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SYMPTOMATOLOGY OF THERAPEUTIC DISEASES

Teaching manual

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The teaching manual presents a detailed analysis of the symptoms and syndromes of therapeutic diseases and describes the principal methods of examination at the patient's bedside.

The manual is intended for international medical students.

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СИМПТОМАТОЛОГИЯ ТЕРАПЕВТИЧЕСКИХ ЗАБОЛЕВАНИЙ

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В учебном пособии представлен подробный анализ симптомов и синдромов терапевтических заболеваний и описаны основные методы обследования у постели больного.
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CONTENTS

UNIT 1. GENERAL EXAMINATION OF THE PATIENT (INSPECTION)

1. The severity of the patient's general condition is determined by the presence and severity of decompensation of the vital functions of the organism. According to this, the doctor decides on urgency and the scope of diagnostic and therapeutic measures and establishes the prognosis. The first impression about the severity of the condition is formed on the basis of the present complaints and the data of general and local inspection. The final impression is formed on the basis of the results of the examination of the internal organs. The functional state of the cardiovascular and respiratory systems and the state of consciousness are of particular importance. The levels of patient's general condition are satisfactory, moderate, severe, extremely severe, agony and clinical death. General condition is defined as ***satisfactory*** if the functions of vital organs are relatively compensated. Patient's condition is defined as ***moderate*** if the disease has led to decompensation of the functions of vital organs, but poses no immediate threat to the patient's life. Such patients are admitted to hospital as there is a risk of the rapid disease progression and development of life-threatening complications. The patient's condition is defined as ***serious*** if decompensation of the functions of vital organs as a result of disease poses a direct threat to the patient's life or can lead to profound disability. Such patients need emergency hospital treatment in the intensive care unit. ***Extremely severe condition*** is defined as the condition when the patient may die in some hours if left without prompt and intensive treatment, the treatment being provided in the resuscitation department.

2. Position of the patient may be

- active: can be noted in mild diseases and in early stages of severe diseases
- passive: can be noted in unconscious condition or in severe weakness
- forced: when the patient occupies the least painful position

3. The state of patient's consciousness. There are the following kinds of consciousness:

Clear consciousness: the patient is aware of himself/herself, place, time and the environment, adequately and easily answers the questions.

Confused consciousness: depressive disorders of consciousness (inhibition);
irritative disorders of consciousness (excitation).

There are three degrees of depressed consciousness:

• Stupor is an inhibited (contused/obtunded) state, which the patient can come out for a short time if you talk to him/her. The patient is poorly oriented in the surroundings, the answers are slow, retarded.

• Sopor (hibernation) is a more profound disorder of consciousness. The patient does not react to other people, anwers the questions in one word, after a loud hail, sensitivity (both sensitive and tactile) is present.

• Coma is a state when a patient is unconscious, not responding to inspection procedure and when addressed, there is a significant diminuition in or absence of unconditioned reflexes.

Kinds of coma are alcoholic, apoplectic, hypoglycemic, uremic and others.

Irritative disorders of consciousness manifest in arousal of the central nervous system in which the patient has delusions not related to the real environment. The most frequent types of such disorders are delirium (delirium tremens in alcoholics – delirium) and hallucinations.

4. The patient's mood. It is necessary to pay attention to the presence of possible emotional disorders in the patient, i.e. apathy, depression, euphoria, emotional lability, irritability, etc.

5. Habitus.

Habitus includes constitution, height and weight.

Constitution (constitution is body build-up) is a sum of anatomical, physiological and psychological characteristics depending on heredity and influence of the environment:

- asthenic (is characterized by prevalence of longitudinal sizes of body)
- hypersthenic (is characterized by prevalence of diametrical sizes of body)
- normosthenic (average)

6. Examination of the head and face.

A change in shape and size of the head may be of diagnostic value. In hydrocephalus, there is excessive enlargement of the skull size. Abnormally small head size (microcephaly) is common in congenital mental hypoplasia. Square head shape, flattened at the top, with marked bumps may indicate past rachitis. Facial expression may depend on age and various pathological conditions. The following kinds of face are distinguished (diagnostic faces):

• ***Facies febrilis*** **(a feverish/flushed face)** is the hyperemic skin characterized by feverish glittering eyes and excited expression.

• ***Facies nephritica*** is pale, doughy/puffy face with puffiness of the upper and lower eyelids, puffiness under the eyes.

• ***Facies mitralis*** is characterized by marked cyanosis of the lips, cyanotic blush on the cheeks as "mitral butterflies".

• ***Corvisart's facies*** is typical of patients with heart failure. The face is doughy/puffy, of yellow pale color. There is marked cyanosis of the lips, nose tip and ear lobes. Parted lips and dull eyes are typical.

• ***"Wax doll" face*** is slightly doughy, very pale, yellowish white and translucent skin characteristic of patients with Addison-Biermer disease (vitamin B12 deficiency).

• ***hippocratic facies*** (hippocratic face) is typical of patients with generalized peritonitis. The face is deadly pale, pinched, with sunken eyes, a pointed nose and with drops of sweat.

• ***Risus sardonicus*** is a persistant grimace, in which the mouth is stretched as if in laughter and the forehead forms folds as in case of sadness or in tetanus.

• ***Parkinson's face*** is characterized by the absence of mimics like in encephalitis.

• ***Lion's face*** is marked by skin of face which gets thick with tubercles like in leprosy.

• ***In endocrine diseases***:

- acromegalia (enlarged nose, chin, brows)

- myxedema (functions of thyroid gland are reduced): the face is edematous/puffy, pale; the palpebral fissures are narrow

-thyreotoxicosis or hyperfunction of the thyroid gland (Basedow's disease) is characterized by a frightened face and exophthalmos

- in Cushing's disease when the face is crescent-shaped, red, with a growing beard and moustache in women.

7. Measuring temperature /Thermometry. Skin temperature mainly reflects the internal temperature of the body. In a healthy person's armpit, temperature ranges from 36.0 to 36.9 degrees Celsius, daily temperature fluctuations do not exceed 0.3 – 0.5 °C. In the mouth and rectum, the temperature is usually higher by 0.5 – 1.0 °C, but does not exceed 37.5 °C. Body temperature in the armpit which is above 36.9 °C as well as a significant difference between morning and evening temperatures (even under normal absolute figures) are considered pathological.

Increased body temperature is called a fever. Depending on the severity of hyperthermia, the following kinds of fever are distinguished:

- subfebrile (37.0 – 37.9 °C),
- febrile (38.0 – 38.9 °C),
- pyretic (39.0 – 41. 0 °C).
- hyperpyretic (more than 41.0 °C).

According to the temperature curve several types of fevers are distinguished:

- *Continuous* (constant) fever (f. continua) is a long, high fever, usually within the 38-39 °C, the daily fluctuations do not exceed 1.0 °C (in pneumonia).
- *Remitting* fever (f. remittens) is a prolonged increase in temperature. The daily fluctuations exceed 1.0 °C, morning minimum is above 37 °C (in purulent diseases).
- *Intermitting* fever (f. intermittens) is an alternation in high and normal temperature for a few days.
- *Hectic* fever (febris hectica) is a pyretic or hyperpyretic fever with daily range to 3-4 °C and lowering to the normal level (in sepsis).
- *Recurrent* fever (f. recurrens) is when 3 – 5 pyretic periods of fever are observed during the illness alternating with periods of normal temperature (recurrent/relapsing fever).
- *Undulate* fever (f. undulans) is smooth alternating periods of rise and fall in temperature (in brucellosis, lymphogranulematosis), which is well tolerated by patients.

8. Examination of the skin.

When examining the skin, attention is usually paid to its color, purity, integrity, moisture, elasticity, the presence of a rash, heamorrages, scarring, etc. The condition of the appendages of hair and nails is also assessed.

Skin color depends on the following factors: 1) thickness and transparency of the skin, the quantity of pigments contained therein; 2) maturity, depth and blood fullness of the skin vessels; 3) erythrocytes and hemoglobin content per unit of blood volume and the degree of saturation of hemoglobin with oxygen. Depending on race and ethnicity, skin color can be pale pink (corporal) or with various shades of yellow, red, brown and black. *Pathological changes in skin color* can be diffuse and localized, transient and steady. The following are the most common:

• ***Pallor*** of the skin often is caused by anemia of any origin and is necessarily accompanied by pallor of the mucous membranes and conjunctiva. Skin pallor without anemia occurs when there is a spasm of peripheral arterioles (aortic defects, kidney disease), redistribution of blood in acute vascular insufficiency, due to sudden profuse bleeding as well as in reduced the transparency of the upper layers of the skin or deeply located vessels. Pallor of the skin may have a peculiar shade in some diseases. It is greenish in iron-deficiency anemia, lemon in hemolysis, coffee with milk in subacute infectious endocarditis. You can sometimes detect congenital skin depigmentation – diffuse (albinism) or focal (vitiligo).

• ***Hyperemia*** (redness of the skin) is caused by enlargement of the peripheral vessels (fever, overheating, after drinking alcohol, in local inflammation of the skin or burns, emotional arousal, hypertensive crisis) or increase in hemoglobin and erythrocytes content in the blood volume unit (erythrocytosis).

• ***Cyanosis*** (blueness/bluish color) develops if the absolute amount of restored hemoglobin in the blood exceeds 40-50 g/L. Cynosis is most commonly observed as a result of: 1) respiratory failure, i.e. impairment of hemoglobin oxygenation in the lungs. With the development of central cyanosis in this case; 2) congestive heart failure, i.e. a slow blood flow at the periphery. The blood becomes oversaturated with restored hemoglobin. Cold, peripheral cyanosis (acrocyanosis) develops.

• ***Jaundice*** is caused by a high concentration of bilirubin in the skin and mucous membranes, with the increase in its content in the blood. De-

pending on the severity, duration and origin of hyperbilirubinemia the skin becomes yellow with different shades: from green (icterus verra) to olive-black (icterus melas) in mechanical jaundice, reddish (icterus ruber) in hepatitis, jaundiced in combination with pallor (icterus albĭcans) in hemolytic jaundice.

• ***Hyperpigmentation*** of the skin can develop as a result of excessive accumulation in its natural pigments (melanin) or deposit of pathological staining substances. Bronze skin is present in Addison's disease (chronic adrenal insufficiency). Haemochromatosis (bronze diabetes) is defined as deposit of iron. Hyperpigmentation can develop in cutaneous porphyria, in metabolic tyrosine and phenylalanine (alkaptonuria), arsenic, silver, gold poisoning. Focal hyperpigmentation (chloasma) is observed in pregnancy.

Skin irritation and hemorrhages are found in many diseases as their important diagnostic sign. There are two main types of skin rashes – inflammatory and hemorrhagic.

Inflammatory rashes include:

• ***Roseola*** is pale pink spots ranging from 1 to 5 mm in diameter which do not usually raise above the surrounding tissue, disappear on pressing and then reappear. Their appearance is connected with dilation of the small vessels. They are characteristic of typhoid fever, syphilis, paratyphoid fever. Roseola often turns to **papula** (a nodule) which is a soft mass raising above the surface of the skin. A papula can resolve completely when being transformed into **pustule** (a mass with pus).

• ***Macula*** is a larger spot up to 2 cm in diameter observed in dermatitis and secondary syphilis.

• ***Erythema*** is a large site of homogeneous skin redness with well-defined margins, observed in food idiosyncrasy, allergic reactions. Erythema nodosum (painful, reddish nodes on the shins) is often the response to tuberculosis, sarcoidosis, rheumatism. Annular erythema is more typical in rheumatism.

• ***Hives (urticaria)*** is reddish-whitish itchy blisters which tend to coalesce. It is observed in allergic reactions.

• ***Herpetic rash*** is vesicles (vesicula) up to 1 cm in diameter, containing liquid that burst after a few days with the formation of a crust on their site.

Most often it occurs on the lips (herpes labialis), the nose (herpes nasalis). Herpetic rash also occurs in herpes zoster.

Skin hemorrhages (hemorrhages) may be of different size, shape and location. They occur in contusions/bruises as well as in a number of diseases characterized by disturbances of hemostasis (thrombocytopenia, coagulopathy, vasculitis). There are ***petechiae*** (bleeding points), ***purpura*** (spots up to 5 mm), ***ecchymosis*** (larger spots). Skin hemorrhages can occur in diseases of the liver, scurvy, sepsis. On being pressed by a subject glass, hemorrhagic rash, unlike inflammatory one, does not go away and never turns pale.

Skin integrity may be impaired as a result of injuries, wounds, burns (erosion, abrasion). Large ulcerous defect occurs in tumour disintegration, tuberculosis, tertiary syphilis, trophic disorders of tissues in diabetes, circulatory failure.

Skin elasticity (turgor) can also have a diagnostic value: decrease in elasticity is indicative of dehydratation of the organism, exhaustion, and also occurs in the elderly. Increase in turgor indicates fluid retention (hidden swelling).

Examination of hair allows you to get important information about some anomalies: pathology of the thyroid gland, anemia. Brittle hair is observed in endocrine disease. Total/general alopecia is observed in some cases of systemic lupus erythematous, hyperthyroidism, radiation sickness. Focal/patchy baldness is observed in syphilis.

When examining the nails, attention is paid to the shape of the nail plate, its color, opacity. Nail plates become concave, lose their luster and transparency in anemia. Transverse nails can occur in a number of metabolic diseases. The nails become dull and brittle. Convex, or crystal, nail shape in combination with enlargement of the terminal finger phalanges (clubbing) is observed in patients with chronic lung disease.

9. Subcutaneous fat.

Subcutaneous fat is examined almost simultaneously with the skin. The development of fatty tissue reflects the patient's fat degree, or nutritional status. It is determined by skinfold thickness as follows: normal skin fold thickness on the anterior surface of the stomach at the level of the navel is 2-3 cm; under the shoulder and the back of the shoulder, it is 1-2 cm.

Nowadays, the Quetelet index (BMI – body mass index) is increasingly used to determine the nutritional status: weight (kg)/height (m^2). In case of satisfactory nutrition, the Quetelet index is 20-24. In obesity, it is more than 24 units. In body mass deficit, the index is less than 20 units.

In addition, attention is paid to the presence of **edema**, which is an important symptom of many diseases of the inner organs. Swelling may be general and local. *General edema* is associated with a common disease and characterized by a symmetric distribution, either in limited areas of the body or throughout the body. *Local edema* is due to any local disorder of blood or lymphatic system and characterized by asymmetrical arrangement (for example, in thrombophlebitis). The severity of edema syndrome (generalized swelling) may be different: hidden swelling, sponginess, marked swelling. The body of an adult human can retain up to 3-5 liters of liquid without marked swelling. When generalized swelling is caused by disorders of a general nature, transudate usually accumulates in the body cavities: ascites (hydroperitoneum), chest (hydrothorax), pericardium (hydropericardium). If swelling spreads over all the hypodermis and is detected in the cavities, this condition is called anasarca.

To detect peripheral edema, the following methods are used:

1) monitoring body weight;

2) measuring the amount of fluid you have taken and excreted;

3) pitting edema (a finger is put into the sweeling of the ankles, legs, sacrum, sternum. Pits, or small depressions, are common in case of edema).

10. Examination of the lymph nodes. The examination of the peripheral lymph nodes is of great importance in the diagnosis of certain diseases of the inner organs (blood system diseases, malignant neoplasms, tuberculosis). Lymph nodes perform filtration and immune function. Normally, peripheral lymph nodes are round or oval structures ranging in size from 5 to 10 mm. They do not protrude above the skin and are not detected on examination.

Examination of the lymph nodes is carried out in the corresponding symmetric fields as follows: chin/mental, submandibular, angle of the mandible, parotid, occipital, cervical posterior, cervical anterior, clavicular superior and inferior, axillary, elbow, inguinal, popliteal. When palpating

the lymph nodes, you should pay attention to their number, size, shape, texture, tenderness, mobility, coherence between themselves and with the surrounding tissue. The condition of the skin above the nodes is visualized: color, temperature, presence of ulceration or fistulas. Small single painless lymph nodes can be normally palpated in the submandibular, inguinal areas and, rarely, in the armpit.

In practice, the doctor comes across the two types of changes of lymph nodes:

1) Lymph nodes of an inflammatory nature. In acute lymphadenitis, the node is soft, sharply painful, the skin above them is hyperemic, hot to the touch. If regional lymphadenitis is caused by inflammation in the underlying limb, lymphangoit, or the track of hyperemia, can be found on the skin. There may be a local enlargement of the regional lymph nodes (in inflammatory diseases of the oral cavity, chronic tonsillitis, tuberculosis) and a widespread systemic lesion (in infectious mononucleosis, brucellosis).

2) Lymph nodes of neoplastic nature. In tumor lesion, the node is dense (consolidated/indurate) and of irregular form (tuberous), subsequently becomes inflexible. Common neoplastic lesion of the lymph nodes is observed in lymphogranulematosis, leukemia. In cancer of the stomach, a local enlargement is noted in metastases into the cervical lymph nodes on the left (Virchow's metastasis). In breast cancer, it is noted in metastases into the axillary lymph nodes.

11. Muscular system.

When examining the muscle development, the doctor assesses the degree of voluntary musclulature, the presence of general or local muscle atrophy, contractures, muscle tone as well as their tenderness on palpation.

The degree of muscle development is directly connected with their volume and bump on symmetric sections. Muscle atrophy often occurs in weakened/debilitated patients, diseases of the nervous system followed by paresis or paralysis, chronic lesions of the joints.Often there is also a muscle contracture defined as a persistent spasm of the muscles adjacent to the joint, the flexor one being the more common. Muscle strength is determined by the ability of the patient to provide active resistance movements (bending or straightening the limbs) or by means of a dynamometer. Muscle tone is checked on touching. Muscle tone is reduced in muscu-

lar atrophy, peripheral paralysis. The muscles become flabby and passive movements in the relevant joints become superfluous. Central paralysis, by contrast, combines with the hypertone of muscles which become dense and well contured. Passive movement is difficult and limited. On palpation, areas of considerable consolidation and tenderness can also be detected in muscular thickness, which points to inflammatory lesions in them (myositis). Foci of stony density in the muscular thickness are formed in the deposit of calcium salts, which is common in hyperparathyroidism.

12. Osteoarticular system. When examining the limbs, attention should be paid to their shape, correct/proper contours and symmetry. Attention is also paid to various kinds of defects, curvatures and other deformities of the skull bones, spine, chest and extremities. The most common spinal curvatures among the pathological bone deformities are:

1) kyphosis – curvature to the back, often with the formation of the hump;

2) lordosis – anterior curvature;

3) scoliosis – lateral curvatures, predominantly in the thoracic part, often combined with kyphosis.

Marked curvatures of the spine deforming the chest often lead to significant disturbances of the heart and lungs function.

Patients who suffered rickets in childhood often have varus (O) or valgus (X) deformities of the lower limbs. On tapping, this can be an important sign of bone tenderness (leukosis/leukemia, pernicious anemia). Local thickening, surface roughness and tenderness of the bone reveal pathology of periosteum (periostitis).

When examining the joints, attention should be paid to their configuration, swelling, tenderness on touching and movements, the condition of the skin covering them. Volume of active and passive movements is determined in the joints. Configuration changes (changes in forms) of the joints can be due to inflammation of periarticular soft tissue. There is a steady increase in volume and smoothing of the contour of the joints – a change in form (*defiguration)*. Inflammatory lesions along with joint defiguration is characterized by local hyperemia and hyperthermia of the covering skin, diffuse tenderness of the joint on palpation, impairment of both active and passive movements. *Deformation/deformity* is a persistent modified

change in the form of the joint caused by destruction of the cartilage and articular surfaces of the bones, development of ankilosis, bone outgrowth, impairment of the muscle and ligament apparatus. Many diseases manifest by the characteristic deformities of the joints (rheumatoid arthritis or "walrus fins").

Damage to the joints is detected by *tenderness* of the joints on touching if it is detected along the joint fissure. On walking, the nature of pain is detected as follows: stressful pain which is a sharp pain in the extreme points of flexion or extension indicating inflammation of the synovial membrane and pain of the same intensity which indicates mechanical changes in the joint. Rough crepitus (crunching) when moving points to lesion of the intraarticular cartilage or bone. Instability of the joints and their excessive mobility may be caused by weakness of ligaments, tendon rupture, articular bursa injury.

UNIT 2. EXAMINATION OF THE PATIENT WITH RESPIRATORY DISEASES

1. Complaints. The main complaints characteristic of respiratory diseases include: cough, hemoptysis, shortness of breath (dyspnea), pain in the chest. The general complaints in pathology of the respiratory system include: fever, chills, increased sweating, weakness, decreased work performance, headaches, sleep disorders, decreased appetite, weight loss.

1.1. Cough (tussis) is a complex reflectory act aimed at protection and self-purification of the airway from foreign bodies, irritating substances and sputum. Cough occurs when the cough center is irritated (in brain tumors) as well as in case of irritation of receptors of the vagal and upper laryngeal nerves that are located in the throat, trachea, bronchi, mediastinum and pleura, (esophagus, aorta), abdominal organs (stomach, intestines, gall bladder).

As for the time of cough onset, we distinguish: 1) morning cough (due to the movement of sputum accumulated per night in the cavities of the lungs and bronchi after getting up). It is common in chronic bronchitis, bronchiectasis, ling abscess. 2) during the day (typical of patients with pneumonia, acute bronchitis); 3) night cough (in tuberculosis, lymphogranulematosis when enlarged mediastinal lymph nodes irritate the trachea bifurcation area during the period of the aroused vagal tone).

As for the periodicity of cough, we distinguish: 1) continuous (occurs in inflammation of the larynx, bronchi, lung cancer of bronchial origin, tuberculosis, metastatic mediastinal lymph nodes); 2) periodic (in the form of single cough attacks in flu, pneumonia, chronic bronchitis).

As for the volume and timbre of cough, we distinguish: 1) loud 'barking' (in whooping cough, compression of the trachea by retrosternal goiter or tumor lesions of the larynx, hysteria); 2) quiet (in the first stage of lobular pneumonia, dry pleurisy, tuberculosis); 3) silent (in ulceration of the vocal cords).

As for the duration of cough, we distinguish: 1) acute (up to 3 months) which is observed in infectious diseases of the respiratory tract (pneumonia, acute bronchitis, pleurisy, whooping cough) and in mechanical obstruction of the bronchus by a foreign body, in inhalation of toxic substances; 2) chronic (more than 3 months) which is observed in chronic respiratory diseases (chronic bronchitis, bronchial asthma, interstitial lung

disease, tumors of the bronchi) and outside the lung lesions (tumors of the mediastinum, aneurysm of the aorta, mitral stenosis, use of some medicines, neuroses).

As for the nature of cough, we distinguish: 1) nonproductive (dry). It is observed in the initial stage of inflammation of the membrane lining of the bronchi and lungs if the large bronchi have extremely viscous sputum in small quantities, in the pleura, mediastinum, and other organs, which have the receptors of the vagus nerve, in the elderly and weakened/debilitated patients characterized by suppression of cough reflex and only a slight cough without sputum discharge; 2) productive (moist). It is accompanied by sputum discharge from the airway passages. It is characterized by sputum discharge after 2-3 coughs of normal force, but with the absence of obvious (objective) evidence of respiratory failure on coughing (shortness of breath and cyanosis); 3) low-productive. It is characterized by relatively prolonged bouts of painful cough with lots of painful excrutiating cough attacks and a little discharge of usually viscous mucopurulent sputum and obvious (objective) evidence of respiratory failure of obstructive type (shortness of breath, cyanosis, swelling of the neck veins).

In case of productive cough, it necessary to check: frequency of sputum production; the amount of simultaneously produced sputum; the amount of sputum within 24 hours; difficulty in sputum production; the body position when the sputum is easier produced; sputum colour; the presence of inclusions in the sputum (blood, dense particles etc.).

1.2. Hemoptysis is discharge of blood with sputum when coughing. The amount of blood in the sputum can be scarce in the form of hardly noticeable streaks and spot inclusions, but may be significant in the form of admixtures of scarlet, foamy blood or dark blood clots. Blood in the sputum is due to destruction of the lung tissue in bronchiectasis, tuberculosis, lung abscess, tumors, airways injuries; diapedesis of erythrocytes in pneumonia ("rusty" sputum), lung infarction, cardiovascular diseases.

1.3. Shortness of breath (dyspnoë) is the subjective feeling of lack of air or difficulty breathing, objectively accompanied by violation of the frequency and depth of breathing rhythm and intensifying the work of the respiratory muscles. Shortness of breath may be physiological (on increased exertion, in high altitudes) and pathological (in diseases of the respiratory organs, cardiovascular system).

The following types of wheezing are distinquished: 1) subjective when the patient has shortness of breath, chest tightness and a lack of air on inhalation, which is not accompanied by objective signs of changes in frequency, depth and rhythm of breathing; 2) objective, which is characterized by real changes in frequency, depth and rhythm of breathing, inhalation and exhalation phases ratio.

As for the preferred difficulty in this or that phase of breathing, we distinguish the following types: 1) inspiratory. It is characterized by difficulty to inhale. The most common causes are the presence of obstacles in the larynx, trachea, pathological processes, accompanied by the squeezing of lung and restriction on excursion of the lungs (hydrothorax, pneumothorax, pronounced deformity of the chest), pathological processes in the lungs accompanied by a decrease in elasticity of the lung tissue (pneumonia), 2) expiratory. It is characterized by difficulty to exhale, indicating the presence of bronchial obstruction in small airways (bronchoconstriction in asthma, mucous edema in acute bronchitis, the presence of viscous exudate in the bronchial lumen in chronic bronchitis) and 3) mixed. It is characterized by difficulty both to inhale and to exhale.

Choking (asthma) is a suddenly ensuing sense of a lack of air accompanied, as a rule, by intensity of breathing unusual for this patient and obvious (objective) signs of respiratory failure (cyanosis, swelling of the neck veins, the use of additional respiratory muscles, the patient's forced position).

1.4. Pain (dolor) in the chest may be of different origin. The appearance of pain is associated with stimulation of pain receptors, many of them are in the pleura, trachea and upper respiratory tract, and are practically absent in the parenchyma of the lung. Chest pain can occur in the development of pathological process:

- on the skin (erysipelas, zoster);
- in muscles (injury, inflammation);
- in intercostal nerves (neuralgia);
- in ribs and the periosteum (bone fractures, periostitis, tumour metastases);
- in pleura (pleuritis);
- in trachea (acute tracheitis);
- in the mediastinal organs (heart, aorta, esophagus);
- radiating pain in diseases of the gastrointestinal tract.

Localisation of pain, its nature, intensity, duration, radiation, connection with the act of breathing, cough, dynamics of the changing situation should be described. Pain in diseases of the respiratory system can be localized in different parts of the thorax and, as a rule, is usually of stabbing nature, intense, worsening on deep breathing, coughing, on patient's bending to a healthy side (due to increased friction of the inflamed pleural layers). Pain becomes less when lying on the affected side (due to decreased respiratory excursions of the thorax).

2. The significance of history. The doctor should pay particular attention to the following parts:

2.1. History of disease development. The doctor needs to find out: 1) How did the disease begin? Acute onset with a sudden rise in temperature, chills, profuse sweating is characteristic of acute pneumonia, lung abscess, acute tracheobronchitis; gradual (with a slow, gradual increase in temperature, sweating) is observed in chronic bronchitis, some forms of pulmonary tuberculosis. 2) What is the onset associated with? – Hypothermia, epidemiological contacts (acute respiratory viral infections, influenza, tuberculosis). 3) Peculiarities of the disease course, including the provided treatment and its effectiveness, complications.

***2.2. History of working and living conditions*.** Attention should be paid to working and living conditions having a bad influence on the respiratory organs: living or working in a raw, poorly heated room, working out-of-doors, systematic inhalation of organic and inorganic dusts (chlororganics, poultry production, flour production, coal dust, silica, asbestos, talc, aluminum etc.).

2.3. Past medical history of the lungs and pleura can cause the development of the present condition.

2.4. Family history. There is a certain genetic predisposition to lung diseases such as asthma, α1-antitrypsin deficiency (primary cause of lung emphysema), cystic fibrosis. You should also find out whether any of the family members has ever been affected by tuberculosis or malignant diseases.

2.5. Allergic history. In some cases, a respiratory disease (e.g. asthma) is closely associated with allergic reactions to foods, medicines, pollen, chemical and perfume products. If allergic reactions occur, identify the type: vasomotor rhinitis, urticaria, Quincke's swelling, bronchial spasm.

2.6. Bad habits: smoking and drinking/alcohol abuse. Smoking can be a cause of respiratory diseases or has a significant impact on its prognosis. It is heavy smokers who are likely to have chronic obstructive bronchitis most often (also called "smoker's bronchitis/cough"), emphysema of the lungs and they are more likely to have cancer of the lungs. It is important for a doctor to know the number of cigarettes smoked per day and the numbers of years. Pneumonia in alcoholics tends to run an adverse course due to impairment of the mechanisms of cellular and humoral immunity.

3. General examination of the patient. When examining patients with diseases of the respiratory organs, attention is paid to the position of the patient. Forced position is often observed: the position with support on the hands in attack of bronchial asthma, the position on the affected side in case of pneumonia, lung abscess, pleurisy etc. In patients with diseases of the lungs, there may be diffuse (central) cyanosis, with blue coloring of the skin and mucous membranes, which develops as a consequence of poor blood oxygenation in the lungs that leads to an increase in the content of reduced hemoglobin in tissues. Swelling of the neck veins is due to increased intrathoracic pressure, poor outflow of blood through the veins into the right atrium and, consequently, increased central venous pressure. This is observed in low-productive cough attacks or in bronchial asthma. A change in the terminal phalanges of fingers (clubbing) is observed in chronic purulent lung diseases (lung abscess, bronchiectasis), diffuse pneumosclerosis. A change in finger and toe nails ("watch glasses") is observed in chronic purulent lung diseases.

4. Examination of the thorax. When inspecting the chest, the physician detects its form, symmetry, the participation of both halves in the act of breathing, and also describes respiratory rate, depth and type of rhythm.

4.1. The form/shape of the chest. There is differentiation between normal and pathological forms/shapes of the thorax (see Table 1). The chest is normal in the proper build-up and corresponds to the constitutional type.

Table 1

Detecting the shape of the normal chest

Options	Normosthenic	Hyperstenic	Asthenic
Anterioposterior ratio and lateral sizes	0,65-0,75	> 0,75	< 0,65
Supra- and subclavian fossae	moderately marked	marked	marked
Angle of connection between the manubrium and body of the sternum (Louis' angle)	clearly marked	considerably marked	abscent
Epigastric angle	approaching 90°	>90 °	<90 °
Direction of the lateral chest divisions	moderately oblique	almost horizontal	vertical, the 10th rib is not attached to the costal arch
Intercostal intervals	equal to the width of the ribs	narrow	expanded
Adjoining of the scapulae/ shoulder blades to the chest	moderately adjoined and are at the same level	tightly adjoined	protruded like wings

Pathological form of thorax may result from congenital abnormalities of bones and various chronic diseases. There are the following pathological forms of the thorax:

- emphysematous (barrel-shaped) chest: anteriorposterior chest diameter comes close to the side (the thorax resembles a barrel, it is wide and short), the direction of the ribs is horizontal. The subclavian fossae are not marked and ajoin the chest closely. Epigastric angle is obtuse. This is observed in lung emphysema and in attack of bronchial asthma.

- paralytic thorax: anterioposterior diameter of the thorax is much less than the lateral one, clavicles/collarbones are sharply outlined and are not symmetrical. The subclavian spaces lag behind, the scapulas do not adjoin the thorax and are located at different levels. Epigastric angle is acute. Atrophy of the thorax muscles is marked. It is observed in patients with chronic diseases of the lungs and pleura, in tuberculosis, Marfan's syndrome, extremely exhausted /debilitated people.

- rachitic thorax (keeled) is like a chicken breast with dramatically increased anterioposterior size due to the protruding breastbone (the keel of the sternum/carina) and there is some thickening in places of transition of the costal cartilages in the bone (rachitic rosary).

- funnel-like chest has a funnel-shaped impression or a deepening in the lower third of the sternum and in the xiphoid process area. This deformation is considered a result of abnormalities of the sternum or long-acting compression (the "cobbler's chest compression").

- navicular thorax has an elongated impression in the middle part of the sternum. It is observed in syringomyelia.

4.2. Detecting symmetry of the right and left halves of the thorax and their participation in the act of breathing. Normally, both halves of the chest are almost the same size and equally participate in the act of breathing. Asymmetry of the chest can be associated with (see Table 2):

- increase in the volume of one half due to the accumulation of fluid or air in the pleural cavity;
- decrease in the volume of one half due to pleural adhesions/commissures, in shrinkage of a large part of the lung due to proliferation of the connective tissue (pneumosclerosis), in the case of lung atelectasis or its lobe due to obstruction or compression from the outside of the draining bronchus, after surgical removal of a part of the lung.

Asymmetry of the thorax is detected on the basis of the following criteria: the clavicles and scapulae are located at the different levels, the supra- and subclavian pits/fossae are not equally marked. A static examination of the chest must be complemented by a dynamic one, which is detected by the degree of participation (synchronicity) of the halves of the thorax in the act of breathing (the movement of angles of the scapulae/shoulder blades). The half lagging in the act of breathing is considered pathological. It should be noted that a lag/delay of one-half of the thorax in the act of breathing is also observed in intercostal neuralgia, fractures of ribs accompanied by marked pain syndrome, which leads to reflex muscle contraction of the affected half of the thorax and unilateral reduction of respiratory movements.

To study the mobility of chest, its circumference is measured on inhalation and exhalation. The difference between these rates reflects its excursion. On quiet breathing, chest excursion does not exceed 2-3 cm. Maxi-

mum chest excursion ranges from 7.0 to 8.5 cm (maximum inspiratory and maximum expiratory). Respiratory excursion is reduced in the presence of pleural adhesions/commissures (after past pleurisy, pneumonia), emphysema, obesity.

4.3. Characteristics of respiration. A healthy person breathes freely through the nose. Breathing through the mouth is observed during pathological conditions of the nasopharynx, i.e. rhinitis, curvature of the nasal septum, etc.

Respiratory rate is determined by monitoring the patient. Counting the number of breaths is made by invisibly observing the movement of the patient's chest or abdominal wall. First, the pulse is counted. Then, the respiratory rate is assessed for not less than one minute. The respiratory and pulse rate ratio is typically 1:4. At rest, an adult healthy human makes from 15 to 18 breaths per minute. Depth of respiration is determined by the volume of inhaled and exhaled air in a quiet state.

There are the following anomalies of respiratory rate and depth:

- *Bradypnea* is pathological slowing down of respiration. It is observed in inhibiton of respiratory center functions (pathological processes in the brain, brain edema, metabolic disorders (uremia, acidosis, morphine poisoning). Rare shallow breathing can occur in severe lung emphysema, a sudden narrowing of the vocal rim (rim of the glottis /rima glottidis) and trachea. The athlete's breathing is usually rare/infrequent but deep.
- *Tachypnea* is increasing number of breaths per minute. It occurs on physical exertion, emotional arousal, fever, anemia, hysteria, pains in the chest (soft breathing), considerable reduction of the respiratory surface of the lungs (atelectasis, pneumonia) and in the presence of obstacles to normal inspiration such as ascites, flatulence, fractured/broken ribs, diseases of the diaphragm. As a rule, tachypnea is accompanied by lower depth of breathing movements.
- *Hyperpnea* is deep frequent respiration (Kussmaul's respiration). It occurs in certain types of anemia, coma.

There are following types of breathing:

- chest – due to contraction of the intercostal muscles (more often in women);
- abdominal – muscle contraction of the diaphragm (more often in men);

- mixed – when the lower divisions of the thorax and upper abdomen are involved in breathing (it is observed in the elderly).

Changes in the type of breathing can be caused by pathological process. For example, women with dry pleurisy due to sharp limitations of movements of the intercostal muscles are present with abdominal (diaphragmatic/phrenic) type of breathing. In men, the transition to the thoracic (costal) type of breathing may occur in acute abdominal processes (perforating stomach or duodenal ulcer, acute cholecystitis).

The rhythm of breathing in a healthy person is regular, respiratory movements are uniform. They are of the same length and the same depth for each breathing movement – inhalation/inspiration and exhalation/expiration. Pause is almost unobtainable. As an exception, there may be slight irregular breathing in healthy people during sleep.

Impairment of respiratory rhythm is most often associated with decreased sensitivity of the respiratory center in severe pathological processes in the brain or toxic effects on the respiratory center. If impairments of respiratory rhythm are repeated in a definite order, such breathing is called periodic.

- Biot's respiration is characterized by rhythmic, but deep breathing movements that alternate at regular intervals in approximately equally long (from several seconds up to half a minute) respiratory pauses. It can be observed in patients with meningitis and agony with profound disorder of cerebral circulation.
- Cheyne - Stokes respiration is observed when after a long (from several seconds up to 1 minute) respiratory pause quiet shallow breathing appears first, which quickly grows in its depth and becomes noisy and reaches its maximum on the 5-7th inhalation movement, and then in the same sequence decreases and ends with the next regular short-term pause. It occurs in disorders of cerebral circulation, cerebral hypoxia, severe intoxications.
- Undulate Grocco's respiration is somewhat like that of Cheyne-Stokes breathing. The only difference is that instead of respiratory pause poor shallow breathing is noted with a subsequent increase in the depth of breathing movements and then with its decline. This kind can be apparently seen as a manifestation of an earlier stage of the same pathological processes that cause Cheyne - Stokes respiration.

Table 2

Interpretation of the results of the chest examination

Symmetry of breathing movements of the thorax on deep breathing	Symmetry of the right and left halves of the thorax on deep breathing	Changes of the intercostal spaces	Syndromes or diseases
There is no lag/delay of one half of the chest on breathing	The rib cage is symmetrical	Without change	Norm
		Expanded	Bronchial obstructive syndrome, emphysema
Lag/delay of one half in the act of breathing	The rib cage is symmetrical	Without change	Infiltrative packing syndrome Cavity syndrome
	Increase in the affected half	Smooth or bulging (Litten's phenomenon)	Hydrothorax Pneumothorax
	Decrease in the affected half	Reduced	Obturative atelectasis Fibrothorax

5. ***Palpation of the thorax.*** Palpation of the thorax is focused on pain, elasticity and voice vibration.

To accurately specify the localization of changes in the chest area, you must know the topography landmarks:

- linea mediana anterior (the anterior midline) is drawn from the top to the bottom through the middle of the sternum/breastbone;
- linea sternalis dextra et sinistra (right and left sternal lines) passes along the left and right margins of the sternum respectively;
- linea parasternalis dextra et sinistra (left and right line near the sternum) are on the halfway between linea medioclavicularis and linea sternalis;
- linea medioclavicularis dextra et sinistra (right and left median clavicular lines) are drawn vertically downward from the middle of the clavicle;
- linea axillaris anterior (anterior axillary line) is drawn on the anterior margin of the armpit on the left and right;

• linea axillaris media (median axillary line) is drawn vertically downwards from the top of the armpit;

• linea axillaris posterior (posterior axillary line) is drawn on the posterior margin of the armpit on the left and right;

• linea scapularis dextra et sinistra (right and left scapular lines) are drawn posteriorly through the lower corners of the scapulae vertically down;

• linea paravertebralis dextra et sinistra (right and left paravertebral lines) are drawn in the middle of the distance between the posterior median and scapular lines;

• linea mediana posterior (the posterior median line) passes through the spinous processes of the vertebrae.

A horizontal topographic landmark is the ribs but it is necessary to take into account that the 1st rib is hidden by the clavicle/collarbone, consequently, the first intercostal space is below the clavicle. Angles of the scapulae, the xiphoid process of the sternum, spinous processes of the vertebrae are also used as landmarks.

5.1. Determining chest pain. Palpation of painful points and zones is carried out both in the normal position (standing or sitting) and at maximum level of inspiration, expiration, bending the torso to the affected and to the healthy side, thereby noting the dynamics of pain. Pressure shouldn't be too strong and fast.

Tenderness can be caused by a lesion of:

• skin and subcutaneous tissue (herpes zoster);

• intercostal muscles (myositis – palpation is painful);

• intercostal nerves (three painful points are identified in the superficial location of the nerve: near the spine, on the lateral surface of the rib cage and near the sternum);

• bone tissue (rib tenderness on the limited area can be attributed to fracture or inflammation of the periosteum. Tenderness of the sternum and ribs is due to bone marrow diseases);

• pleura (tenderness is in the intercostal space in dry pleurisy, but not across the intercostal space).

5.2. Determining thorax elasticity. Elasticity (resistance) of the thorax is identified by its compression in the anterioposterior and lateral directions. Terms of elasticity and resistance characterize the opposite properties of the thorax. Elasticity means chest pliability/compliance to palpa-

tion. Resistance characterizes its resistance to compression. In other words, a decrease in chest elasticity corresponds to its high resistance (or rigidity).

5.3. Determining vocal fremitus/vocal vibration. The phenomenon of vocal vibration (fremitus vocalis) is vibration of the patient's chest when uttering single words containing the sound "R". Low-frequency sound vibrations from the vocal cords pass on through the bronchi to the surface of the thorax. The degree of vibration on the left and right is assessed, which depends on the properties of conductive tissues, i.e. on density of the bronchial tree and ability to pass through it. Normally, vocal vibration is better perceived above the upper divisions of the thorax, it is weaker above its lower parts (see Table 3).

Table 3

Reasons of vocal vibration

Voice vibration	Syndromes
Without change	Norm Spasm of the bronchi
Diminished on the side of a lesion	Hydrothorax, exudative pleurisy Obturative atelectasis Pneumothorax Fibrotorax
Amplified on the side of a lesion	Consolidation syndrome Cavity communicating with the bronchus Compressive atelectasis Pneumosclerosis
Diminished symmetrically	Emphysema Marked obesity Massive musculature

6. Percussion of the lungs. Percussion of the lungs is tapping the chest making the underlying organs to vibrate, their physical characteristics (duration of sound vibrations, their frequency, amplitude and timbre) depending on organ density, elasticity, its structures and content of the air in it.

There are the following kinds of percussion:

1. Strength of percussion blow:

1.1. Loud (depth of penetration percussion blow is 6-7 cm)

1.2. Medium strength (depth of penetration percussion blow is 4-5 cm)

1.3. Quiet (depth of penetration percussion blow is 2-3 cm)

1.4. The quietest or threshold (penetration depth is 1-2 cm)

2. Objectives of percussion:

2.1. Topographic percussion is used to detect the lung borders

2.2. Comparative percussion is used to identify the nature of the pathological changes in the lungs and pleural cavity.

The nature of percussion sound produced by percussion of the lung depends on the density of the underlying tissues, on the ratio between air and dense elements. There are the following kinds of percussion sound:

• ***clear pulmonary*** is a loud, prolonged sound of relatively low frequency detected by percussion of the subclavian, axillary, subscapular areas in a healthy person. In case of clear sound, timbre is rich due to vibrations of elastic structures of the alveoli.

• ***dull*** is a quiet, short sound of high frequency detected by percussion of the lung when there is a predominance of dense elements in the percussion area. The stronger dullness (shortening) of the percussion sound above the lesion focus is, the less its airiness is. The model of a dull sound is a sound detected by percussion of the thigh muscles (femoral sound).

• ***tympanic*** is a loud, prolonged sound of low frequency that possesses a sonorous music tinge detected by percussion of the lung when there is a predominance of air elements in the percussion area. Varieties of tympanic percussion sound are a metal sound and a sound of a cracked pot. Model of tympanic sound is a sound detected by abdominal percussion and percussion of Traube's space.

• ***wooden/bandbox*** is a loud sound of low frequency that appears above the lungs on destruction of the alveolar walls and reduction of lung tissue elasticity (emphysema). It is characterized by a sharp decrease in sound timbre. Model wooden/bandbox sound is a sound detected by percussion of a pillow.

6.1. Topographic percussion of the lung. Objective of the study is to detect the height of the standing of the lung apex (the height level), frontally and at the back, Krönig's field, topography of the lower lung borders on the right and left as well as mobility of the lower border of the lungs (see Table 4).

Rules of topographic percussion:

• Percussion is done precisely according to the topographic lines.

• A finger plessimeter is parallel to the border being detected.

- Percussion blows of moderate strength are made.
- Percussion is carried out from the organ producing a loud sound to the organ producing a dull sound, that is, from a clear sound to a dull one.
- The border of the organ is detected according to the side of the finger plessimeter, facing the side of the organ producing a clear pulmonary sound. **NB!** ***The only exception is detecting mobility of the lower lung border on maximum exhalation.***

Table 4

Indicators of the lung borders location

Indicator	Right lung	Left lung
Lung apex anteriorly	At the level of 3-4 cm above the clavicle	
Lung apex posteriorly	The level of the 7th spinous process of the cervical vertebra	
Krönig's field width	5-6 cm	5-6 cm
The lower lung border:		
l. parasternalis	the 5th intercostal space	is not detected
l. medioclavicularis	6th rib	is not detected
l. axillaris anterior	7th rib	7th rib
l. axillaris media	8th rib	8th rib
l. axillaris posterior	9th rib	9th rib
l. scapularis	10th rib	10th rib
l. paravertebralis	The level of the 11th spinous process of the thoracic vertebra	

Determining mobility of the lung borders. The distance (in cm) between the level of the lower border of the lung which is detected on lag/delay of breathing at the maximum level of deep inhalation and after maximum exhalation level is called excursion of the lung border. Mobility of the lung border can be identified by any of the topographic lines, but it can usually be detected only along the secondary axillary line where it is the greatest. Normally, excursion of the lung border along the linea media axillaris on breathing in is 6-8 cm (on the right and on the left).

Table 5

Interpreting the results of topographic percussion of the lung

Changing the borders of the lung	Causes
The upper borders are raised, expansion of Krönig's field	1. emphysema 2. attack of bronchial asthma
The upper borders are lowered, reduction of the Krönig's field	1. infiltrative processes 2. pneumosclerosis
Bilateral downward displacement of the lung borders	1. emphysema 2. attack of bronchial asthma 3. the low standing of the diaphragm (due to weakened abdominal muscles, ptosis)
Unilateral downward displacement of the lung borders	compensatory emphysema of one lung
Bilateral upward displacement of the the lung borders	maximum height of the aperture as a result of development of ascites, flatulence, pneumoperitoneum
Unilateral upward displacement of the lung borders	1. consolidated lung borders 2. hydrothorax 3. pneumothorax 4. liver disease (tumor, echinococcus) 5. considerable enlargement of the spleen
Reducing the mobility of the lower lung border	1. emphysema 2. hydrothorax 3. pneumothorax 4. massive pleural adhesions/commissures

6.2. Comparative lung percussion. Objective of the study is to identify the nature of pathological changes in the lungs and the pleural cavity and for the diagnosis of bronchial and pulmonary syndromes (see Table 5).

• Rules of comparative percussion:

• Percussion sounds taken on symmetrical sections of thorax are compared;

• As a rule, percussion blows of medium strength are made. Volume of percussion sound may vary depending on the thickness of the subcutaneous tissue, the degree of development of the musculature, depth of location of pathological process and other causes;

• Percussion is performed along the intercostal spaces.

At the end of comparative percussion a conclusion is made about homogeneity of percussion sound above the symmetrical regions of the lungs and its physical characteristics (clear pulmonary, dull, tympanic, dull-tympanic, wooden/bandbox). The results of comparative percussion are given in Table 6.

Table 6

The results of comparative percussion and voice vibration

Percussion sound	Voice vibration	Syndromes and diseases
Clear pulmonary	Without change	Normal Constriction of the bronchi
Dull (short)	Diminution	Hydrothorax Obturative atelectasis Fibrothorax
	Amplification	Consolidation syndrome
Tympanic	Diminution	Pneumothorax
	Amplification	Cavity communicating with the bronchus
Dull-tympanic	Amplification	Compressive atelectasis Initial stages of lung inflammation
Wooden/bandbox	Diminution	Emphysema

7. Auscultation of the lungs. Lung auscultation is listening to acoustic phenomena that occur in the chest in a norm or pathology. It enables to detect sound phenomena that occur in the lungs on breathing, evaluate their character, strength, localization, and relevance to the phase of respiration. On auscultation, it is necessary to assess basic breathing sounds, additional/side breathing sounds and bronchophony.

Basic rules of auscultation:

- A stethoscope is placed alternately on symmetric sections of the chest to the right and left, being tightly pressed to the chest wall.
- 2 – 3 respiratory cycles are listened to on quiet breathing through the nose at each point of auscultation.
- Sounds of low frequency are better heard using a stethoscope without a bell (a membrane), high frequency sounds are better heard with the help of a stethoscope with a bell (a membrane);

• If additional/side breathing sound is present, it is possible to use special techniques for clarifying the nature of sounds: the patient is asked to breathe deeply through the mouth, his/her breathing is listened to on forced inhalation and exhalation, after a cough, lying on the side or back, with a stethoscope being more tightly pressed etc.;

• Detected changes on breathing and additional/side breathing sounds are described, using adopted topographic landmarks.

7.1. Basic respiratory sounds. Normally, two types of respiration are heard above the lungs: vesicular one and physiological bronchial.

Vesicular (alveolar) breathing is heard over nearly the entire surface of the lung tissue and is perceived as a continuous, smooth, soft, blowing sound reminding the sound [f]. Vesicular breathing is due to fluctuation of the expanding alveolar walls. Their sound is well heard on the surface of the thorax as a result of resulting respiratory sounds coming from a large number of alveoli. It is heard on inhalation/breathing in and in the first third part of the exhalation.

Under various physiological and pathological conditions vesicular breathing can vary both in terms of quality and quantity. Quantitative change can be observed either in diminution/weakening or intensification of breathing. Diminution of vesicular breathing may occur:

• In physiological conditions, e.g. in the thick chest wall (well-developed pectoral muscles, obesity);

• On reducing elasticity of the alveoli (pulmonary emphysema, interstitial pulmonary edema, the initial stages of pneumonia);

• On the narrowing of the airways (obturative atelectasis);

• If there are "obstacles" in transmitting the sounds on the surface of the thorax (hydrothorax, pneumothorax);

• On limitation of the thorax mobility (fibrothorax, chest trauma, impairment of the intercostal muscles etc.);

• On superficial breathing in weakened/debilitated (elderly) patients.

Increased vesicular respiration is observed in case of a thin chest (child's breath/breathing in children); in hyperventilation (physical exertion, hyperthermia, hyperthyroidism).

Harsh/coarse breathing is different from normal vesicular in that it does not have smooth, soft breezing character and seems to be uneven, rough. As a result of the narrowing of the bronchi (the swelling of mucous, viscous

exudate in the bronchial lumen, bronchospasm), normal sound on vesicular breathing associated with vibrations of the alveolar walls is mixed with the sound caused by turbulent motion of the air flow in the bronchi, the walls of which are not smooth. Breathing sound becomes harder/tougher in timbre than normal vesicular breathing. It is heard not only during inhalation but also during exhalation.

The cause of interrupted/saccadic breathing is the irregular narrowing of the bronchioles. As a result, the air flow which encounters an obstacle like the bronchial narrowing of different degree first enters one section of the lungs. Then, it enters the other section and so on. Inhalation is intermittent, usually in the 2-3 phases. The most common cause of the uneven narrowing of the bronchioles and interrupted breathing is a lesion of respiratory tuberculous etiology.

Physiological bronchial (laryngotracheal) breathing is rough and loud breathing sound resembling the sound [h] which is heard over the larynx and trachea (as well as in places of their projection on the posterior surface of the chest) at the end of inhalation and during exhalation. Bronchial breathing is due to turbulent flow. It emerges during the passage of air through the vocal rim (rim of the glottis /rima glottidis), which is well distributed through the trachea and bronchi, but then normally becomes extinct by the alveolar lung tissue and is not heard on its surface. Bronchial breathing heard in some part of the chest outside the projection of the larynx and the trachea is considered pathological.

Pathological breathing is heard under the conditions of proper laryngeal breathing on the surface of the chest, i.e. preserved patency/passage ability of the bronchi, the presence of air in the lung cavity connected to the bronchus, lobar lung infiltration/consolidation (inflammation, compressive atelectasis).

A special kind of bronchial respiration is **amphoric breathing** (an amphora is a vessel with a long narrow neck). It is characterized by the presence of metallic tone. Amphoric breathing develops if there is a cavity in the lung that is superficial, not less than 5 cm in diameter, with smooth thin walls, surrounded by infiltrative.

Bronchovesicular breathing. In focal inflammatory lung infiltration, weak bronchial breathing is heard in the field of projection of a small area of the lung tissue on the surface of the lung. The alveoli surrounding this fo-

cus produce sounds of vesicular breathing. Mixing these two sounds leads to the so-called bronchovesicular breathing. Inhalation phase has vesicular features and expiratory phase has the features of bronchial breathing.

7.2. Additional/side breathing sounds.

Râles/ronchi/wheezes represent the most common additional/side breathing sounds that occur in trachea and bronchi pathologies due to movement or fluctuations of pathological secretion in their lumen (see Table 7). The nature of râles/rhonchi depends on several factors: viscosity of secretions, its quantity, localization in bronchi, smoothness of the surface of the bronchi, bronchial obstruction, conductive properties of the lung tissue etc. As for the mechanism of occurrence, dry and moist râles are distinguished.

Depending on the diameter of the affected bronchus, dry wheezing is divided into:

- Low are noted in involvement of the large bronchi, trachea;
- High-pitched are noted in involvement of the small and medium bronchi (in bronchial asthma). Dry low râles are heard in the trachea and bronchi in the presence of viscous sputum in their lumen and high dry râles are present on the narrowing of the lumen of the bronchus due to edema of the mucosa. They resemble long whistling or buzzing.

Dry râles are heard on both inhaling and exhaling, dry high-pitched râles/wheezes are better heard in a horizontal position of a patient and on forced exhalation. The peculiarity of dry low râles is their volatility. They may diminish or disappear after coughing.

Moist râles are heard in the presence of liquid secretion (sputum, blood etc.) in the trachea, bronchi or cavities connected with the bronchial tubes. They resemble the sounds heard on bursting bubbles when air is drawn through the tube in water (cracles). The character of moist râle depends on the diameter of those parts of the airways with moist secretion present in them:

- Large bubbling moist râles are formed in the trachea and large bronchi, in large cavities connected with the bronchial tubes (abscess);
- Medium bubbling moist râles are formed in the medium bronchi (segmental) and in bronchiectasis;
- Fine bubbling moist râles are formed in the small bronchi.

Moist râles can change after coughing. They are heard both on inhalation and exhalation. All moist râles are divided into sonorous/wheezing

and not sonorous. Sound intensity of moist râles is noted when there is inflammatory lung infiltration around the bronchus or cavity (pneumonia, abscess).

Crepitation (crepitatio) is noted in the alveoli with viscous secretions (exudate or blood) in them. Such conditions are common in patients with initial stages of croupous pneumonia, in compressive atelectasis and infarction of the lung. On exhalation, the alveoli and their walls adhere. On inhalation, the adhered walls expand with the characteristic short crackling sound. In contrast to moist fine bubbling râles, crepitation is heard only at inhalation maximum level and does not change after coughing.

Friction rub occurs when rough surfaces of the pleural layers that are changed due to inflammation rub each other on breathing. It resembles crunching snow, creaking leather, rustling paper. Friction rub usually indicates the presence of acute inflammation of the pleural layers in the absence of pleural cavity exudates.

Friction rub is heard on inhaling and exhaling. It does not change after coughing and increases with the pressure made by a stethoscope on the chest and torso bent toward a lesion. Friction rub can be heard on simulating inspiration/inhalation when the vocal rim (rim of the glottis /rima glottidis) is closed ("imaginary" breathing), which is used to differentiate this side breathing sound from râles and crepitation.

7.3. Bronchophony. Bronchophony is a research method which is used to hear whispering speech through a stethoscope applied to the chest. The patient is asked to say in a whisper the words containing affricative/sibilant/hissing sounds ([tʃ]/[ʃ], for example, in Russian, "**ч**а**ш**ка **ч**ая", "**ш**естьдесят **ш**есть". This method is based on the same physical phenomenon as the study of voice vibration is based on. Thus, bronchophony is an acoustic phenomenon that gives you an idea of spreading sound from the vocal cords of the larynx through the air bronchial tract to the surface of the thorax. Normally, the spoken words sound indistictly and like one word, which means normal (negative) bronchophony as vibrations are absorbed by the lung tissue (alveoli).

Under conditions of better conduction of vibrations from the larynx to the surface of the thorax (inflammatory infiltration of the lung tissue, the cavity in the lung connected with the brochus, compressive atelectasis), sounds become recognizable and spoken words are distinct. In these cases,

we can speak about bronchophony amplification in the relevant part of the thorax. When uttering the words in your headphones, you do not hear any sounds, it points to diminishing/weakening bronchophony. Unilateral decrease in voice conduction onto the surface of the chest is observed in air exudative pleurisy, pneumothorax, fibrothorax and obturative atelectasis. Bilateral diminution/weakening of bronchophony on each side is observed in lung emphysema.

Table 7

Characteristic additional/side breathing sounds

Additional / side breathing sounds	Acoustic characteristics	Localization	Conditions of occurrence	Diseases and syndromes
Dry low râles/ wheeze	Low, droning, buzzing, long râles are heard on inhalation and exhalation, change after coughing	Trachea, large and medium bronchi	Filaments, sticky adhesions of viscous sputum	Tracheitis, bronchitis
Pitched dry râles/ wheeze	High-pitched, whistling, long râles are heard on inhalation and exhalation, change after coughing, amplified with forced exhalation	Small bronchi	Viscous sputum, bronchial constriction due to edema of the mucosa and broncho-spasm	Bronchial asthma, bronhiolitis
Moist large bubbling râles/ wheezes (not sonorous)	Various, slightly dull sounds, resembling large bursting bubbles of air, heard on inhalation and exhalation, change after coughing	Trachea, large bronchi	Liquid secretion in the lumen (liquid mucus, transudate, blood)	Pulmonary edema, pulmonary hemorrhage
Moist large bubbling râles/ wheezes (sonorous)	Various, very loud sounds resembling large bursting bubbles of air, heard on inhalation and exhalation, change after coughing	Large cavity in the lung connected with the bronchus	Cavity containing secretion (pus, blood) and air	Lung abscess, tuberculosis

Moist medium bubbling râles/wheeze	Sounds resembling air bubbles bursting, heard on inhalation and exhalation, change after coughing	Medium bronchi, bronchiectasis	Liquid secretions (blood, pus) in the bronchi and bronchiectasis	Bronchiectasis, bronchitis (rarely), pulmonary hemorrhage
Moist fine bubbling râles/ wheezes (not sonorous)	Various, slightly dull sounds that resemble bursting small air bubbles, heard on inhalation and exhalation, slightly modified after coughing	Small bronchi, bronchioles	Liquid mucus in bronchus	Congestion in pulmonary circulation, bronchitis (rarely)
Moist fine bubbling râles/ wheezes (sonorous)	A variety of very loud sounds, resembling small bursting bubbles of air, heard on inhalation and exhalation, slightly modified after coughing	Small bronchi, bronchioles	Liquid mucus in lumen of the bronchus surrounded by infiltrated lung tissue	Pneumonias
Crepitation	Monotonous sounds resembling friction of dry hair, heard only at the height of deep inspiration, does not change after coughing	Alveoli	Transudate or exudate, blood on the walls of alveoli	The initial stages of lung inflammation, compression atelectasis, lung infarction
Friction rub	A variety of sounds, resembling crunching snow, creaking leather, rustling paper (rub), heard on inhalation and exhalation. It does not change after a cough and increases with the pressure made by a stethoscope on the chest. It is clearly heard on "imaginary" breathing.	Visceral layers of the pleura	Inflammation of the visceral layer of the pleura, fibrin deposits on them	Dry pleurisy, pleuropneumonia

8. Major clinical syndromes in pulmonology

8.1. Syndrome of infiltrative consolidation is a pathological condition caused by penetration of cellular elements and various chemicals into the lung tissue and their accumulation there. We distinguish infiltration by leukocytes, lymphocytes, eosinophils, erythrocytes. There also can be tumoral infiltration. **The essence of the syndrome** is reduction of lung tissue airiness in the area with this or that extent of spreading. Subjective manifestations of the syndrome vary depending on the disease that caused infiltration, the degree of inflammatory activity, area and location of lesion.

The most common complaints of patients with pulmonary infiltration are shortness of breath, cough and hemoptysis. Nature of cough depends on stage of development and etiology of infiltration: dry cough occurs usually in the beginning of the development of pulmonary infiltration whereas prolonged cough may indicate tuberculous infiltrations. In inflammatory infiltrations, cough is moist, sputum is mucopurulent, sometimes bloody (pneumonia). When there is destruction of infiltration, hemoptysis (tuberculosis, cancer) may occur. Shortness of breath occurs in major or confluent infiltrations (croupous pneumonia). It characterizes the severity of restrictive ventilation impairments (reduction of respiratory surface) and the degree of respiratory failure. Pain in the chest is noted only in cases when the parietal pleura becomes involved in the pathological process. Pain is deeply localized, worsening on breathing and coughing. Pain is precisely localized ("pleural pain").

On inspection, there is a lag/delay in breathing in the affected half of the thorax. Tachypnoe is present. Physical examination above the focus of a lesion reveals the following:

- palpation reveals amplification of voice vibration;
- percussion reveals dullness of lung sound;
- auscultation reveals bronchial breathing, increased bronchophony.

Bubbling moist râles that are better heard on inspiration as well as a change in nature after coughing are characteristic.

8.2. Syndrome of bronchial obstruction is an abnormal condition of the organism, resulting from impairment of bronchial conductivity and, consequently, difficult and uneven ventilation (limited exhalation rate).

Impairment of bronchial tubes conduction can be caused by spasm of the smooth muscles, swelling of the mucosa in inflammatory phenomena in the lungs, the narrowing of the bronchial lumen by various fluids (sputum, pus, blood, vomitus), tumor, a foreign body squeezing bronchi from outside. Etiologically, we distinguish primary bronchial obstruction syndrome (bronchial asthma) and secondary one (all the other diseases like bronchitis, swelling, pneumoconiosis). As for the course, we distinguish attack-like/paroxysmal and chronic.

Complaints include shortness of breath or attacks of gasping/wheezing of expiratory type, paroxysmal cough. Symptoms of hypercapnia include insomnia and headache. Dry cough is typical of stenosis of the trachea and large bronchi, bronchospasm. Productive cough is observed in diseases accompanied by bronchial mucus hypersecretion, exudates formation. The harder the discharge/excretion of mucus is, the deeper the source is.

On inspection, "oral" respiratory sounds, forced position of leaning forward, with an emphasis on hand are usually revealed. The rib cage can be expanded. Its excursion is reduced. The act of breathing involves the supporting muscles. Bradypnoe is present.

Palpation reveals voice vibration which is not changed. Diminished voice vibration occurs in emphysema.

Percussion reveals pulmonary sound which is not changed, wooden/bandbox sound appears only in emphysema.

Auscultation reveals the most important sign of the bronchial obstruction that is harsh breathing and dry wheezes increasing on forced exhalation.

8.3. Syndrome of increased airiness (lung emphysema) is a pathological condition characterized by the enlargement of the air spaces of the lungs located more distally to the terminal bronchioles and resulting from reduction of the elastic properties of the lung tissue. Circulatory disorders in pulmonary capillary network and destruction of the alveolar septa play a leading role in reducing the elastic properties of the lung tissue. This is supported by:

inhalation of aggressive substances, including tobacco smoke; impairment of the production and activity of surfactants; disturbance in the metabolism of mucopolysaccharides, which is a genetic defect of collagen and elastin; diseases of the respiratory tract accompanied by obstruction of

the small bronchi and increased residual lung volume (primarily chronic bronchitis and bronchial asthma).

Thus, the walls of the bronchioles which have lost their elasticity in lung emphysema serve as a valve, which closes prematurely on exhalation and the alveoli remain constantly inflated due to increased residual air. The dilated alveoli squeeze the lung capillaries, causing trophic changes in the lung tissue, which leads to the destruction of the alveolar septa (to the formation of large air cavities, or bullae). Pathogenetically, we distinguish primary and secondary emphysema.

Complaints are shortness of breath of mixed type and dry cough.

Inspection reveals superficial difficulty breathing (especially on exhalation), with the participation of the supporting muscles, swelling of the neck veins, chest expansion (barrel-shaped), reduced excursion of the chest.

Palpation reveals rigid thorax. Vocal fremitus/vibration is diminished.

Percussion reveals a wooden/bandbox sound, extension of Krönig's field and the height of the lung's apex. The lower lung border is lowered. Decrease in motion of excursion of the lower lung border is marked. Auscultation reveals diminished/weakened vesicular ("wadding"/ "cotton") breathing.

8.4. Atelectasis syndrome is a pathological lung (or a part of it) condition in which the alveoli do not contain air. Atelectasis may be congenital (a lack of surfactant) or acquired. Depending on pathogenic mechanism, acquired atelectasis is divided into obturative, compressive and mixed (parapneumonic). In atelectasis, the respiratory surface area decreases, hypoventilation develops, hemodynamics of the pulmonary circulation is impaired, arterial hypoxemia develops.

8.4.1 Obturation atelectasis is caused by a closure of the bronchus lumen (foreign body aspiration, obstruction with mucus, tumor, lymph node).

Complaints are paroxysmal nocturnal dyspnea, persistent dry cough.

Inspection reveals retraction of the affected part and its lag/delay in the act of breathing, tachypnoe, diffuse cyanosis.

Palpation reveals that vocal fremitus over an area of atelectasis is absent or severely diminished. Percussion over an area of atelectasis reveals a dull percussion sound. The lower lung border on the side of the lesion is higher than on the healthy one. Mobility of the pulmonary border is lim-

ited. Decreased lung volume leads to escalation of negative pressure in the pleural cavity on the side of the lesion and displacement of the mediastinal organs (heart) in the direction of atelectasis. Auscultation above the area of atelectasis reveals dramatic diminution/weakening of vesicular breathing and bronchophony till complete disappearance.

8.4.2 Compression atelectasis (lung collapse) develops due to external compression of the lung tissue in massive pathological processes in the thoracic cavity (aortic arch aneurysm, tumor of the mediastinum) or in accumulation of a large amount of fluid or air in the pleural cavity.

Complaints are shortness of breath of mixed type, dry cough.

Inspection reveals a lag/delay of the affected half in the act of breathing, flatness or bulging of the intercostal spaces on the side of lesion can be observed (exudative pleurisy, pneumothorax). Palpation reveals forced vocal fremitus/vibration over the area of atelectasis.

Percussion reveals a dull percussion sound, the mediastinal organs are displaced to the side of the healthy lung because of an increase in intrathoracic pressure.

Auscultation reveals bronchial breathing, increased bronchophony.

8.5. Cavity syndrome. The air cavity is a localized pathologic process communicating with the bronchus. It results from destructive, degenerative-dystrophic or cystic changes in the lung tissue. Its formation is most commonly observed in infectious lung destruction (abscess, gangrene), tuberculosis of the lungs (caverna), tumour/consolidation disintegration, bronchiectasis.

Complaints are productive cough, more rarely hemoptysis and pain on the side of the lesion worsening with cough. Discharge of a large amount of purulent sputum is common in certain (draining) position of the body.

Inspection reveals a forced position of the patient on the affected side. If there is a large cavity, a lag/delay of the affected part in the act of breathing is noted.

Palpation reveals amplification of vocal fremitus over the cavity.

Percussion. The nature of percussion sound depends on the ratio of fluid and air, its size and depth of location. If the cavity is large and mostly filled with air, a tympanic sound is detected. If the cavity contains both air and fluid (pus) simultaneously, percussion reveals a dull and tympanic sound.

Auscultation reveals bronchial or amphoric breathing. Sound moist mediocre or large bubbling râles are heard.

8.6. Pleural syndrome is a set of symptoms typical of lesions of the pleural layers (inflammation, swelling/tumour/consolidation). Fluid or gas can accumulate in the pleural cavity.

8.6.1 Accumulation of air in the pleural cavity (pneumothorax) is a pathological condition characterized by accumulation of air between the parietal and visceral pleura. Pneumothorax can be unilateral and bilateral, partial and total, open and closed. In open pneumothorax, ambient air being freely inhaled enters the pleural space and comes out of it on exhaling. Valve pneumothorax means difficult evacuation of air on exhalation and its gradual accumulation and increased pressure in the pleural cavity. In closed pneumothorax, a hole through which air enters is closed. The causes of pneumothorax are chest trauma, spontaneous pneumothorax (destruction of the lung tissue in case of abscess, gangrene, rupture of a cavern, rupture of the esophagus), artificial pneumothorax (in the treatment of tuberculosis). Intrapleural pressure increases on accumulation of air in the pleural cavity and lung collapse develops. Respiratory failure develops. As a result of lung hypertenstion, acute right-ventricular failure occurs.

Complaints are sudden chest pain, shortness of breath, dry cough, palpitations. Inspection reveals flatness of intercostal spaces, increase in half of the thorax and its lag/delay in the act of breathing, superficial breathing, fast breathing, diffuse cyanosis.

Palpation reveals sharply diminished or even absent vocal fremitus, the thorax is resistant.

Percussion reveals a loud tympanc percussion sound.

Auscultation reveals sharp weakening/diminution or absence of vesicular breathing over an area of pneumothorax, bronchophony is negative.

8.6.2 Complex of symptoms of fluid accumulation in the pleural cavity is a clinical and radiological laboratory symptom either caused by the fluid that accumulates in the pleural cavity due to the lesion of the pleural tissue lining it or due to general lesions of water and electrolytes exchange in the body. Liquid can be exudate (in inflammation), transudate (effusion of non-inflammatory origin), pus (pyothorax), blood (hemothorax), lymph (hylothorax).

Up to 5-6 liters of liquid can accumulate in the pleural cavity. Less than 100 ml are not clinically detectable, more than 100 ml are detected radiographically, more than 500 ml are detected on physical examination. First, liquid accumulates above the diaphragm. Then, it fills the costal and diaphragmatic /costal phrenic sinus (up to 1.500 ml). Respiratory failure develops due to limitations of lung mobility, compressive atelectasis.

Complaints are shortness of breath, feeling of heaviness in the chest, a feeling of transfusing/flowing liquid movement on the affected side. There may be chest pain and dry cough. The severity of dyspnea depends on the amount of liquid, accumulation rate, reduction extent of respiratory surface of the lung. Inspection reveals a forced position (on the affected side), expanded and flattened intercostal spaces, increased volume of the affected half of the chest and its lag/delay in the act of breathing.

Palpation reveals marked diminution or absence of voice vibration over the area of fluid accumulation, the chest is resistant.

Percussion reveals marked or absolute dullness over the area of fluid accumulation, a dull and tympanic sound is heard above. On topographic percussion, mobility of the lung border is limited.

Auscultation reveals the absence of breathing sounds over the affected area, bronchophony is not performed.

8.7. Pneumonia is an acute infectious inflammatory process involving all structural elements of the lung tissue and a necessary/comlulsory lesion of the lung alveoli. Etiologically, we distinguish bacterial pneumonia (pneumococcal, streptococcal, staphylococcal etc.), viral, fungal etc. According to the clinical and morphological features, pneumonias are divided into lobar (pleuropneumonia), focal (segmental bronchopneumonia), interstitial. According to international consensus (1998), additional characteristic were added to pneumonia classification: community-acquired pneumonia (acquired outside the medical establishment); nosocomial (hospital) pneumonia; pneumonia in patients with immunodeficiency (AIDS, long-term use of immunosuppressants); aspiration pneumonia. As for manifestation of intoxication and respiratory failure and the spread of lesions, we distinguish three degrees of severity of acute pneumonia: mild, moderate and severe.

8.7.1. Lobar pneumonia (croupous pneumonia/pleuropneumonia) is of a hyperergic type of inflammatory reaction, which is marked by a specific

manifestations of clinical symptoms and stage levels of a course (pathomorphological and clinical).

Stage 1 (onset):

• Complaints are acute onset, a sharp increase in body temperature (pyretic temperature), shivering, chills, severe intoxication with a headache accompanied by chest pain on breathing. Cough is at first dry.

• Inspection reveals face hyperemia (often on the affected side), herpetic eruptions, accelerated shallow breathing, with involvement of the additional muscles, cyanosis, a lag/delay of the chest on the affected side.

• Palpation reveals slight amplification of vocal fremitus above the affected area.

• Percussion reveals a dull tympanic sound.

• Auscultation reveals diminished vesicular breathing. The initial crepitation is heard (crepitatio indux).

Stage 2 (the climax of the disease, consolidation):

• Complaints are permanent febrile fever, coughing up "rusty" sputum, later mucopurulent, chest pain on breathing, inspiratory dyspnea.

• Palpation reveals a sharp amplification in vocal fremitus over the affected area.

• Percussion reveals a dull percussion sound.

• Auscultation reveals bronchial breathing, moist bubbling râles, friction rub, positive bronchophony.

Stage 3 (resolution):

• Complaints are critical reduction of fever with profuse sweating.

• Palpation reveals normal vocal fremitus.

• Percussion reveals a dull and tympanic percussion sound, later it becomes a clear pulmonary/pulmonic one.

• Auscultation reveals bronchial breathing changes to harsh one and later vesicular breathing. Crepitation appears again (crepitatio redux). Fine and large moist râles are heard.

Additional data

• Sputum in lobar pneumonia contains a large number of leukocytes, macrophages, erythrocytes ("rusty" sputum) and alveolar epithelium. Bacteriological sputum analysis makes it possible to reveal a causative agent of pneumonia and to determine its sensitivity to antibiotics.

• Laboratory data: leukocytosis, toxic neutrophil granulosis, relative

lymphopenia, acceleration of erythrocyte sedimentation rate (ESR). Analysis of urine reveals mild proteinuria.

• Spirography reveals a reduction of vital capacity of the lungs (VCL) in case of a large focus. Restrictive respiratory insufficiency type develops.

• Complications of lobar pneumonia are: exudative pleurisy; abscess and lung gangrene (in case of inflammation focus destruction, especially in staph and strep pneumonia); postpneumonic pneumosclerosis (in disturbances of fibrin lysis and exudate resorption in the focus of inflammation); acute respiratory insufficiency (in generalized lesion of both lungs); infectious toxic shock.

8.7.2. **Focal pneumonia** is characterized by the development of infectious inflammation in the lung parenchyma and the surrounding bronchi. Often focal pneumonia is preceded by inflammation of the upper respiratory tract, where the infection spreads to the lower divisions. The disease often develops in people with chronic bronchitis, bronchiectasia, in pulmonary circulation stasis, after surgical interventions, injuries to the thorax.

• Complaints are a gradual onset, cough with discharge of a small amount of mucopurulent sputum, mild dyspnea, subfebrile fever, weakness, sweating.

• Examination does not reveal anything special.

• Palpation and percussion reveal signs of lung tissue consolidation (amplified vocal fremitus and shortening of a percussion sound) only if the pneumonic focus is large enough (more than 3 cm) and is located close to the surface of the thorax.

• Auscultation reveals bronchovesicular breathing, moist fine bubbling râles.

• Laboratory data include moderate leukocytosis, leukopenia in viral infection and accelerated ESR.

8.8. Pleurisy is a disease of the pleura of the inflammatory nature accompanied by the deposit of fibrin on the surface of the pleura layers (dry pleurisy) or by exudate accumulation in the pleural cavity (exudative pleurisy). Etiologically, we distinguish infectious pleurisy (bacterial, viral, fungal, tuberculosis, etc.) and non-infectious (in malignant tumors, systemic connective tissue disorders, allergic reactions etc.).

8.8.1. Dry pleurisy

• Complaints are stabbing pain in the chest, worsening on deep breathing, coughing; dry cough, subfebrile fever, sweating.

• Inspection reveals forced position on the affected side, breathing is frequent and shallow with limitation of the respiratory lung excursion.

• Palpation reveals pain on palpation of the trapezius and pectoral muscles (Sternberg's symptom) with apical pleurisies.

• Percussion reveals no pathological changes.

• Auscultation reveals friction rub which is pathognomonic.

8.8.2. Exudative pleurisy

• Complaints are marked shortness of breath, feeling of heaviness in the chest, a feeling

• of transfusing/flowing liquid movement/floating liquid/ on the affected side. Chest pain and dry cough are likely. Weakness and fever are present.

• Inspection reveals forced position on the affected side, expanded and flattened intercostal spaces, increased volume of the affected half of the chest and its lag/delay in the act of breathing.

• Palpation reveals the absence of vocal fremitus on the affected side. The thorax is resistant.

• Percussion reveals total dullness over the area of fluid accumulation, the upper limit of which has a parabola form (Ellis-Damoiseau line).

• Auscultation reveals the absence of breathing sounds over the affected area. Bronchophony is not performed.

8.9. Acute bronchitis is an acute diffuse inflammation of the mucous membranes of the bronchi. Etiological factors include infectious (bacteria, viruses), physical (considerably dry, hot or cold air), chemical (acids, alkali, toxic substances etc.), organic allergens (dust, plant pollen, etc.).

Complaints are dry paroxysmal cough at the disease onset, which later gives way to productive one; expiratory shortness of breath occurs with the involvement in of small bronchi in the pathological process (bronchiolitis).

Inspection, palpation and percussion give little information.

Auscultation reveals harsh breathing, diffused dry râles of different timbre (low in tracheobronchitis, wheezing/whistling in bronchiolitis). Moist non-sonorous râles also appear after liquefaction of sputum.

8.10. Chronic obstructive bronchitis is a diffuse progressive lesion of the bronchi associated with prolonged respiratory irritation by harmful agents, characterized by inflammatory and sclerotic changes in the bronchial wall and accompanied by the restructuring of the secretory apparatus and hypersecretion of mucus. Etiological factors include prolonged smoking, air pollution from industrial wastes (coal, asbestos, silica dust etc.), cold humid climate, a genetic defect α1-antitrpsina and others.

• Complaints are permanent or recurrent cough with discharge of mucopurulent sputum for at least 3 months a year during two or more consecutive years (a compulsory symptom!), expiratory type of breathlessness (changing nature of breathlessness depending on the weather, time of day etc. is typical, shortness of breath which also depends on the conditons). Fever, weakness and sweating are noted on exacerbation.

• Inspection reveals difficult prolonged exhalation, swelling of the neck/jugular veins on exhalation. Thickening of the terminal finger phalanges (clubbing) and changed nails ("crystal glass") are present in long-lasting disease.

• Palpation reveals diminution/weakening of vocal fremitus only with the development of emphysema.

• Percussion reveals wooden/bandbox sound with the development of emphysema.

• Auscultation reveals harsh breathing, moist râles of different caliber on exacerbation. Dry wheezes/râles are also heard.

• Sputum is thick and purulent. It is of yellow and green color. It contains a large number of leukocytes, epithelial cells.

• Spirometry reveals reduced forced expiratory volume (FEV1), maxmum ventilation of the lungs (MVL), reduction of Tiffeneau index, respiratory failure of obstructive type.

8.11. Asthma is a chronic inflammatory recurrent disease with primary lesion of the distal bronchi characterized by their reversible obstruction and hyperreactivity. Compulsory clinical sign of bronchial asthma is a choking attack due to a spasm of the smooth muscles of the bronchi, hypersecretion, edema of the mucous membranes.

The following factors contribute to disease development: non-infectious allergens (pollen, plants, household dust, medications); infectious

allergens, primarily viruses, fungi, mycoplasma; chemical factors (wood, silicate, cotton and other dusts, aerosols, cigarette smoke); physical and meteorological factors (cold moist air, fluctuations in barometric pressure, magnetic field); stress and physical activity; heredity.

• Complaints are choking of expiratory type (exhalation is 2-4 times longer than inhalation, exhalation is difficult, painful), excruciating cough with a small amount of viscous, sticky sputum**.**

• Inspection reveals a forced position with emphasis to the arms with the involvement of supportive muscles in the act of breathing, noisy/wheezy breathing with distant wheezes, swelling of the neck/jugular veins, barrel-shaped chest.

• Palpation reveals rigidity of the thorax, greatly diminished/weakened vocal fremitus in acute emphysema.

• Percussion reveals acute emphysema: the expanding borders of the lungs, wooden/bandbox percussion sound.

• Auscultation reveals harsh breathing, a lot of dry, mostly wheezing/whistling râles, the number of which increases during the expiratory phase.

• Sputum is vitreous, viscous, contains a large number of eosinophils, Charcot-Leyden crystals, Curschmann's spiral.

• Spirography reveals a decrease in FEV1, the Tiffeneau index, positive test with bronchodilators.

8.12. Bronchiectasis

Bronchiectasis is an irreversible pathological enlargement of the bronchus as a result of inflammatory destruction of the bronchial wall. Bronchiectasis is classified according to the anatomical characteristics (saccular, fusiform, cylindrical), according to morphological characteristics – atrophic and hypertrophic, according to pathogenesis – congenital and acquired. Congenital bronchiectasis is due to a variety of pre- and postnatal developmental defects of the tracheobronchial system. Acquired bronchiectasis develops as a result of respiratory infections, airway obstruction, inhalation of aggressive chemicals.

Bronchiectasis can be accompanied by various pathological conditions or can be a manifestation of independent nosologic form, bronchiectasis disease, which is characterized by a chronic purulent process in the lumen of bronchiectasis. The clinical picture of the disease is characterized by the following signs:

• Complaints are cough with a large amount of purulent sputum which is easier discharged in a particular drainage position. Hemoptysis (vomiting blood) is also likely as well as weakness, anorexia, fever.

• Inspection reveals the change in the terminal phalanges of fingers (clubbing) and nails as "watch glasses".

• Palpation revelas non-specific changes, possibly local amplification of vocal fremitus above the large bronchiectasis.

• Percussion revelas a dull tympanic percusison sound over an area of bronchiectasis.

• Auscultation reveals harsh breathing over an area of bronchiectasis, persistent sound moist mediocre-bubbling wheezes.

• Sputum is purulent or mucopurulent, viscous. It contains a large number of white blood cells, red blood cells. The flora is abundant.

• Confirmation of diagnosis is by bronchoscopy, bronchography, tomography.

• Complications are pulmonary bleeding, renal amyloidosis, localized pneumonia, lung abscess, pleurisy/pleural effusion, respiratory failure.

8.13. Lung abscess is a nonspecific inflammation of the lung tissue, resulting in its necrosis (dissolution) in the form of a limited focus and cavity formation. The abscess goes through two periods in its development: before and after rupture (drainage) of cavity.

Clinical symptoms of the first period (before abcess rupture):

• Complaints are high fever, chills, profuse sweat, dry cough. There may be mild shortness of breath and pain of the pleural type in the chest.

• Inspection reveals hyperemia. There may be a lag/delay of the affected part in the act of breathing.

• Palpation reveals diminution/weakening of vocal fremitus.

• Percussion reveals a dull percussion sound.

• Auscultation reveals a local weakening of vesicular breathing.

Clinical symptoms of the second period (after abcess rupture):

• Complaints are cough with a large number of purulent, fetid sputum (by full mouth), decreased body temperature.

• Inspection reveals a probable lag/ delay of the affected part in the act of breathing.

• Palpation reveals amplification of vocal fremitus.

• Percussion reveals a tympanic percussion sound.

• Auscultation reveals bronchial or amphoric breathing, sound moist large bubbling râles.

• Complications are pleural empyema, purulent mediastinitis, pulmonary bleeding, septic shock, formation of chronic abscess.

8.14. Respiratory failure/insufficiency (RF/RI)

Respiratory failure/insufficiency is a condition in which normal arterial blood gas composition is not maintained, or the latter is achieved by abnormal (tense) performance of the external respiration apparatus, resulting in reduced functioning of the organism. According to etiology, RF/RI is divided into primary (impairment of the external respiratory apparatus itself) and secondary (pathology of the other parts of the respiratory system).

The basic mechanisms of the development of respiratory failure/insufficiency:

1. Impairment of the respiratory organs such as the airways and lung parenchyma is called pulmonary respiratory failure.

2. Impairment of the osteomuscular skeleton of the thorax and pleura, impairment of rib mobility (congenital or acquired deformity), lesion of the pleura, impairment of the respiratory muscles (impairment of innervation of the respiratory muscles such as poliomyelitis, and some others – paralysis, tetanus, botulism, curare poisoning)

3. Impairment of breathing regulation includes pathological changes of the central nervous system (tumors, heamorrhages, injury), inhibition of the breathing center by barbiturates, morphine, toxic and other lesions of the brain.

In respiratory pathology, RF/RI is the main clinical syndrome resulting from impairment of the pathophysiological processes of alveolar ventilation, diffusion of gases through the alveolar-capillary membrane and capillary blood perfusion of the lungs.

The obvious causes of the development of respiratory failure/insufficiency are:

1. Obstruction of the large airways (tumor, a foreign body etc);

2. Obstruction of the small airways (bronchial asthma, bronchiolitis);

3. Impairment of restriction of the alveolar tissue (interstitial edema, pleural effusion, pneumothorax, hydrothorax etc.);

4. Reduction of respiratory surface of the lungs (massive inflammation, lung resection, atelectasis);

5. Thickening of the alveolar-capillary membrane (interstitial edema, inflammation of the lung tissue, lung fibrosis);

6. Impairment of pulmonary blood flow (blood stasis in the pulmonary circulation, hypovolemia);

7. Impairment of ventilation-perfusion ratios (chronic obstructive bronchitis, inflammation of the lung, thromboembolism of the smaller branches of the pulmonary artery).

It should be remembered that most of the pathological processes in the lungs are accompanied by impairment of several mechanisms of gas exchange. For example, in pneumonia, restriction impairments are quite typical with simultenous reduction in gas diffusion through the alveolar-capillary membrane. The number of functioning alveoli is also reduced. In chronic bronchitis, along with acute obstructive disorders, there are impairments of ventilation-perfusion ratios due to considerable unevenness of lung ventilation.

There are two types of respiratory failure/insufficiency (see Table 8).

1. Obstructive type of RF/RI occurs due to the narrowing of the airways and an increase in resistance to air movemen. There is both impairment of lung ventilation and that of breathing mechanics. Exhalation becomes difficult, the respiratory muscles work increases. The main causes of obstructive RF/RI are: spasm of smooth muscles of the bronchi, inflammatory infiltration and swelling of mucous membrane of the bronchi, a large amount of viscous secretion in the bronchi, deformation of the bronchi, bronchial tumours, foreign bodies, expiratory collapse of the small bronchial tubes that have no cartilage skeleton.

2. Restrictive type of RF/RI is due to reduction of the lower respiratory surface of the lungs or reduction of their elasticity. The causes of restrictive RF/RI are massive infiltrative changes of the lung tissue, pneumosclerosis, pleural disease, restricted excursion of the lungs (pleuritis, exudative pneumothorax), reduced functioning of the lung parenchyma (lung resection, atelectasis, congenital lung hypoplasia, surfactant activity change), deformation of the thorax and other causes.

Clinical manifestations of RF/RI are quite similar, regardless of the mechanism of their formation. The following are the most significant in clinical practice:

- Shortness of breath;
- Central (diffuse) cyanosis;
- Work intensity of the respiratory muscles;
- Change in respiratory volumes and capacities;
- Intensity of circulation (tachycardia, increased minute volume).

The most important symptom of RF/RI is shortness of breath due to stimulation of the respiratory center. Objectively, shortness of breath is accompanied by a change in frequency, depth and rhythm of breathing. It is possible to determine the nature of breathlessness in obstruction of the small airways (expiration) and restrictive disorders (inspiration) more clearly. Cyanosis in RF/RI is due to a high content of reduced hemoglobin in the blood (over 50 g/L). This is diffuse warm cyanosis which decreases (or disappears) after inhaling pure oxygen for 5-12 min.

Table 8

Clinical differences of obstructive and restrictive RF/RI

Clinical manifestations	Restrictive RI	Obstructive RI
Shortness of breath	Inspiratory	Expiratory, often changes after coughing
Cyanosis	Central	Central, can be more marked in attacks of nonproductive cough
Cough	May be absent	With little sputum or productive, being forced
Auscultation	Changes are not of a typical character	Dry wheezes, worsen or appear on forced exhalation
Spirography data	Reduced VCL and MVL	Significantly reduced FEV1, MVL and the Tiffeneau index

As for the rate of clinical pathophysiological manifestations, we distinguish acute and chronic respiratory failure. The causes of acute RF/RI can be severe mechanical damage, massive bleeding, foreign body aspiration, severe attack of gasping, atelectasis, pulmonary edema, the massive inflammation focus.

9. Methods of functional and laboratory diagnosis

9.1. Spirometry/spirography is a method of graphical change in lung volumes when performing various breathing maneuvers. With the help of it we can estimate: 1) pulmonary volumes and capacities; 2) rates/values of pulmonary ventilation; 3) effectiveness of pulmonary ventilation.

Pulmonary ventilation rates do not have strict constant values. They depend greatly on the constitution/build-up of the body and physical training, height, body mass, age and sex of a person. Therefore, the data obtained are evaluated in comparison with the so-called proper values, taking into account all these data and which are normal for an examined person. Proper values are calculated in accordance with nomograms and formulas, which are based on the definition of the proper basic exchange.

Pulmonary volumes and capacities

1. Respiratory volume (RV) is the volume of air inhaled and exhaled on normal breathing, equal to an average of 500 ml (within the range from 300 to 900 ml). Of it, about 150 ml account for the so-called air of the functional dead space in the larynx, trachea, bronchi. This air does not participate in gas exchange. But we should not forget that air of functional dead space, combining with inhaled air, humidifies and warms it. This is its important physiological role.

2. Expiratory reserve volume equals to 1,500-2,000 ml that a person can exhale if after a normal exhalation a person makes maximum exhalation.

3. Inspiratory reserve volume equals to 1,500-2,000 ml that a person can inhale if after a normal inhalation a person makes maximum inhalation.

4. Vital capacity of lungs (VCL) equivalent to RV, expiratory and inspiratory reserve volumes averaging 3.700 ml is the air that a person is able to breathe out on the deepest exhalation after maximum inhalation. Deviation of actual VCL from the proper one should not exceed ± 15%.

5. Residual volume (RV) is the air remaining in the lungs after maximum exhalation (1,000-1,500 ml) and equals approximately 33% of VCL.

6. Total (maximum) lung capacity (TLC) is a sum of respiratory, reserve (inhalation and exhalation) and residual amounts and is equal to about 5,000-6,000 ml.

Indicators of pulmonary ventilation

1. Respiratory minute volume (RMV) is calculated by multiplying RV

by the respiratory rate per minute. On average, it is 5,000 ml (6-8 L). It is estimated at rest and depends on the level of metabolism in the body.

2. Maximum ventilation (MV, "breathing limit") is the amount of air that can be ventilated through the lungs on the maximum exertion of the respiratory system per minute. It is estimated by spirometry on deep breathing at a rate of about 50 breaths per minute, a normal range is 80-200 l/min.

3. Respiratory reserve (RD) is calculated by the formula RD = MLV – RMV. In healthy individuals, it is equal to 85% of maximum lung ventilation (MLV). In case of respiratory failure, it is reduced to 60-55% and below.

Studying the mechanics of respiratory act allows you to identify the change in inhalation and exhalation ratio, respiratory effort in different phases of respiration and other indicators.

1. *Forced vital capacity (FVC)* is determined using the method of Votchal-Tiffeneau. The measurement is like that carried out in estimating vital capacity of the lungs. Its main condition is the fastest, forced exhalation, the FVC in healthy individuals being 8-11% (100-300 ml) less than vital capacity of the lungs, mainly due to the increased resistance to airflow in the smaller bronchi. Test of Votchal-Tiffeneau primarily reveals obstructive impairments of the airways. In case of bronchitis, bronchial asthma, emphysema and other obstructive conditions the difference between FVC and VCL rises to 1,500 ml or more.

2. *Forced expiratory volume in first second (FEV1)* is also determined. In healthy individuals, it averages 82.7% of VCL. FEV1 is one of the main indicators of lung ventilation function. In case of obstructive impairments, it decreases at the expense of the slowing down of the forced exhalation, and in case of restrictive impairments, it decreases at the expense of reduction of all lung volumes.

3. Normally, index FEV1/VC×100% is calculated. It is the so-called Tiffeneau index which normally exceeds 70%. The Tiffeneau index is reduced in obstructive syndrome (decreased FEV1 at relatively safe VCL). In restrictive syndrome, the Tiffeneau index does not change because all lung volumes reduce proportionally.

9.2. Sputum examination helps determine the nature of the pathological process in the respiratory organs and, in some cases, determine its etiology. Sputum is pathological discharge of the respiratory system that comes

out when you cough. The composition of sputum may include mucus, serous fluid, blood cells, protozoa, rarely helminths and their eggs. It is better to take fresh sputum in the morning, if possible, before meals and after rinsing the mouth. Only sputum to detect Mycobacteria tuberculosis lungs, providing the patient spits out little of it, can be collected within 1-2 days. For collecting sputum, special spittoons with screwing lids and measuring grades are used.

On macroscopic examination, the amount, character, color and consistency of sputum are determined. Amount of sputum can widely range from 10 ml in laryngitis to 500-1,000 ml in tuberculous caverns, bronchiectasia, abscess and lung gangrene. Consistency of sputum is usually viscous, depending on the content of the liquid in the sputum. Sputum viscosity increases with high content of fibrin (in inflammation). Liquid mucus in large quantities is discharged in case of lung edema. Nature of sputum is determined by the main component: mucous (consisting mainly of mucus like in acute bronchitis), purulent (pure purulent sputum is found in case of rupture of lung abscess, more often sputum is mucopurulent), serous (liquid and foamy sputum is blood transudate). Mucous sputum is usually colourless. Turbid sputum is observed in acute bronchitis. Serous mucus is also colorless, liquid, foamy, is observed in lung edema. Mucopurulent sputum, yellow or greenish is excreted in chronic bronchitis, tuberculosis etc. Purely purulent, homogeneous, semi-fluid, greenish-yellow sputum is characteristic of rupture of lung abscess. Bloody sputum can be both pure blood as in case of pulmonary bleeding (tuberculosis, cancer, bronchiectasis) or of mixed nature, for example, mucopurulent with streaks of blood (in bronchiectasis), muco-bloody (infarct lung or stasis in the pulmonary blood circulation), purulent-bloody, semi-fluid, brownish-grey (in gangrene and lung abscess). If the blood from the respiratory tract does not come out immediately, but remains in the respiratory tract for a long period of time, its hemoglobin turns to hemosiderin and gives sputum rusty color characteristic of lobular pneumonia. When standing, sputum may flake. Smell from sputum is often absent. The fetid smell of newly discharged sputum depends on putrid disintegration of tissue (gangrene, cancer) or from decomposition of sputum proteins when they remain in the cavities (abscess). Sputum medium reaction is usually alkaline. It becomes acidic on decomposition and from gastric juice admixtures, which helps differentiate hemoptysis from bloody vomit.

Microscopic examination of sputum is done in both native/natural and dyed materials. In the sputum we can be detect:

• *White blood cells*. Any sputum usually contains small number of them. A large number of neutrophils are found in inflammatory processes; eosinophils appear in the sputum in bronchial asthma.

• *Red blood cells* are found in the sputum in pneumonia ("rusty" sputum), stasis in the pulmonary blood circulation, myocardial infarction or destruction of the lung.

• *Alveolar macrophages* are detected if the material is obtained from the deep divisions of the respiratory tract. They can be colorless (myelin grains), black due to charcoal particles (dust cells) or yellow-brown due to hemosiderin ("cells of heart defects"). Alveolar macrophages are present in small quantities in any sputum, more of them are found in inflammatory diseases.

• *Epithelial cells*. Simple squamous epithelium in the oral cavity, pharynx, larynx or cylindrical epithelium of deeper divisions of the respiratory tract can be found in sputum.

• *Elastic fibers* are found in the sputum in case of lung tissue disintegration, e.g. in abscess, tuberculosis, cancer.

• *Cells of malignant tumors* are revealed in the sputum, especially when the tumour grows endobrochially or disintegrate. They are predominantly large, of ugly form, have a nucleus, and sometimes many nuclei.

• *Curschman's spirals* are casts of the narrowed bronchioles. They are found in sputum in case of bronchial asthma.

• *Charcot-Leyden crystals* are clear crystals of various sizes. They remind a compass arrow in their form. They consist of protein undergoing crystallizaton in disintegration of eosinophils. They are found in the sputum in bronchial asthma.

• *"Lentils"* are small greenish-yellow, dense lumps consisting of calcified elastic fiber, cholesterol crystals and saponifiers. They contain Mycobacterium tuberculosis.

• *Dittrich's plugs* are similar to "lentils" in appearance and composition but do not contain mycobacterium tuberculosis. When crushed they produce fetid smell (in gangrene, chronic abscess, putrid bronchitis);

• *Drusen of actinomycetes* are like small yellowish grains resembling semolina. If pressed under a microscope cover glass in a drop of glycerin or alkali, a druse usually shows a visible central part consisting of mycelium plexus.

Microscopy of stained preparations is performed to examine the microbial flora of sputum and some of its cells.

9.3. Pleural puncture. The pleural cavity of a healthy person has a small amount of fluid, in composition similar to lymph, which facilitates the sliding movements of the pleural layers on breathing. The volume of pleural fluid may increase (effusion) both in the blood disorders and impairment of lymph circulation in the lungs, i.e. non-inflammatory effusion (transudate) and in case of inflammatory changes of the pleura (exudate).

The presence of fluid in the pleural cavity serves as an indicator for diagnostic pleural puncture, which enables you to determine the nature of the fluid and the tactics of treatment of the patient. Pleural puncture (thoracentesis) is applied: 1) for diagnostic purpose, i.e. to determine the nature of pleural fluid and clarify the diagnosis; 2) for the purpose of treatment, i.e. to remove fluid from the pleural cavity and, if necessary, for administering/giving medications.

Technique. Usually the puncture is made below the angle of the scapular (shoulder blade) or in the posterior axillary line in the zone of maximum dullness, determined by percussion beforehand, usually in the seventh or eighth intercostal spaces along the upper margin of the underlying rib because the intercostal vessels pass along the lower margin. For diagnostic purpose, take/withdraw 50-150 ml of fluid and send it for chemical, cytological and bacteriological examination. For therapeutic purposes, withdraw up to 800-1,200 ml if there is accumulation of considerable amount of fluid in the pleural cavity. Removal of more amount of fluid from the pleural cavity leads to a rapid displacement of the mediastinum to the affected side and may be accompanied by a collapse.

On examination of pleural fluid, first of all, define its character, either it is transudate or exudate. Transudate is effusion of non-inflammatory origin which accumulates in the pleural cavity due to common disturbances in water-electrolyte balance in the body. It is clear, colorless or slightly yellowish liquid. Exudate is produced when there is inflammation and reactive processes in the pleura (pleurisy). Physical and chemical examination of pleural fluid is of the utmost importance in distinguishing between transudate and exudate, relative density and protein content being estimated alongside with it (see Table 9). To distinguish between transudate and exudate, the Rivalta test is done: water in a glass cylinder is mixed with 2 or 3 drops of 80% acetic acid. After it, a few drops of fluid under examination are added into the

prepared solution. If the liquid is exudate, you can see white clouds resembling sigarette smoke after the falling drops (serosomucin)

Table 9

Differential diagnostic signs of transudate and exudate

Indicator	Transudate	Exudate
Relative density	Less than 1,015	More than 1,015
Protein,%	Less than 3	More than 3
White blood cells (in field of vision)	Less than 15	More than 15
Rivalta test	Negative	Positive

As for the nature of the exudates, we distinguish: serous, fibrinous, serous-fibrinous, purulent, putrid, bloody and others. The nature of pleural effusion is determined by its cell composition. Physical examination also determines the color and transparancy/opacity of pleural fluid. Serous exudate is lemon-yellow. At the maximum level flakes of fibrin fall into exudate, making it turbid. Turbidity of exudate may also be due to the presence of leukocytes, red blood cells (in hemorrhagic exudate).

Microscopic examination is performed when working with sediment of the pleural fluid by centrifuging. Sediment cells are studied by several methods: by examining native preparations, taking dry smears stained by method of Romanovsky-Gimze. Also, fluorescent microscopy is used when searching for tumour cells as well as histological examination of sediment put in paraffin or cell culture. The number of corpucular elements is counted. A small number of erythrocytes can be in any punctate due to injury in puncture. There are a lot of them in hemorrhagic exudate, e.g. in tumors, trauma, hemorrhagic diathesis. A large number of leukocytes are estimated in bacterial infection of the pleura. Transudate contains not so many leukocytes but it contains a lot of mesothelium cells. On microbiological examination, transudates are usually sterile. Exudates can be sterile, for example, effusion in rheumatic pneumonia, lung cancer. In pleurisy which is caused by pyogenic flora, this pyogenic flora can be already detected on bacterioscopy of Gram-staining smear sample. In other case, a smear culture should be done. All detected microbes are checked for their sensitivity to antibiotics to make the treatment effective.

UNIT 3. EXAMINATION OF THE PATIENT WITH DISEASES OF THE GASTROINTESTINAL TRACT

3.1. Subjective and objective methods of investigation

3.1.1. Complaints. People suffering from diseases of the upper gastrointestinal divisions (esophagus, stomach and duodenum) have a variety of complaints, which are usually divided into local and general.

The main local complaints are those that directly point to the impairment of the esophagus, stomach and intestines:

1. Dysphagia is impairment of the act of swallowing and passage of food through the esophagus.
2. Complaints about the severity and pain in the epigastric area.
3. Dyspeptic complaints are heartburn, regurgitation, nausea, vomiting, bloating (flatulence), diarrhea, constipation.
4. Complaints about appetite disorders.

The main common patient's complaint is weight loss.

3.1.2. History significance. In many cases, a correct interpretation of some passport data as well as medical diseases and life suggests the presence of a specific disease of the stomach or duodenum. Of the greatest diagnostic significance are:

1. Age and sexual dimorphism in the digestive tract diseases (especially the developmental peculiarities and a course of the disease, depending on age and sex).
2. Evaluation of the disease course (alternating periods of exacerbation and remission in ulcers, a steadily progressing course in stomach cancer).
3. Changing nature of complaints (for example, the absence of general association between pain and food intake, appearance of new complaints).
4. Working and living conditions (mental overwork, violations of work and rest, occupational hazards).
5. Past and associated diseases.
6. Heredity.
7. Taking ulcerogenic drugs.
8. Bad regimen and violations in nutrition, bad habits.

3.1.3. Examination of the abdomen. When examining the abdomen, attention is paid to its shape, characteristics of the abdominal wall and motility. Normally, the stomach is slightly convex, the right and left halves

are symmetrical, the navel neither bulges nor is retracted, the costal arches are slightly outlined. Pathologically, a change in the abdomen shape can be observed, either preserving symmetry or not.

The enlargement of the whole abdomen is observed in obesity, meteorism, accumulation of inflammatory or edematous fluid in the abdomen (ascites), the swelling of the abdominal wall etc. Physiological enlargement of the abdomen is pregnancy. The abdomen is mainly enlarged in its middle part in obesity. On palpation, the abdominal wall is thickened, the navel is retracted. In case of flatulence, the abdomen is evenly distended, has the shape of a hemisphere which does not change when moving the patient from horizontal to vertical position. A loud tympanic sound is heard on percussion. The navel bulges in ascites, the shape of the abdomen/belly varies depending on the patient's body position because the fluid is located in the most slopin /oblique sites. In the upright position, it is located in the lower abdominal. In horizontal position, liquid is diffused in the field of flanks and the abdomen becomes flattened (a frog's belly). Total abdominal retraction may be present in malnutrition, profuse diarrhea, prolonged vomiting. The abdominal wall is thin and flaccid. The enlargement of abdominal sections with impairment of its symmetry is observed:

- in considerable enlargement of some organs (splenomegaly),
- in development of large-size tumours in the abdominal cavity or behind it,
- in the presence of exudates,
- in abscess or inflammatory infiltrates,
- in case of expansion of individual divisions of the gastrointestinal tract above a place of obstacles
- in hernias of the anterior abdominal wall.

When examining the abdominal wall, attention is paid to the presence of rashes, hernias, venous network on the skin of the abdomen etc. Hernia can be localized in different parts of the abdomen. Sometimes they are better visualized in vertical position. Extended venous network on the front wall of the abdomen reveals obstruction of blood circulation in the system of the lower hollow and portal veins. It also occurs in digestive pathologies (cirrhosis) and in some other diseases. Pregnancy stretching marks, postoperative scars, pigmentation can be revealed on the abdominal skin. Inspection may also reveal visible peristalsis, epigastric pulsation.

3.1.4. Palpation of the abdomen. General rules of palpation: 1) the patient takes a position on the back with slightly bent legs and torso along with the hands; 2) head of the patient should lie low because a high headrest causes muscle tension of the abdominal wall; 3) the patient should breathe deeply involving the muscles of the abdomen; 4) the doctor should be on the right of the patient, the hands should be warm, nails should be short trimmed. There are two types of palpation: superficial and deep palpation.

Surface-oriented palpation of the abdomen allows you to determine the presence of pain, degree of tension of the abdominal wall, the divergence of rectus muscles of the abdomen, a considerable enlargement of the abdominal organs. Surface palpation is done starting with the left iliac region counterclockwise to the pubis and further up the midline of the abdomen. If you know in advance what portions are painful, begin palpation with a symmetric painless site.

Tenderness in the field of projection of the abdominal organs always points to various pathological processes in them. Skin hyperesthesia is also of diagnostic value. It is revealed both in the area of projection of the affected organ and as reflected pain. In diseases of the stomach body, tenderness on palpation is usually localized in the epigastric area. In pathology of the pyloric portion and duodenum, it is localized in the pyloroduodenal portion and in the point of the diaphragmatic nerve on the left (left phrenicus is a symptom). In ulcers, pain is revealed on tapping the spinal processes of the 7^{th} – 12^{th} thoracic vertebrae (Openkhovskij's points) as well as in the paravertebral points on the right at level of the 7^{th} – 11^{th} thoracic vertebrae (Boas' painful points).

Under pathological conditions, there are two types of tension increase in the abdominal wall: 1) resistance, i.e. resistance of the abdominal wall to the palpating fingers in places corresponding to pathological (inflammatory) process in a deeply located organ; 2) muscular defence/protection is observed where there is inflammation in the abdomen, involving the peritoneum. Unlike resistance, tension of the abdominal wall may reach wood hardiness. Palpation is sharply painful.

To determine divergence of the rectus muscles, put your half-bent fingers of the right hand to midline below the xiphoid process and ask the patient to raise his head. As a result, rectus muscles become tense and if there is their divergence, the examiner's hand falls deep into the abdominal cavity.

Deep methodical sliding palpation of the abdomen enables to perform a topographical demarcation of the abdominal organs, determine size, shape, position, nature of surface, tenderness and mobility of these organs. The essence of the method of deep sliding methodical palpation is while getting the hand into the depth of the abdominal cavity, sliding your fingers along the back of the abdomen to feel the examined organ and palpating it with fingers to determine its characteristics.

Rules of performing sliding deep and methodical palpation: 1) The palm of the right hand is applied above the projection of the organ, with the finger tips being on the same line and located parallel to the long axis of the palpated organ; 2) On palpation, the patient must breathe evenly, deeply, using abdominal breathing type; 3) On inhalation, a skin fold is made by the fingers, shifting the skin in the direction opposite to the direction of palpation; 4) On exhalation, the finger tips should be put inside the abdominal cavity as deeply as possible to its back wall 5) At the end of exhalation, the finger tips slide in the direction perpendicular to the palpated organ, pressing it to the back wall of the abdomen. A tactile impression about the peculiarities of the organ (its properties) is made at this particular moment.

One of the most important conditions for performing deep palpation is knowledge of projection of abdominal cavity organs on the frontal abdominal wall:

- The left hypochondrium, i.e. cardiac portion of the stomach, the cauda/tail of the pancreas, the spleen, the left curvature of the colon.
- The epigastrium, i.e. the stomach, the duodenum, the pancreas, the left liver lobe.
- The right hypochondrium, i.e. the right liver lobe, the gall bladder, the right curvature of the colon.
- The abdominal flanks, i.e. the ascending (right) and the descending (left) colon divisions, a part of loops of the small intestine.
- The umbilical area, i.e. the loops of the small intestine, the transverse colon, the horizontal part of the duodenum, the greater curvature of the stomach, the head of the pancreas.
- The left iliac region, i.e. the sigmoid colon.
- The area above the pubis, i.e. the loops of the small intestine.
- The right iliac region, i.e. the caecum, the terminal ileum division, the appendix.

Usually, the sequence of deep palpation is as follows: the sigmoid colon, the caecum, the appendix and the final part of the small intestine, the ascending and descending colon, the transverse colon, the large curvature of the stomach, the pylorus. Palpating the intestine, we determine its diameter, density, mobility, tenderness, presence of peristalsis. Normally, in the overwhelming majority of cases it is possible to palpate the sigmoid, caecum and transverse colon. The small intestine is usually not palpable because it is extremely movable. The transverse colon is palpated bilaterally. Before palpation it is necessary to detect the lower border of the stomach, since the transverse colon is located 2 – 3 cm below it. The lower border of the stomach can be detected using the method of percussion or auscultation-affriction.

3.1.5. Percussion and auscultation of the abdomen. Percussion of the abdomen of a healthy person reveals a tympanic sound. Percussion of the abdomen is essential for the diagnosis of accumulation of free fluid in the abdominal cavity. Dullness in the lower abdomen is revealed in the vertical position of the patient in the lower abdomen, tympanic sound/tympanitis is revealed above it. When the patient lies on his/her back, dullness is determined in the lateral abdominal parts. Normally, auscultation of the abdomen identifies the recurring sounds of the bowel peristalsis. Intensified intestinal sounds may be due to the accelerated promotion of the intestinal contents, the narrowing in the digestive tract, liquid consistence of the intestinal contents. In case of mechanical obstruction, peristaltic sounds above the place of narrowing become more sonorous, but disappear completely in case of paralytic obstruction.

3.2. Major clinical syndromes

3.2.1. Pain syndrome. Abdominal pain is a vital sign of gastrointestinal pathology. Localization of pain to some extent indicates the affected organ: pains behind the sternum point to the esophagus and cardiac portion of the stomach; pain in the left epigastrium points to the body of the stomach; pain in the right epigastrium points to the pylorus or duodenum; pain in the paraumbilical region points to the small intestine (except the duodenum); pain in the lower abdomen divisions on the left poins to the disease of the left half of the colon. Pain syndrome in the digestive tract diseases is characterized by rhythmic rate (time of onset and meal dependency), periodicity/frequency

(alternation of pain and their absence) and seasonal character (spring/autumn). Constancy of pain indicates the involvement of the submucous layer in the pathological process. Pain intensity is marked by moderate pain in case of gastrointestinal diseases without complications. Violent ("dagger-like/knife-like"/stabbing) pain is common in perforation of the stomach.

Daily rhythmic character of pain (after meal) to a certain extent indicates the location of the pathological process. The sooner the pain appears after meal, the more proximal localization of lesion is.

- Pain during meal makes you think about the problems with the esophagus.
- Increased pain immediately after meal or during 15-20 minutes after meal occurs in diseases of the esophagus and cardiac stomach portion.
- "Early" pain appears 30-60 minutes after meal and reduce after evacuation of food from the stomach. They are common in lesions of the gastric body.
- "Late" pain appears after 1.5 – 2 hours after meal and are regarded as a sign of duodenal or pyloric ulcers as well as duodenitis.
- "Fasting" pain is common in duodenal ulcer.
- "Night" pain appears between 11:00 pm to 3:00 am. They are typical of duodenal ulcer.

3.2.2. Dyspeptic syndrome. Characteristic clinical and laboratory signs of digestive disorders are grouped under the name of dyspeptic syndrome. Depending on the prevailing dysfunctions of the digestive organ, dyspepsia is divided into the following clinical forms: gastric, intestinal, pancreatic and other.

Gastric dyspepsia includes complaints about heartburn, regurgitation, nausea and vomiting. Heartburn is a feeling of burning pain along the esophagus, behind the breastbone/substernal or epigastric area caused by irritation of the esophageal receptors by the regurgitation of food from the stomach. It often occurs when there is increased acid production. Regurgitation is a sudden involuntary discharge of gas from the stomach or esophagus into the oral cavity, sometimes with small portions of the stomach contents. We distinguish air, sour, rotten, food regurgitation. Nausea is an unpleasant feeling in the epigastric area, chest and mouth, often preceding vomiting. It can be accompanied by salivation, skin pallor, increased sweating. Vomiting is a complex reflectory act involving the vomiting centre, leading to expulsion

of the stomach contents through the mouth. Physiologically, vomiting is a protective reaction of the digestive system to toxic or other harmful substances. Source of vomiting reflex may become irritation of the back wall of the pharynx, coronary vessels, peritoneum, mesenteric vessels, bile ducts, cortex. If the patient has vomiting, attention should be paid to the nature of vomitus, time of appearence and causes vomiting. Vomiting in the morning on an empty stomach, with a large amount of mucus is characteristic of chronic gastritis. Vomiting occurs within a few minutes after meal in case of ulcers of the cardiac portion of the stomach. In case of non-complicated ulcer, vomiting is rare, usually very acidic. In problems with evacuation of the stomach content, the vomitus has an unpleasant smell, it is abundant and contains the remains of food eaten long time ago. Hematemesis with color of "coffee grounds" can be present in gastrointestinal bleeding.

Intestinal dyspepsia includes the following symptoms: borborygmi in the abdomen, bloating, diarrhea, constipation. Borborygmi appear in case of increased peristalsis and can be heard from a distance. Flatulence is bloating of the abdomen that occurs due to enhanced gas formation in the intestines and malabsorption of gases. In digestion process, a healthy person makes about 15 liters of gas. Most of them are absorbed by intestinal wall and about 2 liters are excreted/discharged outside. Patients complain of extention and expantion of the abdomen, cramping (intestinal colic). Diarrhea is frequent bowel movements (usually more than 2 times a day) with production of dissolved and sometimes copious bowel movements. Diarrhea is usually associated with accelerating peristalsis of the bowel, reducing intake of water and electrolytes. In stomach diseases diarrhea is usually associated with inadequate secretion of hydrochloric acid. Constipation is a long delay of stool (stool less than once every 2 days) or a great difficulty in discharge of a small amount of feces at usual terms (less than 100 grams per day) without feeling full bowel movement. Constipation is usually associated with the disorder of intestinal motility, presence of obstacles to normal passage of intestinal content, a mismatch between the colon and the volume capacity of intestinal contents. Excessive secretion of hydrochloric acid causes constipation in stomach diseases.

Appetite disorders in patients with gastrointestinal diseases are very common. Loss of appetite can be due to a decrease in secretory and acid production function of the stomach. True anorexia must be distinguished

from cytophobia which is a fear of food intake because of pain (in stomach ulcer). Increased appetite is often observed in patients with duodenum ulcer due to increased acid secretion. Perversion of appetite often happens in stomach cancer, e.g. aversion to meat.

3.2.3. Syndrome of motor and evacuation dysfunction of the stomach is a complex of functional disorders caused by changes in peristalsis (hyper- and hypokinesia) and muscle tone (hyper- and hypotension) of stomach accelerating or slowing down the evacuation of gastric contents.

Hyperkinesia and hypertonicity. Vagus nerve irritation increases motor activity of the stomach and accelerates gastric evacuation. Factor of humoral regulation, gastrin, is of some importance in the development of gastric hyperkinesia. In people with a healthy stomach, hyperkinetic effects can be seen on rough diet intake, irregular diet, taking large quantities of alcohol or certain medicines, psychic shock. One of the most common clinical forms of increased tone of the stomach is the spasm of pylorus. Clinical picture includes sudden spasmodic pain in the epigastric area. Long spasm of the pylorus results in retention food mass in the stomach, which manifests by heaviness, belching, nausea and vomiting with stomach contents as well as signs of irritation of the vagus nerve, i.e. sweating and salivation. When examining the epigastric area, you can see the peristaltic movement. On palpation the pylorus is like a tight band.

Hypokinesia and hypotension. Sympathetic nerve irritation reduces rhythm, contraction strength and the spread speed of peristaltic waves. Acidic and hypertensive solutions, ethanol and fats as chemical agents cause a marked motor inhibition. Gastrointestinal hormones such as secretin and cholecystokinin-pancreosimin inhibit motor activity of the stomach. In healthy people, hypokinesia and hypotension of the stomach can occur in prolonged overeating and taking a lot of fluid, increased fat content in the food, in depression. Reduced peristaltic movements and tone of the stomach cause the openening of the pylorus, so that stomach contents are easily released into the duodenum and thrown back into the stomach. Hypotension of the stomach is typical of persons with asthenic constitution and can develop in general intoxication and hypoxia. The clinical presentation of chronic hypotension of the stomach develops gradually. Patients complain of change in appetite, rapid fullness/saturation. They also complain about systematic belching with rotten feeling, heartburn, especially

in horizontal position (hypotension of the cardiac sphincter), a feeling of fullness and heaviness in the epigastric area which occur more often after a hearty meal. Atonic constipation is often observed.

3.2.4. Secretory stomach dysfunction syndrome is a complex of clinical and laboratory signs of functional disorders of the secretory apparatus of the stomach, i.e. hyper- or hyposecretion of gastric juice, hyper-, hypo- or achlorhydria, gastric achylia. A healthy person produces about 2 litres of gastric juice per 24 hours. Nornally, gastric juice has acidic reaction, its value being estimated by the ratio of acidic and alkaline components of gastric secretion. An acid component is hydrochloric acid. Activating enzymes, the optimum pH for activity of gastric proteinases, bactericidal properties of gastric juice and stimulating pancreatic secretion are due to hydrochloric acid. The main gastric enzyme is pepsin which is produced in the major cells. An alkaline component is mucus in colloidal suspension of bicarbonates and neutral chlorides. It is constantly present in gastric juice. It covers the entire inner surface of the stomach and serves as an important protective factor against harmful action of hydrochloric acid and pepsin. Insoluble mucus is a product of epithelial cell activity. It binds with hydrochloric acid and adsorbs pepsin. Soluble mucus is produced by the mucous cells of the cervix of the gastric glands and, forming a complex with pepsin, contributes to hydrolysis of proteins by this enzyme.

Secretion of gastric juice is due to the influence of specific stimuli. There are three phases in gastric secretion: neurogenic (vagal), stomach (gastrin) and intestinal. Increase in vagal nerve tone is the main stimulus of increased secretion. Increased irritation of the pyloric area and increased production of gastrin occur in impairment of motor action of the stomach and retention of food mass.

Gastric hypersecretion (increasing amount of gastric acid) is typical of people suffering from vagus nerve disorders, in overeating, in taking food which contributes to increased secretion of juice and in smokers. Hypersecretion can be accompanied by increased production of hydrochloric acid, i.e. hyperchlorhydria. Increased juice secretion usually parallels increased acidity, but sometimes there is a mismatch. Patients complain of hunger night pains in the epigastric area, sour belching/regurgitation, heartburn, vomiting. Relief is brought by taking soda/bicarbonate solution and absorbing antacids. Appetite is usually increased, there has been a tendency to constipation.

Hyposecretion of gastric juice is observed when the number or activity of secretory cells is reduced or decreased. It is also observed in irritation of the sympathetic nervous system, as a result of hard muscle work of persons working in noise, vibration, high temperature, etc conditions. Hyposecretion is usually combined with a decrease in acidity of gastric contents.

Absolute achlorhydria is the result of profound structural changes in the glandular stomach apparatus, free hydrochloric acid being absent even after histamine administration. In chronic gastritis, hypochlorhydria is often associated with hypersecretion of mucus. Complete absence of gastric juice and hydrochloric acid and pepsin is called achylia. Organic achylia is the result of irreversible lesions of the glandular stomach apparatus. It occurs in atrophic gastritis, stomach cancer, severe endogenous intoxication. The clinical picture includes decrease of appetite, unpleasant taste in the mouth, belching with rotten feeling, nausea, a feeling of constant heaviness in the epigastric area, a feeling of bloating. Patients poorly tolerate meat. There are also signs of intestinal dyspepsia, achylic diarrhea (especially after taking fats and milk), progressive weight loss, B_{12}-folic acid deficiency anemia.

3.3. Methods of examination of acid-secretion stomach function

3.3.1. Fractional gastric probing. Gastric secretion is examined by fractional probing done by a thin probe on an empty stomach, in basal conditions and after stimulation (see Table 10). To stimulate secretion, some enteral stimuli can be used (cabbage broth, meat broth, coffein or alcoholic breakfast). However, now a parenteral submaximum histamine test is used more often: subcutaneous administration of histamine hydrochloride in a dose of 0.01 mg/kg of body weight (or 0.1 ml per 10 kg of body weight). When performing a fractional examination, first, gastric juice contained in an empty stomach is extracted. Then, 4 portions of basal secretion are obtained within an hour with 15-minute intervals. After stimulation of the gastric glands, the other 4 portions are obtained within 60 minutes. In each extracted portion, we estimate the volume of gastric juice, total acidity (titration of 0.1 M NaOH), free and bound hydrochloric acid as well as debit-hour (production of hydrochloric acid per hour) is calculated: the amountof gastric juice (ml) is multiplied by the free hydrochloric acid (t.u) and is divided by 1,000.

Table 10

Standard values of key indicators of gastric secretion

Indicator	On an empty stomach	Basal secretion	Submaximum secretion
Gastric volume, ml	50-100	50-100	100-140
Total acidity, t.u.	up to 20	40-60	80-100
Free acid, t.u.	0-10	20-40	65-85
Debit-hour of free hydrochloric acid, meq/h	-	1-4	8-12

3.3.2. Intragastric pH measurement. The advantage of this method compared with a fractional probing is the possibility to determine pH within the range of 2.5 – 6.9 and more (see Table 11). Acidity below 2.5 can be determined only by titration method due to low sensitivity of reagents. However, the pH measurement does not allow to estimate volume values but it can be used to examine acid secretion for a lont period of time, using both stimulants (histamine) and secretion blockers (atropine). To measure pH, a probe of 1 – 1. 5 mm in thickness is used. It has two (and more) sensors that register acid secretion in the body of the stomach and alkaline reserve in the pylorus within hours every 10 minutes. Examination is carried out within an hour more when tests are used.

Table 11

Evaluation of pH-metric data

Interpretation	PH of the cardiac part	The pH of the antrum
Norm	1.6-2.0	more than 2.5
Note. Normally the difference between antral pH and pH of the cardiac parts must not be less than 1.5 – 2 units.		
Disturbance of alkalinity		0.9-2.5
Hyperacidity	0.9-1.2	
“Acidic stomach”	0.6-1.2	0.9-2.5
Hypoacidity	2.1-5.0	
Achlorhydria	more than 6.0	

3.3.3. Non-probing methods of examination are used for screening surveys of a large number of people and if probing is contraindicated (severe general condition, varicose veins of the esophagus, esophageal diverticula, aortic aneurysm, high blood pressure, severe organic damage to the cardiovascular system).

Desmoid probing. 0.1 g of methylene blue is placed in a rubber (desmoids) bag and sealed with catgut. The patient swallows a pill on an empty stomach. If gastric juice has hydrochloric acid and pepsin, catgut is digested. Methylene blue enters the lumen of the stomach, is absorbed, enters in the general blood circulation and is excreted with urine afterward, painting it blue. After swallowing a pill, urine is collected within 3, 5 and 20 hours, evaluating the time and intensity of coloring.

The method of ion exchange resins is based on the internal use of pills of ion exchange resins saturated by indicator (quinine, Azur II). The ions of this indicator can be exchanged for an equivalent number of hydrogen ions in the presence of hydrochloric acid (at pH is less than 3.0). The indicator released is then absorbed in the small intestine, enters the bloodstream and is excreted with urine. Evaluation of secretory function is made analyzing the urine staining extent.

Acidotest. After complete emptying of the bladder a patient is given 2 coffein-benzoat sodium tablets. After an hour, the first (control) urine sample is taken. Then a patient is given a dragée of a dye (2.4 diamino-4-etoxyazobensol). The second urine sample is taken within 1.5 hours. Both samples are diluted to 200 ml, acidified with hydrochloric acid. Colorimetric measurement of hydrochloric acid concentration, is carried out using color scale.

3.4. Diseases of the stomach and duodenum

3.4.1. Gastritis: definition, etiology, classification, clinic.

Gastritis is inflammation of the mucous membrane of the stomach. It is one of the most common diseases of the internal organs. According to some data, up to 50% of the population of the developed countries suffers from chronic gastritis. The practical significance of gastritis is determined not only by its incidence, but also because gastritis is a forerunner of other, more threatening diseases, like peptic ulcer and gastric cancer. The main etiological factors of gastritis are divided into exogenous (bad food, al-

cohol and nicotine abuse, the effect of thermal, chemical or other agents, impact of some professional hazards, *Helicobacter pylori* infection (HP), food allergy) and endogenous (neuro-reflex impact, endocrine disorders, hypoxemia, genetic factor). *The Sydney gastritis classification* (1990) can be simply represented as follows (see Table 12):

Table 12

Sydney gastritis classification

Type	Topography	Histology	Endoscopy
Acute Chronic Specific forms	Antral Fundal Pangastritis	Inflammation Activity Atrophy Intestinal metaplasia The infestation of HP	Erythematous Hemorrhagic Reflux gastritis Atrophic Erosions Hyperplasia of folds

Acute gastritis is inflammation of various etiologies affecting the stomach mucosa. It occurs at any age. As a rule, it is possible to trace its relationship to a certain etiological factor. The most common factors of acute gastritis are: use of low quality food, use of very hot, rough etc. food) and chemical (alcohol use, use of salicylates, acids, alkalis). Acute radiation gastritis can develop after radiation therapy.

Morphologically, we distinguish acute catarrhal, corrosive and phlegmonous gastritis.

Clinical symptoms of acute catarrhal gastritis develop within 6-12 hours after exposure to an agent. The signs of gastric dyspepsia: loss of appetite, unpleasant/bad taste in the mouth, pain and heaviness in the epigastrium, excessive salivation, nausea, belching with air and food, vomiting of the stomach contents mixed with mucus and bile. Vomitus is abundant and has fetid odour. Vomiting can repeat. Dizziness and weakness, rise in temperature and soft (loose) stool are common.

In acute corrosive gastritis, intake of concentrated poisons (acids or alkali) immediately causes severe burning pain in the oral cavity, in the esophagus and the stomach. Persistent vomiting brings no relief. The vomitus contains mucus, blood, remains of mucous membranes. Traces of chemical burns are seen on examination of the oral cavity. Painful shock

and hemolysis develop. Necrosis of the whole thickness of the stomach wall with subsequent perforation and peritonitis are possible.

The main symptoms of acute phlegmonous gastritis include high fever, chills, vomiting, pain in the epigastric area. Patient's condition deteriorates rapidly. Vomitus may contain pus. Local peritonitis develops. Prognosis is poor.

Chronic gastritis **(CG)** is a chronic inflammation of the stomach mucus membrane with its restructuring and progressive atrophy, impaired secretion, motor and endocrine functions. Clinical picture of CG in the acute phase is characterized by symptoms of gastric and intestinal dyspepsia, pain in the epigastric area, usually of moderate intensity. An important feature of the CG is a variety of motor and evacuation disorders. CG rarely affects the patient's general condition. Pathological symptoms are not usually detected. The tongue is coated, the abdomen is soft. Deep palpation can reveal moderate diffuse tenderness in the epigastrium. ***Gastritis can be reliably diagnosed only on the basis of the results of histological examination of targeted mucus membrane biopsy material!*** Morphological (histological) changes are not only the criteria for diagnosis but also the severity of chronic gastritis.

Features of clinical manifestations of CG depend, above all, on the acid secreting stomach function. Antral localization, association with *HP* infection and young age of patients are common in chronic gastritis with persistant or increased acid secretion. It manifests by "late" pains, heartburn, acidic/sour belching, a tendency to constipation. Endoscopically, submucosal hemorrhages, erosion, hyperplasia of folds can be often seen against a background of hyperemia and edema of the mucous membrane. Motor disorders are characterized by stasis in the antral part and spasm of the pylorus. CG with low gastric secretion which is most often of autoimmune etiology is characterized by localization in the body of the stomach, marked atrophy of the mucous membrane and patient's old age. A feeling of fullness and heaviness in the epigastrium is present. Pain is of dull character, they appear after meal ("early" pain). Such signs as poor appetite, nausea, belching with air, tendency to diarrhea, diarrhea after taking milk are revealed. Endoscopic examination reveals pallor and thinning of the mucous membrane whereas histological one reveals atrophy of the glandular epithelium. The disease is often of a family nature, combined with a number of autoimmune endocrinopathies, i.e Addison's disease, Hashimo-

to's thyroiditis, diabetes mellitus type 1. Progressive atrophy of the glands is accompanied by a sharp reduction in the secretion of hydrochloric acid, pepsinogen I and Castle's factor. B12-deficient anemia manifests by fatigue, drowsiness, a burning tongue.

Specific forms of gastritis. Specific forms include: hypertrophic gastritis (Ménétrier's disease) which is a rare condition with considerable hypertrophy of the stomach mucus membrane folds, accompanied by anorexia, diarrhea, loss of weight, hypoalbumianemic edema; granulomatous gastritis; lymphocytic gastritis, eosinophilic gastritis, reactive gastritis.

3.4.2. Peptic ulcer: definition, etiology, classification, clinic.

Ulcer disease (UD) or peptic ulcer is a chronic relapsing disease characterized by exacerbations and complications. Its morphological substrate is ulcerative defect of the stomach and/or duodenum mucous membranes. According to various authors, about 6-10% of the population in economically developed countries have ulcer.

Etiology and pathogenesis of UD. Currently, it is widely recognized that UD is a polyetiological disease. It is difficult to point out the leading factor that explains all cases of UD among many others. Various exo- and endogenous factors can play etiological role in their dynamic interaction. A number of specific genetic factors underlying hereditary predisposition to UD have been identified: blood group 0 (1) in ABO system, congenital deficiency of α1-antitrypsin and β2-macroglobulin, which normally provide protection of the stomach and duodenum from aggression of acid and peptic factors, increased blood I pepsinogen as well as in serum and erythrocyte cholinesterase, lack of alkaline phosphatase intestine component in blood, reduction in secretory IgA and the presence of histocompatibility HLA antigens – B5, B15, B35. Psycho-emotional, psychosocial, communicable, nutritional factors etc may also contribute. Bad habits contributing to the development of UD, above all, include smoking and alcohol abuse. UD is twice often in smokers than in non-smokers. It has been clinically and experimentally proved that a number of drugs (aspirin and other nonsteroidal anti-inflammatory drugs, reserpine, glucocorticoids, etc.) can sometimes cause ulceration of the stomach or duodenum in individuals with genetic predisposition. Special attention should be paid to *infectious theory of UD*. Description of *Helicobacter pylori* (HP) in 1983 by B. Marshall and J. Warren and clarification of its role in the development of chron-

ic antral gastritis have been one of the major achievements of scientific research in the field of gastroenterology over the past decades. Nowadays, appearance and recurrence of nearly 100% of duodenal ulcers and more than 70% of stomach ulcers are associated with *Helicobacter pylori* infection of the stomach mucous membrane. Recently, the role of *HP* in the development of nonulcerous dyspepsia and gastric lymphoma has also been proved. Among the mechanisms involving *Helicobacter pylori* and leading to the development of diseases are: toxins and toxic enzymes, inflammation stimulation, changes in physiological functions. *HP* transmission from person to person is the most likely transmission way of infection.

From the modern point of view, ulcerative disease pathogenesis is a result of imbalance between aggression of gastric juice and protection of the stomach and duodenum. Aggressive factors that lead to stomach ulcer are hydrochloric acid and pepsin of gastric juice as well as to mechanical, thermal and chemical lesion of the mucosa. Bile salts, even in small concentrations impair secretion of bicarbonate and can cause impairment of the integrity of the epithelial layer. Protective factors include gastric mucus secretion of alkaline bicarbonate, adequate microcirculation, regeneration of cellular elements.

Clinical syndromes of UD. Clinical picture of exacerbation is characterized by ulcerative pain and dyspeptic syndromes. Pain in UD has a clear rhythmic character (time of onset and association with the meal), frequency (alternating periods of presence of pain and its absence) and seasonal character. The nature of pain may change when taking food or antacids as well as after vomiting. Clinical manifestations of UD, to some extent, depend on the age and sex of the patient. So, juvenile ulcers are characterized by marked pain syndrome, high acidity, frequent relapses. UD in the elderly and senile age goes with moderate pain syndrome but marked peptic disorders – nausea and vomiting.

UD of the stomach body is characterized by early pain in the epigastric area, sometimes on the left of the midline, belching with food, nausea. Appetite is usually present, less frequently it is reduced due to fear of provoking pain. Belching with rotten taste and vomiting often indicate impairment of evacuation of the stomach contents as a result of spasm or inflammatory swelling in the pyloroduodenal area. The tongue is coated (it is of grayish-white color). Pain in the epigastric area is revealed on palpation.

UD of the duodenum: late or night pain that become less after meal, especially milk, with localization in the pyloroduodenal area and radiating to the back; persistent heartburn, sour/acidic belching, vomiting with sour/acidic content, which occurs at pain maximum and brings a considerable relief to the patient. There is tendency to constipation. The tongue is clean. Tenderness is revealed in the epigastrium to the right.

Diagnosis of UD is based on a comprehensive assessment of the patient's complaints and objective research data. A thorough analysis of clinical symptoms of ulcerative disease makes it possible to suspect a disease and to carry out a focused examination of the patient. The most reliable method to confirm or rule out a diagnosis of UD is esophagogastroduodenoscopy (EGD), which has a high resolution, allowing you to detect UD not less than in 95% of cases. Endoscopic gastric and duodenal ulcerative disease criteria are the presence of acute or chronic ulcers, linear or stellate scars of "red" or "white" color, scarring and ulcerative bulb deformation of the duodenum or the stomach. In gastric ulcer, especially newly diagnosed, as well as in the lasting non-scarring and giant ulcers, regardless of localization, esophagogastroduodenoscopy (EGD) should be carried out with target biopsy from the margins and bottom (to exclude malignancy and to assess the degree of inflammation, atrophy or intestinal metaplasia of the stomach mucous membrane). Radiological method of examination has now lost its popularity compared to endoscopy. But it is certainly significant in the assessment of tone and motor-evacuation function of the stomach and duodenum, in estimating the extent of pyloric stenosis as well as in cases where there are relative contraindications for gastroduodenoscopy. X-ray examination is indispensable in the diagnosis of ulcer perforations (gas detection under the diaphragm). A deepening of peptic ulcer niche and low motility of the stomach wall are revealed on penetration.

To identify *Helicobacter pylori* (*HP)* in the stomach mucous membrane, a variety of different methods are used. "Gold standard" in the diagnosis of *Helicobacter pylori* (*HP)* is culture and histological examination of stomach samples. Tests based on the study of urease activity of *HP* (CLO test, CU test, De-NOL test, breathing test), cytological stomach smears, immunological tests are also widely used.

Complications of peptic ulcers

Gastrointestinal bleeding (GB) is one of the most frequent and dangerous complications of UD occuring in 10-15% of patients. Duodenal ulcers

bleed more often than stomach ones. In most cases, gastrointestinal bleeding develops during exacerbation of UD (characterized by the reduction or disappearance of typical pains), but it may be the first sign of the disease in some patients. The clinical picture is made on the basis of data on the rate of bleeding and the amount of blood loss. In mild bleeding, when blood loss does not exceed 350-400 ml, subjective feelings are limited to mild transient nausea, dry mouth, weakness, chills. Blood loss within the range of 10% of the blood circulation volume is easily tolerated by the human organism due to mechanisms of self-regulation and compensation. In case of profuse instant blood loss or repeated bleeding, symptoms of acute post-hemorrhagic anemia appear, i.e. sudden weakness, nausea, sweating, palpitations, dizziness and fainting. The patient turns pale. The skin becomes moist. Pulse is frequent and of small filling. Systolic blood pressure decreases.

Classical signs of GB are: vomiting with fresh blood or gastric contents color of coffee grounds and black tarry stools, melena, which occurs when the bowel evacuation is not less than 80 ml of blood, thirst, dryness in the mouth, dizziness. In vomiting with blood, the source of bleeding often is located in the stomach. Melena is more typical of duodenal bleeding. Tachycardia, decreased blood pressure, fainting and cold sweat with the loss of more than 500 ml of blood develop. More than 1,500 ml of blood loss can lead to collapse and hypovolemic shock. The criterion of bleeding severity of is anemia developing by the end of the first day. Positive Gregersen's reaction persists for 2 weeks. Melena persists from 3 to 5 days.

Perforation of the ulcer in the free abdomen area is often preceded by physical exertion, neuro-mental stress, alcohol, overfilling of the stomach with food. Clinical picture of perforation often develops in exacerbation of ulcer. Increase in pain intensity, appearance of subfebrile temperature, chills, nausea, vomiting precede perforation. The most characteristic symptoms of perforation are suddenly appearing "knife-like" pain that radiates across the abdomen, wooden tension of abdominal muscles, disappearance of liver dullness, dry tongue, dramatically positive peritoneal symptoms. Rapid deterioration of patient's condition till shock development is common. It can be preceded by tachycardia and decreased blood pressure. Peritonitis develops in 6-8 hours after perforation. Diagnosis of perforation becomes clear when X-ray of the abdominal wall shows some gas below the diaphragm.

Penetration is the spread of ulcer outside the walls of the stomach or duodenum into the surrounding organs and tissues. Ulcers of the back and side walls of the duodenal bulbs most often penetrate to the head of pancreas and the liver. Stomach ulcers penetrate to the body of the pancreas. Penetration is accompanied by the development of inflammatory process and the formation of fibrous adhesions between the bodies. The course of UD becomes more severe. Symptoms which are typical of the disease of the adjacent organs involved in penetration start to develop. Penetration in the pancreas is characterized by persistent pain radiating to the back. They don't go away after administration of antacid. They become worse when the patient lies on his back. The patient takes a forced position. Penetration is more common in patients with a long history of ulcer and frequently recurrent course.

Stenosis of the stomach outlet occurs in 6-15% of patients. It can be both organic (post-ulcerative scarring/cicatricial changes) and functional. It occurs when recurrent ulcers are localized in pylorus canal and the duodenal bulb. Pyloric patency is impaired by periulcerative edema and spasm of pylorus. Evident deformation of the pylorus, identified in the phase of remission of UD is of clinical importance. Clinical picture of UD exacerbation in patients with ulcerative pyloric stenosis is characterized by a feeling of fullness and pain in the epigastric area, belching with rotten food, constant vomiting that leads to exhaustion and dehydration, visible peristalsis in the stomach. The severity of symptoms depends on the degree of evacuation function. There are three degrees of pyloric stenosis: compensated, subcompensated and decompensated.

Malignancy of ulcer. Early symptoms correspond to small symptoms syndrome: loss of a daily rhythm of pain like persistent pain, anorexia (aversion to meat food, in particular), increasing weakness, constantly positive Gregersen's reaction, progressive reduction in the acidity of gastric juice. Late signs of malignancy are: persistent weight loss, progressive anemia with a significant increase in ESR. On examination, pallor and earthy hue/tinge of the skin, persistent reduction in gastric acidity, peripheral lymph nodes metastases (Virchow's metastasis) are usually revealed.

Symptomatic ulcers are sharp, often superficial and multiple ulcerations of the stomach and duodenum arising under certain extreme conditions. Symptomatic ulcers include: 1) stress ulcer in common burns

(Curling's ulcers), head injuries, hemorrhages in the brain, neurosurgical operations (Cushing's ulcer), myocardial infarct, sepsis, severe injuries and abdominal operations as well as in critical conditions in patients with severe multiple organ failure; 2) medicinal ulcers that occur when taking certain medications, e.g. nonsteroidal anti-inflammatory, corticosteroi etc.; 3) endocrine ulcers developing during Zollinger-Ellison syndrome and hyperparathyroidism.

3.5. Semiotics of liver diseases

3.5.1. Complaints. Numerous complaints of patients can be divided into hepatic and extrahepatic. The main liver complaints are:

- pain in the right hypochondrium;
- hepatic dyspepsia;
- itchy skin;
- change in color of the skin, urine, feces;
- profuse vomiting blood;
- bloating.

The main non-hepatic complaints are complaints about increased bleeding, hormonal disorders, exhaustion.

Pain in liver diseases is caused by irritation or stretching of hepatic capsules or inflammation of the liver or the peritoneum covering the liver (perihepatitis). Enlargement of the liver, leading to extension of the hepatic capsule is observed in hepatitis, venous stasis in the liver (with right ventricle heart insufficiency). In liver cirrhosis, pain syndrome is most often absent. Liver disease is characterized by moderate, prolonged pain in the right hypochondrium. Pain radiation up to the right and back is typical of liver pathology (right shoulder, scapula, lumbar region).

Pain in pathology of bile ducts system is localized in the right hypochondrium and the gall bladder. They are caused by inflammation, stretching or necrosis of the gall bladder, sphincter muscles spasm. Bile duct diseases are also characterized by pain radiating up to the right and back (along the right diaphragmatic nerve). Calculus-free cholecystitis and hypomotor dyskinesia of the Oddi's sphincter are characterized by prolonged pain, usually of moderate intensity. Biliary colic is acute pain which occurs suddenly and quickly. It becomes extremely severe and untolerable. It can be triggered by taking excessive amount of fatty food, psycho-emotional

stress, physical stress. It is common in gallstone disease (GSD), hypermotor dyskinesia of the Oddi's sphincter.

Hepatic dyspepsia as a complex of symptoms includes decreased appetite (intolerance of fatty foods), bloating, fullness and heaviness in the area of the right hypochondrium, bad, often bitter taste in the mouth, belching with air or bitter taste, nausea, vomiting, bloating and stomach borborygmi, unstable stool. These complaints are due to the disorders of bile secretion and, consequently, the digestion of fats in the gut as well as detoxifying liver dysfunction, reflex changes in gastric secretion and motility disorders of the intestine. Nausea and vomiting of reflex origin can be observed during the attack of biliary colic.

Changes in skin color, urine and feces are clinical manifestations of hyperbilirubinemia. Jaundice is yellow coloration of the skin, mucous membranes and sclera. It is one of the most common signs of liver pathology. To recognize the nature of jaundice, it is important to find out the urine color (dark colour, colour of beer) and feces.

A frequent symptom of liver pathology is itchy skin, the occurrence of which is due to the accumulation of excess bile acids in the blood. Profuse hematemesis is caused by a rupture of the esophageal varicose veins of the lower third of the esophagus in which many anastomoses develop in case of liver cirrhosis. Enlargement of the abdomen in patients with liver diseases is usually associated with ascites or flatulence.

Patients with liver and bile duct diseases may have a lot of general complaints, which can be grouped under single/separate syndromes:

- Asthenic-neurotic – unmotivated weakness, fatigue, irritability, headache, sleep disturbance, decreased physical and mental performance.
- Arthritic – arthralgia, clinical picture of reactive arthritis.
- Subfebrile – prolonged subfebrile periods.
- Encephalopathic – loss of memory and cognitive function, drowsiness, inadequate behavior.
- Cholecysto-cardiac – long dull pain in the cardiac area, beginning after a hearty meal, extrasystoles, depression or inversion of the T wave on ECG.
- Neurosis-like – anxiety, depression, hypochondria, withdrawal, suspiciousness, aggressiveness.
- Hypothalamic – chills, frequent tremor, arterial hypertension, symptoms of angina pectoris, tachycardia, muscle weakness.

5.5.2. Value of history. Whe examining patients with liver disease, constant etiological alertness is required because these algorithms are directly connected with the treatment of patients. Household history: nature of nutrition is of certain significance. Excessive consumption of fats can contribute to the development of cholelithiasis, fatty liver. Poisoning with poisonous mushrooms (gyromitra, amanita muscaria) containing powerful poisons etc. History of working conditions: neuro-mental stress, contact with dichloroethane, compounds of phosphorus, copper, lead, arsenic, beryllium etc. (chronic hepatitis). Past medical history: acute viral hepatitis (chronic hepatitis and cirrhosis), lambliasis/giardiasis (chronic cholecystitis), leptospirosis. Hereditary predisposition is typical of peculiar diseases such as Gilbert's disease, hemochromatosis etc. Medication history (paracetamol, isoniazid, isoniazid, oral contraceptives etc.) is actively studied. Alcohol history: amount and duration of systematic use of alcohol (alcoholic liver disease) is of importance.

3.5.3. General examination of the patient with liver pathology helps to identify a number of characteristic signs. The general condition of the patient can vary from satisfactory to very heavy (decompensated cirrhosis). In liver decompensation, there may be impaired consciousness in the form of marked euphoria or its depression to total loss of consciousness (hepatic coma). Considerable weight loss, sometimes with the development of cachexia, occurs during cirrhosis and malignant tumors of the liver and bile duct.

When examining the skin and mucous membranes, jaundice is of the highest value for diagnosis. It can be of varying intensity and shades: lemon yellow (hemolytic), yellow-red (parenchymal), jaundice with green or even black shade (cholestasis). At first, it is noted on the eyes (sclera), the lower surface of the tongue and the soft palate, then on the palms, soles and, finally, the entire skin. Inspection of the sclera helps to differentiate true (bilirubin) jaundice from exogenous one. It is known that long use of some drugs, carotene (carrots), eating tangerines and oranges in large quantities, working with trinitrotoluol and some acids can cause yellow color ("false" jaundice), but staining of the sclera never occurs. In some cases, the patient may be pale (due to bleeding in portal cirrhosis). Grey-brown or brown color is typical of hemochromatosis (pigment cirrhosis). It is a disease associated with primary or secondary increase in iron intake in the gut and hemosiderin accumulation in different organs and tissues, particularly in the liver and the pancreas.

Examination of the patient's skin (especially with mechanical, rarely with parenchymal jaundice) can reveal traces of scratching due to intense itching. Often scratches are subjected to infection and suppuration. If there is impairment of cholesterol exchange in patients with cirrhosis of the liver, intradermal deposit of cholesterol occurs in the form of yellow plaques, which are especially common on the eyelids (xanthelasma), less often on hands, elbows and feet (xanthoma).

In patients with hepatic failure/insufficiency, the so-called "inconsiderable liver signs" are revealed. Liver palms, or palmar erythema, are symmetric diffuse redness of the skin in the area of thenar and hypothenar. Vascular asterisks are dilation of single skin vessels 2-5 mm in size, in the area of the shoulder girdle (pulsation of vascular asterisks is seen in the place of pressing a glass sample). Gynecomastia is revealed in men, i.e. breast enlargement caused by excess estrogen. In addition, signs of hemorrhagic diathesis (bleeding points, or petechiae) are often detected.

3.5.4. Examination of the abdomen is done in a vertical and horizontal position of the patient. The enlargement of the whole abdomen is observed on accumulation of fluid in the abdomen, i.e. ascite (in liver cirrhosis with portal hypertension). When being examined in an upright position, the patient's abdomen looks flabby as if liquid is flowing down. In a horizontal position, the abdomen looks spread, with its lateral divisions bulged (a "frog's belly"). When examining the patient with ascite in the upright position, a bulging navel above the surface of abdomen can be seen (symptom of a bell) due to increased intra-abdominal pressure. Examination of the abdomen may reveal one more important sign of portal hypertension – the presence of dilated venous network on the anterior abdominal wall (a symptom of "Medusa's head").

In case of enlargement of the liver and marked exhaustion of the patient, right hypochondrium protrusion and that of the epigastric area can be seen. If the abdominal wall is thin, sometimes it may be noted that the area is irregular, bumpy (in tumors and cysts of the liver). The gall bladder can push the abdominal wall only when there is its considerable enlargement, especially in malnourished patients (in dropsy of the gall bladder, common bile duct cancer and cancer of the pancreatic head, pressing the common bile duct (Courvoisier's sign/symptom). In case of considerable spleen enlargement (splenomegaly) and accompanying cirrhosis (hepatolienal syndrome), there is bulging in the area of the left hypochondrium.

3.5.5. Palpation of the liver and gall bladder. Shape, consistency, nature, and pain are determined by palpation of the lower border of the liver. In case of liver enlargement or its ptosis, the front surface of the right liver lobe along the midclavicular line can be felt and its consistency, the nature of the surface and tendeness can be assessed. Some results of palpation of the liver are listed below:

- A sharp border of the liver points to cirrhosis, echinococcus, muscular dystrophy;
- The rounded border of the liver points to blood stasis, amyloidosis;
- The irregular bumpy border points to liver cancer, cirrhosis;
- Dense consistency of the liver borders and surface points to cirrhosis, liver cancer, leukemia, amyloidosis;
- Soft consistency points to muscular dystrophy, venous stasis;
- Painless liver border points to cirrhosis, fatty degeneration, amyloidosis.
- Painful liver border points to hepatitis, liver abscess, venous congestion.
- In echinococcus, single painless elastic swelling on the smooth surface is felt.
- Normally, the gall bladder is not palpated. In diseases of the gall bladder and bile ducts, pain is localized in the right hypochondria, at the gall bladder point (intersection of the outer margin of the right rectus abdominal muscle and the right costal arch). The following symptoms are pathognomonomic in bile duct pathology:
- Kehr's symptom – pain on deep palpation at the gall bladder point;
- Murphy's symptom – sharp pain on palpation at the gall bladder point that occurs on exalation at maximum height;
- Ortner's symptom – sharp tenderness on palpation on tapping the right costal arch with the edge of the palm;
- Mussy'symptom – tenderness on pressing between the crus/leg of the sternocleidomastoid muscle on the right side (right-sided phrenicus symptom);
- The points of the skin hyperesthesia in the right shoulder as well as in the paravertebral points on the right at the level 7^{th} –11^{th} thoracic vertebrae and in the right angle of the scapula.

3.5.6. Percussion of the liver. Liver size can be conveniently and fairly precisely determined by Kurlov's method. On percussion, three liver sizes are determined. Size 1 along the midclavicular line is normally 9±1cm. It corresponds to the maximum diameter of the right liver lobe. Size 2 along the median line is 8±1 cm. Size 3 is determined percussing the left costal arc. It is 7±1 cm and corresponds to the length of the left liver lobe (see Table 13).

Percussion of the abdomen also detects the presence of free fluid in the abdomen (ascites). It is possible when there are at least 1.5 - 2 liters in the abdomen. In vertical position of the patient, dullness is determined in the lower abdomen. Above it, there is tympanitis. When the patient lies on his back, dullness is determined on the abdominal sides. If you turn the patient laterally, there is a change in limit of a dull percussive sound (caused by the movement of fluid in the abdomen).

Table 13

Causes of change in the liver borders

Border	Shift	Causes
Lower	Down	Enlargement of the liver in hepatitis, fatty liver, congestive venous blood Displacement of the non-enlarged liver – visceroptosis, lung emphysema
	Up	Reduction in liver sizes (cirrhosis) High standing of the diaphragm
Upper	Down	Low standing of the diaphragm – visceroptosis, lung emphysema, right-sided hydropneumothorax
	Up	High standing of the diaphragm Shrinkage of the lower border of right lung Lliver cancer, liver echinococcus

3.5.7. Percussion and palpation of the spleen.

The spleen is located deeply in the left hypochondrium, more lateral to the stomach. Percussion and palpation of the spleen are better carried out when the patient lies on the right side. The spleen is projected onto the left side surface of the thorax between the ninth and eleventh ribs. Spleen length corresponds to the movement of the 10^{th} rib. Normally, it is 6-8 cm. The width of the spleen is detected by percussing the line located in the middle of the length (most commonly, this is linea axillaris media). Normally, the width of the spleen is 4-6 cm.

The spleen is also palpated using the position as was described by Sali, with the hand moving along the left costal arch and slightly pressing the left hypochondrium. Normally, the spleen is not palpated. Spleen enlargement occurs in blood diseases, sepsis, cirrhosis of the liver, in certain acute infectious diseases, malaria.

3.6. Basic clinical and laboratory syndromes of liver diseases

3.6.1. Jaundice syndrome

Jaundice (icterus) is one of the main symptoms with diseases of liver and bile ducts. Jaundice is the result of deposit of bile pigment, bilirubin, in the skin and mucous membranes (see Table 14). Bilirubin is formed from blood hemoglobin and is transported by serum albumin (indirect, or unconjugated, bilirubin). The next stage in bilirubin exchage is its conjugation with glucuronic acid in the liver cells (direct conjugated bilirubin). Then, glucuronide bilirubin complex penetrates into the bile ducts and is secreted into the intestines. In the intestine, under the influence of microflora, bilirubin is restored to urobilinogen (gives urine the colour) and stercobilinogen (gives feces the colour).

Table 14

Differential diagnostic signs jaundices

Symptom	Haemolytic (suprahepatic)	Parenchymal (hepatocellular)	Obstructive (subhepatic)
Skin colour	Pale yellow, lemon	Orange, bright yellow	Olive, to black and yellow
Itchy skin	Absent	Occasional moderate	Marked
Direct blood bilirubin	+	+++	++++
Indirect blood bilirubin	++++	+++	+
Urine urobilin	++++	+++	+-
Urine bilirubin	-	++++	++++
Stercobilin	++++	+	-
Additional data	Anemia, reticulocytosis, splenomegaly, hypercholic feces	Urine is yellow-green, hypocholic feces, Asp-AT, ALa-AT ↑↑	Urine of beer colour , acholic feces, steatorrhea, AP ↑↑

Impairments of individual levels of bilirubin exchange cause hyperbilirubinemia. When bilirubin is over 34.2 μmol/L (within normal limits 8.5 – 20.5), yellowish skin, sclera and mucous colouring develop.

Hyperbilirubinemia may occur:

1. Due to increased bilirubin formation in case of excessive hemolysis in hemolytic anemia, massive hemorrhage, suprahepatic (in infarctions) or hemolytic jaundice.

2. Due to direct damage to the hepatic tissue and liver detoxification dysfunction, i.e. parenchymal, or liver (hepatocellular) jaundice.

3. Subhepatic, or mechanical (obstructive) jaundice, develops due to impairment of bile secretion or passage through the intrahepatic or extrahepatic bile ducts.

3.6.2. Portal hypertension syndrome

Liver circulatory system comprises two donating vessels – the portal vein (70-80% of blood) and the hepatic artery, and one carrying vessel – the hepatic vein. Impairment of the outflow through the blood vessels of the portal vein system leads to the development of portal hypertension (normal pressure in the portal vein is 50-115 mm water column). In cirrhosis of the liver, proliferation of the connective tissue at the site of dead liver cells leads to the narrowing or complete obliteration of the part of the liver sinusoids and intrahepatic vessels. Intrahepatic form of portal hypertension develops. In thrombosis or stenosis of the hepatic vein or inferior vena cava, suprahepatic form of portal hypertension develops (Budd-Chiari's syndrome). On squeezing the portal vein or its major branches (splenic vein) by tumors by the enlarged lymph nodes, subhepatic portal hypertension develops.

Clinical manifestations of portal hypertension syndrome are development of portal-caval anastomoses, swelling and ascitic syndrome, hepatolienal syndrome and hepatic (shunt) encephalopathy.

Hepatic portal vein has many anastomoses with the lower and upper hollow veins, expansion of which occurs when portal pressure increases. The following anastomoses are of diagnostic value:

1) in the area of esophageal-gastric plexus – bypass pathway via the left gastric vein, the esophageal plexus and semipair vein into the upper hollow vein. In case of marked portal hypertension, varicose venous nodes appear in the lower part of the esophagus. If they are severely

injured, for example, by food of dense consistency, bleeding may occur in the form of bloody vomiting (a terrible complication of diseases associated with portal hypertension syndrome, which is often the cause of death).

2) between the periumbilical veins and veins of the anterior abdominal wall and the diaphragm carrying blood in the upper and lower hollow vein. Varices around the navel in portal hypertension are dilated and diverge in different directions. They form the original picture called "Medusa's" (caput Medusae).

3) in the area of hemorrhoidal venous plexus – between the lower mesentry veins (portal vein system) and hemorrhoidal veins going into the lower hollow vein. In portal hypertension, hemorrhoidal nodes appear. Their rupture often causes bleeding from the rectum.

Formation of swelling and ascitic syndrome in the portal hypertension in patients with cirrhosis of the liver is due to increased hydrostatic pressure in sinusoids and venules of the liver, decreased oncotic plasma pressure due to hypoproteinemia and also developing sodium and water retention because of insufficient inactivation of angiotensin and aldosterone in the liver.

Blood stasis in the portal vein often leads to the development of hepatolienal syndrome: spleen enlargement (splenomegaly) which may be accompanied by enhancement in its function – hypersplenism. In splenomegaly, patients complain of heaviness and pain in the left hypochondrium. Hypersplenism manifests by thrombocytopenia, leukopenia and moderate anemia. These changes are due to the fact that, as a result of excessively intense activity of the spleen, inhibition of bone marrow blood cell production occurs. Thrombocytopenia often leads to bleeding complications, whereas leukopenia leads to secondary infections.

Under conditions of blood shunting through portcaval anastomoses, detoxifying liver function is absent and shunt hepatic encephalopathy develops, which manifests by various neuro-psychiatric disorders, i.e. increasing muscle tone, pronounced/enhanced reflexes, ataxia as well as psychotic disorders – irritability, euphoria, psychoses, hallucinations etc.

3.6.3. Serum-biochemical syndromes in liver pathology

Syndrome of cytolysis is due to damage to liver cells with marked impairment of the hepatocytic membrane (see Table 15). Syndrome of cytolysis

refers to the basic indicators of pathological process activity in the liver. It is characterized by increased serum concentrations of the following enzymes:

- Aspartate aminotransferase (Asp-AT) – normal to 40 units; 0.1 – 0.45μmol/ (h * L),
- Alanine aminotransferase (ALa-AT) – normal to 40 units; 0.1 –0.68 μmol/(h * L)
- Gamma-glutamyl-transpeptidase (GGTP) – normal to 105 units for men, to 65 units for women; 0.6 – 3.96 μmol/(h * L).

It must be remembered that hyperfermentemia can develop not only in liver pathology, but also in heart disease, myositis etc. The most remarkable and persistent hyperfermentemia is observed in acute viral hepatitis. The highest level of GGTP is observed in alcoholic liver damage.

Mesenchymal inflammatory syndrome is due to increased activity of mesenchymal stromal liver elements. Sedimentation tests are used for its diagnosis. They are thymol (norm 0 – 7 units.) and sulema (norm 1.9 units and more) as well as indicators of γ-globulin serum (norm 8 – 17 g/L or 14 – 21% of the total protein amount). Thymol test is the most informative in acute viral hepatitis whereas sulema one is informative in liver cirrhosis. Considerably high hypergammaglobulinemia is observed in autoimmune processes in the liver.

Cholestasis syndrome is associated with secretion and circulation of bile. It is observed not only in mechanical obstacles, but in hormonal changes (cholestasis of pregnancy). Main indicators of cholestatic syndrome are: serum alkaline phosphatase (norm 50 – 120 U, or 139 – 360 nmol/s*L, 1 – 3 mmol/(h * L)), direct bilirubin (norm is not more than 25% of the total, i.e. not more than 5 μmol/L), gamma-glutamyl transpeptidase.

Hepato-depressive syndrome (small liver failure) manifests in synthetic and detoxifying liver dysfunction without encephalopathy. The indicators include:

The synthetic function of the liver is estimated according to the level of total protein and albumin (35-50 g/L) of blood serum as well as on the level of prothrombin index (80-110%) and the content of cholesterol.

Loading test: bromsulfophthalein according to Rosenthal-White (not more than 5% should remain in the serum in 45 minures after injecting a dye).

Table 15

Biochemical syndromes in liver pathology

Syndrome	Markers	Norm	Changes
Cytolytic	Asp-AT ALa-AT GGTP	7-40 units; 0.1-0.45 μmol/(h * L) to 40 units; 0.1-0.68 μmol/(h * L) 0.6-3.96 μmol/(h * L)	Increased Increased Increased
Mesenchymal inflammatory	Thymol test Sulema test γ-globulin	0-7 units 1,9 units and more 8-17 g/L	Increased Decreased Increased
Cholestatic	Serum alkaline phosphatase Direct bilirubin	50-120 U, or 139-360 nmol/s * L not more than 5 μmol/L	Increased Increased
Hepato-depressive	Serum albumin Prothrombin index Cholesterol Bromsulfophthalein sample	35-50 g/L 80-110% 3.6-6.8 mmol/L Not more than 5% should remain in the serum 45 minutes after the injection of bromsulfophthalein	Decreased Decreased Decreased Dye retention
Shunting	Serum ammonia	21.4-42.8 mmol/L	Increased

Shunting, or bypass, syndrome is the presence of a large amount of substances subjected to conversion in the liver into the general bloodstream as a result of the development of porto-caval anastomoses. Ammonia serum is the most suitable indicator for bypass syndrome (21.4-42.8 mmol/L).

Syndrome of regeneration and tumor growth. Its key indicator is α-fetaprotein (less than 10-25 ng/ml). A significant increase in the concentration of AFP (over 8) is characteristic of hepatocellular carcinoma. A moderate increase (1.5 – 4 above normal) sometimes is observed in regenerative processes in the liver in active cirrhosis or acute viral hepatitis.

3.6.4. Hepatocellular insufficiency syndrome

Complex of symptoms of liver failure is a pathological condition resulting from the profound impairments of numerous and vital liver functions accompanied by neuro-psychiatric disorders, up to the development of hepatic coma.

The leading morphological substrate of hepatocellular failure is dystrophic and necrotic changes in hepatocytes. Hepatocellular failure can be a complication of any diffuse liver damage. The risk of developing hepatocellular insufficiency and hepatic coma increases significantly with alcohol use, barbiturates, narcotic analgesics (morphine, promedol), inhalation narcosis, infections, excess food protein (which leads to processes of decay in the intestine, increased formation and absorption of toxic products into the blood), massive bleeding from the digestive tract, the use of large doses of diuretics etc.

Depending on the nature and severity of the disease, we distinguish acute and chronic liver failure. ***Acute liver failure*** develops within several hours or days in massive necrosis of the liver tissue. It is characterized by a brightly manifested and quickly escalating clinical picture. Acute liver failure occurs in severe forms of viral hepatitis, poisonings (including certain medications), excessive alcohol use. Chronic liver failure occurs in diffuse liver diseases and is characterized by slow, gradual development of clinical manifestations.

Liver failure is primarily characterized by metabolic changes: carbohydrate (disturbance of gluconeogenesis, i.e. hepatogenic hypoglycemia), fat (lower clearance of fatty acids with the development of fatty liver), protein (disturbance of protein synthesis, i.e. hypooncotic swelling, hemorrhagic syndrome), disturbance of urea formation, i.e. hyperammonemia and toxic damage to the central nervous system). Polyhypovitaminosis develops. The liver is one of the most important organs where inactivation of various hormones takes place, so, in liver pathology, a variety of endocrinopathies also develop, such as androgen deficiency in male, disturbance of reproductive function in women.

Despite the diversity of clinical manifestations of liver failure, the main criteria for assessing its severity are severity of encephalopathy, hepato-depressive and hemorrhagic syndromes.

Patients complain of decreased appetite, nausea and vomiting, increasing weakness, rapid change of mood, reduced tolerance to alcohol. “Liver signs” and jaundice are detected on examination. The main symptoms of encephalopathy are patients’ inhibition, inadequacy (euphoria or, on the contrary, mental depression), insomnia at night and drowsiness during the day, sometimes headaches, dizziness, disorientation and short mild fainting. Hemorrhagic syndrome manifests by subcutaneous bleeding, often at an injection site, gum and nasal bleeding. The first (primary) and, especially, the second (decompensated) stages of liver failure are characterized by manifestations of hepato-depressive syndrome.

The third (terminal) stage of liver failure is characterized by gradual development of ***hepatic coma*** (see Table 16). Pathogenesis of hepatic coma results to the almost complete cessation of detoxifying liver function.

Table 16

Clinical manifestations of hepatic coma

Stage	Clinical manifestations
Precoma	Marked symptoms of encephalopathy, i.e. drowsiness, inadequate behavior, disorientation in time. Retarded speech (sopor), stereotypical answers, liver smell, sweating, escalating hemorrhagic syndrome. Adynamia.
Threatening coma	Marked confusion - stupor, incoherent speech. Episodes of sleepiness can alternate with excitement. Flapping tremor of fingertips, convulsions, wandering movements of eyeballs, loss of contact with patients in adequate response to pain appear.
Complete coma	Absence of consciousness. Spontaneous movements and reaction to pain stimuli in the onset of coma may persist, and then disappear. Divergent strabismus. Absence of pupil reactions. Pathological reflexes (Babinski's, grasping, sucking). Muscle rigidity of limbs and occiput. Abnormal breathing – Kussmaul's or Cheyne-Stokes respiration.

3.6.5. Chronic hepatitis

Chronic hepatitis (CH) is a progressive inflammatory liver disease lasting more than 6 months, in which the pathological process takes place without disrupting the normal liver lobular structure. Classification of CH is based on establishing the disease etiology (see Table 17) and inflammatory activity.

Table 17

The main etiological factors of chronic hepatitis

Primary hepatotropic viruses	Inflammation is caused by hepatitis viruses B, C, D
Secondary hepatotropic viruses	Cytomegalovirus, Epstein-Barr virus, Q-fever virus
Toxic compounds	Alcohol chemical compounds
Autoimmune damage	Autoimmune hepatitis
Medicinal drugs	Salicylates, tetracycline
Genetic/metabolic disorders	Wilson's disease, α1-antitrypsin deficiency, hemochromatosis, cystic fibrosis

The main clinical manifestations of chronic hepatitis

Pain in the right subcostal area is pulling, persistent, moderate, typically radiating to the right and upward. Signs of hepatic dyspepsia are nausea, dryness and bitterness in the mouth, reduced appetite. Asthenoneurotic syndrome manifests in all patients by increased fatigue, unmotivated weakness, decreased work productivity, headaches, sleep disorders. Patients often complain of pain in the joints; characterized by prolonged subfebrile fever, especially in viral hepatitis.

Physical examination reveals jaundice, enlargement of the liver, rarely spleen. In a high degree of inflammation, symptoms of liver cell failure are revealed, i.e. hemorrhagic skin, vascular asterisks, palmar erythema. Laboratory data include those of the development of syndromes of cytolysis, mesenchymal inflammation, hyperbilirubinemia, moderately marked hepatic depression, accelarated erythrocyte sedimentation rate.

Criteria for assessing hepatitis activity: low – an increase in ALa-AT, Asp-AT to 5; moderate – with an increase in ALa-AT, Asp-AT to 5-10, high with an increase in ALa-AT, Asp-AT more than 10 times above normal.

3.6.6. Cirrhosis

Cirrhosis of the liver (CL) is a chronic progressive disease characterized by diffuse disease characterized by restructuring the lobular liver structure with the development of signs of functional insufficiency of the organ and formation of portal hypertension (see Table 18). The causes of the disease are largely the same as those causing chronic hepatitis (see Table 17). CL can develop initially, but is usually the outcome of CH. Viral infection and chronic alcohol intoxication are responsible for developing of more than 80% cases of cirrhosis.

For a long time the disease may be clinically asymptomatic (70-80%) or present under the diagnosis of CH. ***NB!*** **Clinical picture of CL is considerably different from that of CH by the signs of portal hypertension and liver cell failure without exacerbation of the disease.** Final diagnosis of CL can be established by means of needle liver biopsy, which helps clarify etiology of the disease (for example, in alcoholic or toxic damage) and determine the degree of histological process activity according to Knodell.

In the stage of compensation or subcompensation, patients complain of increased fatigue, bad tolerance of usual load, bleeding gums, dark colored

urine, bitter taste in the mouth, bloating, vascular asterisks. In the course of time, a dull pain in the right hypochondrium, nausea, weight loss, sexual disorders, jaundice, disturbances of liver function appear. Jaundice in CL (except primary biliary cirrhosis) is parenchymatous. CL is characterized by development of cytolytic, mesenchymal inflammatory and hepatodepressive laboratory syndromes. Clinical picture of decompensated CL is characterized by impairment of the portal circulation, which leads to the development of portal hypertension syndromes (splenomegaly, hypersplenism, heamorrhagic diathesis, swelling) and liver cell failure (up to the development of hepatic coma).

The main complications of portal hypertension associated with Cl are: ascites, spontaneous bacterial peritonitis, bleeding from esophageal varices. Hepatic encephalopathy may be due to both liver cell failure and shunting/bypass syndrome.

Table 18

Classification of severity of liver cirrhosis according to Child-Pugh

Indicator	Points		
	1	2	3
Ascites	No	Mild	Moderate/large
Encephalopathy	No	Mild/moderate	Marked
Bilirubin, μmol/L	< 2	2-3	> 3
Albumin, g/L	> 35	28-35	< 28
Prothrombin time, s	1-3	4-6	> 6.0
Total number of points		CL Class	
5-6		A	
7-9		B	
10-15		C	

• *Chronic viral hepatitis (CVH) and CL* are the most common forms of diffuse liver damage. CVH is the outcome of acute process in 10-12% of hepatitis B, up to 50-60% of cases of hepatitis C, in 70-90% of cases in delta virus coinfections. The disease begins unnoticeably, gradually

manifesting by repeated episodes of mild jaundice and liver enlargement. Asthenic syndrome is very typical. Pain in the liver, muscles and joints, extrahepatic signs, nausea, intolerance of fatty and fried foods are present. Hepatomegaly is detected in most patients, the liver being of moderate density, its boredr being sharp and palpation painful. There is moderate splenomegaly. Remission is accompanied by a decrease in the liver and spleen size. Clinical picture depends in many respects on the degree of activity and phase of viral infection. Exacerbation of chronic viral hepatitis is characterized by hypergammaglobulinemia, hypoalbuminemia, increases in thymol test and transaminotransferase activity (Asp-AT/ ALa-AT ratio is less 1.0). The diagnosis is confirmed by markers of hepatitis viruses. Chronic viral hepatitis may run a long latent coourse, without episodes of activity, with gradual development of CL and the onset of cirrhosis complications (encephalopathy, ascites, gastrointestinal bleeding). According to the literature data, about 50% CVH turn to the CL. Viral CL, as a rule, persists with mild activity. Fermentemia is moderate. Splenomegaly and dysproteinemia are more evident. Complaints are about weakness, pain in the right subcostal area, rapid weight loss. Jaundice appears sporadically in exacerbation process.

• *Alcoholic liver disease (ALD)* is a combination of various impairments of structure and functional ability of the body caused by prolonged systematic use of alcohol. The distinctive feature of ALD is a clear correlation of pathological changes in the organ and an alcohol dose and duration of its use. Clinical and morphological variants of ALD: adaptive alcoholic hepatomegaly, fatty liver/liver steatosis, alcoholic liver fibrosis, chronic alcoholic hepatitis, alcoholic liver cirrhosis. ALD is characterized by predominance of dyspeptic (loss of appetite, possible alternation of diarrhea and constipation, bloating) and pain syndromes. There is jaundice, severe weakness, anorexia, subfebrile fever. The objective study mostly reveals hepatomegaly, pain in the liver on palpation. Alcoholic encephalopathy can develop. Laboratory data are as follows: leukocytosis (10,000 – 26,000), accelerated ESR (40-50 mm/h), anemia, hyperbilirubinemia, transaminases moderately elevated (no more than 3-5 times above normal), Asp-AT/ALa-AT ratio is more than 1, considerable elevation in GGTP.

• *Chronic autoimmune hepatitis* more often affects young women. It is characterized by recurrent jaundice, arthralgia, fever, hepatosplenomegaly,

antibodies to smooth muscles, mitochondria, histiocytes nuclei in the blood. The following extrahepatic manifestations are characteristic: Cushing's type of face, skin striae, generalized lymphadenopathy, Hashimoto's thyroiditis, lupus/lupoid changes. This disease is characterized by rare spontaneous remissions and rapid progression in active CL. Diagnosis of disease is most likely made by a combination of the following symptoms: mostly female gender, lack of data about blood and its components transfusions in the history, recent use of some drugs or alcohol, absence of serological markers of viral hepatitis. The levels of α1-antitrypsin, copper and ceruloplasmin in blood serum are a norm variant. The levels of total globulin, gamma-globulin or immunoglobulin G exceed the norm by more than 1.5 times. Serum titers of antinuclear antibodies exceed 1:80.

• *Primary biliary cirrhosis* is a generalized idiopathic cholestatic liver disease found mainly among middle-aged women (40-60 years, 6-10 times more often than men) and is characterized by the destruction of the bile ducts. The most common clinical signs include itchy skin, dark brown skin pigmentation, jaundice, xanthelasmas, marked hypercholesterolemia. Itchy skin precedes the development of jaundice (the duration time varies from 6 months to 2 years). Itching becomes worse after warm baths and at night. Jaundice is cholestatic, skin is of dirty green color (up to black). Discoloration of feces, elevated level of AP in the blood, direct bilirubin, cholesterol, bile acids are typical. The liver and the spleen are moderately enlarged. Bile colic with fever and chills may appear. Such symptoms as "vascular asterisks" and palmar erythema are rare. In the later stages of the disease patients develop deficiency of fat-soluble vitamins. A serious complication is developing osteodystrophy, i.e. osteoporosis and osteomalacia (softening of bones). Osteoporosis is particularly noted in the spine, ribs and pelvic bones and is often accompanied by compression fractures. Life expectancy of patients with the onset of jaundice averages 2 years.

3.7. Study methods and semiotics of diseases of biliary tract

Diseases of the gall bladder and bile ducts are ones of the most common diseases of the digestive system. This pathology is imporatnt due to social (primary morbidity in most working age, high rates of temporary disability) and medical aspects (with a possible change of functional disorders into organic pathology, involvement of related organs (liver, pancreas, duodenum) in the pathological process.

3.7.1. Duodenal probing makes it possibe to separately obtain bile from the lumen of the duodenum, common bile duct, gallbladder and intrahepatic biliary ducts for subsequent biochemical and microscopic study. Normally, the contents of the duodenum consist of intestinal juice, bile, pancreatic juice, gastric juice and a small amount of mucus. Under pathological conditions, there are also some admixtures like various products accumulating in the bile ducts, the duodenum or the pancreas, so the study of duodenal contents can provide highly valuable data. For performing duodenal probing, a thin dual probe not less than 1.5 m in length is used. The probe has 3 marks: 40 cm marks the distance from the incisors to the cardia, 70 cm marks the distance to the pylorus and 80 cm marks the distance to the Vater's papilla. Probing is carried out on an empty stomach. After inserting a probe into the stomach, a patient is placed on the right side, a pad is put below the waist. Passage of oliva in the duodenum takes approximately 45-60 minutes. Oliva location can be precisely determined by X-rays.

After getting oliva in the duodenum, intestinal contents begin to pour (phase 1): clear light yellow liquid of alkaline reaction, consisting of a mixture of gastric, intestinal and pancreatic juices is portion A (20-30 ml within 20 min). The increase in portion volume (more 40-45 ml) reveals evacuation retention from the duodenum. After collecting the duodenal portion, gall bladder reflex is evoked (phase 2), i.e. gall bladder contraction reflex. 50 ml of warm 25% magnesium sulfate are poured through the probe, with a half of the injected solution being sucked off within 2 minutes. The third phase is a period of closed Oddi's sphincter (3-5 min). Shortening of the closed sphincter phase (less than 3 minutes) indicates hypotonus. If this phase is longer than 6 minutes, it points to hypertonicity of Oddi's sphincter or to the presence of organic obstacles to bile drain. After relaxation of the biliary sphincters, cystic bile discharge starts (phase 4). Portion B is of olive color, with a volume of about 50-60 ml. Within 15-20 minutes cystic bile discharge stops and clear golden yellow liquid starts to flow. This is portion C (10-30 ml within 30 minutes) coming from the intrahepatic bile ducts (phase 5).

Table 19

Standard results of duodenal probing

Indicators	Phase 1, portion A (intestinal contents)	Phase 2, stimulation of gall bladder reflex	Phase 3, the period of Oddi's sphincter closure	Phase 4, the portion in (cystic bile)	Phase 5, portion c (liver bile)
Time, min	Up to 20	2 min	3-5 min	20-30	Up to 30
Volume, ml	20-30	-	-	30-60	10-30
Density	1,008-1,012	-	-	1,016-1,034	1,007-1,010
Transparency	Opalescent	-	-	Transparent	Transparent
Color	Golden-yellow	-	-	Olive	Light lemon
Epithelium	Single	-	-		Single
Leukocytes	Single	-	-	less than10 in the field of vision	Single

Change in color of cystic bile: pale color in dysfunctions of the gall bladder (cholecystitis) or with a decrease in bilirubin secretion with bile (hepatitis, cirrhosis); very dark, almost black color occurs in pathological condensing of bile in the gall bladder (cholelithiasis). Change in transparency of bile (flakes) is observed in inflammatory processes: duodenitis (portion A), cholecystitis (portion B), cholangitis, or inflammatory process in the intrahepatic bile ducts (portion C). Gravity weight of bile points to the density of solids, especially, bilirubin. Decreased density of portion B indicates the gall bladder's inability to concentrate, increased gravity weight indicates condensing of bile. Increased number of epithelial cells and leukocytes in bile indicates the presence of an inflammatory process: portion A – the duodenum, portion B – the gallbladder, portion C – the bile ducts. On microscopic examination of bile vegetative forms of lamblia or eggs of helminths can be detected (see Table 19).

3.8. Chronic a calculous (calculus-free) cholecystitis

Chronic acalculous (calculus-free) cholecystitis is chronic polyetiological inflammatory disease of the gall bladder, usually combined with motor impairments of biliary system. Bacterial infection spreading by hematogenic and lymphogenic routes (rarely ascending from the duodenum) plays a leading role in the development of the disease. A wide variety of sources of infection include ENT diseases, dental diseases, gynecological diseases, kidney diseases and urinary tract diseases (UTD). The predisposing factors include: bile stasis (dyskinesia, pregnancy, obesity, negative emotions, lack of cellulose fibers in food), irregular diet, reflectory influence by GIS organs with inflammatory processes taking place in them, pancreatic-biliary reflux, past acute cholecystitis, intestinal dysbiosis. Clinical symptoms are pain in the right hypochondnrium, with characteristic radiation after taking alcohol, fried and fatty foods, after shaky driving and physical exertion. Pain is accompanied by nausea, vomiting, bitterness and dryness in the mouth, often heartburn and belching air and food. Examination reveals subicterus of the sclerae, tenredness on palpation in the gall bladder area, positive symptoms of irritation of gallbladder. In exacerbation, there is an inflammatory reaction of the blood (leukocytosis, accelerated erythrocyte sedimentation rate. Gastric probing reveals cystic bile of acid reaction, gravity weight is lowered and contains flakes of mucus, a large number of white blood cells, cylindrical epithelium, increased content of sialic acids and aminotranferases. Ultrasound examination reveals thickening of the gall bladder walls.

Dyskinesia of the biliary system is inconsistent contraction of the gall bladder and Oddi's, Lutkens', Mirichi's sphincters, which is due to impairment of interaction between innervation and paracrine systems coordinating the sequence of contraction and relaxation of the gall bladder and sphincters system. Diseases of bile tract are often accompanied by dyskinesias but dyskinesias can occur in the absence of organic changes in the gall bladder and extrahepatic bile ducts. ***NB!*** **The hallmark of dyskinesia is the absence of inflammation signs (fever, leukocytosis, ESR).**

Clinical manifestations are due to a type of dyskinesia. *Hyperkinetic (hypertonic) type* is characterized by sharp colicy pains in the right hypochondrium, radiating to the right shoulder blade (scapula). Pain is short. It can repeat several times per day and can be accompanied by neurological

disorders: sweating, tachycardia, hypotension, weakness, irritability. Pain syndrome can be caused by diet irregularities, psycho-emotional stress. In duodenal probing, there is prolongation of phase time of closed Oddi's sphincter and shortening of cystic bile secretion. *Hypokinetic (hypotonic) type* is characterized by a constant dull, aching pain in the right hypochondrium without clear radiation. In marked dyspepsia, nausea, belching, bitter taste in the mouth, flatulence, constipation are noted. Palpation reveals slight tenderness in the gall bladder area. In duodenal probing, there is shortening of phase of closed sphincter and an increase in the volume of gall bladder bile to 200 ml.

3.9. Research methods and semiotics of diseases of the pancreas

The main complaints of patients with diseases of the pancreas are:

- Pain syndrome
- Syndrome of dyspepsia/dyspeptic syndrome

Pancreatic pain occurs some time after meal. They are localized in the epigastrium, they are of belting character and can radiate to the back. Pain becomes worse when lying on the back, so patients take a forced position – often lying on the right side, or sitting, leaning forward.

Dyspeptic phenomen are due to impairment of normal pancreas juice secretion, low enzyme content in secretion and therefore, impaired digestion of the cavitary process (maldigestion) and development of dysbiosis. Accelerated peristalsis, bloating, borborygmi in the abdomen appear. Maldigestion leads to a decrease in body weight and the appearance of diarrhea (abundant).

Loss of appetite, aversion to meat and oily food, up to the development of pancreatic anorexia are typical. In long-term chronic pancreatitis, symptoms of secondary diabetes (polydipsia, polyuria) develop.

During the examination, the patient can have yellow color and dryness of skin, traces of scratching, weight loss. Signs of obstructive jaundice are due to the squeezing of the common bile duct by the enlarged head of the pancreas. When examining the abdomen, one can reveal atrophy of the subcutaneous fat in the projection area of the pancreas (Grott's sign). Palpation reveals pain in the epigastrium (in the area of Chauffard's and at Desjardin's point) and in the left hypochondrium (at the point of Mayo-Robson).

Laboratory diagnosis of diseases of the pancreas

Enzymatic activity study helps to determine the serum amylase content in the blood (normal value 16-30 g/h * L), lipase (normal value 22-193 u/L), trypsin (normal value 17-67 nmol/s*L). Urine amylase, or diastase, (normally up to 44 mg/L) has some advantages, i.e. the possibility of easily obtaining the material, the possibility of multiple research and detection of minor fluctuations in enzyme activity. The content of fecal elastase 1 is determined by feces examination.

Pancreatic secretion probing is the collection and study of duodenal content after stimulation of pancreatic secretion by exogenous hormones (secretin-cholecystokinine test). You can identify 3 types of pathologic pancreatic secretions: hyposecretion – normal amount of secretion, low content of enzymes (diffuse fibrosis of the pancreas in the later stages of the CP); hypersecretion – normal or increased secretions, high enzyme activity (initial stages of CP); obturation – a significant decrease in enzyme activity, normal or elevated (mechanical block).

Study of feces is carried out in accordance with the standard diet. Patients should not be prescribed any enzyme preparations. Pancreatic dyspepsia (see Table 20) is primarily characterized by steatorrhea (high content of neutral fat), to a lesser extent creatorrhea (increased content of unmodified muscle fibers) and amylorrhea (elevated amount of extra-and intracellular starch).

Table 20

Research of digestive organs in norm and pathology

Syndrome	Normal	Pancreatic dyspepsia	Hepatic dyspepsia	Gastric dyspepsia
Reaction of the medium	Weakly alkaline	Alkaline	Acidic	Alkaline
Muscle fibers	+ - Changed	+++ Without change	+ -	++ Without change
Neutral fat	-	+++	+	-
Fatty acids and soaps (sapo)	+ -	+ -	++	+ -
Starch	+ -	++	+ -	+
Digestable fibers	+ -	++	+ -	++
Connective tissue	-	-	-	++

Chronic pancreatitis (CP) is a chronic inflammatory dystrophic disease of the glandular tissue of the pancreas with impaired passage of its ducts, it causing sclerosis of parenchyma and considerable exo- and endocrine dysfunctions of the pancreas. By etiology, we distinguish primary chronic pancreatits (alcoholic, alimentary, hereditary, metabolic, idiopathic) and secondary (pathology of biliary system, hepatitis and cirrhosis, ulcers, duodenitis, etc.). Activation of pancreatic enzymes (especially trypsin) is one of the leading mechanisms in pathogenesis of chronic pancreatitis. The other pathogenetic mechanism of CP is an increase in pressure in the duodenum, spasm of Oddi's sphincter, which leads to increased pressure in the pancreatic ducts, thickening of pancreatic secretion, formation of protein plugs, glandular tissue edema, and subsequently to its atrophy and replacement by the connective tissue. Great importance in the progress of the disease is attached to autoimmune processes.

Symptoms of CP depend on the clinical form, phase and stage of the disease. In most cases, the main clinical manifestations of CP are: pain in the epigastrium and in the left hypochondrium, dyspeptic disorders, maldigestion and malabsorption syndromes. In the later stages, there are signs of secondary diabetes. Pain is mainly caused by pressure increase in the ducts of the pancreas due to poor secretion outflow. In exacerbation, the enlarged pancreas may exert pressure on the solar plexus, causing extreme pain, so that patients take a forced position. Patients restrict their food intake because of pain. Dyspeptic phenomena are pancreatic anorexia, belching with air, excessive salivation, nausea, vomiting without relief, flatulence, pancreatic diarrheas. Symptoms of obstructive jaundice may appear. Clinical picture also depends on the stage of the disease. Stage 1 is mostly pain syndrome. Stage 2 is extrasecretory dysfunction of the pancreas with the development of malabsorption and maldigestion. Stage 3 is concomitant endocrine disturbances, secondary diabetes mellitus.

UNIT 4. EXAMINATION OF THE PATIENT WITH CARDIOVASCULAR DISEASES

4.1. Subjective and objective methods of investigation

4.1.1. Complaints in diseases of the circulatory system

The main complaints pointing to the pathology of the cardiovascular system are:

- Pain or discomfort in the heart and behind the sternum
- Dyspnea
- Palpitations and interruptions in the work of the heart
- Edema
- Fainting
- Heaviness in the right subcostal area
- Cough and hemoptysis

Chest pain is one of the most frequent symptoms of major heart disease. When asking a patient, it is necessary to check:

- localization of pain
- nature and intensity of pain
- duration of pain
- radiation of pain
- conditions that cause pain
- effect of medicines.

Pain of ischemic origin is caused by impairment of coronary blood flow.

Stenocardia pain occurs in temporary myocardial ischemia. It is localized in the sternum. They are of squeezing or pressing character. In typical cases, the pain is short (not more than 15 minutes), radiating to the left and up (in the left shoulder, arm, shoulder, rarely in the left lower jaw, to the left hypochondrium). Pain is relieved by sublingual (under the tongue) nitroglycerin within 2-5 min.

In myocardial infarction, pain is more intense, accompanied by a feeling of fear, excitement, marked sweating. Pain lasts more than 30 min, but it can also last up to several hours or even days. Pain remains even after taking nitroglycerin.

Myocardial pain across the whole heart area is nagging, aching, prolonged, sometimes become worse on physical exertion. It is not relieved by

nitroglycerin, which indicates a severe diffuse lesion of the heart muscle.

In pericarditis, pain becomes more intense on deep breathing, coughing, swallowing, changing position. Pain often becomes less when a patient leans forward or lying on his abdomen.

Neurogenic pains are located in the area of the heart apex. They are of two types: sharp, short-term, of piercing character and prolonged, aching, lasting for some hours or nearly constant.

Pain in the chest caused by non-cardiac diseases in most cases is different from pain in diseases of the cardiovascular system. In diseases of the lung and pleura, pain is usually located on one lateral side of the chest cavity. It becomes more intense on breathing, coughing, body movements. Diseases of the esophagus and stomach are among the most frequent causes of acid reflux type and burning sensation, which are associated with food intake. Only in case of spasm of the esophagus, pain does not practically differ from spontaneous sternocardia. For the proper diagnosis, it is necessary to make an association between the onset of pain and food intake, pain relief in a sitting position, after taking antacids. Pain caused by lesions of the spine and the chest wall is characterized by their appearence or worsening in body movements. Tenderness is also revealed on palpation of the chest.

Shortness of breath is a subjective feeling of lack of air, accompanied by objective changes in frequency and depth of breathing rhythm. Shortness of breath caused by heart disease, usually first occurs on physical exertion. Ortopnoea (inability to be in a lying position due to shortness of breath) gradually appears with the progression of lesions of the left heart. The so-called **paroxysmal night shortness of breath** can appear later on when the patient suddenly awakes in 2-5 hours after falling asleep due to a lack of air. An attack is over when sitting or standing within 30-45 minutes. Shortness of breath in the right heart damage is not accompanied by ortopnoea. The patient can be in a lying position for a long time. Only tachypnoea and signs of venous stasis in the general blood circulation are noted.

Heartbeat is a result of subjective sensations of heart beats, but they are not always associated with the true frequency of cardiac contractions. The main cause of heart palpitations are paroxysmal and chronic tachyarrythmias. In addition, complaints about palpitations are typical of patients who have increased heart stroke volume, e.g. in anemia, aortic insufficiency, bradycardia.

Impairment of heart work can be caused by arrythmia, atrial fibrillation, 2nd degree atrioventricular or sinoatrial blocks as well as by impairments of nervous regulation of cardiac activity in neurocirculatory dystonia.

Edema is an objective sign of circulatory failure. It appears due to the slowing down of blood flow in the capillaries, increased hydrostatic pressure in the venous line of the general circulation as well as sodium and water retention (activation of renin-angiotensin-aldosterone system).

Edema of cardiac origin is characterized by symmetry. Edema is localized, depending on the body position (edema is mainly in the feet and shin area in a vertical position). Cardiac edema develops, starting from the periphery (ankles, feet, shin). At first, edema appears only in the evening and in the morning it disappears on its own.

Later on, edema starts escalaitng, with fluid accumulation in the serous cavities (abdominal, pleural, pericardial). Anasarca develops. Cardiac edema is combined with acrocyanosis ("colorful" swelling). In marked edema, there are always the other signs of circulatory failure: dilation of the neck veins and increase in the upper level pulsation of the internal jugular vein, enlargement of the liver, heart enlargement.

Fainting can be observed in such diseases as aortal stenosis, cardiomyopathy, primary pulmonary hypertension. Syncope on physical exertion is quite common in these diseases. Loss of consciousness is due to a lack of an adequate increase in cardiac output during physical exercise due to mechanical barriers to the bloodstream. Syncope caused by impairments of rhythm (Morgagni-Adams-Stokes syndrome) is characterized by complete unexpectedness and rapid recovery. Loss of consciousness can also be caused by both tachyarrhythmia and bradyarrythmia. In patients with organic heart lesion (postinfarction cardiosclerosis, myocardium hypertrophy), syncope is often caused by paroxysmal ventricular tachyarrhythmia. In patients without signs of organic heart lesion, syncope is most often caused by sudden bradyarrhythmia (sinus node weakness syndrome or 2nd – 3rd degree atrioventricular blocks) or Wolff-Parkinson-White syndrome (WPW). In WPW syndrome, loss of consciousness can occur due to an attack of atrial fibrillation with very high heart rate (up to 250-300/min and more).

The most common cause of short-term loss of consciousness is vasovagal syncope. Classical vasovagal syncope is related to stressful situation such as pain, fear etc.

Cough and hemoptysis are assotiated with impairment of blood circulation in the lungs. Cough occurs in blood stasis in the lungs (congestive bronchitis) due to the left ventricle failure, when the left ventricle becomes weakened and cannot take all the blood from the pulmonary circulation. Cough is dry, sometimes accompanied by hemoptysis (coughing blood). Coughing blood is typical of cardiac asthma. In this case, foamy sputum of a pink tint is present, which is due to congestive phenomena and increased permeability of the vessel walls. Hemoptysis is often observed in mitral stenosis, myocardial infarction, pulmonary artery thromboembolism.

4.1.2. History taking peculiarities in patients with cardiovascular disease

History of disease development. You need to ask the patient about the following: 1) when and under what circumstances the disease began, describe in detail the first symptoms of the disease; 2) dynamics of symptoms to date, dates of medical care referral; 3) past history – possible information about the results of previous studies and established diagnoses using the patient's medical records (medical certificates, extracts fom case history etc.); 4) results of past treatment.

Past history. You should pay attention to the patient's living conditions (overeating, obesity, excessive salt intake, lifestyle) and adverse working conditions (noise pollution at work, vibration, ultrasound or electromagnetic effects, constant contact with metal organic compounds, benzene etc.).

There is a need to clarify the history of gynecological anamnesis for women (first symptoms of cardiovascular disease can appear during pregnancy and childbirth).

It is necessary to pay attention to the other circumstances that may contribute to heart disease:

- past diseases (it is necessary to know if the patient has ever been affected by rheumatism, tonsillitis, diphtheria, syphilis, which are often complicated by heart disease);
- the presence of diseases accelerating the development of atherosclerosis (diabetes, hypertension);
- hereditary predisposition (heart disease in patient's relatives);
- allergic history;
- bad habits (smoking, alcohol abuse).

4.1.3. General inspection of the cardiac patient

Patients with various heart diseases often occupy a forced position:

- lie on the right side, usually avoid lying on the left because they experience discomfort in the heart area;

- progression of heart failure makes patients occupy the lying position with a high raised head rest of the bed.

In orthopnoea, shortness of breath becomes less due to the outflow of blood to the legs, reduction of stasis in pulmonary circulation and improvement of diaphragm excursion;

- in exudative pericarditis, patients occupy the knee-elbow position or sitting on leaning forward to get some relief;

- in angina, it is a position of sinking (keeping still);

- in myocardial infarction, the patient is restless, rushes, moans, keeps the hand in the heart area;

- in shock, collapse, patients' passive position is noted.

Diagnostic "Corvisart's facies" (Corvisart's face) is typical of patients with heart failure. The face is doughy/puffy, of yellow pale colour, with cyanotic lips, tip of nose, ear lobes, dull eyes, parted lips and an expression of apathy and drowsiness.

Facies mitralis is observed in mitral stenosis: marked cyanosis of the lips, cyanotic blush on the cheeks as "mitral butterflies".

When examining the skin and mucous membranes of patients with cardiovascular disease, attention is paid to a change in their color.

Cyanosis in heart disease appears on the nose, lips, hands, feet **(acrocyanosis)** and is a sign of heart failure. Later, cyanosis becomes more intense and widespread. To differentiate peripheral cyanosis from central (pulmonary), you can perform the following test: 1) inhalation of pure oxygen by the patient within 5-10 minutes leads to disappearance of central cyanosis whereas acrocyanosis does not disappear; 2) massage of the ear lobe within minutes speeds up the bloodstream and causes temporary disappearance of acrocyanosis.

Local cyanosis can be caused by impairment of local blood flow, which often occurs in thrombophlebitis of the limbs when blood flow is poor in the affected vessels.

Hyperemia of the skin is observed in patients with clearly marked hypertension, although there may be other reasons.

Pallor of the skin can be caused by insufficient blood supply of the skin blood vessels, for example, in fainting, collapse, shock, aortic heart defects ("pale heart" defects).

Jaundice in cardiac pathologies may occur as a result of bile stasis in the liver, development of cardiac liver cirrhosis.

In case of protracted septic endocarditis, jaundice can be considerable, but, if combined with the pallor (as a result of anemia associated with hemolysis of red blood cells), it gives the skin a special coloring "coffee with milk".

When examining the fingers in patients with chronic heart failure as well as in protracted septic endocarditis, characteristic changes in their shape are noted, i.e. a "clubbing" symptom and a symptom of "watch glasses".

When examining the subcutaneous adipose tissue in patients with heart disease, edema can be detected. It is one of the signs of heart failure. Edema is located in low-lying areas (on the ankles, feet, shin). Later, with the progression of heart failure it is noticed on the thighs, lower back, buttocks and in the cavity (ascites, hydrothorax, hydropericardium). Massive edema of the body is called anasarca. The limbs are cool to the touch in case of edema in heart disease. Edema in cardiac pathology in contrast to the kidney ones is usually combined with cyanosis. Skin in the swollen areas looks smooth, shiny and thin.

4.1.4. Examination of the heart and vessels

On examination of the heart and major vessels, the patient with diseases of the heart and blood vessels is presented with the following symptoms:

Heart hump (gibbus cardiacus) is bulging of the chest in the heart area associated with a significant increase in its size. Heart hump is detected if the heart became enlarged in childhood when the bones are fairly pliable.

Heart (cardiac) beat is rhythmic bulging during systole of the limited area of the chest to the left of the sternum at the level of 3^{rd} -4^{th} intercostal spaces. It is better detected on lateral inspection, with a thin chest wall. The occurrence of cardiac impulse is due to hypertrophy and dilatation of the right ventricle. It is more often detected on palpation.

Apical (apex) beat is rhythmic bulging of the limited area of the chest wall in the area of the heart apex during systole. The heart apex is a specific

point of support for the contracting heart muscle (due to the concentric arrangement of muscle fibers). During systole, it is tightly pressed to the chest wall, making the apical beat. The apex beat is detected on palpation, a visible apical beat is due to hypertrophy and dilatation of the left ventricle.

Epigastric pulsation is visible bulging of the epigastrium synchronized with heart activity. It may be due to the contraction of the right ventricle, abdominal aortic pulsation, pulsation of liver.

Dilation of the skin veins in the heart area is noted on compression of deep veins by mediastinal tumors, exudative pericarditis.

Tortuous and abruptly acting temporal arteries are observed in patients with essential hypertension and atherosclerosis.

Inspection of the neck in patients with stasis in the systemic circulation reveals swelling of the jugular veins. Positive vein pulse is noted in case of insufficiency of the tricuspid valve. Pulsation of the carotid arteries ("dance of carotids") as well as Musset's symptom (synchronously nodding of head to the beat of pulsation of the carotid artery (a. carotid) are noted in aortic valve insufficiency.

Knotty (nodular) dilatation of the shin veins is characteristic of thrombophlebitis.

4.1.5. Palpation of the heart

Objective is to detect:

- apical beat and assess its characteristics
- the presence of cardiac beat
- epigastric pulsation
- "cat's purr" symptom

Apical beat (AB) is rhythmic pulsation of the thorax in the projection of the apex of the heart that occurs at the expense of the left ventricle contraction. Normally, it is located in the 5^{th} intercostal space 1.5 cm inward from the left midclavicular line. Its square does not exceed 2 cm^2. It is of moderate strength, low and coincides with the pulse in the carotid arteries. Interpretation of data is given in Table1.

Amplitude of the apical beat is amount of pulsation area of the apical beat. Normally, it is 1-2 cm^2. It can be generalized or spread in those cases where the larger heart area due to dilatation of the left ventricle adheres to the anterior chest wall (see Table 21). Extra-cardiac causes may include

pregnancy, tumors of the posterior mediastinum, shrinkage of the lung borders. Limited (concentrated) apical beat occurs when the heart moves away backward from the anterior thoracic wall (overly developed subcutaneous tissue, lung emphysema, a low standing of the diaphragm).

Height of apical beat is detected by the amplitude of the apical beat/stroke at the examiner's fingers. It usually changes in the same direction with its width. The height of the apex beat is directly proportional to the strength of the heart and is inversely proportional to the thickness of the chest wall. On physical exertion, excitement, fever, thyrotoxic goiter and an increase in heart contraction, apical height increases. In obesity, well developed musculature and in exudative pericarditis, apical beat tends to be low.

Strength (force) of the apical beat (AB) is measured by pressure exerted by the heart apex on the palpating fingers. Like the first two properties, the force of beat depends on thickness of the chest and the proximity of the heart to the fingers, but mainly it depends on the strength of left ventricular contraction. Enhanced AB occurs in left ventricle hypertrophy whereas in concentric hypertrophy, the force increases without enhancing its amplitude.

Palpating the apical beat, in addition to its width, height and strength, you may define another property – resistance, i.e. get the idea of the density of the heart muscle. Muscle density of the left ventricle increases significantly in hypertrophy of the left ventricle. Thus, hypertrophy of the left ventricle is characterized by a high, forced, resistant apical beat.

A cardiac impulse/stroke (CI/CS) is detected in the area of the so-called absolute dullness of the heart, formed by the right ventricle. Normally, a CI/CS is not detected; only asthenic patients have barely noticeable pulsation. The appearance of a CI/CS reveals hypertrophy and dilatation of the right ventricle (mitral stenosis, tricuspid valve insufficiency, chronic pulmonary heart).

Epigastric pulsation is better detected at the height of deep breathing in when the heart located on the diaphragm goes slightly down. Epigastric pulsation caused by dilatation of the right ventricle, is determined under the xyphoid process. It increases on deep breathing in when retraction of the abdominal wall is detected. It is better noted when the patient is upright.

Pulsation of the liver can be transmitting when contractions of the hypertrophied right ventricle are transmitted to the liver, or it can be true as in tricuspid valve insuffficiency.

Table 21

Interpretation of data of heart palpation

Changes in AB data		The reasons	Diseases and syndromes
Shift of AB	To the left	Dilatation of the left ventricle	Aortic heart defects Mitral insufficiency Arterial hypertension
		Shift of the mediastinum	Hydro- or pneumothorax on right side Obturative atelectasis on the left side
	Down	Dilatation of the left ventricle	Aortic valve insufficiency Mitral insufficiency Myocarditis
	To the right	Shift of the mediastinum	Obturative atelectasis on the right side Hydro- or pneumothorax on the left side
Change in the height and strength	Forced and high	Hypertrophy of the left ventricle	Aortal heart defects Mitral insufficiency Arterial hypertension
	Weakened and low	Extra-cardiac causes	Emphysema of the lungs Obesity
Square changes	Diffuse	Dilatation of the left ventricle	Aortic heart defects Mitral insufficiency Arterial hypertension
	Concentric and resistant	Concentric hypertrophy of the left ventricle	Aortic mouth stenosis
	Systolic retraction	Adhesive layers of the pericardium	Adhesive pericarditis
	Forced and diffuse	Hypertrophy and dilatation of the right ventricle	Mitral stenosis Tricuspid valve insufficiency Cor pulmonale

The phenomenon of "cat's purr". Sometimes in the precordial area, you can palpate the so-called systolic or diastolic tremor, i.e. a symptom of "cat's purr" caused by concussion of the chest as a result of vibrations that occur with the passage of the blood through the narrowed valve openings. **Diastolic murmur** at the apex of heart occurs in the narrowing of the left atrioventricular opening (mitral stenosis) when during diastolic filling of the left ventricle, the blood from the left atrium, encountering a barrier in the area of the mitral opening, forms a turbulent flow. **Systolic murmur** in the aorta is detected in the 2nd intercostal space to the right of the sternum in case of the narrowing of the aorta mouth.

4.1.6. Study of major vessels and evaluation of heartbeat characteristicss

The ascending part of the aorta is palpated by the finger tips in the 2nd intercostal space. The pulmonary artery trunk is palpated to the left of the sternum. The aortic arch is palpated in the jugular fossa. Normally, by palpating the area of the major vessels, you can determine slight pulsation only in the jugular fossa. Increased pulsation in the jugular fossa can be associated with either an increase in pulse pressure in the aorta in aortic insufficiency, hypertensive disease or aneurysm of the aortic arch. Marked pulsation in the 2nd costal space to the left of the sternum can point to the dilation of the pulmonary artery trunk, which occurs more often as a result of pulmonary arterial hypertension.

In patients with ischemic heart disease, myocardial infarction, it is necessary to carry out an additional study to the left of the sternum at the level of 3rd – 5th ribs, where pathologically limited pulsation is often revealed in the development of left ventricular aneurysm. This should be distinguished from the pulsation of forced cardiac impulse/stroke caused by hypertrophy and dilatation of the right ventricle. In aneurism of the left ventricle, pathological pulsation to the left of the sternum is limited to this area and does not spread to the epigastric area.

Pulse is dilation of the vessels which occurs periodically and simultaneously with left ventricular systole and is visible or determined on palpation. Pulse wave propagation is due to the ability of the walls of the arteries to expand and collapse. Pulse wave velocity varies from 4 to 13 m per second and is vastly superior to the linear speed of the blood flow, which does not exceed 0.5 m per second even in the large arteries.

Pulse feeling can be done on the radial, carotid, temporal artery with 2nd, 3rd and 4th finger tips. Normally, the artery is palpable in the form of a thin elastic tube. With significant deposit of calcium salts in the walls of arteries, they are felt like tight tortuous tubes, sometimes with distinct local thickening.

First, arterial pulse is felt on both hands to reveal a possible unequal filling and pulse rate is estimated on the left and right. *Pulsus differens* is observed in unilateral obliterative diseases of major arteries and in external compression of the large arterial vessels (aortic aneurysm, tumor of the mediastinum, the expansion of the left atrium in mitral stenosis). After comparing the values of pulse on both hands, a detailed study of the pulse on the one hand, usually left, is begun.

The following features of the arterial pulse are identifed:

Pulse rate is normally equals to the number of heartbeats (60 – 90 beats per minute). On accelerating heart contractions (tachycardia) the number of pulse waves increases, accelerated/frequent pulse (tachysphygmy/pulsus frequens) appears. On slowing down (bradycardia) pulse rate becomes rare (bradysphygmy/pulsus rarus). Physiological pulse fluctuations depend on gender (women have by 7-8 beats more), age (child's pulse is more rapid), growth (pulse is rarer in tall people). Pulse rate accelerates after physical exertion, in emotional arousal, during digestion, changing the position from horizontal to vertical. Pathological tachysfigmia is observed in fever, anemia, thyrotoxicosis, alcohol intoxication, heart disease. Pathological bradysfigmia is observed in cachexia, heart conduction disorders (heart blocks), stimulation of the carotid sinus, overdose of digoxin, hypothyrodism, bile colic, kidney colic, lead intoxication. In frequent arhythmic (irregular) heart contractions, single left ventricular systoles may be so weak that the expulsion of the blood into the aorta is absent or will be so slight that pulse wave does not reach the peripheral arteries. The difference between the number of heartbeats and pulse waves, calculated per one minute, is called a pulse deficit (*pulsus deficiens*). *P. deficiens* is characteristic of atrial fibrillation. It may also occur in frequent extrasystole.

Rhythm. In a healthy person, contraction of the heart and pulse waves follow one another at regular intervals, so the pulse is rhythmical - *pulsus regularis*. In heart rhythm disorders, waves follow at different periods of time. Pulse becomes irregular - *pulsus irregularis*.

Tension of pulse is estimated by the force the examiner has to apply for the complete compression of the pulsating artery. Pulse tension depends on systolic blood pressure. Normally, pulse is of moderate tension. The higher the blood pressure, the harder it is to compress the artery. Such pulse is called tense or hard (*pulsus durus*). When pressure is low, artery is easily compressed, pulse is called soft pulse (*pulsus mollis*). Atherosclerotic arteries also give an impression of intense pulse. However, this artery is felt as dense twisted tube.

Filling of pulse reflects the amount of blood that is ejected in systole into the arterial system and causes vibrations of the walls of the arteries. Filling of pulse depends on heart stroke volume, total blood amount in the body and its distribution throughout the body. In sufficient blood supply of the artery, full pulse or *pulsus plenus* is felt. If there is a disorder of the circulatory system, massive blood loss, pulse filling is reduced/ weak (*pulsus vacuus*).

Pulse magnitude. Pulse magnitude is a concept that combines such features as tension and feeling. It depends on the degree of dilation of the artery during systole and its collapse during diastole. This, in turn, depends on pulse filling, fluctuations in blood pressure during systole and diastole and the ability of the arterial wall to elastic dilation. In case of increased blood stroke volume, in large fluctuations in blood pressure in the arteries as well as in reduction of arterial wall tonus the magnitude of pulse waves increases. This pulse is called great pulse (*pulsus magnus*). Great pulse is characterized by high amplitude, so it is also called full/high pulse (*pulsus altus*). Large/full pulse is observed in case of insufficiency of the aortic valve, in thyrotoxic goiter, when pulse wave magnitude increases due to the large difference between the systolic and diastolic blood pressure. Such pulse can appear in fever due to reduction of tonus of the arterial walls.

Decreased stroke volume, small fluctuation amplitude of pressure during systole and diastole, tonus enhancement of the artery wall lead to decreased pulse wave magnitude. Pulse becomes small (*pulsus parvus*). Small pulse is observed in small or slow blood flow in the arterial system: in narrowing of the aorta mouth or the left venous opening, tachycardia, acute cardiac insufficiency. Sometimes in shock, acute heart failure, massive blood loss pulse wave magnitude can be so slight that they are barely estimated. Such pulse is called thready pulse (*pulsus filiformis*).

Pulse form. It depends on the rate of change in pressure in the arterial system during systole and diastole. If during systole a lot of blood is pumped into the aorta and pressure in it rapidly goes up whereas it falls rapidly during diastole, a rapid dilation and collapse of the artery wall are present. This pulse is called quick /rapid pulse (*pulsus celer*), or swift pulse. Swift pulse appears in aortic insufficiency, the pulse being not only swift, but also high (*pulsus altus et celerimus*). Slow pulse (*pulsus tardus*) is associated with a slow increase in pressure in the arterial system and its low fluctuation during the cardiac cycle. Slow pulse is common in stenosis of the aorta. Wave magnitude in aortic stenosis is reduced, so pulse will not only be slow but also small (*pulsus parvus et tardus*).

After feeling the pulse on the radial artery, it is studied in the other vessels, i.e. temporal, carotid, femoral, popliteal arteries, the arteries of the rear foot etc. To estimate the pulse on various arteries is especially necessary if you suspect their lesion (obliterating endarteriitis, arteriosclerosis, thrombosis of blood vessels). Pulse of the carotid arteries should be studied very carefully because of the risk of carotid reflex, leading to a drastic slowing of the heart till cessation of its activity and a significant drop in blood pressure. Clinically, this manifests by dizziness, fainting, convulsions.

Some diseases of the cardiovascular system are characterized by different pulse magnitude on the upper and lower extremities. In the narrowing of the aortic mouth (coarctation), pulse wave magnitude is significantly reduced on the lower extremities whereas in the carotid arteries, the arteries of the upper extremity it remains normal or even increased. In Takayasu's disease ("absence of pulse" disease or pulseless disease), which is characterized by obliterating (obliterans) arteriitis of the large vessels coming from the aortic arch, it is pulsation of the carotid, axillar, brachial and radial arteries that disappears or decreases first.

4.1.7. Percussion of the heart

Objectives are to determine:

- heart size (borders of its relative and absolute dullness)
- width of the vascular bundle
- heart configuration

The heart gives a blunt sound on percussion. But because it is partly covered with the lungs laterally, dullness produced is of two kinds – relative (RHD) and absolute (AHD). The most important is to detect the bor-

ders of relative dullness of heart, i.e. the true borders of the heart, which are covered with lungs.

Detection of relative heart dullness (RHD)

The right, upper and left borders of relative heart dullness are detected successively. Normal values of the location of relative heart dullness borders and their changes in pathological conditions are given in Tables 22 and 23.

Table 22

Normal values of relative heart dullness borders

Value	Localization	The heart division
The right border of the RHD	4th intercostal space on the right, at 1 cm more laterally to the sternum border	Right atrium
The upper border of the RHD	At the level of 3rd rib on the left parasternal line line (linea parasternalis sinistra)	Left atrium
The left border of the RHD	In the 5th intercostal space , at 1.5 cm inward from the left midclavicular line	Left ventricle
The right border of AHD	In the 4th intercostals space along the left border of the sternum	Right ventricle
The upper border of AHD	At the level 4th rib along the left parasternal line (linea parasternalis sinistra)	Right ventricle
The left border of AHD	In the 5th intercostal space, at 1-1.5 cm inward from the left border of RHD	Right ventricle
The vascular bundle	2nd intercostals space, coincides with the borders of the sternum	The aorta is on the right, the pulmonary trunk is on the left.

Measurement of heart diameter. To measure the heart diameter, it is necessary to determine the distance from the right and left borders of RHD to the anterior midline. Normally, they are 3-4 cm and 8-9 cm respectively, with the heart width measuring 11- 13 cm.

Determining absolute heart dullness (AHD). The borders of absolute heart dullness are a part of the heart formed by the right ventricle. Quiet percussion is used to identify the borders of absolute heart dullness, which gives absolutely dull sound. Percussion is performed from the previously established borders of relative heart dullness towards the field of absolute dullness.

Determining the vascular bundle borders. The vascular bundle, formed by the aorta, the upper hollow vein and the pulmonary artery is detected by quiet percussion in the 2nd intercostal space. Normally, the vascular bundle borders coincide with the right and left border of the sternum. Its width does not exceed 5-6 cm.

Table 23

Changes in heart dullness borders

Changes in the heart borders		Causes	Diseases and syndromes
Displacement of the right border of RHD	To the right	Dilation of the right heart divisions	Mitral stenosis Cor pulmonale Tricuspid insufficiency
		Displacement of the mediastinum	Left-sided hydro- or pneumothorax Right side obturative atelectasis
	To the left	"Suspended" ("droplet") heart	Asthenic type of the body
		Displacement of the mediastinum	Left-sided obturative atelectasis Right-sided hydro- or pneumothorax
Displacement of the upper border of RHD	Upward	Dilatation of the left atrium	Mitral stenosis Mitral insufficiency
Displacement of the left border of AHD	To the left	Hypertrophy and dilatation of the left ventricle	Aortic insufficiency Mitral insufficiency Aortic stenosis Arterial hypertension Chronic left ventricle cardiac insufficiency (myogenic dilation)
		Displacement of the mediastinum	Right-sided hydro- or pneumothorax Left-sided obturative atelectasis
		"Lying" heart (in a lying position)	High standing of the diaphragm (ascites, flatulence, obesity)
	To the right	Displacement of the mediastinum	Right-sided obturative atelectasis Left-sided hydro- or pneumothorax

Extension of AHD	Dilatation of the right ventricule	Mitral stenosis Cor pulmonale Tricuspid insufficiency
	Extra-cardiac causes	High standing of the diaphragm Shrinking of the lung borders Tumor of the posterior mediastinum
Reduction of AHB	Extra-cardiac causes	Lung emphysema Pneumothorax "Droplet" heart (in patients with asthenic body built)
Dilation of the vascular bundle	Dilation of the ascending, descending part of the aorta or aortic arch	Arterial hypertension Atherosclerosis of the aorta Aortic aneurysm
	Dilation of the pulmonary artery	High pressure in the pulmonary artery

4.1.8. Auscultation of the heart

Rules of auscultation of the heart

1. You must follow the general rules of auscultation (in silence and in a warm room).

2. Listening is performed in the patient's upright (sitting) or supine position.

3. The examiner stands in front and to the right of the patient.

Sometimes auscultation is carried out before and after physical exertion. On physical exertion, speed of blood flow increases and heart murmurs become more distinct, making it easier to diagnose heart defects.

4. During auscultation of the heart, a patient is periodically asked to take a deep breath in and out and hold breath for 3-5 seconds.

Auscultation is performed in certain sequence. The bell of a stethoscope is consistently put at the points given below (see Table 24). Auscultation of the heart involves a sequential analysis of rhythm, tone and possible heart murmurs.

To master the heart auscultation technique, you must know the phase structures of the cardiac cycle and changes in intracardiac hemodynamics. They are given in Table 25. Normally, heart sounds ate rhythmic (follow

each other at regular intervals), heart rate ranges from 60 to 90 per min. Accelerating rhythm is called tachycardia, slowing down rhythm (less than 60 beats per min) is called bradycardia.

Table 24

Heart auscultation points

Point	Location	Valve
1	the apex beat area	mitral valve
2	the 2nd intercostal space at the right border of the sternum	aortic valve
3	the 2nd intercostal space at the left border of the sternum	valve of a pulmonary artery
4	at the base of the xiphoid process	tricuspid valve
5 (Botkin-Erb point)	point of attachment of the 3rd – 5th ribs to the left border of the sternum	aortic valve (extra point)

The rhythm of heart sounds may be disrupted due to arrhythmia.

Extraordinary heart contractions detected on auscultation followed by elongation of an interval between sounds are called extrasystole. Single extrasystoles are not always a sign of heart failure. In a healthy person, 200-400 extrasystoles can be revealed per day. Particular attention should be paid to frequent group (following one after another) extrasystoles.

Atrial fibrillation is characterized by irregular rhythm ("full" arrhythmia) with unequal volume of blood discharge in the aorta on each heart contraction.

In a healthy person, two basic heart sounds, following one another, separated by pauses are detected on auscultation.

1st heart sound (systolic) occurs at the beginning of systole. It is a total amount of sound effects including the following components:

1. Valvular component caused by vibrations of closed atrioventricular valves; valve component is particularly important for strength and volume of the 1st sound because it consists of high frequency and high amplitude vibrations.

2. Muscular component caused by vibrations in the tense muscles of the ventricles.

3. Vascular component caused by vibrations in the primary divisions of the main vessels in the early period of blood discharge.

4. Atrial component caused by vibrations of the contracting atrial muscle. With this component, in fact, the 1st sound starts because atrial systole precedes ventricular one.

Duration of the 1st sound is 0.09 -0.12 seconds.

Table 25

Structure of the cardiac cycle

Phases of the cardiac cycle		Characteristic of phase
Systole of ventricles	Asynchronous contraction phase	Distribution of electrical excitation through the myocardium, the contraction of some muscle fibres, duration 0.04-0.07 sec
	Isometric contraction phase	Fast and powerful contraction of the myocardium in fully closed valves, accompanied by a sharp increase in intraventricular pressure, duration 0.01-0.05. The beginning of 1st heart sound.
	Ejection phase	Disclosure of venous valves as a result of changes in the pressure gradient between the ventricles and the main vessels, discharge of blood from the cavities of the ventricles.
Diastole of ventricles	Protodiastolic period	Closure of the venous valves as a result of decreased pressure in the ventricles. The beginning of 2nd heart sound.
	Isometric relaxation phase	Active relaxation of the ventricular myocardium in hermetically closed valves, a gradual change in the pressure gradient between the cavities of the ventricles and atria.
	Rapid filling phase	Disclosure of the atrioventricular valves, passive filling of the ventricles with blood. The beginning of 3rd heart sound.
	Slow filling phase	Gradual equalization of pressure between the cavities of atria and ventricles, slowing down blood flow.
Atrial systole		Contraction of atria, actively squeezing blood residue to the ventricles, the gradual closing of the atrioventricular valves. The beginning of 4th heart sound.

2nd heart sound (diastolic) occurs at the beginning of diastole when due to the beginning of ventricular relaxation, pressure in them rapidly falls and becomes less than pressure in the major vessels (the aorta and the pulmonary artery). As a result, the flow of blood in these vessels rushes back, the valves close and within short time (for about 0.05 sec.) they vibrate together with the walls of the aorta and the pulmonary artery. 2nd heart sound includes vibrations of the valves and walls of the aorta and the pulmonary artery. Its duration is 0.05 -0.07 sec.

How to distinguish between 1st and 2nd heart soundson auscultation

1) 1st heart sound is a longer and lower, 2nd one is short and high;

2) 1st heart sound is separated from 2nd by a short pause and 2nd is separated from 1st by a long pause;

3) 1st heart sound is better heard at the apex (1st point of auscultation). 2nd heart sound is better heard on the aorta and the pulmonary artery (2nd and 3rd points of auscultation)

4) 1st heart sound coincides with the apex beat and pulsation of the carotid artery whereas 2nd heart sound does not.

3rd physiological heart sound occurs at the end of rapid filling phase of the ventricles in 0.12-0.18 seconds after 2nd heart sound. It is produced as a result of vibrations in the walls of the ventricles in their rapid filling with blood in the beginning of diastole. Healthy people have a very quiet, weak, low frequent 3rd physiological heart sound. It is heard with difficulty even when a patient lies on his/her left side. It can be heard in children and adolescents due to hyperkinetic circulation type.

4th physiological heart sound occurs during active atrial systole, i.e. immediately before 1st heart sound at the end of diastole. It is due to vibrations of the atrial walls. In healthy people, 4th physiological heart sound is very quiet, low frequent and is rarely heard, mainly in children and adolescents.

To properly interpret the volume changes of heart sounds, it is necessary to know factors determining the normal volume of 1st and 2nd heart sounds.

Volume of 1st heart sound normally depends on the following factors:

1) tightness of the ventricular chamber during isometric contractions, in particular on density of the AV (atrioventricular) valve closure;

2) contraction speed of the ventricles in the phase of isometric contraction, which in turn is determined by the contractile capacity of the cardiac muscle and by amount of ventricular systolic volume (the more filling with blood is, the less contraction speed is);

3) density of structures involved in vibrational motions (primarily on density of the AV valves).

Volume of 2nd heart sound normally depends on the following factors:

1) tightness of closure of the semilunar valves of the aorta and the pulmonary arteries;

2) closing and vibration speed of these valves during the protodiastolic period, which in turn depends on the level of blood pressure in the major vessels and relaxation speed of the myocardial ventricles;

3) density of structures involved in vibrational motions (primarily on density of the semilunar valves as well as the walls of the major vessels).

Sound intensity of a heart sound also depends on the conditions of sound vibrations conductivity, that is on extra-cardiac causes: chest thickness, presence of processes which make the heart distant from the front of the chest wall (lung emphysema, fluid accumulation in the left pleural cavity), improvement of sound vibrations conductivity (shrinking of the lung borders, presence of resonance when large air cavities are located near the heart (a large pulmonary cavern, an enlarged stomach gas cyst), rheological properties of the blood flowing through the heart.

Thus, a change in sound intensity of heart sounds depends on morphological cardiovascular impairmentss and changes in the surrounding tissues that affect the conductivity of sounds from the heart (see Table 26).

Diminution/weakening of the 1st heart sound at the apex of heart occurs in case of mitral valve insufficiency. During ventricular systole a portion of blood goes back to the left atrium as the valve cusps do not fully cover the atrioventricular orifice. The blood pressure on the walls of the ventricle and on the mitral valve cusps is reduced, so the valvular and musclular components of 1st heart sound are significantly diminished/weakened. In case of aortic valve insufficiency, the muscular component of 1st heart sound will also be significantly weakened.

In tricuspid valve insuffucuency, diminution/weakening of 1st heart sound is better detected at the base of the sternal xiphoid process due to diminution/weakening of the valvular and the muscular components of the right ventricle.

Diminution of 1st heart sound at the heart apex can occur in aortal mouth stenosis because its overflow in case of difficulty in emptying of the left ventricle decreases vibration amplitude of the left ventricular wall, leading to diminution/weakening of the musclular component. In diffuse myocardial lesions, the muscular component also becomes diminished/weakened.

Intensification of 1st heart sound at the heart apex occurs in reducing the blood filling of the left ventricle during diastole. Intensification of 1st heart sound is noted in the narrowing of the left atrioventricular orifice when during the diastole less than normal blood volume enters the ventricle from the atrium. So, by the beginning of systole the left ventricular muscle is less extended, more relaxed, which enables it to contract more quickly, causing intensification of 1st heart sound (see Table 26).

In right atrioventricular orifice stenosis, **amplification/intensity of 1st heart sound is heard at the base of the xiphoid process of the sternum**. It is also observed in tachycardia and extrasystole due to insufficient diastolic filling of ventricles.

Diminution/weakening of 2nd heart sound above the aorta occurs in aortic valve insufficiency due to either valve cusps destruction or to reduction of their capacity to fluctuate due to a scar consolidation. 2nd heart sound above the aorta may not be heard if the aortic valve is significantly damaged. Diminution/weakening of 2nd heart sound above the aorta is also observed with a considerable decrease in blood pressure.

Diminution/weakening of 2nd heart sound over the pulmonary trunk appears in pulmonary valve insufficiency and decreased pressure in the pulmonary circulation.

Intensification of 2nd heart sound may be noted above the aorta or the pulmonary trunk.

Emphasis/accent of 2nd heart sound on the aorta occurs when pressure in it increases (in hypertension and symptomatic arterial hypertension, on heavy physical exertion, mental excitation) because at the beginning of diastole as a result of high blood pressure in the aorta leaves of its valve close with greater force.

Emphasis/accent of 2nd heart sound on the pulmonary artery appears with an increase in pressure in the pulmonary circulation (in mitral heart defects) as well as in blood circulation in the lungs and the narrowing the bed of the pulmonary artery (in lung emphysema, pneumosclerosis).

Splitting of heart sounds is a result of unsynchronized single sound components. The main cause of splitting of 1st heart sound is unsynchronized closing and vibration of the mitral and tricuspid valves observed in block of one of the His' bundle branches. Splitting of 2nd heart sound occurs in prolongation of right ventricular systole (mitral heart defects).

Sometimes, in serious myocardial lesions (myocardial infarction, myocarditis etc.) you can hear three, but not two, heart sounds.

The "galloping" rhythm is a three-sound rhythm resembling horse gallop. Protodiastolic "galloping" rhythm (in the presence of pathological 3rd heart sound) is the result of a considerable decrease in ventricular myocardium tone in patients with heart failure, acute myocardial infarction, myocarditis and other serious lesion of the heart muscle. Resistance of the ventricular wall is low, which leads to abnormal vibration of the ventricles at the beginning of diastole.

Table 26

Change in heart sounds in pathology

Change in heart sounds	Causes	Diseases and syndromes
Diminution/ weakening of 1st heart sound	Weakness of ventricle chamber Increased diastolic and systolic volume	Mitral incufficiency
		Aortic insufficiency
		Tricuspid insufficiency
	Increased diastolic volume Decreased rate of emptying of the ventricle	Aortic stenosis
	Lesion of the heart muscle with weakened heart muscle contractility	Hypertrophy of left ventricle Diffuse myocardial lesions
Diminution/ weakening of 2nd heart sound	Loose closure of the valves	Aortic insufficiency
	Fusion of valvular leaves Reduced systolic ejection	Aortic stenosis
	Decreased blood pressure	Arterial hypotension, collapse
Diminution/ weakening of both heart sounds	Decreased myocardial contractile capacity	Myocarditis Myocardiodystrophy Diffuse and focal infarction
	Extra-cardiac causes	Obesity, hydropericardium, lung emphysema, lefd-sided pneumothorax or hydrothorax
Intensification of 1st heart sound	Reduction in filling of the ventricle during diastole	Mitral stenosis – "flapping" tone
		Complete atrioventricular block- "cannon" sound Extrasystolic contraction

Intensification of 2nd heart sound	Increased blood pressure	Arterial hypertension
	Hardening of the aortic valve leaves and the aortic walls	Atherosclerosis of the aorta – "metallic" sound
	Increased pressure in the pulmonary circulation	Mitral heart defects Lung emphysema Obstructive chronic bronchitis
Intensification of both sounds	Growing influence of sympathetic nervous system on the heart	Physical effort Emotional stress Hyperthyroidism, fever, tachycardia
	Extra-cardiac causes	Thin thorax Shrinking of the lung borders Large cavern Enlarged stomach gas cyst Anemia

Presystolic "galloping" rhythm (the presence of pathological 4th heart sound) Pathological 4th heart sound is observed in patients with marked myocardial hypertrophy, post-infarction cardiosclerosis, i.e. under the conditions of increased diastolic myocardial rigidity and abnormal ventricular contractions during atrial sytole.

Summed "galloping" rhythm (3rd and 4th heart sounds coalesce into one) indicates considerable diminution/weakening of myocardial ventricular tone accompanied by tachycardia. That is why it is interpreted as SOS sign – "a cry of the heart for help".

"Quail" rhythm occurs in mitral stenosis (a click of opening of the mitral valve similar to splitting 2nd heart sound combined with "flapping" sound).

Heart murmur is an audible phenomenon heard between sounds during pauses. The origin of murmurs is a change in laminar blood flow in the vessels or inside the heart chambers. Murmurs detected between 1st and 2nd heart sounds are called systolic whereas those heard after 2nd heart sound are called diastolic.

After detection of murmurs on heart auscultation in standard points, you must detect:

1) phase of the cardiac cycle in which murmur is heard (systolic, diastolic, systolodiastolic);

2) duration of murmur (short or long) and what phase of the cardiac cycle it takes place in (protodiastolic, mesodiastolic, presystolic or pandiastolic, early systolic, late systolic or pansystolic)

3) volume of murmur in general (quiet or loud) and volume change in the phase of the cardiac cycle (descending,accelerating, decreasing-accelearting or monotonous);

4) timbre of murmur (blowing, scraping, sawing etc.);

5) the point of maximum murmur volume (*punctum maximum*) and its direction (the left axillary fossa, the carotid and subclavian arteries, the interscapular space);

6) variability of murmur, i.e. dependence of volume, timbre and duration on body position, breathing and physical activity phases.

Organic systolic murmurs can be caused by insufficiency of the atrioventricular heart valves (valvular or muscle origin), stenosis of the aorta mouth and pulmonary artery mouth, heart septal defect and some other causes (see Table 27). Thre hallmarks of organic systolic murmur are volume, duration and harsh timbre. Sometimes it is heard over the entire heart surface. However, its maximum volume and length are always determined at the auscultation point of the valve or the orifice where the murmur has originated form. In addition, organic systolic murmurs often have characteristic areas of spreading. Another feature of such murmurs is their relative stability since they are well heard in different positions of the patient during both respiratory phases and always increase after physical exertion.

Table 27

Points of maximum listening to organic murmurs

The point of maximum listening	Location	Organic heart murmurs	Causes
1	the apex beat area	Systolic	mitral valve insufficiency
		Diastolic	mitral valve stenosis
2	2nd intercostal space at the right border of the sternum	Systolic	stenosis of aorta
		Diastolic	aortic valve insufficiency
3	2nd intercostal space at the left border of the sternum	Systolic	stenosis of pulmonary artery
		Diastolic	pulmonary valve insufficiency
4	at the base of the xiphoid process	Systolic	tricuspid valve insufficiency
		Diastolic	tricuspid valve stenosis

Organic **systolic murmur at the first point** above the heart apex is heard in mitral valve insufficiency. It is of diminishing or band-like character and is usually combined with diminution/weakening or even complete disappearance of 1st heart sound. 3rd heart sound can often be detected as well. The sound becomes louder when the patient lies on his/her left side, on holding breath on breathing in, after physical exertion. The left axillary fossa is a common place of spreading.

Organic **systolic murmur at the second auscultation point** in the second intercostal space at the right border of the sternum is determined in **stenosis of aortic mouth**. Often it is so loud and harsh that it is distinctly heard over the entire heart area and is sometimes felt even on palpation on the sternal manubrium or to the right of it in the form of systolic tremor. Murmur usually spreads over the carotid and subclavian arteries. It is often detected in the interscapular space at the level of the 1st – 3rd thoracic vertebrae. It is of rhombic character and is best heard in a horizontal position on the right side.

Organic **systolic murmur at the fourth auscultation point** at the base of the xiphoid process is typical of tricuspid valve insufficiency, which like mitral insufficiency may be of valvular or muscular origin. Murmur is of diminishing character. It is not always combined with the weakening of 1st heart sound and additional 3rd heart sound. It is heard in 3rd – 4th intercostal space to the right of the sternum, and, unlike other heart murmurs, is amplified in inspiration (Rivero-Corvallo' sign).

Diastolic murmur at the first auscultation point above the tip of the heart in **mitral stenosis** is most often heard in the limited area and does not spread far. Usually it is best detected in vertical position of the patient and after physical exertion. Protodiastolic murmur during passive ventricular filling is of diminishing character and presystolic one (during atrial systole) is escalating.

Quiet, gentle/soft diastolic (presystolic) murumur over the heart apex is sometimes heard in patients with insufficiency of the aortic valve. This murmur is the so-called Flint's murmur (functional mitral stenosis).

Diastolic murmur at the second auscultation point is heard in the second intercostal space at the right border of the sternum indicates **aortic valve insufficiency**. It is usually gentle/soft, blowing, diminishing, "pouring". It is better detected on standing or sitting on leaning forward. At the same time after physical exertion the murmur often diminishes.

Quiet blowing diastolic murmur in the second intercostal space on the left side of the sternum is sometimes heard in patients with hypertension of the pulmonary circulation. This murmur is the murmur of relative insufficiency of the valve of pulmonary artery (Graham Stell's murmur).

Functional systolic murmur is most often heard above the pulmonary artery, the heart apex and the left border of the sternum in the 3rd – 4th intercostal spaces and it is seldom heard above the aorta. They have a wide range of features:

1) they are heard only in the limited area and do not spread anywhere;

2) they are quiet, short-term, blowing. The exceptions are murmurs associated with dysfunction of the papillary muscles and chords because they sometimes have a peculiar musical timbre, which is comparable with the sound of a ringing/clinking or torn string;

3) they are labile as they can change their timbre, volume, and duration. They appear or disappear under the influence of the psycho-emotional and physical stress, in changes the body's position , in different phases of respiration etc;

4) they are not accompanied by a change in 1st and 2nd heart sounds, appearance of additional sounds, expansion of the heart borders and signs of circulatory failure.

Pericardial friction murmur commonly occurs in dry pericarditis and it is the only objective sign of it. The inflamed cusps of pericardium become rough because of the presence of fibrin deposits on their surface. Most commonly, it is detected in the area of absolute heart dullness on the left border of the sternum or above the heart base on the manubrium of the sternum. It is commonly heard in the limited area and does not spread anywhere. It can be quiet or loud. It can resemble rustling, scraping, crispy sound, with this feature being so marked that it can be felt on palpation. Systolic-diastolic-presystolic murmur resulting from ventricular systole, ventricular diastole and active atria contractions is common. It is perceived as sound that occurs near the very surface of the chest wall, pressing a stethoscope intensifying the murmur volume.

4.2. Symptoms of diseases of the cardiovascular system

4.2.1 Arterial hypertension

The term "arterial hypertension" (AH) implies the syndrome of high blood pressure in "hypertension" and "symptomatic arterial hypertension".

The term "hypertension disease" (HD) proposed by Georgij F.Lang in 1948 corresponds to the notion of "essential hypertension".

HD is referred to as a chronic disease. Its main manifestation is AH which not associated with the presence of pathological processes characterized by increased blood pressure due to the known and controlled causes at present (symptomatic AH).

The prevalence of arterial hypertension: HD accounts for 90-95%, symptomatic AH accounts for 5-10% (atherosclerosis of the aorta is 20%, renoparenchymal is 45-55%, renovascular is 1.5-5% 1.5 and endocrine is 3%).

Factors contributing to the development of HD :

- heredity (family history of HD)
- age
- emotional stress
- overweight
- chronic alcohol intoxication
- smoking
- abuse of salt
- lack of physical activity

Increased blood pressure is necessarily stated in patients with a newly diagnosed AH. The extent of arterial hypertension achieved in other patients under treatment is estimated.

Table 28

The classification of blood pressure level (BP)

Category	Systolic BP, mm Hg	Diastolic BP, mm Hg.
Optimal	<120	<80
Normal	<130	<85
High normal	130-139	85-89
1 degree	140- 159	90-99
2 degree	160- 179	100- 109
3 degree	>180	>110

If systolic BP and diastolic BP are of different categories, higher category is assigned.

In patients with hypertension, prognosis depends not only on the level of BP (see Table 28), but also on the level of the target organs involvement as well as the presence of associated clinical conditions. Owing to this, the stratification of patients according to the degree of risk was introduced in the modern classification. Data on risk factors, lesions of the target organs and associated clinical conditions are used to determine the risk of developing cardiovascular complications.

Risk factors (RF) affecting cardiovascular complications:

- Value of pulse pressure (in elderly)
- Age (in men over 55 years, in women over 65 years)
- Smoking
- Total cholesterol > 5 mmol/L, low density cholesterol > 3 mmol/L and high density cholesterol in men < 1 mmol/L and in women < 1.2 mmol/L.
- Impaired glucose tolerance (IGT).
- Family history of early cardiovascular disease (CVD) in men under 55 years and in women under 65 years
- Abdominal obesity (waist circumference in men > 102 cm, in women > 88 cm)

Lesions of the target organs (LTO):

Left ventricular hypertrophy

- ECG: Sokolov-Lyon's sign > 38 mm;
- Echocardiography: index of left ventricle mass > 125 g/m² in men and > 110 g/m² in women

Vessels

- signs of artery wall thickening or atherosclerotic plaques of the major vessels detected by ultrasound examination
- the rate of pulse wave from the carotid to femoral artery > 12 m/s
- ankle/brachial index < 0.9

Kidneys

- a slight rise in serum creatinine: 115-133 mkmol/L (1.3 -1.5 mg/dL) in men or 107-124 μmol/L (1.2 -1.4 mg/dL) in women
- low creatinine clearance < 60 ml/min (Cockcroft formula)
- MAU (microalbuminurea) 30-300 mg/day
- albumin/creatinine ratio in urine > 22 mg/g (2.5 mg/mmol) in men and > 31 mg/g (3.5 mg/mmol) in women

Related clinical conditions (RCC):

- Cerebrovascular disease (ischemic stroke, hemorrhagic stroke, transient ischemic attack)
- Heart disease (myocardial infarction, angina pectoris, coronary revascularization, congestive heart insufficiency)
- Peripheral artery disease
- Hypertensive retinopathy

Low risk group (see Table 29). This group includes men and women under 55 years with 1st degree of arterial hypertension in absence of risk factors and lesions of the target organs. The risk of cardiovascular complications in the next 10 years in this group is less than 15%.

Intermediate risk group. This group includes patients with a wide range of blood pressure fluctuations. The inclusion criterion for this group is the presence of risk factors in the absence of lesion of the target organs and assotiated diseases. In other words, this group includes patients with a slight increase in BP and multiple risk factors and patients with a marked increase in BP. The risk of cardiovascular complications in the next 10 years in this group is 15-20%.

Table 29

Stratification of risk cardio-vascular complications in AH

Risk criteria	**High normal BP**	**1- degree AH**	**2- degree AH**	**3- degree AH**
	130-139/85-89	140-159/90-99	160-179/100-109	180/110 and more
	Risk of cardiovascular complications			
Absence of risk factors (RF)	low risk	low risk	medium risk	high risk
1-2 RF	low risk	medium risk	medium risk	very high risk
> 2 RF, LTO, diabetes mellitus	high risk	high risk	high risk	very high risk
RCC	very high risk	very high risk	very high risk	very high risk

High risk group. This category includes patients with lesions of the target organs, regardless of degree of AH and associated risk factors. This

group includes patients with high normal BP with diabetes mellitus. The risk of cardiovascular complications in the next 10 years in this group of patients is more than 20%.

Very high risk group. This group includes patients with RCC (angina pectoris and/or past myocardial infarction, revascularization operation, cardiac insufficiency, cerebral stroke or transient ischemic attack, chronic renal insufficiency, lesion of the peripheral vessels, II-IV stage retinopathy) regardless of AH degree. The risk of cardiovascular complications in the next 10 years in this group is more than 30%.

In Russia, it is acceptable to state the stage of HD. Stage I suggests the absence of LTO. Stage II suggests the presence of the LTO. Stage III is established in the presence of RCC.

Diagnosis HD is made in two stages. **The first stage** consists in stating AH, **the second stage** is ruling out symptomatic AH. After that the degrees of BP and the risk of AH are determined.

The objectives of examination of patients with AH are:

- to confirm stability in BP increase;
- to rule out AH of secondary character;
- to establish risk factors for developing CVD both curable and incurable;
- to evaluate the presence of lesions of the target organs and related clinical conditions;
- to assess the individual risk of cardiovascular complications.

Patient's complaints

The most common subjective manifestation of AH is headaches. The character of headaches is varied. Some patients have them in the morning on waking up. The other group of patients assotiate headaches with emotional stress and notices their worsening by the end of the working day. Many patients associate the incidence of headaches with changes in meteorological conditions. As a rule, the pain is not so intense. Many patients perceive them as a feeling of heaviness in the head.

Localization of pain also varies: the occiput, temporal area, forehead, or of uncertain localization. Pain intensity and frequency grow with an increase in BP.

Dizziness is less often. Usually it is transient and does not affect the patient's well-being. However, certain patients have severe dizziness accompanied by nausea and vomiting. Although this syndrome is more char-

acteristic of hypertonic crisis, however, its appearance is not always clearly associated with fluctuations of BP.

Neurotic disturbances are the third in this group. They manifest by irritability, fatigue, despondency, sleep disturbance and unstable mood. With the course of disease and rising values of BP, neurotic disturbances occur more and more often, which may be due to influence of the present disease on the patient's condition.

Pain in the heart is also one of the functional disorders. It is described by patients as pain of moderate intensity, localized in the left side of the chest, usually in the heart apex area. Pain usually appears at rest after emotional stress, is not associated with the physical stress and **is not** relieved by nitrates. These pain sensations are not the manifestations of coronary heart disease (CHD) and are usually relieved after administering psychoactive drugs. More often in such cases, patients notice a feeling of heaviness in the heart without a clear association with physical stress and without radiation, controlled by taking nitroglycerin and disappearing after BP returns to the normal level. Apparently, such sensations in their origin do not greatly differ from cardialgia though some researchers do not rule out possible participation in their origin and irritation of the aorta receptors in case of its wall tension in response to a considerable rise in BP.

A relatively small number of patients with uncomplicated AH mention heartbeats which are most often a manifestation of sinus tachycardia or extrasystole.

More specific and more common are complaints about vision disturbances such as black spots/"flies", curved lines and blurred vision. In organic changes of retina (hemorrhage, degenerative changes), there may be persistent vision disturbances, even its total loss (central retinal artery thrombosis).

Finally, some patients point out subjective impairments of nonspecific nature such as weakness, fatiguability, reduced work performance.

When analyzing the disease and life history, pay attention to the following:

- duration of high BP, the symptoms of disease onset, degree and periodicity of high BP, the presence of hypertonic crisis, efficacy and tolerance of the previous antihypertensive treatment, the causes of worsening of the disease;

- patinet's lifestyle, including nutrition (fats and salt consumption), physical activity, personal and psychosocial factors (family, work, education), adherence /compliance to antihypertensive therapy;

- the presence of ischemic heart disease or heart insufficiency, cerebrovascular disease, peripheral vascular disease, diabetes, gout, dyslipidemia, bronchospasm, kidney disease, other diseases and information about medications used to treat present diseases;

- family history of AH, diabetes mellitus, ischemic heart disease, brain stroke and kidney disease;

- bad habits (smoking, alcohol).

Objective examination includes:

measurement of height, body weight, body mass index, measuring waist and hips circumference, calculation of the ratio between waist and hip circumference. Edema of the limbs is noted.

Examination can reveal tortuous and sharply protruding temporal arteries. Hard pulse is usually noted. A number of patients have a tendency to tachycardia. If there is a considerable left ventricular hypertrophy, a raising apical beat is detected. The heart borders are not greatly changed. If dilatation of the left ventricle cavity accompanies the condition, patients with long-lasting AH may have a slight left heart enlargement.

On auscultation, 2nd heart sound emphasis/accent is noted above the aorta. Typical expulsion systolic murmur can be heard in patients in persistent AH or a sharp increase in blood pressure (hypertonic crisis). This sound is constatntly heard in patients with associated atherosclerosis of the ascending aorta segment. Independent systolic murmur above the aorta is due to parietal (near-the-wall) vortical (turbulent) blood movement in the aorta because of rigidity and roughness of inner surface during its expansion during systole.

Marked hypertrophy of the left ventricle and its diastolic dysfunction (impaired relaxation) may cause 4th heart sound due to active reduction of the left atrium when diastolic pressure in the cavity of the left ventricle is high. This tone is most often heard at the moment of a considerable BP increase, making systolic emptying of the left ventricle difficult. The 4th heart sound is most often registered on phonocardiography. The 3rd heart sound and systolic murmur of mitral regurgitation can appear if accompanied by dilatation of left ventricle cavity and impairment of its contractile function.

BP measuring in accordance with international standards:

BP measuring conditions

It is recommended to:

- exclude use of coffee and strong tea within an hour before examination;
- not to smoke within 30 minutes before BP measuring;
- stop taking simpathomimetics, including nasal and eye drops.
- BP is measured at rest within a 5-minute rest. If BP measurement procedure was preceded by a significant physical exertion or emotional arousal, a period of rest should be extended by 15-30 minutes.

Equipment:

• cuff size should match the size of the arm: a rubber inflatable part of the cuff should cover at least 80% of the circumference of the upper arm. The cuff of 12-13 cm in width and 30-35 cm in length (medium size) is used for adults. Large and small cuffs should be available for thick and thin arms.

• a column of mercury or a gauge arrow of the sphygmomanometer should be at zero mark before BP measuring.

The number of measurements:

• To assess the magnitude of BP on every hand, you must perform at least two measurements at intervals of not less than one minute. If difference is more than 5 mmHg, one more measurement is performed. The average value of the last two measurements is taken as a final one.

• To diagnose AH in case of a slight BP increase, a repeated measurement (2-3 times) is performed in a few months;

• In case of a marked BP increase and the presence of lesions of the target organs as well as high and very high risk of cardiovascular complications repeated BP measurements are performed in several days.

Measuring tips:

• Air is quickly pumped into the cuff to pressure value exceeding systolic BP by 20 mm Hg (a marked sign is pulse disappearance)

• BP is measured with accuracy to 2 mm Hg.

• Pressure in the cuff is reduced at a rate of approximately 2 mm Hg per second.

• Pressure value at which 1st heart sound appears corresponds to systolic BP (Korotkoff's 1st sound phase);

• Pressure value at which disappearance of heart sounds (Korotkoff's

5th sound phase) corresponds to diastolic BP. In children, adolescents and young people immediately after physical exertion, in pregnant women and in some pathological conditions in adults, when it is impossible to determine 5th phase, Korotkoff's 4th sound phase should be determined, which is characterized by significant weakening of heart sounds

• If tones are very weak, you should raise your hand and perform several compressive movements with a hand, then repeat measurement, thereby you don't have to firmly squeeze the artery by the stethoscope bell (membrane).

• On primary examination of the patient pressure should be measured on both hands. In the future, measurement is done on the hand where BP is higher.

• In patients over 65 years old, with diabetes mellitus and patients undergoing antihypertensive therapy, BP is measured after standing 2 minutes.

• It is also advisable to measure BP on the feet, especially in patients under 30. Measurement is done using a wide cuff (the same as in people with obesity). The diaphragm of the stethoscope is placed in the popliteal fossa.

All patients are administered blood, creatinine level, cholesterol, triglycerides, HDL and LDL (high and low density lipoproteins) /cholesterol, glucose and urine tests. Degree of dyslipidemia is determined. In patients with nephrosclerosis or malignant AH, moderate proteinuria is observed with minimal changes in the sediment (hyaline cylinders).

Daily monitoring of arterial pressure is conducted to estimate more accurately the level of BP and its changes under the influence of physical and emotional stress, time of day, weather factors and adequate therapy.

ECG criteria for diagnosis of left ventricular hypertrophy: voltage $S_{V1} + R_{V5-6} > 35$ mm (Sokolov-Lyon's criterion) and R aVL + S_{V3} x width of QRS (Cornell's multiplication result) > 2,440 mm x ms.

Echo-cardiography signs of left ventricle hypertrophy: myocardium mass index /height² (> 115 g/m² in men; > 95 g/m² in women).

Specialist advice is needed to clarify the presence of cerebrovascular pathology and to identify the degree of hypertensive retinopathy.

Indications for special studies:

- fairly rapid development of AH severity which previously ran a benign course;

- presence of crises with marked vegetative manifestations;

- sudden development of AH;
- suspected secondary AH.

According to the findings, it is necessary to do potassium and sodium blood test, Nechyporenko blood test, daily urine protein test, urine bacteriuria test, kidney ultrasound etc.

Symptomatic (secondary) arterial hypertension

Symptomatic AH is a symptom of illness or injury of the organs involved in BP regulation. They account for up to 10% of the total number of AH cases. There are more than 50 diseases that are accompanied by AH.

Factors enabling to suspect secondary AH:

- frequent symptoms of secondary AH: muscle weakness, convulsions, spontaneous hypokalemia, episodes of paroxysmal sweating, headaches, anxiety, palpitations (pheochromocytoma), murmur above the renal arteries (stenosis of the renal arteries);
- early-life onset (under 20 years of age);
- BP is higher than 180/110 mm Hg;
- lesions of the target organs: retinopathy of high degree, serum creatinine level is more than 132 mkmol/L;
- family history of kidney disease;
- ineffectiveness of the multidrug therapy;
- acute onset of AH, BP stability and primarily high diastolic blood pressure;
- good tolerance of AH.

Parenchymatous nephrogenic hypertension is caused by humoral renin-angiotensin-aldosterone system. Renal-parenchymatous AH is the most common one among symptomatic AH.

Characteristic signs are:

1) young age;
2) absence of risk factors for developing HD;
3) absence of hypertensive crises;
4) primarily high diastolic BP;
5) relationship between worsening of nephritis and increase in diastolic BP or increase in diastolic blood pressure due to intercurrent infections;
6) progression in blood pressure rise with development of chronic renal insufficiency;

7) identification of functional and anatomic asymmetry of the kidneys;

8) association between AH and urinary syndrome (characteristic changes in urine sediment).

Renovascular hypertension. Renovascular AH develops when the narrowing of the lumen of the renal arteries is more than 70%. Any pathological condition accompanied by constriction of the major kidney arteries (malformations, atherosclerosis of renal artery) causes a rise in blood pressure. Renovascular hypertension is characterized by:

1) prolonged vascular systolic or systolic-diastolic murmur occurring in turbulent blood flow as a result of the narrowing of the renal arter, which is heard on the outer edge of the rectal abdominal muscles 2.5-4 cm above the navel or in costovertebral corners of the lumbar region;

2) lag/delay in kidney contrast on the lesion side in urography;

3) reduction in kidney size by 1 cm on the lesion side (ultrasound data);

4) severity of stenosis (aortography and duplex scanning of kidney vessels data);

5) is characterized by high, mostly diastolic hypertension > 120 mm Hg.

Hemodynamic (cardiovascular) hypertension is caused by lesions of the heart and major vessels:

1) atherosclerosis of the aorta, aorta hypoplasia, aortitis (decreased aorta elasticity);

2) aortic valve insufficiency and atrioventricular block (increased heart stroke volume);

3) polycytemia (BP rises due to increased total peripheral resistance, blood viscosity, circulating blood volume)

4) coarctation of the aorta (BP rises due to a sharp increase in resistance to blood flow at the site of narrowing of the aorta).

Endocrine hypertension. Excessive production and hormone secretion with pressing influence underlies the development of AH. AH in Cushing's disease is caused by excessive production of cortisol, thereby increasing the content of corticosteroids and aldosterone. In pheochromocytoma (a tumor of the adrenal gland) AH is caused by hypersecretion of noradrenaline. It is characterized by a considerable increase in BP with signs of irritation of the vegetative nervous system (agitation, tremors, fever, leukocytosis, hyperglycemia). In hyperthyroidism, under the influence of high content thyroxine in the blood hyperkinetic AH develops, which is due to increased

cardiac output and cardiac propulsive function. In Conn's syndrome (primary aldosteronism) high AH is combined with muscle asthenia, ensuing cramps, hypokalemia, polyuria, reduced tolerance to glucose.

Conn's syndrome

1. Cardiovascular syndrome. AH is constant. Crises are rare and are associated with mechanical pressure of the tumor on the adrenal medulla and release of large amounts of catecholamines. Insignificant hypernatremia (143-147mmol/L), spontaneous hypokalemia (less than 3, 5mmol/L), severe hypokalemia in treatment with diuretics are present.

2. Neuromuscular syndrome includes myasthenia gravis, paresthesia, paresis, convulsions, tetanic cramps worsening in winter and with taking tiazide diuretics.

3. Kidney syndrome includes polydipsia, polyuria, nocturia, isohypostenuria, alkaline reaction urine, primary hyperaldosteronism.

Cushing's disease is a disease accompanied by increased levels of ACTH (adrenocorticotropic hormone) and cortisol due to pituitary adenoma and hyperplasia of cells of the cortex of the adrenal gland. The disease often develops between 20 to 40 years, women being affected 5 times more often than men. Change in ACTH secretion control mechanism underlies the disease.

Cushing's syndrome develops in the adrenal cortex tumors and non-hypophysis localization (lungs, pancreas).

Complaints: general weakness, headache, pain in the spine, frequently in the lumbosacral area, reduced libido, dysmenorrhea.

Examination data: the skin is thin, dry on the face. The skin is of purple-cyanotic colour in the chest and back area. Distinct venous pattern is marked on the chest and limbs. Wide scars/striae of reddish-purple colour appear on the skin of the abdomen, the inner surfaces of the shoulders, hips, in the area of breasts. Skin hyperpigmentation is often noted in places of skin folds, rubbing because of wearing the clothes. There is loss of hair. Excessive deposit of fat in the neck, torso, face, abdomen area is noted. Moon-like face is typical.

AH is one of the early and permanent symptoms in hypercorticism.

Osteoporosis with pathological bone fracture occurs in almost 80% of patients. Most often it affects the lumbar and thoracic parts of the vertebral column, ribs, sternum, bones of the skull. Pustules and trophic skin lesions

followed by the development of secondary immunodeficiency tend to form increasingly. Carbohydrate metabolism is impaired due to reduced glucose tolerance. Sexual disorders are one of the early and regular syndromes. Virile syndrome is marked, i.e. appearance of male and disappearance of female secondary sexual characteristics in women: male body type, facial hair, hair on the abdomen, back, extremities, lower voice timbre, hair loss, disturbance of menstrual cycle up to amenorrhea, breast atrophy, hypertrophy of the clitoris. Hirsutism is a less significant degree of virilization, manifested only male hair growth type.

The following instrumental methods are a valuable diagnostic tool: x-ray, tomography, ultrasonography, radioisotope study as well as determination of hormones in the serum and daily excretion of 17-oxyketosteroids with urine (cortisol, cortisone).

Pheochromocytoma is a tumor of the cerebral layer of the adrenal glands.

In case of adrenaline crisis, increased blood pressure is of short-term nature (often a few minutes) and is accompanied by a feeling of inner restlessness, palpitations, tremor, sweating. In paroxysm, patients note shortness of breath, pain in the heart. Laboratory tests detect hyperglycemia and leucocytosis.

Crises of norepinephrine are characterized by a more gradual, but long persistent rise in blood pressure with less marked vegetative manifestations. Attacks usually occur spontaneously, though sometimes they are caused by physical or emotional stress, palpation of the abdomen, invasive methods of study etc.

Cerebrovascular hypertension is associated with diseases of the brain or spinal cord such as tumor, trauma, cysts of the hypothalamus, encephalitis, hemorrhage in history. Paroxysmal increase in blood pressure, symptoms of lesion of the central nervous system divisions, vegetative dysfucntions, severe cephalgia, epilepsy or diencephalic crisis, typical changes in personality are characteristic.

4.2.2. Atherosclerosis

Risk factors for atherosclerosis

Atherosclerosis is a chronic disease caused by the change in the intima of the arteries due to deposit of lipids in it, lipid plaque formation, the development of connective tissue, which leads to hardening and unequal

narrowing of vessles, disruption of blood flow and the development of dystrophic, necrobiotic and sclerotic processes in the organs.

Risk factors for atherosclerosis

Irreversible:

- age;
- early-life onset in men (by 10 years) compared to women;
- genetic predisposition.

Reversible:

- smoking;
- AH;
- obesity;
- low physical activity;
- emotional stress;

Potentially reversible:

- hyperlipidemia;
- hyperglycemia;
- low level of high density lipoproteins cholesterol (less than 0.9 mmol/L).

In terms of prevention of atherosclerosis and its complications, it is desirable that the level of serum cholesterol should not exceed 5.0 mmol/l; TG – 1.7 mmol/L, low density lipoproteins cholesterol – 3.0 mmol/L and high density lipoproteins cholesterol range 1.0 – 1.89 mmol/L.

Pathogenesis of atherosclerosis

Atherosclerosis is a disease affecting the arteries of elastic type, such as the aorta, iliac vessels as well as the large and medium arteries of muscular type (coronary, carotid, cerebral arteries, arteries of the lower limbs).

Atherogenesis involves a complex of interactions between the vascular wall, corpuscular elements of the blood, biologically active substances dissolved in bloood and a local disruption of blood flow (Virchow's triad). History of research aimed at clarifying the essence of processes underlying atherosclerosis began more than 150 years ago. To date, clear understanding of atherosclerosis as multifactorial disease has been formed, with complex disorders in biochemical, immunological and molecular-genetic processes underlying it.

To date, two hypothesis of formation and development of atherosclerosis dominate: **the hypothesis of "response to damage"** and **lipid-infil-**

tration hypothesis. Numerous studies in the field of lipid study and other areas have shown that both hypotheses in principle do not contradict and largely complement one another.

Hypothesis of "response to damage" formulated by the American researcher Ross focuses on the integrity disruption of the endothelium as an initiating factor of atherosclerotic process. Factors causing damage to the endothelium are very diverse, but the most common are the following: carbon monoxide entering the blood in active and "passive" smoking, increased blood pressure due to either illness or emotional or considerable physical exertion, dyslipidemia, particularly hypercholesterinemia (HC), resulting from either family (hereditary) predisposition or bad habits, primarily dietary errors. As damaging agents we can also mention various viruses and bacteria (most often Chlamydia pneumonia, cytomegalovirus), modified (oxidized) lipoproteins, and a number of other endogenous and exogenous factors. Whatever the agent that has caused damage to the endothelium is, monocytes and platelets adhesion occurs in its place, accompanied by migration of monocytes to the intima.

Progressive thickening of the intima leads to hypoxia within a plaque and in nearby areas of the vessel. Hypoxia is a possible cause of necrotic changes in the plaque core and enhanced plaque vascularization from the vasa vasorum adventitia system. As a result of weakening of the muscloelastic vessel layer in the coronary arteries their remodeling with dilatation takes place, with inner diameter of vessel for some time being supported "normal" until the progressive growth of plaque exceeds the compensatory possibilities of the medial layer of the artery and leads to the progressive narrowing of its lumen. It is at this stage that the plaques become unstable and play a key role in the development of complications of atherosclerosis.

Lipid theory of atherosclerosis was introduced by the Russian scientist N. Anichkov, who showed in his experiments on rabbits in 1913 that adding cholesteol to the normal food of these animals caused changes in the aorta similar to those observed in humans with atherosclerosis. In the future, this scientific evidence was developed in the works of scientists of the United States, England, Germany, Japan and some other countries. Unlike "response to the damage" theory, the supporters of this hypothesis believe that atherosclerosis is triggered by lipid and lipoprotein infiltration

of the intima and subendothelium. With the accumulation of lipids in the core of plaques, there is an increase in its size. As a result a fibrous cover plaque under the action of specific enzymes (elastase, metalloproteinase) becomes thinner and ruptures under certain conditions (increase in blood pressure, considerable physical exertion). The rupture is accompanied by activating a cascade of blood coagulation, platelet aggregation with the formation of a blood clot that blocks the lumen of the vessel. Clinically this process manifests itself, depending on the localization, by unstable angina, myocardial infarction (MI), stroke.

Classification of dyslipoproteinemia

Dyslipoproteinemia is impairment of lipid-content markers ratio levels in serum (see Table 30).

1. Primary (hereditary);

2. Secondary in the presence of other diseases (hypothyroidism, diabetes mellitus, nephrotic syndrome, obstructive diseases of bile ducts).

Fredrickson et al. (1987) described five types of hyperlipoproteinemia based on numerous measurements of their levels. Subsequently, the WHO experts completed the classification (see Table 30).

Table 30

Diagnostic criteria of dyslipoproteinemia

Lipids parameters	mmol/L
Cholesterol	> 5.0
Low density lipoprotein cholesterol	> 3.0
High density lipoprotein cholesterol	< 1.0 (in men), < 1.2 (in women)
Triglycerides	> 1.7

You must also remember that the type of hyperlipidemia of the patient can change from one to the other under the influence of diet, changes in body weight and treatment.

Table 31

Classification of hyperlipidemia

type of hyperlip-idemia	Elevated lipoproteins	choloesterol	triglicerides (TG)	Occurrence	Degree of atherogeniity
Type I	chylomicrons	Norm	++++	<1%	Non-atherogenic
Type IIa	Low density lipoproteins	++	Norm	10%	High
Type IIb	Low and very low density lipoproteins	++	++	40%	High
Type III	Low density lipoproteins	++	+++	<1%	High
Type IV	Very low density lipoproteins	Norm or +	++	45%	Moderate
Type V	Very low density lipo-proteins and chylomicrons	++	++++	5%	Low

The classification also does not take into account the concentration of high density lipoproteins cholesterol, though this value, as it became clear later, significantly affects the likelihood of coronary heart disease (CHD) in patients with hyperlipidemia. Despite these drawbacks, the system of typing is of paramount importance, drawing attention to the nature of metabolic disorders causing hyperlipidemia and allowing to apply a rational approach to diagnosis and treatment. The two most common form variations are type II a и IV (see Table 31).

Hyperlipidemia IIa type is characterized by increased concentration of low density lipoproteins cholesterol and total cholesterol concentration, with triglyceride levels being within the normal range. This phenotype is fairly common among the population and is closely assotiated with the development of coronary atherosclerosis. Family hypercholesterinemia, polygenic hypercholesterinemia, hypothyroidism are those nosologic forms when hyperlipidemia of IIa type often develops .

Hyperlipidemia IV type manifests by a high concentration of very low density lipoproteins and triglycerides. Hyperlipidemia IV phenotype may reflect family hypertriglyceridemia and can also be a frequent manifestation of secondary lipid metabolism disturbances. The nature of mono/polygenous defect of hyperlipidemia IV type remains unclear. In combination with a low concentration of high density lipoproteins this phenotype is highly atherogeinc, especially in patients with diabetes mellitus.

4.2.3. Coronary heart disease

Ischemic heart disease (IHD) is a disease of the myocardium caused by mismatch of coronary blood flow and the needs of the myocardium. Arteriosclerosis of the coronary arteries underlies IHD. However, the development of IHD can be caused by spasms or thrombosis of the unchanged artery. IHD is one of the leading among causes of death among the population. In this country, mortality rate from cardiovascular diseases is 52% among men and 63% among women. IHD and cerebral stroke account for 90% of all deaths from cardiovascular diseases.

Classification of ischemic heart disease

1. Sudden coronary death.
2. Stenocardia.
2.1. Stenocardia of tension.
2.2. Angina of first onset (1-2 months).
2.3. Stable stenocardia I-IV.
2.4. Progressive stenocardia.
2.5. Spontaneous stenocardia.
3. Acute myocardial infarction.
3.1. Macrofocal (transmural).
3.2. Microfocal.
4. Postinfarction cardiosclerosis.
5. Cardiac arrhythmias.
6. Heart failure.

Stenocardia (angina pectoris)

Stenocardia is paroxysmal (attack-like) pain in the heart, which is one of the clinical forms of IHD both in patients with post-infarction heart sclerosis and in patients without myocardial infarction in history.

Pain syndrome in stenocardia meets the following criteria:

1. Localization of pain behind sternum or along the left border of the sternum.

2. Squeezing, crushing nature of pain.

3. Pain of short duration (minutes).

4. Pain radiates to the left shoulder, left arm, left half of the head and neck, lower jaw, interscapular space.

5. The appearance of pain is obviously connected with physical or emotional stress.

6. Quick and controlling effect of taking nitroglycerin.

When asking a patient with stenocardia, it is necessary to ask about the frequency of attacks, the need for taking nitroglycerine, tolerance of physical exercise, duration of disease, family and occupational anamnesis, bad habits.

Functional classes of severity of stable stenorcardia of tension according to the Classification of the Canadian Association of Cardiology (Campeau L, 1976)

I "Everyday physical activity" (walking or climbing stairs) doesn't cause angina. Pain occurs only on very intensive, very quick or prolonged physical exertion.

II "A small restriction of normal physical activity", which means the occurrence of angina pectoris on fast walking or climbing stairs, after a meal, being in the cold environment, the windy weather, during emotional stress or in the first few hours after awakening (while walking a distance of 200 m > (two quarters) on the level or on climbingstai rs for more than one floor at normal pace under normal conditions.

III "A considerable restriction of normal physical activity". Angina occurs as a result of relaxed walking a distance of 100-200 m (from one to two quarters) on the level or on climbing stairs for more than one floor at normal pace under normal conditions.

IV "Inability to perform any physical activity without discomfort" or angina may occur at rest.

Diagnosis of stable stenocardia:

Stage I is to detect if the patient has angina or cardialgia.

Stage II is to detect stenocardia within IHD or of other origin – aor-

tal heart defects, hypertensive disease, coronaritis, coronary abnormalities (the presence of risk factors is suggestive of IHD).

Stage III is to perform laboratory diagnosis of dyslipidemia.

Stage IV is to perform following the instrumental studies:

ECG at rest, ECG during the attack. Myocardial ischemia on ECG usually manifests by the ST segment depression (horizontal or oblique descending) and the T wave changes (the inverted, high pointed/spiky or pseudo-normal T wave). Pseudo-normal is called the transformation of the inverted T wave into a normal one. The ST segment depression and T wave changes usually occur in incomplete occlusion of one of the major coronary arteries.

Bicycle ergometry/veloergometry (VEM) is used to detect latent coronary insufficiency, transient arrhythmias and to establish individual patients' tolerance of physical activity. Physical exertion causes tachycardia, moderate BP increase, increased cardiac output and subsequent need of the myocardium in oxygen. Due to the narrowing of coronary arteries lumen in atherosclerosis this leads to acute coronary insufficiency that is accompanied by corresponding changes on ECG. In VEM procedure, the load is increased gradually every three minutes under taking an ECG, measuring BP level and checking the general condition of the patient.

Clinical criteria for stopping of VEM are attack of angina, a decrease in BP by 20 mm Hg from the initial BP level, an increase in BP to 220/110 and more, marked breathlessness, neurological symptoms (dizziness, abrupt headache), submaximal age cardiac beat rate, the refusal of the patient.

ECG criteria for stopping of VEM are a fall or rise in the ST segment by more than 1 mm, frequent (1:10) extrasystoles, paroxysmal tachycardia, atrial fibrillation, conduction disorders, particular changes in the QRS complex.

Diagnostic criteria in patients with ischemic heart disease when performing VEM are the onset of an attack of angina pectoris and ECG changes, only specific ECG changes.

The test is considered positive if horizontal or oblique descending depression or a rise of the ST segment by more than 1 mm has been estimated.

VEM is contraindicated in acute myocardial infarction, unstable angina, severe heart failure, acute thrombophlebitis, marked respiratory distress. Relative contraindications include the presence of marked AH (220/130

mm Hg and higher), tachycardia of idiopathic origin (more than 100 beats/ min), severe rhythm and conduction disorder, fever and fainting in history.

Holter's monitoring ECG (HM) is used to:

1. clarify the causes of syncopal states, their association with rhythm and conduction disorders;

2. register transient and paroxysmal rhythm and conduction disorders, their diagnosis and assotiation with physical exertion and (or) emotional stress;

3. reveal non-evident/ hidden asymptomatic (painless) forms of coronary disease;

4. evaluate physical capacity of patients and adequacy of locomotor regimen for persons with contraindications for VEM as well as for patients of middle and young age with low tolerance of physical activity;

5. evaluate the effectiveness of antianginal and antiarrhythmic therapy and the opportunity of its correction;

6. evaluate the work of the electric cardiac pacemaker, especially with complex stimulation modes.

There are no contraindications for this method.

Indications for coronary angiography are:

1. the need for verification of ischemic heart disease in the presence of pain syndrome which has not been confirmed by other methods.

2. changes in ECG in the absence of angina.

3. in women during menopause suffering from cardialgia which is resistant to antianginal therapies and drugs (nitroglycerine) in the presence of changes in ECG.

4. in patients with diagnosed ischemic heart disease, post-infarction heart aneurysm and those who are subject to cardiosurgical treatment.

5. acute myocardial infarction, unstable angina/ stenocardia.

6. in case of ineffectiveness of medical treatment of angina of tension (FC III – IV).

7. in suspected developmental anomaly of coronary arteries.

8. in aortic heart defects in patients with IHD.

The role of echocardiography in IHD

1. The study of echocardiography findings of left ventricular dilatation and hypertrophy in dynamics after past acute myocardial infarction (AMI) enables to assess the degree of pathological remodeling of the myocardium (structural-functional restructuring of the myocardium).

2. Clarification of the status of local myocardial contractility in patients with postinfarction cardiosclerosis. Local contractility disorders (hypokinesia, akinesia and dyskinesia) correspond to the areas of necrosis.

3. Determination of integrated myocardial contractility values (score of the left ventricle ejection fraction).

The most characteristic clinical feature of unstable angina is, above all, the appearance of symptoms, completely different from the manifestations of stable angina. This primarily affects the nature of cardinal pain when attacks become more intense and prolonged. Pain may radiate to the places where it was absent before. Pain becomes more frequent and can no longer be controlled by taking nitroglycerin.

Classification of unstable angina (E.Braunwald and C. Hamm, 1998)

There are three severity classes and three forms of unstable angina:

Class I is characterized by progressive angina without **angina at rest**. The risk of death or myocardial infarction within one year is 7.3%.

Class II includes patients with **angina at rest**, which has developed within the previous month (not less than 48 hours). The risk of death or myocardial infarction is about 10.3%.

Class III includes patients with **angina at rest** developed within 48 h (angina rest acute). It is the most difficult in terms of prognosis. The risk of cardiac death or myocardial infarction within 1 year is 10.8%.

Thus, the risk of cardiac death and myocardial infarction increases with the increase of severity class.

Depending on the circumstances, prior to the development of unstable angina, A, B, C forms are distinguished.

Form A is secondary unstable angina that develops under the influence of extracardiac factors that cause an increase in myocardial oxygen requirement and, consequently, ischemia. Extracardiac factors may be anemia, infectious-inflammatory processes, arterial hypertension or hypotonia, emotional stressful situation, thyrotoxicosis, respiratory failure. The risk of cardiac death or myocardial infarction within 1 year in form A is 14.1%.

Form B is primary unstable angina that develops without the influence of extracardiac factors. The risk of cardiac death or myocardial infarction within a year of the first unstable angina is about 8.5%.

Form C is postinfarction angina that occurs within 2 weeks after myocardial infarction. It is unstable postinfarction stenocardia. The risk of cardiac death or myocardial infarction within 1 year is 18.5%.

ECG options in unstable angina pectoris:

1. Frequent pain attacks with a rise in the ST segment and a fall in the T wave amplitude.

2. Frequent pain attacks with depression of the ST segment and tachycardia (hypersympathicotonia).

If ECG changes are absent during the attack, this does not rule out the presence of ischemia.

Acute myocardial infarction

The term "myocardial infarction" implies cardiac muscle necrosis associated with impaired coronary blood flow.

Necrotized part loses its ability to be evoked and becomes electrically inactive, which is accompanied by a decrease in myocardial contractility in the area of necrosis. Exclusion of the myocardium part from blood circulation leads to increased loads on the other divisions of the heart with compensatory hyperfunction, which maintains the systemic circulation.

The main symptom in clinical picture of MI is pain. Pain is usually localized behind the sternum. Pain is of squeezing nature, extremely intense. It radiates to the area of the left scapular, the left shoulder, the left half of the neck and the mandible. Duration of pain is dozens of minutes, sometimes several hours.Taking nitroglycerin and non-narcotic analgesics have no effect. The patient has cyanosis, cold sweat, accelerated pulse of poor filling. Pulse is arrhythmic. Blood pressure is often reduced. A typical clinical picture of MI is observed in 75% of patients. It should be noted that data on physical examination in uncomplicated MI are often not informative.

ECG diagnosis. The main sign of macrofocal MI on ECG is the appearance of the Q wave, which becomes more enhanced (more than 0.04 sec) and deeper (more than 1/4 of the R wave). If the waves Q and S merge into one deep wave, it speaks of transmural lesion of the heart.

Thus, macrofocal non-transmural MI is characterized by a deep Q wave and a small R wave. Macrofocal transmural MI is characterized by the QS complex.

The rise in the ST segment in MI represents a shift upwards from the isoline in the leads to the area of infarction. It occurs when there is complete occlusion of one of the major coronary arteries. Changes in the T wave point to disruption of repolarization process in the ischemic damage area surrounding the area of necrosis.

Microfocal MI is characterized by the development of small foci of necrosis in the heart muscle. In contrast to macrofocal MI, there are no pathological Q waves in small foci of necrosis. Therefore, subendocardial, subepicardial and intramural MI are called "myocardial infarction without Q" (MI without Q).

In patients without changes in ventricular complex, the basic ECG signs are changes in the ST segment or the T wave. With a wide use of thrombolytic therapy (it is effective only in acute MI with the rise in the ST segment) and estimating troponins, such concepts as acute MI with the rise in the ST segment and without rise in the ST segment have appeared. Troponins help to diagnose AMI without rise in the ST segment. In microfocal MI, troponins have the greatest diagnostic value and dynamic monitoring for 3-5 weeks.

Periodicity of macrofocal MI (2007):

Developing MI lasts from 0 to 6 hours. It is characterized by the final formation of necrosis focus. By the end of the period, a pathological Q wave (area of necrosis) is formed. The ST segment merges with the positive T wave. During this period, as a rule, the pain disappears.

Acute period is characterized by the presence of polymorphonuclear leukocytes in the myocardium. It continues from 6 hours to 7 days. ECG shows a gradual fall in the ST segment to isoline with the formation of a negative T wave (area of ischemia).

Scarring period is with mononuclear and fibroblast infiltration. Pathological Q wave persists. The ST segment is in isoline. Reverse dynamics of the T waves is seen. During 5-6 weeks the area of necrosis undergoes the formation of a scar.

Healed MI is a scar tissue without cell infiltration. In this period, the heart adapts to new working conditions (myocardial remodeling).

This is a classic characteristic of the dynamic EEG changes on medical treatment of Q-AMI (without thrombolytic therapy and coronary artery balloon angioplasty).

Determining localization and prevalence of MI. This is done when analyzing ECG records in various areas (see Table 32). Localization of MI depends on the location of critical stenosis of either coronary artery and collateral blood flow.

Table 32

Localization of MI

Septal	V_1-V_3
Apical	V_4
Anterior	I, II, aVL
Lateral	V_5- V_6
Posterior	II, III, aVF

Laboratory diagnosis of IM. In case of myocardial necrosis, the contents of a dead cell enter the general bloodstream and can be detected in blood samples.

1) Heart troponins I and T have the highest specificity and sensitivity. Troponin I is detected in the serum in 3-6 hours after myocardial necrosis. Increase in concentration remains for 10 days. Troponin T has a similar diagnostic value, but remains until 14 days. Normal value of troponins in the serum is not detected. Troponin concentration in the blood is directly proportional to the size of heart necrosis area and reaches the maximum values in extensive transmural MI.

2) creatinphosphokinase activity (MB fractions) increases in four hours after damage to the myocardium, reaches its peak in 18-24 hours and remains at an elevated level for 3-4 days.

3) manifestations of resorbtion and necrotizing syndrome. Leukocytosis in blood is present on the first day and remains during the week. ESR begins to accelerate in 5-7 days. Thus, at the end of 1st week of MI there is overlapping between: reducing leukocytosis and acceleration of erythrocyte sedimentation rate.

4) presence of c-reactive protein is noted as well.

4.2.4. Acquired heart defects

Mitral valve insufficiency

Etiology. Causes of organic forms of mitral valve insufficiency are rheumatism (up to 75% of all cases), infectious endocarditis, atherosclerosis and systemic diseases of connective tissue (systemic lupus erythematosus, systemic scleroderma, dermatomyositis, visceral form of rheumatoid arthritis).

Complaints. Patients complain of *shortness of breath* on physical exertion and *palpitations*. In congestive phenomena, shortness of breath at rest and attacks of cardiac asthma can develop. Some patients have *cough*, either dry or with mucous sputum, sometimes mixed with blood. *Heart pain* is aching, crushing, stabbing. It is not always associated with physical activity.

Inspection and palpation of the heart reveal a forced and diffuse apical beat in the 5th intercostal space outward from the midclavicular line (as a result of hypertrophy and dilatation of the left ventricle) on marked regurgitation.

Percussion reveals displacement of the relative heart dullness to the left and upward.

Auscultation of the heart. 1st heart sound is diminished (absence of a period of valves closing). Systolic murmur above the heart apex merges with 1st heart sound. When a patient lies on the left side (with holding breath on exhaling after previous physical exertion), auscultation symptoms become clearer and the place for the best listening shifts laterally, nearer to the front and even the medial axillary line. If blood pressure is increased in the pulmonary circulation, 1st heart sound emphasis/accent is above the pulmonary artery.

X-ray study. In profound mitral insufficiency, a marked enlargement of the left ventricle and left atrium is detected. Displacement of the contrasted esophagus along the major radial arch is noted.

ECG shows signs of hypertrophy of the left atrium and left ventricle.

PCG shows a decrease in 1st heart sound amplitude merging with systolic murmur. Systolic murmur is recorded immediately after 1st heart sound and fills the whole pause between 1st and 2nd sounds. Q-1 sound interval is from 0.07 to 0.08 sec.

Echocardiography reveals:

1) morphological changes in the valves. The nature and extent of the valves lesion (divergent movement of mitral valve cusps, their thickening and the absence of closure in systole), degree of regurgitation are estimated.

2) degree of hypertrophy and enlargement of cavities of the left atrium and the left ventricle (systolic and diastolic chamber size, wall thickness);

3) effects of hemodynamic disorders are pulmonary hypertension and left ventricular dysfunction (ejection fraction, stroke volume).

Stages of mitral insufficiency:

Stage I. Due to mitral valve insufficiency a part of blood from the left ventricle during systole returns back to the left atrium. Increased blood volume entering the left atrium due to atrial contractions is completely expelled to the left ventricle. Normally, there are no signs of pulmonary hypertension. Clinically, a defect may manifest by systolic murmur in the apex, the patient's condition and work performance are not disturbed.

Stage II is characterized by hypertension of pulmonary blood circulation due to weakened contractility of the left ventricle.

Stage III manifests by right ventricular decompensation with congestive phenomena in the systemic blood circulation.

Mitral stenosis

Etiology. Almost all cases of this defect are the result of rheumatism, which is, as a rule, slowly progressing because apparent "attacks" of rheumatism are not detected in 30-60% of cases the disease. Very rarely, mitral stenosis develops as a result of infectious endocarditis.

Complaints. Patients complain of *shortness of breath* and *palpitations* on physical exertion. In case of a sudden rise in pressure in the lung capillaries, cardiac asthma attack can develop. If attacks of asthma and hemoptysis are frequent, critical mitral stenosis (left AV orifice is 1 cm^2) is detected. There is also *cough*, either dry or with production of a small amount of mucous sputum, sometimes with blood, fatigue, fast weakness on physical exertion. Some disturbances (atrial extrasystole) are observed in the period preceding the development of cardiac fibrillation/arrhythmia. Patients do not often complain about *pain* of aching, stabbing nature, rarely of crushing character.

Inspection. Mitral blush at the background of pale skin as well as cyanosis of the finger tips, nose, ears/auricles are noted in accelerating the degree of stenosis and development of pulmonary hypertension. A "heart" hump can develop. Pulsation in the $3^{rd} – 4^{th}$ intercostal spaces along the left border of the sternum and the epigastrium is noted.

Palpation: in the heart apex area or more laterally *diastolic tremor* ("cat's purring") can be detected .

Percussion: borders of heart are extended upward and to the right.

Auscultation: amplification of the 1^{st} heart sound ("flapping") and the

sound of the opening of the mitral valve above the heart apex in typical cases. This combination creates a characteristic melody – "quail" rhythm". Immediately after the opening sound protodiastolic murmur is heard. This murmur is of low timbre, gradually diminishing in intensity with presystolic increase. Mitral melody is well heard when the patient is standing upright, when holding breath in exhalation phase (after previous physical exertion). Pulse can be reduced on the left hand, resulting from compression of the subclavian artery probably by the enlarged left atrium.

X-ray study reveals the enlargement of the left atrium displacing the contrasted esophagus along the minor radial arch.

ECG data show the signs of the left atrium hypertrophy syndrome. The most informative ones are the appearance of the two-voltage R wave (0.07 sec. and more), extension of the area (negative) of the wave P phase in the lead V1. In pulmonary hypertension, the signs of right ventricular hypertrophy syndrome and later on those of the right atrium hypertrophy syndrome appear.

PCG. When registered above the heart apex, it can detect an amplified fluctuation of the 1st heart sound and retardation of the mitral valve closure, which lengthens the Q-1 sound interval up to 0.07s and more. High frequency sound of "opening" and 2nd sound interval shortening are registered. In the case of an increase in pressure in the lung capillaries, this interval shortens.

The course of disease. According to the evolution of hemodynamic disorders 3 periods are distinguished:

I period - *compensation* of defect by left atrium. Defects are usually detected by chance. Patients do not have complaints. The objective study reveals changes on auscultation and moderate ly enlarged left atrium.

II period - pulmonary hypertension and right ventricular hypertrophy.

III period - right ventricle insufficiency with congestive phenomema in the systemic blood circulation.

Echocardiography reveals:

1) morphological changes in the valves, i.e. the degree of mitral stenosis (area and dimensions of the left atrioventricular foramen/orifice);

2) degree of hypertrophy and expansion of the cavities of the left atrium and the right ventricle (systolic and diastolic chamber size, wall thickness);

3) effects of hemodynamic disturbances - pulmonary hypertension;

4) the presence of specific complications (thrombosis of the left atrium).

Classification of mitral stenosis by echocardiography data (G.M. Solovjov, 1990):

1st degree– critical stenosis: square of mitral foramen 1 – 1.6 cm^2.

2nd degree – marked stenoses: square of mitral foramen 1.7 – 2.2 cm^2.

3rd degree – moderate stenosis: square of mitral foramen 2.3 – 2.9 cm^2.

4th degree – slight stenosis: square of mitral foramen 3 cm^2.

Normally the cross-section of the mitral foramen/orifice is 4 – 6 cm^2.

Aortic valve insufficiency

Etiology. Aortic valve insufficiency develops due to the following causes: 1) rheumatic fever (80% of all cases of aortic insufficiency); 2) infectious endocarditis; 3) syphilis; 4) atherosclerosis of the aorta; 5) congenital anomalies, usually combined with lesion of the other valves.

Complaints. A feeling of enforced carotid artery pulsation and heartbeat appears. They usually become more intense on physical exertion. Pain similar to that of in *angina pectoris*, dizziness, feeling unwell and *tendency to faintness* are common. *Shortness of breath* occurs on reducing left ventricular contractility on physical exercise at first and then at rest, often resembling attacks of heart asthma.

Inspection reveals *skin pallor*. Shaking of the head is noted together with carotid artery pulsation (Musset's symptom). One of the characteristic symptoms is carotid artery pulsation ("dance" of carotids), pulsation of the subclavian artery, pulsation in the jugular fossa (the aortic arch), pulsation of the temporal and brachial arteries. The so-called *capillary pulse* also belongs to this group (change in color intensity of the nail bed).

Palpation. Intensified and diffuse apex beat is noted in the heart area. Apex beat is often in the 6th intercostal space and displaced to the left to the median axillary line due to abrupt dilatation of the left ventricle. On palpation in the jugular fossa pulsation of the aortic arch is distinctly detected. Pulsation of abdominal aorta is noted in the epigastrium.

Expansion of the ascending aorta causes an increase in the diameter of dullness of the vascular bundle. Pulse is high and accelerated, which depends on a rapid rise in pressure in the arterial system and on a rapid fall.

Auscultation of the heart gives the most informative signs. The 1st sound is dull. The 2nd sound diminishes or fades/disappears because of shrinking of the aortic valve cusps. Diminishing degree of the 2nd sound is proportional to the expression of valvular defect.

Diastolic murmur is the major ausultative sign. The murmur occurs immediately after the 2nd heart sound. It diminishes gradually in its intensity towards the end of diastole and has a mild blowing character.

The murmur is best heard in the 3rd – 4th intercostal spaces near the left border of the sternum. Such localization of murmur is typical of aortic insufficiency of rheumatic origin. When heart defect is a result of syphilis, murmur is of low timbre and is best heard in the second intercostal space to the right of the sternum.

Diastolic murmur is best heard in a sitting position with leaning forward during expiratory phase.

Mesodiastolic and/or presystolic murmur (Flint's murmur) is heard at the heart apex. It is caused by the fact that the reverse blood flow during diastole from the aorta into the ventricle is made with significant force and displaces the aortic cusp of the mitral valve, which causes relative stenosis of the mitral orifice.

In case of a marked valve defect, systolic BP increrases and diastolic BP decreases, so that the amplitude of pulse BP becomes enhanced.

X-ray study. The heart increases significantly at the expense of the left ventricle. The heart waist is clearly marked. The shadow of the aorta is usually diffusely enhanced, its pulsation amplitude as well as pulsation of the left ventricle also become enhanced and increase.

ECG signs of left ventricular hypertrophy.

In *rheumatic heart defect or* bacterial endocarditis, valve symptoms appear already in the period of forming the valve defect. There is no considerable dilation of the aorta. Heart failure develops on average several years earlier than aortic failure of another etiology. This is associated with myocardial damage by repeated rheumatic attacks.

In *heart defect of atherosclerotic* etiology, there are no vascular (peripheral) symptoms in particular. Diastolic BP is slightly decreased or not changed. Diastolic murmur is more clearly heard in the 2nd intercostal space on the right. The 2nd heart sound usually remains. Often, there is an accompanying systolic murmur. Hypertrophy of the left ventricle is not marked. There are signs of atherosclerosis of other vessels.

Syphilitic aortic failure is characterized by a combination of ischemic chest pain attacks, diastolic murmur and loud 2nd heart sound at the base of the heart, but this may be not present.

Relative insufficiency of aortic valve, observed in patients with the sharply dilated aorta due to atherosclerosis as well as high arterial hypertension is accompanied by safe or even amplified 2nd heart sound in the 2nd intercostal space on the right.

Echocardiography reveals:

1) morphological changes in the valves. The nature and extent of the valves lesion (divergent movement of mitral valve cusps, their thickening and the absence of closure in systole), degree of regurgitation are estimated;

2) degree of hypertrophy and enlargement the left ventricle (systolic and diastolic chamber size, wall thickness);

3) consequences of hemodynamic impairments – left ventricular dysfunction (ejection fraction, stroke volume).

Aortic stenosis (aortic mouth stenosis)

Etiology. Aortic stenosis is caused by 1) rheumatism; 2) atherosclerosis; 3) infectious endocarditis when a prompt antibiotic treatment prevents the aortic valves from destruction and the present valvular thrombotic clots/deposits are involved in scarring that leads to aortic stenosis; 4) congenital abnormalities of the valve and aortic mouth.

Complaints: *dizziness*, feeling unwell, *fainting*, *crushing pain* in the heart and behind the sternum on exertion. A combination of crushing pain in the heart with dizziness and fainting is quite typical. There is *shortness of breath* in reducing left ventricular contractility, cardiac asthma attacks as well as shortness of breath at rest. There may be increased fatigue.

Inspection reveals skin pallor. Apex beat increases in area and is shifted down (in the 6th intercostal space) and to the left (up to the anterior axillary line), which is due to the development of the left ventricle dilatation.

Palpation reveals systolic tremor in the 2nd intercostal space to the right of the sternum. **Percussion:** In marked poststenotic dilation of the ascending aorta, the expansion of the percussion borders of the vascular bundle is marked. In marked aortic stenosis, a decrease in systolic and pulse blood pressure is observed. Pulse is weak and slow. Bradycardia is frequently noted.

Auscultation. There is diminishing or disappearance of the 2nd heart sound as well as *systolic murmur* of low tembre of various intensity and

duration in the 2nd intercostal space and at Botkin's point. On listening, the murmur is intensified when a patient lies on the right side holding breath in exhalation phase. The murmur is heard in the jugular and supraclavicular fossa in the course of the carotid artery.

X-ray study. In marked defect, there is a progressive enlargement of the left ventricle and subsequently, the left atrium. Later, there is an enlargement of the right ventricle. There is also poststenotic dilation of the aorta. In some cases, there is calcification of aortic valve.

ECG shows signs of left ventricular hypertrophy of varying degrees. Quite early in the left precordial leads there are changes in the ST segment and the T wave.

PCG registers *systolic murmur of rhombic or fusiform form* in typical cases, which starts after a short interval after the 1st heart sound and ending before the beginning of the 2nd heart sound. The epicenter of the murmur is the 2nd intercostal space on the right of the sternum. Aortic component of 2nd heart sound is considerably diminished. In half of the cases a "systolic click" (ejection sound) is noted at the heart apex. These are several short vibrations in 0.04-0.06 seconds after the beginning of the 1st heart sound.

Echocardiography reveals:

1) morphological changes in the valves - the degree of aortic stenosis (area and size of the aortic foramen/orifice), normal cross-section of the aortic foramen/orifice is 3 cm^2;

2) degree of hypertrophy and enlargement of the cavities of the left atrium and left ventricle (systolic and diastolic chamber size, wall thickness);

3) effects of hemodynamic disturbances are pulmonary hypertension and left ventricular dysfunction (ejection fraction, stroke volume).

Tricuspid valve insufficiency

Etiology. The most common cause of this heart defect is rheumatism, infectious endocarditis being much less common. Clinically, it is determined by reduction of emission of blood from the right ventricle and stasis/congestion development in the right atrium and hollow veins/venae cavae.

Complaints. *Shortness of breath* in such patients is moderate because stasis in the pulmonary circulation is decreased and a part of blood is stored in the right parts of the heart and liver. Physical activity is unlikely to be limited to increasing breathlessness, but rather by abrupt weakness. There

are frequent pains in the right hypochondrium and epigastrium, nausea, decreased appetite. Accompanying ascites leads to a feeling of heaviness and distending pain around the abdomen.

Inspection. In most cases, there is *acrocyanosis* with jaundiced hue. Venous congestion is especially evident *in bulging the neck veins and their systolic pulsation*, the so-called *positive vein pulse*. Unlike pulsation of the carotid arteries in case of aortic valve insufficiency, venous pulsation has a lesser amplitude and almost unobtainable. Differentiation becomes easier with simultaneous study of pulse on the radial artery. If arterial pulse is weak and pulsation of the cervical vessels is marked, the latter is due to the venous pulsation.

Palpation reveals a cardiac beat and marked epigastric pulsation.

Percussion reveals displacement of relative heart dullness border to the right which is due to a considerable enlargement of the right ventricle and right atrium.

Auscultation reveals a systolic murmur, which is most clearly heard at the base of the xyphoid process. Sometimes this murmur is better heard in the 5th intercostal space along the midclavicular line. Systolic murmur is usually amplified on maximum inhalation (Rivero-Corvallo's sign), which is explained by increased regurgitation and acceleration of blood flow through the right heart divisions. The 1st heart sound is usually diminished.

X-ray study shows a considerable heart enlargement due to the enlargement of the right atrium and right ventricle.

ECG shows signs of hypertrophy of the right atrium and right ventricle. There is a right ventricle dilatation syndrome in the form of low amplitude of the QRS compex in V1lead and the S wave of enhanced amplitude in V2-6 leads.

Echocardiography reveals:

1) morphological changes in the valves - the nature and extent of valve lesion, divergent movement of the tricuspid valve cusps, their thickening and the absence of closure in systole;

2) degree of hypertrophy and enlargement of the right ventricle (systolic and diastolic size, wall thickness);

3) effects of hemodynamic impairments are right ventricular dysfunction (ejection fraction, stroke volume).

4.2.5. Chronic heart insufficiency (CHI)

E. Braunwald believes that CHI can be defined as "a pathophysiological condition in which the heart dysfunction leads to myocardial failure to pump blood with the speed necessary to meet the metabolic needs of the tissues". The main causes of CHI are coronary heart disease, hypertension and symptomatic arterial hypertension, cardiomyopathy, congenital and acquired heart disease, cardiac arrhythmias.

Classification is divided into two parts (see Table 33). The right one is functional. The left one is structural (morphological). They are specifically located in the form of steps to emphasize originality and independent significance of each of the parts.

Table 33

Classification of CHI

Stages of CHI	Functional classes of CHI (can be changed during the treatment)
I. The initial stage of heart disease (lesion). Hemodynamics is not damaged. Hidden heart failure. Asymptomatic dysfunction of LV	I. No limitation of physical activity: habitual physical activity is not accompanied by fatigue, shortness of breath or palpitations. A patient tolerates increased exercise, but it may be accompanied by shortness of breath and/or slow functional restoration.
II A. Clinically marked disease stage (lesion) of the heart. Hemodynamic disorders in one of the circles of blood circulation, they are moderately marked. Adaptive remodeling of the heart and blood vessels	II. Slight limitation of physical activity: absence of symptoms at rest, habitual physical activity is accompanied by fatigue, shortness of breath or palpitations
II B. Clinically marked disease stage (lesion) of the heart. Marked disturbances in hemodynamics in both blood circles. Maladaptive heart and vascular remodeling	III. Marked limitation of physical activity: absence of symptoms at rest, physical activity is less intense compared with habitual loads and is accompanied by symptom
III. The final stage of heart failure. Marked changes in hemodynamics and heavy (irreversible) structural changes in target organs (heart, lungs, blood vessels, brain, kidneys). The final stage of organs remodeling	IV. Inability to perform any physical activity without discomfort; heart insufficiency symptoms are present at rest and become worse on minimum physical exertion.

Clinical manifestations in the lesion of the right or left heart divisions

Left ventricle failur occurs in lesion and overload of the left heart. Congestion phenomena in the lungs such as shortness of breath, attacks of cardiac asthma and pulmonary edema and their radiographic signs, pulse acceleration develop in mitral heart defect, severe forms of ischemic heart disease, cardiomyopathies, myocarditis. Left ventricle failure in output manifests a decrease in cerebral blood flow (dizziness, blackouts, syncope) and coronary diseases (angina). It is typical of aortic heart defects, coronary heart disease, arterial hypertension, obstructive cardiopathy. Both types of left ventricular failure can be combined with each other.

Right ventricle failure occurs in overload or lesion of the right heart divisions. Congestive right ventricle failure (swelling of the jugular/neck veins, high venous pressure, cyanosis of fingers, nose, ears, chin, enlargement of the liver, slight jaundice, edema of various degree) usually joins congestive left ventricle failure. It is typical of the defects of the mitral and tricuspid valve, constrictive pericarditis, myocarditis, congestive cardiomyopathy, severe ischemic heart disease. With its signs being detected mainly on X-ray and ECG, right ventricle failure of output is characteristic of pulmonary artery stenosis of pulmonary hypertension.

Dystrophic form is the terminal stage of right ventricle failure with cachexia (exhaustion of the whole body), dystrophic changes in the skin (thinning, glittering, flatness of the picture, flabbiness), swelling up to anasarca (total swelling of the skin and body cavities), lower protein (albumin) levels in blood, disturbance of water-salt balance of the body.

Breathlessness of inspiratory type or, according to James Mackenzie, "lust for air", in patients with CHI is of complicated origin. The cause of breathlessness is hypoxemia and increased pressure in the lung capillaries due to blood stasis. A sense of breathlessness depends largely on diffusion capacity of the lungs (the severity of breathlessness depends on the severity of hypoxemia), the reaction of the central nervous system to change in the blood composition (hypoxemia, hypercapnia, acidosis etc.), the condition of the peripheral and respiratory muscles and the patient's weight. Fluid accumulation in the pleural and abdominal cavities contributes to shortness of breath impeding respiratory excursion of the lungs.

Cardiac asthma is an abrupt suffocation/gasping of a "cardiac" patient. An attack of marked cardiogenic breathlessness reaching the point

of suffocation indicates acute left ventricle congestive heart insufficiency. Cardiac asthma can occur at any time of the day, but is more likely to develop at night in a horizontal position. In this case, there is blood discharge from the depot. The so-called hidden edema represented by extracellular fluid accumulated in the tissues during the day mostly by the lower half of the body due to increased venous pressure passes to the blood flow. There is also weakening of the respiratory function, decreasing gas exchange, arousal of the vagus nerve and bronchoconstriction. The patient wakes up (if he could fall asleep before this) usually after nightmares with a feeling of breathlessness, chest tightness, fear of death, and forced to sit in bed. He is afraid to move, holds the bedside, breathes slowly or fast (respiratory movements are not restricted). Often cough with serous mucus develops. On decreasing right ventricular contractility and associated tricuspid valve insufficiency, recurrence of cardiac asthma usually stops or its frequency is reduced, that is, with the progression of heart failure from IIA to IIB stage choking attacks become fewer.

Cough is dry or slightly productive (in patients with acute left ventricular heart failure mucus is usually not viscous, easily produced in the form of liquid or foamy liquid). Cough as a reflex act is due to the swelling of the mucous membrane of the congestive bronchi or irritation of the recurrent nerve or the enlarged left atrium. Blood overflow of the small vessels of the lungs may be accompanied by the diapedesis of red blood cells or even slight hemorrhage and hemoptysis (mucus in blood).

Early fatigue is present in most patients even with initial CHI. Fatigue is associated with impaired blood supply to the skeletal muscles.

Palpitation is interpreted like feeling every heart contraction by the patient. Often palpitation is present in tachycardia (hence a synonym is heart "race") but it can be present in normal heart rate and even in bradycardia. In the initial stages of CHI, heart rate at rest is within normal range. Tachycardia occurs only on physical exercise but, unlike a physiological increase in heart rate in patients with heart failure, it returns to normal not after stopping exercising but in 10 minutes or later. With progression of CHI palpitations and tachycardia are noted at rest. In patients with CHI, tachycardia is due to humoral (activation of the sympathoadrenal system etc) or reflectory (Bainbridge's reflex) effects on the heart, but sometimes it can be associated with taking medicines (e.g. nitrates or calcium antagonists), abuse of strong coffee, tea, tobacco.

Feeling of heaviness in the right subcostal area in patients with right ventricle heart insufficiency usually precedes the appearance of edema because it is the liver which reacts first to the right heart failure. These symptoms are due stretching of the liver capsule in case of overflow of hepatic veins and capillaries with blood. With progression of CHI, prolonged stagnation (muscat liver, cardiac cirrhosis) symptoms of liver dysfunction appear, i.e. yellow color of skin (skin icterus) and mucous membranes. Portal hypertension occurs. First, a patient is worried about the feeling of abdomen bloating/distension and overfilling. Then, a patient observes an enlargement of the abdomen (due to accumulation of ascitic fluid).

Nausea, vomiting, loss of appetite, constipation, bloating/flatulence and other symptoms of gastric and intestinal dyspepsia are almost constant companions of congestive heart failure. Gastrointestinal functions in CHI are impaired as a result of hypoxia and reflex impact.

Patients with CHI complain about **reduced mental performance and decreased mood, irritability, insomnia at night and drowsiness during the day**. These complaints are related to with an early change in functional status of the central nervous system following blood circulation disorder.

Present history. It is important to find out features and sequence of the onset of patient's symptoms. It is always important to note the time of occurrence of each symptom and its association with the suspected time of heart disease, possible provoking factors of first manifestation and exacerbations of CHI, guidelines on treatment interference and its effectiveness.

Past history. You must ask the patient about the physical and intellectual development, past diseases, bad habits, living conditions, adverse factors at work. It is important to get information about the patient's parents, I, II degree relatives, find out their age, diseases, and if they died, from what and at what age.

Objective examination data

Orthopnea is a high degree of shortness of breath with forced (semirecumbent or sitting) position of the patient. Patients with severe CHI often sit in a chair or on the bed, with lowered legs, leaning forward, resting on the back of a chair or they occupy a semi-recumbent position on the cushions (a high headrest is made using several cushions or a rolled mattress). Any attempt to lie exacerbates shortness of breath. Sleepless nights can last

for weeks until the patient gets some relief from therapy. This phenomenon is especially characteristic of the left heart failure. Orthopnea is due to the fact there is blood movement in the vertical position with deposits in the veins of the lower part of the trunk and extremities along with a reduction in venous return to the right atrium and, consequently, the pulmonary blood circulation becomes less full of blood. Respiratory function in the vertical position is improved by creation of better conditions for movement of the diaphragm as well as for the work of the auxiliary respiratory muscles. Orthopnea usually disappears (or becomes much less marked) on escalating right ventricle heart insufficiency secondary to left ventricular heart insufficiency.

"Corvisart's face" is common in refractory chronic cardiac insufficiency. The face is doughy/puffy of yellow pale colour with a bluish shade. Patient's facial expression is apathetic, indifferent, drowsy, with half-closed dull eyes and constantly parted and cyanotic lips.

Cyanosis (skin blueness) is considered a frequent sign of CHI. Cyanosis in patients with CHI is associated with a decrease in blood flow rate and an increase in oxygen absorption by the tissues as well as insufficient blood oxygenation in the pulmonary capillaries, resulting in increased restored blood hemoglobin (it is of blue color). The first manifestations of cyanosis in patients with CHI are called acrocyanosis, i.e. cyanosis of body parts located far from the heart (tip of the nose, earlobes, lips, finger nails, toe nails). Acrocyanosis is due mainly to the slowing of blood flow and, therefore, it is of peripheral character (often referred to as peripheral cyanosis).

Pallor of skin and mucous membranes in patients with CHI can be combined with cyanosis (so-called "pale cyanosis") in aortic heart lesions (aortic stenosis, insufficiency of the aortic valve), collapse, profuse bleeding, infectious endocarditis. In stenosis of the mitral orifice, pallor is combined with violet-red blush on the cheeks, i.e. "mitral butterfly".

Jaundice is yellowish color of the skin and mucous membranes (especially sclera) in patients with severe chronic right ventricle heart failure due to development of congestive fibrosis ("cardiac cirrhosis") in the liver.

Cardiac cachexia. Significant weight loss and cachexia development are noted at advanced CHI stage. In case of treated cardiac decompensation, this indicates the final/ultimate (irreversible) stage of the disease development. Cachexia results from:

1) metabolic activation under the influence of additional work performed by the respiratory muscles;

2) lack of appetite, nausea and vomiting caused by central disorders or long-lasting hepatomegalia;

3) malabsorption in the guts caused by interstitial stasis in the veins.

Registered unintentional weight loss of at least 5 kg or more than 7.5 per cent from the original "net" weight (without edema) during the last 6 months may suggest the development of pathological weight loss.

Edema in patients with CHI can be already determined on general inspection. Dependency of edema on the body position is noted. Edema goes from downward upward. In the early stages when the patient is upright (NB edema in the lying patient start spreading from the lumbar/loin area), there is swelling only on the dorsal surface of the feet. It appears in the evening and disappears in the morning (a typical complaint is "shoes become tight in the evening"). Edema spreads on the ankle joints (near the condyles), then spreads over the shin and above. Further, swelling of the feet become permanent and they spread over the lower abdomen and lower back till anasarca develops. In long-lasting edema, trophic skin changes, fissures, dermatitis develop.

Physical examination of the respiratory organs in patients with chronic heart insufficiency can detect signs of pulmonary congestion with moist and dry râles as well as the presence of fluid in the pleural cavity. Dull moist râles predominantly over the small basal lung divisions (especially on the side on which the patient lies) in patients with left ventricular heart insufficiency are associated with high blood pressure in the lung capillaries and veins as well as with inconsiderable accumulation of secretions in the small bronchi lumen (fine bubbling râles). In addition to the moist râles, patients with extremely marked decompensation of the left ventricle and cardiac asthma may have dry high-pitched râles caused by full blood supply of the bronchial mucosa and accumulation of viscous transudate in the bronchial lumen. Increase in pleural capillary pressure in CHI and penetration of fluid in the pleural cavity leads to accumulation of pleural effusion. It occurs more often in the right pleural cavity than in the left one. This can be determined using ordinary physical diagnostic techniques (lag/delay in the affected part of the chest in the act of breathing, sharp weakening or absence of voice vibration over the fluid accumulation area,

determineation of dull sound or absolute dullness by percussion, diminution/weakening or absence of breath and bronchophony on ausculatation).

Swelling and pulsation of neck vein is present in CHI. Total venous congestion occurs. In patients with CHI pulsation of jugular veins (venous pulse) can be noted in the neack area. If the outflow of venous blood into the right atrium is difficult, vein pulse is detected on usual inspection (the pathological venous pulse).

Palpation and percussion of the heart in patients with CHI can detect signs of cardiomegaly, namely, the shift of the apex beat outwards from the left midclavicular line and below the fifth intercostal space; diffuse (more than 2 sm^2) character of the apex beat; a cardiac beat (pulsation of increased right ventricle on the left side of the sternum spreading to the epigastric region); expanding the borders of relative heart dullness.

Auscultation can reveal tachycardia, arrhythmia, diminished sonority of heart sounds and protodiastolic gallop rhythm caused by pathological 3rd heart sound, which are often heard in CHI. Listening to heart murmurs may be the key to diagnosing heart defect underlying CHI.

Systemic BP is elevated in patients with poorly controlled arterial hypertension, but the later stage of the disease is characterized by low blood pressure with low pulse pressure.

Hepatomegaly is the first symptom of congestive liver and represents the classic manifestation of the right ventricle failure.

At the initial stage of right ventricle heart insufficiency the liver, which is painful on palpation only slightly protrudes from under the costal arch. Its border is round and smooth. The surface is smooth. Its size variability is typical. It is related to hemodynamics condition and treatment efficacy. In the future, the lever can be of a big size, can "fall" below the crest of the iliac bone. The liver border is sharpened, the surface becomes dense. Pain intensity can reduce on palpation.

Ascites in a patient with right ventricle heart insufficiency develops as a result of transudation of fluid from veins in the liver and peritoneum. Usually, massive ascites is diagnosed in patients with tricuspid valve lesion or chronic constrictive pericarditis. In vertical position, the patient's abdomen in marked ascites looks flabby whereas in horizontal position, the abdomen is like a "frog's belly". In the so-called tense ascites, the form of the abdomen does not much depend on the patient's position (the amount of fluid in

the abdomen is so high that it doesn't move). In patient's upright position, the protruded navel makes it possible to distinguish between the enlarged abdomen in ascites and that of in marked obesity.

SELF-TEST "EXAMINATION OF A PATIENT WITH RESPIRATORY DISEASES"

1. Lobar pneumonia is characterized by (more than one answer is possible):

a. moist sonorous fine bubbling rales
b. dull sound
c. vocal fremitus
d. cough with sputum
e. the presence of Charcot-Leyden's crystals in the sputum

2. After disclosure abcess is characterized by (more than one answer is possible):

a. retardation of the affected part of the chest in the act of breathing
b. sputum expectoration in the morning
c. vocal fremitus
d. moist large bubbling râles
e. bronchial breathing

3. Inspiratory dyspnea is observed in (more than one answer is possible):

a. asthma
b. foreign body in the trachea
c. chronic obstructive bronchitis
d. hydrothorax

4. The increase in the volume of one half of the chest is observed in (only one answer is possible):

a. obstructive atelectasis
b. lobar pneumonia
c. pneumosclerosis
d. pneumothorax
e. bronchial asthma

5. Compression atelectasis is characterized by (more than one answer is possible):

a. crepitation
b. dull tympanic sound
c. bronchial breathing
d. expiratory dyspnea
e. bronchophony

6. Induration syndrome of the lung tissue is characterized by (more than one answer is possible):

a. tympanic percussion sound
b. dull percussion sound
c. vocal fremitus
d. bronchophony
e. lag/delay of the affected part of the chest in the act of breathing

7. "Orthopnea" reduces (only one answer is possible):

a. pain in the heart
b. hypertonic crisis
c. edema of the lower extremities
d. dyspnea
e. headache

8. Which is observed in the second stage of lobar pneumonia? (more than one answer is possible)

a. weakened vesicular breathing
b. bronchial breathing
c. bronchophony
d. vocal fremitus
e. dull percussion sound

9. Inspiratory dyspnea is observed in (more than one answer is possible):

a. bronchial asthma
b. massive hydrothorax
c. chronic obstructive bronchitis
d. lobar pneumonia
e. tumor of the trachea

10. Transudation in the pleural cavity occurs in: (more than one answer is possible):

a. tuberculosis
b. heart failure
c. kidney insufficiency
d. chronic bronchitis
e. lobar pneumonia

SELF-TEST "EXAMINATION OF THE PATIENT WITH DISEASES OF GASTROINTESTINAL TRACT"

1. Hyperpigmentation of urine and feces are noted in (only one answer is possible):

a) hemolytic jaundice
b) hepatic jaundice
c) obstructive jaundice

2. Hypoacid state is characterized by (only one answer is possible):

a) heartburn
b) constipation
c) diarrhea

3. Hyperacid state is characterized by (only one answer is possible):

a) dysphagia
b) constipation
c) diarrhea
d) jaundice

4. Cholehemia occurs in (only one answer is possible):

a) hemolytic jaundice
b) cholecystitis
c) obstructive jaundice
d) pancreatitis

5. "Vascular asterisks" are characteristic of (only one answer is possible):

a) cirrhosis of the liver
b) cholecystitis
c) pancreatitis
d) gastritis

6. Hemorrhagic syndrome in liver disease is a manifestation of (only one answer is possible):

a) hepatocellular insufficiency
b) portal hypertension
c) biliary dyskinesia
d) cholangitis

7. Gynecomastia in liver disease is a manifestation of (only one answer is possible):

a) hepatocellular insufficiency

b) portal hypertension
c) biliary dyskinesia
d) cholangitis

8. Splenomegaly in liver disease is a manifestation of (only one answer is possible):

a) hepatocellular insufficiency
b) portal hypertension
c) biliary dyskinesia
d) cholangitis

SELF-TEST "EXAMINATION OF THE PATIENT WITH CARDIOVASCULAR DISEASES"

1. Where is normally the upper boundary of the relative bluntness of the heart? (only one answer is possible)

a) at the level of 2nd rib
b) in the 2nd intercostal space
c) at the level of 3rd rib
d) in the 3rd intercostal space
e) at the level of 4th rib

2. Where pulmonary valve is auscultate? (only one answer is possible)

a) the xiphoid process
b) 2nd intercostal space to the right of the sternum
c) 2nd intercostal space to the left of the sternum
d) at the heart apex
e) at Botkin-Erb point

3. What characterizes a complete atrio-ventricular block? (only one answer is possible)

a) increase in PQ interval
b) absence of the P wave
c) absence of relation between the rhythm of the atria and ventricles
d) normal PQ interval

4. Where the aortais auscultated? (only one answer is possible)

a) the xiphoid process
b) 2nd intercostal space from the sternum to the right

c) 2nd intercostal space to the left in the sternum

d) at the heart apex

5. What is the 1st heart sound in auscultatory picture of the heart in aortic stenosis: (only one answer is possible)

a) 1st amplified sound at the apex

b) 1st diminished sound at the apex

c) 1st sound does not change

6. What murmur is auscultated in aortic stenosis? (only one answer is possible)

a) systolic murmur at the apex

b) diastolic murmur at the apex

c) diastolic murmur at the aorta

d) systolic murmur at the aorta

7. What is the normal width of the vascular bundle? (only one answer is possible)

a) 3-4 cm

b) 5-6 cm

c) 6-8 cm

8. When is there a positive venous pulse? (only one answer is possible)

a) in stenosis of the aorta

b) in mitral valve insufficiency

c) in tricuspid valve insufficiency

d)in aortic insufficiency

Keys to self-test "Examination of the patient with respiratory diseases"

1. a, b, c, d
2. a, b, c, d, e
3. b, d
4. d
5. a, b, c, e
6. b, c, d
7. a
8. b, c, d, e
9. b, d, e
10. b, c

Keys to self-test "Examination of the patient with diseases of gastro-intestinal tract"

1. a
2. c
3. b
4. c
5. a
6. b
7. a
8. b

Keys to self-test "Examination of the patient with cardiovascular diseases"

1. c
2. c
3. c
4. b
5. b
6. d
7. b
8. c

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