appendicitis is typical:

- A. * Obrastsow's sign
- B. Ortner's sign
- C. Homans sign
- D. Sklyarov's sign
- E. Meyo-Robson sign

38. For acute appendicitis typical t° is:

- A. * 38° C
- B. 37° C
- C. subfebrile
- D. 37-39° C
- E. 38-40° C

39. For appendectomy the most suitable surgical access is:
- A. * Volkovich-Dyakonov
 - B. McBurney
 - C. Lenander
 - D. Sprengel
 - E. Kocher
40. For pelvic appendicitis is characteristic the sign:
- A. * Kulenkampf's sign
 - B. Yaure-Rozanov sign
 - C. Sitkovsky's sign
 - D. Obrastsov's sign
 - E. Voskresensky's sign
41. For retrocecal appendicitis is characteristic the sign:
- A. * Yaure-Rozanov sign
 - B. Sitkovsky's sign
 - C. Obrastsov's sign
 - D. Voskresensky's sign
 - E. Kulenkampf's sign
42. For retrocecal appendicitis is characteristic the sign:
- A. * Gabay's sign
 - B. Sitkovsky's sign
 - C. Obrastsov's sign
 - D. Voskresensky's sign
 - E. Kulenkampf's sign
43. For retroperitoneal appendicitis is characteristic the sign:
- A. * Pasternatsky's
 - B. Sitkovsky's
 - C. Yaure-Rozanov
 - D. Rovzing's
 - E. Koer's
44. For the left-side appendicitis is typical:
- A. * The painfulness of the left iliac region
 - B. Expressed pain in a right lumbar area
 - C. Flank tenderness in right lower quadrant
 - D. Clinic of irritation of pelvic organs
 - E. Painfulness of anterior rectal wall and posterior vaginal vault
45. For the pelvic appendicitis is typical:
- A. * Clinic of irritation of pelvic organs
 - B. The painfulness of the left iliac region
 - C. Expressed pain in a right lumbar area
 - D. Flank tenderness in right lower quadrant
 - E. Peritoneal signs
46. For the pelvic appendicitis is typical:
- A. * Painfulness of anterior rectal wall
 - B. The painfulness of the left iliac region
 - C. Expressed pain in a right lumbar area
 - D. Flank tenderness in right lower quadrant

E. Peritoneal signs

47. For the pelvic appendicitis is typical:

- A. * Painfulness of posterior vaginal vault
- B. The painfulness of the left iliac region
- C. Expressed pain in a right lumbar area
- D. Flank tenderness in right lower quadrant
- E. Peritoneal signs

48. For the perforation of appendix is not characteristic:

- A. * Decrease of body temperature
- B. Acute pain in a right iliac area, especially expressed after false improvement
- C. Tension of the abdominal wall at first in a right iliac area, and then spreading on other departments
- D. Increasing swelling of abdomen
- E. Leucocytosis

49. For the retrocaecal appendicitis is not typical:

- A. * Volkovcha-Kocher's sign
- B. delayed diagnostics
- C. late entrance of patients in the hospital
- D. frequent development of destructive forms
- E. weak expressed signs of peritoneal irritation

50. For the retrocecal appendicitis is typical:

- A. * Yaure-Rozanov sign
- B. Rovsing's sign
- C. Blumberg's sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

51. For the retrocecal appendicitis is typical:

- A. * Gabay's sign
- B. Rovsing's sign
- C. Blumberg's sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

52. For the retrocecal appendicitis is typical:

- A. * Pasternatsky's sign
- B. Rovsing's sign
- C. Blumberg's sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

53. For the retrocecal appendicitis is typical:

- A. * Psoas sign
- B. Rovsing's sign
- C. Blumberg's sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

54. For the retrocecal appendicitis is typical:

- A. * Expressed pain in a right lumbar area

- B. Flank tenderness in right lower quadrant
- C. The painfulness of the left iliac region
- D. Clinic of irritation of pelvic organs
- E. Painfulness of anterior rectal wall and posterior vaginal vault

55. For the retroperitoneal appendicitis is typical:

- A. * Flank tenderness in right lower quadrant
- B. Peritoneal signs
- C. The painfulness of the left iliac region
- D. Clinic of irritation of pelvic organs
- E. Painfulness of anterior rectal wall and posterior vaginal vault

56. For the simple appendicitis is typical:

- A. * Rovsing's sign
- B. Blumberg's sign
- C. Yaure-Rozanov sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

57. For the simple appendicitis is typical:

- A. * Sitkovsky's sign
- B. Blumberg's sign
- C. Yaure-Rozanov sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

58. For the simple appendicitis is typical:

- A. * Bartomier's sign
- B. Blumberg's sign
- C. Yaure-Rozanov sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

59. For the simple appendicitis is typical:

- A. * Dunphy's sign
- B. Blumberg's sign
- C. Yaure-Rozanov sign
- D. Voskresensky's sign
- E. Kulenkampf's sign

60. Gabay's sign is typical for:

- A. * acute appendicitis
- B. acute thrombophlebitis
- C. pneumothorax
- D. food poisoning
- E. bleeding ulcer

61. Hyperaemia, thickening, oedema of appendix are the signs of:

- A. * catarrhal appendicitis
- B. phlegmonous appendicitis
- C. gangrenous appendicitis
- D. gangreno-perforating appendicitis
- E. dystrophic appendicitis

62. Hyperemia, fibrino-purulent fur, pus the lumen are the signs of:
- A. * phlegmonous appendicitis
 - B. catarrhal appendicitis
 - C. gangrenous appendicitis
 - D. gangreno-perforative appendicitis
 - E. dystrophic appendicitis
63. In what location of appendix the rectal examination is the most informing?
- A. * Pelvic
 - B. Retrocaecal
 - C. Madian
 - D. Subhepatic
 - E. Retroperitoneal
64. Increased pain with coughing is:
- A. * Dunphy's sign
 - B. Rovsing's sign
 - C. Sitkovsky's sign
 - D. Obrastsov's sign
 - E. Kulenkampf's sign
65. Inexpressive abdominal pain in acute appendicitis is characteristic for:
- A. * elderly patients
 - B. children
 - C. females
 - D. males
 - E. pregnant
66. Inexpressive abdominal pain in acute appendicitis is characteristic for:
- A. * elderly patients
 - B. children
 - C. females
 - D. males
 - E. pregnant
67. Inexpressive leucocytosis in acute appendicitis is characteristic for:
- A. * elderly patients
 - B. females
 - C. children
 - D. pregnant
 - E. males
68. Inexpressive leucocytosis in acute appendicitis is characteristic for:
- A. * elderly patients
 - B. females
 - C. children
 - D. pregnant
 - E. males
69. Inexpressive muscular tension of anterior abdominal wall in acute appendicitis is characteristic for:
- A. * elderly patients
 - B. females
 - C. children

- D. pregnant
- E. males

70. Inexpressive muscular tension of anterior abdominal wall in acute appendicitis is characteristic for:

- A. * elderly patients
- B. females
- C. children
- D. pregnant
- E. males

71. Initially-gangrenous appendicitis differs from inflammatory-gangrenous form mostly developing in persons:

- A. * of elderly age
- B. children of early age
- C. pregnant in the first half of pregnancy
- D. pregnant in the second half of pregnancy
- E. with concomitant diseases

72. Kocher-Volkovitch's sign is typical for:

- A. * acute appendicitis
- B. acute cholecystitis
- C. acute intestinal obstruction
- D. food poisoning
- E. acute pancreatitis

73. Lymphoid hypoplasia determines the peculiarities of the clinical course of appendicitis in:

- A. * children
- B. elderly patients
- C. pregnant
- D. males
- E. females

74. Lymphoid hypoplasia determines the peculiarities of the clinical course of appendicitis in:

- A. * children
- B. elderly patients
- C. pregnant
- D. males
- E. females

75. Microhematuria is typical for such kind of appendicitis:

- A. * retroperitoneal
- B. retrocecal
- C. pelvic
- D. subhepatic
- E. left-side

76. Migration of pain to the right iliac area from epigastric is:

- A. * Kocher-Volkovitch's sign
- B. Rovsing's sign
- C. Sitkovsky's sign
- D. Obrastsov's sign
- E. Kulenkampf's sign

77. Modern method of appendectomy is:
- A. * laparoscopic
 - B. microlaparotomy
 - C. laparocentesis
 - D. laparotomy
 - E. ligation
78. Obrastsov's sign is typical for:
- A. * acute appendicitis
 - B. acute thrombophlebitis
 - C. pneumothorax
 - D. food poisoning
 - E. bleeding ulcer
79. On the line between the anterior-superior process of the iliac bone and umbilicus located the point:
- A. * McBurney's
 - B. Kalk's
 - C. Kehr's
 - D. Lenander's
 - E. Volkovich-Dyakonov
80. Only during the operation is possible the differential diagnostics of acute appendicitis with:
- A. * terminal ileitis
 - B. renal colic
 - C. acute pyelonephritis
 - D. acute paraproctitis
 - E. acute pancreatitis
81. Pain during digital examination of rectum - is:
- A. * Kulenkampf's sign
 - B. Obrastsov's sign
 - C. Voskresensky's sign
 - D. Kocher-Volkovitch's sign
 - E. Sitkovsky's sign
82. Pain during palpation in a lumbar region - is the sign:
- A. * Yaure-Rozanov sign
 - B. Sitkovsky's sign
 - C. Obrastsov's sign
 - D. Voskresensky's sign
 - E. Kulenkampf's sign
83. Pain during palpation in a lumbar region after taking away of the hand is the sign:
- A. * Gabay's sign
 - B. Sitkovsky's sign
 - C. Obrastsov's sign
 - D. Voskresensky's sign
 - E. Kulenkampf's sign
84. Pain during palpation in the Petit triangle - is the sign:
- A. * Yaure-Rozanov sign
 - B. Sitkovsky's sign
 - C. Obrastsov's sign

- D. Voskresensky's sign
 - E. Kulenkampf's sign
85. Pain during percussion by fingers of anterior abdominal wall - is:
- A. * Rozdolsky's sign
 - B. Obrastsow's sign
 - C. Sitkovsky's sign
 - D. Voskresensky's sign
 - E. Kulenkampf's sign
86. Pain during removing of the hand from abdominal wall after its pressing - is:
- A. * Shchotkin-Blumberg's sign
 - B. Rovsing's sign
 - C. Sitkovsky's sign
 - D. Obrastsow's sign
 - E. Kulenkampf's sign
87. Pain during sliding of hand on abdominal wall - is:
- A. * Voskresensky's sign
 - B. Sitkovsky's sign
 - C. Obrastsow's sign
 - D. Kocher-Volkovitch's sign
 - E. Bartomier-Mikhelson's sign
88. Pain in the iliac region during elevation of the leg - is:
- A. * Obrastsow's sign
 - B. Sitkovsky's sign
 - C. Kocher-Volkovitch's sign
 - D. Bartomier-Mikhelson's sign
 - E. Voskresensky's sign
89. Pain in the right iliac region by pushing of the left - is:
- A. * Rovsing's sign
 - B. Sitkovsky's sign
 - C. Obrastsow's sign
 - D. Voskresensky's sign
 - E. Kulenkampf's sign
90. Pain in the right iliac region during palpation of the iliac region on the left side - is:
- A. * Bartomier-Mikhelson's sign
 - B. Sitkovsky's sign
 - C. Obrastsow's sign
 - D. Voskresensky's sign
 - E. Kocher-Volkovitch's sign
91. Pain on extension of right thigh is:
- A. * Psoas sign
 - B. Rovsing's sign
 - C. Sitkovsky's sign
 - D. Obrastsow's sign
 - E. Kulenkampf's sign
92. Painfulness during palpation of Petit triangle is:
- A. * Yaure-Rozanov's sign

- B. Rovsing's sign
- C. Sitkovsky's sign
- D. Obrastsov's sign
- E. Kulenkampf's sign

93. Painfulness of posterior vaginal vault is typical for:

- A. * Pelvic appendicitis
- B. Retrocecal appendicitis
- C. Phlegmonous appendicitis
- D. Simple appendicitis
- E. Left-side appendicitis appendicitis

94. Psoas sign is typical for:

- A. * acute appendicitis
- B. acute thrombophlebitis
- C. pneumothorax
- D. food poisoning
- E. bleeding ulcer

95. Rapid destruction of the appendix in the course of acute appendicitis is characteristic for:

- A. * elderly patients
- B. children
- C. pregnant
- D. males
- E. females

96. Rapid destruction of the appendix in the course of acute appendicitis is characteristic for:

- A. * elderly patients
- B. children
- C. pregnant
- D. males
- E. females

97. Reduced reactivity of the organism influences on the peculiarities of the course of acute appendicitis in:

- A. * elderly patients
- B. females
- C. males
- D. pregnant
- E. children

98. Reduced reactivity of the organism influences on the peculiarities of the course of acute appendicitis in:

- A. * elderly patients
- B. females
- C. males
- D. pregnant
- E. children

99. Rovsing's sign is typical for:

- A. * acute appendicitis
- B. acute cholecystitis
- C. acute intestinal obstruction
- D. food poisoning

- E. acute pancreatitis
100. Rozdolsky's sign is typical for:
- A. * acute appendicitis
 - B. acute thrombophlebitis
 - C. pneumothorax
 - D. food poisoning
 - E. bleeding ulcer
101. Sitkovsky's sign is typical for:
- A. * acute appendicitis
 - B. acute cholecystitis
 - C. acute intestinal obstruction
 - D. food poisoning
 - E. acute pancreatitis
102. Strengthening of pain in right iliac region on the left side - is:
- A. * Sitkovsky's sign
 - B. Obrastsow's sign
 - C. Voskresensky's sign
 - D. Kocher-Volkovitch's sign
 - E. Bartomier-Mikhelson's sign
103. The absence of muscular tenderness is typical for:
- A. * Pelvic appendicitis
 - B. Retrocecal appendicitis
 - C. Phlegmonous appendicitis
 - D. Simple appendicitis
 - E. Left-side appendicitis appendicitis
104. The appendix ends its formation at the age of:
- A. * 7 years
 - B. 6 months
 - C. 1 year
 - D. 3 years
 - E. 3 months
105. The bailer form of appendix is characteristic for:
- A. * new-born
 - B. males
 - C. females
 - D. pregnant
 - E. elderly patients
106. The Bartomier's sign is typical for:
- A. * Simple appendicitis
 - B. Retrocecal appendicitis
 - C. Retroperitoneal appendicitis
 - D. Pelvic appendicitis
 - E. Left-side appendicitis
107. The Blumberg's sign is typical for:
- A. * Phlegmonous appendicitis
 - B. Simple appendicitis

- C. Retrocecal appendicitis
 - D. Retroperitoneal appendicitis
 - E. Pelvic appendicitis
108. The changes in clinical manifestation of acute appendicitis in pregnancy is characterized :
- A. * by the absence of signs of peritoneal irritation
 - B. by the presence of signs of peritoneal irritation
 - C. by the presence of expressed signs of peritoneal irritation
 - D. by displacement of the signs of peritoneal irritation
 - E. by the change of the character of signs of peritoneal irritation
109. The changes of clinical manifestations of acute appendicitis in pregnancy are caused by the displacement of appendix in relation to cecum:
- A. * upword
 - B. lateral
 - C. downword
 - D. medial
 - E. retroperitoneal
110. The changes of clinical manifestations of acute appendicitis in pregnancy are caused by:
- A. * distension of anterior abdominal wall by uterus
 - B. inflammation of uterus
 - C. irritation of anterior abdominal wall by uterus
 - D. compression of appendix by uterus
 - E. inflammation of the right ovarium
111. The changes of clinical manifestations of acute appendicitis in pregnancy are caused by:
- A. * absence of muscular tension of anterior abdominal wall
 - B. absence of tension of the uterus
 - C. presence of tension of the uterus
 - D. expressed muscular tension of anterior abdominal wall
 - E. presence of tension of peritoneum of anterior abdominal wall
112. The clinical manifestation of acute appendicitis does not relate to destructive changes in the appendix in:
- A. * elderly patients
 - B. children
 - C. females
 - D. males
 - E. pregnant
113. The clinical manifestation of acute appendicitis in pregnancy depends on:
- A. * the term of pregnancy
 - B. degree of inflammatory changes
 - C. the relation of appendix to peritoneum
 - D. the duration of appendicitis
 - E. the form of appendicitis
114. The clinical manifestation of appendicular infiltrate is:
- A. * swelling
 - B. the signs of peritoneal irritation
 - C. muscular tension
 - D. high temperature
 - E. leucocytosis

115. The clinical manifestations of acute appendicitis in pregnancy are characterised by the changes of:
- A. * localization of pain
 - B. severity of pain
 - C. irradiation of pain
 - D. duration of pain
 - E. character of pain
116. The clinical manifestations of acute appendicitis in the first trimester of the pregnancy are:
- A. * typical
 - B. atypical
 - C. expressed
 - D. unexpressed
 - E. absent
117. The clinical manifestations of acute appendicitis in the second trimester of the pregnancy are:
- A. * typical
 - B. atypical
 - C. expressed
 - D. unexpressed
 - E. absent
118. The clinical manifestations of acute appendicitis in the third trimester of the pregnancy are:
- A. * atypical
 - B. typical
 - C. expressed
 - D. unexpressed
 - E. absent
119. The conditions, which contribute to the formation of appendicular infiltrate include:
- A. * Phlegmonous changes of appendix
 - B. Chronic appendicitis
 - C. Meckel's diverticulum
 - D. Pylephlebitis
 - E. Perforation of appendix
120. The destructive changes in the appendix don't relate to the clinical manifestation of acute appendicitis in:
- A. * elderly patients
 - B. children
 - C. females
 - D. males
 - E. pregnant
121. The distinctive peculiarities of acute appendicitis in the second half of pregnancy are:
- A. * Weak express of pain syndrome, similar to the ligamentary tension of uterus
 - B. Absence of Volkovcha-Kocher's sign
 - C. Expressed signs of peritoneal irritation
 - D. The express local muscular tension in a right iliac area
 - E. Expressed of Obraztsov's sign
122. The Dunphy's sign is typical for:

- A. * Simple appendicitis
 - B. Retrocecal appendicitis
 - C. Retroperitoneal appendicitis
 - D. Pelvic appendicitis
 - E. Left-side appendicitis
123. The dysuria is typical for:
- A. * Pelvic appendicitis
 - B. Retrocecal appendicitis
 - C. Phlegmonous appendicitis
 - D. Simple appendicitis
 - E. Left-side appendicitis appendicitis
124. The expressed deviation of the differential leukocyte count to the left in acute appendicitis is characteristic for persons :
- A. * elderly patients
 - B. females
 - C. males
 - D. pregnant
 - E. children
125. The expressed pain in a right lumbar area is typical for:
- A. * Retrocecal appendicitis
 - B. Phlegmonous appendicitis
 - C. Simple appendicitis
 - D. Left-side appendicitis appendicitis
 - E. Pelvic appendicitis
126. The Gabay's sign is typical for:
- A. * Retrocecal appendicitis
 - B. Phlegmonous appendicitis
 - C. Simple appendicitis
 - D. Left-side appendicitis appendicitis
 - E. Pelvic appendicitis
127. The most frequent complications of appendicitis are:
- A. * infiltrate, abscess, pilephlebitis, peritonitis
 - B. infiltrate, abscess, thrombophlebitis, hepatitis
 - C. conglomerate, adhesions, cystitis, peritonitis
 - D. infiltrate, conglomerate, hepatitis
 - E. abscess, peritonitis, adhesions, phlebitis
128. The most informative for differentiation of appendicitis with a basal pleurisy is:
- A. * X-ray film
 - B. percussion
 - C. tomography
 - D. auscultation
 - E. bronchoscopy
129. The most informative for differentiation of appendicitis with an epigastric form of myocardial infarction are the changes in:
- A. * ECG
 - B. hemodynamic disturbances
 - C. expressed shortness of breath

- D. auscultation
 - E. tachycardia
130. The most informative for differentiation of appendicitis with food poisoning is:
- A. * frequent vomit
 - B. single vomit
 - C. nausea
 - D. increased peristalsis
 - E. slow peristalsis
131. The most informative for differentiation of appendicitis with gastric phlegmon is:
- A. * esophagogastroscopy
 - B. roentgenoscopy
 - C. palpation
 - D. laparocentesis
 - E. ultrasound examination
132. The most informative for differentiation of appendicitis with intercostal neuralgia is:
- A. * paravertebral blockade
 - B. laparoscopy
 - C. microlaparotomy
 - D. laparocentesis
 - E. peridural blockades
133. The most informative for differentiation of appendicitis with perforative ulcer of duodenum is:
- A. * absence of hepatic dullness
 - B. presence of hepatic dullness by percussion
 - C. absence of the splenic dullness
 - D. presence of a high tympanic sound by percussion
 - E. absence of the gastric dullness
134. The most informative for the differentiation of appendicitis with cholecystitis is:
- A. * ultrasound examination
 - B. X-ray film
 - C. anamnesis
 - D. laparocentesis
 - E. laparoscopy
135. The most informative for the differentiation of appendicitis with intestinal obstruction is:
- A. * X-ray film
 - B. ultrasound examination
 - C. blockade
 - D. laparotomy
 - E. laparoscopy
136. The most informative for the differentiation of appendicitis with pancreatitis is:
- A. * ultrasound examination
 - B. blockades
 - C. laparostomy
 - D. laparoscopy
 - E. X-ray film
137. The most informing method of instrumental diagnostics of acute appendicitis is:

- A. * tomography
 - B. esophagogastroscopy
 - C. colonoscopy
 - D. gastroscopy
 - E. contrasting roentgenoscopy
138. The most informing method of instrumental diagnostics of acute appendicitis is:
- A. * ultrasound examination
 - B. contrasting roentgenoscopy
 - C. gastroscopy
 - D. esophagogastroscopy
 - E. colonoscopy
139. The most prominent clinical sign of chronic appendicitis is:
- A. * pain by deep palpation
 - B. pain by percussion
 - C. pain by superficial palpation
 - D. skin hyperesthesia
 - E. pain by bimanual palpation
140. The omental hypoplasia influences on the peculiarities of the course of acute appendicitis in:
- A. * children
 - B. females
 - C. males
 - D. pregnant
 - E. elderly patients
141. The omental hypoplasia influences on the peculiarities of the course of acute appendicitis in:
- A. * children
 - B. females
 - C. males
 - D. pregnant
 - E. elderly patients
142. The pain all over the whole abdomen in acute appendicitis is characteristic for:
- A. * children
 - B. females
 - C. pregnant
 - D. males
 - E. elderly patients
143. The pain all over the whole abdomen in acute appendicitis is characteristic for:
- A. * children
 - B. females
 - C. pregnant
 - D. males
 - E. elderly patients
144. The painfulness of anterior rectal wall is typical for:
- A. * Pelvic appendicitis
 - B. Retrocecal appendicitis
 - C. Phlegmonous appendicitis

- D. Simple appendicitis
 - E. Left-side appendicitis appendicitis
145. The painfulness of the left iliac region is typical for:
- A. * Left-side appendicitis appendicitis
 - B. Pelvic appendicitis
 - C. Retrocecal appendicitis
 - D. Phlegmonous appendicitis
 - E. Simple appendicitis
146. The Pasternatsky's sign is typical for:
- A. * Retrocecal appendicitis
 - B. Phlegmonous appendicitis
 - C. Simple appendicitis
 - D. Left-side appendicitis appendicitis
 - E. Pelvic appendicitis
147. The peculiarities of the clinical course of appendicitis in children are caused:
- A. * by the bailed form of appendix
 - B. by the tubular form of appendix
 - C. by hypertrophy of appendix
 - D. by atrophy of appendix
 - E. by the spherical form of appendix
148. The peculiarities of the clinical course of appendicitis in children are caused:
- A. * by the bailed form of appendix
 - B. by the tubular form of appendix
 - C. by hypertrophy of appendix
 - D. by atrophy of appendix
 - E. by the spherical form of appendix
149. The pelvic appendicitis manifests by:
- A. * F. tenesmi
 - B. G. spasms
 - C. H. myalgia
150. paresis
- A. J. enuresis
151. The pelvic appendicitis manifests by:
- A. * diarrhea
 - B. vomiting
 - C. constipation
 - D. nausea
 - E. colicks
152. The pelvic appendicitis manifests by:
- A. * dysurination
 - B. dyspepsia
 - C. hyperthermia
 - D. hematuria
 - E. dystrophy
153. The psoas sign is typical for:
- A. * Retrocecal appendicitis

- B. Phlegmonous appendicitis
 - C. Simple appendicitis
 - D. Left-side appendicitis appendicitis
 - E. Pelvic appendicitis
154. The pulling rectal pain is typical for:
- A. * Pelvic appendicitis
 - B. Retrocecal appendicitis
 - C. Phlegmonous appendicitis
 - D. Simple appendicitis
 - E. Left-side appendicitis appendicitis
155. 154. The purulent inflammation of portal vein as the complication of acute appendicitis - is:
- A. * pilephlebitis
 - B. mesadenitis
 - C. tiplitis
 - D. thrombophlebitis
 - E. adnexitis
156. The rapid spread of inflammatory process in acute appendicitis is characteristic for:
- A. * children
 - B. females
 - C. males
 - D. pregnant
 - E. elderly patients
157. The rapid spread of inflammatory process in acute appendicitis is characteristic for:
- A. * children
 - B. females
 - C. males
 - D. pregnant
 - E. elderly patients
158. The removal of appendix from apex - is :
- A. * antegrade appendectomy
 - B. retrograde appendectomy
 - C. retrocecal appendectomy
 - D. antececal appendectomy
 - E. laparoscopic appendectomy
159. The removal of appendix from the base is:
- A. * Retrograde appendectomy
 - B. antegrade appendectomy
 - C. retrocecal appendectomy
 - D. antececal appendectomy
 - E. laparoscopic appendectomy
160. The Rovsing's sign is typical for:
- A. * Simple appendicitis
 - B. Retrocecal appendicitis
 - C. Retroperitoneal appendicitis
 - D. Pelvic appendicitis
 - E. Left-side appendicitis

161. The Rozdolsky's sign is typical for:
- A. * Phlegmonous appendicitis
 - B. Simple appendicitis
 - C. Retrocecal appendicitis
 - D. Retroperitoneal appendicitis
 - E. Pelvic appendicitis
162. The sign of gas migration is called:
- A. * Rovsing's sign
 - B. Kocher's sign
 - C. Sitkovsky's sign
 - D. Bartomier's sign
 - E. Dunphy's sign
163. The Sitkovsky's sign is typical for:
- A. * Simple appendicitis
 - B. Retrocecal appendicitis
 - C. Retroperitoneal appendicitis
 - D. Pelvic appendicitis
 - E. Left-side appendicitis
164. The tenesmi are typical for:
- A. * Pelvic appendicitis
 - B. Retrocecal appendicitis
 - C. Phlegmonous appendicitis
 - D. Simple appendicitis
 - E. Left-side appendicitis appendicitis
165. The undiagnosed destructive appendicitis complicated by:
- A. * infiltrate
 - B. fistula
 - C. adhesions
 - D. bleeding
 - E. colic
166. The Voskresenky's sign is typical for:
- A. * Phlegmonous appendicitis
 - B. Simple appendicitis
 - C. Retrocecal appendicitis
 - D. Retroperitoneal appendicitis
 - E. Pelvic appendicitis
167. The Yaure-Rozanov sign is typical for:
- A. * Retrocecal appendicitis
 - B. Phlegmonous appendicitis
 - C. Simple appendicitis
 - D. Left-side appendicitis appendicitis
 - E. Pelvic appendicitis
168. To the chronic secondary appendicitis belongs:
- A. * residual
 - B. catarrhal
 - C. empyema

- D. phlegmonous
 - E. gangrenous
169. Tumour with fluctuation are the main clinical manifestation of:
- A. * appendicular abscess
 - B. appendicular peritonitis
 - C. appendicular infiltrate
 - D. appendicular mesadenitis
 - E. appendicular typhlitis
170. Typical complications of the appendicitis are:
- A. * infiltrate, abscess, peritonitis, pilephlebitis
 - B. abscess, phlegmon, paraproctitis, pilephlebitis
 - C. infiltrate, gangrene, paraproctitis, pilephlebitis
 - D. abscess, phlegmon, peritonitis, pilephlebitis
 - E. infiltrate, abscess, osteomyelitis, pilephlebitis
171. Voskresenky's sign is typical for:
- A. * acute appendicitis
 - B. acute thrombophlebitis
 - C. pneumothorax
 - D. food poisoning
 - E. bleeding ulcer
172. What clinical picture is typical for appendicitis in children?
- A. * Clinic of destructive forms of appendicitis and intoxication
 - B. Abdominal distension
 - C. Absence of dyspeptic manifestation
 - D. Absence of muscular tenderness
 - E. Clinic of acute intestinal obstruction
173. What complication is typical for acute appendicitis?
- A. * Appendicular infiltrate
 - B. Appendicular bleeding
 - C. Acute intestinal obstruction
 - D. Appendicular-intestinal fistula
 - E. Malignization
174. What complication is typical for acute appendicitis?
- A. * Appendicular abscess
 - B. Appendicular bleeding
 - C. Acute intestinal obstruction
 - D. Appendicular-intestinal fistula
 - E. Malignization
175. What does the Bartomier-Mikhelson's sign mean?
- A. * The increase of pain intensity during the palpation of right iliac area when the patient lies on the left side.
 - B. Increased pain with coughing
 - C. Pain in right lower quadrant during palpation of left lower quadrant
 - D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
176. What does the Blumberg's sign mean?

- A. * The sharp increase of pain quick taking off the hand during palpation of anterior abdominal wall.
 - B. Increased pain with coughing
 - C. Pain in right lower quadrant during palpation of left lower quadrant
 - D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
177. What does the Dunphy's sign mean?
- A. * Increased pain with coughing
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Increase of pain in a right iliac area when the patient lies on the left side
 - D. The increase of pain intensity during the palpation of right iliac area when the patient lies on the left side.
 - E. Migration of pain to the right iliac area from epigastric
178. What does the examination of infant children in acute appendicitis require to use?
- A. * Chloralhydrate enema
 - B. Contrast enema
 - C. Siphon enema
 - D. Cleaning enema
 - E. X-ray with barium swallow
179. What does the Gabay's sign mean?
- A. * Blumberg's sign in Petit triangle
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Migration of pain to the right iliac area from epigastric
 - D. Tapping of lumbar region cause the pain
 - E. Increase of pain in a right iliac area when the patient lies on the left side
180. What does the Gabay's sign mean?
- A. * Blumberg's sign in Petit triangle
 - B. Increased pain with coughing
 - C. Pain in right lower quadrant during palpation of left lower quadrant
 - D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
181. What does the Kocher's sign mean?
- A. * Migration of pain to the right iliac area from epigastric
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Increase of pain in a right iliac area when the patient lies on the left side
 - D. The increase of pain intensity during the palpation of right iliac area when the patient lies on the left side.
 - E. Increased pain with coughing
182. What does the Pasternatsky's sign mean?
- A. * Tapping of lumbar region cause the pain
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Increase of pain in a right iliac area when the patient lies on the left side
 - D. Increased pain with coughing
 - E. Migration of pain to the right iliac area from epigastric
183. What does the psoas sign mean?
- A. * Pain on extension of right thigh
 - B. Increased pain with coughing

- C. Pain in right lower quadrant during palpation of left lower quadrant
 - D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
184. What does the psoas-sign mean?
- A. * Pain on extension of right thigh
 - B. Painfulness during palpation of Petit triangle
 - C. Migration of pain to the right iliac area from epigastric
 - D. Tapping of lumbar region cause the pain
 - E. Increase of pain in a right iliac area when the patient lies on the left side
185. What does the Rovsing's sign mean?
- A. * Pain in right lower quadrant during palpation of left lower quadrant
 - B. Increase of pain in a right iliac area when the patient lies on the left side
 - C. The increase of pain intensity during the palpation of right iliac area when the patient lies on the left side.
 - D. Increased pain with coughing
 - E. Migration of pain to the right iliac area from epigastric
186. What does the Rozdolsky's sign mean?
- A. * Painfulness in a right iliac area during percussion
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Increase of pain in a right iliac area when the patient lies on the left side
 - D. Increased pain with coughing
 - E. Migration of pain to the right iliac area from epigastric
187. What does the Rozdolsky's sign mean?
- A. * Painfulness in a right iliac area during percussion.
 - B. Increased pain with coughing
 - C. Pain in right lower quadrant during palpation of left lower quadrant
 - D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
188. What does the Sitkovsky's sign mean?
- A. * Increase of pain in a right iliac area when the patient lies on the left side
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. The increase of pain intensity during the palpation of right iliac area when the patient lies on the left side.
 - D. Increased pain with coughing
 - E. Migration of pain to the right iliac area from epigastric
189. What does the Voskresenky's sign mean?
- A. * Increase of pain during quick sliding movements by the tips of fingers from epigastric to right iliac area
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Increase of pain in a right iliac area when the patient lies on the left side
 - D. Increased pain with coughing
 - E. Migration of pain to the right iliac area from epigastric
190. What does the Voskresenky's sign mean?
- A. * The increase of pain during quick sliding movements by the tips of fingers from epigastric to right iliac area.
 - B. Increased pain with coughing
 - C. Pain in right lower quadrant during palpation of left lower quadrant

- D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
191. What does the Yaure-Rozanov sign mean?
- A. * Painfulness during palpation of Petit triangle
 - B. Pain in right lower quadrant during palpation of left lower quadrant
 - C. Migration of pain to the right iliac area from epigastric
 - D. Tapping of lumbar region cause the pain
 - E. Increase of pain in a right iliac area when the patient lies on the left side
192. What does the Yaure-Rozanov sign mean?
- A. * Painfulness during palpation of Petit triangle
 - B. Increased pain with coughing
 - C. Pain in right lower quadrant during palpation of left lower quadrant
 - D. Increase of pain in a right iliac area when the patient lies on the left side
 - E. Migration of pain to the right iliac area from epigastric
193. What dyspeptic manifestations are typical for acute appendicitis?
- A. * Single nausea and vomiting
 - B. Constant vomiting and nausea without any relief
 - C. Vomiting by bile without any relief
 - D. Absence of peristalsis
 - E. Constant diarrhea
194. What form of appendicitis the signs of peritoneal irritation are absent in?
- A. * chronic
 - B. calculous
 - C. perforative
 - D. appendicular infiltrate
 - E. appendicular abscess
195. What form of the appendicitis results in the developing of fibrosis of the appendix?
- A. * chronic
 - B. phlegmonous
 - C. catarrhal
 - D. gangrenous
 - E. perforative
196. What form of the appendicitis results in the obliteration of the appendix??
- A. * chronic
 - B. phlegmonous
 - C. catarrhal
 - D. gangrenous
 - E. perforative
197. What is the lethality in acute appendicitis caused by?
- A. * late hospitalization
 - B. tactical errors
 - C. concomitant diseases
 - D. technical errors during an operation
 - E. severity of disease
198. What is the medical tactic of the acute appendicitis in pregnant:
- A. * to operate

- B. to prescribe antibiotics
 - C. to prescribe conservative therapy
 - D. to observe
 - E. to interrupt pregnancy
199. What is the treatment of appendicular infiltrate?
- A. * Conservative treatment
 - B. Draining operation
 - C. Appendectomy
 - D. Hemicolectomy
 - E. Caecostomy
200. What manifestation is predominant for pelvic appendicitis?
- A. * Clinic of irritation of pelvic organs (dysuria, pulling rectal pain, tenesmi)
 - B. Clinic of acute abdomen
 - C. Clinic of retroperitoneal phlegmon
 - D. Clinic of acute intestinal obstruction
 - E. Clinic of acute pancreatitis
201. What manifestation is predominant for retroperitoneal appendicitis?
- A. * Clinic of retroperitoneal phlegmon
 - B. Clinic of acute abdomen
 - C. Dyspeptic syndrome
 - D. Clinic of acute intestinal obstruction
 - E. Clinic of acute pancreatitis
202. What manifestation is typical for pelvic appendicitis?
- A. * Absence of muscular tenderness
 - B. Clinic of retroperitoneal phlegmon
 - C. Clinic of acute intestinal obstruction
 - D. Clinic of acute abdomen
 - E. Clinic of acute pancreatitis
203. What objective manifestations are typical for acute appendicitis?
- A. * Muscular tension in a right iliac area
 - B. Abdominal distension
 - C. Absence of hepatic dullness
 - D. Absence of peristalsis
 - E. Rigidity of anterior abdominal wall
204. What objective manifestations are typical for retrocaecal appendicitis?
- A. * Pain and muscular rigidity in a right iliac area during palpation
 - B. Abdominal distension
 - C. Absence of hepatic dullness
 - D. Clinic of retroperitoneal phlegmon
 - E. Rigidity of anterior abdominal wall
205. What objective manifestations are typical for retrocaecal appendicitis?
- A. * Painfulness of anterior rectal wall and posterior vaginal vault
 - B. Abdominal distension
 - C. Absence of hepatic dullness
 - D. Clinic of retroperitoneal phlegmon
 - E. Rigidity of anterior abdominal wall

206. What sign is typical for phlegmonous appendicitis in contrast to simple appendicitis?
- A. * Blumberg's sign
 - B. Kocher's sign
 - C. Bartomier's sign
 - D. Sitkovsky's sign
 - E. Dunphy's sign
207. What sign is typical for phlegmonous appendicitis in contrast to simple appendicitis?
- A. * Voskresenky's sign
 - B. Sitkovsky's sign
 - C. Bartomier's sign
 - D. Kocher's sign
 - E. Dunphy's sign
208. What sign is typical for retrocaecal appendicitis in contrast to simple appendicitis?
- A. * Pasternatsky's sign
 - B. Kocher's sign
 - C. Bartomier's sign
 - D. Sitkovsky's sign
 - E. Dunphy's sign
209. What sign is typical for retrocaecal appendicitis in contrast to simple appendicitis?
- A. * Psoas sign
 - B. Sitkovsky's sign
 - C. Bartomier's sign
 - D. Kocher's sign
 - E. Dunphy's sign
210. What signs are typical for gangrenous appendicitis in contrast to simple appendicitis?
- A. * Signs of intoxication
 - B. Signs of gas migration
 - C. Retention of stool or single diarrhea
 - D. Muscular tension in a right iliac area
 - E. Single nausea and vomiting
211. What signs are typical for phlegmonous appendicitis in contrast to simple appendicitis?
- A. * Peritoneal signs
 - B. Signs of gas migration
 - C. Signs of pain migration
 - D. Muscular tension in a right iliac area
 - E. Nausea and vomiting
212. Where does the pain arise in the onset of acute appendicitis?
- A. * Epigastric region
 - B. Left iliac region
 - C. Right iliac region
 - D. Left subcostal region
 - E. Right lumbar region
213. Where does the pain irradiate in acute appendicitis?
- A. * Not irradiate
 - B. Lumbar region
 - C. Left iliac region
 - D. Right scapular

- E. Perineum
214. Where is the pain localized in acute appendicitis?
- A. * Right iliac region
 - B. Epigastric region
 - C. Left iliac region
 - D. Left subcostal region
 - E. Right lumbar region
215. Where is the pain localized in left-side appendicitis?
- A. * Left iliac region
 - B. Epigastric region
 - C. Right iliac region
 - D. Left subcostal region
 - E. Right lumbar region
216. Where is the pain localized in retrocaecal appendicitis?
- A. * Right lumbar region
 - B. Right iliac region
 - C. Epigastric region
 - D. Left iliac region
 - E. Left subcostal region
217. Which method of appendectomy is used in children before age 3?
- A. * ligation
 - B. amputation
 - C. retrograde
 - D. antegrade
 - E. laparoscopic
218. Who usually suffer from gangrenous appendicitis?
- A. * People of old age
 - B. Newborns
 - C. Children
 - D. Pregnant women
 - E. Young men
219. Yaure-Rozanov's sign is typical for:
- A. * acute appendicitis
 - B. acute thrombophlebitis
 - C. pneumothorax
 - D. food poisoning
 - E. bleeding ulcer
220. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appear. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
221. A sick on a background the stones in the common bile duct needs icterus:

- A. To the urgent operation
 - B. Conservative treatment
 - C. * To the urgent operation after preoperative preparation
 - D. Cannulations of the abdominal artery
 - E. Plasmapheresis
222. A veritable postcholecystectomy syndrome can be conditioned only
- A. Cicatrical stenosis of the common bile duct
 - B. The stone of the common bile duct not found during an operation
 - C. Stenosis of the large duodenal nipple
 - D. Duodenostasis
 - E. * Decline of tone of sphincter Oddi and expansion of the common bile duct after cholecystectomy
223. A veritable postcholecystectomy syndrome can be conditioned only:
- A. By cicatrical stenosis of the common bile duct
 - B. By the stone of the common bile duct not found during an operation
 - C. By stenosis of large duodenal nipple
 - D. Duodenostasis
 - E. * By the decline of tone the sphincter Oddie and expansion of the common bile duct after cholecystectomy
224. Absolute indication to operative treatment the ulcerous illness is
- A. heavy pain syndrome
 - B. * perforation of ulcer
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. giant ulcers
225. Absolute indication to operative treatment the ulcerous illness is
- A. * voluminous bleeding
 - B. callous ulcers
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. heavy pain syndrome
226. Absolute indication to operative treatment the ulcerous illness is
- A. ulcerous anamnesis more than 10 years
 - B. * bleeding what do not stopped with conservative
 - C. perforation ulcer in anamnesis
 - D. heavy pain syndrome
 - E. relapses more than 3 times per a year
227. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. ulcerous anamnesis more than 10 years
 - C. relapse ulcer after the resection of stomach
 - D. relapses more than 3 times per a year
 - E. * cicatrical-ulcerous stenosis of pylorus
228. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years

- D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
229. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
230. Acute cholecystitis usually begins with
- A. Increases the temperature
 - B. Appearance of vomiting
 - C. * Pains under a rib on the right
 - D. Disorders of chair
 - E. Weights are in an epigastric area
231. Acute cholecystitis usually begins with:
- A. Increases of temperatures
 - B. Appearances of vomiting
 - C. * Pains in right under rib space
 - D. Disorders of defecation
 - E. Weights in the epigastric area
232. Acute pancreatitis with a heavy flow treat in terms:
- A. Ambulatory
 - B. Permanent establishment
 - C. * Department of intensive therapy
 - D. All answers are faithful
 - E. A faithful answer is not present
233. After cholecystectomy drainage is more frequent than all used
- A. By Robson - Vishnevskiy
 - B. By Holsted - Pikovskiy
 - C. * To the couch of the gall-bladder and Winslow foramen
 - D. By Ker
 - E. Combination draining by Pikovskiy and Spasokukotskiy
234. After cholecystostomy drainage more frequent than all used:
- A. By Robson-Vishnevskiy
 - B. By Holsted-Pikovskiy
 - C. * By Spasokukotskiy
 - D. By Ker
 - E. Combination draining by Pikovskiy and Spasokukotskiy
235. After time of origin complications acute pancreatitis select:
- A. Premature and remote
 - B. Urgent and deferred
 - C. Primary and second
 - D. * Early and late
 - E. Any of variants faithful
236. After what operation at ulcerous illness the natural arc of meal is saved on a digestive tract

- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I
 - E. saved after all transferred operations
237. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred
 - E. not saved after all operations
238. All surgical interferences at the destructive forms of acute pancreatitis divide on:
- A. * Early, late, deferred operations
 - B. Primary, second, repeated operations
 - C. Invasion, not invasion operations
 - D. Complicated, operations are not complicated
 - E. Not divided
239. Among acute surgical diseases acute pancreatitis occupies:
- A. First place
 - B. * Third place
 - C. Fifth place
 - D. Second - third place
 - E. Most widespread
240. An intravenous cholecystography are indicated and informing at
- A. Gall-bladder is palpated
 - B. To the icterus
 - C. Peritonitis
 - D. * Calming down attack of the acute cholecystitis
 - E. Cholangitis
241. An intravenous cholecystography is indicated and informing:
- A. At presence of palpation the gall-bladder
 - B. At an icterus
 - C. At peritonitis
 - D. * At the calming down attack of the acute cholecystitis
 - E. At a cholangitis
242. As the first period flowing of acute pancreatitis is named:
- A. * Haemodynamic violations and pancreatic shock
 - B. To functional insufficiency of parenchymatous organs
 - C. Degenerative and festerings complications
 - D. All answers are faithful
 - E. A right answer is not present
243. As the second period flowing of acute pancreatitis is named:
- A. * Haemodynamic violations and pancreatic shock
 - B. To functional insufficiency of parenchymatous organs
 - C. Degenerative and festerings complications
 - D. All answers are faithful
 - E. A right answer is not present

244. As the third period flowing of acute pancreatitis is named:
- A. Haemodynamic violations and pancreatic shock
 - B. * To functional insufficiency of parenchymatous organs
 - C. Degenerative and festering complications
 - D. All answers are faithful
 - E. A right answer is not present
245. At a acute calculary cholecystitis can be used
- A. Extra operation
 - B. Urgent operation
 - C. Medicinal therapy is the planned operation in future
 - D. Only medicinal therapy
 - E. * All are right
246. At a acute calculary cholecystitis can be used:
- A. Extraordinary operation
 - B. Urgent operation
 - C. Conservative therapy is the planned operation in future
 - D. Only conservative therapy
 - E. * All transferred
247. At a frequent „fat” chair with disseminations of undigested meat and permanent thirst, it is foremost necessary to think about:
- A. * Chronic pancreatitis
 - B. Chronic duodenitis
 - C. Chronic hepatocholecystitis
 - D. Ulcerous diseases of duodenum
 - E. Hepatocirrhosis
248. At an acute and chronic cholecystitis application is contra-indicated
- A. Omnoponum
 - B. * Morphine hydrochloride
 - C. No-spa
 - D. Atropine sulfate
 - E. Spazmalgon
249. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
250. At bile-stones diseases cholecystectomy is indication
- A. * In all cases
 - B. At the latent form of disease
 - C. At the clinical signs of disease
 - D. Decline of ability to work
 - E. For patients more senior 55 years
251. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour

- C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
252. At gangrenous cholecystitis is indication
- A. Deferred operation
 - B. Operation in default of effect from the conservative therapy
 - C. Conservative treatment
 - D. * Urgent operation
 - E. A decision-making depends on age of patient
253. At III stage blood loss at the bleeding ulcer the patient loses
- A. over 1000 ml blood
 - B. * over 2000 ml blood
 - C. over 500 ml blood
 - D. over 2500 ml blood
 - E. over 1500 ml blood
254. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
255. At localization the stone in a cystic channel and absence infection the phenomenon is carried by the name
- A. Courvoisier's symptom
 - B. Acute cholecystitis
 - C. Hydrocholecystis
 - D. * Cyst of gall-bladder
 - E. All answers are not right
256. At pancreatitis abscesses and infected necroses execute such operations, except for:
- A. Opening of abscess with draining
 - B. Pancreaticnecrosekvestrektomy
 - C. Pancreaticsekvestrektomy
 - D. Pancreaticsekvestrektomy with laparostomy
 - E. * Total pancreatotomy
257. At the duodenum ulceroperation of choice is
- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
258. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%

259. At the III item of activity hemostasis and III stage blood loss from the I – III stage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
260. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
261. At transferring the stone from a gall-bladder in the common bile duct develops
- A. * Hepatic colic
 - B. Icterus
 - C. Festering cholangitis
 - D. Stenosis papillitis
 - E. Syndrome of Badd – Kiyary
262. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
263. At what disease pain of girdle character is characteristic:
- A. Gastric ulcers
 - B. Acute cholecystitis
 - C. Intestinal impassability
 - D. * Acute pancreatitis
 - E. Acute cystitis
264. At what pathology is absence pulsation of abdominal aorta
- A. * Acute cholecystitis
 - B. Acute pancreatitis
 - C. Perforated ulcer
 - D. Intestinal obstruction
 - E. Acute appendicitis
265. At what pathology is appearance cyanosis the sides of stomach:
- A. * Acute pancreatitis
 - B. Perforated ulcer
 - C. Acute cholecystitis
 - D. Intestinal obstruction
 - E. Acute appendicitis
266. At what pathology is appearance of sickliness in left costal-vertebral coal:
- A. * Acute pancreatitis
 - B. Acute cholecystitis

- C. Perforated ulcer
 - D. Intestinal obstruction
 - E. A right answer is not present
267. Basic method examination the patients with the uncomplicated cholecystitis
- A. Infusion cholegraphy
 - B. ERCP
 - C. * Sonography
 - D. Laparoskopy
 - E. Gastroduodenoscopy
268. Bergman's sign is characteristic for
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
269. Berhstein's sign characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
270. Bile-stones diseases can cause everything, except for
- A. Cystic-duodenal fistula
 - B. Mechanical icterus
 - C. Acute cholecystitis
 - D. * Intra-abdominal bleeding
 - E. Cholangitis
271. Bile-stones diseases is dangerous all transferred, except for
- A. * Development the postnecrotic hepatocirrhosis
 - B. Cancer transformation of gall-bladder
 - C. Second pancreatitis
 - D. Development of destructive cholecystitis
 - E. Mechanical icterus
272. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
273. Blood loss I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
274. Blood loss II stage characterized

- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
275. Blood loos III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
276. Blumberg's sign is
- A. Sickliness at palpation of blind gut in position the patient on the left side
 - B. * Sickliness at the acute tearing away the palpation hand
 - C. Appearance or strengthening the pains in position on the left side
 - D. Percussion sickliness in a right iliac area
 - E. At pressing on a left arm in the left iliac area shove there causes a sickliness in a right iliac area
277. Bonde's sign at acute pancreatitis:
- A. * Swelling the stomach only in epigastrium
 - B. Cyanosis sides of stomach and trunk
 - C. Cyanosis skin of stomach
 - D. Ictericness round a belly-button
 - E. Cyanosis of hands
278. Can not stipulate a mechanical icterus
- A. Cancer the head of pancreas
 - B. * Stone of cystic channel
 - C. Chronic pancreatitis
 - D. Stone the general bilious channel
 - E. Tumour large duodenal papilla
279. Caused a remittent icterus is
- A. The wedged stone of terminal department of the common bile stone
 - B. The tumour of the common bile stone
 - C. The stone of cystic channel
 - D. * The valve stone of the common bile stone
 - E. Stricture of the common bile stone
280. Characteristic complication acute pancreatitis is:
- A. * Pancreonecrosis
 - B. Pylephlebitis
 - C. Hepatocirrhosis
 - D. Veritable pancreas cyst
 - E. Hepatitis
281. Characteristic complication the acute pancreatitis is:
- A. Paranephritis
 - B. Douglas's abscess
 - C. Pylephlebitis
 - D. Cyst of pancreas

- E. * Pancreonecrosis
282. Characteristic laboratory sign of the acute uncomplicated cholecystitis
- A. Diastasia
 - B. * Leukocytosis
 - C. Hypoglycemia
 - D. Glucosuria
 - E. Hyperbilirubinemia
283. Complication of choledocolitiasis
- A. Hydrocholecystis
 - B. Empyema of gall-bladder
 - C. * Icterus, cholangitis
 - D. Chronic active hepatitis
 - E. Perforate cholecystitis, peritonitis
284. Complications of acute cholecystitis
- A. Bleeding
 - B. * Empyema of gall-bladder
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
285. Complications of acute cholecystitis
- A. Bleeding
 - B. * Hepatic-renal insufficiency
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
286. Complications of acute cholecystitis
- A. Bleeding
 - B. * Mechanical icterus
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
287. Complications of acute cholecystitis
- A. Bleeding
 - B. * Hydrocholecystis
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
288. Complications of acute cholecystitis
- A. Bleeding
 - B. * Perforation of gall-bladder
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
289. Complications of acute cholecystitis
- A. Bleeding
 - B. * Peritonitis

- C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
290. Complications of acute cholecystitis
- A. Bleeding
 - B. * Approximately cystic infiltration
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
291. Complications of acute cholecystitis
- A. Bleeding
 - B. * Approximately cystic abscess
 - C. Syndrome of v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
292. Complications of the acute cholecystitis
- A. Bleeding
 - B. * Cholangitis
 - C. Syndrome v. cava sup.
 - D. Artery-venous fistula
 - E. All answers are right
293. Conservative treatment the acute pancreatitis includes:
- A. * Hunger
 - B. Povzner's diet №5
 - C. Povzner's diet №15
 - D. Moderation in a meal
 - E. Morning gymnastics
294. Courvoisier's symptom is
- A. Painless megascopic gall-bladder, patient is not yellow
 - B. Sickly megascopic gall-bladder, patient is not yellow
 - C. * Painless megascopic gall-bladder, patient is yellow
 - D. A gall-bladder don't palpaton
 - E. All answers are not right
295. De-Cerven's sing is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
296. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
297. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is

- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
298. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
299. Early complications the acute pancreatitis is not:
- A. * Fistula of pancreas
 - B. Pancreatic shock
 - C. Collapse
 - D. Ferment peritonitis
 - E. Pancreatic delirious syndrome
300. Eleker's sign is characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
301. ERCP apply at:
- A. Postcholecystectomy syndrome
 - B. Stenosing papillitis
 - C. * Stenosis the supraduodenal department of choledoch
 - D. Stricture the terminal department of choledoch
 - E. Mechanical icterus
302. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- A. tumours
 - B. ulcers
 - C. bleeding polypuses
 - D. erosions
 - E. * changes of evacuation function
303. Etiology of the acute cholecystitis:
- A. * Infection and stagnation of bile
 - B. Stagnation of bile
 - C. An infection in a gall-bladder
 - D. Duodenostasis
 - E. All answers are right
304. For a colic, caused a cholelithiasis, not characteristically
- A. A megalgia in a right side
 - B. Nausea
 - C. * Blumberg's sign in a right side
 - D. Ortner's sign
 - E. Merfi sign

305. For a mechanical icterus, conditioned the stones of the common bile duct , not characteristically
- Symptom of Kurvuazie
 - Increase the direct bilirubine of blood
 - Increase of alkaline phosphathase
 - A considerable increase level the transaminasis in plasma
 - * Absence the stercobiline in an excrement
306. For a patient 48 years clinical picture the acute phlegmon cholecystitis and phenomenon of the local peritonitis. Conservative treatment was conduct. Over 6 hours from a receipt great pains appeared in a stomach, death-damp, pulse is 120 in min, a stomach is tense and acute sickly in all departments, Blumberg's sing is positive. It is decided to make an operation
- * Cholecystectomy, revision the bilious channels, draining the abdominal region
 - Gastrotomy, sewing of acute ulcers
 - Cholecystectomy, draining and sanitization abdominal region
 - Sewing up of the perforated opening, sanitization and draining abdominal region
 - Cholecystectomy and gastrotomy
307. For an acute catarrhal cholecystitis not characteristic:
- Nausea
 - * Ker's sing
 - Merfi sing
 - Absence tension of muscles in right under rib space
 - Mussi sing
308. For an acute catarrhal cholecystitis not characteristically
- Nausea
 - Ker's sing
 - Merfi sing
 - * Tension the muscles in right underrib space
 - Symptom by Myussi
309. For an icterus on soil the stone ofcommon bile duct not characteristically:
- * Urobilinuria
 - Increase of alkaline phosphathase
 - A normal or lowered albumen in blood
 - Increase the bilirubine of blood
 - Normal or moderato enhanceable transaminase
310. For an icterus owing to stones of the common bile duct not characteristic
- * Urobilinuria
 - Increase of alkaline phosphatase
 - A normal or lowered albumen in blood
 - Increase bilirubine of blood
 - Normal or moderato increase of transaminase
311. For bleeding ulcer characteristic sign is
- * pain in an epigastrium;
 - knife-like pain;
 - signs irritation of peritoneum;
 - presence fresh blood in incandescence
 - melena;

312. For bleeding ulcer characteristically
- * melena
 - tension the muscles of front abdominal wall
 - Spazarskiy's sign
 - sickness the back vault of vagina
 - irradiation pain in a shoulder or shoulder-blade
313. For clarification the character icterus and reason of its origin don't used
- Computer tomography
 - * Peroral cholecystocholangiography
 - Throughskin transhepatic cholangiography
 - ERCPG
 - Sonography
314. For clarification character of the icterus and reason of its development is necessary to make:
- X-ray examination of the subhepatic space, infusion cholecystography, ERCP
 - Sonography, ERCP
 - Infusion cholecystography, ERCP
 - * Sonography, infusion cholecystography, ERCP
 - ERCP
315. For diagnostics of acute pancreatitis most informing is:
- * Sonography
 - CT
 - Cholangiography
 - Esophagogastroduodenoscopy
 - Colonoscopy
316. For motion of disease ulcerous illness of middle weight characteristically
- development of complications
 - * relapses 1-2 times per a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year
 - 3 and anymore relapses on a year
317. For pancreonecrosis characteristically is:
- * Rapid progress, strengthening pain, proof enteroplegia, growth haemodynamic violations
 - Rapid progress, diminishing pain, strengthening of моторики of intestine, growth haemodynamic violations
 - Slow progress, strengthening pain, phase of imaginary prosperity.
 - Abortive flow, toxemia, development shock.
 - Appearance light interval
318. For perforated ulcer characteristically
- * tension the muscles of front abdominal wall
 - melena
 - vomiting by coffee-grounds
 - high intestinal impassability
 - vomiting stagnant gastric maintenance
319. For pneumoperitoneum is characteristic symptom
- * Zhober's;
 - Khelatid's;

- C. Podlag's;
 - D. Vigats's;
 - E. Udin's.
320. For the abortive flowing characteristically:
- A. * A process limited to the acute edema with convalescence in 7-8 days
 - B. A process limited to tearing away of the pathologically changed gland
 - C. A process limited to tearing away of the pathologically unchanged gland
 - D. A disease completed so not attaining clinical displays
 - E. Changes from the side of organ are not present
321. For the clinic of acute cholangitis not characteristically
- A. High temperature
 - B. Pains in right under rib space
 - C. Icterus
 - D. Leucocytosis
 - E. * Unsteady liquid stool
322. For the clinic of acute obturation cholangitis not characteristically
- A. Icterus
 - B. Increase of temperature
 - C. * Diminishing sizes the liver
 - D. Leucocytosis
 - E. Increase the liver
323. For the exposure the stone of common bile duct without an icterus used more frequent than all
- A. * Sonography
 - B. Intravenous cholecystocholangiography
 - C. ERCPG
 - D. Peroral cholecystography
 - E. Low blood pressure duodenography
324. For the exposure the stone of the common bile duct not used:
- A. Sonography
 - B. Intravenous cholecystocholohgiography
 - C. ERPKHG
 - D. Transhepatic cholegraphy
 - E. * Low pressure duodenography
325. For the heavy flow of ulcerous illness characteristically
- A. 2 and anymore relapses on a year
 - B. * 3 and anymore relapses on a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 6 and more relapses are on a year
326. For the heavy flow of ulcerous illness characteristically
- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days

327. For what pathology appearance of violet spots on the skin of person and trunk is characteristic:
- A. Perforated ulcer
 - B. Acute cholecystitis
 - C. * Acute pancreatitis
 - D. Intestinal obstruction
 - E. Acute appendicitis
328. From what department degestive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
329. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
330. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
331. Holsted's sing at acute pancreatitis:
- A. Violet spots on face and trunk
 - B. Cyanosis sides of stomach and trunk
 - C. * Cyanosis skin of stomach
 - D. Icteritiousness round a belly-button
 - E. Cyanosys of hands
332. How many is the period of haemodynamic violations and pancreatic shock lasts:
- A. * 1-3 days
 - B. 3-7 days
 - C. More 7 days
 - D. 2 weeks
 - E. 2 hour
333. How many is the period of degenerative and festerings complications lasts:
- A. 1-3 days
 - B. 3-7 days
 - C. * More 7 days
 - D. 2 weeks
 - E. 2 hour
334. How many is the period of functional insufficiency of parenchymatous organs lasts:
- A. 1-3 days
 - B. * 3-7 days

- C. More 7 days
 - D. 2 weeks
 - E. 2 hour
335. How often does the pancreatitis department the general bilious channel pass through the head of pancreas?:
- A. * 80-90 %
 - B. 90-100 %
 - C. 75-85 %
 - D. 50-60 %
 - E. 40-50 %
336. In a gall-bladder stone formation don't promote
- A. Stagnation of bile in a bladder
 - B. Exchange violations
 - C. Inflammatory changes in a gall-bladder
 - D. Diskinetic of the bile excretive ways
 - E. * Violation secretion the pancreas
337. In obedience to classification complications of acute pancreatitis, after etiology and pathogeny, distinguish such complications, except for:
- A. Infectiously inflammatory
 - B. Enzymic
 - C. * Allergic
 - D. Mixed
 - E. Trombogemoragic
338. In the case of bile-stones diseases an urgent operation is indicated at
- A. Occlusion of cystic channel
 - B. Cholecystopancreatitis
 - C. * Perforate cholecystitis
 - D. The mechanical icterus
 - E. The hepatic colic
339. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?
- A. small curvature of stomach;
 - B. back wall of stomach, nearer to small curvature;
 - C. large curvature of stomach
 - D. * cardiac part of stomach;
 - E. pylorus.
340. In what vein is a venous outflow carried out in from a stomach?
- A. * V. Portae;
 - B. V. odd;
 - C. V. pair;
 - D. V. overhead hollow;
 - E. V. lower hollow;
341. Indication to early operative interference at acute pancreatitis is:
- A. Acute oedematous pancreatitis
 - B. Acute pancreatolysis
 - C. * Acute traumatic pancreatitis at the „fresh” break the gland
 - D. Acute fatty pancreatitis

- E. A faithful answer is not present
342. Indication to early operative interference at acute pancreatitis is:
- A. Acute oedematous pancreatitis
 - B. Acute pancreatolysis
 - C. * Acute biliary pancreatitis
 - D. Acute fatty pancreatitis
 - E. Forming of pseudocyst
343. Indication to early operative interference at acute pancreatitis is:
- A. Acute pancreatolysis
 - B. Acute oedematous pancreatitis
 - C. * Progressive multiple organ failure what not added conservative therapy during 48-72 hours
 - D. Acute fatty pancreatitis
 - E. Forming of pseudocyst
344. Indications to special intraoperative examination bilious ways
- A. * A cholangitis, expansion of the common bile duct, plural shallow concrements in a gall-bladder, mechanical icterus in anamnesis
 - B. Cholangitis, expansion of the common bile duct, plural shallow concrements in a gall-bladder
 - C. Expansion of the common bile duct, icterus in the moment of operation, plural shallow concrements in a gall-bladder
 - D. Expansion of the common bile duct, mechanical icterus in anamnesis
 - E. All right
345. Indications to the special intraoperative examination the bilious ways
- A. Expansion the common bile duct, mechanical icterus in anamnesis
 - B. Expansion the common bile duct, icterus in the moment of operation, plural shallow concrements in a gall-bladder
 - C. * All right
 - D. A cholangitis, expansion the common bile duct, plural shallow concrements in a gall-bladder, mechanical icterus in anamnesis
 - E. Cholangitis, expansion the common bile duct, plural shallow concrements in a gall-bladder
346. Intraoperative cholangiographic is not absolutely indicated at
- A. Shallow stone in the common bile duct
 - B. Suspicion on the cancer the large duodenal nipple
 - C. Expansion of the common bile duct
 - D. To the mechanical icterus in anamnesis
 - E. * Switched-off gall-bladder
347. Intraoperative cholangiography at cholecystectomy used for
- A. Exposures of cholangitis
 - B. Researches of peristalsis of the general bilious channel
 - C. Researches the tone of sphincter Oddi
 - D. * Retrograde filling of intrahepatic bilious channels
 - E. Exceptions the concrements in channels
348. Intraoperative cholangiography is absolutely indication at everything, except for:
- A. Presences of shallow stone are in the common bile duct
 - B. Suspicion on the cancer of the large duodenal nipple

- C. Expansions the common bile duct
 - D. Mechanical icterus in anamnesis
 - E. * Switched-off gall-bladder
349. Intraoperative cholangiography at cholecystectomy used for
- A. Researches of peristalsis the general bilious channel
 - B. Retrograde filling of intrahepatic bilious channels
 - C. Researches tone the sphincter Oddi
 - D. * Exceptions concrements in channels
 - E. Exposures cholangitis
350. Intraoperative cholangiography is not indication at
- A. * Finding concrements in the common bile duct at palpation
 - B. Cancer the head of pancreas
 - C. The icterus in anamnesis
 - D. Expansion of common bile duct
 - E. The icterus during an operation
351. Kerte's sign at acute pancreatitis:
- A. Absence pulsation the abdominal aorta
 - B. Sickliness in left costal-vertebral coal
 - C. * Sickliness and proof tension the muscles in an epigastrium with passing to left subcostal area
 - D. Ictericness round a belly-button
 - E. Skin hyperesthesia in projection the gland
352. Kulen's sign at acute pancreatitis:
- A. Violet spots on face and trunk
 - B. Cyanosis sides of stomach and trunk
 - C. Cyanosis skin of stomach
 - D. * Ictericness round a belly-button
 - E. Cyanosis of hands
353. Kurvazie symptoms is not characteristic for
- A. * Acute calculary cholecystitis
 - B. Tumor the head of pancreas
 - C. The pancreatitis
 - D. Tumours large duodenal nipple
 - E. Tumours common bile duct
354. Large ulcer is an ulcer measuring
- A. 1-4 cm
 - B. 1-3 cm
 - C. 3-5 cm
 - D. 2- 6 cm
 - E. * 2-3 cm
355. Little ulcer it is an ulcer measuring
- A. * to 0,5 cm
 - B. 0,5-1 cm
 - C. to 1,0 cm
 - D. 3 to 1,5 cm
 - E. 0,5-1,5 cm

356. Meets the most frequent localization bleeding the digestive tract is
- gullet;
 - stomach;
 - rectum;
 - * duodenum;
 - colon
357. Megascopic sickly gall-bladder, positive Ortner's sign, Obraztsov's sign, Merfi sign, Ker's sign more frequent meet at
- * Acute cholecystitis
 - The hydrocholecystitis
 - Syndrom by Kurvuaz'e
 - Hepatitis
 - Panerkatitis
358. Melena is
- black designed chair
 - * black liquid tarry chair
 - a discoloured liquid excrement
 - foamy stinking emptying of black
 - an excrement designed veined blood
359. Melena is a characteristic sign
- * bleeding ulcer
 - for cicatrical-ulcerous pylorus stenosis
 - perforeted ulcers
 - penetration ulcers
 - malignization ulcers
360. Method intraoperative diagnostics the pathology of bilious ways
- Intravenous cholegraphy
 - * Choledochoscopy
 - Selective celiacography
 - Peroral cholecystography
 - All answers are right
361. Mayo-Robson's sign at acute pancreatitis:
- Absence pulsation the abdominal aorta
 - * Sickliness in left costal-vertebral coal
 - Sickliness and proof tension the muscles in an epigastrium with passing to left subcostal area
 - Icteritiousness round a belly-button
 - Skin hyperesthesia in projection the gland
362. Mondor's sign at acute pancreatitis:
- * Violet spots on face and trunk
 - Cyanosis sides of stomach and trunk
 - Cyanosis skin of stomach
 - Icteritiousness round a belly-button
 - Cyanosys of hands
363. Most informing method at a bleeding ulcer
- survey sciagraphy the organs of abdominal region
 - * EFGDS

- C. sciagraphy the stomach with contrasting
 - D. Sonography
 - E. Laparoskopy
364. 145. Most widespread laparoscopic operation at bile-stone illness:
- A. Cholecystectomy with revision the bilious ways
 - B. Ideal cholecysectomy
 - C. * Choledoholitotomy
 - D. Cholecystectomy
 - E. All answers are right
365. Name classic complications of ulcerous illness
- A. bleeding, pylorostenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pylorostenosis, malignization
 - C. malignization, pylorostenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
366. Name most frequent complication after ERDPH:
- A. * Pancreatitis
 - B. Cholangitis
 - C. Pancreatic sepsis
 - D. Reactive cholecystitis
 - E. Obstructing papillitis
367. Name principal reason the acute pancreatitis:
- A. Trauma the pancreas
 - B. * Bile-stone diseases
 - C. Alimentary factor
 - D. Chronic alcoholic pancreatitis
 - E. Cardiospasm
368. Name the optimum lines the operative interference concerning a acute pancreatitis after the beginning of disease:
- A. 7-8 days
 - B. 1-3 days
 - C. 1-5 days
 - D. 3-5 days
 - E. * Surgical treatment is not indicate
369. Name the optimum volume of operation at acute biliary pancreatitis:
- A. Deleting exsudate from an abdominal region
 - B. Decapsulation pancreas
 - C. Pancreatectomy
 - D. * Sanitization and draining the bilious ways
 - E. Draining the chanel of pancreas
370. Normal indexes the billirubin of blood:
- A. 0,10-0,68 mcmol/l
 - B. * 8,55-20,52 mcmol/l
 - C. 2,50-8,33 mcmol/l
 - D. 3,64-6,76 mcmol/l
 - E. 7,62-12,88 mcmol/l

371. Normal width of the common bile duct
- A. To 0,4 cm
 - B. * 0,5-0,7 cm
 - C. 0,8-1,3 cm
 - D. 1,4-2,0 cm
 - E. Over 2,0 cm
372. On a background choledocholithiasis needs a sick icterus
- A. To the urgent operation
 - B. Medicinal treatment
 - C. * To the urgent operation after preoperative preparation
 - D. Cannulations of the abdominal artery
 - E. Plazmopheresis
373. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
374. pathognomonic symptom at acute pancreatitis is:
- A. Pasternatskiy's sing
 - B. * Kulen's sing
 - C. Ker's sing
 - D. Lenander's sing
 - E. Roving's sing
375. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
376. Patient with a gangrenous cholecystitis is indicating:
- A. * Urgent operation
 - B. Operation at default of effect from conservative therapy
 - C. Conservative treatment
 - D. Deferred operation
 - E. A decision-making depends on age of patient
377. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
378. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer

- E. relapses more than 3 times per a year
379. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
380. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
381. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
382. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
383. Specify the optimum volume operation at acute biliar pancreatitis:
- A. Deleting exsudate from abdominal region
 - B. Decapsulation pancreas
 - C. Pancreatectomy
 - D. * Sanitization and draining bilious ways
 - E. Draining pancreas channel
384. Stone formation in a gall-bladder promote
- A. Infection, stagnation of bile, exchange violations, inflammatory processes in a bubble, constitution, enhanceable amount of bilious acids
 - B. * An infection, stagnation of bile, exchange violations, inflammatory processes in a bubble, sex, constitution
 - C. Stagnation of bile, inflammatory processes in a bubble, inflammatory processes in a bubble, half, enhanceable amount of bilious acids
 - D. An infection, stagnation of bile, exchange violations, inflammatory processes in a bubble, enhanceable amount of bilious acids
 - E. All answers correct
385. Stoneformation in a gall-bladder promote:
- A. * An infection, stagnation of bile, exchange violations, inflammatory processes in a bubble, sex, constitution
 - B. Infection, stagnation of bile, exchange violations, inflammatory processes in a bubble, constitution, raised amount of bilious acids

- C. Stagnation of bile, inflammatory processes in a bubble, inflammatory processes in a bubble, sex, raised amount of bilious acids
 - D. An infection, stagnation of bile, exchange violations, inflammatory processes in a bubble, raised amount of bilious acids
 - E. All answers correct
386. Symptoms perforation the gall-bladder are
- A. * Strengthening of pains, appearance Blumberg's sign on all stomach, irradiation the pain in a right shoulder
 - B. Megascopic sickly gall-bladder
 - C. Positive Ortner's sign
 - D. Positive Obraztsov's sign
 - E. All answers correct
387. The attack of hepatic (bilious) colic arises up:
- A. * Suddenly, acutely
 - B. After a initial period
 - C. Gradually, gradually
 - D. After the protracted starvation
 - E. After supercooling
388. The attack of hepatic (bilious) colic development
- A. * Suddenly, acutely
 - B. After a initial period
 - C. Gradually, gradually
 - D. After the protracted starvation
 - E. After supercooling
389. The characteristic laboratory sign of the acute uncomplicated cholecystitis is:
- A. Diastasia
 - B. * Leucocytosis
 - C. Hypoglycemia
 - D. Glucosuria
 - E. Hyperbilirubinemia
390. The Courvoisier's symptom is not observed at a cancer
- A. Heads of pancreas
 - B. Supraduodenal part of the common bile stone
 - C. Retroduodenal part of general bilious channel
 - D. Large duodenal papilla
 - E. * Gall-bladder
391. The index litogenic bile is determined correlation
- A. Cholesterol, bilirubine and lecithin
 - B. Billirubine, bilious acids and lecithin
 - C. Cholesterol, bilious acids and bilirubine
 - D. * Cholesterol, bilious acids and lecithin
 - E. Billirubine and lecithin
392. The laboratory signs of total pancreonecrosis is:
- A. Growth glucose concentration
 - B. Diminishing maintenance fibrinogenum
 - C. * Growth activity diastase
 - D. Diminishing activity diastase

- E. Growth index AST
393. The leading clinical symptoms the acute pancreatitis is:
- A. * Stomach-ache
 - B. Vomiting by „coffee-grounds”
 - C. Disuria
 - D. Febrile temperature of body
 - E. Lock
394. The liquid painted a bile in an abdominal region is not observed at
- A. * Break pus hydatidoma
 - B. To the protracted mechanical icterus
 - C. Spontaneous bilious peritonitis
 - D. Perforations of gall-bladder
 - E. Perforations the ulcer of duodenum
395. The liquid painted a bile in an abdominal region is not observed at
- A. Perforations the ulcer of duodenum
 - B. Perforations the gall-bladder
 - C. To the protracted mechanical icterus
 - D. * Break the pus hydatidoma
 - E. Spontaneous bilious peritonitis
396. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
397. The patients with a gangrenous cholecystitis is indication
- A. Conservative treatment
 - B. Deferred operation
 - C. A decision-making depends on age of patient
 - D. Operation at default of effect from medicinal therapy
 - E. * Urgent operation
398. The secretory function stomach is carried out the next membrane of stomach
- A. * mucous membrane
 - B. internal muscular layer
 - C. serosal
 - D. mucous submembrane
 - E. external layer
399. The special examination the extrahepatic bilious ways is absolutely indicated at
- A. Suspicion on stenosis of large duodenal papilla, expansion the common bile duct, mechanical icterus in the moment of operation
 - B. Suspicion on stenosis of large duodenal papilla, expansion the common bile duct, to the mechanical icterus in anamnesis, mechanical icterus in the moment of operation
 - C. Expansion the common bile duct, mechanical icterus in the moment of operation
 - D. Shallow stone in the common bile duct, suspicion on stenosis of large duodenal papilla, expansion the common bile duct, mechanical icterus in the moment of operation
 - E. * All right

400. The special research extrahepatic bilious ways is absolutely indicated at:
- A. Shallow stone in common bile duct, suspicion on stenosis the large duodenal papilla, expansion of the common bile duct, mechanical icterus in the moment of operation
 - B. Suspicion on stenosis of large duodenal papilla, expansion of the common bile duct, mechanical icterus in the moment of operation
 - C. At expansion of the common bile duct
 - D. * All right
 - E. All not right
401. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scary-ulcerous stenosis
 - B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
402. To complications of the acute calculary cholecystitis does not attribute:
- A. * Phlebeurysm gullet
 - B. Mechanical icterus
 - C. Cholangitis
 - D. Subhepatic abscess
 - E. Peritonitis
403. To complications of the acute stone cholecystitis does not attribute
- A. * Varicose widening the vein of gullet
 - B. Mechanical icterus
 - C. Cholangitis
 - D. Under hepatic abscess
 - E. Peritonitis
404. To early complications acute pancreatitis attribute:
- A. Shock
 - B. Acute hepatic-kidney insufficiency
 - C. Poured out peritonitis
 - D. Icterus
 - E. * All answers are faithful
405. To the gastric – intestinal bleeding of un ulcerous etiology belong
- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.
406. To the intraoperative methods research the extrahepatic bilious ways does not pertain
- A. Palpation of the common bile duct
 - B. Choledochoscopy
 - C. Intraoperative cholangiography
 - D. Sounding of the common bile duct
 - E. * Intravenous cholangiography
407. To the intraoperative methods of research the extrahepatic bilious ways all behaves, except for:

- A. Palpation of the common bile duct
 - B. Choledochoscopy
 - C. Intraoperative cholangiography
 - D. Soundings of the common bile duct
 - E. * Intravenous holangiography
408. Turner's sign at acute pancreatitis:
- A. Violet spots on face and trunk
 - B. * Cyanosis sides of stomach and trunk
 - C. Cyanosis skin of stomach
 - D. Ictericness round a belly-button
 - E. Cyanosis of hands
409. Udin's sign at a perforated ulcer is
- A. * feeling at palpation shove the gases which penetrate through the perforated opening
 - B. dulling perforated sound in the lateral departments of stomach
 - C. disappearance of hepatic dullness
 - D. irradiation pain in a shoulder or shoulder-blade
 - E. sickliness the back vault of vagina
410. Vomiting coffee-grounds is a characteristic sign
- A. * bleeding ulcer
 - B. penetrative ulcers
 - C. perforated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorus stenosis
411. Vomiting what arose up in 4-6 hours after eating characteristic for
- A. chronic alcoholic gastritis
 - B. cancer and ulcers of cardia
 - C. * pylorus ulcers
 - D. achalasia of gullet
 - E. ulcer and cancer the body of stomach
412. Voskresenskiy's sign at acute pancreatitis:
- A. * Absence pulsation the abdominal aorta
 - B. Sickliness in left costal-vertebral coal
 - C. Sickliness and proof tension the muscles in an epigastrium with passing to left subcostal area
 - D. Ictericness round a belly-button
 - E. Skin hyperesthesia in projection the gland
413. What nosotropic conditionality Voskresenskiy's sign at acute pancreatitis:
- A. * Inflammatory edema of pancreas
 - B. Reflex paresis of colon
 - C. Thrombosis of abdominal aorta
 - D. Embolism of abdominal aorta
 - E. Development of peritonitis
414. What a clinical flow can be at acute pancreatitis:
- A. Easy, middle, heavy
 - B. Acute, chronic
 - C. * Abortive, making progress
 - D. Edema, necrosis

- E. Any variant
415. What basic method the treatment of acute pancreatitis is:
A. Surgical
B. * Conservative
C. Homoeopathic
D. Physical therapy
E. A right answer is not present
416. What cages of pancreas are make glucagon:
A. клетки
B. клетки
C. -клетки
D. None of cages
E. D-клетки
417. What cages of pancreas are make insulin:
A. клетки
B. клетки
C. -клетки
D. None of cages
E. D-клетки
418. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodwnum?
A. * Tarry excrement
B. Presence on the formed excrement of strokes of red blood
C. Littlechanged blood in an excrement
D. Excrement of the raspberry colouring with the admixtures of mucus
E. Acholic excrement
419. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
A. perforation
B. perforation + bleeding
C. pylorus stenosis
D. malignization + penetration
E. * bleeding
420. What complications at a acute pancreatitis is behave to early:
A. * Peritonitis
B. Phlegmon retroperitoneum space
C. Formation of pseudocysts
D. Development of saccharine diabetes
E. Intestinal impassability
421. What complications at a acute pancreatitis is behave to late:
A. Peritonitis
B. * Phlegmon retroperitoneum space
C. Formation of pseudocysts
D. Development of saccharine diabetes
E. Intestinal impassability
422. What external signs are characteristic for the profuse bleeding from a gastric ulcer?

- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
423. What from operations does not execute at surgical treatment complicated acute pancreatitis:
- A. Through draining the stuffing-box bag
 - B. Abdominisation the pancreas
 - C. Omentopankreatopeksiy
 - D. Left-side resection of gland
 - E. * Pancreatojejunostomy
424. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol
 - B. Liciviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
425. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy
 - E. the all transferred does not belong
426. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
427. What hormone of pancreas has influences on metabolism glucose:
- A. * Insulin
 - B. Vasopressin
 - C. Adrenalin
 - D. Somatotropin
 - E. Tiroksin
428. What hormone of pancreas has influences on the exchange of fats:
- A. Insulin
 - B. Glyukagon
 - C. * Lipocainu
 - D. Adrenalin
 - E. Somatotropin
429. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E

- D. estrogens
 - E. STH
430. What is blood supply the body and tail pancreas:
- A. * Splenic artery
 - B. A.gastroduodenalis
 - C. A.gastrica sinistra
 - D. A.cystica
 - E. Variously
431. What is condition hematomesis at acute pancreatitis:
- A. Presence concomitant gastric ulcer
 - B. Presence concomitant gastritis
 - C. * Formation erosions in a stomach
 - D. Violation of microcirculation
 - E. Enzymes in blood
432. What is necrectomy:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. * Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
433. What is pancreas located in relation to a peritoneum:
- A. * Retroperitoneal
 - B. Mesoperitoneal
 - C. Intraperitoneal
 - D. All answers are incorrect
 - E. Variously
434. What is pancreatectomy:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. * Total delete of organ
 - E. There is not a faithful answer
435. What is resection the pancreas:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. * Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
436. What is sequestrotomy:
- A. * Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ

- E. There is not a faithful answer
437. What level diastase answers a acute pancreatitis:
A. 100-160
B. 200-500
C. * 600-1000
D. 1000-1500
E. 10-20
438. What level diastase answers a chronic pancreatitis:
A. * 200-500
B. 100-160
C. 600-1000
D. 1000-1500
E. 10-20
439. What level diastase answers pancreonecrosis:
A. 600-1000
B. 200-500
C. 100-160
D. * 1000-1500
E. 10-20
440. What localization of ulcer is most characteristic for the patients of elderly and senile age
A. * cardial department of stomach
B. overhead third of gullet
C. lower third of gullet
D. bulb of duodewnum
E. small curvature
441. What method diagnostics hte ulcerous illness most informing
A. * esophagogastroduodenoscopy
B. analysis of excrement on the hidden blood
C. X-ray
D. global analysis of blood
E. research of gastric secretion
442. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
A. On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
B. On blood volume
C. * On an arteriotony, pulse, state of patient
D. On a globular volume
E. On the level of thrombocytes
443. What most effective blocker secretion of pancreas at acute pancreatitis:
A. Cyanocobalamin
B. Ubretid
C. Arginine
D. * Sandostatin
E. Benzogeksoniy
444. What most effective treatment the formed uncomplicated cyst is:
A. External draining the cyst

- B. Marsupialization
 - C. Resection the cyst within the limits of the unchanged gland
 - D. * Cysticenterostomy
 - E. Cystogastrostomy
445. What most effective treatment the unformed complicated cyst is:
- A. Conservative treatment
 - B. * External draining cyst
 - C. Resection cyst within the limits of the unchanged gland
 - D. Cysticenterostomy
 - E. Cystogastrostomy
446. What most effective treatment the unformed uncomplicated cyst is:
- A. * Conservative treatment
 - B. External draining cyst
 - C. Resection cyst within the limits of the unchanged gland
 - D. Cysticenterostomy
 - E. Cystogastrostomy
447. What most optimum resort is which used for pathology the pancreas:
- A. Pyatigorsk
 - B. * Morshin
 - C. Kuyal'nik
 - D. Truskavets
 - E. Nemirov
448. What norm diastase is:
- A. * To 160
 - B. To 50
 - C. To 200
 - D. To 300
 - E. To 1200
449. What operation is most often used for localization the formed pseudocyst in the area of tail the pancreas:
- A. Cystojejunostomy on the eliminated loop
 - B. External draining the cyst
 - C. Cystogastrostomy
 - D. * Cystoduodenostomy
 - E. Cystoenteroanastomosis
450. What operation is used for suppuration the pseudocysts of pancreas:
- A. Cystojejunostomy on the eliminated loop
 - B. * External draining the cyst
 - C. Cystogastrostomy
 - D. Cystoduodenostomy
 - E. Cystoenteroanastomosis
451. What operation is used for the pseudocyst of pancreas in the 3th stage of its forming:
- A. * Cystojejunostomy on the eliminated loop
 - B. External draining the cyst
 - C. Cystogastrostomy
 - D. Cystoduodenostomy
 - E. Cystoenteroanastomosis

452. What operation on a pancreas is indicated at the oedematous form the acute pancreatitis:
- A. * Interference on a pancreas is not needed
 - B. Pancreatectomy
 - C. Omentopankreatopeksy
 - D. Abdominisation pancreas
 - E. Marsupialization
453. What preparation does behave to blocker H₂-retseptors?
- A. * tavegil
 - B. obzidan
 - C. hystdol
 - D. cerucal
 - E. oraza
454. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
455. What preparation is applied at violation the extrasecretory function of pancreas at a chronic pancreatitis:
- A. Pyracetam
 - B. Papaverin
 - C. Pantocrin
 - D. * Panzinorm
 - E. Panthenol
456. What preparation is applied at violation the extrasecretory function pancreas at a chronic pancreatitis:
- A. Pyracetam
 - B. Papaverin
 - C. Pantocrin
 - D. * Mezim-forte
 - E. Panthenol
457. What preparation is attributed to the proteases inhibitors:
- A. Garamycin
 - B. Gaviskon
 - C. * Gordox
 - D. Halidor
 - E. Gramicidin
458. What preparation is attributed to the proteases inhibitors:
- A. Tocopherol
 - B. Triampur
 - C. Tagamet
 - D. * Trasylol
 - E. Trypsin
459. What preparation is not attributed to the inhibitors proteases:
- A. Pantripin

- B. Kontrikal
 - C. Gordox
 - D. Trasylol
 - E. * Trypsin
460. What preparations from the cytostatic group use for intensifying the chronic pancreatitis:
- A. Cyanocobalamin
 - B. Methyluracil
 - C. * 5-fluorouracil
 - D. Furadolizon
 - E. Mezimforte
461. What preparations, except for other properties, own yet and a bacteriostatic effect on *Helicobacter pylori*
- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
462. What primary purpose treatment the patient with fatty pancreatonecrosisto the operation is:
- A. Liquidations the pain
 - B. * Disintoxication the organism
 - C. Liquidations crampy the big duodenal papilla
 - D. Declines secretory activity the pancreas
 - E. Improvements microcirculation
463. What primary purpose treatment the patients with the heavy form of hemorrhagic pancreatonecrosis to the operation is:
- A. Liquidations the pain
 - B. * Disintoxication the organism
 - C. Liquidations crampy the big duodenal papilla
 - D. Declines secretory activity the pancreas
 - E. Improvements microcirculation
464. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
465. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
466. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;

- E. 6 – 20 hours
467. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- hypocalcemia
 - * hypokaliemia
 - Hyponatremia
 - Hypercalcemia
 - hyperkaliemia
468. When apply Teylor's method at ulcerous illness
- * at conservative treatment perforated ulcers
 - at conservative treatment sanguifluous ulcers
 - at conservative treatment penetration ulcers
 - at conservative treatment malignization ulcers
 - at conservative treatment of cicatrical pylorostenosis
469. With the purpose preparation to operation the patient with the protracted icterus of bail-stone diseases origin and phenomena hepatic insufficiency, it is necessary to make
- Endoscope papilotomy, throughskin transhepatic draining of hepaticocholedoch, endoscope papilotomy and nosebilliar draining
 - Throughskin transhepatic draining of the hepaticocholedoch, enteropersorption, lymphopersorption or hemopersorption, endoscope papilotomy and nosebilliar draining
 - * All right
 - Laparoscopic cholecystostomy, nosebilliar draining, throughskin transhepatic draining of hepaticocholedoh
 - Laparoscopic cholecystostomy, nosebilliar draining, throughskin transhepatic draining of hepaticocholedoch, endoscope papilotomy and nosebilliar draining
470. With transferring of stone from a gall-bladder in the common bile duct does not develop:
- Hepatic colic
 - Icterus
 - Festering cholangitis
 - Stenotic papillitis
 - * Syndrome by Badd-Khiary
471. A patient 2 hours ago fallen down from the first floor. State heavy. There are signs of the diffusive peritonitis. Survey sciagraphy of stomach is executed. What from roentgenologic symptoms diagnosis of break of cavernous organ will confirm?
- hight standings of diaphragm dome
 - Kloybers bowls
 - * free gas in an abdominal region
 - a free liquid in an abdominal region
 - infiltration in an abdominal region
472. A patient 2 hours ago got a blow in a stomach. Delivered in a urgent clinic in a grave condition with suspicion on the break of cavernous organ. What method of roentgenologic research will allow to specify a diagnosis?
- laparocentzis
 - laparoscopy
 - * sciagraphy of stomach
 - contrasting X-ray examination
 - irrigoscopy

473. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appeared. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- Combination perforation with bleeding
 - Perforation
 - Malignization of ulcer
 - Stenosis of ulcer
 - * Gastroenteric bleeding
474. A patient entered to urgent with closed trauma of stomach. At an inspection are positive symptoms the irritation of peritoneum. The damage of internalss is suspected. What method of diagnostics does use for confirmation the diagnosis?
- angiography
 - X-ray examination
 - Sonography
 - * diagnostic laparotomy
 - diagnostic puncture
475. A patient entered urgent clinic in a grave condition. Pulse is 112 in min., AP - 11060, T - 35,1 C. It was found diagnosis the diffusive peritonitis. What treatment is rotined a patient?
- laparotsentez
 - laparoscopy
 - * operation
 - antibiotic
 - puncture
476. A patient entered urgent in a clinic on 3 days from the beginning of disease. The state is heavy, adynamic. Face of Hippocrates, pulse 138 in 1 min, AP - 8040. A stomach is blown away, moderato sickly at palpation. Shchotkin-Blyumbergs symptom is doubtful. A liquid is determined in the lower departments the stomach. A liquid, stinking excrement departs through gaping sphincter. That does explain the state of patient?
- intestinal impassability
 - * diffusivr peritonitis
 - mesenteric ischemia
 - toxic dilatation of colon
 - acute pancreatitis
477. A patient got the trauma of stomach 3 hours ago. Delivered in a urgent clinic with complaints about tormina. The lines of person are focus. Positive symptoms the irritation of peritoneum. At survey X-ray examination is found out free gas under the dome of diaphragm. A diagnosis is set: break of cavernous organ. What type of treatment is most expedient?
- cold on a stomach
 - laparotsentez
 - laparoscopy
 - * operative
 - antibiotic
478. A patient grumbles about great pain in a stomach, which arose up suddenly 6 hours ago. The diagnosis of peritonitis is set. What symptom is most characteristic for this diagnosis?
- increases of temperature
 - tachycardia
 - leucocytosis
 - tension of muscles the abdominal wall

- E. * irritations of peritoneum
479. A patient suffers a stenocardia. Became ill suddenly after the physical loading. Complaints about pain in a epigastric area, which spread on the right half of stomach. Positive Shchotkin-Blyumbergs symptom. Rectal – overhang and painfulness the front wall of rectum. It is not discovered the free gas on the survey sciagram of stomach. What is most reliable diagnosis?
- A. abdominal form of heart attack the myocardium
 - B. acute cholecystitis
 - C. acute pancreatitis
 - D. * peritonitis
 - E. peritonitis of apendicular character
480. A prophylaxis and treatment of postoperative enteroplegias at peritonitis is
- A. gastrotomy
 - B. gastrointestinotomy
 - C. * nasogastrointestinal intubation
 - D. intubation of abdominal region
 - E. intubation of stuffing-box bag
481. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
482. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
483. Acute pancreatitis with a heavy flow treat in terms:
- A. Ambulatory
 - B. Permanent establishment
 - C. * Department of intensive therapy
 - D. All answers are faithful
 - E. A faithful answer is not present
484. After time of origin complications acute pancreatitis select:
- A. Premature and remote
 - B. Urgent and deferred
 - C. Primary and second
 - D. * Early and late
 - E. Any of variants faithful
485. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I
 - E. saved after all transferred operations

486. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred
 - E. not saved after all operations
487. Agents, which cause peritonitis, can be all except for:
- A. urines at the break of urinary bladder
 - B. tables of contents the stomach during the perforation of ulcer
 - C. to blood at the trauma of stomach
 - D. biles during the perforation of gall-bladder
 - E. * air in an abdominal region after laparoscopy research
488. All surgical interferences at the destructive forms of acute pancreatitis divide on:
- A. * Early, late, deferred operations
 - B. Primary, second, repeated operations
 - C. Invasion, not invasion operations
 - D. Complicated, operations are not complicated
 - E. Not divided
489. Among acute surgical diseases acute pancreatitis occupies:
- A. First place
 - B. * Third place
 - C. Fifth place
 - D. Second - third place
 - E. Most widespread
490. As the first period flowing of acute pancreatitis is named:
- A. * Haemodynamic violations and pancreatic shock
 - B. To functional insufficiency of parenchymatous organs
 - C. Degenerative and festerings complications
 - D. All answers are faithful
 - E. A right answer is not present
491. As the second period flowing of acute pancreatitis is named:
- A. * Haemodynamic violations and pancreatic shock
 - B. To functional insufficiency of parenchymatous organs
 - C. Degenerative and festerings complications
 - D. All answers are faithful
 - E. A right answer is not present
492. As the third period flowing of acute pancreatitis is named:
- A. Haemodynamic violations and pancreatic shock
 - B. * To functional insufficiency of parenchymatous organs
 - C. Degenerative and festerings complications
 - D. All answers are faithful
 - E. A right answer is not present
493. Aseptic inflammation of peritoneum can be caused:
- A. by a collibacillus
 - B. by staphylococuss
 - C. * by pancreatitis juice

- D. by an abscess
 - E. by intestinal maintenance
494. At a subdiaphragmatic abscess in a clinical picture characteristically all following, except for:
- A. declines the respiratory excursion of lights.
 - B. high standing of diaphragm dome.
 - C. concord pleurisy.
 - D. basale atelectasis of lights.
 - E. * blood spitting
495. At a subhepatic abscess can take place all, except for:
- A. pains in a thorax with an irradiation in a supraclavicular area
 - B. reactive pleurisy
 - C. * Courvoisier's symptom
 - D. Senator's symptom
 - E. Dyushen's symptom
496. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
497. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
498. At festering peritonitis the disorder of hemodynamics not conditioned:
- A. by the decline of volume the circulatory blood
 - B. * by the increase of volume the circulatory blood
 - C. by the decline of tone the vascular wall
 - D. by the change of properties the hemorheologys
 - E. by violation of cardiovascular activity
499. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
500. At pancreatitis abscesses and infected necroses execute such operations, except for:
- A. Opening of abscess with draining
 - B. Pancreaticnecrosekvestrektomy
 - C. Pancreaticsekvestrektomy
 - D. Pancreaticsekvestrektomy with laparostomy
 - E. * Total pancreatotomy
501. At peritonitis intestinal impassability develops, as a rule:

- A. mechanical
 - B. dynamic
 - C. spastic
 - D. * paralytic
 - E. mixed
502. At peritonitis of violation the proteometabolism characterized:
- A. by the increase of concentration the albumen
 - B. by the increase of concentration the globulins
 - C. diminishing of concentration the albumen
 - D. * diminishing of the albumen - globulins coefficient
 - E. by the increase of the albumen - globulins coefficient
503. At suspicion on a subdiaphragmatic abscess the followings methods of diagnostics are rotined, except for:
- A. * laparoscopy.
 - B. Sonography.
 - C. X-ray examination of thorax.
 - D. X-ray examination of abdominal region.
 - E. computer tomography
504. At suspicion on the abscess of Duglas space all followings methods of diagnostics are rotined, except for:
- A. rectal inspection.
 - B. * proctoscopy.
 - C. Sonography.
 - D. computer tomography.
 - E. vaginal research
505. At the duodenum ulceroperation of choice is
- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
506. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
507. At the III item of activity hemostasis and III sage blood loss from the I – III sage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
508. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);

- B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
509. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
510. At what disease pain of girdle character is characteristic:
- A. Gastric ulcers
 - B. Acute cholecystitis
 - C. Intestinal impassability
 - D. * Acute pancreatitis
 - E. Acute cystitis
511. At what pathology is absence pulsation of abdominal aorta
- A. * Acute cholecystitis
 - B. Acute pancreatitis
 - C. Perforated ulcer
 - D. Intestinal obstruction
 - E. Acute appendicitis
512. At what pathology is appearance cyanosis the sides of stomach:
- A. * Acute pancreatitis
 - B. Perforated ulcer
 - C. Acute cholecystitis
 - D. Intestinal obstruction
 - E. Acute appendicitis
513. At what pathology is appearance of sickliness in left costal-vertebral coal:
- A. * Acute pancreatitis
 - B. Acute cholecystitis
 - C. Perforated ulcer
 - D. Intestinal obstruction
 - E. A right answer is not present
514. Atypical clinical motion of postoperative peritonitis is conditioned setting in a postoperative period:
- A. spasmolytic
 - B. anticoagulant
 - C. * anaesthetic
 - D. analeptics
 - E. cardiac
515. Bergman's sign is characteristic for
- A. * bleeding ulcer
 - B. for cicatricial-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers

516. Berhstein's sign characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
517. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
518. Blood loss I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
519. Blood loss II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
520. Blood loss III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
521. Bonde's sign at acute pancreatitis:
- A. * Swelling the stomach only in epigastrium
 - B. Cyanosis sides of stomach and trunk
 - C. Cyanosis skin of stomach
 - D. Ictericness round a belly-button
 - E. Cyanosis of hands
522. Characteristic complication acute pancreatitis is:
- A. * Pancreonecrosis
 - B. Pylephlebitis
 - C. Hepatocirrhosis
 - D. Veritable pancreas cyst
 - E. Hepatitis
523. Characteristic complication the acute pancreatitis is:
- A. Paranephritis
 - B. Douglas's abscess
 - C. Pylephlebitis

- D. Cyst of pancreas
 - E. * Pancreonecrosis
524. Conservative treatment the acute pancreatitis includes:
- A. * Hunger
 - B. Povzner's diet №5
 - C. Povzner's diet №15
 - D. Moderation in a meal
 - E. Morning gymnastics
525. De-Cerven's sign is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignitized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
526. Decision role in differential diagnostics of peritonitis and acute vascular purpura is a symptom:
- A. * hemorrhagic rash on a skin
 - B. acute pain in a stomach
 - C. systole noise above the abdominal department of aorta
 - D. absence of pulsation of abdominal department of aorta
 - E. melena at rectal research
527. Decision role in differential diagnostics of peritonitis and break the bone of pelvis is:
- A. to appoint antibiotics
 - B. to appoint anaesthetic
 - C. to execute laparocentthesis
 - D. * to execute laparoscopy
 - E. to execute survey sciagraphy of abdominal region
528. Decision role in differential diagnostics the peritonitis and dissecting aneurysm of aorta , there is a symptom:
- A. aperistalsis
 - B. acute pain in a stomach
 - C. * systole noise above the abdominal department of aorta
 - D. absence of pulsation of abdominal department of aorta
 - E. melena at rectal research
529. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
530. Diffusive festering peritonitis can be investigation of all transferred diseases, except for:
- A. perforations Meckel's diverticulum
 - B. destructive appendicitis
 - C. * stenosis of large duodenal nipple
 - D. Richter strangulation of hernia
 - E. acute intestinal impassability

531. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- * Bergman's sign
 - Spazarskiy's sign
 - Mendel's sign
 - De Keven's sign
 - Eleker's sign
532. Draining the abdominal region is conducted at:
- phlegmonous appendicitis without exudation
 - * gangrenous appendicitis with exudation
 - phlegmonous appendicitis with serosal exudation odourless
 - there is not a right answer
 - catarrhal appendicitis
533. Duration the period of primary shock at a perforated ulcer
- * 3-6 hours
 - 6-12 hours
 - 1-3 hours
 - 12-24 hours
 - 24-36 hours
534. Early complications the acute pancreatitis is not:
- * Fistula of pancreas
 - Pancreatic shock
 - Collapse
 - Ferment peritonitis
 - Pancreatic delirious syndrome
535. Eleker's sign is characteristic for
- * perforated ulcers
 - bleeding ulcer
 - penetrated ulcers
 - malignized ulcers
 - for cicatricial-ulcerous pylorostenosis
536. ERCP apply at:
- Postcholecystectomy syndrome
 - Stenosing papillitis
 - * Stenosis the supraduodenal department of choledoch
 - Stricture the terminal department of choledoch
 - Mechanical icterus
537. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- tumours
 - ulcers
 - bleeding polypuses
 - erosions
 - * changes of evacuation function
538. Fibrinogenous impositions on a peritoneum are not at peritonitis:
- * serosal
 - fibrinogenous
 - festering
 - putrid

- E. excrement
539. For a patient, suffering ulcerous illness, sudden great pain in an epigastrium and unclear symptoms of peritonitis appeared. During subsequent days the display of these symptoms diminished, the state of patient had become better. It is possible to suppose at such clinic
- typical perforation the ulcer
 - * covered perforation
 - preperforative state
 - intensifying of ulcerous illness
 - there is not a right answer
540. For a perforation declivous organ all is characteristic in a free abdominal region, except for:
- acute began pains.
 - wooden belly.
 - collapse.
 - * oliguria.
 - tachycardia.
541. For bleeding ulcer characteristic sign is
- * pain in an epigastrium;
 - knife-like pain;
 - signs irritation of peritoneum;
 - presence fresh blood in incandescence
 - melena;
542. For bleeding ulcer characteristically
- * melena
 - tension the muscles of front abdominal wall
 - Spazarskiy's sing
 - sickliness the back vault of vagina
 - irradiation pain in a shoulder or shoulder-blade
543. For diagnostics of acute pancreatitis most informing is:
- * Sonography
 - CT
 - Cholangiography
 - Esophagogastroduodenoscopy
 - Colonoscopy
544. For motion of disease ulcerous illness of middle weight characteristically
- development of complications
 - * relapses 1-2 times per a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year
 - 3 and anymore relapses on a year
545. For pancreonecrosis characteristically is:
- * Rapid progress, strengthening pain, proof enteroplegia, growth haemodynamic violations
 - Rapid progress, diminishing pain, strengthening of моторики of intestine, growth haemodynamic violations
 - Slow progress, strengthening pain, phase of imaginary prosperity.
 - Abortive flow, toxemia, development shock.
 - Appearance light interval

546. For perforative appendicitis characteristically
- tension the muscles of front abdominal wall
 - there is the sudden strengthening of stomach-aches
 - rapid growth of clinical picture the peritonitis
 - Razdolskuy's symptom
 - * all transferred
547. For perforated ulcer characteristically
- * tension the muscles of front abdominal wall
 - melena
 - vomiting by coffee-grounds
 - high intestinal impassability
 - vomiting stagnant gastric maintenance
548. For peritonitis in the first 24 hours not typical
- aperistalsis intestine
 - * Kullenkamp's symptom
 - Tachycardia
 - dry language
 - tension the muscles of abdominal wall and positive Shchetkin-Blyumberg's symptom
549. For peritonitis there is a not characteristic symptom:
- Shchetkin-Blyumberg's symptom
 - Voskresenskiy's symptom
 - Kulenkampf's symptom
 - * Moebius's symptom
 - Krymov's symptom
550. For pneumoperitoneum is characteristic symptom
- * Zhober's;
 - Khelatid's;
 - Podlag's;
 - Vigats's;
 - Udin's.
551. For the abortive flowing characteristically:
- * A process limited to the acute edema with convalescence in 7-8 days
 - A process limited to tearing away of the pathologically changed gland
 - A process limited to tearing away of the pathologically unchanged gland
 - A disease completed so not attaining clinical displays
 - Changes from the side of organ are not present
552. For the heavy flow of ulcerous illness characteristically
- 2 and anymore relapses on a year
 - * 3 and anymore relapses on a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year
 - 6 and more relapses are on a year
553. For the heavy flow of ulcerous illness characteristically
- * development of complications
 - seasonal exacerbation more not frequent 1-2 times per a year
 - 1-2 relapse on a year

- D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days
554. For the late stage of peritonitis all is characteristic, except for:
- A. swelling of stomach
 - B. hypovolemia
 - C. disappearance of intestinal noises
 - D. hypoproteinemia
 - E. * increased peristalsis
555. For the reactive stage of festering peritonitis not characteristically:
- A. acute sickliness the stomach at palpation
 - B. positive Shchetkin-Blyumberg's symptom
 - C. tension of muscles the front abdominal wall
 - D. * face of «Hippocrates»
 - E. tachycardia
556. For the terminal stage of peritonitis not characteristically:
- A. tachycardia
 - B. * bradycardia
 - C. hyperthermia
 - D. falling of arteriotony
 - E. dynamic intestinal impassability
557. For what pathology appearance of violet spots on the skin of person and trunk is characteristic:
- A. Perforated ulcer
 - B. Acute cholecystitis
 - C. * Acute pancreatitis
 - D. Intestinal obstruction
 - E. Acute appendicitis
558. For what purpose in treatment of diffusive festering peritonitis does execute nasointestinal intubation?
- A. account of losses the liquid through a gastroenteric highway.
 - B. control of electrolyte composition the intestinal maintenance
 - C. * prophylaxis of intestinal impassability
 - D. stimulation of the intestinal peristalsis
 - E. suppression of the intestinal peristalsis
559. From what department degestive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
560. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm

561. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
562. Holsted's sign at acute pancreatitis:
- A. Violet spots on face and trunk
 - B. Cyanosis sides of stomach and trunk
 - C. * Cyanosis skin of stomach
 - D. Ictericity round a belly-button
 - E. Cyanosis of hands
563. How is the diagnosis of the general peritonitis set to the operation?
- A. roentgenologic
 - B. anamnestic
 - C. by laboratory determination the signs of inflammatory reaction
 - D. * on clinical signs
 - E. on the level secretion the gastric juice
564. How many is the period of haemodynamic violations and pancreatic shock lasts:
- A. * 1-3 days
 - B. 3-7 days
 - C. More 7 days
 - D. 2 weeks
 - E. 2 hour
565. How many is the period of degenerative and festering complications lasts:
- A. 1-3 days
 - B. 3-7 days
 - C. * More 7 days
 - D. 2 weeks
 - E. 2 hour
566. How many is the period of functional insufficiency of parenchymatous organs lasts:
- A. 1-3 days
 - B. * 3-7 days
 - C. More 7 days
 - D. 2 weeks
 - E. 2 hour
567. In a patient with pains in an epigastric area, what was displaced from a right iliac area. There was nausea and single vomiting. A patient accepted an analgin and put a hot-water bottle to the stomach, pains calmed down whereupon. On 2 days pains recommenced, spread on all stomach, the frequent vomiting appeared. The state of patient is heavy. Consciousness entangled. Euphoria. Pulse 128 in min, AP - 95/60. Language is dry. A stomach is tense in all departments. Temperature 37,2. Leucocytes in blood of 18? 109/l. Diagnosis
- A. * acute appendicitis, terminal stage of peritonitis
 - B. typhoid, perforation of typhoidal ulcer
 - C. perforation gastric ulcer
 - D. gastric bleeding
 - E. there is not a right answer

568. In classic motion of peritonitis select the stages:
- early, intermediate, late
 - reactive, intermediate, late
 - toxic, intoxication, terminal
 - * reactive, toxic, terminal
 - reactive, toxic, late
569. In the dynamics of acute peritonitis it is necessary positive to consider
- * decline of amount the leucocytes
 - increase of amount the leucocytes
 - leucocytosis with the change of leukocytic formula to the left
 - growth of the leukocytic index intoxication
 - leukopenia
570. In treatment of the diffusive peritonitis of appendicitis origin a basic value has
- * all transferred
 - antibacterial therapy
 - correction water-electrolyte violations
 - removal of source of peritonitis
 - sanitization of the abdominal region
571. In treatment of the diffusive peritonitis of appendicitis origin a basic value has
- correction water-electrolyte violations
 - sanitization of abdominal region
 - removal the source of peritonitis
 - * all answers are faithful
 - antibacterial therapy
572. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?
- small curvature of stomach;
 - back wall of stomach, nearer to small curvature;
 - large curvature of stomach
 - * cardiac part of stomach;
 - pylorus.
573. In what vein is a venous outflow carried out in from a stomach?
- * V. Portae;
 - V. odd;
 - V. pair;
 - V. overhead hollow;
 - V. lower hollow;
574. Indication to early operative interference at acute pancreatitis is:
- Acute pancreatolysis
 - Acute oedematous pancreatitis
 - * Progressive multiple organ failure what not added conservative therapy during 48-72 hours
 - Acute fatty pancreatitis
 - Forming of pseudocyst
575. Kerte's sing at acute pancreatitis:
- Absence pulsation the abdominal aorta

- B. Sickliness in left costal-vertebral coal
 - C. * Sickliness and proof tension the muscles in an epigastrium with passing to left subcostal area
 - D. Icteritiousness round a belly-button
 - E. Skin hyperesthesia in projection the gland
576. Kulen's sign at acute pancreatitis:
- A. Violet spots on face and trunk
 - B. Cyanosis sides of stomach and trunk
 - C. Cyanosis skin of stomach
 - D. * Icteritiousness round a belly-button
 - E. Cyanosis of hands
577. Laparostomy at the diffusive peritonitis used with a purpose
- A. * to repeated sanitization the abdominal region
 - B. extracorporal dialysis
 - C. hemosorption
 - D. laparoscopy
 - E. stimulations the peristalsis
578. Large ulcer is an ulcer measuring
- A. 1-4 cm
 - B. 1-3 cm
 - C. 3-5 cm
 - D. 2- 6 cm
 - E. * 2-3 cm
579. Least answers a subhepatic abscess:
- A. Senator's symptom
 - B. Dyushen's symptom
 - C. Liten's symptom
 - D. * Rovzing's symptom
 - E. Shchotkin-Blyumberg's symptom
580. Little ulcer it is an ulcer measuring
- A. * to 0,5 cm
 - B. 0,5-1 cm
 - C. to 1,0 cm
 - D. 3 to 1,5 cm
 - E. 0,5-1,5 cm
581. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
582. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood

583. Melena is a characteristic sign
- * bleeding ulcer
 - for cicatrical-ulcerous pylorus stenosis
 - perforeted ulcers
 - penetration ulcers
 - malignization ulcers
584. Mayo-Robson's sign at acute pancreatitis:
- Absence pulsation the abdominal aorta
 - * Sickliness in left costal-vertebral coal
 - Sickliness and proof tension the muscles in an epigastrium with passing to left subcostal area
 - Icteritiousness round a belly-button
 - Skin hyperesthesia in projection the gland
585. Middle laparotomy must be conducted at:
- * diffusive peritonitis
 - local unlimited peritonitis
 - abscess of Duglas space
 - periappendiceal infiltration
 - acute appendicitis
586. Mondor's sign at acute pancreatitis:
- * Violet spots on face and trunk
 - Cyanosis sides of stomach and trunk
 - Cyanosis skin of stomach
 - Icteritiousness round a belly-button
 - Cyanosys of hands
587. Most frequent reason of peritonitis
- posoperative complication
 - acute trauma of stomach
 - acute cholecystitis
 - * acute appendicitis
 - acute intestinal impassability
588. Most informing method at a bleeding ulcer
- survey sciagraphy the organs of abdominal region
 - * EFGDS
 - sciagraphy the stomach with contrasting
 - Sonography
 - Laparoskopy
589. Name class complications of ulcerous illness
- bleeding, pyloristhenosis, second pancreatitis, malignization, perforation
 - * perforation, penetration, bleeding, pyloristhenosis, malignization
 - malignization, pyloristhenosis, penetration, anaemia, perforation
 - bleeding, perforation, second pancreatitis, anaemia, malignization
 - perforation, peritonitis, pancreatitis, bleeding, penetration
590. Name most frequent complication after ERDPH:
- * Pancreatitis
 - Cholangitis

- C. Pancreatic sepsis
 - D. Reactive cholecystitis
 - E. Obstructing papillitis
591. Name principal reason the acute pancreatitis:
- A. Trauma the pancreas
 - B. * Bile-stone diseases
 - C. Alimentary factor
 - D. Chronic alcoholic pancreatitis
 - E. Cardiospasm
592. Name the optimum volume of operation at acute biliary pancreatitis:
- A. Deleting exsudate from an abdominal region
 - B. Decapsulation pancreas
 - C. Pancreatectomy
 - D. * Sanitization and draining the bilious ways
 - E. Draining the chanel of pancreas
593. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
594. pathognomonic symptom at acute pancreatitis is:
- A. Pasternatskiy's sing
 - B. * Kulen's sing
 - C. Ker's sing
 - D. Lenander's sing
 - E. Roving's sing
595. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
596. Peritonitis does not develop at the next form of acute appendicitis
- A. * catarrhal
 - B. phlegmonous
 - C. gangrenous
 - D. perforative
 - E. gangrenous-perforative
597. Postoperative peritonitis is characterized a flow:
- A. typical
 - B. * atypical
 - C. stormy
 - D. with the expressed pain reaction
 - E. with the expressed intoxication
598. Preoperated complication of acute appendicitis

- A. * diffusive peritonitis
 - B. intra-abdominal bleeding
 - C. suppuration of wound
 - D. evisceration wounds
 - E. there are not a right answer
599. Preoperative preparation for patients from peritonitis does not provide:
- A. corrections the violations cardiovascular system
 - B. * laparocentzis
 - C. declines the intoxication
 - D. corrections the exchange violations
 - E. struggle with hy hypovolemia
600. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
601. Relaparotomy at diffusive festering peritonitis pursues:
- A. * repeated revision of abdominal region
 - B. repeated laparoscopy of abdominal region
 - C. repeated endoscopy of abdominal region
 - D. normalization the function of gall-bladder
 - E. normalization the function of urinary bladder
602. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
603. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
604. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
605. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct

606. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
607. Specific symptom of perforation declivous organ in a free abdominal region is:
- A. high leucocytosis.
 - B. absence of intestinal noises.
 - C. * pneumoperitoneum.
 - D. positive symptoms of irritation the peritoneum.
 - E. dulling of the percussion sound in the gently sloping places of abdominal region
608. Specify a criterion which grounds the choice of middle laoarotomy access at the deffusion festering peritonitis:
- A. minimum trauma the abdominal wall
 - B. minimum cut
 - C. * valuable revision the abdominal region.
 - D. minimum blood loss.
 - E. minimum level of infecting the wound
609. Specify obligatory measures which are conducted during an operation concerning widespread fibrinopurulent peritonitis:
- A. removal the source of peritonitis.
 - B. sanitization of the abdominal region.
 - C. decompression of intestine.
 - D. draining of abdominal region.
 - E. * all answers are faithful
610. Specify possible reasons of the pseudoperitoneal syndrome:
- A. uremia.
 - B. porphyria.
 - C. diabetic crisis.
 - D. nephrocolic.
 - E. * all transferred
611. Specify reason of use the derivatives of metronidasoli as an obligatory component of antibacterial therapy of widespread peritonitis?
- A. * removal of anaerobic microflora.
 - B. removal of mycotic flora.
 - C. removal of microflora of aerobic.
 - D. prophylaxis of intestinal worm invasion.
 - E. prophylaxis of widespread candidosis
612. Specify the most rational way the introduction of antibacterial preparations in treatment of diffusive festering peritonitis:
- A. peroral
 - B. hypodermic
 - C. intramuscular
 - D. * intravenous
 - E. intraperitoneal

613. Specify the optimum volume operation at acute biliar pancreatitis:
- Deleting exsudate from abdominal region
 - Decapsulation pancreas
 - Pancreatectomy
 - * Sanitization and draining bilious ways
 - Draining pancreas channel
614. Symptom of the toxic «scissors» at peritonitis it:
- increase of temperature and pulse
 - diminishing of temperature and pulse
 - * diminishing of temperature and increase of pulse
 - increase of temperature and diminishing of pulse
 - increase of temperature and diminishing of breathing frequency
615. Symptom of toxic «scissors» at the peritinitis it is correlation:
- * pulse and temperature
 - arteriotony and pulse
 - arteriotony and temperature
 - rectal and axillar temperature
 - rate breathings and temperatures
616. Tactic of surgeon at periappendiceal mas:
- operative treatment
 - * conservative treatment, systematic looking after a patient
 - there is not a right answer
 - supervision
 - punction
617. Tension of abdominal wall and stage peritonitis at acute appendicitis
- * absents, a stomach is swollen - terminal
 - absents, a stomach is not swollen - terminal
 - expressed, a stomach is not swollen - terminal
 - absents, a stomach is swollen - toxic
 - absents, a stomach is swollen – initial
618. Tension of muscles the right iliac area at the perforation of duodenal ulcer is explained
- by development of the diffusive peritonitis
 - by reflex connections through medullispinal nerves
 - * flowing down of gastric content in a right lateral channel
 - entering of air abdominal region
 - viscero0visceral connections with a vermicular appendix
619. The best method of treatment the subhepatic abscess is:
- thoracolaparotomy
 - lumbotomy
 - double-stage transpleural approach
 - laparotomy by Fedorov
 - * extrapleural extra-peritoneal method
620. The best variant the treatment of subhepatic abscess is:
- conservative treatment
 - extra-peritoneal section and draining
 - laparotomy, section and tamponing of cavity
 - * punction of abscess by a thick needle under control Sonography

- E. all transferred right
621. The complex treatment of festering peritonitis does not provide for:
- delete of primary hearth
 - * vagotomy
 - correction the metabolic violations
 - adequate therapy by antibiotics
 - struggle of paresis the intestine
622. The diagnostics criteria of the anaerobic peritonitis is
- stormy progress of disease
 - heavy festering intoxication
 - expressed enteroplegia
 - abundant amount of exsudate green-brown colors
 - * all is transferred
623. The exsudate painted blood in an abdominal region is observed always, except for:
- * tubercular peritonitis
 - violations of extra-uterine pregnancy
 - mesenteric ischemia
 - acute pancreatitis
 - twisted oothecoma
624. The favourable result of treatment the patients with peritonitis provide:
- early surgical interference
 - adequate operation
 - intravenous antibacterial therapy
 - methods of extracorporal dialysis
 - * all is transferred
625. The laboratory signs of total pancreonecrosis is:
- Growth glucose concentration
 - Diminishing maintenance fibrinogenum
 - * Growth activity diastase
 - Diminishing activity diastase
 - Growth index AST
626. The leading clinical symptoms the acute pancreatitis is:
- * Stomach-ache
 - Vomiting by „coffee-grounds”
 - Disuria
 - Febrile temperature of body
 - Lock
627. The leading symptom of peritonitis is:
- stomach-ache
 - enteroplegia
 - swelling of stomach
 - * symptoms the irritation of peritoneum
 - symptoms of the intestinal impassability
628. The methods of disintoxication at peritonitis are not:
- lymphosorbtion
 - hemisorption

- C. enterosorbition
 - D. plasmapheresis
 - E. * antibiotic
629. The most informing method the instrumental diagnostics of peritonitis is:
- A. * survey sciagraphy of abdominal region
 - B. lasparoscopy
 - C. angiography
 - D. gastroscopy
 - E. colonoscopy
630. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
631. The secretory function stomach is carried out the next membrane of stomach
- A. * mucous membrane
 - B. internal muscular layer
 - C. serosal
 - D. mucous submembrane
 - E. external layer
632. The sign of the diffusive peritonitis is not
- A. * visible peristalsis
 - B. absence the peristaltic intestinal noises
 - C. sickliness of stomach at palpation in all departments
 - D. even tension the muscles of front abdominal wall
 - E. positive Shchetkin-Blyumberg's symptom
633. There is vomiting at peritonitis, as a rule:
- A. single
 - B. episodic
 - C. * frequent
 - D. abundant
 - E. scanty
634. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scarry-ulcerous stenosis
 - B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
635. To early complications acute pancreatitis attribute:
- A. Shock
 - B. Acute hepatic-kidney insufficiency
 - C. Poured out peritonitis
 - D. Icterus
 - E. * All answers are faithful
636. To the gastric – intestinal bleeding of un ulcerous etiology belong

- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.
637. To the initial phase the peritonitis of appendicle origin does not behave
- A. sickliness the pelvic peritoneum at rectal research
 - B. tachycardia
 - C. * noticeable electrolyte changes
 - D. tendency to growth leucocytosis
 - E. tension of muscles the abdominal wall
638. To the late stage of peritonitis of appendicle origin does not behave
- A. dehydration
 - B. swelling of stomach
 - C. * increased peristalsis
 - D. hypoproteinemia
 - E. disappearance of intestinal noises
639. To the local isolated peritonitis does not attribute:
- A. subdiaphragmatic abscess
 - B. subhepatic abscess
 - C. interintestinal abscess
 - D. * primary idiopathic peritonitis
 - E. abscess cystic-rectal spaces
640. Turner's sing at acute pancreatitis:
- A. Violet spots on face and trunk
 - B. * Cyanosis sides of stomach and trunk
 - C. Cyanosis skin of stomach
 - D. Icteritiousness round a belly-button
 - E. Cyanosys of hands
641. Udin's sing at a perforated ulcer is
- A. * feeling at palpation shove the gases which penetrate through the perforated opening
 - B. dulling perforated sound in the lateral departments of stomach
 - C. disappearance of hepatic dullness
 - D. irradiation pain in a shoulder or shoulder-blade
 - E. sickliness the back vault of vagina
642. Vomiting coffee-grounds is a characteristic sign
- A. * bleeding ulcer
 - B. penetrative ulcers
 - C. perforated ulcers
 - D. malignizated ulcers
 - E. for cicatrical-ulcerous pylorus stenosis
643. Vomiting what arose up in 4-6 hours after eating characteristic for
- A. chronic alcoholic gastritis
 - B. cancer and ulcers of cardia
 - C. * pylorus ulcers
 - D. achalasia of gullet
 - E. ulcer and cancer the body of stomach

644. Voskresenskiy's sign at acute pancreatitis:
- * Absence pulsation the abdominal aorta
 - Sickliness in left costal-vertebral coal
 - Sickliness and proof tension the muscles in an epigastrium with passing to left subcostal area
 - Ictericity round a belly-button
 - Skin hyperesthesia in projection the gland
645. What nosotropic conditionality Voskresenskiy's sign at acute pancreatitis:
- * Inflammatory edema of pancreas
 - Reflex paresis of colon
 - Thrombosis of abdominal aorta
 - Embolism of abdominal aorta
 - Development of peritonitis
646. What a clinical flow can be at acute pancreatitis:
- Easy, middle, heavy
 - Acute, chronic
 - * Abortive, making progress
 - Edema, necrosis
 - Any variant
647. What basic method the treatment of acute pancreatitis is:
- Surgical
 - * Conservative
 - Homoeopathic
 - Physical therapy
 - A right answer is not present
648. What cages of pancreas are make glucagon:
- клетки
 - клетки
 - клетки
 - None of cages
 - D-клетки
649. What cages of pancreas are make insulin:
- клетки
 - клетки
 - клетки
 - None of cages
 - D-клетки
650. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodenum?
- * Tarry excrement
 - Presence on the formed excrement of strokes of red blood
 - Littlechanged blood in an excrement
 - Excrement of the raspberry colouring with the admixtures of mucus
 - Acholic excrement
651. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age

- A. perforation
 - B. perforation + bleeding
 - C. pylorus stenosis
 - D. malignization + penetration
 - E. * bleeding
652. What complications at a acute pancreatitis is behave to early:
- A. * Peritonitis
 - B. Phlegmon retroperitoneum space
 - C. Formation of pseudocysts
 - D. Development of saccharine diabetes
 - E. Intestinal impassability
653. What complications at a acute pancreatitis is behave to late:
- A. Peritonitis
 - B. * Phlegmon retroperitoneum space
 - C. Formation of pseudocysts
 - D. Development of saccharine diabetes
 - E. Intestinal impassability
654. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
655. What from operations does not execute at surgical treatment complicated acute pancreatitis:
- A. Through draining the stuffing-box bag
 - B. Abdominisation the pancreas
 - C. Omentopankreatopeksiy
 - D. Left-side resection of gland
 - E. * Pancreatojejunostomy
656. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol
 - B. Licviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
657. What from the transferred diseases can be reason of the pseudoperitoneal syndrome?
- A. dissecting aneurysm of the abdominal part of aorta.
 - B. Extraperitoneal haematoma.
 - C. Nephrolithiasis, nephrocolic
 - D. Thrombosis the pelvis veins
 - E. * All transferred
658. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy

- E. the all transferred does not belong
659. What from the transferred operations on the stomach organ protective is
- * selective proximal vagotomy
 - resection by Bilrot I
 - resection by Bilrot II
 - gastrectomy
 - all are transferred
660. What hormone of pancreas has influences on metabolism glucose:
- * Insulin
 - Vasopressin
 - Adrenalin
 - Somatotropin
 - Tiroksin
661. What hormone of pancreas has influences on the exchange of fats:
- Insulin
 - Glyukagon
 - * Lipocainu
 - Adrenalin
 - Somatotropin
662. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- * ACTH
 - epidermal factor of growth
 - prostaglandin E
 - estrogens
 - STH
663. What is blood supply the body and tail pancreas:
- * Splenic artery
 - A.gastroduodenalis
 - A.gastrica sinistra
 - A.cystica
 - Variously
664. What is condition hematomesis at acute pancreatitis:
- Presence concomitant gastric ulcer
 - Presence concomitant gastritis
 - * Formation erosions in a stomach
 - Violation of microcirculation
 - Enzymes in blood
665. What is necrectomy:
- Delete the necrotic area within the limits of nonviable fabrics
 - * Delete the necrotic area within the limits of healthy fabrics
 - Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - Total delete of organ
 - There is not a faithful answer
666. What is pancreas located in relation to a peritoneum:

- A. * Retroperitoneal
 - B. Mesoperitoneal
 - C. Intraperitoneal
 - D. All answers are incorrect
 - E. Variously
667. What is pancreatectomy:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. * Total delete of organ
 - E. There is not a faithful answer
668. What is resection the pancreas:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. * Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
669. What is sequestrotomy:
- A. * Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
670. What level diastase answers a acute pancreatitis:
- A. 100-160
 - B. 200-500
 - C. * 600-1000
 - D. 1000-1500
 - E. 10-20
671. What level diastase answers a chronic pancreatitis:
- A. * 200-500
 - B. 100-160
 - C. 600-1000
 - D. 1000-1500
 - E. 10-20
672. What level diastase answers pancreonecrosis:
- A. 600-1000
 - B. 200-500
 - C. 100-160
 - D. * 1000-1500
 - E. 10-20
673. What localization of ulcer is most characteristic for the patients of elderly and senile age
- A. * cardial department of stomach
 - B. overhead third of gullet

- C. lower third of gullet
 - D. bulb of duodenum
 - E. small curvature
674. What method diagnostics the ulcerous illness most informing
- A. * esophagogastroduodenoscopy
 - B. analysis of excrement on the hidden blood
 - C. X-ray
 - D. global analysis of blood
 - E. research of gastric secretion
675. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- A. On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - B. On blood volume
 - C. * On an arteriotomy, pulse, state of patient
 - D. On a globular volume
 - E. On the level of thrombocytes
676. What most effective blocker secretion of pancreas at acute pancreatitis:
- A. Cyanocobalamin
 - B. Ubretid
 - C. Arginine
 - D. * Sandostatin
 - E. Benzogeksoniy
677. What most effective treatment the unformed complicated cyst is:
- A. Conservative treatment
 - B. * External draining cyst
 - C. Resection cyst within the limits of the unchanged gland
 - D. Cysticenterostomy
 - E. Cystogastrostomy
678. What most effective treatment the unformed uncomplicated cyst is:
- A. * Conservative treatment
 - B. External draining cyst
 - C. Resection cyst within the limits of the unchanged gland
 - D. Cysticenterostomy
 - E. Cystogastrostomy
679. What most optimum resort is which used for pathology the pancreas:
- A. Pyatigorsk
 - B. * Morshin
 - C. Kuyal'nik
 - D. Truskavets
 - E. Nemirov
680. What must be done in the case of development the posoperative peritonitis?
- A. to appoint antibiotics
 - B. to appoint anaesthetic
 - C. to execute laparocenthezis
 - D. to execute lasparoscopy
 - E. * to execute laparotomy

681. What norm diastase is:
- * To 160
 - To 50
 - To 200
 - To 300
 - To 1200
682. What operation is most often used for localization the formed pseudocyst in the area of tail the pancreas:
- Cystojejunostomy on the eliminated loop
 - External draining the cyst
 - Cystogastrostomy
 - * Cystoduodenostomy
 - Cystoenteroanastomosis
683. What operation is used for suppuration the pseudocysts of pancreas:
- Cystojejunostomy on the eliminated loop
 - * External draining the cyst
 - Cystogastrostomy
 - Cystoduodenostomy
 - Cystoenteroanastomosis
684. What operation is used for the pseudocyst of pancreas in the 3th stage of its forming:
- * Cystojejunostomy on the eliminated loop
 - External draining the cyst
 - Cystogastrostomy
 - Cystoduodenostomy
 - Cystoenteroanastomosis
685. What operation on a pancreas is indicated at the oedematous form the acute pancreatitis:
- * Interference on a pancreas is not needed
 - Pancreatectomy
 - Omentopankreatopeksy
 - Abdominisation pancreas
 - Marsupialization
686. What preparation does behave to blocker H2-retseptors?
- * tavegil
 - obzidan
 - hystdol
 - cerucal
 - oraza
687. What preparation does behave to blocker of muscarine receptors of coating cages?
- cymetidin
 - eglonin
 - * gastocepini
 - etimsiloli
 - vinilin
688. What preparation is applied at violation the extrasecretory function of pancreas at a chronic pancreatitis:
- Pyracetam
 - Papaverin

- C. Pantocrin
 - D. * Panzinorm
 - E. Panthenol
689. What preparation is applied at violation the extrasecretory function pancreas at a chronic pancreatitis:
- A. Pyracetam
 - B. Papaverin
 - C. Pantocrin
 - D. * Mezim-forte
 - E. Panthenol
690. What preparation is attributed to the proteases inhibitors:
- A. Tocopherol
 - B. Triampur
 - C. Tagamet
 - D. * Trasylol
 - E. Trypsin
691. What preparation is not attributed to the inhibitors proteases:
- A. Pantripin
 - B. Kontrikal
 - C. Gordox
 - D. Trasylol
 - E. * Trypsin
692. What preparations from the cytostatic group use for intensifying the chronic pancreatitis:
- A. Cyanocobalamin
 - B. Methyluracil
 - C. * 5-fluorouracil
 - D. Furadolizon
 - E. Mezimforte
693. What preparations, except for other properties, own yet and a bacteriostatic effect on *Helicobacter pyloris*
- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
694. What primary purpose treatment the patient with fatty pancreatonecrosisto the operation is:
- A. Liquidations the pain
 - B. * Disintoxication the organism
 - C. Liquidations crampy the big duodenal papilla
 - D. Declines secretory activity the pancreas
 - E. Improvements microcirculation
695. What primary purpose treatment the patients with the heavy form of hemorrhagic pancreatonecrosis to the operation is:
- A. Liquidations the pain
 - B. * Disintoxication the organism
 - C. Liquidations crampy the big duodenal papilla
 - D. Declines secretory activity the pancreas

- E. Improvements microcirculation
696. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
697. What surgical pathology is a characteristic symptom of toxic «scissors» ?
- A. appendicitis
 - B. cholecystitis
 - C. pancreatitis
 - D. * peritonitis
 - E. mesadenitis
698. What symptom is most characteristic in the initial phase of peritonitis:
- A. sickliness and overhanding of pelvic peritoneum at rectal research.
 - B. dulling in declivous places at percussion of stomach
 - C. * tension of muscles the front abdominal wall.
 - D. swelling of stomach
 - E. melena
699. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
700. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
701. What type of peritonitis are fibrinogenous impositions at on a parietal and visceral peritoneum?
- A. at serosal.
 - B. at festering.
 - C. at fibrinogenous
 - D. at putrid.
 - E. * at all
702. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia
703. When apply Teylor's method at ulcerous illness

- A. * at conservative treatment perforated ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pylorostenosis
704. Widespread festering peritonitis is investigation of all above-stated diseases, except for:
- A. perforative ulcers of duodenum
 - B. phlegmonous cholecystitis
 - C. * hydrocholecystiss
 - D. destructive pancreatitis
 - E. volvulus of sigmoid bowel
705. With what disease above all things is it necessary to differentiate the acute peritonitis?
- A. Hepar-kidney syndrome
 - B. * ischemic abdominal syndrome
 - C. adrenogenital syndrome
 - D. Horner's syndrome
 - E. diencephalic syndrome
706. A patient 2 hours ago fallen down from the first floor. State heavy. There are signs of the diffusive peritonitis. Survey sciagraphy of stomach is executed. What from roentgenologic symptoms diagnosis of break of cavernous organ will confirm?
- A. hight standings of diaphragm dome
 - B. Kloybers bowls
 - C. * free gas in an abdominal region
 - D. a free liquid in an abdominal region
 - E. infiltration in an abdominal region
707. A patient 2 hours ago got a blow in a stomach. Delivered in a urgent clinic in a grave condition with suspicion on the break of cavernous organ. What method of roentgenologic research will allow to specify a diagnosis?
- A. laparocentzis
 - B. laparoscopy
 - C. * sciagraphy of stomach
 - D. contrasting X-ray examination
 - E. irrigoscopy
708. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intansive, but weakness and dizziness were appeare. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
709. A patient 59 years old grumbles about permanent pains in an epigastrium, givings in the back, bad appetite, weakness, becoming thin. Pains appeared 3 months ago and in the beginning disturbed little, but grew gradually, during the last week there is vomiting, bringing a facilitation. Patient of the lowered feed, pale. The turgor of skin is lowered. A stomach is pulled in, palpation is sickly in an epigastrium, peripheral lymphonoduss are not megascopic. Roentgenologic is a defect of filling in area of back wall of pyloric department of stomach. At

a fibroscopy deep ulcer of back wall of stomach is with overpeering uneven edges. Possible complication

- A. malignization
- B. penetration, germination in surrounding organs
- C. perforation
- D. * stenosis
- E. bleeding

710. A patient delivered in a clinic with complaints about great pain after a breastbone and between shoulder-blades, arising up during the massive vomiting. A patient has low blood pressure, temperature is 39,5; leucocytosis - 20000. At x-ray photography research found the accumulation air and liquid in the left pleura cavity and mediastinum. Preliminary diagnosis

- A. * perforation of gullet
- B. subdiaphragmatic abscess
- C. acute pneumonia with the parapneumonic empyema of pleura
- D. perforation the gastric ulcer
- E. heart attack of myocardium

711. A patient entered urgent in a clinic on 3 days from the beginning of disease. The state is heavy, adynamic. Face of Hippocrates, pulse 138 in 1 min, AP - 8040. A stomach is blown away, moderato sickly at palpation. Shchotkin-Blyumbergs symptom is doubtful. A liquid is determined in the lower departments the stomach. A liquid, stinking excrement departs through gaping sphincter. That does explain the state of patient?

- A. intestinal impassability
- B. * diffusiv peritonitis
- C. mesenteric ischemia
- D. toxic dilatation of colon
- E. acute pancreatitis

712. A patient got the trauma of stomach 3 hours ago. Delivered in a urgent clinic with complaints about tormina. The lines of person are focus. Positive symptoms the irritation of peritoneum. At survey X-ray examination is found out free gas under the dome of diaphragm. A diagnosis is set: break of cavernous organ. What type of treatment is most expedient?

- A. cold on a stomach
- B. laparotsentez
- C. laparoscopy
- D. * operative
- E. antibiotic

713. A patient grumbles about great pain in a stomach, which arose up suddenly 6 hours ago. The diagnosis of peritonitis is set. What symptom is most characteristic for this diagnosis?

- A. increases of temperature
- B. tachycardia
- C. leucocytosis
- D. tension of muscles the abdominal wall
- E. * irritations of peritoneum

714. A patient suffers a stenocardia. Became ill suddenly after the physical loading. Complaints about pain in a epigastric area, which spread on the right half of stomach. Positive Shchotkin-Blyumbergs symptom. Rectal – overhang and painfulness the front wall of rectum. It is not discovered the free gas on the survey sciagram of stomach. What is most reliable diagnosis?

- A. abdominal form of heart attack the myocardium
- B. acute cholecystitis
- C. acute pancreatitis

- D. * peritonitis
 - E. peritonitis of apendicular character
715. A prophylaxis and treatment of postoperative enteroplegias at peritonitis is
- A. gastrotomy
 - B. gastrointestinotomy
 - C. * nasogastrointestinal intubation
 - D. intubation of abdominal region
 - E. intubation of stuffing-box bag
716. A stomach-ache develops suddenly at (complication of ulcerous illness)
- A. bleeding
 - B. malignization
 - C. stenosis
 - D. penetration
 - E. * perforations
717. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
718. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
719. Acute cholecystitis usually begins with
- A. Increases the temperature
 - B. Appearances the vomiting
 - C. * Pains under a rib on the right
 - D. Disorders of chair
 - E. Weights are in a epigastria area
720. Agents, which cause peritonitis, can be all except for:
- A. urines at the break of urinary bladder
 - B. tables of contents the stomach during the perforation of ulcer
 - C. to blood at the trauma of stomach
 - D. biles during the perforation of gall-bladder
 - E. * air in an abdominal region after laparoscopy research
721. All surgical interferences at the destructive forms of acute pancreatitis divide on:
- A. * Early, late, deferred operations
 - B. Primary, second, repeated operations
 - C. Invasion, not invasion operations
 - D. Complicated, operations are not complicated
 - E. Not divided
722. An intravenous cholecystography are indicated and informing at
- A. Gall-bladder is palpated

- B. To the icterus
 - C. Peritonitis
 - D. * Calming down attack of the acute cholecystitis
 - E. Cholangitis
723. Appearance „splashing sound” in acute intestinal obstruction is caused:
- A. * By the accumulation of liquid and gases in the afferent loop of intestine
 - B. By the accumulation of liquid and gases in the efferent loop of intestine
 - C. By the presence of liquid in the abdominal cavity
 - D. By the presence of free gas in the abdominal cavity
 - E. By the presence of free gas and liquid in the abdominal cavity
724. Aseptic inflammation of peritoneum can be caused:
- A. by a collibacillus
 - B. by staphylococuss
 - C. * by pancreatitis juice
 - D. by an abscess
 - E. by intestinal maintenance
725. At a perforation gastric ulcer, vomiting blood is
- A. often
 - B. very often
 - C. it is never
 - D. * rarely
 - E. there is not a right answer
726. At a subdiaphragmatic abscess in a clinical picture characteristically all following, except for:
- A. declines the respiratory excursion of lights.
 - B. high standing of diaphragm dome.
 - C. concord pleurisy.
 - D. basale atelectasis of lights.
 - E. * blood spitting
727. At a subhepatic abscess can take place all, except for:
- A. pains in a thorax with an irradiation in a supraclavicular area
 - B. reactive pleurisy
 - C. * Courvoisier's symptom
 - D. Senator's symptom
 - E. Dyushen's symptom
728. At an acute and chronic cholecystitis application is contra-indicated
- A. Omnoponum
 - B. * Morphine hydrochloride
 - C. No-spa
 - D. Atropine sulfate
 - E. Spazmalgon
729. At festering peritonitis the disorder of hemodynamics not conditioned:
- A. by the decline of volume the circulatory blood
 - B. * by the increase of volume the circulatory blood
 - C. by the decline of tone the vascular wall
 - D. by the change of properties the hemorheologys
 - E. by violation of cardiovascular activity

730. At localization the stone in a cystic channel and absence infection the phenomenon is carried by the name
- A. Courvoisier's symptom
 - B. Acute cholecystitis
 - C. Hydrocholecystis
 - D. * Cyst of gall-bladder
 - E. All answers are not right
731. At operations on a stomach sometimes by mistake bandage an additional hepatic artery, that, in same queue, can result to necrosis the segment, sectors or even stakes of liver. An additional hepatic artery more frequent walks away from an artery
- A. general hepatic
 - B. * left stomach
 - C. splenic
 - D. overhead mesenteric
 - E. all answers are faithful
732. At pancreatitis abscesses and infected necroses execute such operations, except for:
- A. Opening of abscess with draining
 - B. Pancreaticnecrsekvestrektomy
 - C. Pancreaticsekvestrektomy
 - D. Pancreaticsekvestrektomy with laparostomy
 - E. * Total pancreatotomy
733. At percussion in the first clock after perforation the ulcer more possibly
- A. * dulling in the gently sloping places of stomach
 - B. expansion of percussion border of liver
 - C. tympanitis in left subcosctal area
 - D. expansion of percussion border the spleen
 - E. there is not a right answer
734. At perforated ulcer the pulled wooden belly is determined in the stage of peritonitis
- A. * reactive
 - B. terminal
 - C. toxic
 - D. terminal-toxic
 - E. there is not a right answer
735. At peritonitis intestinal impassability develops, as a rule:
- A. mechanical
 - B. dynamic
 - C. spastic
 - D. * paralytic
 - E. mixed
736. At peritonitis of violation the proteometabolism characterized:
- A. by the increase of concentration the albumen
 - B. by the increase of concentration the globulins
 - C. diminishing of concentration the albumen
 - D. * diminishing of the albumen - globulins coefficient
 - E. by the increase of the albumen - globulins coefficient
737. At suspicion on a duodenal ulcer conduct above all things

- A. research of gastric secretion
 - B. * EGDS
 - C. X-ray examination organs of abdominal region
 - D. determination the level of gastrin the whey blood
 - E. cholecystography
738. At suspicion on a subdiaphragmatic abscess the followings methods of diagnostics are rotined, except for:
- A. * laparoscopy.
 - B. Sonography.
 - C. X-ray examination of thorax.
 - D. X-ray examination of abdominal region.
 - E. computer tomography
739. At suspicion on the abscess of Duglas space all followings methods of diagnostics are rotined, except for:
- A. rectal inspection.
 - B. * proctoscopy.
 - C. Sonography.
 - D. computer tomography.
 - E. vaginal research
740. At the perforate ulcer of duodenum used more frequent
- A. * sewing up of the perforate opening
 - B. sewing up + gastroenteroanastomosis
 - C. resection of stomach
 - D. resection of stomach for a shutdown
 - E. different types of vagotomy in combination with the economy resection of stomach and other draining operations
741. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
742. Atypical clinical motion of postoperative peritonitis is conditioned setting in a postoperative period:
- A. spasmolytic
 - B. anticoagulant
 - C. * anaesthetic
 - D. analeptics
 - E. cardiac
743. Basic method examination the patients with the uncomplicated cholecystitis
- A. Infusion cholegraphy
 - B. ERCP
 - C. * Sonography
 - D. Laparoskopy
 - E. Gastroduodenoscopy
744. Blood loos I stage characterized such indexes
- A. Ht 48-44, Hb 120

- B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
745. Blood loos II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-50, Hb 130
746. Blood loos III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
747. Can not stipulate a mechanical icterus
- A. Cancer the head of pancreas
 - B. * Stone of cystic channel
 - C. Chronic pancreatitis
 - D. Stone the general bilious channel
 - E. Tumour large duodenal papilla
748. Change in the analysis of blood at a perforete ulcer
- A. leucopenia
 - B. anaemia
 - C. eosinophilia
 - D. * leucocytosis with a neutrophilic change
 - E. there is not a right answer
749. Characteristic laboratory sign of the acute uncomplicated cholecystitis
- A. Diastasuria
 - B. * Leykocytosis
 - C. Hypoglycemia
 - D. Glucosuria
 - E. Hyperbillirubinemia
750. Conservative treatment of intestinal obstruction is indicated in all cases, except for:
- A. * Torsion
 - B. Spastic obstruction
 - C. Paralytic obstruction
 - D. Coprostasis
 - E. There is no right answer
751. Courvoisier's symptom is
- A. Painless megascopic gall-bladder, patient is not yellow
 - B. Sickly megascopic gall-bladder, patient is not yellow
 - C. * Painless megascopic gall-bladder, patient is yellow
 - D. A gall-bladder don't palpaton
 - E. All answers are not right

752. Decision role in differential diagnostics of peritonitis and acute vascular purpura is a symptom:
- A. * hemorrhagic rash on a skin
 - B. acute pain in a stomach
 - C. systole noise above the abdominal department of aorta
 - D. absence of pulsation of abdominal department of aorta
 - E. melena at rectal research
753. Decision role in differential diagnostics of peritonitis and break the bone of pelvis is:
- A. to appoint antibiotics
 - B. to appoint anaesthetic
 - C. to execute laparocenthezis
 - D. * to execute laparoscopy
 - E. to execute survey sciagraphy of abdominal region
754. Decision role in differential diagnostics the peritonitis and dissecting aneurysm of aorta , there is a symptom:
- A. aperistalsis
 - B. acute pain in a stomach
 - C. * systole noise above the abdominal department of aorta
 - D. absence of pulsation of abdominal department of aorta
 - E. melena at rectal research
755. Dehidratation of the organism in acute intestinal obstruction most rapidly develops in:
- A. * Torsion of small intestine
 - B. Torsion of sygmoid intestine
 - C. Tumours of rectum with the phenomena of obstruction
 - D. Obturation large intestinal obstruction
 - E. Ileocaecal invagination
756. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
757. Diffusive festering peritonitis can be investigation of all transferred diseases, except for:
- A. perforations Meckel's diverticulum
 - B. destructive appendicitis
 - C. * stenosis of large duodenal nipple
 - D. Richter strangulation of hernia
 - E. acute intestinal impassability
758. Direct sign of ulcer at x-ray research
- A. violation of evacuation from a stomach
 - B. change of tone of stomach
 - C. form the stomach as "sand-glasses"
 - D. * symptom of "niche"
 - E. defect of filling
759. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- A. * Bergman's sing
 - B. Spazarskiy's sing

- C. Mendel's suing
 - D. De Keven's sing
 - E. Eleker's sing
760. Draining the abdominal region is conducted at:
- A. phlegmonous appendicitis without exudation
 - B. * gangrenous appendicitis with exudation
 - C. phlegmonous appendicitis with serosal exudation odourless
 - D. there is not a right answer
 - E. catarrhal appendicitis
761. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
762. During the examination of patient with acute intestinal obstruction: the Zege-Maitenphel's and "Obuhov's hospital" symptoms are positive. What type of obstruction it is characteristic for?
- A. * Sigmoid intestine torsion
 - B. Ileocaecal invagination
 - C. Obturation by the tumour of ascending intestine
 - D. Torsion of small intestine
 - E. All mentioned is correct
763. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- A. tumours
 - B. ulcers
 - C. bleeding polypuses
 - D. erosions
 - E. * changes of evacuation function
764. Fibrinogenous impositions on a peritoneum are not at peritonitis:
- A. * serosal
 - B. fibrinogenous
 - C. festering
 - D. putrid
 - E. excrement
765. For a patient 35 years old "knife-like pain" in an epigastrium, appearing suddenly hour back. Pale, pulse is 50, T-36,9 °C. A stomach is tense, as a board. In anamnesis an ulcer of duodenum is during 5 years. Complication came
- A. malignization
 - B. stenosis
 - C. penetration
 - D. * perforation
 - E. bleeding
766. For a patient, suffering ulcerous illness, sudden great pain in an epigastrium and unclear symptoms of peritonitis appeared. During subsequent days the display of these symptoms diminished, the state of patient had become better. It is possible to suppose at such clinic
- A. typical perforation the ulcer

- B. * covered perforation
 - C. preperforative state
 - D. intensifying of ulcerous illness
 - E. there is not a right answer
767. For a perforation declivous organ all is characteristic in a free abdominal region, except for:
- A. acute began pains.
 - B. wooden belly.
 - C. collapse.
 - D. * oliguria.
 - E. tachycardia.
768. For a perforate gastric ulcer in the first six clock typical
- A. great sudden pains in a stomach, frequent vomiting, swelling of stomach, disappearance the hepatic dullness, "sickle" under the right dome of diaphragm
 - B. frequent vomiting, swelling of stomach, disappearance the hepatic dullness, "sickle" under the right dome of diaphragm
 - C. great sudden pains in a stomach, wooden belly, swelling of stomach
 - D. * great sudden pains in a stomach, wooden belly, disappearance the hepatic dullness, "sickle" under the right dome of diaphragm
 - E. combinations of signs are equivalent
769. For acute intestinal obstruction the followings types of operations are possible, except for:
- A. * Gastrostomy
 - B. Right-side hemicolectomy
 - C. Resections of intestine
 - D. Colostomy
 - E. Hartmann's operation
770. For bleeding ulcer characteristic sign is
- A. * pain in an epigastrium;
 - B. knife-like pain;
 - C. signs irritation of peritoneum;
 - D. presence fresh blood in incandescence
 - E. melena;
771. For bleeding ulcer characteristically
- A. * melena
 - B. tension the muscles of front abdominal wall
 - C. Spazarskiy's sign
 - D. sickliness the back vault of vagina
 - E. irradiation pain in a shoulder or shoulder-blade
772. For clarification character of the icterus and reason of it development is necessary to make:
- A. X-ray examination of the subhepatic space, infusion cholecystography, ERCP
 - B. Sonography, ERCP
 - C. Infusion cholecystography, ERCP
 - D. * Sonography, infusion cholecystography, ERCP
 - E. ERCP
773. For clarification diagnosis of perforate ulcer used
- A. Gastroscopy
 - B. X-ray of abdominal region
 - C. * X-ray of abdominal region, after gastroscopy, then X-ray of abdominal region

- D. CT
 - E. There is not a right answer
774. For differentiation of acute appendicitis with the covered perforate ulcer useful
- A. gastroduodenoscopy, X-ray of abdominal region, laparoscopy
 - B. gastroduodenoscopy, X-ray of abdominal region, Sonography of abdominal region
 - C. survey X-ray of abdominal region, Sonography of abdominal region, irrigoscopy
 - D. X-ray of abdominal region, irrigoscopy
 - E. * X-ray of abdominal region, laparoscopy
775. For intestinal obstruction caused by invagination is characteristic:
- A. * Bloody discharges from rectum
 - B. The wave-like abdominal pain
 - C. Tumorous formation in a right iliac area by palpation
 - D. Bolus-like feces
 - E. Atony of rectal sphincter
776. For motion of disease ulcerous illness of middle weight characteristically
- A. development of complications
 - B. * relapses 1-2 times per a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 3 and anymore relapses on a year
777. For paralytic intestinal obstruction is characteristic:
- A. * Delay of stool and gases and acute distension of abdomen
 - B. Asymmetry of abdomen
 - C. The expressed wave-like pain in the abdominal cavity
 - D. Zege-Maitenphel's sign
 - E. Effusion in the abdominal cavity
778. For perforative appendicitis characteristically
- A. tension the muscles of front abdominal wall
 - B. there is the sudden strengthening of stomach-aches
 - C. rapid growth of clinical picture the peritonitis
 - D. Razdolskuy's symptom
 - E. * all transferred
779. For perforated ulcer characteristically
- A. * tension the muscles of front abdominal wall
 - B. melena
 - C. vomiting by coffee-grounds
 - D. high intestinal impassability
 - E. vomiting stagnant gastric maintenance
780. For peritonitis in the first 24 hours not typical
- A. aperistalsis intestine
 - B. * Kullenkamp's symptom
 - C. Tachycardia
 - D. dry language
 - E. tension the muscles of abdominal wall and positive Shchetkin-Blyumberg's symptom
781. For peritonitis there is a not characteristic symptom:
- A. Shchetkin-Blyumberg's symptom

- B. Voskresenskiy's symptom
 - C. Kulenkampf's symptom
 - D. * Moebius's symptom
 - E. Krymov's symptom
782. For pneumoperitoneum is characteristic symptom
- A. * Zhober's;
 - B. Khelatid's;
 - C. Podlag's;
 - D. Vigats's;
 - E. Udin's.
783. For the heavy flow of ulcerous illness characteristically
- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days
784. For the high small intestinal obstruction is not characteristic:
- A. * Zege-Maitenphel's sign
 - B. The wave-like abdominal pain
 - C. Splashing sound" (Sklyarov's sign)
 - D. Multiple vomiting
 - E. Cloyber's cups on X-ray
785. For the late stage of peritonitis all is characteristic, except for:
- A. swelling of stomach
 - B. hypovolemia
 - C. disappearance of intestinal noises
 - D. hypoproteinemia
 - E. * increased peristalsis
786. For the low large intestinal obstruction all is characteristic, except for:
- A. * Rapid dehydration
 - B. Delay of stool
 - C. Appearances of Cloyber's cups
 - D. Gradual progression of symptoms
 - E. Abdominal distension
787. For the reactive stage of festering peritonitis not characteristically:
- A. acute sickliness the stomach at palpation
 - B. positive Shchetkin-Blyumberg's symptom
 - C. tension of muscles the front abdominal wall
 - D. * face of «Hippocrates»
 - E. tachycardia
788. For the terminal stage of peritonitis not characteristically:
- A. tachycardia
 - B. * bradycardia
 - C. hyperthermia
 - D. falling of arteriotony
 - E. dynamic intestinal impassability

789. For the treatment measures in obturation intestinal obstruction all mentioned belong, except:
- A. * Prescribing of medicines which increase intestinal peristalsis
 - B. Prescribing of spasmolytics
 - C. Performing of siphon enema
 - D. Introducing of nasogastral tube for intestinal decompression
 - E. Corrections of water-electrolytes disturbances
790. For what purpose in treatment of diffusive festering peritonitis does execute nasointestinal intubation?
- A. account of losses the liquid through a gastroenteric highway.
 - B. control of electrolyte composition the intestinal maintenance
 - C. * prophylaxis of intestinal impassability
 - D. stimulation of the intestinal peristalsis
 - E. suppression of the intestinal peristalsis
791. From what department degestive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
792. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
793. Hectic fever is possible at
- A. the uncomplicated ulcer
 - B. bleeding from an ulcer
 - C. perforations of ulcer in the first clock
 - D. * penetration
 - E. cicatrice-ulcerous stenosis
794. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
795. Hepatic dullness is not determined at
- A. break of spleen
 - B. * perforations of gastric ulcer
 - C. break of bud
 - D. volvulus of stomach
 - E. mesenterial ishemia
796. How is the diagnosis of the general peritonitis set to the operation?
- A. roentgenologic
 - B. anamnestic

- C. by laboratory determination the signs of inflammatory reaction
 - D. * on clinical signs
 - E. on the level secretion the gastric juice
797. In a patient with pains in a epigastric area, what was displaced from a right iliac area. There was nausea and single vomiting. A patient accepted an analgin and put a hot-water bottle to the stomach, pains calmed down whereupon. On 2 days pains recommenced, spread on all stomach, the frequent vomiting appeared. The state of patient is heavy. Consciousness entangled. Euphoria. Pulse 128 in min, AP - 95/60. Language is dry. A stomach is tense in all departments. Temperature 37,2. Leucocytes in blood of 18? 109/l. Diagnosis
- A. * acute appendicitis, terminal stage of peritonitis
 - B. typhoid, perforation of typhoidal ulcer
 - C. perforation gastric ulcer
 - D. gastric bleeding
 - E. there is not a right answer
798. In a patient, suffering ulcerous illness, sudden great pain in an epigastrium and unclear symptoms of peritonitis appeared. During subsequent days the display of these symptoms diminished, the state of patient had become better. It is possible to suppose at such clinic
- A. typical perforation of ulcer
 - B. * covered perforation
 - C. preperforete state
 - D. intensifying the ulcerous illness
 - E. there is not a right answer
799. In an induction centre the sick is delivered with the attacks of cramps. Many years suffers ulcerous illness. Lately the daily vomiting appeared practically by the eaten food. Exhausted, in a эпигастральной area pigmentation of skin. A capotement is determined. Diagnosis
- A. malignization
 - B. bleeding
 - C. * stenosis
 - D. perforation
 - E. penetration
800. In classic motion of peritonitis select the stages:
- A. early, intermediate, late
 - B. reactive, intermediate, late
 - C. toxic, intoxication, terminal
 - D. * reactive, toxic, terminal
 - E. reactive, toxic, late
801. In patient of 82 years old with acute intestinal obstruction caused by the tumour of sigmoid intestine, who entered in late terms, the most rational tactic of treatment is the following:
- A. * Preparing for the operation during 2-3 hours with following performance of Hartmann's operation
 - B. Examination and performance of operation in 48-72 hours
 - C. Only conservative therapy
 - D. Infusion therapy and repeated siphon enemas
 - E. Urgent operation with performance of sigmoid intestine resection and anastomosis "end to end"
802. In the dynamics of acute peritonitis it is necessary positive to consider
- A. * decline of amount the leucocytes
 - B. increase of amount the leucocytes

- C. leucocytosis with the change of leukocytic formula to the left
 - D. growth of the leukocytic index intoxication
 - E. leukopenia
803. In the moment of perforation the gastric or duodenum ulcer meets most often
- A. * suddenly arising up megalgia
 - B. cramp-like pain
 - C. noncommunicative, moderate pain
 - D. liquid chair
 - E. tachycardia
804. In treatment of the diffusive peritonitis of appendicitis origin a basic value has
- A. correction water-electrolyte violations
 - B. sanitization of abdominal region
 - C. removal the source of peritonitis
 - D. * all answers are faithful
 - E. antibacterial therapy
805. In treatment of ulcerous illness the stomach and duodenum executed only on urgent indications
- A. stomach resection by Bilrot-II
 - B. * sewing up of the perforate opening
 - C. selective-proximal vagotomy
 - D. trunk vagotomy with a pyloroplasty
 - E. stomach resection by Bilrot-I
806. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?
- A. small curvature of stomach;
 - B. back wall of stomach, nearer to small curvature;
 - C. large curvature of stomach
 - D. * cardiac part of stomach;
 - E. pylorus.
807. In what type of intestinal obstruction one of the symptom is the red water after a cleaning enema:
- A. * Intestinal infarction
 - B. Paralytic
 - C. Spastic
 - D. Torsion of small intestine
 - E. Invagination
808. In what type of intestinal obstruction one of the symptom will be a blood discharge from rectum:
- A. * Invagination
 - B. Paralytic
 - C. Spastic
 - D. Torsion of small intestine
 - E. Intestinal infarction
809. In what vein is a venous outflow carried out in from a stomach?
- A. * V. Portae;
 - B. V. odd;
 - C. V. pair;

- D. V. overhead hollow;
 - E. V. lower hollow;
810. Indication to early operative interference at acute pancreatitis is:
- A. Acute pancreatolysis
 - B. Acute oedematous pancreatitis
 - C. * Progressive multiple organ failure what not added conservative therapy during 48-72 hours
 - D. Acute fatty pancreatitis
 - E. Forming of pseudocyst
811. Indications to special intraoperative examination bilious ways
- A. * A cholangitis, expansion of the common bile duct, plural shallow concrements in a gall-bladder, mechanical icterus in anamnesis
 - B. Cholangitis, expansion of the common bile duct, plural shallow concrements in a gall-bladder
 - C. Expansion of the common bile duct, icterus in the moment of operation, plural shallow concrements in a gall-bladder
 - D. Expansion of the common bile duct, mechanical icterus in anamnesis
 - E. All right
812. Intraoperative cholangiography at cholecystectomy used for
- A. Researches of peristalsis the general bilious channel
 - B. Retrograde filling of intrahepatic bilious channels
 - C. Researches tone the sphincter Oddi
 - D. * Exceptions concrements in channels
 - E. Exposures cholangitis
813. Laparostomy at the diffusive peritonitis used with a purpose
- A. * to repeated sanitization the abdominal region
 - B. extracorporal dialysis
 - C. hemosorption
 - D. laparoscopy
 - E. stimulations the peristalsis
814. Large intestine obstruction is more frequently caused:
- A. * By the malignant tumours of intestine
 - B. Foreign bodies
 - C. By the adhesions of abdominal cavity
 - D. By gall-stones
 - E. Helminths
815. Large ulcer is an ulcer measuring
- A. 1-4 cm
 - B. 1-3 cm
 - C. 3-5 cm
 - D. 2- 6 cm
 - E. * 2-3 cm
816. Least answers a subhepatic abscess:
- A. Senator's symptom
 - B. Dyushen's symptom
 - C. Liten's symptom
 - D. * Rovzing's symptom

- E. Shchotkin-Blyumberg's symptom
817. Middle laparotomy must be conducted at:
- * diffusive peritonitis
 - local unlimited peritonitis
 - abscess of Douglas space
 - periappendiceal infiltration
 - acute appendicitis
818. Most frequent reason of peritonitis
- posoperative complication
 - acute trauma of stomach
 - acute cholecystitis
 - * acute appendicitis
 - acute intestinal impassability
819. Most guarantee against the recedive of ulcer during an operation concerning ulcerous diseases of duodenum gives
- selective-proximal vagotomy
 - trunks vagotomy with a pyloroplasty
 - * resection no less than a 2/3 stomach
 - antrumectomy with selective vagotomy
 - veritable antrumectomy
820. Most informing method diagnostics the perforate ulcers
- X-ray examination
 - Sonography
 - EGDS
 - * laparoscopy
 - laparocentezis
821. 116. Most widespread laparoscopic operation at bile-stone illness:
- Cholecystectomy with revision the bilious ways
 - Ideal cholecysectomy
 - * Choledoholitotomy
 - Cholecystectomy
 - All answers are right
822. Name the most important method of investigation in the diagnostic of „acute intestinal obstruction“:
- * Research of barium passage trough intestine
 - Plain X-ray of abdominal cavity
 - Fibrogastroduodenoscopy
 - Laparoscopy
 - Biochemical blood analysis
823. Normal width of the common bile duct
- To 0,4 cm
 - * 0,5-0,7 cm
 - 0,8-1,3 cm
 - 1,4-2,0 cm
 - Over 2,0 cm
824. Numbers of complications the ulcerous illness

- A. * 5
 - B. 4
 - C. 1
 - D. 3
 - E. 2
825. Pain in the left shoulder can be rather at
- A. acute cholecystitis
 - B. perforations the ulcer of duodenum
 - C. * perforations the gastric ulcer
 - D. mesenteric lymphadenitis
 - E. there is not a right answer
826. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
827. Patient with a gangrenous cholecystitis is indicating:
- A. * Urgent operation
 - B. Operation at default of effect from conservative therapy
 - C. Conservative treatment
 - D. Deferred operation
 - E. A decision-making depends on age of patient
828. Penetration of gastric content in an abdominal region possibly at
- A. bleeding
 - B. stenosis
 - C. penetration
 - D. malignixation
 - E. * perforations
829. Peritonitis does not develop at the next form of acute appendicitis
- A. * catarrhal
 - B. phlegmonous
 - C. gangrenous
 - D. perforatiove
 - E. gangrenous-perforatiove
830. Postoperative peritonitis is characterized a flow:
- A. typical
 - B. * atypical
 - C. stormy
 - D. with the expressed pain reaction
 - E. with the expressed intoxication
831. Preoperated complication of acute appendicitis
- A. * diffusive peritonitis
 - B. intra-abdominal bleeding
 - C. suppuration of wound
 - D. evisceration wounds
 - E. there are not a right answer

832. Preoperative preparation for patients from peritonitis does not provide:
- A. corrections the violations cardiovascular system
 - B. * laparocentzis
 - C. declines the intoxication
 - D. corrections the exchange violations
 - E. struggle with hy hypovolemia
833. Rational operation at the subcompensated ulcerous stenosis of pylorus
- A. antrumectomy
 - B. * resection 2/3 stomach
 - C. front gastroenteroanastomosis
 - D. gastrectomy
 - E. selective proximal vagotomy
834. Relaparotomy at difuusive festering peritonitis pursues:
- A. * repeated revision of abdominal region
 - B. repeated laparoscopy of abdominal region
 - C. repeated endoscopy of abdominal region
 - D. normalization the function of gall-bladder
 - E. normalization the function of urinary bladder
835. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
836. Specific symptom of perforation declivous organ in a free abdominal region is:
- A. high leucocytosis.
 - B. absence of intestinal noises.
 - C. * pneumoperitoneum.
 - D. positive symptoms of irritation the peritoneum.
 - E. dulling of the percussion sound in the gently sloping places of abdominal region
837. Specify a criterion which grounds the choice of middle laoarotomy access at the deffusion festering peritonitis:
- A. minimum trauma the abdominal wall
 - B. minimum cut
 - C. * valuable revision the abdominal region.
 - D. minimum blood loss.
 - E. minimum level of infecting the wound
838. Specify obligatory measures which are conducted during an operation concerning widespread fibrinopurulent peritonitis:
- A. removal the source of peritonitis.
 - B. sanitization of the abdominal region.
 - C. decompression of intestine.
 - D. draining of abdominal region.
 - E. * all answers are faithful
839. Specify possible reasons of the pseudoperitoneal syndrome:
- A. uremia.

- B. porphyria.
 - C. diabetic crisis.
 - D. nephrocolic.
 - E. * all transferred
840. Specify reason of use the derivatives of metronidasoli as an obligatory component of antibacterial therapy of widespread peritonitis?
- A. * removal of anaerobic microflora.
 - B. removal of mycotic flora.
 - C. removal of microflora of aerobic.
 - D. prophylaxis of intestinal worm invasion.
 - E. prophylaxis of widespread candidosis
841. Specify the most rational way the introduction of antibacterial preparations in treatment of diffusive festering peritonitis:
- A. peroral
 - B. hypodermic
 - C. intramuscular
 - D. * intravenous
 - E. intraperitoneal
842. Sudden and painful pain with localization in the middle departments of stomach with an irradiation in the back more characteristic for
- A. heart attack the myocardium
 - B. * break aneurysm the aorta
 - C. bilious colic
 - D. perforate ulcers
 - E. nephrocolic
843. Symptom of the toxic «scissors» at peritonitis it:
- A. increase of temperature and pulse
 - B. diminishing of temperature and pulse
 - C. * diminishing of temperature and increase of pulse
 - D. increase of temperature and diminishing of pulse
 - E. increase of temperature and diminishing of breathing frequency
844. Symptom of toxic «scissors» at the peritinitis it is correlation:
- A. * pulse and temperature
 - B. arteriotony and pulse
 - C. arteriotony and temperature
 - D. rectal and axillar temperature
 - E. rate breathings and temperatures
845. Tactic of family doctor during the covered perforation of ulcer
- A. * urgent hospitalization in surgical permanent establishment
 - B. planned hospitalization in surgical permanent establishment
 - C. supervision on to the house
 - D. hospitalization in therapeutic permanent establishment
 - E. there is not a right answer
846. Tactic of surgeon at periappendiceal mas:
- A. operative treatment
 - B. * conservative treatment, systematic looking after a patient
 - C. there is not a right answer

- D. supervision
 - E. puncture
847. Tension of abdominal wall and stage peritonitis at acute appendicitis
- A. * absents, a stomach is swollen - terminal
 - B. absents, a stomach is not swollen - terminal
 - C. expressed, a stomach is not swollen - terminal
 - D. absents, a stomach is swollen - toxic
 - E. absents, a stomach is swollen – initial
848. Tension of muscles the right iliac area at the perforation of duodenal ulcer is explained
- A. by development of the diffusive peritonitis
 - B. by reflex connections through medullispinal nerves
 - C. * flowing down of gastric content in a right lateral channel
 - D. entering of air abdominal region
 - E. viscerovisceral connections with a vermicular appendix
849. Tension the muscles of stomach in an initial period of perforation ulcer
- A. absents
 - B. * visible
 - C. sickliness under the left shoulder-blade
 - D. appears at palpation
 - E. there is not a right answer
850. The attack of hepatic (bilious) colic development
- A. * Suddenly, acutely
 - B. After a initial period
 - C. Gradually, gradually
 - D. After the protracted starvation
 - E. After supercooling
851. The best variant the treatment of subhepatic abscess is:
- A. conservative treatment
 - B. extra-peritoneal section and draining
 - C. laparotomy, section and tamponing of cavity
 - D. * puncture of abscess by a thick needle under control Sonography
 - E. all transferred right
852. The complex treatment of festering peritonitis does not provide for:
- A. delete of primary hearth
 - B. * vagotomy
 - C. correction the metabolic violations
 - D. adequate therapy by antibiotics
 - E. struggle of paresis the intestine
853. The development of paralytic intestinal obstruction is caused by all mentioned except for:
- A. * Leaden poisoning
 - B. Peritonitis
 - C. Acute pancreatitis
 - D. Retroperitoneal hematoma
 - E. Disorders of mesenterial circulation of blood
854. The diagnostic measures which immediately performed in suspicion on acute intestinal obstruction include everything, except:

- A. * Angiography of abdominal cavity
 - B. Auscultation of abdomen
 - C. Plain X-ray of abdominal cavity
 - D. Introducing of nasogastal tube for the decompression of intestine
 - E. Palpation of abdomen, digital examination of rectum
855. The diagnostics criteria of the anaerobic peritonitis is
- A. stormy progress of disease
 - B. heavy festering intoxication
 - C. expressed enteroplegia
 - D. abundant amount of exsudate green-brown colors
 - E. * all is transferred
856. The exsudate painted blood in an abdominal region is observed always, except for:
- A. * tubercular peritonitis
 - B. violations of extra-uterine pregnancy
 - C. mesenteric ischemia
 - D. acute pancreatitis
 - E. twisted oothecoma
857. The favourable result of treatment the patients with peritonitis provide:
- A. early surgical interference
 - B. adequate operation
 - C. intravenous antibacterial therapy
 - D. methods of extracorporeal dialysis
 - E. * all is transferred
858. The index litogenic bile is determined correlation
- A. Cholesterol, billirubine and lecithin
 - B. Billirubine, bilious acids and lecithin
 - C. Cholesterol, bilious acids and bilirubine
 - D. * Cholesterol, bilious acids and lecithin
 - E. Billirubine and lecithin
859. The leading symptom of peritonitis is:
- A. stomach-ache
 - B. enteroplegia
 - C. swelling of stomach
 - D. * symptoms the irritation of peritoneum
 - E. symptoms of the intestinal impassability
860. The liquid painted a bile in an abdominal region is not observed at
- A. * Break pus hydatidoma
 - B. To the protracted mechanical icterus
 - C. Spontaneous bilious peritonitis
 - D. Perforations of gall-bladder
 - E. Perforations the ulcer of duodenum
861. The methods of disintoxication at peritonitis are not:
- A. lymphosorbtion
 - B. hemosorption
 - C. enterosorbtion
 - D. plasmapheresis
 - E. * antibiotic

862. The most frequent cause of small intestine mechanical obstruction is:
- * Adhesions of abdominal cavity
 - Gall-stones
 - Foreign bodies
 - Tumours
 - Helmints
863. The most informing method the instrumental diagnostics of peritonitis is:
- * survey sciagraphy of abdominal region
 - lasparoscopy
 - angiography
 - gastroscopy
 - colonoscopy
864. The patient of 62 years old grumbles about a weakness, fatigue ability, sometimes moderate pains and sense of weight in an anticardium, vomiting after-meal. Sick about 6 months. The pallor of skin covers, sickliness and compression is marked in a epigastric area, a reaction with a benzidine is acutely positive, haemoglobin is 52 g/l, eras. 2,5. At x-ray research pyloric department of stomach the defect of filling is determined 3x2 cm, displaced at palpation.
- Complication
- * bleeding, anaemia
 - stenosis
 - penetration, germination in surrounding organs
 - perforation
 - malignization
865. The sign of the diffusive peritonitis is not
- * visible peristalsis
 - absence the peristaltic intestinal noises
 - sickliness of stomach at palpation in all departments
 - even tension the muscles of front abdominal wall
 - positive Shchetkin-Blyumberg's symptom
866. The special research extrahepatic bilious ways is absolutely indicated at:
- Shallow stone in common bile duct, suspicion on stenosis the large duodenal papilla, expansion of the common bile duct, mechanical icterus in the moment of operation
 - Suspicion on stenosis of large duodenal papilla, expansion of the common bile duct, mechanical icterus in the moment of operation
 - At expansion of the common bile duct
 - * All right
 - All not right
867. The ways distribution of gastroenteric content during the perforation of ulcer depend on
- anatomic structure of the lateral ductings
 - locations of stomach
 - localizations of the perforate opening
 - * only transferred
 - forms and locations of transversal rim bowel
868. There is vomiting at peritonitis, as a rule:
- single
 - episodic
 - * frequent

- D. abundant
 - E. scanty
869. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scarry-ulcerous stenosis
 - B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
870. To determine the viability of strangulated intestine it is necessary to be oriented on the followings signs, except:
- A. * Presence of strangulation groove
 - B. Color of intestine
 - C. Presence of peristalsis
 - D. The pulsation of mesenteric vessels
 - E. All answers are incorrect
871. To the gastric – intestinal bleeding of un ulcerous etiology belong
- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.
872. To the initial phase the peritonitis of appendicle origin does not behave
- A. sickliness the pelvic peritoneum at rectal research
 - B. tachycardia
 - C. * noticeable electrolyte changes
 - D. tendency to growth leucocytosis
 - E. tension of muscles the abdominal wall
873. To the late stage of peritonitis of appendicle origin does not behave
- A. dehydration
 - B. swelling of stomach
 - C. * increased peristalsis
 - D. hypoproteinemia
 - E. disappearance of intestinal noises
874. To the local isolated peritonitis does not attribute:
- A. subdiaphragmatic abscess
 - B. subhepatic abscess
 - C. interintestinal abscess
 - D. * primary idiopathic peritonitis
 - E. abscess cystic-rectal spaces
875. Udin's sing at a perforated ulcer is
- A. * feeling at palpation shove the gases which penetrate through the perforated opening
 - B. dulling perforated sound in the lateral departments of stomach
 - C. disappearance of hepatic dullness
 - D. irradiation pain in a shoulder or shoulder-blade
 - E. sickliness the back vault of vagina
876. Ulcerous diaeases behaves to the diseases

- A. innate
 - B. because of alcoholism
 - C. because of the broken circulation of blood
 - D. * chronic recurrent
 - E. traumatic
877. What nosotropic conditionality Vosresenky's sign at acute pancreatitis:
- A. * Inflammatory edema of pancreas
 - B. Reflex paresis of colon
 - C. Thrombosis of abdominal aorta
 - D. Embolism of abdominal aorta
 - E. Development of peritonitis
878. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodenum?
- A. * Tarry excrement
 - B. Presence on the formed excrement of strokes of red blood
 - C. Littlechanged blood in an excrement
 - D. Excrement of the raspberry colouring with the admixtures of mucus
 - E. Acholic excrement
879. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- A. perforation
 - B. perforation + bleeding
 - C. pylorus stenosis
 - D. malignization + penetration
 - E. * bleeding
880. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
881. What from operations does not execute at surgical treatment complicated acute pancreatitis:
- A. Through draining the stuffing-box bag
 - B. Abdominisation the pancreas
 - C. Omentopankreatopeksiy
 - D. Left-side resection of gland
 - E. * Pancreatojejunostomy
882. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol
 - B. Liciviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
883. What from the transferred diseases can be reason of the pseudoperitoneal syndrome?
- A. dissecting aneurysm of the abdominal part of aorta.
 - B. Extraperitoneal haematoma.

- C. Nephrolithiasis, nephrocolic
 - D. Thrombosis the pelvis veins
 - E. * All transferred
884. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy
 - E. the all transferred does not belong
885. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
886. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E
 - D. estrogens
 - E. STH
887. What is necrectomy:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. * Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
888. What is pancreatectomy:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. * Total delete of organ
 - E. There is not a faithful answer
889. What is resection the pancreas:
- A. Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics
 - C. * Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
890. What is sequestrotomy:
- A. * Delete the necrotic area within the limits of nonviable fabrics
 - B. Delete the necrotic area within the limits of healthy fabrics

- C. Delete part of organ with his transversal cutting within the limits of the changed fabrics
 - D. Total delete of organ
 - E. There is not a faithful answer
891. What kind of operation is more rationally to perform for the patient with the cancer of caecum, complicated by acute intestinal obstruction in early terms of the disease:
- A. * Right-side hemicolectomy with ileotransversoanastomosis
 - B. Formation of ileostomy
 - C. Formation of caecostomy
 - D. Hartmann's operation
 - E. Mikulich's operation
892. What localization of ulcer is most characteristic for the patients of elderly and senile age
- A. * cardial department of stomach
 - B. overhead third of gullet
 - C. lower third of gullet
 - D. bulb of duodewnum
 - E. small curvature
893. What method diagnostics hte ulcerous illness most informing
- A. * esophagogastroduodenoscopy
 - B. analysis of excrement on the hidden blood
 - C. X-ray
 - D. global analysis of blood
 - E. research of gastric secretion
894. What most effective blocker secretion of pancreas at acute pancreatitis:
- A. Cyanocobalamin
 - B. Ubretid
 - C. Arginine
 - D. * Sandostatin
 - E. Benzogeksoniy
895. What most effective treatment the unformed complicated cyst is:
- A. Conservative treatment
 - B. * External draining cyst
 - C. Resection cyst within the limits of the unchanged gland
 - D. Cysticenterostomy
 - E. Cystogastrostomy
896. What most effective treatment the unformed uncomplicated cyst is:
- A. * Conservative treatment
 - B. External draining cyst
 - C. Resection cyst within the limits of the unchanged gland
 - D. Cysticenterostomy
 - E. Cystogastrostomy
897. What must be done in the case of development the posoperative peritonitis?
- A. to appoint antibiotics
 - B. to appoint anaesthetic
 - C. to execute laparocenthezis
 - D. to execute lasparoscopy
 - E. * to execute laparotomy

898. What operation is most often used for localization the formed pseudocyst in the area of tail the pancreas:
- A. Cystojejunostomy on the eliminated loop
 - B. External draining the cyst
 - C. Cystogastrostomy
 - D. * Cystoduodenostomy
 - E. Cystoenteroanastomosis
899. What operation is used for suppuration the pseudocysts of pancreas:
- A. Cystojejunostomy on the eliminated loop
 - B. * External draining the cyst
 - C. Cystogastrostomy
 - D. Cystoduodenostomy
 - E. Cystoenteroanastomosis
900. What operation is used for the pseudocyst of pancreas in the 3th stage of its forming:
- A. * Cystojejunostomy on the eliminated loop
 - B. External draining the cyst
 - C. Cystogastrostomy
 - D. Cystoduodenostomy
 - E. Cystoenteroanastomosis
901. What preparation does behave to blocker H2-retseptors?
- A. * tavegil
 - B. obzidan
 - C. hystdol
 - D. cerucal
 - E. oraza
902. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
903. What preparation is applied at violation the extrasecretory function of pancreas at a chronic pancreatitis:
- A. Pyracetam
 - B. Papaverin
 - C. Pantocrin
 - D. * Panzinorm
 - E. Panthenol
904. What preparations from the cytostatic group use for intensifying the chronic pancreatitis:
- A. Cyanocobalamin
 - B. Methyluracil
 - C. * 5-fluorouracil
 - D. Furadolizon
 - E. Mezimforte
905. What preparations, except for other properties, own yet and a bacteriostatic effect on Helicobacter pyloris

- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
906. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
907. What surgical pathology is a characteristic symptom of toxic «scissors» ?
- A. appendicitis
 - B. cholecystitis
 - C. pancreatitis
 - D. * peritonitis
 - E. mesadenitis
908. What symptom is most characteristic in the initial phase of peritonitis:
- A. sickliness and overhanding of pelvic peritoneum at rectal research.
 - B. dulling in declivous places at percussion of stomach
 - C. * tension of muscles the front abdominal wall.
 - D. swelling of stomach
 - E. melena
909. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
910. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
911. What type of peritonitis are fibrinogenous impositions at on a parietal and visceral peritoneum?
- A. at serosal.
 - B. at festering.
 - C. at fibrinogenous
 - D. at putrid.
 - E. * at all
912. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia

- D. Hypercalcemia
 - E. hyperkalemia
913. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforated ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pyloristhenosis
914. Widespread festering peritonitis is investigation of all above-stated diseases, except for:
- A. perforative ulcers of duodenum
 - B. phlegmonous cholecystitis
 - C. * hydrocholecystitis
 - D. destructive pancreatitis
 - E. volvulus of sigmoid bowel
915. With what disease above all things is it necessary to differentiate the acute peritonitis?
- A. bronchitis
 - B. enterorrhagia
 - C. anaemia
 - D. * acute vascular purpura
 - E. endotoxemia
916. The "light intervals" is characteristic for such phase of acute intestinal obstruction:
- A. Ileus scream"
 - B. Intoxications
 - C. Terminal
 - D. Initial manifestations
 - E. False improvement
917. A typical sign for invagination in irrigoscopy is:
- A. Cockades"
 - B. Candles"
 - C. Rat tail"
 - D. Spizharny's sign
 - E. Bartomier-Mikhelson's sign
918. Acute intestinal obstruction according to the level of obstruction is divided on:
- A. * Small intestinal, large intestinal
 - B. Small intestinal, large intestinal, caecal
 - C. High, low, middle
 - D. Long, short
 - E. High, small intestinal, large intestinal
919. Acute intestinal obstruction according to the origin is divided on:
- A. * Dynamic and mechanical
 - B. Dynamic and paralytic
 - C. Dynamic, spastic and strangulation
 - D. Strangulation and spastic
 - E. Mechanical and paralytic
920. Appearance „splashing sound" in acute intestinal obstruction is caused:
- A. * By the accumulation of liquid and gases in the afferent loop of intestine

- B. By the accumulation of liquid and gases in the efferent loop of intestine
 - C. By the presence of liquid in the abdominal cavity
 - D. By the presence of free gas in the abdominal cavity
 - E. By the presence of free gas and liquid in the abdominal cavity
921. Arterial mesenteric acute intestinal obstruction belongs to:
- A. * obturation
 - B. Strangulation
 - C. Paralytic
 - D. spastic
 - E. Mixed
922. Bloody discharge during enema in acute intestinal obstruction is the sign of:
- A. * Hemodynamic intestinal obstruction as the result of mesenteric thrombosis
 - B. Obturation intestinal obstruction
 - C. Strangulation intestinal obstruction
 - D. Adhesive intestinal obstruction
 - E. Hemorrhoids
923. Choose the correct algorithm of operative intervention for the III stage of acute intestinal obstruction:
- A. * Laparotomy, liquidation of the source of peritonitis, intestinal intubation, sanation of abdominal cavity, suturing of the abdomen or laparostomy
 - B. Laparotomy, liquidation of obstruction, intestinal intubation, sanation of abdominal cavity, suturing of the abdomen
 - C. Laparotomy, liquidation of obstruction, intestinal intubation, sanation of abdominal cavity, laparostomy
 - D. Laparotomy, liquidation of obstruction, intestinal intubation, sanation of abdominal cavity, suturing of the abdomen
 - E. Laparotomy, liquidation of obstruction, liquidation of peritonitis, sanation of abdominal cavity, suturing of the abdomen
924. Choose the correct algorithm of the operative intervention for the II stage of acute intestinal obstruction :
- A. * Laparotomy, liquidation of obstruction, intestinal intubation, sanation of abdominal cavity, suturing of the abdomen
 - B. Laparotomy, liquidation of the source of peritonitis, sanation of abdominal cavity, suturing of the abdomen
 - C. Laparotomy, liquidation of obstruction, sanation of abdominal cavity, suturing of the abdomen
 - D. Laparotomy, liquidation of obstruction, intestinal intubation, sanation of abdominal cavity, laparostomy
 - E. Laparotomy, liquidation of obstruction, liquidation of the source of peritonitis, intestinal intubation, sanation of abdominal cavity, suturing of the abdomen
925. Choose the operation, which is not performed in intestinal obstruction, caused by cancer of sigmoid intestine:
- A. * Collateral ileotransversoanastomosis
 - B. Obstructive resection (Hartmann's operation)
 - C. Colostomy
 - D. Resection of sigmoid intestine with anastomosis "end to end"
 - E. Resection of sigmoid intestine with closed anastomosis and temporal transversostomy

926. Choose the type of acute intestinal obstruction which is characterized by excretion of blood from anus:
- A. * Invagination of iliac intestine in caecum
 - B. Paralytic
 - C. Spastic
 - D. Volvulus of small intestine
 - E. Intestinal infarction
927. Conservative treatment of intestinal obstruction is indicated in all cases, except for:
- A. * Torsion
 - B. Spastic obstruction
 - C. Paralytic obstruction
 - D. Coprostasis
 - E. There is no right answer
928. Dehydration of the organism in acute intestinal obstruction most rapidly develops in:
- A. * Torsion of small intestine
 - B. Torsion of sigmoid intestine
 - C. Tumours of rectum with the phenomena of obstruction
 - D. Obturation large intestinal obstruction
 - E. Ileocaecal invagination
929. Describe the Kloiber's cups in large intestinal obstruction:
- A. * Not wide, high, single
 - B. Wide, not high, multiple
 - C. Not characteristic
 - D. Wide, not high, with folds
 - E. Of different size, localization
930. Describe the Kloiber's cups in small intestinal obstruction:
- A. * Wide, not high, multiple
 - B. Not wide, high, single
 - C. Not characteristic
 - D. Wide, not high, with folds
 - E. Of different size, localization
931. Disorders, resulting in spastic acute intestinal obstruction:
- A. * All mentioned
 - B. Hysteria
 - C. Lead colics
 - D. Neuroses
 - E. None of mentioned
932. During the examination of patient with acute intestinal obstruction: the Zege-Maitenphel's and "Obuhov's hospital" symptoms are positive. What type of obstruction it is characteristic for?
- A. * Sigmoid intestine torsion
 - B. Ileocaecal invagination
 - C. Obturation by the tumour of ascending intestine
 - D. Torsion of small intestine
 - E. All mentioned is correct
933. During the revision of the site of obstruction an afferent loop looks like:
- A. * Dilated and overfull intestinal content

- B. Spastic
 - C. Collapsed
 - D. The same as efferent loop
 - E. The diagnostics is not a necessary
934. Dynamic intestinal obstruction is divided on:
- A. * Spastic, paralytic
 - B. Strangulation, obturation, mixed
 - C. Strangulation, spastic, paralytic
 - D. Mechanical, spastic, paralytic
 - E. Mechanical and paralytic
935. For acute intestinal obstruction the followings types of operations are possible, except for:
- A. * Gastrostomy
 - B. Right-side hemicolectomy
 - C. Resections of intestine
 - D. Colostomy
 - E. Hartmann's operation
936. For intestinal obstruction caused by invagination is characteristic:
- A. * Bloody discharges from rectum
 - B. The wave-like abdominal pain
 - C. Tumorous formation in a right iliac area by palpation
 - D. Bolus-like feces
 - E. Atony of rectal sphincter
937. For paralytic intestinal obstruction is characteristic:
- A. * Delay of stool and gases and acute distension of abdomen
 - B. Asymmetry of abdomen
 - C. The expressed wave-like pain in the abdominal cavity
 - D. Zege-Maitenphel's sign
 - E. Effusion in the abdominal cavity
938. For performance of siphon enema in acute intestinal obstruction it is necessary to prepare:
- A. * 10 – 15 litres and more of warm water
 - B. 500 ml. of cold water
 - C. 1 litre of mineral water with gas
 - D. 5 litres of mineral water without gas
 - E. 500 ml. of warm water
939. For stimulation of peristalsis of intestine used:
- A. * Proserin
 - B. No-spa
 - C. Analgin
 - D. Droperidol
 - E. Dimedrol
940. For strangulation is not typical:
- A. * Normal body temperature
 - B. Tension of abdominal wall
 - C. Leucocytosis
 - D. Frequent vomit
 - E. Wahl's symptom

941. For strangulation is not typical:
- * Leucopenia
 - Tension of abdominal wall
 - Frequent vomit
 - Body temperature 37,5°C and higher
 - Wahl's symptom
942. For the differential diagnostic of acute intestinal obstruction with perforative gastric ulcer it is necessary to perform above all:
- * Plain X-ray of abdominal cavity
 - Pneumogastrography
 - Roentgenoscopy of the abdomen
 - Gastroscopy
 - Laparoscopy
943. For the high small intestinal obstruction is not characteristic:
- * Zege-Maitenphel's sign
 - The wave-like abdominal pain
 - Splashing sound" (Sklyarov's sign)
 - Multiple vomiting
 - Cloyber's cups on X-ray
944. For the initial stage of acute strangulation intestinal obstruction the most frequent first symptom is:
- * Primary reflex vomiting and pain shock
 - Appearance of outpouching of intestinal loops on abdominal wall (visible peristalsis)
 - Strengthening of peristalsis
 - Delay of stool and gases
 - Lost of peristalsis
945. For the low large intestinal obstruction all is characteristic, except for:
- * Rapid dehydration
 - Delay of stool
 - Appearances of Cloyber's cups
 - Gradual progression of symptoms
 - Abdominal distension
946. For the patient of 72 years old, who entered in late term with acute intestinal obstruction caused by the tumour of sigmoid intestine, the most acceptable tactic is the following:
- * Preparation to the operation for 2-3 hours with following Hartmann's operation
 - Inspection and performance of operation in 48-72 hours
 - Only conservative therapy
 - Infusion therapy and repeated siphon enemas
 - Urgent operation with resection of the intestine and anastomosis „end to end”
947. For the torsion of small intestine is not characteristic:
- * Zege-Maitenphel's sign
 - Asymmetry of abdomen
 - Splashing sound”
 - Multiple vomiting
 - The wave-like abdominal pain
948. For the treatment measures in obturation intestinal obstruction all mentioned belong, except:
- * Prescribing of medicines which increase intestinal peristalsis

- B. Prescribing of spasmolytics
 - C. Performing of siphon enema
 - D. Introducing of nasogastral tube for intestinal decompression
 - E. Corrections of water-electrolytes disturbances
949. For what type of acute intestinal obstruction is possible the "syndrome of minor signs"?
- A. * Obturation
 - B. Spastic
 - C. Strangulation
 - D. Invagination
 - E. All types
950. For which type of intestinal obstruction is characteristic the absence of Sklyarov's, Wahl's, Kywul's signs?
- A. * Spastic
 - B. Strangulation
 - C. Obturation
 - D. Invagination
 - E. None of mentioned
951. If the disease begins from a sudden „knife” pain, it is characteristically for:
- A. * Perforative gastric ulcer
 - B. Spastic acute intestinal obstruction
 - C. Volvulus
 - D. Peritonitis
 - E. Acute pancreatitis
952. If the high level of normal hematocrit is 40 %, what level of fluid must be infused on every 5 % increase in acute intestinal obstruction?
- A. * 1000 ml. of liquid
 - B. 200 ml. of liquid
 - C. 2500 ml. of liquid
 - D. 100 ml. of liquid
 - E. Transfusion of liquid is not indicated
953. In acute intestinal obstruction the basic X-ray sign is:
- A. * Air-fluid levels, Kloiber's cups
 - B. Expressed limitation of mobility of the right dome of diaphragm
 - C. Diffusely dilated loops of bowels
 - D. Free gas in the abdomen
 - E. Sklyarov's sign
954. In case of large intestine obstruction, the surgeon must begin the intraoperative revision:
- A. * From cecum to the rectosigmoid part of colon
 - B. From cecum to the level of obstruction
 - C. From the level of obstruction to the rectosigmoid part of colon
 - D. A revision is not necessary
 - E. A revision is not performed
955. In case of small intestine obstruction, the surgeon must begin the intraoperative revision:
- A. * From Treitz ligament to ileocecal angle
 - B. From Treitz ligament to the level of obstruction
 - C. From the level of obstruction to ileocecal angle
 - D. A revision is not necessary

- E. A revision is not performed
956. In early period of acute small intestinal obstruction in the organism of patient observed all, except:
- A. * Decrease of hematocrit
 - B. Dehydration
 - C. Increase of hematocrit
 - D. Decrease of blood potassium
 - E. There is no correct answer
957. In paralytic intestinal obstruction the stimulation of peristalsis of intestine is performed:
- A. * Only after infusion therapy and correction of hypovolemia
 - B. Depending on age
 - C. Contra-indicated
 - D. Depending on a clinical situation
 - E. Not indicated
958. In patient 75 years old two days ago aroused up the volvulus of sigmoid intestine. On operation was found out its necrosis, acute distension of the colon. What is the optimal variant of the operation?
- A. * Resection of sigmoid intestine with formation of colostomy
 - B. Resection of sigmoid intestine with anastomosis „end to end”
 - C. Resection of sigmoid intestine with anastomosis "side to side"
 - D. Sigmoidostomy
 - E. All answers are incorrect
959. In patient of 42 years old after the considerable physical exertion appeared spastic pain in the whole abdomen, nausea, double vomiting, distension of abdomen, retention of gases. What disease such clinical picture is characteristic for?
- A. * Acute intestinal obstruction
 - B. Perforative gastric ulcer
 - C. Acute pancreatitis
 - D. Acute appendicitis
 - E. Acute gangrenous cholecystitis
960. In patient of 82 years old with acute intestinal obstruction caused by the tumour of sigmoid intestine, who entered in late terms, the most rational tactic of treatment is the following:
- A. * Preparing for the operation during 2-3 hours with following performance of Hartmann's operation
 - B. Examination and performance of operation in 48-72 hours
 - C. Only conservative therapy
 - D. Infusion therapy and repeated siphon enemas
 - E. Urgent operation with performance of sigmoid intestine resection and anastomosis "end to end"
961. In patient was diagnosed acute intestinal obstruction caused by cancer of caecum. During an urgent operation was revealed, that regional lymph nodes are not enlarged, the distant metastases of the tumour are absent. What is the tactic?
- A. * Right-side hemicolectomy
 - B. Caecostomy
 - C. Resection of caecum and ascending intestine
 - D. Hartmann's operation
 - E. Ileostomy

962. In the initial stages of obturation colon obstruction the most valuable is the following medical tactic:
- A. * First conservative treatment and if it is failed – immediate operation
 - B. Urgent operation, that allows to prevent necrosis of intestine and its perforation
 - C. Performance of operation in 48-72 hours after inspection and establishment of the cause of intestinal obstruction
 - D. Direct performance of operation with formation of colostomy or enterostomy
 - E. Direct performance of Hartmann's operation
963. In what case the drainage of the abdominal cavity is inadvisable in operative treatment of acute intestinal obstruction?
- A. * None of mentioned cases
 - B. In formation of anastomosis
 - C. In formation of haematoma
 - D. In formation of stoma
 - E. In all these cases
964. In what type of intestinal obstruction one of the symptom is the red water after a cleaning enema:
- A. * Intestinal infarction
 - B. Paralytic
 - C. Spastic
 - D. Torsion of small intestine
 - E. Invagination
965. In what type of intestinal obstruction one of the symptom will be a blood discharge from rectum:
- A. * Invagination
 - B. Paralytic
 - C. Spastic
 - D. Torsion of small intestine
 - E. Intestinal infarction
966. Increased and loud peristaltic noises in early period of the disease are characteristic for:
- A. * mechanical intestinal obstruction
 - B. paralytic intestinal obstruction
 - C. Perforative gastric ulcer
 - D. Gangrenous cholecystitis
 - E. Thrombosis of mesenteric vessels
967. Invagination much more frequent in:
- A. * Children
 - B. Pregnant
 - C. Elderly people
 - D. Teenagers
 - E. Does not depend on age
968. Is a fecal vomit is characteristic sign for acute intestinal obstruction?
- A. * Yes, in III phase
 - B. Yes, in I phase
 - C. Never
 - D. Always
 - E. In obturation acute intestinal obstruction

969. Is it reasonable to perform a paranephral blockade in acute intestinal obstruction?
- A. * Yes
 - B. No
 - C. Yes, except strangulation
 - D. Yes, except obturation
 - E. Contraindicated
970. Is obligatory the consultation of anesthesiologist at suspicion on acute intestinal obstruction:
- A. * Yes
 - B. No
 - C. Only anaesthesist
 - D. Only internist
 - E. Only one of them
971. Is obligatory the digital examination of rectum at suspicion on acute intestinal obstruction?
- A. * Yes
 - B. No, if you know that acute intestinal obstruction is of obturative origin
 - C. Yes, if you know that acute intestinal obstruction is of obturative origin
 - D. No
 - E. Yes, except for children, pregnant
972. Is obligatory the X-ray examination at suspicion on acute intestinal obstruction?
- A. * Yes
 - B. No, if you know that acute intestinal obstruction is of obturative origin
 - C. Yes, if you know that acute intestinal obstruction is of obturative origin
 - D. No
 - E. Yes, except for children and pregnant
973. Is the intestinal obstruction suitable in the treatment of ²⁻²² stage of acute intestinal obstruction?
- A. * Yes
 - B. No
 - C. Only in the case of formation of anastomosis
 - D. Only in strangulation acute intestinal obstruction
 - E. Only in obturation acute intestinal obstruction
974. Is the programmable laparostomy suitable in the treatment of ²⁻²² stage of acute intestinal obstruction?
- A. * No
 - B. Yes
 - C. Only in the case of formation of anastomosis
 - D. Only in strangulation acute intestinal obstruction
 - E. Only in obturation acute intestinal obstruction
975. Large intestine obstruction is more frequently caused:
- A. * By the malignant tumours of intestine
 - B. Foreign bodies
 - C. By the adhesions of abdominal cavity
 - D. By gall-stones
 - E. Helminths
976. Mechanical intestinal obstruction is divided on:
- A. * Strangulation, obturation, mixed
 - B. Mechanical, spastic, paralytic

- C. Strangulation, spastic, paralytic
 - D. Spastic, paralytic
 - E. Mechanical and paralytic
977. Mechanism of the development of intestinal obstruction, caused by gall-stones:
- A. * Stones produce bile acids which irritate the bowel causing its spasm
 - B. Stones mechanically irritate the wall of the bowel and causing its spasm
 - C. Stones secrete toxic substances which irritate the wall of the bowel causing its spasm
 - D. Stones obturate the lumen of the bowel
 - E. All answers are correct
978. Name a radical operation in the volvulus of sigmoid colon:
- A. * A resection of sigmoid colon in any modifications
 - B. Mesosygmoplication
 - C. Gartman's operation
 - D. Mesosygmopexia
 - E. Detorsion of volvulus
979. Name a tactical error during performance of operation for intestinal obstruction:
- A. * Refuse of intestine intubation
 - B. Resection of necrotic part of bowel
 - C. Lavage of abdominal cavity
 - D. Draining of abdominal cavity
 - E. Liquidation of obstruction
980. Name duration of conservative treatment of acute intestinal obstruction in the stage of compensation?
- A. * 5-7 days
 - B. 1-2 days
 - C. 12-24 hours
 - D. To 12 hours
 - E. not less than 2 weeks
981. Name the causes of intestinal obstruction:
- A. * All mentioned causes
 - B. Long mesentery of small or large intestine which results in the mobility of their loops
 - C. Tumours of the abdominal cavity and retroperitoneal space
 - D. None of mentioned
 - E. Adhesions of abdominal cavity
982. Name the character of peristalsis in the onset of the acute intestinal obstruction:
- A. * Hyperperistalsis
 - B. Normal peristalsis
 - C. Absent
 - D. Variable
 - E. Heard only in regions upper the obstruction
983. Name the leading signs of acute intestinal obstruction:
- A. * Wave-like pain, vomiting, delay of gases and stool
 - B. Knife-like" pain, wooden abdomen, proper anamnesis
 - C. Knife-like" pain, wooden abdomen, vomiting
 - D. Wave-like pain, anaemia
 - E. Nausea, loss of appetite, metallic taste in the mouth

984. Name the method of examination which is not obligatory in acute intestinal obstruction:
- A. * All are obligatory
 - B. General blood analysis
 - C. General urine analysis
 - D. Coagulogramm
 - E. Electrolytes
985. Name the methods of intestinal intubation:
- A. * All types are acceptable, depending on a clinical situation
 - B. Through gastrostoma
 - C. Through ceco- or appendicostoma
 - D. Through the rectum
 - E. Nasogasral
986. Name the methods of operative treatment of acute intestinal obstruction for preventing of its relapse:
- A. * Operations of Noble and Child-Pott
 - B. Gartman's operation
 - C. Anisevich operation
 - D. Kirsh operation
 - E. Zeremin-cummel operation
987. Name the most frequent causes of obturation intestinal obstruction:
- A. * All mentioned
 - B. Scar and inflammatory stricture
 - C. Foreign bodies
 - D. Helminths
 - E. Tumours
988. Name the most frequent form of colon volvulus:
- A. * Volvulus of sygmoid colon
 - B. Volvulus of cecum
 - C. Volvulus of ascending colon
 - D. Volvulus of appendix
 - E. Volvulus of descending colon
989. Name the most important method of investigation in the diagnostic of „acute intestinal obstruction“:
- A. * Research of barium passage trough intestine
 - B. Plain X-ray of abdominal cavity
 - C. Fibrogastroduodenoscopy
 - D. Laparoscopy
 - E. Biochemical blood analysis
990. Name the most severe form of strangulation intestinal obstruction:
- A. * Nodulus
 - B. Volvulus
 - C. Adhesive intestinal obstruction
 - D. Invagination
 - E. None of the mentioned forms
991. Name the operation of choice in intestinal obstruction caused by solid colon tumour of hepatic angle (T3N0M0):

- A. * Right-side hemicolectomy with formation of ileotransversoanastomosis and obligatory intestinal intubation
 - B. Right-side hemicolectomy without intestinal intubation
 - C. Collateral anastomosis without the removal of tumour
 - D. A resection of hepatic angle of colon with ascendotransversoanastomosis
 - E. Formation of ileostomy in the right iliac region
992. Necrosis of intestine is possible in all variants of intestinal obstruction, except for:
- A. * Obturation of small intestine by gall-stone
 - B. Torsion of small intestine
 - C. Nodulus
 - D. Hernia strangulation
 - E. Acute mesenteric obstruction
993. Optimal access in the operative treatment of acute intestinal obstruction is:
- A. * Middle laparotomy
 - B. Phaneuil's
 - C. Vinkelman's
 - D. Fedorov's
 - E. Right pararectal
994. Select the phases of the clinical course of acute intestinal obstruction:
- A. „Iliac scream”, intoxication, terminal
 - B. Initial, to development
 - C. Initial, intoxications, „iliac scream”
 - D. „Iliac scream”, purulent, septic complications
 - E. Acute onset, false improvement, peritonitis
995. Stool and gases in volvulus of small intestine:
- A. * Is possible in case of high localization of obstruction
 - B. Is possible after digital examination of rectum
 - C. Impossible
 - D. Is possible in case of low localization of obstruction
 - E. Is possible after washing out of the stomach
996. Tactic of treatment of acute sigmoid torsion without the visual changes of intestine consists of:
- A. * Detorsion of sigmoid intestine with mesosigmoplication
 - B. Colostomy
 - C. Resection with primary anastomosis
 - D. Hartmann's operation
 - E. Liquidation of torsion
997. The "trident", "crescent" signs are characteristic for such type of acute intestinal obstruction, as:
- A. * Invagination
 - B. Spastic
 - C. Obturation
 - D. Strangulation
 - E. All kinds
998. The absolute indication for operative treatment of acute intestinal obstruction°:
- A. * III phase of the course of acute intestinal obstruction
 - B. II phase of the course of acute intestinal obstruction

- C. I phase of the course of acute intestinal obstruction
 - D. The prolonged anamnesis of acute intestinal obstruction
 - E. Dynamic acute intestinal obstruction
999. The acute obstruction of duodeno-jejunal junction is characterized by:
- A. * Vomiting by bile
 - B. The phenomena of paralytic intestinal obstruction
 - C. Diffuse distension of abdomen
 - D. Delay of stool and gases
 - E. Tenesmi
1000. The air-fluid levels (Kloiber's cups) are not characteristic for such type of acute intestinal obstruction, as:
- A. * Spastic
 - B. Paralytic
 - C. Obturation
 - D. Invagination
 - E. All kinds
1001. The cause of obturation intestinal obstruction includes all, except:
- A. * Torsion of intestinal mesentery
 - B. Inflammatory adhesions
 - C. Gall-stones
 - D. Invagination
 - E. Compression by tumour
1002. The contributory factor of the development of obturation is:
- A. * Stool stones
 - B. Long intestinal mesentery
 - C. Adhesions in abdominal cavity
 - D. All of mentioned
 - E. None of mentioned
1003. The contributory factor of the development of strangulation is:
- A. * Long intestinal mesentery
 - B. Stool stones
 - C. Gall-stones
 - D. Tumour
 - E. None of mentioned
1004. The criteria of the efficiency of gastrointestinal tract passage renewal during conservative therapy of acute intestinal obstruction is:
- A. * Pulling of gases and stool
 - B. Normalization of rectal temperature
 - C. Absence of Shchotkin-Blumberg's sign
 - D. Feeling of heartburn
 - E. None of mentioned
1005. The criterion of the conservative therapy efficiency of acute intestinal obstruction is:
- A. * Absence of Sklyarov's sign
 - B. Absence of Shchotkin-Blumberg's sign
 - C. Absence of Sitkovsky's sign
 - D. Normalization of rectal temperature
 - E. None of mentioned

1006. The decompression of gastrointestinal tract includes everything, except:
- A. * Lavage of abdominal cavity
 - B. Endoscopic intubation
 - C. Enterotomy with aspiration
 - D. Washing of the stomach
 - E. Performing of siphon enema
1007. The development of paralytic intestinal obstruction is caused by all mentioned except for:
- A. * Lead poisoning
 - B. Peritonitis
 - C. Acute pancreatitis
 - D. Retroperitoneal hematoma
 - E. Disorders of mesenteric circulation of blood
1008. The diagnosis of acute intestinal obstruction is established on the basis of:
- A. * Character of pain and roentgenologic signs
 - B. Anamnesis and laboratory information
 - C. Anamnesis, clinical research and laboratory information
 - D. Clinical course of the disease
 - E. Only by roentgenologic signs
1009. The diagnostic measures which immediately performed in suspicion on acute intestinal obstruction include everything, except:
- A. * Angiography of abdominal cavity
 - B. Auscultation of abdomen
 - C. Plain X-ray of abdominal cavity
 - D. Introducing of nasogastric tube for the decompression of intestine
 - E. Palpation of abdomen, digital examination of rectum
1010. The dividing of mechanical intestinal obstruction is based on:
- A. * Compression of mesenteric vessels
 - B. Compression of bowel loops
 - C. Involvement in a tumour process
 - D. Degree of adhesions
 - E. Medical tactic
1011. The efficiency of conservative measures for acute intestinal obstruction are determined by clinical changes except:
- A. * Decrease of height of Menber's cups on X-ray
 - B. Appearance of stool and gases
 - C. Diminishing of distension of the abdomen
 - D. Loss of peristalsis
 - E. Diminishing of pain intensity
1012. The first phase of the clinical course of acute intestinal obstruction lasts:
- A. * To 12 hours
 - B. To 2 hours
 - C. To 1 day
 - D. More than 1 day
 - E. To 1 hour
1013. The Grekov's sign in acute intestinal obstruction is:
- A. * Gaping of anus

- B. Good heard cardiac tones during auscultation of the abdomen
 - C. Dullness in the lower regions
 - D. Sound of falling drop
 - E. Noise of splash
1014. The I phase of the clinical course of acute intestinal obstruction is:
- A. Ileus scream"
 - B. Intoxications
 - C. Terminal
 - D. Initial manifestations
 - E. False improvement
1015. The II phase of the clinical course of acute intestinal obstruction is:
- A. * Intoxications
 - B. Terminal
 - C. Ileus scream"
 - D. Initial manifestations
 - E. False improvement
1016. The III phase of the clinical course of acute intestinal obstruction is:
- A. * Terminal
 - B. Intoxications
 - C. Ileus scream"
 - D. Initial manifestations
 - E. False improvement
1017. The indication for cecopexia in the operative treatment of invagination is:
- A. * For the prophylaxis of relapses
 - B. For self desinvagination
 - C. For better desinvagination
 - D. Is not indicated
 - E. Not performed
1018. The indication for operative treatment of acute intestinal obstruction is:
- A. * Mechanical acute intestinal obstruction in inefficient conservative treatment
 - B. I phase of the course of acute intestinal obstruction
 - C. II phase of the course of acute intestinal obstruction
 - D. The prolonged anamnesis of acute intestinal obstruction
 - E. Mechanical acute intestinal obstruction
1019. The Kloiber's cups on X-ray examination are:
- A. * A presence of gas and levels of fluid in the loops of bowel
 - B. Presence of gallstones in the loops of bowel
 - C. A presence of foreign bodies in the loops of bowel
 - D. A presence of intestinal content in the loops of bowel
 - E. A presence of stool stones in the loops of bowel
1020. The leading signs in acute intestinal obstruction are:
- A. * Wave-like pain, ðâîðà, delay of gases and stool
 - B. Knife-like" pain, wooden abdomen, proper anamnesis
 - C. Knife-like" pain, wooden abdomen, vomiting
 - D. Wave-like pain, anaemia
 - E. Nausea, loss of appetite, metallic taste in the mouth

1021. The Loteyssen's sign in acute intestinal obstruction is:
- A. * Good heard cardiac tones during auscultation of the abdomen
 - B. Noise of splash
 - C. Dullness in the lower regions
 - D. Sound of falling drop
 - E. Gaping of anus
1022. The manifestation of the Anshuts sign in large intestinal obstruction is:
- A. * Considerable meteorism in the right iliac region
 - B. Visible peristalsis of intestine
 - C. Sound of intestinal splash”
 - D. Sound of falling drop”
 - E. A metallic sound over the dilated bowel
1023. The manifestation of the Babuk's sign is:
- A. * A presence of blood after the repeated siphon enema
 - B. Periodic appearance of wave-like pain in the abdomen
 - C. Tenesmi during palpation of elastic tumour in the abdomen
 - D. Bleeding from the rectum
 - E. The presence of the fluid level in abdominal cavity
1024. The manifestation of the Cruvelew's sign is:
- A. * Bleeding from the rectum
 - B. Tenesmi during palpation of elastic tumour in the abdomen
 - C. A presence of the blood after the repeated siphon enema
 - D. Periodic appearance of wave-like pain in the abdomen
 - E. A presence of solitary level in abdominal cavity
1025. The manifestation of the Kywul's sign in acute intestinal obstruction is:
- A. * A metallic sound over the dilated bowel
 - B. Noise of intestinal splash
 - C. Sound of falling drop”
 - D. Gaping of anus
 - E. Sounds of the beginning, quiet of the end”
1026. The manifestation of the Rush's sign is:
- A. * Tenesmi during palpation of elastic tumour in the abdomen
 - B. Periodic appearance of wave-like pain in the abdomen
 - C. A presence of the blood after the repeated siphon enema
 - D. Bleeding from the rectum
 - E. A presence of solitary level in abdominal cavity
1027. The manifestation of the Shlange's sign in acute intestinal obstruction is:
- A. * Peristalsis of the bowel which arises up after palpation of the abdomen
 - B. Sounds of the beginning, quiet of the end”
 - C. Sound of falling drop”
 - D. Noise of intestinal splash
 - E. Gaping of anus
1028. The manifestation of the Simagin's sign is:
- A. * A presence of solitary level in abdominal cavity
 - B. Tenesmi during palpation of elastic tumour in the abdomen
 - C. A presence of the blood after the repeated siphon enema
 - D. Bleeding from the rectum

- E. Periodic appearance of wave-like pain in the abdomen
1029. The manifestation of the Tiliyax's sign is:
- A. * Periodic appearance of wave-like pain in the abdomen
 - B. Tenesmi during palpation of elastic tumour in the abdomen
 - C. A presence of blood after the repeated siphon enema
 - D. Bleeding from the rectum
 - E. A presence of solitary level in abdominal cavity
1030. The manifestation of the Wahl's sign in acute intestinal obstruction is:
- A. * Limited elastic formation in the abdomen
 - B. A metallic sound over the dilated bowel
 - C. Sound of falling drop”
 - D. Gaping of anus
 - E. Noise of intestinal splash
1031. The method of choice in sigmoid intestine torsion can be such operations, except:
- A. * Nobble's operation
 - B. Resection of sigmoid intestine with anastomosis "end to end"
 - C. Hartmann's operation
 - D. Mesosigmoplication after Gagen-Thorn
 - E. All answers are correct
1032. The most characteristic manifestation of the tumour obturation of colon is:
- A. * Chronic intestinal obstruction
 - B. Acute intestinal obstruction
 - C. Dynamic intestinal obstruction
 - D. Paralytic intestinal obstruction
 - E. Wooden abdomen
1033. The most frequent cause of small intestine mechanical obstruction is:
- A. * Adhesions of abdominal cavity
 - B. Gall-stones
 - C. Foreign bodies
 - D. Tumours
 - E. Helminths
1034. The most frequent cause of the large intestinal obstruction is:
- A. * Tumours
 - B. Invagination
 - C. Volvulus
 - D. Hemorrhoids of IV degree
 - E. Errors in the diet
1035. The most frequent cause of the mechanical intestinal obstruction is:
- A. * Adhesions
 - B. Tumours of small intestine
 - C. Internal hernia
 - D. Invagination
 - E. Dull trauma of abdomen
1036. The most frequently the sygmoid volvulus arises in:
- A. * Elderly patients with frequent constipations
 - B. Females with menstrual arrest

- C. Children
 - D. Elderly patients people with permanent diarrhea
 - E. New-borns
1037. The nodulus involves in the process:
- A. * Not less than two parts of intestine
 - B. One part of intestine
 - C. One or more parts of intestine
 - D. All parts of intestine
 - E. Parietal peritoneum
1038. The nodulus requires:
- A. * Untie the knot, if impossible – resection of the bowel
 - B. Resection of the bowel
 - C. Untie the knot
 - D. To perform the stoma. The second stage the resection of the bowel
 - E. None of mentioned
1039. The percussion in acute intestinal obstruction reveals:
- A. * Tympanic sound
 - B. No changes
 - C. Dull sound
 - D. Tympanic sound in the region of liver
 - E. Dullness in lower sites
1040. The peritonitis, caused by perforation of duodenal ulcer is characterised by such type of obstruction:
- A. * Paralytic
 - B. Spastic
 - C. Strangulation
 - D. There is no characteristic type
 - E. The obstruction can not develop in this case
1041. The peritonitis, caused by perforation of gastric ulcer is characterised by such type of obstruction:
- A. * Paralytic
 - B. Spastic
 - C. Strangulation
 - D. There is no characteristic type
 - E. The obstruction can not develop in this case
1042. The positive Gregersen's reaction is the most typical for such form of intestinal obstruction:
- A. * obturation of tumour origin
 - B. spastic
 - C. strangulation
 - D. adhesive
 - E. characteristic for all mentioned
1043. The purpose of conservative therapy in compensated acute intestinal obstruction:
- A. * All mentioned
 - B. Preoperative preparation
 - C. Treating
 - D. Detoxication
 - E. Diagnostic

1044. The purpose of conservative therapy in decompensated acute intestinal obstruction:
- A. * Preoperative preparation
 - B. Treating
 - C. Detoxication
 - D. All mentioned
 - E. None of mentioned
1045. The raspberry jelly-like feces are characteristic for:
- A. * Invagination
 - B. Stenosis of pilorus
 - C. Meckel's diverticulum
 - D. Chronic appendicitis
 - E. Chronic enterocolitis
1046. The Samarina's sign does not include:
- A. * Excess sodium in the blood plasma
 - B. Erythrocytosis
 - C. Leucocytosis
 - D. Hypoproteinemia
 - E. Drop in the chloride content of the blood serum
1047. The Samarina's sign does not include:
- A. * Hyperproteinemia
 - B. Leucocytosis
 - C. Erythrocytosis
 - D. Decreased potassium in the blood plasma
 - E. Hypovitaminosis
1048. The sign of what disease is the expressed abdominal pain, which does not relieve after intake of spasmolytics and analgetics?
- A. * Acute disturbance of mesenteric bloodflow
 - B. Chronic enterocolitis
 - C. Acute appendicitis
 - D. Acute cholecystitis
 - E. Chronic pancreatitis
1049. The similar signs of the clinical manifestation of acute intestinal obstruction and perforation ulcer are:
- A. * Acute course with sudden intensive pain and muscular tension of the abdomen
 - B. The abdominal pain with irradiation in the right shoulder-blade and shoulder
 - C. Slowly-progressive course with gradual increasing pain
 - D. Presence of diarrhea
 - E. Absence of general signs
1050. The similar signs of the clinical manifestation of acute pancreatitis and acute intestinal obstruction are:
- A. * Presence of the signs of intoxication and repeated vomiting
 - B. Presence of diarrhea
 - C. Positive Mayo-Robson sign
 - D. A high level of urine diastase
 - E. Presence of constipation
1051. The Sklyarov's sign in acute intestinal obstruction is:

- A. * Noise of splash
 - B. Good heard cardiac tones during auscultation of the abdomen
 - C. Dullness in the lower regions
 - D. Sound of falling drop
 - E. Gaping of anus
1052. The Spasokukotsky's sign in acute intestinal obstruction is:
- A. * Sound of falling drop
 - B. Good heard cardiac tones during auscultation of the abdomen
 - C. Dullness in the lower regions
 - D. Noise of splash
 - E. Gaping of anus
1053. The treatment of patients with strangulation acute intestinal obstruction which accompanied by the manifestations of peritonitis must include:
- A. * 2 hours of conservative treatment, then operative
 - B. To 12 hours conservative treatment, then operative
 - C. Immediately operative without conservative
 - D. Conservative in ambulatory conditions
 - E. During the first days conservative with the gradual increase of volume infusion
1054. The tumour obturation of cecum requires:
- A. * Right-side hemicolectomy
 - B. Resection of cecum
 - C. Cecostomy
 - D. Only ileostomy
 - E. Only intubation of small intestine
1055. To detect the presence of "sequestral fluid" in the lumen of the bowel in mechanical intestinal obstruction is possible by means of:
- A. * Ultrasound examination
 - B. X-ray examination
 - C. Irrigography
 - D. Fibrocolonoscopy
 - E. Digital examination of rectum
1056. To determine the viability of strangulated intestine it is necessary to be oriented on the followings signs, except:
- A. * Presence of strangulation groove
 - B. Color of intestine
 - C. Presence of peristalsis
 - D. The pulsation of mesenteric vessels
 - E. All answers are incorrect
1057. To differentiate acute pancreatitis with acute intestinal obstruction used such methods of examination, except:
- A. * General analysis of blood
 - B. Determination of amylase in blood
 - C. Determination of diastase in urine
 - D. Plain X-ray of abdominal cavity
 - E. All answers are correct
1058. To the criteria of permanent renewal of the gastrointestinal tract passage as efficiency of conservative treatment belongs:

- A. * Absence of stagnant content in the stomach
 - B. Absence of Shchotkin-Blumberg's sign
 - C. Normalization of rectal temperature
 - D. Feeling of heartburn
 - E. None of mentioned
1059. To the method of early diagnostics of acute intestinal obstruction belongs:
- A. * Plain X-ray of abdominal cavity
 - B. Laparoscopy
 - C. Ultrasound examination of abdominal cavity
 - D. Irrigography
 - E. Colonoscopy
1060. Treatment of patients with acute intestinal obstruction in the stage of decompensation must be:
- A. * 2-4 hours of conservative, then operative
 - B. To 24 hours of conservative, then operative
 - C. Immediately operative
 - D. During the first days conservative treatment with the gradual increase of volume of infusion
 - E. Conservative in ambulatory conditions
1061. What among mentioned is correct in relation to the diagnostics of acute intestinal obstruction:
- A. * All answers are correct
 - B. The typical roentgenologic signs appear in 3-6 hours from the onset of mechanical intestinal obstruction
 - C. In strangulation obstruction the X-ray with barium is contra-indicated
 - D. More higher the obstruction, more quick the vomiting arise up
 - E. In operated on the abdominal organs patients the cause of the obstruction is represented by adhesions, and in not operated – by tumours of intestine
1062. What among the mentioned types of intestinal obstruction has primary vascular origin:
- A. * Mesenteric obstruction
 - B. Arteriomesenteric obstruction
 - C. Adhesive
 - D. Strangulation
 - E. Obturation
1063. What are the causes of the dynamic intestinal obstruction:
- A. * All answers are correct
 - B. Leaden colic
 - C. Uremia
 - D. Pancreonecrosis
 - E. Peritonitis
1064. What are the Kloiber's cups?
- A. * Horizontal air-fluid levels
 - B. Gas bubble of the stomach
 - C. Folds of intestine
 - D. Gas sickles under the domes of diaphragm
 - E. None of mentioned

1065. What are the measures of resection of nonviable region of bowel in thrombosis of mesenteric vessels?
- A. * 30-40 sm. of afferent and 15-20 sm. of efferent part
 - B. 10-15 sm. of afferent and efferent part
 - C. Within the limits of visible healthy tissues
 - D. 30 sm. of afferent and efferent part
 - E. 20 sm. afferent and efferent part
1066. What belongs to the clinical signs of invagination intestinal obstruction?
- A. * All mentioned signs
 - B. The periodic abdominal pain
 - C. A presence of elastic, slightly painful, mobile formation in abdominal cavity
 - D. Appearance of blood in a stool
 - E. None of mentioned signs
1067. What can be the cause of mechanical intestinal obstruction?
- A. * All mentioned
 - B. Strangulated hernia
 - C. Scar strictures
 - D. Adhesions, tumours
 - E. Drainage mistakes
1068. What changes in laboratory indexes are characteristic for adhesive intestinal obstruction?
- A. * Hypoproteinemia
 - B. Lymphocytosis
 - C. Excess sodium in the blood plasma
 - D. Increased diastase
 - E. No changes
1069. What changes in laboratory indexes are characteristic for nodulus?
- A. * Hyponatremia
 - B. Lymphocytosis
 - C. Leucopenia
 - D. Increased diastase
 - E. No changes
1070. What changes in laboratory indexes are characteristic for spastic intestinal obstruction?
- A. * No changes
 - B. Leucopenia
 - C. Lymphocytosis
 - D. Excess sodium in the blood plasma
 - E. Increased diastase
1071. What complication mainly influences on the choice of operation:
- A. * Perforation and peritonitis
 - B. Bleeding
 - C. Obstruction of intestine
 - D. Distant metastases
 - E. Ulceration
1072. What does acute intestinal obstruction, caused by a tumour obturation, require?
- A. * Operative intervention
 - B. Liquidations of the tumour by chemotherapy
 - C. Liquidations of the tumour by radiotherapy

- D. Surgery only after chemotherapy
 - E. Only symptomatic treatment
1073. What does not belong to conservative therapy of acute intestinal obstruction?
- A. * Liquidation of hypervolemia
 - B. Decompression of gastrointestinal tract
 - C. The struggle against abdominal-pain shock
 - D. Detoxication
 - E. Correction of microcirculation
1074. What does not belong to conservative therapy of acute intestinal obstruction?
- A. * Liquidation of hypervolemia
 - B. Decompression of gastrointestinal tract
 - C. A struggle against abdominal-pain shock
 - D. Detoxication
 - E. Correction of microcirculation
1075. What does not belong to the fight against abdominal-pain shock?
- A. * Performing of siphon enema
 - B. Paranephral novocaine blockade
 - C. Neuroleptanalgesia
 - D. Peridural anaesthesia
 - E. Spasmolytic therapy
1076. What does the appearance of the signs of peritoneal irritation and the thrombosis of mesenteric vessels mean?
- A. * Necrosis of all layers of the bowel wall
 - B. Necrosis of muscular layer of this segment of bowel
 - C. About necrosis of serous membrane of this segment of bowel
 - D. Necrosis of mucus membrane of this segment of bowel
 - E. About paresis of intestine
1077. What does the decompression of gastro-intestinal tract include?
- A. * Stomach wash out, introduction of nasogastral zond, and performing of cleaning enemas
 - B. Only washing out of the stomach
 - C. Only introduction of nasogastral zond
 - D. Only performing of cleaning enemas
 - E. Introduction of rectal mirror
1078. What does the positive Mondor's sign in acute intestinal obstruction mean?
- A. Sounds of the beginning, quiet of the end”
 - B. Noise of intestinal splash
 - C. Frequent uncontrollable vomiting
 - D. Limited elastic formation in the abdomen
 - E. Gaping of anus
1079. What does volvulus mean?
- A. * Torsion of the bowel with its mesentery along longitudinal axis
 - B. Torsion of the bowel with the mesentery of another loop
 - C. Invagination of one part of the bowel in another
 - D. Obturation of the bowel lumen
 - E. Torsion of the bowel with its mesentery along transverse axis

1080. What is not characteristic for acute high intestinal obstruction:
- A. * Distension of abdomen in the first hours of the disease
 - B. Rapid decrease of the volume of circulating blood
 - C. Frequent prolonged vomiting
 - D. Rapid dehydration
 - E. Wave-like pain
1081. What is not typical for the high small intestinal obstruction:
- A. * Early even distension of abdomen
 - B. A presence of Cloyber's cups in the upper half of abdomen
 - C. Early vomiting
 - D. Wave-like pains
 - E. Rapid worsening of the patient condition
1082. What is the aim of conservative therapy in compensated acute intestinal obstruction?
- A. * Treating
 - B. Preoperative preparation
 - C. Detoxication
 - D. All mentioned
 - E. None of mentioned
1083. What is the aim of conservative therapy in decompensated acute intestinal obstruction?
- A. * Preoperative preparation
 - B. Treating
 - C. Detoxication
 - D. All mentioned
 - E. None of mentioned
1084. What is the aim of the operative treatment of volvulus if the bowel „alive“?
- A. * Detorsion, decompression, fixing to the abdominal wall
 - B. Detorsion, resection, fixing to the abdominal wall
 - C. Detorsion, dilation, decompression, fixing to the abdominal wall
 - D. Detorsion, dilation, decompression
 - E. Decompression, fixing to the abdominal wall
1085. What is the character of peristalsis at the beginning of acute intestinal obstruction:
- A. * Hyperperistalsis
 - B. Normal peristalsis
 - C. Absent
 - D. Variable
 - E. Heard only in the region higher to obstruction
1086. What is the definition of intestinal obstruction?
- A. * Complete or partial disturbance of passage through intestinal tract
 - B. Syndrome of acute vomiting
 - C. Constipation
 - D. Absence of stool
 - E. Disturbances of defecation
1087. What is the drawback of Gartman's operation?
- A. * Formation of stoma
 - B. Volvulus of mesentery
 - C. Development of early adhesive intestinal obstruction
 - D. A long term of patient stay in the hospital

- E. Death of patient
1088. What is the essence of arterial mesenteric intestinal obstruction?
- A. * superior mesenteric artery compresses the duodenum
 - B. duodenum compresses the superior mesenteric artery
 - C. acute intestinal obstruction on the background of mesenteric thrombosis
 - D. mesenteric thrombosis caused by obstruction
 - E. duodenum compresses inferior mesenteric artery
1089. What is the forced patient's position with embolism of mesenteric vessels?
- A. * Knee-elbow or on-side position with flexed legs
 - B. On abdomen
 - C. Semi-sitting position
 - D. On back with flexed legs
 - E. Lotus position
1090. What is the Gartman's operation in cancer of the left side of colon or rectum with obturative intestinal obstruction?
- A. * A resection of the cancered segment of bowel with suturing of distal end and formation of the stoma of proximal end of the bowel
 - B. A resection of the cancered segment with formation of primary anastomosis
 - C. Anterior resection of rectum
 - D. Formation of stoma
 - E. Formation of transversostoma
1091. What is the mechanism of gall-stones entrance in the lumen of small intestine which results in acute intestinal obstruction:
- A. * As a result of the bed sore in the walls of gallbladder and bowel which adjoins to him
 - B. From bile ducts through the Vater's papilla
 - C. Creates in the lumen of small intestine by itself
 - D. All variants are correct
 - E. None of variants is correct
1092. What is the most frequent localization of invagination:
- A. * the region of cecum
 - B. splenic angle
 - C. hepatic angle
 - D. Rectosigmoid angle
 - E. Patients with the Led's syndrome
1093. What is the volvulus?
- A. * Torsion of the bowel with its mesentery along longitudinal axis
 - B. Torsion of the bowel with the mesentery of another loop
 - C. Invagination of one part of the bowel in another
 - D. Obturation of the bowel lumen
 - E. Torsion of the bowel with its mesentery along transverse axis
1094. What kind of acute intestinal obstruction the invagination belongs to?
- A. * Mixed
 - B. Paralytic
 - C. Volvulus
 - D. Strangulation
 - E. Dynamic

1095. What kind of acute intestinal obstruction the nodule belongs to?
- A. * Strangulation
 - B. Paralytic
 - C. Volvulus
 - D. Dynamic
 - E. Mixed
1096. What kind of operation is more rationally to perform for the patient with the cancer of caecum, complicated by acute intestinal obstruction in early terms of the disease:
- A. * Right-side hemicolectomy with ileotransversoanastomosis
 - B. Formation of ileostomy
 - C. Formation of caecostomy
 - D. Hartmann's operation
 - E. Mikulich's operation
1097. What of mentioned is considered to be the morphological signs of dynamic intestinal obstruction?
- A. * All mentioned signs
 - B. Slight thickening of intestinal wall
 - C. Edema of intestinal tissues
 - D. A presence of fluid and gases in the lumen of the bowel
 - E. None of mentioned
1098. What organ diseases results in the embolism of mesenteric vessels?
- A. * Heart
 - B. Blood
 - C. Stomach
 - D. Liver
 - E. Lungs
1099. What pathology the Gray-Turner's sign is characteristic for?
- A. * Acute pancreatitis
 - B. Perforative gastric ulcer
 - C. Spastic acute intestinal obstruction
 - D. Volvulus
 - E. Peritonitis
1100. What pathology the Mayo-Robson's sign is characteristic for?
- A. * Acute pancreatitis
 - B. Perforative gastric ulcer
 - C. Spastic acute intestinal obstruction
 - D. Volvulus
 - E. Peritonitis
1101. What pathology the phrenicus sign is characteristic for?
- A. * Perforative gastric ulcer
 - B. Spastic acute intestinal obstruction
 - C. Volvulus
 - D. Peritonitis
 - E. Acute pancreatitis
1102. What pathology the Rovsing's sign is characteristic for?
- A. * Appendicitis

- B. Spastic acute intestinal obstruction
 - C. Perforative gastric ulcer
 - D. Volvulus
 - E. Acute cholecystitis
1103. What pathology the Spizharny's sign is characteristic for?
- A. * Perforative gastric ulcer
 - B. Spastic acute intestinal obstruction
 - C. Volvulus
 - D. Peritonitis
 - E. Acute pancreatitis
1104. What precedes the development of intestinal obstruction of gall-stones origin?
- A. * Attack of biliary colic and clinic of acute cholecystitis
 - B. Constipation
 - C. Diarrhea
 - D. Clinics of acute pancreatitis
 - E. Nausea and vomiting
1105. What signs are typical for intestinal obstruction?
- A. * Wahl's, Kywul's, Sklyarov's, Grekov's, Spasokukotsky's
 - B. Jobber's, Spizharny's, Rattner's
 - C. Grekov's, Murphy's, Georgievsky's, Mussi
 - D. Rovsing's, Sitkovsky's, Rozdolsky's, Bartomier-Mikhelson's, Obrastsow's, Shchotkin-Blumberg
 - E. Mozart's, Beethoven's
1106. What treatment is indicated in gall-stones intestinal obturation?
- A. * Only operative
 - B. Only conservative
 - C. Operative in the case of the development of peritonitis
 - D. Treatment is not required
 - E. Tactic depends on the size of stone
1107. What treatment tactic of acute intestinal obstruction, caused by a tumour obturation is required?
- A. * Operative intervention
 - B. Liquidation of tumour by a chemotherapy
 - C. Liquidations of tumour by radiotherapy
 - D. Operative intervention only after chemotherapy
 - E. Only symptomatic treatment
1108. What type of vomit is characteristic for acute intestinal obstruction?
- A. * Frequent, without relief
 - B. Single, with relief
 - C. With relief
 - D. Not characteristically
 - E. Double
1109. When is the intubation zond removed from intestine after the operation for acute intestinal obstruction?
- A. * On the next day after appearance of peristalsis, but not later than on 7th day
 - B. On the 5th day
 - C. On the 4th day

- D. On the 3th day
 - E. On desire of the patient
1110. When is the operative intervention for acute intestinal obstruction accompanied by the drainage of abdominal cavity?
- A. * In all mentioned cases
 - B. In formation of stoma
 - C. In increased bleeding during dissecting of adhesions
 - D. In formation of anastomosis
 - E. None of mentioned cases
1111. Where the pain irradiates in acute intestinal obstruction?
- A. * The irradiation is not characteristic
 - B. In lumbar region
 - C. In the right shoulder
 - D. In shoulder-blade
 - E. In testicle
1112. Which form of intestinal obstruction belongs the retrograde incarceration of small intestine:
- A. * Strangulation
 - B. Richter's hernia
 - C. Litre's hernia
 - D. Paralytic
 - E. Obturative
1113. Which of the phase of acute intestinal obstruction the „Gippokrath face” is characteristic for?
- A. * III
 - B. I
 - C. II
 - D. Not characteristic
 - E. In all
1114. Which type of acute intestinal obstruction is connected with previous operations:
- A. * Strangulation
 - B. Spastic
 - C. Obturation
 - D. Invagination
 - E. All kinds
1115. Which type of strangulation intestinal obstruction is characterised by the Rush's, Babuck's signs:
- A. * Invagination
 - B. Nodulus
 - C. Adhesive intestinal obstruction
 - D. Obturation by gall-stones
 - E. Obturation by tumour
1116. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appeared. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer

- D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
1117. A peristalsis is absent as a rule in:
- A. * peritonitis
 - B. paraproctitis
 - C. appendicitis
 - D. colitis
 - E. cholecystitis
1118. A peritoneal cavity is closed in:
- A. * males
 - B. females
 - C. children
 - D. persons of elderly age
 - E. young persons
1119. A peritoneal cavity is opened in:
- A. * females
 - B. males
 - C. children
 - D. persons of elderly age
 - E. young persons
1120. A peritoneum consists of sheets:
- A. * visceral, parietal
 - B. parietal
 - C. visceral
 - D. visceral, extraorganic
 - E. parietal, extraorganic
1121. A presence of peritonitis is the indication for the operation:
- A. * absolute
 - B. relative
 - C. conditional
 - D. no operation required
 - E. planned
1122. A programmed laparostomy is indicated in peritoneal endotoxiosis of:
- A. * IIIA or IV degree
 - B. II degree
 - C. IIIA-B degree
 - D. I stage
 - E. V stage
1123. Abdominal, infracostal, retropleural accesses are used for the drainage of the abscess:
- A. * anterior subphrenic
 - B. back subhepatic
 - C. anterior subhepatic
 - D. interintestinal
 - E. back subphrenic
1124. Abscess is the form of peritonitis:
- A. * focal

- B. diffuse
- C. total
- D. general
- E. unfocal

1125. Absence of diaphragm excursion in breathing is characteristic for the abscess:
- A. * subphrenic
 - B. pelvic
 - C. appendicular
 - D. Douglas space
 - E. iliac
1126. Absolute indication to operative treatment the ulcerous illness is
- A. heavy pain syndrome
 - B. * perforation of ulcer
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. giant ulcers
1127. Absolute indication to operative treatment the ulcerous illness is
- A. * voluminous bleeding
 - B. callous ulcers
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. heavy pain syndrome
1128. Absolute indication to operative treatment the ulcerous illness is
- A. ulcerous anamnesis more than 10 years
 - B. * bleeding what do not stopped with conservative
 - C. perforation ulcer in anamnesis
 - D. heavy pain syndrome
 - E. relapses more than 3 times per a year
1129. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. ulcerous anamnesis more than 10 years
 - C. relapse ulcer after the resection of stomach
 - D. relapses more than 3 times per a year
 - E. * cicatricial-ulcerous stenosis of pylorus
1130. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
1131. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct

1132. According to microbial character the peritonitis is distinguished:
- A. * aerobic, anaerobic, mixed
 - B. aerobic, clostridial, mixed
 - C. anaerobic, nonclostridial, mixed
 - D. bacteroid, streptococcal, mixed
 - E. aerobic, staphylococcal, mixed
1133. According to the course the peritonitis is distinguished:
- A. * acute, chronic, subacute
 - B. fulminant, acute, chronic
 - C. acute, torpid, subacute
 - D. acute, subacute, fulminant
 - E. acute, progressive, subacute
1134. According to the severity the peritonitis is distinguished:
- A. * slight, moderate, severe, grave, terminal
 - B. slight, moderate, severe, terminal
 - C. slight, moderate, severe
 - D. slight, moderate, severe, terminal, agonal
 - E. slight, moderate, severe, agonal
1135. According to the spread the peritonitis is distinguished:
- A. * local, diffuse, general
 - B. furunculus, diffuse, general
 - C. furunculus, diffuse, total
 - D. local, furunculus, general
 - E. local, diffuse, furunculus
1136. After the operation for peritonitis performed:
- A. * intestinal intubation
 - B. fixing of the intestine
 - C. stimulation of the intestine
 - D. dilation of the intestine
 - E. removal of the intestine
1137. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I
 - E. saved after all transferred operations
1138. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred
 - E. not saved after all operations
1139. Ambulatory treatment after the operation for diffuse peritonitis lasts:
- A. * 1,5-4 months
 - B. 2-3 weeks
 - C. 1-2 months

- D. 10-15 days
 - E. 6 months
1140. Among causes of death rate from acute surgical diseases peritonitis possesses the place:
- A. * first
 - B. second
 - C. third
 - D. fourth
 - E. fifth
1141. Appearance of pain during percussion of anterior abdominal wall in peritonitis is the sign:
- A. * Rozdolsky's
 - B. Spasokukotsky's
 - C. Shchotkin-Blumberg
 - D. Kulenkampf's
 - E. Voskresensky's
1142. Appearance of pain during sliding of fingers on anterior abdominal wall in peritonitis is the sign:
- A. * Voskresensky's
 - B. Sitkovsky's
 - C. Rovsing's
 - D. Rozdolsky's
 - E. Ortner's
1143. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
1144. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
1145. At III stage blood loss at the bleeding ulcer the patient loses
- A. over 1000 ml blood
 - B. * over 2000 ml blood
 - C. over 500 ml blood
 - D. over 2500 ml blood
 - E. over 1500 ml blood
1146. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
1147. At the duodenum ulceroperation of choice is

- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
1148. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
1149. At the III item of activity hemostasis and III stage blood loss from the I – III stage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1150. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1151. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
1152. Basic role in pathogenesis of peritonitis belongs:
- A. * to cytokines
 - B. to lymphocytes
 - C. to enzymes
 - D. to monocytes
 - E. to leucocytes
1153. Bergman's sign is characteristic for
- A. * bleeding ulcer
 - B. for cicatricial-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1154. Berhstein's sign characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers

- D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
1155. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
1156. Blood loos I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
1157. Blood loos II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
1158. Blood loos III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
1159. Cryptogenic peritonitis - is:
- A. * primary
 - B. subacute
 - C. acute
 - D. secondary
 - E. chronic
1160. De-Cerven's sing is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
1161. Deep palpation in peritonitis is impossible in the stage:
- A. * first
 - B. second
 - C. third
 - D. fourth
 - E. fifth
1162. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's

- B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
1163. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
1164. Distinguished such operative accesses for subphrenic abscesses:
- A. * peritoneal, retroperitoneal
 - B. retroperitoneal
 - C. peritoneal
 - D. pleural
 - E. pleural, retropleural
1165. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
1166. During one day the inflamed peritoneum can absorb a volume of fluid to:
- A. * 70 litres
 - B. 20 litres
 - C. 40 litres
 - D. 50 litres
 - E. 10 litres
1167. Eleker's sign is characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
1168. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- A. tumours
 - B. ulcers
 - C. bleeding polypuses
 - D. erosions
 - E. * changes of evacuation function
1169. For bleeding ulcer characteristic sign is
- A. * pain in an epigastrium;
 - B. knife-like pain;
 - C. signs irritation of peritoneum;
 - D. presence fresh blood in incandescence
 - E. melena;

1170. For bleeding ulcer characteristically
- * melena
 - tension the muscles of front abdominal wall
 - Spazarskiy's sign
 - sickliness the back vault of vagina
 - irradiation pain in a shoulder or shoulder-blade
1171. For motion of disease ulcerous illness of middle weight characteristically
- development of complications
 - * relapses 1-2 times per a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year
 - 3 and anymore relapses on a year
1172. For perforated ulcer characteristically
- * tension the muscles of front abdominal wall
 - melena
 - vomiting by coffee-grounds
 - high intestinal impassability
 - vomiting stagnant gastric maintenance
1173. For pneumoperitoneum is characteristic symptom
- * Zhober's;
 - Khelatid's;
 - Podlag's;
 - Vigats's;
 - Udin's.
1174. For subphrenic abscess is characteristic the X-ray sign:
- * displacement of the diaphragm upward
 - thinning of the diaphragm
 - thickening of the diaphragm
 - displacement of the diaphragm downward
 - displacement of the diaphragm is absent
1175. For the abscess of Douglas space used surgical access:
- * rectal
 - subpubic
 - perineal
 - suprapubic
 - obturatorial
1176. For the heavy flow of ulcerous illness characteristically
- 2 and anymore relapses on a year
 - * 3 and anymore relapses on a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year
 - 6 and more relapses are on a year
1177. For the heavy flow of ulcerous illness characteristically
- * development of complications
 - seasonal exacerbation more not frequent 1-2 times per a year
 - 1-2 relapse on a year
 - liquid, but protracted exacerbation

- E. exacerbation duration more than 10 days
1178. For the parietal peritoneum of anterior and back abdominal wall is characteristic:
- A. * exudation
 - B. imbibition
 - C. salivation
 - D. proliferation
 - E. resorbtion
1179. For the parietal peritoneum of diaphragm is characteristic:
- A. * resorbtion
 - B. imbibition
 - C. proliferation
 - D. exudation
 - E. salivation
1180. For the parietal peritoneum of lateral wall of the abdomen is characteristic:
- A. * resorbtion
 - B. imbibition
 - C. salivation
 - D. exudation
 - E. proliferation
1181. For the terminal stage of peritonitis is characteristic:
- A. * face of Gippokrath
 - B. cyanosys of the abdomen
 - C. frog belly
 - D. Caput Medusae"
 - E. tension of the abdomen
1182. Forced diuresis belongs to:
- A. * intracorporal methods of detoxication
 - B. extracorporal methods of detoxication
 - C. programmed laparostomy
 - D. peritoneal lavage
 - E. peritoneal dialysis
1183. Frequent painful urination is characteristic for such abscess:
- A. * pelvic
 - B. paraappendicular
 - C. iliac
 - D. appendicular
 - E. retroperitoneal
1184. From what department degestyive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
1185. Giant ulcer is an ulcer measuring
- A. over 4,5 cm

- B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
1186. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
1187. How many anatomic regions are involved in inflammatory process in local peritonitis?
- A. * one
 - B. 2 – 3
 - C. 3 – 6
 - D. 6 – 9
 - E. all regions
1188. How many layers does the peritoneum of mesentery of large intestine consist of?
- A. * 4
 - B. 3
 - C. 5
 - D. 6
 - E. 7
1189. How many layers does the peritoneum of mesentery of small intestine consist of?
- A. * 7
 - B. 4
 - C. 5
 - D. 6
 - E. 3
1190. How many sites of the drainage of abdominal cavity used in peritonitis?
- A. * four
 - B. two
 - C. three
 - D. one
 - E. five
1191. How to prevent the development of peritonitis in the penetrate wound of the abdomen on the prehospital stage?
- A. * to impose an aseptic bandage and inject antibiotics
 - B. to impose an aseptic bandage and inject cardiac medicines
 - C. to anesthetize the wound by novocaine. aseptic bandage
 - D. to inject antibiotics
 - E. to put a cold on the abdomen
1192. If the inflammatory process in peritonitis spreads on 2-3 anatomic regions it is named:
- A. * diffuse
 - B. local
 - C. total
 - D. general
 - E. widespread

1193. Impaired fibrinolysis is the base of pathogenesis of peritonitis:
- A. * focal
 - B. unfocal
 - C. total
 - D. general
 - E. total
1194. In case of involvement of 3 – 6 anatomic regions in inflammatory process the peritonitis is named:
- A. * diffuse
 - B. focal
 - C. local
 - D. general
 - E. total
1195. In diffuse peritonitis the inflammatory process spreads on anatomic regions:
- A. * more than 6
 - B. from 2 to 3
 - C. from 3 to 6
 - D. 1
 - E. 9
1196. In interintestinal abscess used such medical tactic:
- A. * opening of the abscess with drainage
 - B. detoxication therapy
 - C. antiinflammatory therapy
 - D. puncture of the abscess with drainage
 - E. antibiotics
1197. In paraappendicular abscess used such operative access:
- A. * retroperitoneal
 - B. McBurney
 - C. pararectal
 - D. middle
 - E. Volkovich-Dyakonov
1198. In peritonitis in intestine prevails:
- A. * secretion
 - B. exudation
 - C. absorption
 - D. resorbtion
 - E. excretion
1199. In peritonitis the development of polyorganic insufficiency results from the lesion of:
- A. * parenchymatous organs
 - B. respiratory organs
 - C. connective tissue organs
 - D. hollow organs
 - E. genitourinary organs
1200. In peritonitis the first organ which is affected by the toxins from a parietal peritoneum is:
- A. * lungs
 - B. spleen

- C. kidneys
 - D. liver
 - E. heart
1201. In peritonitis the II degree of endotoxemia requires:
- A. * intracorporeal methods of detoxication
 - B. extracorporeal methods of detoxication
 - C. programmed laparotomy
 - D. peritoneal lavage
 - E. peritoneal dialysis
1202. In peritonitis the main cause of metabolic disturbances is:
- A. * hypoxia
 - B. venous dysfunction
 - C. acidosis
 - D. alkalosis
 - E. arterial dysfunction
1203. In peritonitis the most optimal volume of the operation is:
- A. * minimal
 - B. maximal
 - C. complex
 - D. extended
 - E. radical
1204. In peritonitis the toxins from a visceral peritoneum affect first of all:
- A. * liver
 - B. spleen
 - C. kidneys
 - D. lungs
 - E. heart
1205. In peritonitis the toxins spread from a visceral peritoneum through:
- A. * portal vein
 - B. superior cava vein
 - C. inferior cava vein
 - D. hepatic vein
 - E. splenic vein
1206. In peritonitis, the formation of the pathological "third space" takes place through the sequestration of the fluid into:
- A. * abdominal cavity, retroperitoneal space
 - B. lumen of intestine, muscles
 - C. lumen of intestine, parenchymatous organs
 - D. parenchymatous organs, muscles
 - E. lumen of intestine, subcutaneous tissue
1207. In subphrenic abscess the retraction of intercostal spaces during deep breath in is the sign:
- A. * Litten's
 - B. De Gardin's
 - C. De Cervan's
 - D. Dushen's
 - E. Senator's

1208. In the case of operation for acute peritonitis performed:
- A. * wide laparotomy
 - B. laparostomy
 - C. microlaparotomy
 - D. laparocentesis
 - E. laparotomy after Volkovich-Dyakonov
1209. In the widespread forms of peritonitis after the resection of the bowel the most optimal operation is:
- A. * Formation of stoma
 - B. anastomosis " end to side "
 - C. collateral anastomosis
 - D. anastomosis " side to side "
 - E. anastomosis " end to end "
1210. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?
- A. small curvature of stomach;
 - B. back wall of stomach, nearer to small curvature;
 - C. large curvature of stomach
 - D. * cardiac part of stomach;
 - E. pylorus.
1211. In what vein is a venous outflow carried out in from a stomach?
- A. * V. Portae;
 - B. V. odd;
 - C. V. pair;
 - D. V. overhead hollow;
 - E. V. lower hollow;
1212. Interintestinal abscesses complicates as a rule:
- A. * by the diffuse peritonitis, intestinal fistula, obstruction, pilephlebitis
 - B. by the diffuse peritonitis intestinal fistula, ulcer, pilephlebitis
 - C. by the diffuse peritonitis, intestinal fistula, ulcer, gangrene
 - D. By the diffuse peritonitis, intestinal fistula, ulcer, phlegmon
 - E. By the diffuse peritonitis, intestinal fistula, ulcer, paraproctitis
1213. Interintestinal abscesses formed after peritonitis are named:
- A. * residual
 - B. chronic
 - C. recurrent
 - D. remittent
 - E. acute
1214. Large ulcer is an ulcer measuring
- A. 1-4 cm
 - B. 1-3 cm
 - C. 3-5 cm
 - D. 2- 6 cm
 - E. * 2-3 cm
1215. Late subphrenic abscesses arise up as a rule after:
- A. * 30-60 days
 - B. 10-15 days

- C. 15-20 days
 - D. 7-10 days
 - E. 60-90 days
1216. Little ulcer it is an ulcer measuring
- A. * to 0,5 cm
 - B. 0,5-1 cm
 - C. to 1,0 cm
 - D. 3 to 1,5 cm
 - E. 0,5-1,5 cm
1217. Low virulence of microbe flora on the background of normal reactivity of the organism predetermines the peritonitis:
- A. * focal
 - B. diffuse
 - C. local
 - D. total
 - E. general
1218. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
1219. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood
1220. Melena is a characteristic sign
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorus stenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1221. Mesotelocytes of peritoneum are located in such layer:
- A. * superficial
 - B. deep
 - C. middle
 - D. vascular
 - E. nervous
1222. Most informing method at a bleeding ulcer
- A. survey sciagraphy the organs of abdominal region
 - B. * EFGDS
 - C. sciagraphy the stomach with contrasting
 - D. Sonography
 - E. Laparoscopy

1223. Name classic complications of ulcerous illness
- A. bleeding, pylorostenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pylorostenosis, malignization
 - C. malignization, pylorostenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
1224. Name correct classification of pelvic abscesses:
- A. * recto-uterine, vesico-rectal, paravesical, retrorectal
 - B. recto-uterine, ischio-rectal, pelviorectal, retrorectal
 - C. recto-uterine, vesico-rectal, pelviorectal, retrorectal
 - D. recto-uterine, paravesical, pelviorectal, retrorectal
 - E. recto-uterine, paravesical, ischio-rectal, retrorectal
1225. Name etiologic classification of peritonitis:
- A. * traumatic, postoperative, perforating, inflammatory
 - B. lymphogenous, postoperative, perforating, inflammatory
 - C. contact, postoperative, perforating, inflammatory
 - D. reactive, postoperative, perforating, inflammatory
 - E. hematogenous, postoperative, perforating, inflammatory
1226. Nasogastrintestinal intubation in peritonitis is used for:
- A. * decompression of intestine
 - B. relaxation of intestine
 - C. enteral feeding
 - D. compression of intestine
 - E. stimulation of intestine
1227. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
1228. Pain in peritonitis is caused by the lesion of:
- A. * parietal peritoneum
 - B. anterior abdominal wall
 - C. back abdominal wall
 - D. internal organs
 - E. visceral peritoneum
1229. Painful, dense infiltrate in the abdomen determined by palpation are the signs of the abscess:
- A. * interintestinal
 - B. retrocecal
 - C. subphrenic
 - D. subhepatic
 - E. pelvic
1230. Patient with gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision

- D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
1231. Percussion sign of subphrenic abscess:
- A. * expanding of hepatic dullness
 - B. diminishing of hepatic dullness
 - C. absence of hepatic dullness
 - D. impaired hepatic dullness
 - E. tympanic sound over the liver
1232. Peritonitis is treated:
- A. * urgent operation
 - B. antibiotic therapy
 - C. detoxication therapy
 - D. no treatment required
 - E. conservative therapy
1233. Peritonitis resulting from surgical diseases, damages is called:
- A. * secondary
 - B. residual
 - C. primary
 - D. chronic
 - E. recurrent
1234. Pleural, retropleural operative accesses are used for the drainage of the abscess:
- A. * back subphrenic
 - B. subnephral
 - C. subhepatic
 - D. superior subphrenic
 - E. anterior subhepatic
1235. Polyorganic insufficiency is the main cause of the death of patients of:
- A. * peritonitis
 - B. cholecystitis
 - C. ulcers
 - D. myocardial infarction
 - E. appendicitis
1236. Preoperative preparation of patients with acute peritonitis must be:
- A. * individual and lasts not more than 2 hours
 - B. complex and lasts not more than 5 hours
 - C. depending on the patients condition to the complete correction of the vital functions
 - D. complex and to last not more than 6 hours
 - E. individual and lasts not more than 8 hours
1237. Programed laparapertion is:
- A. * formation of the provisional sutures on the wound with a further revision and sanation of abdominal cavity
 - B. leaving of open wound with its washing by antiseptics
 - C. suturing only of peritoneum with a further revision and sanation of abdominal cavity
 - D. suturing only of aponeurosis with a further revision and sanation of abdominal cavity
 - E. suturing of wound with a further revision of abdominal cavity
1238. Radical operation at a bleeding gastric ulcer and duodenum consists in

- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
1239. Rational operative access in peritonitis is:
- A. * median laparotomy
 - B. lower-middle laparotomy
 - C. upper-middle laparotomy
 - D. pararectal
 - E. lumbar
1240. Rectal infiltrate with fluctuation is determined in:
- A. * pelvic abscess
 - B. appendicular infiltrate
 - C. appendicular abscess
 - D. pelvic infiltrate
 - E. iliac abscess
1241. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
1242. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
1243. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
1244. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
1245. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.

1246. Subphrenic space is limited:
- A. * by a diaphragm, transverse colon, walls of the abdomen
 - B. by a diaphragm, small intestine, walls of the abdomen
 - C. by a diaphragm, gastro-colic ligament, walls of the abdomen
 - D. by a diaphragm, liver, walls of the abdomen
 - E. by a diaphragm, small omentum, walls of the abdomen
1247. Such stages of peritonitis are distinguished:
- A. * reactive, toxic, terminal
 - B. reactive, toxic, serous
 - C. toxic, fibrinous, terminal
 - D. acute, fibrinous, terminal
 - E. shock, toxic, terminal
1248. The recession between rectum and uterus is the space of:
- A. * Douglas
 - B. Dushen's
 - C. Dragsten's
 - D. Dumbadze
 - E. De Cervan's
1249. The recession between urinary bladder and rectum is named the space of:
- A. * Douglas
 - B. Dushen's
 - C. Dragsten's
 - D. Dumbadze
 - E. De Cervan's
1250. The abdominal cavity is named the space between:
- A. * anatomic structures which contain organs and tissues
 - B. peritoneum and abdominal wall
 - C. parietal and visceral peritoneum
 - D. organs and abdominal wall
 - E. diaphragm and pelvis
1251. The abscess of abdominal cavity is limited at first:
- A. * by demarcation bulwark from leucocytes
 - B. by demarcation bulwark from lymphocytes
 - C. by demarcation bulwark from fibroblasts
 - D. by demarcation bulwark from monocytes
 - E. by pyogenic membrane
1252. The abscess of Douglas is localized in space:
- A. * recto-vesicle
 - B. paravesicle
 - C. retrorectal
 - D. antevesicle
 - E. recto-colica
1253. The abscesses of abdominal cavity are divided on:
- A. * subphrenic, pelvic, interintestinal
 - B. subphrenic, rectal, subhepatic
 - C. subphrenic, interintestinal, subhepatic

- D. subphrenic, rectal, subhepatic
 - E. supraphrenic, pelvic, interintestinal
1254. The absence of somatic innervation of pelvic peritoneum in peritonitis predetermines:
- A. * The absence of muscular tension of the abdomen
 - B. The swelling of the abdomen
 - C. The wooden abdomen
 - D. The rigidity of muscles of anterior abdominal wall
 - E. The retraction of the abdomen
1255. The absorbal ability of the diaphragm predetermines formation of abscess:
- A. * subphrenic
 - B. interintestinal
 - C. pelvic
 - D. appendicular
 - E. supraphrenic
1256. The base of pathogenesis of the focal peritonitis is:
- A. * increased activity of the coagulative system
 - B. absence of activity of the coagulative system
 - C. impaired activity of the coagulative system
 - D. activity of the coagulative system is not changed
 - E. presence of activity of the coagulative system
1257. The cavity between organs and abdominal wall is called:
- A. * peritoneal
 - B. abdominal
 - C. pelvic
 - D. retroperitoneal space
 - E. interintestinal
1258. The clinic of pelvic abscess is often accompanied :
- A. * by absence of tension of anterior abdominal wall
 - B. by tension of anterior abdominal wall
 - C. by painfulness of anterior abdominal wall
 - D. by a wooden abdomen
 - E. swelling of the abdomen
1259. The connective tissue membrane has such abscess of the abdominal cavity:
- A. * formed
 - B. primary
 - C. early
 - D. not formed
 - E. loose
1260. The connective tissue membrane is formed in peritonitis:
- A. * secondary focal
 - B. diffuse
 - C. general
 - D. primary focal
 - E. total
1261. The diarrhea often is the sign of such abscess:
- A. * pelvic

- B. paraappendicular
 - C. iliac
 - D. appendicular
 - E. retroperitoneal
1262. The difference of more than 2° N between the morning and evening temperatures occurs in:
- A. * subphrenic abscess
 - B. diffuse peritonitis
 - C. general peritonitis
 - D. subphrenic infiltrate
 - E. local peritonitis
1263. The disturbance of protein metabolism in peritonitis is represented by:
- A. * diminishing of albumins, increasing of globulins
 - B. increasing of albumins and globulins
 - C. increasing of albumins, diminishing of globulins
 - D. diminishing of albumins and globulins
 - E. lbumins, globulins is not changed
1264. The draining of Douglas space is performed through:
- A. * rectum
 - B. foramen obturatorium
 - C. perineum
 - D. sacrum region
 - E. anterior abdominal wall by retroperitoneal
1265. The euphoric state of patient in peritonitis arises up as a rule in the stage:
- A. * II
 - B. I
 - C. III
 - D. IV
 - E. V
1266. The focal peritonitis is limited by such anatomic structures:
- A. * omentum, mesentery and the wall of bowel
 - B. liver, adhesions
 - C. appendix, cecum
 - D. adhesions, vessels, the wall of bowel
 - E. adhesions, round ligament of liver
1267. The frequency of what pathogenic flora increases in peritonitis:
- A. * anaerobic nonclostridial
 - B. aerobic clostridial
 - C. aerobic nonclostridial
 - D. anaerobic clostridial
 - E. aerobic
1268. The general surface of peritoneum is:
- A. 2 - 3 m²
 - B. 1 - 2 m²
 - C. 0,5 - 1 m²
 - D. 3 - 4 m²
 - E. 5 - 6 m²

1269. The immobility of back bone at walking of patient in peritonitis is the sign:
- A. * Senator's
 - B. Dushen's
 - C. Lithen's
 - D. Mussi
 - E. Voskresensky's
1270. The immobility of back bone in a subphrenic abscess is the sign:
- A. * Senator's
 - B. Dushen's
 - C. Lithen's
 - D. Sitkovsky's
 - E. Spasokukotsky's
1271. The increased peristalsis is characteristic for such abscess:
- A. * interintestinal
 - B. subphrenic
 - C. subhepatic
 - D. retrocecal
 - E. subsplenic
1272. The infection in peritonitis in the most cases is:
- A. * aerobico-anaerobic
 - B. anaerobic
 - C. nonclostridial
 - D. clostridial
 - E. aerobic
1273. The infection in peritonitis in the most cases represented by:
- A. * E. coli
 - B. Bacteroids
 - C. Pneumococcus
 - D. Pseudomonas aeruginosa
 - E. Protheus
1274. The interintestinal abscesses formed near the primary source of infection are named:
- A. * primary
 - B. lymphogenous
 - C. metastatic
 - D. hematogenous
 - E. secondary
1275. The involvement of epigastric region during breath in and its outpouching during breath out in peritonitis is the sign:
- A. * Dushen's
 - B. Lithen's
 - C. Senator
 - D. Mussi
 - E. Voskresensky's
1276. The irradiation of pain in the lumbar region is characteristic for such abscess:
- A. * subphrenic
 - B. retrocecal
 - C. retroperitoneal

- D. appendicular
 - E. subhepatic
1277. The main cause of the development of peritonitis is:
- A. * infection
 - B. trauma
 - C. shock
 - D. impaired immunity
 - E. impaired resistance
1278. The main spread of infection in peritonitis is:
- A. * hematogenous and lymphogenous
 - B. exogenous
 - C. lymphogenous
 - D. hematogenous
 - E. endogenous
1279. The middle thickness of peritoneum is:
- A. * 0,7 – 1,1 mm.
 - B. 0,5-1,0 mm.
 - C. 0,3 – 0,5 mm.
 - D. 1 - 2 mm.
 - E. 2 - 3 mm.
1280. The morphologic structure of peritoneum:
- A. * connective tissue
 - B. fibrous
 - C. serous
 - D. visceral
 - E. parietal
1281. The most information instrumental method of diagnostics of peritonitis is:
- A. * laparoscopy
 - B. ultrasound examination
 - C. roentgenoscopy
 - D. laparocentesis
 - E. computer tomography
1282. The most informative additional method of examination in peritonitis is:
- A. * plain X-ray
 - B. irrigography
 - C. esophagogastroscopy
 - D. barium swallow
 - E. coprologic examination
1283. The most informative instrumental method of diagnostics of peritonitis is:
- A. * laparoscopy
 - B. ultrasound examination
 - C. thermography
 - D. esophagogastroscopy
 - E. barium swallow
1284. The most informative instrumental method of diagnostics of peritonitis is:
- A. * laparocentesis

- B. Rectoscopy
 - C. rheovasography
 - D. thermography
 - E. roentgenoscopy
1285. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
1286. The organs are fixed around the site of inflammation in the focal peritonitis by:
- A. * fibrin
 - B. exudate
 - C. transsudate
 - D. prothrombin
 - E. thrombin
1287. The painfulness during palpation in a supraclavicular region in subphrenic abscess is the sign:
- A. * Mussi-Georgievsky
 - B. Bartomier-Mikhelson's
 - C. Yaure-Rozanov's
 - D. Shchotkin-Blumberg's
 - E. Rovsing's
1288. The painfulness of anterior wall of rectum is the sign:
- A. * Kulenkampf's
 - B. Krymov's
 - C. Kocher
 - D. Culen's
 - E. Crown's
1289. The paralysis of microcirculation in peritonitis develops in such stage:
- A. * terminal
 - B. toxic
 - C. reactive
 - D. acute
 - E. subacute
1290. The participation of anterior abdominal wall in the act of breathing in peritonitis:
- A. * does not take part
 - B. takes part partly
 - C. takes part
 - D. takes part doubtfully
 - E. takes part fastly
1291. The peritonitis caused by the contamination of the peritoneum by hematogenous and lymphogenous way is called:
- A. * primary
 - B. chronic
 - C. acute
 - D. secondary

- E. recurrent
1292. The peritonitis develops in such concentration of microorganisms in 1 ml. of exudate:
- A. * 1?107
 - B. 1?103
 - C. 1?105
 - D. 1?101
 - E. 1?109
1293. The peritonitis of 3? degree requires:
- A. * extracorporal methods of detoxication
 - B. laparocentesis
 - C. programmed laparostomy
 - D. laparoscopy
 - E. lavage
1294. The peritonitis of IV degree requires:
- A. * programmed laparostomy
 - B. laparoscopy
 - C. lavage
 - D. extracorporal methods of detoxication
 - E. laparocentesis
1295. The retraction of epigastric region during breath in and its outpouching during breath out in subphrenic abscess is the sign:
- A. * Dushen's
 - B. Lithen's
 - C. De Cervan's
 - D. De Gardin's
 - E. Senator's
1296. The secretory function stomach is carried out the next membrane of stomach
- A. * mucous membrane
 - B. internal muscular layer
 - C. serosal
 - D. mucous submembrane
 - E. external layer
1297. The separation by tissue structures parallel to the development of inflammatory process leads to the form of peritonitis:
- A. * the primary focal
 - B. the secondary diffuse
 - C. diffuse
 - D. the secondary focal
 - E. the primary diffuse
1298. The signs of peritoneal irritation are absent in such abscess:
- A. * pelvic
 - B. paraappendicular
 - C. interintestinal
 - D. subphrenic
 - E. appendicular
1299. The stages of the operative treatment of peritonitis:

- A. * liquidation of the cause, sanation, drainage of the abdominal cavity
 - B. laparotomy, sanation, drainage of the abdominal cavity
 - C. liquidation of the cause, drainage of abdominal cavity, suturing of wound
 - D. laparotomy, liquidation of the cause, drainage of abdominal cavity
 - E. liquidation of the cause, sanation
1300. The subphrenic abscess is treated by:
- A. * opening and drainage of abscess
 - B. puncture, washing out by antiseptics
 - C. resection of abscess
 - D. opening of abscess, washing out by antiseptics
 - E. puncture
1301. The subphrenic abscesses are caused:
- A. * by negative pressure during excursion of diaphragm
 - B. by the inflammatory reaction of diaphragm
 - C. by the presence of free space
 - D. by positive pressure during excursion of diaphragm
 - E. by position of patient
1302. The tenesmi often are the signs of such abscess:
- A. * pelvic
 - B. paraappendicular
 - C. iliac
 - D. retroperitoneal
 - E. appendicular
1303. The tense resistance of anterior abdominal wall in peritonitis is the sign:
- A. * Mondor's
 - B. Voskresensky's
 - C. Kulenkampf's
 - D. Shchotkin-Blumberg
 - E. Pasternatsky's
1304. The toxic phase of peritonitis above all things is caused by:
- A. * endotoxins
 - B. exotoxins
 - C. toxins
 - D. polytoxins
 - E. monotoxins
1305. The toxins in peritonitis spread from a parietal peritoneum through:
- A. * inferior cava vein
 - B. superior cava vein
 - C. portal vein
 - D. hepatic vein
 - E. splenic vein
1306. The vascular layer of peritoneum is located under:
- A. * superficial
 - B. serous
 - C. deep
 - D. fibrous
 - E. nervous

1307. The wooden abdomen in peritonitis is characteristic for the stage:
- A. * first
 - B. third
 - C. fourth
 - D. second
 - E. fifth
1308. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scarry-ulcerous stenosis
 - B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
1309. To the focal peritonitis belongs:
- A. * infiltrate of abdominal cavity
 - B. diffuse, limited
 - C. total, unlimited
 - D. total
 - E. general
1310. To the gastric – intestinal bleeding of un ulcerous etiology belong
- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.
1311. To the local peritonitis belongs:
- A. * abscess
 - B. diffuse
 - C. general
 - D. furunculus
 - E. total
1312. Udin's sing at a perforated ulcer is
- A. * feeling at palpation shove the gases which penetrate through the perforated opening
 - B. dulling perforated sound in the lateral departments of stomach
 - C. disappearance of hepatic dullness
 - D. irradiation pain in a shoulder or shoulder-blade
 - E. sickliness the back vault of vagina
1313. Vasoconstriction of microcirculation in peritonitis develops in such stage:
- A. * reactive
 - B. toxic
 - C. terminal
 - D. acute
 - E. subacute
1314. Vasodilatation of microcirculation in peritonitis develops in such stage:
- A. * toxic
 - B. reactive
 - C. terminal

- D. acute
 - E. subacute
1315. Vomiting coffee-grounds is a characteristic sign
- A. * bleeding ulcer
 - B. penetrative ulcers
 - C. perforated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorus stenosis
1316. Vomiting in peritonitis:
- A. * does not relief
 - B. relief
 - C. does not change the state of patient
 - D. deteriorates the state of patient
 - E. improves the state of patient
1317. Vomiting what arose up in 4-6 hours after eating characteristic for
- A. chronic alcoholic gastritis
 - B. cancer and ulcers of cardia
 - C. * pylorus ulcers
 - D. achalasia of gullet
 - E. ulcer and cancer the body of stomach
1318. What are the changes, which don't require the drainage of abdominal cavity in peritonitis:
- A. * A presence of inflammatory changes of the peritoneum + absence of exudate
 - B. intestinal content in abdominal cavity + possibility of anastomosis leakage
 - C. presence of turbid exudate + presence of pus in abdominal cavity + presence of inflammatory infiltrate
 - D. inflammatory changes of peritoneum + presence of turbid exudate
 - E. inflammatory changes of peritoneum + presence of pus in abdominal cavity F. a presence of pus in abdominal cavity + infection of abdominal cavity
1319. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodwnum?
- A. * Tarry excrement
 - B. Presence on the formed excrement of strokes of red blood
 - C. Littlechanged blood in an excrement
 - D. Excrement of the raspberry colouring with the admixtures of mucus
 - E. Acholic excrement
1320. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- A. perforation
 - B. perforation + bleeding
 - C. pylorus stenosis
 - D. malignization + penetration
 - E. * bleeding
1321. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement

- D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
1322. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol
 - B. Liciviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
1323. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy
 - E. the all transferred does not belong
1324. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
1325. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E
 - D. estrogens
 - E. STH
1326. What is the treatment tactic in pelvic abscess?
- A. * opening and drainage of the abscess,
 - B. antibiotics
 - C. antiinflammatory therapy
 - D. puncture of the abscess, drainage
 - E. detoxication therapy
1327. What is the treatment tactic in subphrenic abscess?
- A. * operative treatment
 - B. antiinflammatory medicines
 - C. conservative treatment
 - D. antibiotics
 - E. contemplate tactic
1328. What is the volume of fluid contained in peritoneal cavity?
- A. * 10 ml.
 - B. 100 ml.
 - C. 30 ml.
 - D. 40 ml.
 - E. 50 ml.

1329. What kind of abdomen is characteristic for the reactive stage of peritonitis?
- A. * wooden
 - B. soft
 - C. bloated
 - D. rigidit
 - E. retracted
1330. What localization of ulcer is most characteristic for the patients of elderly and senile age
- A. * cardial department of stomach
 - B. overhead third of gullet
 - C. lower third of gullet
 - D. bulb of duodewnum
 - E. small curvature
1331. What method diagnostics hte ulcerous illness most informing
- A. * esophagogastroduodenoscopy
 - B. analysis of excrement on the hidden blood
 - C. X-ray
 - D. global analysis of blood
 - E. research of gastric secretion
1332. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- A. On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - B. On blood volume
 - C. * On an arteriotony, pulse, state of patient
 - D. On a globular volume
 - E. On the level of thrombocytes
1333. What phase of peritonitis lasts after 72 hours?
- A. * terminal
 - B. hemodynamic
 - C. polyorganic
 - D. toxic
 - E. reactive
1334. What phase of peritonitis lasts for 24 hours?
- A. * reactive
 - B. toxic
 - C. terminal
 - D. hemodynamic
 - E. polyorganic
1335. What phase of peritonitis lasts from 24 to 72 hours?
- A. * toxic
 - B. hemodynamic
 - C. reactive
 - D. terminal
 - E. polyorganic
1336. What preparation does behave to blocker H2-retseptors?
- A. * tavegil
 - B. obzidan
 - C. hystdol

- D. cerucal
 - E. oraza
1337. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
1338. What preparations, except for other properties, own yet and a bacteriostatic effect on *Helicobacter pylori*
- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
1339. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
1340. What stage of peritonitis is characterised besides general manifestation the clinic of intestinal obstruction?
- A. * second
 - B. first
 - C. third
 - D. fourth
 - E. fifth
1341. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
1342. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
1343. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia

1344. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforated ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pylorostenosis
1345. Which stage of peritonitis is characterized by endotoxication?
- A. * toxic
 - B. terminal
 - C. reactive
 - D. hemodynamic
 - E. polyorganic
1346. Which stage of peritonitis is characterized by infection generalization?
- A. * terminal
 - B. toxic
 - C. hemodynamic
 - D. reactive
 - E. polyorganic
1347. Which stage of peritonitis is characterized by the reaction of the organism on infecting of abdominal cavity?
- A. * reactive
 - B. toxic
 - C. terminal
 - D. polyorganic
 - E. hemodynamic
1348. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appeared. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
1349. Absolute indication to operative treatment the ulcerous illness is
- A. heavy pain syndrome
 - B. * perforation of ulcer
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. giant ulcers
1350. Absolute indication to operative treatment the ulcerous illness is
- A. * voluminous bleeding
 - B. callous ulcers
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. heavy pain syndrome
1351. Absolute indication to operative treatment the ulcerous illness is

- A. ulcerous anamnesis more than 10 years
 - B. * bleeding what do not stopped with conservative
 - C. perforation ulcer in anamnesis
 - D. heavy pain syndrome
 - E. relapses more than 3 times per a year
1352. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. ulcerous anamnesis more than 10 years
 - C. relapse ulcer after the resection of stomach
 - D. relapses more than 3 times per a year
 - E. * cicatrical-ulcerous stenosis of pylorus
1353. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
1354. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
1355. Acquire hernia could be all, except:
- A. Neurological
 - B. Traumatic
 - C. Recurrent
 - D. Postoperative
 - E. * Post-natal
1356. Acute ileus arises up at presence in the hernia sac:
- A. Urinary bladder
 - B. Salpinx
 - C. Omentum
 - D. Subcutaneous fatty tissue
 - E. * Loops of bowel
1357. After the method of Kukudzhanov it is performed hernioplasty of:
- A. Umbilical hernia
 - B. Femoral and inguinal hernias
 - C. Epigastroceles
 - D. Femoral hernia
 - E. * Inguinal hernia
1358. After the method of Meyo it is performed hernioplasty of:
- A. Femoral and inguinal hernias
 - B. Epigastroceles
 - C. Femoral hernia
 - D. Inguinal hernia
 - E. * Umbilical hernia

1359. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I
 - E. saved after all transferred operations
1360. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred
 - E. not saved after all operations
1361. Any hernia of abdomen consists of:
- A. Hernia sack
 - B. Hernia sack and content
 - C. Hernia content
 - D. Hernia gate and hernia sack
 - E. * Hernia gate, hernia sack, hernia content
1362. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
1363. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
1364. At III stage blood loss at the bleeding ulcer the patient loses
- A. over 1000 ml blood
 - B. * over 2000 ml blood
 - C. over 500 ml blood
 - D. over 2500 ml blood
 - E. over 1500 ml blood
1365. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
1366. At incarcerated hernia after the dissection of hernia sack, the next manipulation is:
- A. Finish operation
 - B. Sequence of manipulations are not important

- C. To perform puncture of hernia sack and delete hernia water
 - D. To cut a incarceration ring
 - E. * To cut a hernia sack
1367. At incarceration hernia microcirculation disorders could be:
- A. In abdominal cavity
 - B. In a hernia gate
 - C. In a hernia sack
 - D. In a distal bowel
 - E. * In a proximal bowel
1368. At incarceration of diaphragmatic hernia more frequent arises up:
- A. Syndrome of respiratory insufficiency
 - B. Dyspepsia
 - C. Intoxication syndrome
 - D. Pain a syndrome
 - E. * Pain shock with violation of cardiac rhythm
1369. At incarceration of hernia the most deep changes arise up in:
- A. In all simultaneously
 - B. In the peritoneum
 - C. In the serosa
 - D. In the muscular tunic
 - E. * In the mucus tunic
1370. At left-side non-reducible femoral hernia is used:
- A. Methods of Martinov, Postempsky
 - B. Methods of Martinov, Zhirar, Spasokukotsky, Kimbarovsky
 - C. Methods of Bassini, Kukudzhanov, Postempsky
 - D. Methods of Sapezhko, Meyo
 - E. * Methods of Bassini, Rudzhi, Parlavecho
1371. At the duodenum ulceroperation of choice is
- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
1372. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
1373. At the III item of activity hemostasis and III sage blood loss from the I – III sage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)

1374. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1375. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
1376. Auscultative sign of presence in the hernia sack of loop of bowel:
- A. Drum shot
 - B. Noise of falling drop
 - C. Clang
 - D. Absence of peristaltic waves
 - E. * Presence of peristaltic waves
1377. Auscultative sign of presence in the hernia sack of omentum is:
- A. Drum shot
 - B. Noise of falling drop
 - C. Clang
 - D. Presence of peristaltic waves
 - E. * Absence of peristaltic waves
1378. Autoplastic methods of surgical treatment of femoral hernia:
- A. Method of Spasokukotsky
 - B. Method of Meyo
 - C. Method of Rudzhi
 - D. Methods of Lockwood and Abrazhanov
 - E. * Method Caravanov
1379. Bergman's sing is characteristic for
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1380. Berhtein's sing characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
1381. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions

- D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
1382. Blood loss I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
1383. Blood loss II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
1384. Blood loss III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
1385. By Bassini method the plastic are performed:
- A. All of the listed hernia
 - B. Umbilical hernia
 - C. Only inguinal hernia
 - D. Only femoral hernia
 - E. * Inguinal and femoral hernia
1386. By Spasokukocky method treated:
- A. A method can be applied at all hernias
 - B. Umbilical hernia
 - C. Femoral hernia
 - D. Direct inguinal hernia
 - E. * Oblique inguinal hernia
1387. By the method of Postempsky it is performed hernioplasty of:
- A. Femoral and inguinal hernias
 - B. Epigastroceles
 - C. Femoral hernia
 - D. Umbilical hernia
 - E. * Inguinal hernia
1388. By the method of Sapezhko it is performed hernioplasty of:
- A. Femoral and inguinal hernias
 - B. Epigastroceles
 - C. Femoral hernia
 - D. Inguinal hernia
 - E. * Umbilical hernia
1389. Congenital inguinal hernia could be?
- A. Upper and lower

- B. Combined
 - C. Oblique and direct
 - D. Direct
 - E. * Oblique
1390. De-Cerven's sign is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
1391. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
1392. Differential diagnostics of femoral hernia is performed with:
- A. Lymphadenitis
 - B. Inguinal hernia
 - C. Varicosity
 - D. Cold abscess
 - E. * All listed
1393. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
1394. Duration of dynamic supervision if incarcerated hernia is replaced:
- A. 2 hours
 - B. 1 hour
 - C. 12 hours
 - D. 6 hours
 - E. * 24 hours
1395. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
1396. During Postempsky method:
- A. Forming of double-layer of white line of abdomen
 - B. Suturing of defect of anterior abdominal wall
 - C. Strengthening of posterior wall of inguinal channel
 - D. Strengthening of anterior wall of inguinal channel
 - E. * The closing of inguinal interval

1397. During retrograde incarceration:
- A. Large part of bowel is jammed
 - B. All of bowel is jammed
 - C. Only small part of bowel is jammed
 - D. Jammed part of loop which located in a hernia sack
 - E. * Jammed part of bowel, located in the abdominal cavity
1398. During what operation is performed suturing between the ligament of Kuper, by the vagina of direct muscle of abdomen and aponeurosis of transversal muscle:
- A. The plastic by Bassini
 - B. Method of Postempsky
 - C. Method of Martinov
 - D. Method of Spasokukotsky
 - E. * Method of Kukudzhanov
1399. Eleker's sign is characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
1400. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- A. tumours
 - B. ulcers
 - C. bleeding polypuses
 - D. erosions
 - E. * changes of evacuation function
1401. For a hernioplasty by skins materials is used:
- A. Method of Martinov
 - B. Method of Rudzhi
 - C. Method of Meyo
 - D. Method of Postempsky
 - E. * Method of Janov
1402. For bleeding ulcer characteristic sign is
- A. * pain in an epigastrium;
 - B. knife-like pain;
 - C. signs irritation of peritoneum;
 - D. presence fresh blood in incandescence
 - E. melena;
1403. For bleeding ulcer characteristically
- A. * melena
 - B. tension the muscles of front abdominal wall
 - C. Spazarskiy's sign
 - D. sickliness the back vault of vagina
 - E. irradiation pain in a shoulder or shoulder-blade
1404. For closing of hernia gate from the femoral side is used:
- A. Method of Spasokukocky
 - B. Method of Meyo
 - C. Method Caravanov

- D. Method of Rudzhi
 - E. * Methods of Lokvud and Abrazhanov
1405. For diaphragmatic hernia a typical symptom is:
- A. Negative cough sign
 - B. Nausea
 - C. Pain
 - D. Positive cough sign
 - E. * Feeling of grumbling in a thorax on the side of hernia
1406. For hernioplasty of wide femoral fascia is used:
- A. Method of Martinov
 - B. Method of Zhirar
 - C. Method of Postempsky
 - D. Method of Meyo
 - E. * Method of Kirschner
1407. For motion of disease ulcerous illness of middle weight characteristically
- A. development of complications
 - B. * relapses 1-2 times per a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 3 and anymore relapses on a year
1408. For perforated ulcer characteristically
- A. * tension the muscles of front abdominal wall
 - B. melena
 - C. vomiting by coffee-grounds
 - D. high intestinal impassability
 - E. vomiting stagnant gastric maintenance
1409. For pneumoperitoneum is characteristic symptom
- A. * Zhober's;
 - B. Khelatid's;
 - C. Podlag's;
 - D. Vigats's;
 - E. Udin's.
1410. For strengthening of anterior wall of inguinal channel is used:
- A. Method of Kukudzhanov
 - B. Method of Postempsky
 - C. Method of Bassini, Kukudzhanov
 - D. Method of Cherni, Ru
 - E. * Methods of Martinov, Zhirar, Spasokukotsky
1411. For strengthening of posterior wall of inguinal channel is used:
- A. Methods of Kirschner
 - B. Methods of Postempsky
 - C. Methods of Cherni, Ru
 - D. Methods of Martinov, Zhirara, Spasokukotsky
 - E. * Method of Bassini, Kukudzhanov
1412. For the heavy flow of ulcerous illness characteristically
- A. 2 and anymore relapses on a year

- B. * 3 and anymore relapses on a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 6 and more relapses are on a year
1413. For the heavy flow of ulcerous illness characteristically
- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days
1414. From what department degestiyve tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
1415. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
1416. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
1417. Hernioplasty by Kimbarovsky is mean:
- A. Use of alloplastic material
 - B. Strengthening of lateral wall of inguinal channel
 - C. Narrowing of inguinal channel
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Renewing the inguinal channel to it normal state
1418. Hernioplasty by Kukudzhanov is mean:
- A. Use of alloplastic material
 - B. Strengthening of lateral wall of inguinal channel
 - C. Narrowing of inguinal channel
 - D. Strengthening of anterior wall of inguinal channel
 - E. * Strengthening of posterior wall of inguinal channel
1419. Hernioplasty by Martinov is mean:
- A. Use of alloplastic material
 - B. Strengthening of lateral wall of inguinal channel
 - C. Narrowing of inguinal channel
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Strengthening of anterior wall of inguinal channel

1420. Hernioplasty by Rudzhi is mean:
- A. Strengthening of lateral wall of inguinal channel
 - B. Narrowing of inguinal channel
 - C. Strengthening of anterior wall of inguinal channel
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Closing of hernia gate from the side of femoral channel
1421. Hernioplasty by Zhirar is mean:
- A. Use of alloplastic material
 - B. Strengthening of lateral wall of inguinal channel
 - C. Narrowing of inguinal channel
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Strengthening of anterior wall of inguinal channel
1422. How many days patients stays in hospital after hernioplasty:
- A. 17 days
 - B. 14 days
 - C. 12 days
 - D. 10 days
 - E. * 2-5 days
1423. If a bowel is jammed in a hernia sack, the diameter will increase in:
- A. None of the listed part
 - B. Remote loop of bowel
 - C. Loop in a hernia sack
 - D. Distal loop of bowel
 - E. * Proximal loop of bowel
1424. If after dissecting of incarceration ring there are signs of necrosis of bowel, surgeon must:
- A. To perform the resection of bowel in the distance 50 cm
 - B. To perform the resection of bowel in the distance 60 cm
 - C. To perform the resection of bowel in the distance 70 cm
 - D. To replace a bowel in an abdominal cavity
 - E. * To perform the resection of bowel in the distance 25-30 cm proximally and 10-15 cm distally
1425. If during introduction of finger to the superficial ring of inguinal channel the pulsation of lower epigastric artery is determined laterally to the hernia swelling, its mean:
- A. Incarcerated inguinal hernia
 - B. Femoral hernia
 - C. Umbilical hernia
 - D. Oblique inguinal hernia
 - E. * Direct inguinal hernia
1426. If during introduction of finger to the superficial ring of inguinal channel the pulsation of lower epigastric artery is determined medially to the hernia swelling, its mean:
- A. Incarcerated inguinal hernia
 - B. Umbilical hernia
 - C. Direct inguinal hernia
 - D. Femoral hernia
 - E. * Oblique inguinal hernia
1427. If hernia, complicated by phlegmon, is jammed:

- A. Performed hernioplasty by Kukudzhanov
 - B. Performed hernioplasty by Spasokukocky
 - C. Performed hernioplasty by Sapezhko
 - D. Performed hernioplasty by Bassini
 - E. * A hernioplasty is not performed
1428. If in a hernia sack organs compressed with lost of function, necrosis, its mean:
- A. Damage of hernia
 - B. Tumor of hernia
 - C. Nonreducible hernia
 - D. Inflammation of hernia
 - E. * Incarceration of hernia
1429. In case of incarceration of loop of bowel, above the hernia sack will be:
- A. Absence of sounds
 - B. Clang
 - C. Sonorous sound
 - D. Dull sound
 - E. * Tympanit
1430. In the case of direct inguinal hernia lower epigastric vessels are located:
- A. Behind a hernia sack
 - B. Higher hernia sack
 - C. Medially from a hernia sack
 - D. Below hernia sack
 - E. * Lateral to hernia sack
1431. In the case of incarceration of omentum above the hernia sack will be:
- A. Absence of sounds
 - B. Clang
 - C. Sonorous sound
 - D. Tympanit
 - E. * Dull sound
1432. In the case of negative cough sign will be:
- A. Tumor of hernia
 - B. Inflammation of hernia
 - C. Coprostasis
 - D. Nonreducible hernia
 - E. * Incarceration of hernia
1433. In the case of oblique inguinal hernia lower epigastric vessels are located:
- A. Behind a hernia sack
 - B. Lateral to hernia sack
 - C. Higher hernia sack
 - D. Below hernia sack
 - E. * Medially from a hernia sack
1434. In the case of the incarcerated hernia a main symptom is:
- A. Nausea and vomit
 - B. Bleeding
 - C. Negative cough sign
 - D. High temperature
 - E. * Pain in the hernia swelling

1435. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?
- A. small curvature of stomach;
 - B. back wall of stomach, nearer to small curvature;
 - C. large curvature of stomach
 - D. * cardiac part of stomach;
 - E. pylorus.
1436. In what vein is a venous outflow carried out in from a stomach?
- A. * V. Portae;
 - B. V. odd;
 - C. V. pair;
 - D. V. overhead hollow;
 - E. V. lower hollow;
1437. Incarcerated hernia does not complicated by:
- A. Acute ileus
 - B. Peritonitis
 - C. Necrosis of hernia sack
 - D. Phlegmon of hernia sack
 - E. * Lymphadenitis
1438. Incarcerated hernia must be treated:
- A. Conservative treatment
 - B. Dynamic supervision
 - C. By reducing of hernia
 - D. Planned operation
 - E. * Urgent operation
1439. Large ulcer is an ulcer measuring
- A. 1-4 cm
 - B. 1-3 cm
 - C. 3-5 cm
 - D. 2- 6 cm
 - E. * 2-3 cm
1440. Little ulcer it is an ulcer measuring
- A. * to 0,5 cm
 - B. 0,5-1 cm
 - C. to 1,0 cm
 - D. 3 to 1,5 cm
 - E. 0,5-1,5 cm
1441. Lower epigastric vessels at oblique inguinal hernia is located:
- A. Behind hernia sack
 - B. Laterally to hernia sack
 - C. Higher to hernia sack
 - D. Below to hernia sack
 - E. * Medially to hernia sack
1442. Maydlya hernia is:
- A. Unreducible hernia
 - B. Incarceration of sliding hernia

- C. Incarceration of diverticulum of Meckel
 - D. Wall incarceration
 - E. * Retrograde incarceration
1443. Measure of resection of the incarcerated bowel:
- A. 10-15 cm distally
 - B. 25-30 cm proximally
 - C. Within the limits of visible healthy tissue
 - D. 15-20 cm proximally and 5-10 cm distally
 - E. * 25-30 cm proximally and 10-15 cm distally
1444. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
1445. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood
1446. Melena is a characteristic sign
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorus stenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1447. Method of examination of incarcerated hernia is:
- A. Colonoscopy
 - B. Endoscopy
 - C. CT
 - D. Sonography
 - E. * X-Ray examination
1448. Methods of operative treatment of the left-side oblique reducible inguinal hernia:
- A. Methods of Sapezhko, Meye
 - B. Methods of Abrazhanov, Caravanov
 - C. Methods of Rudzhi, Parlavecho, Praksin
 - D. Methods of Bassini, Kukudzhinov, Postempsky
 - E. * Methods of Martinov, Zhirar-Spasokukocky-Kimbarovsky
1449. Most frequent form of incarceration:
- A. All listed meets rare
 - B. All listed meets often
 - C. Incarceration is mixed
 - D. Excrement incarceration
 - E. * Elastic incarceration
1450. Most informing method at a bleeding ulcer

- A. survey sciagraphy the organs of abdominal region
 - B. * EFGDS
 - C. sciagraphy the stomach with contrasting
 - D. Sonography
 - E. Laparoscopy
1451. Name classic complications of ulcerous illness
- A. bleeding, pylorostenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pylorostenosis, malignization
 - C. malignization, pylorostenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
1452. Negative cough sign means:
- A. Tumor of hernia
 - B. Inflammation of hernia
 - C. Coprostasis
 - D. Nonreducible hernia
 - E. * Incarceration of hernia
1453. Nonreducible hernia arises because of:
- A. Necrosis
 - B. Coprostasis
 - C. Peristalsis
 - D. An acute jamming of hernia content in the hernia gate
 - E. * Adhesion process between hernia content and hernia sack
1454. Objective sign of the incarcerated hernia:
- A. Bleeding
 - B. Positive cough sign
 - C. Vomit
 - D. Pain
 - E. * Negative cough sign
1455. On the method of the plastic by Bassini is performed:
- A. Strengthening of medial wall of inguinal channel
 - B. Suturing of defect of anterior abdominal wall
 - C. Strengthening of lateral wall of inguinal channel
 - D. Strengthening of anterior wall of inguinal channel
 - E. * Creation or strengthening of posterior wall of inguinal channel
1456. Opening, through which goes out direct inguinal hernia:
- A. White line of abdomen
 - B. Femoral channel
 - C. Lateral inguinal fossa
 - D. Left inguinal fossa
 - E. * Medial inguinal fossa
1457. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfannenstiel's access

1458. Operation which narrow inguinal channel without its opening:
- A. Method of Spasokukotsky, Kimbarovsky
 - B. Method of Bassini, Kukudzhanov
 - C. Method of Ru
 - D. Method of Martinov, Zhirar
 - E. * Method of Postempsky
1459. Operation, which is used at right nonreducible femoral hernia:
- A. Methods of Martinov, Postempsky
 - B. Methods of Bassini, Kukudzhanov, Postempsky
 - C. Methods of Martinov, Jirar, Spasokukocky, Kimbarovsky
 - D. Methods of Sapezhko, Meyo
 - E. * Methods of Bassini, Rudzhi, Parlavecho
1460. Operations of narrowing of inguinal channel without it opening is named by:
- A. By Spasokukotsky, Kimbarovsky
 - B. By Bassini, Kukudzhanov
 - C. By Martinov, Zhirar
 - D. By Postempsky
 - E. * By Cherni, Ru
1461. Operations of renewing of inguinal channel is named by:
- A. Method of Lockwood
 - B. Method of Kirschner
 - C. Method of Postempsky
 - D. Method of Cherni, Ru
 - E. * Method of Martinov, Zhirar
1462. Operations with liquidation of inguinal channel is named by:
- A. Method of Lockwood
 - B. Method of Kirschner
 - C. Method of Cherni, Ru
 - D. Method of Martinov, Zhirar
 - E. * Method of Postempsky
1463. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
1464. Principle of Janov method is:
- A. Strengthening of upper wall of inguinal channel
 - B. Strengthening of anterior wall of inguinal channel
 - C. Strengthening of lateral wall of inguinal channel
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Use for hernioplasty patch of skin
1465. Principle of Kirschner method is:
- A. Strengthening of upper wall of inguinal channel
 - B. Strengthening of anterior wall of inguinal channel
 - C. Strengthening of lateral wall of inguinal channel

- D. Strengthening of posterior wall of inguinal channel
 - E. * Used for hernioplasty of wide femoral fascia
1466. Principle of Lexer method at umbilical hernia is:
- A. Vertical cut on middle line
 - B. Removing of the umbilicus
 - C. Horizontal hernioplasty
 - D. Transversal hernioplasty
 - E. * Placing of circle sutures around the edges of defect
1467. Principle of Lockwood method is:
- A. Strengthening of upper wall of inguinal channel
 - B. Strengthening of anterior wall of inguinal channel
 - C. Strengthening of lateral wall of inguinal channel
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Closing of femoral channel by sewing inguinal ligament to the periosteum of pubic bone
1468. Principle of Zhirar method is:
- A. Use of alloplastic material
 - B. Strengthening of lateral wall of inguinal channel
 - C. Narrowing of inguinal channel without its opening
 - D. Strengthening of posterior wall of inguinal channel
 - E. * Strengthening of anterior wall of inguinal channel
1469. Principles of Meyo method:
- A. Strengthening of posterior wall of inguinal channel
 - B. Strengthening of anterior wall of inguinal channel
 - C. Strengthening of lateral wall of inguinal channel
 - D. Formation of double-layer of white line
 - E. * Suturing of defect of anterior abdominal wall in the area of umbilical ring by U-shape sutures
1470. Purpose of the using of Kukudzhanov method:
- A. Closing of hernia gate is from the side of femoral channel
 - B. Strengthening of lateral wall of inguinal channel
 - C. Narrowing of inguinal channel is without its opening
 - D. Strengthening of anterior wall of inguinal channel
 - E. * Strengthening of posterior wall of inguinal channel
1471. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
1472. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year

1473. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
1474. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
1475. Signs of non-complicated hernia are all, except:
- A. Nausea
 - B. Constipation
 - C. Swelling
 - D. Vomit
 - E. * Bleeding from hernia
1476. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
1477. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
1478. Strengthening of posterior wall of inguinal channel performed at the hernioplasty by:
- A. Martinov
 - B. Kimbarovsky
 - C. Rudzhi
 - D. Meyo
 - E. * Bassini
1479. Swelling in the area of lateral fossa is means:
- A. Umbilical hernia
 - B. Epigastroceles
 - C. Direct inguinal hernia
 - D. Femoral hernia
 - E. * Oblique inguinal hernia
1480. Swelling in the area of medial fossa is means:
- A. Umbilical hernia
 - B. Epigastroceles
 - C. Oblique inguinal hernia
 - D. Femoral hernia

- E. * Direct inguinal hernia
1481. Swelling of anterior abdominal wall in the area of lateral fossa could be at:
- A. Direct and oblique inguinal hernia
 - B. Epigastroceles
 - C. Direct inguinal hernia
 - D. Femoral hernia
 - E. * Oblique inguinal hernia
1482. Swelling of anterior abdominal wall in the area of medial fossa could be at:
- A. Direct and oblique inguinal hernia
 - B. Epigastroceles
 - C. Femoral hernia
 - D. Oblique inguinal hernia
 - E. * Direct inguinal hernia
1483. Tactic of doctor, if during hospitalization incarcerated hernia is replaced:
- A. Nothing to do
 - B. Conservative treatment
 - C. To discharge patient
 - D. Urgent surgical treatment
 - E. * Hospitalization, supervision
1484. Tactic of surgeon during replacing of the incarcerated hernia during operation:
- A. Finishing of operation
 - B. Draining of abdominal cavity
 - C. Laparotomy
 - D. Hernioplasty without revision of nearest organs
 - E. * Operative treatment with the revision of nearest organs
1485. The „symptom of flowing” is characteristic for:
- A. Epigastroceles
 - B. Umbilical hernia
 - C. Inguinal hernia
 - D. Femoral hernia
 - E. * Diaphragmatic hernia
1486. The anterior wall of inguinal channel is:
- A. Inguinal ligament
 - B. Lower edge of internal oblique and transversal muscles of abdomen
 - C. Direct muscle of abdomen
 - D. Transversal fascia
 - E. * Aponeurosis of external oblique muscle of abdomen
1487. The best method for differential diagnostics of acute hydroxy of testicle with the incarcerated inguinal hernia is:
- A. Colonoscopy
 - B. Endoscopy
 - C. CT
 - D. Sonography
 - E. * Diaphanoscopy
1488. The effective method of prophylaxis of incarceration of hernia is:
- A. Changing of job

- B. Diet
 - C. Anti-spastic drugs
 - D. Antibiotics
 - E. * The planned operations
1489. The elastic incarceration develops:
- A. Languidly
 - B. With high speed
 - C. Too slowly
 - D. Gradually
 - E. * Suddenly
1490. The excrement incarceration arises up at:
- A. Sneeze
 - B. Cough
 - C. Physical training
 - D. Bleeding
 - E. * Decreasing of peristalsis
1491. The excrement incarceration develops:
- A. Too slowly
 - B. Suddenly
 - C. With high speed
 - D. Languidly
 - E. * Gradually
1492. The external wall of femoral channel is:
- A. Transversal fascia
 - B. Os pubis
 - C. Lakunar ligament
 - D. Inguinal ligament
 - E. * Femoral vein
1493. The internal wall of femoral channel is formed by:
- A. Transversal fascia of abdomen
 - B. By the inguinal ligament
 - C. By a femoral vein
 - D. By the horizontal branch of pubic bone
 - E. * By the edge of Jimbernat ligament
1494. The lower wall of inguinal channel is formed by:
- A. Pubic bone
 - B. Transversal fascia of abdomen
 - C. Aponeurosis of external oblique muscle of abdomen
 - D. Lower edge of internal oblique and transversal muscles of abdomen
 - E. * Inguinal ligament
1495. The method of Kukudzhanov is performed at hernioplasty of:
- A. Umbilical hernia
 - B. Femoral and inguinal hernia
 - C. Epigastroceles
 - D. Femoral hernia
 - E. * Inguinal hernia

1496. The method of Spasokukotsky is performed at hernioplasty of:
- A. Umbilical hernia
 - B. Umbilical hernia
 - C. Femoral hernia
 - D. Direct inguinal hernia
 - E. * Oblique inguinal hernia
1497. The methods of hernioplasty with using of synthetic materials:
- A. Fixation of graft for the edges of defect
 - B. Fixation of graft between peritoneum and aponeurosis layer (inlay technic)
 - C. Fixation of graft under autoplasty (sublay technic)
 - D. Fixation of graft above autoplasty (onlay technic)
 - E. * All listed methods
1498. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
1499. The objective sign of hernia is:
- A. Vomit
 - B. Nausea
 - C. Constipation
 - D. Pain
 - E. * Swelling
1500. The posterior wall of femoral channel is:
- A. Transversal fascia of abdomen
 - B. Edge of Jimbernat ligament
 - C. Femoral vein
 - D. Inguinal ligament
 - E. * Horizontal branch of pubic bone
1501. The posterior wall of inguinal channel is:
- A. Pubic bone
 - B. Inguinal ligament
 - C. Lower edge of internal oblique and transversal muscles of abdomen
 - D. Aponeurosis of external oblique muscle of abdomen
 - E. * Transversal fascia of abdomen
1502. The resection of the loop of incarcerated bowel in hernia gate is indicated:
- A. At presence of all of the listed signs
 - B. In default of pulsation of mesentery vessels
 - C. In default of discoloration
 - D. In default of peristalsis
 - E. * At presence of any of the listed signs
1503. The retrograde incarceration arises up at presence of:
- A. Adhesion in a hernia sack
 - B. Wrong form of hernia gate
 - C. Narrow hernia gate
 - D. Negative pressure in a hernia sack

- E. * Wide hernia gate
1504. The retrograde incarceration looks like :
- A. Letters of F
 - B. Letters of R
 - C. Letters of B
 - D. Letters of G
 - E. * Letters of W
1505. The Richter incarceration arises up, when in a hernia sack located:
- A. Part of urinary bladder
 - B. Loop of bowel
 - C. Lateral wall of bowel
 - D. Part of mesentery
 - E. * Part of wall of bowel
1506. The Rudzhi method is use for:
- A. Oblique and direct inguinal hernia
 - B. Direct inguinal hernia
 - C. Acquired oblique inguinal hernia
 - D. Congenital oblique inguinal hernia
 - E. * Femoral hernia
1507. The second stage of operation at incarcerated hernia:
- A. Sequence of manipulations not important
 - B. Section of hernia sack
 - C. Fixing of jammed organ
 - D. Puncture of hernia sack
 - E. * Scission of incarceration ring
1508. The secretory function stomach is carried out the next membrane of stomach
- A. * mucous membrane
 - B. internal muscular layer
 - C. serosal
 - D. mucous submembrane
 - E. external layer
1509. The signs of non-complicated hernia are all, except of:
- A. Nausea
 - B. Constipation
 - C. Swelling
 - D. Vomit
 - E. * Bleeding from hernia
1510. The sizes of hernia gate at small hernia:
- A. Over 20 cm
 - B. Over 15 cm
 - C. To 15 cm
 - D. To 8 cm
 - E. * To 2 cm
1511. The upper wall of inguinal channel is:
- A. Pubic bone
 - B. Transversal fascia of abdomen

- C. Inguinal ligament
 - D. Aponeurosis of external oblique muscle of abdomen
 - E. * Lower edge of internal oblique and transversal muscles of abdomen
1512. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scarry-ulcerous stenosis
 - B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
1513. To the gastric – intestinal bleeding of un ulcerous etiology belong
- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.
1514. Udin's sing at a perforated ulcer is
- A. * feeling at palpation shove the gases which penetrate through the perforated opening
 - B. dulling perforated sound in the lateral departments of stomach
 - C. disappearance of hepatic dullness
 - D. irradiation pain in a shoulder or shoulder-blade
 - E. sickliness the back vault of vagina
1515. Vomiting coffee-grounds is a characteristic sign
- A. * bleeding ulcer
 - B. penetrative ulcers
 - C. perforated ulcers
 - D. malignizated ulcers
 - E. for cicatrical-ulcerous pylorus stenosis
1516. Vomiting what arose up in 4-6 hours after eating characteristic for
- A. chronic alcoholic gastritis
 - B. cancer and ulcers of cardia
 - C. * pylorus ulcers
 - D. achalasia of gullet
 - E. ulcer and cancer the body of stomach
1517. Wall of inguinal channel, which is strengthened at the plastic by Bassini:
- A. Anterior and posterior
 - B. Lower
 - C. Upper
 - D. Anterior
 - E. * Posterior
1518. What additional symptom, which will arise up at incarceration of sliding inguinal hernia, the wall of which is an urinary bladder:
- A. Absence of other symptoms
 - B. Bleeding
 - C. Symptom of sexual weakness
 - D. Intoxication symptom
 - E. * Disuria

1519. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodenum?
- A. * Tarry excrement
 - B. Presence on the formed excrement of strokes of red blood
 - C. Littlechanged blood in an excrement
 - D. Excrement of the raspberry colouring with the admixtures of mucus
 - E. Acholic excrement
1520. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- A. perforation
 - B. perforation + bleeding
 - C. pylorus stenosis
 - D. malignization + penetration
 - E. * bleeding
1521. What complications can be at incarceration of omentum:
- A. Peritonitis
 - B. Necrosis of hernia sack
 - C. Intestinal obstruction
 - D. Phlegmon of hernia sack
 - E. * A thrombosis of vessels of omentum and embolism of portal vein
1522. What diameter of hernia gate at large hernia:
- A. Over 8 cm
 - B. Over 14 cm
 - C. Over 12 cm
 - D. Over 10 cm
 - E. * Over 4 cm
1523. What diameter of hernia gate at middle hernia:
- A. Over 8 cm
 - B. Over 14 cm
 - C. Over 12 cm
 - D. Over 10 cm
 - E. * Up to 4 cm
1524. What does form the anterior wall of femoral channel?
- A. Transversal fascia
 - B. Pubic bone
 - C. Lakunar ligament
 - D. Femoral vein
 - E. * Inguinal ligament
1525. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
1526. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol

- B. Liciviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
1527. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy
 - E. the all transferred does not belong
1528. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
1529. What hernia has mesoperitoneal organ as part of hernia sack wall:
- A. Umbilical hernia
 - B. Oblique inguinal hernia
 - C. Femoral hernia
 - D. Direct inguinal hernia
 - E. * Sliding hernia
1530. What hernia is operated by Sapezhko method?
- A. Direct inguinal hernia
 - B. Only at hernia of white line
 - C. Only umbilical hernia
 - D. Only femoral hernia
 - E. * Umbilical and white line
1531. What hernia tissue can be the tumors origin?
- A. Never arise
 - B. Hernia sack
 - C. Membrane of hernia
 - D. Hernia content
 - E. * All listed elements of hernia
1532. What hernioplasty is indicated in case of phlegmon of hernia sack:
- A. Meyo hernioplasty
 - B. Bassini hernioplasty
 - C. Hernioplasty with draining of wound
 - D. Performed by any methods
 - E. * Contra-indicated
1533. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E
 - D. estrogens
 - E. STH

1534. What is contra-indicated for patients with coprostitis?
A. Enemas
B. Antispastic
C. Anaesthetic
D. Easy local massage
E. * Purgative drugs
1535. What is external hernia of abdomen:
A. An output of organs through the damaged abdominal wall
B. Jamming of organs
C. Inflammations of organs
D. An output of organs outside of peritoneum
E. * Output of organs, covered by peritoneum under a skin
1536. What is incarcerated hernia:
A. Jamming of content of hernia is from the side of abdominal cavity
B. Inflammation of hernia
C. Jamming of content of hernia by the wall of hernia sack
D. Jamming of content of hernia by surrounding tissue
E. * A jamming of content of hernia in its gate
1537. What is initial femoral hernia?
A. Swelling goes out outside of internal femoral ring
B. Swelling goes out outside superficial fascia
C. Swelling passes all anatomic structure of femoral channel
D. Swelling does not go outside superficial fascia
E. * Swelling does not go outside of internal femoral ring
1538. What is internal hernia of abdomen:
A. An output of organs through the damaged abdominal wall
B. Jamming of organs
C. Inflammations of organs
D. An output of organs outside of peritoneum
E. * The output of organs through the natural folds of peritoneum within the limits of abdominal cavity
1539. What is Lihtenshtein method?
A. Hernioplasty with painter net
B. Hernioplasty with metallic net
C. Hernioplasty with arachnoidite net
D. Hernioplasty with polyethylene net
E. * Hernioplasty with mesh material "Ethicon"
1540. What is Littre hernia ?
A. Nonreducible hernia
B. Incarceration of sliding hernia
C. Antegrade incarceration
D. Retrograde incarceration
E. * Incarceration of Mekkel diverticul
1541. What is more frequent forms wall at sliding left-side inguinal hernia?
A. Stomach
B. Transverse colon

- C. Urinary bladder
 - D. Small intestine
 - E. * Sigmoid colon
1542. What is more frequent forms wall at sliding right-side inguinal hernia?:
- A. Stomach
 - B. Transverse colon
 - C. Urinary bladder
 - D. Small intestine
 - E. * Urinary bladder
1543. What is not characteristic for determination of viability of incarcerated bowel:
- A. Presence of peristalsis
 - B. Presence of pulsation of mesentery vessels
 - C. Absence of strangulation furrow
 - D. Renewing of the natural color of organ
 - E. * Presence of strangulation furrow
1544. What is orifice of hernia sack?
- A. Its widest part
 - B. Its upper part
 - C. Its lower part
 - D. Distal part of hernia sack
 - E. * Part of hernia sack, which connected with abdominal cavity
1545. What is principle of Lexer method at umbilical hernia?
- A. Vertical cut on middle line
 - B. Removing of the umbilicus
 - C. Horizontal hernioplasty
 - D. Transversal hernioplasty
 - E. * Placing of circle sutures around the edges of defect
1546. What is Richter's hernia?
- A. Nonreducible hernia
 - B. Incarceration of sliding hernia
 - C. Incarceration of diverticulum of Meckel
 - D. Retrograde incarceration
 - E. * A hernia in which only a portion of the wall of the intestine is involved.
1547. What is the condition of expression and character of development of pathological changes at incarceration hernia?:
- A. Degree of jamming of organ by the incarcerated ring
 - B. Structure of jammed organ
 - C. State of vessels
 - D. Duration of incarceration
 - E. * All listed signs
1548. What is the first sign of the false incarceration?
- A. Intestinal obstruction
 - B. Peritonitis
 - C. Intoxication syndrome
 - D. Pain in the hernia
 - E. * Pain in the abdominal cavity

1549. What is the first step of operation at the incarcerated hernia?
- A. Estimation of viability of jammed organ
 - B. Conservative treatment
 - C. Dissecting of jamming ring
 - D. Puncture of hernia sack.
 - E. * Quick opening of hernia sack and fixing of jammed organ.
1550. What is the internal wall of femoral channel?:
- A. Transversal fascia of abdomen
 - B. Inguinal ligament
 - C. Femoral vein
 - D. Horizontal branch of pubic bone
 - E. * Edge of Jimbernat ligament
1551. What is the most frequent complication of incarcerated hernia:
- A. Colitis
 - B. Tumor of colon
 - C. Pancreatitis
 - D. Peptic ulcer
 - E. * Necrosis of hernia sack
1552. What is the principle of inlay technic of the hernioplasty of postoperative hernia:
- A. All are transferred methods
 - B. Fixation of graft for the edges of defect
 - C. Fixation of graft above autoplasty
 - D. Fixation of graft under autoplasty
 - E. * Fixation of graft between peritoneum and aponeurosis layer
1553. What is the principle of onlay technic of the hernioplasty of postoperative hernia:
- A. All are transferred methods
 - B. Fixation of graft for the edges of defect
 - C. Fixation of graft between peritoneum and aponeurosis layer
 - D. Fixation of graft under autoplasty
 - E. * Fixation of graft above autoplasty
1554. What is the principle of sublay technic of the hernioplasty of postoperative hernia:
- A. All are transferred methods
 - B. Fixation of graft for the edges of defect
 - C. Fixation of graft between peritoneum and aponeurosis layer
 - D. Fixation of graft above autoplasty
 - E. * Fixation of graft under autoplasty
1555. What is the superior wall of inguinal channel?
- A. Pubic bone
 - B. Transversal fascia of abdomen
 - C. Inguinal ligament
 - D. Aponeurosis of external oblique muscle of abdomen
 - E. * Lower edge of internal oblique and transversal muscles of abdomen
1556. What is the time of disability after treatment of incarcerated hernia?:
- A. 14 days
 - B. 1 week
 - C. 4-6 days
 - D. 12 weeks

- E. * 4-6 weeks
1557. What localization of ulcer is most characteristic for the patients of elderly and senile age
- * cardial department of stomach
 - overhead third of gullet
 - lower third of gullet
 - bulb of duodenum
 - small curvature
1558. What method diagnostics the ulcerous illness most informing
- * esophagogastroduodenoscopy
 - analysis of excrement on the hidden blood
 - X-ray
 - global analysis of blood
 - research of gastric secretion
1559. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - On blood volume
 - * On an arteriotomy, pulse, state of patient
 - On a globular volume
 - On the level of thrombocytes
1560. What method is used for closing of hernia gate from the side of femoral channel:
- Method of Spasokukotsky
 - Method of Meyo
 - Method Caravanov
 - Methods of Lockwood and Abrazhanov
 - * Method of Rudzhi
1561. What methods of hernioplasty at postoperative hernia?:
- Aponeurotic hernioplasty
 - Alloplasty by a tantal mesh
 - Alloplasty by lavsan suture
 - Muscle-aponeurotic
 - * All listed
1562. What name of method of closing of femoral channel by sewing of inguinal ligament to the periosteum of pubic bone?
- Method Caravanov
 - Method of Spasokukotsky
 - Method of Meyo
 - Method of Rudzhi
 - * Method of Lockwood
1563. What name of the symptoms of the incarcerated hernia:
- Nausea and vomit
 - Pain in the hernia swelling
 - Negative cough sign
 - Nonreducible swelling
 - * All are transferred signs
1564. What preparation does behave to blocker H2-retseptors?

- A. * tavegil
 - B. obzidan
 - C. hystdol
 - D. cerucal
 - E. oraza
1565. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
1566. What preparations, except for other properties, own yet and a bacteriostatic effect on *Helicobacter pylori*
- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
1567. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
1568. What signs of viability of the jammed loop of bowel is used?:
- A. By a tint
 - B. By a color
 - C. By the presence of pulsation of vessels
 - D. By the presence of peristalsis
 - E. * Used all signs
1569. What stages has by medial vascular-lacunar femoral hernia?
- A. Incomplete, complete
 - B. Initial, complete
 - C. Incomplete, complete, eventual
 - D. First, second, third
 - E. * Initial, incomplete, complete
1570. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
1571. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;

- E. 6 – 20 hours
1572. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia
1573. What wall of inguinal channel is strengthened by Bassini hernioplasty:
- A. Anterior and posterior
 - B. Inferior
 - C. Superior
 - D. Anterior
 - E. * Posterior
1574. What wall of inguinal channel is strengthened at the plastic by Kukudzhanov:
- A. Anterior and posterior
 - B. Inferior
 - C. Superior
 - D. Anterior
 - E. * Posterior
1575. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforated ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pylorostenosis
1576. When could be false incarceration of hernia?
- A. At incarceration of diverticul of Meckel
 - B. At inflammation of hernia
 - C. At jamming of the organ in a hernia sack
 - D. At the partial incarceration of wall of organ
 - E. * At the acute diseases of organs of abdominal cavity
1577. When lower epigastric vessels is located laterally to hernia sack?
- A. Epigastroceles
 - B. Umbilical hernia
 - C. Oblique inguinal hernia
 - D. Femoral hernia
 - E. * Direct inguinal hernia
1578. When lower epigastric vessels is located medially to hernia sack?
- A. Epigastroceles
 - B. Umbilical hernia
 - C. Direct inguinal hernia
 - D. Femoral hernia
 - E. * Oblique inguinal hernia
1579. With the purpose of strengthening of anterior wall of inguinal channel it is performed:
- A. Method of Kirschner

- B. Method of Postempsky
 - C. Method of Bassini, Kukudzhanov
 - D. Method of Ru
 - E. * Methods of Martinov, Zhirar, Spasokukotsky
1580. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appeared. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
1581. A patient, 32 years, 4 hours ago has "knife-like" abdominal pain. Diagnosis?
- A. Acute appendicitis.
 - B. Acute ileus.
 - C. Acute pancreatitis.
 - D. Gangrenous cholecystitis.
 - E. * All of answers are incorrect.
1582. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
1583. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
1584. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I
 - E. saved after all transferred operations
1585. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred
 - E. not saved after all operations
1586. Among the symptoms of perforative peptic ulcer one is indicated wrong:
- A. Positive symptom of Schetkin-Blumberg.
 - B. Disappearance of hepatic sound.
 - C. There is a knife-like pain.

- D. Tension of muscles of anterior abdominal wall.
 - E. * Vomiting is not facilitate.
1587. An absolute indication to the operation at peptic ulcerous disease are:
- A. Persistent duodeno-gastric reflux with gastritis and peptic ulcer
 - B. Presence of genetic predisposition to peptic ulcerous disease
 - C. Combination of gigant gastric and duodenal peptic ulcers
 - D. Large peptic ulcer of pylorus with possible development of stenosis
 - E. * Penetrative peptic ulcer with formation of pathological fistula
1588. An intraduodenal brake effect of acid secretion is realized through:
- A. Duodeno-gastric reflux
 - B. Decreasing of pancreatic secretion
 - C. Increasing of pancreatic secretion
 - D. Increasing of bile production
 - E. * Increasing of secretine production
1589. An operation at a duodenal peptic ulcer is not performed at:
- A. Development of malignancy
 - B. Development of perforation
 - C. Development of stenosis
 - D. Massive bleeding
 - E. * Formation of peptic ulcer after anti-inflammation non-steroid drug
1590. Appearance of disgust for meat is the most characteristic complaint of patient with:
- A. Peptic peptic ulcer
 - B. Pancreatitis
 - C. peptic ulcerous disease of stomach
 - D. Uremic gastritis
 - E. * Cancer of stomach
1591. At a large gastric peptic ulcer the best method of treatment is:
- A. Selective proximal vagotomy.
 - B. Gastrectomy.
 - C. Selective vagotomy and pyloroplasty by Finney.
 - D. Trunk vagotomy with excision of peptic ulcer.
 - E. * Resection of stomach.
1592. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
1593. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
1594. At determination of indications to surgical treatment of peptic ulcerous disease not important:

- A. Duration of disease
 - B. Duration of remission
 - C. Efficiency of conservative therapy
 - D. Frequency of relapses
 - E. * Expressed of inflammation in region of pylorus and duodenum
1595. At determination of indications to the operation of peptic ulcerous disease there it is important:
- A. Sizes of antral part of stomach
 - B. Secretion
 - C. Evacuation function of stomach and duodenum
 - D. X-Ray examination of stomach and duodenum
 - E. * Information of endoscopic examination of peptic ulcer and its localization
1596. At determination of indications to the operation of peptic ulcerous disease there it is not important:
- A. Duodeno-gastric reflux
 - B. Gastric secretion
 - C. Pathogenesis of disease
 - D. Complications of peptic ulcerous disease
 - E. * Predisposition to dumping-syndrome
1597. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
1598. At often relapse of peptic ulcers of duodenum it is possible to suspect:
- A. Bleeding
 - B. Perforation
 - C. Penetration
 - D. Tumor
 - E. * Syndrome of Zollinger-Ellison.
1599. At patient 40 years old 5 hours ago the perforation of gastric peptic ulcer is happened. What is the best surgical treatment:
- A. Gastrectomy.
 - B. Vagotomy and pyloroplasty.
 - C. Vagotomy and sewing of peptic ulcer.
 - D. Antrumectomy.
 - E. * Classic resection 2/3 stomach.
1600. At patients with peptic ulcerous disease the risk of the gastro-duodenal bleeding is most high at:
- A. All of cases
 - B. Malignancy
 - C. Perforations of peptic ulcer
 - D. Stenosis of pylorus
 - E. * Penetration of peptic ulcer to the omentum
1601. At peptic ulcerous disease of stomach more frequent in all:
- A. Gastritis is expressed less than at a duodenal peptic ulcer.

- B. Malignity more rare, than at a duodenal peptic ulcer.
 - C. low pH in antral part
 - D. The motor function of stomach is enhanceable in an interdigestive period
 - E. * Surgical treatment is used considerably more frequent, than at a duodenal peptic ulcer.
1602. At peptic ulcerous disease:
- A. All of answers are faithful
 - B. Increased motility of duodenum
 - C. Considerably more frequent, than at a gastric peptic ulcer, surgical treatment is used
 - D. Decreased motility of duodenum
 - E. * Considerably more frequent, than at a gastric peptic ulcer, conservative treatment is used
1603. At perforative gastric peptic ulcer method of operation is determined from:
- A. Age of patient
 - B. Time from the moment of perforation
 - C. Degree of peritonitis
 - D. Localization of the perforation
 - E. * All of listed
1604. At suspicion of duodenal peptic ulcer is performed:
- A. Examination of gastric secretion
 - B. X-Ray of organs of abdominal cavity
 - C. Cholecystography
 - D. Determination of level of gastrin in blood
 - E. * Esophagogastroduodenoscopy
1605. At suspicion on the perforative peptic ulcer of stomach the first examination must be:
- A. Laparoscopy
 - B. Angiography
 - C. Urgent esophagogastroduodenoscopy
 - D. X-Ray of stomach with barium
 - E. * X-Ray of abdominal region
1606. At the duodenum ulceroperation of choice is
- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
1607. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
1608. At the III item of activity hemostasis and III sage blood loss from the I – III sage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);

- D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1609. At the relapse of the peptic ulcerous gastro-duodenal bleeding is performed:
- A. Conservative therapy
 - B. Colonoscopy
 - C. Sonography
 - D. Planned surgical treatment
 - E. * Urgent operation
1610. At the threat of relapse of the peptic ulcerous gastro-duodenal bleeding is recommended:
- A. Conservative therapy
 - B. Colonoscopy
 - C. Sonography
 - D. Planned surgical treatment
 - E. * Urgent operation
1611. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1612. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
1613. At which disease will be tension of muscles of anterior abdominal wall?
- A. At pylorostenosis
 - B. At hernia
 - C. At appendicitis
 - D. At chronic gastritis
 - E. * Right answer not present
1614. Belching by bright red blood which increased at a cough is characteristic for:
- A. Syndrome of Randyu - Osler
 - B. Bleeding gastric peptic ulcer
 - C. Syndrome of Mellori - Weiss
 - D. Tumor of cardiac part
 - E. * Pulmonary bleeding
1615. Bergman's sign is characteristic for
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1616. Berhstein's sign characteristic for

- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
1617. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
1618. Blood loss I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
1619. Blood loss II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
1620. Blood loss III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
1621. By the most credible reason of development peptic ulcer by decreasing of resistance of mucous tunic of stomach is:
- A. Hormonal changes in organism
 - B. Deficit of plastic and biochemically active substances
 - C. Local ischemia of gastro-duodenal mucous membrane
 - D. Metabolic changes in organism
 - E. * Chronic gastritis
1622. Choice of method of operative treatment at the perforative peptic ulcer complicated by peritonitis?
- A. Selective proximal vagotomy
 - B. Pyloroplasty with vagotomy
 - C. Antrumectomy with vagotomy
 - D. Subtotal resection of stomach
 - E. * Sewing of the perforative opening
1623. Clinical sign of perforative peptic ulcer in the first 6 hours of disease is not characterized by:
- A. By absence of vomiting.
 - B. By disappearance of hepatic sound.

- C. By a "knife-like" abdominal pain.
 - D. Wooden belly.
 - E. * Diarrhea.
1624. Conservative therapy at perforative peptic ulcer is prescribed only at:
- A. Combination of peptic ulcerous disease of stomach and duodenum
 - B. High degree of operating risk
 - C. Absence in patient peptic ulcerous anamnesis
 - D. Old age of patients
 - E. * Absence of possibility of urgent operation
1625. De-Cerven's sign is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
1626. Decreasing of gastric acid secretion comes at duodenal pH:
- A. 6,0
 - B. 3,0
 - C. 4,0
 - D. 5,0
 - E. * 2,5 and below
1627. Decreasing of pain and appearance of "melena" at duodenal peptic ulcer is characteristic for:
- A. Penetration to pancreas
 - B. Pyloroduodenal stenosis
 - C. Malignancy
 - D. Perforations of peptic ulcer
 - E. * Bleeding
1628. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
1629. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
1630. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours

1631. During transformation of gastric peptic ulcer to cancer will be such clinical signs: 1. Decreasing of pain. 2. Appearance of anemia. 3. Change of appetite. 4. Absent of "niche" at X-Ray examination of stomach. Choose correct combination of answers:
- 1 and 3.
 - Only 4.
 - 2,3.
 - 1,2,3.
 - * 1,2,3,4.
1632. Eleker's sign is characteristic for
- * perforated ulcers
 - bleeding ulcer
 - penetrated ulcers
 - malignized ulcers
 - for cicatricial-ulcerous pylorostenosis
1633. Endoscopic examination does not diagnose:
- Stenosis of pylorus
 - Type of gastritis
 - Cancer of stomach
 - Syndrome of Mellori - Weiss
 - * Syndrome of Zollinger - Ellison
1634. Endoscopy does not allow:
- To perform pH- metry
 - To inform about cardiac sphincter and pylorus
 - To inform about mucous membrane of gullet, stomach and duodenum
 - To inform about peptic ulcerous defect and define its localization
 - * To define the degree of duodenogastric reflux
1635. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- tumours
 - ulcers
 - bleeding polypuses
 - erosions
 - * changes of evacuation function
1636. Etiopatogenetic method of operation at the uncomplicated duodenal peptic ulcer is:
- Selective vagotomy
 - trunk vagotomy
 - combination of antrumectomy with trunk vagotomy
 - ideal antrumectomy with trunk vagotomy
 - * SPV (selective proximal vagotomy)
1637. For bleeding ulcer characteristic sign is
- * pain in an epigastrium;
 - knife-like pain;
 - signs irritation of peritoneum;
 - presence fresh blood in incandescence
 - melena;
1638. For bleeding ulcer characteristically
- * melena
 - tension the muscles of front abdominal wall

- C. Spazarskiy's sign
 - D. sickliness the back vault of vagina
 - E. irradiation pain in a shoulder or shoulder-blade
1639. For chronic gastro-duodenal peptic ulcers is not characteristic:
- A. Possibility of develop different complications
 - B. Dense edges
 - C. Different sizes of peptic ulcerous defect
 - D. Penetration
 - E. * Absence of convergence of folds of mucous membrane
1640. For decompensated stenosis of pylorus is characteristic: 1. Vomiting by food which eaten the day before. 2. Tension of muscles of abdominal wall. 3. Decrease of diuresis. 4. "Splash sound" in abdomen on an empty stomach. 5. Delay of barium in a stomach more than 24 hours. Choose correct combination of answers:
- A. 1,2.
 - B. 1,2,3,4.
 - C. 1,2,4,5.
 - D. 1,2,4.
 - E. * 1,3,4,5.
1641. For decompensated stenosis of pylorus is not characteristic:
- A. Delay of barium in a stomach more than 24 hours on X-Ray
 - B. Vomiting by food, eaten a day before
 - C. Oliguria
 - D. Splash sound" in a abdomen on an empty stomach
 - E. * Tension of muscles of abdominal wall
1642. For decompensated stenosis of pylorus is not characteristic:
- A. Delay of barium in stomach for more than 24 hours
 - B. Splash sound in empty stomach
 - C. Anuria
 - D. Vomiting by food, eaten the day before
 - E. * Tension of muscles of abdominal wall
1643. For motion of disease ulcerous illness of middle weight characteristically
- A. development of complications
 - B. * relapses 1-2 times per a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 3 and anymore relapses on a year
1644. For perforative gastric peptic ulcer in the first 6 hours is not characteristic:
- A. Gas under the diaphragm
 - B. Acute stomach
 - C. Absent of hepatic sound
 - D. Wooden belly
 - E. * Swelling of stomach
1645. For perforative gastro-duodenal peptic ulcer is characteristic:
- A. General weakness
 - B. Vomiting
 - C. Spastic pain
 - D. Gradual growth of pain syndrome

- E. * Acute pain in epigastrium
1646. For perforated ulcer characteristically
- A. * tension the muscles of front abdominal wall
 - B. melena
 - C. vomiting by coffee-grounds
 - D. high intestinal impassability
 - E. vomiting stagnant gastric maintenance
1647. For pneumoperitoneum is characteristic symptom
- A. * Zhober's;
 - B. Khelatid's;
 - C. Podlag's;
 - D. Vigats's;
 - E. Udin's.
1648. For successful surgical treatment of duodenal peptic ulcer is necessarily:
- A. Sonography
 - B. Angiography
 - C. Examination of liver
 - D. Colonoscopy
 - E. * Estimation of the state of pylorus
1649. For successful surgical treatment of duodenal peptic ulcer not necessarily:
- A. Examination of duodenal permeability
 - B. Determination of gastrin production
 - C. Estimation of the state of pylorus
 - D. Examination of gastric secretion
 - E. * Determination of etiology of peptic ulcer
1650. For the bleeding peptic ulcer not characteristic:
- A. Decreasing of blood volume
 - B. Melena
 - C. Decreasing of hemoglobin
 - D. Vomiting by color of coffee-grounds
 - E. * Increasing of pain in stomach
1651. For the bleeding peptic ulcer the followings signs are characteristic: 1. There is increasing of pain syndrome. 2. Vomiting by "coffee-grounds". 3. There is decreasing pain syndrome. 4. Bradicardia. 5. Melena. Choose correct combination of answers:
- A. 1,3,5.
 - B. 3,4,5.
 - C. 2,3,4.
 - D. 1,2,5.
 - E. * 2,3,5.
1652. For the heavy flow of ulcerous illness characteristically
- A. 2 and anymore relapses on a year
 - B. * 3 and anymore relapses on a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 6 and more relapses are on a year
1653. For the heavy flow of ulcerous illness characteristically

- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days
1654. For what complications of peptic ulcer is characteristic convergences of folds of mucous membrane:
- A. Bleeding
 - B. Stenosis
 - C. Perforation
 - D. Penetration
 - E. * Malignancy
1655. From what department digestive tract development more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial part;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
1656. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
1657. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
1658. How to explain the reason of appearance of tension of muscles in the right iliac region in patients with perforative peptic ulcer:
- A. Viscero-visceral reflex.
 - B. Developing of peritonitis.
 - C. Reflex through the spinal nerves.
 - D. An accumulation of air in abdominal region.
 - E. * Flowing of gastric content to the right lateral channel
1659. How to find the source of the gastro-duodenal bleeding:
- A. Sonography
 - B. X-Ray examination of stomach
 - C. Nasogastric probe
 - D. Laparoscopy
 - E. * Endoscopy
1660. If patient with perforative gastric peptic ulcer is refuse from the operation, it is prescribed:
1. Cleaning the stomach by cold water.
 2. Long-term naso-gastric aspiration.
 3. Stimulation of

intestine. 4. Antibiotic therapy. 5. Position of Trendelenburg. Choose correct combination of answers:

- A. 2,3,5.
- B. 1,2,5.
- C. 1,4.
- D. 2,3,4,5.
- E. * 2,4.

1661. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?

- A. small curvature of stomach;
- B. back wall of stomach, nearer to small curvature;
- C. large curvature of stomach
- D. * cardiac part of stomach;
- E. pylorus.

1662. In what vein is a venous outflow carried out in from a stomach?

- A. * V. Portae;
- B. V. odd;
- C. V. pair;
- D. V. overhead hollow;
- E. V. lower hollow;

1663. Indications to surgical treatment of the uncomplicated peptic ulcerous disease are: 1. Low localization of peptic ulcer. 2. Long time of peptic ulcerous anamnesis with the frequent relapse. 3. Young age of patient. 4. Ineffective conservative treatment more than 3 months. Choose correct combination of answer.

- A. 1,2,3,4.
- B. 1,4.
- C. 2,3,4.
- D. 1,3.
- E. * 2,4.

1664. Isolated selective proximal vagotomy is performed at:

- A. Gastro-duodenal bleeding.
- B. Malignancy.
- C. Perforative peptic ulcer
- D. Duodenal peptic ulcer with subcompensated stenosis of pylorus.
- E. * Duodenal chronic peptic ulcer without the stenosis.

1665. Large ulcer is an ulcer measuring

- A. 1-4 cm
- B. 1-3 cm
- C. 3-5 cm
- D. 2- 6 cm
- E. * 2-3 cm

1666. Little ulcer it is an ulcer measuring

- A. * to 0,5 cm
- B. 0,5-1 cm
- C. to 1,0 cm
- D. 3 to 1,5 cm
- E. 0,5-1,5 cm

1667. Maximal Histamin test at peptic ulcerous disease is used for:
- A. Estimations of efficiency of H-2 blockers.
 - B. Determinations of base acid production.
 - C. Determinations of function of antrum part of stomach.
 - D. Examinations of motility of stomach.
 - E. * Examinations of humoral phase of gastric secretion.
1668. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
1669. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood
1670. Melena is a characteristic sign
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorus stenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1671. Most informing method at a bleeding ulcer
- A. survey sciagraphy the organs of abdominal region
 - B. * EFGDS
 - C. sciagraphy the stomach with contrasting
 - D. Sonography
 - E. Laparoskopy
1672. Most physiological method of resection of stomach:
- A. Resection by Raykhel-Polia
 - B. Bilioth-II in modification by Hakker-Balfur
 - C. Bilioth-II in modification by Hofmeister-Finsterer
 - D. Resection in modification by Ru
 - E. * Bilrot-I
1673. Name class complications of ulcerous illness
- A. bleeding, pyloristhenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pyloristhenosis, malignization
 - C. malignization, pyloristhenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
1674. Name the most characteristic symptom of chronic gastric peptic ulcer:
- A. Weakness
 - B. Pain in 1,5-2 hours after food intake
 - C. Heartburn
 - D. Vomiting

- E. * Pain in 30 minutes after food intake
1675. Name the most characteristic symptom of chronic peptic ulcer:
- A. Vomiting
 - B. Pain in 2,5-3 hours after food intake
 - C. Pain in 30 minutes after food intake
 - D. Nightly pain
 - E. * Heartburn, pain in 1,5-2 hours after food intake
1676. Name the most informative method of examination at dumping-syndrome:
- A. Colonoscopy
 - B. Examination of volume of circulatory blood
 - C. Endoscopy
 - D. CT
 - E. * X-Ray with barium
1677. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
1678. Operation of choice at peptic ulcerous disease with violation of duodenal passage is:
- A. SPV without the special correction of the duodenal passage
 - B. SPV with duodeno-jejuno anastomosis
 - C. Resection of stomach (antrumectomy) with vagotomy by Gofmeyster-Finsterer
 - D. Resection of stomach (antrumectomy) with vagotomy by Bilroth-1
 - E. * Resection of stomach (antrumectomy) with vagotomy by Ru
1679. Operative treatment of patient with a duodenal peptic ulcer is performed in cases of: 1. There are often relapses of disease, 2. A disease is complicated by bleeding, 3. A disease is complicated by stenosis 4. A disease is complicated by perforation of peptic ulcer 5. A disease is complicated by penetration. Choose correct combination of answers:
- A. 1,2
 - B. 3,4
 - C. 2,3
 - D. 1,4
 - E. * 1,2,3,4,5.
1680. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
1681. Patients with the compensated pyloroduodenal stenosis without the signs of active peptic ulcer:
- A. Operated after 2-monthly course of intensive antipeptic ulcer therapy
 - B. Does not need surgical treatment
 - C. Operated only in the case of progress of stenosis
 - D. Need surgical treatment in the case of intensifying of peptic ulcerous disease
 - E. * Need obligatory surgical treatment

1682. Penetrative gastric and duodenal peptic ulcer can result of: 1. abscess of abdominal region 2. pylephlebitis, 3. fistule 4. acute pancreatitis 5. bleeding. Choose correct combination of answers:
- A. 1,2,3,4,5
 - B. 1,3,5.
 - C. 1,2,3
 - D. 2,3,4.
 - E. * 3,4,5.
1683. Planning of operation on duodenal peptic ulcer, there it is not important:
- A. Examination of function of duodenum
 - B. X-Ray of stomach
 - C. Endoscopic examination of lung
 - D. Examine of gastric secretion
 - E. * Examination of secretion of pancreas
1684. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
1685. Rare complication of peptic ulcer is:
- A. Cicatricial deformation of bowel
 - B. Penetration
 - C. Bleeding
 - D. Perforation
 - E. * Malignancy
1686. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
1687. Relative indications to surgical treatment of peptic ulcerous disease:
- A. Atypical perforation of peptic ulcer
 - B. Malignant regeneration of peptic ulcer
 - C. Stenosis of pylorus
 - D. Relapse of the peptic ulcerous bleeding after endoscopic hemostasis
 - E. * Low bulb peptic ulcers
1688. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
1689. Selective proximal vagotomy belongs to
- A. palliative

- B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
1690. Selective proximal vagotomy is not indicated for:
- A. peptic ulcers with subcompensated stenosis
 - B. Duodenal peptic ulcer, complicated by bleeding
 - C. Uncomplicated duodenal peptic ulcer
 - D. Perforative peptic ulcer
 - E. * Gastric and duodenal peptic ulcer
1691. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
1692. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
1693. Spastic pain in epigastric region could be at
- A. Ascitis
 - B. Acute ileus
 - C. Hypertension
 - D. Chronic bronchitis
 - E. * Peptic peptic ulcer
1694. Specify factors, which determine the choice of method of operation at the perforative peptic ulcer of stomach: 1. Presence of peritonitis. 2. Time from the moment of perforation of peptic ulcer. 3. Qualification of surgeon. 4. General state and age of patient. 5. Sizes of peptic ulcer. Choose correct combination of answers:
- A. All of answers are correct.
 - B. 1,2,3,5.
 - C. 1,2,4.
 - D. 1,3,4,5.
 - E. * 1,2,3,4.
1695. Specify physiopathology changes characteristic for decompensated stenosis of pylorus: 1. Hypervolemia. 2. Anaemia. 3. Metabolic alkalosis. 4. Hypovolemiya. 5. Acidosis. Choose correct combination of answers:
- A. 2,3,4.
 - B. 4,5.
 - C. 1,3.
 - D. 2,4.
 - E. * 3,4.
1696. Specify the most characteristic symptoms of peptic ulcerous stenosis of pylorus:
- A. Weakness

- B. Constipation, vomiting
 - C. Constipation
 - D. Vomiting
 - E. * Filling of weight in the epigastrium
1697. Specify the most informative method of examination at a peptic ulcer:
- A. Colonoscopy
 - B. CT
 - C. Determine the gastric secretion
 - D. Sonography
 - E. * Endoscopy
1698. Syndrome of Mellori-Veys is:
- A. hemorrhagic erosive gastroduodenitis
 - B. Varicosity of cardiac part, complicated by bleeding
 - C. Bleeding from mucous membrane.
 - D. Bleeding peptic ulcer from diverticulum of Meckel
 - E. * Fissure in a cardiac part of stomach with bleeding
1699. Syndrome of Zollinger-Ellison is?
- A. Hepatitis
 - B. Cholecystitis
 - C. Diabetes
 - D. Hyperthyroidism
 - E. * Tumor of pancreas
1700. Tension of muscles at right iliac region at the perforation of duodenal peptic ulcer is explained by:
- A. By viscerovisceral reflex
 - B. By development of peritonitis
 - C. Bleeding
 - D. Entering of air to abdominal cavity
 - E. * Flowing of gastric content to the right lateral channel
1701. The best method of diagnostics of perforative peptic ulcers is:
- A. X-Ray
 - B. Endoscopy
 - C. Laparocentesis
 - D. Sonography
 - E. * Laparoscopy
1702. The best method of examination of gastric secretion is:
- A. by using a cabbage juice
 - B. by using an insulin
 - C. by using a coffee
 - D. by using a food
 - E. * by using Histaminum
1703. The best method of intraoperative control after vagotomy:
- A. MRI
 - B. CT
 - C. Sonography
 - D. X-Ray
 - E. * Intra gastric ??-metry by the special ??-tube

1704. The characteristic clinical signs of cancer of cardiac part of stomach is:
- A. Pain in epigastrium
 - B. Belch
 - C. Weakness
 - D. Sense of weight in epigastrium
 - E. * Dysphagia
1705. The characteristic of X-Ray signs of malignancy of stomach is:
- A. Rigidity of wall of stomach
 - B. Strengthening of peristalsis
 - C. Defect of filling
 - D. Niche"
 - E. * Convergence of folds of mucous membrane of stomach
1706. The clinic of decompensated pyloroduodenal stenosis is characterized:
- A. By a "splash sound" on an empty stomach.
 - B. By the hypovolemia.
 - C. Delay of barium in a stomach to 24 hours.
 - D. By the decrease of mass of body.
 - E. * All of answers are correct.
1707. The compensated stage of piloroduodenal stenosis is characterized by: 1. "Splash sound" on an empty stomach. 2. Vomiting at mornings. 3. By the delay of barium in a stomach more than 12 hours. 4. By the hypovolemia. 5. General weakness. Choose correct combination of answers:
- A. 1,2,3.
 - B. All of answers are correct.
 - C. 1,4,5.
 - D. 1,3,4.
 - E. * All of answers are wrong.
1708. The diet of Meylengraft is based:
- A. All listed is not right
 - B. On mechanical defense of mucous membrane of stomach
 - C. On providing of high-calorie food
 - D. On suppression of secretion of gastric juice
 - E. * All of listed is right
1709. The dumping-syndrome conditioned:
- A. By dilatation of stomach
 - B. By psycho state of patients
 - C. By hormonal changes
 - D. By the result of intestinal hyperosmose
 - E. * By the genetically determined reaction of organism on food products
1710. The gastric secretion in healthy persons:
- A. Brake
 - B. Inert
 - C. Asthenic
 - D. Excitable
 - E. * Normal
1711. The high risk of the gastro-duodenal bleeding has patients with:
- A. In all of cases.

- B. Malignancy.
 - C. Perforation of peptic ulcer.
 - D. Stenosis of pylorus.
 - E. * Penetration of peptic ulcer to the omentum.
1712. The highest level of acidity is observed at peptic ulcer in:
- A. Cardiac part of stomach
 - B. Body of stomach
 - C. Bottom of stomach
 - D. Antral part
 - E. * Pyloric part
1713. The initial process of peptic ulcer formation is depended from:
- A. With decreasing of secretion of pancreas
 - B. With reverse diffusion of hydrogen ions
 - C. With violation of acid-neutralization function of duodenum
 - D. With decompensation of antral acid-neutralization function
 - E. * Balance between the factors of defence and aggression of gastro-duodenal region
1714. The morning vomiting by “acid” is characterized by?
- A. Hepatitis
 - B. Cholecystitis
 - C. Diabetes
 - D. Hyperthyroidism
 - E. * To peptic ulcerous disease
1715. The most frequent complication of penetrative gastric peptic ulcer is:
- A. Perforation
 - B. Development of stenosis of pylorus
 - C. formation of fistula
 - D. Malignancy
 - E. * Bleeding
1716. The most typical complication of peptic ulcer of posterior wall of duodenum is:
- A. Stenosis.
 - B. Malignancy.
 - C. Penetration to the head of pancreas.
 - D. Perforation.
 - E. * Bleeding.
1717. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
1718. The operation of choice at the perforative peptic ulcer of stomach in the stage of festering peritonitis is:
- A. Antrumectomy
 - B. SPV with sewing of perforation
 - C. Resection of stomach
 - D. Excision of peptic ulcer with vagotomy and pyloroplasty
 - E. * Sewing of perforation

1719. The reliable X-Ray sign of perforation of gastro-duodenal peptic ulcer is:
- Absent of gas in the stomach
 - Kloyber's "cup"
 - Gas in the intestine
 - High location of diaphragm
 - * Presence of free gas in abdominal region
1720. The secretory function stomach is carried out the next membrane of stomach
- * mucous membrane
 - internal muscular layer
 - serosal
 - mucous submembrane
 - external layer
1721. The symptoms of perforative gastric peptic ulcer are: 1. "Knife-like" pain. 2. Wooden belly. 3. Frequent vomiting. 4. Disappearance of hepatic sound. 5. Arterial hypertension. Choose correct combination of answers:
- 1,5.
 - 4,5.
 - 2,3.
 - 1,3,4.
 - * 1,2,4.
1722. The typical ways of metastasis of tumor of antrum part on large curvature of stomach is:
- Lymphatic nodes of pancreas
 - Mesenteric
 - Spleen
 - Paraesophageal lymphatic nodes
 - * Liver
1723. To absolute indication to operative interference at ulcerous illness does not belong
- * scarry-ulcerous stenosis
 - perforation of ulcer
 - profuse bleeding
 - diameter ulcer a more than 3 cm
 - bleeding what does not stopped with conservative
1724. To the gastric – intestinal bleeding of un ulcerous etiology belong
- * Mallory-Weiss syndrome;
 - hemorrhagic erosive gastritis;
 - diseases by Randyu – Oslera – Vebera;
 - Menetrie's sing;
 - all answers are correct.
1725. Udin's sing at a perforated ulcer is
- * feeling at palpation shove the gases which penetrate through the perforated opening
 - dulling perforated sound in the lateral departments of stomach
 - disappearance of hepatic dullness
 - irradiation pain in a shoulder or shoulder-blade
 - sickliness the back vault of vagina
1726. Vomiting coffee-grounds is a characteristic sign
- * bleeding ulcer

- B. penetrative ulcers
 - C. perforated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorus stenosis
1727. Vomiting what arose up in 4-6 hours after eating characteristic for
- A. chronic alcoholic gastritis
 - B. cancer and ulcers of cardia
 - C. * pylorus ulcers
 - D. achalasia of gullet
 - E. ulcer and cancer the body of stomach
1728. What are indications to operative treatment of peptic ulcer disease. 1. Perforation. 2. Penetration. 3. Stenosis of pylorus. 4. Malignancy. 5. Gastro-duodenal bleeding. Correct will be:
- A. Only 1.
 - B. 1,4,5.
 - C. 1,3,5.
 - D. 1,5.
 - E. * 1,2,3,4,5.
1729. What are indications to urgent operative treatment of peptic ulcer disease. 1. Perforation. 2. Penetration. 3. Stenosis of pylorus. 4. Malignancy. 5. Gastro-duodenal bleeding. Correct will be:
- A. 1,2,3,4,5.
 - B. Only 1.
 - C. 1,4,5.
 - D. 1,3,5.
 - E. * 1,5.
1730. What are the normal indexes of Histaminum-stimulated pH of body and antral parts of stomach?
- A. body 5,0-7,0, antrum part of stomach 5-7
 - B. body 3,0-4,0, antrum part of stomach 4-5
 - C. body 8,9-10, antrum part of stomach 1,5-2,5
 - D. body 0,5-0,7, antrum part of stomach 1-2
 - E. * body 1,2-1,6, antrum part of stomach from 5 and higher
1731. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodenum?
- A. * Tarry excrement
 - B. Presence on the formed excrement of strokes of red blood
 - C. Littlechanged blood in an excrement
 - D. Excrement of the raspberry colouring with the admixtures of mucus
 - E. Acholic excrement
1732. What complication of peptic ulcerous disease has disappearance of pain in an epigastrium and appearance characteristic melena?
- A. Penetration of peptic ulcer to the pancreas.
 - B. Malignancy of peptic ulcer.
 - C. Pyloroduodenal stenosis.
 - D. Perforation of peptic ulcer.
 - E. * Bleeding from an peptic ulcer.

1733. What complication of peptic ulcerous disease of stomach is characteristic tension of abdominal muscles ?
- Stenosis.
 - Bleeding .
 - Penetration of peptic ulcer in a pancreas.
 - Covered perforation.
 - * Perforation in a free abdominal cavity.
1734. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- perforation
 - perforation + bleeding
 - pylorus stenosis
 - malignization + penetration
 - * bleeding
1735. What complications of peptic ulcer disease: 1 Penetration, 2 Perforation, 3 Stenosis, 4 Bleeding, 5 Malignancy
- 1,3.
 - 1,3,4.
 - 2,3,4.
 - 1,2,3,4.
 - * 1,2,3,4,5
1736. What examination is performed at suspicion on the perforation of peptic ulcer:
- Colonoscopy.
 - Laparoscopy.
 - Gastroduodenoscopy.
 - Sonography.
 - * X-Ray of abdominal cavity
1737. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - Tarry darkly-cherry chair
1738. What factors is stipulate for the state of patient with peptic ulcerous piloro-duodenal stenosis?
- Hypovolemia
 - Low temperature
 - Arterial hypertension
 - High temperature
 - * Hypovolemia
1739. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- * trichopol
 - Licviroton
 - oxiferiskorbon
 - atropine
 - pirinzsipin

1740. What from the listed do not important at choice method of operative treatment at the cancer of stomach?
- A. Histological structure of tumor
 - B. Prevalence of tumor
 - C. Weight of patients
 - D. Age of patients
 - E. * Duration of disease
1741. What from the listed reasons is main in development of duodeno-gastric reflux?
- A. Tumor of stomach
 - B. peptic ulcerous disease
 - C. Stenosis of pylorus
 - D. Inflammation of bile ducts
 - E. * Violation of duodenal permeability
1742. What from the methods of hemostasis at gastro-duodenal bleeding is most effective?
- A. Transfusion of small doses of blood
 - B. Antispastic drugs
 - C. Antiinflammation drugs
 - D. Intravenous introduction of Vicasol
 - E. * Endoscopic coagulation of bleeding
1743. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy
 - E. the all transferred does not belong
1744. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
1745. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E
 - D. estrogens
 - E. STH
1746. What includes method of Teylor at treatment of perforative peptic ulcer: 1. Putting probe in a stomach, 2 Permanent aspiration of gastric content, 3 Antibiotic therapy, 4 Desintoxication therapy.
- A. 1,3.
 - B. 1,3,4.
 - C. 2,3,4.
 - D. 1,2,3,4.
 - E. * All listed

1747. What indicated at a gastric peptic ulcer, which diagnosed during X-Ray examination of stomach:
- Immediate operation - resection of stomach.
 - Protracted conservative treatment,
 - Periodically X-Ray control
 - Operative treatment
 - * Gastroscopy with biopsy and treatment depending on its result
1748. What is basic methods of diagnostics at suspicion of the perforative gastric peptic ulcer: 1. X-Ray of stomach with barium. 2. Survey X-Ray of abdominal region. 3. Gastroduodenoscopy. 4. Sonography of abdominal region. 5. Laparoscopy. Choose correct combination of answers:
- All answers are correct.
 - 1,3,5.
 - 1,3.
 - 2,3,4.
 - * 2,5.
1749. What is characteristic for compensated pyloroduodenal stenosis :
- Severe condition of patients
 - Permanent pain in epigastric region
 - Constipation
 - Frequent vomiting
 - "Hungry" pain in epigastric region
1750. What is criteria of adequate preoperative preparation at patient with decompensated peptic ulcerous stenosis of pylorus: 1. Level of diuresis. 2. Indexes of volume of circulatory blood. 3. Level of hematocrit. 4. Indexes of electrolytes of blood. Choose correct combination of answers:
- All of answers are wrong.
 - 3 and 4.
 - 2,3,4.
 - 2,4.
 - * All of answers are correct.
1751. What is important in pathogenesis of peptic ulcerous disease of stomach: 1. Decreasing of motility of stomach. 2. Duodeno-gastric reflux. 3. Pancreatitis. 4. Violation of protective properties of mucous membrane. 5. Peritonitis. Choose correct combination of answers.
- 1,2,3,4,5.
 - 1,3,4
 - 1,2,3.
 - 2,4,5.
 - * 1,2,4.
1752. What is indicated at bleeding peptic ulcer of body of stomach and small degree of operating risk:
- Excision of peptic ulcer
 - Sewing of bleeding peptic ulcer with a pyloroplasty and vagotomy
 - Excision of bleeding peptic ulcer with SPV
 - Excision of bleeding peptic ulcer with a pyloroplasty vagotomy
 - * Segmental resection of stomach with a bleeding peptic ulcer with omeprazole
1753. What is indicated for the patient with decompensated peptic ulcerous stenosis of pylorus and convulsive syndrome:
- All of answers are true.

- B. Gastroduodenostomy.
 - C. Urgent gastrostomy.
 - D. Urgent resection of stomach.
 - E. * Resection of stomach in the planned order after the correction of general state.
1754. What is most frequent vagotomy is used:
- A. Posterior trunk.
 - B. Anterior trunk
 - C. Selective
 - D. Trunk
 - E. * SPV
1755. What is normal indexes of stomach pH in a body and in the antrum part of stomach?
- A. body 0,3-0,5, antrum part of stomach 1,1-1,9
 - B. body 0,3-0,5, antrum part of stomach 1,1-1,9
 - C. body 4,0-5,0, antrum part of stomach 1,0-1,5
 - D. body 0,8-1,0, antrum part of stomach 1,5-2,5
 - E. * body 1,6-2,2, antrum part of stomach from 5 and higher
1756. What is not characteristic for perforative peptic ulcer:
- A. Knife-like pain
 - B. Disappearance of hepatic sound
 - C. General weakness
 - D. Wooden belly
 - E. * Spastic pain in abdomen
1757. What is not complication of peptic ulcerous disease:
- A. Pyloroduodenal stenosis.
 - B. Gastro-duodenal bleeding.
 - C. Perforation of peptic ulcer.
 - D. Penetration of peptic ulcer.
 - E. * Malignancy of peptic ulcer.
1758. What is not indication to the operation at a duodenal peptic ulcer:
- A. Penetration of peptic ulcer.
 - B. Failure of conservative therapy.
 - C. Stenosis of pylorus.
 - D. Bleeding from peptic ulcer.
 - E. * Localization of peptic ulcers in a bulb of duodenum.
1759. What is pain localization at peptic ulcer of small curvature of stomach?
- A. In back
 - B. In left inguinal region
 - C. Near a umbilicus
 - D. In right inguinal region
 - E. * In epigastrium
1760. What is phases of perforation: 1 shock, 2 peritonitis, 3 pain phase, 4 “imaginary prosperity”, 5 recovery.
- A. 2,4,5.
 - B. 3,1,5.
 - C. 3,2,5.
 - D. 1,2,3.
 - E. * 1,4,2.

1761. What is prescribed for patients with decompensated stenosis of pylorus before operation: 1. Blood transfusion. 2. Solutions of glucose. 3. Solutions of Ringer. 4. Introduction of ions of potassium. 5. Introduction of osmotic diuretics. Choose correct combination of answers?
- A. 3,4,5.
 - B. 1,3,4.
 - C. 1,2,3.
 - D. 1,4,5.
 - E. * 2,3,4.
1762. What is prescribed for the patient with gastro-duodenal bleeding: 1. Permanent aspiration of gastric content. 2. X-Ray of stomach. 3. Gastroduodenoscopy. 4. Laparoscopy. 5. Determination of hemoglobin. Choose correct combination of answers:
- A. 3,4,5.
 - B. 1,3,4,5
 - C. 3,4,5
 - D. 1,2,3,4
 - E. * 1,3,5
1763. What is the best method of surgical treatment of peptic ulcerous disease, complicated by subcompensated stenosis of pylorus:
- A. Subtotal resection of stomach with a large and small omentum.
 - B. Anterior gastroenterostomy.
 - C. Selective proximal vagotomy.
 - D. Gastroduodenoanastomosis.
 - E. * Selective proximal vagotomy in combination with a pyloroplasty by Finney.
1764. What is the best method of treatment of patient, 28 years, with a duodenal peptic ulcer, complicated by subcompensated piloro-duodenal stenosis:
- A. Posterior gastroenteroanastomosis.
 - B. Trunk vagotomy.
 - C. Subtotal resection of stomach.
 - D. Selective proximal vagotomy.
 - E. * Selective proximal vagotomy in combination with a draining operation.
1765. What is the most typical complication of peptic ulcer with penetration to the head of pancreas:
- A. Reflux.
 - B. Stenosis of cardiac part of stomach.
 - C. Malignancy.
 - D. Perforation.
 - E. * Acute pancreatitis.
1766. What is the reasons of formation of peptic peptic ulcers after resection of stomach:
- A. Acute ileus
 - B. Pancreatitis
 - C. Syndrome of Zollinger - Ellison
 - D. Economy resection of stomach
 - E. * Is not resected antrum part of stomach
1767. What is treatment of peptic peptic ulcer disease with cicatrices and deformation of duodenum
- A. Conservative treatment
 - B. Antibiotic.

- C. Antispastic
 - D. H-2 blockers
 - E. * Operative treatment.
1768. What is triad of Mondor: 1 peptic ulcerous anamnesis, 2 knife-like pain, 3 wooden belly, 4 local peritonitis, 5 the shock state of patient
- A. 2,3,4.
 - B. 1,4,5.
 - C. 2,3,5.
 - D. 1,3,5.
 - E. * 1,2,3.
1769. What is used for diagnostics of perforative peptic ulcers:
- A. X-Ray
 - B. Endoscopy
 - C. Laparocentesis
 - D. Sonography
 - E. * Laparoscopy
1770. What localization of ulcer is most characteristic for the patients of elderly and senile age
- A. * cardial department of stomach
 - B. overhead third of gullet
 - C. lower third of gullet
 - D. bulb of duodewnum
 - E. small curvature
1771. What method diagnostics hte ulcerous illness most informing
- A. * esophagogastroduodenoscopy
 - B. analysis of excrement on the hidden blood
 - C. X-ray
 - D. global analysis of blood
 - E. research of gastric secretion
1772. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- A. On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - B. On blood volume
 - C. * On an arteriotony, pulse, state of patient
 - D. On a globular volume
 - E. On the level of thrombocytes
1773. What operation is performed after the hour after the perforation of gastric peptic ulcer:
- A. Any of the listed operations
 - B. Vagotomy with a pyloroplasty
 - C. Closing of perforative peptic ulcer
 - D. Antrumectomy
 - E. * Classic resection of 2/3 stomach
1774. What operation is performed at decompensated stenosis of pylorus in old patients?
- A. Subtotal resection of stomach
 - B. Resection of stomach
 - C. Pyloroplasty with vagotomy
 - D. Antrumectomy with vagotomy
 - E. * Gastroenteroanastomosis

1775. What operation is performed for patient, 43 years, with bleeding peptic ulcer of antrum part of stomach:
- Gastrectomy.
 - Conservative treatment.
 - Proximal vagotomy.
 - Excision of peptic ulcer with trunk vagotomy.
 - * Resection of stomach.
1776. What operation is performed in patient with the peptic ulcer, penetrated to the pancreas:
- Pyloroplasty by Finney
 - Selective proximal vagotomy.
 - Vagotomy and draining operation.
 - Distal subtotal resection of stomach.
 - * Resection 2/3 stomach
1777. What preparation does behave to blocker H₂-retseptors?
- * tavegil
 - obzidan
 - hystdol
 - cerucal
 - oraza
1778. What preparation does behave to blocker of muscarine receptors of coating cages?
- cymetidin
 - eglonin
 - * gastocepini
 - etimsiloli
 - vinilin
1779. What preparations, except for other properties, own yet and a bacteriostatic effect on Helicobacter pyloris
- * all are transferred preparations
 - Almagel
 - Vinylin
 - De-nol
 - Clarithromycin
1780. What products are recommended in the diet of № 1?
- * hen in a steam kind
 - pancakes
 - raw egg-white
 - bread rye fresh
 - acute cheeses
1781. What special methods of examination need to be applied at differentiation of acute appendicitis with the covered perforative peptic ulcer? 1. Gastroduodenoscopy. 2. X-Ray. 3. Sonography of abdominal region. 4. Laparoscopy. 5. Sciagraphy of stomach with the sulfate of barium. Choose correct combination of answers:
- Only 2 and 5.
 - Only 2 and 4.
 - 1,2,3.
 - 2,3,5.
 - * 1,2,4.

1782. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
1783. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
1784. What treatment of peptic ulcer, complicated by non-stopped gastro-duodenal bleeding?
- A. Introduction of probe of Blekmora.
 - B. Embolization of gastric and gastro-duodenal arteries.
 - C. Operation at the relapse of bleeding.
 - D. Conservative treatment.
 - E. * Urgent operation.
1785. What treatment of the covered perforation of peptic ulcer:
- A. Laparoscopic draining of abdominal cavity.
 - B. Treatment by method of Teylor.
 - C. Operation in the case of ineffective conservative treatment.
 - D. Conservative treatment.
 - E. * Urgent operation.
1786. What treatment of the perforative peptic ulcer after 15 hours from perforation?
- A. Antrumectomy
 - B. Gastroenteroanastomosis.
 - C. Resection of stomach.
 - D. Vagotomy with a pyloroplasty by Finney.
 - E. * Sewing of perforation.
1787. What treatment of the perforative peptic ulcer of antrum part of stomach.
- A. Antrumectomy.
 - B. Sewing of peptic ulcer and trunk vagotomy.
 - C. Resection 3/4 stomach with a large and small omentum.
 - D. Sewing of peptic ulcer.
 - E. * Resection of 2/3 stomach.
1788. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia
1789. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforated ulcers
 - B. at conservative treatment sanguifluous ulcers

- C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pyloristhenosis
1790. When conservative treatment could be performed at perforative peptic ulcer?
- A. Bleeding peptic ulcer
 - B. If a atypical perforation
 - C. At the clinical picture of general peritonitis
 - D. In patients with tuberculosis
 - E. * If patient does not want operation
1791. When could be "rotten" belch ?
- A. At cholecystitis
 - B. At pancreatitis
 - C. At peptic ulcerative disease of stomach
 - D. At peptic ulcerative disease of duodenum
 - E. * At the cancer of stomach
1792. When could be symptom of fluctuation in abdominal cavity?
- A. At pancreatitis
 - B. At peptic ulcerative disease of stomach
 - C. At peptic ulcerative disease of duodenum
 - D. At chronic gastritis
 - E. * Ascitis
1793. When does a melena appear ?
- A. At pancreatitis
 - B. At peptic ulcerative disease of stomach
 - C. At peptic ulcerative disease of duodenum
 - D. At bleeding in the abdominal cavity
 - E. * At gastro-duodenal bleeding
1794. When is observed tension of muscles of stomach ?
- A. At pancreatitis
 - B. At peptic ulcerative disease of stomach
 - C. At peptic ulcerative disease of duodenum
 - D. At chronic gastritis
 - E. * At inflammation of peritoneum
1795. When small curvature of stomach is palpated?
- A. At gastritis
 - B. At decreasing of stomach
 - C. At increasing of stomach
 - D. In a norm
 - E. * At gastropnoia
1796. Where is located inferior measure of stomach?
- A. Below umbilicus on 3-4 cm
 - B. Above umbilicus on 7-8 cm
 - C. At the level of umbilicus
 - D. Below umbilicus on 2-3 cm
 - E. * Above umbilicus on 2-3 cm
1797. Which clinical sign is not characteristic for dumping-syndrome:

- A. A diarrhoea after eating
 - B. Pain in an epigastrium after food intake
 - C. Weakness, dizziness, hard beet filling after the food intake
 - D. A loss of weight, general weakness
 - E. * Vomiting by the eaten food
1798. Which disease is characterized by disappearance of pain in the epigastrium?
- A. Pancreatitis.
 - B. Cholecystitis.
 - C. Pyloroduodenal stenosis.
 - D. Perforation of peptic ulcer.
 - E. * Bleeding from an peptic ulcer.
1799. Which disease is characterized by melena?
- A. Penetration.
 - B. Malignancy .
 - C. Stenosis.
 - D. Perforation .
 - E. * Bleeding from an peptic ulcer.
1800. Which drug is prescribed at the gastro-duodenal bleeding:
- A. Vicalinum.
 - B. Baralgin
 - C. Paracetamol
 - D. Aspirin
 - E. * Dicinon.
1801. Which groups of preparations is prescribed for treatment of peptic ulcerous disease
- A. Antibiotics
 - B. Antiacid preparations
 - C. Histamin blockers
 - D. H-2 blockers
 - E. * Non-steroid antiinflammation drugs.
1802. Which symptoms is absent at perforative gastric peptic ulcer:
- A. Severe state of patient
 - B. Disappearance of hepatic sound.
 - C. Knife-like" pain
 - D. Wooden belly.
 - E. * Frequent vomiting.
1803. Why could be disappearance of hepatic sound at a perforative gastric peptic ulcer?
- A. A presence of liquid in abdominal cavity.
 - B. Swelling of intestine.
 - C. Interposition of intestinal loops between a liver and diaphragm.
 - D. High location of diaphragm.
 - E. * A presence of free gas in an abdominal cavity.
1804. Why during the operations at peptic ulcerous disease resection of 2/3 of stomach is performed:
- A. All of answers are correct.
 - B. All of answers are wrong.
 - C. By the features of blood supply of stomach.
 - D. By the features of lymph supply of stomach.

- E. * By the necessity to remove zones with hastrine and acid production.
1805. Wooden belly is characteristic for:
- A. Acute ileus
 - B. Colitis
 - C. Pancreatitis
 - D. Volvulus
 - E. * Perforative peptic ulcer
1806. A acute cholecystitis usually begins from:
- A. Paine in the left hypohondrium
 - B. Disorders of chair
 - C. High temperature
 - D. Vomiting
 - E. * Paine in the right hypohondrium
1807. A liquid in the cysts of pancreas:
- A. Milk-white
 - B. Green
 - C. Hemorrhagic
 - D. Brown grey
 - E. * Transparent or rather yellow
1808. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intansive, but weakness and dizziness were appeare. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
1809. A primary purpose of treatment of patients with fatty pancreonecrosis before operation is:
- A. Improvement of microcirculation
 - B. Decrease of secretion of pancreas
 - C. Decrease of secretion of stomach
 - D. Analgesia
 - E. * Desintoxication of organism
1810. A remittent icterus is caused:
- A. By the stricture of choledochus
 - B. Peptic ulcer disease
 - C. By the stone in cystic duct
 - D. By the tumor of choledochus
 - E. * By the valve stone of choledochus
1811. Absolute indication to operative treatment the ulcerous illness is
- A. heavy pain syndrome
 - B. * perforation of ulcer
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. giant ulcers
1812. Absolute indication to operative treatment the ulcerous illness is

- A. * voluminous bleeding
 - B. callous ulcers
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. heavy pain syndrome
1813. Absolute indication to operative treatment the ulcerous illness is
- A. ulcerous anamnesis more than 10 years
 - B. * bleeding what do not stopped with conservative
 - C. perforation ulcer in anamnesis
 - D. heavy pain syndrome
 - E. relapses more than 3 times per a year
1814. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. ulcerous anamnesis more than 10 years
 - C. relapse ulcer after the resection of stomach
 - D. relapses more than 3 times per a year
 - E. * cicatrical-ulcerous stenosis of pylorus
1815. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
1816. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
1817. According to time of origin of complications of acute pancreatitis is divided to:
- A. All true
 - B. All false
 - C. Primary and secondary
 - D. Urgent and non-urgent
 - E. * Early and late
1818. After what develops postnecrotic cysts of pancreas?
- A. Acute pancreatitis, edematous form
 - B. Chronic indurative pancreatitis
 - C. Chronic pseudotumor- pancreatitis
 - D. Chronic pancreatitis
 - E. * Acute pancreatitis, pancreonecrosis
1819. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I

- E. saved after all transferred operations
1820. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred
 - E. not saved after all operations
1821. An optimum volume of operation is at a acute biliary pancreatitis:
- A. Draining of bed of pancreas.
 - B. Removing of exudates from abdominal cavity;
 - C. Pancreatectomy;
 - D. Encapsulation of pancreas;
 - E. * Draining of bilious ways;
1822. An unreal pancreatitis cyst contains:
- A. Sinovial liquid
 - B. Gastric juice
 - C. Serous liquid
 - D. Rudiments of teeth, hairs, nails
 - E. * Blood, pancreatitis juice, products of necrosis of pancreas
1823. As a rule, a pseudocyst contains:
- A. Water
 - B. Lymph
 - C. Pus
 - D. Bile
 - E. * Pancreatic juice
1824. At a acute and chronic cholecystitis contra-indicated drugs:
- A. Baralginum
 - B. Atropinum sulfate
 - C. Nospanum
 - D. Omnoponum
 - E. * Morphinum
1825. At a pseudo-tumorous pancreatitis a basic symptom is:
- A. Portal hypertension
 - B. Suppuration
 - C. Pseudocysts
 - D. Paine
 - E. * Intensive mechanical jaundice
1826. At acute pancreatitis with heavy motion a patient must be treated in:
- A. True answer is absent
 - B. All of answers are true
 - C. Home
 - D. Surgical department
 - E. * Department of intensive therapy
1827. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases

- C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
1828. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
1829. At gallstone disease cholecystectomy is performed:
- A. At young persons
 - B. At old patients
 - C. At presence of clinical signs of disease
 - D. At the latent form of disease
 - E. * Always
1830. At III stage blood loss at the bleeding ulcer the patient loses
- A. over 1000 ml blood
 - B. * over 2000 ml blood
 - C. over 500 ml blood
 - D. over 2500 ml blood
 - E. over 1500 ml blood
1831. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
1832. At lung complication of acute pancreatitis respiratory insufficiency is characterized:
- A. By absence of all listed symptoms
 - B. By a frequent superficial breath
 - C. Acrocyanosis
 - D. By the short breath
 - E. * By the presence of all listed symptoms
1833. At the complicated pancreatitis conservative therapy indicated for:
- A. Decreasing of secretion of stomach
 - B. Decreasing of secretion of pancreas
 - C. Treatment of shock
 - D. Decreasing of pain
 - E. * All listed true
1834. At the complicated pancreatitis, bleeding could be from vessels, except:
- A. Left gastric artery
 - B. Gastro-duodenal artery
 - C. Splenic vein
 - D. Splenic artery
 - E. * Hepatic artery
1835. At the destructive forms of acute pancreatitis all surgical operations are divided on:

- A. Not divided
 - B. With complications, without complications
 - C. Invasive, non-invasive operations
 - D. Primary, secondary, repeated operations
 - E. * Early, late, delay operation
1836. At the duodenum ulceroperation of choice is
- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
1837. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
1838. At the III item of activity hemostasis and III sage blood loss from the I – III sage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1839. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
1840. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
1841. Bergman's sing is characteristic for
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1842. Berhtein's sing characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers

- D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
1843. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
1844. Blood loos I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
1845. Blood loos II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
1846. Blood loos III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
1847. Blood supply of body and tail of pancreas is:
- A. Variously
 - B. A.cystica
 - C. A.gastrica sinistra
 - D. A.gastroduodenalis
 - E. * Splenic artery
1848. By localization complications of acute pancreatitis are divided to:
- A. Pancreatic
 - B. Ekstraabdominal
 - C. Intraabdominal
 - D. Parapancreatic
 - E. * All of answers are true
1849. Cells of pancreas, which are makes glucagone:
- A. Z-cells
 - B. Y-cells
 - C. X-cells
 - D. W-cells
 - E. * B-cells
1850. Cells of pancreas, which are makes insulin:
- A. Z-cells

- B. Y-cells
 - C. X-cells
 - D. W-cells
 - E. * B-cells
1851. Classification of clinical passing of acute pancreatitis:
- A. All true
 - B. Edema, necrosis
 - C. Easy, middle, heavy
 - D. Acute, chronic
 - E. * Abortive, progressive
1852. Clinical signs of acute pancreatitis:
- A. Constipation
 - B. Vomiting by blood
 - C. Melena
 - D. Vomiting by „coffee-grounds”
 - E. * There is a pain in the epigastria
1853. Complaints of the patient with pseudo-tumorous pancreatitis:
- A. Yellow color of the skin
 - B. Icterus
 - C. Dispeptic syndrome
 - D. Pain in the epigastric region
 - E. * All of answers are true
1854. Complication of choledocholitis is:
- A. Perforative cholecystitis, peritonitis
 - B. Chronic hepatitis
 - C. Hydrocholecystitis
 - D. Empyema of gall-bladder
 - E. * Icterus, cholangitis
1855. Complications of acute pancreatitis is not:
- A. Omentobursitis
 - B. Biliary hypertension
 - C. Fermentativ peritonitis
 - D. Pylephlebitis
 - E. * Phlegmon of retroperitoneal space
1856. De-Cerven's sign is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatrical-ulcerous pylorostenosis
1857. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom

1858. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
1859. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
1860. Early complication at acute pancreatitis is:
- A. Acute ileus
 - B. Development of diabetes
 - C. Formation of pseudocysts
 - D. Phlegmon of retroperitoneal space
 - E. * Peritonitis
1861. Early complication of acute pancreatitis is not:
- A. True answer is absent
 - B. Enzymes peritonitis
 - C. Collapse
 - D. Pancreatic shock
 - E. * Fistula of pancreas
1862. Eleker's sign is characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
1863. Endoscopic papillosphincterotomy is indicated at such disease, as:
- A. Mechanical jaundice
 - B. Hepatitis
 - C. Postcholecystectomy syndrome
 - D. Peptic ulcer
 - E. * Stenosis of supraduodenal part of choledochus
1864. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- A. tumours
 - B. ulcers
 - C. bleeding polypuses
 - D. erosions
 - E. * changes of evacuation function
1865. Ferment's peritonitis can arise up in the case of disease of such organs of abdominal cavity:
- A. Stomach
 - B. Gall-bladder
 - C. Liver
 - D. Spleen

- E. * Pancreas
1866. Ferment's peritonitis in patients with acute pancreatitis develops:
- A. After 72 hours
 - B. In 5-6 days
 - C. In 12-15 hours
 - D. In 6 hours from the beginning of disease
 - E. * In the period of 24-48 hours
1867. Fermentativ shock at the complicated pancreatitis more frequent arises up at:
- A. Chronic pancreatic fistula
 - B. Abscess of pancreas
 - C. To the edema of pancreas
 - D. Local necrosis of pancreas
 - E. * Subtotal or total necrosis of pancreas
1868. For a cholangitis the most characteristic combination of symptoms: 1) icterus 2) fever 3) anaemia 4) leucocytosis 5) peritonitis
- A. 2,3,5
 - B. 2,5
 - C. 3,4,5
 - D. 1,2,3
 - E. * 1,2,4
1869. For a mechanical icterus, with choledolithiasis, not characteristic:
- A. absence of stercobilin in stool
 - B. hypertermia
 - C. increase of alkaline phosphatase
 - D. Increasing of direct bilirubin of blood
 - E. * An acute increasing of level of amylase is in plasma
1870. For acute cholangitis not characteristic:
- A. High temperature
 - B. Leucocytosis
 - C. Icterus
 - D. Pain in right hypochondrium
 - E. * Unsteady liquid stool
1871. For acute cholangitis not characteristic:
- A. increase of liver
 - B. leucocytosis with shift of formula to the left
 - C. icterus
 - D. increase of temperature
 - E. * decreasing of sizes of liver
1872. For bleeding ulcer characteristic sign is
- A. * pain in an epigastrium;
 - B. knife-like pain;
 - C. signs irritation of peritoneum;
 - D. presence fresh blood in incandescence
 - E. melena;
1873. For bleeding ulcer characteristically
- A. * melena

- B. tension the muscles of front abdominal wall
 - C. Spazarskiy's sign
 - D. sickliness the back vault of vagina
 - E. irradiation pain in a shoulder or shoulder-blade
1874. For clarification of character of icterus and its reason of origin not used:
- A. Sonography
 - B. ERCP
 - C. transcutaneous transhepatic cholangiography
 - D. CT
 - E. * intravenous cholecystocholangiography
1875. For motion of disease ulcerous illness of middle weight characteristically
- A. development of complications
 - B. * relapses 1-2 times per a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 3 and anymore relapses on a year
1876. For patient with gangrenous cholecystitis it is indicated:
- A. Conservative treatment
 - B. Without operation
 - C. Tactic depends from age
 - D. An operation is deferred
 - E. * Urgent operation
1877. For perforated ulcer characteristically
- A. * tension the muscles of front abdominal wall
 - B. melena
 - C. vomiting by coffee-grounds
 - D. high intestinal impassability
 - E. vomiting stagnant gastric maintenance
1878. For pneumoperitoneum is characteristic symptom
- A. * Zhober's;
 - B. Khelatid's;
 - C. Podlag's;
 - D. Vigats's;
 - E. Udin's.
1879. For the heavy flow of ulcerous illness characteristically
- A. 2 and anymore relapses on a year
 - B. * 3 and anymore relapses on a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 6 and more relapses are on a year
1880. For the heavy flow of ulcerous illness characteristically
- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days

1881. Forming of pancreatic infiltrate is depended from:
- A. Toxic influence
 - B. Autoimmune inflammation
 - C. Septic inflammation
 - D. Allergic reaction
 - E. * Aseptic inflammation
1882. Frequency of hepatic insufficiency at complicated acute pancreatitis:
- A. In 95 % patients
 - B. In 2 % patients
 - C. In 75 % patients
 - D. In 100 % patients
 - E. * In 25 % patients
1883. From what department degestive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
1884. Gallstone disease is complicated by all of listed, except:
- A. Mechanical jaundice
 - B. Development of destructive cholecystitis
 - C. Secondary pancreatitis
 - D. Cancer of gall-bladder
 - E. * Development of cirrhosis of liver
1885. Gallstone disease is not complicated:
- A. Cholangitis
 - B. Fistula
 - C. By acute cholecystitis
 - D. By mechanical icterus
 - E. * Intra-abdominal bleeding
1886. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
1887. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
1888. Holsted symptom at acute pancreatitis is:
- A. Cyanosis of hands
 - B. Yellow skin around umbilicus
 - C. Violet spots are on face and body

- D. Cyanosis of lateral surfaces of abdomen and body
 - E. * Cyanosis of skin of abdomen
1889. Hormone of pancreas which is responsible for metabolic of fat:
- A. Somatotropinum
 - B. Adrenalin
 - C. Insulin
 - D. Glukagon
 - E. * Lipocainum
1890. How often pancreatic part of common bile duct pass through the head of pancreas?
- A. 40-50 %
 - B. 30-40 %
 - C. 25-35 %
 - D. 10-20 %
 - E. * 80-90 %
1891. If patient has frequent „fatty stool” with undigested meat, it could be:
- A. Cirrhosis of liver
 - B. Ulcerous disease of duodenum
 - C. Chronic hepatitis
 - D. Chronic duodenitis
 - E. * Chronic pancreatitis
1892. In case of acute pancreatitis bleeding could be to:
- A. Intestine (at internal fistula)
 - B. Abdominal cavity
 - C. Wound
 - D. External fistula
 - E. * At all listed variants
1893. In case of purulent inflammation of the pseudocysts of pancreas is used:
- A. Cystoenteroanastomosis
 - B. Cystoduodenostomy
 - C. Cystogastrostomy
 - D. Cystoenterostomy
 - E. * External draining of cyst
1894. In relation to peritoneum pancreas is located:
- A. All of answers are correct
 - B. All of answers are incorrect
 - C. Intraperitoneally
 - D. Mesoperitoneally
 - E. * Retroperitoneally
1895. In the case of gallstone disease urgent operation is indicated:
- A. At a hepatic colic
 - B. At mechanical joundice
 - C. At occlusion of cystic duct
 - D. At Cholecysto-pancreatitis
 - E. * At perforative cholecystitis
1896. In the case of surgical treatment of the complicated acute pancreatitis does not used:
- A. Right-side resection of gland

- B. Left-side resection of gland
- C. Omentopancreatopexy
- D. Abdominisation of pancreas
- E. * Pankreatojejunostomy

1897. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?

- A. small curvature of stomach;
- B. back wall of stomach, nearer to small curvature;
- C. large curvature of stomach
- D. * cardiac part of stomach;
- E. pylorus.

1898. In what vein is a venous outflow carried out in from a stomach?

- A. * V. Portae;
- B. V. odd;
- C. V. pair;
- D. V. overhead hollow;
- E. V. lower hollow;

1899. Intraoperative cholangiography is not indicated:

- A. At icterus during the operation
- B. At dilatation of choledochus
- C. At presence of icterus in anamnesis
- D. At tumor of head of pancreas
- E. * At a single large stone in the common bile duct

1900. Irradiation of pain to the back could be in case of:

- A. Acute cystitis
- B. Gastric ulcers
- C. Acute ileus
- D. Acute cholecystitis
- E. * Acute pancreatitis

1901. Large ulcer is an ulcer measuring

- A. 1-4 cm
- B. 1-3 cm
- C. 3-5 cm
- D. 2- 6 cm
- E. * 2-3 cm

1902. Late complications at acute pancreatitis are:

- A. Acute ileus
- B. Shock
- C. Renal insufficiency
- D. Peritonitis
- E. * Phlegmon of retroperitoneal space

1903. Late complications of acute pancreatitis are:

- A. Cysts and fistula of pancreas
- B. Abscesses of abdominal cavity
- C. Phlegmon retroperitoneal tissue
- D. Festering pancreatitis and parapancreatitis
- E. * All of answers are true

1904. Little ulcer it is an ulcer measuring
- A. * to 0,5 cm
 - B. 0,5-1 cm
 - C. to 1,0 cm
 - D. 3 to 1,5 cm
 - E. 0,5-1,5 cm
1905. Lung complications is includes:
- A. Bronchial asthma
 - B. Pulmonary insufficiency
 - C. Abscess of lights
 - D. Right-side pleurisies and pneumonias
 - E. * Left-side pleurisies and pneumonias
1906. Main reason of acute pancreatitis is:
- A. Achalasia
 - B. Chronic alcoholic pancreatitis
 - C. Alimentary factor
 - D. Trauma of pancreas
 - E. * Gallstone disease
1907. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
1908. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood
1909. Melena is a characteristic sign
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorus stenosis
 - C. perforeted ulcers
 - D. penetration ulcers
 - E. malignization ulcers
1910. Method of instrumental examination of pseudocysts is:
- A. Biopsy
 - B. Endoscopy
 - C. Colonoscopy
 - D. Sciagraphy of organs of abdominal cavity
 - E. * Sonography of organs of abdominal cavity
1911. Mondor symptom at acute pancreatitis is:
- A. Cyanosis of hands
 - B. Yellow skin around umbilicus
 - C. Cyanosis of skin of abdomen

- D. Cyanosis of lateral surfaces of abdomen and body
 - E. * Violet spots are on face and body
1912. Most informing method at a bleeding ulcer
- A. survey sciagraphy the organs of abdominal region
 - B. * EFGDS
 - C. sciagraphy the stomach with contrasting
 - D. Sonography
 - E. Laparoscopy
1913. Mostly a intoxication psychosis can arise up at abuse of:
- A. Smoking
 - B. Medicines
 - C. Drugs
 - D. Fatty food
 - E. * Alcohol
1914. Name class complications of ulcerous illness
- A. bleeding, pylorostenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pylorostenosis, malignization
 - C. malignization, pylorostenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
1915. Name specific complications of acute pancreatitis in early and late postoperative periods:
- A. Phlegmon of retroperitoneal space
 - B. Pseudocyst of pancreas
 - C. Fistula of pancreas
 - D. Bleeding
 - E. * All of answers are true
1916. Necrectomy of pancreas is mean:
- A. True answer is absent
 - B. Complete removing of pancreas
 - C. Removing of part of pancreas with its transversal cutting
 - D. Removing of necrotic area within the measures of necrotic tissue
 - E. * Removing of necrotic area is within the limits of healthy tissue
1917. Normal indexes of білірубіну of blood:
- A. 60,6-80,5 mmol/l
 - B. 40,6-60,5 mmol/l
 - C. 20,6-40,5 mmol/l
 - D. 0-1,6 mmol/l
 - E. * 1,7-20,5 mmol/l
1918. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
1919. Pancreatectomy is mean:
- A. True answer is absent

- B. Removing of necrotic area within the measures of necrotic tissue
 - C. Removing of necrotic area is within the limits of healthy tissue
 - D. Removing of part of pancreas with its transversal cutting
 - E. * Complete removing of pancreas
1920. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
1921. Principle of conservative treatment of fistula of pancreas:
- A. Conservative treatment is non-effective
 - B. Improvement of outflow of bile
 - C. Increase of regeneration
 - D. Increase of pancreatic secretion
 - E. * Decrease of pancreatic secretion
1922. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
1923. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
1924. Resection of pancreas is mean:
- A. True answer is absent
 - B. Complete removing of pancreas
 - C. Removing of necrotic area within the measures of necrotic tissue
 - D. Removing of necrotic area is within the limits of healthy tissue
 - E. * Removing of part of pancreas with its transversal cutting
1925. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
1926. Result of hypersecretion of pancreas can be the spasm of sphincter:
- A. Heyster
 - B. Vestfal
 - C. Mirizzi
 - D. Lutkins
 - E. * Oddi

1927. Sekvestrectomy of pancreas is mean:
- A. True answer is absent
 - B. Complete removing of pancreas
 - C. Removing of part of pancreas with its transversal cutting
 - D. Removing of necrotic area is within the limits of healthy tissue
 - E. * Removing of necrotic area within the measures of necrotic tissue
1928. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
1929. Septic complications of acute pancreatitis is indication to:
- A. Analgesic treatment
 - B. Antibiotic treatment
 - C. Conservative treatment
 - D. Sonography
 - E. * Operation
1930. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
1931. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
1932. Specify indication to early operative treatment at acute pancreatitis:
- A. Forming of pseudocyst
 - B. Acute fatty pancreatitis
 - C. Acute edematous pancreatitis
 - D. Acute pancreatolysis
 - E. * Acute traumatic pancreatitis
1933. Specify indication to early operative treatment at acute pancreatitis:
- A. Forming of pseudocyst
 - B. Acute fatty pancreatitis
 - C. Acute oedematous pancreatitis
 - D. Acute pancreatolysis
 - E. * Acute biliary pancreatitis
1934. Specify one of symptoms, what not characteristic for hydropsy of gallbladder:
- A. Absence of peritoneal symptoms
 - B. palpable gall-bladder
 - C. Increase of gall-bladder
 - D. Pains in right hypohondrium

- E. * Icterus
1935. Specify the best therapy of parapancreatic infiltrate:
- A. Operative treatment
 - B. Antispastic
 - C. Analgesic treatment
 - D. Desintoxication
 - E. * Antibiotic
1936. Specify the most dangerous complication of pancreonecrosis:
- A. Pseudocyst of pancreas
 - B. Diabetes
 - C. Inflammation
 - D. Fibrosis of pancreas
 - E. * Bleeding
1937. Specify the most effective treatment of the formed non-complicated cyst:
- A. Cystogastrostomy
 - B. External draining of cyst
 - C. A resection of cyst
 - D. Conservative treatment
 - E. * Cystoenteroanastomosis
1938. Specify the most effective treatment of the non-formed complicated cyst:
- A. Cystogastrostomy
 - B. Cystoenteroanastomosis
 - C. A resection of cyst
 - D. Conservative treatment
 - E. * External draining of cyst
1939. Specify the norm of diastase:
- A. Up to 10
 - B. Up to 30
 - C. Up to 20
 - D. Up to 50
 - E. * Up to 160
1940. Specify what pathology is reason of development of pseudocyst of pancreas:
- A. Liver cirrhosis
 - B. Peptic ulcer
 - C. Diabetes
 - D. Acute cholecystitis
 - E. * Acute pancreatitis
1941. Symptom of Bonde at acute pancreatitis is:
- A. Cyanosis of hands
 - B. Yellow skin around umbilicus
 - C. Cyanosis of skin of abdomen
 - D. Cyanosis of lateral surfaces of abdomen and body
 - E. * Swelling of abdomen only in the epigastric area
1942. Symptom of Kerte at a acute pancreatitis it:
- A. Hyper seniti of skin in the projection of gland
 - B. Yellow skin around umbilicus

- C. Absence of pulsation of abdominal aorta
 - D. Pain in left costal-vertebral area
 - E. * Pain and proof tension of muscles in the epigastria with irradiation to left hypochondria
1943. Symptom of Kulen at acute pancreatitis is:
- A. Cyanosis of hands
 - B. Violet spots are on face and body
 - C. Cyanosis of skin of abdomen
 - D. Cyanosis of lateral surfaces of abdomen and body
 - E. * Yellow skin around umbilicus
1944. Symptom of Meyo-Robson at acute pancreatitis is:
- A. Hyper seniti of skin in the projection of gland
 - B. Yellow skin around umbilicus
 - C. Pain and proof tension of muscles in the epigastria with irradiation to left hypochondria
 - D. Absence of pulsation of abdominal aorta
 - E. * Pain in left costal-vertebral area
1945. Symptom of Voskresensky at acute pancreatitis is:
- A. Hyper seniti of skin is in the projection of gland
 - B. Yellow skin around umbilicus
 - C. Pain and tension of muscles in the epigastria
 - D. Pain in the left costal-vertebral area
 - E. * Absence of pulsation of abdominal aorta
1946. The basic method ofexamination of acute cholecystitis is:
- A. Gastroduodenoscopy
 - B. Laparoscopy
 - C. Cholegraphy
 - D. Endoscopy
 - E. * Sonography of gall-bladder
1947. The best time of operative treatment at acute pancreatitis after beginning of disease is:
- A. 7-8 days
 - B. 3-4 days
 - C. 4-5 days
 - D. 1-3 days
 - E. * Surgical treatment is not indicated
1948. The diameter of ductus choledochus is :
- A. over 2,0 cm
 - B. 1,6-2,0 cm
 - C. 1,1-1,5 cm
 - D. to 0,5 cm
 - E. * 0,6-1,0 cm
1949. The early bleeding at the complicated acute pancreatitis are more frequent:
- A. In the first minute
 - B. During the first hour
 - C. During the first minute
 - D. At a few first hours
 - E. * In a few first days

1950. The early bleeding at the complicated pancreatitis is stopped by using:
- A. Cold on the abdomen
 - B. Hot-water bottles on the abdomen
 - C. Rest and cold on the abdomen
 - D. Operative treatment
 - E. * Ordinary haemostatic drugs
1951. The first period of acute pancreatitis has the name:
- A. A right answer is absent
 - B. All of answers are true
 - C. Degenerative and festering complications
 - D. Functional insufficiency of parenchyma's organs
 - E. * Hemodynamic violations and pancreatic shock
1952. The most frequent complication after ERCP is:
- A. Chronic hepatitis
 - B. Reactive cholecystitis
 - C. Pancreatic sepsis
 - D. Cholangitis
 - E. * Pancreatitis
1953. The most informing method for diagnostics of acute pancreatitis is:
- A. Colonoscopy
 - B. Duodenodcopy
 - C. Endoscopy
 - D. ECG
 - E. * Sonography
1954. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
1955. The period of degenerative complications has:
- A. 2 hours
 - B. 2 weeks
 - C. 1-3 days
 - D. 3-7 days
 - E. * over 7 days
1956. The period of functional insufficiency of abdominal organs has:
- A. 2 hours
 - B. 2 weeks
 - C. over 7 days
 - D. 1-3 days
 - E. * 3-7 days
1957. The period of hemodynamic violations and pancreatic shock has:
- A. 2 hours
 - B. 2 weeks
 - C. over 7 days

- D. 3-7 days
 - E. * 1-3 days
1958. The principle of operation at acute biliary pancreatitis:
- A. Draining of parapancreatic tissue
 - B. Removing of fluid from abdominal cavity
 - C. Pancreatectomy
 - D. Decapsulation of pancreas
 - E. * Draining of bile ducts
1959. The reason of development of mechanical jaundice can be all, except
- A. Stenosis of duodenal papilla
 - B. Stricture of the choledochus
 - C. Stone in proximal part of choledochus
 - D. Increase of head of pancreas
 - E. * To the stone in the area of neck of gall-bladder
1960. The second period of acute pancreatitis has the name:
- A. A right answer is absent
 - B. All of answers are true
 - C. Degenerative and festering complications
 - D. Hemodynamic violations and pancreatic shock
 - E. * Functional insufficiency of parenchyma's organs
1961. The secretory function stomach is carried out the next membrane of stomach
- A. * mucous membrane
 - B. internal muscular layer
 - C. serosal
 - D. mucous submembrane
 - E. external layer
1962. The symptom of Curvuasie is not observed at cancer of:
- A. Head of pancreas
 - B. Duodenal papilla
 - C. Retroduodenal part of common bile duct
 - D. Supraduodenal part of choledochus
 - E. * Gall-bladder
1963. The symptoms of intoxication psychosis at the complicated acute pancreatitis is:
- A. Visual hallucinations
 - B. Aggression
 - C. Apathy
 - D. Hypodynamia
 - E. * Disorientation
1964. The third period of acute pancreatitis has the name:
- A. A right answer is absent
 - B. All of answers are true
 - C. Hemodynamic violations and pancreatic shock
 - D. Functional insufficiency of parenchyma's organs
 - E. * Degenerative and festering complications
1965. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scarry-ulcerous stenosis

- B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
1966. To the gastric – intestinal bleeding of un ulcerous etiology belong
- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.
1967. Total pancreonecrosis is characterized by:
- A. Increasing of AST
 - B. Decreasing of activity of diastase
 - C. Increasing of ALT
 - D. Decreasing of AST
 - E. * Increasing of activity of diastase
1968. Turner symptom at acute pancreatitis is:
- A. Cyanosis of hands
 - B. Yellow skin around umbilicus
 - C. Cyanosis of skin of abdomen
 - D. Violet spots are on face and body
 - E. * Cyanosis of lateral surfaces of abdomen and body
1969. Udin's sing at a perforated ulcer is
- A. * feeling at palpation shove the gases which penetrate through the perforated opening
 - B. dulling perforated sound in the lateral departments of stomach
 - C. disappearance of hepatic dullness
 - D. irradiation pain in a shoulder or shoulder-blade
 - E. sickliness the back vault of vagina
1970. Vomiting by „coffee-grounds” at acute pancreatitis is predefined:
- A. By presence of enzymes in blood
 - B. By violation of microcirculation
 - C. By the presence of concomitant gastric ulcer
 - D. By the presence of concomitant gastritis
 - E. * By formation of erosions in a stomach
1971. Vomiting coffee-grounds is a characteristic sign
- A. * bleeding ulcer
 - B. penetrative ulcers
 - C. perforated ulcers
 - D. malignizated ulcers
 - E. for cicatrical-ulcerous pylorus stenosis
1972. Vomiting what arose up in 4-6 hours after eating characteristic for
- A. chronic alcoholic gastritis
 - B. cancer and ulcers of cardia
 - C. * pylorus ulcers
 - D. achalasia of gullet
 - E. ulcer and cancer the body of stomach

1973. What are complications of acute pancreatitis:
- A. Phlegmon of retroperitoneal space
 - B. Biliary hypertension
 - C. Omentobursitis
 - D. Fermentativ peritonitis
 - E. * All indicated complication
1974. What are cysts of pancreas ?:
- A. Traumatic
 - B. Inflammatory
 - C. After echinococcus
 - D. Real and unreal
 - E. * All of answers are true
1975. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodwnum?
- A. * Tarry excrement
 - B. Presence on the formed excrement of strokes of red blood
 - C. Littlechanged blood in an excrement
 - D. Excrement of the raspberry colouring with the admixtures of mucus
 - E. Acholic excrement
1976. What combination of clinical symptoms does explain the syndrome of Curvuasie?
- A. an icterus, enlarged liver
 - B. absence of stool, pain, appearance of formation in abdominal region
 - C. icterus, local peritoneal phenomena
 - D. increase of liver, hydro-peritoneum, expansion of veins of front abdominal wall
 - E. * A painless enlarged gall-bladder in combination with icterus
1977. What complication of acute pancreatitis?
- A. Paranepritis
 - B. Cyst of pancreas
 - C. Pylephlebitis
 - D. Abscess of Duglas space
 - E. * Pancreonecrosis
1978. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- A. perforation
 - B. perforation + bleeding
 - C. pylorus stenosis
 - D. malignization + penetration
 - E. * bleeding
1979. What components is absent in urine at a mechanical jaundice?
- A. All are present
 - B. Bilious acids.
 - C. Not direct bilirubin.
 - D. Direct bilirubin.
 - E. * Urobilin.
1980. What does the pseudocyst of pancreas behave to?:
- A. All of answers are true
 - B. Symptom of acute pancreatitis

- C. Congenital pathology of pancreas
 - D. Early complication of acute pancreatitis
 - E. * Late complication of acute pancreatitis
1981. What drug is used at chronic pancreatitis with violation of the external function of pancreas?
- A. Panthenol
 - B. Pyracetamum
 - C. Pantocrinum
 - D. Papaverin
 - E. * Panzinorm
1982. What drugs from cytostatic group is used in acute pancreatitis:
- A. Mezimforte
 - B. Baralgin
 - C. Creon
 - D. Motilium
 - E. * 5-ftoruracyl
1983. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
1984. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol
 - B. Liciviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
1985. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy
 - E. the all transferred does not belong
1986. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
1987. What hormone of pancreas responsible for metabolism of glucose:
- A. Tiroksin
 - B. Somatotropinum
 - C. Adrenalin
 - D. Vasopressinum
 - E. * Insulin

1988. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E
 - D. estrogens
 - E. STH
1989. What indications for surgical treatment of cholecystitis?
- A. Dispeptic syndrome
 - B. presence of pancreatitis
 - C. Concomitant changes in a liver
 - D. Anamnesis of disease
 - E. * Presence of stones in the gall-bladder
1990. What is complication of acute pancreatitis:
- A. Hepatitis
 - B. Cyst of pancreas
 - C. Cirrhosis of liver
 - D. Pylephlebitis
 - E. * Pankreonekrosis
1991. What is conservative treatment before operation in patients with severe form of hemorrhagic pancreonecrosis:
- A. Improvement of microcirculation
 - B. Decrease of secretion of pancreas
 - C. Decrease of secretion of stomach
 - D. Analgesia
 - E. * Desintoxication of organism
1992. What is early complications of acute pancreatitis?
- A. Shock
 - B. Jaundice
 - C. Peritonitis
 - D. Acute hepatic-renal insufficiency
 - E. * All of answers are true
1993. What is included in conservative treatment of acute pancreatitis?
- A. Morning exercises
 - B. High caloric diet
 - C. A diet by Pevzner N15
 - D. A diet by Pevzner N5
 - E. * Hunger
1994. What is local symptoms of retroperitoneal phlegmon at complicated acute pancreatitis?
- A. Hyperemia of tissue
 - B. Swelling of tissue
 - C. Tension of lumbar muscles
 - D. Pain during palpation on the left hypochondrium
 - E. * All of symptoms true
1995. What is mechanism of Voskresensky symptom at acute pancreatitis:
- A. Development of peritonitis
 - B. Embolism of abdominal aorta

- C. Thrombosis of abdominal aorta
 - D. Reflex-paresis of colon
 - E. * Inflammatory edema of pancreas
1996. What is not inhibitors of protease:
- A. Pantripin
 - B. Trasilol
 - C. Gordoxum
 - D. Kontrikal
 - E. * Tebris
1997. What is result of pancreatic infiltrate:
- A. Development of pseudocyst
 - B. Distribution of process with development of peritonitis
 - C. Formation of capsule
 - D. Quick disappear
 - E. * Slow (during 1,5-3 month) disappear
1998. What is the basic method of treatment of acute pancreatitis:
- A. Diet
 - B. Physical therapy
 - C. Homoeopathic
 - D. Surgical
 - E. * Conservative
1999. What is the basic methods of diagnostics of postnecrotic cysts:
- A. Laparoscopy
 - B. ERCP
 - C. Colonoscopy
 - D. Endoscopy
 - E. * Sonography and CT
2000. What is the best method of examination of pancreatic infiltrate:
- A. Sonography
 - B. X-Ray
 - C. Biochemical blood test
 - D. Palpation
 - E. * Sonography
2001. What is the best resort which is used for pathology of pancreas?
- A. Nemirov
 - B. Truskavets
 - C. Kuyal'nik
 - D. P'yatigorsk
 - E. * Morshin
2002. What is the complications of pseudocyst of pancreas:
- A. Fistula
 - B. Bleeding
 - C. Perforation
 - D. Suppuration
 - E. * All is true
2003. What is the inhibitors of protease:

- A. Trypsinum
 - B. Tocopherolum
 - C. Loroxon
 - D. Tebris
 - E. * Trasilol
2004. What is the inhibitors of protease:
- A. Gramicidine
 - B. Loroxon
 - C. Garamycine
 - D. Motilium
 - E. * Gordox
2005. What is the method of treatment of chronic calculus cholecystitis?
- A. Conservative therapy
 - B. Antispastic drugs
 - C. Lithothripsy
 - D. Cholecystostomy
 - E. * Cholecystectomy
2006. What is the most effective treatment of the non-formed non-complicated cyst:
- A. Cystogastrostomy
 - B. Cystoenteroanastomosis
 - C. A resection of cyst
 - D. External draining of cyst
 - E. * Conservative treatment
2007. What is the most frequent reason of development of mechanical jaundice?
- A. Metastases of tumor into the liver
 - B. Peptic ulcer
 - C. Cancer of head of pancreas
 - D. Stricture of extrahepatic bile ducts
 - E. * Choledocholitis
2008. What is the reason of late complications of acute pancreatitis?
- A. By violation of local blood flow
 - B. Obstruction of pancreatic ducts
 - C. Development of aseptic inflammation
 - D. Enzymes
 - E. * Infection
2009. What localization of ulcer is most characteristic for the patients of elderly and senile age
- A. * cardial department of stomach
 - B. overhead third of gullet
 - C. lower third of gullet
 - D. bulb of duodewnum
 - E. small curvature
2010. What method diagnostics hte ulcerous illness most informing
- A. * esophagogastrroduodenoscopy
 - B. analysis of excrement on the hidden blood
 - C. X-ray
 - D. global analysis of blood
 - E. research of gastric secretion

2011. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- A. On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - B. On blood volume
 - C. * On an arteriotomy, pulse, state of patient
 - D. On a globular volume
 - E. On the level of thrombocytes
2012. What method of examination is most informative at the estimation of pathology of bile ducts?
- A. CT
 - B. Sonography
 - C. transcutaneous transhepatic cholangiography
 - D. intravenous cholangiography
 - E. * ERCP
2013. What method of examination is most informative for diagnostics of calculus cholecystitis?
- A. ERCP
 - B. Endoscopy
 - C. X-Ray
 - D. Laparoscopy
 - E. * Sonography
2014. What operation is indicated at the edematous form of acute pancreatitis:
- A. Marsupialisation
 - B. Abdominisation of pancreas
 - C. Omentopancreatopexy
 - D. Pancreatectomy
 - E. * Operation is not needed
2015. What operation is not performed at pancreatic abscesses and infected necrosis?
- A. Draining of abscess
 - B. Pancreato-necro-sekvestrectomy with laparostomy
 - C. Pancreato-sekvestrectomy
 - D. Pancreato-necro-sekvestrectomy
 - E. * Total pancreatectomy
2016. What operation is performed at localization of the formed pseudocyst in the tail of pancreas:
- A. Cistoenteroanastomosis
 - B. Cistoenterostomy
 - C. Conservative treatment
 - D. External draining of cyst
 - E. * Resection of tail of pancreas
2017. What operation is performed at the pseudocyst of pancreas in III stage usually:
- A. Conservative treatment
 - B. Cistoduodenostomy
 - C. Cystogastrostomy
 - D. External draining of cyst
 - E. * Cistoenterostomy
2018. What pathology is characterized by presence of plenty of hemorrhagic exudates with high ferment activity in the abdominal cavity?

- A. Destructive cholecystitis
 - B. Perforation of ulcer
 - C. Hepatitis
 - D. Cirrhosis of liver
 - E. * Pancreonecrosis
2019. What place does occupy an acute pancreatitis among acute surgical diseases?
- A. It is most widespread
 - B. Second place
 - C. Fifth place
 - D. First place
 - E. * Third place
2020. What preparation does behave to blocker H₂-retseptors?
- A. * tavegil
 - B. obzidan
 - C. hystdol
 - D. cerucal
 - E. oraza
2021. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
2022. What preparations, except for other properties, own yet and a bacteriostatic effect on *Helicobacter pyloris*
- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
2023. What procedure is prescribed for patients with plenty of hemorrhagic exudates with high ferment activity in the abdominal cavity?
- A. Conservative treatment
 - B. Computer examination of organs of abdominal cavity
 - C. X-Ray of organs of abdominal cavity
 - D. Sonography of organs of abdominal cavity
 - E. * Laparotomy
2024. What procedure must be performed at the postnecrotic cysts of pancreas:
- A. Omentopancreatopexy
 - B. Pancreatotomy
 - C. Necrectomy
 - D. Pancreatectomy
 - E. * Puncture and external draining of cyst
2025. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white

- D. bread rye fresh
 - E. acute cheeses
2026. What symptom is typical for a acute pancreatitis?
- A. Rovzing symptom
 - B. Lenander symptom
 - C. Ker symptom
 - D. Pasternacky symptom
 - E. * Kulen symptom
2027. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
2028. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
2029. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia
2030. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforeted ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pyloristenosis
2031. When could be intoxication psychosis at acute pancreatitis?
- A. In 2 weeks
 - B. On the 9-11 days
 - C. On the 6-8 days
 - D. On the first day
 - E. * On the 2-3 days
2032. When do patients have late complications of acute pancreatitis?
- A. 1-2 days
 - B. 2-3 days
 - C. 5-6 days
 - D. 3-4 days
 - E. * 10-12 days
2033. Which drug is applied at chronic pancreatitis:

- A. Panthenol.
- B. Pyracetamum;
- C. Pantocrinum;
- D. Papaverin;
- E. * Creon

2034. Which pathology characterized by absence of pulsation of abdominal aorta (Voskresensky symptom):

- A. Acute appendicitis
- B. Acute ileus
- C. Peptic ulcer
- D. Acute cholecystitis
- E. * Acute pancreatitis

2035. Which pathology characterized by appearance of cyanosis of lateral surfaces of abdomen (symptom of Turner):

- A. Acute appendicitis
- B. Acute ileus
- C. Acute cholecystitis
- D. Peptic ulcer
- E. * Acute pancreatitis

2036. Which pathology characterized by appearance of pain in left costal-vertebral area (symptom of Meyo-Robson):

- A. Acute appendicitis
- B. Acute ileus
- C. Acute cholecystitis
- D. Peptic ulcer
- E. * Acute pancreatitis

2037. Which pathology characterized by violet spots on the skin and body:

- A. Acute appendicitis
- B. Acute ileus
- C. Peptic ulcer
- D. Acute cholecystitis
- E. * Acute pancreatitis

2038. A frequent liquid stool is the first sign of:

- A. haemorrhoids
- B. Fissures of anus
- C. Proctosigmoiditis
- D. * UUC
- E. All of answers are correct

2039. A method of choice at surgical treatment of unspecific ulcerative colitis

- A. * proctocolectomy
- B. hemicolectomy
- C. resection of colon
- D. colectomy
- E. application of colostomy.

2040. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appeared. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is

- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
2041. A patient after the electroscission of polypus of sigmoid bowel has bleeding. What is tactic?
- A. * it is performed hemostatic therapy
 - B. it is performed operative treatment
 - C. concervative treatment
 - D. laparotomy
 - E. laparoscopy
2042. A patient after the electroscission of polypus of sigmoid bowel has stomach-aches. What complication can be?
- A. * perforation
 - B. bleeding
 - C. malignancy
 - D. toxic dilatation
 - E. penetration
2043. A patient with 10-years anamnesis of unspecific ulcerative colitis has periodic swelling of abdomen, feeling of the incomplete emptying, worsening of the general state. On irrigography is absent haustration with circular narrowing in sigmoid bowel. What complication patient has?
- A. * A regeneration to the cancer
 - B. Toxic dilatation
 - C. Bleeding
 - D. Perforation
 - E. Nothing
2044. A patient with 10-years anamnesis of unspecific ulcerative colitis has periodic swelling of abdomen, feeling of the incomplete emptying, worsening of the general state. On irrigography is absent haustration with circular narrowing in sigmoid bowel. What operation is prescribed?
- A. * Colproctectomy
 - B. Resection of sigmoid bowel
 - C. Left-side hemicolectomy
 - D. Sigmoidostomy
 - E. Right-side hemicolectomy
2045. A presence of mucus and pus in stool is characteristic for:
- A. Proctosigmoiditis
 - B. Cancer of rectum
 - C. UUC
 - D. All of answers are wrong
 - E. * All of answers are correct
2046. Absolute indication for the surgical treatment of unspecific ulcerative colitis
- A. * bleeding, perforation, toxic dilatation
 - B. absence of effect from conservative treatment
 - C. frequent diarrhea, loss of weight
 - D. a pain syndrome
 - E. adhesion
2047. Absolute indication to operative treatment the ulcerous illness is

- A. heavy pain syndrome
 - B. * perforation of ulcer
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. giant ulcers
2048. Absolute indication to operative treatment the ulcerous illness is
- A. * voluminous bleeding
 - B. callous ulcers
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. heavy pain syndrome
2049. Absolute indication to operative treatment the ulcerous illness is
- A. ulcerous anamnesis more than 10 years
 - B. * bleeding what do not stopped with conservative
 - C. perforation ulcer in anamnesis
 - D. heavy pain syndrome
 - E. relapses more than 3 times per a year
2050. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. ulcerous anamnesis more than 10 years
 - C. relapse ulcer after the resection of stomach
 - D. relapses more than 3 times per a year
 - E. * cicatrical-ulcerous stenosis of pylorus
2051. Absolute indication to operative treatment the ulcerous illness is
- A. relapses more than 2 one time per a year
 - B. * malignization ulcers
 - C. ulcerous anamnesis more than 10 years
 - D. heavy pain syndrome, proof heartburn
 - E. relapse ulcer after vagotomy
2052. Absolute sign of unstable hemostasis
- A. * profluvium blood from a vessel;
 - B. absence blood in a stomach and bulb of duodenum;
 - C. presence light blood and faltungs of blood in a stomach;
 - D. all answers are correct;
 - E. all answers are not correct
2053. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- A. Valter-Braun's gastroenterostomy
 - B. not saved after any operation
 - C. resection by Bilrot II
 - D. * resection by Bil'rot I
 - E. saved after all transferred operations
2054. After what operation innervation of pyloric department of stomach is saved
- A. * selective vagotomy
 - B. barrel vagotomy
 - C. selective proximal vagotomy
 - D. at all transferred

- E. not saved after all operations
2055. An anal itch is a sign of:
- A. Insufficiency of sphincter of anus
 - B. Mycotic lesion of skin of coccyx
 - C. The hidden diabetes
 - D. Intestinal worm invasion
 - E. * All of answers are correct
2056. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
2057. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
2058. At colonoscopy for a patient 60 years old was remoted the polypus of sigmoid bowel in the distance 35 cm from anus. Histologically was found a microinvasive cancer an it apex. On it basis it is not found the cancer's cell. What is tactic of doctor?
- A. * Repeated colonoscopies every 3 months
 - B. Radial therapy
 - C. The resection of sigmoid bowel
 - D. Chemotherapy
 - E. Nothing
2059. At Crohn disease is damaged
- A. * all parts of gastrointestinal tract
 - B. only small intestine
 - C. only colon
 - D. only rectum
 - E. only sigmoid bowel
2060. At III stage blood loss at the bleeding ulcer the patient loses
- A. over 1000 ml blood
 - B. * over 2000 ml blood
 - C. over 500 ml blood
 - D. over 2500 ml blood
 - E. over 1500 ml blood
2061. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
2062. At the duodenum ulceroperation of choice is

- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
2063. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
2064. At the III item of activity hemostasis and III stage blood loss from the I – III stage blood loss indicated
- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
2065. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
2066. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
2067. At what degrees of unspecific ulcerative colitis is indicated hormonal preparations
- A. * heavy degrees
 - B. easy degrees
 - C. middle degrees
 - D. to chronic
 - E. chronic relapse
2068. At what disease could be histologically unspecific granuloma
- A. * Crohn disease
 - B. unspecific ulcerative colitis
 - C. cancer of rectum
 - D. poliposis
 - E. diverticulosis
2069. Bergman's sign is characteristic for
- A. * bleeding ulcer
 - B. for cicatricial-ulcerous pylorostenosis
 - C. perforated ulcers

- D. penetratration ulcers
 - E. malignization ulcers
2070. Berhtein's sing characteristic for
- A. * perforeted ulcers
 - B. bleeding ulcer
 - C. penetratrated ulcers
 - D. malignizated ulcers
 - E. for cicatrical-ulcerous pylorostenosis
2071. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
2072. Blood loos I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100
 - E. Ht 44-40, Hb 110 /?
2073. Blood loos II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
2074. Blood loos III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
2075. Characteristic complications of unspecific ulcerative colitis
- A. * bleeding, perforation, toxic dilatation of bowels
 - B. pancreatitis, cholecystitis
 - C. adhesion
 - D. perforation, penetration
 - E. peritonitis
2076. Classification of unspecific ulcerative colitis by its clinical management
- A. * acute, fulminating, chronic continuous and relapse
 - B. proctitis, proctosygmoiditis
 - C. left-side and total colitis
 - D. easy, middle, heavy
 - E. easy, heavy
2077. Classification of unspecific ulcerative colitis by its distribution
- A. * proctitis, proctosygmoiditis, left-side and total colitis

- B. acute, quick
 - C. chronic continuous and relapse
 - D. left-side and total colitis
 - E. easy, middle, heavy
2078. Clinical symptoms of the unspecific ulcerative colitis
- A. * pain, diarrhea, loss of weight
 - B. icterus, hydroperitoneum
 - C. increasing of liver and spleen
 - D. pain, obesity
 - E. pain, icterus, hydroperitoneum
2079. De-Cerven's sign is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
2080. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
2081. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is
- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
2082. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
2083. During rectoscopy is found the endoscopic symptom of "roadway". What disease?
- A. Unspecific ulcerative colitis
 - B. * Crohn disease of rectum
 - C. Dysentery
 - D. Salmonellosis
 - E. Syndrome of irritation of colon
2084. Eleker's sign is characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis

2085. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- tumours
 - ulcers
 - bleeding polypuses
 - erosions
 - * changes of evacuation function
2086. Features of surgical treatment of anaerobic paraproctitis
- * opening by wide cuts
 - ligature method
 - operation of Gabriel
 - an operation by Rizhik-Bobroviy
 - by Milligan-Morgan and Gabriel.
2087. For bleeding ulcer characteristic sign is
- * pain in an epigastrium;
 - knife-like pain;
 - signs irritation of peritoneum;
 - presence fresh blood in incandescence
 - melena;
2088. For bleeding ulcer characteristically
- * melena
 - tension the muscles of front abdominal wall
 - Spazarskiy's sing
 - sickliness the back vault of vagina
 - irradiation pain in a shoulder or shoulder-blade
2089. For motion of disease ulcerous illness of middle weight characteristically
- development of complications
 - * relapses 1-2 times per a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year
 - 3 and anymore relapses on a year
2090. For perforeted ulcer characteristically
- * tension the muscles of front abdominal wall
 - melena
 - vomiting by coffee-grounds
 - high intestinal impassability
 - vomiting stagnant gastric maintenance
2091. For pneumoperitoneum is characteristic symptom
- * Zhober's;
 - Khelatid's;
 - Podlag's;
 - Vigats's;
 - Udin's.
2092. For the heavy flow of ulcerous illness characteristically
- 2 and anymore relapses on a year
 - * 3 and anymore relapses on a year
 - 4 and anymore relapses on a year
 - 5 and more relapses are on a year

- E. 6 and more relapses are on a year
2093. For the heavy flow of ulcerous illness characteristically
- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days
2094. For what disease characteristic symptom of "water-pipe"?
- A. * Unspecific ulcerative colitis.
 - B. Crohn disease.
 - C. Pseudopoliposis.
 - D. Diverticulosis.
 - E. Spastic colitis.
2095. For what disease is characteristic symptom of "roadway"?
- A. * Crohn disease.
 - B. Amebioz.
 - C. Spastic colitis.
 - D. Cancer of large intestine.
 - E. Unspecific ulcerative colitis.
2096. For which diseases of large intestine characteristic symptom of the "shot target"
- A. * unspecific ulcerative colitis
 - B. diverticulosis
 - C. poliposis
 - D. cancer
 - E. Crohn disease
2097. From what department degestive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
2098. From what part of gastrointestinal tract is bleeding when presence of cherry-colour blood in the stool
- A. * colon
 - B. stomach and duodenum
 - C. rectum
 - D. duodenum
 - E. small intestine
2099. From what part of intestine is most often begins unspecific ulcerative colitis?
- A. * From the rectum.
 - B. From the ascending part of colon.
 - C. From the transverse part of colon.
 - D. From the descent part of colon.
 - E. From the terminal part of small intestine.
2100. From what tissue anal papilla are formed from?

- A. From epithelial tissue
 - B. * From connective tissue
 - C. From lymphoid tissue
 - D. From muscular tissue
 - E. From mucus
2101. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
2102. Haemorrhoid's nodes do not fall out at
- A. * I stage
 - B. II stage
 - C. III stage
 - D. External nodes
 - E. Internal nodes
2103. Haemorrhoid's nodes fall out and not replaced
- A. * III stage
 - B. I stage
 - C. II stage
 - D. External nodes
 - E. Internal nodes
2104. Haemorrhoid's nodes fall out during defecation and replaced
- A. * II stage
 - B. I stage
 - C. III stage
 - D. External nodes
 - E. Internal nodes
2105. Haemorrhoidectomy is complicated by cicatricial stricture of rectum. What next operation is indicated?
- A. * dosed sphincterotomy with sewing mucus of rectum to the perianal skin
 - B. anal bougienage
 - C. dosed sphincterotomy
 - D. hemorrhoidectomy
 - E. colostomy
2106. Haemorrhoids complicated by bleeding is indication for
- A. * urgent operation
 - B. planned operation
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. therapy not performed
2107. Haemorrhoids complicated by thrombosis is indication for
- A. * urgent operation
 - B. planned operation
 - C. conservative therapy
 - D. physiotherapeutic procedure

- E. therapy not performed
2108. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
2109. How many physiology flexures has rectum?
- A. 1
 - B. * 2
 - C. 3
 - D. 4
 - E. 5
2110. Hyperbaric oxygenation in a postoperative period is used at:
- A. * Anaerobic paraproctitis
 - B. To the anal fissure
 - C. Epithelial coccygeal
 - D. Haemorrhoids
 - E. Cancer of rectum
2111. In the perianal area patient has the slight swelling, red skins, soft infiltrate. What is the diagnosis?
- A. * Acute paraproctitis
 - B. Anal fissure
 - C. Haemorrhoids
 - D. Cancer of rectum
 - E. Proctopolypus
2112. In what amount of physiologic solution does dissolve medicines for medical micro-enemas?
- A. * 80 ml.
 - B. 200 ml.
 - C. 250 ml.
 - D. 300 ml
 - E. 400 ml
2113. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?
- A. small curvature of stomach;
 - B. back wall of stomach, nearer to small curvature;
 - C. large curvature of stomach
 - D. * cardiac part of stomach;
 - E. pylorus.
2114. In what vein is a venous outflow carried out in from a stomach?
- A. * V. Portae;
 - B. V. odd;
 - C. V. pair;
 - D. V. overhead hollow;
 - E. V. lower hollow;
2115. Indication to the operation of haemorrhoidectomy is

- A. * repeated thrombosis of haemorrhoids nodes
 - B. portal hypertension
 - C. second stage of non-complicated haemorrhoids
 - D. first stage of non-complicated haemorrhoids
 - E. itch
2116. Indication to the operation of haemorrhoidectomy is
- A. * thrombosis of haemorrhoids nodes
 - B. portal hypertension
 - C. pain
 - D. itch
 - E. discomfort
2117. Large ulcer is an ulcer measuring
- A. 1-4 cm
 - B. 1-3 cm
 - C. 3-5 cm
 - D. 2- 6 cm
 - E. * 2-3 cm
2118. Little ulcer it is an ulcer measuring
- A. * to 0,5 cm
 - B. 0,5-1 cm
 - C. to 1,0 cm
 - D. 3 to 1,5 cm
 - E. 0,5-1,5 cm
2119. Mark the disease of colon, which characterised by such complications as bleeding, formation of stricture, perforation, toxic dilatation, malignancy:
- A. Haemorrhoids
 - B. Fissure
 - C. Polipus
 - D. * Unspecific ulcerative colitis
 - E. Paraproctitis
2120. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
2121. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood
2122. Melena is a characteristic sign
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorus stenosis
 - C. perforated ulcers
 - D. penetration ulcers

- E. malignization ulcers
2123. Method of surgical treatment of acute submucous proctitis
- A. * by Rizhik-Bobroviy
 - B. opening of abscess by a radial cut
 - C. ligature method
 - D. Operation of Rizhikh-1.
 - E. by Milligan-Morgan.
2124. Method of surgical treatment of anal fissure
- A. * cutting of fissure
 - B. by Milligan-Morgan.
 - C. suturing of fissure
 - D. by Kyumel-Zerenin.
 - E. by Kenyu-Milse.
2125. Method of surgical treatment of haemorrhoids
- A. * by Milligan-Morgan.
 - B. by Bebkok
 - C. by Narat
 - D. by Gabriel.
 - E. Operation of Blinnichev.
2126. Most informing method at a bleeding ulcer
- A. survey sciagraphy the organs of abdominal region
 - B. * EFGDS
 - C. sciagraphy the stomach with contrasting
 - D. Sonography
 - E. Laparoscopy
2127. Name classic complications of ulcerous illness
- A. bleeding, pylorostenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pylorostenosis, malignization
 - C. malignization, pylorostenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
2128. On irrigography is found the symptoms of «water-pipe», «shot through target». What is diagnosis?
- A. * Unspecific ulcerative colitis
 - B. Crohn disease
 - C. Dysentery
 - D. Salmonellosis
 - E. Food toxicoinfection
2129. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
2130. Operation which performed after the cutting of fissure of rectum
- A. * by Gabriel

- B. by Milligan-Morgan.
 - C. sphincterotomy
 - D. by Kyumel-Zerenin.
 - E. by Kenyu-Miles.
2131. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
2132. Patient 27 years old has stomach-ache, liquid stool up to 10 times per days with mucus and blood, weakness, weight lost. On the irrigigraphy is narrowing of transvers colon. What diagnosis?
- A. Dysentery.
 - B. Polypus of small intestine.
 - C. * Cancer of transvers colon.
 - D. Spastic colitis.
 - E. Unspecific ulcerative colitis
2133. Patient 50 years old has permanent pain in the anus, frequent defecation with blood, lost of appetite, weight lost, weakness. What examination is prescribed?
- A. * A biopsy with histological examination
 - B. Radio-active scan
 - C. Selective angiography
 - D. Doplerography
 - E. Sonography
2134. Patient 59 years old has suspicion of the tumour of ascending part of colon. What method of examination is the best?
- A. * A colonoscopy with a biopsy
 - B. Irrigography
 - C. Survey sciagraphy
 - D. Sonography
 - E. Endoscopy
2135. Patient 72 years old has acute pain in the left half of abdomeb, nausea, delay of stool and gases. He is ill 6 hours. No mucus and blood in stool, not weight lost . Pulse 84 per 1 min. Peristaltic noises is increased periodically. On the X-Ray of organs of abdominal region is present the Kloyber's cup in the left half of abdomen. What diagnosis ?
- A. * Invagination of sygmoid bowel
 - B. Crohn disease
 - C. Poliposis
 - D. Unspecific ulcerative colitis
 - E. Diverticulosis
2136. Patient during the act of defecation has paine in anal channel, red blood in the stool. What disease?
- A. * Anal fissure
 - B. Haemorrhoids
 - C. Acute paraproctitis
 - D. Cancer of rectum
 - E. Proctopolypus

2137. Patient has poliposis of right half of colon. What is treatment?
- A. * right hemicolectomy
 - B. conservative treatment
 - C. stoma
 - D. by Milligan-Morgan.
 - E. by Gabriel.
2138. Patient has a blood in the first portions of stool. What disease is possible?
- A. * Cancer of rectum.
 - B. Haemorrhoids.
 - C. Fissure.
 - D. Paraproctitis.
 - E. Fistula.
2139. Patient has a general weakness, presence of dark blood in the stool. At a rectoscopy on 11 cm from anus is found the circular narrowing of rectum. What diagnosis?
- A. * Cancer of rectum
 - B. Proctopolypus
 - C. Acute paraproctitis
 - D. Chronic paraproctitis
 - E. Acute proctitis
2140. Patient has a red blood at the end of defecation. What disease is possible?
- A. * Haemorrhoids and fissure of mucus of rectum.
 - B. Gastric and duodenal ulcers.
 - C. Cancer of rectum.
 - D. Paraproctitis.
 - E. Fistula.
2141. Patient has anal fissure of mucus of rectum with periodic pains. Pregnancy 16 weeks. Tactic of surgeon?
- A. * an operation - cutting of fissure after birth of child
 - B. an operation by Milligan-Morgan
 - C. an operation by Gabriel
 - D. cutting of fissure
 - E. an operation by Kenu-Miles
2142. Patient has anterior mucosal prolapse of rectum of the III stage and complete prolapse of uterus. What operation is indicated?
- A. * By Kumel-Zerenin, amputation of uterus
 - B. by Kenu-Miles
 - C. by Tartu
 - D. by Rizhikh-1
 - E. by Milligan-Morgan
2143. Patient has bleeding from a colon as a result of complication of unspecific ulcerative colitis. What operation is indicated?
- A. * proctocolectomy
 - B. suturing of bleeding area of bowel
 - C. resection of bowel
 - D. colectomy
 - E. colostomy

2144. Patient has cicatricial narrowing of sigmoid bowel with intestinal obstruction as complication of unspecific ulcerative colitis. What operation is indicated?
- A. * proctocolectomy
 - B. resection of the narrowed area of bowel
 - C. colectomy
 - D. colostomy
 - E. Operation by Kenyu-Miles.
2145. Patient has diarrhea up to 25-30 times per days with blood, has weight lost, general weakness, periodic stomach-ache. He is ill during 1,5 month. What diagnosis?
- A. * Unspecific ulcerative colitis
 - B. colitis
 - C. Pseudopoliposis
 - D. Diverticulosis
 - E. Spastic colitis
2146. Patient has intersphincteric fistula and external haemorrhoids. What operation is performed?
- A. * By Milligan-Morgan and Gabriel.
 - B. By Milligan-Morgan.
 - C. By Gabriel.
 - D. Operation of Blinnichev.
 - E. Operation of Rizhikh-1.
2147. Patient has long-term ulcer of rectum. In anamnesis white plague. What is previous diagnosis?
- A. * tuberculosis of rectum
 - B. haemorrhoids
 - C. paraproctitis
 - D. fistula
 - E. fissure of rectum
2148. Patient has melena. What is the source of bleeding?
- A. * Stomach and duodenum.
 - B. Rectum.
 - C. Colon.
 - D. Small intestine.
 - E. Sigmoid bowel.
2149. Patient has paraproctitis and fistula in pararectal area. Also it is present fruzi of actinomicete. What diagnosis?
- A. * actinomicosis
 - B. haemorrhoids
 - C. paraproctitis
 - D. fistula
 - E. fissure of rectum
2150. Patient has perforation of colon as complication of unspecific ulcerative colitis. What operation is indicated
- A. * proctocolectomy
 - B. suturing of the perforative hole
 - C. resection of area of bowel
 - D. colectomy
 - E. colostomy

2151. Patient has poliposis of left half of colon. What is treatment?
- A. * left-side hemicolectomy
 - B. conservative treatment
 - C. stoma
 - D. by Milligan-Morgan.
 - E. by Gabriel.
2152. Patient has polypus of sigmoid colon with signs of malignancy. What treatment?
- A. * resection of area of bowel with polypus
 - B. electroscission
 - C. cutting of polypus
 - D. criodestruction
 - E. conservative
2153. Patient has polypus on wide leg on 15 sm from anus. What treatment?
- A. * removal of polypus by laparotomy, rectotomy
 - B. electroscission
 - C. conservative treatment
 - D. ligating
 - E. criodestruction
2154. Patient has proctopolypus on 15 sm from anus with the signs of mamalignancy
- A. * anterior resection of rectum
 - B. electroscission
 - C. cutting of polypus
 - D. criodestruction
 - E. conservative
2155. Patient has small sizes proctopolypus in sygmoid bowel and pregnancy 8 weeks. Tactic of surgeon?
- A. * it is removing polypus after birth of child
 - B. an operation by Milligan-Morgan
 - C. an operation by Gabriel
 - D. cutting of polypus during pregnancy
 - E. an operation by By Kenu-Miles
2156. Patient has swelling from an anal channel during the act of defecation, without paine, with fresh blood after defecation. Previous diagnosis?
- A. Anal fissure
 - B. * Haemorrhoids
 - C. Acute paraproctitis
 - D. Cancer of rectum
 - E. Proctopolypus
2157. Patient has the combined haemorrhoids and pregnancy 8 weeks. Tactic of surgeons?
- A. * it is performed operative treatment after birth of child
 - B. an operation by Gabriel
 - C. an operation by Milligan-Morgan
 - D. by Rizhikh-1
 - E. by Kenu-Miles
2158. Patient has the combined haemorrhoids with bleeding and pregnancy 9 weeks. Tactic of surgeon?
- A. * an operation by Milligan-Morgan

- B. an operation by Gabriel
 - C. it is performed operative treatment after birth of child
 - D. by Rizhikh-1
 - E. by Kenu-Miles
2159. Patient has the III stage anterior mucosal prolapse of rectum. What operation is indicated?
- A. * by Kyumel-Zerenin
 - B. by Kenyu-Miles
 - C. by Milligan-Morgan
 - D. by Gabriel
 - E. by Rizhikh-1
2160. Patient has toxic dilatation as complication of unspecific ulcerative colitis. What operation is indicated?
- A. * proctocolectomy
 - B. resection of dilatated area of colon
 - C. colectomy
 - D. colostomy
 - E. an operation by Kenu-Miles
2161. Patient has ulcer of rectum by duration near two months. The reaction of Wassermann is positive. Previous diagnosis
- A. * venereal lymphogranuloma
 - B. haemorrhoids
 - C. paraproctitis
 - D. fistula
 - E. fissure of rectum
2162. Patients with haemorrhoids has blood in the stool
- A. * during defecation
 - B. before defecation
 - C. after defecation
 - D. constantly
 - E. never
2163. Presence of blood in the stool is characteristically for:
- A. haemorrhoids
 - B. UUC
 - C. Cancer of colon
 - D. Fissures of anus
 - E. * All of answers are correct
2164. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
2165. Reasons of origin of fissures of rectum
- A. * constipations, diarrhoea
 - B. cancer of rectum
 - C. portal hypertension
 - D. ulcerous disease

- E. varicose disease
2166. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
2167. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
2168. Roentgenological signs of unspecific ulcerative colitis
- A. * symptom of "water-pipe"
 - B. symptom of "niche"
 - C. defect of filling
 - D. symptom of «roadway»
 - E. bowls of Kloyber
2169. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation
 - D. resection
 - E. does not belong to any group
2170. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
2171. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
2172. Small amount of blood in stool could has patients with:
- A. * Poliposis
 - B. Colitis
 - C. Pancreatitis
 - D. Peptic ulcer disease
 - E. Appendicitis
2173. Solution of atropine sulfate is used, before operation with a purpose
- A. * of block of peripheral M-cholinoreceptors
 - B. providing of the adequate anaesthetizing

- C. increase of vagus activity
 - D. increasing of frequency of pulse
 - E. decreasing of frequency of pulse
2174. Surgical treatment by the method of cutting of fistula of rectum with cutting of skin and subcutaneous tissue in the type of triangle
- A. * for Gabriel
 - B. for Rizhik-Bobroviy
 - C. ligature method
 - D. by Milligan-Morgan.
 - E. Operation of Rizhikh-1.
2175. Surgical treatment of paraproctitis by the method of desection of fistula of rectum with cutting of skin and mucus in the type of triangle
- A. * by Rizhik-Bobrov
 - B. by Gabriel
 - C. ligature method
 - D. by Milligan-Morgan.
 - E. Operation of Rizhikh-1.
2176. The medicinal “constipation” is used at operations
- A. * on a rectum
 - B. on a small intestine
 - C. on a liver
 - D. on a stomach
 - E. on a duodenum
2177. The nosotropic mechanisms bleedingness at ulcerous illness is
- A. * all answers are correct.
 - B. permanent hyperemia all system of stomach
 - C. different degree dystrophy of superficial layers the mucus shell
 - D. accumulation the central mucopolysaccharides
 - E. hypoplastic, dystrophic processes
2178. The secretory function stomach is carried out the next membrane of stomach
- A. * mucous membrane
 - B. internal muscular layer
 - C. serosal
 - D. mucous submembrane
 - E. external layer
2179. To absolute indication to operative interference at ulcerous illness does not belong
- A. * scarry-ulcerous stenosis
 - B. perforation of ulcer
 - C. profuse bleeding
 - D. diameter ulcer a more than 3 cm
 - E. bleeding what does not stopped with conservative
2180. To the gastric – intestinal bleeding of un ulcerous etiology belong
- A. * Mallory-Weiss syndrome;
 - B. hemorrhagic erosive gastritis;
 - C. diseases by Randyu – Oslera – Vebera;
 - D. Menetrie's sing;
 - E. all answers are correct.

2181. Udin's sign at a perforated ulcer is
- * feeling at palpation show the gases which penetrate through the perforated opening
 - dulling perforated sound in the lateral departments of stomach
 - disappearance of hepatic dullness
 - irradiation pain in a shoulder or shoulder-blade
 - sickliness the back vault of vagina
2182. Vomiting coffee-grounds is a characteristic sign
- * bleeding ulcer
 - penetrative ulcers
 - perforated ulcers
 - malignized ulcers
 - for cicatrical-ulcerous pylorus stenosis
2183. Vomiting what arose up in 4-6 hours after eating characteristic for
- chronic alcoholic gastritis
 - cancer and ulcers of cardia
 - * pylorus ulcers
 - achalasia of gullet
 - ulcer and cancer the body of stomach
2184. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodnum?
- * Tarry excrement
 - Presence on the formed excrement of strokes of red blood
 - Littlechanged blood in an excrement
 - Excrement of the raspberry colouring with the admixtures of mucus
 - Acholic excrement
2185. What complication of unspecific ulcerative colitis is indication to urgent surgery?
- Malignizaciya
 - Bleeding
 - Acute toxic dilatation
 - * Perforation
 - Diarrhea
2186. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- perforation
 - perforation + bleeding
 - pylorus stenosis
 - malignization + penetration
 - * bleeding
2187. What disease is damage the superficial layer of wall of bowel
- * unspecific ulcerative colitis
 - Crohn disease
 - diverticulosis
 - poliposis
 - haemorrhoids
2188. What does mean a term "haemorrhoids"?
- Varicose enlargement of haemorrhoidal veins

- B. Spasm of anal sphincter
 - C. * Bleeding
 - D. Inflammation of paraperctal tissue
 - E. Inflammation of anal channel
2189. What does subserve to development of haemorrhoidal thrombosis?
- A. Constipation
 - B. Diarrhea
 - C. * Spasm of sphincter
 - D. Criptitis
 - E. Papillitis
2190. What drug has purgative action?
- A. * Fenolftalein.
 - B. Aspirine.
 - C. Ftalasol.
 - D. Proserin.
 - E. Biphicol.
2191. What drug is decreased frequency of defecation?
- A. * Immodium.
 - B. Kofeol.
 - C. Karbolen.
 - D. Sulfate of magnesium.
 - E. Prozerin.
2192. What drug is used for fistulography?
- A. * Iodlipol.
 - B. Bilignost.
 - C. Verografin.
 - D. Sulfate of barium.
 - E. Methylene bluing.
2193. What drug is used for irrihography?
- A. * Sulfate of barium.
 - B. Cardiotrast.
 - C. Bilignost.
 - D. Iodlipol.
 - E. Methylene.
2194. What drugs is used to decrease meteorism?
- A. * Espumisan.
 - B. Norsulfazolum.
 - C. Vaseline oil.
 - D. Sulfate of magnesium.
 - E. Prozerin.
2195. What enema is used at intestinal obstruction?
- A. * Siphon enema.
 - B. Cleansing.
 - C. Microenema.
 - D. Purgative.
 - E. Does not used.

2196. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - Tarry darkly-cherry chair
2197. What form of unspecific ulcerative colitis is most dangerous?
- * Fulminating.
 - Acute.
 - Chronic recurrent.
 - Chronic continuous.
 - Recurrent.
2198. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- * trichopol
 - Licviroton
 - oxiferiskorbon
 - atropine
 - pirinzsipin
2199. What from the transferred operations does not belong to organ protective
- trunk vagotomy
 - * resection by Bilrot II
 - selective vagotomy
 - selective proximal vagotomy
 - the all transferred does not belong
2200. What from the transferred operations on the stomach organ protective is
- * selective proximal vagotomy
 - resection by Bilrot I
 - resection by Bilrot II
 - gastrectomy
 - all are transferred
2201. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- * ACTH
 - epidermal factor of growth
 - prostaglandin E
 - estrogens
 - STH
2202. What instrumental examination is performed at jamming of haemorrhoidal nodes
- * examination is not performed
 - rectometer
 - irrigoscopy
 - irrigography
 - colonoscopy
2203. What is from listed has hemostatic effect
- * blood
 - red corpuscles

- C. Haemodesum
 - D. physiologic solution
 - E. glucose
2204. What is indication to surgical treatment of anal fissure
- A. * chronic passing
 - B. acute passing
 - C. perforation
 - D. bleeding
 - E. malignancy
2205. What is location of fistula chanal at intersphincteric paraproctitis?
- A. * Between mucus and sphincter
 - B. Passes through sphincter
 - C. Located after sphincter
 - D. All are true
 - E. All are false
2206. What is location of fistula chanal at the transsphincteric paraproctitis?
- A. Between mucus and sphincter
 - B. * Passes through sphincter
 - C. Located after sphincter
 - D. All is true
 - E. All is false
2207. What is the basic examination of patients with disease of rectosigmoid area?
- A. * Rectoromanoscopy.
 - B. X-Ray.
 - C. Sonography.
 - D. Digital examination of rectum.
 - E. Irrigography.
2208. What is the complication after haemorrhoidectomy
- A. * stricture of anus
 - B. proctosigmoiditis
 - C. cancer of rectum
 - D. intestinal obstruction
 - E. peritonitis
2209. What is the complication of surgical treatment of anal fissures
- A. * insufficiency of anal sphincter
 - B. anterior mucosal prolapse of rectum
 - C. cancer of rectum
 - D. malignancy
 - E. Crohn disease
2210. What is the contra-indication for the colproctectomy at a unspecific ulcerative colitis:
- A. * A perforation of colon bowel
 - B. Acute toxic dilatation
 - C. Bleeding
 - D. Malignizaciya
 - E. Stenosis
2211. What is the operation of choice at a unspecific ulcerative colitis:

- A. Resection of rectum
 - B. * proctocolectomy
 - C. Left-side hemicolectomy
 - D. Right-side hemicolectomy
 - E. Resection of sigmoid bowel
2212. What is the operation of choice at the unspecific ulcerative colitis?
- A. * Proctocolonectomy with ileostomy.
 - B. Bypassed loop anastomosis.
 - C. Subtotal colectomy.
 - D. Resection of colon.
 - E. Application of colostomy.
2213. What is the reason of origin of acute proctitis?
- A. Trauma of rectum
 - B. * Micro-injury of rectum mucus with the damage of crypt
 - C. Proctopolypus
 - D. Anal fissure
 - E. Cancer of rectum
2214. What is the reason of relapse of proctitis?
- A. An operation is done not enough radically
 - B. Infection of wound
 - C. * The internal opening is not removed
 - D. Anaerobic infection
 - E. Wrong conservative treatment
2215. What is the typical localization of anal fissure
- A. * on 6 hr.
 - B. on 12 hr.
 - C. on 3 hr.
 - D. on 9 hr.
 - E. on 2 hr.
2216. What kind of enemas is applied at preparation a patient to the operation?
- A. * Cleaning.
 - B. Siphon.
 - C. Microenema.
 - D. Purgative.
 - E. Does not used.
2217. What layers of the bowel are damaged at Crohn disease
- A. * all of layers
 - B. mucus
 - C. submucous and muscle
 - D. submucous
 - E. muscle
2218. What localization of ulcer is most characteristic for the patients of elderly and senile age
- A. * cardial department of stomach
 - B. overhead third of gullet
 - C. lower third of gullet
 - D. bulb of duodenum
 - E. small curvature

2219. What medicines is used for increasing the frequency of defecation ?
- A. * Prozerin.
 - B. Sulfate of magnesium.
 - C. Immodium.
 - D. Karbolen.
 - E. Digestal.
2220. What medicines is used for treatment of unspecific ulcerative colitis?
- A. * Sulfasalazinum.
 - B. Ampicillin.
 - C. Nospanum, papaverini.
 - D. Aspirine.
 - E. Furazolidonum.
2221. What method diagnostics hte ulcerous illness most informing
- A. * esophagogastroduodenoscopy
 - B. analysis of excrement on the hidden blood
 - C. X-ray
 - D. global analysis of blood
 - E. research of gastric secretion
2222. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- A. On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - B. On blood volume
 - C. * On an arteriotony, pulse, state of patient
 - D. On a globular volume
 - E. On the level of thrombocytes
2223. What method of examination is used to confirm the diagnosis of Crohn disease?
- A. * A biopsy is with histological examination
 - B. CT
 - C. Sciagraphy
 - D. Sonography
 - E. Rectoscoopy
2224. What method of operative treatment is used at the I - IV stage of complicated extrasphincteric fistula?
- A. * Ligature method.
 - B. Rizhikh-1.
 - C. Rizhikh-2.
 - D. Gabriel.
 - E. Cutting and suturing of fistula.
2225. What operation is performed at a acute shoe-shaped paraproctitis ?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2226. What operation is performed at an anal fissure?
- A. * Cutting of fissure with sphincterotomy.

- B. Suturing of fissure.
 - C. Operation of Milligan-Morgan.
 - D. Operation of Gabriel.
 - E. Operation of Nobl.
2227. What operation is performed at haemorrhoids complicated by bleeding
- A. * urgent
 - B. planned
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. not performed
2228. What operation is performed at haemorrhoids complicated by trombosis
- A. * urgent
 - B. planned
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. not performed
2229. What operation is performed at haemorrhoids?
- A. * Operation of Milligan-Morgan.
 - B. Rizhikh-1.
 - C. Rizhikh-2.
 - D. Operation of Gabriel.
 - E. Operation of Blinnichev.
2230. What operation is performed at patient with fissure and polypus?
- A. * Cutting of fissure with polypectomy.
 - B. Cutting of fissure.
 - C. By Milligan-Morgan.
 - D. By Gabriel.
 - E. Operation of Blinnichev.
2231. What operation is performed at rectocaele?
- A. * by Kumel-Zerenin.
 - B. Kenu-Miles.
 - C. Milligan-Morgan.
 - D. Gabriel.
 - E. Operation of Blinnichev.
2232. What operation is performed at the acute subcutaneous paraproctitis?
- A. Opening of abscess with suturing
 - B. * Cutting of abscess (by Gabriel)
 - C. Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2233. What operation is performed at the Crohn disease?
- A. * A resection of bowel within the limits of healthy tissues.
 - B. Proctocolonectomy.
 - C. Subtotal colectomy.
 - D. Resection of large and small intestine.
 - E. Application of colostomy.

2234. What operation is performed at the extrasphincteric paraproctitis?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2235. What operation is performed at the ischiorectal paraproctitis?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2236. What operation is performed at the retrorectal paraproctitis?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2237. What operation is performed at the transsphincteric paraproctitis?
- A. Cutting of fistula chanal (by Gabriel)
 - B. * Cutting of fistula chanal with the partial suturing of bottom of wound
 - C. Cutting of fistula chanal with the ligature conduction
 - D. Cutting of fistula with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2238. What operation is radical at haemorrhoids?
- A. Suturing of haemorrhoidal nodes
 - B. Operation by Gabriel
 - C. * Operation by Milligan-Morgan
 - D. Sclerotherapy
 - E. Conservative treatment
2239. What operation is used for pararectal fistula?
- A. * Operation of Gabriel.
 - B. Operation of Milligan-Morgan.
 - C. Operation of Gagen-Torn.
 - D. Operation of Nobl.
 - E. A sphincterectomy.
2240. What part of colon is damaged by cancer most often:
- A. * Sigmoid colon
 - B. Caecum
 - C. Ascending part
 - D. Descending part
 - E. Rectum
2241. What part of intestine of most often is a pathological process localized in at Crohn disease?
- A. * Terminal part of small intestine.
 - B. Rectum.
 - C. Ascending part of colon bowel.
 - D. Lumbar part of colon bowel.

- E. Sigmoid bowel.
2242. What patients with poliposis of colon could has in stool:
- A. Nothing
 - B. * Small amount of blood
 - C. Undigested meal
 - D. Presence of part of polypuses
 - E. Melena
2243. What preparation does behave to blocker H2-retseptors?
- A. * tavegil
 - B. obzidan
 - C. hystdol
 - D. cerucal
 - E. oraza
2244. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
2245. What preparations, except for other properties, own yet and a bacteriostatic effect on Helicobacter pyloris
- A. * all are transferred preparations
 - B. Almagel
 - C. Vinylin
 - D. De-nol
 - E. Claritromycin
2246. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
2247. What stool has patients with poliposis of colon:
- A. Not changed
 - B. Constipations
 - C. * Diarrhea
 - D. White
 - E. Melena
2248. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
2249. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;

- B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
2250. What triad of symptoms is characteristic for an anal fissure
- A. * pain during defecation, spasm of sphincter, bleeding
 - B. pain before defecation, constipations, bleeding
 - C. diarrhea, bleeding, weight loss
 - D. anaemia, diarrhea, pain
 - E. bleeding, anaemia, diarrhea
2251. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia
2252. What volume of blood lost at a unspecific ulcerative colitis does consider complications?
- A. To 50 ml
 - B. 50-100 ml
 - C. 150-200 ml
 - D. 200-300 ml
 - E. * 300 ml and more
2253. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforated ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pyloristensis
2254. Where are anal papilla usually located?
- A. In any area of anal channel
 - B. * Only above and lower from dentata line
 - C. In sygmoid colon
 - D. In any area of rectum
 - E. In perianal region
2255. Which preparation is used for treatment of unspecific ulcerative colitis
- A. * sulfasalasine
 - B. fenolftaleine
 - C. cerucal
 - D. analgin
 - E. ketanov
2256. With the purpose of preventing of anaerobic infection at surgical treatment of paraproctitis is used
- A. * hydrogen peroxide
 - B. furacilini
 - C. iodine solution
 - D. rivanol

- E. alcohol
2257. A frequent liquid stool is the first sign of:
- A. haemorrhoids
 - B. Fissures of anus
 - C. Proctosigmoiditis
 - D. * UUC
 - E. All of answers are correct
2258. A ligature method is used at treatment of paraproctitis
- A. * extrasphincteric fistula
 - B. acute submucous fistula
 - C. intersphincteric fistula
 - D. ischiorectal
 - E. retrorectal
2259. A patient 40 years old, suffered ulcerous diseases of stomach. Last 2 days the pain became less intensive, but weakness and dizziness were appeared. Rose from a bed and lost consciousness. Pale. There are insignificant pains in epigastrium. It is
- A. Combination perforation with bleeding
 - B. Perforation
 - C. Malignization of ulcer
 - D. Stenosis of ulcer
 - E. * Gastroenteric bleeding
2260. A patient after the electroscission of polypus of sigmoid bowel has bleeding. What is tactic?
- A. * it is performed hemostatic therapy
 - B. it is performed operative treatment
 - C. conservative treatment
 - D. laparotomy
 - E. laparoscopy
2261. A patient after the electroscission of polypus of sigmoid bowel has stomach-aches. What complication can be?
- A. * perforation
 - B. bleeding
 - C. malignancy
 - D. toxic dilatation
 - E. penetration
2262. A presence of mucus and pus in stool is characteristic for:
- A. Proctosigmoiditis
 - B. Cancer of rectum
 - C. UUC
 - D. All of answers are wrong
 - E. * All of answers are correct
2263. Absolute indication to operative treatment the ulcerous illness is
- A. heavy pain syndrome
 - B. * perforation of ulcer
 - C. relapses more than 2 one time per a year
 - D. ulcerous anamnesis more than 10 years
 - E. giant ulcers

2264. Absolute indication to operative treatment the ulcerous illness is
- * voluminous bleeding
 - callous ulcers
 - relapses more than 2 one time per a year
 - ulcerous anamnesis more than 10 years
 - heavy pain syndrome
2265. Absolute indication to operative treatment the ulcerous illness is
- ulcerous anamnesis more than 10 years
 - * bleeding what do not stopped with conservative
 - perforation ulcer in anamnesis
 - heavy pain syndrome
 - relapses more than 3 times per a year
2266. Absolute indication to operative treatment the ulcerous illness is
- relapses more than 2 one time per a year
 - ulcerous anamnesis more than 10 years
 - relapse ulcer after the resection of stomach
 - relapses more than 3 times per a year
 - * cicatricial-ulcerous stenosis of pylorus
2267. Absolute indication to operative treatment the ulcerous illness is
- relapses more than 2 one time per a year
 - * malignization ulcers
 - ulcerous anamnesis more than 10 years
 - heavy pain syndrome, proof heartburn
 - relapse ulcer after vagotomy
2268. Absolute sign of unstable hemostasis
- * profluvium blood from a vessel;
 - absence blood in a stomach and bulb of duodenum;
 - presence light blood and faltungs of blood in a stomach;
 - all answers are correct;
 - all answers are not correct
2269. After what operation at ulcerous illness the natural arcade of meal is saved on a digestive tract
- Valter-Braun's gastroenterostomy
 - not saved after any operation
 - resection by Bilrot II
 - * resection by Bil'rot I
 - saved after all transferred operations
2270. After what operation innervation of pyloric department of stomach is saved
- * selective vagotomy
 - barrel vagotomy
 - selective proximal vagotomy
 - at all transferred
 - not saved after all operations
2271. An anal itch is a sign of:
- Insufficiency of sphincter of anus
 - Mycotic lesion of skin of coccyx
 - The hidden diabetes

- D. Intestinal worm invasion
 - E. * All of answers are correct
2272. At a chronic paraproctitis is performed:
- A. * Planned operation.
 - B. Urgent operation.
 - C. Conservative treatment.
 - D. Emergency operation.
 - E. Nothing
2273. At beginning bleeding from an ulcer
- A. * pain diminishes
 - B. pain increases
 - C. there is knife-like pain
 - D. character of pain does not change
 - E. girdle pain
2274. At bleeding emergency operative interferences are executed
- A. * to 3 hours
 - B. to 1,5 hour
 - C. to 6 hours
 - D. to 8 hours
 - E. 6 – 12 hours
2275. At III stage blood loss at the bleeding ulcer the patient loses
- A. over 1000 ml blood
 - B. * over 2000 ml blood
 - C. over 500 ml blood
 - D. over 2500 ml blood
 - E. over 1500 ml blood
2276. At III stage blood loss at the bleeding ulcer the patient loses
- A. more than 25 % blood volume
 - B. * more than 30 % blood volume
 - C. more than 20 % blood volume
 - D. more than 15 % blood volume
 - E. more than 35 % blood volume
2277. At the duodenum ulceroperation of choice is
- A. * resection by Bilrot I
 - B. resection by Bilrot II
 - C. resection of duodenum
 - D. selective proximal vagotomy
 - E. sewing up of ulcer
2278. At the gastroenteric bleeding the middle degree of blood loss is diagnosed at next indexes
- A. Hb below 80 g/l
 - B. * Hb 80-100 g/l
 - C. red corpuscles below $2,5 \cdot 10^{12}/l$
 - D. red corpuscles of $3,5-4,0 \cdot 10^{12}/l$
 - E. Ht below 25%
2279. At the III item of activity hemostasis and III sage blood loss from the I – III sage blood loss indicated

- A. * urgent operations (6 – 12 hours);
 - B. emergency operations (to 3 hours);
 - C. exigent operations (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
2280. At the V item activity of hemostasis and at the recurrent bleeding of the I – III item of blood loss indicated
- A. * emergency operations (to 3 hours);
 - B. urgent operations (6 – 12 hours);
 - C. exigent (12 – 24 hours);
 - D. early deferred (24 – 72 hours);
 - E. planned operations (4 – 10 days)
2281. At ulcerous illness can a bleeding source be
- A. artery;
 - B. veins;
 - C. shallow vessels and ulcers;
 - D. all answers are not correct.
 - E. * all answers are correct
2282. At what disease could be histologically unspecific granuloma
- A. * Crohn disease
 - B. unspecific ulcerative colitis
 - C. cancer of rectum
 - D. poliposis
 - E. diverticulosis
2283. Bergman's sign is characteristic for
- A. * bleeding ulcer
 - B. for cicatricial-ulcerous pylorostenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
2284. Berhstein's sign characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
2285. Bleeding ulcer, complicated blood loss III stage degrees, requires
- A. * blood and its components transfusions
 - B. transfusion of salt solutions
 - C. transfusion of colloid solutions
 - D. transfusion of salt and colloid solutions
 - E. infusion therapy does not need
2286. Blood loss I stage characterized such indexes
- A. Ht 48-44, Hb 120
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 31-23, Hb 80
 - D. * Ht 38-32, Hb 100

- E. Ht 44-40, Hb 110 /?
2287. Blood loos II stage characterized
- A. * Ht 23 and below, Hb 50 and below
 - B. Ht 31-23, Hb 80
 - C. Ht 44-40, Hb 110
 - D. Ht 48-44, Hb 120
 - E. Ht 48-44, Hb 120
2288. Blood loos III stage characterized a degree such indexes
- A. * Ht 31-23, Hb 80
 - B. Ht 23 and below, Hb 50 and below
 - C. Ht 38-32, Hb 100
 - D. Ht 44-40, Hb 110
 - E. Ht 48-44, Hb 120
2289. Classification of haemorrhoids by the clinical passing
- A. * complicated, non-complicated
 - B. I, II, III stage
 - C. external, internal
 - D. I, II stage
 - E. all are false
2290. Classification of haemorrhoids by the degrees
- A. * I, II, III of the stage
 - B. external, internal
 - C. complicated, non-complicated
 - D. I, II stage
 - E. all are false
2291. Classification of haemorrhoids by the localizations
- A. * external, internal
 - B. I, II, III of the stage
 - C. complicated, non-complicated
 - D. I, II stage
 - E. all are false
2292. Classification of paraproctitis depending on activity of inflammatory process
- A. * acute, chronic
 - B. banal, specific, posttraumatic
 - C. perianal, submucous, ischiorectal, pelviorectal, retrorectal
 - D. front, lateral, back
 - E. Intersphincteric, transsphincteric, extrasphincteric
2293. Classification of paraproctitis depending on etiologic sign
- A. * banal, specific, posttraumatic
 - B. acute, chronic
 - C. perianal, submucos, ischiorectal, pelviorectal, retrorectal
 - D. front, lateral, back
 - E. Intersphincteric, transsphincteric, extrasphincteric
2294. Classification of paraproctitis depending on fistula localisation
- A. * Intersphincteric, transsphincteric, extrasphincteric
 - B. banal, specific, posttraumatic

- C. acute, chronic
 - D. front, lateral, back
 - E. perianal, submucous, Ischiorectal, pelvirectal, retrorectal
2295. Classification of paraproctitis depending on localizations
- A. * perianal, submucos, Ischiorectal, pelvirectal, retrorectal
 - B. banal, specific, posttraumatic
 - C. acute, chronic
 - D. front, lateral, back
 - E. Intersphincteric, transsphincteric, extrasphincteric
2296. Clinical manifestation of acute paraproctitis
- A. * pain, high temperature
 - B. enterorrhagia
 - C. diarrhea
 - D. constipations
 - E. vomiting
2297. Complication of haemorrhoids
- A. * thromboses, bleeding, paraproctitis
 - B. Crohn disease
 - C. portal hypertension
 - D. perforation
 - E. malignancy
2298. Complication of surgical treatment of anal fissures
- A. * insufficiency of anal sphincter
 - B. perforation
 - C. cancer of rectum
 - D. malignancy
 - E. Crohn disease
2299. Contr-indication to the operation of haemorrhoidectomy is
- A. * portal hypertension
 - B. bleeding
 - C. repeated thrombosis
 - D. pain
 - E. itch
2300. De-Cerven's sign is characteristic for
- A. bleeding ulcer
 - B. * perforated ulcers
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
2301. Diet at bleeding gastric and duodenum ulcers
- A. * Meulengracht's
 - B. 1 by Pevznerom
 - C. 5 by Pevznerom
 - D. 15 by Pevznerom
 - E. 7 by Pevznerom
2302. Disappearance or diminishing the pain with beginning of bleeding from an ulcer is

- A. * Bergman's sign
 - B. Spazarskiy's sign
 - C. Mendel's sign
 - D. De Keven's sign
 - E. Eleker's sign
2303. Duration the period of primary shock at a perforated ulcer
- A. * 3-6 hours
 - B. 6-12 hours
 - C. 1-3 hours
 - D. 12-24 hours
 - E. 24-36 hours
2304. During rectoscopy is found the endoscopic symptom of "roadway". What disease?
- A. Unspecific ulcerative colitis
 - B. * Crohn disease of rectum
 - C. Dysentery
 - D. Salmonellosis
 - E. Syndrome of irritation of colon
2305. Eleker's sign is characteristic for
- A. * perforated ulcers
 - B. bleeding ulcer
 - C. penetrated ulcers
 - D. malignized ulcers
 - E. for cicatricial-ulcerous pylorostenosis
2306. Esophagogastroduodenoscopy can find out next changes in a stomach, except for
- A. tumours
 - B. ulcers
 - C. bleeding polypuses
 - D. erosions
 - E. * changes of evacuation function
2307. Features of surgical treatment of anaerobic paraproctitis
- A. * opening by wide cuts
 - B. ligature method
 - C. operation of Gabriel
 - D. an operation by Rizhik-Bobroviy
 - E. by Milligan-Morgan and Gabriel.
2308. For bleeding ulcer characteristic sign is
- A. * pain in an epigastrium;
 - B. knife-like pain;
 - C. signs irritation of peritoneum;
 - D. presence fresh blood in incandescence
 - E. melena;
2309. For bleeding ulcer characteristically
- A. * melena
 - B. tension the muscles of front abdominal wall
 - C. Spazarskiy's sign
 - D. sickliness the back vault of vagina
 - E. irradiation pain in a shoulder or shoulder-blade

2310. For motion of disease ulcerous illness of middle weight characteristically
- A. development of complications
 - B. * relapses 1-2 times per a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 3 and anymore relapses on a year
2311. For perforated ulcer characteristically
- A. * tension the muscles of front abdominal wall
 - B. melena
 - C. vomiting by coffee-grounds
 - D. high intestinal impassability
 - E. vomiting stagnant gastric maintenance
2312. For pneumoperitoneum is characteristic symptom
- A. * Zhober's;
 - B. Khelatid's;
 - C. Podlag's;
 - D. Vigats's;
 - E. Udin's.
2313. For the heavy flow of ulcerous illness characteristically
- A. 2 and anymore relapses on a year
 - B. * 3 and anymore relapses on a year
 - C. 4 and anymore relapses on a year
 - D. 5 and more relapses are on a year
 - E. 6 and more relapses are on a year
2314. For the heavy flow of ulcerous illness characteristically
- A. * development of complications
 - B. seasonal exacerbation more not frequent 1-2 times per a year
 - C. 1-2 relapse on a year
 - D. liquid, but protracted exacerbation
 - E. exacerbation duration more than 10 days
2315. For what disease characteristic symptom of "water-pipe"?
- A. * Unspecific ulcerative colitis.
 - B. Crohn disease.
 - C. Pseudopoliposis.
 - D. Diverticulosis.
 - E. Spastic colitis.
2316. For what disease is characteristic symptom of "roadway"?
- A. * Crohn disease.
 - B. Amebioz.
 - C. Spastic colitis.
 - D. Cancer of large intestine.
 - E. Unspecific ulcerative colitis.
2317. For which diseases of large intestine characteristic symptom of the "shot target"?
- A. * unspecific ulcerative colitis
 - B. diverticulosis
 - C. poliposis

- D. cancer
 - E. Crohn disease
2318. From what department degestive tract developmentp more frequent than all the bleeding at the Mallory-Weiss syndrome
- A. gastric fundus
 - B. * cardial pert;
 - C. pyloric department;
 - D. from duodenal;
 - E. from a thick intestine
2319. From what part of gastrointestinal tract is bleeding when presence of cherry-colour blood in the stool
- A. * colon
 - B. stomach and duodenum
 - C. rectum
 - D. duodenum
 - E. small intestine
2320. From what part of intestine is most often begins unspecific ulcerative colitis?
- A. * From the rectum.
 - B. From the ascending part of colon.
 - C. From the transverse part of colon.
 - D. From the descent part of colon.
 - E. From the terminal part of small intestine.
2321. From what tissue anal papilla are formed from?
- A. From epithelial tissue
 - B. * From connective tissue
 - C. From limphoid tissue
 - D. From muscular tissue
 - E. From mucus
2322. Giant ulcer is an ulcer measuring
- A. over 4,5 cm
 - B. * over 3 cm
 - C. over 4 cm
 - D. over 5 cm
 - E. over 3,5 cm
2323. Haemorrhoid's nodes do not fall out at
- A. * I stage
 - B. II stage
 - C. III stage
 - D. External nodes
 - E. Internal nodes
2324. Haemorrhoid's nodes fall out and not replaced
- A. * III stage
 - B. I stage
 - C. II stage
 - D. External nodes
 - E. Internal nodes

2325. Haemorrhoid's nodes fall out during defecation and replaced
- A. * II stage
 - B. I stage
 - C. III stage
 - D. External nodes
 - E. Internal nodes
2326. Haemorrhoidectomy is complicated by cicatricial stricture of rectum. What next operation is indicated?
- A. * dosed sphincterotomy with sewing mucus of rectum to the perianal skin
 - B. anal bougienage
 - C. dosed sphincterotomy
 - D. hemorrhoidectomy
 - E. colostomy
2327. Haemorrhoids complicated by bleeding is indication for
- A. * urgent operation
 - B. planned operation
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. therapy not performed
2328. Haemorrhoids complicated by trombosis is indication for
- A. * urgent operation
 - B. planned operation
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. therapy not performed
2329. Hemobilia is
- A. * all answers are correct;
 - B. bleeding the bilious ways and liver;
 - C. bleeding the general bilious channel;
 - D. bloody clot in the big duodenal papilla;
 - E. all answers are not correct.
2330. How many physiology flexures has rectum?
- A. 1
 - B. * 2
 - C. 3
 - D. 4
 - E. 5
2331. Hyperbaric oxygenation in a postoperative period is used at:
- A. * Anaerobic paraproctitis
 - B. To the anal fissure
 - C. Epithelial coccygeal
 - D. Haemorrhoids
 - E. Cancer of rectum
2332. In the perianal area patient has the slight swelling, red skins, soft infiltrate. What is the diagnosis?
- A. * Acute paraproctitis
 - B. Anal fissure

- C. Haemorrhoids
- D. Cancer of rectum
- E. Proctopolypus

2333. In what amount of physiologic solution does dissolve medicines for medical micro-enemas?

- A. * 80 ml.
- B. 200 ml.
- C. 250 ml.
- D. 300 ml
- E. 400 ml

2334. In what area of stomach practically never is not origin of ulcers, or it is extraordinarily rarely?

- A. small curvature of stomach;
- B. back wall of stomach, nearer to small curvature;
- C. large curvature of stomach
- D. * cardiac part of stomach;
- E. pylorus.

2335. In what vein is a venous outflow carried out in from a stomach?

- A. * V. Portae;
- B. V. odd;
- C. V. pair;
- D. V. overhead hollow;
- E. V. lower hollow;

2336. Indication to the operation of haemorrhoidectomy is

- A. * thrombosis of haemorrhoids nodes
- B. portal hypertension
- C. pain
- D. itch
- E. discomfort

2337. Indication to the operation of haemorrhoidectomy is

- A. * repeated thrombosis of haemorrhoids nodes
- B. portal hypertension
- C. second stage of non-complicated haemorrhoids
- D. first stage of non-complicated haemorrhoids
- E. itch

2338. Large ulcer is an ulcer measuring

- A. 1-4 cm
- B. 1-3 cm
- C. 3-5 cm
- D. 2- 6 cm
- E. * 2-3 cm

2339. Little ulcer it is an ulcer measuring

- A. * to 0,5 cm
- B. 0,5-1 cm
- C. to 1,0 cm
- D. 3 to 1,5 cm
- E. 0,5-1,5 cm

2340. Mark the disease of colon, which characterised by such complications as bleeding, formation of stricture, perforation, toxic dilatation, malignancy:
- A. Haemorrhoids
 - B. Fissure
 - C. Polipus
 - D. * Unspecific ulcerative colitis
 - E. Paraproctitis
2341. Meets the most frequent localization bleeding the digestive tract is
- A. gullet;
 - B. stomach;
 - C. rectum;
 - D. * duodenum;
 - E. colon
2342. Melena is
- A. black designed chair
 - B. * black liquid tarry chair
 - C. a discoloured liquid excrement
 - D. foamy stinking emptying of black
 - E. an excrement designed veined blood
2343. Melena is a characteristic sign
- A. * bleeding ulcer
 - B. for cicatrical-ulcerous pylorus stenosis
 - C. perforated ulcers
 - D. penetration ulcers
 - E. malignization ulcers
2344. Method of surgical treatment of acute submucous paraproctitis
- A. * by Rizhik-Bobroviy
 - B. opening of abscess by a radial cut
 - C. ligature method
 - D. Operation of Rizhikh-1.
 - E. by Milligan-Morgan.
2345. Method of surgical treatment of anal fissure
- A. * cutting of fissure
 - B. by Milligan-Morgan.
 - C. suturing of fissure
 - D. by Kyumel-Zerenin.
 - E. by Kenyu-Milse.
2346. Method of surgical treatment of haemorrhoids
- A. * by Milligan-Morgan.
 - B. by Bebkok
 - C. by Narat
 - D. by Gabriel.
 - E. Operation of Blinnichev.
2347. Most informing method at a bleeding ulcer
- A. survey sciagraphy the organs of abdominal region
 - B. * EFGDS
 - C. sciagraphy the stomach with contrasting

- D. Sonography
 - E. Laparoscopy
2348. Name classic complications of ulcerous illness
- A. bleeding, pylorostenosis, second pancreatitis, malignization, perforation
 - B. * perforation, penetration, bleeding, pylorostenosis, malignization
 - C. malignization, pylorostenosis, penetration, anaemia, perforation
 - D. bleeding, perforation, second pancreatitis, anaemia, malignization
 - E. perforation, peritonitis, pancreatitis, bleeding, penetration
2349. On irrigography is found the symptoms of «water-pipe», «shot through target». What is diagnosis?
- A. * Unspecific ulcerative colitis
 - B. Crohn disease
 - C. Dysentery
 - D. Salmonellosis
 - E. Food toxicoinfection
2350. Operating access at operations on a stomach
- A. * Upper-middle laparotomy
 - B. Lower-middle laparotomy
 - C. Pararectum access
 - D. Volokovich-Dyakonov's access
 - E. Pfanenhtil's access
2351. Operation which performed after the cutting of fissure of rectum
- A. * by Gabriel
 - B. by Milligan-Morgan.
 - C. sphincterotomy
 - D. by Kyumel-Zerenin.
 - E. by Kenyu-Miles.
2352. Patient which the gastroenteric bleeding in house is necessary
- A. * To send a patient in surgical permanent establishment
 - B. To appoint rest, enter Cacl, vicasol
 - C. To wash a stomach, appoint a cold, rest of supervision
 - D. To send a patient in a therapeutic gastroenterology separation
 - E. A right answer absents
2353. Patient 27 years old has stomach-ache, liquid stool up to 10 times per days with mucus and blood, weakness, weight lost. On the irrigography is narrowing of transvers colon. What diagnosis?
- A. Dysentery.
 - B. Polypus of small intestine.
 - C. * Cancer of transvers colon.
 - D. Spastic colitis.
 - E. Unspecific ulcerative colitis
2354. Patient 50 years old has permanent pain in the anus, frequent defecation with blood, lost of appetite, weight lost, weakness. What examination is prescribed?
- A. * A biopsy with histological examination
 - B. Radio-active scan
 - C. Selective angiography
 - D. Doplerography

E. Sonography

2355. Patient 59 years old has suspicion of the tumour of ascending part of colon. What method of examination is the best?
- A. * A colonoscopy with a biopsy
 - B. Irrigography
 - C. Survey sciagraphy
 - D. Sonography
 - E. Endoscopy
2356. Patient 72 years old has acute pain in the left half of abdomen, nausea, delay of stool and gases. He is ill 6 hours. No mucus and blood in stool, not weight lost. Pulse 84 per 1 min. Peristaltic noises are increased periodically. On the X-Ray of organs of abdominal region is present the Kloyber's cup in the left half of abdomen. What diagnosis?
- A. * Invagination of sigmoid bowel
 - B. Crohn disease
 - C. Poliposis
 - D. Unspecific ulcerative colitis
 - E. Diverticulosis
2357. Patient during the act of defecation has pain in anal channel, red blood in the stool. What disease?
- A. * Anal fissure
 - B. Haemorrhoids
 - C. Acute paraproctitis
 - D. Cancer of rectum
 - E. Proctopolypus
2358. Patient has poliposis of right half of colon. What is treatment?
- A. * right hemicolectomy
 - B. conservative treatment
 - C. stoma
 - D. by Milligan-Morgan.
 - E. by Gabriel.
2359. Patient has a blood in the first portions of stool. What disease is possible?
- A. * Cancer of rectum.
 - B. Haemorrhoids.
 - C. Fissure.
 - D. Paraproctitis.
 - E. Fistula.
2360. Patient has a general weakness, presence of dark blood in the stool. At a rectoscopy on 11 cm from anus is found the circular narrowing of rectum. What diagnosis?
- A. * Cancer of rectum
 - B. Proctopolypus
 - C. Acute paraproctitis
 - D. Chronic paraproctitis
 - E. Acute proctitis
2361. Patient has a red blood at the end of defecation. What disease is possible?
- A. * Haemorrhoids and fissure of mucus of rectum.
 - B. Gastric and duodenal ulcers.
 - C. Cancer of rectum.

- D. Paraproctitis.
 - E. Fistula.
2362. Patient has anal fissure of mucus of rectum with periodic pains. Pregnancy 16 weeks. Tactic of surgeon?
- A. * an operation - cutting of fissure after birth of child
 - B. an operation by Milligan-Morgan
 - C. an operation by Gabriel
 - D. cutting of fissure
 - E. an operation by Kenu-Miles
2363. Patient has anterior mucosal prolapse of rectum of the III stage and complete prolapse of uterus. What operation is indicated?
- A. * By Kumel-Zerenin, amputation of uterus
 - B. by Kenu-Miles
 - C. by Tartu
 - D. by Rizhikh-1
 - E. by Milligan-Morgan
2364. Patient has bleeding from a colon as a result of complication of unspecific ulcerative colitis. What operation is indicated?
- A. * proctocolectomy
 - B. suturing of bleeding area of bowel
 - C. resection of bowel
 - D. colectomy
 - E. colostomy
2365. Patient has cicatricial narrowing of sigmoid bowel with intestinal obstruction as complication of unspecific ulcerative colitis. What operation is indicated?
- A. * proctocolectomy
 - B. resection of the narrowed area of bowel
 - C. colectomy
 - D. colostomy
 - E. Operation by Kenyu-Miles.
2366. Patient has diarrhea up to 25-30 times per days with blood, has weight lost, general weakness, periodic stomach-ache. He is ill during 1,5 month. What diagnosis?
- A. * Unspecific ulcerative colitis
 - B. colitis
 - C. Pseudopoliposis
 - D. Diverticulosis
 - E. Spastic colitis
2367. Patient has intersphincteric fistula and external haemorrhoids. What operation is performed?
- A. * By Milligan-Morgan and Gabriel.
 - B. By Milligan-Morgan.
 - C. By Gabriel.
 - D. Operation of Blinnichev.
 - E. Operation of Rizhikh-1.
2368. Patient has long-term ulcer of rectum. In anamnesis white plague. What is previous diagnosis?
- A. * tuberculosis of rectum
 - B. haemorrhoids

- C. proctitis
 - D. fistula
 - E. fissure of rectum
2369. Patient has melena. What is the source of bleeding?
- A. * Stomach and duodenum.
 - B. Rectum.
 - C. Colon.
 - D. Small intestine.
 - E. Sigmoid bowel.
2370. Patient has proctitis and fistula in perirectal area. Also it is present fruzi of actinomicete. What diagnosis?
- A. * actinomycosis
 - B. haemorrhoids
 - C. proctitis
 - D. fistula
 - E. fissure of rectum
2371. Patient has perforation of colon as complication of unspecific ulcerative colitis. What operation is indicated
- A. * proctocolectomy
 - B. suturing of the perforative hole
 - C. resection of area of bowel
 - D. colectomy
 - E. colostomy
2372. Patient has poliposis of left half of colon. What is treatment?
- A. * left-side hemicolectomy
 - B. conservative treatment
 - C. stoma
 - D. by Milligan-Morgan.
 - E. by Gabriel.
2373. Patient has polypus of sigmoid colon with signs of malignancy. What treatment?
- A. * resection of area of bowel with polypus
 - B. electroscission
 - C. cutting of polypus
 - D. criodestruction
 - E. conservative
2374. Patient has polypus on wide leg on 15 sm from anus. What treatment?
- A. * removal of polypus by laparotomy, rectotomy
 - B. electroscission
 - C. conservative treatment
 - D. ligating
 - E. criodestruction
2375. Patient has proctopolypus on 15 sm from anus with the signs of mamalignancy
- A. * anterior resection of rectum
 - B. electroscission
 - C. cutting of polypus
 - D. criodestruction
 - E. conservative

2376. Patient has small sizes proctopolypus in sygmoid bowel and pregnancy 8 weeks. Tactic of surgeon?
- * it is removing polypus after birth of child
 - an operation by Milligan-Morgan
 - an operation by Gabriel
 - cutting of polypus during pregnancy
 - an operation by By Kenu-Miles
2377. Patient has swelling from an anal channel during the act of defecation, without paine, with fresh blood after defecation. Previous diagnosis?
- Anal fissure
 - * Haemorrhoids
 - Acute paraproctitis
 - Cancer of rectum
 - Proctopolypus
2378. Patient has the combined haemorrhoids and pregnancy 8 weeks. Tactic of surgeons?
- * it is performed operative treatment after birth of child
 - an operation by Gabriel
 - an operation by Milligan-Morgan
 - by Rizhikh-1
 - by Kenu-Miles
2379. Patient has the combined haemorrhoids with bleeding and pregnancy 9 weeks. Tactic of surgeon?
- * an operation by Milligan-Morgan
 - an operation by Gabriel
 - it is performed operative treatment after birth of child
 - by Rizhikh-1
 - by Kenu-Miles
2380. Patient has the III stage anterior mucosal prolapse of rectum. What operation is indicated?
- * by Kyumel-Zerenin
 - by Kenyu-Miles
 - by Milligan-Morgan
 - by Gabriel
 - by Rizhikh-1
2381. Patient has toxic dilatation as complication of unspecific ulcerative colitis. What operation is indicated?
- * proctocolectomy
 - resection of dilatated area of colon
 - colectomy
 - colostomy
 - an operation by Kenu-Miles
2382. Patient has ulcer of rectum by duration near two months. The reaction of Wassermann is positive. Previous diagnosis
- * venereal lymphogranuloma
 - haemorrhoids
 - paraproctitis
 - fistula
 - fissure of rectum

2383. Patients with haemorrhoids has blood in the stool
- A. * during defecation
 - B. before defecation
 - C. after defecation
 - D. constantly
 - E. never
2384. Presence of blood in the stool is characteristically for:
- A. haemorrhoids
 - B. UUC
 - C. Cancer of colon
 - D. Fissures of anus
 - E. * All of answers are correct
2385. Radical operation at a bleeding gastric ulcer and duodenum consists in
- A. sewing vessels on a draught;
 - B. * vagotomy or resection the stomach;
 - C. sewing vessels in an ulcer;
 - D. gastroenteroanastomosis;
 - E. all answers are faithful
2386. Reasons of origin of fissures of rectum
- A. * constipations, diarrhoea
 - B. cancer of rectum
 - C. portal hypertension
 - D. ulcerous disease
 - E. varicose disease
2387. Relative absolute indication to operative treatment ulcerous illness is
- A. * penetration of ulcer
 - B. ulcerous anamnesis more than 15 years
 - C. malignization ulcers
 - D. perforation of ulcer
 - E. relapses more than 3 times per a year
2388. Resection of stomach by Bilrot II belongs to
- A. * radical operation
 - B. palliative operation
 - C. draining operations
 - D. organ protect operation
 - E. does not belong to any group
2389. Roentgenological signs of unspecific ulcerative colitis
- A. * symptom of "water-pipe"
 - B. symptom of "niche"
 - C. defect of filling
 - D. symptom of «roadway»
 - E. bowls of Kloyber
2390. Selective proximal vagotomy belongs to
- A. palliative
 - B. draining
 - C. * organ protect operation

- D. resection
 - E. does not belong to any group
2391. Signs of stable hemostasis
- A. * absence blood in a stomach and duodenum bulb;
 - B. presence the light blood and faltungs of blood in a stomach;
 - C. profluvium blood from a vessel;
 - D. all answers correct;
 - E. all answers are not correct
2392. Signs of unstable hemostasis
- A. * the pulsation of vessel is determined;
 - B. the bottom ulcer is covered a fibrin;
 - C. profluvium blood from a vessel;
 - D. all answers are correct;
 - E. all answers are not correct.
2393. Small amount of blood in stool could has patients with:
- A. * Poliposis
 - B. Colitis
 - C. Pancreatitis
 - D. Peptic ulcer disease
 - E. Appendicitis
2394. Solution of atropine sulfate is used, before operation with a purpose
- A. * of block of peripheral M-cholinoreceptors
 - B. providing of the adequate anaesthetizing
 - C. increase of vagus activity
 - D. increasing of frequency of pulse
 - E. decreasing of frequency of pulse
2395. Surgical treatment by the method of cutting of fistula of rectum with cutting of skin and subcutaneous tissue in the type of triangle
- A. * for Gabriel
 - B. for Rizhik-Bobroviy
 - C. ligature method
 - D. by Milligan-Morgan.
 - E. Operation of Rizhikh-1.
2396. Surgical treatment of paraproctitis by the method of desection of fistula of rectum with cutting of skin and mucus in the type of triangle
- A. * by Rizhik-Bobrov
 - B. by Gabriel
 - C. ligature method
 - D. by Milligan-Morgan.
 - E. Operation of Rizhikh-1.
2397. The medicinal “constipation” is used at operations
- A. * on a rectum
 - B. on a small intestine
 - C. on a liver
 - D. on a stomach
 - E. on a duodenum

2398. The nosotropic mechanisms bleedingness at ulcerous illness is
- * all answers are correct.
 - permanent hyperemia all system of stomach
 - different degree dystrophy of superficial layers the mucus shell
 - accumulation the central mucopolysaccharides
 - hypoplastic, dystrophic processes
2399. The secretory function stomach is carried out the next membrane of stomach
- * mucous membrane
 - internal muscular layer
 - serosal
 - mucous submembrane
 - external layer
2400. To absolute indication to operative interference at ulcerous illness does not belong
- * scarry-ulcerous stenosis
 - perforation of ulcer
 - profuse bleeding
 - diameter ulcer a more than 3 cm
 - bleeding what does not stopped with conservative
2401. To the gastric – intestinal bleeding of un ulcerous etiology belong
- * Mallory-Weiss syndrome;
 - hemorrhagic erosive gastritis;
 - diseases by Randyu – Oslera – Vebera;
 - Menetrie's sing;
 - all answers are correct.
2402. Udin's sing at a perforated ulcer is
- * feeling at palpation shove the gases which penetrate through the perforated opening
 - dulling perforated sound in the lateral departments of stomach
 - disappearance of hepatic dullness
 - irradiation pain in a shoulder or shoulder-blade
 - sickliness the back vault of vagina
2403. Vomiting coffee-grounds is a characteristic sign
- * bleeding ulcer
 - penetrative ulcers
 - perforated ulcers
 - malignized ulcers
 - for cicatrical-ulcerous pylorus stenosis
2404. Vomiting what arose up in 4-6 hours after eating characteristic for
- chronic alcoholic gastritis
 - cancer and ulcers of cardia
 - * pylorus ulcers
 - achalasia of gullet
 - ulcer and cancer the body of stomach
2405. What colouring of chair is most characteristic for bleeding from the ulcer of stomach and duodwnum?
- * Tarry excrement
 - Presence on the formed excrement of strokes of red blood
 - Littlechanged blood in an excrement

- D. Excrement of the raspberry colouring with the admixtures of mucus
 - E. Acholic excrement
2406. What complication of unspecific ulcerative colitis is indication to urgent surgery?
- A. Malignizaciya
 - B. Bleeding
 - C. Acute toxic dilatation
 - D. * Perforation
 - E. Diarrhea
2407. What complication the ulcerous illness of stomach is most characteristically for the patients of elderly and senile age
- A. perforation
 - B. perforation + bleeding
 - C. pylorus stenosis
 - D. malignization + penetration
 - E. * bleeding
2408. What disease is damage the superficial layer of wall of bowel
- A. * unspecific ulcerative colitis
 - B. Crohn disease
 - C. diverticulosis
 - D. poliposis
 - E. haemorrhoids
2409. What does mean a term "haemorrhoids"?
- A. Varicose enlargement of haemorrhoidal veins
 - B. Spasm of anal sphincter
 - C. * Bleeding
 - D. Inflammation of paraperctal tissue
 - E. Inflammation of anal channel
2410. What does subserve to development of haemorrhoidal thrombosis?
- A. Constipation
 - B. Diarrhea
 - C. * Spasm of sphincter
 - D. Criptitis
 - E. Papillitis
2411. What drug has purgative action?
- A. * Fenolftalein.
 - B. Aspirine.
 - C. Ftalasol.
 - D. Proserin.
 - E. Biphicol.
2412. What drug is decreased frequency of defecation?
- A. * Immodium.
 - B. Kofeol.
 - C. Karbolen.
 - D. Sulfate of magnesium.
 - E. Prozerin.
2413. What drug is used for fistulography?

- A. * Iodlipol.
 - B. Bilignost.
 - C. Verografin.
 - D. Sulfate of barium.
 - E. Methylene bluing.
2414. What drug is used for irrihography?
- A. * Sulfate of barium.
 - B. Cardiotrast.
 - C. Bilignost.
 - D. Iodlipol.
 - E. Methylene.
2415. What drugs is used to decrease meteorism?
- A. * Espumisan.
 - B. Norsulfazolum.
 - C. Vaseline oil.
 - D. Sulfate of magnesium.
 - E. Prozerin.
2416. What enema is used at intestinal obstruction?
- A. * Siphon enema.
 - B. Cleansing.
 - C. Microenema.
 - D. Purgative.
 - E. Does not used.
2417. What external signs are characteristic for the profuse bleeding from a gastric ulcer?
- A. Vomiting by the littlechanged blood, excrement of the raspberry colouring
 - B. * Vomiting by the littlechanged blood, tarry darkly-cherry chair
 - C. Vomiting by a complete mouth by dark blood with clots, black formed excrement
 - D. Vomiting on the type of "coffee-grounds", presence on the formed incandescence of strokes of red blood
 - E. Tarry darkly-cherry chair
2418. What form of unspecific ulcerative colitis is most dangerous?
- A. * Fulminating.
 - B. Acute.
 - C. Chronic recurrent.
 - D. Chronic continuous.
 - E. Recurrent.
2419. What from preparations has the expressed bacteriostatic action on Helicobacter pyloris
- A. * trichopol
 - B. Liciviroton
 - C. oxiferiskorbon
 - D. atropine
 - E. pirinzsipin
2420. What from the transferred operations does not belong to organ protective
- A. trunk vagotomy
 - B. * resection by Bilrot II
 - C. selective vagotomy
 - D. selective proximal vagotomy

- E. the all transferred does not belong
2421. What from the transferred operations on the stomach organ protective is
- A. * selective proximal vagotomy
 - B. resection by Bilrot I
 - C. resection by Bilrot II
 - D. gastrectomy
 - E. all are transferred
2422. What hormones undertake the protective operating on the mucous membrane of stomach, except for
- A. * ACTH
 - B. epidermal factor of growth
 - C. prostaglandin E
 - D. estrogens
 - E. STH
2423. What instrumental examination is performed at jamming of haemorrhoidal nodes
- A. * examination is not performed
 - B. rectometer
 - C. irrigoscopy
 - D. irrigography
 - E. colonoscopy
2424. What is from listed has hemostatic effect
- A. * blood
 - B. red corpuscles
 - C. Haemodesum
 - D. physiologic solution
 - E. glucose
2425. What is indication to surgical treatment of anal fissure
- A. * chronic passing
 - B. acute passing
 - C. perforation
 - D. bleeding
 - E. malignancy
2426. What is location of fistula chanal at intersphincteric paraproctitis?
- A. * Between mucus and sphincter
 - B. Passes through sphincter
 - C. Located after sphincter
 - D. All are true
 - E. All are false
2427. What is location of fistula chanal at the transsphincteric paraproctitis?
- A. Between mucus and sphincter
 - B. * Passes through sphincter
 - C. Located after sphincter
 - D. All is true
 - E. All is false
2428. What is the basic examination of patients with disease of rectosigmoid area?
- A. * Rectoromanoscopy.

- B. X-Ray.
 - C. Sonography.
 - D. Digital examination of rectum.
 - E. Irrigography.
2429. What is the complication after haemorrhoidectomy
- A. * stricture of anus
 - B. proctosygmoiditis
 - C. cancer of rectum
 - D. intestinal obstruction
 - E. peritonitis
2430. What is the complication of surgical treatment of anal fissures
- A. * insufficiency of anal sphincter
 - B. anterior mucosal prolapse of rectum
 - C. cancer of rectum
 - D. malignancy
 - E. Crohn disease
2431. What is the contra-indication for the colproctectomy at a unspecific ulcerative colitis:
- A. * A perforation of colon bowel
 - B. Acute toxic dilatation
 - C. Bleeding
 - D. Malignizaciya
 - E. Stenosis
2432. What is the operation of choice at a unspecific ulcerative colitis:
- A. Resection of rectum
 - B. * proctocolectomy
 - C. Left-side hemicolectomy
 - D. Right-side hemicolectomy
 - E. Resection of sygmoid bowel
2433. What is the operation of choice at the unspecific ulcerative colitis?
- A. * Proctocolonectomy with Ileostomy.
 - B. Bypassed loop anastomosis.
 - C. Subtotal colectomy.
 - D. Resection of colon.
 - E. Application of colostomy.
2434. What is the reason of origin of acute paraproctitis?
- A. Trauma of rectum
 - B. * Micro-injury of rectum mucus with the damage of crypt
 - C. Proctopolypus
 - D. Anal fissure
 - E. Cancer of rectum
2435. What is the reason of relapse of paraproctitis?
- A. An operation is done not enough radically
 - B. Infection of wound
 - C. * The internal opening is not removed
 - D. Anaerobic infection
 - E. Wrong conservative treatment

2436. What is the typical localization of anal fissure
- * on 6 hr.
 - on 12 hr.
 - on 3 hr.
 - on 9 hr.
 - on 2 hr.
2437. What kind of enemas is applied at preparation a patient to the operation?
- * Cleaning.
 - Siphon.
 - Microenema.
 - Purgative.
 - Does not used.
2438. What layers of the bowel are damaged at Crohn disease
- * all of layers
 - mucus
 - submucous and muscle
 - submucous
 - muscle
2439. What localization of ulcer is most characteristic for the patients of elderly and senile age
- * cardial department of stomach
 - overhead third of gullet
 - lower third of gullet
 - bulb of duodewnum
 - small curvature
2440. What medicines is used for increasing the frequency of defecation ?
- * Prozerin.
 - Sulfate of magnesium.
 - Immodium.
 - Karbolon.
 - Digestal.
2441. What medicines is used for treatment of unspecific ulcerative colitis?
- * Sulfasalazinum.
 - Ampicillin.
 - Nospanum, papaverini.
 - Aspirine.
 - Furazolidonum.
2442. What method diagnostics hte ulcerous illness most informing
- * esophagogastrroduodenoscopy
 - analysis of excrement on the hidden blood
 - X-ray
 - global analysis of blood
 - research of gastric secretion
2443. What method is it orientation possible to define the volume of hemorrhage on at the acute gastroenteric bleeding?
- On maintenance red corpuscles, haemoglobin, Ht, thrombocytes
 - On blood volume
 - * On an arteriotony, pulse, state of patient

- D. On a globular volume
 - E. On the level of thrombocytes
2444. What method of examination is used to confirm the diagnosis of Crohn disease?
- A. * A biopsy is with histological examination
 - B. CT
 - C. Sciagraphy
 - D. Sonography
 - E. Rectoscopy
2445. What method of operative treatment is used at the I - IV stage of complicated extrasphincteric fistula?
- A. * Ligature method.
 - B. Rizhikh-1.
 - C. Rizhikh-2.
 - D. Gabriel.
 - E. Cutting and suturing of fistula.
2446. What operation is performed at a acute shoe-shaped paraproctitis ?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2447. What operation is performed at an anal fissure?
- A. * Cutting of fissure with sphincterotomy.
 - B. Suturing of fissure.
 - C. Operation of Milligan-Morgan.
 - D. Operation of Gabriel.
 - E. Operation of Nobl.
2448. What operation is performed at haemorrhoids complicated by bleeding
- A. * urgent
 - B. planned
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. not performed
2449. What operation is performed at haemorrhoids complicated by trombosis
- A. * urgent
 - B. planned
 - C. conservative therapy
 - D. physiotherapeutic procedure
 - E. not performed
2450. What operation is performed at haemorrhoids?
- A. * Operation of Milligan-Morgan.
 - B. Rizhikh-1.
 - C. Rizhikh-2.
 - D. Operation of Gabriel.
 - E. Operation of Blinnichev.
2451. What operation is performed at patient with fissure and polypus?

- A. * Cutting of fissure with polypectomy.
 - B. Cutting of fissure.
 - C. By Milligan-Morgan.
 - D. By Gabriel.
 - E. Operation of Blinnichev.
2452. What operation is performed at rectocoele?
- A. * by Kumel-Zerenin.
 - B. Kenu-Miles.
 - C. Milligan-Morgan.
 - D. Gabriel.
 - E. Operation of Blinnichev.
2453. What operation is performed at the acute subcutaneous paraproctitis?
- A. Opening of abscess with suturing
 - B. * Cutting of abscess (by Gabriel)
 - C. Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2454. What operation is performed at the Crohn disease?
- A. * A resection of bowel within the limits of healthy tissues.
 - B. Proctocolonectomy.
 - C. Subtotal colectomy.
 - D. Resection of large and small intestine.
 - E. Application of colostomy.
2455. What operation is performed at the extrasphincteric paraproctitis?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2456. What operation is performed at the ischiorectal paraproctitis?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2457. What operation is performed at the retrorectal paraproctitis?
- A. Opening of abscess with suturing
 - B. Cutting of abscess (by Gabriel)
 - C. * Opening of abscess with the ligature conduction
 - D. Cutting of abscess with shifting of mucus to distal part of rectum
 - E. Conservative treatment
2458. What operation is performed at the transsphincteric paraproctitis?
- A. Cutting of fistula chanal (by Gabriel)
 - B. * Cutting of fistula chanal with the partial suturing of bottom of wound
 - C. Cutting of fistula chanal with the ligature conduction
 - D. Cutting of fistula with shifting of mucus to distal part of rectum
 - E. Conservative treatment

2459. What operation is radical at haemorrhoids?
- A. Suturing of haemorrhoidal nodes
 - B. Operation by Gabriel
 - C. * Operation by Milligan-Morgan
 - D. Sclerotherapy
 - E. Conservative treatment
2460. What operation is used for pararectal fistula?
- A. * Operation of Gabriel.
 - B. Operation of Milligan-Morgan.
 - C. Operation of Gagen-Torn.
 - D. Operation of Nobl.
 - E. A sphincterectomy.
2461. What part of colon is damaged by cancer most often:
- A. * Sigmoid colon
 - B. Caecum
 - C. Ascending part
 - D. Descending part
 - E. Rectum
2462. What part of intestine of most often is a pathological process localized in at Crohn disease?
- A. * Terminal part of small intestine.
 - B. Rectum.
 - C. Ascending part of colon bowel.
 - D. Lumbar part of colon bowel.
 - E. Sigmoid bowel.
2463. What patients with poliposis of colon could has in stool:
- A. Nothing
 - B. * Small amount of blood
 - C. Undigested meal
 - D. Presence of part of polypuses
 - E. Melena
2464. What preparation does behave to blocker H2-retseptors?
- A. * tavegil
 - B. obzidan
 - C. hystdol
 - D. cerucal
 - E. oraza
2465. What preparation does behave to blocker of muscarine receptors of coating cages?
- A. cymetidin
 - B. eglonin
 - C. * gastocepini
 - D. etimsiloli
 - E. vinylin
2466. What preparations, except for other properties, own yet and a bacteriostatic effect on Helicobacter pyloris
- A. * all are transferred preparations
 - B. Almagel

- C. Vinylin
 - D. De-nol
 - E. Claritromycin
2467. What products are recommended in the diet of № 1?
- A. * hen in a steam kind
 - B. pancakes
 - C. raw egg-white
 - D. bread rye fresh
 - E. acute cheeses
2468. What stool has patients with poliposis of colon:
- A. Not changed
 - B. Constipations
 - C. * Diarrhea
 - D. White
 - E. Melena
2469. What syndrome is characteristic for hemorrhagic erosive gastritis?
- A. * ulcerous
 - B. hemorrhagic;
 - C. pain;
 - D. all answers are correct;
 - E. all answers are not correct.
2470. What time urgent operations are executed at acute bleeding
- A. * 6 – 12 hours;
 - B. 6 – 10 hours;
 - C. 6 – 8 hours
 - D. 6 – 14 hours;
 - E. 6 – 20 hours
2471. What triad of symptoms is characteristic for an anal fissure
- A. * pain during defecation, spasm of sphincter, bleeding
 - B. pain before defecation, constipations, bleeding
 - C. diarrhea, bleeding, weight loss
 - D. anaemia, diarrhea, pain
 - E. bleeding, anaemia, diarrhea
2472. What violation of mineral exchange is characteristic for patients with ulcerous illness of stomach and intestine
- A. hypocalcemia
 - B. * hypokaliemia
 - C. Hyponatremia
 - D. Hypercalcinemia
 - E. hyperkaliemia
2473. What volume of blood lost at a unspecific ulcerative colitis does consider complications?
- A. To 50 ml
 - B. 50-100 ml
 - C. 150-200 ml
 - D. 200-300 ml
 - E. * 300 ml and more

2474. When apply Teylor's method at ulcerous illness
- A. * at conservative treatment perforeted ulcers
 - B. at conservative treatment sanguifluous ulcers
 - C. at conservative treatment penetration ulcers
 - D. at conservative treatment malignization ulcers
 - E. at conservative treatment of cicatrical pyloristenosis
2475. Where are anal papilla usually located?
- A. In any area of anal channel
 - B. * Only above and lower from dentata line
 - C. In sygmoid colon
 - D. In any area of rectum
 - E. In perianal region
2476. Which preparation is used for treatment of unspecific ulcerative colitis
- A. * sulfasalasine
 - B. fenolftaleine
 - C. cerucal
 - D. analgin
 - E. ketanov
2477. With the purpose of preventing of anaerobic infection at surgical treatment of paraproctitis is used
- A. * hydrogen peroxide
 - B. furacilini
 - C. iodine solution
 - D. rivanol
 - E. alcohol
2478. Aberrant goiter is:
- A. * The goiter of additional gland
 - B. Dislocation of the goiter
 - C. The goiter with increased function
 - D. The goiter with decreased function
 - E. The goiter with normal function
2479. Among the complication of a postoperative period for thyrotoxicosis is:
- A. * Air embolism
 - B. Cretinism
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2480. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. * Thyroid storm
 - B. Cretinism
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2481. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. * The damage of laryngeal nerve
 - B. Cretinism
 - C. Lerishe's syndrome

- D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2482. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. * Asphyxia
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2483. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. * Parathyroid tetany
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2484. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. * Bleeding
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2485. Basedow's disease is:
- A. * Thyrotoxicosis
 - B. Wooden thyroiditis
 - C. Purulent thyroiditis
 - D. Autoimmune thyroiditis
 - E. Mixedema
2486. Diffuse goiter with hyperthyroidism is called:
- A. * Grave's disease
 - B. Hashimoto disease
 - C. De Kerven disease
 - D. Riedel's disease
 - E. Raynaud's disease
2487. Diffuse goiter with hyperthyroidism is called:
- A. * Basedow's disease
 - B. Hashimoto disease
 - C. De Kerven disease
 - D. Riedel's disease
 - E. Raynaud's disease
2488. Ectopic goiter is:
- A. * Dislocation of the goiter
 - B. The goiter of additional gland
 - C. The goiter with increased function
 - D. The goiter with decreased function
 - E. The goiter with normal function
2489. For the clinical manifestation of retrosternal is typical:
- A. * Dyspnea

- B. Sleepiness
- C. Hypomnesia
- D. Excessive sweating
- E. Tremor of arms

2490. For the clinical manifestation of retrosternal is typical:

- A. * Hoarseness
- B. Sleepiness
- C. Hypomnesia
- D. Excessive sweating
- E. Tremor of arms

2491. For the clinical manifestation of retrosternal is typical:

- A. * Distended veins of neck
- B. Sleepiness,
- C. Hypomnesia
- D. Excessive sweating
- E. Tremor of arms

2492. For the clinical manifestation of the damage of laryngeal nerve is typical:

- A. * Hoarseness
- B. Diarrhea
- C. Cramps
- D. Fever to 40°C
- E. Anemia

2493. For the clinical manifestation of the damage of laryngeal nerve is typical:

- A. * Aponia
- B. Diarrhea
- C. Cramps
- D. Fever to 40°C
- E. Anemia

2494. For the clinical manifestation of the parathyroid tetany is typical:

- A. * Cramps
- B. Aponia
- C. Diarrhea
- D. Fever to 40°C
- E. Anemia

2495. For the clinical manifestation of thyroid storm is typical:

- A. * Excitement, up to psychosis and coma
- B. Hoarseness
- C. Cramps
- D. Aponia
- E. Anemia

2496. For the clinical manifestation of thyroid storm is typical:

- A. * Tachycardia (pulse rate – 150-200 per minute)
- B. Hoarseness
- C. Cramps
- D. Aponia
- E. Anemia

2497. For the clinical manifestation of thyroid storm is typical:
- A. * Complete arrhythmia
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
2498. For the clinical manifestation of thyroid storm is typical:
- A. * Fever to 40°C
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
2499. For the clinical manifestation of thyroid storm is typical:
- A. * Hyperemia of the face, neck, limbs
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
2500. For the clinical manifestation of thyroid storm is typical:
- A. * Extremely sweating
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
2501. For the clinical manifestation of thyroid storm is typical:
- A. * Diarrhea
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
2502. For the laboratory disturbances of Basedow's disease is typical:
- A. * Increased level of triiodothyronine
 - B. Decreased level of triiodothyronine
 - C. Increased level of glucocorticoids
 - D. Decreased level of glucocorticoids
 - E. Decreased level of insulin
2503. For the laboratory disturbances of Basedow's disease is typical:
- A. * Increased level of thyroxine
 - B. Decreased level of triiodothyronine
 - C. Increased level of glucocorticoids
 - D. Decreased level of glucocorticoids
 - E. Decreased level of insulin
2504. For the laboratory disturbances of thyrotoxicosis is typical:
- A. * Increased level of lipid metabolism
 - B. Decreased level of carbohydrate metabolism
 - C. Decreased level of lipid metabolism
 - D. Decreased level of protein metabolism

- E. Decreased level of all kinds of metabolism
2505. For the laboratory disturbances of thyrotoxicosis is typical:
- A. * Increased level of protein metabolism
 - B. Decreased level of carbohydrate metabolism
 - C. Decreased level of lipid metabolism
 - D. Decreased level of protein metabolism
 - E. Decreased level of all kinds of metabolism
2506. For the laboratory disturbances of thyrotoxicosis is typical:
- A. * Increased level of carbohydrate metabolism
 - B. Decreased level of carbohydrate metabolism
 - C. Decreased level of lipid metabolism
 - D. Decreased level of protein metabolism
 - E. Decreased level of all kinds of metabolism
2507. For the thyrotoxicosis is typical:
- A. * Mebius' sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
2508. For the thyrotoxicosis is typical:
- A. * Graefe's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
2509. For the thyrotoxicosis is typical:
- A. * Dalrymple's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
2510. For the thyrotoxicosis is typical:
- A. * Kocher's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
2511. For thyrotoxicosis is typical:
- A. * Stellwag's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
2512. Goiter which localized on the back of the tongue is called:
- A. * Ectopic goiter
 - B. Aberrant goiter

- C. Typical
- D. Presternal
- E. Retrosternal

2513. Goiter which occurs in biogeochemical regions with iodine deficiency in environment is called:

- A. * Endemic goiter
- B. Sporadic goiter
- C. Thyrotoxicosis
- D. Myxedema
- E. Atypical goiter

2514. Goiter which occurs in unendemic regions is called:

- A. * Sporadic goiter
- B. Endemic goiter
- C. Thyrotoxicosis
- D. Myxedema
- E. Atypical goiter

2515. Grave's disease is:

- A. * Thyrotoxicosis
- B. Wooden thyroiditis
- C. Purulent thyroiditis
- D. Autoimmune thyroiditis
- E. Mixedema

2516. How is the sign, which is characterized by a weakness of convergence named by author?

- A. * Mebius' sign
- B. Stellwag's sign
- C. Graefe's sign
- D. Dalrymple's sign
- E. Kocher's sign

2517. How is the sign, which is characterized by a wide palpebral fissure named by author?

- A. * Dalrymple's sign
- B. Mebius' sign
- C. Stellwag's sign
- D. Graefe's sign
- E. Kocher's sign

2518. How is the sign, which is characterized by infrequent winking named by author?

- A. * Stellwag's sign
- B. Graefe's sign
- C. Mebius' sign
- D. Dalrymple's sign
- E. Kocher's sign

2519. How is the sign, which is characterized by retraction of the upper eyelid at prompt change of view named by author?

- A. * Kocher's sign
- B. Dalrymple's sign
- C. Mebius' sign
- D. Stellwag's sign
- E. Graefe's sign

2520. How is the sign, which is characterized by the upper lid lag when the patient looks downward named by author?
- A. * Graefe's sign
 - B. Mebius' sign
 - C. Stellwag's sign
 - D. Dalrymple's sign
 - E. Kocher's sign
2521. In case of euthyroid goiter the patient mainly complains of:
- A. * Neck deformity
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
2522. In case of euthyroid goiter the patient mainly complains of:
- A. * Difficult breathing
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
2523. In case of euthyroid goiter the patient mainly complains of:
- A. * Difficult swallowing
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
2524. In case of euthyroid goiter the patient mainly complains of:
- A. * Sudden attacks of cough
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
2525. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Excessive sweating
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2526. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Irritability
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2527. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Heartbeat
 - B. Leg edemas

- C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2528. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Tremor of arms
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2529. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Sleeplessness
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2530. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Feeling of fever
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2531. In case of hyperthyroid goiter the patient mainly complains of:
- A. * Loss of weight
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2532. In case of hypothyroid goiter the patient mainly complains of:
- A. * Sleepiness
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2533. In case of hypothyroid goiter the patient mainly complains of:
- A. * General weakness
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2534. In case of hypothyroid goiter the patient mainly complains of:
- A. * Malaise
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2535. In case of hypothyroid goiter the patient mainly complains of:

- A. * Hypomnesia
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2536. In case of hypothyroid goiter the patient mainly complains of:
- A. * Dry skin
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2537. In case of hypothyroid goiter the patient mainly complains of:
- A. * Constipations
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2538. In case of hypothyroid goiter the patient mainly complains of:
- A. * Leg edemas
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
2539. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Excessive sweating
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2540. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Irritability
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2541. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Heartbeat
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2542. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Tremor of arms
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation

2543. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Sleeplessness
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2544. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Feeling of fever
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2545. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Loss of weight
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2546. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Palpitation
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2547. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Exophthalmos
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2548. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. * Tremor
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
2549. Intrathoracic localization of goiter is called:
- A. * Ectopic goiter
 - B. Aberrant goiter
 - C. Typical
 - D. Presternal
 - E. Retrosternal
2550. The "woody" goiter is a:
- A. * Riedel's goiter
 - B. Hashimoto's goiter
 - C. De Kerven thyroiditis

- D. Grave's disease
 - E. Basedow's disease
2551. The autoimmune thyroiditis is a:
- A. * Hashimoto's goiter
 - B. De Kerven thyroiditis
 - C. Riedel's goiter
 - D. Grave's disease
 - E. Basedow's disease
2552. The constipation is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2553. The Dalrymple's sign is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2554. The Dalrymple's sign is:
- A. * Wide palpebral fissure
 - B. Infrequent winking
 - C. The upper lid lag when the patient looks downward
 - D. A weakness of convergence
 - E. Retraction of the upper eyelid at prompt change of view
2555. The De Kerven thyroiditis is a:
- A. * Purulent thyroiditis
 - B. Autoimmune thyroiditis
 - C. Fibrous thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
2556. The development of aphonia in early postoperative period after thyroid surgery is the manifestation of:
- A. * The damage of laryngeal nerve
 - B. Thyroid storm
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2557. The development of complete arrhythmia in early postoperative period after thyroid surgery is the manifestation of:
- A. * Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome

2558. The development of cramps in early postoperative period after thyroid surgery is the manifestation of:
- A. * Parathyroid tetany
 - B. The damage of laryngeal nerve
 - C. Thyroid storm
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2559. The development of excitement, up to psychosis and coma in early postoperative period after thyroid surgery is the manifestation of:
- A. * Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Air embolism
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2560. The development of extremely sweating, diarrhea in early postoperative period after thyroid surgery is the manifestation of:
- A. * Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2561. The development of fever to 40°C in early postoperative period after thyroid surgery is the manifestation of:
- A. * Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2562. The development of hoarseness in early postoperative period after thyroid surgery is the manifestation of:
- A. * The damage of laryngeal nerve
 - B. Thyroid storm
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2563. The development of hyperemia of the face, neck, limbs in early postoperative period after thyroid surgery is the manifestation of:
- A. * Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
2564. The development of tachycardia (pulse rate – 150-200 per minute) in early postoperative period after thyroid surgery is the manifestation of:
- A. * Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Parathyroid tetany
 - D. Adrenal insufficiency

- E. Itsenko-Cushing syndrome
2565. The difficult breathing is a clinical manifestation of the patient with:
- A. * Euthyroid goiter
 - B. Hypothyroidism
 - C. Thyrotoxicosis
 - D. Esophageal achalasia
 - E. Gastric ulcer
2566. The difficult swallowing is a clinical manifestation of the patient with:
- A. * Euthyroid goiter
 - B. Hypothyroidism
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Empyema
2567. The dry skin is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2568. The edemas is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2569. The excessive sweating is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2570. The excessive sweating is a clinical manifestation of the patient with:
- A. * Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2571. The exophthalmos is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2572. The feeling of fever is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema

- C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2573. The feeling of fever is a clinical manifestation of the patient with:
- A. * Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2574. The fibrous thyroiditis is a:
- A. * Riedel's goiter
 - B. Hashimoto's goiter
 - C. De Kerven thyroiditis
 - D. Grave's disease
 - E. Basedow's disease
2575. The general weakness is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2576. The goiter of additional gland is called:
- A. * Aberrant goiter
 - B. Ectopic goiter
 - C. Typical
 - D. Presternal
 - E. Retrosternal
2577. The Graefe's sign is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2578. The Graefe's sign is:
- A. * The upper lid lag when the patient looks downward
 - B. Infrequent winking
 - C. A weakness of convergence
 - D. Wide palpebral fissure
 - E. Retraction of the upper eyelid at prompt change of view
2579. The Hashimoto's goiter is a:
- A. * Autoimmune thyroiditis
 - B. Fibrous thyroiditis
 - C. Purulent thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
2580. The heartbeat is a clinical manifestation of the patient with:

- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2581. The heartbeat is a clinical manifestation of the patient with:
- A. * Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2582. The hypomnesia is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2583. The increased level of carbohydrate metabolism is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2584. The increased level of lipid metabolism is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2585. The increased level of protein metabolism is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2586. The irritability is a clinical manifestation of the patient with:
- A. * Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2587. The Kocher's sign is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum

2588. The Kocher's sign is:
- A. * Retraction of the upper eyelid at prompt change of view
 - B. Wide palpebral fissure
 - C. Infrequent winking
 - D. The upper lid lag when the patient looks downward
 - E. A weakness of convergence
2589. The loss of weight is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2590. The loss of weight is a clinical manifestation of the patient with:
- A. * Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2591. The malaise is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2592. The Mebius' sign is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2593. The Mebius' sign is:
- A. * A weakness of convergence
 - B. Infrequent winking
 - C. The upper lid lag when the patient looks downward
 - D. Wide palpebral fissure
 - E. Retraction of the upper eyelid at prompt change of view
2594. The neck deformity is a clinical manifestation of the patient with:
- A. * Euthyroid goiter
 - B. Hypothyroidism
 - C. Thyrotoxicosis
 - D. Esophageal achalasia
 - E. Empyema
2595. The palpitation is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter

- D. Gastric ulcer
 - E. Esophageal diverticulum
2596. The palpitation is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2597. The purulent thyroiditis is a:
- A. * De Kerven thyroiditis
 - B. Hashimoto's goiter
 - C. Riedel's goiter
 - D. Grave's disease
 - E. Basedow's disease
2598. The Riedel's goiter is a:
- A. Woody" goiter
 - B. Autoimmune thyroiditis
 - C. Purulent thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
2599. The Riedel's goiter is a:
- A. * Fibrous thyroiditis
 - B. Autoimmune thyroiditis
 - C. Purulent thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
2600. The sleepiness is a clinical manifestation of the patient with:
- A. * Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2601. The Stellwag's sign is typical for:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2602. The Stellwag's sign is:
- A. * Infrequent winking
 - B. The upper lid lag when the patient looks downward
 - C. A weakness of convergence
 - D. Wide palpebral fissure
 - E. Retraction of the upper eyelid at prompt change of view
2603. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. * Goiter of IV-V degree

- B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Goiter with hypothyroidism
2604. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. * Nodular transformation of toxic goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Goiter with hypothyroidism
2605. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. * Thyrotoxicosis
 - B. Obesity
 - C. Leriche's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
2606. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. * Severe forms of thyrotoxicosis
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Goiter with hypothyroidism
2607. The treatment of de Kerven thyroiditis is a:
- A. * Drainage of suppurative focus
 - B. Glycocorticoids
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil
2608. The treatment of Hashimoto's goiter is a:
- A. * Glycocorticoids
 - B. Drainage of suppurative focus
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil
2609. The treatment of Hashimoto's goiter is a:
- A. * Thyroidectomy
 - B. Drainage of suppurative focus
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil
2610. The treatment of Riedel's goiter is a:
- A. * Thyroidectomy
 - B. Drainage of suppurative focus
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil

2611. The tremor is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2612. The tremor of arms is a clinical manifestation of the patient with:
- A. * Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2613. The tremor of arms is a clinical manifestation of the patient with:
- A. * Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
2614. What belongs to the I stage of thyrotoxicosis?
- A. * Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - B. Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - C. Thyrotoxic lesion of viscera
 - D. Nonreversible dystrophy of organs and systems
 - E. Asymptomatic course
2615. What belongs to the II stage of thyrotoxicosis?
- A. * Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - B. Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - C. Thyrotoxic lesion of viscera
 - D. Nonreversible dystrophy of organs and systems
 - E. Asymptomatic course
2616. What belongs to the III stage of thyrotoxicosis?
- A. * Thyrotoxic lesion of viscera
 - B. Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - C. Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - D. Nonreversible dystrophy of organs and systems
 - E. Asymptomatic course
2617. What belongs to the IV stage of thyrotoxicosis?
- A. * Nonreversible dystrophy of organs and systems
 - B. Thyrotoxic lesion of viscera
 - C. Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - D. Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - E. Asymptomatic course
2618. What complication is characteristic for the goiter?
- A. * Strumitis
 - B. Laringospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema

- E. Atypical goiter
2619. What complication is characteristic for the goiter?
- A. * Hemorrhage into the gland
 - B. Laryngospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
2620. What complication is characteristic for the goiter?
- A. * Malignancy
 - B. Laryngospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
2621. What complication is characteristic for the goiter?
- A. * Asphyxia
 - B. Laryngospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
2622. What diseases should be the thyrotoxicosis differentiated with?
- A. * Rheumatic disease
 - B. Obesity
 - C. Leriche's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
2623. What diseases should be the thyrotoxicosis differentiated with?
- A. * Chroniosepsis
 - B. Obesity
 - C. Leriche's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
2624. What diseases should be the thyrotoxicosis differentiated with?
- A. * Diencephalic lesions
 - B. Obesity
 - C. Leriche's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
2625. What diseases should be the thyrotoxicosis differentiated with?
- A. * Thyroid cancer
 - B. Obesity
 - C. Leriche's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
2626. What diseases should be the thyrotoxicosis differentiated with?
- A. * Encephalitis
 - B. Obesity

- C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
2627. What form of thyrotoxicosis is classified as a mild?
- A. * Pulse rate less than 100 beat/min
 - B. Pulse rate 100-120 beat/min
 - C. Pulse rate 120-140 beat/min
 - D. Pulse rate 140-160 beat/min
 - E. Pulse rate more than 160 beat/min
2628. What form of thyrotoxicosis is classified as a mild?
- A. * Loss weight less than 3-5 kg
 - B. Loss weight 5-8 kg
 - C. Loss weight 8-10 kg
 - D. Loss weight 10-15 kg
 - E. Loss weight exceeds 15 kg
2629. What form of thyrotoxicosis is classified as a mild?
- A. * Increase of basal metabolism to 30 %
 - B. Increase of basal metabolism on 30-50 %
 - C. Increase of basal metabolism more than 50 %
 - D. Decrease of basal metabolism to 30 %
 - E. Decrease of basal metabolism more than 50 %
2630. What form of thyrotoxicosis is classified as a moderate?
- A. * Pulse rate 100-120 beat/min
 - B. Pulse rate less than 100 beat/min
 - C. Pulse rate 120-140 beat/min
 - D. Pulse rate 140-160 beat/min
 - E. Pulse rate more than 160 beat/min
2631. What form of thyrotoxicosis is classified as a moderate?
- A. * Loss weight 5-10 kg
 - B. Exceed of weight more than 1-2 kg
 - C. Exceed of weight more than 3-5 kg
 - D. Loss weight 10-15 kg
 - E. Loss weight exceeds 15 kg
2632. What form of thyrotoxicosis is classified as a moderate?
- A. * Increase of basal metabolism on 30-50 %
 - B. Increase of basal metabolism to 30 %
 - C. Increase of basal metabolism more than 50 %
 - D. Decrease of basal metabolism to 30 %
 - E. Decrease of basal metabolism more than 50 %
2633. What form of thyrotoxicosis is classified as severe?
- A. * Pulse rate more than 120 beat/min
 - B. Pulse rate less than 40 beat/min
 - C. Pulse rate 40-80 beat/min
 - D. Pulse rate 80-100 beat/min
 - E. Pulse rate 100-120 beat/min
2634. What form of thyrotoxicosis is classified as severe?

- A. * Loss weight exceeds 10 kg
 - B. Loss weight less than 1-2 kg
 - C. Loss weight 2-3 kg
 - D. Exceed of weight more than 1-2 kg
 - E. Exceed of weight more than 3-5 kg
2635. What form of thyrotoxicosis is classified as severe?
- A. * Increase of basal metabolism more than 50 %
 - B. Increase of basal metabolism on 30-50 %
 - C. Increase of basal metabolism to 30 %
 - D. Decrease of basal metabolism to 30 %
 - E. Decrease of basal metabolism more than 50 %
2636. What form of thyrotoxicosis is related with the increase of basal metabolism more than 50 %?
- A. * Severe
 - B. Mild
 - C. Moderate
 - D. Subclinic
 - E. Asymptomatic
2637. What form of thyrotoxicosis is related with the increase of basal metabolism on 30-50 %?
- A. * Moderate
 - B. Mild
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
2638. What form of thyrotoxicosis is related with the increase of basal metabolism to 30 %?
- A. * Mild
 - B. Moderate
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
2639. What form of thyrotoxicosis is related with the loss weight 5-10 kg?
- A. * Moderate
 - B. Mild
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
2640. What form of thyrotoxicosis is related with the loss weight less than 3-5 kg?
- A. * Mild
 - B. Moderate
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
2641. What form of thyrotoxicosis is related with the loss weight more than 10 kg?
- A. * Severe
 - B. Moderate
 - C. Mild
 - D. Subclinic

- E. Asymptomatic
2642. What group of medicines does Lithium carbonate belong to?
- A. * Thyrostatic agents
 - B. Antibiotics
 - C. Anticoagulants
 - D. Antiaggregants
 - E. Vitamines
2643. What group of medicines does Mercasolil belong to?
- A. * Thyrostatic agents
 - B. Antibiotics
 - C. Anticoagulants
 - D. Antiaggregants
 - E. Vitamines
2644. What is the 0 degree of goiter?
- A. * The thyroid gland is not palpated;
 - B. The isthmus of the gland is noticeable during swallowing and could be palpated;
 - C. Entire gland is noticeable during swallowing and could be palpated;
 - D. The enlargement of gland results in evident thickening of neck ("a thick neck");
 - E. The gland considerably enlarged, and sharply deforms neck
2645. What is the cause of thyrotoxicosis?
- A. * Autoimmune disturbances
 - B. Atherosclerotic changes
 - C. Calcium metabolism disturbances
 - D. Renal insufficiency
 - E. Pulmonary emphysema
2646. What is the contributing factor which causes the lung abscess?
- A. * Excessive calcium, deficiency of bromine in environment
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Suprarenal insufficiency
 - D. Rheumatism, endocarditis
 - E. Lack of vitamin C
2647. What is the contributing factor which causes the lung abscess?
- A. * Lack of cobalt, and zinc
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Suprarenal insufficiency
 - D. Rheumatism, endocarditis
 - E. Lack of vitamin C
2648. What is the degree of goiter when entire gland is noticeable during swallowing and could be palpated?
- A. * I
 - B. II
 - C. V
 - D. III
 - E. IV
2649. What is the degree of goiter when the enlargement of gland results in evident thickening of neck ("a thick neck")?

- A. * III
- B. I
- C. V
- D. II
- E. IV

2650. What is the degree of goiter when the enlargement reaches excessive size (goiter of major sizes)?

- A. * V
- B. IV
- C. III
- D. I
- E. II

2651. What is the degree of goiter when the gland considerably enlarged, and sharply deforms neck?

- A. * IV
- B. III
- C. I
- D. V
- E. II

2652. What is the degree of goiter when the isthmus of the gland is noticeable during swallowing and could be palpated?

- A. * I
- B. V
- C. II
- D. III
- E. IV

2653. What is the degree of goiter when the thyroid gland is not palpated?

- A. * 0
- B. II
- C. I
- D. III
- E. IV

2654. What is the endemic goiter characterized by?

- A. * Goiter which occurs in biogeochemical regions with iodine deficiency in environment
- B. Goiter which occurs in unendemic regions
- C. Goiter lesion of both lobes
- D. Goiter of atypical localization
- E. Goiter with changed function

2655. What is the I degree of goiter?

- A. * The isthmus of the gland is noticeable during swallowing and could be palpated;
- B. The thyroid gland is not palpated;
- C. Entire gland is noticeable during swallowing and could be palpated;
- D. The enlargement of gland results in evident thickening of neck ("a thick neck");
- E. The gland considerably enlarged, and sharply deforms neck

2656. What is the I stage of thyrotoxicosis?

- A. * Neurotic

- B. Neurohormonal
 - C. Visceropathic
 - D. Cachectic
 - E. Asymptomatic
2657. What is the II degree of goiter?
- A. * Entire gland is noticeable during swallowing and could be palpated;
 - B. The thyroid gland is not palpated;
 - C. The isthmus of the gland is noticeable during swallowing and could be palpated;
 - D. The enlargement of gland results in evident thickening of neck ("a thick neck");
 - E. The gland considerably enlarged, and sharply deforms neck
2658. What is the II stage of thyrotoxicosis?
- A. * Neurohormonal
 - B. Neurotic
 - C. Visceropathic
 - D. Cachectic
 - E. Asymptomatic
2659. What is the III degree of goiter?
- A. * The enlargement of gland results in evident thickening of neck ("a thick neck");
 - B. The thyroid gland is not palpated;
 - C. The isthmus of the gland is noticeable during swallowing and could be palpated;
 - D. Entire gland is noticeable during swallowing and could be palpated;
 - E. The gland considerably enlarged, and sharply deforms neck
2660. What is the III stage of thyrotoxicosis?
- A. * Visceropathic
 - B. Neurohormonal
 - C. Neurotic
 - D. Cachectic
 - E. Asymptomatic
2661. What is the indication for the conservative treatment of the goiter?
- A. * The goiter of I degree
 - B. The retrosternal ectopy of thyroid gland
 - C. The aberrant goiter
 - D. The goiter of IV degree
 - E. The goiter with secondary hyperthyroidism
2662. What is the indication for the conservative treatment of the goiter?
- A. * The goiter of II degree
 - B. The retrosternal ectopy of thyroid gland
 - C. The aberrant goiter
 - D. The goiter of IV degree
 - E. The goiter with secondary hyperthyroidism
2663. What is the indication for the operative treatment of the goiter?
- A. * Nodular goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter

2664. What is the indication for the operative treatment of the goiter?
- A. * Mixed forms of goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2665. What is the indication for the operative treatment of the goiter?
- A. * The sings of neck compression by goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2666. What is the indication for the operative treatment of the goiter?
- A. * The goiter with secondary hyperthyroidism
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2667. What is the indication for the operative treatment of the goiter?
- A. * The goiter with suspicion on malignancy
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2668. What is the indication for the operative treatment of the goiter?
- A. * The goiter of additional thyroid glands
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2669. What is the indication for the operative treatment of the goiter?
- A. * The aberrant goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2670. What is the indication for the operative treatment of the goiter?
- A. * The intrathoracic goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2671. What is the indication for the operative treatment of the goiter?
- A. * The retrosternal ectopy of thyroid gland
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree

- E. Uncomplicated hypothyroid goiter
2672. What is the indication for the operative treatment of the goiter?
- A. * The complicated goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
2673. What is the IV degree of goiter?
- A. * The gland considerably enlarged, and sharply deforms neck;
 - B. The enlargement reaches excessive size (goiter of major sizes)
 - C. The isthmus of the gland is noticeable during swallowing and could be palpated;
 - D. Entire gland is noticeable during swallowing and could be palpated;
 - E. The enlargement of gland results in evident thickening of neck ("a thick neck")
2674. What is the IV degree of goiter?
- A. * The enlargement reaches excessive size (goiter of major sizes)
 - B. The gland considerably enlarged, and sharply deforms neck;
 - C. The isthmus of the gland is noticeable during swallowing and could be palpated;
 - D. Entire gland is noticeable during swallowing and could be palpated;
 - E. The enlargement of gland results in evident thickening of neck ("a thick neck")
2675. What is the IV stage of thyrotoxicosis?
- A. * Cachectic
 - B. Visceropathic
 - C. Neurohormonal
 - D. Neurotic
 - E. Asymptomatic
2676. What is the main treatment of noncomplicated goiter?
- A. * Conservative treatment
 - B. Segmentectomy
 - C. Lobectomy
 - D. Thyroidectomy
 - E. Hemithyroidectomy
2677. What is the most informative in the diagnostic of thyrotoxicosis?
- A. * Thyroid hormone concentration
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
2678. What is the most informative in the diagnostic of thyrotoxicosis?
- A. * Serum thyroidstimulating antibodies
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
2679. What is the most informative in the diagnostic of thyrotoxicosis?
- A. * Detecting of basal metabolism
 - B. General blood analysis

- C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
2680. What is the most informative in the diagnostic of thyrotoxicosis?
- A. * Serum iodine-binding globulin concentration
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
2681. What is the most informative in the diagnostic of thyrotoxicosis?
- A. * Serum thyroidstimulating hormone of hypophysis
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
2682. What is the predominant factor which causes the goiter?
- A. * Lack of iodine
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Suprarenal insufficiency
 - D. Rheumatism, endocarditis
 - E. Lack of vitamin C
2683. What is the sporadic goiter characterized by?
- A. * Goiter which occurs in unendemic regions
 - B. Goiter which occurs in biogeochemical regions with iodine deficiency in environment
 - C. Goiter lesion of both lobes
 - D. Goiter of atypical localization
 - E. Goiter with changed function
2684. What medicines are used for the treatment of goiter?
- A. * Triiodothyronine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
2685. What medicines are used for the treatment of goiter?
- A. * Inorganic iodine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
2686. What medicines are used for the treatment of goiter?
- A. * Thyroidine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
2687. What medicines are used for the treatment of goiter?

- A. * Thyroxine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
2688. What medicines belong to thyrostatic agents?
- A. * Lithium carbonate
 - B. Euphyllin
 - C. Vasaprostan
 - D. Nicotine acid
 - E. Dextrin
2689. What medicines belong to thyrostatic agents?
- A. * Mercasolil
 - B. Euphyllin
 - C. Vasaprostan
 - D. Nicotine acid
 - E. Dextrin
2690. What operation is performed in endemic goiter?
- A. * Subfascial resection of thyroid gland
 - B. Segmentectomy
 - C. Lobectomy
 - D. Thyroidectomy
 - E. Hemithyroidectomy
2691. What stage of thyrotoxicosis correlates with the marked signs of thyrotoxicosis and noticeably enlarged thyroid?
- A. * II
 - B. I
 - C. III
 - D. IV
 - E. 0
2692. What stage of thyrotoxicosis correlates with the nonreversible dystrophy of organs and systems?
- A. * IV
 - B. III
 - C. II
 - D. I
 - E. 0
2693. What stage of thyrotoxicosis correlates with the onset of the disease, slight enlargement of thyroid gland?
- A. * I
 - B. II
 - C. III
 - D. IV
 - E. 0
2694. What stage of thyrotoxicosis correlates with the thyrotoxic lesion of viscera?
- A. * III
 - B. II

- C. I
- D. IV
- E. 0

SITUATIONAL TASKS

1. A diarrhea is not obligatory, but possible in acute appendicitis. In what cases diarrhea confirms the inflammation of appendix?
 - A. * In pelvic appendicitis
 - B. On the first day of the disease
 - C. In fever
 - D. In retrocaecal appendicitis
 - E. In retroperitoneal appendicitis

2. A patient complains of a pain in a right iliac area. At first pain appeared in epigastrium, then relocated in a right iliac area. During examination muscular tension of anterior abdominal wall and symptoms of peritoneal irritation are detected in a right iliac area. Leucocytes- 8×10^9 . General urine analysis within norm. Your diagnosis.
 - A. * Acute appendicitis
 - B. Right-side renal colic
 - C. Appendicular infiltrate
 - D. Meckel's diverticulum
 - E. Perforative ulcer

3. A patient complains of a pain in the lower abdomen, mostly on the right side, dizziness. The mentioned signs appeared suddenly, at night. Last menstruation 2 weeks ago. During examination the skin is pale, pulse 92 /min., t -36°C , AP 100/60 mm Hg. The abdomen is slightly tense, painful in lower areas. The signs of peritoneal irritation are insignificant. Hb-98 g/l. Your diagnosis?
 - A. * Ovarian apoplexy
 - B. Renal colic
 - C. Acute appendicitis
 - D. Intestinal obstruction
 - E. Bleeding gastric ulcer

4. A patient was operated 9 days ago for destructive appendicitis with pelvic position. As a complication – the abscess of Douglas space was developed. At digital rectal examination the infiltrate was detected with softening in center. Tactic of surgeon?
 - A. * Punction of the abscess with draining
 - B. Relaparotomy, draining of the abscess
 - C. Presacral antibiotic-novocaine blockade
 - D. Warm small enemas, suppositories with antibiotics
 - E. Electrophoresis with antibiotics on abdomen

5. A patient was operated for acute appendicitis. The condition deteriorated. An abdominal pain appeared, a temperature rose to 39°C . The signs of peritoneal irritation are absent. A jaundice joined. What complication is need to think about?
 - A. * Pylephlebitis
 - B. Diffuse peritonitis
 - C. Subphrenic abscess
 - D. Cholecystitis
 - E. Appendicular infiltrate

6. Among the ways of penetration of infection in appendix in acute appendicitis the most frequent is enterogenic. What microorganisms are revealed more frequent?
- * E. Colli
 - Staphylococci
 - Streptococci
 - Bacteroids
 - Koch's mycobacterii
7. During the operation for acute appendicitis, you revealed a dense appendicular infiltrate. What is your tactic?
- * To close the wound of the abdominal wall and prescribe antibiotics and local cold
 - Under general anesthesia perform a median laparotomy and appendectomy
 - To mobilize the appendix from inflammatory infiltrate and perform appendectomy
 - Perform disclosure and drainage of the infiltrate
 - All the answers wrong
8. For the acute appendicitis, complicated by appendicular infiltrate, in contrast to the tumour of caecum, is characteristic:
- * Tendency to diminishing of the tumour in the process of supervision
 - Long-term anamnesis
 - Excretion of blood from rectum
 - Curvassier's sign
 - Frequent partial intestinal obstruction in anamnesis
9. For the differential diagnostics of acute appendicitis with the urology diseases is not used
- * Irrigoscopy
 - Urography
 - Cystochromoscopy
 - X-ray of kidneys
 - Urine analysis
10. In patient during the operation for acute destructive appendicitis the appendicular infiltrate was diagnosed. What the further tactic of surgeon?
- * Appendectomy not performed, draining the abdominal cavity
 - Appendectomy not performed, to suit the wound
 - Performed appendectomy
 - Performed a cystostomy
 - To remove the infiltrate
11. In patient who underwent the operation of appendectomy on the 5th day at rectal examination was found the inflammatory infiltrate in small pelvis without the signs of abscessing. Choose the necessary medical tactic:
- * Conservative treatment
 - Punction of infiltrate
 - Punction of infiltrate and draining.
 - Discharging of infiltrate
 - Operative treatment.
12. In the diagnostics of pelvic appendicitis the most valuable is:
- * rectal and vaginal examination
 - laboratory analyses
 - laparocentesis
 - laparoscopy
 - colonoscopy

13. In the patient after appendectomy on the 5th day appeared the hectic fever, leucocytosis. In the depths of the pelvis appeared moderate pain, disuric phenomenon, tenesmi. What additional method of investigation you will begin with to diagnose the complication?
- * A digital rectal examination
 - Cystochromoscopy
 - Repeat tests of blood and urine
 - Rectoromanoscopy
 - Repeat irrigoscopy
14. In the patient on the 7th day after the operation for gangrenous appendicitis, was developed a fever, pain in the rectum, tenesmi, frequent and painful urination. During rectal examination revealed the infiltrate in pelvis. During 3 days after the treatment, which included warm enemas and antibiotics, the patient's condition had not improved. Repeat examination revealed a softening of rectal infiltration. Temperature had hectic nature. What is the treatment?
- * Drainage of pelvic abscess through the rectum
 - Antibiotics, physiotherapy, hyperbaric oxygen therapy
 - Drainage of pelvic abscess through retroperitoneal access
 - Laparotomy, opening abscess
 - Laparoscopic drainage of abscess
15. In the patient was diagnosed the retroperitoneal phlegmon as a complication of acute appendicitis. What is the cause of the phlegmon?
- * Retroperitoneal location of appendix
 - Subhepatic location of appendix
 - Local peritonitis in the right iliac region
 - Medial location of appendix
 - Location of appendix laterally from caecum
16. In the patient, aged 18, on the 7th day after the appendectomy developed a pelvic abscess. What will you do?
- * Drain the abscess through the front wall of the rectum
 - Massive antibiotic therapy
 - Drain the abscess by low-median access and drainage of small pelvis
 - Drain the abscess through the right iliac region
 - Drain the abscess by a right-side extraperitoneal access
17. In the patient, aged 40, on the 2nd day after the appendectomy for gangrenous appendicitis, developed intestinal paresis, fever, appeared pain in the right half of abdomen, enlarged liver and jaundice, signs of peritoneal irritation. What complications of acute appendicitis you can think about?
- * Pylephlebitis
 - Peritonitis
 - Subphrenic abscess
 - Intraintestinal abscess
 - Subhepatic abscess
18. In the patient, aged of 72, 14 hours ago appeared a permanent pain in a right iliac area. For last 2 hours the pain diminished. Acute appendicitis is diagnosed. What morphological form of acute appendicitis would result in diminishing of pain intensity?
- * Gangrenous
 - Phlegmonous
 - Catarrhal
 - Perforative

- E. Empyema of appendix
19. In the patient, on the 7th day after appendectomy was developed a pelvic abscess. What is your tactic?
- A. * Abscess drainage through the front wall of the rectum
 - B. To prescribe a massive doses of antibiotic
 - C. To drain the abscess by low-median access and drain a small pelvis
 - D. To drain the abscess by Volkovych-Dyakonov access
 - E. To drain the abscess by right-side extraperitoneal access
20. In the patient, who was operated for gangrenous appendicitis, on the 2nd day after the operation developed paresis of the intestine, jaundice, appeared fever and pain in the right side of abdomen. No signs of peritoneal irritation. What kind of complication of the disease developed?
- A. * Pylephlebitis
 - B. Subphrenic abscess
 - C. Peritonitis
 - D. Intraintestinal abscess
 - E. Subhepatic abscess
21. Is suspected for the patient's appendix. For the differential diagnosis of acute appendicitis with a perforated ulcer, 12 duodenal ulcer you apply?
- A. * The examination abdominal radiography
 - B. X-ray of gastrointestinal tract, irrigoscopy
 - C. Irrigoscopy, irrigography
 - D. Laparocentesis, colonoscopy
 - E. X-ray of the stomach with barium sulfate
22. On examination of the patient, the therapist suspected acute appendicitis, but is not confident in the diagnosis. The disease began six hours ago. What to do?
- A. * Urgent hospitalization of the patient in the surgical department
 - B. To recommend the surgeon consultation
 - C. Urgently send the patient to the clinic for further laboratory examination
 - D. As for the short time since the beginning of the disease, to recommend conservative treatment. The calm, local cold, antibiotics
 - E. Dynamic observation of patient in outpatient department
23. On the 3rd day after appendectomy you made the diagnosis - the diffuse peritonitis. What is your tactic?
- A. * To perform laparotomy
 - B. To enhance antibacterial therapy
 - C. To prescribe spasmolytics, analgetics
 - D. To perform angiographic examination
 - E. To perform laparoscopy
24. Patient B. was examined by urgent surgeon, who suspected acute appendicitis. The examination was prescribed. Which of the following investigations is the least helpful in the diagnosis of acute appendicitis?
- A. * Laparocentesis
 - B. Laboratory research (especially leukocytes)
 - C. Rectal examination
 - D. Axillary and rectal thermometry
 - E. The clinical examination to determine protective muscular tension.

25. Patient K. has the diagnosis: diffuse purulent peritonitis of appendicular origin. For the patient is indicated:
- A. * All mentioned is correct
 - B. Appendectomy and sanation of the abdominal cavity
 - C. Correction of fluid and electrolyte disorders
 - D. Antibacterial therapy
 - E. Total parenteral nutrition for 1-2 days after operation
26. The patient was diagnosed the diffuse peritonitis of appendicular origin. In this case used:
- A. * All mentioned
 - B. Median laparotomy
 - C. Appendectomy
 - D. Abdominal lavage
 - E. Drainage of the abdominal cavity
27. The patient was operated for acute phlegmonous appendicitis and diffuses peritonitis by means of the access to the right iliac region. On the 20th day after the operation was diagnosed right-side subphrenic abscess. What could cause its occurrence?
- A. * Was not used Fovler's position after the operation
 - B. Was not used Trendelenburg's position after the operation
 - C. Was not used Finsterer's position after the operation
 - D. Was not used Billroth 's position after the operation
 - E. Was not performed the tamponade of abdominal cavity
28. The patient, 76 years old, with myocardial infarction, is suspected for acute destructive appendicitis. What is your tactic?
- A. * Urgent operation
 - B. Monitoring and operation if the peritonitis develops
 - C. Prescription of massive doses of broad-spectrum antibiotics and if this therapy is ineffective – operation
 - D. Operation if the diagnosis confirms by laparoscopy
 - E. All the answers are wrong
29. The patient, aged 76, with concomitant pneumonia, was diagnosed phlegmonous appendicitis. What is your tactic?
- A. * Urgent operation
 - B. Monitoring and operation if the peritonitis develops
 - C. Prescription of massive doses of broad-spectrum antibiotics and if this therapy is ineffective – operation
 - D. Operation if the diagnosis confirms by laparoscopy
 - E. All the answers are wrong
30. The patient, is pregnant for 32 weeks, has stayed in the surgical department for 18 hours. During dynamic observation it is impossible to rule out acute appendicitis. What is your tactic?
- A. * Patient need to undergo the operation
 - B. Continue to observe the patient
 - C. Carry out abdominal ultrasonography
 - D. With gynecologist to perform abortion, and then appendectomy
 - E. To perform laparoscopy and to operate if the diagnosis confirms
31. The patient's T, at the ambulatory reception is suspected for acute appendicitis. What is advisable to do?
- A. * Immediately hospitalization of the patient
 - B. To prescribe spasmolytics and repeat examination of the patient in 4-6 hours

- C. The control of the dynamics of body temperature and leucocytosis for 12-24 hours
 - D. To prescribe the anti-inflammatory therapy and repeat examination on the next day
 - E. Observed the patient ambulatory and hospitalized in case of deterioration
32. The point through which the Volkovich-Dyakonov access is performed located on the line between anterior-superior process of the iliac bone and umbilicus:
- A. * between external and middle third
 - B. between external and internal third
 - C. in the internal third
 - D. in the middle
 - E. in external third
33. The pregnant woman (22 weeks) is delivered in urgent clinic with complaints of abdominal pain. The onset was acute, a day ago. Pain appeared in epigastrium, then in the right half of abdomen. There was a single vomiting. The abdomen painful above the right iliac area. Signs of peritoneal irritation are insignificant. Leucocytosis – 21?10⁹. What is the most probable diagnosis?
- A. * Acute appendicitis
 - B. Toxemia of pregnancy
 - C. Threatened abortion
 - D. Acute pancreatitis
 - E. Mesenteric thrombosis
34. You diagnosed and prescribed conservative treatment of appendicular infiltrate. What are the signs of abscessing of appendicular infiltrate?
- A. * All answers correct
 - B. Maintaining a high hectic temperature
 - C. Restoring or increase of pain
 - D. Signs of peritoneal irritation
 - E. Increase of leukocytosis with a shift to the left, the increase of ESR
35. You diagnosed in patient the acute appendicitis. What method of diagnostic is suitable?
- A. * All mentioned is correct
 - B. Laparoscopy
 - C. Clinical analysis of blood
 - D. Rectal examination
 - E. Thermography
36. You have diagnosed the appendicular infiltrate in the patient. What is the most expedient conservative treatment in first three days?
- A. * Antibiotics
 - B. Analgesics
 - C. U.H.F.-therapy
 - D. The warm on right iliac region
 - E. Parenteral use of proteolytic enzymes
37. You have diagnosed the gangrenous acute appendicitis in the patient. What symptoms are the basic for the diagnose?
- A. * Decrease of pain
 - B. Increase of pain
 - C. A sudden increase of pain in the right iliac region
 - D. Bradycardia
 - E. Mayo-Robson's sign in the right iliac region

38. You have to operate the patient with a typical picture of acute phlegmonous appendicitis. What access for appendectomy is the most suitable in this case?
- * Volkovych-Diakonov's access
 - Low-median laparotomy
 - Right-side pararectal access
 - Right-side transrectal incision
 - Transverse laparotomy above navel
39. You suspect in patient the chronic appendicitis. Which of the following methods of investigation can confirm the diagnosis?
- * Irrigoscopy
 - Colonoscopy
 - Laparocentesis
 - Rectoromanoscopy
 - X-ray of the abdominal cavity
40. You suspect the abscess of Douglas' pouch in the patient. What investigations should be carried out for its diagnosis?
- * A digital rectal examination
 - Rectoromanoscopy
 - Colonoscopy
 - Laparoscopy
 - Laparocentesis
41. You suspected acute appendicitis in patient. Diagnostic program includes:
- * All answers correct
 - Thorough anamnesis
 - To exclude all diseases that can simulate acute abdominal pathology
 - Rectal examination in men and vaginal additional examination in women
 - Laboratory tests
42. You suspected acute appendicitis. For the differential diagnosis of acute appendicitis from the right-side renal colic you apply:
- * Prescription of spasmolytics, excretory urography
 - Prescription of narcotic analgetics
 - Laparocentesis, laparoscopy
 - Fluoroscopy gastrointestinal, irrigoscopy
 - Angiography of renal arteries
43. Patient S., aged 56, was admitted to hospital with a diagnosis: acute catarrhal cholecystitis. What symptoms are not typical for this diagnosis?
- nausea
 - symptom Kera
 - * all responses venry
 - lack of muscle tension in the right hypochondrium
 - Musso symptom
44. During surgery at cholelithiasis detected wrinkled gallbladder filled with stones and advanced up to 2.5 cm common bile duct. Patients should
- * make cholecystectomy
 - perform cholecystectomy, then holangiography
 - immediately make an audit of cholecystectomy and duct
 - impose cholecystitis
 - duodenotomy make the audit of the major duodenal papilla

45. Patient A., 43, was admitted to hospital with a diagnosis: chronic cholecystitis calculary. What is the main method of research in this patient?
- A. infusion hography,
 - B. oral cholecystography,
 - C. * SONOGRAPHY
 - D. scanning of the liver,
 - E. computed tomography
46. Patient A., aged 45, was admitted to hospital with jaundice in the background cholelithias. The patient is indicate:
- A. emergency surgery
 - B. conservative treatment
 - C. * emergency operation after the preoperative
 - D. catheterization of the celiac artery
 - E. plasmapheresis
47. Patient A., aged 45, was admitted to hospital with jaundice in the background cholelithias. The patient is shown:
- A. emergency surgery
 - B. conservative treatment
 - C. * there is no right answer
 - D. catheterization of the celiac artery
 - E. plasmapheresis
48. Patient D., 63, operated by acute calculouse cholecystitis. Intraoperatively diagnosed gallbladder with concrements. What operation is indicated the patient?
- A. cholecystostomy
 - B. * cholecystectomy from cervical
 - C. cholecystectomy from the bottom
 - D. cholecystitis-enterovirus anastomosis
 - E. cholecystectomy with drainage choledochitis by Halstead-Pikovsky
49. Patient M., aged 56, was admitted to the hospital with a diagnosis: acute gangrenous cholecystitis. The patient is shown:
- A. conservative treatment
 - B. delayed operation
 - C. decision depends on the age of the patient
 - D. operation with no effect of conservative treatment
 - E. * Emergency operation
50. Patient M., aged 56, was admitted to the hospital with a diagnosis: acute gangrenous cholecystitis. The patient is shown:
- A. conservative treatment
 - B. delayed operation
 - C. decision depends on the age of the patient
 - D. operation with no effect of conservative treatment
 - E. * there is no right answer
51. Patient S., 41, was admitted to hospital with a diagnosis: obstructive jaundice. To diagnose the causes of jaundice is the subject of a more reliable:
- A. oral cholecystography
 - B. intravenous cholecystocholangiography
 - C. * retrograde cholangiography

- D. liver scintigraphy
 - E. direct hepatosplenography
52. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What analgesics is contraindicated?
- A. omnopon
 - B. * morphine hydrochloride
 - C. no-spa
 - D. spazgan
 - E. spazmalgon, baralgin
53. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What analgesics is contraindicated?
- A. omnopon
 - B. * no right answer
 - C. no-spa
 - D. spazgan
 - E. spazmalgon, baralgin
54. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What method is most informativve to clarify the diagnosis in the reception-office?
- A. infusion hology,
 - B. oral cholecystography,
 - C. * SONOGRAPHY
 - D. scanning of the liver,
 - E. computed tomography
55. Patient S., aged 56, was admitted to the hospital with a diagnosis: acute catarrhal cholecystitis. What symptoms are not typical for this diagnosis?
- A. nausea
 - B. Ker's sing
 - C. * Murphy's sing
 - D. absence of muscle tension in the right hypochondrium
 - E. Musso's sing
56. Patient V., aged 45, was admitted to hospital with a diagnosis: chronic cholecystitis. What is a reliable radiological signs of chronic cholecystitis?
- A. weak fluoroscopic shadow of the gall bladder
 - B. sharply increased, not reduced by giving choleretic breakfast
 - C. shadow of the gall bladder
 - D. disconnected" gallbladder
 - E. shadow of suspicion on concretions in the gall bladder at a reduced by 1 / 3 after the administration of choleretic breakfast
57. Patients after cholecystectomy in the immediate postoperative period gradually increases jaundice, these operating cholangiography not indicate the pathology of the bile ducts. The most likely cause of jaundice
- A. serum hepatitis
 - B. common bile duct stone
 - C. hemolytic jaundice
 - D. * operating choledochal injury (ligation)
 - E. all of the above

58. Patients after cholecystectomy in the immediate postoperative period gradually increases jaundice, these operating cholangiography not indicate the pathology of the bile ducts. The most likely cause of jaundice
- A. serum hepatitis
 - B. common bile duct stone
 - C. hemolytic jaundice
 - D. * There is no right answer
 - E. All of the above
59. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * abdominal X-ray
 - B. angiography
 - C. Doppler
 - D. celiocentesis
 - E. vulvotsentez
60. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * endoscopic retrograde cholangiography
 - B. angiography
 - C. doppler
 - D. laparocentesis
 - E. vulvocentesis
61. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * ultrasound abdominal
 - B. angiography
 - C. doppler
 - D. laparocentesis
 - E. vulvocentesis
62. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * intravenous cholecystocholangiography
 - B. angiography
 - C. doppler
 - D. laparocentesis
 - E. vulvocentesis
63. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic holegraphy
 - E. * hypotonic doudenography
64. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic holegraphy
 - E. * all answer are right

65. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- ultrasound
 - intravenous cholecystocholangiography
 - ERPHG
 - transhepatic holergraphy
 - * endoscopy
66. The patient K., 50 years old, six months after cholecystectomy performed at the calculouse chronic cholecystitis, again began to appear in the right hypochondrium pain, occasionally accompanied by yellowing of the sclera. Ultrasound examination of the abdominal cavity obvious pathology of the extrahepatic bile duct could not be detected. Which of the following methods is most informative for diagnosis in this case?
- infusion holergraphy,
 - oral cholecystography,
 - * endoscopic retrograde cholangiopancreatography,
 - scanning of the liver,
 - computed tomography
67. The patient K., 50 years old, six months after cholecystectomy performed at the calculouse chronic cholecystitis, again began to appear in the right hypochondrium pain, occasionally accompanied by yellowing of the sclera. Which of the following methods is most informative for diagnosis in this case?
- infusion holergraphy,
 - oral cholecystography,
 - * SONOGRAPHY
 - scanning of the liver,
 - computed tomography
68. The patient was diagnosed during surgery iatrogenic injury extrarenal bile ducts. What operation is indicated?
- closure of the injury duct separate atraumatic suture needle
 - suturing of the duct on the T-shaped drainage
 - closure of the duct on Γ -shaped drainage
 - imposition biliodigestiv anastomosis
 - * any of the above
69. The patient with suspected narrowing of the distal common bile duct for diagnosis you must:
- * endoscopic retrograde cholangiography
 - echocardiography
 - laparocentesis
 - certain fraction of bilirubin
 - definition level of blood flow in truncus coeliacus
70. The patient with suspected narrowing of the distal common bile duct for diagnosis you must:
- * there is no right answer
 - echo kardioskopiyu
 - laparocentesis
 - certain fraction of bilirubin
 - definition level of blood flow in truncus coeliacus
71. Patient R., 67 years old, enrolled in urgent hospital with a diagnosis: acute pancreatitis. What medications is necessary to appoint a patient?
- * Spasmodicals

- B. Venotonics
- C. Prostaglandins
- D. Peripheral vasodilators
- E. Hypnotics

72. Patient P., aged 68, was admitted to hospital with clinical acute pancreatitis. Which complications might arise?
- A. Thyrotoxicosis
 - B. Pyelonephritis
 - C. Wrapping Sigma
 - D. * Enzymatic peritonitis
 - E. Urination disorders
73. At a frequent „fat” chair with disseminations of undigested meat and permanent thirst, it is foremost necessary to think about:
- A. * Chronic pancreatitis
 - B. Chronic duodenitis
 - C. Chronic hepatocholecystitis
 - D. Ulcerous diseases of duodenum
 - E. Hepatocirrhosis
74. How often does the pancreatitis department the general bilious channel pass through the head of pancreas?:
- A. * 80-90 %
 - B. 90-100 %
 - C. 75-85 %
 - D. 50-60 %
 - E. 40-50 %
75. In obedience to classification complications of acute pancreatitis, after etiology and pathogeny, distinguish such complications, except for:
- A. Infectiously inflammatory
 - B. Enzymic
 - C. * Allergic
 - D. Mixed
 - E. Trombogemoragic
76. In operation for acute abdomen intraoperatively revealed a hemorrhagic effusion and foci of fat necrosis in the peritoneum. What kind of illness should think?
- A. rupture of the liver
 - B. * acute pancreatitis
 - C. hollow organ injury
 - D. mesenteric thrombosis
 - E. perforated gastric ulcer
77. In operation for acute abdomen intraoperatively revealed a hemorrhagic effusion and foci of fat necrosis in the peritoneum. What kind of illness should think?
- A. rupture of the liver
 - B. * there is no right answer
 - C. hollow organ injury
 - D. mesenteric thrombosis
 - E. perforated gastric ulcer

78. Name the optimum lines the operative interference concerning a acute pancreatitis after the beginning of disease:
- A. 7-8 days
 - B. 1-3 days
 - C. 1-5 days
 - D. 3-5 days
 - E. * Surgical treatment is not indicate
79. Patient K, aged 45, entered the reception room with suspected acute pancreatitis. What kind of research required to clarify the diagnosis?
- A. * Ultrasound
 - B. CT
 - C. MRI
 - D. Doppler
 - E. Scintigraphy
80. Patient K., aged 39, admitted to hospital with clinical acute pancreatitis. Which analgesic should not be used?
- A. Analgin
 - B. Baralgin
 - C. Ketanov
 - D. * Can all
 - E. Keterolak
81. Patient K., aged 39, admitted to hospital with clinical acute pancreatitis. Which analgesic should not be used?
- A. Analgin
 - B. Baralgin
 - C. Ketanov
 - D. * Morphine
 - E. Keterolak
82. Patient K., aged 45, entered the reception room with suspected acute pancreatitis. What kind of research required to clarify the diagnosis?
- A. * there is no right answer
 - B. CT
 - C. MRI
 - D. Doppler
 - E. Scintigraphy
83. Patient P., aged 68, was admitted to hospital with clinical acute pancreatitis. Which complications might arise?
- A. Thyrotoxicosis
 - B. Pyelonephritis
 - C. Wrapping Sigma
 - D. * Omental abscess
 - E. Urination disorders
84. Patient P., aged 68, was admitted to hospital with clinical acute pancreatitis. Which complications might arise?
- A. Thyrotoxicosis
 - B. Pyelonephritis
 - C. Wrapping Sigma
 - D. * Retroperitoneal phlegmon

E. Urination disorders

85. Patient P., aged 68, was admitted to hospital with clinical acute pancreatitis. Which complications might arise?
- A. Thyrotoxicosis
 - B. Pyelonephritis
 - C. Wrapping Sigma
 - D. * No right answer
 - E. Urination disorders
86. Patient R., 67 years old, enrolled in urgent hospital with a diagnosis: acute pancreatitis. What medications is necessary to appoint a patient?
- A. * Cytostatics
 - B. Venotonics
 - C. Prostaglandins
 - D. Peripheral vasodilators
 - E. Hypnotics
87. Patient S., aged 65, went to urgent hospital with a diagnosis: acute pancreatitis. What medications is necessary to appoint a patient?
- A. * H2 - Blockers
 - B. Venotonics
 - C. prostaglandins
 - D. Peripheral vasodilators
 - E. Hypnotics
88. Patient S., aged 65, went to urgent hospital with a diagnosis: acute pancreatitis. Which disease is necessary to differentiate this pathology?
- A. * Acute cholecystitis
 - B. Thyrotoxicosis
 - C. Thrombophlebitis of subcutaneous veins
 - D. Hernia
 - E. Cystitis
89. Patient S., aged 65, went to urgent hospital with a diagnosis: acute pancreatitis. Which disease is necessary to differentiate this pathology?
- A. * Thrombosis of mesenteric vessels
 - B. Thyrotoxicosis
 - C. Thrombophlebitis of subcutaneous veins
 - D. Hernia
 - E. Cystitis
90. Patient S., aged 65, went to urgent hospital with a diagnosis: acute pancreatitis. Which disease is necessary to differentiate this pathology?
- A. * Perforated ulcer
 - B. Thyrotoxicosis
 - C. Thrombophlebitis of subcutaneous veins
 - D. Hernia
 - E. Cystitis
91. Patient T., age 37, was admitted to hospital with a diagnosis: acute pancreatitis. For the dektoksikatsii patient displayed:
- A. * Plasmapheresis
 - B. Perirenal blockade

- C. Application antisecretion drugs
 - D. Epidural anesthesia
 - E. Using antispasmodics
92. Patient T., age 37, was admitted to hospital with a diagnosis: acute pancreatitis. For the dektoksikatsii patient displayed:
- A. * Lymphsorbition
 - B. Perirenal blockade
 - C. Application antisecretion drugs
 - D. Epidural anesthesia
 - E. Using antispasmodics
93. Patient with acute pancreatitis revealed a sharp cyanosis of the face. It is a symptom of the author?
- A. * Lagermf's
 - B. Mondor's
 - C. Halstead's
 - D. Gray – Turner's
 - E. Kulen's
94. Patient with acute pancreatitis revealed cyanosis lateral parts of abdominal skin. It is a symptom of the author?
- A. Lagermf's
 - B. Mondor's
 - C. Halstead's
 - D. * Gray – Turner's
 - E. Kulen's
95. Patient with acute pancreatitis revealed cyanosis of skin of the abdomen. It is a symptom of the author?
- A. Lagermf's
 - B. Mondor's
 - C. * Halstead's
 - D. Gray – Turner's
 - E. Kulen's
96. The patient admitted to hospital with a diagnosis: biliary pancreatitis. What type of treatment is shown to the patient.
- A. Conservative treatment in hospital
 - B. Outpatient
 - C. * Surgical treatment
 - D. Observation of the surgeon at home
 - E. Treatment not indicated
97. The patient admitted to hospital with a diagnosis: biliary pancreatitis. What type of treatment is shown to the patient.
- A. Conservative treatment in hospital
 - B. Outpatient
 - C. * No right answer
 - D. Observation of the surgeon at home
 - E. Treatment not indicated
98. The patient after intake of alcohol appeared repeated vomiting, epigastric pain of herpes character. Diagnosis

- A. Mallory-Weiss syndrome
 - B. Gastric ulcer
 - C. bleeding from varices of the esophagus
 - D. Crohn diseases
 - E. * acute pancreatitis
99. The patient after intake of alcohol appeared repeated vomiting, epigastric pain of herpes character. Diagnosis
- A. Mallory-Weiss syndrome
 - B. Gastric ulcer
 - C. bleeding from varices of the esophagus
 - D. Crohn diseases
 - E. * there is no right answer
100. The patient with acute pancreatitis during the study revealed the absence of pulsation of the abdominal aorta in the epigastrium. It is a symptom of the author?
- A. Lagermf's
 - B. Mondor's
 - C. Halstead's
 - D. Gray – Turner's
 - E. * Voskresenky
101. The patient with acute pancreatitis revealed purple spots on the face and body. It is a symptom of the author?
- A. Lagermf's
 - B. * Mondor's
 - C. Halstead's
 - D. Gray – Turner's
 - E. Kulen's
102. The patient with acute pancreatitis showed a yellow color of the skin near the navel. It is a symptom of the author?
- A. Lagermf's
 - B. Mondor's
 - C. Halstead's
 - D. Gray – Turner's
 - E. * Kulen's
103. 3 hours before admission, the patient appeared "stabbing" pain in the epigastric region. After 1 hour epigastric pain had decreased slightly, but the patient notes that there were sharp pains in the right iliac region. What kind of complications of peptic ulcer can think of?
- A. * Perforation of gastric ulcer
 - B. Malignancy ulcers
 - C. Pyloristhenosis
 - D. Ulcer Penetration
 - E. All answers are correct
104. A man 46 years complain for the expressed general weakness, attacks of palpitation, pain in an epigastrium, which arise up in 10-15 minutes after the reception of meal. During 10 years is ill ulcerative disease of stomach. 2 years ago the executed resection of stomach after Bilroth-II. At inspected a stomach is soft. Sickly in an epigastrium and pyloro-duodenal area. What complication most probably did arise up for a patient?
- A. * Dumping is a syndrome
 - B. Cholecystitis

- C. Peptic gastric ulcer
 - D. Chronic pancreatitis
 - E. Gastritis of stump of stomach
105. A patient 76 years old came to the hospital on seventh twenty-four hours from the beginning of disease with complaints about pain in right hypochondrium, weakness, repeated vomiting, temperature up to 38 degrees. At an examination is a general of middle weight. Pallor. An enlarged gall-bladder is palpated in right hypochondrium where tension is examined. What method of treatment?
- A. * Cholecystostomy under sonography control
 - B. Diet therapy
 - C. Conservative treatment
 - D. Spasmolytics
 - E. Analgesics
106. Choose the most frequent reason of relapse of ulcer after an organ-saving operation with vagotomy at the adequate decline of acid production after an operation:
- A. * syndrome of Zollinger-Ellison
 - B. incomplete vagotomy
 - C. duodeno-gastric reflux of bile
 - D. gastritis of the operated stomach
 - E. excessive use of alcohol
107. During an operation for a patient 68 years old concerning an acute destructive cholecystitis certain presence of festering cholangitis. At the revision of choledochus the not found concretions and sign of stenosis duodenal to the nipple. Cholecystectomy is executed. How is it necessary to make off an operation?
- A. * To execute the external catchment of choledochus
 - B. To impose supraduodenal choledochoduodenostomy
 - C. To execute a transduodenal choledochoduodenostomy
 - D. To execute a choledochojejunostomy
 - E. To drain an abdominal cavity
108. During an operation on occasion of acute calculus cholecystitis the extended general biliary channel is founded. After cholecystectomy through stump of cystic duct is performed cholangiography. On X-Ray of hepatic is founded general bile ducts. In the distal part of choledochus a precipice of contrasting matter is with even edges. A contrast does not act in a duodenum. How to estimate data of intraoperative cholangiography?
- A. * Present of stones in the distal part of choledochus
 - B. Stenosis of duodenal papilla
 - C. Cicatrix narrowing of distal part of choledochus
 - D. Cholecystitis
 - E. Tumour of head of pancreas
109. For a 38-year-old patient after carried two years ago back cholecystectomy recommenced attacks of hepatic colic with turning of skin covers yellow. A postcholecystectomy syndrome is diagnosed. What is most frequent reason of this situation?
- A. * Leave during the first operation concretions
 - B. Stenosis papillitis
 - C. Duodenostasis
 - D. Diskinesia of bile ducts
 - E. Cholestasis

110. For a patient the decompensate stenosis of pyloro-bulb area is on a background 10 years of ulcerative anamnesis. In the state of middle weight he acted with complaints about the frequent vomiting and belch rotten. Marks the expressed weakness. Exhausted. After stabilizing of metabolic indexes and general a patient gave a consent to operative treatment. There are endoscopic signs of the expressed gastritis in all parts of stomach. Choose the method of operation :
- A. * a resection of stomach by Bilioth II
 - B. vagotomy is with an operation which drains a stomach
 - C. imposition of gastrostomy by Vitsel
 - D. selective proximal vagotomy
 - E. a fundoplication by Nissen
111. For a patient 45 years, in 2 years after cholecystectomy there were pains in right hypochondrium, icteric and itch of skin, bitter taste in a company. At sonography of choledohus a 1,2 cm, in distal part located stone. What is the diagnosis?
- A. * Choledoholitiasis. Obturative icterus
 - B. Cholangitis. Mechanical icterus
 - C. Acute pancreatitis. Mechanical icterus
 - D. Postcholecystectomy syndrome
 - E. Tumour of head of pancreas. Mechanical icterus
112. For a patient 48 years with a giant gastric ulcer with recrudescence motion (has ulcerative disease during 11 years) a histamine-reflectory an-acidity is founded. What medical tactic?
- A. * Conservative treatment in hospital and further sanatorium-resort treatment
 - B. Stationary treatment
 - C. Anti-ulcerative therapy is before a planned operation
 - D. Urgent operation
 - E. Stationary conservative treatment during 8 weeks
113. For a patient 60 years icterus during 3 weeks, intensity of it began without pain grows. A stomach at palpation is soft. Positive symptom of Kurvuasie. At sonography of bilious ways expansion of gall-bladder is marked and choledohus. What primary cause of these changes.
- A. * Cancer of head of pancreas
 - B. Bile-stone disease
 - C. Chronic pancreatitis
 - D. Infectious hepatitis
 - E. Cancer of liver.
114. For a patient which carried the resection of stomach a year ago, the attacks of weakness, which arose up on an empty stomach or in 15-20 minutes after the reception of meal, appeared. Attacks were accompanied by a muscle weakness, headache, decline of sugar of blood, decline of arteriotony, sometimes by dizziness. What complication did arise up for a patient?
- A. * Hypoglycaemia syndrome
 - B. Food allergy
 - C. Dumping-syndrome
 - D. Postresection asthenia
 - E. Syndrome of small stomach
115. For a patient C., 58 years, increased feed, after will attack pains the icteric of skin and conjunctiva of sclera, discoloured excrement, darkening of urine appeared in right hypochondrium. A stomach is exaggerated, at palpation sickly in right hypochondrium, a enlarged liver is palpated on a 2-3 cm. In blood general bilirubin 90 mmole/L, direct 60 micromole/L. What is the best examination method?
- A. * Retrograde cholangiopancreatography

- B. Intravenous cholegraphy
 - C. Transcutaneous cholegraphy
 - D. Transcutaneous transhepatic cholegraphy
 - E. Sonography of hepatic region
116. For a patient, which carried an operation, resection of stomach, concerning ulcerative disease, there was a relapse (endoscopy data). What reason of relapse?
- A. * all is transferred
 - B. incomplete vagotomy;
 - C. leave at a resection part of antrum;
 - D. economy resection of stomach;
 - E. syndrome of Zollinger-Ellison;
117. For a patient, which carried the resection of stomach for Bilroth-II concerning ulcerative disease of duodenum is marked complaints about a acute weakness, attacks of palpitation, waves of heat to the face and dizziness at the reception of sweet and suckling meal. Deficit of b.w. - 15kr. On X-Ray of stump of stomach of small sizes with evacuation of barium in the extended efferent bowel. What operatively interference does need it was to be before done to the patient?
- A. * SPV with a pyloroplasty
 - B. Jejunogastroplasy
 - C. Resection of stomach by Rou
 - D. Trunk vagotomy
 - E. Resection of stomach
118. For the exhausted man of 42, which 15 years suffers ulcerative disease of duodenum, during the last two weeks the massive vomiting takes place every evening. At X-Ray stenosis of goalkeeper of stomach is founded. The above-mentioned state became complicated by cramps. what complication did arise up for a patient?
- A. * Tetany
 - B. Bleeding
 - C. Perforation
 - D. Penetration
 - E. Malignancy
119. For the patient of 32 after carried cholecystectomy the attacks of hepatic colic, which are accompanied by turning of skin canopies yellow, recommenced in 6 months. At sonography examination the leave concrements of 2 x is founded 1cm in distal part of choledohus. What treatment for a patient will be optimal?
- A. * Endoscopic duodenocholedohoscopy with papillosphincterotomy
 - B. Conservative therapy by spasmolitics
 - C. Lithotripsy
 - D. Choledolithotomy
 - E. Choledohoduodenoanastomosis
120. For the patient of 63, in a year after cholecystectomy with choledoho-duodenal anastomosis, there were pains in right hypohondrium, icterus, temperature, fever, dryness and bitter taste. What is the previous diagnosis?
- A. * Cholangitis
 - B. Acute pancreatitis
 - C. Duodenitis. Mechanical icterus
 - D. Stricture of choledohus
 - E. Hepatitis is with transformation in a cirrhosis

121. In the ward the patient brought in an unconscious state, with periodic bouts of clonic seizures. According to relatives, the patient for many years suffered from a disease of the stomach. Last month the patient had copious vomiting every day, and he lost considerable weight. On examination, the patient exhausted, dehydrated, in the epigastric region has pigmentation, is determined by the splashing. What method of research to clarify the diagnosis shows the patient after stabilization of the state?
- ECG
 - CT
 - MRI
 - * X-ray study of the abdominal cavity with the passage of barium
 - Ultrasound
122. Operative treatment with a duodenal ulcer it is indicated a patient in those cases when: 1. often there are relapses of disease, 2. a disease becomes complicated continuous bleeding, 3. there is pyloro-duodenal stenosis 4. there is a perforation of ulcer, 5. ulcer penetrates in the head of pancreas, giving the frequent intensifying and phenomena of pancreatitis. Choose the best composition:
- * all true.
 - 1,4
 - 2,3
 - 3,4
 - 1,2
123. Patient 57 years, during the last 10 years has a chronic calculus cholecystitis. Intensifying of disease take place 1-2 times on a year. The last attack began 8 days ago. The icteric skin and sclera appeared in 2 days. During sonography of inspection of organs of abdominal region: thickness of wall of gall-bladder up to 5 mm, present concrements 0,5 to 1,0 cm in a diameter. A width of choledohus is a 1,2 cm, in it supraduodenal part a stone is founded up to 1,0 cm. What complication of gallbladder disease did arise up for a patient?
- * Choledoholitis. Mechanical icterus
 - Acute cholangitis
 - Acute calculus cholecystitis
 - Choledoho-duodenal fistula
 - Cancer of bilious ducts
124. Patient of 51, operated a year ago concerning the perforative ulcer of duodenum. Done barrel vagotomy is with excision of ulcer. Complain for periodic appearance of liquid stool, which arises up suddenly (for two three times on twenty-four hours within a week), whereupon normalized independently. Define a diagnosis.
- * Postvagotomy diarrhoea
 - Chronic pancreatitis
 - Dumping is a syndrome
 - Chronic colitis
 - Poliposis of colon
125. Patient S., 27 years old admitted to clinic with the diagnosis: perforated ulcer. What study should be carried out in the first place?
- ECG
 - CT
 - MRI
 - * Survey radiography of the abdominal cavity
 - Ultrasound

126. The patient 32 years, two years ago passed cholecystectomy concerning gallbladder disease. There were many shallow concrements in a gallbladder. During six months the attacks of hepatic colic recurred 3 times. Two days ago back after an attack, turning of skin and sclera appeared yellow. Bilirubin of 90 mmole/L. What examination most effective in diagnose?
- A. * sonography of pancreatoduodenal area
 - B. X-Ray of gastrointestinal tract
 - C. Endoscopy
 - D. Cholangiography
 - E. Angiography
127. The patient of 42 four years ago carried the resection of stomach after Hoffmaister-Finsterer concerning the ulcer of duodenum with penetration in a pancreas. During the last year marks pain in an epigastrium and right hypochondrium. A pain syndrome is accompanied massive vomiting by stagnant content with plenty of bile. After vomiting pain is decreased. The repeated treatment of improvement did not bring. A patient became thin on 12 kg. What is the diagnosis?
- A. * Syndrome of afferent loop
 - B. Tumour of stump of stomach
 - C. Chronic pancreatitis
 - D. Relapse of ulcer
 - E. Peptic ulcer of gastroenteroanastomosis
128. The patient of 62 complains in the presence of icterus, itch of skin, light excrement, wet dark. Objectively: a sclera and skin is icteric. An enlarge, painless gallbladder is palpated. Blood test: general bilirubin of 85 mmol/L. What method of examination more expedient in all to use for clarification of diagnosis?
- A. * Retrograde cholangiopancreatography
 - B. Duodenal sounding
 - C. Survey sciagraphy of organs of abdominal region
 - D. Oral cholecystography
 - E. Cholegraphy
129. The patient of H., 62, acted with complaints about pain in the overhead half of stomach after the food intake, heartburn, nausea, decline of mass of body on 15 kg for a year, constipations. Three to the year that is why operated concerning a perforative gastric ulcer, executed resection of stomach. In a year after an operation pain recommenced in the overhead half of stomach, heartburn, periodically there was vomiting by a meal, the state of patient became worse in course of time. Your previous diagnosis?
- A. * Peptic ulcer of gastroenteroanastomosis
 - B. Syndrome of afferent loop
 - C. Cancer of stump of stomach
 - D. Gastritis of the operated stomach
 - E. Chronic pancreatitis
130. The patient with a history of ulcerative while examining the abdomen indicated the disappearance of liver dullness. What is the diagnosis should be suspected in a patient?
- A. Perforated ulcer
 - B. Malignancy ulcers
 - C. * Ulcer bleeding
 - D. Ulcer Penetration
 - E. All answers are correct
131. The patient with a history of ulcerative with X-ray determined by a deep niche, located on the back wall near the small curvature of the antrum. What diagnoses are in the patient?
- A. Perforated ulcer

- B. Malignancy ulcers
 - C. Ulcer bleeding
 - D. * Penetration ulcers
 - E. All answers are correct
132. The patient, 30 years old, was admitted to the surgical department after 2 hours from the moment of perforation of gastric ulcer. The diagnosis beyond doubt, but the patient categorically refused the operation. Your tactics.
- A. Outpatient
 - B. Forced to operate on a patient
 - C. * No right answer
 - D. Transfer the patient in therapy department
 - E. Translate patient infectious disease clinic
133. The patient, 33 years old, with no ulcer history admitted to the surgical department with the diagnosis: perforated gastric ulcer. In the operation revealed perforation hole 0,5 ? 0,5 cm on the lesser curvature of the stomach, without inflammatory infiltration around. What should be the volume of transactions?
- A. Resection of gastric Billroth I
 - B. Resection of gastric Billroth II
 - C. * No right answer
 - D. The operation is not shown
 - E. Gastrostomy
134. The patient, 34 years old, 6 years ago was silent ulcer perforation duodenum. Two years after this the patient felt well and do not appealed. Then came the pain, typical of peptic diseases. What method of research is the most informative?
- A. ECG
 - B. * ENDOSCOPY
 - C. X-ray
 - D. CT
 - E. MRI
135. The patient, 35 years old, admitted to emergency room, diagnosed a bleeding stomach ulcer. Where the patient must treatment?
- A. Therapeutic department
 - B. Surgery
 - C. * Intensive Care Unit
 - D. Orthopedics
 - E. Day hospital
136. The patient, 35 years old, was admitted to the surgical department with ulcerative gastric hemorrhage. What medications is necessary to appoint a patient?
- A. * Preparations of blood
 - B. Antibiotics
 - C. Vasodilators
 - D. Prostaglandins
 - E. Veintonics
137. The patient, 36 years old, 12 years suffer stomach ulcer Conservative treatment is not effective. What tactics are indicating for treating a patient?
- A. Continue medical treatment
 - B. * No right answer
 - C. Outpatient

- D. Infusion therapy
 - E. Not require treatment
138. The patient, 40 years old, long-suffering with stomach ulcer, said that the last 2 days the pain had become less intense, but at the same time it has been increasing weakness, dizziness. In morning for a few seconds to lose consciousness. On examination, pulse 100 in 1 min. The patient was pale. In the epigastric region is very little pain. No symptoms of irritation of the peritoneum. Where You sent for treatment patient?
- A. Therapeutic department
 - B. Surgery
 - C. * Intensive Care Unit
 - D. Orthopedics
 - E. Day hospital
139. The patient, 42 years old, gastric ulcer in existence for 10 years. After treatment in hospital advanced remission. Three months ago, the patient appeared pain in the lumbar region, sometimes wearing herpes character. What study should be used to refine the diagnosis?
- A. * X-ray study of the digestive tract with barium
 - B. Total blood
 - C. Urinalysis
 - D. Rheovasography
 - E. Doppler
140. The patient, 42 years old, gastric ulcer in existence for 10 years. After treatment in hospital advanced remission. Three months ago, the patient appeared pain in the lumbar region, sometimes wearing herpes character. What study should be used to refine the diagnosis?
- A. * No right answer
 - B. Total blood
 - C. Urinalysis
 - D. Rheovasography
 - E. Doppler
141. The patient, aged 39, suffering from stomach ulcer, recently had a feeling of heaviness in the epigastric region, which is usually only after vomiting. The patient has lost much weight. What method of diagnosis should be used to refine the diagnosis?
- A. ECG
 - B. Ultrasound
 - C. * X-ray study of the abdominal cavity with the passage of barium
 - D. CT
 - E. MRI
142. The patient, aged 48, suffering for years with stomach ulcer, said that the pain had not become dependent on food intake, have become permanent and less intense. Noted weakness, malaise, fatigue. Lost 5 kg. ESR 40 mm / hour. What kind of complications the peptic ulcer can think of?
- A. Perforated ulcer
 - B. * Malignancy ulcers
 - C. Pyloristhenosis
 - D. Ulcer Penetration
 - E. All answers are correct
143. Woman 55 years, complains on pain in right hypochondrium, temperature up to 39C, darkening of colour of urine. Objectively: skin and visible mucous membranes icteric, liver enlarged, dense, sickly at palpation. In the global analysis of blood of increase of ESR,

neutrophilic leukocytosis, shift to the left, increase of level of bilirubin, ALT, AST. Sonography are stones of gall-bladder. What complication did arise up for this patient?

- A. * Cholangitis
- B. Empyema of gall-bladder
- C. Chronic cholecystitis
- D. Acute cholecystitis
- E. Sepsis

144. Woman of 53, three years ago back carried cholecystectomy concerning a acute calculus cholecystitis. After operation already four times were twinges in right hypochondrium, which lasted 6-8 hours, passed after spasmolytics. After the second attack marked turning of skin and sclera yellow. What examination will be most informing for imputing of diagnosis?

- A. * Sonography
- B. X-Ray of stomach
- C. Endoscopy
- D. Cholangiography
- E. Thermography

145. After the clinical and instrumental research you have diagnosed acute intestinal obstruction, and during laparotomy revealed that it is caused by volvulus. The operation is performed after 3 hours of the disease onset. The incarcerated gut is of dark color, peristalsis is absent. What is the most appropriate surgical tactics?

- A. * To perform intestinal detorsion, resection of the incarcerated region with anastomosis
- B. To perform intestinal detorsion with intestinoplication
- C. To perform intestinal detorsion with enterostomy
- D. To perform intestinal detorsion with intubation
- E. To perform intestinal detorsion

146. Female 50 years old, who had been operated for uterine fibroma (hysterectomy), entered with complaints of abdominal pain, vomiting. On the X-ray of abdomen revealed the signs of small bowel obstruction. What is the cause of obstruction?

- A. * Adhesive disease
- B. Strangulated inguinal hernia
- C. Carcinoma of the colon
- D. Cancer of the small intestine
- E. Diverticulitis

147. In patient 18 years old, appeared cramping abdominal pain, vomiting, constipation and gas delay. On abdominal X-ray revealed "Kloiber's cups. What kind of acute abdominal disease can we speak about?

- A. * Intestinal obstruction
- B. Flatulence
- C. Bad preparation of the patient for examination
- D. Enterocolitis
- E. Helminthic invasion

148. In the clinic entered the patient, aged 72, complaining of severe cramping abdominal pain, constipation and gas delay. Was a single vomiting. The disease began 5 hours ago. From anamnesis: last year was suffering from constipation. On examination: skin is pale, pulse - 116/min, AP -110/80 mm Hg. Heart sounds are weak. The tongue is dry. The abdomen is asymmetrical. A marked flatulence in the left iliac region. Positive Sklyarov's, Kivul's signs, "the falling drop sound". Rectal ampoule is empty, enlarged, with reduced sphincter tone. On

X-ray - high Kloiber's cups. During the enema fluid flows back, increasing the pain. What is the primary diagnosis?

- A. * Acute obstructive large intestine obstruction
- B. Acute obstructive small intestinal obstruction
- C. Spastic ileus of large intestine
- D. Paralytic ileus
- E. Abscess of Douglas' pouch

149. In the department of surgery entered the patient who complained of cramping abdominal pain, which appeared suddenly 3 hours ago, repeated vomiting. 6 hours ago ate a pound of walnuts. On examination: abdomen is distended symmetrically, on auscultation - strengthening the peristaltic tones. On X-ray of the abdominal cavity multiple horizontal air-fluid levels (the width of the horizontal level of the fluid is greater than the height of the gas). What is your primary diagnosis?

- A. * Acute obstructive intestinal obstruction
- B. Acute obstructive large intestine obstruction
- C. Acute paralytic intestinal obstruction
- D. Acute large intestine paralytic ileus
- E. Chronic paralytic intestinal obstruction

150. In the patient A., 79 years old, was diagnosed acute intestinal obstruction and concomitant cardiac pathology. Pulse 103/min AP - 95/60 mm. Hg. After stabilization of hemodynamic parameters the patient was taken for operation, where. revealed a volvulus of sigmoid colon. After detorsion the bowel is dark, peristalsis and vascular pulsation is absent. What is the optimal operation should be done in this case?

- A. * Resection of the sigmoid colon with colostomy
- B. Resection of the sigmoid colon with primary anastomosis
- C. Transanal intubation of the colon
- D. Drainage of the abdominal cavity
- E. Colectomy

151. In the patient was diagnosed the 3rd phase of acute intestinal obstruction with the signs of multiple organ failure and lack of volume of circulating fluid to 25 %. Where does the fluid accumulate first of all?

- A. * In the lumen of the intestine
- B. In the abdominal cavity
- C. In the pleural cavity
- D. In the tissues of the body
- E. In the intercellular space

152. In the patient, who underwent the operation for peritonitis of appendicular origin, on the 4th day appeared flatulence, constipation and gas delay. By percussion tympanitis all over the abdomen, by auscultation - peristalsis is absent, pain is not determined. What is the cause for this condition.

- A. * Paralytic ileus
- B. Strangulated ileus
- C. The abscess of abdominal cavity
- D. Pylephlebitis
- E. Thrombosis of mesenteric vessels

153. In the surgical department entered the patient with complaints of sudden cramping pain in the abdomen, its asymmetry, which arose suddenly after a food abuse, delay of stool and gases. By palpation the abdomen is soft, without signs of peritoneal irritation. In the abdominal cavity

in the left half detected the movable, painful, tumor formation, by elastic consistency. What is the primary diagnosis?

- A. * Sigmoid volvulus
- B. Obstipation syndrome
- C. Acute pancreatitis
- D. Stomach tumor
- E. Rectal cancer

154. In the surgical department in urgent way entered the woman 25 years old with the signs of acute intestinal obstruction. From anamnesis: the patient had operation for ovarian the right apoplexy. The last 1,5 years after the operation flatulence and pain constantly bothered the patient. What has caused the intestinal obstruction in the patient.

- A. * Adhesions in the abdominal cavity
- B. Diverticulum of ileum
- C. Dolihosigma
- D. Appendicular infiltrate
- E. Tumor of the colon

155. In the university hospital entered the patient with a diagnosis of intestinal obstruction. From anamnesis: weight loss (10 kg for 3 months), weakness and occasionally blood in the stool. What is the cause of obstruction?

- A. * Colon cancer
- B. Adhesive disease of peritoneum
- C. Helminthic invasion
- D. Ulcerative colitis
- E. Coprosthesis

156. On abdominal X-ray of the patient is determined several sites of enlightenment of hemispheric shape with the clear horizontal level. What causes such X-ray picture?

- A. * Intestinal obstruction
- B. Perforated ulcer
- C. Flatulence
- D. Colon cancer
- E. Acute pancreatitis

157. Patient 43 years old, who had been operated for acute cholecystitis, entered the clinic with complaints of intense cramping abdominal pain, repeated vomiting and absence of stool. AP - 90/60 mm Hg. The abdomen is moderately distended, soft, slightly painful. Sklyarov's sign is positive Shchetkin - Blumberg sign is slightly positive. What is the primary diagnosis?

- A. * Acute adhesive intestinal obstruction
- B. Perforation of malignant tumor of the stomach
- C. Mesenteric thrombosis
- D. Acute pancreatitis
- E. Perforation of colon intestine

158. Patient 55 years complains of severe cramping abdominal pain, nausea, vomiting, constipation and gas delay. Has been ill for 6 hours. Had been operated for traumatic injury of spleen. On examination: pulse 84/min. The tongue is moderately wet. The abdomen distended with asymmetry due to the increase in the left half. Defined the "splashing sound". Peristaltic sounds periodically amplified. On X-ray of the abdominal cavity - Kloiber's cups. What is the primary diagnosis?

- A. * Acute adhesive intestinal obstruction
- B. Obstructive ileus (tumor genesis)
- C. Thrombosis of mesenteric vessels

- D. Ulcerative colitis
 - E. Sigmoid volvulus
159. Patient B, aged 68, entered the University Hospital after 2 days from the onset of the disease with signs of acute intestinal obstruction. On X-ray - high Kloiber's cups. During enema the fluid flows back, increasing the pain. Indicate the level of intestinal obstruction.
- A. * Low large intestine obstruction
 - B. High small intestinal obstruction
 - C. Low small intestinal obstruction
 - D. Obstruction of the initial part of the colon
 - E. Strangulated ileus
160. Patient K., aged 23, was hospitalized with complaints of nausea, vomiting, cramping pain in the right half of the abdomen. Appendectomy in anamnesis. Pulse - 96/min. AP - 110/70 mm Hg. Abdomen distended, asymmetrical due to protrusion of the right half, over which is determined "splashing sound". The peristalsis strengthened. Signs of peritoneal irritation are absent. Rectal examination: sphincter tone maintained, ampoule dilated. Your diagnosis?
- A. * Acute adhesive intestinal obstruction
 - B. Acute obstructive ileus
 - C. Acute pancreatitis
 - D. Acute adnexitis
 - E. Food poisoning
161. Patient M, 66 years old, arrived from the district hospital at 10 o'clock with complaints of severe abdominal pain, nausea, constipation and gas delay. The disease started suddenly, at 4 o'clock in the morning, with strong cramping abdominal pain. After one hour of the onset the patient entered to the district hospital, and after 5 hours transferred to the regional surgical department. After properly preparing the patient underwent the operation the acute intestinal obstruction. After wide laparotomy, the nodule was found out. Intestinal loops in knot of a dark-brown color, peristalsis and vascular pulsation is absent. What kind of acute intestinal obstruction you deal with?
- A. * Strangulated
 - B. Obstructive
 - C. Spastic
 - D. Mixed
 - E. Paralytic
162. Patient M., aged 25, was hospitalized with complaints of nausea, vomiting, cramping pain in the right half of the abdomen. In anamnesis appendectomy. Pulse 96 per minute, AP 110/70 mm Hg. The abdomen is distended, asymmetrical due to protrusion of the right half, where determined a sign of intestinal splash. The peristalsis is strengthened. Signs of peritoneal irritation are absent. Rectal examination: sphincter tone is lowered, ampoule dilated. What is the primary diagnosis?
- A. * Acute adhesive intestinal obstruction
 - B. Acute obstructive ileus
 - C. Acute pancreatitis
 - D. Acute adnexitis
 - E. Food poisoning
163. Patient R. 38 years old, three years ago underwent appendectomy. After eating about 2 hours ago appeared cramping abdominal pain, flatulence, gas delay. On examination: the patient of moderate condition, restless, changing body position, had double-pointed vomiting. The tongue is dry. Pulse - 110/min. The abdomen distended, soft, painful. Rectal examination: the rectum is empty, the walls are soft, not painful. On X-ray: multiple Kloiber's cups,

pneumatization of small intestine. The patient was made a diagnosis: adhesive intestinal obstruction. What, in your opinion, should the treatment start with?

- A. * With conservative treatment (decompression of the stomach, cleansing enemas, analgesic and antispasmodic therapy)
 - B. With immediate operation
 - C. With gastric lavage and subsequent operation
 - D. With enema and subsequent immediate operation
 - E. No measures required (expectant tactics)
164. Patient S., 30 years complains of cramping abdominal pain, nausea, vomiting, delay of stool and gases. Has been ill for 4 hours. A year ago was performed gastric resection for duodenal ulcer. The patient's condition is grave. Pulse -100/min. The tongue is dry. Abdomen distended, asymmetrical, soft. By percussion - tympanitis. By auscultation - intestinal tones are absent. On the X-ray of abdominal cavity - the Kloiber's cups. What is the primary diagnosis?
- A. * Acute adhesive intestinal obstruction
 - B. Strangulated postoperative hernia
 - C. Malignant tumor of small intestine
 - D. Appendicular infiltrate
 - E. Colon cancer
165. The man 40 years old, entered the hospital with complaints of severe spastic abdominal pain, nausea, vomiting by intestinal content, abdominal distention, delay of stool and gases. Has been ill for 4 hours. Pulse 110 beats/min. The tongue is dry and furred. The abdomen distended asymmetrically - increased upper half, soft by palpation, painful. By auscultation determined active peristaltic sounds with a metallic tone, splashing sound, gurgling. What is the diagnosis?
- A. * Acute intestinal obstruction
 - B. Acute destructive pancreatitis
 - C. Acute destructive cholecystitis
 - D. Acute erosive gastritis
 - E. Acute non-specific colitis
166. The patient 18 years old, was hospitalized in a university hospital in urgent way with the signs of acute intestinal obstruction. From anamnesis 1 year ago she was operated for acute destructive appendicitis. Recently bothered abdominal distension and pain. Which of the following etiological factors resulted in intestinal obstruction?
- A. * Adhesions in the abdominal cavity
 - B. Dolihosigma
 - C. Diverticulum of ileum
 - D. Food disorders
 - E. Helminthic invasion
167. The patient 25 years old, complains of cramping abdominal pain, nausea, vomiting. The onset was sudden, 3 hours ago. Pulse 108 beats/min. The abdomen is asymmetric - right iliac region is hollowed. By palpation in the right iliac region dense cylindrical formation which is moderately painful. Rectal examination determined the content, resembling a "raspberry".
Diagnosis?
- A. * Acute intestinal obstruction
 - B. Acute destructive pancreatitis
 - C. Duodenal bleeding ulcer
 - D. Rectal polyp
 - E. Perforated ulcer
168. The patient 32 years old, who had been operated for perforated ulcer complains of intense cramping pain in the abdomen. On examination: skin is pale. PS - 98 per 1 min. AP - 100/70

- mm Hg. The abdomen is asymmetric, tense and painful in the epigastrium and the right half. Positive Valya's sign. By percussion tympanitis, hepatic dullness is preserved. On X-ray: Kloiber's cups, pneumatization of the intestine. What is your primary diagnosis?
- A. * Adhesive intestinal obstruction
 - B. Perforated duodenal ulcer
 - C. Acute destructive cholecystitis
 - D. Acute destructive appendicitis
 - E. Acute pancreatitis
169. The patient 74 years old, complains of the severe cramping pain in the left side of the abdomen, nausea, constipation and gas delay. Has been ill for 8 hours. During the last 20 years is suffering from constipations. Refuses admixtures of mucus and blood in the stool. Pulse - 82/min. The tongue is moderately wet. Abdomen greatly distended, asymmetrical due to the increase of the left half. Determined the splashing sound. On X-ray of the abdominal cavity Kloiber's cups in the left abdomen with a dilatated colon loop over them. On rectal examination the sign Obukhovskiy hospital. What is the primary diagnosis?
- A. * Sigmoid volvulus
 - B. Obstructive ileus (tumor genesis)
 - C. Adhesive ileus
 - D. Thrombosis of mesenteric vessels
 - E. Ulcerative colitis
170. The patient aged 55, complains of acute pain in the epigastric region, frequent vomiting, without any relief, a general weakness. On examination: pale skin, tongue is dry. Pulse rate - 110/min. AP - 80/40 mm Hg. The abdomen is hollowed, soft by palpation, moderately tense in the epigastrium, signs of peritoneal irritation are absent. On X-ray - Kloiber's cups in the left half. Has been ill for a day. What kind of pathology should be suspected?
- A. * High intestinal obstruction
 - B. Large intestine obstruction
 - C. Perforated duodenal ulcer
 - D. Acute pancreatitis
 - E. Acute cholecystitis
171. The patient complains of cramping pain in the upper abdomen, nausea, vomiting. The tongue is wet. The abdomen is soft, moderately tense in the epigastrium. Determined dilated intestinal loop, which peristalses by palpation. By percussion - tympanic sound with a metallic tone. On the X-ray of abdomen - Kloiber's cups. What is the primary diagnosis?
- A. * Acute intestinal obstruction
 - B. Acute pancreatitis
 - C. Food poisoning
 - D. Acute cholecystitis
 - E. Acute gastroenterocolitis
172. The patient entered with complaints of cramping abdominal pain, vomiting, delay of stool and gases. The patient's condition is severe. Pulse - 105 per 1 min. The tongue is dry. Abdomen distended, asymmetrical. By percussion - tympanitis, visible by eye peristalsis. Positive Valya's sign. Shchetkin-Blumberg sign is slightly positive. What is your diagnosis?
- A. * Intestinal obstruction
 - B. Acute pancreatitis
 - C. Mesenteric thrombosis
 - D. Acute cholecystitis
 - E. Acute appendicitis

173. During the operation for acute appendicitis, you revealed a dense appendicular infiltrate. What is your tactic?
- * To close the wound of the abdominal wall and prescribe antibiotics and local cold
 - Under general anesthesia perform a median laparotomy and appendectomy
 - To mobilize the appendix from inflammatory infiltrate and perform appendectomy
 - Perform disclosure and drainage of the infiltrate
 - All the answers wrong
174. In the patient after appendectomy on the 5th day appeared the hectic fever, leucocytosis. In the depths of the pelvis appeared moderate pain, disuric phenomenon, tenesmi. What additional method of investigation you will begin with to diagnose the complication?
- * A digital rectal examination
 - Cystochromoscopy
 - Repeat tests of blood and urine
 - Rectoromanoscopy
 - Repeat irrigoscopy
175. In the patient on the 7th day after the operation for gangrenous appendicitis, was developed a fever, pain in the rectum, tenesmi, frequent and painful urination. During rectal examination revealed the infiltrate in pelvis. During 3 days after the treatment, which included warm enemas and antibiotics, the patient's condition had not improved. Repeat examination revealed a softening of rectal infiltration. Temperature had hectic nature. What is the treatment?
- * Drainage of pelvic abscess through the rectum
 - Antibiotics, physiotherapy, hyperbaric oxygen therapy
 - Drainage of pelvic abscess through retroperitoneal access
 - Laparotomy, opening abscess
 - Laparoscopic drainage of abscess
176. In the patient was diagnosed the retroperitoneal phlegmon as a complication of acute appendicitis. What is the cause of the phlegmon?
- * Retroperitoneal location of appendix
 - Subhepatic location of appendix
 - Local peritonitis in the right iliac region
 - Medial location of appendix
 - Location of appendix laterally from caecum
177. In the patient, aged 18, on the 7th day after the appendectomy developed a pelvic abscess. What will you do?
- * Drain the abscess through the front wall of the rectum
 - Massive antibiotic therapy
 - Drain the abscess by low-median access and drainage of small pelvis
 - Drain the abscess through the right iliac region
 - Drain the abscess by a right-side extraperitoneal access
178. In the patient, aged 40, on the 2nd day after the appendectomy for gangrenous appendicitis, developed intestinal paresis, fever, appeared pain in the right half of abdomen, enlarged liver and jaundice, signs of peritoneal irritation. What complications of acute appendicitis you can think about?
- * Pylephlebitis
 - Peritonitis
 - Subphrenic abscess
 - Intraintestinal abscess
 - Subhepatic abscess

179. In the patient, on the 7th day after appendectomy was developed a pelvic abscess. What is your tactic?
- A. * Abscess drainage through the front wall of the rectum
 - B. To prescribe a massive doses of antibiotic
 - C. To drain the abscess by low-median access and drain a small pelvis
 - D. To drain the abscess by Volkovich-Dyakonov access
 - E. To drain the abscess by right-side extraperitoneal access
180. In the patient, who was operated for gangrenous appendicitis, on the 2nd day after the operation developed paresis of the intestine, jaundice, appeared fever and pain in the right side of abdomen. No signs of peritoneal irritation. What kind of complication of the disease developed?
- A. * Pylephlebitis
 - B. Subphrenic abscess
 - C. Peritonitis
 - D. Intraintestinal abscess
 - E. Subhepatic abscess
181. Is suspected for the patient's appendix. For the differential diagnosis of acute appendicitis with a perforated ulcer, 12 duodenal ulcer you apply?
- A. * The examination abdominal radiography
 - B. X-ray of gastrointestinal tract, irrigoscopy
 - C. Irrigoscopy, irrigography
 - D. Laparocentesis, colonoscopy
 - E. X-ray of the stomach with barium sulfate
182. On examination of the patient, the therapist suspected acute appendicitis, but is not confident in the diagnosis. The disease began six hours ago. What to do?
- A. * Urgent hospitalization of the patient in the surgical department
 - B. To recommend the surgeon consultation
 - C. Urgently send the patient to the clinic for further laboratory examination
 - D. As for the short time since the beginning of the disease, to recommend conservative treatment. The calm, local cold, antibiotics
 - E. Dynamic observation of patient in outpatient department
183. On the 3rd day after appendectomy you made the diagnosis - the diffuse peritonitis. What is your tactic?
- A. * To perform laparotomy
 - B. To enhance antibacterial therapy
 - C. To prescribe spasmolytics, analgetics
 - D. To perform angiographic examination
 - E. To perform laparoscopy
184. Patient after surgery for diffuse peritonitis antibiotics. What is the way the drug is best?
- A. oral
 - B. subcutaneous
 - C. intramuscular
 - D. * intravenous
 - E. intraperitoneal
185. Patient B. was examined by urgent surgeon, who suspected acute appendicitis. The examination was prescribed. Which of the following investigations is the least helpful in the diagnosis of acute appendicitis?
- A. * Laparocentesis

- B. Laboratory research (especially leukocytes)
 - C. Rectal examination
 - D. Axillary and rectal thermometry
 - E. The clinical examination to determine protective muscular tension.
186. Patient D., aged 39, was admitted to the surgical department with the diagnosis: diffuse peritonitis. What does determines the severity of peritonitis?
- A. * No right answer
 - B. Patient weight
 - C. The growth of the patient
 - D. Body mass index
 - E. All true
187. Patient K. has the diagnosis: diffuse purulent peritonitis of appendicular origin. For the patient is indicated:
- A. * All mentioned is correct
 - B. Appendectomy and sanation of the abdominal cavity
 - C. Correction of fluid and electrolyte disorders
 - D. Antibacterial therapy
 - E. Total parenteral nutrition for 1-2 days after operation
188. Patient N., aged 45, was admitted to the surgical department with the diagnosis: biliary peritonitis. Treatment.
- A. * Operative treatment
 - B. Conservative treatment
 - C. Outpatient care
 - D. Treatment is not required
 - E. Fluid management
189. Patient received an abdominal trauma 3 hours ago. Fetched in emergency clinic complaining of acute pain in stomach. Positive symptoms irritation of the peritoneum. What method of diagnosis should be used to refine the diagnosis.
- A. ECG
 - B. rheovasography
 - C. * X-ray of the abdominal cavity
 - D. doppler
 - E. thermometry
190. Patient received an abdominal trauma 3 hours ago. Fetched in emergency clinic complaining of acute pain in stomach. Positive symptoms irritation of the peritoneum. When the review X-ray revealed free gas under the dome of the diaphragm. Installed diagnosis: breaking the body cavity. Treatment.
- A. cold on the stomach
 - B. laparocentesis
 - C. laparoscopy
 - D. * operation
 - E. antibiotic
191. Patient S., aged 35, was admitted to the surgical department with the diagnosis: appendicular peritonitis. Treatment.
- A. * Operative treatment
 - B. Conservative treatment
 - C. Outpatient care
 - D. Treatment is not required

- E. Fluid management
192. Patient S., aged 35, was admitted to the surgical department with the diagnosis: diffuse peritonitis. What determines the severity of peritonitis?
- A. * The degree of intoxication
 - B. Patient weight
 - C. The growth of the patient
 - D. Body mass index
 - E. All true
193. Patient transported to hospital with a diagnosis: sub-diaphragmatic abscess. What tactics of treatment is most correct?
- A. conservative treatment
 - B. extraperitoneal incision and drainage
 - C. laparotomy, dissection, and backfilling the cavity
 - D. * thick needle puncture of an abscess under ultrasound control
 - E. all of the above is true
194. Patient transported to hospital with a diagnosis: subdiaphragmatic abscess. What access to the abscess should be used?
- A. thoracolumbotomy
 - B. lumbotomy
 - C. two-stage transpleural approach
 - D. Fedorov's laparotomy
 - E. * Extrapleural extraperitoneal method
195. Patient transported to hospital with suspected sub-diaphragmatic abscess. What method of research can clarify the diagnosis?
- A. ECG
 - B. doppler
 - C. * ultrasound
 - D. contrast fluoroscopy GIT
 - E. irrigoscopy
196. Patient transported to hospital with suspected sub-diaphragmatic abscess. What method of research can clarify the diagnosis?
- A. ECG
 - B. doppler
 - C. * radiography of the abdominal cavity
 - D. contrast fluoroscopy GIT
 - E. irrigoskopiya
197. Patient transported to hospital with the diagnosis: diffuse peritonitis. Which surgical approach should be used?
- A. Adrectal access right
 - B. Adrectal access to the left
 - C. Access below the navel
 - D. * Median laparotomy
 - E. No right answer
198. Patient 2 hours ago fell from the second floor. His condition is grave. There are signs of diffuse peritonitis. Completed radiography abdomen. Which of the radiological signs confirm the diagnosis of rupture cavity body?
- A. highstand dome of the diaphragm

- B. Kloiber's bowls
 - C. * free gas in the abdominal cavity
 - D. free fluid in the abdominal cavity
 - E. infiltration in the abdominal cavity
199. Patients 2 hours ago fell from the second floor. His condition is grave. There are signs of diffuse peritonitis. What investigation method can eliminate the damage of a hollow organ?
- A. ECG
 - B. rheovasography
 - C. * radiography of the abdominal cavity
 - D. doppler
 - E. thermometry
200. Patients 2 hours ago was hit in the stomach. Fetched in emergency hospital in serious condition with a suspected rupture cavity body. What method of research will clarify the diagnosis?
- A. ECG
 - B. Doppler
 - C. * X-ray abdomen
 - D. contrast fluoroscopy GIT
 - E. irrigoscopy
201. Patients 2 hours ago was hit in the stomach. Fetched in emergency hospital in serious condition with a suspected rupture cavity body. What method of research will clarify the diagnosis?
- A. ECG
 - B. doppler
 - C. * laparocentesis
 - D. contrast fluoroscopy GIT
 - E. irrigoscopy
202. Patients 2 hours ago was hit in the stomach. Fetched in emergency hospital in serious condition. When radiography diagnosed gap cavity body. Your tactics.
- A. cold on the stomach
 - B. laparocentesis
 - C. laparoscopy
 - D. * operation
 - E. antibiotic
203. The patient 47 years old admitted to the clinic with complaints of pain in the right hypochondrium, nausea, vomiting, body temperature, 38.3. Pain in 3 days ago after receiving a fatty meal. Pulse 112 in 1 min. tongue dry. Skin and sclera subicterichny. In the right hypochondrium defined sharply painful tense rounded education 12h8h6 see positive symptoms Ortner, Murphy, Kera. Symptom Shchetkin-Blumberg positive throughout the abdomen. Which research method should be used?
- A. * Ultrasound
 - B. ECG
 - C. Rheovasography
 - D. Doppler
 - E. Thermometry
204. The patient admitted in emergency hospital in serious condition. Pulse 112 per minute., AP - 110 60, T - 35, 1 °C. Installed diagnosis diffuse peritonitis. Which research method should be used to clarify the source of peritonitis?

- A. ECG
 - B. Rheovasography
 - C. * Ultrasound
 - D. Doppler
 - E. Thermometry
205. The patient admitted in emergency hospital in serious condition. Pulse 112 per minute., AP - 110 60., T-35, 1 °C. Installed diagnosis diffuse peritonitis. What treatment is indicated the patient?
- A. laparocentesis
 - B. laparoscopy
 - C. * surgery
 - D. antibiotic
 - E. puncture
206. The patient admitted with blunt abdominal trauma. Examination - positive symptoms irritation of the peritoneum. Suspected damage to internal organs. What method of diagnosis should be used?
- A. angiography
 - B. rheovasography
 - C. * ultrasound
 - D. ECG
 - E. EEG
207. The patient admitted with blunt abdominal trauma. Examination - positive symptoms of irritation of the peritoneum. Suspected damage to internal organs. What method of diagnosis should be used?
- A. * laparocentesis
 - B. angiography
 - C. rheovasography
 - D. ECG
 - E. EEG
208. The patient complains of severe abdominal pain, which occurred suddenly 6 hours ago. Installed diagnosis of peritonitis. Which symptom is most typical for this diagnosis?
- A. temperature rise
 - B. tachycardia
 - C. leukocytosis
 - D. tension of the muscles of the abdominal wall
 - E. * irritation of the peritoneum
209. The patient complains of severe abdominal pain, which occurred suddenly 6 o'clock ago. Installed diagnosis of peritonitis. Which research method should be used to clarify the source of peritonitis?
- A. * Ultrasound
 - B. ECG
 - C. Rheovasography
 - D. Doppler
 - E. Thermometry
210. The patient during surgery revealed fibropurulent peritonitis. What activities should be undertaken?
- A. removal or delimitation source of peritonitis.
 - B. reorganization of the abdominal cavity.

- C. decompression of the intestine.
 - D. drainage of the abdominal cavity.
 - E. * all answers are correct
211. The patient operated on for acute appendicitis at day 4 after surgery developed peritonitis. Your tactics?
- A. Antibiotic
 - B. Assign analgesics
 - C. Laparocentesis
 - D. Laparoscopy
 - E. * Operation
212. The patient operated on for diffuse peritonitis. In the postoperative period appointed metranidazol. With what's aim was appointed the drug?
- A. * removal of anaerobic microflora.
 - B. removal of fungal flora.
 - C. elimination of the aerobic microflora.
 - D. prevention of helminthic infestation.
 - E. prevention of disseminated candidiasis
213. The patient was admitted to the surgical department with suspected diffuse peritonitis. Which diseases should be a differential diagnosis?
- A. hepato-renal syndrome
 - B. * abdominal ischemic syndrome
 - C. adrenogenital syndrome
 - D. Horner's syndrome
 - E. diencephalic syndrome
214. The patient was diagnosed the diffuse peritonitis of appendicular origin. In this case used:
- A. * All mentioned
 - B. Median laparotomy
 - C. Appendectomy
 - D. Abdominal lavage
 - E. Drainage of the abdominal cavity
215. The patient was operated for acute phlegmonous appendicitis and diffuses peritonitis by means of the access to the right iliac region. On the 20th day after the operation was diagnosed right-side subphrenic abscess. What could cause its occurrence?
- A. * Was not used Fovler's position after the operation
 - B. Was not used Trendelenburg's position after the operation
 - C. Was not used Finsterer's position after the operation
 - D. Was not used Billroth 's position after the operation
 - E. Was not performed the tamponade of abdominal cavity
216. The patient, 76 years old, with myocardial infarction, is suspected for acute destructive appendicitis. What is your tactic?
- A. * Urgent operation
 - B. Monitoring and operation if the peritonitis develops
 - C. Prescription of massive doses of broad-spectrum antibiotics and if this therapy is ineffective – operation
 - D. Operation if the diagnosis confirms by laparoscopy
 - E. All the answers are wrong

217. The patient, aged 76, with concomitant pneumonia, was diagnosed phlegmonous appendicitis. What is your tactic?
- A. * Urgent operation
 - B. Monitoring and operation if the peritonitis develops
 - C. Prescription of massive doses of broad-spectrum antibiotics and if this therapy is ineffective – operation
 - D. Operation if the diagnosis confirms by laparoscopy
 - E. All the answers are wrong
218. The patient, is pregnant for 32 weeks, has stayed in the surgical department for 18 hours. During dynamic observation it is impossible to rule out acute appendicitis. What is your tactic?
- A. * Patient need to undergo the operation
 - B. Continue to observe the patient
 - C. Carry out abdominal ultrasonography
 - D. With gynecologist to perform abortion, and then appendectomy
 - E. To perform laparoscopy and to operate if the diagnosis confirms
219. The patient's T, at the ambulatory reception is suspected for acute appendicitis. What is advisable to do?
- A. * Immediately hospitalization of the patient
 - B. To prescribe spasmolytics and repeat examination of the patient in 4-6 hours
 - C. The control of the dynamics of body temperature and leucocytosis for 12-24 hours
 - D. To prescribe the anti-inflammatory therapy and repeat examination on the next day
 - E. Observed the patient ambulatory and hospitalized in case of deterioration
220. You diagnosed and prescribed conservative treatment of appendicular infiltrate. What are the signs of abscessing of appendicular infiltrate?
- A. * All answers correct
 - B. Maintaining a high hectic temperature
 - C. Restoring or increase of pain
 - D. Signs of peritoneal irritation
 - E. Increase of leukocytosis with a shift to the left, the increase of ESR
221. You diagnosed in patient the acute appendicitis. What method of diagnostic is suitable?
- A. * All mentioned is correct
 - B. Laparoscopy
 - C. Clinical analysis of blood
 - D. Rectal examination
 - E. Thermography
222. You have diagnosed the appendicular infiltrate in the patient. What is the most expedient conservative treatment in first three days?
- A. * Antibiotics
 - B. Analgesics
 - C. U.H.F.-therapy
 - D. The warm on right iliac region
 - E. Parenteral use of proteolytic enzymes
223. You have diagnosed the gangrenous acute appendicitis in the patient. What symptoms are the basic for the diagnose?
- A. * Decrease of pain
 - B. Increase of pain
 - C. A sudden increase of pain in the right iliac region
 - D. Bradycardia

- E. Mayo-Robson's sign in the right iliac region
224. You have to operate the patient with a typical picture of acute phlegmonous appendicitis. What access for appendectomy is the most suitable in this case?
- A. * Volkovych-Diakonov's access
 - B. Low-median laparotomy
 - C. Right-side pararectal access
 - D. Right-side transrectal incision
 - E. Transverse laparotomy above navel
225. You suspect in patient the chronic appendicitis. Which of the following methods of investigation can confirm the diagnosis?
- A. * Irrigoscopy
 - B. Colonoscopy
 - C. Laparocentesis
 - D. Rectoromanoscopy
 - E. X-ray of the abdominal cavity
226. You suspect the abscess of Douglas' pouch in the patient. What investigations should be carried out for its diagnosis?
- A. * A digital rectal examination
 - B. Rectoromanoscopy
 - C. Colonoscopy
 - D. Laparoscopy
 - E. Laparocentesis
227. You suspected acute appendicitis in patient. Diagnostic program includes:
- A. * All answers correct
 - B. Thorough anamnesis
 - C. To exclude all diseases that can simulate acute abdominal pathology
 - D. Rectal examination in men and vaginal additional examination in women
 - E. Laboratory tests
228. You suspected acute appendicitis. For the differential diagnosis of acute appendicitis from the right-side renal colic you apply:
- A. * Prescription of spasmolytics, excretory urography
 - B. Prescription of narcotic analgetics
 - C. Laparocentesis, laparoscopy
 - D. Fluoroscopy gastrointestinal, irrigoscopy
 - E. Angiography of renal arteries
229. Patient S., 27 years old admitted to clinic with the diagnosis: perforated ulcer. What study should be carried out in the first place?
- A. ECG
 - B. CT
 - C. MRI
 - D. * Survey radiography of the abdominal cavity
 - E. Ultrasound
230. Patient A., 43, was admitted to hospital with a diagnosis: chronic cholecystitis calculary. What is the main method of research in this patient?
- A. Ainfusion holegraphy,
 - B. oral cholecystography,
 - C. * SONOGRAPHY

- D. scanning of the liver,
 - E. computed tomography
231. Patient A., aged 45, was admitted to hospital with jaundice in the background cholelithias. The patient is indicate:
- A. emergency surgery
 - B. conservative treatment
 - C. * emergency operation after the preoperative
 - D. catheterization of the celiac artery
 - E. plasmapheresis
232. Patient A., aged 45, was admitted to hospital with jaundice in the background cholelithias. The patient is shown:
- A. emergency surgery
 - B. conservative treatment
 - C. * there is no right answer
 - D. catheterization of the celiac artery
 - E. plasmapheresis
233. Patient D., 63, operated by acute calculouse cholecystitis. Intraoperatively diagnosed gallbladder with concrements. What operation is indicated the patient?
- A. cholecystostomy
 - B. * cholecystectomy from cervical
 - C. cholecystectomy from the bottom
 - D. cholecystitis-enterovirus anastomosis
 - E. cholecystectomy with drainage choledochitis by Halstead-Pikovsky
234. Patient D., aged 39, was admitted to the surgical department with the diagnosis: diffuse peritonitis. What does determines the severity of peritonitis?
- A. * No right answer
 - B. Patient weight
 - C. The growth of the patient
 - D. Body mass index
 - E. All true
235. Patient M., aged 56, was admitted to the hospital with a diagnosis: acute gangrenous cholecystitis. The patient is shown:
- A. conservative treatment
 - B. delayed operation
 - C. decision depends on the age of the patient
 - D. operation with no effect of conservative treatment
 - E. * Emergency operation
236. Patient M., aged 56, was admitted to the hospital with a diagnosis: acute gangrenous cholecystitis. The patient is shown:
- A. conservative treatment
 - B. delayed operation
 - C. decision depends on the age of the patient
 - D. operation with no effect of conservative treatment
 - E. * there is no right answer
237. Patient N., aged 45, was admitted to the surgical department with the diagnosis: biliary peritonitis. Treatment.
- A. * Operative treatment

- B. Conservative treatment
 - C. Outpatient care
 - D. Treatment is not required
 - E. Fluid management
238. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What analgesics is contraindicated?
- A. omnopon
 - B. * no right answer
 - C. no-spa
 - D. spazgan
 - E. spasmalgon, baralgin
239. Patient S., aged 35, was admitted to the surgical department with the diagnosis: appendicular peritonitis. Treatment.
- A. * Operative treatment
 - B. Conservative treatment
 - C. Outpatient care
 - D. Treatment is not required
 - E. Fluid management
240. Patient S., aged 35, was admitted to the surgical department with the diagnosis: diffuse peritonitis. What does determines the severity of peritonitis?
- A. * The degree of intoxication
 - B. Patient weight
 - C. The growth of the patient
 - D. Body mass index
 - E. All true
241. Patient S., aged 56, was admitted to the hospital with a diagnosis: acute catarrhal cholecystitis. What symptoms are not typical for this diagnosis?
- A. nausea
 - B. B. Ker's sing
 - C. * Murphy's sing
 - D. absence of muscle tension in the right hypochondrium
 - E. Musso's sing
242. The patient 47 years old admitted to the clinic with complaints of pain in the right hypochondrium, nausea, vomiting, body temperature, 38.3. Pain in 3 days ago after receiving a fatty meal. Pulse 112 in 1 min. tongue dry. Skin and sclera subikterichny. In the right hypochondrium defined sharply painful tense rounded education 12h8h6 see positive symptoms Ortner, Murphy, Kera. Symptom Shchetkin-Blumberg positive throughout the abdomen. Which research method should be used?
- A. * Ultrasound
 - B. ECG
 - C. Rheovasography
 - D. Doppler
 - E. Thermometry
243. The patient admitted in emergency hospital in serious condition. Pulse 112 per minute., AP - 110 60., T-35, 1 °C. Installed diagnosis diffuse peritonitis. What treatment is indicated the patient?
- A. laparocentesis
 - B. laparoscopy

- C. * surgery
 - D. antibiotic
 - E. puncture
244. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic holecography
 - E. * hypotonic doudenography
245. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic holecography
 - E. * all answer are right
246. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic holecography
 - E. * endoscopy
247. The patient complains of severe abdominal pain, which occurred suddenly 6 hours ago. Installed diagnosis of peritonitis. Which symptom is most typical for this diagnosis?
- A. temperature rise
 - B. tachycardia
 - C. leukocytosis
 - D. tension of the muscles of the abdominal wall
 - E. * irritation of the peritoneum
248. The patient complains of severe abdominal pain, which occurred suddenly 6 o'clock ago. Installed diagnosis of peritonitis. Which research method should be used to clarify the source of peritonitis?
- A. * Ultrasound
 - B. ECG
 - C. Rheovasography
 - D. Doppler
 - E. Thermometry
249. The patient with a history of ulcerative with X-ray determined by a deep niche, located on the back wall near the small curvature of the antrum. What diagnoses are in the patient?
- A. Perforated ulcer
 - B. Malignancy ulcers
 - C. Ulcer bleeding
 - D. * Penetration ulcers
 - E. All answers are correct

250. The patient, 34 years old, 6 years ago was silent ulcer perforation duodenum. Two years after this the patient felt well and do not appealed. Then came the pain, typical of peptic diseases. What method of research is the most informative?
- A. ECG
 - B. * ENDOSCOPY
 - C. X-ray
 - D. CT
 - E. MRI
251. The patient, 36 years old, 12 years suffer stomach ulcer Conservative treatment is not effective. What tactics are indicating for treating a patient?
- A. Continue medical treatment
 - B. * No right answer
 - C. Outpatient
 - D. Infusion therapy
 - E. Not require treatment
252. The patient, 36 years old, 12 years suffer stomach ulcer. Conservative treatment is not effective. What tactics are indicating for treating a patient?
- A. Continue medical treatment
 - B. * Surgical treatment
 - C. Outpatient
 - D. Infusion therapy
 - E. Not require treatment
253. The patient, 36 years old, 12 years suffer stomach ulcer. It is treated routinely in the clinic. What method of research is most informative?
- A. ECG
 - B. * ENDOSCOPY
 - C. X-ray
 - D. CT
 - E. MRI
254. The patient, 40 years old, long-suffering with stomach ulcer, said that the last 2 days the pain had become less intense, but at the same time it has been increasing weakness, dizziness. In morning for a few seconds to lose consciousness. On examination, pulse 100 in 1 min. The patient was pale. In the epigastric region is very little pain. No symptoms of irritation of the peritoneum. What complication of peptic ulcer you can suspect?
- A. Perforated ulcer
 - B. Malignancy ulcers
 - C. * Ulcer bleeding
 - D. Ulcer Penetration
 - E. All answers are correct
255. The patient, 42 years old, gastric ulcer in existence for 10 years. After treatment in hospital advanced remission. Three months ago, the patient appeared pain in the lumbar region, sometimes wearing herpes character. What study should be used to refine the diagnosis?
- A. * X-ray study of the digestive tract with barium
 - B. Total blood
 - C. Urinalysis
 - D. Rheovasography
 - E. Doppler

256. The patient, 42 years old, gastric ulcer in existence for 10 years. After treatment in hospital advanced remission. Three months ago, the patient appeared pain in the lumbar region, sometimes wearing herpes character. What study should be used to refine the diagnosis?
- * No right answer
 - Total blood
 - Urinalysis
 - Rheovasography
 - Doppler
257. A man 46 years complain for the expressed general weakness, attacks of palpitation, pain in an epigastrium, which arise up in 10-15 minutes after the reception of meal. During 10 years is ill ulcerative disease of stomach. 2 years ago the executed resection of stomach after Bilroth- 2. At inspected a stomach is soft. Sickly in an epigastrium and pyloro-duodenal area. What complication most probably did arise up for a patient?
- * Dumping is a syndrome
 - Cholecystitis
 - Peptic gastric ulcer
 - Chronic pancreatitis
 - Gastritis of stump of stomach
258. A man 46 years complain for the expressed general weakness, attacks of palpitation, pain in an epigastrium, which arise up in 10-15 minutes after the reception of meal. During 10 years is ill ulcerative disease of stomach. 2 years ago the executed resection of stomach after Bilroth-II. At inspected a stomach is soft. Sickly in an epigastrium and pyloro-duodenal area. What complication most probably did arise up for a patient?
- * Dumping is a syndrome
 - Cholecystitis
 - Peptic gastric ulcer
 - Chronic pancreatitis
 - Gastritis of stump of stomach
259. A patient 76 years acted to the hospital on seventh twenty-four hours from the beginning of disease with complaints about pain in right hypochondrium, weakness, repeated vomiting, temperature up to 38 degrees. At a examination is a general of middle weight. Pallor. A enlarged gall-bladder is palpated in right hypochondrium where tension is examined. What method of treatment?
- * Cholecystostomy under sonography control
 - Diet therapy
 - Conservative treatment
 - Spasmolitics
 - Analgesics
260. Choose the most frequent reason of relapse of ulcer after a organ-saving operation with vagotomy at the adequate decline of acid production after an operation:
- * syndrome of Zollinger-Elisson
 - incomplete vagotomy
 - duodeno-gastric reflux of bile
 - gastritis of the operated stomach
 - excessive use of alcohol
261. During an operation for a patient 68 years concerning a acute destructive cholecystitis certain presence of festering cholangitis. At the revision of choledohus the not found concrements and sign of stenosis duodenal to the nipple. Cholecystectomy is executed. How is it necessary to make off an operation?

- A. * To execute the external catchment of choledohus
 - B. To impose supraduodenal choledohoduodenostomy
 - C. To execute a transduodenal choledohoduodenostomy
 - D. To execute a choledochojejunostomy
 - E. To drain an abdominal cavity
262. During an operation on occasion of acute calculus cholecystitis the extended general bilious channel is founded. After cholecystectomy through stump of cystic duct is performed cholangiography On X-Ray of hepatic is founded general bile ducts. In the distal part of choledohus a precipice of contrasting matter is with even edges. A contrast does not act in a duodenum. How to estimate data of intraoperative cholangiography?
- A. * Present of stones in the distal part of choledohus
 - B. Stenosis of duodenal papilla
 - C. Cicatrice narrowing of distal part of choledohus
 - D. Cholecystitis
 - E. Tumour of head of pancreas
263. For a 38-years-old patient after carried two years ago back cholecystectomy recommenced attacks of hepatic colic with turning of skin covers yellow. A postcholecystectomy syndrome is diagnosed. What is most frequent reason of this situation?
- A. * Leave during the first operation concrements
 - B. Stenosis papillitis
 - C. Duodenostasis
 - D. Diskinesia of bile ducts
 - E. Cholestasis
264. For a patient the decompensate stenosis of pyloro-bulb area is on a background 10 years of ulcerative anamnesis. In the state of middle weight he acted with complaints about the frequent vomiting and belch rotten. Marks the expressed weakness. Exhausted. After stabilizing of metabolic indexes and general a patient gave a consent to operative treatment. There are endoscopic signs of the expressed gastritis in all parts of stomach. Choose the method of operation :
- A. * a resection of stomach by Bilioth II
 - B. vagotomy is with an operation which drains a stomach
 - C. imposition of gastrostomy by Vitsel
 - D. selective proximal vagotomy
 - E. a fundoplication by Nissen
265. For a patient 45 years, in 2 years after cholecystectomy there were pains in right hypochondrium, icteric and itch of skin, bitter taste in a company. At sonography of choledohus a 1,2 cm, in distal part located stone. What is the diagnosis?
- A. * Choledoholitiasis. Obturative icterus
 - B. Cholangitis. Mechanical icterus
 - C. Acute pancreatitis. Mechanical icterus
 - D. Postcholecystectomy syndrome
 - E. Tumour of head of pancreas. Mechanical icterus
266. For a patient 48 years with a giant gastric ulcer with recrudescence motion (has ulcerative disease during 11 years) a histamine-reflectory an-acidity is founded. What medical tactic?
- A. * Conservative treatment in hospital and further sanatorium-resort treatment
 - B. Stationary treatment
 - C. Anti-ulcerative therapy is before a planned operation
 - D. Urgent operation
 - E. Stationary conservative treatment during 8 weeks

267. For a patient 60 years icterus during 3 weeks, intensity of it began without pain grows. A stomach at palpation is soft. Positive symptom of Kurvuasie. At sonography of bilious ways expansion of gall-bladder is marked and choledohus. What primary cause of these changes.
- * Cancer of head of pancreas
 - Bile-stone disease
 - Chronic pancreatitis
 - Infectious hepatitis
 - Cancer of liver.
268. For a patient which carried the resection of stomach a year ago, the attacks of weakness, which arose up on an empty stomach or in 15-20 minutes after the reception of meal, appeared. Attacks were accompanied by a muscle weakness, headache, decline of sugar of blood, decline of arteriotony, sometimes by dizziness. What complication did arise up for a patient?
- * Hypoglycaemia syndrome
 - Food allergy
 - Dumping-syndrome
 - Postresection asthenia
 - Syndrome of small stomach
269. For a patient C., 58 years, increased feed, after will attack pains the icteric of skin and conjunctiva of sclera, discoloured excrement, darkening of urine appeared in right hypochondrium. A stomach is exaggerated, at palpation sickly in right hypochondrium, a enlarged liver is palpated on a 2-3 cm. In blood general bilirubin 90 mmole/L, direct 60 micromole/L. What is the best examination method?
- * Retrograde cholangiopancreatography
 - Intravenous cholegraphy
 - Transcutaneous cholegraphy
 - Transcutaneous transhepatic cholegraphy
 - Sonography of hepatic region
270. For a patient, which carried an operation, resection of stomach, concerning ulcerative disease, there was a relapse (endoscopy data). What reason of relapse?
- * all correct
 - incomplete vagotomy;
 - leave at a resection part of antrum;
 - economy resection of stomach;
 - syndrome of Zollinger-Elisson;
271. For a patient, which carried the resection of stomach for Bilroth-II concerning ulcerative disease of duodenum is marked complaints about a acute weakness, attacks of palpitation, waves of heat to the face and dizziness at the reception of sweet and suckling meal. Deficit of b.w. - 15kr. On X-Ray of stump of stomach of small sizes with evacuation of barium in the extended efferent bowel. What operatively interference does need it was to be before done to the patient?
- SPV with a pyloroplasty
 - * Jejunogastroplasy
 - Resection of stomach by Rou
 - Trunk vagotomy
 - Resection of stomach
272. For the exhausted man of 42, which 15 years suffers ulcerative disease of duodenum, during the last two weeks the massive vomiting takes place every evening. At X-Ray stenosis of

goalkeeper of stomach is founded. The above-mentioned state became complicated by cramps. what complication did arise up for a patient?

- A. * Tetany
- B. Bleeding
- C. Perforation
- D. Penetration
- E. Malignancy

273. For the patient of 32 after carried cholecystectomy the attacks of hepatic colic, which are accompanied by turning of skin canopies yellow, recommenced in 6 months. At sonography examination the leave concrements of 2 x is founded 1cm in distal part of choledohus. What treatment for a patient will be optimal?

- A. * Endoscopic duodenocholedohoscopy with papillosphincterotomy
- B. Conservative therapy by spasmolitics
- C. Lithotripsy
- D. Choledoholithotomy
- E. Choledohoduodenoanastomosis

274. For the patient of 63, in a year after cholecystectomy with choledoho-duodenal anastomosis, there were pains in right hypochondrium, icterus, temperature, fever, dryness and bitter taste. What is the previous diagnosis?

- A. * Cholangitis
- B. Acute pancreatitis
- C. Duodenitis. Mechanical icterus
- D. Stricture of choledohus
- E. Hepatitis is with transformation in a cirrhosis

275. Operative treatment with a duodenal ulcer it is indicated a patient in those cases when: 1. often there are relapses of disease, 2. a disease becomes complicated continuous bleeding, 3. there is pyloro-duodenal stenosis 4. there is a perforation of ulcer, 5. ulcer penetrates in the head of pancreas, giving the frequent intensifying and phenomena of pancreatitis. Choose the best composition:

- A. * all true.
- B. 1,4
- C. 2,3
- D. 3,4
- E. 1,2

276. Patient 57 years, during the last 10 years has a chronic calculus cholecystitis. Intensifying of disease take place 1-2 times on a year. The last attack began 8 days ago. The icteric skin and sclera appeared in 2 days. During sonography of inspection of organs of abdominal region: thickness of wall of gall-bladder up to 5 mm, present concrements 0,5 to 1,0 cm in a diameter. A width of choledohus is a 1,2 cm, in it supraduodenal part a stone is founded up to 1,0 cm. What complication of gallbladder disease did arise up for a patient?

- A. * Choledoholitiasis. Mechanical icterus
- B. Acute cholangitis
- C. Acute calculus cholecystitis
- D. Choledoho-duodenal fistula
- E. Cancer of bilious ducts

277. Patient of 51, operated a year ago concerning the perforative ulcer of duodenum. Done barrel vagotomy is with excision of ulcer. Complain for periodic appearance of liquid stool, which arises up suddenly (for two three times on twenty-four hours within a week), whereupon normalized independently. Define a diagnosis.

- A. * Postvagotomy diarrhoea
 - B. Chronic pancreatitis
 - C. Dumping is a syndrome
 - D. Chronic colitis
 - E. Poliposis of colon
278. The patient 32 years, two years ago passed cholecystectomy concerning gallbladder disease. There were many shallow concrements in a gallbladder. During six months the attacks of hepatic colic recurred 3 times. Two days ago back after an attack, turning of skin and sclera appeared yellow. Bilirubin of 90 mmole/L. What examination most effective in diagnose?
- A. * sonography of pancreatoduodenal area
 - B. X-Ray of gastrointestinal tract
 - C. Endoscopy
 - D. Cholangiography
 - E. Angiography
279. The patient of 42 four years ago carried the resection of stomach after Hoffmaister-Finsterer concerning the ulcer of duodenum with penetration in a pancreas. During the last year marks pain in an epigastrium and right hypochondrium. A pain syndrome is accompanied massive vomiting by stagnant content with plenty of bile. After vomiting pain is decreased. The repeated treatment of improvement did not bring. A patient became thin on 12 kg. What is the diagnosis?
- A. * Syndrome of afferent loop
 - B. Tumour of stump of stomach
 - C. Chronic pancreatitis
 - D. Relapse of ulcer
 - E. Peptic ulcer of gastroenteroanastomosis
280. The patient of 62 complains in the presence of icterus, itch of skin, light excrement, wet dark. Objectively: a sclera and skin is icteric. An enlarge, painless gallbladder is palpated. Blood test: general bilirubin of 85 mmol/L. What method of examination more expedient in all to use for clarification of diagnosis?
- A. * Retrograde cholangiopancreatography
 - B. Duodenal sounding
 - C. Survey sciagraphy of organs of abdominal region
 - D. Oral cholecystography
 - E. Cholegraphy
281. The patient of H., 62, acted with complaints about pain in the overhead half of stomach after the food intake, heartburn, nausea, decline of mass of body on 15 kg for a year, constipations. Three to the year that is why operated concerning a perforative gastric ulcer, executed resection of stomach. In a year after an operation pain recommenced in the overhead half of stomach, heartburn, periodically there was vomiting by a meal, the state of patient became worse in course of time. Your previous diagnosis?
- A. * Peptic ulcer of gastroenteroanastomosis
 - B. Syndrome of afferent loop
 - C. Cancer of stump of stomach
 - D. Gastritis of the operated stomach
 - E. Chronic pancreatitis
282. Woman 55 years, complains on pain in right hypochondrium, temperature up to 39C, darkening of colour of urine. Objectively: skin and visible mucous membranes icteric, liver enlarged, dense, sickly at palpation. In the global analysis of blood of increase of ESR, neutrophilic leukocytosis, shift to the left, increase of level of bilirubin, ALT, AST. Sonography are stones of gall-bladder. What complication did arise up for this patient?

- A. * Cholangitis
 - B. Empyema of gall-bladder
 - C. Chronic cholecystitis
 - D. Acute cholecystitis
 - E. Sepsis
283. Woman of 53, three years ago back carried cholecystectomy concerning a acute calculus cholecystitis. After operation already four times were twinges in right hypochondrium, which lasted 6-8 hours, passed after spasmolitics. After the second attack marked turning of skin and sclera yellow. What examination will be most informing for imputing of diagnosis?
- A. * Sonography
 - B. X-Ray of stomach
 - C. Endoscopy
 - D. Cholangiography
 - E. Thermography
284. Patient S., aged 56, was admitted to hospital with a diagnosis: acute catarrhal cholecystitis. What symptoms are not typical for this diagnosis?
- A. nausea
 - B. symptom Kera
 - C. * all responses venrny
 - D. lack of muscle tension in the right hypochondrium
 - E. Musso symptom
285. During surgery at cholelithiasis detected wrinkled gallbladder filled with stones and advanced up to 2.5 cm common bile duct. Patients should
- A. * make cholecystectomy
 - B. perform cholecystectomy, then holangiography
 - C. immediately make an audit of cholecystectomy and duct
 - D. impose cholecystitis
 - E. duodenotomy make the audit of the major duodenal papilla
286. Patient A., 43, was admitted to hospital with a diagnosis: chronic cholecystitis calculary. What is the main method of research in this patient?
- A. infusion holegraphy,
 - B. oral cholecystography,
 - C. * SONOGRAPHY
 - D. scanning of the liver,
 - E. computed tomography
287. Patient A., aged 45, was admitted to hospital with jaundice in the background cholelithias. The patient is indicate:
- A. emergency surgery
 - B. conservative treatment
 - C. * emergency operation after the preoperative
 - D. catheterization of the celiac artery
 - E. plasmapheresis
288. Patient A., aged 45, was admitted to hospital with jaundice in the background cholelithias. The patient is shown:
- A. emergency surgery
 - B. conservative treatment
 - C. * there is no right answer
 - D. catheterization of the celiac artery

- E. plasmapheresis
289. Patient D., 63, operated by acute calculous cholecystitis. Intraoperatively diagnosed gallbladder with concretions. What operation is indicated the patient?
- A. cholecystostomy
 - B. * cholecystectomy from cervical
 - C. cholecystectomy from the bottom
 - D. cholecystitis-enterovirus anastomosis
 - E. cholecystectomy with drainage choledochitis by Halstead-Pikovsky
290. Patient M., aged 56, was admitted to the hospital with a diagnosis: acute gangrenous cholecystitis. The patient is shown:
- A. conservative treatment
 - B. delayed operation
 - C. decision depends on the age of the patient
 - D. operation with no effect of conservative treatment
 - E. * Emergency operation
291. Patient M., aged 56, was admitted to the hospital with a diagnosis: acute gangrenous cholecystitis. The patient is shown:
- A. conservative treatment
 - B. delayed operation
 - C. decision depends on the age of the patient
 - D. operation with no effect of conservative treatment
 - E. * there is no right answer
292. Patient S., 41, was admitted to hospital with a diagnosis: obstructive jaundice. To diagnose the causes of jaundice is the subject of a more reliable:
- A. oral cholecystography
 - B. intravenous cholecystocholangiography
 - C. * retrograde cholangiography
 - D. liver scintigraphy
 - E. direct hepatosplenography
293. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What analgesics is contraindicated?
- A. omnopon
 - B. * morphine hydrochloride
 - C. no-spa
 - D. spazgan
 - E. spazmalgon, baralgin
294. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What analgesics is contraindicated?
- A. omnopon
 - B. * no right answer
 - C. no-spa
 - D. spazgan
 - E. spazmalgon, baralgin
295. Patient S., 42 years old, entered the clinic with a diagnosis: acute calculary cholecystitis. What method is most informative to clarify the diagnosis in the reception-office?
- A. infusion holecystography,
 - B. oral cholecystography,

- C. * SONOGRAPHY
 - D. scanning of the liver,
 - E. computed tomography
296. Patient S., aged 56, was admitted to the hospital with a diagnosis: acute catarrhal cholecystitis. What symptoms are not typical for this diagnosis?
- A. nausea
 - B. Ker's sign
 - C. * Murphy's sign
 - D. absence of muscle tension in the right hypochondrium
 - E. Musso's sign
297. Patient V., aged 45, was admitted to hospital with a diagnosis: chronic cholecystitis. What is a reliable radiological sign of chronic cholecystitis?
- A. weak fluoroscopic shadow of the gall bladder
 - B. sharply increased, not reduced by giving choleretic breakfast
 - C. shadow of the gall bladder
 - D. disconnected" gallbladder
 - E. shadow of suspicion on concretions in the gall bladder at a reduced by 1 / 3 after the administration of choleretic breakfast
298. Patients after cholecystectomy in the immediate postoperative period gradually increase jaundice, these operating cholangiography not indicate the pathology of the bile ducts. The most likely cause of jaundice
- A. serum hepatitis
 - B. common bile duct stone
 - C. hemolytic jaundice
 - D. * operating choledochal injury (ligation)
 - E. all of the above
299. Patients after cholecystectomy in the immediate postoperative period gradually increase jaundice, these operating cholangiography not indicate the pathology of the bile ducts. The most likely cause of jaundice
- A. serum hepatitis
 - B. common bile duct stone
 - C. hemolytic jaundice
 - D. * There is no right answer
 - E. All of the above
300. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * abdominal X-ray
 - B. angiography
 - C. Doppler
 - D. celiocentesis
 - E. vulvotsentz
301. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * endoscopic retrograde cholangiography
 - B. angiography
 - C. doppler
 - D. laparocentesis
 - E. vulvocentesis

302. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * ultrasound abdominal
 - B. angiography
 - C. doppler
 - D. laparocentesis
 - E. vulvocentesis
303. Patients with suspected calculous cholecystitis for diagnosing stones in the gallbladder indicate:
- A. * intravenous cholecystocholangiography
 - B. angiography
 - C. doppler
 - D. laparocentesis
 - E. vulvocentesis
304. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic hography
 - E. * hypotonic doudenography
305. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic hography
 - E. * all answer are right
306. The patient admitted to hospital with suspected choledocolithiasis. What method of diagnosis is not used in this case?
- A. ultrasound
 - B. intravenous cholecystocholangiography
 - C. ERPHG
 - D. transhepatic hography
 - E. * endoscopy
307. The patient K., 50 years old, six months after cholecystectomy performed at the calculouse chronic cholecystitis, again began to appear in the right hypochondrium pain, occasionally accompanied by yellowing of the sclera. Ultrasound examination of the abdominal cavity obvious pathology of the extrahepatic bile duct could not be detected. Which of the following methods is most informative for diagnosis in this case?
- A. infusion hography,
 - B. oral cholecystography,
 - C. * endoscopic retrograde cholangiopancreatography,
 - D. scanning of the liver,
 - E. computed tomography
308. The patient K., 50 years old, six months after cholecystectomy performed at the calculouse chronic cholecystitis, again began to appear in the right hypochondrium pain, occasionally

accompanied by yellowing of the sclera. Which of the following methods is most informative for diagnosis in this case?

- A. infusion holecystography,
 - B. oral cholecystography,
 - C. * SONOGRAPHY
 - D. scanning of the liver,
 - E. computed tomography
309. The patient was diagnosed during surgery iatrogenic injury extrarenal bile ducts. What operation is indicated?
- A. closure of the injury duct separate atraumatic suture needle
 - B. suturing of the duct on the T-shaped drainage
 - C. closure of the duct on Г-shaped drainage
 - D. imposition biliodigestiv anastomosis
 - E. * any of the above
310. The patient with suspected narrowing of the distal common bile duct for diagnosis you must:
- A. * endoscopic retrograde cholangiography
 - B. echocardiography
 - C. laparocentesis
 - D. certain fraction of bilirubin
 - E. definition level of blood flow in truncus coeliacus
311. The patient with suspected narrowing of the distal common bile duct for diagnosis you must:
- A. * there is no right answer
 - B. echo kardiometriya
 - C. laparocentesis
 - D. certain fraction of bilirubin
 - E. definition level of blood flow in truncus coeliacus
312. A patient 30 years complain for the unpleasant feeling in the area of rectum and periodic bleeding at the end of act of defecation, fall off haemorrhoidal knots at defecation. He is ill a few years. The state is satisfactory. At anoscopy enlarged haemorrhoidal knots are determined at 11 hour. What operation is radical in this case?
- A. * Operation of Milligan-Morgan
 - B. Ligation of haemorrhoidal knots
 - C. Operation of Habriel
 - D. Sclerosing injections
 - E. Conservative treatment
313. A patient 36 years complain for pain in a crotch, fever, high temperature. He is ill a 5 days. A disease began acutely. State of middle weight. At a examination slight swelling of buttock on the right, pain at palpation. At the finger inspection of rectum acutely sickly compression, that swelling in a rectum. For a patient diagnosis?
- A. * a acute ishiorectal paraproctitis
 - B. acute submucous paraproctitis
 - C. acute pelviorectal paraproctitis
 - D. extrasphincteric fistula
 - E. thrombosis
314. A patient 36 years complain for pain in a crotch, fever, high temperature. He is ill a 5 days. A disease began acutely. State of middle weight. At a examination slight swelling of buttock on

the right, pain at palpation. At the finger inspection of rectum acutely sickly compression, that swelling in a rectum. What treatment is indicated to the patient?

- A. * opening of paraproctitis
 - B. operation after Habriel
 - C. conservative
 - D. operation after Ryzhykh- 1
 - E. an operation is after Kenu-Mailce
315. A patient 38 years complain for pain in the area of anus, fever. He is ill a 5 days. A disease began acutely. In anamnesis an operation is concerning a acute paraproctitis 2 years ago. At a examination on the left from anus slight swelling, turning, pain at palpation. In the area of scar of 2cm from anus point opening with festering excretions. What treatment is indicated to the patient?
- A. * operation after Habriel
 - B. opening of paraproctitis
 - C. conservative
 - D. operation after Ryzhykh- 1
 - E. an operation is after Kenu-Mailce
316. A patient 38 years complain for pain in the area of anus, fever. He is ill a 5 days. A disease began acutely. In anamnesis an operation is concerning a acute paraproctitis 2 years ago. At a examination on the left from anus slight swelling, turning, pain at palpation. In the area of scar of 2cm from anus point opening with festering excretions. What diagnosis?
- A. * recrudescant paraproctitis
 - B. ishiorectal paraproctitis
 - C. pelviorectal paraproctitis
 - D. submucous paraproctitis
 - E. thrombosis
317. A patient 60 years complain for pain in an anal area, high temperature of body, diarrhoea with constipations, periodically appearance of blood in incandescence. What sign testify in behalf on a acute paraproctitis in this case ?
- A. * pain, high temperature of body
 - B. enterorrhagia
 - C. diarrhoea
 - D. constipations
 - E. only pain
318. A patient with 10-years anamnesis of unspecific ulcerative colitis has periodic swelling of abdomen, feeling of the incomplete emptying, worsening of the general state. On irrigography is absent haustration with circular narrowing in sigmoid bowel. What complication patient has?
- A. * A regeneration to the cancer
 - B. Toxic dilatation
 - C. Bleeding
 - D. Perforation
 - E. Nothing
319. A patient with 10-years anamnesis of unspecific ulcerative colitis has periodic swelling of abdomen, feeling of the incomplete emptying, worsening of the general state. On irrigography is absent haustration with circular narrowing in sigmoid bowel. What operation is prescribed?
- A. * Colproctectomy
 - B. Resection of sigmoid bowel
 - C. Left-side hemicolectomy
 - D. Sigmoidostomy

- E. Right-side hemicolectomy
320. A patient B., 35 years, complains for a periodic pain in abdomen, frequent (up to 16 - 18 on twenty-four hours) unexecuted stool with mucus and blood. An unspecific ulcerative colitis is diagnosed. The sudden stopping of diarrhoea came on a background of heavy intoxication. The indicated changes of signs of disease means:
- A. efficiency of conservative therapy
 - B. * toxic dilatation
 - C. misdiagnosis
 - D. perforation
 - E. hypovolemia
321. At colonoscopy for a patient 60 years old was removed the polypus of sigmoid bowel in the distance 35 cm from anus. Histologically was found a microinvasive cancer an it apex. On it basis it is not found the cancer's cell. What is tactic of doctor?
- A. * Repeated colonoscopies every 3 months
 - B. Radial therapy
 - C. The resection of sigmoid bowel
 - D. Chemotherapy
 - E. Nothing
322. For a patient 25 years acute horseshoe paraproctitis is diagnosed. What operation is indicated ?
- A. * Excision of abscess is with realization of ligature
 - B. Excision of abscess is with imposition of sutures
 - C. Excision of abscess (as Habriel)
 - D. Excision of abscess is with moving of mucous membrane of distal part of rectum
 - E. Conservative treatment
323. For a patient 25 years three month ago the ulcer of rectum is diagnosed. At a complex examination – Wassermann test is positive. What is diagnosis
- A. * venereal limphogranuloma
 - B. hemorrhoids
 - C. paraproctitis
 - D. fistula
 - E. crack of rectum
324. For a patient 26 years a acute ishiorectal paraproctitis is diagnosed. What operation is indicated ?
- A. * Excision of abscess is with realization of ligature
 - B. Excision of abscess is with imposition of sutures
 - C. Excision of abscess (by Habriel)
 - D. Excision of abscess is with moving of mucous membrane of distal part of rectum
 - E. Conservative treatment
325. For a patient 27 years a acute subcutaneus paraproctitis is diagnosed. What operation is indicated?
- A. * Excision of abscess (as Habriel)
 - B. Excision of abscess is with imposition of sutures
 - C. Excision of abscess is with realization of ligature
 - D. Excision of abscess is with moving of mucous membrane of distal part of rectum
 - E. Conservative treatment

326. For a patient 28 years a chronic transsphincteric paraproctitis is diagnosed. What operation is indicated ?
- A. * Excision of fistula is with the partial suturing of bottom of wound
 - B. Excision of fistula (as Habriel)
 - C. Excision of fistula is with realization of ligature
 - D. Excision of fistula is with moving of mucous membrane of distal part of rectum
 - E. Conservative treatment
327. For a patient 29 years a chronic extrasphincteric paraproctitis is diagnosed. What operation is indicated ?
- A. * Excision of fistula is with realization of ligature
 - B. Excision of fistula is with imposition of deaf sutures
 - C. Excision of fistula (as Habriel)
 - D. Excision of fistula is with moving of mucous membrane of distal part of rectum
 - E. Conservative treatment
328. For a patient a 23 combined hemorrhoids became complicated by bleeding. Pregnancy of 9 weeks. Tactic?
- A. * operation after Milligan-Morgan
 - B. operation after Habriel
 - C. to conduct operative intervention of after birth of child
 - D. after Ryzhykh- 1
 - E. for Kenu-Mailce
329. For a patient an unspecific ulcerative colitis is diagnosed. Prescribed anti-recurrent treatment. What is preparation of choice in treatment of disease?
- A. NSAID
 - B. Lipofundinum
 - C. Smecta
 - D. * Sulfasalasin
 - E. Salbutamololum
330. For a patient intersphincteric fistula of i is 30 years diagnosed external hemorrhoids. He is ill about 5 years. What operation is indicated?
- A. * After Milligan-Morgan and Habriel
 - B. By Milligan-Morgan.
 - C. By Habriel.
 - D. Operation Blinnichev.
 - E. Operation Ryzhykh- 1.
331. For a patient R. 58 years diagnosed fall-out of rectum of III stage. A capacity and quality of life of patient is bad. What is operation in this case?
- A. * Kumel-Zerenin.
 - B. Milligan-Morgan.
 - C. Kenu-Mailce.
 - D. Habriel.
 - E. Operation Blinnichev.
332. For a patient the combined hemorrhoids, complicated by a thrombosis, is diagnosed. A patient is disturbed by pain, discomfort, itch. What is the indication to the operation in this case
- A. * thrombosis of haemorrhoidal nodes
 - B. age
 - C. pain
 - D. itch

- E. discomfort
333. For a patient the unspecific ulcerative colitis of middle weight is diagnosed. It is prescribed prednisolon. What most optimal dose can be used in treatment of disease?
- A. 10-20 mg
 - B. 30-40 mg
 - C. * 60-80 mg
 - D. 35 mg.
 - E. 100 mg.
334. For a patient E., 67p., the perforation of bowel came on a background of unspecific ulcerative colitis. What operative treatment is indicated for the patient?
- A. * Suturing of perforation and ileostomy
 - B. Proximal colostomy
 - C. Total colectomy and ileostomy
 - D. Resection of segment of bowel with perforation
 - E. Conservative treatment
335. For a patient K., 37 years, an unspecific ulcerative colitis is diagnosed. Prescribe conservative treatment:
- A. Diet therapy (exception of milk and dairies)
 - B. Vitamin therapy
 - C. Sulphanilamide
 - D. Desintoxication therapy
 - E. * All listed are true
336. For a patient K., 37p., an unspecific ulcerative colitis is diagnosed. What does not prescribed at unspecific ulcerative colitis for treatment?
- A. Antibiotics
 - B. * Purgatives
 - C. Vitamins
 - D. Desintoxication therapy
 - E. Hormonal preparations
337. For a patient M., 45p., a unspecific ulcerative colitis is diagnosed. It is conducted irrigography. On a sciagram the characteristic sign of this disease is founded. It is a symptom:
- A. index finger"
 - B. roadways"
 - C. water-pipe"
 - D. half of month"
 - E. niches"
338. For a patient M., 45p., it is diagnosed unspecific ulcerative colitis, phase of remission. What is preparation of choice in treatment of disease?
- A. antiagregant
 - B. cytostatics
 - C. vitamins of group In
 - D. * preparations of 5-aminosalicile acid
 - E. hormones
339. For a patient C., 44 years., erosive proctitis is diagnosed. What is preparation of choice in treatment of disease?
- A. Antibiotics
 - B. Hormonal preparations

- C. * Sulphosalaso-drugs
 - D. Enzymes preparations
 - E. Vitamins
340. For a patient C., 45 years, poliposis of colon and rectum is diagnosed. A diagnosis is confirmed by irrigography. What symptom is characteristic for the indicated disease?
- A. water-pipe"
 - B. shot through target"
 - C. a specific symptom is absent
 - D. cockades"
 - E. roadways".
341. For a patient, 39p., an unspecific ulcerative colitis is diagnosed. In the case of development of complications, which complication does not need operative treatment?
- A. * Bleeding
 - B. Toxic megacolon
 - C. Water-electrolyte changes
 - D. Malignancy
 - E. Perforation
342. For the patient of 22 during 2 years it is diagnosed combined haemorrhoid. On this time - pregnancy 8 weeks. Tactic?
- A. * to conduct operative treatment after birth of child
 - B. operation after Habriel
 - C. operation after Milligan-Morgan
 - D. after Ryzhykh- 1
 - E. for Kenu-Mailce
343. For the patient of 24 years on posterior anal is founded fissure of mucous rectum. Pregnancy of 16 weeks. Tactic of surgeon.
- A. * Cut off fissure after birth of child
 - B. operation after Milligan-Morgan
 - C. operation after Habriel
 - D. cut off fissure
 - E. operation after Kenu-Mailce
344. For the patient of 60 year during last 5 years it is diagnosed fall-out of rectum. On this time it is II degree. Quality of life of patient is severe. What operation is indicated?
- A. * Kumel-Zerenin.
 - B. Milligan-Morgan.
 - C. Kenu-Mailce.
 - D. Habriel.
 - E. Operation Blinnichev.
345. On irrigography is found the symptoms of «water-pipe», «shot through target». What is diagnosis?
- A. * Unspecific ulcerative colitis
 - B. Crohn disease
 - C. Dysentery
 - D. Salmonellosis
 - E. Food toxicoinfection
346. Patient of Б., 30 years, a previous diagnosis is: Poliposis of colon. What most reliable method of diagnostics of polypus's of colon is indicated to the patient?:

- A. X-Ray with sulphate of barium
 - B. Irrigoscopy
 - C. * Colonoscopy
 - D. Examination of excrement
 - E. Laparoscopy
347. Patient of K, 45 years, diagnosis: unspecified ulcerative colitis. What treatment is indicated to the patient?
- A. Complete parenterally feed
 - B. Total colectomy with an ileostomy
 - C. Subtotal colectomy with ileostomy
 - D. * All are true
 - E. All are false
348. Patient 27 years old has stomach-ache, liquid stool up to 10 times per days with mucus and blood, weakness, weight lost. On the irrigigrahy is narrowing of transvers colon. What diagnosis?
- A. * Cancer of transvers colon.
 - B. Dysentery.
 - C. Polypus of small intestine.
 - D. Spastic colitis.
 - E. Unspecific ulcerative colitis
349. Patient 40 years complines for pain during time of defecation, spasm of sphincter, bleeding. These symptoms are characteristic for ?
- A. * anal fissure
 - B. proctopolypus
 - C. unspecific ulcerative colitis
 - D. shrine of rectum
 - E. a right answer is not
350. Patient 50 years old has permanent pain in the anus, frequent defecation with blood, lost of appetite, weight lost, weakness. What examination is prescribed?
- A. * A biopsy with histological examination
 - B. Radio-active scan
 - C. Selective angiography
 - D. Doplerography
 - E. Sonography
351. Patient 59 years old has suspicion of the tumor of ascending part of colon. What method of examination is the best?
- A. * A colonoscopy with a biopsy
 - B. Irrigography
 - C. Survey sciagraphy
 - D. Sonography
 - E. Endoscopy
352. Patient 72 years old has acute pain in the left half of abdomeb, nausea, delay of stool and gases. He is ill 6 hours. No mucus and blood in stool, not weight lost . Pulse 84 per 1 min. Peristaltic noises is increased periodically. On the X-Ray of organs of abdominal region is present the Kloyber's cup in the left half of abdomen. What diagnosis ?
- A. * Invagination of sigmoid bowel
 - B. Crohn disease
 - C. Poliposis

- D. Unspecific ulcerative colitis
 - E. Diverticulosis
353. Patient during the act of defecation has pain in anal channel, red blood in the stool. What disease?
- A. * Anal fissure
 - B. Haemorrhoids
 - C. Acute paraproctitis
 - D. Cancer of rectum
 - E. Proctopolypus
354. Patient has a general weakness, presence of dark blood in the stool. At a rectoscopy on 11 cm from anus is found the circular narrowing of rectum. What diagnosis?
- A. * Cancer of rectum
 - B. Proctopolypus
 - C. Acute paraproctitis
 - D. Chronic paraproctitis
 - E. Acute proctitis
355. Patient has diarrhea up to 25-30 times per days with blood, has weight lost, general weakness, periodic stomach-ache. He is ill during 1,5 month. What diagnosis?
- A. * Unspecific ulcerative colitis
 - B. colitis
 - C. Pseudopoliposis
 - D. Diverticulosis
 - E. Spastic colitis
356. Patient has swelling from an anal channel during the act of defecation, without pain, with fresh blood after defecation. Previous diagnosis?
- A. * Haemorrhoids
 - B. Anal fissure
 - C. Acute paraproctitis
 - D. Cancer of rectum
 - E. Proctopolypus
357. Patient M., 42 years, complains for moderate pain in abdomen, frequent stool up to 15 times on twenty-four hours. Temperature - 38C. Abdomen is mildly enlarged, painful in the colon. Rectally: enlarged, mildly sickly haemorrhoidal knots, dark blood. Rectoscopy: mucous membrane of colon with hyperaemia, filling out, covered by erosions. In general blood test is anaemia, ESR - a 54 mm/hour. What is drug used in treatment of disease?
- A. Anti-diarrhoea preparations
 - B. Sulphate of magnesium
 - C. * Sulphosalazo-drugs
 - D. Salbutamol
 - E. Moriamin
358. Patient of B., 38 years, entered with complaints of moderate stomach-ache, diarrhoea up to 17 times on twenty-four hours with blood, pus, mucus. Temperature of body is 38 C. Stomach is inflated, painful in the colon projection. Per rectum: dark blood is founded. It is anaemia, ESR - a 42 mm/hour. What is diagnosis?
- A. Enterocolitis
 - B. Crohn's disease.
 - C. Diverticulosis
 - D. * Unspecific ulcerative colitis

- E. Specific ulcerative colitis
359. Patient of B. in 35, complain for periodic pain in a right inguinal area, increase of T to 38C., general weakness, diarrhoea with the admixtures of blood. At a rectoscopy is a "symptom of roadway". What is the most credible diagnosis for a patient?
- Disease of Hirshprung
 - Dysentery
 - Unspecific ulcerative colitis
 - * Crohn's disease
 - Enterocolitis
360. Patient of M., 66 years, became ill suddenly: pain in the left inguinal area, temperature 38 C. At examination insignificant tension of muscle and pain in the left inguinal area. Specify the diagnosis.
- Stenosis of sigmoid bowel
 - * Diverticulitis of sigmoid bowel
 - Poliposis of colon
 - Volvulus of sigmoid bowel
 - Crohn's disease
361. Patient M., 45 years, passed irrigography. Clinically and on the sciagram a toxic megacolon is founded. For what disease these changes is characteristic?
- Crohn's disease
 - Disease of Hirshprung
 - To the syndrome of Gardner
 - To the syndrome of Paits-Egers
 - * Unspecific ulcerative colitis
362. Patient H. 32 years, complains for periodic pain in a right inguinal area, increase of temperature to 38C., general weakness, diarrhoea with the admixtures of blood. At a rectoscopy an edema, hyperaemia, plural erosions, ulcers, festering and necrotizing raid of mucous membrane of intestine, is founded. What possible diagnosis?
- Enterocolitis
 - * Unspecific ulcerative colitis
 - Poliposis
 - Crohn's disease
 - Diverticulitis.
363. Patient, 70 years, during the last 3 months disturb constipation. During the last 2 weeks was absent of stool during 3-4 days. Patient is used purgative drugs. One week ago was bleeding from rectum – up to 200 ml of the fresh blood. Lost of weight up to 10 kg, an appetite is bad. At examination the general state is middle weight. During palpation of abdomen tumour-like formation is palpated in the left iliac area by size 6x8 cm. At percussion - tympanitis. It is not founded any pathology at digital examination of rectum. What previous diagnosis?
- Diverticulitis of sigmoid bowel
 - Megacolon
 - * Tumour of the left half of colon
 - Volvulus of sigmoid bowel
 - No right answer.
364. The patient 32 years complain for the expressed pain in the area of anus, which arises up at the end of act of defecation, admixtures of blood in incandescence. He is ill for a year. Pains were intensive at first, intensity diminished then. At a examination on the back commissure of

anal ring longitudinal linear wound a 21 cm, pale-grey, with the hypertrophied roller.

Operation, that indicated to the patient?

- A. * Cut off the fissure with dosed sphincterotomy.
- B. Suturing of fissure.
- C. Operation Milligan-Morgan.
- D. Operation Habriel.
- E. Operation Noblja.

365. The patient 32 years complain for the expressed pain in the area of anus, which arises up at the end of act of defecation, admixtures of blood in stool. He is ill for a year. Pains were intensive at first, intensity diminished then. At a examination on the back commissure of anal ring longitudinal linear wound 21 cm, pale-grey, with the hypertrophied roller. Your diagnosis?

- A. * Posterior anal fissure
- B. proctopolypus
- C. unspecific ulcerative colitis
- D. cancer of rectum
- E. hemorrhoids

366. The patient of 28 years complain for the unpleasant feeling in the area of rectum and periodic bleeding at the end of act of defecation. He is ill a few years. The state is satisfactory. Palpation of stomach - without pathology. At anoscopy the haemorrhoidal knot are determined at 11 hour, blood with the edema of mucous membrane. Haemoglobin is 100 /L, red corpuscles 2,7 * 10¹². What is a diagnosis?

- A. * Internal haemorrhoid, bleeding, anaemia.
- B. Gastric ulcer, bleeding, anaemia.
- C. Cancer of rectum, bleeding, anaemia.
- D. Unspecific ulcerative colitis, bleeding, anaemia.
- E. Poliposis, bleeding, anaemia.

367. The patient of 29 years complain for the unpleasant feeling in the area of rectum and periodic bleeding at the end of act of defecation. He is ill a few years. The state is satisfactory. Palpation of stomach - without pathology. It is not founded at the examination of anus and finger inspection of pathological structure. At anoscopy the haemorrhoidal knot are determined at 11 hour, blood with the edema of mucous membrane. Haemoglobin is 100 /L; red corpuscles 2,7 * 10¹². Treatment?

- A. * Operation Milligan-Morgan.
- B. Ryzhykh- 1.
- C. Ryzhykh- 2.
- D. Operation Habriel.
- E. Operation Blinnichev.

368. The patient of 33 complain for the unpleasant feeling in the area of rectum and periodic bleeding at the end of act of defecation, fall off haemorrhoidal knots at defecation. He is ill a few years. The state is satisfactory. At anoscopy are enlarged haemorrhoidal knots and opening of incomplete intersphincteric fistula determined. What operation is radical in this case?

- A. * After Milligan-Morgan and Habriel.
- B. For Milligan-Morgan.
- C. For Habriel.
- D. Operation Blinnichev.
- E. Operation Ryzhykh- 1.

369. The patient of 33 years complain for the expressed pains in the area of anus, that arise up at the end of act of defecation, admixtures of blood in incandescence. He is ill for a year. Pains were intensive at first, intensity diminished then. At a examination on the back commissure of

anal ring longitudinal linear wound a 21 cm, pale-grey, with the hypertrophied roller. On a 2 cm higher line of comb polypus on a narrow leg diameter by 0.5 cm. What operations at posterior anal fissure with polypus?

- A. * Cut off of fissure and removal of polypus
- B. Cut off of fissure.
- C. For Milligan-Morgan.
- D. For Habriel.
- E. Operation Blinnichev.

370. The patient of 43 after the operation of haemorrhoidectomy had cicatrice stricture of anus. What operation is indicated?

- A. * dosed sphincterotomy and suturing of mucous rectum to perianal skin
- B. bougie of rectum from suturing of mucous rectum to perianal skin
- C. dosed sphincterotomy
- D. hemorrhoidectomy
- E. colostomy

371. The patient of B., 47 years, entered to surgical department with complaints for pain in the left inguinal area, weight lost. Objectively: T- 38 C. Stomach troubles, painful at palpation in the left inguinal area. At rectoscopy hyperaemia and deep cracks of mucous membrane, ulcer, stenosis is founded. What is preparation of choice in treatment of disease?

- A. Anticoagulants
- B. * 5-ASK, sulfasalasin, NSAID
- C. NSAID
- D. mercaptopurine
- E. Smecta, Imodium.

372. The patient M., 38 years, entered to surgical department with complaints for periodic pain in a right inguinal area, which arises up after the reception of meal, weight lost. Objectively: T- 38 C. Stomach is subinflated, painful at palpation in the left inguinal area. At rectoscopy is hyperaemia and deep cracks of mucous membrane, ulcer, stenosis. What is the diagnosis of patient?

- A. Dysentery
- B. Disease of Hirshprung.
- C. * Crohn's disease
- D. Unspecific ulcerative colitis
- E. Salmonellas

373. The patient C., in 46 years, entered with complaints of pain in the left inguinal area, diarrhoea up to 20-30 times on twenty-four hours, with the admixtures of blood and mucus. T - 38 °C. Per rectum is a mucous with hyperaemia, swelling, covered by the ulcers of different size, with bleeding. What disease?

- A. Diverticulums
- B. Polyps
- C. Poliposis
- D. Diverticulosis
- E. * Unspecific ulcerative colitis

374. To the patient K., 62 years, roentgenologically and on colonoscopy is diagnosed unspecific ulcerative colitis. What method of treatment of this patient?

- A. * enemas with sulfasalasin
- B. enemas with smecta
- C. enemas with NSAID
- D. enemas with cytostatics

- E. enemas with a celandine
375. A man 50 years of asymptomatic mixed goiter. At the first stage, it should appoint
- A. thyroid hormones to suppress the function of cancer
 - B. propylthiouracil
 - C. subtotal thyroidectomy
 - D. radioiodine
 - E. * only observation
376. In patient K, aged 26, noted a relapse of hyperthyroidism after medical treatment. Your tactics.
- A. Continue medical treatment
 - B. * Surgical treatment
 - C. Outpatient
 - D. Treatment is not required
 - E. Is no right answer
377. On examination, the patient was 32 years reveal the formation of the left lobe of the thyroid gland size 4x6 cm, painless at palpation. What additional diagnostic method to assign?
- A. Radiography of the neck
 - B. * Thyroid gland
 - C. Doppler
 - D. Rheovasography
 - E. EEG
378. On examination, the patient was 32 years reveal the formation of the left lobe of the thyroid gland size 4x6 cm, painless at palpation. What analysis should be performed in order to clarify the diagnosis?
- A. Total blood
 - B. Urinalysis
 - C. Immunogram
 - D. * Thyroid hormones
 - E. Protein fraction
379. Patient D, aged 39, admitted to the hospital with the diagnosis: diffuse toxic goiter. What study be done.
- A. ENDOSCOPY
 - B. Is no right answer
 - C. Rheovasography
 - D. * Investigation of iodine hormones in the blood serum
 - E. Doppler
380. Patient J., 57, was admitted to the surgical clinic with a diagnosis: euthyroid nodular goiter. Choose the correct treatment option.
- A. enucleation
 - B. medication
 - C. subtotal resection of the thyroid gland
 - D. * resection of the thyroid gland with maximal preservation of healthy tissue and routine histological examination
 - E. excision of the node with the routine histological examination
381. Patient K, aged 49, was admitted to the hospital with the diagnosis: diffuse toxic goiter. What study be done.
- A. ENDOSCOPY

- B. Rheovasography
 - C. * Scanning of the thyroid gland
 - D. Doppler
 - E. There is no correct answer
382. Patient K., aged 45, lives in the area of iodine deficiency, was admitted to the clinic with complaints of enlarged thyroid gland. What is the most likely diagnosis in a patient?
- A. * goiter
 - B. acute strumitis
 - C. sporadic goiter
 - D. epidemic goiter
 - E. mass thyrotoxicosis
383. Patient M, 39 years old, was admitted to the hospital with the diagnosis: diffuse toxic goiter. What study be done.
- A. ENDOSCOPY
 - B. * Thyroid gland
 - C. Rheovasography
 - D. Doppler
 - E. Is no right answer
384. Patient M., aged 35, lives in the area of iodine deficiency, was admitted to the clinic with complaints of enlarged thyroid gland. What is the most likely diagnosis in a patient?
- A. acute strumitis
 - B. * there is no right answer
 - C. sporadic goiter
 - D. epidemic goiter
 - E. mass thyrotoxicosis
385. Patient O., aged 39, on the diffuse toxic goiter performed subtotal resection of the thyroid gland. One day the patient became restless, twitching of facial muscles appeared convulsive reduction of hands. Treatment.
- A. * the introduction of calcium chloride intravenously
 - B. introduction Seduxen
 - C. introduction of iodine
 - D. infusion therapy
 - E. introduction of calcium chloride oral
386. Patient O., aged 39, on the diffuse toxic goiter performed subtotal resection of the thyroid gland. One day the patient became restless, twitching of facial muscles appeared convulsive reduction of hands. What mated complication of surgery?
- A. Iodine deficiency
 - B. Lack of thyroid tissue
 - C. * Removal of parathyroid glands
 - D. Increased thyroid hormone
 - E. Increased parathyroid hormone
387. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. At SONOGRAPHY: tissue homogeneous, tissue hypertrophy hyper. The most likely diagnosis
- A. Hashimoto struma
 - B. * toxic goiter
 - C. Acute thyroiditis

- D. nodular goiter
 - E. goiter De Quervain
388. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. At SONOGRAPHY: tissue homogeneous, tissue hypertrophy hyper. The most likely diagnosis
- A. Hashimoto's thyroiditis
 - B. * there is no right answer
 - C. Acute thyroiditis
 - D. nodular goiter
 - E. goiter De Quervain
389. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. What additional diagnostic method to assign?
- A. Radiography of the neck
 - B. * Thyroid gland
 - C. Doppler
 - D. Reovazogrifyu
 - E. EEG
390. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. What analysis should be performed in order to clarify the diagnosis?
- A. Total blood
 - B. Urinalysis
 - C. * Thyroid hormones
 - D. Protein fraction
 - E. Immunogram
391. Patients after resection of the thyroid having convulsions, symptoms by Chvostek and Trousseau. What a complication arose in a patient?
- A. * there is no right answer
 - B. laryngeal nerve injury
 - C. residual effects of hyperthyroidism
 - D. thyrotoxic crisis
 - E. hypothyroidism
392. Patients after resection of the thyroid having convulsions, symptoms by Chvostek and Trousseau. What a complication is arose in a patient?
- A. laryngeal nerve injury
 - B. * hypoparathyreosis
 - C. residual effects of hyperthyroidism
 - D. thyrotoxic crisis
 - E. hypothyroidism
393. The patient complaints of excessive sweating, hand tremor, exophthalmos. The most likely diagnosis.
- A. Goiter
 - B. Tireodit
 - C. Thyrotoxicosis
 - D. * Strumil

- E. Is no right answer
394. The patient diagnosed with an aberrant goiter. Refine the definition of aberrant goiter.
- A. metastases of thyroid cancer in the liver
 - B. atypical location of the thyroid gland
 - C. * is a cancer of the thyroid gland
 - D. all true
 - E. all wrong
395. The patient diagnosed with nodular euthyroid goiter left lobe of the thyroid gland. What is the optimal treatment option.
- A. excision of the node with the routine histological examination
 - B. enucleation site
 - C. subtotal thyroidectomy
 - D. * hemistrumectomy or resection of the lobe of the thyroid gland
 - E. conservative treatment thyroidin
396. The patient lives in the area of endemic iodine. What can be used to prevent goitre?
- A. merkasalil
 - B. vaccination
 - C. iodine
 - D. improving the social life of the population
 - E. * iodination salt
397. The patient S., 546 years old, diagnosed with goiter II degree. What is characteristic of the III degree of increase in thyroid gland?
- A. giant goiter
 - B. determined only by palpation
 - C. * visible swallowing
 - D. visible only when swallowing
 - E. determined only on ultrasound
398. The patient S., 61, suffering for 2 years nodular goiter, recently noted a rapid increase in the node. On scanning image identified a "cold node". Specify the most probable cause of this condition
- A. * node malignancy
 - B. cystic degeneration of the node
 - C. autoimmune
 - D. all true
 - E. hemorrhage site
399. The patient S., 61, suffering for 2 years nodular goiter, recently noted a rapid increase in the node. What method of diagnosis is the most informative?
- A. X-ray of the neck
 - B. * scanning with radioactive iodine
 - C. Doppler
 - D. Rheovasography
 - E. EEG
400. The patient V., age 56, diagnosed with goiter of third degree. What is characteristic of the III degree of increase in thyroid gland?
- A. giant goiter
 - B. determined only by palpation
 - C. * visible without swallowing

- D. visible only when swallowing
- E. determined only on ultrasound

401. The patient's 43 years revealed an increase in the left lobe of the thyroid gland. When scanning in this region found a hot site. Diagnosis.

- A. diffuse non-toxic goiter
- B. toxic goiter
- C. multinodular toxic goiter
- D. * nodular toxic goiter
- E. non-toxic nodular goiter

402. The patient's 60 years in the last 3 months has been rapidly increasing dense mass in the left lobe of the thyroid gland. Effects of hyperthyroidism is not. At thyroid scan revealed a cold junction. Preliminary diagnosis

- A. * thyroid cancer
- B. lipoma of the thyroid gland
- C. cyst
- D. metastasis of lung cancer
- E. thyroid cyst

MODULE 2. THORACIC, CARDIOVASCULAR AND ENDOCRINE SURGERY.

Text test questions

1. What is the abscessing pneumonia characterized by?
 - A. *Multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs
 - B. Purulent destruction of pulmonary tissue within 1 segment with formation of cavity, filled by pus
 - C. Purulent, necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma
 - D. Diffuse purulent, ichorous necrosis more than lobe without the tendency to defined demarcation
 - E. Accumulation of pus in a pleural cavity
2. What is the lung abscess characterized by?
 - A. *Purulent destruction of pulmonary tissue within 1 segment with formation of cavity, filled by pus
 - B. Multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs
 - C. Purulent, necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma
 - D. Diffuse purulent, ichorous necrosis more than lobe without the tendency to defined demarcation
 - E. Accumulation of pus in a pleural cavity
3. What is the lung gangrenous abscess characterized by?
 - A. *Purulent, necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma
 - B. Multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs
 - C. Purulent destruction of pulmonary tissue within 1 segment with formation of cavity, filled by pus
 - D. Diffuse purulent, ichorous necrosis more than lobe without the tendency to defined demarcation
 - E. Accumulation of pus in a pleural cavity
4. What is the lung gangrene characterized by?
 - A. *Diffuse purulent, ichorous necrosis more than lobe without the tendency to defined demarcation
 - B. Multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs
 - C. Purulent destruction of pulmonary tissue within 1 segment with formation of cavity, filled by pus
 - D. Purulent, necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma
 - E. Accumulation of pus in a pleural cavity
5. Multiple destructive foci 0,3-0,5 cm in size within 1-2 segments of lungs are called:
 - A. *Abscessing pneumonia
 - B. Lung abscess
 - C. Lung gangrenous abscess
 - D. Lung gangrene
 - E. Bronchoectatic disease
6. Purulent destruction of pulmonary tissue within 1 segment with formation of cavity, filled by pus is called:
 - A. *Lung abscess
 - B. Abscessing pneumonia
 - C. Lung gangrenous abscess
 - D. Lung gangrene
 - E. Bronchoectatic disease

7. Purulent, necrosis of a pulmonary tissue within 2-3 segments, detached from adjacent pulmonary parenchyma is called:
 - A. *Lung gangrenous abscess
 - B. Lung abscess
 - C. Abscessing pneumonia
 - D. Lung gangrene
 - E. Bronchoectatic disease
8. Diffuse purulent, ichorous necrosis beyond the lobe without the tendency to defined demarcation is called:
 - A. *Lung gangrene
 - B. Lung gangrenous abscess
 - C. Lung abscess
 - D. Abscessing pneumonia
 - E. Bronchoectatic disease
9. What type of the lung gangrene is considered to be limited?
 - A. *The lesion within 1 lobe
 - B. The lesion within 1 segment
 - C. The total lesion of 1 lung
 - D. The lesion of 2 lungs
 - E. The lesion of lungs and pleura
10. What is the I stage of lung abscess?
 - A. *Necrotic pneumonia
 - B. Asymptomatic
 - C. Destruction and rejection
 - D. Cleaning and cicatrization
 - E. Gangrenous
11. What is the II stage of lung abscess?
 - A. *Destruction and rejection
 - B. Asymptomatic
 - C. Necrotic pneumonia
 - D. Cleaning and cicatrization
 - E. Gangrenous
12. What is the III stage of lung abscess?
 - A. *Cleaning and cicatrization
 - B. Asymptomatic
 - C. Necrotic pneumonia
 - D. Destruction and rejection
 - E. Gangrenous
13. What stage of lung abscess correlates with the necrotic pneumonia?
 - A. *I
 - B. II
 - C. III
 - D. IV
 - E. V
14. What stage of lung abscess correlates with the destruction and rejection?
 - A. *II
 - B. I
 - C. III
 - D. V
 - E. IV
15. What stage of lung abscess correlates with the cleaning and cicatrization?
 - A. *III
 - B. II
 - C. I

- D. IV
 - E. V
16. What is the predominant factor which causes the lung abscess?
 - A. *Disturbances of bronchial permeability with the development of atelectasis
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Pulmonary hypertension
 - D. Rheumatism, endocarditis
 - E. Myocardial infarction
 17. What is the predominant factor which causes the lung abscess?
 - A. *Infection in a pulmonary tissue
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Pulmonary hypertension
 - D. Rheumatism, endocarditis
 - E. Myocardial infarction
 18. What is the predominant factor which causes the lung abscess?
 - A. *Regional disturbances of pulmonary blood supply with a further necrosis of parenchyma
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Pulmonary hypertension
 - D. Rheumatism, endocarditis
 - E. Myocardial infarction
 19. What acute complication is characteristic for lung abscess?
 - A. *Pulmonary bleeding
 - B. Emphysema
 - C. Rib fracture
 - D. Malignancy
 - E. Esophageal bleeding
 20. What acute complication is characteristic for lung abscess?
 - A. *Pyopneumothorax
 - B. Rib fracture
 - C. Emphysema
 - D. Malignancy
 - E. Esophageal bleeding
 21. What complication is characteristic for lung abscess?
 - A. *Pleural empyema
 - B. Esophageal bleeding
 - C. Rib fracture
 - D. Emphysema
 - E. Malignancy
 22. What complication is characteristic for lung abscess?
 - A. *Sepsis
 - B. Esophageal bleeding
 - C. Rib fracture
 - D. Emphysema
 - E. Malignancy
 23. What complication is characteristic for lung abscess?
 - A. *Bronchogenic dissemination
 - B. Esophageal bleeding
 - C. Rib fracture
 - D. Emphysema
 - E. Malignancy
 24. For the clinical manifestation of lung abscess is typical:
 - A. *Chest pain
 - B. Abdominal pain

- C. Paralysis of intercostal nerve
 - D. Edema of legs
 - E. Dilated cervical veins
25. For the clinical manifestation of lung abscess is typical:
- A. *Dyspnea
 - B. Abdominal pain
 - C. Paralysis of intercostal nerve
 - D. Edema of legs
 - E. Dilated cervical veins
26. For the clinical manifestation of lung abscess is typical:
- A. *Fever to 39-40°C
 - B. Abdominal pain
 - C. Paralysis of intercostal nerve
 - D. Edema of legs
 - E. Dilated cervical veins
27. For the clinical manifestation of lung abscess is typical:
- A. *Troubling cough
 - B. Abdominal pain
 - C. Paralysis of intercostal nerve
 - D. Edema of legs
 - E. Dilated cervical veins
28. For the clinical manifestation of lung abscess is typical:
- A. *Intoxication
 - B. Abdominal pain
 - C. Paralysis of intercostal nerve
 - D. Edema of legs
 - E. Dilated cervical veins
29. For the clinical manifestation of lung abscess is typical:
- A. *Troubling cough with foul-smelling sputum
 - B. Abdominal pain
 - C. Paralysis of intercostal nerve
 - D. Edema of legs
 - E. Dilated cervical veins
30. The fever to 39-40°C is typical for:
- A. *Lung abscess
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Lung cyst
31. The chest pain is typical for:
- A. *Lung abscess
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Lung cyst
32. The intoxication is typical for:
- A. *Lung abscess
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Lung cyst
33. The troubling cough with foul-smelling sputum is typical for:
- A. *Lung abscess
 - B. Bronchitis

- C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Lung cyst
34. What is revealed in acute lung abscess by percussion?
- A. *Blunted sound
 - B. Bandbox sound
 - C. Tympanic sound
 - D. Clear sound
 - E. Pulmonary sound
35. The blunted sound by percussion is typical for:
- A. *Lung abscess
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pneumothorax
 - E. Lung cyst
36. The blunted sound by percussion is typical for:
- A. *Pneumonia
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pneumothorax
 - E. Lung cyst
37. The blunted sound by percussion is typical for:
- A. *Lung gangrene
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pneumothorax
 - E. Lung cyst
38. What is revealed in the I stage of acute lung abscess by auscultation?
- A. *Bronchial breathing with moist rales
 - B. Vesicular breathing
 - C. Amphoric breathing with moist rales
 - D. Harsh breathing with dry rales
 - E. The breathing isn't auscultated
39. What is revealed in the II stage (after draining) of acute lung abscess by auscultation?
- A. *Amphoric breathing with moist rales
 - B. Vesicular breathing
 - C. Bronchial breathing with moist rales
 - D. Harsh breathing with dry rales
 - E. The breathing isn't auscultated
40. What is revealed in the I stage of acute lung abscess by X-ray?
- A. *Rounded shadow with irregular contour
 - B. Rounded cavity with air-fluid level
 - C. Expressed fibrosis
 - D. Intensive shadow of a considerable area of lung with cavities and fluid levels
 - E. One or several cavities with a thick, dense pyogenic sheath
41. What is revealed in the II stage (after draining) of acute lung abscess by X-ray?
- A. *Rounded cavity with air-fluid level
 - B. Rounded shadow with irregular contour
 - C. Expressed fibrosis
 - D. Intensive shadow of a considerable area of lung with cavities and fluid levels
 - E. One or several cavities with a thick, dense pyogenic sheath
42. What is revealed in the III stage of acute lung abscess by X-ray?
- A. *Expressed fibrosis
 - B. Rounded shadow with irregular contour

- C. Rounded cavity with air-fluid level
 - D. Intensive shadow of a considerable area of lung with cavities and fluid levels
 - E. One or several cavities with a thick, dense pyogenic sheath
43. What is revealed in lung gangrene by X-ray?
- A. *Intensive shadow of a considerable area of lung with cavities and fluid levels
 - B. Rounded shadow with irregular contour
 - C. Rounded cavity with air-fluid level
 - D. Expressed fibrosis
 - E. One or several cavities with a thick, dense pyogenic sheath
44. What is revealed in chronic lung abscess by X-ray?
- A. *One or several cavities with a thick, dense pyogenic sheath
 - B. Rounded shadow with irregular contour
 - C. Rounded cavity with air-fluid level
 - D. Expressed fibrosis
 - E. Intensive shadow of a considerable area of lung with cavities and fluid levels
45. The rounded shadow with irregular contour on X-ray is typical for:
- A. *I stage of acute lung abscess
 - B. II stage of acute lung abscess
 - C. III stage of acute lung abscess
 - D. Lung emphysema
 - E. Lung cyst
46. The rounded cavity with air-fluid level on X-ray is typical for:
- A. *II stage of acute lung abscess
 - B. I stage of acute lung abscess
 - C. III stage of acute lung abscess
 - D. Lung emphysema
 - E. Lung cyst
47. The expressed fibrosis on X-ray is typical for:
- A. *III stage of acute lung abscess
 - B. II stage of acute lung abscess
 - C. I stage of acute lung abscess
 - D. Lung emphysema
 - E. Lung cyst
48. The intensive shadow of a considerable area of lung with cavities with sequesters and fluid levels on X-ray is typical for:
- A. *Lung gangrene
 - B. III stage of acute lung abscess
 - C. II stage of acute lung abscess
 - D. Lung emphysema
 - E. Lung cyst
49. One or several cavities with a thick, dense pyogenic sheath on X-ray is typical for:
- A. *Chronic lung abscess
 - B. Lung gangrene
 - C. Acute lung abscess
 - D. Lung emphysema
 - E. Lung cyst
50. When the lung abscess is considered to be chronic?
- A. *In 6-8 weeks after the onset
 - B. In 10 days after the onset
 - C. In 3-4 weeks after the onset
 - D. In 6-8 months after the onset
 - E. In 1 year after the onset
51. What kind of X-ray shadow is typical for acute lung abscess before draining?
- A. *Rounded shadow with considerable perifocal infiltration

- B. Homogeneous spherical shadow with regular edge on the background of intact pulmonary tissue
 - C. Heterogeneous shadow with calcifications, excentric destruction and regular edge
 - D. Homogeneous spherical shadow with irregular edge and phenomena of lymphangitis (corona maligna)
 - E. Heterogeneous shadow with destruction, displaced in the upper lobes, with fibrosis, petrifications in adjacent tissue, peribronchial lymphadenitis
52. What kind of X-ray shadow is typical for lung cyst?
- A. *Homogeneous spherical shadow with regular edge on the background of intact pulmonary tissue
 - B. Rounded shadow with considerable perifocal infiltration
 - C. Heterogeneous shadow with calcifications, excentric destruction and regular edge
 - D. Homogeneous spherical shadow with irregular edge and phenomena of lymphangitis (corona maligna)
 - E. Heterogeneous shadow with destruction, displaced in the upper lobes, with fibrosis, petrifications in adjacent tissue, peribronchial lymphadenitis
53. What kind of X-ray shadow is typical for tuberculoma?
- A. *Heterogeneous shadow with calcifications and regular edge
 - B. Homogeneous spherical shadow with regular edge on the background of intact pulmonary tissue
 - C. Rounded shadow with considerable perifocal infiltration
 - D. Homogeneous spherical shadow with irregular edge and phenomena of lymphangitis (corona maligna)
 - E. Heterogeneous shadow with destruction, displaced in the upper lobes, with fibrosis, petrifications in adjacent tissue, peribronchial lymphadenitis
54. What kind of X-ray shadow is typical for peripheral lung cancer?
- A. *Homogeneous spherical shadow with irregular edge and phenomena of lymphangitis (corona maligna)
 - B. Homogeneous spherical shadow with regular edge on the background of intact pulmonary tissue
 - C. Rounded shadow with considerable perifocal infiltration
 - D. Heterogeneous shadow with calcifications and regular edge
 - E. Heterogeneous shadow with destruction, displaced in the upper lobes, with fibrosis, petrifications in adjacent tissue, peribronchial lymphadenitis
55. What kind of X-ray shadow is typical for tubercular cavern?
- A. *Heterogeneous shadow with destruction, displaced in the upper lobes, with fibrosis, petrifications in adjacent tissue, peribronchial lymphadenitis
 - B. Homogeneous spherical shadow with regular edge on the background of intact pulmonary tissue
 - C. Rounded shadow with considerable perifocal infiltration
 - D. Heterogeneous shadow with calcifications and regular edge
 - E. Homogeneous spherical shadow with irregular edge and phenomena of lymphangitis (corona maligna)
56. Homogeneous spherical shadow with regular edge on the background of intact pulmonary tissue on X-ray is typical for:
- A. *Lung cyst
 - B. Tuberculoma
 - C. Peripheral lung cancer
 - D. Tubercular cavern
 - E. Lung emphysema
57. Heterogeneous shadow with calcifications and regular edge on X-ray is typical for:
- A. *Tuberculoma
 - B. Lung cyst
 - C. Peripheral lung cancer

- D. Tubercular cavern
 - E. Lung emphysema
58. Homogeneous spherical shadow with irregular edge and phenomena of lymphangitis (corona maligna) on X-ray is typical for:
- A. *Peripheral lung cancer
 - B. Tuberculoma
 - C. Lung cyst
 - D. Tubercular cavern
 - E. Lung emphysema
59. Heterogeneous shadow with destruction, displaced in the upper lobes, with fibrosis, petrifications in adjacent tissue, peribronchial lymphadenitis on X-ray is typical for:
- A. *Tubercular cavern
 - B. Peripheral lung cancer
 - C. Tuberculoma
 - D. Lung cyst
 - E. Lung emphysema
60. The clinical dynamics of lung abscess which is characterized by prompt positive clinical, roentgenological and laboratory dynamics and recovery after the adequate treatment regards to the:
- A. *Favorable course
 - B. Non-progressive course
 - C. Progressing course
 - D. Incapsulated process
 - E. Complicated course
61. The clinical dynamics of lung abscess which is characterized by transforming of the process into the chronic form due to poor drainage of the suppurative focus and permanent purulent intoxication regards to the:
- A. *Non-progressive course
 - B. Favorable course
 - C. Progressing course
 - D. Incapsulated process
 - E. Complicated course
62. The clinical dynamics of lung abscess which is characterized by expansion of the zone of necrosis and destruction with transforming in gangrene regards to the:
- A. *Progressing course
 - B. Non-progressive course
 - C. Favorable course
 - D. Incapsulated process
 - E. Complicated course
63. The clinical dynamics of lung abscess which is characterized by the partial or complete obstruction of the draining bronchus combined with satisfactory resistance of the organism regards to the:
- A. *Incapsulated process
 - B. Progressing course
 - C. Non-progressive course
 - D. Favorable course
 - E. Complicated course
64. The clinical dynamics of lung abscess which is characterized by different kinds of complications regards to the:
- A. *Complicated course
 - B. Incapsulated process
 - C. Progressing course
 - D. Non-progressive course
 - E. Favorable course

65. What disease doesn't result in pulmonary bleeding?
- A. *Pleurisy
 - B. Lung gangrene and abscess
 - C. Lung cancer
 - D. Tuberculosis
 - E. Bronchiectatic disease
66. What pulmonary hemorrhage is classified as I degree?
- A. *Less than 300 ml
 - B. 300-500 ml
 - C. 500-700 ml
 - D. 700-1000 ml
 - E. More than 1500 ml
67. What pulmonary hemorrhage is classified as II degree?
- A. *500-700 ml
 - B. 50-100 ml
 - C. 100-300 ml
 - D. 700-1000 ml
 - E. More than 1500 ml
68. What pulmonary hemorrhage is classified as III degree?
- A. *More than 700 ml
 - B. 50-100 ml
 - C. 100-300 ml
 - D. 300-500 ml
 - E. 500-700 ml
69. The pulmonary hemorrhage less than 300 ml is classified as:
- A. *I degree
 - B. 0 degree
 - C. II degree
 - D. III degree
 - E. IV degree
70. The pulmonary hemorrhage within 500-700 ml is classified as:
- A. *II degree
 - B. I degree
 - C. 0 degree
 - D. III degree
 - E. IV degree
71. The pulmonary hemorrhage more than 700 ml is classified as:
- A. *III degree
 - B. II degree
 - C. I degree
 - D. 0 degree
 - E. IV degree
72. The coughing out of the bloody sputum without hemodynamic disturbances are characteristic for such degree of pulmonary bleeding:
- A. *I degree
 - B. III degree
 - C. II degree
 - D. 0 degree
 - E. IV degree
73. The decrease of arterial pressure on 20-30 mm Hg is characteristic for such degree of pulmonary bleeding:
- A. *II degree
 - B. I degree
 - C. III degree

- D. 0 degree
 - E. IV degree
74. The amount of hemoglobin within 60-80 g/l is characteristic for such degree of pulmonary bleeding:
- A. *II degree
 - B. I degree
 - C. III degree
 - D. 0 degree
 - E. IV degree
75. The tachycardia to 100 beats/min is characteristic for such degree of pulmonary bleeding:
- A. *II degree
 - B. I degree
 - C. III degree
 - D. 0 degree
 - E. IV degree
76. The decrease of arterial pressure to 40-60 mm Hg is characteristic for such degree of pulmonary bleeding:
- A. *III degree
 - B. II degree
 - C. I degree
 - D. 0 degree
 - E. IV degree
77. The rapid (more than 100-120 beats/min), small, thread pulse is characteristic for such degree of pulmonary bleeding:
- A. *III degree
 - B. II degree
 - C. I degree
 - D. 0 degree
 - E. IV degree
78. The amount of hemoglobin less than 50-60 g/l is characteristic for such degree of pulmonary bleeding:
- A. *III degree
 - B. II degree
 - C. I degree
 - D. 0 degree
 - E. IV degree
79. What is the main method of lung abscess sanitation?
- A. *Microtracheostomy
 - B. Transpleural
 - C. Transesophageal
 - D. Endovascular
 - E. Thoracotomy
80. What is the main treatment of noncomplicated acute lung abscess?
- A. *Conservative treatment
 - B. Segmentectomy
 - C. Lobectomy
 - D. Pneumonectomy
 - E. Pleural puncture
81. What medicine drug belongs to broad spectrum antibiotics?
- A. *Ceftriaxon, Ciprinol
 - B. Euphyllin, No-spa
 - C. Vasaprostan, Alprostan
 - D. Nicotine acid, Heparin
 - E. Detrlex, Venoplant

82. What medicine drug belongs to broad spectrum antibiotics?
- A. *Tebris, Ciprinol
 - B. Dimedrol, Suprastin
 - C. Vasaprostan, Alprostan
 - D. Nicotine acid, Heparin
 - E. Detrlex, Venoplant
83. What medicine drug belongs to broad spectrum antibiotics?
- A. *Zanocin
 - B. Naclofen
 - C. Vasaprostan
 - D. Nicotine acid
 - E. Omeprasol
84. What medicine used for the treatment of pulmonary bleeding?
- A. *Aminocapronic acid
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
85. What medicine used for the treatment of pulmonary bleeding?
- A. *Calcium chloridi
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
86. What medicine used for the treatment of pulmonary bleeding?
- A. *Dicinon
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
87. What medicine used for the treatment of pulmonary bleeding?
- A. *Vitamin K
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
88. What medicine used for the treatment of pulmonary bleeding?
- A. *Benzohexonium
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
89. What is the indication for operative treatment of acute abscess of lungs?
- A. *Pulmonary bleeding of II- III degree
 - B. Decompensation of the vital organs
 - C. Bilateral purulent destruction of lungs
 - D. Incurable malignant tumours
 - E. Pulmonary hypertension
90. What is the indication for operative treatment of acute abscess of lungs?
- A. *Progression of the process despite appropriate therapy
 - B. Decompensation of the vital organs
 - C. Bilateral purulent destruction of lungs
 - D. Incurable malignant tumours
 - E. Pulmonary hypertension

91. What is the indication for operative treatment of acute abscess of lungs?
- A. *Tense pyopneumothorax, which is failed to liquify by the draining of pleural space
 - B. Decompensation of the vital organs
 - C. Bilateral purulent destruction of lungs
 - D. Incurable malignant tumours
 - E. Pulmonary hypertension
92. What is the indication for operative treatment of acute abscess of lungs?
- A. *Impossibility to rule out the suspicion on a malignant tumour
 - B. Decompensation of the vital organs
 - C. Bilateral purulent destruction of lungs
 - D. Incurable malignant tumours
 - E. Pulmonary hypertension
93. What operation is performed in complicated acute lung abscess?
- A. *Pneumonectomy, bilobectomy, lobectomy
 - B. Draining of a pleural space
 - C. Transthoracic drainage of the abscess
 - D. Bronchial plastics
 - E. Transplantation of lungs
94. What is the main treatment of chronic lung abscess?
- A. *Pneumonectomy, bilobectomy, lobectomy
 - B. Draining of a pleural space
 - C. Transthoracic drainage of the abscess
 - D. Transplantation of lungs
 - E. Conservative treatment
95. The pneumonectomy is indicated for:
- A. *Chronic lung abscess
 - B. Pneumonia
 - C. Pulmonary hypertension
 - D. Lung emphysema
 - E. Pleural empyema
96. The pneumonectomy is indicated for:
- A. *Complicated lung abscess
 - B. Pneumonia
 - C. Pulmonary hypertension
 - D. Lung emphysema
 - E. Pleural empyema
97. The pneumonectomy is indicated for:
- A. *Lung gangrene
 - B. Pneumonia
 - C. Pulmonary hypertension
 - D. Lung emphysema
 - E. Pleural empyema
98. The pneumonectomy is indicated for:
- A. *Lung cancer
 - B. Pneumonia
 - C. Pulmonary hypertension
 - D. Lung emphysema
 - E. Pleural empyema
99. The pneumonectomy is indicated for:
- A. *Tuberculosis cavern
 - B. Pneumonia
 - C. Pulmonary hypertension
 - D. Lung emphysema
 - E. Pleural empyema

100. What is the cause of pleural empyema?
 - A. *Destructive processes of lungs
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
101. What is the cause of pleural empyema?
 - A. *Abscesses of abdominal cavity
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
102. What is the cause of pleural empyema?
 - A. *Open and closed damages of chest
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
103. What is the cause of pleural empyema?
 - A. *Operative approaches on thoracic organs
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
104. What sign is not typical for pleural empyema?
 - A. *Hemoptysis
 - B. Pain
 - C. Dyspnea
 - D. Cough
 - E. Intoxication
105. For the clinical manifestation of pleural empyema is typical:
 - A. *Chest pain
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
106. For the clinical manifestation of pleural empyema is typical:
 - A. *Dyspnea
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
107. For the clinical manifestation of pleural empyema is typical:
 - A. *Cough with sputum
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
108. For the clinical manifestation of pleural empyema is typical:
 - A. *Intoxication
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins

109. What is revealed in pleural empyema by percussion?
- A. *Blunted sound
 - B. Clear sound
 - C. Bandbox sound
 - D. Metallic ringing
 - E. Tympanic sound
110. The blunted sound by percussion is typical for:
- A. *Pleural empyema
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pneumothorax
 - E. Lung cyst
111. The blunted sound by percussion is typical for:
- A. *Pleurisy
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pneumothorax
 - E. Lung cyst
112. What is revealed in pleural empyema by auscultation?
- A. *The breathing isn't auscultated
 - B. Vesicular breathing
 - C. Amphoric breathing with moist rales
 - D. Bronchial breathing with moist rales
 - E. Harsh breathing with dry rales
113. The absence of breathing sounds by auscultation is typical for:
- A. *Pleurisy
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Pneumonia
114. The absence of breathing sounds by auscultation is typical for:
- A. *Pleural empyema
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Pneumonia
115. What is revealed in wide-spread pleural empyema by X-ray?
- A. *Intensive homogeneous shadow in a basal parts with oblique upper contour
 - B. Rounded shadow with irregular contour
 - C. Rounded cavity with air-fluid level
 - D. Intensive homogeneous shadow in a basal parts with horizontal upper contour
 - E. Lung atelectasis
116. What is the most informative in differential diagnostic of pleural empyema with pleuropneumonia?
- A. *Pleural puncture
 - B. X-ray examination
 - C. Auscultation
 - D. Clinical manifestation
 - E. Sputum analysis
117. What is the most informative in the diagnostic of pleural empyema?
- A. *Pleural puncture
 - B. General blood analysis
 - C. Auscultation
 - D. Clinical manifestation

- E. Sputum analysis
118. The swelled soft tissues of supraclavicular region are typical for the:
- A. *Apical empyema
 - B. Paracostal empyema
 - C. Paramediastinal empyema
 - D. Basal empyema
 - E. Postoperative empyema
119. The restricted thoracic excursion with severe chest pain are typical for the:
- A. *Paracostal empyema
 - B. Apical empyema
 - C. Paramediastinal empyema
 - D. Basal empyema
 - E. Postoperative empyema
120. The heart pain is typical for the:
- A. *Paramediastinal empyema
 - B. Paracostal empyema
 - C. Apical empyema
 - D. Basal empyema
 - E. Postoperative empyema
121. The pain in subcostal area, which increases at respiration is typical for the:
- A. *Paramediastinal empyema
 - B. Paracostal empyema
 - C. Apical empyema
 - D. Basal empyema
 - E. Postoperative empyema
122. What is the typical method of treatment of focal empyema?
- A. *Pleural puncture
 - B. Drainage of pleural space
 - C. Thoracotomy
 - D. Pneumonectomy, bilobectomy, lobectomy
 - E. Conservative treatment
123. What is the typical method of treatment of spread empyema?
- A. *Drainage of pleural space
 - B. Pleural puncture
 - C. Thoracotomy
 - D. Pneumonectomy, bilobectomy, lobectomy
 - E. Conservative treatment
124. Where is the drainage of pleural space in spread empyema performed?
- A. *VII intercostal space, scapular line
 - B. II intercostal space, midclavicular line
 - C. II intercostal space, scapular line
 - D. IV intercostal space, anterior axillary line
 - E. VII intercostal space, midclavicular line
125. What is the typical method of treatment of chronic empyema?
- A. *Pleurectomy, decortication of lung
 - B. Drainage of pleural space
 - C. Thoracotomy
 - D. Pneumonectomy, bilobectomy, lobectomy
 - E. Conservative treatment
126. The pleurectomy is indicated for:
- A. *Chronic empyema
 - B. Complicated lung abscess
 - C. Pneumonia
 - D. Pulmonary hypertension

- E. Lung emphysema
127. The decortication of lung is indicated for:
- A. *Chronic empyema
 - B. Complicated lung abscess
 - C. Pneumonia
 - D. Pulmonary hypertension
 - E. Lung emphysema
128. What is the cause of pyopneumothorax?
- A. *Lung abscess
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
129. What is the cause of pyopneumothorax?
- A. *Lung gangrene
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
130. What is the cause of pyopneumothorax?
- A. *Suppurative cyst of lung
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
131. What is the cause of pyopneumothorax?
- A. *Abscessing pneumonia
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
132. What is the cause of pyopneumothorax?
- A. *Subphrenic abscess
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
133. What is the cause of pyopneumothorax?
- A. *Chest trauma
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
134. What is the cause of pyopneumothorax?
- A. *Bronchiectatic disease
 - B. Obstructive bronchitis
 - C. Pulmonary embolism
 - D. Bronchial asthma
 - E. Pulmonary emphysema
135. For the clinical manifestation of pyopneumothorax is typical:
- A. *Chest pain
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia

- E. Dilated cervical veins
136. For the clinical manifestation of pyopneumothorax is typical:
- A. *Dyspnea
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
137. For the clinical manifestation of pyopneumothorax is typical:
- A. *Cough with sputum
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
138. For the clinical manifestation of pyopneumothorax is typical:
- A. *Intoxication
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
139. For the clinical manifestation of pyopneumothorax is typical:
- A. *Hectic fever
 - B. Vomiting
 - C. Regurgitation
 - D. Dysphagia
 - E. Dilated cervical veins
140. What is revealed in pyopneumothorax by percussion?
- A. *Blunt sound over the exudate and bandbox sound over the region of collapsed lung
 - B. Pulmonary sound
 - C. Bandbox sound over the whole lung
 - D. Blunted sound over the whole lung
 - E. Metallic ringing
141. What is revealed in pyopneumothorax by auscultation?
- A. *The breathing isn't auscultated
 - B. Vesicular breathing
 - C. Amphoric breathing with moist rales
 - D. Bronchial breathing with moist rales
 - E. Harsh breathing with dry rales
142. What is revealed in pyopneumothorax by X-ray?
- A. *Intensive homogeneous shadow in a basal parts with horizontal upper contour
 - B. Rounded shadow with irregular contour
 - C. Rounded cavity with air-fluid level
 - D. Intensive homogeneous shadow in a basal parts with oblique upper contour
 - E. Lung atelectasis
143. What is the typical method of treatment of restricted pyopneumothorax?
- A. *Pleural puncture
 - B. Drainage of pleural space
 - C. Thoracotomy
 - D. Pneumonectomy, bilobectomy, lobectomy
 - E. Conservative treatment
144. What is the typical method of treatment of pyopneumothorax?
- A. *Drainage of pleural space
 - B. Pleural puncture
 - C. Thoracotomy
 - D. Pneumonectomy, bilobectomy, lobectomy

- E. Conservative treatment
145. Where is performed the drainage of pleural space in pyopneumothorax?
- A. *II intercostal space, midclavicular line and VII intercostal space, scapular line simultaneously
 - B. II intercostal space, midclavicular line
 - C. II intercostal space, scapular line
 - D. VII intercostal space, midclavicular line
 - E. VII intercostal space, scapular line
146. What is the early complication of chest trauma?
- A. *Pneumothorax
 - B. Posttraumatic pneumonia
 - C. Posttraumatic pleurisy
 - D. Lung abscess
 - E. Pleural empyema
147. What is the early complication of chest trauma?
- A. *Hemothorax
 - B. Lung abscess
 - C. Pleural empyema
 - D. Posttraumatic pneumonia
 - E. Posttraumatic pleurisy
148. What is the early complication of chest trauma?
- A. *Traumatic shock
 - B. Lung abscess
 - C. Pleural empyema
 - D. Posttraumatic pneumonia
 - E. Posttraumatic pleurisy
149. What is the late complication of chest trauma?
- A. *Pleural empyema
 - B. Pneumothorax
 - C. Hemothorax
 - D. Mediastinal emphysema
 - E. Traumatic shock, asphyxia
150. What is the late complication of chest trauma?
- A. *Posttraumatic pneumonia
 - B. Pneumothorax
 - C. Hemothorax
 - D. Mediastinal emphysema
 - E. Traumatic shock, asphyxia
151. What is the chief clinical manifestation of noncomplicated rib fracture?
- A. *Pain
 - B. Dyspnea
 - C. Hemoptysis
 - D. Shock
 - E. Vomiting
152. What is revealed in noncomplicated rib fracture by auscultation?
- A. *Diminished vesicular breathing
 - B. The breathing isn't auscultated
 - C. Amphoric breathing with moist rales
 - D. Bronchial breathing with moist rales
 - E. Harsh breathing with dry rales
153. What kind of X-ray picture is typical for noncomplicated rib fracture?
- A. *Break in continuity of bone fragments of ribs
 - B. Exudate in pleural space
 - C. Collapse of the lung

- D. Lung atelectasis
 - E. Heterogeneous lung shadow with destruction
154. What is the chief clinical manifestation of floating rib fracture?
- A. *Shock
 - B. Pain
 - C. Dyspnea
 - D. Hemoptysis
 - E. Vomiting
155. What is the chief objective sign of floating rib fracture?
- A. *Paradoxical respiratory movements of chest
 - B. Crepitation of ribs
 - C. Hematoma of the chest wall
 - D. Hemoptysis
 - E. Subcutaneous emphysema
156. What is revealed in floating rib fracture by auscultation?
- A. *Diminished vesicular breathing
 - B. The breathing isn't auscultated
 - C. Amphoric breathing with moist rales
 - D. Bronchial breathing with moist rales
 - E. Harsh breathing with dry rales
157. What kind of X-ray picture is typical for floating rib fracture?
- A. *Multiple rib fracture with deformity of the chest
 - B. Lung emphysema
 - C. Spheric shadow of the lung
 - D. Lung atelectasis
 - E. Heterogeneous lung shadow with destruction
158. What is the typical treatment of noncomplicated rib fracture?
- A. *Novocaine block
 - B. External fixation of ribs
 - C. Intramedullary costal osteosynthesis;
 - D. Mechanical ventilation with positive end-expiratory pressure
 - E. Thoracotomy
159. What is the first aid of floating rib fracture?
- A. *Fixation of floating segment
 - B. Analgesics
 - C. Spasmolytics
 - D. Hemostatic drugs
 - E. Antibiotics
160. What is the main treatment of floating rib fracture?
- A. *Skeletal extraction
 - B. Pleural puncture
 - C. Pneumonectomy
 - D. Resection of lung
 - E. Decortication of lung
161. What is the main treatment of floating rib fracture?
- A. *Intramedullary costal osteosynthesis
 - B. Pleural puncture
 - C. Pneumonectomy
 - D. Resection of lung
 - E. Decortication of lung
162. What is the main treatment of floating rib fracture?
- A. *Mechanical ventilation with positive end-expiratory pressure
 - B. Pleural puncture
 - C. Pneumonectomy

- D. Resection of lung
 - E. Decortication of lung
163. What type of Novocaine block is used for the treatment of floating rib fracture?
- A. *Vagosympathetic block
 - B. Paraneprhal block
 - C. Spinal block
 - D. Epidural anesthesia
 - E. Lung root dlock
164. What type of Novocaine block is used for the treatment of floating rib fracture?
- A. *Alcohol - novocaine block of the site of fracture
 - B. Paraneprhal block
 - C. Spinal block
 - D. Epidural anesthesia
 - E. Lung root dlock
165. What type of Novocaine block is used for the treatment of floating rib fracture?
- A. *Paravertebral block
 - B. Paraneprhal block
 - C. Spinal block
 - D. Epidural anesthesia
 - E. Lung root dlock
166. Partial pneumothorax means:
- A. *Collapse of lung to 1/3 of its volume
 - B. No collapse of lung
 - C. Collapse of lung to 2/3 of its volume
 - D. Collapse of lung more than 2/3 of its volume
 - E. Total collapse of lung
167. Subtotal pneumothorax means:
- A. *Collapse of lung to 2/3 of its volume
 - B. No collapse of lung
 - C. Collapse of lung to 1/3 of its volume
 - D. Collapse of lung more than 2/3 of its volume
 - E. Total collapse of lung
168. Total pneumothorax means:
- A. *Collapse of lung more than 2/3 of its volume
 - B. No collapse of lung
 - C. Collapse of lung to 1/3 of its volume
 - D. Collapse of lung to 2/3 of its volume
 - E. Collapse of lung to 1/2 of its volume
169. The collapse of lung in pneumothorax from 1/3 to 2/3 of its volume is called:
- A. *Subtotal pneumothorax
 - B. Partial pneumothorax
 - C. Total pneumothorax
 - D. Bilateral pneumothorax
 - E. Paradoxal pneumothorax
170. The collapse of lung in pneumothorax less than 1/3 of its volume is called:
- A. *Partial pneumothorax
 - B. Subtotal pneumothorax
 - C. Total pneumothorax
 - D. Bilateral pneumothorax
 - E. Paradoxal pneumothorax
171. The collapse of lung in pneumothorax more than 2/3 of its volume is called:
- A. *Total pneumothorax
 - B. Partial pneumothorax
 - C. Subtotal pneumothorax

- D. Bilateral pneumothorax
 - E. Paradoxal pneumothorax
172. What is the most dangerous pneumothorax?
- A. *Valvular
 - B. Subtotal closed
 - C. Total closed
 - D. Subtotal open
 - E. Total open
173. What is the chief clinical manifestation of pneumothorax?
- A. *Dyspnea
 - B. Vomiting
 - C. Hemoptysis
 - D. Shock
 - E. Heart failure
174. What is the chief manifestation of valvular pneumothorax?
- A. *Shock
 - B. Vomiting
 - C. Melena
 - D. Hemoptysis
 - E. Heart failure
175. What is the chief clinical manifestation of pneumothorax?
- A. *Chest pain
 - B. Vomiting
 - C. Hemoptysis
 - D. Melena
 - E. Heart failure
176. What is revealed in pneumothorax by percussion?
- A. *Bandbox sound over the whole lung
 - B. Pulmonary sound
 - C. Blunted sound over the whole lung
 - D. Metallic ringing
 - E. Blunt sound over the region of collapsed lung
177. The bandbox sound by percussion is typical for:
- A. *Pneumothorax
 - B. Lung gangrene
 - C. Pneumonia
 - D. Pleural empyema
 - E. Lung abscess
178. What is revealed in pneumothorax by auscultation?
- A. *The breathing isn't auscultated
 - B. Vesicular breathing
 - C. Amphoric breathing with moist rales
 - D. Bronchial breathing with moist rales
 - E. Harsh breathing with dry rales
179. The absence of breathing sound by auscultation is typical for:
- A. *Pneumothorax
 - B. Chronic bronchitis
 - C. Pneumonia
 - D. Lung emphyzema
 - E. Lung abscess
180. What is revealed in pneumothorax by X-ray?
- A. *Lung collapse
 - B. Lung atelectasis
 - C. Rounded cavity with air-fluid level

- D. Intensive homogeneous shadow in a basal parts with horizontal upper contour
E. Intensive homogeneous shadow in a basal parts with oblique upper contour
181. The lung collapse by X-ray is typical for:
A. *Pneumothorax
B. Chronic bronchitis
C. Pneumonia
D. Lung emphyzema
E. Lung abscess
182. The lung collapse by X-ray is typical for:
A. *Pneumothorax
B. Lung cancer
C. Pneumonia
D. Tuberculosis
E. Lung empyema
183. What is the treatment of partial pneumothorax?
A. *Pleural puncture
B. Pleural drainage
C. Thoracotomy
D. Pneumonectomy, bilobectomy, lobectomy
E. Conservative treatment
184. What is the treatment of subtotal and total pneumothorax?
A. *Pleural drainage
B. Pleural puncture
C. Thoracotomy
D. Pneumonectomy, bilobectomy, lobectomy
E. Conservative treatment
185. The pleural drainage is used for the treatment of:
A. *Pneumothorax
B. Chronic bronchitis
C. Pneumonia
D. Lung emphyzema
E. Lung abscess
186. The pleural drainage is used for the treatment of:
A. *Pyopneumothorax
B. Chronic bronchitis
C. Pneumonia
D. Lung emphyzema
E. Lung abscess
187. The pleural drainage is used for the treatment of:
A. *Pleural empyema
B. Chronic bronchitis
C. Pneumonia
D. Lung emphyzema
E. Lung abscess
188. What is the first aid in closed pneumothorax?
A. *It doesn't require first aid measures
B. Pleural drainage
C. Compression bandage with closure of the wound
D. Artificial respiration
E. Intubation
189. What is the first aid in open pneumothorax?
A. *Compression bandage with closure of the wound
B. It doesn't require first aid measures
C. Pleural drainage

- D. Artificial respiration
 - E. Intubation
190. What is the first aid in valvular pneumothorax?
- A. *Pleural drainage (to transform into open)
 - B. It doesn't require first aid measures
 - C. Compression bandage with closure of the wound
 - D. Artificial respiration
 - E. Intubation
191. Where the drainage of pleural space in pneumothorax is performed?
- A. *II intercostal space, midclavicular line
 - B. II intercostal space, scapular line
 - C. IV intercostal space, anterior axillary line
 - D. VII intercostal space, midclavicular line
 - E. VII intercostal space, scapular line
192. The pleural drainage in II intercostal space along midclavicular line is used for the treatment of:
- A. *Pneumothorax
 - B. Pleural empyema
 - C. Pneumonia
 - D. Pleurisy
 - E. Hemothorax
193. The pleural drainage in VII intercostal space along scapular line is used for the treatment of:
- A. *Pleural empyema
 - B. Pneumothorax
 - C. Chronic bronchitis
 - D. Pneumonia
 - E. Lung emphysema
194. What is the indication for operative treatment of pneumothorax?
- A. *Valvular
 - B. Subtotal closed
 - C. Total closed
 - D. Subtotal open
 - E. Total open
195. The small hemothorax means:
- A. *Loss less 10 % of volume of circulating blood
 - B. Loss of 10-20 % of volume of circulating blood
 - C. Loss of 20-40 % of volume of circulating blood
 - D. Loss of 40-60 % of volume of circulating blood
 - E. Loss more than 60 % of volume of circulating blood
196. The moderate hemothorax means:
- A. *Loss of 10-20 % of volume of circulating blood
 - B. Loss less 10 % of volume of circulating blood
 - C. Loss of 20-40 % of volume of circulating blood
 - D. Loss of 40-60 % of volume of circulating blood
 - E. Loss more than 60 % of volume of circulating blood
197. The great hemothorax means:
- A. *Loss of 20-40 % of volume of circulating blood
 - B. Loss less 10 % of volume of circulating blood
 - C. Loss of 10-20 % of volume of circulating blood
 - D. Loss of 40-60 % of volume of circulating blood
 - E. Loss more than 60 % of volume of circulating blood
198. The total hemothorax means:
- A. *Loss more than 40 % of volume of circulating blood
 - B. Loss of 20-30 % of volume of circulating blood

- C. Loss less 10 % of volume of circulating blood
 - D. Loss of 10-20 % of volume of circulating blood
 - E. Loss of 30-40 % of volume of circulating blood
199. The bloodloss to 10 % of volume of circulating blood relates to:
- A. *Small hemothorax
 - B. Moderate hemothorax
 - C. Great hemothorax
 - D. Total hemothorax
 - E. Coagulated hemothorax
200. The bloodloss of 10-20 % of volume of circulating blood relates to:
- A. *Moderate hemothorax
 - B. Small hemothorax
 - C. Great hemothorax
 - D. Total hemothorax
 - E. Coagulated hemothorax
201. The bloodloss of 20-40 % of volume of circulating blood relates to:
- A. *Great hemothorax
 - B. Moderate hemothorax
 - C. Small hemothorax
 - D. Total hemothorax
 - E. Coagulated hemothorax
202. The bloodloss more than 40 % of volume of circulating blood relates to:
- A. *Total hemothorax
 - B. Moderate hemothorax
 - C. Small hemothorax
 - D. Great hemothorax
 - E. Coagulated hemothorax
203. What is the chief clinical manifestation of hemothorax?
- A. *Clinic of internal bleeding
 - B. Pain
 - C. Vomiting
 - D. Hemoptysis
 - E. Intoxication
204. What is revealed in hemothorax by percussion?
- A. *Blunted sound
 - B. Clear sound
 - C. Bandbox sound
 - D. Tympanic sound
 - E. Metallic ringing
205. The blunted sound by percussion is typical for:
- A. *Hemothorax
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pneumothorax
 - E. Lung cyst
206. The absence of breathing sounds by auscultation is typical for:
- A. *Hemothorax
 - B. Bronchitis
 - C. Lung emphysema
 - D. Lung abscess
 - E. Pneumonia
207. What is revealed in hemothorax by auscultation?
- A. *The breathing isn't auscultated
 - B. Vesicular breathing

- C. Amphoric breathing with moist rales
 - D. Bronchial breathing with moist rales
 - E. Harsh breathing with dry rales
208. What is revealed in hemothorax by X-ray?
- A. *Intensive homogeneous shadow in a basal parts with oblique upper contour
 - B. Lung atelectasis
 - C. Rounded shadow with irregular contour
 - D. Rounded cavity with air-fluid level
 - E. Intensive homogeneous shadow in a basal parts with horizontal upper contour
209. Where is the level of the X-ray shadow in small hemothorax?
- A. *Shadow observed only in the region of sinus
 - B. Up to scapular angle
 - C. Up to III rib
 - D. Complete shadow of a pleural space
 - E. The shadow is absent
210. Where is the level of the X-ray shadow in moderate hemothorax?
- A. *Up to scapular angle
 - B. Shadow observed only in the region of sinus
 - C. Up to III rib
 - D. Complete shadow of a pleural space
 - E. The shadow is absent
211. Where is the level of the X-ray shadow in great hemothorax?
- A. *Up to III rib
 - B. Shadow observed only in the region of sinus
 - C. Up to scapular angle
 - D. Complete shadow of a pleural space
 - E. The shadow is absent
212. Where is the level of the X-ray shadow in total hemothorax?
- A. *Complete shadow of a pleural space
 - B. Up to scapular angle
 - C. Shadow observed only in the region of sinus
 - D. Up to III rib
 - E. The shadow is absent
213. The X-ray shadow in hemothorax observed only in the region of sinus relates to:
- A. *Small hemothorax
 - B. Moderate hemothorax
 - C. Great hemothorax
 - D. Total hemothorax
 - E. Coagulated hemothorax
214. The X-ray shadow in hemothorax up to scapular angle relates to:
- A. *Moderate hemothorax
 - B. Small hemothorax
 - C. Great hemothorax
 - D. Total hemothorax
 - E. Coagulated hemothorax
215. The X-ray shadow in hemothorax up to III rib relates to:
- A. *Great hemothorax
 - B. Small hemothorax
 - C. Moderate hemothorax
 - D. Total hemothorax
 - E. Coagulated hemothorax
216. The complete X-ray shadow of a pleural space in hemothorax relates to:
- A. *Total hemothorax
 - B. Small hemothorax

- C. Moderate hemothorax
 - D. Great hemothorax
 - E. Coagulated hemothorax
217. What method is the most informative in differential diagnostic of hemothorax with pleurisy?
- A. *Pleural puncture
 - B. Clinical manifestation
 - C. Sputum analysis
 - D. Auscultation
 - E. X-ray examination
218. What method is the most informative in the diagnostic of hemothorax?
- A. *Pleural puncture
 - B. General blood analysis
 - C. Sputum analysis
 - D. Auscultation
 - E. X-ray examination
219. What is the sign of continuity of pleural bleeding?
- A. *The property of pleural blood to form the clot
 - B. Data of general blood analysis
 - C. Data of biochemical blood analysis
 - D. Data of coagulogram
 - E. Clinical manifestation
220. What test is used to determine the continuity of pleural bleeding?
- A. *Revilour-Gregar's test
 - B. Troyanov-Trendelenburg's test
 - C. Talman's test
 - D. Mayo-Pratt's test
 - E. Delbe-Pertess test (marching test)
221. The Revilour-Gregar's test is used in the diagnostics of:
- A. *Pleural bleeding
 - B. Lung abscess
 - C. Pleural empyema
 - D. Pneumothorax
 - E. Deep vein thrombosis
222. The property of pleural blood to form the clot is called:
- A. *Revilour-Gregar's test
 - B. Troyanov-Trendelenburg's test
 - C. Talman's test
 - D. Mayo-Pratt's test
 - E. Delbe-Pertess test (marching test)
223. What sign shows that the pleural bleeding is stopped?
- A. *The pleural blood doesn't form the clot
 - B. Data of general blood analysis
 - C. Data of biochemical blood analysis
 - D. Data of coagulogram
 - E. Clinical manifestation
224. What does the clotted hemothorax result in?
- A. *Pleural empyema
 - B. Dyspnea
 - C. Hemoptysis
 - D. Obliteration of pleural space
 - E. Cardiac tamponade
225. What kind of hemothorax is treated by pleural aspiration?
- A. *Small
 - B. Great

- C. Total
 - D. Clotted
 - E. Continuing hemothorax
226. What medicine used for the treatment of hemothorax?
- A. *Aminocapronic acid
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
227. What medicine used for the treatment of hemothorax?
- A. *Calcium chloridi
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
228. What medicine used for the treatment of hemothorax?
- A. *Dicinon
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
229. What medicine used for the treatment of hemothorax?
- A. *Vitamin K
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
230. What pathology is the indication for operative treatment?
- A. *Great and total hemothorax
 - B. Noncomplicated rib fracture
 - C. Closed partial pneumothorax
 - D. Closed total pneumothorax
 - E. Subcutaneous emphysema
231. What pathology is the indication for operative treatment?
- A. *Clotted hemothorax
 - B. Noncomplicated rib fracture
 - C. Subcutaneous emphysema
 - D. Closed partial pneumothorax
 - E. Closed total pneumothorax
232. What pathology is the indication for operative treatment?
- A. *Continuing hemothorax
 - B. Closed partial pneumothorax
 - C. Closed total pneumothorax
 - D. Noncomplicated rib fracture
 - E. Subcutaneous emphysema
233. What is the main cause of mediastinal emphysema?
- A. *Disruptions of trachea, bronchi
 - B. Rib fracture
 - C. Pneumothorax
 - D. Hemothorax
 - E. Mediastinal tumours
234. What does the mediastinal emphysema result in?
- A. *Cardiac tamponade
 - B. Hemoptysis

- C. Pleural empyema
 - D. Pneumothorax
 - E. Lung atelectasis
235. What is the treatment of mediastinal emphysema?
- A. *Drainage of anterior mediastinum
 - B. Conservative treatment
 - C. Drainage of pleural cavity
 - D. Novocaine block
 - E. Pericardial puncture
236. Where is located the first anatomical narrowing of esophagus?
- A. *The site of pharyngoesophageal junction
 - B. The site of crossing with left bronchus
 - C. The site of crossing with aorta
 - D. The site of passing through diaphragm
 - E. The site of cardia
237. Where is located the second anatomical narrowing of esophagus?
- A. *The site of crossing with left bronchus
 - B. The site of pharyngoesophageal junction
 - C. The site of crossing with aorta
 - D. The site of passing through diaphragm
 - E. The site of cardia
238. Where is located the third anatomical narrowing of esophagus?
- A. *The site of passing through diaphragm
 - B. The site of pharyngoesophageal junction
 - C. The site of crossing with aorta
 - D. The site of crossing with left bronchus
 - E. The site of cardia
239. Where is located the first physiological narrowing of esophagus?
- A. *The site of crossing with aorta
 - B. The site of pharyngoesophageal junction
 - C. The site of crossing with left bronchus
 - D. The site of passing through diaphragm
 - E. The site of cardia
240. Where is located the second physiological narrowing of esophagus?
- A. *The site of cardia
 - B. The site of pharyngoesophageal junction
 - C. The site of crossing with aorta
 - D. The site of crossing with left bronchus
 - E. The site of passing through diaphragm
241. The site of pharyngoesophageal junction is:
- A. *The first anatomical narrowing of esophagus
 - B. The second anatomical narrowing of esophagus
 - C. The third anatomical narrowing of esophagus
 - D. The first physiological narrowing of esophagus
 - E. The second physiological narrowing of esophagus
242. The site of crossing with left bronchus is:
- A. *The second anatomical narrowing of esophagus
 - B. The first anatomical narrowing of esophagus
 - C. The third anatomical narrowing of esophagus
 - D. The first physiological narrowing of esophagus
 - E. The second physiological narrowing of esophagus
243. The site of passing through diaphragm is:
- A. *The third anatomical narrowing of esophagus
 - B. The first anatomical narrowing of esophagus

- C. The second anatomical narrowing of esophagus
 - D. The first physiological narrowing of esophagus
 - E. The second physiological narrowing of esophagus
244. The site of crossing with aorta is:
- A. *The first physiological narrowing of esophagus
 - B. The first anatomical narrowing of esophagus
 - C. The second anatomical narrowing of esophagus
 - D. The third anatomical narrowing of esophagus
 - E. The second physiological narrowing of esophagus
245. The site of cardia is:
- A. *The second physiological narrowing of esophagus
 - B. The first anatomical narrowing of esophagus
 - C. The second anatomical narrowing of esophagus
 - D. The third anatomical narrowing of esophagus
 - E. The first physiological narrowing of esophagus
246. What is the mechanism of formation of pulsion diverticula?
- A. *Herniation of the esophageal wall proximal to anatomical narrowing
 - B. Inflammatory changes of paraesophageal tissues
 - C. Dilatation of esophagus caused by achalasia
 - D. Changes of esophagus caused by chemical burns
 - E. Changes of esophagus caused by reflux esophagitis
247. What is the mechanism of formation of traction diverticula?
- A. *Inflammatory changes of paraesophageal tissues
 - B. Herniation of the esophageal wall proximal to anatomical narrowing
 - C. Dilatation of esophagus caused by achalasia
 - D. Changes of esophagus caused by chemical burns
 - E. Changes of esophagus caused by reflux esophagitis
248. What sign is not typical for Zenker's diverticula?
- A. *Dyspnea
 - B. Salivation
 - C. Cervical dysphagia
 - D. Difficult swallowing
 - E. Cough
249. Zenker's diverticulum is:
- A. *Pharyngoesophageal diverticulum
 - B. Bifurcational diverticulum
 - C. Epiphrenic diverticulum
 - D. Paraaortal diverticulum
 - E. Multiple diverticula
250. What is the main objective manifestation of Zenker's diverticula?
- A. *Compressible mass on the left side of the neck
 - B. Signs of achalasia
 - C. Cyanosis of the upper part of body
 - D. Esophago-bronchial fistula with aspiration pneumonia
 - E. Lung atelectasis
251. What can bifurcational diverticula result in?
- A. *Esophago-bronchial fistula with aspiration pneumonia
 - B. Signs of achalasia
 - C. Cyanosis of the upper part of body
 - D. Compressible mass on the left side of the neck
 - E. Coarctation of aorta
252. What is the main objective manifestation of epiphrenic diverticula?
- A. *Signs of achalasia
 - B. Cyanosis of the upper part of body

- C. Compressible mass on the left side of the neck
 - D. Esophago-bronchial fistula with aspiration pneumonia
 - E. Lung atelectasis
253. For the clinical manifestation of esophageal diverticulum is typical:
- A. *Salivation
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
254. For the clinical manifestation of esophageal diverticulum is typical:
- A. *Cervical dysphagia
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
255. For the clinical manifestation of esophageal diverticulum is typical:
- A. *Difficult swallowing
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
256. For the clinical manifestation of esophageal diverticulum is typical:
- A. *Compressible mass in the neck
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
257. For the clinical manifestation of esophageal diverticulum is typical:
- A. *Gurgling sound while eating
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
258. For the clinical manifestation of esophageal diverticulum is typical:
- A. *Foul-smell from the mouth
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
259. For the clinical manifestation of esophageal diverticulum is typical:
- A. *The sign "of a wet pillow"
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
260. What is the main method of diagnostic of esophageal diverticula?
- A. *X-ray examination with barium swallow
 - B. Pleural puncture
 - C. Ultrasound examination
 - D. Plain X-ray examination of the chest
 - E. Irrigoscopy
261. What is the main method of diagnostic of esophageal diverticula?
- A. *Esophagogastroduodenoscopy
 - B. Pleural puncture

- C. Ultrasound examination
 - D. Plain X-ray examination of the chest
 - E. Irrigoscopy
262. What complication is typical for esophageal diverticula?
- A. *Bleeding
 - B. Obstructive jaundice
 - C. Intestinal obstruction
 - D. Myocardial infarction
 - E. Lung atelectasis
263. What complication is typical for esophageal diverticula?
- A. *Perforation into pleural space
 - B. Myocardial infarction
 - C. Pancreatitis
 - D. Obstructive jaundice
 - E. Intestinal obstruction
264. What complication is typical for esophageal diverticula?
- A. *Diverticulitis
 - B. Obstructive jaundice
 - C. Intestinal obstruction
 - D. Myocardial infarction
 - E. Lung atelectasis
265. What complication is typical for esophageal diverticula?
- A. *Malignancy
 - B. Obstructive jaundice
 - C. Intestinal obstruction
 - D. Myocardial infarction
 - E. Lung atelectasis
266. What disease should be the diverticulitis differentiated from?
- A. *Angina pectoris
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
267. What is the typical treatment of complicated Zenker's diverticula?
- A. *Surgical treatment
 - B. Spasmolytics
 - C. Analgetics
 - D. Nonsteroid antiinflammatory drugs
 - E. Antibiotics
268. What is the indication for conservative treatment of bifurcational diverticula?
- A. *Asymptomatic course
 - B. Frequently recurrent diverticulites
 - C. Perforation
 - D. Esophago-bronchial fistula
 - E. Suspicion on malignancy
269. What operation is performed in esophageal diverticula?
- A. *Resection of diverticulum
 - B. Esophagomyotomy
 - C. Esophagogastric anastomosis
 - D. Extirpation of esophagus
 - E. Esophageal plastics by intestine
270. What surgical access should be applied for Zenker's diverticula?
- A. *Cervical access along the anterior border of the sternocleidomastoid muscle
 - B. Left-sided anterolateral thoracotomy in IV intercostal space

- C. Right-sided posterolateral thoracotomy in IV intercostal space
 - D. Left-sided posterolateral thoracotomy in VII intercostal space
 - E. Right-sided anterolateral thoracotomy in VII intercostal space
271. What surgical access should be applied for bifurcational diverticula?
- A. *Right-sided posterolateral thoracotomy in IV intercostal space
 - B. Cervical access along the anterior border of the sternocleidomastoid muscle
 - C. Left-sided anterolateral thoracotomy in IV intercostal space
 - D. Left-sided posterolateral thoracotomy in VII intercostal space
 - E. Right-sided anterolateral thoracotomy in VII intercostal space
272. What surgical access should be applied for epiphrenal diverticula?
- A. *Left-sided posterolateral thoracotomy in VII intercostal space
 - B. Cervical access along the anterior border of the sternocleidomastoid muscle
 - C. Left-sided anterolateral thoracotomy in IV intercostal space
 - D. Right-sided posterolateral thoracotomy in IV intercostal space
 - E. Right-sided anterolateral thoracotomy in VII intercostal space
273. What is the characteristic feature of achalasia of the cardia?
- A. *Failure of the lower esophageal sphincter to relax
 - B. Spasm of the lower esophageal sphincter
 - C. Cicatrical changes after the burn
 - D. Anorexia
 - E. Esophageal gaping
274. The failure of the lower esophageal sphincter to relax is called:
- A. *Achalasia
 - B. Chaliasia
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
275. What is the cause of achalasia?
- A. *Disturbance of innervation of esophagus
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatrical changes after the burn of esophagus
276. What is the cause of achalasia?
- A. *Psycho-emotional trauma
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatrical changes after the burn of esophagus
277. What is the cause of achalasia?
- A. *Influence of vegetotrophic substances on muscular fibers
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatrical changes after the burn of esophagus
278. Who mostly suffer from achalasia?
- A. *Young women
 - B. Young men
 - C. Old men
 - D. Old women
 - E. Both old men and women
279. What is the characteristic feature of the I stage of achalasia?
- A. *Functional spasm without esophageal dilation
 - B. Asymptomatic

- C. Constant spasm with a moderate esophageal dilation and maintained peristalsis
 - D. Cicatricial changes with expressed esophageal dilation, the peristalsis is absent
 - E. Considerable esophageal dilation with S-shaped elongation.
280. What is the characteristic feature of the II stage of achalasia?
- A. *Constant spasm with a moderate esophageal dilation and maintained peristalsis
 - B. Asymptomatic
 - C. Functional spasm without esophageal dilation
 - D. Cicatricial changes with expressed esophageal dilation, the peristalsis is absent
 - E. Considerable esophageal dilation with S-shaped elongation.
281. What is the characteristic feature of the III stage of achalasia?
- A. *Cicatricial changes with expressed esophageal dilation, the peristalsis is absent
 - B. Asymptomatic
 - C. Functional spasm without esophageal dilation
 - D. Constant spasm with a moderate esophageal dilation and maintained peristalsis
 - E. Considerable esophageal dilation with S-shaped elongation.
282. What is the characteristic feature of the IV stage of achalasia?
- A. *Considerable esophageal dilation with S-shaped elongation.
 - B. Cicatricial changes with expressed esophageal dilation, the peristalsis is absent
 - C. Asymptomatic
 - D. Functional spasm without esophageal dilation
 - E. Constant spasm with a moderate esophageal dilation and maintained peristalsis
283. Functional spasm without esophageal dilation relates to such stage of achalasia:
- A. *I
 - B. II
 - C. III
 - D. IV
 - E. V
284. Constant spasm with a moderate esophageal dilation and maintained peristalsis relates to such stage of achalasia:
- A. *II
 - B. I
 - C. III
 - D. IV
 - E. V
285. Cicatricial changes with expressed esophageal dilation with absent peristalsis relates to such stage of achalasia:
- A. *III
 - B. I
 - C. II
 - D. IV
 - E. V
286. Considerable esophageal dilation with S-shaped elongation relates to such stage of achalasia:
- A. *IV
 - B. I
 - C. II
 - D. III
 - E. V
287. For the clinical manifestation of esophageal achalasia is typical:
- A. *Dysphagia
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"

288. For the clinical manifestation of esophageal achalasia is typical:
- A. *Regurgitation
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
289. For the clinical manifestation of esophageal achalasia is typical:
- A. *Splashing sounds and gurgling
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
290. For the clinical manifestation of esophageal achalasia is typical:
- A. *Nocturnal cough
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
291. For the clinical manifestation of esophageal achalasia is typical:
- A. *Pain and sense of tightness in the chest
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
292. For the clinical manifestation of esophageal achalasia is typical:
- A. *Loss of weight
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
293. What does dysphagia mean?
- A. *Disturbances of swallowing
 - B. Pain behind breastbone
 - C. Absence of appetite
 - D. Esophageal vomiting
 - E. Vomiting with blood
294. What is the I stage of dysphagia?
- A. *Disturbances of solid food passage
 - B. Asymptomatic
 - C. Disturbances of semisolid food passage
 - D. Disturbances of liquid food passage
 - E. No passage of food
295. What is the II stage of dysphagia?
- A. *Disturbances of semisolid food passage
 - B. Asymptomatic
 - C. Disturbances of solid food passage
 - D. Disturbances of liquid food passage
 - E. No passage of food
296. What is the III stage of dysphagia?
- A. *Disturbances of liquid food passage
 - B. Asymptomatic
 - C. Disturbances of solid food passage
 - D. Disturbances of semisolid food passage
 - E. No passage of food

297. What is the IV stage of dysphagia?
A. *No passage of food
B. Asymptomatic
C. Disturbances of solid food passage
D. Disturbances of semisolid food passage
E. Disturbances of liquid food passage
298. Disturbances of solid food passage relates to such stage of dysphagia:
A. *I
B. II
C. III
D. IV
E. V
299. Disturbances of semisolid food passage relates to such stage of dysphagia:
A. *II
B. I
C. III
D. IV
E. V
300. Disturbances of liquid food passage relates to such stage of dysphagia:
A. *III
B. II
C. I
D. IV
E. V
301. No passage of any food relates to such stage of dysphagia:
A. *IV
B. III
C. II
D. I
E. V
302. What is the roentgenological sign of achalasia?
A. *"Rat tail" sign
B. Filling defects
C. "Niche" sign
D. "Bell" sign
E. Blunt His angle
303. What is the roentgenological sign of esophageal cancer?
A. *Filling defects
B. "Rat tail" sign
C. "Bird-beak" sign
D. "Bell" sign
E. Blunt His angle
304. "Rat tail" is the X-ray sign of:
A. *Achalasia
B. Esophageal cancer
C. Esophageal diverticulum
D. Pylorostenosis
E. Intestinal obstruction
305. "Bird-beak" is the X-ray sign of:
A. *Achalasia
B. Esophageal cancer
C. Esophageal diverticulum
D. Pylorostenosis
E. Intestinal obstruction

306. What is the main method of diagnostic of esophageal achalasia?
A. *X-ray examination with barium swallow
B. Pleural puncture
C. Ultrasound examination
D. Plain X-ray examination of the chest
E. Irrigoscopy
307. What is the main method of diagnostic of esophageal achalasia?
A. *Esophagogastroduodenoscopy
B. Pleural puncture
C. Ultrasound examination
D. Plain X-ray examination of the chest
E. Irrigoscopy
308. What is the main treatment of the I stage of achalasia?
A. *Diet, conservative treatment
B. Cardiodilatation
C. Esophagomyotomy (Heller's operation)
D. Esophagogastroanastomosis (Hellerovskiy's operation)
E. Esophageal plastics by intestine
309. What is the main treatment of the II stage of achalasia?
A. *Cardiodilatation
B. Diet, conservative treatment
C. Esophagomyotomy (Heller's operation)
D. Esophagogastroanastomosis (Hellerovskiy's operation)
E. Esophageal plastics by intestine
310. What is the main treatment of the III stage of achalasia?
A. *Esophagomyotomy (Heller's operation)
B. Diet, conservative treatment
C. Cardiodilatation
D. Esophagogastroanastomosis (Hellerovskiy's operation)
E. Esophageal plastics by intestine
311. What is the main treatment of the IV stage of achalasia?
A. *Esophagogastroanastomosis (Hellerovskiy's operation)
B. Diet, conservative treatment
C. Cardiodilatation
D. Esophagomyotomy (Heller's operation)
E. Esophageal plastics by intestine
312. Diet, conservative treatment is indicated for such stage of achalasia:
A. *I
B. II
C. III
D. IV
E. V
313. Cardiodilatation is indicated for such stage of achalasia:
A. *II
B. I
C. III
D. IV
E. V
314. Esophagomyotomy is indicated for such stage of achalasia:
A. *III
B. II
C. I
D. IV
E. V

315. Heller's operation is indicated for such stage of achalasia:
- A. *III
 - B. II
 - C. I
 - D. IV
 - E. V
316. Esophagogastronomy is indicated for such stage of achalasia:
- A. *IV
 - B. III
 - C. II
 - D. I
 - E. V
317. Hellerovskiy's operation is indicated for such stage of achalasia:
- A. *IV
 - B. III
 - C. II
 - D. I
 - E. V
318. Hellerovskiy's operation is used for the treatment of:
- A. *Esophageal achalasia
 - B. Esophageal ulcer
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
319. Esophagogastronomy is used for the treatment of:
- A. *Esophageal achalasia
 - B. Esophageal ulcer
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
320. Heller's operation is used for the treatment of:
- A. *Esophageal achalasia
 - B. Esophageal cancer
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
321. Esophagogastronomy is used for the treatment of:
- A. *Esophageal achalasia
 - B. Esophageal ulcer
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
322. Esophagomyotomy is used for the treatment of:
- A. *Esophageal achalasia
 - B. Esophageal cancer
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
323. Cardiomyotomy is used for the treatment of:
- A. *Esophageal achalasia
 - B. Esophageal cancer
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction

324. Heller's operation is:
- A. *Esophagomyotomy
 - B. Resection of the stomach
 - C. Cardiodilatation
 - D. Esophagogastranastomosis
 - E. Esophageal plastics by intestine
325. Helerovsky's operation is:
- A. *Esophagogastranastomosis
 - B. Esophagomyotomy
 - C. Resection of the stomach
 - D. Cardiodilatation
 - E. Esophageal plastics by intestine
326. Esophagogastranastomosis is:
- A. *Helerovsky's operation
 - B. Heller's operation
 - C. Bilroth's operation
 - D. Cocher's operation
 - E. Lerishe's operation
327. Esophagomyotomy is:
- A. *Heller's operation
 - B. Helerovsky's operation
 - C. Bilroth's operation
 - D. Cocher's operation
 - E. Lerishe's operation
328. What is the most often cause of cicatrical esophageal stricture?
- A. *Chemical burns
 - B. Thermal burns
 - C. Radial burns
 - D. Esophagitis
 - E. Peptic ulcers
329. What is the I stage of morphological changes of esophageal burns?
- A. *Stage of acute esophagitis
 - B. Asymptomatic
 - C. Stage of chronic esophagitis
 - D. Stage of cicatrical stricture of esophagus
 - E. Stage of late complications
330. What is the II stage of morphological changes of esophageal burns?
- A. *Stage of chronic esophagitis
 - B. Asymptomatic
 - C. Stage of acute esophagitis
 - D. Stage of cicatrical stricture of esophagus
 - E. Stage of late complications
331. What is the III stage of morphological changes of esophageal burns?
- A. *Stage of cicatrical stricture of esophagus
 - B. Asymptomatic
 - C. Stage of acute esophagitis
 - D. Stage of chronic esophagitis
 - E. Stage of late complications
332. What is the IV stage of morphological changes of esophageal burns?
- A. *Stage of late complications
 - B. Asymptomatic
 - C. Stage of acute esophagitis
 - D. Stage of chronic esophagitis
 - E. Stage of cicatrical stricture of esophagus

333. What is the I degree of esophageal burns?
- A. *Superficial burn with the damage of epithelial layer of esophagus;
 - B. The burn with the damage of entire mucosa of esophagus;
 - C. The burn damage of all layers of esophagus;
 - D. The spread of postburn necrosis on paraesophageal tissue and adjacent organs.
 - E. Asymptomatic
334. What is the II degree of esophageal burns?
- A. *The burn with the damage of entire mucosa of esophagus;
 - B. Superficial burn with the damage of epithelial layer of esophagus;
 - C. The burn damage of all layers of esophagus;
 - D. The spread of postburn necrosis on paraesophageal tissue and adjacent organs.
 - E. Asymptomatic
335. What is the III degree of esophageal burns?
- A. *The burn damage of all layers of esophagus;
 - B. Superficial burn with the damage of epithelial layer of esophagus;
 - C. The burn with the damage of entire mucosa of esophagus;
 - D. The spread of postburn necrosis on paraesophageal tissue and adjacent organs.
 - E. Asymptomatic
336. What is the IV degree of esophageal burns?
- A. *The spread of postburn necrosis on paraesophageal tissue and adjacent organs.
 - B. Superficial burn with the damage of epithelial layer of esophagus;
 - C. The burn with the damage of entire mucosa of esophagus;
 - D. The burn damage of all layers of esophagus;
 - E. Asymptomatic
337. Superficial burn with the damage of epithelial layer of esophagus relates to such degree of esophageal burns:
- A. *I
 - B. II
 - C. III
 - D. IV
 - E. V
338. The burn with the damage of entire mucosa of esophagus relates to such degree of esophageal burns:
- A. *II
 - B. I
 - C. III
 - D. IV
 - E. V
339. The burn damage of all layers of esophagus relates to such degree of esophageal burns:
- A. *III
 - B. II
 - C. I
 - D. IV
 - E. V
340. The spread of postburn necrosis on paraesophageal tissue and adjacent organs relates to such degree of esophageal burns:
- A. *IV
 - B. III
 - C. II
 - D. I
 - E. V
341. How long has been the risk of esophageal bleeding after the burn?
- A. *1-2 months
 - B. 2-3 days

- C. 10-20 days
 - D. 1-2 years
 - E. 2 years and more
342. How long is formed the esophageal stricture after the burn?
- A. *1-2 years
 - B. 2-3 days
 - C. 10-20 days
 - D. 1-2 months
 - E. 2 years and more
343. What is the roentgenological sign of the esophageal burn of mild degree?
- A. *Free passage of barium with maintained peristalsis
 - B. Filling defects without peristalsis
 - C. "Rat tail" sign with stagnation of barium
 - D. "Bird-beak" sign without peristalsis
 - E. Dilated esophagus with sites of constriction and weak peristalsis
344. What is the roentgenological sign of the esophageal burn of moderate degree?
- A. *Dilated esophagus with sites of constriction and weak peristalsis
 - B. Filling defects without peristalsis
 - C. "Rat tail" sign with stagnation of barium
 - D. "Bird-beak" sign without peristalsis
 - E. Free passage of barium with maintained peristalsis
345. What is the main clinical manifestation of the esophageal burn of severe degree?
- A. *Clinic of shock
 - B. Clinic of reflux-esophagitis
 - C. Clinic of acute abdomen
 - D. Clinic of hepatic insufficiency
 - E. Clinic of respiratory insufficiency
346. What is the main method of diagnostic of esophageal stricture?
- A. *X-ray examination with barium swallow
 - B. General blood analysis
 - C. Ultrasound examination
 - D. Plain X-ray examination of the chest
 - E. Esophagogastroduodenoscopy
347. What solution is used for washing out of acid esophageal burn?
- A. *Sodium hydrocarbonatis solution
 - B. Antiseptic solution
 - C. Antibiotic solution
 - D. Glucose solution
 - E. Vinegar solution
348. What solution is used for washing out of alkaline esophageal burn?
- A. *Vinegar solution
 - B. Antiseptic solution
 - C. Antibiotic solution
 - D. Glucose solution
 - E. Sodium hydrocarbonatis solution
349. What is the main prophylaxis of esophageal stricture after the chemical burn?
- A. *Esophageal bougienage
 - B. Spasmolytics
 - C. Parenteral feeding
 - D. Pneumocompression
 - E. Gastrostomy
350. What complication is typical for esophageal burn?
- A. *Disturbances of epiglottic valve
 - B. Esophageal diverticulum

- C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
351. What complication is typical for esophageal burn?
- A. *Pyloric stenosis
 - B. Esophageal diverticulum
 - C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
352. What complication is typical for esophageal burn?
- A. *Esophageal stricture
 - B. Esophageal diverticulum
 - C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
353. What complication is typical for esophageal burn?
- A. *Gastrointestinal bleeding
 - B. Esophageal diverticulum
 - C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
354. What complication is typical for esophageal burn?
- A. *Mediastinitis
 - B. Esophageal diverticulum
 - C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
355. What complication is typical for acute stage of esophageal burn?
- A. *Shock
 - B. Esophageal diverticulum
 - C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
356. What complication is typical for acute stage of esophageal burn?
- A. *Renal insufficiency
 - B. Esophageal diverticulum
 - C. Obstructive jaundice
 - D. Intestinal obstruction
 - E. Leriche's syndrome
357. What kind of operation is performed for liquidation of esophageal stricture?
- A. *Esophageal plastic
 - B. Esophagostomy
 - C. Gastrostomy
 - D. Resection of the esophagus
 - E. Resection of the stomach
358. What kind of esophageal plastic doesn't exist?
- A. *Retroperitoneal
 - B. Subcutaneous
 - C. Retrosternal
 - D. Intrapleural
 - E. Mediastinal
359. What organ is used for esophageal plastic?
- A. *Large intestine
 - B. Part of vein

- C. Prosthetic graft
 - D. Xenotransplantat
 - E. Lyophilized esophagus
360. What is the predominant manifestations of sliding diaphragmatic hernia?
- A. *Reflux-esophagitis
 - B. Achalasia
 - C. Dysphagia
 - D. Cardiospasm
 - E. Pylorostenosis
361. What is the chief clinical manifestations of reflux-esophagitis?
- A. *Heartburn
 - B. Achalasia
 - C. Dysphagia
 - D. Vomiting
 - E. Coughing
362. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *Pain behind breastbone
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
363. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *Heartburn
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
364. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *Belching by air
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
365. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *Regurgitation
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
366. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *The sign of "lacing shoes"
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
367. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *Nausea and vomiting
 - B. Dyspnea
 - C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
368. For the clinical manifestation of sliding diaphragmatic hernia is typical:
- A. *Dysphagia
 - B. Dyspnea

- C. Cyanosis of the upper part of body
 - D. Retention of stool and gases
 - E. Vomiting by "coffee masses"
369. What is the roentgenological sign of sliding diaphragmatic hernia?
- A. *Blunt His angle
 - B. Filling defects
 - C. "Rat tail" sign
 - D. "Bird-beak" sign
 - E. Esophageal dilatation
370. What is the roentgenological sign of sliding diaphragmatic hernia?
- A. *Lack of air bubble of the stomach
 - B. Esophageal dilatation
 - C. Filling defects
 - D. "Rat tail" sign
 - E. "Bird-beak" sign
371. What is the roentgenological sign of sliding diaphragmatic hernia?
- A. *"Bell" sign
 - B. "Rat tail" sign
 - C. Filling defects
 - D. "Bird-beak" sign
 - E. Esophageal dilatation
372. "Bell"-sign is the X-ray sign of:
- A. *Sliding diaphragmatic hernia
 - B. Achalasia
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
373. Lack of air bubble of the stomach is the X-ray sign of:
- A. *Sliding diaphragmatic hernia
 - B. Achalasia
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
374. Blunt His angle is the X-ray sign of:
- A. *Sliding diaphragmatic hernia
 - B. Achalasia
 - C. Esophageal diverticulum
 - D. Pylorostenosis
 - E. Intestinal obstruction
375. What is the typical clinic of paraesophageal diaphragmatic hernia?
- A. *Asymptomatic
 - B. Achalasia
 - C. Dysphagia
 - D. Pylorostenosis
 - E. Reflux-esophagitis
376. What is the most often complication of paraesophageal diaphragmatic hernia?
- A. *Strangulation
 - B. Bleeding
 - C. Dysphagia
 - D. Pylorostenosis
 - E. Reflux-esophagitis
377. What is the most often complication of sliding diaphragmatic hernia?
- A. *Bleeding
 - B. Strangulation

- C. Dysphagia
 - D. Pylorostenosis
 - E. Reflux-esophagitis
378. What is the most often complication of sliding diaphragmatic hernia?
- A. *Peptic stricture of esophagus
 - B. Strangulation
 - C. Dysphagia
 - D. Pylorostenosis
 - E. Reflux-esophagitis
379. What is the most often complication of sliding diaphragmatic hernia?
- A. *Malignancy
 - B. Strangulation
 - C. Dysphagia
 - D. Pylorostenosis
 - E. Reflux-esophagitis
380. What drugs are used for suppression of gastric secretion?
- A. *Blockers of proton pump
 - B. Spasmolytics
 - C. Adrenoblockers
 - D. Blockers of calcium channel
 - E. Anticoagulants
381. What drugs are used for suppression of gastric secretion?
- A. *H2-histamin blockers
 - B. Spasmolytics
 - C. Adrenoblockers
 - D. Blockers of calcium channel
 - E. Anticoagulants
382. What drugs are used for suppression of gastric secretion?
- A. *Omeprasol, Nexium
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Dextrin, Venoplant
383. What drugs are used for suppression of gastric secretion?
- A. *Lansa, Ultop
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Dextrin, Venoplant
384. What drugs are used for suppression of gastric secretion?
- A. *Esomeprasol, Omez
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Dextrin, Venoplant
385. What drugs are used for suppression of gastric secretion?
- A. *Quamatel, Ranitidin
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Dextrin, Venoplant
386. What drugs belong to blockers of proton pump?
- A. *Esomeprasol, Omez
 - B. Ceftriaxon, Ciprinol

- C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Detrex, Venoplast
387. What drugs belong to blockers of proton pump?
- A. *Lansa, Ultop
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Detrex, Venoplast
388. What drugs belong to blockers of proton pump?
- A. *Omeprasol, Nexium
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Detrex, Venoplast
389. What drugs belong to H₂-histamin blockers?
- A. *Quamatel, Ranitidin
 - B. Ceftriaxon, Ciprinol
 - C. Euphyllin, No-spa
 - D. Nicotine acid, Heparin
 - E. Detrex, Venoplast
390. Quamatel, Ranitidin belong to:
- A. *H₂-histamin blockers
 - B. Blockers of proton pump
 - C. Adrenoblockers
 - D. Blockers of calcium channel
 - E. Anticoagulants
391. Omeprasol, Nexium belong to:
- A. *Blockers of proton pump
 - B. H₂-histamin blockers
 - C. Adrenoblockers
 - D. Blockers of calcium channel
 - E. Anticoagulants
392. Lansan, Ultop belong to:
- A. *Blockers of proton pump
 - B. H₂-histamin blockers
 - C. Adrenoblockers
 - D. Blockers of calcium channel
 - E. Anticoagulants
393. What disease should be the sliding diaphragmatic hernia differentiated from?
- A. *Angina pectoris
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
394. What disease should be the sliding diaphragmatic hernia differentiated from?
- A. *Peptic ulcer
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
395. What disease should be the sliding diaphragmatic hernia differentiated from?
- A. *Lung atelectasis
 - B. Pancreatitis

- C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
396. What disease should be the sliding diaphragmatic hernia differentiated from?
- A. *Pleurisy
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
397. What disease should be the sliding diaphragmatic hernia differentiated from?
- A. *Pneumonia
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
398. What disease should be the sliding diaphragmatic hernia differentiated from?
- A. *Hypochromic anemia
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
399. What is the main treatment of sliding diaphragmatic hernia?
- A. *Conservative treatment
 - B. Esophagostomy
 - C. Esophageal plastic
 - D. Resection of the esophagus
 - E. Resection of the stomach
400. What is the main treatment of sliding diaphragmatic hernia?
- A. *Cruroplasty with Nissen's fundoplication
 - B. Esophagostomy
 - C. Esophageal plastic
 - D. Resection of the esophagus
 - E. Resection of the stomach
401. What type of operation is used for paraesophageal diaphragmatic hernia?
- A. *Cruroplasty
 - B. Esophagostomy
 - C. Resection of the esophagus
 - D. Resection of the stomach
 - E. Cruroplasty with Nissen's fundoplication
402. What type of operation is used for sliding diaphragmatic hernia?
- A. *Cruroplasty with Nissen's fundoplication
 - B. Esophagostomy
 - C. Cruroplasty
 - D. Resection of the esophagus
 - E. Resection of the stomach
403. Cruroplasty with Nissen's fundoplication is used for the treatment of:
- A. *Sliding diaphragmatic hernia
 - B. Esophageal achalasia
 - C. Esophageal cancer
 - D. Esophageal diverticulum
 - E. Pylorostenosis
404. Nissen's fundoplication is used for prevention of:
- A. *Reflux-esophagitis
 - B. Intestinal obstruction

- C. Esophageal cancer
 - D. Esophageal diverticulum
 - E. Pylorostenosis
405. For prevention of reflux-esophagitis is used:
- A. *Nissen's fundoplication
 - B. Hellerovskiy's operation
 - C. Heller's operation
 - D. Cocher's operation
 - E. Lerishe's operation
406. What is the roentgenological sign of diaphragmatic relaxation?
- A. *High standing of diaphragmatic dome
 - B. Filling defect
 - C. "Rat tail" sign
 - D. Sign of "nishe"
 - E. Pneumoperitoneum
407. What is the X-ray sign of diaphragmatic relaxation?
- A. *Restriction of diaphragmatic excursion
 - B. Filling defect
 - C. "Rat tail" sign
 - D. Sign of "nishe"
 - E. Pneumoperitoneum
408. What is the X-ray sign of diaphragmatic relaxation?
- A. *Inflection of abdominal part of esophagus
 - B. Filling defect
 - C. "Rat tail" sign
 - D. Sign of "nishe"
 - E. Pneumoperitoneum
409. What disease should be the diaphragmatic relaxation differentiated from?
- A. *Diaphragmatic elevation
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
410. What disease should be the diaphragmatic relaxation differentiated from?
- A. *Pneumothorax
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
411. What disease should be the diaphragmatic relaxation differentiated from?
- A. *Pyopneumothorax
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
412. What disease should be the diaphragmatic relaxation differentiated from?
- A. *Pleurisy
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
413. What disease should be the diaphragmatic relaxation differentiated from?
- A. *Diaphragmatic hernia
 - B. Pancreatitis

- C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
414. What disease should be the diaphragmatic relaxation differentiated from?
- A. *Esophageal cancer
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
415. What operation is performed for diaphragmatic relaxation?
- A. *Phrenoplasty
 - B. Esophagomyotomy
 - C. Cruroplasty
 - D. Cruroplasty with Nissen's fundoplication
 - E. Esophageal plastics by intestine
416. Phrenoplasty is used for the treatment of:
- A. *Diaphragmatic relaxation
 - B. Sliding diaphragmatic hernia
 - C. Esophageal achalasia
 - D. Esophageal diverticulum
 - E. Pylorostenosis
417. What is the cause of acute mediastinitis?
- A. *Perforation of esophagus
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatricial changes after the burn of esophagus
418. What is the cause of acute mediastinitis?
- A. *Chemical burns of esophagus
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatricial changes after the burn of esophagus
419. What is the cause of acute mediastinitis?
- A. *Injuries of trachea
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatricial changes after the burn of esophagus
420. What is the cause of acute mediastinitis?
- A. *Injuries of bronchi
 - B. Ischemia of esophagus
 - C. Tumour growth of esophagus
 - D. Diverticula of esophagus
 - E. Cicatricial changes after the burn of esophagus
421. For the clinical manifestation of acute mediastinitis is typical:
- A. F. *Hectic temperature
 - B. G. Vomiting
 - C. H. Retention of stool and gases
 - D. Portal hypertension
 - E. J. Obstructive jaundice
422. For the clinical manifestation of acute mediastinitis is typical:
- A. *Dyspnea
 - B. Vomiting

- C. Retention of stool and gases
 - D. Portal hypertension
 - E. Obstructive jaundice
423. For the clinical manifestation of acute mediastinitis is typical:
- A. *Profuse sweating
 - B. Vomiting
 - C. Retention of stool and gases
 - D. Portal hypertension
 - E. Obstructive jaundice
424. For the clinical manifestation of acute mediastinitis is typical:
- A. *Dysphagia
 - B. Vomiting
 - C. Retention of stool and gases
 - D. Portal hypertension
 - E. Obstructive jaundice
425. For the clinical manifestation of acute mediastinitis is typical:
- A. *Constant cough
 - B. Vomiting
 - C. Retention of stool and gases
 - D. Portal hypertension
 - E. Obstructive jaundice
426. For the clinical manifestation of acute mediastinitis is typical:
- A. *Hoarseness
 - B. Vomiting
 - C. Retention of stool and gases
 - D. Portal hypertension
 - E. Obstructive jaundice
427. For the clinical manifestation of acute mediastinitis is typical:
- A. *Change of cardiac rhythm
 - B. Vomiting
 - C. Retention of stool and gases
 - D. Portal hypertension
 - E. Obstructive jaundice
428. The fever to 39-40°C is typical for:
- A. *Acute mediastinitis
 - B. Sliding diaphragmatic hernia
 - C. Esophageal achalasia
 - D. Esophageal cancer
 - E. Esophageal diverticulum
429. The dyspnea is typical for:
- A. *Acute mediastinitis
 - B. Sliding diaphragmatic hernia
 - C. Esophageal achalasia
 - D. Esophageal cancer
 - E. Esophageal diverticulum
430. The chest pain is typical for:
- A. *Acute mediastinitis
 - B. Sliding diaphragmatic hernia
 - C. Esophageal achalasia
 - D. Esophageal cancer
 - E. Esophageal diverticulum
431. The severe intoxication is typical for:
- A. *Acute mediastinitis
 - B. Sliding diaphragmatic hernia

- C. Esophageal achalasia
 - D. Esophageal cancer
 - E. Esophageal diverticulum
432. The dysphagia is typical for:
- A. *Acute mediastinitis
 - B. Bronchitis
 - C. Lung emphysema
 - D. Pulmonary hypertension
 - E. Lung cyst
433. What is the roentgenological sign of acute mediastinitis?
- A. *Widening of mediastinum, shadowing of its anterior
 - B. Filling defect
 - C. The sign of "bell"
 - D. Lack of air bubble of the stomach
 - E. High standing of diaphragmatic dome
434. What sign is typical for anterior mediastinitis?
- A. *Intensifying of pain during percussion of breast bone
 - B. Throbbing chest pain with irradiation in interscapular region
 - C. Intensifying of pain during vertebral pressing
 - D. Intensifying of pain at swallowing
 - E. Sign of compression of azygos and hemiazygos veins
435. What sign is typical for anterior mediastinitis?
- A. *Intensifying of pain when head is unbent back
 - B. Throbbing chest pain with irradiation in interscapular region
 - C. Intensifying of pain at swallowing
 - D. Swelling above clavicle
 - E. Sign of compression of azygos and hemiazygos veins
436. What sign is typical for anterior mediastinitis?
- A. *Signs of compression of superior vena cava
 - B. Throbbing chest pain with irradiation in interscapular region
 - C. Intensifying of pain at swallowing
 - D. Swelling above clavicle
 - E. Sign of compression of azygos and hemiazygos veins
437. What sign is typical for posterior mediastinitis?
- A. *Throbbing chest pain with irradiation in interscapular region
 - B. Intensifying of pain during percussion of breast bone
 - C. Intensifying of pain when head is unbent back
 - D. Occurrence of swelling in the region of jugular fossa
 - E. Signs of compression of superior vena cava
438. What sign is typical for posterior mediastinitis?
- A. *Intensifying of pain during vertebral pressing
 - B. Intensifying of pain during percussion of breast bone
 - C. Intensifying of pain when head is unbent back
 - D. Occurrence of swelling in the region of jugular fossa
 - E. Signs of compression of superior vena cava
439. What sign is typical for posterior mediastinitis?
- A. *Sign of compression of azygos and hemiazygos veins
 - B. Intensifying of pain during percussion of breast bone
 - C. Intensifying of pain when head is unbent back
 - D. Occurrence of swelling in the region of jugular fossa
 - E. Signs of compression of superior vena cava
440. What disease should be the acute mediastinitis differentiated from?
- A. *Pyopneumothorax
 - B. Diaphragmatic hernia

- C. Intestinal obstruction
 - D. Cholecystitis
 - E. Esophageal achalasia
441. What disease should be the acute mediastinitis differentiated from?
- A. *Acute pneumonia
 - B. Diaphragmatic hernia
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Esophageal achalasia
442. What disease should be the acute mediastinitis differentiated from?
- A. *Pleural empyema
 - B. Diaphragmatic hernia
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Esophageal achalasia
443. What disease should be the acute mediastinitis differentiated from?
- A. *Exsudative pericarditis
 - B. Diaphragmatic hernia
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Esophageal achalasia
444. What operation is performed for acute mediastinitis?
- A. *Mediastinotomy
 - B. Esophagomyotomy
 - C. Thoracotomy
 - D. Laparotomy
 - E. Phrenoplasty
445. Mediastinotomy is used for the treatment of:
- A. *Acute mediastinitis
 - B. Sliding diaphragmatic hernia
 - C. Esophageal achalasia
 - D. Esophageal diverticulum
 - E. Pylorostenosis
446. Which of the following statements about microalbuminuria is true?
- A. To be of clinical value, microalbuminuria must be measured in a time 12- to 24-hour
 - B. *Microalbuminuria is a cardiovascular risk factor that is independent of traditional Framingham risk factors
 - C. Microalbuminuria is a predictor of risk only in patients with diabetes
 - D. Microalbuminuria is present when the “spot” urine albumin-to-creatinine ration is >500 mg/l
 - E. All of the above statements are correct
447. All of the following are associated with hyperviscosity in the setting of Eisenmenger's syndrome except:
- A. *Coronary artery ectasia
 - B. Erythrocytosis
 - C. Visual disturbances
 - D. Paresthesias
 - E. Thrombosis and bleeding
448. All of the following physical examination findings are usually associated with ostium Secundary except:
- A. Precordial heave
 - B. Fixed split S2
 - C. *Lateral and inferior displacement of the apex beat
 - D. Soft systolic ejection murmur in the second left intercostal space

- E. Normal S1
449. All of the following statements regarding patent ductus arteriosus (PDA) are true except:
- A. *The majority of cases close spontaneously after infancy
 - B. There is a higher incidence in mothers who acquired rubella during pregnancy
 - C. A decrease in the duration and intensity of the murmur has a poor prognostic implication
 - D. Left ventricular hypertrophy precedes RV hypertrophy
 - E. If it is uncorrected, approximately one third of patients die by the age of 40 years
450. Congenital MR is commonly encountered in all of the following conditions except:
- A. *Cor triatriatum
 - B. Ostium primum ASD
 - C. Coarctation of the aorta
 - D. Congenitally corrected TGA
 - E. Subaortic stenosis
451. Coronary sinus ASD is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. *Left superior vena cava
452. Each of the following statements regarding splitting of the second heart sound is true except:
- A. Severe pulmonic valvular stenosis is associated with a softened P2
 - B. Delayed closure of the pulmonic valve with inspiration contributes to physiologic splitting of S2
 - C. Fixed splitting of S2 is the auscultatory hallmark of an ostium Secondary atrial septal defect
 - D. Paradoxical splitting of S2 is expected in patients with a right ventricular electronically paced rhythm
 - E. *Right bundle branch block is associated with paradoxical splitting of S2
453. In which of the following cases is surgical correction recommended?
- A. Asymptomatic small VSD to decrease risk of endocarditis
 - B. PDA with severe pulmonary hypertension
 - C. *Asymptomatic subaortic stenosis with severe aortic valve insufficiency
 - D. Coarctation of the aorta with a transcoarctation gradient of 20 mmHg item
 - E. Small ASD to prevent paradoxical embolization
454. Which adult congenital disorder corresponds to the following physical examination findings: wide pulse pressure, prominent LV impulse, and a continuous machinery murmur enveloping S2
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. *Patent ductus arteriosus
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
455. Which congenital disorder corresponds with the following chest radiography findings: prominent central PAs (possible calcifications) and peripheral PA pruning?
- A. *Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
456. Which lifestyle change has been shown to produce the biggest reduction in systolic blood pressure?
- A. *10-kg weight loss
 - B. Dietary sodium reduction
 - C. Moderation of alcohol consumption

- D. Change to a vegetarian diet
 - E. Magnesium supplement
457. Which of the following syndromes is associated with pulmonary arterio-venous fistula?
- A. Williams syndrome
 - B. *Weber-Osler-Rendu syndrome
 - C. Bland-Garland-White syndrome
 - D. Kartagener's syndrome
 - E. Crouzon's syndrome
458. A 30-year-old man with Eisenmenger's syndrome and irreversible pulmonary hypertension caused by untreated VSD is at risk for developing symptoms and signs of hyperviscosity. All of the following are associated with hyperviscosity syndrome except
- A. *Coronary artery ectasia
 - B. Erythrocytosis
 - C. Visual disturbances
 - D. Paresthesias
 - E. Thrombosis and bleeding
459. A 34-year-old female with hypertension is considering becoming pregnant. Which of the following medications would be absolutely CONTRAINDICATED to control her BP during pregnancy?
- A. Methyldopa
 - B. Metoprolol
 - C. Labetalol
 - D. *Captopril
 - E. Nifedipine
460. A 48-year-old male with diabetes mellitus, hypertension, and hyperlipidemia presents to the ER with hypertensive emergency. His mean arterial pressure is 150 mmHg. Which medications would be most appropriate therapy for this patient?
- A. *Nitroprusside
 - B. Enteral metoprolol
 - C. Fenoldopam
 - D. Intravenous nitroglycerine
 - E. Any of the above
461. A young hypertensive patient has serum potassium 2.8 mEq/l and increased aldosterone level with decreased plasma renin activity. The diagnosis is:
- A. Renal artery stenosis
 - B. Ectopic ACTH syndrome
 - C. *Conn syndrome
 - D. Liddle syndrome
 - E. Cushing syndrome
462. All of the following are characteristic findings of ostium primum atrial septal defect (ASD) except:
- A. Precordial heave
 - B. Fixed split S2
 - C. *Right axis deviation
 - D. Systolic ejection murmur
 - E. Prominent pulmonary vascular markings on CXR
463. All of the following are indications for surgical closure of an ASD except:
- A. Significant symptoms in a 65-year-old
 - B. RV dysfunction
 - C. *Pulmonary vascular resistance >15
 - D. An asymptomatic 20-year-old with a Qp/Qs of 1.7 with no pulmonary hypertension
 - E. RV enlargement
464. All of the following are risk factors for hypertension, except
- A. Increased body weight

- B. Family history of hypertension
 - C. Excessive intake of sodium
 - D. *Regular use of one glass of wine per month
 - E. Cigarette smoking
465. All of the following characteristics are typical of hypertensive crisis except:
- A. Diastolic BP >120 mmHg
 - B. Retinal hemorrhages
 - C. *Constriction of cerebral arterioles with decreased vascular permeability
 - D. Proteinuria
 - E. Microangiopathic hemolytic anemia
466. ?All of the following findings are suggestive of LVH except:
- A. S in V1 + R in V5 or V6 > 35 mm
 - B. R in aVL > 11 mm
 - C. R in aVF > 20 mm
 - D. R in I + S in III > 25 mm
 - E. *R in aVR > 8 mm
467. All of the following findings are suggestive of RVH except:
- A. R in V1=7 mm (15 mm with RBBB)
 - B. R in V1 + S in V5 or V6 > 10 mm
 - C. R < S in V6
 - D. R or R' in V1
 - E. *S in V3 + R in aVL > 28 mm (20 mm for women)
468. All of the following statements concerning hypertension are true, except
- A. In a western adult population the prevalence of hypertension exceeds 20%
 - B. Hypertension is a major risk factor for cardiovascular and cerebrovascular disease
 - C. The pathophysiology of hypertension differs in black adults compared to South Asians and Europeans
 - D. *People of African descent commonly have a high renin type hypertension
 - E. An increase in potassium intake may significantly reduce blood pressure in hypertensive patients
469. All of the following statements concerning target organ damage are true except
- A. Microalbuminuria is a sensitive marker of hypertension-induced renal damage
 - B. *An increase in serum creatinine when antihypertensive therapy is intensified is a sign of progressive renal deterioration
 - C. Electrocardiography should be part of all routine assessment of subjects with high BP
 - D. Echocardiography is much more sensitive than electrocardiography in diagnosing left ventricular hypertrophy
 - E. Grades 1 and 2 arteriolar retinal changes do not have a significant prognostic value
470. All of the following statements regarding Ebstein's anomaly are true except
- A. An ASD or PFO is present in up to 80% of patients
 - B. The cardinal feature is an apically displaced tricuspid valve resulting in atrialization of ventricular tissue
 - C. WPW syndrome is common in these patients and multiple tracts can exist
 - D. *A bicuspid aortic valve is commonly present
 - E. A "sail sound" is a common finding on physical examination
471. All of the following statements regarding the effects of maneuvers on the auscultation of cardiac murmurs are true except:
- A. In patent ductus arteriosus (PDA), the diastolic phase of the murmur is intensified by isometric handgrip
 - B. *The murmur of hypertrophic obstructive cardiomyopathy becomes softer with standing or during a Valsalva strain maneuver
 - C. The murmur of a ventricular septal defect (VSD) increases with isometric handgrip
 - D. Sudden squatting or isometric handgrip increases the diastolic murmur of aortic regurgitation

- E. The mid-diastolic and presystolic murmurs of mitral stenosis become louder with exercise
472. Bacterial endocarditis prophylaxis is indicated in all adults who have the following congenital heart disease except:
- A. VSD
 - B. Coarctation of the aorta
 - C. *Secondary ASD
 - D. Hypertrophic obstructive cardiomyopathy
 - E. PDA
473. Besides pulmonary valve stenosis, which of the following is the most common associated cardiac defect present in patients with PA stenosis?
- A. *VSD
 - B. ASD
 - C. Coarctation of the aorta
 - D. PDA
 - E. Bicuspid aortic valve
474. Choose the adult congenital disorder corresponding to the following physical examination findings: wide pulse pressure, prominent LV impulse, and a continuous machinery murmur enveloping S2:
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. *Patent ductus arteriosus
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
475. Choose the adult congenital disorder corresponding to the following physical examination findings: weak or delayed femoral pulses, harsh systolic murmur in the back, and a systolic ejection click in the aortic area
- A. Eisenmenger's syndrome
 - B. *Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
476. Choose the adult congenital disorder corresponding to the following physical examination findings: RV lift with a loud systolic ejection murmur along the left sternal border, with a single S2
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
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477. Choose the adult congenital disorder corresponding to the following physical examination findings: loud S1, holosystolic murmur in left sternal border, systolic ejection click, and hepatomegaly
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. *Ebstein's anomaly
 - E. Tetralogy of Fallot
478. Choose the adult congenital disorder corresponding to the following physical examination findings: cyanosis, digital clubbing, loud P2, and a variable Graham-Steel murmur
- A. *Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly

- E. Tetralogy of Fallot
479. Choose the case in which surgical correction is recommended?
- A. Asymptomatic small VSD to decrease risk of endocarditis
 - B. PDA with severe pulmonary hypertension
 - C. *Asymptomatic subaortic stenosis with severe aortic valve insufficiency
 - D. Coarctation of the aorta with a transcoarctation gradient of 20 mmHg
 - E. Small ASD to prevent paradoxical embolization
480. Choose the congenital disorder compatible with the following chest radiography findings: posterior rib notching and a "reverse E" or "3" sign
- A. Eisenmenger's syndrome
 - B. *Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
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481. Choose the congenital disorder compatible with the following chest radiography findings: pulmonary plethora, prominent ascending aorta, proximal PA dilatation, and opacity at the confluence of the aortic knob and descending aorta
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. *PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
482. Choose the congenital disorder compatible with the following chest radiography findings: marked cardiomegaly, severe right atrial enlargement, and normal lung fields
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. *Ebstein's anomaly
 - E. Tetralogy of Fallot
483. Choose the congenital disorder compatible with the following chest radiography findings: prominent central PAs (possible calcifications) and peripheral PA pruning?
- A. *Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
484. Choose the congenital disorder compatible with the following chest radiography findings: right aortic arch, RV enlargement, and a "boot-shaped" heart?
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. *Tetralogy of Fallot
485. Each of the following statements regarding the effects of maneuvers on the auscultation of cardiac murmurs is true except:
- A. Ductus arteriosus (PDA), the diastolic phase of the murmur is intensified by isometric handgrip
 - B. *The murmur of hypertrophic obstructive cardiomyopathy becomes softer with standing or during a Valsalva strain maneuver
 - C. The murmur of a ventricular septal defect (VSD) increases with isometric handgrip
 - D. Sudden squatting or isometric handgrip increases the diastolic murmur of aortic regurgitation
 - E. The mid-diastolic and presystolic murmurs of mitral stenosis become louder with exercise

486. If there is suspicion of pheochromocytoma, the first line drug is:
- A. Sodium-nitroprusside
 - B. Nifedipine
 - C. *Phentolamine
 - D. Verapamil
 - E. Enalapril
487. In patients of African descent the drug of choice for initial treatment of hypertension is:
- A. Verapamil
 - B. Atenolol
 - C. Perindopril
 - D. *Hydrochlorothiazide
 - E. Amlodipine
488. In the elderly with systolic hypertension antihypertensive therapy should be initiated if SBP
- A. 120 mmHg
 - B. 130 mmHg
 - C. 140 mmHg
 - D. *160 mmHg
 - E. 170 mmHg
489. Infective endocarditis prophylaxis is indicated in all adults who have the following congenital heart disease except:
- A. VSD
 - B. Coarctation of the aorta
 - C. *Secondary ASD
 - D. Hypertrophic obstructive cardiomyopathy
 - E. PDA
490. Noonan's syndrome is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. *Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
491. Of the following pairs, choose one which is incorrectly matched with the indication for therapy in hypertension?
- A. ACE inhibitor – diabetic nephropathy
 - B. Beta-blocker – coronary artery disease
 - C. Calcium channel blocker – angina pectoris
 - D. Diuretics – heart failure
 - E. *Loop diuretic – gout
492. Of the following syndromes, which one is associated with pulmonary arterio-venous fistula?
- A. Williams syndrome
 - B. *Weber-Osler-Rendu syndrome
 - C. Bland-Garland-White syndrome
 - D. Kartagener's syndrome
 - E. Crouzon's syndrome
493. Optimal blood pressure (BP) is defined as a BP level of
- A. *120/80 mmHg
 - B. 130/80 mmHg
 - C. 140/90 mmHg
 - D. 160/100 mmHg
 - E. 125/80 mmHg
494. Ostium primum ASD is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. Supravalvular pulmonic stenosis
 - C. *Cleft mitral valve

- D. Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
495. Physicians should diagnose isolated office hypertension (so-called “white-coat hypertension”) whenever office BP is $\geq 140/90$ mmHg at several visits, while 24-h ambulatory BP is
- A. 160/100 mmHg
 - B. 140/90 mmHg
 - C. 130/85 mmHg
 - D. *125/80 mmHg
 - E. 120/70 mmHg
496. Renin-dependent hypertension includes:
- A. Primary hyperaldosteronism
 - B. Essential hypertension
 - C. *Renovascular hypertension
 - D. Pheochromocytoma
 - E. Cushing syndrome
497. Sinus venosus ASD is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. *Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
498. Target organs in hypertension include all of the following except
- A. Brain and eyes
 - B. Heart
 - C. Kidneys
 - D. Peripheral arteries
 - E. *Liver
499. Tetralogy of Fallot includes all of the following lesions except
- A. A ventricular septal defect
 - B. An overriding aorta
 - C. *An atrial septal defect
 - D. Right ventricular outflow obstruction
 - E. Right ventricular hypertrophy
500. The best medication for the treatment of isolated systolic hypertension in the elderly:
- A. ACE-inhibitor and diuretic
 - B. *Dihydropyridine calcium-channel blocker and diuretic
 - C. Beta-blocker and diuretic
 - D. Non-dihydropyridine calcium-channel blocker and diuretic
 - E. Beta- and alpha blocker
501. The congenital mitral regurgitation is commonly encountered in all of the following conditions except:
- A. *Cor triatriatum
 - B. Ostium primum ASD
 - C. Coarctation of the aorta
 - D. Congenitally corrected TGA
 - E. Subaortic stenosis
502. The coronary sinus ASD is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. *Persistent left superior vena cava
503. The following cardiovascular malformations are all associated with congenital rubella except:

- A. PDA
 - B. PA stenosis
 - C. *Ebstein's anomaly
 - D. Tetralogy of Fallot
 - E. Coarctation of the aorta
504. The following drug combinations are effective to treat hypertension except
- A. Diuretics + beta-blockers
 - B. Beta-blockers + dihydropyridine calcium channel blockers
 - C. Diuretics + ACE-inhibitors/ARB
 - D. ACE-inhibitors/ARB + calcium channel blockers
 - E. *Beta-blockers + ACE-inhibitors/ARB
505. The following medications can be used in pregnancy except
- A. Methyl dopa
 - B. Labetalol
 - C. *ACE-inhibitor / ARB
 - D. Hydralazine
 - E. Nifedipine
506. The following statements regarding patent ductus arteriosus (PDA) are true except:
- A. *The majority of cases close spontaneously after infancy
 - B. There is a higher incidence in mothers who acquired rubella during pregnancy
 - C. A decrease in the duration and intensity of the murmur has a poor prognostic implication
 - D. Left ventricular hypertrophy precedes RV hypertrophy
 - E. If it is uncorrected, approximately one third of patients die by the age of 40 years
507. The Noonan's syndrome is commonly associated with:
- A. Supravalvular aortic stenosis
 - B. *Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
508. The ostium primum ASD is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. Supravalvular pulmonic stenosis
 - C. *Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
509. The ostium Secundary ASD is usually associated with the following physical examination findings except:
- A. Precordial heave
 - B. Fixed split S2
 - C. *Lateral and inferior displacement of the apex beat
 - D. Soft systolic ejection murmur in the second left intercostal space
 - E. Normal S1
510. The proportion of essential (primary) hypertension among all hypertension causes is as high as
- A. 25-30%
 - B. 40-45%
 - C. 60-65%
 - D. 70-75%
 - E. *90-95%
511. The sinus venosus ASD is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. *Anomalous pulmonary venous drainage

- E. Persistent left superior vena cava
512. The timing of an "innocent" murmur is usually:
- A. Early systolic
 - B. Presystolic
 - C. *Midsystolic
 - D. Holosystolic
 - E. Early diastolic
513. The Williams syndrome is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. *Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
514. Thiazide diuretics can contribute to each of the following metabolic effects except:
- A. Hypomagnesemia
 - B. *Hypouricemia
 - C. Hypercalcemia
 - D. Hypercholesterolemia
 - E. Hyponatremia
515. What is the correct feature of the hypertensive urgency?
- A. It is always a life-threatening situation
 - B. Patient must be hospitalized
 - C. *Patients can be managed as outpatients
 - D. Therapy should not be given orally
 - E. None of the above
516. What is the most common coexisting congenital anomaly in patients with coarctation of the aorta?
- A. Cleft mitral valve
 - B. *Bicuspid aortic valve
 - C. Ebstein's anomaly
 - D. VSD
 - E. PDA
517. When present, each of the following heart sounds occurs shortly after S2 except:
- A. Opening snap
 - B. Third heart sound
 - C. *Ejection clic
 - D. Tumor plop
 - E. Pericardial knock
518. Which adult congenital disorder corresponds to the following physical examination findings: cyanosis, digital clubbing, loud P2, and a variable Graham-Steel murmur
- A. *Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
519. Which adult congenital disorder corresponds to the following physical examination findings: RV lift with a loud systolic ejection murmur along the left sternal border, with a single S2
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. *Tetralogy of Fallot
520. Which BP profile below identifies the patient with the highest risk for development of cardiovascular complications?

- A. 160/90 mmHg
 - B. *160/65 mmHg
 - C. 140/100 mmHg
 - D. 130/90 mmHg
 - E. 120/70 mmHg
521. Which condition is an absolute contraindication to pregnancy?
- A. Surgically corrected transposition of great arteries (TGA)
 - B. Congenitally corrected TGA
 - C. Ebstein's anomaly
 - D. *Eisenmenger's syndrome
 - E. Status post Fontan operation
522. Which congenital disorder corresponds with the following chest radiography findings: pulmonary plethora, prominent ascending aorta, proximal PA dilatation, and opacity at the confluence of the aortic knob and descending aorta
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 - B. Coarctation of the aorta
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523. Which congenital disorder corresponds with the following chest radiography findings: posterior rib notching and a "reverse E" or "3" sign
- A. Eisenmenger's syndrome
 - B. *Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
524. Which congenital disorder corresponds with the following chest radiography findings: right aortic arch, RV enlargement, and a "boot-shaped" heart?
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. *Tetralogy of Fallot
525. Which congenital disorder corresponds with the following chest radiography findings: marked cardiomegaly, severe right atrial enlargement, and normal lung fields
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. *Ebstein's anomaly
 - E. Tetralogy of Fallot
526. Which lifestyle change has been shown to produce the biggest reduction in systolic blood pressure?
- A. *10-kg weight loss
 - B. Dietary sodium reduction
 - C. Moderation of alcohol consumption
 - D. Change to a vegetarian diet
 - E. Magnesium supplement
527. Which of the following is NOT a characteristic findings of ostium primum atrial septal defect (ASD)?
- A. Precordial heave
 - B. Fixed split S2
 - C. *Right axis deviation
 - D. Systolic ejection murmur
 - E. Prominent pulmonary vascular markings on CXR

528. Which of the adult congenital disorders corresponds to the following physical examination findings: weak or delayed femoral pulses, harsh systolic murmur in the back, and a systolic ejection click in the aortic area
- A. Eisenmenger's syndrome
 - B. *Coarctation of the aorta
 - C. PDA
 - D. Ebstein's anomaly
 - E. Tetralogy of Fallot
529. Which of the adult congenital disorders corresponds to the following physical examination findings: loud S1, holosystolic murmur in left sternal border, systolic ejection click, and hepatomegaly
- A. Eisenmenger's syndrome
 - B. Coarctation of the aorta
 - C. PDA
 - D. *Ebstein's anomaly
 - E. Tetralogy of Fallot
530. Which of the following antihypertensive agents is incorrectly matched with the indication for therapy?
- A. ACE inhibitor – diabetic nephropathy
 - B. Beta-blocker – coronary artery disease
 - C. Calcium channel blocker – angina pectoris
 - D. *Hydrochlorothiazide – diabetes mellitus
 - E. Loop diuretic – heart failure
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 - C. Calcium channel blocker – angina pectoris
 - D. *Hydrochlorothiazide – diabetes mellitus
 - E. Loop diuretic – heart failure
533. Which of the following antihypertensive agents is a known cause of autoimmune hemolytic anemia?
- A. Metoprolol
 - B. *Methyldopa
 - C. Captopril
 - D. Losartan
 - E. Monoxidine
534. Which of the following cardiovascular malformations is NOT associated with congenital rubella?
- A. PDA
 - B. PA stenosis
 - C. *Ebstein's anomaly
 - D. Tetralogy of Fallot
 - E. Coarctation of the aorta
535. Which of the following diagnostic studies is most likely to demonstrate the cause of the headaches?
- A. MRI of the head

- B. MRI of the kidney
 - C. *MRI of the thorax
 - D. 24-h urinary 5-HIAA
 - E. 24-h urinary free cortisol
536. Which of the following is an absolute contraindication to pregnancy?
- A. Surgically corrected transposition of great arteries (TGA)
 - B. Congenitally corrected TGA
 - C. Ebstein's anomaly
 - D. *Eisenmenger's syndrome
 - E. Status post Fontan operation
537. Which of the following is NOT an indication for surgical closure of an ASD?
- A. Significant symptoms in a 65-year-old
 - B. RV dysfunction
 - C. *Pulmonary vascular resistance >15 Wood units that does not diminish with vasodilators
 - D. An asymptomatic 20-year-old with a Qp/Qs of 1,7 with no pulmonary hypertension
 - E. RV enlargement
538. Which of the following is the most common associated cardiac defect present in patients with pulmonary stenosis?
- A. *VSD
 - B. ASD
 - C. Coarctation of the aorta
 - D. PDA
 - E. Bicuspid aortic valve
539. Which of the following pairs of medical conditions and antihypertensive medications would be incorrect to use in a patient with essential hypertension?
- A. Beta-blocker and a history of myocardial infarction
 - B. Alpha-blocker and prostatic hypertrophy
 - C. *Thiazide diuretic and gout
 - D. Amlodipine and heart failure
 - E. ACE-inhibitor and diabetes mellitus
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 - B. Alpha-blocker and prostatic hypertrophy
 - C. *Thiazide diuretic and gout
 - D. Amlodipine and heart failure
 - E. ACE-inhibitor and diabetes mellitus
541. Which of the following statements regarding antihypertensive agents and atrial fibrillation (AF) is true?
- A. *Losartan has been shown to decrease new-onset AF more effectively than atenolol
 - B. Valsartan has been shown to decrease new-onset AF more effectively than amlodipine
 - C. Atenolol has been shown to decrease new-onset AF more effectively than captopril
 - D. Nifedipine has been shown to decrease new-onset AF more effectively than diltiazem
 - E. All of the above are false
542. Which of the following statements regarding Ebstein's anomaly is NOT correct?
- A. An ASD or PFO is present in up to 80% of patients
 - B. The cardinal feature is an apically displaced tricuspid valve resulting in atrialization of ventricular tissue
 - C. WPW syndrome is common in these patients and multiple tracts can exist
 - D. *A bicuspid aortic valve is commonly present
 - E. A "sail sound" is a common finding on physical examination
543. Which of the following statements regarding prevention of stroke is correct?
- A. Treatment of systolic hypertension does not decrease the risk of stroke in patients older than age 60

- B. Hypertension should not be a target of secondary prevention after an ischemic stroke because elevated BP is desirable to maintain adequate cerebral perfusion
 - C. *Treatment with HMG-CoA reductase inhibitors reduces the risk of recurrent stroke
 - D. The combination of aspirin and clopidogrel is superior to aspirin alone for prevention of recurrent stroke
 - E. None of the above
544. Which of the following syndromes and cardiac anomalies are incorrectly matched?
- A. Trisomy 21 – atrioventricular canal defects
 - B. Noonan syndrome – pulmonic stenosis
 - C. Holt-Oram syndrome – atrial septal defects
 - D. Marfan syndrome – mitral valve prolapse
 - E. *Williams syndrome – ventricular septal defects
545. Which of these statements is true regarding renin-angiotensin system-blocking agents?
- A. Less effective when combined with a diuretic than when used alone
 - B. More effective in patients of African descent than in white patients
 - C. *Preserve kidney function in addition to lowering blood pressure
 - D. Are the first-line antihypertensive medications in pregnancy
 - E. All of the above
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547. Which parameter of arterial blood pressure (BP) yields the best prognostic information in patients at risk for cardiovascular disease?
- A. Systolic BP
 - B. Diastolic BP
 - C. Mean arterial BP
 - D. *Pulse pressure
 - E. None
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 - E. ACE-inhibitor and diabetes mellitus
576. Which of the following statements regarding antihypertensive agents and atrial fibrillation (AF) is true?
- A. *Losartan has been shown to decrease new-onset AF more effectively than atenolol
 - B. Valsartan has been shown to decrease new-onset AF more effectively than amlodipine
 - C. Atenolol has been shown to decrease new-onset AF more effectively than captopril
 - D. Nifedipine has been shown to decrease new-onset AF more effectively than diltiazem
 - E. All of the above are false
577. Which of the following statements regarding Ebstein's anomaly is NOT correct?
- A. An ASD or PFO is present in up to 80% of patients
 - B. The cardinal feature is an apically displaced tricuspid valve resulting in atrialization of ventricular tissue
 - C. WPW syndrome is common in these patients and multiple tracts can exist
 - D. *A bicuspid aortic valve is commonly present
 - E. A "sail sound" is a common finding on physical examination
578. Which of the following statements regarding prevention of stroke is correct?
- A. Treatment of systolic hypertension does not decrease the risk of stroke in patients older than age 60
 - B. Hypertension should not be a target of secondary prevention after an ischemic stroke because elevated BP is desirable to maintain adequate cerebral perfusion
 - C. *Treatment with HMG-CoA reductase inhibitors reduces the risk of recurrent stroke
 - D. The combination of aspirin and clopidogrel is superior to aspirin alone for prevention of recurrent stroke
 - E. None of the above
579. Which of the following syndromes and cardiac anomalies are incorrectly matched?
- A. Trisomy 21 – atrioventricular canal defects
 - B. Noonan syndrome – pulmonic stenosis
 - C. Holt-Oram syndrome – atrial septal defects
 - D. Marfan syndrome – mitral valve prolapse
 - E. *Williams syndrome – ventricular septal defects
580. Which of these statements is true regarding renin-angiotensin system-blocking agents?
- A. Less effective when combined with a diuretic than when used alone
 - B. More effective in patients of African descent than in white patients
 - C. *Preserve kidney function in addition to lowering blood pressure
 - D. Are the first-line antihypertensive medications in pregnancy
 - E. All of the above
581. Which of these statements is true regarding renin-angiotensin system-blocking agents?
- A. Less effective when combined with a diuretic than when used alone
 - B. More effective in patients of African descent than in white patients
 - C. *Preserve kidney function in addition to lowering blood pressure
 - D. Are the first-line antihypertensive medications in pregnancy
 - E. All of the above
582. Which parameter of arterial blood pressure (BP) yields the best prognostic information in patients at risk for cardiovascular disease?
- A. Systolic BP
 - B. Diastolic BP
 - C. Mean arterial BP
 - D. *Pulse pressure

- E. None
583. Williams syndrome is commonly associated with...
- A. Supravalvular aortic stenosis
 - B. *Supravalvular pulmonic stenosis
 - C. Cleft mitral valve
 - D. Anomalous pulmonary venous drainage
 - E. Persistent left superior vena cava
584. One of the causes of sick sinus syndrome is:
- A. *Idiopathic degenerative fibrotic infiltration
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
585. One of the causes of sick sinus syndrome is:
- A. *Amyloidosis
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
586. One of the causes of sick sinus syndrome is:
- A. *Arteritis
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
587. One of the causes of sick sinus syndrome is:
- A. *Cardiomyopathies
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
588. One of the causes of sick sinus syndrome is:
- A. *Collagen vascular disease
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
589. One of the causes of sick sinus syndrome is:
- A. *Diphtheria
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
590. One of the causes of sick sinus syndrome is:
- A. *Familial sinoatrial node disorders
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
591. One of the causes of sick sinus syndrome is:
- A. *Fatty replacement
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia

- E. Parkinson's disease
592. One of the causes of sick sinus syndrome is:
A. *Friedreich's ataxia
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
593. One of the causes of sick sinus syndrome is:
A. *Hemochromatosis
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
594. One of the causes of sick sinus syndrome is:
A. *Myocardial infarction
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
595. One of the causes of sick sinus syndrome is:
A. *Muscular dystrophy
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
596. One of the causes of sick sinus syndrome is:
A. *Myocarditis
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
597. One of the causes of sick sinus syndrome is:
A. *Pericarditis
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
598. One of the causes of sick sinus syndrome is:
A. *Rheumatic fever
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
599. One of the causes of sick sinus syndrome is:
A. *Sarcoidosis
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
600. One of the causes of sick sinus syndrome is:
A. *Hyperkalemia
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia

- E. Parkinson's disease
601. One of the causes of sick sinus syndrome is:
A. *Digitalis intake
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
602. One of the causes of sick sinus syndrome is:
A. *Calcium channel blockers intake
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
603. One of the causes of sick sinus syndrome is:
A. *Beta blockers intake
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
604. One of the causes of sick sinus syndrome is:
A. *Sympatholytic agents intake
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
605. One of the causes of sick sinus syndrome is:
A. *Antiarrhythmics intake
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
606. One of the causes of sick sinus syndrome is:
A. *Sinoatrial nodal artery deficiency intake
B. Ulcerative diseases of stomach and duodenum
C. Aortic aneurysm
D. Cardiac achalasia
E. Parkinson's disease
607. For the clinical manifestation of sick sinus syndrome is typical:
A. *Dementia
B. Exophthalmus
C. Weight loss
D. Pain in the epigastric region
E. Profuse vomiting
608. For the clinical manifestation of sick sinus syndrome is typical:
A. *Lethargy
B. Exophthalmus
C. Weight loss
D. Pain in the epigastric region
E. Profuse vomiting
609. For the clinical manifestation of sick sinus syndrome is typical:
A. *Nocturnal wakefulness
B. Exophthalmus
C. Weight loss
D. Pain in the epigastric region

- E. Profuse vomiting
610. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Lightheadedness
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
611. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Syncope or pre-syncope
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
612. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Angina pectoris
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
613. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Arterial thromboemboli
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
614. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Cerebrovascular accident
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
615. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Congestive heart failure
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
616. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Dementia
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
617. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Palpitations
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
618. For the clinical manifestation of sick sinus syndrome is typical:
- A. *Dizziness
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region

- E. Profuse vomiting
619. What clinical sign is not typical for sick sinus syndrome:
- A. *Profuse vomiting
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
620. What clinical sign is not typical for sick sinus syndrome:
- A. *Regurgitation
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
621. What clinical sign is not typical for sick sinus syndrome:
- A. *Melena
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
622. What clinical sign is not typical for sick sinus syndrome:
- A. *Defans of abdominal wall
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
623. What clinical sign is not typical for sick sinus syndrome:
- A. *Wooden abdomen
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
624. What clinical sign is not typical for sick sinus syndrome:
- A. *Pneumoperitoneum
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
625. What clinical sign is not typical for sick sinus syndrome:
- A. *Vomiting by bile
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
626. What clinical sign is not typical for sick sinus syndrome:
- A. *Exophthalmus
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure
 - E. Palpitations
627. What clinical sign is not typical for sick sinus syndrome:
- A. *Pain in epigastric region
 - B. Nocturnal wakefulness
 - C. Syncope or pre-syncope
 - D. Congestive heart failure

- E. Palpitations
628. What clinical sign is not typical for sick sinus syndrome:
A. *Pain in right iliac region
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
629. What clinical sign is not typical for sick sinus syndrome:
A. *Retention of stool and gases
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
630. What clinical sign is not typical for sick sinus syndrome:
A. *Dysphagia
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
631. What clinical sign is not typical for sick sinus syndrome:
A. *Intermittent claudication
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
632. What clinical sign is not typical for sick sinus syndrome:
A. *Leriche's syndrome
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
633. What clinical sign is not typical for sick sinus syndrome:
A. *Trophic ulcers on foot
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
634. What clinical sign is not typical for sick sinus syndrome:
A. *Toes gangrene
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
635. What clinical sign is not typical for sick sinus syndrome:
A. *Muscular contracture of limbs
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
636. What clinical sign is not typical for sick sinus syndrome:
A. *Cough with blood sputum
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure

- E. Palpitations
637. What clinical sign is not typical for sick sinus syndrome:
A. *Signs of hypothyroidism
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
638. What clinical sign is not typical for sick sinus syndrome:
A. *Signs of hyperthyroidism
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
639. What clinical sign is not typical for sick sinus syndrome:
A. *Upper lid lag when the patient looks downward
B. Nocturnal wakefulness
C. Syncope or pre-syncope
D. Congestive heart failure
E. Palpitations
640. To supraventricular bradyarrhythmia in sick sinus syndrome belongs:
A. *Sinus bradycardia
B. Paroxysmal supraventricular tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Atrial tachycardia
641. To supraventricular bradyarrhythmia in sick sinus syndrome belongs:
A. *Sinus arrest with or without junctional escape
B. Paroxysmal supraventricular tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Atrial tachycardia
642. To supraventricular bradyarrhythmia in sick sinus syndrome belongs:
A. *Sinoatrial exit block
B. Paroxysmal supraventricular tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Atrial tachycardia
643. To supraventricular bradyarrhythmia in sick sinus syndrome belongs:
A. *Ectopic atrial bradycardia
B. Paroxysmal supraventricular tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Atrial tachycardia
644. To supraventricular bradyarrhythmia in sick sinus syndrome belongs:
A. *Atrial fibrillation with slow ventricular response
B. Paroxysmal supraventricular tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Atrial tachycardia
645. To supraventricular tachyarrhythmia in sick sinus syndrome belongs:
A. *Paroxysmal supraventricular tachycardia
B. Sinus bradycardia
C. Sinus arrest with or without junctional escape
D. Sinoatrial exit block

- E. Ectopic atrial bradycardia
646. To supraventricular tachyarrhythmia in sick sinus syndrome belongs:
- A. *Atrial flutter
 - B. Sinus bradycardia
 - C. Sinus arrest with or without junctional escape
 - D. Sinoatrial exit block
 - E. Ectopic atrial bradycardia
647. To supraventricular tachyarrhythmia in sick sinus syndrome belongs:
- A. *Atrial fibrillation
 - B. Sinus bradycardia
 - C. Sinus arrest with or without junctional escape
 - D. Sinoatrial exit block
 - E. Ectopic atrial bradycardia
648. To supraventricular tachyarrhythmia in sick sinus syndrome belongs:
- A. *Atrial tachycardia
 - B. Sinus bradycardia
 - C. Sinus arrest with or without junctional escape
 - D. Sinoatrial exit block
 - E. Ectopic atrial bradycardia
649. Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
650. Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented belongs to the such class for permanent pacemaker implantation:
- A. *IIa
 - B. I
 - C. IIb
 - D. III
 - E. IV
651. Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake belongs to the such class for permanent pacemaker implantation:
- A. *IIb
 - B. IIa
 - C. I
 - D. III
 - E. IV
652. Sick sinus syndrome in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate less than 40 bpm) is a consequence of long-term drug treatment belongs to the such class for permanent pacemaker implantation:
- A. *III
 - B. IIb
 - C. IIa
 - D. I
 - E. IV
653. Sick sinus syndrome with symptomatic bradycardia caused by nonessential drug therapy belongs to the such class for permanent pacemaker implantation:
- A. *III
 - B. IIb
 - C. IIa

- D. I
 - E. IV
654. Sick sinus syndrome in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate belongs to the such class for permanent pacemaker implantation:
- A. *III
 - B. IIb
 - C. IIa
 - D. I
 - E. IV
655. To the class I as the indications for permanent pacemaker implantation in sick sinus syndrome belong:
- A. *Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - B. Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented
 - C. Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake
 - D. Sick sinus syndrome with symptomatic bradycardia caused by nonessential drug therapy
 - E. Sick sinus syndrome in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate
656. To the class IIa as the indications for permanent pacemaker implantation in sick sinus syndrome belong:
- A. *Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented
 - B. Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - C. Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake
 - D. Sick sinus syndrome with symptomatic bradycardia caused by nonessential drug therapy
 - E. Sick sinus syndrome in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate
657. To the class IIb as the indications for permanent pacemaker implantation in sick sinus syndrome belong:
- A. *Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake
 - B. Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - C. Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented
 - D. Sick sinus syndrome with symptomatic bradycardia caused by nonessential drug therapy
 - E. Sick sinus syndrome in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate
658. To the class III as the indications for permanent pacemaker implantation in sick sinus syndrome belong:
- A. *Sick sinus syndrome with symptomatic bradycardia caused by nonessential drug therapy
 - B. Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - C. Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented
 - D. Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake
 - E. Sick sinus syndrome with symptomatic tachycardia

659. To the class III as the indications for permanent pacemaker implantation in sick sinus syndrome belong:
- A. *Sick sinus syndrome in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate
 - B. Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake
 - C. Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - D. Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented
 - E. Sick sinus syndrome with symptomatic tachycardia
660. To the class III as the indications for permanent pacemaker implantation in sick sinus syndrome belong:
- A. *Sick sinus syndrome in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate less than 40 bpm) is a consequence of long-term drug treatment
 - B. Sick sinus syndrome in minimally symptomatic patients with chronic heart rate less than 30 bpm while awake
 - C. Sick sinus syndrome with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
 - D. Sick sinus syndrome occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association has not been documented
 - E. Sick sinus syndrome with symptomatic tachycardia
661. One of the causes of AV-block of the I degree is:
- A. *Professional sport
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
662. One of the causes of AV-block is:
- A. *Myocardial infarction
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
663. One of the causes of AV-block is:
- A. *Angina pectoris
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
664. One of the causes of AV-block is:
- A. *Viral myocarditis
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
665. One of the causes of AV-block is:
- A. *Rheumatic fever
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
666. One of the causes of AV-block is:

- A. *Infectious mononucleosis
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
667. One of the causes of AV-block is:
- A. *Sarcoidosis
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
668. One of the causes of AV-block is:
- A. *Amyloidosis
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
669. One of the causes of AV-block is:
- A. *Malignant tumours
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
670. One of the causes of AV-block is:
- A. *Beta blockers intake
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
671. One of the causes of AV-block is:
- A. *Calcium channel blockers intake
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
672. One of the causes of AV-block is:
- A. *Digitalis intake
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
673. One of the causes of AV-block is:
- A. *Leva's disease
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
674. One of the causes of AV-block is:
- A. *Lenegra's disease
 - B. Ulcerative diseases of stomach and duodenum
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Parkinson's disease
675. Leva's disease is:

- A. *Idiopathic heart stroma sclerosis
 - B. Idiopathic conducting system sclerosis
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Sarcoidosis
676. Lenegra's disease is:
- A. *Idiopathic conducting system sclerosis
 - B. Idiopathic heart stroma sclerosis
 - C. Aortic aneurysm
 - D. Cardiac achalasia
 - E. Sarcoidosis
677. Idiopathic conducting system sclerosis is:
- A. *Lenegra's disease
 - B. Leva's disease
 - C. Parkinson's disease
 - D. Mobitz disease
 - E. Wenckebach disease
678. First-degree AV block is characterized by:
- A. *Prolongation of the PR interval on the ECG more than 0.20 s
 - B. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
679. Mobitz I second-degree AV block is characterized by:
- A. *Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - B. Prolongation of the PR interval on the ECG more than 0.20 s
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
680. Wenckebach block is characterized by:
- A. *Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - B. Prolongation of the PR interval on the ECG more than 0.20 s
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
681. Mobitz II second-degree AV block is characterized by:
- A. *Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - B. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - C. Prolongation of the PR interval on the ECG more than 0.20 s
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
682. Third-degree AV block is characterized by:

- A. *No supraventricular impulses are conducted to the ventricles
 - B. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - C. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - D. Prolongation of the PR interval on the ECG more than 0.20 s
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
683. AV dissociation is characterized by:
- A. *Rhythm identified by atrial and ventricular activation occurring from different pacemakers
 - B. No supraventricular impulses are conducted to the ventricles
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - E. Prolongation of the PR interval on the ECG more than 0.20 s
684. Prolongation of the PR interval on the ECG more than 0.20 s is:
- A. *First-degree AV block
 - B. Mobitz I second-degree AV block
 - C. Mobitz II second-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation
685. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause is:
- A. *Mobitz I second-degree AV block
 - B. First-degree AV block
 - C. Mobitz II second-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation
686. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause is:
- A. *Wenckebach AV block
 - B. First-degree AV block
 - C. Mobitz II second-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation
687. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles is:
- A. *Mobitz II second-degree AV block
 - B. Mobitz I second-degree AV block
 - C. First-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation
688. No supraventricular impulses are conducted to the ventricles is:
- A. *Third-degree AV block
 - B. Mobitz II second-degree AV block
 - C. Mobitz I second-degree AV block
 - D. First-degree AV block
 - E. AV dissociation
689. Rhythm identified by atrial and ventricular activation occurring from different pacemakers is:
- A. *AV dissociation
 - B. Third-degree AV block

- C. Mobitz II second-degree AV block
 - D. Mobitz I second-degree AV block
 - E. First-degree AV block
690. First-degree AV block is characterized by:
- A. *Slowed AV conduction
 - B. Partial conduction of supraventricular impulses to the ventricles
 - C. Complete failure of AV conduction
 - D. Independent impulses from atriums and ventricles
 - E. Paroxysmal tachycardia
691. Second-degree AV block is characterized by:
- A. *Partial conduction of supraventricular impulses to the ventricles
 - B. Slowed AV conduction
 - C. Complete failure of AV conduction
 - D. Independent impulses from atriums and ventricles
 - E. Paroxysmal tachycardia
692. Third-degree AV block is characterized by:
- A. *Complete failure of AV conduction
 - B. Partial conduction of supraventricular impulses to the ventricles
 - C. Slowed AV conduction
 - D. Independent impulses from atriums and ventricles
 - E. Paroxysmal tachycardia
693. AV dissociation is characterized by:
- A. *Independent impulses from atriums and ventricles
 - B. Partial conduction of supraventricular impulses to the ventricles
 - C. Slowed AV conduction
 - D. Complete failure of AV conduction
 - E. Paroxysmal tachycardia
694. Slowed AV conduction is typical for:
- A. *First-degree AV block
 - B. Second-degree AV block
 - C. Third-degree AV block
 - D. AV dissociation
 - E. Paroxysmal tachycardia
695. Partial conduction of supraventricular impulses to the ventricles is typical for:
- A. *Second-degree AV block
 - B. First-degree AV block
 - C. Third-degree AV block
 - D. AV dissociation
 - E. Paroxysmal tachycardia
696. Complete failure of AV conduction is typical for:
- A. *Third-degree AV block
 - B. First-degree AV block
 - C. Second-degree AV block
 - D. AV dissociation
 - E. Paroxysmal tachycardia
697. Independent impulses from atriums and ventricles is typical for:
- A. *AV dissociation
 - B. Third-degree AV block
 - C. First-degree AV block
 - D. Second-degree AV block
 - E. Paroxysmal tachycardia
698. No supraventricular impulses are conducted to the ventricles is:
- A. *Third-degree AV block
 - B. Mobitz II second-degree AV block

- C. Mobitz I second-degree AV block
 - D. First-degree AV block
 - E. AV dissociation
699. ECG changes of the first-degree AV block are characterized by:
- A. *Prolongation of the PR interval on the ECG more than 0.20 s
 - B. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
700. ECG changes of the Mobitz I second-degree AV block are characterized by:
- A. *Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - B. Prolongation of the PR interval on the ECG more than 0.20 s
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
701. ECG changes of the Mobitz II second-degree AV block are characterized by:
- A. *Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - B. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - C. Prolongation of the PR interval on the ECG more than 0.20 s
 - D. No supraventricular impulses are conducted to the ventricles
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
702. ECG changes of the third-degree AV block are characterized by:
- A. *No supraventricular impulses are conducted to the ventricles
 - B. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - C. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - D. Prolongation of the PR interval on the ECG more than 0.20 s
 - E. Rhythm identified by atrial and ventricular activation occurring from different pacemakers
703. ECG changes of the AV dissociation are characterized by:
- A. *Rhythm identified by atrial and ventricular activation occurring from different pacemakers
 - B. No supraventricular impulses are conducted to the ventricles
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause
 - E. Prolongation of the PR interval on the ECG more than 0.20 s
704. Prolongation of the PR interval more than 0.20 s is the ECG sign of:
- A. *First-degree AV block
 - B. Mobitz I second-degree AV block
 - C. Mobitz II second-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation

705. Progressive prolongation of the PR interval with the subsequent occurrence of a single nonconducted P wave that results in a pause is the ECG sign of:
- A. *Mobitz I second-degree AV block
 - B. First-degree AV block
 - C. Mobitz II second-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation
706. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles is the ECG sign of:
- A. *Mobitz II second-degree AV block
 - B. Mobitz I second-degree AV block
 - C. First-degree AV block
 - D. Third-degree AV block
 - E. AV dissociation
707. No supraventricular impulses are conducted to the ventricles is the ECG sign of:
- A. *Third-degree AV block
 - B. Mobitz II second-degree AV block
 - C. Mobitz I second-degree AV block
 - D. First-degree AV block
 - E. AV dissociation
708. Rhythm identified by atrial and ventricular activation occurring from different pacemakers is the ECG sign of:
- A. *AV dissociation
 - B. Third-degree AV block
 - C. Mobitz II second-degree AV block
 - D. Mobitz I second-degree AV block
 - E. First-degree AV block
709. For the clinical manifestation of third-degree AV block is typical:
- A. *Dizziness
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
710. For the clinical manifestation of third-degree AV block is typical:
- A. *Lightheadedness
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
711. For the clinical manifestation of third-degree AV block is typical:
- A. *Syncope or pre-syncope
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
712. For the clinical manifestation of third-degree AV block is typical:
- A. *Morgagni-Adams-Stokes syndrome
 - B. Exophthalmus
 - C. Weight loss
 - D. Pain in the epigastric region
 - E. Profuse vomiting
713. For the clinical manifestation of first-degree AV block is typical:
- A. *Asymptomatic course
 - B. Morgagni-Adams-Stokes syndrome

- C. Syncope or pre-syncope
 - D. Lightheadedness
 - E. Dizziness
714. For the clinical manifestation of second-degree AV block is typical:
- A. *Asymptomatic course
 - B. Morgagni-Adams-Stokes syndrome
 - C. Syncope or pre-syncope
 - D. Lightheadedness
 - E. Dizziness
715. Syncope or pre-syncope syndrome in AV block is called:
- A. *Morgagni-Adams-Stokes syndrome
 - B. Leriche's syndrome
 - C. Mobitz syndrome
 - D. Wenckebach syndrome
 - E. Leva's disease
716. Morgagni-Adams-Stokes syndrome is
- A. *Syncope or pre-syncope syndrome in AV block
 - B. Prolongation of the PR interval on the ECG more than 0.20 s
 - C. Constant PR interval followed by sudden failure of a P wave to be conducted to the ventricles
 - D. Lightheadedness in AV block
 - E. Association of AV block with myocardial infarction
717. Third-degree AV block at any anatomic level associated with bradycardia and symptoms presumed to be due to AV block belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
718. Third-degree AV block at any anatomic level associated with arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
719. Third-degree AV block at any anatomic level associated with documented periods of asystole 3.0 seconds belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
720. Third-degree AV block at any anatomic level associated with documented any escape rate <40 beats per minute (bpm) in awake, symptom-free patients belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV

721. Third-degree AV block at any anatomic level after catheter ablation of the AV junction belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
722. Postoperative third-degree AV block at any anatomic level that is not expected to resolve belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
723. Third-degree AV block at any anatomic level associated with myotonic muscular dystrophy belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
724. Third-degree AV block at any anatomic level associated with Erb's dystrophy (limb-girdle dystrophy) belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
725. Second-degree AV block regardless of type or site of block, with associated symptomatic bradycardia belongs to the such class for permanent pacemaker implantation:
- A. *I
 - B. IIa
 - C. IIb
 - D. III
 - E. IV
726. Asymptomatic third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster belongs to the such class for permanent pacemaker implantation:
- A. *IIa
 - B. I
 - C. IIb
 - D. III
 - E. IV
727. Asymptomatic type II second-degree AV block belongs to the such class for permanent pacemaker implantation:
- A. *IIa
 - B. I
 - C. IIb
 - D. III
 - E. IV
728. Asymptomatic type I second-degree AV block at intra- or infra-His levels found incidentally at electrophysiological study for other indications belongs to the such class for permanent pacemaker implantation:
- A. *IIa
 - B. I

- C. IIb
 - D. III
 - E. IV
729. First-degree AV block with symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary AV pacing belongs to the such class for permanent pacemaker implantation:
- A. *IIa
 - B. I
 - C. IIb
 - D. III
 - E. IV
730. Marked first-degree AV block (>0.30 second) in patients with LV dysfunction and symptoms of congestive heart failure in whom a shorter AV interval results in hemodynamic improvement belongs to the such class for permanent pacemaker implantation:
- A. *IIb
 - B. IIa
 - C. I
 - D. III
 - E. IV
731. Asymptomatic first-degree AV block belongs to the such class for permanent pacemaker implantation:
- A. *III
 - B. IIb
 - C. IIa
 - D. I
 - E. IV
732. Asymptomatic type I second-degree AV block at the supra-His (AV node) level belongs to the such class for permanent pacemaker implantation:
- A. *III
 - B. IIb
 - C. IIa
 - D. I
 - E. IV
733. AV block which expected to resolve and unlikely to recur (eg, drug toxicity, Lyme disease) belongs to the such class for permanent pacemaker implantation:
- A. *III
 - B. IIb
 - C. IIa
 - D. I
 - E. IV
734. A main direct danger for a patient with acute bleeding is
- A. deficit of hemoglobin
 - B. *Hypovolemia
 - C. Hypoproteinemia
 - D. coagulopathy
 - E. deficit of fibrinogen
735. A most important for the life of patient through possible transformation in ventricles fibrillation is presented by arrhythmia
- A. ventricle premature beats more than 20 in a minute
 - B. *ventricle tachycardia
 - C. arrhythmias, that unite with lengthening of intraventricular conductivity blockade of the left leg of bunch of Giss
 - D. arrhythmia
 - E. all answers are correct

736. At a patient with concomitant cardiac insufficiency acutely the expressed anemia. Which from offered remedies for transfusion prevails?
- *erithromass
 - fresh blood
 - frozen plasma
 - there are all wrong answers
 - all answers are faithful
737. Endotracheally it is possible to enter the following drugs, except for:
- noradrenalin
 - adrenalin
 - lidocain
 - atropine
 - *oxybutirat
738. A complete atrioventricular block is characterised
- by absence of conducting of impulses from an autriums to ventricles
 - by a idioventricoular rhythm
 - by the attacks Morgani - Adams - Stocs
 - all answers are faithful
 - *all answers are right
739. A doctor decided to perform the defibrillation to patient with arrhythmia, using a synchronizer. The digit of defibrillator must be synchronized:
- *with wave Q
 - with the descending phase of R
 - by an T
 - consideration of phase of ECG-complex not important
 - there is no right answer
740. A leading symptom for the diagnosis of stop of circulation of blood is:
- wide pupils, that are irresponsive on light
 - absence of consciousness
 - absence of breathing
 - *absence of pulse on a carotid
 - cyanosis
741. At anafilactic shock obligatory medicine are:
- *providing of permeability of respiratory tracts and adequate
 - imobilisation extremities
 - infousion therapy
 - analgesia
 - injection of vazopresors
742. At development of signs of toxic action of digoxinum medical treatment includes intravenous injection of
- verapamilum
 - *lidocainum
 - calcium chloride
 - all answers are wrong
 - faithful all answers
743. At which types of arrhythmia intravenous injection of potassium is effective?
- Ventricular tahycardia
 - Nodular tahycardia
 - Ventricular extrasystolia
 - Supraventricular arrhythmia
 - *At all of indicated types of arrhythmia
744. Cardiotoxic properties of hypercalciemia is taken off by application:
- Adrenalin
 - Coffeinum

- C. Ephedrine
 - D. *Molar solution of lactat natrium
 - E. 10 % solution of glucose
745. Cardiotoxicity action of hypocalcemia treated by application:
- A. adrenalin
 - B. ephedrine
 - C. *preparations of calcium
 - D. a 10% solution of glucose
 - E. corticosteroid
746. For the hypovolemia is not typical:
- A. reduction of volume of circulatory blood
 - B. decline of BP, tachycardia
 - C. reduction of shock volume and cardiac troop landing
 - D. *rise of CVP
 - E. decline of pressure of filling of the left ventricle
747. For the prophylaxis of carcinogenic shock it does not use at the acute heart attack follow
- A. beta-adrenoblocers
 - B. anaesthetizing, sedation
 - C. inhalation O₂
 - D. beta-adrenostimulators
 - E. *all answers are not complete
748. Hypotension at anaphylactic shock develops as a result of
- A. *increase of permeability of vessels and loss of volume of intravascular liquid
 - B. losses of likable tone
 - C. free prostaglandin
 - D. bradycardia
 - E. all are right
749. In the case of tamponade of heart is not observed
- A. rise of central vein pressure
 - B. *noise of systole
 - C. cyanosis
 - D. paradoxical pulse
 - E. rise of pulmonary capillary pressure
750. In the origin of hyperdynamic type of circulation of blood at patients with a sepsis, acute bleeding the following reactions take part:
- A. acute tamponade of heart
 - B. shunting of circulation
 - C. *violations of microcirculation, disorders of tissue exchange and accumulations of vasoactive metabolities
 - D. faithful all answers
 - E. there is no right answer
751. Intravenous injection of morphine at the carcinogenic edema of lungs can be attained the following positive effects:
- A. increase of irritating of myocardium
 - B. decline of retroactive power of myocardium
 - C. spasm of coronal vessels
 - D. *faithful all answers
 - E. right all answers
752. Lengthening of interval P-Q more than 0,2 sec is observed at:
- A. Fibrillation of auricles
 - B. Sine bradycardia
 - C. Alternuous pulse
 - D. *AV block 1
 - E. Bigemina

753. Medical treatment of anaphylactic shock includes first time the use of
- *adrenalin
 - antihistaminic preparations
 - neuroplegic
 - All answers are wrong
 - faithful all answers
754. Properties of blood reology are fixed:
- By viscosity of blood
 - By physical and chemical stability of suspension of uniform elements
 - By the state of vessels wall
 - *All answers are correct
 - There is no right answer
755. Signs of local myocarditis recovering are represented best of all by the changes of wave
- P
 - Q
 - T
 - *R
 - S
756. Stretched neck veins in standing position are observed at
- tamponade of heart
 - tense pneumothorax
 - to pulmonary embolism
 - *faithful all answers
 - all answers are wrong
757. The acute carcinogenic edema of lungs is conditioned most of all:
- by the rise of hydrostatical pressure in pulmonary capillaries
 - by the promoted pulmonary capillary permeability
 - *by insufficiency of the left ventricle
 - faithful all answers
 - All answers are wrong
758. The basic sign of heart death is:
- agonal complexes
 - mechanical asistolia with saving of electric systole
 - electric asistolia during 30 min, not looking on cordial - pulmonary reanimation and proper medical therapy
 - *isoelectric line on ECG during 15 min
 - all answers are faithful
759. The dosage of bicarbonate of sodium in case of stopping of heart makes:
- the first injection 1 mmol/Kg
 - the first injection 2 mmol/Kg
 - the repeated injections 0.5 meq/cg every 10 mines
 - *approximately 100 ml for each min of clinical death
 - faithful all answers
760. The forcing function of heart is supported in case:
- At wrong situation of electrodes
 - At inadequate ventilation of lungs
 - acidosis
 - When the previous massage of heart was uneffective
 - *All answers are right
761. The injection of the following medicines may prevent the attack of paroxysmal tahycardia, except of :
- *Isoproterenol
 - Mezatonum
 - Digitalis and hinidinum

- D. Metocsamium
 - E. Morphynum
762. The most expressed positive inotropic effect at cardiogenic shock is observed at injection of:
- A. noradrenalinum
 - B. *dopaminum
 - C. digocsinum
 - D. isadrinum
 - E. ephedrinum
763. The most frequent reason of embolism is:
- A. Trauma
 - B. Cardial insufficiency
 - C. Aneurism
 - D. Atherosclerosis
 - E. *Fibrillation of atrium
764. The most important ECG-sign of subendocardial myocardial ischemia is:
- A. *rise of segment S-T higher to the isoline line more than on 2 mm
 - B. decline of segment S-T below to the isoline line more than on 2 mm
 - C. inversion of T
 - D. there are all wrong answers
 - E. all answers are faithful
765. The permission to use lidocainum in the process of cardiac-pulmonary reanimation are:
- A. *fibrillation of ventricles
 - B. electro mechanics dissociation
 - C. fibrillation of atriums
 - D. right all answers
 - E. wrong all answers
766. The satisfactory oxygen capacity of blood is provided by hematocrit, not below
- A. 20-25%
 - B. *30%
 - C. 35%
 - D. 40%
 - E. 45%
767. To arrhythmias, which are accompanied by acute cardiac insufficiency, belong:
- A. fibrillation of ventricles
 - B. complete atrioventricular block
 - C. high-frequency trembling of atriums
 - D. faithful all answers
 - E. *right all answers
768. To medicines which have most vasodilatational and lowering postload, belong:
- A. nitrates
 - B. *ganglioblocers
 - C. nitroprousid
 - D. morfin
 - E. faithful all answers
769. What medicine is used for medical treatment of atrio-ventricular block III?
- A. Lidocain
 - B. Inderal
 - C. Novocainamid
 - D. *Isuprel
 - E. There is no right answer
770. What must be applied from following during haemotransfusion for saving of blood properties, that take part in coagulation?
- A. Warmed-up blood
 - B. Calcium

- C. Oxygen
 - D. Blood of two-week remoteness
 - E. *Fresh blood
771. What type of hypoxia changes greatly oxygen saturation of arterial blood ?
- A. Circulatory
 - B. Anemic
 - C. *Hypoxic
 - D. Tissue
 - E. There is no right answer
772. At medical treatment of paroxysmal tachycardia it is not prescribed to apply
- A. antagonists of beta-adrenoreceptors
 - B. electric cardioversion
 - C. pressure on a carotid sine
 - D. lidocain
 - E. *atropin
773. Characteristic signs for different types of shocks are:
- A. Oppression of consciousness
 - B. *Lowering of arterial pressure
 - C. Lowering of shock index Algovera
 - D. Lowering TSVD
 - E. Oppression of breathing
774. Choose the most optimum therapy of anaphylactic shock
- A. *Adrenalin, polyglucin, prednisolone IV
 - B. Dimedrol, prednisolon, cordiaminum, - IV
 - C. Atropin, pipolphenum, prednisolon, - IV
 - D. Polyhluvin, calcium chloride, hydrocortyson, - IV
 - E. Antihistaminic orally
775. Deep wave Q may be the sign of
- A. MI
 - B. Ischemia of myocardium
 - C. Hypokaliemia
 - D. Hyperpotassiumemia
 - E. *All answers are wrong
776. Deep wave Q may be the sign of
- A. Necrosis of myocardium
 - B. Ischemia of myocardium
 - C. Hypokaliemia
 - D. Hyperpotassiumemia
 - E. *All answers are wrong
777. Electrocardiostimulation is not prescribed at:
- A. to the complete atrioventricular block
 - B. syndrome of weakness of sine knot with the attacks Morgani-Adams-Stocs
 - C. ciliary arrhythmia with rare contraction of ventricles
 - D. *bradycardia, by the caused intoxication by cardiac glucoside
 - E. all answers are faithful
778. For cardiogenic shock is not characteristic:
- A. *Decreasing of central venous pressure
 - B. Stagnation in the small circle of blood
 - C. Low BP
 - D. Diminishing of arteriotony
 - E. Decline of diuresis
779. For treatment of pulmonary edema of cardiac origin the doctor should prescribe
- A. *Narcotic analgetic
 - B. Solution of euphyllinum

- C. Diuretic - MANNITOL
 - D. Diuretic - veroshpiron
 - E. Calcium chloride
780. Heparin behaves to the group;
- A. *Enkefalins
 - B. Endorfins
 - C. Eucasinoids
 - D. A superoxyde anion
 - E. There is no right answer
781. High vein pressure, hypotension and acute circulation insufficiency are observed in the case of:
- A. tense pneumathorax
 - B. embolisms of pulmonary artery
 - C. to vein air embolism
 - D. wrong all answers
 - E. *right all answers
782. Hypovolaemic shock is determined in:
- A. *Reduction of body weight
 - B. By the decline of hemokoncentrative indexes
 - C. By paresis of peripheral vessels
 - D. By hypoglycemia
 - E. By growth of central venous pressure
783. Medical treatment of patient with acute left side ventricular insufficiency includes
- A. ALV at the permanent promoted pressure
 - B. infusion of nitroglycerinum
 - C. inhibitor of phosphodiesterase, furosemide
 - D. all answers are faithful
 - E. *All answers are wrong
784. Medicine of choice at paroxysmal supraventricular tachycardia is:
- A. digoxin
 - B. lidocain
 - C. *isoptin
 - D. novocainamid
 - E. polarized mixture
785. Removing a pain syndrome at the urgent call concerning the acute heart attack of myocardium is possible simpler than all:
- A. *by injection of narcotic and unnarcotic analgetic
 - B. epidural analgesia
 - C. by inhalation of nitrous oxide, xenon with O₂(1:1)
 - D. there is no right answer
 - E. all answers are faithful
786. The depression of ST segment means
- A. Hypoxia of myocardium
 - B. Hypokaliemia
 - C. Hyperpotassiumemia
 - D. *Necrosis of myocardium
 - E. All answers are wrong
787. The depression of T wave may mean
- A. Hypoxia of myocardium
 - B. Fibrillation of atriums
 - C. *Hyperpotassiumemia
 - D. Necrosis of myocardium
 - E. All answers are wrong
788. The depression of T wave means

- A. Hypoxia of myocardium
 - B. Hypokaliemia
 - C. *Hyperpotassiumemia
 - D. Necrosis of myocardium
 - E. All answers are wrong
789. The elevation of ST segment means
- A. *Hypoxia of myocardium
 - B. Hypokaliemia
 - C. Hyperpotassiumemia
 - D. Necrosis of myocardium
 - E. All answers are wrong
790. The insufficiency of left ventricle can be caused :
- A. *by insufficient flow of blood to the heart
 - B. by the overload volume of blood
 - C. by the decline of refractive power of myocardium
 - D. wrong all answers
 - E. faithful all answers
791. The main ECG sign of transmural MI is
- A. Elevation of T wave
 - B. Depression of ST segment
 - C. *Pathological QS wave
 - D. Pathological S wave
 - E. All answers are wrong
792. The most important sign of acute heart attack of myocardium during anesthesia are:
- A. Hypotensia
 - B. *changes of ECG, arrhythmia
 - C. increase of pulse pressure
 - D. All answers are wrong
 - E. All answers are right
793. The stop of heart during asynchronous defibrillation is linked
- A. with direct damaging of myocardium
 - B. with the hit of impulse on the P wave of ECG
 - C. with the hit of impulse on ascending part of T wave
 - D. *with the hit of impulse on descending part of T wave
 - E. all answers are faithful
794. What assertion is incorrect for traumatic shock?
- A. *Sharp decreases of shock index
 - B. A syndrome of low cardiac ejection
 - C. Centralization of circulation of blood develops
 - D. CVP goes down
 - E. Disturbances of coagulation
795. ?What remedy does dilate veins?
- A. *Nitroglicerine
 - B. Analgin
 - C. Strophanthine
 - D. Dopamine
 - E. Anaprilin
796. What remedy does not dilate veins?
- A. Nitrosorbid
 - B. Analgin
 - C. Strophanthine
 - D. *Dopamine
 - E. Anaprilin
797. Which of medicines are most contra-indicated at ventricular tahycardia

- A. Lidocain
 - B. Propranolol
 - C. Novocainamid
 - D. *Isuprel
 - E. Metaraminol
798. Which of the following changes on ECG is typical for hypercaliemia?
- A. fibrillation of ventricles
 - B. fibrillation of auricles
 - C. *High and narrow wave T
 - D. Decline of indent T and appearance of indent V
 - E. Decline of segment S-T
799. You can use for treatment of ischemic heart disease
- A. *Nitrates
 - B. Stabisol
 - C. Manitol
 - D. Arduan
 - E. All answers are wrong
800. You will use for treatment of ischemic heart disease
- A. Sustac
 - B. Stabisol
 - C. Manitol
 - D. Arduan
 - E. *All answers are wrong
801. You will use for treatment of ischemic heart disease
- A. *Nitrong
 - B. Stabisol
 - C. Manitol
 - D. Arduan
 - E. All answers are wrong
802. What is the endemic goiter characterized by?
- A. *Goiter which occurs in biogeochemical regions with iodine deficiency in environment
 - B. Goiter which occurs in unendemic regions
 - C. Goiter lesion of both lobes
 - D. Goiter of atypical localization
 - E. Goiter with changed function
803. What is the sporadic goiter characterized by?
- A. *Goiter which occurs in unendemic regions
 - B. Goiter which occurs in biogeochemical regions with iodine deficiency in environment
 - C. Goiter lesion of both lobes
 - D. Goiter of atypical localization
 - E. Goiter with changed function
804. Goiter which occurs in biogeochemical regions with iodine deficiency in environment is called:
- A. *Endemic goiter
 - B. Sporadic goiter
 - C. Thyrotoxicosis
 - D. Myxedema
 - E. Atypical goiter
805. Goiter which occurs in unendemic regions is called:
- A. *Sporadic goiter
 - B. Endemic goiter
 - C. Thyrotoxicosis
 - D. Myxedema
 - E. Atypical goiter

806. What is the 0 degree of goiter?
A. *The thyroid gland is not palpated;
B. The isthmus of the gland is noticeable during swallowing and could be palpated;
C. Entire gland is noticeable during swallowing and could be palpated;
D. The enlargement of gland results in evident thickening of neck ("a thick neck");
E. The gland considerably enlarged, and sharply deforms neck;
807. What is the I degree of goiter?
A. *The isthmus of the gland is noticeable during swallowing and could be palpated;
B. The thyroid gland is not palpated;
C. Entire gland is noticeable during swallowing and could be palpated;
D. The enlargement of gland results in evident thickening of neck ("a thick neck");
E. The gland considerably enlarged, and sharply deforms neck;
808. What is the II degree of goiter?
A. *Entire gland is noticeable during swallowing and could be palpated;
B. The thyroid gland is not palpated;
C. The isthmus of the gland is noticeable during swallowing and could be palpated;
D. The enlargement of gland results in evident thickening of neck ("a thick neck");
E. The gland considerably enlarged, and sharply deforms neck;
809. What is the III degree of goiter?
A. *The enlargement of gland results in evident thickening of neck ("a thick neck");
B. The thyroid gland is not palpated;
C. The isthmus of the gland is noticeable during swallowing and could be palpated;
D. Entire gland is noticeable during swallowing and could be palpated;
E. The gland considerably enlarged, and sharply deforms neck;
810. What is the IV degree of goiter?
A. *The gland considerably enlarged, and sharply deforms neck;
B. The enlargement reaches excessive size (goiter of major sizes)
C. The isthmus of the gland is noticeable during swallowing and could be palpated;
D. Entire gland is noticeable during swallowing and could be palpated;
E. The enlargement of gland results in evident thickening of neck ("a thick neck");
811. What is the IV degree of goiter?
A. *The enlargement reaches excessive size (goiter of major sizes)
B. The gland considerably enlarged, and sharply deforms neck;
C. The isthmus of the gland is noticeable during swallowing and could be palpated;
D. Entire gland is noticeable during swallowing and could be palpated;
E. The enlargement of gland results in evident thickening of neck ("a thick neck");
812. What is the degree of goiter when the thyroid gland is not palpated?
A. *0
B. II
C. I
D. III
E. IV
813. What is the degree of goiter when the isthmus of the gland is noticeable during swallowing and could be palpated?
A. *I
B. V
C. II
D. III
E. IV
814. What is the degree of goiter when entire gland is noticeable during swallowing and could be palpated?
A. *I
B. II
C. V

- D. III
 - E. IV
815. What is the degree of goiter when the enlargement of gland results in evident thickening of neck ("a thick neck")?
- A. *III
 - B. I
 - C. V
 - D. II
 - E. IV
816. What is the degree of goiter when the gland considerably enlarged, and sharply deforms neck?
- A. *IV
 - B. III
 - C. I
 - D. V
 - E. II
817. What is the degree of goiter when the enlargement reaches excessive size (goiter of major sizes)?
- A. *V
 - B. IV
 - C. III
 - D. I
 - E. II
818. What is the predominant factor which causes the goiter?
- A. *Lack of iodine
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Suprarenal insufficiency
 - D. Rheumatism, endocarditis
 - E. Lack of vitamin C
819. What is the contributing factor which causes the lung abscess?
- A. *Excessive calcium, deficiency of bromine in environment
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Suprarenal insufficiency
 - D. Rheumatism, endocarditis
 - E. Lack of vitamin C
820. What is the contributing factor which causes the lung abscess?
- A. *Lack of cobalt, and zinc
 - B. Increased cholesterol, dyslipoproteinemia
 - C. Suprarenal insufficiency
 - D. Rheumatism, endocarditis
 - E. Lack of vitamin C
821. Goiter which localized on the back of the tongue is called:
- A. *Ectopic goiter
 - B. Aberrant goiter
 - C. Typical
 - D. Presternal
 - E. Retrosternal
822. Intrathoracic localization of goiter is called:
- A. *Ectopic goiter
 - B. Aberrant goiter
 - C. Typical
 - D. Presternal
 - E. Retrosternal
823. The goiter of additional gland is called:

- A. *Aberrant goiter
 - B. Ectopic goiter
 - C. Typical
 - D. Presternal
 - E. Retrosternal
824. Ectopic goiter is:
- A. *Dislocation of the goiter
 - B. The goiter of additional gland
 - C. The goiter with increased function
 - D. The goiter with decreased function
 - E. The goiter with normal function
825. Aberrant goiter is:
- A. *The goiter of additional gland
 - B. Dislocation of the goiter
 - C. The goiter with increased function
 - D. The goiter with decreased function
 - E. The goiter with normal function
826. What complication is characteristic for the goiter?
- A. *Strumitis
 - B. Laringospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
827. What complication is characteristic for the goiter?
- A. *Hemorrhage into the gland
 - B. Laringospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
828. What complication is characteristic for the goiter?
- A. *Malignancy
 - B. Laringospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
829. What complication is characteristic for the goiter?
- A. *Asphyxia
 - B. Laringospasm
 - C. Pneumothorax
 - D. Mediastinal emphysema
 - E. Atypical goiter
830. In case of euthyroid goiter the patient mainly complains of:
- A. *Neck deformity
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
831. In case of euthyroid goiter the patient mainly complains of:
- A. *Difficult breathing
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
832. In case of euthyroid goiter the patient mainly complains of:

- A. *Difficult swallowing
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
833. In case of euthyroid goiter the patient mainly complains of:
- A. *Sudden attacks of cough
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
834. For the clinical manifestation of retrosternal is typical:
- A. *Dyspnea
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
835. For the clinical manifestation of retrosternal is typical:
- A. *Hoarseness
 - B. Sleepiness
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
836. For the clinical manifestation of retrosternal is typical:
- A. *Distended veins of neck
 - B. Sleepiness,
 - C. Hypomnesia
 - D. Excessive sweating
 - E. Tremor of arms
837. The neck deformity is a clinical manifestation of the patient with:
- A. *Euthyroid goiter
 - B. Hypothyroidism
 - C. Thyrotoxicosis
 - D. Esophageal achalasia
 - E. Empyema
838. The difficult breathing is a clinical manifestation of the patient with:
- A. *Euthyroid goiter
 - B. Hypothyroidism
 - C. Thyrotoxicosis
 - D. Esophageal achalasia
 - E. Gastric ulcer
839. The difficult swallowing is a clinical manifestation of the patient with:
- A. *Euthyroid goiter
 - B. Hypothyroidism
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Empyema
840. In case of hypothyroid goiter the patient mainly complains of:
- A. *Sleepiness
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
841. In case of hypothyroid goiter the patient mainly complains of:

- A. *General weakness
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
842. In case of hypothyroid goiter the patient mainly complains of:
- A. *Malaise
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
843. In case of hypothyroid goiter the patient mainly complains of:
- A. *Hypomnesia
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
844. In case of hypothyroid goiter the patient mainly complains of:
- A. *Dry skin
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
845. In case of hypothyroid goiter the patient mainly complains of:
- A. *Constipations
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
846. In case of hypothyroid goiter the patient mainly complains of:
- A. *Leg edemas
 - B. Difficult breathing
 - C. Neck deformity
 - D. Excessive sweating
 - E. Tremor of arms
847. The sleepiness is a clinical manifestation of the patient with:
- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
848. The general weakness is a clinical manifestation of the patient with:
- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
849. The malaise is a clinical manifestation of the patient with:
- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
850. The hypomnesia is a clinical manifestation of the patient with:

- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
851. The dry skin is a clinical manifestation of the patient with:
- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
852. The edemas is a clinical manifestation of the patient with:
- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
853. The constipation is a clinical manifestation of the patient with:
- A. *Hypothyroid goiter
 - B. Euthyroid goiter
 - C. Thyrotoxicosis
 - D. Gastric ulcer
 - E. Esophageal diverticulum
854. In case of hyperthyroid goiter the patient mainly complains of:
- A. *Excessive sweating
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
855. In case of hyperthyroid goiter the patient mainly complains of:
- A. *Irritability
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
856. In case of hyperthyroid goiter the patient mainly complains of:
- A. *Heartbeat
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
857. In case of hyperthyroid goiter the patient mainly complains of:
- A. *Tremor of arms
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
858. In case of hyperthyroid goiter the patient mainly complains of:
- A. *Sleeplessness
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
859. In case of hyperthyroid goiter the patient mainly complains of:

- A. *Feeling of fever
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
860. In case of hyperthyroid goiter the patient mainly complains of:
- A. *Loss of weight
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
861. The irritability is a clinical manifestation of the patient with:
- A. *Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
862. The heartbeat is a clinical manifestation of the patient with:
- A. *Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
863. The excessive sweating is a clinical manifestation of the patient with:
- A. *Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
864. The tremor of arms is a clinical manifestation of the patient with:
- A. *Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
865. The feeling of fever is a clinical manifestation of the patient with:
- A. *Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
866. The loss of weight is a clinical manifestation of the patient with:
- A. *Hyperthyroid goiter
 - B. Hypothyroid goiter
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
867. What is the main treatment of noncomplicated goiter?
- A. *Conservative treatment
 - B. Segmentectomy
 - C. Lobectomy
 - D. Thyroidectomy
 - E. Hemithyroidectomy
868. What medicines are used for the treatment of goiter?

- A. *Inorganic iodine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
869. What medicines are used for the treatment of goiter?
- A. *Thyroidine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
870. What medicines are used for the treatment of goiter?
- A. *Thyroxine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
871. What medicines are used for the treatment of goiter?
- A. *Triiodothyronine
 - B. Heparin
 - C. Vasaprostan
 - D. Fenillin
 - E. Omeprasol
872. What is the indication for the operative treatment of the goiter?
- A. *Nodular goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
873. What is the indication for the operative treatment of the goiter?
- A. *Mixed forms of goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
874. What is the indication for the operative treatment of the goiter?
- A. *The sings of neck compression by goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
875. What is the indication for the operative treatment of the goiter?
- A. *The goiter with secondary hyperthyroidism
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
876. What is the indication for the operative treatment of the goiter?
- A. *The goiter with suspicion on malignancy
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
877. What is the indication for the operative treatment of the goiter?

- A. *The goiter of additional thyroid glands
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
878. What is the indication for the operative treatment of the goiter?
- A. *The aberrant goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
879. What is the indication for the operative treatment of the goiter?
- A. *The intrathoracic goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
880. What is the indication for the operative treatment of the goiter?
- A. *The retrosternal ectopy of thyroid gland
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
881. What is the indication for the operative treatment of the goiter?
- A. *The complicated goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Uncomplicated hypothyroid goiter
882. What is the indication for the conservative treatment of the goiter?
- A. *The goiter of I degree
 - B. The retrosternal ectopy of thyroid gland
 - C. The aberrant goiter
 - D. The goiter of IV degree
 - E. The goiter with secondary hyperthyroidism
883. What is the indication for the conservative treatment of the goiter?
- A. *The goiter of II degree
 - B. The retrosternal ectopy of thyroid gland
 - C. The aberrant goiter
 - D. The goiter of IV degree
 - E. The goiter with secondary hyperthyroidism
884. What operation is performed in endemic goiter?
- A. *Subfascial resection of thyroid gland
 - B. Segmentectomy
 - C. Lobectomy
 - D. Thyroidectomy
 - E. Hemithyroidectomy
885. Diffuse goiter with hyperthyroidism is called:
- A. *Grave's disease
 - B. Hashimoto disease
 - C. De Kerven disease
 - D. Riedel's disease
 - E. Raynaud's disease
886. Diffuse goiter with hyperthyroidism is called:

- A. *Basedow's disease
 - B. Hashimoto disease
 - C. De Kerven disease
 - D. Riedel's disease
 - E. Raynaud's disease
887. Basedow's disease is:
- A. *Thyrotoxicosis
 - B. Wooden thyroiditis
 - C. Purulent thyroiditis
 - D. Autoimmune thyroiditis
 - E. Mixedema
888. Grave's disease is:
- A. *Thyrotoxicosis
 - B. Wooden thyroiditis
 - C. Purulent thyroiditis
 - D. Autoimmune thyroiditis
 - E. Mixedema
889. What is the cause of thyrotoxicosis?
- A. *Autoimmune disturbances
 - B. Atherosclerotic changes
 - C. Calcium metabolism disturbances
 - D. Renal insufficiency
 - E. Pulmonary emphysema
890. What belongs to the I stage of thyrotoxicosis?
- A. *Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - B. Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - C. Thyrotoxic lesion of viscera
 - D. Nonreversible dystrophy of organs and systems
 - E. Asymptomatic course
891. What belongs to the II stage of thyrotoxicosis?
- A. *Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - B. Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - C. Thyrotoxic lesion of viscera
 - D. Nonreversible dystrophy of organs and systems
 - E. Asymptomatic course
892. What belongs to the III stage of thyrotoxicosis?
- A. *Thyrotoxic lesion of viscera
 - B. Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - C. Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - D. Nonreversible dystrophy of organs and systems
 - E. Asymptomatic course
893. What belongs to the IV stage of thyrotoxicosis?
- A. *Nonreversible dystrophy of organs and systems
 - B. Thyrotoxic lesion of viscera
 - C. Marked sings of thyrotoxicosis, the thyroid is noticeably enlarged in size
 - D. Onset of thyrotoxicosis, slight enlargement of thyroid gland
 - E. Asymptomatic course
894. What stage of thyrotoxicosis correlates with the onset of the disease, slight enlargement of thyroid gland?
- A. *I
 - B. II
 - C. III
 - D. IV
 - E. 0

895. What stage of thyrotoxicosis correlates with the marked signs of thyrotoxicosis and noticeably enlarged thyroid?
- A. *II
 - B. I
 - C. III
 - D. IV
 - E. 0
896. What stage of thyrotoxicosis correlates with the thyrotoxic lesion of viscera?
- A. *III
 - B. II
 - C. I
 - D. IV
 - E. 0
897. What stage of thyrotoxicosis correlates with the nonreversible dystrophy of organs and systems?
- A. *IV
 - B. III
 - C. II
 - D. I
 - E. 0
898. What is the I stage of thyrotoxicosis?
- A. *Neurotic
 - B. Neurohormonal
 - C. Visceropathic
 - D. Cachectic
 - E. Asymptomatic
899. What is the II stage of thyrotoxicosis?
- A. *Neurohormonal
 - B. Neurotic
 - C. Visceropathic
 - D. Cachectic
 - E. Asymptomatic
900. What is the III stage of thyrotoxicosis?
- A. *Visceropathic
 - B. Neurohormonal
 - C. Neurotic
 - D. Cachectic
 - E. Asymptomatic
901. What is the IV stage of thyrotoxicosis?
- A. *Cachectic
 - B. Visceropathic
 - C. Neurohormonal
 - D. Neurotic
 - E. Asymptomatic
902. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Excessive sweating
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
903. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Irritability
 - B. Leg edemas
 - C. Hypomnesia

- D. Neck deformity
 - E. Constipation
904. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Heartbeat
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
905. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Tremor of arms
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
906. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Sleeplessness
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
907. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Feeling of fever
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
908. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Loss of weight
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
909. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Palpitation
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
910. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Exophthalmos
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
911. In case of thyrotoxicosis goiter the patient mainly complains of:
- A. *Tremor
 - B. Leg edemas
 - C. Hypomnesia
 - D. Neck deformity
 - E. Constipation
912. The palpitation is a clinical manifestation of the patient with:
- A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter

- D. Gastric ulcer
- E. Esophageal diverticulum
- 913. The palpitation is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 914. The exophthalmos is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 915. The tremor is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 916. The heartbeat is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 917. The excessive sweating is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 918. The tremor of arms is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 919. The feeling of fever is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 920. The loss of weight is a clinical manifestation of the patient with:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 921. For the laboratory disturbances of Basedow's disease is typical:
 - A. *Increased level of triiodothyronine
 - B. Decreased level of triiodothyronine
 - C. Increased level of glucocorticoids

- D. Decreased level of glucocorticoids
 - E. Decreased level of insulin
922. For the laboratory disturbances of Basedow's disease is typical:
- A. *Increased level of thyroxine
 - B. Decreased level of triiodothyronine
 - C. Increased level of glucocorticoids
 - D. Decreased level of glucocorticoids
 - E. Decreased level of insulin
923. For the laboratory disturbances of thyrotoxicosis is typical:
- A. *Increased level of protein metabolism
 - B. Decreased level of carbohydrate metabolism
 - C. Decreased level of lipid metabolism
 - D. Decreased level of protein metabolism
 - E. Decreased level of all kinds of metabolism
924. For the laboratory disturbances of thyrotoxicosis is typical:
- A. *Increased level of carbohydrate metabolism
 - B. Decreased level of carbohydrate metabolism
 - C. Decreased level of lipid metabolism
 - D. Decreased level of protein metabolism
 - E. Decreased level of all kinds of metabolism
925. For the laboratory disturbances of thyrotoxicosis is typical:
- A. *Increased level of lipid metabolism
 - B. Decreased level of carbohydrate metabolism
 - C. Decreased level of lipid metabolism
 - D. Decreased level of protein metabolism
 - E. Decreased level of all kinds of metabolism
926. The increased level of protein metabolism is typical for:
- A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
927. The increased level of lipid metabolism is typical for:
- A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
928. The increased level of carbohydrate metabolism is typical for:
- A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
929. The Graefe's sign is typical for:
- A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
930. The Stellwag's sign is typical for:
- A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter

- D. Gastric ulcer
- E. Esophageal diverticulum
- 931. The Mebius' sign is typical for:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 932. The Dalrymple's sign is typical for:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 933. The Kocher's sign is typical for:
 - A. *Thyrotoxicosis
 - B. Mixedema
 - C. Euthyroid goiter
 - D. Gastric ulcer
 - E. Esophageal diverticulum
- 934. The Graefe's sign is:
 - A. *The upper lid lag when the patient looks downward
 - B. Infrequent winking
 - C. A weakness of convergence
 - D. Wide palpebral fissure
 - E. Retraction of the upper eyelid at prompt change of view
- 935. The Mebius' sign is:
 - A. *A weakness of convergence
 - B. Infrequent winking
 - C. The upper lid lag when the patient looks downward
 - D. Wide palpebral fissure
 - E. Retraction of the upper eyelid at prompt change of view
- 936. The Stellwag's sign is:
 - A. *Infrequent winking
 - B. The upper lid lag when the patient looks downward
 - C. A weakness of convergence
 - D. Wide palpebral fissure
 - E. Retraction of the upper eyelid at prompt change of view
- 937. The Dalrymple's sign is:
 - A. *Wide palpebral fissure
 - B. Infrequent winking
 - C. The upper lid lag when the patient looks downward
 - D. A weakness of convergence
 - E. Retraction of the upper eyelid at prompt change of view
- 938. The Kocher's sign is:
 - A. *Retraction of the upper eyelid at prompt change of view
 - B. Wide palpebral fissure
 - C. Infrequent winking
 - D. The upper lid lag when the patient looks downward
 - E. A weakness of convergence
- 939. How is the sign, which is characterized by the upper lid lag when the patient looks downward named by author?
 - A. *Graefe's sign
 - B. Mebius' sign

- C. Stellwag's sign
 - D. Dalrymple's sign
 - E. Kocher's sign
940. How is the sign, which is characterized by infrequent winking named by author?
- A. *Stellwag's sign
 - B. Graefe's sign
 - C. Mebius' sign
 - D. Dalrymple's sign
 - E. Kocher's sign
941. How is the sign, which is characterized by a weakness of convergence named by author?
- A. *Mebius' sign
 - B. Stellwag's sign
 - C. Graefe's sign
 - D. Dalrymple's sign
 - E. Kocher's sign
942. How is the sign, which is characterized by a wide palpebral fissure named by author?
- A. *Dalrymple's sign
 - B. Mebius' sign
 - C. Stellwag's sign
 - D. Graefe's sign
 - E. Kocher's sign
943. How is the sign, which is characterized by retraction of the upper eyelid at prompt change of view named by author?
- A. *Kocher's sign
 - B. Dalrymple's sign
 - C. Mebius' sign
 - D. Stellwag's sign
 - E. Graefe's sign
944. For the thyrotoxicosis is typical:
- A. *Graefe's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
945. For the thyrotoxicosis is typical:
- A. *Mebius' sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
946. For thyrotoxicosis is typical:
- A. *Stellwag's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
947. For the thyrotoxicosis is typical:
- A. *Dalrymple's sign
 - B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
948. For the thyrotoxicosis is typical:
- A. *Kocher's sign

- B. Homan's sign
 - C. Lovenberg's sign
 - D. Mondor's sign
 - E. Murphy's sign
949. What form of thyrotoxicosis is classified as a mild?
- A. *Pulse rate less than 100 beat/min
 - B. Pulse rate 100-120 beat/min
 - C. Pulse rate 120-140 beat/min
 - D. Pulse rate 140-160 beat/min
 - E. Pulse rate more than 160 beat/min
950. What form of thyrotoxicosis is classified as a moderate?
- A. *Pulse rate 100-120 beat/min
 - B. Pulse rate less than 100 beat/min
 - C. Pulse rate 120-140 beat/min
 - D. Pulse rate 140-160 beat/min
 - E. Pulse rate more than 160 beat/min
951. What form of thyrotoxicosis is classified as severe?
- A. *Pulse rate more than 120 beat/min
 - B. Pulse rate less than 40 beat/min
 - C. Pulse rate 40-80 beat/min
 - D. Pulse rate 80-100 beat/min
 - E. Pulse rate 100-120 beat/min
952. What form of thyrotoxicosis is classified as a mild?
- A. *Loss weight less than 3-5 kg
 - B. Loss weight 5-8 kg
 - C. Loss weight 8-10 kg
 - D. Loss weight 10-15 kg
 - E. Loss weight exceeds 15 kg
953. What form of thyrotoxicosis is classified as a moderate?
- A. *Loss weight 5-10 kg
 - B. Exceed of weight more than 1-2 kg
 - C. Exceed of weight more than 3-5 kg
 - D. Loss weight 10-15 kg
 - E. Loss weight exceeds 15 kg
954. What form of thyrotoxicosis is classified as severe?
- A. *Loss weight exceeds 10 kg
 - B. Loss weight less than 1-2 kg
 - C. Loss weight 2-3 kg
 - D. Exceed of weight more than 1-2 kg
 - E. Exceed of weight more than 3-5 kg
955. What form of thyrotoxicosis is classified as a mild?
- A. *Increase of basal metabolism to 30 %
 - B. Increase of basal metabolism on 30-50 %
 - C. Increase of basal metabolism more than 50 %
 - D. Decrease of basal metabolism to 30 %
 - E. Decrease of basal metabolism more than 50 %
956. What form of thyrotoxicosis is classified as a moderate?
- A. *Increase of basal metabolism on 30-50 %
 - B. Increase of basal metabolism to 30 %
 - C. Increase of basal metabolism more than 50 %
 - D. Decrease of basal metabolism to 30 %
 - E. Decrease of basal metabolism more than 50 %
957. What form of thyrotoxicosis is classified as severe?
- A. *Increase of basal metabolism more than 50 %

- B. Increase of basal metabolism on 30-50 %
 - C. Increase of basal metabolism to 30 %
 - D. Decrease of basal metabolism to 30 %
 - E. Decrease of basal metabolism more than 50 %
958. What form of thyrotoxicosis is related with the increase of basal metabolism on 30-50 %?
- A. *Moderate
 - B. Mild
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
959. What form of thyrotoxicosis is related with the increase of basal metabolism to 30 %?
- A. *Mild
 - B. Moderate
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
960. What form of thyrotoxicosis is related with the increase of basal metabolism more than 50 %?
- A. *Severe
 - B. Mild
 - C. Moderate
 - D. Subclinic
 - E. Asymptomatic
961. What form of thyrotoxicosis is related with the loss weight less than 3-5 kg?
- A. *Mild
 - B. Moderate
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
962. What form of thyrotoxicosis is related with the loss weight 5-10 kg?
- A. *Moderate
 - B. Mild
 - C. Severe
 - D. Subclinic
 - E. Asymptomatic
963. What form of thyrotoxicosis is related with the loss weight more than 10 kg?
- A. *Severe
 - B. Moderate
 - C. Mild
 - D. Subclinic
 - E. Asymptomatic
964. What is the most informative in the diagnostic of thyrotoxicosis?
- A. *Thyroid hormone concentration
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
965. What is the most informative in the diagnostic of thyrotoxicosis?
- A. *Serum iodine-binding globulin concentration
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
966. What is the most informative in the diagnostic of thyrotoxicosis?

- A. *Serum thyroidstimulating hormone of hypophysis
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
967. What is the most informative in the diagnostic of thyrotoxicosis?
- A. *Serum thyroidstimulating antibodies
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
968. What is the most informative in the diagnostic of thyrotoxicosis?
- A. *Detecting of basal metabolism
 - B. General blood analysis
 - C. X-ray examination with barium
 - D. Coagulogram
 - E. X-ray examination of the neck
969. What diseases should be the thyrotoxicosis differentiated with?
- A. *Rheumatic disease
 - B. Obesity
 - C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
970. What diseases should be the thyrotoxicosis differentiated with?
- A. *Chroniosepsis
 - B. Obesity
 - C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
971. What diseases should be the thyrotoxicosis differentiated with?
- A. *Diencephalic lesions
 - B. Obesity
 - C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
972. What diseases should be the thyrotoxicosis differentiated with?
- A. *Thyroid cancer
 - B. Obesity
 - C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
973. What diseases should be the thyrotoxicosis differentiated with?
- A. *Encephalitis
 - B. Obesity
 - C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
974. What medicines belong to thyrostatic agents?
- A. *Mercasolil
 - B. Euphyllin
 - C. Vasaprostan
 - D. Nicotine acid
 - E. Detrlex
975. What medicines belong to thyrostatic agents?

- A. *Lithium carbonate
 - B. Euphyllin
 - C. Vasaprostan
 - D. Nicotine acid
 - E. Detrlex
976. What group of medicines does Mercasolil belong to?
- A. *Thyrostatic agents
 - B. Antibiotics
 - C. Anticoagulants
 - D. Antiaggregants
 - E. Vitamines
977. What group of medicines does Lithium carbonate belong to?
- A. *Thyrostatic agents
 - B. Antibiotics
 - C. Anticoagulants
 - D. Antiaggregants
 - E. Vitamines
978. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. *Thyrotoxicosis
 - B. Obesity
 - C. Lerishe's syndrome
 - D. Paget-Shretter's syndrome
 - E. Achalasia
979. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. *Severe forms of thyrotoxicosis
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Goiter with hypothyroidism
980. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. *Goiter of IV-V degree
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Goiter with hypothyroidism
981. The subtotal subfascial resection of the thyroid gland is indicated for:
- A. *Nodular transformation of toxic goiter
 - B. The goiter of 0 degree
 - C. The goiter of I degree
 - D. The goiter of II degree
 - E. Goiter with hypothyroidism
982. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. *Thyroid storm
 - B. Cretinism
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
983. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. *The damage of laryngeal nerve
 - B. Cretinism
 - C. Lerishe's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
984. Among the typical complication of a postoperative period for thyrotoxicosis is:

- A. *Asphyxia
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
985. Among the complication of a postoperative period for thyrotoxicosis is:
- A. *Air embolism
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
986. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. *Parathyroid tetany
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
987. Among the typical complication of a postoperative period for thyrotoxicosis is:
- A. *Bleeding
 - B. Cretinism
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
988. The development of excitement, up to psychosis and coma in early postoperative period after thyroid surgery is the manifestation of:
- A. *Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Air embolism
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
989. The development of tachycardia (pulse rate – 150-200 per minute) in early postoperative period after thyroid surgery is the manifestation of:
- A. *Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
990. The development of complete arrhythmia in early postoperative period after thyroid surgery is the manifestation of:
- A. *Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
991. The development of fever to 40°C in early postoperative period after thyroid surgery is the manifestation of:
- A. *Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
992. The development of hyperemia of the face, neck, limbs in early postoperative period after thyroid surgery is the manifestation of:
- A. *Thyroid storm

- B. The damage of laryngeal nerve
 - C. Leriche's syndrome
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
993. The development of extremely sweating, diarrhea in early postoperative period after thyroid surgery is the manifestation of:
- A. *Thyroid storm
 - B. The damage of laryngeal nerve
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
994. The development of hoarseness in early postoperative period after thyroid surgery is the manifestation of:
- A. *The damage of laryngeal nerve
 - B. Thyroid storm
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
995. The development of aphonia in early postoperative period after thyroid surgery is the manifestation of:
- A. *The damage of laryngeal nerve
 - B. Thyroid storm
 - C. Parathyroid tetany
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
996. The development of cramps in early postoperative period after thyroid surgery is the manifestation of:
- A. *Parathyroid tetany
 - B. The damage of laryngeal nerve
 - C. Thyroid storm
 - D. Adrenal insufficiency
 - E. Itsenko-Cushing syndrome
997. For the clinical manifestation of thyroid storm is typical:
- A. *Excitement, up to psychosis and coma
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
998. For the clinical manifestation of thyroid storm is typical:
- A. *Tachycardia (pulse rate – 150-200 per minute)
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
999. For the clinical manifestation of thyroid storm is typical:
- A. *Complete arrhythmia
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
1000. For the clinical manifestation of thyroid storm is typical:
- A. *Fever to 40°C
 - B. Hoarseness
 - C. Cramps

- D. Aphonia
 - E. Anemia
1001. For the clinical manifestation of thyroid storm is typical:
- A. *Hyperemia of the face, neck, limbs
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
1002. For the clinical manifestation of thyroid storm is typical:
- A. *Extremely sweating
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
1003. For the clinical manifestation of thyroid storm is typical:
- A. *Diarrhea
 - B. Hoarseness
 - C. Cramps
 - D. Aphonia
 - E. Anemia
1004. For the clinical manifestation of the damage of laryngeal nerve is typical:
- A. *Hoarseness
 - B. Diarrhea
 - C. Cramps
 - D. Fever to 40°C
 - E. Anemia
1005. For the clinical manifestation of the damage of laryngeal nerve is typical:
- A. *Aphonia
 - B. Diarrhea
 - C. Cramps
 - D. Fever to 40°C
 - E. Anemia
1006. For the clinical manifestation of the parathyroid tetany is typical:
- A. *Cramps
 - B. Aphonia
 - C. Diarrhea
 - D. Fever to 40°C
 - E. Anemia
1007. The purulent thyroiditis is a:
- A. *De Kerven thyroiditis
 - B. Hashimoto's goiter
 - C. Riedel's goiter
 - D. Grave's disease
 - E. Basedow's disease
1008. The autoimmune thyroiditis is a:
- A. *Hashimoto's goiter
 - B. De Kerven thyroiditis
 - C. Riedel's goiter
 - D. Grave's disease
 - E. Basedow's disease
1009. The fibrous thyroiditis is a:
- A. *Riedel's goiter
 - B. Hashimoto's goiter
 - C. De Kerven thyroiditis

- D. Grave's disease
- E. Basedow's disease
- 1010. The "woody" goiter is a:
 - A. *Riedel's goiter
 - B. Hashimoto's goiter
 - C. De Kerven thyroiditis
 - D. Grave's disease
 - E. Basedow's disease
- 1011. The Riedel's goiter is a:
 - A. *"Woody" goiter
 - B. Autoimmune thyroiditis
 - C. Purulent thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
- 1012. The Riedel's goiter is a:
 - A. *Fibrous thyroiditis
 - B. Autoimmune thyroiditis
 - C. Purulent thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
- 1013. The Hashimoto's goiter is a:
 - A. *Autoimmune thyroiditis
 - B. Fibrous thyroiditis
 - C. Purulent thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
- 1014. The De Kerven thyroiditis is a:
 - A. *Purulent thyroiditis
 - B. Autoimmune thyroiditis
 - C. Fibrous thyroiditis
 - D. Thyrotoxicosis
 - E. Nodular goiter
- 1015. The treatment of de Kerven thyroiditis is a:
 - A. *Drainage of suppurative focus
 - B. Glycocorticoids
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil
- 1016. The treatment of Hashimoto's goiter is a:
 - A. *Glycocorticoids
 - B. Drainage of suppurative focus
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil
- 1017. The treatment of Hashimoto's goiter is a:
 - A. *Thyroidectomy
 - B. Drainage of suppurative focus
 - C. Radioactive iodine
 - D. Resection of thyroid gland
 - E. Mercasolil
- 1018. The treatment of Riedel's goiter is a:
 - A. *Thyroidectomy
 - B. Drainage of suppurative focus
 - C. Radioactive iodine

- D. Resection of thyroid gland
 - E. Mercasolil
1019. What vessels are usually affected by atherosclerosis obliterans?
- A. *Aorta and arteries
 - B. Arterioles
 - C. Microcirculatory bed
 - D. Superficial veins
 - E. Deep veins
1020. What is the morphological basis of atherosclerotic lesions?
- A. *The accumulation of lipids in the intima
 - B. Thrombosis
 - C. Inflammatory process
 - D. Embolism
 - E. Aneurysm
1021. What is the main cause of atherosclerotic lesions?
- A. *Hypercholesterolemia, dyslipoproteinemia
 - B. Infection
 - C. Trauma
 - D. Rheumatism, endocarditis
 - E. Myocardial infarction
1022. What does apply to the first stage of atherosclerotic lesions according to the classification by Fontane?
- A. *Full compensation
 - B. Asymptomatic ran
 - C. Functional circulatory insufficiency
 - D. Limb ischemia at rest
 - E. Destruction of tissue
1023. What does belong to the second stage of atherosclerotic lesions according to the classification by Fontane?
- A. *Functional circulatory insufficiency
 - B. Asymptomatic ran
 - C. Full compensation
 - D. Limb ischemia at rest
 - E. Destruction of tissue
1024. What does belong to the third stage of atherosclerotic lesions according to the classification by Fontane?
- A. *Limb ischemia at rest
 - B. Asymptomatic ran
 - C. Full compensation
 - D. Functional circulatory insufficiency
 - E. Destruction of tissue
1025. What does belong to the fourth stage of atherosclerotic lesions according to the classification by Fontane?
- A. *Destruction of tissue
 - B. Asymptomatic ran
 - C. Full compensation
 - D. Functional circulatory insufficiency
 - E. Limb ischemia at rest
1026. What is the most typical feature of the first stage of atherosclerotic lesions?
- A. *Cooling of the lower extremities
 - B. Fever
 - C. Intermittent claudication
 - D. Gangrene
 - E. Pain at rest

1027. What is the most typical sign of the second stage of atherosclerotic lesions?
- A. *Intermittent claudication
 - B. Cooling of the lower extremities
 - C. Fever
 - D. Gangrene
 - E. Pain at rest
1028. What is the most typical feature of the third stage of atherosclerotic lesions?
- A. *Pain at rest
 - B. Fever
 - C. Cooling of the lower extremities
 - D. Intermittent claudication
 - E. Gangrene
1029. What is the most typical feature of the fourth stage of atherosclerotic lesions?
- A. *Gangrene
 - B. Pain at rest
 - C. Fever
 - D. Cooling of the lower extremities
 - E. Intermittent claudication
1030. What is characterized by intermittent claudication?
- A. *Pain in the muscles of his legs when walking, which disappears after rest
 - B. Ischialgia, lumbago
 - C. Constant pain in the joints
 - D. Pain along the superficial veins
 - E. Edema of lower extremities
1031. Intermittent claudication is characterized by:
- A. *Pain in the lower extremities
 - B. Heartache
 - C. Arthralgia
 - D. Dizziness
 - E. Edema of lower extremities
1032. Intermittent claudication is characterized for:
- A. *Atherosclerosis of the lower extremities
 - B. Deep vein thrombosis
 - C. Pancreatitis
 - D. Varicose
 - E. Cholecystitis
1033. To what stage of atherosclerotic lesions is characteristic intermittent claudication up to 1000 meters?
- A. *II
 - B. IIA
 - C. IIB
 - D. III
 - E. IV
1034. To what stage of atherosclerotic lesions is characteristic intermittent claudication before 500 meters?
- A. *IIA
 - B. I
 - C. III
 - D. IIB
 - E. IV
1035. To what stage of atherosclerotic lesions characteristic of intermittent claudication before 200 meters?
- A. *IIB
 - B. I

- C. IIA
 - D. IV
 - E. III
1036. To what stage of atherosclerotic lesions is characteristic of intermittent claudication 25-50 meters?
- A. *III
 - B. I
 - C. IIA
 - D. IV
 - E. IIB
1037. To what stage of atherosclerotic lesions is characteristic the pain at rest?
- A. *III
 - B. I
 - C. IIA
 - D. IV
 - E. IIB
1038. To what stage of atherosclerotic lesions characteristic dry trophic ulcer?
- A. *III
 - B. I
 - C. IIA
 - D. IV
 - E. IIB
1039. To what stage of atherosclerotic lesions is characteristic the necrosis and gangrene?
- A. *IV
 - B. III
 - C. I
 - D. IIA
 - E. II B
1040. What is the main cause of limb swelling in patients with atherosclerosis obliterans?
- A. *Permanent seating position to relieve pain
 - B. Deep vein thrombosis
 - C. Infection, abscess
 - D. Arterial thrombosis
 - E. Heart failure
1041. Where is the most frequent location of venous ulcers with obliterating atherosclerosis?
- A. *At the tip of the toes
 - B. In the lower third of the lower extremities
 - C. In the upper third of the lower extremities
 - D. On the back of the knee
 - E. At the hip
1042. Leriche syndrome is:
- A. *Occlusion the bifurcation of abdominal aorta
 - B. Stenosis the brachiocephalic trunk
 - C. Renal artery stenosis
 - D. Stenosis of the abdominal trunk
 - E. Stenosis of pulmonary artery
1043. Leriche syndrome is characterized by:
- A. *Atherosclerotic lesions
 - B. Obliterative endarteritis
 - C. Varicose
 - D. Phlebemphraxis
 - E. Lymphedema
1044. Leriche is characterized by:
- A. *Atherosclerotic lesions

- B. Acute cholecystitis
 - C. Acute pancreatitis
 - D. Ileus
 - E. Appendicular infiltrate
1045. For Leriche syndrome is characterized by:
- A. *Intermittent claudication
 - B. Angina
 - C. Dizziness
 - D. Oedema of lower extremities
 - E. Extension of saphenous veins
1046. For Leriche syndrome is characterized by:
- A. *The absence of pulsations in the lower extremities
 - B. Hyperbilirubinemia
 - C. Ascites
 - D. Oedema of lower extremities
 - E. Extension of saphenous veins
1047. For Leriche syndrome is characterized by:
- A. *Hypercholesterolemia
 - B. Hyperbilirubinemia
 - C. Increased blood amylase
 - D. Leukocytosis
 - E. Anemia
1048. For Leriche syndrome is characteristic X-ray symptoms:
- A. *Occlusion of the terminal aorta
 - B. Occlusion of terminal part of the inferior vena cava
 - C. Occlusion of the superior vena cava
 - D. Dysplasia arteries
 - E. Dysplasia veins
1049. For Leriche syndrome is characteristic X-ray symptoms:
- A. *Occlusion of the terminal aorta
 - B. Cloiber's bowls
 - C. Pneumoperitoneum
 - D. The symptom of "niche"
 - E. Detelectasis
1050. At Leriche syndrome patient has complains on:
- A. *Intermittent claudication
 - B. Pain in the heart
 - C. Oedema of lower extremities
 - D. Extension of saphenous veins
 - E. Dizziness
1051. At Leriche syndrome patient has complains on:
- A. *Melosalgia
 - B. Pain in the heart
 - C. Pain during urination
 - D. Tenesmus
 - E. Pain in the epigastric area
1052. At what level is absent arterial pulsation at the Leriche syndrome?
- A. *Femoral artery
 - B. Posterior tibial artery
 - C. Dorsal artery of foot
 - D. Popliteal artery
 - E. Common carotid artery
1053. What complication is caused by atherosclerosis obliterans?
- A. *Acute arterial thrombosis

- B. Arteriorrhexis
 - C. Acute venous thrombosis
 - D. Phlegmon
 - E. Superficial thrombophlebitis
1054. What complication is caused by atherosclerosis obliterans?
- A. *Aneurysm
 - B. Arteriorrhexis
 - C. Acute venous thrombosis
 - D. Phlegmon
 - E. Superficial thrombophlebitis
1055. Which clinical sign is typical for abdominal aortic aneurysm?
- A. *Systolic noise over a pulsating formation in the abdominal cavity
 - B. Abdominal pain
 - C. Leukopenia
 - D. Diarrhea
 - E. Tension of abdominal wall
1056. Which clinical sign is typical for the rupture abdominal aortic aneurysm?
- A. *Hemorrhagic shock
 - B. Leukopenia
 - C. Systolic noise over a pulsating formation in the abdominal cavity
 - D. Diarrhea
 - E. Tension of abdominal wall
1057. Which clinical sign is typical bundle abdominal aortic aneurysm?
- A. *They expressed the pain radiating to the loin
 - B. Pukes bile
 - C. Systolic noise over the formation of a pulsating abdominal
 - D. Diarrhea
 - E. Voltage anterior abdominal wall
1058. What is the treatment of uncomplicated aortic aneurysm?
- A. *Aortic prosthesis
 - B. Saphenectomy
 - C. Conservative treatment
 - D. Ligation of the aorta
 - E. Profundoplastic
1059. What is the treatment gap abdominal aortic aneurysm?
- A. *Endovascular prostheses
 - B. Saphenectomy
 - C. Conservative treatment
 - D. Ligation of the aorta
 - E. Profundoplastic
1060. Which of the instrumental methods of investigation is the most informative at obliterating atherosclerosis?
- A. *Ultrasound
 - B. ECG
 - C. Spirography
 - D. Urography
 - E. Target biopsy
1061. In obliterating atherosclerosis determined by:
- A. *Lenel-Lavestin's symptom
 - B. Homan's symptom
 - C. Ortner's symptom
 - D. Rovzing's symptom
 - E. Babinski symptom
1062. The pulsation of the femoral artery is determined by:

- A. *By the middle of the inguinal ligament;
 - B. By the middle line above the stomach and the navel;
 - C. By the mid-popliteal fossa with slightly bent limbs in the knee;
 - D. Between the back-bottom edge of the medial bone and Achilles tendon;
 - E. Between I and II metatarsals.
1063. The pulsation of the abdominal aorta is determined by:
- A. *On the middle line above the stomach and the navel;
 - B. On the middle of the inguinal ligament;
 - C. On the middle popliteal fossa with slightly bent limbs in the knee;
 - D. Between lowback edge of the medial bone and Achilles tendon;
 - E. Between I and II metatarsals.
1064. The pulsation of the popliteal artery is defined:
- A. *On the middle popliteal fossa with slightly bent limbs in the knee;
 - B. On the middle line above the stomach and the navel;
 - C. On the middle of the inguinal ligament;
 - D. Between the back-bottom edge of the medial bone and Achilles tendon;
 - E. Between I and II metatarsals.
1065. The pulsation of the posterior tibial artery is determined by:
- A. *Between the back-bottom edge of the medial bone and Achilles tendon;
 - B. On the middle popliteal fossa with slightly bent limbs in the knee;
 - C. On the middle line above the stomach and the navel;
 - D. On the middle of the inguinal ligament;
 - E. Between I and II metatarsals.
1066. Ripple dorsal artery of foot is determined by:
- A. *Between I and II metatarsals.
 - B. Between lowback edge of the medial bone and Achilles tendon;
 - C. On the middle popliteal fossa with slightly bent limbs in the knee;
 - D. On the middle line above the stomach and the navel;
 - E. On the middle of the inguinal ligament;
1067. What kind of ankle pressure is characterize the critical ischemia?
- A. *Less than 50 mm Hg.
 - B. Less than 140 mm Hg.
 - C. Less than 70 mm Hg.
 - D. Less than 90 mm Hg.
 - E. Less than 110 mm Hg.
1068. What kind of ankle pressure is characterized the II stage of the chronic ischemia?
- A. *Less than 90 mm Hg.
 - B. Less than 50 mm Hg.
 - C. Less than 140 mm Hg.
 - D. Less than 70 mm Hg.
 - E. Less than 110 mm Hg.
1069. What kind of ankle index corresponds to II stage of the chronic ischemia?
- A. *Less than 0,9.
 - B. Less than 0,5.
 - C. 1,0.
 - D. More than 1,0.
 - E. More than 1,5.
1070. What kind of ankle index corresponds to III stage of the chronic ischemia?
- A. *Less than 0,5.
 - B. Less than 0,9.
 - C. 1,0.
 - D. More than 1,0.
 - E. More than 1,5.
1071. Which of the X-ray methods is the most informative at obliterating atherosclerosis?

- A. *Arteriography
 - B. Abdominal radiography
 - C. Chest radiography
 - D. Radiography limb
 - E. Phlebography
1072. For atherosclerotic lesions at arteriography is characterized by:
- A. *Segmental occlusion of the arteries
 - B. Occlusion of terminal part of the inferior vena cava
 - C. Occlusion of the superior vena cava
 - D. Dysplasia arteries
 - E. Diffuse stenosis of small arteries
1073. What is the main distinctive feature between atherosclerosis and endarteritis obliterans?
- A. *The level of arterial pulsation
 - B. Pain syndrome
 - C. Trophic ulcers
 - D. Changes in coagulation
 - E. Skin color
1074. What method of research is the most informative in the differential diagnosis between atherosclerosis and endarteritis obliterans?
- A. *Angiography
 - B. ECG
 - C. Biochemical analysis of blood
 - D. Complete blood
 - E. Target biopsy
1075. What is the main distinctive feature between atherosclerosis and lumbosacral radiculitis?
- A. *Arterial pulsation in the lower extremities
 - B. Pain syndrome of the lower extremities
 - C. The color of the skin of the lower extremities
 - D. Cold extremities
 - E. Paresthesias of lower extremities
1076. At what level is no ripple at lumbosacral radiculitis?
- A. *Stored at all levels of
 - B. Calf arteries
 - C. Popliteal artery
 - D. Femoral artery
 - E. Aorta
1077. Which method to study is the most informative in the differential diagnosis between atherosclerosis and diabetic angiopathy?
- A. *Biochemical analysis of blood
 - B. Complete blood
 - C. Koagulogramme
 - D. Immunogramma
 - E. Urinalysis
1078. Which clinical sign is not typical for diabetic angiopathy?
- A. *No pulsation of femoral artery
 - B. Necrosis of the fingers on the lower extremity
 - C. Trophic ulcers on the foot
 - D. Phlegmon of the foot
 - E. Paresthesias
1079. What are the indications for conservative therapy of obliterative atherosclerosis?
- A. *I-II stage of chronic arterial insufficiency
 - B. Not shown at all
 - C. III-IV stage of chronic arterial insufficiency
 - D. Leriche syndrome

- E. Arterial thrombosis
1080. Which drugs has affect on atherogenesis?
- A. *Cholestyramine
 - B. Trental
 - C. Vasaprostan
 - D. Nicotinic acid
 - E. Aspirin
1081. Which drug does belongs to antiaggregants?
- A. *Pentoksiphilin
 - B. Cholestyramine
 - C. Vasaprostan
 - D. Nicotinic acid
 - E. Papaverine
1082. What are the indications for surgical arterial reconstruction?
- A. *II-III stage of chronic arterial insufficiency
 - B. No evidence
 - C. I-II stage of chronic arterial insufficiency
 - D. Gangrene of the lower extremity
 - E. Phlegmon of the lower extremity
1083. Which operation is performed with Leriche syndrome?
- A. *Aorto-femoral bypass
 - B. Lumbar sympathectomy
 - C. Intimectomy
 - D. Resection of the arteries
 - E. Artery ligation
1084. Which operation is performed at Leriche syndrome?
- A. *Right answer is absent
 - B. Bypass thick intestinal anastomosis
 - C. Gastrectomy
 - D. Cholecystectomy
 - E. Saphenectomy
1085. What are the indications for endarterectomy:
- A. *Isolated segmental occlusion of the artery
 - B. Leriche syndrome
 - C. Multi-storey artery occlusion
 - D. Calcinosis artery
 - E. Occlusive disease
1086. What is the most common operation at atherosclerotic occlusion of the femoral artery?
- A. *Autogenous vein bypass
 - B. Lumbar sympathectomy
 - C. Intimectomy
 - D. Resection of the arteries
 - E. Artery ligation
1087. What is the localization of arterial occlusion requires prothundoplastic?
- A. *Bifurcation of the common femoral artery
 - B. Bifurcation of the aorta
 - C. Bifurcation of common iliac artery
 - D. Trifurcation popliteal artery
 - E. Bifurcation carotid artery
1088. What is the complication of arterial reconstruction does not require repeated surgical intervention?
- A. *Deep vein thrombosis
 - B. Arterial thrombosis
 - C. Arterial bleeding

- D. Injection of synthetic graft
 - E. The increase in lower limb ischemia
1089. What are indication to amputation at obliterating atherosclerosis?
- A. *Gangrene of the lower extremity
 - B. Leriche
 - C. I-II stage of ischemia
 - D. II-III stage of ischemia
 - E. Arterial bleeding
1090. Which artery are usually affects occlusive endarteritis?
- A. *Calf arteries
 - B. Aorta
 - C. Iliac arteries
 - D. Femoral artery
 - E. Deep femoral artery
1091. What factors play a very significant role in pathogenesis the obliterative endarteritis?
- A. *Chronic intoxication
 - B. Hyperlipidemia
 - C. Triglyceridemia
 - D. Vascular injury
 - E. Thrombophlebitis
1092. What is the morphological basis of obliterative endarteritis?
- A. *Intimal hyperplasia
 - B. Atheroma
 - C. Embolism
 - D. Aneurysm
 - E. Arteriovenous fistula
1093. What is the fourth stage of obliterating endarteritis?
- A. *Ulcer-necrotic
 - B. Asymptomatic
 - C. Coronary
 - D. Trophic changes
 - E. Gangrenous
1094. What is the main feature of obliterative endarteritis?
- A. *Intermittent claudication
 - B. Muscle contraction
 - C. Fever
 - D. Arthritic pain
 - E. Ishalgia
1095. What is the cause of intermittent claudication?
- A. *Muscle ischemia
 - B. Trauma
 - C. Ishalgia
 - D. Muscle contraction
 - E. Arthritic pain
1096. The most typical localization of intermittent claudication at occlusive disease is?
- A. *Foot
 - B. Stifle
 - C. Hip
 - D. Hip
 - E. Stomach
1097. Cooling stop is characteristic:
- A. *Surface thrombophlebitis
 - B. Deep thrombophlebitis
 - C. Obliterative endarteritis

- D. Lymphostasis
- E. Postthrombotic syndrome
- 1098. Blanching of the skin foot is characteristic:
 - A. *Obliterative endarteritis
 - B. Deep thrombophlebitis
 - C. Surface thrombophlebitis
 - D. Postthrombotic syndrome
 - E. Phlegmon of the foot
- 1099. What is the typical location the venous ulcers at occlusive disease?
 - A. *At fingertips
 - B. In the lower third of the lower extremities
 - C. In the upper third of the lower extremities
 - D. On the back of the knee
 - E. At the hip
- 1100. What is the typical sign for the I stage of obliterating endarteritis?
 - A. *Cooling of the lower extremities
 - B. Intermittent claudication
 - C. Fever
 - D. Pain at rest
 - E. Gangrene
- 1101. What is the typical sign for II stage the obliterative endarteritis?
 - A. *Intermittent claudication
 - B. Fever
 - C. Cooling of the lower extremities
 - D. Gangrene
 - E. Pain at rest
- 1102. What is the typical sign for the III stage of obliterating endarteritis?
 - A. *Pain at rest
 - B. Cooling of the lower extremities
 - C. Fever
 - D. Intermittent claudication
 - E. Gangrene
- 1103. What is the typical sign for the IV stage of obliterating endarteritis?
 - A. *Gangrene
 - B. Cooling of the lower extremities
 - C. Fever
 - D. Pain at rest
 - E. Intermittent claudication
- 1104. What is the most frequent complication the obliterative endarteritis?
 - A. *Arterial thrombosis and gangrene of the extremities
 - B. Bleeding
 - C. Blindness
 - D. Chylorrhea
 - E. Aneurysm
- 1105. What is the main goal of therapy at obliterating endarteritis?
 - A. *Renewal or improvement of capillary circulation
 - B. Resumption pass vein
 - C. Resumption of the entrance of lymph
 - D. Resumption pass arteries
 - E. Improving the innervation of the lower extremity
- 1106. For obliterative endarteritis is characterized by:
 - A. *Intermittent claudication
 - B. Angina
 - C. Dizziness

- D. Oedema of lower extremities
- E. Extension of saphenous veins
- 1107. For obliterative endarteritis is characterized by:
 - A. *The pulse absent on the feet
 - B. Hyperbilirubinemia
 - C. Ascites
 - D. Oedema of lower extremities
 - E. Extension of saphenous veins
- 1108. For obliterative endarteritis is characteristic X-ray symptoms:
 - A. *Diffuse stenosis of small arteries
 - B. Occlusion of terminal part of the inferior vena cava
 - C. Occlusion of the superior vena cava
 - D. Arteries dysplasia
 - E. Veins dysplasia
- 1109. For obliterative endarteritis is characteristic X-ray symptoms:
 - A. *No right answer
 - B. Bowls Kloiber
 - C. Pneumoperitoneum
 - D. The symptom of "niche"
 - E. Detelectasis
- 1110. At occlusive disease patient complains on:
 - A. *Intermittent claudication
 - B. Pain in the heart
 - C. Oedema of lower extremities
 - D. Extension of saphenous veins
 - E. Dizziness
- 1111. At obliterating endarteritis patient complains of:
 - A. *Melosalgia
 - B. Pain in the heart
 - C. Pain during urination
 - D. Tenesmus
 - E. Pain in the epigastric area
- 1112. At what level is absent arterial pulsation at obliterative endarteritis ?
 - A. *Arteries foot
 - B. Femoral artery
 - C. Popliteal artery
 - D. Common carotid artery
 - E. Abdominal aorta
- 1113. Which group of drugs are pentoxifyllinum?
 - A. *Antiagrigant
 - B. Antispasmodic
 - C. Antihistamines
 - D. Stimulants metabolism
 - E. Narcotic analgesics
- 1114. Which group of drugs are vasaprostan?
 - A. *Prostaglandins
 - B. Antiagrigant
 - C. Antispasmodic
 - D. Antihistamines
 - E. Stimulants metabolism
- 1115. Which operation is performed at obliterative endarteritis ?
 - A. *Lumbar sympathectomy
 - B. Intimectomy
 - C. Bypass grafting

- D. Resection of the arteries
 - E. Artery ligation
1116. Which factor is the leader in the development of atherosclerosis?
- A. *Dyslipoproteinemia.
 - B. Diabetes.
 - C. Suprarenalism.
 - D. Frequent hypothermia.
 - E. Smoking.
1117. Which factor is the leader in the development of obliterative endarteritis?
- A. *Hypothermia, intoxication.
 - B. Hypercholesterolemia.
 - C. Diabetes.
 - D. Violations electrolytic exchange.
 - E. Suprarenalism.
1118. At what age is the greatest risk of ill atherosclerosis obliterans?
- A. *Older than 40 years.
 - B. In 20 - 60 years.
 - C. In 19 - 25 years.
 - D. In 30 - 35 years.
 - E. In 35 - 39 years.
1119. At what age is the greatest risk of ill obliterative endarteritis?
- A. *Up to 40 years.
 - B. 41 - 50 years.
 - C. 51 - 60 years.
 - D. In 10 - 15 years.
 - E. Over 60 years.
1120. At atherosclerosis obliterans first affected:
- A. *Arteries, aorta.
 - B. Arteriovenous shunt vessels.
 - C. Capillaries.
 - D. Arteries of medium diameter.
 - E. Small arteries.
1121. At obliterative endarteritis first affected:
- A. *Peripheral arteries.
 - B. Inguinal artery.
 - C. Aorta.
 - D. Ventral trunk.
 - E. The upper and lower mesenteric artery.
1122. At what disease you can auscultated systolic murmur on the major arteries?
- A. *In obliterating atherosclerosis.
 - B. When occlusive disease.
 - C. With varicose veins.
 - D. In acute venous thrombosis shins.
 - E. When ileofemoralnom venous thrombosis.
1123. With the defeat of what artery atherosclerosis can develops Leriche syndrome?
- A. *Bifurcation of the aorta, common iliac arteries.
 - B. Popliteal artery.
 - C. Arteries of the lower leg.
 - D. Ventral trunk.
 - E. Inferior mesenteric artery.
1124. What kind of reconstructive operations on the vessels are carried out with Leriche syndrome?
- A. *Aorto-femoral prosthesis or bypass surgery.
 - B. Operation Linton or Kokkett.

- C. Leriche's operation.
 - D. Troyanov-Trendelenburg's operation, Babcock's operation.
 - E. Embolectomy
1125. What kind of reconstructive operations on the major arteries are carried out with obliterating atherosclerosis?
- A. *Endarterectomy, bypass surgery or prosthetic arteries.
 - B. Leriche's operation.
 - C. Lumbar sympathectomy.
 - D. Palm's operation .
 - E. Troyanov-Trendelenburg's operation.
1126. What operations are conducted in obliterating endarterite lower extremities?
- A. *Lumbar sympathectomy, Leriche's operation.
 - B. Embolectomy
 - C. Saphenectomy.
 - D. Thrombectomy.
 - E. Intimectomy
1127. What kind of manipulation to be done vascular prostheses infection?
- A. *Remove the prosthesis.
 - B. Catheterization subclavian vein.
 - C. Catheterization great saphenous vein.
 - D. Fasciotomy.
 - E. Necrectomy.
1128. Named the arteries that catheterization for aortography with bilateral Leriche syndrome?
- A. *Brachial artery.
 - B. Total n artery.
 - C. Thigh iliac artery.
 - D. Subclavian artery.
 - E. Rear leg artery
1129. Aorto-occlusive disease at arteriogram characterized by:
- A. *Uniform narrowing of the lumen of the arteries of the lower extremities.
 - B. Occlusion of peripheral arteries.
 - C. Uneven narrowing of the lumen of the arteries of the lower extremities.
 - D. Occlusion of collateral arteries.
 - E. Occlusion of capillaries.
1130. Lumbar sympathectomy is accompanied by:
- A. *Removing the spasm of precapillary sphincter .
 - B. Decrease in prothrombin index.
 - C. Normalization of glucose.
 - D. Increased protein content in blood serum.
 - E. Normalization of bilirubin in the blood serum.
1131. To improve the microcirculation provide drugs:
- A. *Nicotinic acid, reopolyglukine.
 - B. Diphenhydramine, suprastin.
 - C. Anaprilin, lineotol.
 - D. Cytitone, lobeline.
 - E. Amidopyrine, analgin.
1132. What drugs have antisclerotic action?
- A. *Clofibrate, linetol, parmidin.
 - B. Cytitone, lobeline.
 - C. Fenilin, Omefin.
 - D. Nicotinic acid.
 - E. Diphenhydramine, suprastin.
1133. What methods are used to reduce the concentration of cholesterol and lipoproteins in the blood serum?

- A. *Plasmapheresis, hemosorption.
 - B. Artificial diuresis.
 - C. Hemodilution.
 - D. Transfusion of blood
 - E. Introduction vasorostana
1134. At what level are removed ganglia at the lumbar sympathectomy?
- A. *L3 - L4.
 - B. S2 - S3.
 - C. L1.
 - D. Th 10-17
 - E. Th 8-9.
1135. How soon after the appointment of showing its effect indirect anticoagulants?
- A. *After 12 - 48 hours.
 - B. After 4 hours.
 - C. After 72 hours.
 - D. After 56 hours.
 - E. 46-56 hours.
1136. Specify non-pharmacological methods of correcting blood coagulation system.
- A. Hemodilution.
 - B. Hemodialysis.
 - C. Hemosorption.
 - D. Artificial diuresis.
 - E. Limfosorbtsiya.
1137. What are the contrast agents used for angiography?
- A. *Triyodtrast, verografín.
 - B. Methylene blue.
 - C. Barium sulfate.
 - D. Alprostan.
 - E. Vasoprostan.
1138. Catheterization of the aorta is performed by:
- A. *Seldinger.
 - B. Billroth.
 - C. Kocher
 - D. Pirogov
 - E. Shalimov
1139. What complications may arise during percutaneous catheterization of the aorta by Seldinger?
- A. *Bleeding, thrombosis, embolism.
 - B. Acute thrombophlebitis leg veins.
 - C. Relaxation of the diaphragm.
 - D. Thrombosis of the subclavian vein.
 - E. Endocarditis.
1140. At occlusion what arteries can develops Leriche syndrome:
- A. *Occlusion of the aortic bifurcation, common iliac arteries
 - B. Occlusion of inferior mesenteric artery
 - C. Occlusion of the subclavian and brachial arteries
 - D. Occlusion of the popliteal artery and lower leg
 - E. Occlusion of the internal iliac arteries
1141. At occlusion what arteries can develops unilateral Leriche syndrome?
- A. *Occlusion of the external and common iliac arteries
 - B. Occlusion of the internal iliac arteries
 - C. Occlusion of the aortic bifurcation
 - D. Occlusion of the deep femoral artery
 - E. Occlusion of popliteal artery

1142. For aorto-arteriography using contrast agents:
- A. *Triumbrast, verografin, urotrast
 - B. Seabar
 - C. Bilignost
 - D. Holevid
 - E. Iodognost
1143. Aorto-arteriography by percutaneous catheterization of the aorta through a peripheral artery is developed:
- A. *Seldingerom
 - B. Petrovsky
 - C. Suharev
 - D. Pokrovsky
 - E. Vishnevsky
1144. For aorto-arteriografii with bilateral Leriche syndrome conducted puncture:
- A. *Brachialis artery
 - B. Femoral artery
 - C. Popliteal artery
 - D. External iliac artery
 - E. Cubital vein
1145. At obliterating atherosclerosis affected:
- A. *Aorta and arteries
 - B. Small and small arteries
 - C. Arterivenoznye shunts
 - D. Kommunikantni vein
 - E. Arterioles
1146. At obliterating endartereiite affected:
- A. *Peripheral artery
 - B. Arteriovenous shunts
 - C. Kommunikantnye vein
 - D. Aorta and arteries
 - E. Sural vein
1147. The leading factor in the development of atherosclerotic lesions is:
- A. *Violation of cholesterol-lipid
 - B. Violation of protein metabolism
 - C. Suprarenalism
 - D. Improving the function of the sympathetic system
 - E. Violation of mineral metabolism
1148. B-lipoproteins show:
- A. *Antiplatelet effect
 - B. Atherogenic effect
 - C. Spasmolytic
 - D. Antiatherogenic effect
 - E. Surfactant effect
1149. Alpha-lipoproteins has:
- A. *Atherogenic effect
 - B. Antiatherogenic effect
 - C. Anticoagulant activity
 - D. Antiplatelet effect
 - E. Surfactant effect
1150. At obliterative atherosclerosis of lower limb arteries performed:
- A. *Bypass surgery, prostheses, endarterectomy
 - B. Leriche's operation, Oppel
 - C. Linton's operation
 - D. Babcock's operation

- E. Troyanov – Trandelenburg’s operation
1151. The most severe complication after reconstructive operations on the major arteries are:
- A. *Bleeding
 - B. Suppuration
 - C. Thrombosis
 - D. Chylorrhea
 - E. Phlebeurysm
1152. At occlusive disease of the lower extremities performed :
- A. *Lumbar sympathectomy, Leriche's operation
 - B. Linton’s operation
 - C. Bypass surgery
 - D. Intimectomy
 - E. Troyanov – Trandelenburg’s operation
1153. What are the indications for lumbar sympathectomy at obliterating endarterite?
- A. *Stage II
 - B. Stage IV
 - C. Gangrene of the lower extremity
 - D. Deep venous thrombosis
 - E. The duration of reactive hyperemia was more than 3 minutes
1154. At segmental occlusion of the bifurcation of the femoral artery what operation is performed:
- A. *Endarterectomy of the femoral artery
 - B. Bifurcation bypass surgery
 - C. Iliac-femoral bypass surgery
 - D. Saphenectomy
 - E. Artery ligation
1155. At segmental occlusion of the superficial femoral artery what operation is performed:
- A. *Autogenous vein bypass
 - B. Endarterectomy of the femoral artery
 - C. Iliac-femoral bypass surgery
 - D. Saphenectomy
 - E. Artery ligation
1156. At segmental occlusion of the popliteal artery what operation is performed:
- A. *Autogenous vein bypass
 - B. Endarterectomy of the femoral artery
 - C. Iliac-femoral bypass surgery
 - D. Saphenectomy
 - E. Artery ligation
1157. At segmental occlusion of the iliac artery what operation is performed:
- A. *Iliac-femoral aloshuntirovanie
 - B. Autogenous vein bypass
 - C. Endarterectomy of the femoral artery
 - D. Saphenectomy
 - E. Artery ligation
1158. Endarterectomy is performed at:
- A. *Atherosclerosis obliterans
 - B. Occlusive disease
 - C. Varicose
 - D. Deep vein thrombosis
 - E. Lymphedema
1159. Iliac-femoral bypass surgery performed at:
- A. *Atherosclerosis obliterans
 - B. Occlusive disease
 - C. Varicose
 - D. Deep vein thrombosis

- E. Lymphedema
1160. Femoropopliteal autovenous bypass surgery is performed at :
- A. *Atherosclerosis obliterans
 - B. Occlusive disease
 - C. Varicose
 - D. Deep vein thrombosis
 - E. Lymphedema
1161. Aorto-femoral bifurcation bypass surgery performed at:
- A. *Atherosclerosis obliterans
 - B. Occlusive disease
 - C. Varicose
 - D. Deep vein thrombosis
 - E. Lymphedema
1162. Decompression osteoperforation performed at:
- A. *Atherosclerosis obliterans
 - B. Coxarthrosis
 - C. Varicose
 - D. Deep vein thrombosis
 - E. Lymphedema
1163. Sympathectomy performed at:
- A. *Occlusive disease
 - B. Atherosclerosis obliterans
 - C. Varicose
 - D. Deep vein thrombosis
 - E. Lymphedema
1164. What is the normal number of platelets in the blood?
- A. *180-320
 - B. 20-40
 - C. 40-120
 - D. 420-650
 - E. 600-1000
1165. What phase hemocoagulation inhibits heparin:
- A. *Only the formation of thrombin
 - B. No right answer
 - C. Only the formation of fibrin
 - D. Only the formation of thromboplastin
 - E. It operates on the phase of thrombotic
1166. By indirect anticoagulants include:
- A. *Preparation of 4-oksikumarina
 - B. Preparations of alkaloids
 - C. Fibrinolytic drugs
 - D. Pentoksifilin
 - E. Thrombolytic drugs
1167. Absolute contraindications to anticoagulation attributes is:
- A. *Bleeding of any location, hemorrhagic diathesis
 - B. Aplastic anemia
 - C. Respiratory failure
 - D. Cardiovascular insufficiency
 - E. Lack of cerebral circulation
1168. Methods control of coagulation and fibrinolytic systems of blood:
- A. *Coagulogramm
 - B. Complete blood count
 - C. Determination of the rate of local blood flow
 - D. Blood count

- E. Biochemical analysis of blood
1169. What vessels are usually affected by atherosclerosis obliterans?
A. *Aorta and arteries
B. Arterioles
C. Microcirculatory bed
D. Superficial veins
E. Deep veins
1170. What is the morphological basis of atherosclerotic lesions?
A. *The accumulation of lipids in the intima
B. Thrombosis
C. Inflammatory process
D. Embolism
E. Aneurysm
1171. What is the main cause of atherosclerotic lesions?
A. *Hypercholesterolemia, dyslipoproteinemia
B. Infection
C. Trauma
D. Rheumatism, endocarditis
E. Myocardial infarction
1172. What principle is the basis classification of ischemic tissues in acute arterial obstruction?
A. *Clinical manifestations of ischemic tissues.
B. The residual blood flow in the limbs.
C. No ripple on the main arteries.
D. Segmental principle
E. The level of occlusion
1173. Specify non-pharmacological methods of correcting blood coagulation system.
A. Hemodilution.
B. Hemodialysis.
C. Hemosorption.
D. Artificial diuresis.
E. Limphosorbtsion.
1174. What are the contrast agents used for angiography?
A. *Triiodtrast, verografin.
B. Barium sulfate.
C. Methylene blue.
D. Alprostan.
E. Vasoprostan.
1175. In stage III acute obstruction of major arteries embolectomy completed:
A. *Fasciotomy
B. Operation Linton
C. Troyanov-Trendelenburg's operation
D. Modelung's operation
E. Kokket's operation
1176. The direct anticoagulant is
A. Heparin.
B. Phenilin, Omephin.
C. Nicotinic acid.
D. Streptokinase.
E. Acetylsalicylic acid.
1177. In case of overdose of heparin used:
A. *Protamine sulfate.
B. Rheopolyglucine.
C. Albumin.
D. Phibrinolysin.

- E. 10% glucose solution.
1178. For embolectomy used:
- *Fogarty probe .
 - Dzhadkins catheter .
 - Babcock's vein stripper.
 - Nelaton's probe .
 - Blekmor's probe
1179. Best results embolectomy occur during an operation in the first place:
- *6 hours.
 - 48 hours.
 - 8:00.
 - 24 hours.
 - 12 hours.
1180. What are the methods embolectomy arteries?
- *Direct and indirect embolectomy .
 - Fasciotomy.
 - Intimtrombektomiya.
 - Shunting.
 - Endarterectomy.
1181. Indicate the surgical treatment acute thrombosis of major arteries.
- Intimtrombectomy, thrombectomy, bypass surgery.
 - Embolectomy.
 - Fasciotomy.
 - Sympathectomy.
 - Leriche's operation.
1182. Types embolectomy :
- *Direct, indirect
 - Retrograde
 - Full, partial
 - Open, half closed, closed
 - Rising
1183. The most frequent cause of arterial thrombosis is:
- *Blood disease
 - Obliterating atherosclerosis
 - Trauma
 - Aneurysm
 - Diabetes mellitus
1184. To angiographic signs embolisation of major arteries relates:
- *Uniform narrowing of major vessels
 - Breaking contrast, poorer collateral channel, the absence of signs of atherosclerosis
 - The dramatic expansion of the main vessel
 - Pronounced collateral blood flow
 - Phlebeurysm
1185. To angiographic signs of acute thrombosis the major arteries include:
- *Aneurysmal expansion of arteries
 - Weak collateral blood flow
 - Slanted posted line break contrast, unequal contours of the arteries, developed collateral network
 - Uneven, serrated contours artery
 - Phlebeurysm
1186. Severe pallor of skin is characteristic for:
- *Artery Embolism
 - Varicose
 - Deep phlebothrombosis

- D. Postthrombophlebitic syndrome
 - E. Phlegmon limb
1187. Expressed cold extremities is characteristic for:
- A. *Arterial thrombosis
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombophlebitic syndrome
 - E. Phlegmon limb
1188. Full anesthesia limbs is characteristic for:
- A. *Arterial thrombosis
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombophlebitic syndrome
 - E. Phlegmon limb
1189. The absence pulsations on the extremities is characteristic for :
- A. *Arterial thrombosis
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
1190. Embolectomy performed at:
- A. *Artery Embolism
 - B. Varicose
 - C. Deep vein thrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
1191. Trombintimectomy performed at:
- A. *Arterial thrombosis
 - B. Deep vein thrombosis
 - C. Artery Embolism
 - D. Subcutaneous thrombophlebitis
 - E. Varicose
1192. Autovenous bypass grafting is performed when:
- A. *Arterial thrombosis
 - B. Deep vein thrombosis
 - C. Artery Embolism
 - D. Subcutaneous thrombophlebitis
 - E. Varicose
1193. Thrombolytic therapy is indicated for:
- A. *Arterial thrombosis
 - B. Extremity lymphedema
 - C. Artery Embolism
 - D. Subcutaneous thrombophlebitis
 - E. Varicose
1194. Acute myocardial infarction is a risk factor:
- A. *Artery Embolism
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
1195. Bacterial endocarditis is a risk factor:
- A. *Artery Embolism
 - B. Arterial thrombosis
 - C. Varicose

- D. Phlegmon limb
- E. Postthrombotic syndrome
- 1196. Rheumatic heart disease is a risk factor:
 - A. *Artery Embolism
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1197. Abdominal aortic aneurysm is a risk factor for:
 - A. *Artery Embolism
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1198. Atrial fibrillation is a risk factor:
 - A. *Artery Embolism
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1199. Cardiac fibrillation is a risk factor:
 - A. *Artery Embolism
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1200. Obliterating atherosclerosis is a risk factor:
 - A. *Arterial thrombosis
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1201. Closed trauma limb is risk factor:
 - A. *Arterial thrombosis
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1202. Systemic lupus erythematosus is a risk factor:
 - A. *Arterial thrombosis
 - B. Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1203. Acute leukemia is a risk factor:
 - A. *Arterial thrombosis
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1204. Extravascular compression is a risk factor:
 - A. *Arterial thrombosis
 - B. Artery Embolism
 - C. Varicose

- D. Phlegmon limb
 - E. Postthrombotic syndrome
1205. Severe pallor of skin is characteristic for:
- A. *No right answer
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
1206. Expressed cold extremities is characteristic for:
- A. *No right answer
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
1207. Full anesthesia limbs characteristic:
- A. *No right answer
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
1208. The absence pulsations on the extremities artery is characteristic for:
- A. *No right answer
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Postthrombotic syndrome
 - E. Phlegmon limb
1209. Embolectomy performed at:
- A. *No right answer
 - B. Varicose
 - C. Deep phlebothrombosis
 - D. Posttromboflebitichnogo syndrome
 - E. Phlegmon limb
1210. Trombintimectomy performed at:
- A. *No right answer
 - B. Deep vein thrombosis
 - C. Artery Embolism
 - D. Subcutaneous thrombophlebitis
 - E. Varicose
1211. Autovenous bypass grafting is performed when:
- A. *No right answer
 - B. Deep vein thrombosis
 - C. Artery Embolism
 - D. Subcutaneous thrombophlebitis
 - E. Varicose
1212. Thrombolytic therapy is indicated for:
- A. *No right answer
 - B. Extremity lymphedema
 - C. Artery Embolism
 - D. Subcutaneous thrombophlebitis
 - E. Varicose
1213. Acute myocardial infarction is a risk factor:
- A. *No right answer
 - B. Arterial thrombosis
 - C. Varicose

- D. Phlegmon limb
- E. Postthrombotic syndrome
- 1214. Bacterial endocarditis is a risk factor:
 - A. *No right answer
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1215. Rheumatic heart disease is a risk factor:
 - A. *No right answer
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1216. Abdominal aortic aneurysm is a risk factor:
 - A. *No right answer
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1217. Atrial fibrillation is a risk factor:
 - A. *No right answer
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1218. Cardiac fibrillation is a risk factor:
 - A. *No right answer
 - B. Arterial thrombosis
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1219. Obliterating atherosclerosis is a risk factor:
 - A. *No right answer
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1220. Closed trauma limb risk factor:
 - A. *No right answer
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1221. Systemic lupus erythematosus is a risk factor:
 - A. *No right answer
 - B. Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1222. Acute leukemia is a risk factor:
 - A. *No right answer
 - B. Artery Embolism
 - C. Varicose

- D. Phlegmon limb
- E. Postthrombotic syndrome
- 1223. Extra vessels artery compression is a risk factor:
 - A. *Arterial thrombosis
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1224. Angiography is a procedure risk factor:
 - A. *Arterial thrombosis
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1225. Angiography is a procedure risk factor:
 - A. *No right answer
 - B. Artery Embolism
 - C. Varicose
 - D. Phlegmon limb
 - E. Postthrombotic syndrome
- 1226. What drugs belong to a direct anticoagulant?
 - A. *Fraxiparine,
 - B. Sinkumar,
 - C. Streptokinase,
 - D. Trental,
 - E. Diclofenac,
- 1227. What drugs belong to the indirect anticoagulants?
 - A. *Sinkumar
 - B. Fraxiparine
 - C. Streptokinase
 - D. Trental
 - E. Diclofenac
- 1228. What drugs belong to fibrinolytics?
 - A. *Streptokinase, urokinase
 - B. Heparin
 - C. Sinkumar, fenilin
 - D. Trental, Pentoksifilin
 - E. Diclofenac, Naklofen
- 1229. What is initial dose Streptokinase?
 - A. *250000 units
 - B. 5000
 - C. 10000 points
 - D. 100000 units
 - E. 1000000 Units
- 1230. What drug is used in the overdose of heparin?
 - A. *Protamine sulfate
 - B. Fraxiparine
 - C. Streptokinase
 - D. Trental
 - E. Diclophenac
- 1231. Which drug is used in the overdose of heparin?
 - A. *No right answer
 - B. Fraxiparine
 - C. Streptokinase

- D. Trental
 - E. Diclophenac
1232. What drug is used in an overdose of streptokinase?
- A. *Aminocaproic acid, trasylol
 - B. Fraxiparine, Clexane
 - C. Protamine sulfate
 - D. Trental, Pentoksifilin
 - E. Diclofenac, Naklofen
1233. What is normal prothrombin index?
- A. *85-100%
 - B. 10-20%
 - C. 30-60%
 - D. 50-70%
 - E. 100-120%
1234. What are the indicators of coagulation control dosing of anticoagulants?
- A. *Prothrombin index
 - B. The number of platelets in the blood
 - C. The level of plasma fibrinogen
 - D. Trombotest
 - E. Plasma recalcification time
1235. What is the normal level of plasma fibrinogen?
- A. *2-4 g / l
 - B. 6.8 grams / liter
 - C. 10-16 grams / liter
 - D. 30-50 grams / liter
 - E. 75-100 grams / liter
1236. What are the indicators of coagulation control dosing of thrombolytics?
- A. *The level of plasma fibrinogen
 - B. The number of platelets in the blood
 - C. Prothrombin index
 - D. Thrombotest
 - E. Plasma recalcification time
1237. What are the normal values of plasma recalcification time?
- A. *60-120 seconds
 - B. 0.5-2 seconds
 - C. 10-40 seconds
 - D. 40-60 seconds
 - E. 4-8 minutes
1238. What are the normal rates thrombotest?
- A. *IV-V degree
 - B. I-II degree
 - C. II-III degree
 - D. VI-VIII degree
 - E. X-XII level
1239. What is the normal number of platelets in the blood?
- A. *180-320 10⁹ / l
 - B. 20-40 10⁹ / l
 - C. 40-120 10⁹ / l
 - D. 420-650 10⁹ / l
 - E. 600-1000 10⁹ / l
1240. What phase hemocoagulation inhibits heparin:
- A. *Only the formation of thrombin
 - B. No right answer
 - C. Only the formation of fibrin

- D. Only the formation of thromboplastin
- E. It operates on the phase of thrombotic
- 1241. Indirect anticoagulants include:
 - A. *Preparation of 4-oksikumarina
 - B. Preparations of alkaloids
 - C. Fibrinolytic drugs
 - D. Pentoksiphylin
 - E. Thrombotic drugs
- 1242. The absolute contraindication of anticoagulants include:
 - A. *Bleeding of any location, hemorrhagic diathesis
 - B. Aplastic anemia
 - C. Respiratory failure
 - D. Cardiovascular insufficiency
 - E. Lack of cerebral circulation
- 1243. Activators of fibrinolysis include:
 - A. *Nicotinic acid, Complamin
 - B. Phenilin, Omefin
 - C. Gastrotsepin, Venter
 - D. Vitamin B
 - E. Vitamins A, C
- 1244. Preparations thrombolytic action are:
 - A. *Phibrinolysin, celiasa, streptokinase
 - B. Strophanthus
 - C. Tselonid, lantozid
 - D. Rheopolyglucine, poliglucin
 - E. Trental
- 1245. Methods of control the coagulation and fibrinolytic systems of blood:
 - A. *Coagulogramm
 - B. Complete blood
 - C. Determination of the rate of local blood flow
 - D. Blood count
 - E. Biochemical analysis of blood
- 1246. At bleeding caused by anticoagulants of indirect action shall appoint:
 - A. *Vicasol
 - B. Pipolphen
 - C. Digitoxin
 - D. Hydrocortisone
 - E. Pentoksiphilin
- 1247. Clinical signs the embolism arteries in the first hours is
 - A. Severe pain in the limbs
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
- 1248. Clinical signs the embolism arteries in the first hours
 - A. Cold extremities
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
- 1249. Clinical signs the arteries embolism in the first hours is
 - A. Severe weakness of the affected limb
 - B. Dizziness
 - C. Nausea

- D. Limb gangrene
 - E. Dystonia
1250. Clinical signs the arteries embolism in the first hours is
- A. Paleness of the skin of the affected limb
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1251. Clinical signs the arteries embolism in the first hours is
- A. The deterioration of the picture subcutaneous veins of the affected limb
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1252. Clinical signs the arteries embolism in the first hours is
- A. Changing the surface sensitivity of the affected limb
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1253. Clinical signs the arteries embolism in the first hours is
- A. Changing the deep sensitivity of the affected limb
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1254. Clinical signs the arteries embolism in the first hours is
- A. Violation of the function of the affected limb
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1255. Clinical signs the arteries embolism in the first hours is
- A. The disappearance of pulsation of the arteries to the level of obstruction
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1256. Clinical signs the arteries embolism in the first hours is
- A. No right answer
 - B. Dizziness
 - C. Nausea
 - D. Limb gangrene
 - E. Dystonia
1257. At occlusion of the bifurcation aorta region of ischemia captures
- A. Both legs and lower abdomen
 - B. Lower limb to crural arch
 - C. Lower extremity to the middle third of the thigh
 - D. Lower extremity to the knee
 - E. Foot
1258. When occlusion of the bifurcation of the aorta region of ischemia captures
- A. No right answer
 - B. Lower limb to crural arch
 - C. Lower extremity to the middle third of the thigh

- D. Lower extremity to the knee
- E. Foot
- 1259. At occlusion of iliac artery ischemia region captures
 - A. Lower limb to crural arch
 - B. Both legs and lower abdomen
 - C. Lower extremity to the middle third of the thigh
 - D. Lower extremity to the knee
 - E. Foot
- 1260. When occlusion of iliac artery ischemia region captures
 - A. No right answer
 - B. Both legs and lower abdomen
 - C. Lower extremity to the middle third of the thigh
 - D. Lower extremity to the knee
 - E. Foot
- 1261. At occlusion of the femoral artery ischemia region captures
 - A. Lower extremity to the middle third of the thigh
 - B. Lower limb to crural arch
 - C. Both legs and lower abdomen
 - D. Lower extremity to the knee
 - E. Foot
- 1262. At occlusion of the femoral artery ischemia region captures
 - A. No right answer
 - B. Lower limb to crural arch
 - C. Both legs and lower abdomen
 - D. Lower extremity to the knee
 - E. Foot
- 1263. At occlusion of the popliteal artery ischemia region captures
 - A. Lower extremity to the middle third of the thigh
 - B. Lower limb to crural arch
 - C. Both legs and lower abdomen
 - D. Lower extremity to the knee
 - E. Foot
- 1264. At occlusion of the popliteal artery ischemia region captures
 - A. No right answer
 - B. Lower extremity to the middle third of the thigh
 - C. Lower limb to crural arch
 - D. Both legs and lower abdomen
 - E. Foot
- 1265. At occlusion of the popliteal artery ischemia region captures
 - A. Lower extremity to the knee
 - B. Lower extremity to the middle third of the thigh
 - C. Lower limb to crural arch
 - D. Both legs and lower abdomen
 - E. Foot
- 1266. At occlusion of the subclavian artery ischemia region captures
 - A. All hand
 - B. Arm to the upper third of the shoulder
 - C. Hand until the middle third of the shoulder
 - D. Both hands
 - E. Brush
- 1267. When occlusion of the subclavian artery ischemia region captures
 - A. No right answer
 - B. Arm to the upper third of the shoulder
 - C. Hand until the middle third of the shoulder

- D. Both hands
 - E. Brush
1268. When occlusion of the axillary artery ischemia region captures
- A. Arm to the upper third of the shoulder
 - B. All hand
 - C. Hand until the middle third of the shoulder
 - D. Both hands
 - E. Brush
1269. At occlusion of the axillary artery ischemia region captures
- A. No right answer
 - B. All hand
 - C. Hand until the middle third of the shoulder
 - D. Both hands
 - E. Brush
1270. At occlusion of the brachial artery area of ischemia captures
- A. Hand until the middle third of the shoulder
 - B. All hand
 - C. Arm to the upper third of the shoulder
 - D. Both hands
 - E. Brush
1271. At occlusion of the brachial artery area of ischemia captures
- A. No right answer
 - B. All hand
 - C. Arm to the upper third of the shoulder
 - D. Both hands
 - E. Brush
1272. How many days can occur secondary deep vein thrombosis limb?
- A. *3-6 days
 - B. 1-2 days
 - C. 7-8 days
 - D. 7-10 days
 - E. 14 days
1273. How many days can occur secondary deep vein thrombosis limb?
- A. *No right answer
 - B. 1-2 days
 - C. 7-8 days
 - D. 7-10 days
 - E. 14 days
1274. Arterial thrombosis developed on the background:
- A. *Atherosclerotic lesions
 - B. Ulcer
 - C. Chronic pancreatitis
 - D. Ulcerative colitis
 - E. Chronic gastritis
1275. Arterial thrombosis developed on the background:
- A. *Obliterative endarteritis
 - B. Ulcer
 - C. Chronic pancreatitis
 - D. Ulcerative colitis
 - E. Chronic gastritis
1276. Arterial thrombosis developed on the background:
- A. *Nonspecific aortoarteriitis
 - B. Ulcer
 - C. Chronic pancreatitis

- D. Ulcerative colitis
- E. Chronic gastritis
- 1277. Arterial thrombosis developed on the background:
 - A. *Infectious Diseases
 - B. Ulcer
 - C. Chronic pancreatitis
 - D. Ulcerative colitis
 - E. Chronic gastritis
- 1278. Arterial thrombosis developed on the background:
 - A. *Erythrocytosis
 - B. Ulcer
 - C. Chronic pancreatitis
 - D. Ulcerative colitis
 - E. Chronic gastritis
- 1279. Arterial thrombosis developed on the background:
 - A. *Hypercoagulation
 - B. Ulcer
 - C. Chronic pancreatitis
 - D. Ulcerative colitis
 - E. Chronic gastritis
- 1280. How long is the reversible phase change?
 - A. *2-3 hours
 - B. 3-5 hours
 - C. 5-6 hours
 - D. 7-8 hours
 - E. 10-12 hours
- 1281. How long is the reversible phase change?
 - A. *No right answer
 - B. 3-5 hours
 - C. 5-6 hours
 - D. 7-8 hours
 - E. 10-12 hours
- 1282. How long is the growth phase of irreversible changes
 - A. 5-6 hours
 - B. 2-3 hours
 - C. 3-5 hours
 - D. 7-8 hours
 - E. 10-12 hours
- 1283. How long is the growth phase of irreversible changes
 - A. No right answer
 - B. 2-3 hours
 - C. 3-5 hours
 - D. 7-8 hours
 - E. 10-12 hours
- 1284. For the diagnosis of embolism of arteries of extremities most informative method is:
 - A. *Doppler ultrasound
 - B. Rheovasography
 - C. Radiography limbs
 - D. ECG
 - E. Echocardiography
- 1285. To diagnose thrombosis of arteries most informative method is:
 - A. *Doppler ultrasound
 - B. Rheovasography
 - C. Radiography limbs

- D. ECG
 - E. Echocardiography
1286. For the diagnosis embolism of arteries the extremities most informative method is:
- A. *Arteriography
 - B. Rheovasography
 - C. Radiography limbs
 - D. ECG
 - E. Echocardiography
1287. For the diagnosis embolism of arteries the extremities most informative method is:
- A. *No right answer
 - B. Rheovasography
 - C. Radiography limbs
 - D. ECG
 - E. Echocardiography
1288. At arteries embolism differential diagnosis must be with:
- A. *Arterial spasm
 - B. Deep vein thrombosis
 - C. Lymphadenitis
 - D. Coxarthrosis
 - E. Strangulated hernia
1289. When embolism arteries differential diagnosis must be with:
- A. *Acute heart failure on a background of obliterating arterial diseases
 - B. Deep vein thrombosis
 - C. Lymphadenitis
 - D. Coxarthrosis
 - E. Strangulated hernia
1290. At arteries embolism differential diagnosis must be with:
- A. *Stratification of abdominal aortic aneurysm
 - B. Deep vein thrombosis
 - C. Lymphadenitis
 - D. Coxarthrosis
 - E. Strangulated hernia
1291. At arteries embolism differential diagnosis must be with:
- A. *Acute transverse poliomyelitis
 - B. Deep vein thrombosis
 - C. Lymphadenitis
 - D. Coxarthrosis
 - E. Strangulated hernia
1292. When embolism arteries differential diagnosis must be with:
- A. *No right answer
 - B. Deep vein thrombosis
 - C. Lymphadenitis
 - D. Coxarthrosis
 - E. Strangulated hernia
1293. Absolute contraindication for surgical treatment of embolism is:
- A. *Agony state of patients
 - B. Angina
 - C. Transient ischemic attack
 - D. Fracture limb
 - E. Heart disease
1294. Absolute contraindication for surgical treatment of embolism is:
- A. *Total contracture limb
 - B. Angina
 - C. Transient ischemic attack

- D. Fracture limb
- E. Heart disease
- 1295. Absolute contraindication for surgical treatment of embolism is:
 - A. *Limb gangrene
 - B. Angina
 - C. Transient ischemic attack
 - D. Fracture limb
 - E. Heart disease
- 1296. Absolute contraindication for surgical treatment of embolism is:
 - A. *Critically ill patients with a mild degree of ischemia
 - B. Angina
 - C. Transient ischemic attack
 - D. Fracture limb
 - E. Heart disease
- 1297. Relative contraindications to operation at a mild degree of acute limb ischemia and the absence of its progression is
 - A. Acute myocardial infarction
 - B. Angina
 - C. Varicose disease
 - D. PTFS
 - E. Angiodysplasia
- 1298. Relative contraindications to operation at a mild degree of acute limb ischemia and the absence of its progression is
 - A. Insult
 - B. Angina
 - C. Varicose disease
 - D. PTFS
 - E. Angiodysplasia
- 1299. Relative contraindications to operation at a mild degree of acute limb ischemia and the absence of its progression is
 - A. Inoperable tumor
 - B. Angina
 - C. Varicose disease
 - D. PTFS
 - E. Angiodysplasia
- 1300. Relative contraindications to operation at a mild degree of acute limb ischemia and the absence of its progression is
 - A. No right answer
 - B. Angina
 - C. Varicose disease
 - D. PTFS
 - E. Angiodysplasia
- 1301. For elimination spasm in acute arterial insufficiency using:
 - A. *Papaverine
 - B. Alprostan
 - C. Heparin
 - D. Warfarin
 - E. Dimexide
- 1302. For elimination spasm in acute arterial insufficiency using:
 - A. *No-shpa
 - B. Alprostan
 - C. Heparin
 - D. Warfarin
 - E. Dimexide

1303. The elimination of spasm in acute arterial insufficiency using:
- A. *Halidor
 - B. Alprostan
 - C. Heparin
 - D. Warfarin
 - E. Dimexide
1304. Radical operation in acute arterial insufficiency include:
- A. *Embolintimtrmbectomy
 - B. Thoracic sympathectomy
 - C. Transverse sympathectomy
 - D. Periarterial simpaectomy
 - E. Amputation
1305. Radical operation in acute arterial insufficiency include:
- A. *Intimectomy
 - B. Thoracic sympathectomy
 - C. Transverse sympathectomy
 - D. Periarterial simpaectomy
 - E. Amputation
1306. Radical operation in acute arterial insufficiency include:
- A. *Shunt surgery
 - B. Thoracic sympathectomy
 - C. Transverse sympathectomy
 - D. Periarterial sympathectomy
 - E. Amputation
1307. Radical operation in acute arterial insufficiency include:
- A. *Resection of the artery with prosthetic
 - B. Thoracic sympathectomy
 - C. Transverse sympathectomy
 - D. Periarterial sympathectomy
 - E. Amputation
1308. Radical operation in acute arterial insufficiency include:
- A. *Plasticity of arterial wall with patches
 - B. Thoracic sympathectomy
 - C. Transverse sympathectomy
 - D. Periarterial sympathectomy
 - E. Amputation
1309. To palliative surgery for acute arterial insufficiency include:
- A. *Periarterial sympaectomy
 - B. Plasticity of arterial wall with patches
 - C. Resection artery prosthetic
 - D. Shunt surgery
 - E. Endarterectomy
1310. To palliative surgery for acute arterial insufficiency include:
- A. *Transverse sympathectomy
 - B. Plasticity of arterial wall with patches
 - C. Resection artery prosthetic
 - D. Shunt surgery
 - E. Endarterectomy
1311. To palliative surgery for acute arterial insufficiency include:
- A. *Amputation
 - B. Plasticity of arterial wall with patches
 - C. Resection artery prosthetic
 - D. Shunt surgery
 - E. Endarterectomy

1312. Types embolectomy
- A. Straight
 - B. Oblique
 - C. Transverse
 - D. Ring
 - E. No right answer
1313. Types embolectomy
- A. Indirect
 - B. Oblique
 - C. Transverse
 - D. Ring
 - E. No right answer
1314. For secondary prevention thrombosis during the operation in the vascular bed is introduced
- A. Heparin
 - B. Solcoseryl
 - C. Vicasol
 - D. Actovegin
 - E. No-shpa
1315. For secondary prevention of thrombosis during the operation in the vascular bed is introduced
- A. No right answer
 - B. Solcoseryl
 - C. Vicasol
 - D. Actovegin
 - E. No-shpa
1316. For secondary prevention of thrombosis during the operation in the vascular bed is introduced at a dose of heparin:
- A. *5000
 - B. 10000
 - C. 1000
 - D. 7000
 - E. 9000
1317. Shunting operations include
- A. Femoropopliteal bypass
 - B. Endarterectomy
 - C. Saphenectomy
 - D. Fasciotomy
 - E. Ganglionectomy
1318. Shunting operations include
- A. Femoropopliteal bypass autovenous
 - B. Endarterectomy
 - C. Saphenectomy
 - D. Fasciotomy
 - E. Ganglionectomy
1319. What volume of blood flows through superficial veins?
- A. *10-15 %
 - B. 5 %
 - C. 30-40 %
 - D. 70-80 %
 - E. 90 %
1320. Where does a great saphenous vein drain in?
- A. *Femoral vein
 - B. The veins of shin
 - C. Popliteal vein

- D. External iliac vein
 - E. Vena cava inferior
1321. Where does a small saphenous vein drain in?
- A. *Popliteal vein
 - B. Vena cava inferior
 - C. External iliac vein
 - D. Femoral vein
 - E. The veins of shin
1322. The localization of great saphenous vein inflow is:
- A. *2-3 sm. below inguinal ligament
 - B. In upper third of the lower extremity
 - C. In a popliteal fossa
 - D. In lower third of thigh
 - E. In a lumbar area
1323. The localization of small saphenous vein inflow is:
- A. *In a popliteal fossa
 - B. In upper third of the lower extremity
 - C. In lower third of thigh
 - D. 2-3 sm. below inguinal ligament
 - E. In a lumbar area
1324. What veins belongs to the superficial system?
- A. *Small and great saphenous vein
 - B. The veins of shin
 - C. Superficial and deep femoral vein
 - D. Brachial veins
 - E. Elbow and radial veins
1325. What veins belongs to the deep system?
- A. *V. radialis
 - B. Small saphenous vein
 - C. Great saphenous vein
 - D. V. basilica
 - E. V. cephalica
1326. What factor prevails in the development of primary varicosity?
- A. *Weakness of connecting tissue of vessels
 - B. Arterio-venous fistulas
 - C. Venous hypoplasia
 - D. Diabetes mellitus
 - E. Obliterative atherosclerosis
1327. What are the hormonal changes contribute to the development of varicosity?
- A. *Pregnancy
 - B. Diabetes mellitus
 - C. Thyrotoxicosis
 - D. Miksedema
 - E. Insufficiency of suprarenal glands
1328. What is the pathological basis of the development of chronic venous insufficiency?
- A. *Venous hypertension
 - B. Block of lymph outflow
 - C. Arterial ischemia
 - D. Hypertension
 - E. Disturbance of innervation
1329. What is the cause of hypertension in the venous system of lower extremities?
- A. *Venous valvular incompetence
 - B. Arterial ischemia
 - C. Hypertension

- D. Block of lymph outflow
 - E. Disturbance of innervation
1330. What does the venous valvular incompetence result in?
- A. *Venous hypertension
 - B. Arterial ischemia
 - C. Hypertension
 - D. Disturbance of innervation
 - E. Disturbance of lymph outflow
1331. What is the characteristic sign of the I stage of varicosity?
- A. *Heavy sensation
 - B. Transitory edema
 - C. Permanent edema
 - D. Hyperpigmentation
 - E. Trophic ulcer
1332. What is the characteristic sign of the IIA stage of varicosity?
- A. *Transitory edema
 - B. Heavy sensation
 - C. Permanent edema
 - D. Hyperpigmentation
 - E. Trophic ulcer
1333. What is the characteristic sign of the IIB stage of varicosity?
- A. *Hyperpigmentation
 - B. Heavy sensation
 - C. Transitory edema
 - D. Open trophic ulcer
 - E. Healed trophic ulcer
1334. What is the characteristic sign of the IIB stage of varicosity?
- A. *Permanent edema
 - B. Heavy sensation
 - C. Transitory edema
 - D. Open trophic ulcer
 - E. Healed trophic ulcer
1335. What is the characteristic sign of the III stage of varicosity?
- A. *Trophic ulcer
 - B. Heavy sensation
 - C. Transitory edema
 - D. Permanent edema
 - E. Hyperpigmentation
1336. What stage of chronic venous insufficiency is characterized by the sign of "heavy sensation"?
- A. *I
 - B. 0
 - C. IIA
 - D. IIB
 - E. III
1337. What stage of chronic venous insufficiency is characterized by the sign of a transitory edema?
- A. *IIA
 - B. 0
 - C. I
 - D. IIB
 - E. III
1338. What stage of chronic venous insufficiency is characterized by the sign of a permanent edema?

- A. *IIB
 - B. 0
 - C. I
 - D. IIA
 - E. III
1339. What stage of chronic venous insufficiency is characterized by the sign of a hyperpigmentation?
- A. *IIB
 - B. 0
 - C. I
 - D. IIA
 - E. III
1340. What stage of chronic venous insufficiency is characterized by the sign of lipodermatosclerosis?
- A. *IIB
 - B. 0
 - C. I
 - D. IIA
 - E. III
1341. What stage of chronic venous insufficiency is characterized by the sign of opened trophic ulcer?
- A. *III
 - B. 0
 - C. I
 - D. IIA
 - E. IIB
1342. What stage of chronic venous insufficiency is characterized by the sign of the healed trophic ulcer?
- A. *III
 - B. 0
 - C. I
 - D. IIA
 - E. IIB
1343. Which stage of chronic venous insufficiency is characterized by valvular incompetence of superficial veins?
- A. *I
 - B. 0
 - C. IIA
 - D. IIB
 - E. III
1344. Which stage of chronic venous insufficiency is characterized by valvular incompetence of superficial and perforative veins?
- A. *II
 - B. 0
 - C. I
 - D. III
 - E. IV
1345. Which stage of chronic venous insufficiency is characterized by valvular incompetence of superficial, perforative and deep veins?
- A. *IIB-III
 - B. 0
 - C. I
 - D. IIA
 - E. IV

1346. For varicosity of lower extremities is typical:
- A. *Heavy sensation
 - B. Intermittent claudication
 - C. Rest pains
 - D. Disappearance of sensation
 - E. Disappearance of movements
1347. For varicosity of lower extremities is typical:
- A. *Transitory edema of lower extremities
 - B. Intermittent claudication
 - C. Rest pains
 - D. Disappearance of sensation
 - E. Disappearance of movements
1348. For varicosity of lower extremities is typical:
- A. *Varicose veins
 - B. Cyanosis of the lower extremity
 - C. Pallor of the lower extremity
 - D. Disappearance of sensation
 - E. Disappearance of movements
1349. For varicosity of lower extremities is typical:
- A. *Lipodermatosclerosis of shin
 - B. Cyanosis of the lower extremity
 - C. Pallor of the lower extremity
 - D. Disappearance of sensation
 - E. Disappearance of movements
1350. For varicosity of lower extremities is typical:
- A. *Trophic ulcer of shin
 - B. Cyanosis of the lower extremity
 - C. Pallor of the lower extremity
 - D. Disappearance of sensation
 - E. Disappearance of movements
1351. For varicosity of lower extremities is typical:
- A. *Hyperpigmentation of shin
 - B. Cyanosis of the lower extremity
 - C. Pallor of the lower extremity
 - D. Disappearance of sensation
 - E. Disappearance of movements
1352. Heavy sensation of lower extremities is characteristic for:
- A. *Varicosity
 - B. Atherosclerosis obliterans
 - C. Thrombangiitis obliterans
 - D. Leriche's syndrome
 - E. Embolisms of femoral artery
1353. The transitory edema of lower extremities is characteristic for:
- A. *Varicosity
 - B. Atherosclerosis obliterans
 - C. Thrombangiitis obliterans
 - D. Leriche's syndrome
 - E. Embolisms of femoral artery
1354. Lipodermatosclerosis of the lower third of shin is characteristic for:
- A. *Varicosity
 - B. Atherosclerosis obliterans
 - C. Thrombangiitis obliterans
 - D. Leriche's syndrome
 - E. Embolisms of femoral artery

1355. The hyperpigmentation of the lower third of shin is characteristic for:
- A. *Varicosity
 - B. Atherosclerosis obliterans
 - C. Thrombangiitis obliterans
 - D. Leriche's syndrome
 - E. Embolisms of femoral artery
1356. The trophic ulcer of the lower third of shin is characteristic for:
- A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Thrombangiitis obliterans
 - D. Leriche's syndrome
 - E. Embolisms of femoral artery
1357. What test is used for determination of valvular incompetence of superficial veins?
- A. *Troyanov-Trendelenburg's test
 - B. Talman's test
 - C. Pratt's test
 - D. Mayo-Pratt's test
 - E. Delbe-Pertess test (marching test)
1358. What test is used for determination of valvular incompetence of perforative veins?
- A. *Pratt's test
 - B. Troyanov-Trendelenburg's test
 - C. Gackenbruch's test
 - D. Homan's test
 - E. Delbe-Pertess test (marching test)
1359. What test is used for the estimation of the passability of deep veins?
- A. *Delbe-Pertess test (marching test)
 - B. Troyanov-Trendelenburg's test
 - C. Gackenbruch's test
 - D. Pratt's test
 - E. Talman's test
1360. The Troyanov-Trendelenburg's test is used in the diagnostics of:
- A. *Valvular incompetence of superficial veins
 - B. Valvular incompetence of perforative veins
 - C. Passability of deep veins
 - D. Deep vein thrombosis
 - E. Lymphedema of the extremity
1361. The Talman's test is used in the diagnostics of:
- A. *Valvular incompetence of perforative veins
 - B. Valvular incompetence of superficial veins
 - C. Passability of deep veins
 - D. Deep vein thrombosis
 - E. Lymphedema of the extremity
1362. The Pratt's test is used in the diagnostics of:
- A. *Valvular incompetence of perforative veins
 - B. Valvular incompetence of superficial veins
 - C. Passability of deep veins
 - D. Deep vein thrombosis
 - E. Lymphedema of the extremity
1363. The Mayo-Pratt's test is used in the diagnostics of:
- A. *Passability of deep veins
 - B. Valvular incompetence of perforative veins
 - C. Valvular incompetence of superficial veins
 - D. Deep vein thrombosis
 - E. Lymphedema of the extremity

1364. The Delbe-Pertess test (marching test) is used in the diagnostics of:
- A. *Passability of deep veins
 - B. Valvular incompetence of perforative veins
 - C. Valvular incompetence of superficial veins
 - D. Deep vein thrombosis
 - E. Lymphedema of the extremity
1365. What complication is typical for varicosity?
- A. *Subcutaneous thrombophlebitis
 - B. Arterial thrombosis
 - C. Paresis
 - D. Lymphostasis
 - E. Gangrene
1366. More frequently the subcutaneous thrombophlebitis develops as a result of:
- A. *Varicosity
 - B. Atherosclerosis obliterans
 - C. Lymphedema of lower extremities
 - D. Acute appendicitis
 - E. Acute cholecystitis
1367. What complication is typical for varicosity?
- A. *Trophic ulcer
 - B. Arterial thrombosis
 - C. Paresis
 - D. Plegia
 - E. Gangrene
1368. What is the basic method of the diagnostics of arterio-venous fistulas?
- A. *Phlebography
 - B. Coagulogram
 - C. Delbe-Pertess test (marching test)
 - D. Ultrasound examination
 - E. Arteriography
1369. What is the basic method of the diagnostics of venous angiodysplasia?
- A. *Phlebography
 - B. Coagulogram
 - C. Delbe-Pertess test (marching test)
 - D. Ultrasound examination
 - E. Arteriography
1370. A phlebography is used for the diagnostics of:
- A. *Arterio-venous fistulas
 - B. Atherosclerosis obliterans
 - C. Thrombangiitis obliterans
 - D. Lymphedema
 - E. Gynaecological pathology
1371. A phlebography is used for the diagnostics of:
- A. *Venous angiodysplasia
 - B. Atherosclerosis obliterans
 - C. Thrombangiitis obliterans
 - D. Lymphedema
 - E. Gynaecological pathology
1372. What method is used for the diagnostics of valvular incompetence of perforative and deep veins?
- A. *Ultrasound duplex scanning
 - B. Coagulogram
 - C. Reovasography
 - D. Ultrasound doppler examination

- E. Arteriography
1373. What method is used for the estimation of the passability of deep veins?
- A. *Ultrasound examination
 - B. Coagulogram
 - C. Reovasography
 - D. ECG
 - E. Arteriography
1374. What operation is performed for varicosity?
- A. *Saphenectomy
 - B. Thrombectomy
 - C. Ligation of vein
 - D. Endarterectomy
 - E. Femoro-popliteal by-passing
1375. What treatment is indicated in varicosity of the IIA stage?
- A. *Saphenectomy
 - B. Conservative treatment
 - C. Sclerotherapy
 - D. Troyanov-Trendelenburg's operation
 - E. Saphenectomy with the subfascial ligation of perforative veins (Linton's operation)
1376. What treatment is indicated for a noncomplicated varicosity?
- A. *Saphenectomy
 - B. Conservative treatment
 - C. Sclerotherapy
 - D. Troyanov-Trendelenburg's operation
 - E. Saphenectomy with the subfascial ligation of perforative veins (Linton's operation)
1377. To the real causes of varicosity relapse regard:
- A. *Remaining of main trunk, long stump of great saphenous vein
 - B. Thrombosis of vena cava inferior
 - C. Arterio-venous fistula
 - D. Insufficiency of the ostial valve
 - E. Thrombosis of iliac veins
1378. To the functional tests for the detection of valvular incompetence of superficial veins used:
- A. *Troyanov-Trendelenburg's test
 - B. Delbe-Pertess test (marching test)
 - C. Oppel's test
 - D. Pratt's test
 - E. Talman's test
1379. To the functional tests for the detection of valvular incompetence of perforative veins used:
- A. *Talman's test
 - B. Delbe-Pertess test (marching test)
 - C. Troyanov-Trendelenburg's test
 - D. Gackenbruch's test
 - E. Oppel's test
1380. To the functional tests for the detection of valvular incompetence of deep veins used:
- A. *Delbe-Pertess test (marching test)
 - B. Troyanov-Trendelenburg's test
 - C. Gackenbruch's test
 - D. Oppel's test
 - E. Talman's test
1381. What is the sequence of the saphenectomy performance?
- A. *Troyanov-Trendelenburg's operation, Beacock's, Linton's
 - B. Linton's operation, Cocketh's, Beacock's
 - C. Narath's operation, Beacock's, Cocketh's
 - D. Beacock's operation, Linton's, Troyanov-Trendelenburg's

- E. Bebcock's operation, Narath's, Cocketh's operation
1382. A maximal amount of perforative veins is located in:
- A. *Lower third of shin
 - B. Upper third of shin
 - C. Middle third of shin
 - D. Area of thigh
 - E. Lower third of thigh
1383. The contraindication for saphenectomy in varicosity is:
- A. *Obstruction of deep veins
 - B. Insufficiency of valves of perforative veins
 - C. Insufficiency of the ostial valve
 - D. Diffuse type of great saphenous vein
 - E. Valvular incompetence of sural veins
1384. For the saphenectomy operation used:
- A. *Bebcock's catheter
 - B. Blackmore's zond
 - C. Fogarti's catheter
 - D. Nelaton's catheter
 - E. Nasogastric zond
1385. What operations are performed on the perforative veins of lower extremities:
- A. *Linton's, Cocketh's operation
 - B. Bebcock's, Narath's
 - C. Troyanov-Trendelenburg's
 - D. Madelung's
 - E. Felder's
1386. To the indirect anticoagulants regard the medicines:
- A. *Fenilin, Omephin, Pelentan.
 - B. Tiklid, Parmidin.
 - C. Papaverin, Platyphyllin.
 - D. Nicotine acid.
 - E. Miscleronum, Lypoic acid.
1387. To the direct anticoagulants belongs:
- A. *Heparin.
 - B. Fenilin, Omephin.
 - C. Nicotine acid.
 - D. Streptokinase.
 - E. Aspirin.
1388. Saphenectomy is performed for:
- A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
1389. Troyanov-Trendelenburg's operation is performed for:
- A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
1390. The operation crossectomy is performed for:
- A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis

- E. Obliterative thrombangiitis
- 1391. Linton's operation is performed for:
 - A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
- 1392. Troyanov-Trendelenburg's operation is:
 - A. *Ligation of saphenofemoral junction
 - B. Removal of the main trunk of great saphenous vein
 - C. Suprafascial ligation of perforative veins
 - D. Subfascial ligation of perforative veins
 - E. Removal of collaterals of great saphenous vein
- 1393. Narath's operation is:
 - A. *Removal of collaterals of great saphenous vein
 - B. Ligation of saphenofemoral junction
 - C. Removal of the main trunk of great saphenous vein
 - D. Suprafascial ligation of perforative veins
 - E. Subfascial ligation of perforative veins
- 1394. Bebbcock's operation is:
 - A. *Removal of the main trunk of great saphenous vein
 - B. Removal of collaterals of great saphenous vein
 - C. Ligation of saphenofemoral junction
 - D. Suprafascial ligation of perforative veins
 - E. Subfascial ligation of perforative veins
- 1395. Cocketh's operation is:
 - A. *Suprafascial ligation of perforative veins
 - B. Removal of collaterals of great saphenous vein
 - C. Ligation of saphenofemoral junction
 - D. Removal of the main trunk of great saphenous vein
 - E. Subfascial ligation of perforative veins
- 1396. Linton's operation is:
 - A. *Subfascial ligation of perforative veins
 - B. Removal of collaterals of great saphenous vein
 - C. Ligation of saphenofemoral junction
 - D. Removal of the main trunk of great saphenous vein
 - E. Suprafascial ligation of perforative veins
- 1397. The Narath's operation is performed for:
 - A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
- 1398. The Bebbcock's operation is performed for:
 - A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
- 1399. The Cocketh's operation is performed for:
 - A. *Varicosity
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis

- E. Obliterative thrombangiitis
1400. The indication for the sclerotherapy is:
- A. *Reticular varicosity
 - B. Obliterative atherosclerosis
 - C. Thrombangiitis obliterans
 - D. Thrombosis of deep veins
 - E. Lymphedema of the extremity
1401. The indication for the sclerotherapy is:
- A. *Teleangiectasy
 - B. Obliterative atherosclerosis
 - C. Thrombangiitis obliterans
 - D. Thrombosis of deep veins
 - E. Lymphedema of the extremity
1402. The indication for the sclerotherapy is:
- A. *Relapse of varicosity
 - B. Obliterative atherosclerosis
 - C. Thrombangiitis obliterans
 - D. Thrombosis of deep veins
 - E. Lymphedema of the extremity
1403. The method of choice of the treatment of reticular varicosity is:
- A. *Sclerotherapy
 - B. Linton's operation
 - C. Saphenectomy
 - D. Endarterectomy
 - E. Autovenous by-passing
1404. The method of choice of the treatment of teleangiectasy is:
- A. *Sclerotherapy
 - B. Linton's operation
 - C. Saphenectomy
 - D. Endarterectomy
 - E. Autovenous by-passing
1405. The method of choice of the treatment of relapse of varicosity is:
- A. *Sclerotherapy
 - B. Linton's operation
 - C. Saphenectomy
 - D. Endarterectomy
 - E. Autovenous by-passing
1406. For the sclerotherapy used:
- A. *Fibro vein
 - B. Triumbrast
 - C. Verographin
 - D. Barium sulfate
 - E. Bilignost
1407. After saphenectomy the elastic compression is used for:
- A. *2-3 months
 - B. 3 days
 - C. 7 days
 - D. 2 weeks
 - E. 3-4 weeks
1408. For the treatment of the varicosity of the I degree the elastic stocks and bandages are used of the:
- A. *I degree of compression
 - B. II degree of compression
 - C. III degree of compression

- D. IV degree of compression
 - E. The compression stocks are not used
1409. For the treatment of varicosity of II degree the elastic stocks and bandages are used of the:
- A. *II degree of compression
 - B. I degree of compression
 - C. III degree of compression
 - D. IV degree of compression
 - E. The compression stocks are not used
1410. After saphenectomy the elastic stocks and bandages are used of the:
- A. *II degree of compression
 - B. I degree of compression
 - C. III degree of compression
 - D. IV degree of compression
 - E. The compression stocks are not used
1411. In patients with a trophic ulcer the elastic stocks and bandages are used of the:
- A. *III degree of compression
 - B. II degree of compression
 - C. I degree of compression
 - D. IV degree of compression
 - E. The compression stocks are not used
1412. Endotelon is used for the treatment of:
- A. *Varicosity
 - B. Ulcerous disease
 - C. Uterine bleeding
 - D. Atherosclerosis obliterans
 - E. Hypertensive disease
1413. Detralelex is used for the treatment of:
- A. *Varicosity
 - B. Ulcerous disease
 - C. Uterine bleeding
 - D. Atherosclerosis obliterans
 - E. Hypertensive disease
1414. Flebodia is used for the treatment of:
- A. *Varicosity
 - B. Ulcerous disease
 - C. Uterine bleeding
 - D. Atherosclerosis obliterans
 - E. Hypertensive disease
1415. Troksevasin is used for the treatment of:
- A. *Varicosity
 - B. Ulcerous disease
 - C. Uterine bleeding
 - D. Atherosclerosis obliterans
 - E. Hypertensive disease
1416. Why the saphenectomy does always begins with the ligation of saphenofemoral junction?
- A. *To prevent pulmonary embolism
 - B. To prevent bleeding
 - C. To prevent saphenofemoral reflux
 - D. To insert easy a venous extractor
 - E. To perform the sclerotherapy
1417. What is the basic sign of subcutaneous thrombophlebitis?
- A. *Painful cord along a great saphenous vein
 - B. Trophic ulcer
 - C. A gangrene of toes

- D. Absence of pulsation
 - E. Edema of the lower extremity
1418. Painful cord along a great saphenous vein is characteristic for:
- A. *Subcutaneous thrombophlebitis
 - B. Lymphedema
 - C. Atherosclerosis obliterans
 - D. Thrombangiitis obliterans
 - E. Thrombosis of deep veins
1419. Hyperemia and infiltrate along a great saphenous vein is characteristic for:
- A. *Subcutaneous thrombophlebitis
 - B. Lymphedema
 - C. Atherosclerosis obliterans
 - D. Thrombangiitis obliterans
 - E. Thrombosis of deep veins
1420. What are the clinical manifestations of subcutaneous thrombophlebitis?
- A. *Painful infiltrate and hyperemia along a great saphenous vein
 - B. Pale cold extremity, pulsation is absent
 - C. The hot cyanotic edematous lower extremity
 - D. The pale edematous lower extremity
 - E. Gangrene of toes and trophic ulcer
1421. What is the main danger of subcutaneous thrombophlebitis?
- A. *Pulmonary embolism
 - B. Venous insufficiency
 - C. Trophic ulcer
 - D. A gangrene of toes
 - E. Paralysis
1422. To the pulmonary embolism can lead:
- A. *Subcutaneous thrombophlebitis
 - B. Lymphedema
 - C. Obliterative atherosclerosis
 - D. Obliterative thrombangiitis
 - E. Raynaud's phenomenon
1423. Development of edema of the extremity in the case of subcutaneous thrombophlebitis specifies on:
- A. *Lesion of deep veins
 - B. Infection
 - C. Cardiac insufficiency
 - D. Kidney insufficiency
 - E. Development of lymphostasis
1424. What is the typical treatment of subcutaneous thrombophlebitis?
- A. *Surgical treatment
 - B. Anticoagulants
 - C. Thrombolytics
 - D. Spasmolytics
 - E. Vitamins
1425. What is the main cause of the development of subcutaneous thrombophlebitis?
- A. *Varicosity
 - B. Arterio-venous fistulas
 - C. Venous hypoplasia
 - D. Diabetes mellitus
 - E. Obliterative atherosclerosis
1426. What is the background of the development of subcutaneous thrombophlebitis?
- A. *Inflammatory reaction of venous wall
 - B. Atherosclerotic plaque

- C. Diabetic angiopathy
 - D. Lipidemia
 - E. Anemia
1427. What factor contributes to the development of subcutaneous thrombophlebitis?
- A. *Trauma
 - B. Diabetes mellitus
 - C. Obliterative atherosclerosis
 - D. Anemia
 - E. Hemophilia
1428. What medicinal medicines can result in formation of subcutaneous thrombophlebitis?
- A. *Contraceptives
 - B. Anticoagulants
 - C. Fibrinolytics
 - D. Antiaggregants
 - E. Nonsteroid anti-inflammatory medicines
1429. Mondor's disease is:
- A. *Venous thrombosis of front chest wall
 - B. Ileofofemoral venous thrombosis
 - C. Thrombosis of subclavian vein
 - D. Thrombosis of vena cava inferior
 - E. Thrombosis of vena cava superior
1430. Venous thrombosis of front chest wall is:
- A. *Mondor's disease
 - B. Paget-Shretter's syndrome
 - C. Badda-Chiari's syndrome
 - D. Raynaud's phenomenon
 - E. Disease of Madelung
1431. A subcutaneous thrombophlebitis often associated with the following disease:
- A. *Obliterative thromboangitis
 - B. Diabetes mellitus
 - C. Obliterative atherosclerosis
 - D. Nonspecific aorto-arteriitis
 - E. Hemophilia
1432. A subcutaneous thrombophlebitis often associated with the following disease:
- A. *Varicosity
 - B. Diabetes mellitus
 - C. Obliterative atherosclerosis
 - D. Nonspecific aorto-arteriitis
 - E. Hemophilia
1433. What is the typical treatment of subcutaneous thrombophlebitis?
- A. *Surgical treatment
 - B. Anticoagulants
 - C. Thrombolytics
 - D. Spasmolytics
 - E. Vitamins
1434. What operation is performed for a subcutaneous thrombophlebitis?
- A. *Saphenectomy
 - B. Trombintimectomy
 - C. Autovenous by-passing
 - D. Prosthetic graft repairing of vessel
 - E. Amputation
1435. Saphenectomy is performed for:
- A. *Subcutaneous thrombophlebitis
 - B. Thrombosis of deep veins

- C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
1436. Troyanov-Trendelenburg's operation is performed for:
- A. *Subcutaneous thrombophlebitis
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
1437. The operation crosssectomy is performed for:
- A. *Subcutaneous thrombophlebitis
 - B. Thrombosis of deep veins
 - C. Lymphedema
 - D. Obliterative atherosclerosis
 - E. Obliterative thrombangiitis
1438. What are the indications for an urgent operation?
- A. *Acute ascending thrombophlebitis
 - B. Subcutaneous thrombophlebitis of shin
 - C. Deep venous thrombosis of shin
 - D. Paget-Shretter's syndrome
 - E. Badda-Chiari's syndrome
1439. A testimony for an urgent saphenectomy is:
- A. *Acute ascending thrombophlebitis
 - B. Subcutaneous thrombophlebitis of shin
 - C. Deep venous thrombosis of shin
 - D. Paget-Shretter's syndrome
 - E. Badda-Chiari's syndrome
1440. What is the acute ascending thrombophlebitis?
- A. *Localization of blood clot in saphenofemoral junction
 - B. Total thrombotic lesion of vein
 - C. Partial thrombotic lesion of vein
 - D. Localization of blood clot in a subclavian vein
 - E. Localization of blood clot in the veins of front chest wall
1441. What is the main cause of the development of thrombosis of deep veins?
- A. *Subcutaneous thrombophlebitis
 - B. Varicosity
 - C. Arterio-venous fistulas
 - D. Diabetes mellitus
 - E. Obliterative atherosclerosis
1442. What factor contributes to the development of thrombosis of deep veins?
- A. *Operating trauma
 - B. Diabetes mellitus
 - C. Obliterative atherosclerosis
 - D. Anemia
 - E. Hemophilia
1443. What factor contributes to the development of thrombosis of deep veins?
- A. *Trauma
 - B. Diabetes mellitus
 - C. Obliterative atherosclerosis
 - D. Anemia
 - E. Hemophilia
1444. Localization of blood clot in saphenofemoral junction is:
- A. *Acute ascending thrombophlebitis
 - B. Ileo-femoral venous thrombosis

- C. Thrombosis of deep veins
 - D. Paget-Shretter's syndrome
 - E. Badda-Chiari's syndrome
1445. What are the clinical manifestations of deep venous thrombosis of shin?
- A. *Edema of shins with cyanosis
 - B. A total edema of the lower extremity with cyanosis
 - C. Edema of both lower extremities
 - D. Pale lower extremity, pulsation is absent
 - E. Varicosity of shin with a trophic ulcer
1446. What is the main danger of subcutaneous thrombophlebitis?
- A. *Pulmonary embolism
 - B. Venous insufficiency
 - C. Trophic ulcer
 - D. A gangrene of toes
 - E. Paralysis
1447. The Homan's sign is typical for:
- A. *Thrombosis of deep veins
 - B. Subcutaneous thrombophlebitis
 - C. Varicosity
 - D. Obliterative atherosclerosis
 - E. Diabetes mellitus
1448. Homan's sign is:
- A. *Pain in the muscles of shin, which increases by back flexing of foot
 - B. Intermittent claudication at physical exertion
 - C. Absence of pulsation in a cold environment
 - D. Symptom of caught shoe
 - E. Absence of hepatic dullness
1449. How is the symptom named by author, when back flexing of foot increases the pain in the muscles of shin?
- A. *Homan's sign
 - B. Lovenberg's sign
 - C. Mondor's sign
 - D. Murphy's sign
 - E. Paget-Shretter's syndrome
1450. The Lovenberg's sign is typical for:
- A. *Thrombosis of deep veins
 - B. Obliterative atherosclerosis
 - C. Diabetes mellitus
 - D. Subcutaneous thrombophlebitis
 - E. Varicosity
1451. Lovenberg's sign is:
- A. *Pain in the muscles of shin after imposition of cuff with pressure 80-100 mm of Hg.
 - B. Intermittent claudication at physical exertion
 - C. Absence of pulsation in a cold environment
 - D. Absence of hepatic dullness
 - E. Pain in the muscles of shin, caused by the back flexing of foot
1452. What are the clinical manifestations of ileofemoral venous thrombosis?
- A. *A total edema of the lower extremity with cyanosis
 - B. Edema of shins with cyanosis
 - C. Edema of both lower extremities
 - D. Pale lower extremity, pulsation is absent
 - E. Varicosity of shin with a trophic ulcer
1453. Intermittent claudication is characteristic for:
- A. *Atherosclerosis of lower extremities

- B. Thrombosis of deep veins
 - C. Pancreatitis
 - D. Varicosity
 - E. Cholecystitis
1454. Dilating pain in extremity is characteristic for:
- A. *Thrombosis of deep veins
 - B. Teleangiektaziy
 - C. Retikulyarnogo varicosity
 - D. Mikotichnogo of lesion
 - E. Diseasees of Madelunga
1455. Edema of the extremity is characteristic for:
- A. *Thrombosis of deep veins
 - B. Teleangiectasia
 - C. Reticular varicosity
 - D. Mycotic lesion
 - E. Madelung's disease
1456. Cyanosis of the skin of extremity is characteristic for:
- A. *Thrombosis of deep veins
 - B. Teleangiectasia
 - C. Reticular varicosity
 - D. Mycotic lesion
 - E. Madelung's disease
1457. Intermittent claudication is characteristic for:
- A. *Atherosclerosis obliterans
 - B. Thrombosis of deep veins
 - C. Teleangiectasia
 - D. Reticular varicosity
 - E. Madelung's disease
1458. In thrombosis of deep veins the patients complain on:
- A. *Dilating pain in extremity
 - B. Intermittent claudication
 - C. Coldness of the extremity
 - D. Varicosity
 - E. Presence of trophic ulcer
1459. In thrombosis of deep veins the patients complain on:
- A. *Edema of extremities
 - B. Intermittent claudication
 - C. Coldness of the extremity
 - D. Poblidninnya of the extremity
 - E. Presence of trophic ulcer
1460. In thrombosis of deep veins the patients complain on:
- A. *Cyanosis of the skin of the extremity
 - B. Intermittent claudication
 - C. Coldness of the extremity
 - D. Pale extremity
 - E. Presence of trophic ulcer
1461. What is the classic clinical triad for a deep venous thrombosis?
- A. *Pain, edema, cyanosis of the extremity
 - B. Pale lower extremity, absent pulsation, edema
 - C. The varicosity of the lower extremity, trophic ulcer, edema
 - D. Pain, paralysis, cyanosis
 - E. Pain, paralysis, trophic ulcer
1462. What is the characteristic clinical sign of deep venous thrombosis?
- A. *Edema of extremities

- B. Pulsation is absent
 - C. Trophic ulcer
 - D. Paralysis
 - E. Gangrene
1463. What are the basic collaterals for compensation of venous outflow in thrombosis of distal segment of vena cava inferior?
- A. *Azygos and hemiazygos veins
 - B. Lumbar veins
 - C. Femoral veins
 - D. Kidney veins
 - E. Vena cava superior
1464. What are the clinical manifestations of thrombosis of distal segment of vena cava inferior?
- A. *Edema of lower half of body and lower extremities
 - B. Trophic ulcers of lower extremities
 - C. Gangrene of lower extremities
 - D. Kidney insufficiency
 - E. Insufficiency of liver
1465. Edema of lower extremities is characteristic for:
- A. *Thrombosis of vena cava inferior
 - B. Leriche's syndrome
 - C. Aortic aneurysm
 - D. Paget-Shretter's syndrome
 - E. Thrombosis of vena cava superior
1466. Disturbance of sensation is characteristic for:
- A. *Embolisms of the artery
 - B. Varicosity
 - C. Thrombosis of deep veins
 - D. Subcutaneous thrombophlebitis
 - E. Lymphedema
1467. Paresis of the extremity is characteristic for:
- A. *Arterial thrombosis
 - B. Varicosity
 - C. Thrombosis of deep veins
 - D. Subcutaneous thrombophlebitis
 - E. Lymphedema
1468. Plegia of the extremity is characteristic for:
- A. *Embolisms of the artery
 - B. Varicosity
 - C. Thrombosis of deep veins
 - D. Subcutaneous thrombophlebitis
 - E. Lymphedema
1469. Absence of pulsation on extremity is characteristic for:
- A. *Embolisms of the artery
 - B. Varicosity
 - C. Thrombosis of deep veins
 - D. Subcutaneous thrombophlebitis
 - E. Postphlebitic syndrome
1470. Muscular contracture of the extremity is characteristic for:
- A. *Embolisms of the artery
 - B. Varicosity
 - C. Thrombosis of deep veins
 - D. Subcutaneous thrombophlebitis
 - E. Postphlebitic syndrome
1471. Development of kidney insufficiency is characteristic for:

- A. *Thrombosis of renal segment of vena cava inferior
 - B. Thrombosis of distal segment of vena cava inferior
 - C. Thrombosis of hepatic segment of vena cava inferior
 - D. Paget-Shretter's syndrome
 - E. Thrombosis of vena cava superior
1472. Development of Badda-Chiari's syndrome is characteristic for:
- A. *Thrombosis of hepatic segment of vena cava inferior
 - B. Thrombosis of renal segment of vena cava inferior
 - C. Thrombosis of distal segment of vena cava inferior
 - D. Paget-Shretter's syndrome
 - E. Thrombosis of vena cava superior
1473. What are the clinical manifestations of thrombosis of kidney segment of vena cava inferior?
- A. *Kidney insufficiency
 - B. Trophic ulcers of lower extremities
 - C. Edema of lower half of body and lower extremities
 - D. Gangrene of lower extremities
 - E. Insufficiency of liver
1474. What are the clinical manifestations of thrombosis the hepatic segment of vena cava inferior?
- A. *Badda-Chiari's syndrome
 - B. Trophic ulcers of lower extremities
 - C. Edema of lower half of body and lower extremities
 - D. Gangrene of lower extremities
 - E. Kidney insufficiency
1475. Badda-Chiari's syndrome is:
- A. *Thrombosis of hepatic veins
 - B. Ileo-femoral venous thrombosis
 - C. Thrombosis of subclavian vein
 - D. Thrombosis of kidney veins
 - E. Thrombosis of vein of gate
1476. What are the clinical manifestations of Badda-Chiari's syndrome?
- A. *Portal hypertension
 - B. Trophic ulcer of lower extremities
 - C. Respiratory insufficiency
 - D. Gangrene of lower extremities
 - E. Hematuria
1477. Thrombosis of axillary and subclavian vein is:
- A. *Paget-Shretter's syndrome
 - B. Badda-Chiari's syndrome
 - C. Cushing's syndrome
 - D. Leriche's syndrome
 - E. Syndrome of Morgani-Adams-Stokes
1478. Paget-Shretter's syndrome is:
- A. *Thrombosis of axillary and subclavian vein
 - B. Occlusion of bifurcation of aorta
 - C. Thrombosis of vena cava superior
 - D. Thrombosis of hepatic veins
 - E. Thrombosis of vena cava inferior
1479. In Paget-Shretter's syndrome the patients complain on:
- A. *Edema of upper extremity
 - B. Intermittent claudication
 - C. Coldness of the extremity
 - D. Pale extremity
 - E. Presence of trophic ulcer

1480. In Paget-Shretter's syndrome the patients complain on:
- A. *Edema of upper extremity
 - B. Edema of the lower extremity
 - C. Edema of both lower extremities
 - D. Edema of lower half of body
 - E. Pulsation of neck veins
1481. In Paget-Shretter's syndrome the patients complain on:
- A. *Cyanosis of upper extremity
 - B. Cyanosis of the lower extremity
 - C. Varicose veins of the lower extremity
 - D. Cyanosis of lower half of body
 - E. Varicose veins of front abdominal wall
1482. What is the main cause of Paget-Shretter's syndrome?
- A. *Compression of subclavian vein in costo-clavicular space
 - B. Compression of common iliac vein by a tumour
 - C. Atherosclerotic lesion of arteries of upper extremities
 - D. Atherosclerotic lesion of carotid
 - E. Aortic aneurysm
1483. Compression of subclavian vein is a risk factor for:
- A. *Paget-Shretter's syndrome
 - B. Badda-Chiari's syndrome
 - C. Cushing's syndrome
 - D. Leriche's syndrome
 - E. Morgani-Adams-Stokes syndrome
1484. Implantation of pace-maker is a risk factor for:
- A. *Paget-Shretter's syndrome
 - B. Badda-Chiari's syndrome
 - C. Cushing's syndrome
 - D. Leriche's syndrome
 - E. Morgani-Adams-Stokes syndrome
1485. The mastectomy operation is a risk factor for:
- A. *Paget-Shretter's syndrome
 - B. Badda-Chiari's syndrome
 - C. Cushing's syndrome
 - D. Leriche's syndrome
 - E. Morgani-Adams-Stokes syndrome
1486. What are the clinical manifestations of Paget-Shretter's syndrome?
- A. *Edema of upper extremity with cyanosis
 - B. A pale hand, pulsation is absent
 - C. Pale lower extremity, pulsation is absent
 - D. Varicosity of the lower extremity with a trophic ulcer
 - E. Edema of both lower extremities with cyanosis
1487. What is the main cause of the syndrome of vena cava superior?
- A. *Tumour of mediastinum
 - B. Subcutaneous thrombophlebitis
 - C. Deep venous thrombosis of lower extremities
 - D. Tumour of retroperitoneal space
 - E. Tumour of abdominal region
1488. What are the clinical manifestations of the syndrome of vena cava superior?
- A. *Edema of upper half of body and hands with cyanosis
 - B. Trophic ulcer of upper extremities
 - C. Edema of lower half of body and lower extremities with cyanosis
 - D. Absence of pulsation of carotids
 - E. Absence of pulsation of arteries of upper extremities

1489. What is the level of the absent pulsation in thrombosis of deep veins?
- A. *Preserved on all levels
 - B. Tibial arteries
 - C. Popliteal artery
 - D. Femoral artery
 - E. Aorta
1490. Which among the roentgenologic methods of diagnostics is the most informative for thrombosis of deep veins?
- A. *Phlebography
 - B. Arteriography
 - C. X-ray examination of abdomen
 - D. X-ray examination with barium
 - E. X-ray examination of the extremity
1491. Which among the methods of diagnostics is the most informative for thrombosis of deep veins?
- A. *Ultrasound examination
 - B. Arteriography
 - C. X-ray examination of abdomen
 - D. X-ray examination of chest
 - E. X-ray examination of the extremity
1492. What method is used for the diagnostics of thrombosis of deep veins?
- A. *Ultrasound duplex scanning
 - B. Coagulogram
 - C. Reovasography
 - D. Ultrasound Doppler examination
 - E. Arteriography
1493. What are the indications for endarterectomy:
- A. *Obliterative atherosclerosis
 - B. Subcutaneous thrombophlebitis
 - C. Leriche's syndrome
 - D. Thrombosis of deep veins
 - E. Varicosity
1494. What group of medicinal drugs the heparin belongs to?
- A. *Direct anticoagulants
 - B. Indirect anticoagulants
 - C. Thrombolytics
 - D. Fibrinolytics
 - E. Antiinflammatory drugs
1495. What group of medicinal drugs the Klexan belongs to?
- A. *Direct anticoagulants
 - B. Indirect anticoagulants
 - C. Thrombolytics
 - D. Fibrinolytics
 - E. Antiinflammatory drugs
1496. What group of medicinal drugs the Sincumar belongs to?
- A. *Indirect anticoagulants
 - B. Direct anticoagulants
 - C. Thrombolytics
 - D. Fibrinolytics
 - E. Antiinflammatory drugs
1497. What group of medicinal drugs the Fenillin belongs to?
- A. *Indirect anticoagulants
 - B. Direct anticoagulants
 - C. Thrombolytics

- D. Fibrinolytics
 - E. Antiinflammatory drugs
1498. What group of medicinal drugs the Actilise belongs to?
- A. *Thrombolytics
 - B. Indirect anticoagulants
 - C. Direct anticoagulants
 - D. Venotonics
 - E. Antiinflammatory drugs
1499. What group of medicinal drugs the Streptokinase belongs to?
- A. *Thrombolytics
 - B. Indirect anticoagulants
 - C. Direct anticoagulants
 - D. Venotonics
 - E. Antiinflammatory drugs
1500. What group of medicinal drugs the Detralex belongs to?
- A. *Venotonics
 - B. Thrombolytics
 - C. Indirect anticoagulants
 - D. Direct anticoagulants
 - E. Antiinflammatory drugs
1501. What group of medicinal drugs the Venoplant belongs to?
- A. *Venotonics
 - B. Thrombolytics
 - C. Indirect anticoagulants
 - D. Direct anticoagulants
 - E. Antiinflammatory drugs
1502. What medical drugs belong to the direct anticoagulants?
- A. *Heparin
 - B. Syncumarum, Fenilin
 - C. Streptokinase, Urokinase
 - D. Trentalum, Pentoxiphyllin
 - E. Diklofenak, Naklofen
1503. What medical drugs belong to the direct anticoagulants?
- A. *Fraxiparin, Kleksan
 - B. Syncumarum, Fenilin
 - C. Streptokinase, Urokinase
 - D. Trentalum, Pentoxiphyllin
 - E. Diklofenak, Naklofen
1504. What medical drugs belong to the indirect anticoagulants?
- A. *Syncumarum, Fenilin
 - B. Fraxiparin, Kleksan
 - C. Streptokinase, Urokinase
 - D. Trentalum, Pentoxiphyllin
 - E. Diklofenak, Naklofen
1505. What medical drugs belong to fibrinolytics?
- A. *Streptokinase, Urokinase
 - B. Heparin
 - C. Syncumarum, Fenilin
 - D. Trentalum, Pentoxiphyllin
 - E. Diklofenak, Naklofen
1506. What is the dose of heparin for the treatment of thrombophlebitis?
- A. *5000-10000 Units each 4-6 hours
 - B. 1000-2000 Units each 4-6 hours
 - C. 1000-2000 Units daily

- D. 2500 Units every hour
 - E. 20000-40000 Units each 12 hours
1507. What is the initial dose of Streptokinase?
- A. *250000 Units
 - B. 5000 Units
 - C. 10000 Units
 - D. 100000 Units
 - E. 1000000 Units
1508. What drugs are used in the overdose of heparin?
- A. *Protamine sulfate
 - B. Fraxiparin, Kleksan
 - C. Streptokinase, Urokinase
 - D. Trentalum, Pentoxiphyllin
 - E. Diklofenak, Naklofen
1509. What drugs are used in the overdose of Streptokinase?
- A. *Aminocapronic acid, Trasyololum
 - B. Fraxiparin, Kleksan
 - C. Protamine sulfate
 - D. Trentalum, Pentoxiphyllin
 - E. Diklofenak, Naklofen
1510. What medical drugs belong to venotonics?
- A. *Detralex, Flebodia
 - B. Heparin
 - C. Syncumarum, Fenilin
 - D. Papaverin, Nospanum
 - E. Diklofenak, Naklofen
1511. What are the normal measures of prothrombine index?
- A. *85-100 %
 - B. 10-20 %
 - C. 30-60 %
 - D. 50-70 %
 - E. 100-120 %
1512. What prothrombine index must be during the treatment of venous thrombosis?
- A. *50-70 %
 - B. 10-20 %
 - C. 30-40 %
 - D. 85-100 %
 - E. 100-120 %
1513. What indexes of coagulogram control the dosage of anticoagulants?
- A. *Prothrombine index
 - B. The amount of thrombocytes in the blood
 - C. Level of blood fibrinogen
 - D. Thrombotest
 - E. Time of recalcification
1514. What is the normal level of blood fibrinogen?
- A. *2-4 g/l
 - B. 6-8 g/l
 - C. 10-16 g/l
 - D. 30-50 g/l
 - E. 75-100 g/l
1515. What indexes of coagulogram control the dosage of thrombolytics?
- A. *Level of blood fibrinogen
 - B. The amount of thrombocytes in the blood
 - C. Prothrombine index

- D. Thrombotest
- E. Time of recalcification
- 1516. What is the norm of time of recalcification?
 - A. *60-120 seconds
 - B. 0.5-2 seconds
 - C. 10-40 seconds
 - D. 40-60 seconds
 - E. 4-8 minutes
- 1517. What are the normal measures of the thrombotest?
 - A. *IV-V degree
 - B. I-II degree
 - C. II-III degree
 - D. VI-VIII degree
 - E. X-XII degree
- 1518. What is the normal amount of thrombocytes in blood?
 - A. *180-320?10⁹/l
 - B. 20-40?10⁹/l
 - C. 40-120?10⁹/l
 - D. 420-650?10⁹/l
 - E. 600-1000?10⁹/l
- 1519. What method is used for the diagnostics of deep venous thrombosis?
 - A. *Ultrasound doppler examination
 - B. General and biochemical analyses of blood
 - C. Coagulogram
 - D. Reovasography
 - E. Arteriography
- 1520. What treatment is used for the deep venous thrombosis of shin?
 - A. *Conservative treatment
 - B. Thrombectomy
 - C. Saphenectomy
 - D. Autovenous by-passing
 - E. Prosthetic graft repairing of vessel
- 1521. What treatment is used for ileofemoral venous thrombosis?
 - A. *Conservative treatment
 - B. Saphenectomy
 - C. Thrombectomy
 - D. Autovenous by-passing
 - E. Prosthetic graft repairing of vessel
- 1522. Iliofemoral prosthetic by-passing is performed for:
 - A. *Obliterative atherosclerosis
 - B. Postphlebotic syndrome
 - C. Varicosity
 - D. Thrombosis of deep veins
 - E. Subcutaneous thrombophlebitis
- 1523. Femoro-popliteal autovenous by-passing is performed for:
 - A. *Obliterative atherosclerosis
 - B. Postphlebotic syndrome
 - C. Varicosity
 - D. Thrombosis of deep veins
 - E. Subcutaneous thrombophlebitis
- 1524. Bifurcation aorto-femoral prosthetic by-passing is performed for:
 - A. *Obliterative atherosclerosis
 - B. Postphlebotic syndrome
 - C. Varicosity

- D. Thrombosis of deep veins
 - E. Subcutaneous thrombophlebitis
1525. Decompression osteoperforation is performed for:
- A. *Obliterative atherosclerosis
 - B. Postphlebitic syndrome
 - C. Varicosity
 - D. Thrombosis of deep veins
 - E. Subcutaneous thrombophlebitis
1526. A sympathectomy is performed for:
- A. *Obliterative thrombangiitis
 - B. Obliterative atherosclerosis
 - C. Varicosity
 - D. Thrombosis of deep veins
 - E. Subcutaneous thrombophlebitis
1527. When is the operation possible in ileofemoral venous thrombosis?
- A. *Duration of disease less than 5 days
 - B. Duration of disease 7-14 days
 - C. Duration of disease 21-28 days
 - D. Duration of disease of 2-3 months
 - E. In a "cold" period
1528. What is the level of arterial pulsation absence in the thrombosis of deep veins?
- A. *Preserved on all levels
 - B. Arteries of foot
 - C. Femoral artery
 - D. Popliteal artery
 - E. Common carotid
1529. What method of prophylaxis of pulmonary embolism is used during the operation for ileofemoral venous thrombosis?
- A. *Introduction of ballon catheter to the vena cava inferior during the operation
 - B. The elastic bandage during the operation
 - C. Application of anticoagulants during the operation
 - D. Application of fibrinolytics during the operation
 - E. Introduction of ballon catheter to the vena cava superior during the operation
1530. What method of prophylaxis of pulmonary embolism is used during the operation for ileofemoral venous thrombosis?
- A. *Introduction of cava-filters to the vena cava inferior during the operation
 - B. Application of anticoagulants during the operation
 - C. Application of fibrinolytics during the operation
 - D. The elastic bandage during the operation
 - E. Introduction of ballon catheter to the vena cava superior during the operation
1531. When after a deep venous thrombosis do we make the diagnosis of postphlebitic syndrome?
- A. *In 6 months
 - B. In 10-14 days
 - C. In 1 month
 - D. Through 2 months
 - E. In 1 year
1532. What are the clinical manifestations of postphlebitic syndrome?
- A. *The expressed edema, secondary varicosity
 - B. Subcutaneous thrombophlebitis
 - C. Arterial ischemia
 - D. Contracture of joints
 - E. Paralysis
1533. What is the cause of postphlebitic syndrome?
- A. *Thrombosis of deep veins

- B. Arterial embolism
 - C. Arterial thrombosis
 - D. Subcutaneous thrombophlebitis
 - E. Varicosity
1534. The pallor of the skin is characteristic for:
- A. *Embolisms of the artery
 - B. Varicosity
 - C. Deep vein thrombosis
 - D. Postphlebitic syndrome
 - E. Phlegmon of the extremity
1535. A coldness of the extremity is characteristic for:
- A. *Arterial thrombosis
 - B. Varicosity
 - C. Deep vein thrombosis
 - D. Postphlebitic syndrome
 - E. Phlegmon of the extremity
1536. What is the base of postphlebitic syndrome?
- A. *Valvular incompetence
 - B. Venous occlusion
 - C. Arterial occlusion
 - D. Nervous damage
 - E. Gangrene of the extremity
1537. What clinical form of postphlebitic syndrome does not exist?
- A. *Gangrenous
 - B. Sclerotic
 - C. Varicose
 - D. Edematous
 - E. Ulcerous
1538. What is the typical follow up of deep venous thrombosis?
- A. *Recanalization of blood clot with valvular incompetence
 - B. Complete obliteration of vein
 - C. Partial obliteration of vein
 - D. Varicose expansion
 - E. Arterio-venous fistula
1539. What are the clinical manifestations of postphlebitic syndrome?
- A. *Signs of venous insufficiency
 - B. Signs of nervous damage
 - C. Signs of vascular dystonia
 - D. Signs of arterial insufficiency
 - E. Limitation of movements of the lower extremity
1540. What medicines belong to thrombolytic medicines?
- A. *Fibrinolysin, Streptokinase.
 - B. Nicotine acid.
 - C. Papaverin, Platyphyllin.
 - D. Fenilin, Omefin.
 - E. Vasaprostan
1541. What clinical sign is characteristic for a postphlebitic syndrome?
- A. *Edema of leg
 - B. Absence of pulsation
 - C. Paralysis
 - D. Gangrene
 - E. Absence of sensation
1542. What is the characteristic sign of the I stage of postphlebitic syndrome?
- A. *Transitory edema

- B. Permanent edema
 - C. Hyperpigmentation
 - D. Trophic ulcer
 - E. Gangrene
1543. What is the characteristic sign of the II stage of postphlebitic syndrome?
- A. *Hyperpigmentation
 - B. Heavy sensation
 - C. Transitory edema
 - D. Open trophic ulcer
 - E. Healed trophic ulcer
1544. What is the characteristic sign of the II stage of postphlebitic syndrome?
- A. *Permanent edema
 - B. Heavy sensation
 - C. Transitory edema
 - D. Open trophic ulcer
 - E. Healed trophic ulcer
1545. What is the characteristic sign of the III stage of postphlebitic syndrome?
- A. *Trophic ulcer
 - B. Heavy sensation
 - C. Transitory edema
 - D. Permanent edema
 - E. Hyperpigmentation
1546. What clinical form of postphlebitic syndrome is characterized by absence of varicosity?
- A. *Sclerotic
 - B. Varicose
 - C. Edematous
 - D. Ulcerous
 - E. Necrotizing
1547. What clinical form of postphlebitic syndrome is characterized by the secondary varicosity?
- A. *Varicose
 - B. Sclerotic
 - C. Edematous
 - D. Ulcerous
 - E. Necrotizing
1548. What clinical form of postphlebitic syndrome is caused by a venous obstruction?
- A. *Edematous
 - B. Sclerotic
 - C. Varicose
 - D. Ulcerous
 - E. Necrotizing
1549. What is the physiology norm of fibrinogen in the blood serum?
- A. *2.2 - 4.2 g/l.
 - B. 5.5 - 7.5 g/l.
 - C. 5.6 - 8.0 g/l.
 - D. 5.9 - 9.2 g/l.
 - E. 7.8 - 5.5 g/l.
1550. What is the physiology norm of prothrombine index?
- A. *80 - 100 %.
 - B. 40 - 60 %.
 - C. 110 - 130 %.
 - D. 15 - 45 %.
 - E. 0 - 10 %.
1551. To the absolute contraindications for the usage of anticoagulants belongs:
- A. *Bleeding of any localization, hemorrhagic diateses

- B. Aplastic anemia
 - C. Respiratory insufficiency
 - D. Cardiac insufficiency
 - E. Insufficiency of cerebral blood circulation
1552. To the activators of Fibrinolysis belong:
- A. *Nicotinic acid, Complamine
 - B. Fenilin, Omefin
 - C. Gastrocepin, Venter
 - D. Vitamins B
 - E. Vitamins A, C.
1553. Medicines of antiaggregant activity:
- A. *Reopolyglucin, Trental, aspirin, курантіл
 - B. Omefin, Fenillin, Pelentan
 - C. Cinarizin, Sermion, Solcoseril
 - D. Fentalamin, Tropaphen
 - E. Simvastatin
1554. Medicines of thrombolytic action:
- A. *Fibrinolysin, Streptokinase
 - B. Korglucon, strophanthin
 - C. Celonid, Lantozid
 - D. Reopolyglucin, polyhybrid
 - E. Trentalum, Pentoxiphyllin
1555. The embolectomy is performed for:
- A. *Embolisms of the artery
 - B. Varicosity
 - C. Thrombosis of deep veins
 - D. Postphlebotic syndrome
 - E. Phlegmon of the extremity
1556. Trombintimectiony is performed for:
- A. *Arterial thrombosis
 - B. Thrombosis of deep veins
 - C. Embolisms of the artery
 - D. Subcutaneous thrombophlebitis
 - E. Varicosity
1557. The collateral autovenous by-passing is performed for:
- A. *Arterial thrombosis
 - B. Thrombosis of deep veins
 - C. Embolisms of the artery
 - D. Subcutaneous thrombophlebitis
 - E. Varicosity
1558. Methods of control of the coagulative and fibrinolytic systems of blood:
- A. *Coagulogram
 - B. General blood analysis
 - C. Determination of speed of local blood stream
 - D. Hemogram
 - E. Biochemical blood test
1559. In the overdosing of heparin enter:
- A. *1% solution of Protamini sulfate
 - B. Cimiton, Lobeline
 - C. Amitriptilin
 - D. 2,5% solution of Aminasini
 - E. Adrenalin
1560. In bleeding, caused by the indirect anticoagulants prescribed:
- A. *Vikasol

- B. Pipolphenum
 - C. Digitoksin
 - D. Hidrokortizon
 - E. Petoksifilin
1561. In fibrinolytic bleeding prescribed:
- A. *Aminokapronovu acid
 - B. 1 % solution of Ombeni, 0,5 % solution of hydrocortison, prednisolon
 - C. 0,1 % solution of nicotine acid
 - D. 5 % solution of аскорбінової acid
 - E. AlprostThe
1562. What is the purpose of the usage of thrombolytic and anticoagulating therapy:
- A. *Lysis of blood clot, prophylaxis of distribution of blood clot
 - B. Improvement of function of liver
 - C. To development of collateral net
 - D. A removal of the spasm on peripheral vessels
 - E. Improvement of microcirculation
1563. Name the unpharmacological methods of correction of the coagulative system of blood.
- A. Hemodilution.
 - B. Hemodialysis.
 - C. Hemosorbtion.
 - D. A forced diuresis.
 - E. Lymphosorbtion.
1564. Where does a small saphenous vein drain?
- A. *In a popliteal vein.
 - B. In a great saphenous vein.
 - C. In a femoral vein.
 - D. In a general iliac vein.
 - E. In a vena cava inferior.
1565. What method is used for the diagnostics of valvular incompetence in postphlebitic syndrome?
- A. *Ultrasound duplex scanning
 - B. Coagulogram
 - C. Reovasography
 - D. Ultrasound doppler examination
 - E. Arteriography
1566. What method is used for the estimation of the passability of deep veins in postphlebitic syndrome?
- A. *Ultrasound examination
 - B. Coagulogram
 - C. Reovasography
 - D. ECG
 - E. Arteriography
1567. What treatment is indicated for a postphlebitic syndrome?
- A. *Saphenectomy
 - B. Conservative treatment
 - C. Scelerotherapy
 - D. Troyanov-Trendelenburg's operation
 - E. Saphenectomy with the subfascial ligation of perforative veins (Linton's operation)
1568. The trophic ulcer of the lower third of shin is characteristic for:
- A. *Postphlebitic syndrome
 - B. Thrombosis of deep veins
 - C. Thrombangiitis obliterans
 - D. Leriche's syndrome
 - E. Embolisms of femoral artery

1569. The basic source of emboli in pulmonary embolism is:
- A. *System of vena cava inferior
 - B. Subclavian artery
 - C. Left ventricle
 - D. Femoral artery
 - E. Abdominal part of aorta
1570. The cause of pulmonary embolism is:
- A. *Thrombosis of deep veins of lower extremities
 - B. Bacterial endocarditis
 - C. Myocardial infarction
 - D. Abdominal aortic aneurysm
 - E. Obliterative atherosclerosis of lower extremities
1571. To massive pulmonary embolism belongs:
- A. *Occlusion of basic trunk of pulmonary artery and its branches
 - B. Embolism of lobe branches of pulmonary artery
 - C. Embolism of segmental branches of pulmonary artery
 - D. Embolism of lobe branches of pulmonary artery with iliofemoral vein thrombosis
 - E. Embolism of segmental branches of pulmonary artery with subcutaneous thrombophlebitis
1572. Which among the symptoms is characteristic for pulmonary embolism?
- A. *Pain behind breastbone
 - B. Regurgitation
 - C. Intermittent claudication
 - D. Paradoxical breathing
 - E. Syndrome of thoracic outlet
1573. Which among the symptoms is characteristic for pulmonary embolism?
- A. *Dyspnoea
 - B. Regurgitation
 - C. Intermittent claudication
 - D. Paradoxical breathing
 - E. Syndrome of thoracic outlet
1574. Which among the symptoms is characteristic for pulmonary embolism?
- A. *Shock
 - B. Regurgitation
 - C. Intermittent claudication
 - D. Paradoxical breathing
 - E. Syndrome of thoracic outlet
1575. EKG change in pulmonary embolism is characterized by:
- A. *The overload of right parts of heart
 - B. The overload of left parts of heart
 - C. Heart premature beats
 - D. Atrial fibrillation
 - E. By the total block of left leg of His bundle
1576. A typical roentgenologic sign of pulmonary embolism is:
- A. *Wedge-shaped atelectasis
 - B. Rounded shadow
 - C. Pneumothorax
 - D. Caverns
 - E. "Rat tail" sign
1577. A typical roentgenologic sign of pulmonary embolism is:
- A. *Pleural effusion
 - B. Rounded shadow
 - C. Pneumothorax
 - D. Caverns

- E. "Rat tail" sign
1578. Which among roentgenologic signs is not characteristic for pulmonary embolism?
- *Rounded shade
 - Wedge-shaped atelectasis
 - Pleural effusion
 - Dilatation of right ventricle
 - High standing of diaphragm dome
1579. Which among instrumental methods is the most informative for the diagnostic of pulmonary embolism?
- *Angiopneumography
 - X-ray examination of chest
 - Ultrasound examination
 - ECG
 - EchoCG
1580. Massive pulmonary embolism is characterized by the following clinic:
- *Phenomena of shock
 - Infarction pneumonia
 - Hemoptysis
 - Pleurisy
 - Asymptomatic course
1581. Which among symptoms is not characteristic for infarction pneumonia?
- *Ring-like shade
 - Wedge-shaped atelectasis
 - Hemoptysis
 - Pleural effusion
 - Increase body t°
1582. For differentiation between pulmonary embolism and myocardial infarction is used:
- *ECG
 - Ultrasound examination
 - X-ray examination of chest
 - Fibrogastroscopy
 - Spirography
1583. Which among echocardiographic signs is not characteristic for pulmonary embolism?
- *Dilated, hypokinetic left ventricle
 - Dilated, hypokinetic right ventricle
 - Dilatation of proximal pulmonary arteries
 - The increase of bloodflow velocity of tricuspid regurgitation $>3-7$ m/s
 - Disturbance of bloodsource from a right ventricle
1584. The direct signs of pulmonary embolism on the angiopneumography are:
- *Defects of filling of vessels
 - Dilatation of pulmonary artery trunk and its large branches
 - Asymmetry of filling of vessels by a contrast substance
 - The stasis of contrast
 - Pleural effusion
1585. The direct signs of pulmonary embolism on the angiopneumography are:
- *«Amputation» of vessel with dilatation proximally to occlusion
 - Dilatation of pulmonary artery trunk and its large branches
 - Asymmetry of filling of vessels by a contrast substance
 - The stasis of contrast
 - Pleural effusion
1586. The direct signs of pulmonary embolism on the angiopneumography are:
- *Oligemia
 - Dilatation of pulmonary artery trunk and its large branches
 - Asymmetry of filling of vessels by a contrast substance

- D. The stasis of contrast
 - E. Pleural effusion
1587. To the laboratory methods which confirm the pulmonary embolism belong:
- A. *Determination of D-dimer
 - B. General blood analysis
 - C. Determination of cholinesterase activity
 - D. Determination of circulatory immune complexes
 - E. Determination of antiphospholipid factor
1588. For the control of anticoagulating therapy in pulmonary embolism used:
- A. *The partial activated thromboplastine time
 - B. Fibrinogen of blood
 - C. Thrombotest
 - D. Tolerance of blood serum to heparin
 - E. Time of recalcification
1589. The initial dose of heparin in the treatment of pulmonary embolism:
- A. *10-20 thousands Unites
 - B. 2,5-5 thousands Unites
 - C. 1-2,5 thousands Unites
 - D. 40-60 thousands Unites
 - E. 100 thousands Unites
1590. What index of the partial activated thromboplastine time, which confirms the efficiency of anticoagulating therapy?
- A. *Increase in 1,5-2 times
 - B. Does not change
 - C. Diminish in 1,5-2 times
 - D. Diminish in 3-5 times
 - E. Diminish in 10-15 times
1591. Medicine of choice of thrombolytic therapy in pulmonary embolism is:
- A. *Aktilise
 - B. Heparin
 - C. Fibrinolysin
 - D. Fraxiparin
 - E. Clexan
1592. What is the dose of Aktilise is used in the treatment of pulmonary embolism?
- A. *100 mg.
 - B. 5 mg.
 - C. 10 mg.
 - D. 500 mg.
 - E. 1000 mg.
1593. The indication for application of thrombolytic therapy is:
- A. *Pulmonary embolism
 - B. Hemorrhagic shock
 - C. Suspicion on dissection of aorta
 - D. Severe traumas or great surgical operations within 2 weeks
 - E. Septic endocarditis
1594. The indication for application of thrombolytic therapy is:
- A. *Deep vein thrombosis
 - B. Hemorrhagic shock
 - C. Suspicion on dissection of aorta
 - D. Severe traumas or great surgical operations within 2 weeks
 - E. Septic endocarditis
1595. What diseases are not contraindication for the thrombolytic therapy?
- A. *Acute myocardial infarction
 - B. Hemorrhagic shock

- C. Suspicion on dissection of aorta
 - D. Severe traumas or great surgical operations within 2 weeks
 - E. Septic endocarditis
1596. What diseases are not contraindication for thrombolytic therapy?
- A. *Acute ileofemoral venous thrombosis
 - B. Hemorrhagic shock
 - C. Suspicion on dissection of aorta
 - D. Severe traumas or great surgical operations within 2 weeks
 - E. Septic endocarditis
1597. In relapse pulmonary embolism with a prophylactic purpose used:
- A. *Implantation of cava-filters
 - B. Direct anticoagulants
 - C. Indirect anticoagulants
 - D. Thrombolytics
 - E. Profundoplastiks
1598. What operation is performed in patients with pulmonary embolism?
- A. *Thrombectomy from a pulmonary artery
 - B. Ligation of pulmonary artery
 - C. Prosthetic graft repairing of pulmonary artery
 - D. Resection of pulmonary artery
 - E. Pneumonectomy

Situational tasks

1. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, lung sounds by percussion, by auscultation crackling rales over the lower lobe of the left lung. On X-ray of the chest expressed infiltration of lung tissue with areas of enlightenment in the center. What is the primary diagnosis?
 - A. *Abscessing pneumonia.
 - B. Pleural empyema.
 - C. Acute lung abscess.
 - D. Pyopneumothorax.
 - E. Bronchiectatic disease.
2. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, the shortening of percussion sound over the lower lobe, by auscultation weakened breathing with amphoric sound, crackling rales. On X-ray of the chest expressed infiltration of lung tissue with enlightenment in the center with fluid level. What is the primary diagnosis?
 - A. *Acute lung abscess.
 - B. Pleural empyema.
 - C. Abscessing pneumonia.
 - D. Pyopneumothorax.
 - E. Bronchiectatic disease.
3. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest. Has been ill for 2 months, the onset is caused by undercooling. The lag of the left half of the chest during breathing, the shortening of percussion sound over the lower lobe, by auscultation weakened breathing with amphoric sound. On X-ray of the chest the destruction cavity with the fibrous capsule in the projection of lower lobe of the left lung, infiltration of lung tissue is not determined. What is the primary diagnosis?
 - A. *Chronic lung abscess.
 - B. Pleural empyema.

- C. Acute lung abscess
 - D. Abscessing pneumonia.
 - E. Pyopneumothorax.
4. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, the shortening of percussion sound over the lower lobe, by auscultation weakened breathing with amphoric sound. On the X-ray of chest the destruction cavity with the fluid level, with a clear thin-walled capsule in the projection of the lower lobe of the left lung. Infiltration of lung tissue is not determined. What is the primary diagnosis?
 - A. *Suppurative cyst of the lung.
 - B. Pleural empyema.
 - C. Acute lung abscess
 - D. Abscessing pneumonia.
 - E. Pyopneumothorax.
 5. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, over the left lobe a dull percussion sound, by auscultation the breathing is absent. On X-ray of the chest the shadow in the basal parts of the left lung with an oblique upper level along Damuazo's line. What is the primary diagnosis?
 - A. *Pleural empyema.
 - B. Acute lung abscess
 - C. Chronic lung abscess.
 - D. Suppurative cyst of the lung.
 - E. Pyopneumothorax.
 6. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest, dyspnea. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, the shortening of percussion sound over the lower lobe, by auscultation the breathing is absent. On X-ray of the chest the shadow in the basal parts of the left lung with a horizontal fluid level and enlightenment over it. Is visible the edge of collapsed lung. What is the primary diagnosis?
 - A. *Limited pyopneumothorax.
 - B. Acute lung abscess
 - C. Chronic lung abscess.
 - D. Pleural empyema.
 - E. Total pyopneumothorax.
 7. Complaints of cough with foul-smelling purulent sputum with streaks of blood, increased body temperature to 40°C, pain in the left half of the chest, dyspnea at rest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, with a shortening of the pulmonary percussion sound, by auscultation moist rales over the left lung. On X-ray of the chest expressed infiltration of the left lung with multiple sites of destruction. What is the primary diagnosis?
 - A. *Gangrene of the lung.
 - B. Pleural empyema.
 - C. Acute lung abscess
 - D. Abscessing pneumonia.
 - E. Pyopneumothorax.
 8. Complaints of cough with foul-smelling purulent sputum with streaks of blood, increased body temperature to 40°C, pain in the left half of the chest, dyspnea at rest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, with a shortening of the pulmonary percussion sound, by auscultation moist rales over the lower lobe of the left lung. On X-ray of the chest expressed infiltration of left lung tissue with a giant cavity in the lower lobe with the level of the fluid. What is the primary diagnosis?
 - A. *Gangrenous abscess of lung.
 - B. Pleural empyema.

- C. Acute lung abscess
 - D. Abscessing pneumonia.
 - E. Gangrene of the lung.
9. Complaints of cough with purulent sputum, increased body temperature to 39°C, pain in the left half of the chest. Has been ill for 2 weeks, the onset is caused by undercooling. The lag of the left half of the chest during breathing, the shortening of percussion sound over the lower lobe, by auscultation weakened breathing. On X-ray of the chest paracostal fusiform shadow in the projection of the left lower lobe of the lung. What is the primary diagnosis?
- A. *Limited empyema.
 - B. Wide-spread pleural empyema.
 - C. Acute lung abscess
 - D. Chronic lung abscess.
 - E. Pyopneumothorax.
10. In the patient on the fourth day after the chest trauma on X-ray - heterogeneous shadow in the lower lobe. By puncture received a small amount of light yellow fluid with blood clots. What treatment are the best for the patient?
- A. *Drainage of the pleural cavity
 - B. Operational - lung decortication
 - C. Daily puncture
 - D. Resorbed therapy
 - E. Antibacterial therapy
11. In the patient, 48 years old, on the seventh day after the onset of a moderate pain in the chest, severe cough, fever to 39°C appeared the bad-smell sputum. The patient's condition remains grave, with expectoration more than 600 ml of gray-green sputum, and hectic fever. On X-ray – on the background of the heterogeneous shadow of the lower lobe of right lung revealed a cavity with a horizontal level of fluid. What is the primary diagnosis?
- A. *Abscess of the right lung
 - B. Gangrene of the right lung
 - C. Acute abscess of the right lung
 - D. Suppuration cyst of the right lung
 - E. Suppuration tuberculous cavern
12. In the patient, 35 years old, during the physical exertion appeared severe pain in the left half of the chest. Objectively: the patient is covered with cold sweat, dyspnea, pain during inspiration. By auscultation: vesicular breathing on the right side, on the left - is absent. Tachycardia, pulse 100 beats/min. What is the primary diagnosis?
- A. *Spontaneous pneumothorax
 - B. Angina pectoris
 - C. Acute myocardial infarction
 - D. Left-sided pleurisy
 - E. Pneumonia
13. The patient A., age 37, entered with complaints of cough with purulent sputum to 150 ml per day, pain in the right half of the chest, fever to 38°C. Has been ill for two weeks. The day before the entrance to the clinic during cough attack expectorated to 300 ml of purulent bad-smell sputum. On examination: a shortening of the pulmonary percussion sound under the right scapula, and the weakening of vesicular breathing. What is the primary diagnosis?
- A. *Acute lung abscess
 - B. Acute bronchitis
 - C. Exacerbation of chronic abscess
 - D. Exacerbation of bronchoectatic disease
 - E. Pleural empyema
14. Patient A., aged 42, had been treated for two months for an acute abscess of the upper lobe of right lung without improvement. The treatment: intramuscular injection of antibiotics, sulfanilamidns drugs. Remains the cough with purulent sputum to 80-100 ml per day, fever (37,6°C). What is the primary diagnosis?

- A. *Chronic lung abscess
 - B. Acute abscess of the right lung
 - C. Tuberculous cavern
 - D. Peripheral lung cancer
 - E. Suppurative cyst of lung
15. The patient, 78 years old, entered with complaints of pain in the left half of the chest, coughing, with daily 80 ml of mucopurulent sputum, fever to 37,2°C. The X-rays of the lower lobe of right lung revealed a cavity with irregular internal border and outside spicules with minor infiltration around. What is the primary diagnosis?
- A. *Hollow form of lung cancer
 - B. Chronic lung abscess
 - C. Suppuration cyst of lung
 - D. Fibro-cavernous tuberculosis
 - E. Limited empyema
16. The patient has a pyogenic lung abscess, which was complicated by repeated bleeding. The patient is undergoing the operative treatment. What antibiotics are the most suitable for preoperative prophylaxis?
- A. *Cephalosporins.
 - B. Penicillin.
 - C. Macrolides.
 - D. Aminoglycosides.
 - E. Fluorohinolones.
17. The patient with bilateral hydrothorax has undergone the repeat pleural puncture of both sides. After the last puncture felt the deterioration, fever, pain in the chest. Therapeutist on the next day during pleural puncture on the right obtained the pus. What is the mechanism of acute right-side empyema?
- A. *Contact-aspirating.
 - B. Lymphogenous.
 - C. Hematogenous.
 - D. Implantation.
 - E. Airborne.
18. The patient has the pyogenic lung abscess, which was complicated by bleeding. What medicines are the most suitable to stop the bleeding?
- A. *Vitamin K.
 - B. Anticoagulants.
 - C. Antibiotics.
 - D. Antiaggregants.
 - E. Prostaglandins.
19. The patient has the lung abscess, which was complicated by bleeding. What medicines are the most suitable to stop the bleeding?
- A. *Dicynon.
 - B. Heparin.
 - C. Penicillin.
 - D. Courantil.
 - E. Alprostan.
20. The patient has the lung abscess, which was complicated by bleeding to 200 ml. How this bleeding is classified?
- A. *I degree
 - B. 0 degree
 - C. II degree
 - D. III degree
 - E. IV degree
21. The patient received chest trauma 2 hours ago. Complains of the severe pain in the right half of the chest, dyspnea at rest. On examination: the lag of the right half of the chest during breathing,

- crepitation along the V-VI ribs on the right side, by percussion - tympanic sound, by auscultation - breathing is absent. What is the primary diagnosis?
- A. *Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic pneumothorax.
 - B. Closed chest trauma. Fractures of V-VI ribs on the right side.
 - C. Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic hemothorax.
 - D. Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic subcutaneous emphysema.
 - E. Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic complicated hemothorax.
22. The patient received chest trauma 2 hours ago. Complains of the severe pain in the right half of the chest, dyspnea at rest. On examination: the lag of the right half of the chest during breathing, crepitation along the V-VI ribs on the right side, by percussion - tympanic sound, by auscultation - breathing is absent. What additional examination is the most suitable?
- A. *Chest X-ray with contrast of the stomach
 - B. Plain X-ray of abdominal cavity
 - C. Esophagogastrosocopy
 - D. Computer tomography
 - E. Tomography of the chest
23. The patient received chest trauma 2 hours ago. Complains of the severe pain in the right half of the chest, dyspnea at rest. On examination: the lag of the right half of the chest during breathing, crepitation along the V-VI ribs on the right side, by percussion - tympanic sound, by auscultation - breathing is absent. The primary diagnosis: Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic pneumothorax. What is the typical treatment of rib fracture?
- A. *Novocaine block
 - B. External fixation of ribs
 - C. Intramedullary costal osteosynthesis;
 - D. Mechanical ventilation with positive end-expiratory pressure
 - E. Thoracotomy
24. The patient received chest trauma 2 hours ago. Complains of the severe pain in the right half of the chest, dyspnea at rest. On examination: the lag of the right half of the chest during breathing, crepitation along the V-VI ribs on the right side, by percussion - tympanic sound, by auscultation - breathing is absent. The primary diagnosis: Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic pneumothorax. What is the treatment of pneumothorax?
- A. *Pleural drainage
 - B. Pleural puncture
 - C. Thoracotomy
 - D. Pneumonectomy, bilobectomy, lobectomy
 - E. Conservative treatment
25. The patient received chest trauma 2 hours ago. Complains of the severe pain in the right half of the chest, dyspnea at rest. On examination: the lag of the right half of the chest during breathing, crepitation along the V-VI ribs on the right side, by percussion - tympanic sound, by auscultation - breathing is absent. The primary diagnosis: Closed chest trauma. Fractures of V-VI ribs on the right side. Posttraumatic pneumothorax. Where the drainage of pleural space in pneumothorax is performed?
- A. *II intercostal space, midclavicular line
 - B. II intercostal space, scapular line
 - C. IV intercostal space, anterior axillary line
 - D. VII intercostal space, midclavicular line
 - E. VII intercostal space, scapular line
26. The patient received chest trauma 14 days ago. Complains of a moderate pain in the right half of the chest, dyspnea, fever up to 38,5°C. Lag of the right half of the chest during breathing,

narrowing of the intercostal spaces. By percussion - a shortening of the percussion sound over the right lung, by auscultation - weakening of breathing. What is the primary diagnosis?

- A. *Right-side suppurative hemothorax.
 - B. Right-side posttraumatic pneumonia.
 - C. Right-side hemothorax.
 - D. Right-side pyopneumothorax.
 - E. Consolidated rib fractures.
27. Was pressed by the truck to the wall. Complains of the expressed dyspnea, difficult breathing, chest pain. On examination the expressed cyanosis. The frequency of respiratory movements - 26-28 per 1 min. Unstable hemodynamics. The chest is deformed, abnormal mobility of the front wall. The swelling of soft tissues of the neck with crepitation. What is the primary diagnosis?
- A. *Mediastinal emphysema.
 - B. Posttraumatic pneumothorax.
 - C. Posttraumatic hemothorax.
 - D. Posttraumatic pneumonia
 - E. Subcutaneous emphysema.
28. Was pressed by the truck to the wall. Complains of the expressed dyspnea, difficult breathing, chest pain. On examination the expressed cyanosis. The frequency of respiratory movements - 26-28 per 1 min. Unstable hemodynamics. The chest is deformed, abnormal mobility of the front wall. The swelling of soft tissues of the neck with crepitation. The primary diagnosis: Mediastinal emphysema. What is the treatment of mediastinal emphysema?
- A. *Drainage of anterior mediastinum
 - B. Conservative treatment
 - C. Drainage of pleural cavity
 - D. Novocaine block
 - E. Pericardial puncture
29. Was pressed by the truck to the wall. Complains of the expressed dyspnea, difficult breathing, chest pain. On examination the expressed cyanosis. The frequency of respiratory movements - 26-28 per 1 min. Unstable hemodynamics. The chest is deformed, abnormal mobility of the front wall. The swelling of soft tissues of the neck with crepitation. The primary diagnosis: Mediastinal emphysema. What is the main cause of mediastinal emphysema?
- A. *Disruptions of trachea, bronchi
 - B. Rib fracture
 - C. Pneumothorax
 - D. Hemothorax
 - E. Mediastinal tumours
30. Was pressed by the truck to the wall. Complains of the expressed dyspnea, difficult breathing, chest pain. On examination the expressed cyanosis. The frequency of respiratory movements - 26-28 per 1 min. Unstable hemodynamics. The chest is deformed, abnormal mobility of the front wall. The swelling of soft tissues of the neck with crepitation. The primary diagnosis: Mediastinal emphysema. What does the mediastinal emphysema result in?
- A. *Cardiac tamponade
 - B. Hemoptysis
 - C. Pleural empyema
 - D. Pneumothorax
 - E. Lung atelectasis
31. Female patient, 62 years old, was got in accident. On examination was detected the region of the right half of the chest, which disengages during inspiration. What are the most appropriate therapeutic measures?
- A. *External fixation of a floating area
 - B. Introduction of narcotic analgetics
 - C. Vagosympathetic block by Vishnevsky
 - D. Tight chest bandage

- E. Paravertebral blockade
32. In the patient after the accident with multiple rib fracture during the puncture of pleural cavity received the gastric content. What additional examination is the most suitable?
- A. *Chest X-ray with contrast of the stomach
 - B. Plain X-ray of abdominal cavity
 - C. Esophagogastroscope
 - D. Computer tomography
 - E. Tomography of the chest
33. In the patient on the fourth day after the chest trauma on X-ray - heterogeneous shadow in the lower lobe. By puncture received a small amount of light yellow fluid with blood clots. What treatment are the best for the patient?
- A. *Drainage of the pleural cavity
 - B. Operational - lung decortication
 - C. Daily puncture
 - D. Resorbed therapy
 - E. Antibacterial therapy
34. In the patient after blunt chest trauma with a sternum fracture appeared the weakness, hypotension, cyanosis of the upper half of the body, distension of the neck veins. By pleural puncture the content is absent. Pulse 120 beats per min, rhythmic, weakened. What is the primary diagnosis?
- A. *Cardiac tamponade
 - B. Pulmonary embolism
 - C. Contusion of the heart
 - D. Acute myocardial infarction
 - E. Coagulated hemopericardium
35. The patient entered in 3 hours after the injury with expressed subcutaneous emphysema of the upper half of the body, dyspnea, tachycardia, pulse - 120 beats/min. On X-ray the pneumothorax was found out with significantly enlargement of the mediastinum in both sides. What is the first aid?
- A. *Drainage of the anterior mediastinum
 - B. Puncture of the pleural cavity
 - C. Drainage of the pleural cavity
 - D. Thoracoscopy
 - E. Thoracotomy
36. The patient entered in 3 hours after the injury with expressed subcutaneous emphysema of the upper half of the body, dyspnea, tachycardia, pulse - 120 beats/min. On X-ray the pneumothorax was found out with significantly enlargement of the mediastinum in both sides. What is the primary diagnosis?
- A. *Mediastinal emphysema.
 - B. Posttraumatic pneumothorax.
 - C. Posttraumatic hemothorax.
 - D. Posttraumatic pneumonia
 - E. Subcutaneous emphysema.
37. Patient S., 25 years old, entered the hospital after the chest trauma. During clinical and X-ray examination was diagnosed the left-side tension pneumothorax. What is the first aid?
- A. *Drainage of the pleural cavity
 - B. Intravenous infusion
 - C. Oxygenotherapy
 - D. Intubation
 - E. Analgesics
38. Patient S., 25 years old, entered the hospital after the chest trauma. During clinical and X-ray examination was diagnosed the left-side tension pneumothorax. Where the drainage of pleural space in pneumothorax is performed?
- A. *II intercostal space, midclavicular line

- B. II intercostal space, scapular line
 - C. IV intercostal space, anterior axillary line
 - D. VII intercostal space, midclavicular line
 - E. VII intercostal space, scapular line
39. Patient S., 35 years old, entered the hospital after the chest trauma. During clinical and X-ray examination was diagnosed the left-side hemothorax. Where the drainage of pleural space in hemothorax is performed?
- A. *VII intercostal space, scapular line
 - B. II intercostal space, midclavicular line
 - C. II intercostal space, scapular line
 - D. IV intercostal space, anterior axillary line
 - E. VII intercostal space, midclavicular line
40. Patient S., 35 years old, entered the hospital after the chest trauma. During clinical and X-ray examination was diagnosed the left-side hemothorax. What method is the most informative in the diagnostic of hemothorax?
- A. *Pleural puncture
 - B. General blood analysis
 - C. Sputum analysis
 - D. Auscultation
 - E. X-ray examination
41. Patient S., 35 years old, entered the hospital after the chest trauma. During clinical and X-ray examination was diagnosed the left-side hemothorax. What test is used to determine the continuity of pleural bleeding?
- A. *Revilour-Greguar's test
 - B. Troyanov-Trendelenburg's test
 - C. Talman's test
 - D. Mayo-Pratt's test
 - E. Delbe-Pertess test (marching test)
42. In the patient, 35 years old, during the physical exertion appeared severe pain in the left half of the chest. Objectively: the patient is covered with cold sweat, dyspnea, pain during inspiration. By auscultation: vesicular breathing on the right side, on the left - is absent. Tachycardia, pulse 100 beats/min. What is the primary diagnosis?
- A. *Spontaneous pneumothorax
 - B. Angina pectoris
 - C. Acute myocardial infarction
 - D. Left-sided pleurisy
 - E. Pneumonia
43. 54 years old patient complains of dysphagia. Two years ago noticed on the left side of neck the appearance of protrusion after eating, vomiting by food, night cough. Began to lose his weight. On X-ray of esophagus with barium at the level of the clavicle was revealed the depot of barium like chicken egg by the size and shape. What is the most probable diagnosis?
- A. *Diverticulum of the esophagus
 - B. Esophagotraheal fistula
 - C. Esophageal cancer
 - D. Stenosis of the esophagus
 - E. Esophageal achalasia
44. 54 years old patient complains of dysphagia. Two years ago noticed on the left side of neck the appearance of protrusion after eating, vomiting by food, night cough. Began to lose his weight. On X-ray of esophagus with barium at the level of the clavicle was revealed the depot of barium like chicken egg by the size and shape. What complication is probable for this disease?
- A. *Diverticulitis
 - B. Obstructive jaundice
 - C. Intestinal obstruction
 - D. Myocardial infarction

- E. Lung atelectasis
45. 54 years old patient complains of dysphagia. Two years ago noticed on the left side of neck the appearance of protrusion after eating, vomiting by food, night cough. Began to lose his weight. On X-ray of esophagus with barium at the level of the clavicle was revealed the depot of barium like chicken egg by the size and shape. What is the typical treatment of this disease?
- A. *Surgical treatment
 - B. Spasmolytics
 - C. Analgetics
 - D. Nonsteroid antiinflammatory drugs
 - E. Antibiotics
46. 54 years old patient complains of dysphagia. Two years ago noticed on the left side of neck the appearance of protrusion after eating, vomiting by food, night cough. Began to lose his weight. On X-ray of esophagus with barium at the level of the clavicle was revealed the depot of barium like chicken egg by the size and shape. What operation is performed in this disease?
- A. *Resection of diverticulum
 - B. Esophagomyotomy
 - C. Esophagogastric anastomosis
 - D. Extirpation of esophagus
 - E. Esophageal plastics by intestine
47. To the hospital entered a man in the critical condition: acrocyanosis, dyspnea, subcutaneous emphysema on the neck and upper part of body. Complains of severe pain behind the breastbone and epigastrium. The body temperature of 38,9°C, pulse 130 beats/min, blood pressure 80/50 mm Hg. From anamnesis 6 years ago after drinking appeared the vomit, which resulted in the signatic. What is the primary diagnosis?
- A. *Spontaneous rupture of esophagus
 - B. Incarceration of paraesophageal hernia
 - C. Spontaneous pneumothorax
 - D. Pulmonary embolism
 - E. Perforated ulcer
48. 38-year-old woman complains of difficulty passing of food through esophagus, periodic vomiting. Has been ill for 1,5 years. Last 6 months notes appearance of food on the pillow during sleep. Lost 15 kg of body weight. There were constipations, stool once in 3-4 days. On plain X-ray film the absence of the gas bubble of the stomach. What is the most probable diagnosis?
- A. *Achalasia of esophagus
 - B. Peptic stenosis of the esophagus
 - C. Esophageal cancer
 - D. Diverticulum of the esophagus
 - E. Sliding esophageal hernia
49. 38-year-old woman complains of difficulty passing of food through esophagus, periodic vomiting. Has been ill for 1,5 years. Last 6 months notes appearance of food on the pillow during sleep. Lost 15 kg of body weight. There were constipations, stool once in 3-4 days. On plain X-ray film the absence of the gas bubble of the stomach. What stage of dysphagia relates to such manifestations?
- A. *II
 - B. I
 - C. III
 - D. IV
 - E. V
50. 38-year-old woman complains of difficulty passing of food through esophagus, periodic vomiting. Has been ill for 1,5 years. Last 6 months notes appearance of food on the pillow during sleep. Lost 15 kg of body weight. There were constipations, stool once in 3-4 days. On plain X-ray film the absence of the gas bubble of the stomach. What is the roentgenological sign of this disease in contrast X-ray with barium?

- A. *"Rat tail" sign
 - B. Filling defects
 - C. "Niche" sign
 - D. "Bell" sign
 - E. Blunt His angle
51. 38-year-old woman complains of difficulty passing of food through esophagus, periodic vomiting. Has been ill for 1,5 years. Last 6 months notes appearance of food on the pillow during sleep. Lost 15 kg of body weight. There were constipations, stool once in 3-4 days. On plain X-ray film the absence of the gas bubble of the stomach. What is the main method of diagnostic of this disease?
- A. *X-ray examination with barium swallow
 - B. Pleural punctere
 - C. Ultrasound examination
 - D. Plain X-ray examination of the chest
 - E. Irrigoscopy
52. On X-ray of the esophagus in the right lateral projection in the middle third on the front wall was found out the additional shadow, of round shape with smooth contours to 2 cm in diameter. What is the most probable diagnosis?
- A. *Diverticulum of the esophagus
 - B. Achalasia of the esophagus
 - C. Esophageal cancer
 - D. Chemical burn of the esophagus
 - E. Diaphragmatic hernia
53. The tool dilation of burn and peptic stricture of the esophagus has a risk of perforation with the development of purulent mediastinitis and pleural empyema. What is the least dangerous method for perforation should be applied in the first attempt of dilation of the stricture?
- A. *Dilatation of the stricture by balloon dilatator with a stable diameter of the cylinder.
 - B. Bouginage under the control of esophagoscope.
 - C. Bouginage along the metal conductor.
 - D. The blind bouginage under local anesthesia.
 - E. Bouginage under the control of X-ray
54. Female complains of difficult passing of food through esophagus, vomiting by unchanged food, regurgitation in night and weight loss. Anamnesis about 10 years. On X-ray study revealed achalasia of esophagus of the IV stage with S-shaped deformation. What is the optimal treatment?
- A. *Operation esophagocardiomyotomy with plastic by the stomach fundus.
 - B. Cardiodilatation by hard probe.
 - C. Cardiodilatation balloon probe.
 - D. Operation esophago-fundoanastomosis by Heyrovsky.
 - E. Resection of the cardia with esophageal anastomosis.
55. Female complains of difficult passing of food through esophagus, vomiting by unchanged food, regurgitation in night and weight loss. Anamnesis about 10 years. On X-ray study revealed achalasia of esophagus of the IV stage. What is the characteristic feature of the IV stage of this disease?
- A. *Considerable esophageal dilation with S-shaped elongation.
 - B. Cicatrical changes with expressed esophageal dilation, the peristalsis is absent
 - C. Asymptomatic
 - D. Functional spasm without esophageal dilation
 - E. Constant spasm with a moderate esophageal dilation and maintained peristalsis
56. Patient 52 entered the clinic with complaints of complete obstruction of the esophagus, salivation, weakness, t-38,7°C. Dysphagia has been for 8 days, after the swallowing of piece of the meat with bone. On X-ray the barium delays at the middle third of the esophagus. On fibroesophagoscopy was found a wedged bone with hyperemia and edema of the mucous membrane, covered by fibrin. What is the optimal treatment strategy in this case?

- A. *Surgical treatment: thoracotomy, esophagotomy, removal of foreign body (bone), suturing of the esophagus + gastrostomy.
 - B. Endoscopic removal of foreign body by rigid esophagoscope
 - C. Pushing of foreign body in the stomach by bougie.
 - D. Removal of foreign body by the Fogarty's probe
 - E. Removal of foreign body by fiberoptic endoscope.
57. Among the methods of esophageal plastic the most physiologic and safe modern method is:
- A. *Isoperistaltic plastic by tube of the greater curvature of the stomach after the extirpation of the esophagus through a cervical-laparotomy access.
 - B. Large intestine plastic in antiperistaltic position of the transplant.
 - C. Large intestine plastic in isoperistaltic position of the transplant.
 - D. Large intestine plastic with a skin flap.
 - E. Large intestine plastic by ileocecal segment.
58. The patient has the postburn stenosis of the esophagus. After the next bouginage felt the fever, tachycardia, pain behind the breastbone. On X-ray: the horizontal level of fluid in the posterior mediastinum. What is the most probable diagnosis?
- A. *Acute posterior mediastinitis.
 - B. Acute anterior mediastinitis.
 - C. Diverticulum of the esophagus.
 - D. Acute pleural empyema.
 - E. Paraesophageal hernia.
59. Complaints of burning, pain behind the breastbone, loss of weight. Has been ill for 7 months. Last 2 weeks noticed difficult passing of solid food. On contrasting X-ray was diagnosed: filling defect of lower thoracic part of the esophagus, a "niche" sign of the lesser curvature of stomach. What is the most probable diagnosis?
- A. *Gastric ulcer
 - B. Paraesophageal hernia
 - C. Decompensated pyloric stenosis
 - D. Sliding esophageal hernia
 - E. Peptic duodenal ulcer
60. Complaints of the pain behind the breastbone, difficult passage of solid food, weight loss, dizziness. Has been ill for 3 months. Last 2 days disturbs the vomiting after fluid food, the stagnation of fluid food. On EGDS severe narrowing of the esophagus, rigidity of the walls, hyperemic mucosa without folds. What is the most probable diagnosis?
- A. *Esophageal cancer
 - B. Sliding esophageal hernia
 - C. Paraesophageal hernia
 - D. Reflux esophagitis
 - E. Varicose veins of the esophagus
61. In the patient six months ago appeared the complaints of pain behind the sternum and a strong burning sensation in the esophagus. Sometimes observed dysphagia. On X-ray examination found the presence of diverticulum of the left wall of esophagus at the level of tracheal bifurcation 3?4 cm, just below the aortic arch. The patient was not treated. What tactics of treatment should be choused?
- A. *Right-side thoracotomy, diverticulectomy.
 - B. Left-side thoracotomy, diverticulectomy.
 - C. Right-side thoracotomy, resection of the esophagus.
 - D. Large intestine plastic of esophagus
 - E. Large intestine plastic of esophagus
62. The woman aged 52 complains of pain behind the breastbone, difficult passing of solid food through esophagus, increased salivation. The doctor advised 0,1 % solution of atropine before eating. After 3 days on X-rays no pathology was revealed. The doctor should do for this patient:
- A. *Send to fibroesophagoscopy
 - B. Allow the job

- C. Control visit after 2 months
 - D. Treatment by spasmolytics
 - E. Send to ECG
63. The woman, 38 years old, complains of difficulty passage of solid meal on esophagus, vomiting by undigested food, night regurgitation (sign of „wet pillow“), loss of weight. Has been ill for 10 years. On X-ray examination with barium the sign of "rat tail", dilation of the esophagus to 6 cm with maintained peristalsis. What stage of achalasia is there in this patient?
- A. *II
 - B. I
 - C. 0
 - D. III
 - E. IV
64. 53 year old man complains of recurrent pain behind the breastbone, heartburn, especially in the horizontal position. Sometimes the burning pain behind the sternum occurs after hot or spicy food. Two weeks ago was vomiting by blood and lost of consciousness. Has entered the hospital after repeated gastric bleeding. What is the most probable diagnosis?
- A. *Sliding esophageal hernia, reflux esophagitis
 - B. Diverticulum of the esophagus
 - C. Varicose veins of the esophagus
 - D. Mallory-Weiss syndrome
 - E. Crohn's disease.
65. 53 year old man complains of recurrent pain behind the breastbone, heartburn, especially in the horizontal position. Sometimes the burning pain behind the sternum occurs after hot or spicy food. Two weeks ago was vomiting by blood and lost of consciousness. Has entered the hospital after repeated gastric bleeding. What roentgenological sign confirms the pathology?
- A. *"Bell" sign
 - B. "Rat tail" sign
 - C. Filling defects
 - D. "Bird-beak" sign
 - E. Esophageal dilatation
66. 53 year old man complains of recurrent pain behind the breastbone, heartburn, especially in the horizontal position. Sometimes the burning pain behind the sternum occurs after hot or spicy food. Two weeks ago was vomiting by blood and lost of consciousness. Has entered the hospital after repeated gastric bleeding. What drugs are used for the treatment of this pathology?
- A. *Blockers of proton pump
 - B. Spasmolytics
 - C. Adrenoblockers
 - D. Blockers of calcium channel
 - E. Anticoagulants
67. 53 year old man complains of recurrent pain behind the breastbone, heartburn, especially in the horizontal position. Sometimes the burning pain behind the sternum occurs after hot or spicy food. Two weeks ago was vomiting by blood and lost of consciousness. Has entered the hospital after repeated gastric bleeding. What disease should be this pathology differentiated from?
- A. *Peptic ulcer
 - B. Pancreatitis
 - C. Intestinal obstruction
 - D. Cholecystitis
 - E. Bronchial asthma
68. 53 year old man complains of recurrent pain behind the breastbone, heartburn, especially in the horizontal position. Sometimes the burning pain behind the sternum occurs after hot or spicy food. Two weeks ago was vomiting by blood and lost of consciousness. Has entered the hospital after repeated gastric bleeding. What is the main treatment of this pathology?
- A. *Conservative treatment
 - B. Esophagostomy

- C. Esophageal plastic
 - D. Resection of the esophagus
 - E. Resection of the stomach
69. 53 year old man complains of recurrent pain behind the breastbone, heartburn, especially in the horizontal position. Sometimes the burning pain behind the sternum occurs after hot or spicy food. Two weeks ago was vomiting by blood and lost of consciousness. Has entered the hospital after repeated gastric bleeding. What type of operation is used for treatment of this pathology?
- A. *Cruroplasty with Nissen's fundoplication
 - B. Esophagostomy
 - C. Cruroplasty
 - D. Resection of the esophagus
 - E. Resection of the stomach
70. After the birth of the child appeared the signs of respiratory failure. By auscultation on the left side the breathing is absent. On X-ray expressed mediastinal shift to the right, the presence of protrusion on the left side. What is the most probable diagnosis?
- A. *Left-sided diaphragmatic hernia
 - B. Hypoplasia of right lung
 - C. Polycystosis of the left lung
 - D. Relaxation of the diaphragm
 - E. Esophageal stricture
71. The patient, 45 years old, complains of retrosternal pain, which increase at night, heartburn, belching. Has been ill for 4 months. What research is the most appropriate?
- A. *X-ray contrast study in Trendelenburg's position.
 - B. Plain chest X-ray
 - C. Fibroesophagogastroduodenoscopy
 - D. Plain X-ray of the abdominal cavity.
 - E. Computer tomography
72. The patient, 45 years old, complains of retrosternal pain, which increase at night, heartburn, belching. Has been ill for 4 months. What is the most probable diagnosis?
- A. *Sliding esophageal hernia, reflux esophagitis
 - B. Diverticulum of the esophagus
 - C. Varicose veins of the esophagus
 - D. Mallory-Weiss syndrome
 - E. Crohn's disease.
73. What is the most wide-spread cause of the peptic stricture of esophagus associated with reflux-esophagitis?
- A. *Sliding esophageal hernia.
 - B. Prolonged nasogastric intubation in the esophagus.
 - C. Short stay nasogastric intubation.
 - D. Frequent vomiting of pregnancy.
 - E. Achalasia of the esophagus
74. X-ray signs: the "bell" sign, blunt Hiss angle, absence of gas bubble of the stomach are characteristic for:
- A. *Sliding esophageal hernia.
 - B. Paraesophageal hernia.
 - C. Relaxation of the diaphragm.
 - D. Malignant tumor of the esophagus.
 - E. Achalasia of the esophagus
75. The patient has the pain behind the breastbone, heartburn, which increases after the meal. Has been ill for 6 months. For 5 days has a black chair. On X-ray examination revealed the "bell" sign, blunt Hiss angle, absence of gas bubble of the stomach. What is the most probable diagnosis?
- A. *Sliding esophageal hernia.
 - B. Paraesophageal hernia.

- C. Relaxation of the diaphragm.
 - D. Malignant tumor of the esophagus.
 - E. Achalasia of the esophagus
76. Complaints of heaviness in the epigastric region, nagging pain after eating. When EGDS pathology identified. On X-ray examination pronounced gas bubble of the stomach is visible to the level III intercostal space on the left. What is the most probable diagnosis?
- A. *Relaxation of the diaphragm.
 - B. Paraesophageal hernia.
 - C. Sliding esophageal hernia.
 - D. Malignant tumor of the esophagus.
 - E. Achalasia of the esophagus.
77. The patients with sliding esophageal hernia mostly complain of:
- A. *Heartburn, pain behind the breastbone.
 - B. Difficult passage of food, loss of weight.
 - C. The pain behind the breastbone, difficulty in food passage.
 - D. Pain in the epigastric region with irradiation into the right hypochondrium.
 - E. Pain in the epigastric region with irradiation into the back.
78. Complications paraesophageal hernias of esophagus:
- A. *Incarceration
 - B. Malignancy.
 - C. Esophageal stricture.
 - D. Dysphagia.
 - E. Reflux esophagitis.
79. In the patient at night appeared a severe pain in the epigastric region, nausea, vomiting. 6 months ago was diagnosed paraesophageal hernia. What complication arose?
- A. *Incarceration
 - B. Malignancy.
 - C. Esophageal stricture.
 - D. Dysphagia.
 - E. Reflex esophagitis.
80. In the patient at night appeared a severe pain in the epigastric region, nausea, vomiting. 6 months ago was diagnosed paraesophageal hernia. What type of operation is indicated for this patient?
- A. *Cruroplasty
 - B. Esophagostomy
 - C. Resection of the esophagus
 - D. Resection of the stomach
 - E. Cruroplasty with Nissen's fundoplication
81. Complaints of heartburn, pain behind the breastbone, regurgitation of food. Has been ill for 2 months. On X-ray examination revealed the "bell" sign, blunt Hiss angle, absence of gas bubble of the stomach. Your tactics?
- A. *Conservative therapy.
 - B. Bougienage of esophagus.
 - C. Surgical intervention.
 - D. Large-intestinal plastic of esophageal cardia.
 - E. Small-intestinal plastic of esophageal cardia.
82. The patient has the postburn stenosis of the esophagus. After the next bougienage felt the fever, tachycardia, pain behind the breastbone. On X-ray: the horizontal level of fluid in the posterior mediastinum. What is the most probable diagnosis?
- A. *Acute posterior mediastinitis.
 - B. Acute anterior mediastinitis.
 - C. Diverticulum of the esophagus.
 - D. Acute pleural empyema.
 - E. Paraesophageal hernia.

83. Complaints of burning, pain behind the breastbone. Has been ill for 4 months. On contrasting X-ray was diagnosed: the "bell" sign, the absence of gas bubble of the stomach, blunt Hiss angle. What is the most probable diagnosis?
- *Sliding esophageal hernia
 - Paraesophageal hernia
 - Gastric ulcer
 - Peptic duodenal ulcer
 - Decompensated pyloric stenosis
84. 5 hours ago, after the bouginage of esophageal cicatrice stricture appeared the pain behind the breastbone, a feeling of compression, subcutaneous crepitation on the neck, fever to 38,5°C. On examination the weakening of the heart tones by auscultation. On plain X-ray of chest – mediastinal emphysema, mediastinal extension of the shadows. What is the most probable diagnosis?
- *Acute mediastinitis
 - Reflux esophagitis
 - Sliding esophageal hernia
 - Paraesophageal hernia
 - Achalasia of the esophagus
85. A 34-year-old female with hypertension is considering to become a pregnant. Which of the following medications would be absolutely contraindicated to control her BP during pregnancy?
- Methyldopa
 - Metoprolol
 - Labetalol
 - *Captopril
 - Nifedipine
86. A 34-year-old male with isolated essential hypertension came to clinic and it was found high BP of 180/100 mm Hg after failure of behavioral modifications. What is the most appropriate next step?
- Start hydrochlorothiazide
 - *Start hydrochlorothiazide and lisinopril
 - Repeat BP in 4 weeks
 - Start amlodipine
 - Start doxazosin
87. A 34-year-old male with isolated essential hypertension came to clinic and it was found high BP of 180/100 mm Hg item after failure of behavioral modifications. What is the most appropriate next step?
- Start hydrochlorothiazide
 - *Start hydrochlorothiazide and lisinopril
 - Repeat BP in 4 weeks
 - Start amlodipine
 - Start doxazosin
88. A 40-year-old diabetic patient with a blood pressure (BP) of 145/90 mm Hg item and proteinuria. Which BP profile represents the best therapeutic goal for this patient?
- 160/90
 - 140/90
 - 130/85
 - *125/75
 - 140/85
89. A 40-year-old diabetic patient presents with a blood pressure (BP) of 145/90 mm Hg item and proteinuria. What is the best medication for the initial management of this patient's hypertension?
- Calcium channel blockers
 - Beta blockers
 - *ACE-inhibitors / angiotensin receptor blockers

- D. Alpha blockers
 - E. Diuretics
90. A 42-year-old female with chronic obstructive pulmonary disease is found on multiple office visits to have elevated BP measurements. Which of the following medications is contraindicated?
- A. Hydrochlorothiazide
 - B. Metoprolol
 - C. Lisinopril
 - D. *None of above
 - E. All of the above
91. A 42-year-old male comes in for a routine physical examination. He is noted to have impaired glucose methabolism, and a BP of 135/85 mmHg. What is the best treatment plan for this individual?
- A. *Aggressive lifestyle modification
 - B. Institute thiazide diuretic regimen
 - C. No treatment at this time
 - D. Initiate an ACE-inhibitor
 - E. Initiate a beta-blocker
92. A 45-year-old male comes in for a routine physical examination. He is noted to have impaired glucose methabolism, and a BP of 140/85 mmHg. What is the best treatment plan for this individual?
- A. *Aggressive lifestyle modification
 - B. Institute thiazide diuretic regimen
 - C. No treatment at this time
 - D. Initiate an ACE-inhibitor
 - E. Initiate a beta-blocker
93. A 48-year-old male with diabetes mellitus, hypertension, and hyperlipidemia has a hypertensive emergency. His arterial pressure is 150/100 mmHg item. Which medications would be most appropriate therapy for this patient?
- A. *Nitroprusside
 - B. Enteral metoprolol
 - C. Fenoldopam
 - D. Intravenous nitroglycerine
 - E. Any of the above
94. ?A 48-year-old obese male with hypertension, dyslipidemia, and diabetes mellitus presents to the outpatient clinic for his yearly physical. He has refused medications in the past, but now is willing to consider treatment. His BP is 145/95 mmHg with a HR of 80 bpm. His laboratory data are significant for the presence of microalbuminuria. Which of the following medications would be the most appropriate?
- A. Carvedilol
 - B. Methyldopa
 - C. *Lisinopril
 - D. Chlorthalidone
 - E. Terazosin
95. A 48-year-old obese male with hypertension, dyslipidemia, and diabetes mellitus presents to the outpatient clinic for his yearly physical. He has refused medications in the past, but now is willing to consider treatment. His BP is 145/95 mmHg with a HR of 80 bpm. His laboratory data are significant for the presence of microalbuminuria. Which of the following medications would be the most appropriate?
- A. Carvedilol
 - B. Methyldopa
 - C. *Lisinopril
 - D. Chlorthalidone
 - E. Terazosin

96. A 56-year-old male on hydralazine, hydrochlorothiazide, lisinopril, and metoprolol begins to develop a malar rash and arthralgias. Which of the above antihypertensive agents is known to cause drug-induced lupus?
- Hydrochlorothiazide
 - Lisinopril
 - *Hydralazine
 - Metoprolol
 - None of the above
97. A 53-year-old male on hydralazine, hydrochlorothiazide, lisinopril, and metoprolol begins to develop a malar rash and arthralgias. Which of the above antihypertensive agents is known to cause drug-induced lupus?
- Hydrochlorothiazide
 - Lisinopril
 - *Hydralazine
 - Metoprolol
 - None of the above
98. A 60-year old white man consults you with a headache. Examination data are unremarkable, except that the blood pressure is raised and subsequent measurements confirm readings of 170/106 mmHg. He is obese, smokes 15 cigarettes per day and drinks 2 pints of beer per day. His investigations reveal a cholesterol of 6 mmol/l with a normal blood sugar and electrolyte profile and normal renal function. There is no evidence of target organ damage.
- This man needs immediate treatment with antihypertensive drugs, aspirin and a statin
 - *Initiate antihypertensive therapy with two-drug combination
 - At this age it is far more important to control the hyperlipidaemia than the BP
 - This man's blood pressure could be wholly ascribed to his alcohol intake
 - This patient can be observed and counseled on maintaining a reasonable BMI
99. A 62-year-old man with isolated essential hypertension, currently taking hydrochlorothiazide 25 mg PO daily, comes to you for his first clinic visit. He notes that his BP at home is always less than 140/80 mmHg, but in clinic it is always at least 155/95 mmHg. What is the next step?
- Increase dose of thiazide
 - Addition of second antihypertensive medication
 - *Do nothing as he has white coat hypertension
 - Evaluate for secondary causes of hypertension
 - Start metoprolol
100. A 62-year-old man with isolated essential hypertension, currently taking hydrochlorothiazide 25 mg PO daily, comes to you for his first clinic visit. He notes that his BP at home is always less than 140/80 mmHg item, but in clinic it is always at least 155/95 mmHg. What is the next step?
- Increase dose of thiazide
 - Addition of second antihypertensive medication
 - *Do nothing as he has white coat hypertension
 - Evaluate for secondary causes of hypertension
 - Start metoprolol
101. At a routine examination, an asymptomatic 46-year-old man is found to have a BP of 150/110 mmHg item, but no other abnormalities are present. What do you do next?
- Reassure the patient and repeat the physical examination in 12 months
 - Initiate antihypertensive therapy
 - *Obtain repeated BP recordings in your office and/or the patient's home or work site
 - Hospitalize patient for renal arteriography
 - Order a 24-h ambulatory BP monitoring
102. For the case below, select the most significant adverse effect of the antihypertensive and/or cardiac agent in question: a 45-year old female has been on diuretic, but BP remains elevated at 145/95, leading to the proposed addition of lisinopril. Which key potential adverse effect should be discussed?

- A. Increased triglyceride levels
 - B. Peripheral edema
 - C. Lupus-like syndrome
 - D. *Cough
 - E. Gynecomastia
103. For the case below, select the most significant adverse effect of the antihypertensive and/or cardiac agent in question: a 58-year old male truck driver has significant hypertension, still not controlled on a diuretic plus calcium channel blocker. Clonidine is being considered as the next medication, but in this patients is concerned by sedation, sexual dysfunction.
- A. Increased triglyceride levels
 - B. Cough
 - C. Gynecomastia
 - D. *Rebound hypertension
 - E. Urinary retention
104. For the case below, select the most significant adverse effect of the antihypertensive and/or cardiac agent in question: a 68-year old male with advanced chronic heart failure and BP 145/90 will have spironolactone therapy, but should be informed about possible side effect.
- A. Increased triglyceride levels
 - B. Peripheral edema
 - C. Lupus-like syndrome
 - D. Cough
 - E. *Gynecomastia
105. In patients with a history of stroke or TIA the preferred drug combination is:
- A. *ACE-inhibitor and diuretic
 - B. Calcium-channel blocker and beta-blocker
 - C. Beta-blocker and diuretic
 - D. Angiotensin receptor blocker and beta blocker
 - E. Beta- and alpha blocker
106. The initial antihypertensive medication recommended for patients who have no compelling indications or contraindications is
- A. ACE-inhibitor
 - B. Calcium-channel blocker
 - C. Diuretics
 - D. Beta blocker
 - E. *Any of the above
107. To reduce the patient's cardiovascular morbidity and mortality, which therapy would you prescribe?
- A. Hydralazine
 - B. Atenolol
 - C. *Losartan
 - D. Doxazosin
 - E. Clonidine
108. What is the appropriate course of action regarding the patient's antihypertensive therapy?
- A. Advise a low-sodium diet
 - B. *Finish doxazosin therapy and consider an alternative agent
 - C. Advise high dietary intake of calcium and potassium
 - D. Increase the doxazosin to 4 mg a day
 - E. Advise magnesium supplements
109. You see a diabetic patient presents with BP readings that are 155/95 or higher. All of the following statements about the treatment of this patient's hypertension are correct EXCEPT:
- A. Pharmacologic blockade of the renin-angiotensin system reduces the risk of both microvascular and macrovascular events
 - B. Aggressive BP control reduces cardiovascular events more in diabetics than in nondiabetics

- C. *Calcium channel blockers show no benefit in reducing cardiovascular events
 - D. The goal BP for this patient is <130/80 mmHg
 - E. All the above statements are correct
110. During the operation concerning strangular impassability of bowels, which conducted under general anaesthesia of i/v with ALV, at a patient the stop of cardiac diyal-nosti happened 50 years in the moment of mesenretium streching by the surgeon. What would prevent the stop of heart in this case?
- A. *i/v injection atropin
 - B. i/v injection of cardiac glycozidis
 - C. Deepening of general anaesthesi
 - D. Additional injection of relaxants
 - E. Additional injection droperidol
111. Patient 30 years after a road-transport failure complaints of the acute tahypno Ob-ly: a skin is pale, cyanosi Hypodermic emphysema in the region of thorax, stomach, right side of the neck. Auscultative: breathing on the right side is not conducted; pulse – 130/min., AP – 80/60 mm.mercury., CVP – 140 mm wt., FB – 30 /min., Ht – 0,27, Hb – 90 g/l. Subsequent therapy must include above all things:
- A. *punction of pleura cavity.
 - B. Urgent ALV
 - C. Massive infusion therapy of the crystalloid solutions
 - D. Infusion of dofamin, 2-5 mcg/cg/min
 - E. 100% oxygen
112. Patient has BP - 80/40 mm mercury, pulse - 120 per min, shock index for him:
- A. *120/80=1,5
 - B. 80+40)120=1
 - C. 80/120=0,67
 - D. 120/40=3
 - E. Not determined, as blood loss is unknown
113. Patient N., 47 years, treated in the hospital with the diagnosis: carbuncle of right kidney. Suddenly general condition deteriorated, body temperature 39.50 C, skin cold, humid, consciousness kept, expressed choking. Breath deep, noisy, 26/min. Pulse - 110/min., SC - 90/60 mm.mercury., oliguri Which complications we can think about?
- A. *Infectious-toxic shock
 - B. Vascular dystonia
 - C. Epilepsy
 - D. Anaphylactic shock
 - E. Orthostatic collapse
114. Patient, 40 years, with the trauma of both thighs is delivered from the scene of accident by a “passing transport”. Objectively: cyanosys, rubor of the lower half of the neck, tahypnoe, AP 60/40 mm mer st., HBA=120 /min, in lungs moist wheezes, diuresis – 20 ml/h., Nb 100 g/l. Which from the offered diagnoses most reliable?
- A. *Fatty embolism
 - B. Traumatic shock
 - C. Hemorrhagic shock
 - D. Pain shock
 - E. Tromboembolism
115. The patient 60 years is delivered in the department of intensive therapy with a diagnosis: bite of bee, anafilactic shock.. Which medicine will be primary and most effective?
- A. Adrenalin
 - B. Calcium the chloride
 - C. Prednizolon
 - D. Dimedrol
 - E. Suprastin

116. The patient 25 years is hospitalized in the surgical department with a diagnosis: penetrable wound of abdominal region. Objectively: it is excited, skin covers and visible mucous shells are pale; peripheral pulse of the weak filling, frequent, AP – 110/60 mm mercury Positive symptom of "desolation" of peripheral hypodermic vein Diuresis is lower How to characterize this state?
- The compensated shock
 - Preagonia
 - Circulating shock
 - Agony
 - Irreversible shock
117. The patient 36 years with ulcerous of gaster in anamnesis is hospitalized in the surgical department with complaints on vomiting by "coffee-grounds", diarrhea, moderately expressed thirst. Objectively: a skin is pale, covered by a death-damp, a tongue is dry, AP – 80/60 mm rt.st., HBA – 120/min., BF – 28/min., diuresis – 25 ml/h. Blood test: Era - 2,8 10¹²/l, Hb – 98 g/l. What will be most expedient in medical treatment:
- *solutions with colloid
 - 5% solution of glucose
 - Whole blood
 - Red corpuscles mass
 - Colloid solutions with red corpuscles mass
118. 118. Patient 48 years the second day in dpartment of intensive therapy concerning the acute front-partition heart attack of myocardium. During a review "wheezing" suddenly, non-permanent tonic constricting of muscles is marked, pupils are extended, pulse on carotis not palpitat What doctor have to do in the first place?
- Triple Safar method
 - Record of ECG
 - Cardial hit in the area of heart
 - Artificial respiration by the method of Silvester
 - Intracardial injection of adrenalin with an atropine
119. The patient entered the department with a diagnosis: acute intestinal impassability. Complaints: insignificant thirst, dizziness at an attempt to get up from a be At a review: patient apathetical, turgor is lowered, eyeballs are soft, tongue is dry with crack Pulse – 110 min., AP – 80 /60 mm of mer item, diuresis – 25 ml /h. Electrolyte composition: Na⁺ - 142 mmol/l, C⁺ - 4 mmol/l, glucose – 6 mmol/l, urea – 7 mmol/l. What variant of infusion is most expedient during operation?
- *Transfusion of crystalloid
 - Transfusion of solution of glucos
 - Transfusion of albumen.
 - Transfusion of native plasm
 - Transfusion of poliglucin.
120. The patient, 28 years, 2 hours ago fell down from the ground floor of hous Sopor, pale, there are the plural scratches of face, the lacerated hemorrhagic wounds on the left forearm. The closed break of the left shoulder and thigh. Pulse –110, Lc-10T/l, Hb – 100, AP – 90/40 mm of rt. item In the blood test: red corpuscles – 3,5 g/l. What infouziyniy serednic does not need to be used for medical treatment of shock?
- *5% solution of glucose
 - Solution of crystalloids
 - Solutions gelatin
 - Solutions of calcium
 - Solution of albumen
121. Anafilactic shock appeared at a patient. The state heavy and progressively gets wors HBA – 110 in a 1 minute, AP – 60/30 mm mercury. Prescribe medicine for the rescue of life of patient above all things?
- *Adrenalin.

- B. Chloride of calcium.
 - C. Prednizolon.
 - D. Dofamin.
 - E. Suprastin.
122. At a patient 20 years on a background the injection of vitamin B1 suddenly there was excitation, fear of death, falling of AP to 50 mm rt.st., hard breathing. Which of medicine it is necessary to inject firstly?
- A. *Adrenalin
 - B. Prednizolon
 - C. Calcium
 - D. Dimedrol
 - E. Eufilin
123. At a patient 60 years with the third day after an extirpation uterus acute insufficiency of breathing developed suddenly, a skin became at first cyanotic, and then ash-colored color. Tachypnoe, cough with bloody sputum, retrosternal pain. BP – 100/70 mm. mercury, HR – 120, BR – 32 in 1 min., CVP – 300 mm wt.col. What most reliable reason of worsening of the state of patient ?
- A. *Tromboemboliya of pulmonary artery
 - B. Bleeding
 - C. Pain shock
 - D. Hypostatic pneumonia
 - E. Heart attack of myocardium
124. At a patient in the ward of intensive therapy you marked appearance on the monitor of fibrillation of ventricule Your first actions?
- A. *To conduct defibrillation three times
 - B. To inject adrenalin
 - C. To inject a chloride
 - D. To begin the closed massage of heart
 - E. To inject lidocain
125. At a patient with the acute heart attack of myocardium best of all to warn relapsing fibrillation of ventricles with :
- A. cordaronum
 - B. lidocainum
 - C. ornidinum
 - D. *electrocardiostimulation
 - E. there is no right answer
126. At a patient with the acute heart attack of myocardium in the region of partition on a 5th day after the brief episode of loss of consciousness there is reduction of frequency of pulse to 32 in a minut BP - 80/40 mm Consciousness at the level of sopor. He immediately needs :
- A. to put right a craniotserebral hypothermia, to enter lasics, prednisoloni, tserebrolizin
 - B. to enter an atropine, eoufilin, to begin infouziyo of aloupenta
 - C. *to conduct urgent cardiostimoulation
 - D. all answers are faithful
 - E. there is no right answer
127. At a patient, carried to a 2 year ago the heart attack of myocardium, the acute decline of cholecystities planned cholecystectomy, signs of electric instability of myocardium . Actions of anaesthesiologist must include:
- A. injection of prednisoloni, lidocaini, hyperventilation, take the ECG
 - B. *injection of dopamini, after stabilisation of BP - nitroglycerine + infusion therapy under the control CVP, conducting of neurovegetative defence, take the ECG
 - C. injection of streptodecasol, stream infusion of reopoliglyocinum, injection of lidocainum, increase of dose of analgetics
 - D. correctly A) and C)
 - E. all answers are faithful

128. At a patient, that is found on medical treatment in the therapeutic department, the sudden stopping of circulation of blood happened. Medical personnel began the reanimation measure. Define the most rational way of injection of adrenalin for renewal of heart abbreviations in default of vein access:
- *To enter to a 3 ml solution of adrenalin in a trache
 - To enter to a 1 ml solution of adrenalin in muscle
 - To enter adrenalin in muscle, multiplying a dose in 3 times
 - The intracardial injection.
 - Adrenalin can be not entered
129. At a patient, that is found under the permanent electrocardiographic supervision, microwave fibrillation of myocardium and diagnosed clinical death developed. It is necessary to do:
- to inject the solution of calcium in cor
 - *to conduct high-voltage electric defibrillation
 - to inject solution of atropine in cor
 - to inject solution of adrenalin in cor
 - All answers are wrong
130. At the patient operated concerning the festering peritonitis caused by perforation of gastric ulcer, in a postoperation period appeared: high temperature, frequency of breathing 35/min., AP – 70/40 mm of mercury, diuresis -20 ml/h, temperature of body to 39 °C, leucocytosis. Transfusion during 12 hours 1,8 of a 0,9% solution of chlorure sodium and 0,8 of solution of reopoligluin did not improve general condition. Central vein pressure – 130 mm wt.st. For stabilization of hemodynamics will be optimum infusion:
- *Dopamin
 - Mezaton
 - Adrenalin
 - Noradrenalin
 - Ephedrine
131. At the ventilated patient with the edema of lungs at low pressure and septic shock intravenous infusion 7,5 mcg/kg/min dopamin will increase
- RaO₂, saturation of oxygen of the mixed vein blood, consumption of oxygen
 - diuresis
 - *cardiac systolic volume
 - right A) and B)
 - faithful all answers
132. In 2 hours after renewal of cardiac activity at a patient, that carried the sudden stop of heart on a background hemorrhagic shock (blood lost near 2,5 l) and is found on ALV, unstable hemorrhagia (AP – 80/40 – 90/60 mm of mercury, tachycardia) is marked, central vein pressure – 5 mm wt.st. It is related to:
- *By Hypovolume syndrome
 - By cardiac insufficiency
 - By the inadequate interchange of gases
 - Vasoplegia
 - By the inadequate anaesthetizing
133. In a clinic a patient with the traumatic tearing of both lower extremities off at the level of knee-joints is delivered. A patient is extremely inert, languid, pale, pulse 140 bpm, threadlike, AP 500. On both lower extremities there are the imposed plait. Bleeding at the receipt is not present. From the words of doctor of first-aid, lost about 3 litres of blood in place of event. What principal reason of heavy of the state of Patient?
- Acute hemorrhag
 - Pain shock.
 - Ishemia of extremities as a result of application of tourniquet
 - Fatty embolism
 - acute kidney insufficiency

134. In a clinic a patient with the traumatic tearing of both lower extremities off at the level of knee-joints is delivered. A patient is extremely inert, languid, pale, pulse 140 bpm, threadlike, AP 500. On both lower extremities there are the imposed plaits. Bleeding at the receipt is not present. From the words of doctor of first-aid, lost about 3 litres of blood in place of event. What principal reason of heavy of the state of Patient?
- *acute hemorrhage
 - Pain shock.
 - Ishemia of extremities as a result of application of tourniquet
 - Fatty embolism
 - acute kidney insufficiency
135. It is typical for:
- *Negative CVT
 - Erroneous puncture of artery
 - Right-side pneumothorax
 - Edema of lungs
 - Hypodermic emphysema
136. On a 4 day after incompatible (on a group) blood transfusion at a patient acutely reduced diuresis, anuria developed, the common state became worse acutely, arterial pressure rose. At laboratory research: creatinin plasma – 680 μmol/l, urea of plasma - 24 mmol/l. What illness and what stage of illness it follows to think about in the first place?
- Acute kidney insufficiency, anuria
 - Anaphylactic shock, acute kidney insufficiency, anuria
 - Hemotransfusion shock, postrenal acute kidney insufficiency, anuria
 - Posthemorrhagic acute kidney insufficiency, anuria
 - acute interstitial nephritis, postrenal anuria
137. Patient 20 years for verification of the functional state of kidneys the X-ray examination with v/v injection of contrast is conducted. At the end of injection the state of patient acutely became worse, the shortness of breath, hyperemia of skin, itch appeared. AP – 60/20 mm of mercury, HBA – 132/min. A similar research was conducted 3 months ago, such effects were not observed. What most reliable diagnosis?
- Medicinal anaphylactic shock
 - Acute kidney insufficiency
 - Thromboembolism of pulmonary artery
 - Stress on the conducted manipulation
 - Heart attack myocardium
138. Patient 38 years, native plasma was poured. At the end of infusion the state became worse: Patient confused, excited, cyanosis, hypersalivation. Breathing frequency 36 on 1 min., AP – 70/40 mm of mercury, whistling dry wheeze. Which from the following medicines must be injected firstly?
- *Adrenalin.
 - Eufilin.
 - Suprastin.
 - Noradrenalin.
 - Prednisolone.
139. Patient 40 years with the acute gastro-intestinal bleeding a canned blood was poured in a volume 400 ml after conducting of all tests on compatibility. After hemotransfusion the state of patient became worse, appeared head pains and pains in muscles at the temperature of body rose to 38,8. What can explain the state of patient?
- Pyrogenic reaction of middle heavy
 - By development of hemotransfusion shock
 - *Allergic reaction
 - By development of bacterial-toxic shock
 - By air embolism

140. Patient 40 years with the acute gastro - intestinal bleeding a canned blood was poured in a volume 400 ml after conducting of all tests on compatibility. After hemotransfusion the state of patient became worse, appeared head pains and pains in muscles at the temperature of body rose to 38,8 What can explain the state of patient?
- Pirogenic reaction of middle heavy
 - *By development of hemotransfusion shock
 - Allergic reaction
 - By development of bacterial-toxic shock
 - By air embolism
141. Patient 62 years the third day of presence in department of intensive therapy concerning the acute transmural heart attack of myocardium of front-partition localization. At night woked up from a suffocating cough, feeling of fear and trouble At a review: cyanosis, FB – 30 after 1 min., HBA – 132/ min., a rhythm is correct, tones of heart are deaf, accent II tone above a pulmonary artery, AP – 180/110 mm mercury. There is the loosened breathing above lungs with the far of moist wheezes in lower fate What probably became the reason of worsening of the state?
- *Edema of lungs
 - Embolism of pulmonary artery
 - The repeated heart attack myocardium
 - Hypertensive crisis
 - Attack of bronchial asthma
142. Patient L is hospitalized in gynecological department with the temperature of 39 degrees C, with complaints of pain in the bottom of stomach, vomit, diarrhea . Criminal abortion have been done 4 days before AP 80/60, breathing is difficult, psychosomatic excitation. Symptom of Schotkin-blumberg is positive Uterus is enlarged as on 9 weeks of pregnancy, limitedly mobile, painful Pus with blood appears Your Diagnosis?
- *septic shock
 - Perforation of uterus
 - Pelvic peritonitis
 - Acute appendicitis
 - Acute adnexia inflammation
143. Patient N., 28 years old 6 day after the complicated birth The clinical hematological signs of subacute disseminated intravascular coagulation syndrome developed after skin hemorrhage and uterine bleeding. The state of patient is very bad blood: Er-2,7 of T/l, Hb-78 of gm/l, CI - 0,93, L-4,7 of Gm/l, thrombocytes-88 of gm/l, time of blood clotting - 16 min, prothrombin time - 25 sec, ethanol test +, fibrinogen-1,4 gramme/l, What preparations should be prescribed ?
- *frozen plasma
 - Heparinum
 - Reopoliglycin
 - Cryoprecipitate
 - U-aminokapric acid
144. Patient N., 40 years, groom. In anamnesis there is an allergy to nonsteroidal antiinflammatory. After injection of antitetanus on a method Besredko concerning the hammered wound of right shin, through 20 minutes, there was an acute weakness, laboured breath, through 10 minutes, loss of consciousness What mechanism of development of anaphylactic form of illness?
- Sensitization to the albumen of horse whey
 - Low quality of horse whey
 - Breach of the technique of PPS injection
 - Presence in anamnesis of medical allergy
 - Infection of whey
145. Patient N., 40 years, groom. In anamnesis there is an allergy to nonsteroidal antiinflammatory. After injection of antytetanus on a method Besredko concerning the hammered wound of right shin, through 20 minutes, there was an acute weakness, laboured breath,

through 10 minutes, loss of consciousness. What mechanism of development of anaphylactic form of illness?

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 - B. Low quality of horse whey
 - C. Breach of the technique of PPS injection
 - D. Presence in anamnesis of medical allergy
 - E. Infection of whey
146. Person 48 years, patient by the heart attack of myocardium, suddenly lost consciousness, breathing and palpitation. On ECG of high-wave fibrillation of ventricle. Conducted defibrillation. Did not pick up normal cardiac activity. What medicine needs to be entered for the rise of sensuality to defibrillation?
- A. *Amiodaron
 - B. Propranolon
 - C. Lidocain
 - D. Strofantin
 - E. Atropini sulfati
147. Pharmacological medicine, that diminish the (afterload) left ventricle at a patient with the acute heart attack of myocardium, are not included
- A. nitroglycerine
 - B. *strophantine
 - C. nitroprussid sodium
 - D. esmolol (brevibloc)
 - E. nifedipinum
148. Sick 46 years treated oneself in a therapeutic department with pneumonia of lower lobe of right lung. Planned antibacterial therapy - amoxiclav. After 40 min after intramuscular injection of duty dose, the patient felt dizziness, pain behind a breastbone. AT 60/40 mm Hg, pulse, - 120 a min., rhythmical. During examination of lungs: wheezes under both lung. Temperature is 38,5. What is worsening of the condition related to?
- A. *Anaphylactic shock
 - B. Infectious toxic shock
 - C. Collapse
 - D. Tromboembolia of pulmonary artery
 - E. Infectious shock
149. The patient 20 years old, delivered to ambulance department on the 2nd day of illness in a grave condition: temperature of body 39°C, symptoms of intoxication are expressed. On extremities, trunk, buttocks, present hemorrhagic rash as eczema with necrosis in the center. One day before cut his leg. Now has the wound in that place. In 2 hours the decline of AP is registered from 100/70 to 60/30 mm of Hg, diffused cyanosis. Application of prednisolon of 120 mg and reopolyglucin did not give any effect. What complication does it follow to think about?
- A. acute sub renal failure
 - B. *Septic shock
 - C. hypovolemic shock
 - D. Hemorrhagic shock
 - E. Respirator distress syndrom of adults
150. The patient 32 years have infusion of native plasma. At the end of infusion the state became worse: disorientation, cyanosis, excitation, appeared hypersalivation, tachypnoea, AP = 70/40 mm Hg, in lungs – the dissipated dry wheeze. What medicine must be injected firstly?
- A. *Adrenalin.
 - B. Suprastin.
 - C. Hidrocortizon.
 - D. Dopamin.
 - E. Eufilin.
151. To patient P., 50 years, with an unspecified ulcerous colitis with the purpose of correction of anaemia transfusion of self-group blood 500 ml A(II) the Rh(-) was conducted. A doctor went out

from a chamber after conducting of necessary tests before hemotransfusion. In 20 minutes he was quickly asked to the patient. Patient without consciousness. The cyanosis of upper body part. Irregular breathing with the selection of a plenty of foamy, with the admixtures of blood, phlegm. Pulse on peripheries and arterial pressure are not determined. Tones of heart are deaf, unrhythmical. An ampoule and transfusion system is empty. What complication arose up as a result of hemotransfusion?

- A. *Air embolism of pulmonary artery
 - B. Tromboembolism of pulmonary artery
 - C. Edema of lungs
 - D. Heart attack of myocardium
 - E. Syndrome of massive hemotransfusion
152. To the patient 45 years with suspicion on cholecystitis the rentgencontrast i/v is quickly injecte. Tahycardiya, arterial hypotension, cyanosis, shortness of breath appeared, acute swelling of veins of neck, extension of liver, CVT to 200 mm wt.st. acute insufficiency of what part of the cardial-vascular system is observed at a patient?
- A. *Right ventricle of heart
 - B. The left ventricle of heart
 - C. Both ventricles of heart
 - D. Vessels
 - E. Uneffective heart
153. A man 50 years of asymptomatic mixed goiter. At the first stage, it should appoint
- A. *only observation
 - B. thyroid hormones to suppress the function of cancer
 - C. propylthiouracil
 - D. subtotal thyroidectomy
 - E. radioiodine
154. In patient K, aged 26, noted a relapse of hyperthyroidism after medical treatment. Your tactics.
- A. *Surgical treatment
 - B. Continue medical treatment
 - C. Outpatient
 - D. Treatment is not required
 - E. Is no right answer
155. In patients after operations on the thyroid gland have complaints about the shortness of breath, lethargy, convulsions. What is the cause of these clinical manifestations?
- A. *lower calcium concentration
 - B. lower content of iodothyronine
 - C. increasing concentrations of glucose
 - D. increasing concentration of potassium
 - E. no right answer
156. On examination, the patient was 32 years reveal the formation of the left lobe of the thyroid gland size 4x6 cm, painless at palpation. What additional diagnostic method to assign?
- A. *Thyroid gland
 - B. Radiography of the neck
 - C. Doppler
 - D. Rheovasography
 - E. EEG
157. On examination, the patient was 32 years reveal the formation of the left lobe of the thyroid gland size 4x6 cm, painless at palpation. What analysis should be performed in order to clarify the diagnosis?
- A. *Thyroid hormones
 - B. Total blood
 - C. Urinalysis
 - D. Immunogram

- E. Protein fraction
158. Patient D, aged 39, admitted to the hospital with the diagnosis: diffuse toxic goiter. What study be done.
- A. *Investigation of iodine hormones in the blood serum
 - B. EFGDS
 - C. Is no right answer
 - D. Rheovasography
 - E. Doppler
159. Patient J., 57, was admitted to the surgical clinic with a diagnosis: euthyroid nodular goiter. Choose the correct treatment option.
- A. *resection of the thyroid gland with maximal preservation of healthy tissue and routine histological examination
 - B. enucleation
 - C. medication
 - D. subtotal resection of the thyroid gland
 - E. excision of the node with the routine histological examination
160. Patient K, aged 49, was admitted to the hospital with the diagnosis: diffuse toxic goiter. What study be done.
- A. *Scanning of the thyroid gland
 - B. EFGDS
 - C. Rheovasography
 - D. Doppler
 - E. There is no correct answer
161. Patient K., aged 45, lives in the area of iodine deficiency, was admitted to the clinic with complaints of enlarged thyroid gland. What is the most likely diagnosis in a patient?
- A. *goiter
 - B. acute strumitis
 - C. sporadic goiter
 - D. epidemic goiter
 - E. mass thyrotoxicosis
162. Patient M, 39 years old, was admitted to the hospital with the diagnosis: diffuse toxic goiter. What study be done.
- A. *Thyroid gland
 - B. EFGDS
 - C. Rheovasography
 - D. Doppler
 - E. Is no right answer
163. Patient M., aged 35, lives in the area of iodine deficiency, was admitted to the clinic with complaints of enlarged thyroid gland. What is the most likely diagnosis in a patient?
- A. *there is no right answer
 - B. acute strumitis
 - C. sporadic goiter
 - D. epidemic goiter
 - E. mass thyrotoxicosis
164. Patient O., aged 39, on the diffuse toxic goiter performed subtotal resection of the thyroid gland. One day the patient became restless, twitching of facial muscles appeared convulsive reduction of hands. Treatment.
- A. *the introduction of calcium chloride intravenously
 - B. introduction Seduxen
 - C. introduction of iodine
 - D. infusion therapy
 - E. introduction of calcium chloride oral

165. Patient O., aged 39, on the diffuse toxic goiter performed subtotal resection of the thyroid gland. One day the patient became restless, twitching of facial muscles appeared convulsive reduction of hands. What mated complication of surgery?
- *Removal of parathyroid glands
 - Iodine deficiency
 - Lack of thyroid tissue
 - Increased thyroid hormone
 - Increased parathyroid hormone
166. ?Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. At USD: tissue homogeneous, tissue hypertrophy hyper. The most likely diagnosis
- *Toxic goiter
 - Hashimoto struma
 - Acute thyroiditis
 - nodular goiter
 - goiter De Quervain
167. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. At USD: tissue homogeneous, tissue hypertrophy hyper. The most likely diagnosis
- *there is no right answer
 - Hashimoto's thyroiditis
 - Acute thyroiditis
 - nodular goiter
 - goiter De Quervain
168. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. What additional diagnostic method to assign?
- *Thyroid gland
 - Radiography of the neck
 - Doppler
 - Reovazography
 - EEG
169. Patient S., 43, in the last 5 months of worry tearfulness, irritability, fatigue, progressive weight loss. On palpation the thyroid gland increased to III class., painless. Pulse 110-120 in minute, regular, blood pressure - 150/80. What analysis should be performed in order to clarify the diagnosis?
- *Total blood
 - Urinalysis
 - Thyroid hormones
 - Protein fraction
 - Immunogram
170. Patients after resection of the thyroid gland have cramps, Hvosstek and Trousseau symptoms. What complication is the patient?
- *gipoparatireosis
 - laryngeal nerve injury
 - residual effects of thyrotoxicosis
 - thyrotoxic crisis
 - hypothyroidism
171. Patients after resection of the thyroid having convulsions, symptoms by Chvostek and Trousseau. What a complication arose in a patient?
- *there is no right answer
 - laryngeal nerve injury

- C. residual effects of hyperthyroidism
 - D. thyrotoxic crisis
 - E. hypothyroidism
172. Patients after resection of the thyroid having convulsions, symptoms by Chvostek and Trousseau. What a complication is arose in a patient?
- A. *hypoparathyreosis
 - B. laryngeal nerve injury
 - C. residual effects of hyperthyroidism
 - D. thyrotoxic crisis
 - E. hypothyroidism
173. The patient complaints of excessive sweating, hand tremor, exophthalmos. The most likely diagnosis.
- A. *Strumit
 - B. Goiter
 - C. Tireodit
 - D. Thyrotoxicosis
 - E. Is no right answer
174. The patient diagnosed nodular nontoxic goiter. What operation is indicated the patient?
- A. *resection of the affected lobe with histological examination
 - B. conservative treatment of thyroxine
 - C. removal of the affected lobe, isthmus and central lymph node dissection
 - D. enucleation site
 - E. subtotal thyroidectomy
175. The patient diagnosed nodular nontoxic goiter. What operation is indicated the patient?
- A. *No right answer
 - B. conservative treatment of thyroxine
 - C. removal of the affected lobe, isthmus and central lymph node dissection
 - D. enucleation site
 - E. subtotal thyroidectomy
176. The patient diagnosed thyrotoxicosis. Which of the following symptoms suggests the pathology?
- A. *Exophthalmos
 - B. Peritoneal signs
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
177. The patient diagnosed thyrotoxicosis. Which of the following symptoms suggests the pathology?
- A. *Graefe symptom
 - B. Peritoneal signs
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
178. The patient diagnosed thyrotoxicosis. Which of the following symptoms suggests the pathology?
- A. *Mobius symptom
 - B. Peritoneal signs
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
179. The patient diagnosed thyrotoxicosis. Which of the following symptoms suggests the pathology?
- A. *Tachycardia
 - B. Peritoneal signs

- C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
180. The patient diagnosed thyrotoxicosis. Which of the following symptoms suggests the pathology?
- A. *Tremor of the upper limbs
 - B. Peritoneal signs
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
181. The patient diagnosed with an aberrant goiter. Refine the definition of aberrant goiter.
- A. *cancer of the thyroid gland
 - B. metastases of thyroid cancer in the liver
 - C. atypical location of the thyroid gland
 - D. all true
 - E. all wrong
182. The patient diagnosed with autoimmune thyroiditis. Which of the following symptoms characteristic of this disease?
- A. *increase and thickening of the thyroid gland, hypothyroidism
 - B. increase and thickening of the thyroid gland, fever
 - C. increase and thickening of the thyroid gland, hypothyroidism, fever
 - D. increase and thickening of the thyroid gland,
 - E. thyrotoxicosis, fever
183. The patient diagnosed with nodular euthyroid goiter left lobe of the thyroid gland. What is the optimal treatment option.
- A. *hemistrumectomy or resection of the lobe of the thyroid gland
 - B. excision of the node with the routine histological examination
 - C. enucleation site
 - D. subtotal thyroidectomy
 - E. conservative treatment thyroidin
184. The patient lives in the area of endemic iodine. What can be used to prevent goitre?
- A. merkasalil
 - B. vaccination
 - C. iodine
 - D. improving the social life of the population
 - E. *iodination salt
185. The patient made thyreoidectomy. Which of the following complications may occur in a patient?
- A. *Pneumathemia
 - B. Onychomycosis
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
186. The patient made thyreoidectomy. Which of the following complications may occur in a patient?
- A. *Damage to the trachea
 - B. Onychomycosis
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
187. The patient made thyreoidectomy. Which of the following complications may occur in a patient?
- A. *Damage to the recurrent laryngeal nerve
 - B. Onychomycosis

- C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
188. The patient made thyroidectomy. Which of the following complications may occur in a patient?
- A. *Bleeding from the wound
 - B. Onychomycosis
 - C. Trophic ulcer
 - D. Limb gangrene
 - E. No right answer
189. The patient S., 54 years old, diagnosed with goiter II degree. What is characteristic of the III degree of increase in thyroid gland?
- A. *visible swallowing
 - B. giant goiter
 - C. determined only by palpation
 - D. visible only when swallowing
 - E. determined only on ultrasound
190. The patient S., 61, suffering for 2 years nodular goiter, recently noted a rapid increase in the node. On scanning image identified a "cold node". Specify the most probable cause of this condition
- A. *node malignancy
 - B. cystic degeneration of the node
 - C. autoimmune
 - D. all true
 - E. hemorrhage site
191. The patient S., 61, suffering for 2 years nodular goiter, recently noted a rapid increase in the node. What method of diagnosis is the most informative?
- A. *scanning with radioactive iodine
 - B. X-ray of the neck
 - C. Doppler
 - D. Rheovasography
 - E. EEG
192. The patient underwent surgery on the thyroid gland. Which of the following post-operative complications can sports a patient?
- A. *No right answer
 - B. Ischemia of the upper extremities
 - C. Ischemia of lower extremities
 - D. Ileus
 - E. Gastric
193. The patient underwent surgery on the thyroid gland. Which of the following postoperative complications may be sports in the patient?
- A. *Hypocalcemia
 - B. Ischemia of the upper extremities
 - C. Ischemia of lower extremities
 - D. Ileus
 - E. Gastric
194. The patient underwent surgery on the thyroid gland. Which of the following postoperative complications may be sports in the patient?
- A. *Paresis of the recurrent laryngeal nerve
 - B. Ischemia of the upper extremities
 - C. Ischemia of lower extremities
 - D. Ileus
 - E. Gastric

195. The patient underwent surgery on the thyroid gland. Which of the following postoperative complications may be sports in the patient?
- *Thyrototoxic crisis
 - Ischemia of the upper extremities
 - Ischemia of lower extremities
 - Ileus
 - Gastric
196. The patient V., age 56, diagnosed with goiter of third degree. What is characteristic of the III degree of increase in thyroid gland?
- *visible without swallowing
 - giant goiter
 - determined only by palpation
 - visible only when swallowing
 - determined only on ultrasound
197. The patient was admitted with the diagnosis: diffuse toxic goiter. What operation is indicated the patient?
- *subtotal resection of the thyroid gland
 - thyroidectomy
 - hemistrumectomy
 - strumectomy
 - no right answer
198. The patient was admitted with the diagnosis: diffuse toxic goiter. What operation is indicated the patient?
- *No right answer
 - thyroidectomy
 - hemistrumectomy
 - strumectomy
 - no right answer
199. The patient was planned to study the thyroid gland with radioactive iodine-131. How much time should not use iodine and thyreostatics?
- *30 days
 - 50 days
 - 40 days
 - 10 days
 - 20 days
200. The patient was planned to study the thyroid gland with radioactive iodine-131. How much time should not use iodine and thyreostatics?
- *No right answer
 - 50 days
 - 40 days
 - 10 days
 - 20 days
201. The patient's 43 years revealed an increase in the left lobe of the thyroid gland. When scanning in this region found a hot site. Diagnosis.
- *nodular toxic goiter
 - diffuse non-toxic goiter
 - toxic goiter
 - multinodular toxic goiter
 - non-toxic nodular goiter
202. The patient's 60 years in the last 3 months has been rapidly increasing dense mass in the left lobe of the thyroid gland. Effects of hyperthyroidism is not. At thyroid scan revealed a cold junction. Preliminary diagnosis
- *thyroid cancer
 - lipoma of the thyroid gland

- C. cyst
 - D. metastasis of lung cancer
 - E. thyroid cyst
203. A Patient admitted to the clinic with a diagnosis: obliterating atherosclerosis of lower extremities, chronic arterial ischemia I stage. The most informative type of research vessels are:
- A. *Ultrasound examination
 - B. Rheovasography
 - C. Thermometry
 - D. Palpation identification of artery pulsation
 - E. Venogram
204. A patient admitted to the clinic with a diagnosis: obliterating atherosclerosis of lower extremities, chronic arterial ischemia I stage. The most informative type of research vessels are:
- A. *Arteriography
 - B. Rheovasography
 - C. Thermometry
 - D. Palpation identification of artery pulsation
 - E. Venogram
205. A patient admitted to the clinic with a diagnosis: obliterating atherosclerosis of lower extremities, chronic arterial ischemia I stage. The most informative type of research vessels are:
- A. *No right answer
 - B. Rheovasography
 - C. Thermometry
 - D. Palpation identification of artery pulsation
 - E. Venogram
206. A patient admitted with complaints of pain in the lower extremities during the passage of more than 1000 m. What is the stage of chronic arterial insufficiency in the patient?
- A. *I
 - B. II A
 - C. II B
 - D. III
 - E. IV
207. A patient admitted with complaints of pain in the lower extremities during the passage of 300 - 400 m. What is the stage of chronic arterial insufficiency in the patient?
- A. *II A
 - B. I
 - C. II B
 - D. III
 - E. IV
208. A patient admitted with complaints of pain in the lower extremities during the passage of 200 m. What is the stage of chronic arterial insufficiency is the patient?
- A. *II B
 - B. I
 - C. II A
 - D. III
 - E. IV
209. A patient admitted with complaints of pain at rest. What stage of chronic arterial insufficiency has the patient?
- A. *III
 - B. I
 - C. II A
 - D. II B
 - E. IV
210. A patient admitted with complaints of pain from intermittent claudication, and impotence. What is the diagnosis can be suspected in a patient?

- A. *Leriche syndrome
 - B. Obliterating endarteritis
 - C. Deep vein thrombosis leg
 - D. Iliac femoral thrombosis
 - E. Coarctation of aorta
211. A patient admitted with complaints of pain in the muscles of the buttocks and waist. What is the diagnosis can be suspected in a patient?
- A. *Leriche syndrome
 - B. Obliterating endarteritis
 - C. Deep vein thrombosis leg
 - D. Iliac femoral thrombosis
 - E. Coarctation of aorta
212. At examination the patient was diagnosed the absence pulse on femoral arteries. What is the diagnosis can be suspected in a patient?
- A. *Leriche syndrome
 - B. Obliterating endarteritis
 - C. Deep vein thrombosis leg
 - D. Iliac femoral thrombosis
 - E. Coarctation of aorta
213. The patient 53 years old admitted to hospital with suspected atherosclerosis arteries of lower extremities. Differential diagnosis should be with:
- A. *Obliterative endarteritis
 - B. Deep vein thrombosis of lower leg
 - C. Iliac vein thrombosis
 - D. Varicosity
 - E. Thrombophlebitis superficial veins
214. The patient 53 years old admitted to hospital with suspected atherosclerosis of arteries of lower extremities. Differential diagnosis should be with:
- A. *Diabetic angiopathy
 - B. Deep vein thrombosis of lower leg
 - C. Iliac vein thrombosis
 - D. Varicosity
 - E. Thrombophlebitis superficial veins
215. The patient aged 53 years admitted to hospital with suspected atherosclerosis of arteries of lower extremities. Differential diagnosis should be with:
- A. *Sciatica
 - B. Deep vein thrombosis of lower leg
 - C. Thrombosis of iliac vein
 - D. Varicosity
 - E. Thrombophlebitis superficial veins
216. The patient admitted to hospital with complaints of pain at rest. What should be used to block pain?
- A. *Epidural block
 - B. No-shpa
 - C. Trental
 - D. Rheopolyglucin
 - E. Solcosery
217. The patient admitted to hospital with complaints of pain at rest. What should be used to block pain?
- A. *Paravertebral sympathetic blockade
 - B. No shpa
 - C. Trental
 - D. Rheopolyglucine
 - E. Solcoseryl

218. Patient is in hospital with diagnosis: obliterating atherosclerosis of arteries lower extremities, chronic arterial insufficiency II stage. To improve the rheological properties of blood should be used:
- A. *Rheopoliglucin
 - B. Seduksen
 - C. Actovegin
 - D. Niacin
 - E. Papaverine
219. Patient is in hospital with a diagnosis: arterial occlusive disease of the lower extremities, chronic arterial insufficiency II stage. To improve the rheological properties of blood should be used:
- A. *Trental
 - B. Seduksen
 - C. Actovegin
 - D. Niacin
 - E. Papaverine
220. Patient is in hospital with a diagnosis: obliterating atherosclerosis of arteries lower extremities, chronic arterial insufficiency II stage. To remove vasospasm should be used:
- A. *Papaverine
 - B. Rheopoliglikin
 - C. Seduksen
 - D. Actovegin
 - E. Niacin
221. Patient to detect arterial ischemia need to be functional tests:
- A. *Oppel
 - B. Troyanov
 - C. Sidorenko
 - D. Stepanova
 - E. Kokket
222. Patient to detect arterial ischemia need to be functional tests:
- A. *Goldflam
 - B. Troyanov
 - C. Sidorenko
 - D. Stepanova
 - E. Kokket
223. Patient to detect arterial ischemia need to be functional tests:
- A. *Panchenko
 - B. Troyanov
 - C. Sidorenko
 - D. Stepanova
 - E. Kokket
224. The patient 47 years old complained on pain in the calf muscles during walking. What is this symptom:
- A. *Intermittent claudication
 - B. Troyanov
 - C. Oppel
 - D. Panchenko
 - E. Kokket
225. At doppler ultrasound study in the patients revealed atherosclerotic changes in arteries. What method of diagnosis must be pursued to clarify the localization process?
- A. *Arteriography
 - B. Thermometry
 - C. Radiography of limbs
 - D. Radiography of the chest cavity

- E. ECG
226. The patient admitted to hospital with a diagnosis: embolism the left popliteal artery. What diseases can cause embolism?
- A. *Rheumatic heart disease
 - B. Acute pancreatitis
 - C. Acute cholecystitis
 - D. Ulcer
 - E. Obliterate endarteritis
227. The patient admitted to hospital with a diagnosis: embolism the left popliteal artery . What is the most probable cause of embolism?
- A. *Myocardial infarction
 - B. Acute pancreatitis
 - C. Acute cholecystitis
 - D. Ulcer
 - E. Obliterate endarteritis
228. The patient admitted to hospital with a diagnosis: embolism the right popliteal artery . What is the most probable cause of embolism?
- A. *Cardiac aneurysm
 - B. Acute pancreatitis
 - C. Acute cholecystitis
 - D. Ulcer
 - E. Obliterate endarteritis
229. The patient admitted to hospital with a diagnosis: embolism the right popliteal artery . What is the most probable cause of embolism?
- A. *Aneurysm of femoral artery
 - B. Acute pancreatitis
 - C. Acute cholecystitis
 - D. Ulcer
 - E. Obliterate endarteritis
230. The patient admitted to hospital with a diagnosis: embolism the left popliteal artery . What is the most probable cause of embolism?
- A. *Septic endocarditis
 - B. Acute pancreatitis
 - C. Acute cholecystitis
 - D. Ulcer
 - E. Obliterate endarteritis
231. The patient admitted to hospital with suspected embolism the left popliteal artery. What is the symptom will testify in favor of embolism?
- A. *Severe pain in the limbs
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
232. *The patient admitted to hospital with suspected embolism right popliteal artery. What is the symptom will testify in favor of embolism?
- A. *Cold extremities
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
233. The patient admitted to hospital with suspected embolism the right popliteal artery. What is the symptom will testify in favor of embolism?
- A. *Acute weakness in the limbs
 - B. Filling saphenous veins

- C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
234. The patient admitted to hospital with suspected embolism left popliteal artery. What is the symptom will testify in favor of embolism?
- A. *Pale skin limbs
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
235. The patient admitted to hospital with suspected embolism, right femoral artery. What is the symptom will testify in favor of embolism?
- A. *The deterioration of the picture subcutaneous veins of the affected limb
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
236. The patient admitted to hospital with suspected embolism the right femoral artery. What is the symptom will testify in favor of embolism?
- A. *Changing the surface sensitivity on the affected limb
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
237. The patient admitted to hospital with suspected embolism the right femoral artery. What is the symptom will testify in favor of embolism?
- A. *Changing a deep sensitivity to the affected limb
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
238. In the patient admitted to hospital with suspected embolism the right femoral artery. What is the symptom will testify in favor of embolism?
- A. *The disappearance of pulsation with the level of the femoral artery
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
239. In the patient admitted to hospital with suspected embolism the right popliteal artery. What is the symptom will testify in favor of embolism?
- A. *The disappearance of pulsation with the level of the popliteal artery
 - B. Filling saphenous veins
 - C. Trophic ulcer leg
 - D. Varicose saphenous veins
 - E. Reticular varicose
240. In the patient zone of ischemia grabs both legs and lower abdomen. What are level of occlusion?
- A. *Bifurcation of the aorta
 - B. Iliac arteries
 - C. Femoral artery
 - D. Popliteal artery
 - E. Arteries foot
241. In the patient zone of ischemia captures the lower extremity to the inguinal ligament. What are level of occlusion?

- A. *Iliac arteries
 - B. Bifurcation of the aorta
 - C. Femoral artery
 - D. Popliteal artery
 - E. Arteries foot
242. In the patient zone of ischemia captures the lower extremity to the middle third of the thigh. What are level of occlusion?
- A. *Femoral artery
 - B. Bifurcation of the aorta
 - C. Iliac arteries
 - D. Popliteal artery
 - E. Arteries foot
243. In the patient zone of ischemia captures the lower extremity to the knee joint. What are level of occlusion?
- A. *Popliteal artery
 - B. Bifurcation of the aorta
 - C. Iliac arteries
 - D. Femoral artery
 - E. Arteries foot
244. In the patient zone of ischemia captures the whole hand. What are level of occlusion?
- A. *Subclavian artery
 - B. Axillary artery
 - C. Brachial artery
 - D. Ulnar artery
 - E. Arteries hand
245. The patient zone of ischemia grabs his hand to the upper third of the shoulder. What are level of occlusion?
- A. *Axillary artery
 - B. Subclavian artery
 - C. Brachial artery
 - D. Ulnar artery
 - E. Arteries hand
246. In the patient zone of ischemia grabs his hand to the middle third of the shoulder. What are level of occlusion?
- A. *Brachial artery
 - B. Axillary artery
 - C. Subclavian artery
 - D. Ulnar artery
 - E. Arteries hand
247. In the patient zone of ischemia grabs his hand to the elbow joint. What are level of occlusion?
- A. *Ulnar artery
 - B. Axillary artery
 - C. Subclavian artery
 - D. Brachial artery
 - E. Arteries hand
248. A patient admitted to hospital complaining of a sharp intense pain in the limbs. This symptom is typical for
- A. *Embolism
 - B. Thrombosis
 - C. Atherosclerotic lesions
 - D. Varicose
 - E. Postthrombotic syndrome

249. A patient admitted to hospital complaining of pain in the limbs with a gradual increase in intensity. This symptom is typical for
- A. *Thrombosis
 - B. Embolism
 - C. Atherosclerotic lesions
 - D. Varicose
 - E. Postthrombotic syndrome
250. The patient admitted to hospital with a stab wound femoral artery. Which type of injury is this damage?
- A. *Open injury
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Fire damage
251. The patient was admitted to hospital with a knife wound femoral artery. Which type of injury is this damage?
- A. *Open injury
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Fire damage
252. The patient admitted to hospital with chopped wound in the femoral artery. Which type of injury is this damage?
- A. *Open injury
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Fire damage
253. The patient admitted to hospital with a shattered wound in the femoral artery. Which type of injury is this damage?
- A. *Open injury
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
254. The patient admitted to hospital with a gunshot wound in the femoral artery. Which type of injury is this damage?
- A. *Open injury
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
255. The patient admitted to hospital with external bleeding in the femoral artery. Which type of injury is this damage?
- A. *Fresh wounds
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
256. The patient was admitted to hospital in a state of shock and damage in the femoral artery in history. Which type of injury is this damage?
- A. *Fresh wounds
 - B. Closed injury
 - C. Mixed damage

- D. Combined damage
 - E. Through damage
257. The patient was admitted to hospital with gangrene and limb damage in the femoral artery in history. Which type of injury is this damage?
- A. *Fresh wounds
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
258. The patient admitted to hospital pulsating hematoma and injuries to the femoral artery in history. Which type of injury is this damage?
- A. *Complicated wounds
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
259. The patient was admitted to hospital with bruising and damage to suppuration in the femoral artery in history. Which type of injury is this damage?
- A. *Complicated wounds
 - B. Closed injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
260. The patient admitted to hospital with a blunt injury in the femoral artery in history. Which type of injury is this damage?
- A. *Closed injury
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
261. The patient admitted to hospital with a compression of the femoral artery in history. Which type of injury is this damage?
- A. *Closed injury
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
262. The patient admitted to the hospital with damage to only the femoral artery. Which type of injury is this damage?
- A. *Isolated damage
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
263. The patient admitted to the hospital with damage to the femoral artery and internal organs. Which type of injury is this damage?
- A. *Combined injuries
 - B. Joint damage
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
264. The patient admitted to the hospital with damage to the popliteal artery in the first day after injury. To what this type of injury is damage?
- A. *Recent damage

- B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
265. The patient admitted to the hospital with damage to the popliteal artery on the second day after injury. To what this type of injury is damage?
- A. *Recent damage
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
266. The patient admitted to hospital with damage to the popliteal artery on the fourth day after injury. To what this type of injury is damage?
- A. *Complicated injuries
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
267. The patient admitted to the hospital with damage to the popliteal artery on the seventh day after injury. To what this type of injury is damage?
- A. *Complicated injuries
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
268. The patient admitted to the hospital with damage to the popliteal artery in the second week after injury. To what this type of injury is damage?
- A. *Complicated injuries
 - B. Open injury
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
269. A patient admitted to hospital with clinical symptoms of external bleeding in axillary artery. To whom this type of injury is damage?
- A. *Open injury
 - B. Complicated injuries
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
270. A patient admitted to hospital with clinical symptoms of external bleeding in axillary artery. To whom this type of injury is damage?
- A. *Open injury
 - B. Complicated injuries
 - C. Mixed damage
 - D. Combined damage
 - E. Through damage
271. A patient admitted to hospital with knife wounds in the superficial femoral artery. Which research method can give the most accurate information?
- A. *Vascular ultrasound
 - B. Thermometry
 - C. Radiography of the lower extremity
 - D. Radiography of abdominal
 - E. Rheovasography

272. A patient admitted to hospital with knife wounds in the superficial femoral artery. Which research method can give the most accurate information?
- A. *Angiography
 - B. Thermometry
 - C. Radiography of the lower extremity
 - D. Radiography of abdominal
 - E. Rheovasography
273. A patient admitted to hospital with knife wounds in the femoral artery. Which research method can give the most accurate information?
- A. *There is no correct answer
 - B. Thermometry
 - C. Radiography of the lower extremity
 - D. Radiography of abdominal
 - E. Rheovasography
274. A patient admitted to hospital with a diagnosis: Varicose veins the left lower extremity. At the examination revealed only varicose saphenous veins of legs without edema. What stage of varicose veins is in a patient?
- A. *I
 - B. II A
 - C. II B
 - D. III
 - E. IV
275. A patient admitted to hospital with a diagnosis: Varicose veins left lower extremity. At the examination revealed varicose saphenous veins of lower leg with swelling and pasty. What stage of varicose veins is in a patient?
- A. *II A
 - B. I
 - C. II B
 - D. III
 - E. IV
276. A patient admitted to hospital with a diagnosis: Varicose veins left lower extremity. At the examination revealed varicose saphenous veins with leg edema, pasty, and induration of the skin ulcer in the lower third of the leg. What stage of varicose veins is in a patient?
- A. *III
 - B. II A
 - C. I
 - D. II B
 - E. IV
277. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency I degree. What kind of treatment the patient is the best?
- A. *Elastic compression
 - B. Surgical treatment
 - C. Antihypertensive therapy
 - D. Anticoagulant therapy
 - E. Anticholesterol therapy
278. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency II degree. What kind of treatment the patient is the best?
- A. *Surgical treatment
 - B. Elastic compression
 - C. Antihypertensive therapy
 - D. Anticoagulant therapy
 - E. Anticholesterol therapy
279. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency III degree. What kind of treatment the patient is the best?

- A. *Surgical treatment
 - B. Elastic compression
 - C. Antihypertensive therapy
 - D. Anticoagulant therapy
 - E. Anticholesterol therapy
280. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency of II degree. What drug with venotonics properties should be appoint?
- A. *Detralex
 - B. Aspirin
 - C. Pentoksifilin
 - D. Heparin
 - E. Warphrin
281. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency of II degree. Which drug with venotonics properties should be appoint?
- A. *Phlebodia
 - B. Aspirin
 - C. Pentoksifilin
 - D. Heparin
 - E. Warphrin
282. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency of II degree. Which drug with venotonics properties should be appoint?
- A. *Detralex
 - B. Aspirin
 - C. Pentoksifilin
 - D. Heparin
 - E. Warphrin
283. A patient admitted to hospital with a diagnosis: Varicose veins left lower limb, chronic venous insufficiency of II degree. Which drug with antiplatelet properties should be designated?
- A. *Aspirin
 - B. Detralex
 - C. Pentoksifilin
 - D. Heparin
 - E. Warphrin
284. A patient admitted to hospital with a diagnosis: Varicose veins, right lower extremity, chronic venous insufficiency of II degree. Which drug with antiplatelet properties should be designated?
- A. *Cardimagnil
 - B. Detralex
 - C. Pentoksiphilin
 - D. Heparin
 - E. Warphrin
285. The patient admitted to the hospital with a diagnosis: Varicose disease of the right lower limb, chronic venous insufficiency III degree. Which of the following methods gives the most accurate information about the state of the venous system?
- A. *Vascular ultrasound
 - B. Radiography of the affected limb
 - C. Rheovasography
 - D. Thermometry
 - E. ECG
286. The patient admitted to the hospital with a diagnosis: Varicose disease of the right lower limb chronic venous insufficiency III degree. Which of the following methods gives the most accurate information about the state of the venous system?
- A. *Phlebography
 - B. Radiography of the affected limb

- C. Rheovasography
 - D. Thermometry
 - E. ECG
287. The patient admitted to the hospital with a diagnosis: Varicose disease of the right lower limb chronic venous insufficiency III degree. Which of the following methods gives the most accurate information about the state of communicative veins?
- A. *Vascular ultrasound
 - B. Radiography of the affected limb
 - C. Rheovasography
 - D. Thermometry
 - E. ECG
288. The patient admitted to the hospital with a diagnosis: Varicose disease of the right lower limb chronic venous insufficiency III degree. Which of the following methods gives the most accurate information about the state of communicative veins?
- A. *No right answer
 - B. Radiography of the affected limb
 - C. Rheovasography
 - D. Thermometry
 - E. ECG
289. The patient admitted to the hospital with a diagnosis: Varicose disease of the right lower limb chronic venous insufficiency III degree. Which of the following methods gives the most accurate information about the state of the venous system?
- A. *Flebotometry
 - B. Radiography of the affected limb
 - C. Rheovasography
 - D. Thermometry
 - E. ECG
290. The patient admitted to the hospital with a diagnosis: Varicose disease of the left lower limb, chronic venous insufficiency of Article III. Which of the following methods gives the most accurate information about the state of the venous system?
- A. *Functional Tests
 - B. Radiography of the affected limb
 - C. Rheovasography
 - D. Thermometry
 - E. ECG
291. The patient admitted to hospital with a diagnosis: Varicose disease, right lower extremity, chronic venous insufficiency III degree. Which diseases should be differentiate this disease?
- A. *Congenital arteriovenous fistula
 - B. Diabetic angiopathy
 - C. Atherosclerosis obliterans
 - D. Obliterative endarteritis
 - E. Raynaud's disease
292. The patient admitted to hospital with a diagnosis: Varicose disease, right lower extremity, chronic venous insufficiency III degree. Which diseases should be differentiate this disease?
- A. *Obtained arteriovenous fistula
 - B. Diabetic angiopathy
 - C. Atherosclerosis obliterans
 - D. Obliterative endarteritis
 - E. Raynaud's disease
293. The patient admitted to hospital with a diagnosis: Varicose disease, right lower extremity, chronic venous insufficiency III degree. Which diseases should be differentiate this disease?
- A. *Venous angiodysplasias
 - B. Diabetic angiopathy
 - C. Atherosclerosis obliterans

- D. Obliterative endarteritis
 - E. Raynaud's disease
294. The patient admitted to hospital with a diagnosis: Varicose disease, right lower extremity, chronic venous insufficiency III degree. Which diseases should be differentiate this disease?
- A. *No right answer
 - B. Diabetic angiopathy
 - C. Atherosclerosis obliterans
 - D. Obliterative endarteritis
 - E. Raynaud's disease
295. The patient admitted to hospital with a diagnosis: Varicose disease left lower limb, chronic venous insufficiency II drgree. What is a contraindication to surgical treatment?
- A. *Obstruction of deep veins
 - B. Chronic bronchitis
 - C. Past history of pneumonia
 - D. Past history of angina
 - E. Cholelithiasis
296. The patient admitted to hospital with a diagnosis: Varicose disease left lower limb, chronic venous insufficiency II degree. What is a contraindication to surgical treatment?
- A. *Angina
 - B. Chronic bronchitis
 - C. Past history of pneumonia
 - D. Past history of angina
 - E. Cholelithiasis
297. The patient admitted to hospital with a diagnosis: Varicose disease left lower limb, chronic venous insufficiency II degree. What is a contraindication to surgical treatment?
- A. *Heart failure
 - B. Chronic bronchitis
 - C. Past history of pneumonia
 - D. Past history of angina
298. The patient complains on burning pain in the limbs and the presence of painful dense strand of progress varicose veins in the leg. What is the most likely diagnosis in a patient?
- A. *Thrombophlebitis of subcutaneous veins
 - B. Deep vein thrombosis
 - C. Obliterating atherosclerosis
 - D. Occlusive disease
 - E. Diabetic angiopathy
299. The patient complains of burning pain in the limbs and the presence of painful dense strand of progress varicose veins in the leg with the transition to the lower third of the thigh. What is the most likely diagnosis in a patient?
- A. *Ascending thrombophlebitis of subcutaneous veins
 - B. Deep vein thrombosis
 - C. Obliterating atherosclerosis
 - D. Occlusive disease
 - E. Diabetic angiopathy
300. The patient complains of burning pain in the limbs and the presence of painful dense strand of progress varicose veins in the leg with the transition to the lower and middle third of the thigh. What is the most likely diagnosis in a patient?
- A. *Ascending thrombophlebitis of subcutaneous veins
 - B. Deep vein thrombosis
 - C. Obliterating atherosclerosis
 - D. Occlusive disease
 - E. Diabetic angiopathy

301. The patient complains of burning pain in the limbs and the presence of painful dense strand of progress varicose veins on the back of the tibia. What is the most likely diagnosis in a patient?
- *Small saphenous vein thrombophlebitis
 - Deep vein thrombosis
 - Obliterating atherosclerosis
 - Occlusive disease
 - Diabetic angiopathy
302. The patient complains on frequent thrombophlebitis in the zone of varicose great saphenous vein. What is the most likely diagnosis?
- *Migrating thrombophlebitis
 - Ascending thrombophlebitis
 - Deep vein thrombosis
 - Postthrombotic syndrome
 - Erysipelas
303. The patient complains on frequent thrombophlebitis in the zone of varicose great saphenous vein. What is the most likely diagnosis?
- *No right answer
 - Ascending thrombophlebitis
 - Deep vein thrombosis
 - Postthrombotic syndrome
 - Erysipelas
304. 304. The patient admitted to hospital with a diagnosis: Varicose veins left lower extremity. Acute ascending thrombophlebitis of subcutaneous veins. Tactics?
- *Surgical treatment
 - Conservative treatment
 - Physiotherapy
 - Sanatorium treatment
 - Treatment is not required
305. The patient admitted to hospital with a diagnosis: Varicose saphenous veins left lower extremity. Acute ascending thrombophlebitis of subcutaneous veins. Tactics?
- *No right answer
 - Conservative treatment
 - Physiotherapy
 - Sanatorium treatment
 - Treatment is not required
306. The patient underwent surgery for acute thrombophlebitis of subcutaneous veins. Which of the following drugs should be appoint after surgery?
- *Ciprinol
 - Vitamin C
 - Vitamin
 - Atenolol
 - Vasilip
307. The patient underwent surgery for acute thrombophlebitis of subcutaneous veins. Which of the following drugs should appoint after surgery?
- *Dikloberl
 - Vitamin C
 - Vitamin
 - Atenolol
 - Vasilip
308. The patient underwent surgery for acute thrombophlebitis of subcutaneous veins. Which of the following drugs should appoint after surgery?
- *Detralex
 - Vitamin C

- C. Vitamin
 - D. Atenolol
 - E. Vasilip
309. The patient underwent surgery for acute thrombophlebitis of subcutaneous veins. How long a patient should be advised to use elastic compression?
- A. *3 months.
 - B. 1 month.
 - C. 1 year
 - D. 6 months.
 - E. 2 weeks
310. The patient underwent surgery for acute thrombophlebitis of subcutaneous veins. How long a patient should be advised to use elastic compression?
- A. *There is no correct answer.
 - B. 1 month.
 - C. 1 year
 - D. 6 months.
 - E. 2 weeks
311. The patient complains on pain in the right leg, increases with foot movements. What is most likely diagnosis?
- A. *Thrombosis of the popliteal vein
 - B. Thrombosis of the femoral vein
 - C. Thrombosis of the iliac vein
 - D. Thrombosis of the inferior vena cava
 - E. Vein thrombosis aksilyarnoy
312. The patient complains on pain in the right leg, increases with foot movements. What research method is most reliable for further diagnosis?
- A. *Vascular ultrasound
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
313. The patient complains on pain in the right leg, increases with foot movements. What research method is most reliable for further diagnosis?
- A. *Phlebography
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
314. The patient complains on pain in the right leg and thigh, reinforced at moving foot and the knee joint. What is most likely diagnosis?
- A. *Thrombosis of superficial femoral vein
 - B. Thrombosis of the popliteal vein
 - C. Thrombosis of the iliac vein
 - D. Thrombosis of the vena cava inferior
 - E. Thrombosis Axillary vein
315. The patient complains on pain in the right leg hip, increases with movements in the foot and the knee joint. What research method is most reliable for further diagnosis?
- A. *Vascular ultrasound
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
316. The patient complains on pain in the right leg hip, increases with movements in the foot and the knee joint. What research method is most reliable for further diagnosis?

- A. *Phlebography
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
317. The patient complains on pain in the right leg and thigh, reinforced at moving foot and the knee joint. There is hypercyanotic color skin limbs. What is most likely diagnosis?
- A. *Thrombosis of common femoral vein
 - B. Thrombosis of the popliteal vein
 - C. Thrombosis of the iliac vein
 - D. Thrombosis of the inferior vena cava
 - E. Thrombosis Axyllary vein
318. The patient complains on pain in the right leg and thigh, reinforced at moving foot and the knee joint. There is hypercyanotic color skin limbs. What research method is most reliable for further diagnosis?
- A. *Phlebography
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
319. The patient complains on pain in the right leg and thigh, reinforced at moving foot and the knee joint. There is hypercyanotic color skin limbs. What research method is most reliable for further diagnosis?
- A. *Vascular ultrasound
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
320. The patient complains on pain in the right leg and thigh, reinforced at moving foot and the knee joint. There is hypercyanotic color skin limbs. Swelling of limbs throughout and on the buttocks What is most likely diagnosis?
- A. *Thrombosis of the iliac-femoral segment
 - B. Thrombosis of the popliteal vein
 - C. Thrombosis of the iliac vein
 - D. Thrombosis of the inferior vena cava
 - E. Thrombosis Axyllary vein
321. The patient complains on pain in the right leg and thigh, reinforced at moving foot and the knee joint. There is hypercyanotic color skin limbs. Swelling of limbs throughout and on the buttocks. What research method is most reliable for further diagnosis?
- A. *Vascular ultrasound
 - B. Radiography of the affected limb
 - C. Ultrasonography of the abdomen
 - D. Radiography of the chest cavity
 - E. ECG
322. The patient admitted to the hospital with a diagnosis deep vein thrombosis left lower extremity. The patient suddenly began to worry shortness of breath, retrosternal pain. What complication can be suspected in a patient?
- A. *Pulmonary embolism
 - B. Superficial thrombophlebitis
 - C. Ulcer
 - D. Pharyngitis
 - E. Pleurisy

323. The patient admitted to the hospital with a diagnosis deep vein thrombosis left lower extremity. The patient suddenly began to worry shortness of breath, retrosternal pain. What method diagnosis should be used to refine the diagnosis?
- A. *ECG
 - B. Reovazography
 - C. Densitometry
 - D. Ultrasound of internal organs
 - E. Ultrasound vascular limb
324. The patient admitted to the hospital with a diagnosis deep vein thrombosis left lower extremity. The patient suddenly began to worry shortness of breath, pain with localized behind the breastbone, a feeling of fear. What complication can be suspected in a patient?
- A. *Pulmonary embolism
 - B. Superficial thrombophlebitis
 - C. Ulcer
 - D. Pharyngitis
 - E. Pleurisy
325. The patient admitted to the hospital with a diagnosis deep vein thrombosis left lower extremity. The patient suddenly began to worry shortness of breath, retrosternal pain. What method diagnosis should be used to refine the diagnosis?
- A. *Radiography of the chest
 - B. Reovazogrifiya
 - C. Densitometry
 - D. Ultrasound of internal organs
 - E. Ultrasound vascular limb
326. The patient suddenly appeared retrosternal pain, dyspnea, collapse with increased sweating, and a sharp V neck veins. Which version of PE is in this patient?
- A. *Acute course
 - B. Subacute course
 - C. Recidivism during
 - D. Mixed
 - E. Combined
327. In the patient suspected pulmonary embolism. Which of the following methods investigation is required to clarify the diagnosis?
- A. *Study of blood coagulation
 - B. Complete blood
 - C. Urinalysis
 - D. Determine the level of uric acid
 - E. Confirmation is not required
328. In the patient suspected pulmonary embolism. Which of the following methods investigation is required to clarify the diagnosis?
- A. *ECG
 - B. Complete blood
 - C. Urinalysis
 - D. Determine the level of uric acid
 - E. Confirmation is not required
329. In the patient suspected pulmonary embolism. Which of the following methods investigation is required to clarify the diagnosis?
- A. *Radiography of the chest cavity
 - B. Complete blood
 - C. Urinalysis
 - D. Determine the level of uric acid
 - E. Confirmation is not required
330. In the patient suspected pulmonary embolism. Which of the following methods investigation is required to clarify the diagnosis?

- A. *Angiopulmonography
 - B. Complete blood
 - C. Urinalysis
 - D. Determine the level of uric acid
 - E. Confirmation is not required
331. In the patient suspected pulmonary embolism. Which of the following methods investigation is required to clarify the diagnosis?
- A. *Lung scintigraphy
 - B. Complete blood
 - C. Urinalysis
 - D. Determine the level of uric acid
 - E. Confirmation is not required
332. In the patient suspected pulmonary embolism. Which of the following methods investigation is required to clarify the diagnosis?
- A. *Study of blood coagulation
 - B. Complete blood
 - C. Urinalysis
 - D. Determine the level of uric acid
 - E. Echocardiography
333. In the patient suspected pulmonary embolism. Done radiography of the chest cavity. Which of the following symptoms are indicate pulmonary embolism?
- A. *The rise of the dome diaphragm
 - B. Pulmonary fibrosis root
 - C. Availability Calcinates
 - D. Increased vascular pattern
 - E. No right answer
334. In the patient suspected pulmonary embolism. Done radiography of the chest cavity. Which of the following symptoms are indicate pulmonary embolism?
- A. *Pleural effusion in the sinuses
 - B. Pulmonary fibrosis root
 - C. Availability Calcinates
 - D. Increased vascular pattern
 - E. No right answer
335. In the patient suspected pulmonary embolism. Done radiography of the chest cavity. Which of the following symptoms are indicate pulmonary embolism?
- A. *The wedge-shaped shadow atelectases
 - B. Pulmonary fibrosis root
 - C. Availability Calcinates
 - D. Increased vascular pattern
 - E. No right answer
336. In the patient suspected pulmonary embolism. Done radiography of the chest cavity. Which of the following symptoms are indicate pulmonary embolism?
- A. *Breaking vessels near the root of the lungs
 - B. Pulmonary fibrosis root
 - C. Availability Calcinates
 - D. Increased vascular pattern
 - E. No right answer
337. In the patient suspected pulmonary embolism. Done radiography of the chest cavity. Which of the following symptoms are indicate pulmonary embolism?
- A. *Hyperperfusion of the contralateral lung
 - B. Pulmonary fibrosis root
 - C. Availability Calcinates
 - D. Increased vascular pattern
 - E. No right answer

338. In the patient suspected pulmonary embolism. Done radiography of the chest cavity. Which of the following symptoms are indicate pulmonary embolism?
- *Dilation of the right ventricle
 - Pulmonary fibrosis root
 - Availability Calcinates
 - Increased vascular pattern
 - No right answer
339. In the patient suspected pulmonary embolism. Echocardiography. Which of the following symptoms are indicate pulmonary embolism?
- *Advanced, hypokinetic right ventricle
 - Normal right ventricle
 - Spasm of the proximal pulmonary arteries
 - Relationships right ventricle / left ventricle is not broken
 - No right answer
340. In the patient suspected pulmonary embolism. Echocardiography. Which of the following symptoms are indicate pulmonary embolism?
- *Increased ratio of right ventricle / left ventricle
 - Normal right ventricle
 - Spasm of the proximal pulmonary arteries
 - Relationships right ventricle / left ventricle is not broken
 - No right answer
341. In the patient suspected pulmonary embolism. Echocardiography. Which of the following symptoms are indicate pulmonary embolism?
- *Dilation of proximal pulmonary arteries
 - Normal right ventricle
 - Spasm of the proximal pulmonary arteries
 - Relationships right ventricle / left ventricle is not broken
 - No right answer
342. In the patient suspected pulmonary embolism. Echocardiography. Which of the following symptoms are indicate pulmonary embolism?
- *Increasing the speed of blood tricuspid regurgitation $> 3.7 \text{ m / s}$
 - Normal right ventricle
 - Spasm of the proximal pulmonary arteries
 - Relationships right ventricle / left ventricle is not broken
 - No right answer
343. In the patient suspected pulmonary embolism. Echocardiography. Which of the following symptoms are indicate pulmonary embolism?
- *Violation of blood flow from the right ventricle
 - Normal right ventricle
 - Spasm of the proximal pulmonary arteries
 - Relationships right ventricle / left ventricle is not broken
 - No right answer
344. In the patient suspected pulmonary embolism. Echocardiography. Which of the following symptoms indicate pulmonary embolism?
- *Advanced vena cava inferior, which has persisted for inspiration
 - Normal right ventricle
 - Spasm of the proximal pulmonary arteries
 - Relationships right ventricle / left ventricle is not broken
 - No right answer
345. Patients with suspected pulmonary embolism made angiopulmonography. Which of the following symptoms is direct?
- *Vascular filling defects
 - Asymmetry of vascular contrast material filling
 - Slow progress or stasis of contrast

- D. Increased pulmonary artery trunk and its major branches
- E. No right answer