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The main methods of examination of patients in clinic internal diseases

Manual for the third-year students of the international faculty

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The textbook is devoted to the discipline definition, the problems of diagnosis, and fundamentals of diagnostic process: history taking, inspection, and physical examination of the respiratory, cardiovascular, digestive systems.

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MODULE 1. THE BASIC METHODS OF EXAMINATION OF PATIENTS IN CLINIC OF INTERNAL DISEASES

Theme 1. **The Scheme of a Case History. An Anamnestic Part of a Case History.**

Medical case history: its basic sections and rules of drawing up. A technique of inquiry of the patient, its diagnostic value, its carrying out in view of individual, intellectual and of psychological features of the patient. The basic structural parts of the anamnesis (a passport part, inquiry on organs and systems, the anamnesis of a life). A role of the Ukrainian and Russian scientist-clinicists in development of professional art of inquiry of the patient.

The contents: *Medical case history*: its basic sections and rules of drawing up. A technique of inquiry of the patient, it's diagnostic value, it's carrying out in view of individual, intellectual and of psychological features of the patient. The basic structural parts of the anamnesis (a passport part, inquiry on organs and systems, the anamnesis of a life). A role of the Ukrainian-clinicists in development of professional art of inquiry of the patient.

The medical case history includes several parts, such as:

1. Passport part
2. Complaints
3. Interrogation of systems
4. Anamnesis of disease
5. Anamnesis of life
6. Objective examination
7. Suppositional diagnosis
8. Facts of additional investigation
9. Terminal clinical diagnosis
10. Treatment
11. The dairy observation
12. Epicrisis

Pain or nausea is subjective symptoms experienced by the patient. These sensations reflect objective changes that occur in the patient's body. Signs of the disease that are revealed by the physician during his examination of the patient, e.g. jaundice or enlarged liver, are objective symptoms of the disease. It is almost impossible to diagnose a disease by only one symptom. A correct diagnosis can only be established by investigating several symptoms. Most incorrect diagnoses are the result of an insufficient examination. The main requirement is therefore a thorough and systematic examination of the patient. A correct diagnosis can be established if the physician follows a definite plan in his examinations.

The examination begins with an interview. The patient tells his complaints which often are of no less importance than a thorough objective examination of the patient. Some diseases are diagnosed almost exclusively by the patient's complaints. *Angina pectoris* for example, is frequently diagnosed almost entirely from the character of pain in the region of the heart. Cholelithiasis is diagnosed by attacks of pain in the right upper abdominal quadrant. A detailed questioning of the patient concerning the time of the onset of the disease, its early symptoms (until the time of medical examination) is even more important in establishing a correct diagnosis. All this information is usually called anamnesis morbi, i.e. remembering the present disease by the patient, as distinct from anamnesis vitae which is the history of previous diseases of the patient. Stages of development of the present disease are traced back while collecting an anamnesis from the history of the disease as given by the patient himself and also from the information supplied by his relatives.

Another stage of examination is objective examination of the patient's condition at the present time (status praesens). This examination includes various diagnostic procedures (inspection, measuring temperature, percussion, auscultation, palpation, laboratory tests, X-ray examination, etc.) and reveals changes in the patient's body and deviations from normal structure and function of various organs that could not be sensed by the patient himself.

As a rule, a patient undergoes repeated examinations during his observation by medical personnel. These examinations reveal subjective and objective changes in the state of the patient, the disease progression, and the efficacy of the therapy given. This is information on the course of the disease (decursus morbi).

All information obtained by questioning the patient and by objective examinations, information on the course of the disease and the prescribed treatment are recorded to make a history of the case. A complete diagnosis (i.e. the main disease and the accompanying diseases and complications, if any) are given on the first page. At the end of the history record, when observation of the patient is over, a conclusion or epicrisis should be given, where the special character of the disease and the result of the treatment should be described.

Interrogation of systems

The art of collecting a correct anamnesis is not easy. The reliability of complaints related by patients varies. Some patients forget to mention the most important symptoms while others tell on unimportant and irrelevant details. The history will therefore be incomplete if the patient is allowed to tell the history of his illness by himself (unguided by the physician). It should be remembered that some patients may be shy and do not readily talk about some diseases (e.g. venereal) or harmful habits (alcoholism).

In order to collect information that might be actually important for a correct diagnosis, the physician should know the symptoms of the disease and the character of its progress. These are the subject matter of special pathology and therapy of internal diseases. The physician must learn the art of correct inquiring. The science of diagnostics concerns the art of the correct and systematic collecting of anamnestic data. The physician first collects general information about the patient: his name, age, place of birth, and occupation. Age is important in the development of some diseases, e.g. essential hypertension, atherosclerosis, and malignant tumors that commonly develop in the aged. Occupation and social status of the patient are often responsible for the onset of the disease (e.g. poisoning, chills, etc.).

The next step is a systematic and thorough functional inquiry of the patient according to a predetermined scheme.

The Present Complaints

The main complaints of the patient should first be determined. If the patient complains of retrosternal pain, the character and exact location of this pain, its focus and intensity should be determined; the time of the onset, and possible causes that provoked the pain (strain, cough, taking food, etc.) should be established. The patient should be asked which remedies remove this pain. Other complaints should also be analysed. In pneumonia, for example, the patient would normally complain of weakness, high temperature, side pain (pleurodynia), and cough; he would note that the onset of the disease was marked a few days ago by a sudden chill and pricking in the side when coughing and breathing deeply.

The study of the main complaints can often lead the examiner to a conclusion concerning the general character of the disease, e.g. high body temperature would normally indicate an infectious process, cough and expectorated sputum indicate possible disease of the lungs. Knowledge of the exact time of the onset of the disease is informative of the character of the disease (acute or chronic).

The inquiry should not be limited to these main points. So as not to omit any symptoms and determine the functional condition of all the organs (status functionalis) the patient should be questioned according to a specially outlined scheme. Changes in the patient's general state should be established (loss of weight, fever, weakness, edema, headache). The condition of the respiratory system (cough, expectoration of sputum and blood, pain in the throat) should also be established. Next is the cardiovascular system (tachycardia, dyspnoea, heart pain, swelling of feet). Then follows the gastrointestinal system (appetite, swallowing, vomiting, epigastric pain, etc.).

The condition of the nervous system is established by asking the patient about his subjective condition, his sleep, irritability or indifferent attitude to the surroundings, weakness, excitement, headache, state of consciousness and the main senses. The patient should be asked about his conduct, responses to external stimuli, his attitude to work and his associates. This is necessary to establish the special properties of his higher nervous activity at the present time and in the past, and the type of his

nervous system according to Pavlov's classification. The inquiry at this stage gives the physician information concerning the condition of various organs and systems of the patient (respiration, blood circulation, digestion, urinary function, motor function, nervous system, etc.).

Anamnesis of the Disease

Exact answers should be obtained from the patient concerning the following aspects of his present disease (anamnesis morbi): (1) the time of the onset of the disease; (2) the character of the first symptoms; (3) the course of the disease; (4) examinations and their results, if any; (5) treatment, if any, and its efficacy. The answers to these questions may give the physician the necessary information on the present disease.

The history of the disease should include information concerning the onset of the disease and its development until the present. The patient's general condition before the disease should first be determined and the causes that might have provoked the disease established wherever possible. The patient should be questioned in detail about the first signs of the disease and the chronology of their development (dynamics), about relapses or exacerbations, remissions and their duration. If the patient was examined during an exacerbation of the disease by some other physician, the results should be studied. Excess verbosity of the patient should be prevented, because the results of the examinations and treatment only are important (therapy with cardiac glycosides, vasodilators, diuretics, antibiotics, hormones, etc.). Motives for hospitalization should also be determined (exacerbation of the disease, verification of the diagnosis, etc.).

Anamnesis of Life

The past history is often very important for establishing the character, the cause, and conditions for the onset of the disease. Anamnesis vitae is a history or a medical biography of the patient in every period of his life (infancy, childhood, adolescence, and maturity).

Collecting the anamnesis begins with the general biographical information. Birth place is important, because some diseases (e.g. endemic goiter) usually predominate in one locality and are not met in others. The age of he parents is also important. The patient should be asked if he was born at term, if there were other children in the family, if he was breast fed or artificially; the age at which the patient began walking and talking is important, and the patient should inform the physician if he had marred signs of rickets during his childhood. This information is important to evaluate the patient's health at birth and during childhood. Conditions of life in childhood and adolescence and health during these periods of life are important information. It is necessary to find out if the patient's physical and mental development was retarded and what was his progress at school. The time of sex maturity should be determined. Women should report the number of pregnancies and parturitions, and the course of labor.

Social conditions are important, for the health of people. The patient should inform the physician on the conditions of his housing (separate apartment, hostel, country house, illumination, the presence of dampness, if any, hygienic conditions, etc.). The composition of the family is important: large or small family, their health, well-being, income etc. Malnutrition is an important factor for the onset of some diseases. The patient should be asked if his diet is sufficiently rich in vegetables. fruits, etc. The way in which the patient spends his leisure time is also important. The patient should report on the time he sleeps, rests, walks in the fresh air, and what sports and exercises he goes in for.

Unfavorable labor conditions and industrial hazards (some harmful dusts) are important, for they may cause bronchial asthma and chronic diseases of the bronchi and lungs. Strong noise, vibration, high ambient temperature, drafts, and cold (work in the open) can cause pathology. Industrial poisoning by mercury, lead, carbon monoxide and other harmful agents, and also exposure to radiation (improper safety measures) may also cause disease. The working schedule is also important. Establishing whether there are unfavorable industrial factors helps the physician give recommendations for organization of the patient's work.

Past illnesses are also important. Some infectious diseases, such as measles or scarlet fever, do not recur because of acquired immunity, while other diseases, such as rheumatism or erysipelas, tend to recur. Rheumatism or diphtheria often provoke heart diseases. Nephropathy often develops after scarlet fever, and incompetence of heart valves often results from the previous endocarditis.

It should be remembered that the patient may not know about his past diseases. Therefore in dubious cases the physician should ask the patient whether he had certain symptoms by which a suspected past disease might manifest itself (e.g. prolonged fevers, swelling of and pain in the joints are characteristic of rheumatism, general edema indicates kidney disease, attacks of right hypochondriac pain may be the cause of the gall bladder disease, etc.). Contacts with infectious patients are important, especially in the presence of epidemics (e.g. influenza).

Family history. Health of the parents, sisters or brothers is often informative. If some of the family have had tuberculosis, the other members of the family may also develop tuberculosis. Syphilis may be transmitted by an intrauterine route. By comparing the pathology of the patient with diseases of his relatives, the physician can make a conclusion on the role of hereditary factors in the development or origin of the disease.

Life of man is tightly connected with the environment, and pathology always depends on external effects. Harmful environmental factors may affect the patient's offspring: his children may be predisposed to some diseases. But this predisposition does not obligatory provoke the disease. Special conditions are usually required for the disease to develop, and if these special factors are absent the person will not develop the disease. Moreover, if conditions favour, the person may strengthen his health and eradicate the hereditary predisposition to an illness.

Hereditary or familial (genotypical) and non-hereditary (paratypical) diseases are distinguished. But this classification is only conventional. As genetics progresses it becomes more obvious that some diseases that would be considered to be resistant to the hereditary factors, are actually genotypical diseases. Internists mainly deal with diseases that are not usually transmitted to the offspring but merely predispose them to these diseases (e.g. essential hypertension, atherosclerosis, cholelithiasis, etc.). Under certain environmental conditions this predisposition may enable the person to develop the disease. It should be remembered that the inherited character may have varying expressivity, or hereditary disease may develop n one member of the family only, or it may be inherited by an offspring after several generations, or it may develop only in family members of one sex (e.g. only males develop hemophilia which is transmitted from a grandfather to a grandson through a healthy daughter). The onset of certain hereditary diseases is sometimes erroneously attributed to an external factor, which was actually only the stimulus that provoked the disease.

In order to establish the hereditary character of a disease, the familial factors should be first given a thorough clinical-genealogical analysis. For the sake of convenience, genealogical schemes should be made out, using he special conventional symbols.

According to the adopted terminology, the patient is called a proband. His brothers and sisters are given in the order of their birth, from left to right. The Roman numerals are used to designate (at the left) successive generations. Each member of the generation is designated by an Arabic numeral. Symbols designating the proband's relatives, who were affected by the same disease, are shaded. The diagram must include data concerning he disease occurring in both parental lines of the proband.

Three main types of inheritance have been established. The first, autosomal-dominant type, is the most prevalent. It is characterized by full penetrance of the mutant gene. In this type of inheritance the disease is directly transmitted from both parents to their offspring with 50 per cent of both sexes being affected. Those who do not inherit the mutant gene have normal offspring. If penetrance of the mutant gene is incomplete, the direct order of inheritance is more difficult to detect. Suspicion that an inherited disease is dominantly transmitted can be verified by analysis of the offspring from repeated marriages (affected children from each marriage). The direct order of inheritance occurs, for example, in subjects with anatomical abnormalities (of the internal organs included).

Heterozygote carriage of the recessive gene in autosomal-recessive inheritance does not cause the disease, which only develops in homozygote carriers. A blood relationship between parents is often revealed in recessive inheritance (otherwise difficult to reveal). Many enzymopathies, certain diseases of the nervous system, etc. are often inherited by the autosomal-recessive type.

Theme 2. General Inspection of the Patient. Inspection of Separate Parts of a Body.

Technique of carrying out of the general inspection of the patient. Definition of the general condition of the patient (a version of the general conditions of the patient and their criteria), an estimation of a condition of its consciousness (types of disturbances of consciousness), gaits (versions of a gait at a various pathology), positions in a bed (active, passive, forced and their kinds). A body build and the basic criteria of normal constitutional types. A skin, its properties (color, turgor, humidity, temperature, elements of a rash, nevuses, scars, cicatrixes) and pathological changes; an estimation of a condition of hair and fingernails. A hypodermic fat (fatness, distribution, types of an obesity), a condition of muscles and of a locomotorium. Sequence of a palpation of lymph nodes. Diagnostic value of the signs received during the general inspection of the patient. Technique of carrying out and sequence of inspection of a head and a neck, extremities and trunks, a stomach and a thorax. Diagnostic value of the signs received during inspection of separate parts of a body of the patient.

The contents: The patient should be examined in the daytime, because electric light will mask any yellow coloring of the skin and the sclera. In addition to direct light, which outlines the entire body and its separate parts, side light will also be useful to reveal pulsation on the surface of the body (the apex beat), respiratory movements of the chest, peristalsis of the stomach and the intestine. During the general inspection, the physician should pay attention to the open parts of the patient's body, the head, the face and the neck.

Inspection technique. The body should be inspected by successively uncovering the patient and examining him in direct and side light. The trunk and the chest are better examined when the patient is in a vertical posture. When the abdomen is examined, the patient may be either in the erect (upright) or supine (dorsal) position. The examination should be carried out according to a special plan, since the physician can miss important signs that otherwise could give a clue for the diagnosis (e.g. liver palm or spider angiomata which are characteristic of cirrhosis of the liver).

The appearance of the patient is described: general condition, consciousness, carriage, gait, position in bed, expression of the face, constitution, stature, skin, visible mucosa, and conjuctiva, subcutaneous fat, edemas, muscles, bones, joints, lymph nodes.

The general condition of the patient is characterized by the following signs: consciousness and the psyche, posture and body-built.

Consciousness. It can be clear or deranged. Depending on the degree of disorder, the following psychic states are differentiated.

1. **Stupor.** The patient cannot orient himself to the surroundings, he gives delayed answers. The state is characteristic of contusion and in some cases poisoning.
2. **Sopor**. This is an unusually deep sleep from which the patient recovers only for short periods of time when called loudly, or roused by an external stimulus. The reflexes are preserved. The state can be observed some infectious diseases and at the initial stage of acute uremia.
3. **Coma.** The comatose state is the full loss of consciousness with complete absence of response to external stimuli, with the absence of reflexes, and deranged vital functions. The causes of coma are quite varied but the loss of consciousness in a coma of any etiology is connected with the cerebral cortex dysfunction caused by some factors, among which the most important are disordered cerebral circulation and anoxia. Edema of the brain and its membranes, increased intracranial pressure, effect of toxic substances on the brain tissue, metabolic and hormone disorders, and also upset acid-base equilibrium are also very important for the onset of coma. Coma may occur suddenly or develop gradually, through various stages of consciousness disorders. The period that precedes the onset of a complete coma is called the precomatose state. The following forms of coma are most common.

Alcoholic coma. The face is cyanotic, the pupils are dilated, the respiration shallow, the pulse low and accelerated, the arterial pressure is low; the patient has alcohol on his breath.

Apoplexic coma (due to cerebral haemorrhage). The face is red, breathing is slow, deep, noisy, the pulse is full and rare.

Hypoglycaemic coma can develop during insulin therapy for diabetes.

Diabetic (hyperglycemic) coma occurs in non-treated diabetes mellitus.

Hepatic coma develops in acute and subacute dystrophy and necrosis of the liver parenchyma, and at the final stage of liver cirrhosis.

Uraemic coma develops in acute toxic and terminal stages of various chronic diseases of the kidneys.

Epileptic coma. The face is cyanotic, there are clonic and tonic convulsions, the tongue is bitten. Uncontrolled urination and defecation. The pulse is frequent, the eye-balls are moved aside, the pupils are dilated, breathing is hoarse.

4. **Irritative disorders of consciousness** may also develop. These are characterized by excitation of the central nervous system in the form of hallucinations, delirium (delirium furibundum due to alcoholism; in pneumonia, especially in alcoholics; quiet delirium in typhus, etc.).

General inspection can also give information on other psychic disorders that may occur in the patient (depression, apathy).

Attention should be paid to *gait* and *carriage*. In healthy person the carriage is straight, the gait is steady. An unsteady gate is observed in blood loss, nervous breakdown, high fever, cerebellar tumors, etc. A specific gait can be seen in some diseases. For example, in patients with ascites the carriage is proud, the upper part of the body is reclined, the abdomen is jutted out. In hemiplegia (paralysis of one half of the body) or paralysis of one lower extremity, the patient draws the leg making circular movements to the front and inside. In tabes dorsalis the gait is ataxic. The extremity is thrown out forward, having put it down the patient continues to feel for a stable support. In coccitis (inflammation of the coxofemoral joint) the leg is thrown out forward with the movement of the whole pelvis without bending in the hip joint. Similar gait is sometimes observed in lumbosacral radiculitis.

The posture or attitude of the patient is often indicative of his general tone, the degree of muscle development, and sometimes of his occupation and habits. Most patients with grave diseases or with psychic depression are often stooped. Erect posture, easy gait, and free and unconstrained movements indicate the normal condition of the body.

Position of the patient. It can be active, passive, or forced.

1) The patient is *active* if the disease is relatively mild or at the initial stage of a grave disease. The patient readily changes his posture depending on circumstances. But it should be remembered that excessively sensitive or alert patients would often lie in bed without prescription of the physician.

2) *Passive posture* is observed with unconscious patients or in rare cases, with extreme asthenia. The patient is motionless, his head and the limbs hang down by gravity, the body slips down from the pillows to the foot end of the bed.

3) *Forced posture* is often assumed by the patient to relieve or remove pain, cough, dyspnoea. For example, the sitting position relieves orthopnoea: dyspnoea becomes less aggravating in cases with circulatory insufficiency. The relief that the patient feels is associated with the decreased volume of circulating blood hi the sitting position (some blood remains in the lower limbs and the cerebral circulation is thus improved). Patients with dry pleurisy, lung abscess, or bronchiectasis prefer to lie on the affected side. Pain relief in dry pleurisy can be explained by the limited movement of the pleural layers when the patient lies on the affected side. If a patient with lung abscess or bronchiectasis lies on the healthy side, coughing intensifies because the intracavitary contents penetrate the bronchial tree. And quite the reverse, the patient cannot lie on the affected side if the ribs are fractured because pain intensifies if the affected side is pressed against the bed. The patient with cerebrospinal meningitis would usually lie on his side with his head thrown back and the thighs and legs flexed on the abdomen. Patients with angina pectoris and intermittent claudication prefer to stand upright. The patient is also erect (standing or sitting) during attacks of bronchial asthma. He would lean against the edge of the table or the chair back, with the upper part of the body slightly inclined forward. Auxiliary respiratory muscles are more active in this posture. The supine posture is characteristic of strong pain in the abdomen (acute appendicitis, perforated ulcer of the stomach or duodenum). The prone position (lying with the face down) is characteristic of patients with tumors of the pancreas and gastric ulcer (if the posterior wall of the stomach is affected). Pressure of the pancreas on the solar plexus is lessened in this posture.

The concept of habitus includes the body-build, i.e. constitution, height, and body weight. **Constitution** (L *constituero* to set up) is the combination of functional and morphological bodily features that are based on the inherited and acquired properties, and that account for the body response to endo- and exogenic factors. The classification differentiates between the following three main constitutional types: asthenic, hypersthenic, and normosthenic.

The *asthenic* constitution is characterized by a considerable predominance of the longitudinal over the transverse dimensions of the body by the dominance of the limbs over the trunk, of the chest over the abdomen. The heart and the parenchymatous organs are relatively small, the lungs are elongated, the intestine is short, the mesenterium long, and the diaphragm is low. Arterial pressure is lower than in hypersthenics; the vital capacity of the lungs is greater, the secretion and peristalsis of the stomach, and also the absorptive power of the stomach and intestine are decreased; the hemoglobin and red blood cells counts, the level of cholesterol, calcium, uric acid, and sugar in the blood are also decreased. Adrenal and sexual functions are often decreased along with thyroid and pituitary hyperfunction.

The *hypersthenic constitution* is characterized by the relative predominance of the transverse over the longitudinal dimensions of the body (compared with the normosthenic constitution). The trunk is relatively long, the limbs are short, the abdomen is large, the diaphragm stands high. All internal organs except the lungs are larger than those in asthenics. The intestine is longer, the walls are thicker, and the capacity of the intestine is larger. The arterial pressure is higher; haemoglobin and red blood cell count and the content of cholesterol are also higher; hypermobility and hypersecretion of the stomach are more normal. The secretory and the absorptive function of the intestine are high. Thyroid hypofunction is common, while the function of the sex and adrenal glands is slightly increased.

Normosthenic constitution is characterized by a well proportioned make-up of the body and is intermediate between the asthenic and hypersthenic constitutions.

The colour, elasticity, and moisture of the skin, eruptions and scars are important.

The *colour of the skin* depends on the blood filling of cutaneous vessels, the amount and quality of pigment, and on the thickness and translucency of the skin. Pallid skin is connected with insufficiency of blood circulation in the skin vessels due to their spasms of various etiology or acute bleeding, accumulation of blood in dilated vessels of the abdominal cavity in collapse, and in anemia. In certain forms of anemia, the skin is specifically pallid: with a characteristic yellowish tint in Addison-Biermer anemia, with a greenish tint in chlorosis, earth-like in malignant anemia, brown or ash-coloured in malaria, cafe au lair in subacute septic endocarditis. Pallid skin can also be due to its low translucency and considerable thickness; this is only apparent anemia, and can be observed in healthy subjects.

Red colour of the skin can be transient in fever or excess exposure to heat; persistent redness of the skin can occur in subjects who are permanently exposed to high temperatures, and also in erythraemia. Cyanotic skin can be due to hypoxia in circulatory insufficiency, in chronic pulmonary diseases, etc. Yellowish colour of the skin and mucosa can be due to upset secretion of bilirubin by the liver or due to increased haemolysis. Dark red or brown skin is characteristic of adrenal insufficiency. Hyperpigmentation of the breast nipples and the areola, in women, pigmented patches on the face and the white line on the abdomen are signs of pregnancy. When silver preparations are taken for a long time, the skirl becomes grey on the open parts of the body (argyria). Foci of depigmentation of the skin (vitiligo) also occur.

The skin can be wrinkled due to the loss of elasticity in old age, in prolonged debilitating diseases and in excessive loss of water.

Elasticity and turgor of the skin can be determined by pressing a fold of skin (usually on the abdomen or the extensor surface of the arm) between the thumb and the forefinger. The fold disappears quickly on normal skin when the pressure is released while in cases with decreased turgor, the fold persists for a long period of time.

Moist skin and excess perspiration are observed in drop of temperature in patients recovering from fever and also in some diseases such as tuberculosis, diffuse toxic goitre, malaria, suppuration, etc. Dry skin can be due to a great loss of water, e.g. in diarrhea or persistent vomiting (toxicosis of pregnancy, organic pylorostenosis).

Eruptions on the skin vary in shape, size, colour, persistence, and spread. The diagnostic value of eruptions is great in some infections such as measles, German measles, chicken- and smallpox, typhus, etc.

Roseola is a rash-like eruption of 2-3 mm patches, which disappears when pressed. This is due to local dilatation of the vessels. Roseola is a characteristic symptom of typhoid fever, paratyphus, louse-borne typhus, and syphilis.

Erythema is a slightly elevated hyperaemic portion of the skin with distinctly outlined margins. Erythema develops in some persons hypersensitive to strawberries, eggs, and canned crabs. Erythema can develop after taking quinine, nicotinic acid, after exposure to a quartz lamp, and also in some infectious diseases, such as erysipelas and septic diseases.

Weals (urticaria, nettle rash) appear on the skin as round or oval itching lesions resembling those, which appear on the skin bitten by stinging nettle. These eruptions develop as an allergic reaction.

Herpetic lesions are small vesicles 0.5 to 1 cm in size. They are filled with transparent fluid, which later becomes cloudy. Drying crusts appear in several days at the point of the collapsed vesicles. Herpes would normally affect the lips (herpes labialis, or cold sore) and the ala nasi (herpes nasalis). Less frequently herpetic lesions appear on the chin, forehead, cheeks, and ears. Herpetic lesions occur in acute lobar pneumonia, malaria, and influenza.

Purpura is a haemorrhage into the skin occurring in Werlhoff's disease, haemophilia, scurvy, capillarotoxicosis, and longstanding mechanical jaundice. The lesions vary in size from small pointed haemorrhages (petechiae) to large black and blue spots (ecchymoses). Lesions of the skin are quite varied in character when they appear as allergic manifestations.

Desquamation of the skin is of great diagnostic value. It occurs in debilitating diseases and many skin diseases. Scars on the skin, e.g. on the abdomen and the hips, remain after pregnancy (striae gravidarum), in Itsenko-Cushing disease, and in extensive oedema. Indented stellar scars, tightly connected with underlying tissues, are characteristic of syphilitic affections. Postoperative scars indicate surgical operations in past history.

Cirrhosis of the liver is often manifested by development of specific vascular stellae (telangiectasia). This is a positive sign of this disease.

Abnormal growth of hair is usually due to endocrine diseases. Abnormally excessive growth of hair (hirsutism, hypertrichosis) can be congenital, but more frequently it occurs in adrenal tumours (Itsenko-Cushing sydrome) and tumors of the sex glands. Deficient hair growth is characteristic of myxoedema, liver cirrhosis, eunuchoidism, and infantilism. Hair is also affected in some skin diseases.

Nails become excessively brittle in myxoedema, anemia and hypovitaminosis, and can also be found in some fungal diseases of the skin. Flattened and thickened nails are a symptom of acromegaly. Nails become rounded and look like watch glass in bronchiectasis, congenital heart diseases and some other affections.

Subcutaneous fat can be normal or to various degrees excessive or deficient. The fat can be distributed uniformly or deposited in only certain parts of the body. Its thickness is assessed by palpation. Excessive accumulation of subcutaneous fat (adiposis) can be due to either exogenic (overfeeding, hypodynamia, alcoholism, etc.) or endogenic factors (dysfunction of sex glands, the thyroid, or pituitary gland). Insufficient accumulation of subcutaneous fat may result from constitutional factors (asthenic type), malnutrition, or alimentary dysfunction. Excessive wasting is referred to as cachexia, and may occur in prolonged intoxication, chronic infections (tuberculosis), malignant newgrowths, diseases of the pituitary, thyroid and pancreas, and in some psychological disorders as well. Weighing the patient gives additional information about his diet and is an objective means in following up on the patient's weight changes during the treatment of obesity or cachexia.

Edema can be caused by penetration of fluid through the capillary walls and its accumulation in tissues. Accumulated fluid may be congestive (transudation) or inflammatory (exudation). Local edema is a result of some local disorders in the blood or lymph circulation; it is usually associated with thrombosis of the veins, that is, compression of the veins by tumors or enlarged lymph nodes. General edema associated with diseases of the heart, kidneys or other organs is characterized by general distribution of edema throughout the entire body (anasarca) or by symmetrical localization in limited regions of the body. These phenomena can be due to the patient lying on one side. If edema is generalized and considerable, transudate may accumulate in the body's cavities: in the abdomen (ascites), pleural cavity (hydrothorax) and in the pericardium (hydropericardium). Examination reveals

swollen glossy skin. The specific relief features of the edema-affected parts of the body disappear due to the leveling of all irregularities on the body surface. Stretched and tense skin appears transparent in edema, and is especially apparent on loose subcutaneous tissues (the eyelids, the scrotum, etc.). In addition to observation, edema can also be revealed by palpation. When pressed by the finger, the edematous skin overlying bones (external surface of the leg, malleolus, loin, etc.) remains depressed for 1-2 minutes after the pressure is released.

During examination of the *muscular system* the physician should assess its development, which depends on the patient's occupation, his sporting habits, etc. Local atrophy of muscles, especially muscles of the extremities, is diagnostically important. Atrophy can be determined by measuring the girth of the symmetrical muscles of both extremities. Determination of muscular strength and detection of functional muscular disturbances (cramps) are also important for diagnosis. Muscular dysfunction may occur in renal insufficiency (eclampsia), disorders of the liver (hepatic insufficiency), affections of the central nervous system (meningitis), tetanus, cholera, etc.

Defects (deformities or bulging) of the *bones* of the skull, chest, spine, and the extremities, may be revealed by external inspection. But in many cases palpation is necessary. Peripheral bones of the extremities (of the fingers, toes), cheek bones or the mandible grow abnormally in acromegaly. Rachitic changes occur in the form of the so-called pigeon breast, rachitic rosary (beading at the junction of the ribs with the cartilages), deformities of the lower extremities, etc. Tuberculotic lesions (the so-called haematogenic osteomyelitis) are localized mainly in the epiphyses of the bones, with formation of fistulae through which pus is regularly discharged. Multiple affections of the flat bones of the skeleton (the skull included) that can be seen radiographically as round light spots (bone tissue defects) are typical of myeloma. Diseases of the spine cause deformation of the spinal column and the chest. Considerable deformities of the spine (kyphosis, scoliosis) can cause dysfunction of the thoracic organs.

When examining *the joints* attention should be paid to their shapes, articulation, tenderness in active or passive movements, edema, and hyperemia of the adjacent tissues. Multiple affections of large joints are characteristic of exacerbated rheumatism. Rheumatoid arthritis affects primarily small joints of the hands with their subsequent deformation. Metabolic polyarthritides, e.g. in gout, are characterized by thickening of the terminal phalanges of the fingers and toes (so-called Heberden's nodes). Monarthritis (affection of one joint) would be usually observed in tuberculosis and gonorrhoea.

Normal *lymph nodes* cannot be detected visually or by palpation. Depending on the character of the process, their size varies from that of a pea to that of an apple. In addition to simple inspection, the physician should resort to palpation in order to make a conclusion on the condition of the lymphatic system. Attention should be paid to the size of the lymph nodes, their tenderness, mobility, consistency and adherence to the skin. Submandibular, axillary, cervical, supraclavicular, and inguinal lymph nodes are commonly enlarged. Submandibular nodes swell in the presence of inflammation in the mouth. Chronic enlargement of the cervical lymph nodes is associated with development of tuberculosis in them, which is characterized by purulent foci with subsequent formation of fistulae and immobile cicatrices.

Cancer of the stomach and, less frequently, cancer of the intestine can metastasize into the lymph nodes of the neck (on the left). The axillary lymph nodes are sometimes enlarged in mammary cancer. In the presence of metastases the lymph nodes are firm, their surface is rough, palpation is painless. Tenderness of a lymph node in palpation and reddening of the overlying skin indicates inflammation in the node. Systemic enlargement of the lymph nodes is observed in lympholeukaemia, lymphogranulomatosis, and lymphosarcomatosis. In lymphatic leukemia and lymphogranuloma the nodes fuse together but do not suppurate. Puncture or biopsy of the lymph nodes is required to diagnose complicated cases.

Changes in the size and shape of the *head* can give diagnostic clues. Excessive growth of the skull occurs in hydrocephalus. An abnormally small head is typical of microcephalus, which is also marked by mental under development. A square head, flattened on top, with prominent frontal tubers, can indicate congenital syphilis or rickets in past history. The position of the head is also important in diagnosing cervical myositis or spondylarthritis. Involuntary movements of the head (tremor) are characteristic of parkinsonism. Rhythmical movements of the head in synchronism with the cardiac pulse are characteristic of aortic incompetence (Musset's sign). The presence of scars on the head may suggest the cause of persistent

headache. It is necessary to find out whether the patient has vertigo, which is typical particularly for Meniere's syndrome, or epileptiform attacks.

Countenance. The facial expression can indicate the mental composure and various psychic and somatic conditions. It also depends on age and sex and can therefore give diagnostic clues when diagnosing some endocrine disorders (woman-like expression in men and masculine features in women). The following changes in the face are diagnostically essential:

1. A puffy face is observed in general edema characteristic of renal diseases; local venous congestion in frequent fits of suffocation and cough; compression of lymph ducts in extensive effusion into the pleural and pericardial cavity, in tumor of mediastinum, enlarged mediastinal lymph nodes, adhesive mediastinopericarditis, compressed superior vena cava (Stokes' collar).
2. Corvisart's fades is characteristics of cardiac insufficiency. The face is edematous, pale yellowish, with a cyanotic hue. The mouth is always half open, the lips are cyanotic, the eyes are dull and the eyelids sticky.
3. Facies febrilis is characterized by hyperaemic skin, sparkling eves and excited expression. There are special features of facies febrilis characteristic of some infectious diseases: feverish redness in acute lobar pneumonia (more pronounced on the side of the affected lung); general hyperemia of the puffy face is characteristic of louse-borne typhus, the sclera is injected (“rabbit eye”); slightly icteric yellow colour is characteristic of typhoid fever. Tuberculosis patients with fever have “burning” eyes on an exhausted and pale face with blush localized on the cheeks. An immobile face is characteristic of septic fever; the face pale, sometimes slightly yellowish.
4. Face and its expression are altered in various endocrine disorders: face with enlarged prominent parts (such as nose, chin, and cheek bones) and enlarged hands are characteristic of acromegalia (hands become enlarged in some pregnancies); myxedematous face indicates thyroid hypofunction: the face may be uniformly puffy with edematous mucosa, narrowed eye slits, the face features smoothed down, the hair is absent on the outward portions of the eyebrow, the presence of a blush on a pale face resembles the appearance of a doll; facies basedovica this is the face of a patient with thyroid hyperfunction: the face is lively with widened eye slits and abnormally sparkling eyes, the eyes are protruded and face looks as if frightened; an intense red, moon-like glittering face with a beard and mustaches in women is characteristic of the Itsenko-Cushing disease.
5. Facies leonjina with nodular thickening of the skin under the eyes and over the brown, with flattened nose is observed in leprosy.
6. Parkinson’s mask (or facies) is an amimic face characteristic of encephalitis patients.
7. A slightly puffy wax-doll, very pale face with a yellowish tint, and seemingly translucent skin, is characteristic of Addison-Biermer anemia.
8. Risus sardonicus with a semblance of a grin occurs in tetanus patients: the mouth widens as in laughter, while the skin folds on the forehead express grief.
9. Facies Hippocratica (first described by Hippocrates) is associated with collapse in grave diseases of the abdominal organs (diffuse peritonitis, perforated ulcer of the stomach or duodenum, rupture of the gall bladder). The face is characterized by sunken eyes, pinched nose, deadly livid and cyanotic skin, which is sometimes covered with large drops of cold sweat.
10. Asymmetric movements of facial muscles indicate a history of cerebral hemorrhage or facial neuritis.

Inspection of the *eyes* and eyelids can reveal some essential diagnostic signs. Edema of the eyelids, especially of the lower eyelids, is the first indication of acute nephritis; it is also observed in anemia, frequent attacks of cough, and deranged sleep; edema of the eyelids can also occur in the morning in healthy persons as well.

The colour of the eyelids is important. The eyelids are dark in diffuse toxic goitre and Addison disease. Xanthomas indicate deranged cholesterol metabolism. A dilated eye slit with the eyelids that do not close is characteristic of paralysis of the facial nerve; persistent drooping of the upper eyelid (ptosis) is an important sign of some affections of the nervous system. Narrowing of the eye slit occurs in myxoedema and general edema of the face. Exophthalmos (protrusion of the eyeball) is observed in thyrotoxicosis, retrobulbar tumors, and also in strong myopia. Recession of the eyeball in the orbit (enophthalmos) is typical of myxoedema and is an important sign of "peritoneal face". Unilateral

recession of the eye into the orbit attended by narrowing of the eye slit, drooping of the upper eyelid and narrowing of the pupil, is the Horner's (Bernard-Homer) syndrome caused by the affection of the pupil sympathetic enervation of the same side (due to various causes).

The shape of the pupils, their symmetry, response to light, accommodation and convergence, and also their "pulsation" are of great diagnostic significance in certain diseases. Abnormally contracted pupil (miosis) is observed in uraemia, tumors and intracranial haemorrhages, and in morphine poisoning. Enlargement of the pupil (mydriasis) occurs in comatose states (except uraemic coma) and cerebral haemorrhages, and also in atropine poisoning. Anisocoria (unequal size of the pupils) occurs in some affections of the nervous system. Squinting results from paralysis of the ocular muscles due to lead poisoning, botulism, diphtheria, affections of the brain and its membranes (syphilis, tuberculosis, meningitis, cerebral haemorrhage).

The size of the *nose* may attract attention providing some diagnostic signs, e.g. it has an abnormal size in acromegaly, or its shape deviates from the normal in rhinoscleroma. The nose may be sunken as a result of syphilis in the past history (saddle nose). Soft tissues of the nose are disfigured in lupus.

When inspecting the *mouth* attention should be paid to its shape (symmetry of the angles, permanently open mouth), the colour of the lips, eruption on the lips (cold sores, herpes labialis), and the presence of fissures. The oral mucosa should also be inspected (for the presence of aphthae, pigmentation, Filatov-Koplik spots, thrush, contagious aphthae of the foot and mouth disease, hemorrhage). Marked changes in the gums can be observed in some diseases (such as pyorrhoea, acute leukaemia, diabetes mellitus, and scurvy) and poisoning (with lead or mercury). The teeth should be examined for the absence of defective shape, size, or position. The absence of many teeth is very important in the etiology of some alimentary diseases. Caries is the source of infection and can affect some other organs.

Disordered movement of the tongue may indicate nervous affections, grave infections and poisoning. Marked enlargement of the tongue is characteristic of myxoedema and acromegaly; less frequently it occurs in glossitis. Some diseases are characterized by the following abnormalities of the tongue: the tongue is clear, red, and moist in ulcer; crimson-red in scarlet fever; dry, with a brown coat and grooves in grave poisoning and infections; coated in the centre and at the root, but clear at the tip and margins in typhoid fever; smooth tongue without papillae (as if polished) is characteristic of Addison-Biermer disease. The glassy tongue is characteristic of gastric cancer, pellagra, sprue, and ariboflavinosis; local thickening of the epithelium is characteristic of smokers (leucoplakia). Local pathological processes, such as ulcers of various etiology, scars, traces left from tongue biting during epileptic fits, etc., are also suggestive of certain diseases.

During inspection of the *neck* attention should be paid to pulsation of the carotid artery (aortic incompetence, thyrotoxicosis), swelling and pulsation of the external jugular veins (tricuspid valve insufficiency), enlarged lymph nodes (tuberculosis, lympholeukaemia, lymphogranulomatosis, cancer metastases), diffuse or local enlargement of the thyroid gland (thyrotoxicosis, simple goitre, malignant tumor).

Examination of the *extremities* can reveal varicosity of the veins, edema, changes in the skin, muscles, tremor of the extremities, deformities, swelling and hyperaemia of the joints, ulcers, and scars. Diseases of the central nervous system (tumors, cerebral hemorrhage) and also of the peripheral nervous system can cause atrophy and paralysis of the muscles.

Hippocrates fingers or clubbing of the terminal phalanges of the fingers and toes are important diagnostic ally. The changed shape of the nails resembles hour glass. This symptom is characteristic of prolonged diseases of the lung (chronic purulent processes), heart (subacute septic endocarditis, congenital heart defects) and liver (cirrhosis). Periodically occurring vascular spasms in the extremities cause the development of the symptom known as the dead finger, transient pallor of the fingers and toes, which is characteristic of Raynaud's disease. Prolonged spasms of blood vessels can cause gangrene of the fingers.

When examining the legs, attention should be paid to possible flat foot. Saber shins occur in rickets and sometimes in syphilis. Uneven thickening of the leg bones indicates periostitis, which can sometimes be of syphilitic etiology.

The intermediate control of mastering of the substantial module 1.

***Theme 3*. Main Complaints of the Patients with Lung Diseases.**

The following complaints and their details. Disease and life history. Static and dynamic examination of the chest. The basic topographical areas and reference points on a surface of the chest. Normal and pathological shape of the chest. Pathological types of respiration (Cheyne-Stokes, Biot´s, Kussmaul´s, Grocco) and their characteristics, courses. Chest palpation, voice resonance.

The contents: Complaints. *Cough* may be dry, without sputum, and moist, during which various amounts of sputum of different quality are expectorated. Some diseases are attended only by dry cough, e.g. laryngitis, dry pleurisy or compression of the main bronchi by the bifurcation lymph nodes (tuberculosis, lymphogranulomatosis, cancer metastases, etc.). Bronchitis, pulmonary tuberculosis, pneumosclerosis, abscess, or bronchogenic cancer of the lungs can be first attended by dry cough, which will then turn into moist one with expectoration of the sputum.

If a patient complains of cough with sputum, the physician should try to determine the amount of sputum expectorated during one fit and during the entire day; it is also important to know the time of the day during which the sputum is expectorated and the position of the body at which cough is provoked; the colour, odour, and other properties of sputum are also important. Morning cough is characteristic of patients with chronic bronchitis, bronchiectasis, lung abscess, and cavernous tuberculosis of the lungs. The sputum accumulates during the night sleep in the lungs and the bronchi, but as the patient gets up, the sputum moves to the neighboring parts of the bronchi to stimulate the reflexogenic zones of the bronchial mucosa. This causes cough and expectoration of the sputum. The amount of the sputum expectorated during the morning may amount to two thirds of the entire daily expectoration. Depending on the gravity of the inflammatory process in patients with mentioned diseases, the daily amount of the expectorated sputum may vary from 10-15 ml to as much as 2 liters. In unilateral bronchiectasis, sputum may be better expectorated in a definite posture, for example, on the right side with bronchiectasis in the left lung, and vice versa. If bronchiectasis is found in the anterior region of the lungs, expectoration is easier in the supine position, and if in the posterior parts, in the prone position.

Dyspnea is a symptom, not a sign, and is one of several sensations a patient may describe. A healthy person notes the increased ventilation required during exercise but does not interpret it as being particularly unpleasant unless extreme. Unpleasant or worrisome awareness that a small amount of exercise leads to a disproportionately large increase in ventilation is a common type of dyspnea, usually described as breathlessness or shortness of breath on exertion. At high altitude, a healthy person notes a similar disproportionately large increase in ventilation resulting from exertion and finds it limiting but usually not otherwise unpleasant.

Other sensations include awareness of increased muscular effort required to expand the chest during inspiration or to expel air from the lungs, sensations of fatigue in the respiratory muscles, awareness of a delay in air leaving the lungs during expiration, the uncomfortable sensation that an inspiration is urgently needed before expiration is completed, and various sensations most often described as tightness in the chest. The last can probably include awareness of collapse or hyperinflation of lung units, obstruction of airways, and distortion or displacement of the lungs, mediastinum, diaphragm, or chest wall.

Afferent impulses to the brain that generate the sensation of dyspnea come from many different sites, such as the lungs, articulations of the rib cage, and the respiratory muscles, including the diaphragm. Peripheral and central chemoreceptors provide part of the sensory input that appears to be involved in dyspnea, either directly or indirectly; other visceral, neural, and emotional stimuli may also participate. The two major causes of pulmonary dyspnea are a restrictive defect with low compliance of the lungs or chest wall and an obstructive defect with increased resistance to airflow. Patients with restrictive dyspnea (due to pulmonary fibrosis or chest deformities) are usually comfortable at rest but intensely dyspneic when exertion causes pulmonary ventilation to approach their greatly limited breathing capacity. In obstructive dyspnea (in chronic obstructive pulmonary disease or asthma), increased ventilatory effort induces dyspnea even at rest, and breathing is labored and retarded, especially during expiration; this type of dyspnea always worsens during effort and exercise.

Physical findings may help determine the cause (pleural effusion, pneumothorax, and sometimes interstitial lung disease). The signs of emphysema, bronchitis, and asthma are frequently helpful in defining the nature and severity of the underlying obstructive lung disease. Pulmonary function testing can provide numeric values for any restriction or airflow obstruction present.

Orthopnea is respiratory discomfort that occurs while the patient is supine, impelling him to sit up. It is precipitated by an increase in venous return of blood to a failing left ventricle that cannot handle the increased preload. Of less importance is the increased effort of breathing in the supine position.

In evaluating *chest pain,* the first task-not always easy is to differentiate respiratory pain from pain related to other systems. Most noncardiac chest pain arises from the pleura or the chest wall. Pleuritic pain is typically made worse by deep breathing or coughing and may be controlled by immobilization of the chest wall; eg, the patient may hold his side, avoid deep breathing, or suppress his cough. The patient can usually identify the site of pleuritic pain. Over time it may move from one site to another. If a pleural effusion develops, the pain may disappear as the inflamed pleural surfaces are separated. A friction rub is often associated with pleuritic pain, but either may occur alone.

Pain arising from the chest wall may be exacerbated by deep breathing or coughing, but it can usually be distinguished by localized tenderness. Although some tenderness may be present with pleuritic pain (eg, in pneumococcal pneumonia), it is usually slight, poorly localized, and elicited only by deep pressure. Chest wall trauma or a broken rib is often obvious from the history, but torn muscle fibers or even a rib fracture can result from severe coughing. A tumor infiltrating the chest wall may cause local pain or, if it involves intercostal nerves, referred pain. Herpes zoster, before the eruption appears, may present as puzzling chest pain. Physical examination and chest x-rays can usually determine the cause.

Anamnesis. When questioning the patient the physician should determine the time the disease began. Acute onset is characteristic of acute pneumonia, especially acute lobar pneumonia. Pleurisy begins more gradually. A non-manifest onset and a prolonged course are characteristic of pulmonary tuberculosis and cancer. The onset of many diseases may be provoked by chills (bronchitis, pleurisy, pneumonia).

Determining epidemiological conditions is very important for establishing the cause of the disease. Thus influenzal pneumonia often occurs during epidemic outbreaks of influenza. Establishing contacts with tuberculosis patients is also very important. Specific features of the course of the disease and the therapy given (and its efficacy) should then be established.

When collecting the life anamnesis, the physician should pay attention to conditions under which the patient lives and works. Damp premises with inadequate ventilation or work in the open (builders, truck drivers, agricultural workers, etc.) can become the cause of acute inflammation of the lungs with more frequent conversion into chronic diseases. Some dusts are harmful and cause bronchial asthma. Coal dust causes a chronic disease of the lungs called anthracosis. Regular exposure to silica dust (cements, pottery, etc.) causes silicosis, the occupational fibrosis of the lungs.

The patient should give a detailed report of his past diseases of the lungs or pleura, which help the physician, establish connections between the present disease and diseases of the past history.

Examination of the chest should be done according to a definite plan. The general configuration of the chest should the next step is to define the type, rhythm and frequency of breathing, respiratory movements of the left and right shoulder blades, and of the shoulder girdle, and involvement of the accessory respiratory muscles in the breathing act. The patient should be better examined in the upright (standing or sitting) position with the chest being naked. Illumination of the body should be uniform.

The shape of the chest may be normal or pathological. A normal chest is characteristic of healthy persons with regular body built. Its right and left sides are symmetrical, the clavicles and the shoulder blades should be at one level and the supraclavicular fossae equally pronounced on both sides. Since all people with normal constitution are conventionally divided into three types, the chest has different shape in accordance with its constitutional type. Pathological shape of the chest may be the result of congenital bone defects and of various chronic diseases (emphysema of the lungs, rickets, tuberculosis).

Normal form of the chest.

1. *Normosthenic (conical) chest* in subjects with normosthenic constitution resembles a truncated cone whose bottom is formed by well-developed muscles of the shoulder girdle and is directed upward. The anteroposterior (sternovertebral diameter of the chest is smaller than the lateral (transverse) one, and the supraclavicular fossae are slightly pronounced. There is a distinct angle between the sternum and the manubrium (angulus Ludowici); the epigastric angle nears 90°. The ribs are derately inclined as viewed from the side; the shoulder blades closely fit to the chest and are at the same level; the chest is about the same height as the abdominal part of the trunk.

2. *Hypersthenic chest* in persons with hypersthenic constitution has the shape a cylinder. The anteroposterior diameter is about the same as the transverse one; the supraclavicular fossae are absent (level with the chest). the manubriosternal angle is indistinct; the epigastric angle exceeds 90°; the ribs in the lateral parts of the chest are nearly horizontal, the intercostal space is narrow, the shoulder blades closely fit to the chest, the thoracic part of the trunk is smaller than the abdominal one.

3. *Asthenic chest* in persons with asthenic constitution is elongated, narrow both the anteroposterior and transverse diameter are smaller than normal); the chest is flat. The supra- and subclavicular fossae are distinctly pronounced. There is no angle between the sternum and the manubrium: the sternal bone and the manubrium make a straight "plate". The epigastric angle is less than 90°. The ribs are more vertical at the sides, the tenth ribs are not attached to the costal arch (costa decima fluctuens); the intercostal spaces are wide, the shoulder blades are winged (separated from the chest), the muscles of the shoulder girdle are underdeveloped, the shoulders are sloping, the chest is longer than the abdominal part of the trunk.

Pathological chest. 1. *Emphysematous (barrell-like) chest* resembles a hypersthenic chest in its shape, but differs from it by a barrel-like configuration, prominence of the chest wall, especially in the posterolateral regions, the intercostal spaces are enlarged. This type of chest is found in chronic emphysema of the, lungs, during which, elasticity of the lungs decreases while the volume of the lungs increases, the lungs seem to be as if at the inspiration phase. Natural expiration is therefore difficult not only during movements but also at rest (expiratory dyspnoea is found). Active participation of accessory respiratory muscles in the respiratory act (especially m. sternocleidomastoideus and m. trapezius), depression of the intercostal space, elevation of the entire chest during inspiration and relaxation of the respiratory muscles and lowering of the chest to the initial position during expiration become evident during examination of emphysema patients.

2. *Paralytic chest* resembles the asthenic chest. It is found in emaciated patients, in general asthenia it often occurs in grave chronic diseases, more commonly in pulmonary tuberculosis and pneumosclerosis, in which fibrous tissue contract the lungs and diminishes their weight due to the progressive chronic inflammation. During examination of patients with paralytic chest, marked atrophy of the chest muscles and asymmetry of the clavicles and dissimilar depression of the supraclavicular fossae can be observed along with typical signs of asthenic chest. The shoulder blades are not at one level either, and their movements during breathing are asynchronous.

3. *Rachitic chest (keeled or pigeon chest).* It is characterized by a markedly greater anteroposterioir diameter (compared with the transverse diameter) due to the prominence of the sternum (which resembles the keel of a boat.) The anterolateral surfaces of the chest are as if pressed on both sides and therefore the ribs meet at an acute angle at the sternal bone, while the costal cartilages thicken like beads at points of their transition to bones (rachitic beads). As a rule, these beads can be palpated after rickets only in children and youths.

4. *Funnel chest* can occur in normosthenic, hypersthenic or asthenic subjects; it has a funnel-shaped depression in the Tower part of the sternum. This deformity can be regarded as a result of abnormal development of the sternum or prolonged compressing effect. In older times this chest would be found in shoemaker adolescents. The mechanism of formation of the funnel chest was explained by the permanent pressure of the chest against the shoe; the funnel chest was therefore formerly called cobbler chest.

5. *Foveated chest* is almost the same as the funnel chest except that the depression is found mostly in the upper and the middle parts of the anterior surface of the chest. This abnormality occurs in syringomyelia, a rare disease of the spinal cord. The chest may be abnormal in subjects with various

deformities of the spine, which arise as a result of injuries, tuberculosis of the spine, rheumatoid arthritis (Bekhterev's disease), etc.

6. Four types of spine deformities are distinguished: *scoliotic chest* is observed in lateral curvature of the spine; *kyphotic chest* is excessive forward and backward curvature of the spine; *kyphoscoliotic chest* is combination of the lateral and forward curvature of the spine; backward curvature (lordosis) of the spine may result in *lordotic chest.* Scoliosis is the most frequently occurring deformity of the spine. It mostly develops in school children due to bad habitual posture. Kyphoscoliosis occurs less frequently. Lordosis only occurs in rare cases. Curvature of the spine, especially kyphosis, lordosis, and kyphoscoliosis cause marked deformation of the chest to change the physiological position of the lungs and the heart and thus interfere with their normal functioning.

The shape of the chest can readily change due to enlargement or diminution of one half of the chest (asymmetry of the chest). These changes can be transient or permanent.

The enlargement of the volume of one half of the chest can be due to escape of considerable amounts of fluid as the result of inflammation (exudate) or non-inflammatory fluid (transudate) into the pleural cavity, or due to penetration of air inside the chest in injuries (pneumothorax). Leveling or protrusion of the intercostal spaces, asymmetry of the clavicles and the shoulder blades and also unilateral thoracic lagging can be observed during examination of the enlarged part of the chest. The chest assumes normal shape after the air or fluid is removed from the pleural cavity.

One part of the chest may diminish due to (1) pleural adhesion or complete closure of the pleural slit after resorption of effusion (after prolonged presence of the fluid in the pleural cavity); (2) contraction of a considerable portion of the lung due to growth of connective tissue (pneumosclerosis) after acute or chronic inflammatory processes, such as acute lobar pneumonia (with subsequent carnification of the lung), lung infarction, pulmonary abscess, tuberculosis, etc.; (3) resection of a part or the entire lung; (4) atelectasis (collapse of the lung or its portion) that may occur due to closure of the lumen in a large bronchus by a foreign body or a tumor growing into the lumen of the bronchus and causing its obturation. The closure of the air passage into the lung with subsequent resorption of air from the alveoli and a decrease in the volume of the lung diminish the corresponding half of the chest. The chest thus becomes asymmetrical, the shoulder of the affected side lowers, the clavicle and the scapula lower as well, and their movements during deep respiration become slower and limited; the supra- and subclavicular fossae become more depressed, the intercostal spaces decrease in size or become invisible. The marked depression of the supraclavicular fossae on one side often depends on the diminution of the apex of a fibrosis-affected lung.

Respiratory movements of the chest should be examined during inspection of the patient. In physiological conditions they are performed by the contraction of the main respiratory muscles: intercostal muscles, muscles of the diaphragm, and partly the abdominal wall muscles. The so-called accessory respiratory muscles (mm. sternocleidomastoideus, trapezius, pectoralis major and minor, etc.) arc actively involved in the respiratory movements in pathological condition associated with difficult breathing.

The type, frequency, depth and rhythm of respiration can be determined by carefully observing the chest and abdomen. Respiration can be costal (thoracic), abdominal, or mixed type.

Thoracic (costal) respiration. Respiratory movements are carried out mainly by the contraction of the intercostal muscles. The chest markedly broadens and slightly rises during inspiration, while during expiration it narrows and slightly lowers. This type of breathing is known as costal and is mostly characteristic of women.

Abdominal respiration. Breathing is mainly accomplished by the diaphragmatic muscles; during the inspiration phase the diaphragm contracts and lowers to increase rarefaction in the chest and to suck in air into the lungs. The intraabdominal pressure increases accordingly to displace anteriorly the abdominal wall. During expiration the muscles are relaxed, the diaphragm rises, and the abdominal wall returns to the initial position. The type of respiration is also called diaphragmatic and characteristic of men.

Mixed respiration. The respiratory movements are carried out simultaneously by the diaphragm and the intercostal muscles. In physiological conditions this respiration sometimes occurs in aged persons and in some pathological conditions of the respiratory apparatus and the abdominal viscera. For example, in women with dry pleurisy, pleural adhesion, myositis and thoracic radiculitis, the contractile activity of the intercostal muscles decreases and the respiratory movements are carried out by the

accessory movements of the diaphragm. In extensive pleural adhesion, lung emphysema, and in strong pain in the chest due to acute inflammation of the intercostal muscles or nerves, respiration is temporarily carried out by the diaphragmatic muscles exclusively. Mixed respiration occurs in men with underdeveloped diaphragmatic muscles, in diaphragmatitis, acute cholecystitis, perforating ulcer of the stomach or the duodenum.

Respiration rate. Respiration rate can be determined by counting the movements of the chest or the abdominal wall, with the patient being unaware of the procedure. The pulse rate should first be taken and then the respiration rate. The number of respiratory movements in a healthy, adult at rest should be 16 to 20 per minute, in the newborn 40-45, this rate gradually decreasing with age. The respiration rate decreases during sleep to 12-14 per minute, while under physical load, emotional excitement, or after heavy meals the respiration rate increases.

The respiration rate alters markedly in some pathological conditions. The causes of accelerated respiration may be narrowing of the lumen of small bronchi due to spasms or diffuse inflammation of their mucosa (bronchiolitis occurring mostly in children), which interfere with normal passage of air into the alveoli; decreased respiratory surface of the lungs due to their inflammation and tuberculosis, in collapse or atelectasis of the lung due to its compression (pleurisy with effusion, hydrothorax, pneumothorax, tumor of mediastinum), in obturation or compression of the main bronchus by a tumor, in thrombosis or embolism of the pulmonary artery, in pronounced emphysema, when the lung is overfilled with blood or in a case of lung edema in certain cardiovascular diseases; insufficient depth of breathing (superficial respiration) which can be due to difficult contractions of the intercostal muscles or the diaphragm in acute pain (dry pleurisy, acute myositis, intercostal neuralgia, rib fracture, or tumor metastasis into the ribs), in a sharp increase in the intraabdominal pressure and high diaphragm (ascites, meteorism, late pregnancy), and finally in hysteria.

Pathological deceleration of respiration occurs in functional inhibition of the respiratory centre and its decreased excitability. It can be due to increased intracranial pressure in patients with cerebral tumor, meningitis, cerebral haemorrhage, or edema of the brain, and also due to the toxic effect on the respiratory centre when toxic substances are accumulated in the blood, e.g. in uraemia, hepatic or diabetic coma, and in certain acute infectious diseases.

Respiration depth. The depth of breathing is determined by the volume of the inhaled and exhaled air at rest. This volume varies in an adult from 300 to (500 ml on the average). Depending on depth, breathing can be either deep or superficial. Superficial (shallow) breathing often occurs in pathologically accelerated respiration when the length of the inspiration and the expiration phases becomes short. Deep and slow breatting is, on the con trary, associated in most cases with pathological deceleration of the respiration rate. Deep and slow respiration, with marked respiratory movements, is sometimes attended by noisy sounds. This is *Kussmaul's respiration* occurring in deep coma. In some pathological conditions, however, rare respiration can be shallow, while accelerated breathing deep. Rare superficial respiration can occur in sharp inhibition of the respiratory centre, pronounced lung emphysema, and sharp narrowing of the vocal slit or the trachea. Respiration becomes accelerated and deep in high fever and marked anemia.

Respiration rhythm. Respiration of a healthy person is rhythmic, of uniform depth and equal length of the inspiration and expiration phases. Rhythm of the respiratory centre can be inhibited in some types of edema. Derangement of the respiratory function can cause edema in which a series of respiratory movements alternates with a pronounced (readily detectable) elongation of the respiratory pause (lasting from a few seconds to a minute) or a temporary arrest of respiration (apnoea). This respiration is known as periodic.

Biot's respiration is characterized by rhythmic but deep respiratory movements, which alternate (at approximately regular intervals) with long respiratory pauses (from few seconds to half a minute). Biot's respiration occurs in meningitis patients and in agony with disorders of cerebral circulation.

Cheyne-Stokes' respiration is characterized by periods (from few seconds to a minute) of cessation of respiration, followed by noiseless shallow respiration, which quickly deepens, becomes noisy to attain its maximum at the 5-7th inhalation, and then gradually slows down to end with a new short respiratory pause. During such pauses, the patient often loses his sense of orientation in the surroundings or even

faints, to recover from the unconscious condition after respiratory movements are restored. This respiratory disorder occurs in diseases causing acute or chronic insufficiency of cerebral circulation and brain hypoxia, and also in heavy poisoning. More frequently this condition develops during sleep and is more characteristic of aged persons with marked atherosclerosis of the
cerebral arteries.

Undulant (wave-like) Grocco's respiration somewhat resembles Cheyne-Stokes' respiration except that a weak shallow respiration occurs instead of the respiratory cause with subsequent deepening of the respiratory movement, follow by slowing down. This type of arrhythmic dyspnoea can probably be regarded as the early stages of the same pathological processes, which are responsible for Cheyne-Stokes respiration.

Palpation is used as an additional means of examination to verify findings of observation (the shape of the chest, its dimensions, respiratory movements), for determining local or profuse tenderness of the chest, its elasticity (resilience), vocal resonance, pleural friction and sounds of fluid in the pleural cavity.

Palpation should be done by placing the palms on the symmetrical (left and right) parts of the chest. This examination helps follow the respiratory excursions and deviation of the chest movements from their normal course. The epigastric angle is determined by palpation as well. The thumbs should be pressed tightly against the costal arch, their tips resting against the xiphoid process (ensiform cartilage).

Palpation is used to locate pain in the chest and its irradiation. For example, in rib fracture, pain is localized over a limited site, namely at the point of the fracture. Displacement (carefull) of bone fractures will be attended in this case by a specific sound (crunch). Inflammation of the intercostal nerves and muscles also causes pain, but it can be felt during palpation over the entire intercostal space. Such pain is called superficial. It is intensified during deep breathing, when the patient bends to the affected side, or lies on this side.

Resilience or elasticity of the chest is determined by exerting pressure of the examining hands from the front to the sides of the chest or on the back and the sternum, and also by palpation of the intercostal spaces. The chest of a healthy person is elastic, plaint and yields under the pressure. In the presence of pleurisy with effusion, or pleural tumor, the intercostal space over the affected site becomes rigid. Rigidity of the chest increases in general in the aged due to ossification of the costal cartilages, development of the lung emphysema, and also with filling of both pleural cavities with fluid. Increased resistance of the chest can then be felt during examining the chest by compression in both the anteroposterior and lateral directions.

Palpation is used for determining the strength of voice conduction to the chest surface – *voice resonance* (fremitus pectoralis). The palms of the hands are placed on the symmetrical parts of the chest and the patient is asked to utter loudly a few words (with the letter 'r' in them to intensify vibration). The voice should be as low as possible since voice vibrations are better transmitted by the air column in the trachea and the bronchi to the chest wall in this case. Fremitus vocalis can also be determined by one hand as well: the palm of the examining hand should be placed alternately on the symmetrical parts of the chest.

Vocal resonance of about the same intensity in the symmetrical parts of the chest of a healthy person. Vocal vibrations are louder in the upper parts of the chest and softer in its lower parts. Moreover, voice conduction is better in men with low voice and thin chest; the vibrations are weaker in women and children with higher voice (and also in persons with the well developed subcutaneous fat tissues). Vocal resonance can be stronger or weaker (or in some cases it can even be impalpable) in pathological conditions of the respiratory organs. In focal affections, vocal resonance becomes unequal over symmetrical parts of the chest.

Vocal resonance is **intensified**, when a part of the lung or its whole lobe becomes airless and more uniform (dense) because of a pathological process. According to the laws of physics, dense and uniform bodies conduct sound better than loose and non-uniform. Induration (consolidation) can be due to various causes, such as acute lobar pneumonia, pulmonary infarction, tuberculosis, etc. Vocal

resonance is also intensified in the presence in the pulmonary tissue of an air cavity communicated with the bronchus.

Vocal resonance becomes **weaker**, when liquid or gas are accumulated in the pleural cavity; they separate the lung from the chest wall to absorb voice vibrations propagating from the vocal slit along the bronchial tree; in complete obstruction of the bronchial lumen by a tumor which in terferes with normal conduction of sound waves to the chest wall; in asthenic emaciated patients (with weak voice); insignificant thickening of the chest wall in obesit. Low-frequency vibrations due to pleural friction (friction fremitus) in dry pleurisy, crepitation sounds characteristic of subcutaneous emphysema of the lungs, vibration of the chest in dry, low (low-pitch buzzing) rales can also be determined by palpation.

***Theme 4*. Percussion as a Method of Physical Examination of the Lungs. Comparative Percussion of the Lungs**.

History of percussion invention and its development. Role of percussion in definition of a condition of the lungs. Percussion rules. Types of percussion, technique of percussion. The characteristics of the percussion sound. Definition, technique of comparative percussion, sequence and diagnostic significance of the received results. Their purpose and diagnostic significance. Courses of formation of a dull, tympanic, deadened tympanic, bandbox percussion sound on the lungs.

The contents: Percussion (L *percutere* to strike through) was first proposed by an Austrian physician Auenbrugger in 1761. Tapping various parts of the human body produces sounds by which one can learn about the condition of the underlying organs. The organs or tissues lying beneath the percussed area begin vibrating and these vibrations are transmitted to the surrounding air whose vibration is perceived by our ears as sounds. Liquids and airless tissues give dull sounds, which can be heard with difficulty, such as the sound of a percussed femur (femoral sound). Airless organs and also liquids cannot therefore be differentiated by percussion. The properties of each particular sound obtained by percussion of the chest or the abdomen, and differing from the femoral sound, depend on the amount of air or gas enclosed within the chest or abdomen. The difference in the sounds of percussed lungs, liver, spleen, heart, stomach and other organs depends on (a) the different amount of gas or air inside or round the percussed organ; (b) tension of the tissue; and (c) different strength of the percussion stroke transmitted to this gas or air.

Main rules of percussion.

1. The patient should be in a comfortable posture and relaxed. The best position is standing or sitting. Patients with grave diseases should be percussed in the lying position. When the patient is percussed from his back, he should be sitting on a chair, his face turned to the chair back. The head should be slightly bent forward, his arms should rest against his lap. In this position muscle relaxation is the greatest and percussion thus becomes more easy.
2. The room should be warm and protected from external noise.
3. The physician should be in a comfortable position as well.
4. A pleximeter or the middle finger of the left hand, which is normally used in the finger-to-finger percussion, should be pressed tightly to the examined surface. The neighbouring fingers should be somewhat set apart and tightly pressed to the patient's body. This is necessary to delimit propagation of vibrations arising during percussion. The physician's hands should be warm.
5. The percussion sound should be produced by the tapping movement of the hand alone. The sound should be short and distinct. Tapping should be uniform, the force of percussion strokes depending on the object being examined.
6. In topographic percussion, the finger or the pleximeter should be placed parallel to the anticipated border of the organ. Organs giving resonant note should be examined first: the ear will better detect changes in sound intensity. The border is marked by the edge of the pleximeter directed toward the zone of the more resonant sounds.

Percussion is done by tapping with a plexor (hammer) on a pleximeter placed on the body, or by a finger on another finger. This is *mediate* percussion. In *immediate* percussion the examined part of the body is struck directly by the soft tip of the index finger. To make tapping stronger, the index finger may be first held by the side of the middle finger and then released. This method was proposed by Obraztsov. Its advantage is that the striking finger feels the resistance of the examined part of the body.

Percussion is done with a slightly flexed middle finger on the dorsal side of the second phalanx of the middle finger of the opposite hand, which is pressed tightly against the examined part of the body. Percussion should be done by the movement of the wrist alone without involving the forearm into the movement. Striking intensity should be uniform, blows must be quick and short, directed perpendicularly to the intervening finger. Tapping should not be strong.

Sounds obtained by percussion differ in strength (clearness), pitch, and tone. Sounds may be strong and clear (resonant) or soft and dull; they may be high or low, and either tympanic or non-tympanic (and with metallic tinkling).

Resonance (clearness) of the percussion sound largely depends on the vibration amplitude: the stronger the tapping the louder is the sound; uniform strength of tapping is therefore required. A louder sound will be heard during percussion of an organ containing greater amount of air. In healthy persons resonant and clear sounds are heard in percussion of thoracic and abdominal organs filled with gas or air (lungs, stomach, intestine).

Soft or dull sound is heard during percussion of the chest and the abdominal wall overlying airless organs (liver, heart, spleen), and also during percussion of muscles (femoral sound). Resonant and clear sound will become soft if the amount of air decreases inside the lung or if liquid is accumulated between the lungs and the chest wall (in the pleural cavity).

The pitch of the sound depends on the vibration frequency: the smaller the volume of the examined organ, the higher the vibration frequency, hence the higher the pitch. Percussion of healthy lungs in children gives higher sounds than in adults. The sound of a lung containing excess air (emphysema) is lower than that of a healthy lung. This sound is called bandbox. Normal vibration frequency of a healthy lung during percussion is 109-130 per second, while in emphysema the frequency decreases to 70-80 c/s. Quite the opposite, if the pulmonary tissue becomes more consolidated, the frequency increases to 400 c/s and more.

The *tympanic* sound resembles the sound of a drum (hence its name: Gk *tympanon* drum). Tympany differs from a non-tympanic sound by higher regularity of vibrations and therefore it approaches a musical tone, while a non-tympanic sound includes many aperiodic vibrations and sounds like noise. A tympanic sound appears when the tension in the wall of an air-containing organ decreases. Tympany can be heard during percussion of the stomach and the intestine of healthy people. Tympany is absent during percussion of healthy lungs, but if the tension in the pulmonary tissue decreases, tympanic sounds can be heard. This occurs in incomplete compression of the lung by the pleural effusion, in inflammation or edema of the lung (the percussion sound then becomes dull tympanic). A tympanic sound can also be heard if air cavities are formed in the lungs or when air penetrates the pleural cavity. Tympany is heard over large caverns and in open pneumothorax (the sound is resonant).

Since air-filled organs produce resonant percussion sounds and airless organs give *dull sounds*, the difference between these sounds helps locate the borders between these organs (e.g. between the lungs and the liver, the lungs and the heart, etc.).

Topographic percussion is used to determine the borders, size and shape of organs. Comparison of sounds on symmetrical points of the chest is called *comparative percussion.*

Tapping strength can vary depending on the purpose of the examination. Loud percussion (with a normal force of tapping), light, and lightest (threshold) percussion are differentiated. The heavier the percussion stroke, the greater is the area and depth to which the tissues are set vibrating, and hence the more resonant is the sound. In heavy or deep percussion, tissues lying at a distance of 4-7 cm from the pleximeter are involved. In light or surface percussion the examined zone has the radius of 2- 4 cm. Heavy percussion should therefore be used to examine deeply located organs, and light percussion for examining superficial organs.

Light percussion is used to determine the size and borders of various organs (liver, lungs and heart). The lightest percussion is used to determine absolute cardiac dullness. The force of the percussion stroke should be the slightest (at the threshold of sound perception). The Goldscheider method is often used for this purpose, the middle finger (flexor) of the right hand is used to tap the middle finger of the left hand flexed at the second phalanx and placed at a right angle touching the surface only with the soft tip of the terminal phalanx (pleximeter).

Comparative percussion should be carried out on exactly symmetrical parts of the body. A certain sequence is followed in comparative percussion. Percussion sounds over the lung apices (in the front) on the symmetrical points of the chest are first compared; the pleximeter finger is placed parallel to the clavicle. The plexor finger is then used to strike the clavicle, which is used as a pleximeter in this case. During percussion of the lungs below the clavicle, the pleximeter finger is placed in the interspace at the strictly symmetrical points of the left and right sides of the chest. The percussion sounds are compared only to the level of the 4th rib along the medioclavicular line (and medially). The heart lying below this level changes the percussion sound. For comparative percussion of the axillary region, the patient should raise his arms and clamp the hands at the back of the head. Comparative percussion of the lungs on the back begins with suprascapular areas. The pleximeter finger is placed horizontally while during percussion of the regions between the scapulae, the pleximeter should be vertical. The patient should cross his arms on the chest to displace the scapulae anteriorly (away from the backbone). During percussion of the points lying below the scapulae, the pleximeter should again be horizontal; in the interspace it should be placed parallel to the ribs.

Percussion sounds of the lungs of a healthy person cannot be of equal strength, length or pitch even if the percussion blows are uniform at symmetrical points. This depends on the mass and thickness of the pulmonary layer and also on the influence of the adjacent organs on the percussion sound. It is softer and shorter:

- over the right upper lobe because it is located somewhat below the left (due to the shorter right upper bronchus) and also because of the better-development of the muscles of the apt side of the shoulder girdle;
- in the second and third interspace on the left, because of the closer location of the heart;
- over the upper lobes of the lung (compared with the lower lobes) because of the varying thickness of pneumatic pulmonary tissue;
- in the right axillary region (compared with the left one) because of the closer location of the liver.

The difference in percussion sounds here depends on me tact that the diaphragm and the lung border on the left with the stomach whose bottom is filled with air and gives a loud tympanic sound during percussion (Traube's semilunar space).The percussion sound in the left axillary region is therefore louder and higher (with tympanic character) because of the resonant effect of the stomach.

The percussion sound can change in pathological processes because of the decreased content or full absence of air in a part of the lung and because of the pleural fluid (transudate, effusion, blood), increased airiness of the lung tissue, and the presence of air in the pleural cavity (pneumothorax).

The amount of air in the lungs decreases: in the pneumosclerosis fibrous-focal tuberculosis; pleural adhesion or oblitaration of pleural cavity which interferes with normal distension of the lung during inspiration; the difference in the percussion sound will be more pronounced at the inspiration level and weaker during expiration; lobular and especially confluent pneumonia, in which pulmonary tissue alternates with consolidations; considerable edema of the lungs, especially in the anteriolateral regions due to insufficient contractility of the left ventricle; compression of the pulmonary tissue by the pleural fluid (compression atelectasis) above the fluid level; complete obstruction of the large bronchus with a tumor and gradual resorption of air from the lungs below the closure of the lumen (obstructive atelectasis).

Clear pulmonary sounds become shoter and higher (i.e. duller) in the mentioned pathological conditions. If this conditions are attended by decreased tension in the elastic elements of the pulmonary tissue, e.g. in the presence of compression or obstructive atelectasis, the sound of the atelectasis zone decode dull with a tympanic tone. This sound can also be heard during percussion of the patient with acute lobar pneumonia at its first stage, when the alveoli of the affected lobe, in addition to air, contain also a small amount of fluid.

A complete absence of air in the entire lobe of the lung or its part (segment) is observed in the following cases: acute lobar pneumonia at the consolidation stage, when the alveoli are filled with the inflammatory exudates containing fibrin; formation in the lung of a large cavity, which is filled with the

inflammatory-fluid (sputum, pus, echinococcous acid, etc.), or heterogeneous airless tissue (tumor); accumulation of fluid in the pleural cavity (transudate, exudate, blood).

Percussion over airless parts of the lung or over fluid accumulated in the pleural cavity a soft short and high sound that is called dull or by analogy with the percussion sounds of airless organs and tissue (liver, muscles), liver dullness. But the absolute dullness identical to the percussion sound of the liver can only be heard in the presence of a large amount of fluid in the pleural cavity.

The amount of air the lung increases in emphysema. The percussion sound in lung emphysema is louder that the dull tympanic sound because of the increased airiness of the pulmonary tissue and decreased elasticity of the tense pulmonary tissues; but the tympanic character as preserved. The percussion sound resembles the one produced by a stroke on a box; hence the name *bandbox sound*.

The amount of air held inside the lung increases with formation in it of a smooth-wall cavity filled with air and communicated with the bronchus (abscess, tuberculotic cavern). The percussion sound over this area will be tympanic. If the cavity is small and situated deeply in the chest, vibrations of the pulmonary tissue will not reach this cavity and no tympanic sound will be heard.

Such a cavity will only be revealed by roentgenoscopy. The sound over a very large smooth-wall cavity in the lung will be tympanic, resembeling a stroke on a metal (*metallic percussion sound*). If this cavity is located superficially and is communicated with the bronchus through a narrow slit, the percussion sound will be soft and will resemble that of a cracked pot (hence the name – *cracked-pot sound*).

***Theme 5*. Topographic Percussion of the Lungs**.

Basic topographic chest lines. Basic tasks and sequence of topographic percussion of the lungs. Determination of the height of the apices of the lungs anterior and posterior, Kroenig´s fields, lower border of the lungs, mobility of the lungs border. Traube´s space, its value at pathology of the lungs.

The contents: To designate the location of the revealed normal or pathological findings it is convenient to use vertical (ordinates) and horizontal (abscissas) lines. The ribs can play the role of abscissas, the vertical lines drown through the definite points on the chest can serve as ordinates.

The lines are as follows:

1. Anterior median line (*l. mediana*) going vertically through the middle of the chest.
2. Right and left sternal lines (l. sternalis dextra et sinistra) going along the both edges of the breastbone.
3. Right and left parasternal lines (*l. parasternalis dextra et sinistra*) going vertically between the two above mentioned.
4. Right and left medioclavicular lines (*l. medioclaviculare dextra et sinistra*) going through the middle of the both collarbones.
5. Right and left anterior axillary lines (*l. axillare anterior dextra et sinistra*) going through the anterior edges of the armpits.
6. Right and left middle axillary lines (*l. axillare medius dextra et sinistra*) going through the middle edges of the armpits.
7. Right and left posterior axillary lines (*l. axillare posterior dextra et sinistra*) going vertically through the posterior edges of the armpits.
8. Right and left scapular lines (*l. scapulare dextra et sinistra*) going vertically through the angles of the shoulder blades.
9. Right and left paravertebral lines (*l. paravertebrale dextra et sinistra*) going vertically between the scapular line and the line going through the processes of the vertebrae.

As these lines go through easily recognizable points they can be determined mentally. If a change is noticed not in the place of crossing the rib and one of the lines, the distance to the nearest line in centimeters is determined. The ribs are easily counted beginning from the second rib on the front, its cartilage is attached to the breastbone at the level of the so-called angulus Ludovici (the angle between the manubrium of sternum and its body). The rib of this angle is easily detected when drawing a line with a finger downward along the breastbone. It is frequently seen like a roller in the upper portion of the sternum. It is also easy to find the 7th rib as this is the last rib attached to the sternum with its cartilage. Vertebral processes are used to orient on back, the process of the 7th cervical vertebra is felt as it is prominent when the head is bend forward. If three vertebrae are prominent together,

the middle one is the 7th cervical vertebra. Besides, the orienting points can be clavicles, the axis of the shoulder blade, is lower angle, xiphoid process as well as fossae on the chest (supra- and subclavicular).

Topographic percussion is used for determining: the upper borders of the lungs or the upper level of their apices and their width (Kroenigs area); the lower borders of the lungs, and variation mobility of the lower border of the lung.

The position of the *upper borders* (apices) of the lungs is determined both anteriorly and posteriorly. In order to locate the apex of the lung, the pleximeter finger is placed parallel to the clavicle and percussion is effected from the middle upwards and slightly medially, to dullness. The upper level of the apices in healthy persons is 3 - 4 cm. The upper posterior border of the lungs is always determined by their position with respect to the spinous process of the 7th cervical vertebra. The pleximeter finger is placed over the supraspinous fosse parallel to the scapular spine and stroked from the middle. The pleximeter finger is moved gradually upward to the point located 3 - 4 cm laterally to the spinous process of the 7th cervical vertebra, at its level, and percussion is then continued until dullness. Normal height of the lung, apices (posterior) is about at the level of the spinous process of the 7th cervical vertebra.

The so-called *Kroenig's area* is a band of clear resonance over the lung apices. The width of these areas is determined by the low anterior border of the trapezius muscle and is (on an average) 5-6 cm wide, but its width can vary from 3 to 8 cm. The anterior border of the trapezius muscle divide the Kroenig area into its anterior field, which extends to the clavicle, and the posterior one that widens toward the supraspinous fossae. Light or subliminal percussion is used to determine the width of the lung apex. The pleximeter finger is held over the middle portion of m. trapezius, perpendicular to its anterior margin, and percussion is first carried out medially, and then laterally, to dullness. The distance between the points of transition of the clear pulmonary resonance to dullness is measured in centimeters.

The upper border of the lungs and the width of the Kroenig area can vary depending on the amount of air in the apices. If the amount of air is high (which may be due to emphysema) the apices increase in size and move upwards. The Kroenig area widens accordingly. The presence of connective tissue in the lung apex (which usually develops *during infiammation as* in tuberculosis or pneumonia or inflammatory infiltration) decreases the airiness of the pulmonary tissue. The upper border of the lung thus lowers and the width of the Kroenig area decreases.

To outline the *lower borders* of the lungs their percussion is carried out in the downward direction along conventional vertical topographical lines. The lower border of the right lung is first determined anteriorly along the parasternal and the medioclavicular lines, then laterally along the anterior, medial and posterior axillary lines, and posteriorly along the scapular and paraspinal lines. The lower border of the left lung is determined only laterally, by the three axillary lines, and posteriorly by the scapular and paraspinal lines. The lower border of the left lung is not determined anteriorly because of the presence of the heart. The pleximeter finger is placed in the interspaces, parallel to the ribs, and the plexor finger produces slight and uniform strokes over it. Percussion of the chest is usually begun anteriorly, from the second and third costal interspace (with the patient in the lying or upright position). The examination of the lateral surface of the chest is performed from the axillary fossae (arm pit). The patient either sits or stands with the hands behind the back of the head. The examination ends with the posterior percussion from the seventh costal interspace, or from the scapular angle, which ends at the seventh rib.

The lower border of the right lung is as a rule at the point of transition of the clear pulmonary resonance to dullness (lung-liver border). In exceptional cases, when air is present in the abdominal cavity (e.g. in perforation of gastric or duodenal ulcer), liver dullness may disappear. The clear pulmonary resonance will then convert to tympany. The lower border of the left lung by the anterior and midaxillary lines is determined by the transition of clear pulmonary resonance to dull tympany. This is explained by the contact between the lower surface of the lung (through the diaphragm) and a small airless organ, such as the spleen and the fundus of the stomach, which give tympany (Traube's space).

Normal Lower Border of the Lungs

Percussion point	*Right lung*	*Left lung*
Parasternal line	6th intercostal space	4th rib

Midclavicular line	6th intercostal space	4th rib
Anterior axillary line	7th intercostal space	7th intercostal space
Midaxillary line	8th intercostal space	8th intercostal space
Posterior axillary line	8th intercostal space	8th intercostal space
Scapular line	9th rib	9th rib
Paraspinal line	Spinous process of the 11th thoracic vertebra	

The position of the border varies depending on the constitutional properties of the body. The lower border of the lungs in asthenic persons is slightly lower than in normosthenics and is found at the interspace (rather than on the rib as in normosthenics) whereas this border is slightly higher in hypersthenic persons. The lower border of the lungs rises temporary during late pregnancy.

The position of the lower border of the lungs can vary in various pathological conditions that develop in the lungs, the pleura, the diaphragm, and the abdominal viscera. The border can both rise and lower from the normal level. This displacement can be uni- or bilateral.

Bilateral lowering of the lower border of the lungs can occur in acute and chronic dilation of the lungs (attack of bronchial asthma and emphysema of the lungs, respectively) and also in sudden weakening of the tone, of the abdominal muscles and lowering of the abdominal viscera (splanchnoptosis). Unilateral lowering of the lower border of the lungs can be due to vicarious (compensatory) emphysema of one lung with inactiva-hemiparesis of the diaphragm).

The elevation of the lower border of the lungs is usually unilateral and occurs in shriveling of the lung due to development of connective tissue (pneumosclerosis); complete obstruction of the lower lobe bronchus by a tumor which causes gradual collapse of the lung, atelectasis: accumulation of fluid or air in the pleural cavity which displace the lung upwards and medially toward the root: marked enlargement of the liver (cancer, echinococcosis), or of the spleen (chronic myeloleukaemia). Bilateral elevation of the lower borders of the lungs occurs in the presence of large amounts of fluid (ascites) or air in the abdomen due to an acute perforation of gastric or duodenal ulcer, and also in acute meteorism.

After determining the lower border of the lungs at rest, *respiratory mobility* of pulmonary borders should be determined by percussion during forced inspiration and expiration. This mobility is called active, and is usually measured by the difference in the position of the lower border of the lungs between the two extremes. Measurements are done by line axillary. The normal variation of the lower border of the lungs is 6-8cm.

The respiratory mobility of the lungs is determined as follows. The lower border of the lungs in normal respiration is first determined and marked by a dermograph. The patient is then asked to make a forced inspiration and to keep breath at the height. The pleximeter finger should at this moment be held at the lower border of the lung (determined earlier). Percussion is now continued by moving the pleximeter downwards to complete dullness, where the second mark should be made by a dermograph at the upper edge of the pleximeter finger. The patient is then asked to exhale maximum air from the lungs and to keep breath again. The percussion is now continued in the upward direction until the clear vesicular resonance appears. The third dermographic mark should be made at the point where relative dullness is heard. The distance between the extreme marks is measured. It corresponds to the maximum respiratory mobility. The patient in a grave condition is unable to keep breath and another method is recommended to determine the respiratory mobility of the lungs.

After marking the lower border of the lung in quiet breathing, the patient is asked to make deep inhalation and exhalation without keeping breath. Percussion should be continuous during deep breathing and the pleximeter finger should gradually move downwards. First the percussion sound is loud and low during inhalation and soft and high during exhalation. Soon a point is attained where the sounds become of the same pitch and strength during both inhalation and exhalation. This point is the lower border of the lung at forced inspiratortion. The lower border at forced exhalation is determined in the same way.

Respiratory mobility of the lower border of the lungs is diminished in inflammatory infiltration or congestive plethora of the lungs, decreased elasticity of the pulmonory tissue (emphysema), profuse pleural effusion, and in pleural adhesion or obliteration.

The so-called passive respiratory mobility is determined in some diseases. This is the mobility of the lung borders during changes in the posture. When the patient changes his posture from the upright to horizontal one, the lower border of the lungs descends some 2 cm, while the lower border of the right lung of a patient lying on his left side may lower 3-4 cm. In pathological conditions, e.g. in pleural adhesion, the variation of the lower border of the lungs is markedly limited.

***Theme 6*. Auscultation of the Lungs. The Methods of Auscultation of the Lungs. Main Respiratory Sounds.**

History of auscultation invention. Auscultation rules. Instructions for using a stethoscope and phonendoscope. The methods of realization of comparative auscultation of the lungs. Main respiratory sounds: vesicular and bronchial respiration (mechanism of formation, changes). Bronchophony and its diagnostic significance.

The contents: Auscultation (L *auscultare* to listen) means listening to sounds inside to body. Auscultation is *immediate (direct)* when the examiner presses his in to the patient's body, or *mediate (indirect,* or instrumental). Auscultation was first developed by the French physician Laennec in 1816. In 1819 it was described and introduced into medical practice. Laennec also invented the first stethoscope. He substantiated the clinical value of auscultation by checking its results during section. He described and named almost all the auscultative sounds (vesicular, bronchial respiration, crepitation, murmurs). Thanks to Laennec, auscultation soon became an important method for diagnostication of lung and heart disease and was acknowledged throughout the world.

The development of auscultation technique is connected with improvement of the stethoscope (Piorri, Yanovsky, and others), invention of the binaural stethoscope (Filatov and others), invention of the phonendoscope, and the study of the physical principles of auscultation (Skoda, Ostroumov, Obraztsov, and others). Elaboration of methods for recording sounds (phonography) that arise in various organs has become a further development of auscultation. The graphic record of heart sounds was first made in 1894 by Einthoven. Improved phonographic technique made it possible to solve many important auscultation problems and showed the importance of this diagnostic method.

Like percussion, auscultation of the lungs should be carried out according to a plan. Stethoscope or phonendoscope should be placed in strictly symmetrical points of the right and left sides of the chest. Auscultation begins with the anterior wall of the chest, from its upper part, in the supra- and subclavicular regions, and then the stethoscope should be moved downward and laterally. The lungs are then auscultated in the same order from the posterior wall of the chest and in the axillary regions. In order to increase the area of auscultation between the scapulae, the patient should be asked to cross his arms on the chest and in this way to displace his shoulder-blades laterally from the spine, while for convenience of auscultation of the axillary regions he should place his hands on the back of the head.

The posture does not matter, but the patient should better sit up on a stool with his hands on the laps. The patient may stand, but the physician should remember that deep breathing (hyperventilation of the lungs) may cause vertigo and the patient may faint. Bearing this in mind, and also to ensure a tight contact between the stethoscope and the skin (especially if a one-piece stethoscope is used) the physician should always use his free hand to support the patient on the side opposite to the point of application of the stethoscope bell gradually increasing. This sound can be simulated by pronouncing the sound during inspiration, or by drawing tea from a saucer alveolar walls still. A shorter second phase of the vesicular breathing, which is heard only during the first third of the expiration phase, because vibrations of elastic alveolar walls are quickly dampened by the decreasing tension of the alveolar walls.

Vesicular breathing appearance is caused by vibrations of extending elastic alveolar walls, heard the whole inspiration and only during the first third of the expiration phase. Normal vesicular breathing is better heard over the anterior-surface of the chest, in the axillary regions and below the scapulae. The largest masses of the pulmonary tissue are located. While carrying out comparative auscultation, it should be remembered that the expiration sounds are louder and longer in the right lung due to a better

conduction of the laryngeal sounds by the right main bronchus, which is shorter and wider. The respiratory sound sometimes becomes bronchovesicular over the right apex; or it may be mixed due to more superficial and horizontal position of the right apical bronchus.

Alterations in vesicular breathing. Vesicular breathing can vary, i.e. it may be louder or softer for both physiological and pathological reasons. Physiological weakening of vesicular breathing occurs in patients with fill muscles or subcutaneous fat.

Physiological intensification of vesicular breathing may be observed in patients with underdeveloped muscles or subcutaneous fat. Intensified vesicular breathing is characteristic of children with a think wall, good elastics interalveolar septa. This respiration is called "puerile respiration" (L puer-child). Vesicular respiration is intensified during exercise; respiratory movements become deeper and more frequent. Physiological changes in vesicular respiration always involve both parts of the chest, and respiratory sounds are equally intensified or weakened at the symmetrical points of the chest.

In pathology, alterations in vesicular breathing may be both uni- and bilateral, or else only over one lobe of the lung. Respiratory sounds become weaker or inaudible at all; or they may be intensified. Alterations in vesicular respiration in such cases depend on the amount of intact alveoli and the properties of their walls, the amount of air contained in them, on the length and strength of the expiration and inspiration phases, and finally on the conditions of sound conduction from the vibrating elastic elements of the pulmonary tissue to the surface of the chest.

Pathologically decreased vesicular respiration can be due to a significantly diminished number of the alveoli because of atrophy and gradual degradation of the interalveolar septa and formation of the larger vesicles incapable collapsing during expiration. This pathological condition is observed in pulmonary emphysema, at which the remaining alveoli are no longer elastic; their walls become incapable of quick distention and do not give sufficiently strong vibrations.

Decreased vesicular breathing can be due to inflammation and swelling of the walls in a part of the lung and decreased amplitude of their vibration during inspiration, which is characteristic of early acute lobar pneumonia. During the second stage of this disease, the alveoli of the affected part of the lung become filled with effusion and vesicular breathing becomes inaudible in this region. Vesicular breathing can be decreased also in insufficient delivery of air to the alveoli through the air ways because of their mechanical obstruction (e.g. by a tumor). Air admission to the alveoli can be decreased in a patients with a markedly weakened inspiration phase (as a result of the inflammation of the respiratory muscles, intercostals nerves, rib fracture, extreme asthenia of the patient and adynamia).

Vesicular respiration decreases also due to obstructed conduction of sound waves from the source of vibration (alveolar walls) to the chest surface, as, for example, in thickening of the pleural layers or accumulation of air or fluid in the pleural cavity. If the amount of fluid or air in the pleural cavity is great, respiratory sounds are not heard. Conduction of sound to .the surface of the chest may be absent in atelectasis of the lung due to complete obstruction of the lumen in the large bronchus.

Abnormally increased vesicular breathing can be heard during expiration during both respiratory phases. Increased expiration depends on obstruction to the air passage through small bronchi or their contracted lumen (inflammatory edema of the mucosa, bronchospasm). Expiration becomes louder and longer.

Deeper vesicular breathing, during which the inspiration and expiration phases are intensified, is called harsh. It occurs in marked and non-uniform narrowing of the lumen in small bronchi and bronchioles due to inflammatory edema of their mucosa (bronchitis).

Another type of pathological respiration is interrupted cogwheel respiration. This vesicular respiration is characterized by short jerky inspiration efforts interrupted by short pauses between them; the expiration is usually normal. Interrupted breathing also occurs in non-uniform contraction of the respiratory muscles, e.g. when a patient is auscultated in a cold room, or when he has nervous trembling, or diseases of the respiratory muscles, etc. Interrupted breathing over a limited part of the lung indicates difficult passage of air from small bronchi to the alveoli in this region and an even unfolding of the alveoli. Interrupted breathing indicates pathology in fine bronchi and is more frequently heard at the apices of the lungs during their tuberculous infiltration.

Bronchial breathing. Respiratory sounds known as bronchial or tubular breathing arise in the larynx and trachea as air passes through the vocal slit. As air is inhaled, it passes through the vocal slit to enter wider trachea where it is set in vortex-type motion. Sound waves thus generated propagate along the air column throughout the entire bronchial tree. Sounds generated by the vibration of these waves are harsh. During expiration, air also passes through the vocal slit to enter a wider space of tie larynx where it is set in a vortex motion. But since the vocal slit is narrower during expiration, the respiratory sound becomes louder, harsher and longer. This type of breathing is called laryngotracheal. Bronchial breathing is well heard in physiological cases over the larynx and trachea, at points of projection of the tracheal bifurcation anteriorly, over the manubrium sterni, at the point of its junction with the sternum, and posteriorly in the interscapular space at the level of the 3rd and 4th thoracic vertebrae). Bronchial breathing is not heard over the other arts of the chest because of large masses of the pulmonary tissue found between the bronchi and the chest wall.

Bronchial breathing can be heard instead of vesicular over the chest in pulmonary pathology. This breathing is called *pathological bronchial respiration*. It is conducted to the surface of the chest wall only under certain conditions, the main one being indurations of the pulmonary tissue where the alveoli are filled with effusion (acute lobar pneumonia, tuberculosis), with blood (lung infarction), and due to compression of the alveoli by the air and fluid accumulated in pleural cavity, and compression of the lung against its root (compression atelectasis). In such cases the alveolar walls do not vibrate, while consolidated airless pulmonary tissue becomes a good conductor of sound waves in laryngotracheal respiration to the surface of the chest wall. Lungs may be consolidated due to replacement of the inflammatory pulmonary tissue by connective tissue (pneumosclerosis, carnification of the lung lobe, which sometimes occurs in acute lobar pneumonia due to growth of connective tissue into the inflamed lobe of the lung).

Depending on degree of induration, its size and location in the lung, pathological bronchial breathing may have different intensity and pitch. If the induration is large and superficial, loud bronchial breathing is heard. It is heard as if near the ear; the pitch of the sound is higher in this case. Bronchial breathing can be heard in acute lobar pneumonia at its second stage (affection of the entire lobe of the lung). If a segment of a lung is indurated, and the affection is deep seated, breathing will be weaker and the pitch lower. This sound can be heard in lobular pneumonia if several foci are close to one another or fuse together to form a large focus of induration (confluent pneumonia). Especially soft and low sounds are heard in patients with compression atelectasis. The sound resembles an echo, as if entering the physician's ear from a far off source.

Pathological bronchial respiration can be heard if an empty cavity is formed in the lung (abscess, cavern) and it is communicated with the bronchus. Consolidation of pulmonary tissue round the focus facilitates conduction of sound waves of laryngotracheal breathing to the surface of the chest wall, the more so that the sound is intensified in the resonant cavity and that at the moment of air passage from the narrow bronchus the air is set in a vortex motion.

Amforic respiration arises in the presence of a smooth-wall cavity (not less than 5-6cm in diameter) communicated with a large bronchus. Because of a strong resonance additional high overtones appear along with the main low-pitch laryngotracheal breathing. These overtones alter the man tone of the bronchial respiratory sound. Sounds of this kind can be produced by blowing over the mouth of an empty glass or clay air. This altered bronchial breathing is therefore called amphoric.

Metallic respiration differs from both bronchial and amphoric. It is loud and high, and resembles the sound produced when apiece of metal is struck. Metallic respiration is heard in open pneumotorax when the air of the pleural cavity communications with the external air.

Stenotic respiration is exaggerated laryngotracheal breathing, which is heard in cases with narrowed trachea or a large bronchus (due to a tumor); it is heard mainly at points where physiological bronchial breathing is normally heard.

Bronchovesicular or mixed respiration heard in lobular pneumonia or infiltrative tuberculosis, and also in pneumosclerosis, with foci of consolidated tissue being seated deeply in the pulmonary tissue and far from one another. Mixed breathing, when the inspiration phase is characteristic of vesicular breathing and the expiration phase of bronchial breathing, is often heard in such cases instead of weak bronchial breathing.

Bronchophony. This is the voice conduction by the larynx to the chest, is determined by auscultation. But as distinct from vocal fremitus, the words containing sounds 'r' or 'ch' are whispered

during auscultation. In physiological conditions, voice conducted to the outer surface of the chest is hardly audible on either side of the chest in symmetrical points. Exaggerated bronchophony suggests consolidation of the pulmonary tissue and also cavities in the lungs which act as resonators to intensify the sounds. Bronchophony is more useful than vocal fremitus in revealing consolidation foci in the lungs of a patient with soft and high voice.

Theme 7. **Auscultation of the Lungs: Accessory Respiratory Murmurs.**

Classification of accessory respiratory murmurs (rales, crepitation, pleura friction rub). Mechanism of formation and classification of dry and moist rales. Mechanism of formation of crepitation, pleura friction rub. Discrimination of accessory respiratory murmurs. Mechanism of formation of additional respiratory phenomena (succussio Hippocratis, dripping sound) and their diagnostic significance.

The contents: Adventitious sounds are rales, crepitation and pleural friction. Rales arise in pathology of he trachea, bronchi, or if cavern is formed in the affected lung. Rales are classified as dry (rhonchi) and moist rales.

Dry rales are rhonchi, may be due to various causes. The main one is constriction of the lumen in the bronchi. Constriction may be total (in bronchial asthma) non-uniform (in bronchitis), or local (in tuberculosis tumor of the bronchus). Dry rales can be due to:

1) spasms of smooth muscles of the bronchi during fits bronchial asthma;
2) swelling of the bronchial mucosa during its inflammation;
3) accumulation of viscous sputum in the bronchi which adheres to the wall of the bronchus its narrows its lumen;
4) formation if fibrouse tissue in the walls of separate bronchi and in the pulmonary tissue with subsequent alteration of their architectonics (bronchiectasis, pneumosclerosis);
5) vibration of viscous sputum in the lumen of large and medium size bronchi during inspiration and expiration: being viscous, the sputum can be drawn (by the aire stream) into threads, which adhere to the opposite walls of the bronchi and vibrate like strings.

Dry rales are heard during inspiration and expiration and vary greatly in their loudness, tone and pitch. According to the quality and pitch of the sounds produced, dry rales are divided into sibilant (high-pitched and whistling sounds) and sonorous rales (low-pitched and sonoring sounds). High-pitched rales are produced when the lumen of the small bronchi is narrowed, while low-pitched sonorous rales are generated in stenosis of medium calibre and large calibre bronchi or when viscous sputum is accumulated in their lumen.

Moist rales are generated because of accumulation of liquid secretion (sputum, edematous fluid, blood) in the bronchi through which air passes. Air bubbles pass through the liquid secretion of the bronchial lumen and collapse to produce the specific cracking sound. This sound can be simulated by bubbling air through water using a fine tube. Moist rales are heard during both the inspiration and expiration but since the air velocity is higher during inspiration moist rales will be better heard at this respiratory phase.

Depending on the calibre of bronchi where rales are generated, they are classified as fine, medium and coarse bubbling rales. Fine bubbles rales generated in fine bronchi and are percepted by the ear as short multiple sounds. Kales originating in the finest bronchi and bronchioles are similar to crepitation from which they should be differentiated. Medium bubbles rales are produced in bronchi of a medium size and coarse bubbles rales in large caliber bronchi, in large bronchiectases, and in pulmonary cavities (abscess, cavern) containing liquid secretions and communicating with the large bronchus. Large bubbling rales are characterized by a lower and louder sound.

Moist rales originating in superficially located large cavities (5-6 cm and over in diameter) may acquire a metallic character. If segmentary bronchiectases or cavities are formed in the lung, rales can usually be heard over a limited area of the chest. Chronic bronchitis or marked congestion in the lungs associated with failure of the left chambers of the heart is as a rule attended by bilateral moist rales of various calibre, which occur at the symmetrical points of the lungs.

Depending on the character of the pathology in the lungs, moist rales are subdivided into **consonating** or crackling as **non-consonating** bubbling rales. Consonating moist rales are heard in the presence obliquid secretions in the bronchi surrounded by airless consolidated pulmonary tissue or in lung cavities with smooth

waifs surrounded by consolidated pulmonary tissue. The cavity it self acts as a resonator to intensify moist rales. Moist consonating rales are heard as if just outside the ear. Consonating rales in the lower portions of the lungs suggest inflammation of the pulmonary tissue surrounding the bronchi. Consonating rales heard in the subclavicular or subscapular regions indicate tuberculous infiltration or cavern in the lung.

Non-consonating rales are heard in inflammation of bronchial mucosa (bronchitis) or acute edema of the lung due to the failure of the left chambers of the heart.

The so-called falling-drop sound (gutta cadens) can be heard by auscultation. It can occur large cavities of the lungs or at the base of the pleural cavity, which contain liquid pus or air the patient changes his posture from recumbent to upright position or vice versa. Tenacious quid containing pus sticks to the surface of the cavity and as the patient changes his position gathers in drops, which fall one after another into the liquid (sputum or pus) accumulated at le bottom.

Crepitation. As distinct from rales, crepitation originates in the alveoli. Some authors erroneously classify these, sound as crepitant and subcrepitant rales. Crepitation is a slight crackling sound that can be imitated by dubbing a lock of hair. The main condition for generation of crepitation is accumulation of a small amount of liquid secretion of the alveoli. During expiration the alveoli stick together, while during inspiration the alveolar walls separated with difficulty and only at the end of the inspiratory movement. Crepitation is therefore only heard during the height of inspiration. In other words, crepitation is the sound produced by many alveoli during their simultaneous reinflation.

Crepitation is mainly heard in inflammation of the pulmonary tissue, at the first (initial) and third (final) stages of acute lobar pneumonia, then the alveoli contain small amounts of inflammatory exudate, in pulmonary tuberculosis, lungs infarction, and finally in congesons that develop due to insufficient contractile function of the left ventricular myocardium or in marked stenosis of the left venous orifice of it heart and in compressive atelectasis. By its acoustic properties, crepitation can often resemble moist fine lies that are produced m fine bronchi or bronchioles filled with liquid secretion. Differential diagnostic or these rales and crepitation are as follows: moist fine rales are heard during both inspiration and expiration; they can be intensified had disappear after coughing, while crepitation can only be heard at the height of inspiration or not does it change after coughing.

Pleural friction sound. In physiological conditions visceral and parietal years of the pleura are constantly "lubricated" by a capillary layer of noiseless. Various pathological conditions alter the physical properties of the pleural surfaces and their friction against one another becomes more intense to generate a peculiar adventitious noise, known as the pleural friction sound. Fibrin is deposited in inflamed pleura to make its surface through; moreover, cicatrices, commissures, and bands are formed between pleural layers at the focus of inflammation. Tuberculosis or cancer are also responsible for the friction sounds.

Pleural friction sounds are heard during both inspiration and expiration. Intensity, or loudness, length, and over which they are heard differentiate the sounds. During early dry pleurisy the sounds are soft can be imitated by rubbing silk or fingers in the close vicinity of the ear. The character of pleural friction sound is altered during the active course of dry pleurisy. It can resemble crepitation or fine bubbling rales sometimes crackling of snow. In pleurisy with effusion, during the period of rapid resorption of exudate, the friction sound becomes coarser due to passive deposits on the pleural surfaces. The time during which pleural friction sound can be heard varies with diseases. For example, in rheumatic pleurisy pleural friction is only heard during a few hours; after a period of quiescence it reappears. Pleural friction persists for a week and over in dry pleurisy of tuberculous etiology and pleurisy with effusion at the stage of resorption. Pleural friction sounds can be heard in some patients for years after pleurisy because of large cicatrices and roughness of the pleural surfaces.

The point over which pleural friction can be heard depends on the focus of inflammation. Most frequently it is heard in the inferolateral parts of the chest, where the lungs are most mobile during respiration. In rare cases this sound can be heard over the lung apices, when they are affected by tuberculosis with involvement of the pleural membranes.

Pleural friction sounds can be differentiated from fine bubbling rales and crepitation by the following signs:

1) the character of rales is altered or rales can disappear for a short time after coughing, while pleural friction sound does not change in these conditions;

2) when a stethoscope is prested tighter against the chest, the pleural friction sound is intensified, while rales do not change;

3) crepitation is only heard at the height of inspiration;

4) if a patient moves his diaphragm in and out while his mouth and nose are closed, the sound produced by the friction of the pleura due to the movement of the diaphragm can be heard, while rales and crepitation cannot because there is no air movement in the bronchi.

Succusion (Hippocratic) sound. This is the splashing sound heard in the chest of a patient with hydropneumothorax, i.e. when serous fluid and air are accumulated in the pleura cavity. The sound was first described by Hippocrates, hence the name. The sound can be identified by auscultation: the physician presses his ear against the chest of the patient and then shakes the patient suddenly. The splashing sounds are sometimes heard by the patient himself during abrupt movements.

Dripping sound also appears in hydro- or pneumothorax, sometimes in a large cavern. It appears when the drops of fluid drop from the upper cupola of the cavity on the surface of the fluid. It appears if the patient sits up. Similar bell sound may appear due to a good resonance at formation of moist riles in a large cavity with smooth walls.

Theme 8. Questioning and External Examination of the Patients with the Disorders of Cardiovascular System.

The basic physical methods of examination of cardiovascular system (questioning, external examination, palpation, percussion, auscultation). The sequence of determining and detail in of patients with the disorders of cardiovascular system. Taking a case history and past history. External examination of the patients with the disorders of cardiovascular system. Independent work with patients. The information in the notebooks.

The contents: Main complaints of the patients with heart diseases are pains in the heart area, palpitation, dyspnea, edema. It is important to know every detail. When the patients feels the *pain in the heart*, it is necessary to know the place of the pain, radiation, recurrence, character, duration, if this is effected by drugs, emotional stress, physical load, accompanying phenomena. The patients are not usually able to describe the peculiarities of the attack of pain. The physician should strictly follow the algorithm of the complaint study.

The most significant and frequent cause of pain in the precordial area associated with a heart disease is myocardium hypoxia resulting from discrepancies between the requirements of the cardiac muscle in oxygen and capabilities of the coronary circulation to meet them.

This pain develops in angina of effort, rest angina, myocardial infarction and may be the only sign of the disease. The pain is typical and the correct questioning allows making a diagnosis.

It is necessary to establish the place of pain. The patient should be asked to show with his finger the place or the region of pain. Typical anginal pain is localized behind the breastbone, commonly behind its body, sometimes behind its lower third, behind the xiphoid process, in the epigastric area and the manubrium. The pain rarely begins on the left of the sternum in the area of the heart apex. Typical anginal pains in the area of the left scapula, in the interscapular region, under one or both collarbones, in the left radiocarpal joint, jaw have also been reported.

Radiation of the pain is noted in 50 % of the patients with coronary artery disease. The pain usually radiates to the left arm at a various distance distally along the inner surface of the arm and forearm, to the elbow, wrist, 5th and 4th fingers, frequently the pain radiates to the both extremities and very rarely the pain is felt in the right upper extremity, the left or right side of the neck, lower jaw, chin, pharynx, ear, abdomen, lumbar area. Unusual radiation of the pain may suggest myocardial infarction.

Recurrence is a characteristic sign of ischemic coronary pains: they appear suddenly, usually without precursors, in the majority of cases they become worse within several seconds reaching their full intensity, and disappear quickly.

The pain in agnina lasts about 3 minutes, rarely 5-10 minutes. If the pain lasts >15 min this may indicate myocardial infarction, pericarditis or it is not associated with the heart disease.

The attacks of pain in coronary artery disease occur at various intervals: in severe cases up to 30 times a day, the remission between the periods of exacerbation may last for weeks and even years. Increased frequency or duration of angina attacks, when they begin at rest, is the sign of the disease progress or represents a danger of myocardial infarction.

The character of the pain is diagnostically significant: in coronary artery disease the pain is squeezing, pressing, the patient complains of feeling of weigh or pressure preventing free breathing behind the breastbone. The phenomenon of "breathing deceleration", when the patient stops on walking, is equivalent to angina. The pain is sometimes accompanied by feeling of fear, horror (angor praecordialis).

The conditions in which the pain develops are frequently decisive for making diagnosis: the pain is caused by the factors, which increase the requirement of the heart muscle in oxygen. The attack usually occurs with physical exertion and is quickly relieved by rest. The pain may appear on any physical exercise, but most frequently on fast walking for a long time, on walking upstairs, with cold, wind, after meals, emotions. The pain in light cases develops on walking upstairs, running, quick walking. In medium-severity cases the pain develops during the first 300 meters of walk. In severe cases the pain may develop on slow walking. The pain decreases and even disappears after stopping. In some cases the pain disappears suddenly, in the others gradually, sometimes not simultaneously behind the breastbone and the places of radiation. The attack of angina frequently develops after a quiet sleep. But sometimes the attack of pain develops in a lying position, especially at night (decubital angina). This attacks are longer (30 min and more), intensive, the pain decreases when the patients tries to sit up. Frequently these attacks appear in hypertensive patients, in cor pulmonale, valve defect, heart failure, they are associated with increase of minute heart volume in a lying position. An important sign of angina if the pain is relieved with nitroglycerin.

During the attack the patient is quiet, the breathing may be slow and superficial, the face is pale gray, the lips are cyanotic. The objective cardiac findings are normal, arterial pressure may be slightly elevated, the ECG demonstrates horizontal decline of ST segment, decreased T wave amplitude, negative T wave.

The changes in minute heart volume, blood flow velocity, venous pressure, arteriovenous gradient of oxygen saturation have not been revealed. Increased pulmonary capillary pressure during the attack has been described, this may be the cause of "respiration deceleration" phenomenon.

After the attack the patient feels better and can do his work without limitations.

In myocardial infarction the pain is severe, persistent (>30 min, frequently for several hours, so-called "status anginosus"). The intensity of the pain ranges from moderate dull pressure at the beginning of the attack to an unbearable pain, the patient becomes restless, expression of anxiety and horror appear on the face, he tries to change the position. The pain is localized behind the breastbone, near the xiphoid process, in the epigastric area, sometimes in the hypochondrium, radiates to one or both extremities, neck, back of the head, lower jaw, one or both shoulder blades, interscapular area, hypogastric areas. The pain is frequently accompanied by nausea, flatulence, vomiting, sings of intestinal obstruction (status gastralgicus). The pain in angina is associated with temporary reversible ischemia of the heart muscle.

The pain in the precordial area can be caused by dissecting aorta aneurysm. It occurs suddenly, is described as pulling, tearing, excruciating, pulsating, it is frequently preceded by dyspnea or collapse. The location, intensity, radiation of pain depend on location, size and direction of the dissection. A substernal pain usually develops with affection of the thoracic aorta. In this case the pain may be felt in the precordial area, in the anterior chest wall, in the interscapular space and radiate to the shoulders, body, abdomen, lumbar and inguinal areas, lower extremities. The pain is frequently accompanied by shock, collapse, dyspnea, vomiting, deorientation, and excitation. The pain is constant, is not relieved by narcotics administration; fatal outcome is frequent. The pathogenesis of the pain is not clear. This is associated with tissue ischemia resulting from disturbances in atrial blood inflow, with dilation of the exterior layer of the aorta.

Short pains in the precordial area on physical exertion may occur in congenital heart defects with overload of the right ventricle. These pains frequently disappear at rest, relieved by nitroglycerin and are associated with transitory ischemia of the hypertrophic right ventricle.

Piercing penetrating constant pains in the area of the heart are frequently caused by myocarditis, cardiomyopathy with considerable heart enlargement. These pains are attributed to a sudden extension of the pericardium with acute dilation of the heart.

The pain in dry pericarditis develops only in simultaneous affection of the parietal layer of the pericardium and the neighboring phrenic or costal pleura. This pain may be acute, sharp, sometimes dull, constant or intermittent, it increases on coughing, swallowing, changes of the position, it may be felt under the breastbone in the precordial area, left side of the back, and interscapular area depending on pleura affection (in front or behind the pericardium).

In exudative pericarditis the pain is more frequent than in dry one, is attributed to distention of the pericardial sac and increase of intrapericardial pressure. This pain is frequently felt behind the breastbone, radiates to the epigastric area, may be of various intensity ranging from feeling of pressure and weight to a cutting, piercing, tearing pain relieved by a sitting position and bending down.

Angina pain may frequently accompany aortic heart defects and hypertrophy of the left ventricle and is due to reduced filling of the coronary arteries in insufficiency of aortic valves (felt during diastole) or in aortic stenosis (felt during systole). The pain behind the breastbone and in the precordial area occurs in mitral stenosis resulting from reduction of the cardiac output and marked hypertrophy of the right ventricle.

Palpitation (palpitatio, cardiopalmus) is sensation of heartbeat, which may be observed not only at accelerated but also in normal as well as decelerated heart activity.

Tachycardia and palpitation are different phenomena. Increased heart rate is not felt by healthy persons. Sensation of heartbeat usually accompanies accelerated and increased heart contractions, this is frequently observed in persons with increased nervous sensitivity on pronounced physical load, quick walking upstairs, drinking large amounts of black coffee, alcohol, smoking. These are the cases of physiological palpitation. It is observed in asthenic constitution, in flatulence, aerophobia, phrenic hernia, vegetovascular dystonia.

Palpitation is a frequent sign of aortic valve incompetence, mitral stenosis, hyperthyrosis, Itsenko-Cushing disease, hypertension, anemia, fever.

Palpitation accompanies different disorders of the rhythm. Solitary beats at various intervals, sometimes one, two, three, and more beats, accompanied by feeling of sinking heart, arrest, lack of air are characteristic for preliminary heart contractions, i.e. extrasystolic arrhythmia, which is often is not a consequence of anorganic heart disease but occurs in regulation disorders. It is said that the more unpleasant is the feeling of extrasystole, the more probable is their functional origin.

Short or long attacks of palpitation can result from paroxysmal tachycardia or atrial flatter. Paroxysmal supraventricular or ventricular tachycardia occur with frequency >150 beats per minute in an attack-like manner with a distinct onset and end, regular heart activity; they last form several minutes to hours and even days. The attacks appear at various intervals.

The rhythm of paroxysms of atrial flatter is irregular, the diagnosis is easily made by objective study during the attack. During the attack-free period the patient can be asked to illustrate the heart activity tapping on a hard surface with a finger. It should be emphasized that diagnostic significance of the complaint on palpitation is decreased by the frequency of this sensation in healthy persons and in those without cardiac diseases as well as the fact how often the patient does not have unpleasant sensations even in marked disorders of the rhythm (constant ciliary arrhythmia, frequent extrasystole caused by organic heart diseases) as well as powerful beats of the enlarged heart causing thrill of the whole anterior chest wall.

Dyspnea (dyspnoe) difficult breathing accompanied by sensation of lack of air, increased respiratory effort, which are evident to the patient.

Normal breathing is not accompanied by difficulties and unpleasant sensations even when it is accelerated due to exercise or emotions (tachypnoe, hyperpnoe).

Edema can accompany hyperpnea, hypopnea, tachypnea, bradypnea, hyperventilation, hypoventilation. It can be only a subjective symptom, is discovered by history taking, and thus depends on the ability of the patient to describe his/her sensations. The sensation of dyspnea is associated with participation of the auxiliary muscles in the act of respiration, which appears after minute respiratory volume exceeds a definite critical value, dyspnea threshold. A healthy person feels dyspnea when minute respiratory volume increases 4 times, >30 % of the vital capacity of the lungs participate in respiration (in normal conditions this is 10—20 %). Anaerobic metabolism prevails in the muscles and excess of lactic acid results in metabolic acidosis, the cause of centrogenic dyspnea.

The amount of physical load at which the patient lacks air depends on the age, sex, body weight, physical development, the state of the nervous system, etc. Dyspnea of effort may disappear in healthy persons with gradual increase of physical load, reduction of the body weight.

The patients with heart diseases reach dyspnea threshold on significantly lower physical exercise then healthy ones. Cardiac dyspnea appears at blood congestion in the pulmonary veins resulting in reduction of the pulmonary tissue elasticity. Various factors may lead to increase in the blood volume in the pulmonary system: disturbances in blood outflow from the pulmonary veins to the left atrium and left ventricle at narrowed left atrioventricular orifice, reduction of diastolic relaxation of the left ventricle in constrictive pericarditis as well as in decreased blood ejection from the left ventricle at decreased contractile function of the left ventricle.

On physical exercise congestion in the pulmonary system increases due to increased inflow of the blood to the right ventricle and increase in the blood amount in the pulmonary vessels. Therefore, dyspnea is more pronounced in left-ventricle insufficiency and decreases in right-ventricle insufficiency.

At first, dyspnea appears only on physical effort, later at rest when the patient is lying (orthopnea) and, at last it does not disappear even in an upright position. In right ventricle incompetence, dyspnea occurs only at marked congestion in the general system and is associated with disturbances of blood gas composition (chronic cor pulmonale).

In primary sclerosis of the pulmonary artery (Ayerza's disease) and stenosis of the pulmonary artery dyspnea appears on minimal physical effort and is associated with reflex stimulation of the respiratory center. Sudden severe dyspnea is characteristic for pulmonary embolism, accompanied by development of the so-called cor pulmonale and right ventricle incompetence with blood congestion in the general system. Dyspnea in this case is associated with reflex influence.

Thus, the following types of dyspnea associated with heart disease can be distinguished:

1) exercise dyspnea;
2) orthopnea, decubital dyspnea (in decubitus);
3) constant dyspnea;
4) fit-like dyspnea:
a) short attacks of nocturnal dyspnea;
b) cardiac asthma;
c) acute pulmonary edema.

Dyspnea in cardiac diseases is frequently mixed with difficulty in inspiration and expiration as well as involvement of the auxiliary respiratory muscles in the act of respiration.

Exercise dyspnea is always a sign of blood congestion in the lungs. It is accompanied by accelerated superficial breathing (superficial polypnea).

Later dyspnea appears when the patient takes a lying position (orthopnea), increases when the patient is lying on the left side (trepopnea). To relieve the dyspnea the patients have to put several pillows and even sleep in a sitting position.

Orthopnea is sometimes the first sign of blood congestion in the lungs and can develop in other pathological states, reducing vital capacity of the lungs (pneumonia, pleurisy, pneumothorax).

The degree of congestion in the lungs, at which dyspnea occurs, depends on individual sensitivity. Orthopnea decreases in advanced cardiac failure with marked brain hypoxia and development of right ventricle incompetence.

Constant dyspnea in progressing cardiac failure increases with the minimal movement of the patient, both in the morning and at night. In patients with constant dyspnea and considerable edema the prognosis is better that in cases when edema is absent.

Fit-like dyspnea occurs mainly at night without an evident stimulus, it increases rapidly and is accompanied by anxiety and fear of death. This sign is called paroxysmal dyspnea, spontaneous dyspnea, and cardiac asthma. The term "cardiac asthma" is frequently used to define this type of dyspnea as this is associated with cardiac diseases and manifests by respiration disturbances and physical findings characteristic for bronchial asthma (pronounced expiration dyspnea, wheezing, distance rales, mucous sputum discharge, sometimes slightly stained with blood, the patient takes a characteristic forced position (sitting, resting the hands on the chair, knees, bed).

The duration and severity of asthma attacks vary. The attack may last from 30 minutes to several hours, in mild cases it manifests by increased respiratory rate accompanied by feeling of anxiety, lack of air, in severe cases asphyxia with bronchospastic syndrome, marked cyanosis, cold perspiration, tachycardia, seething boiling sounds going from the patient's throat (tracheal rales), the cough becomes worse and is accompanied by foamy rosy sputum (development of acute pulmonary edema results in arterial pressure drop and may cause death). But in favorable cases asphyxia and dyspnea subside gradually. Weakness and fatigue remain.

Milder attack of cardiac asthma may disappear in an upright position, when the patient sits up, after release of gases, belching, urination. But the attacks of dyspnea and asphyxia can recur during a night, every night, or be solitary, which do not recur at all and the patient does not pay attention to them. That is why thorough and accurate history taking is very important for diagnosis.

As it has been already mentioned, the pathogenesis of dyspnea in cardiac diseases is associated with increased blood volume in the pulmonary veins. Increased pressure in the pulmonary veins and capillaries results in interstitial edema development, elasticity of the lung tissue decreases as well as its extension during inspiration and contraction during expiration. The function of the external respiration is also affected by considerable dilation of the capillaries, reduction of the lumen of the pulmonary alveoli. Ventilation deficiency is compensated by accelerated breathing (Hering-Breuer reflex, increased activity of the respiratory center with the impulses from the peripheral receptors of the pulmonary tissue). The degree of the vital capacity redaction and the edema severity correlate: when vital capacity of the lungs is reduced by 10-30 %, dyspnea of effort develops, but usual activity is tolerated without difficulty; when vital capacity of the lungs is reduced by 30-60% dyspnea develops on minimal physical exercise; when vital capacity of the lungs decreases by >60 %, dyspnea at rest develops.

Overstrain of the respiratory muscles and involvement of the auxiliary muscles are accompanied by unpleasant sensations of pressure in the chest, neck, epigastric area.

In a recumbent position pulmonary congestion increases because the blood from the lower extremities and abdominal organs moves to the organs and vessels of the chest, blood inflow to the right heart increases, pulmonary ventilation becomes worse due to a high position of the diaphragm and reduction of its motility. Decreased dyspnea with the body bent down is associated with increased pressure in the inferior vena cava, which results in blood congestion in the lower portion of the body and decreased blood inflow to the right ventricle.

Cardiac asthma also results from acute increase in the pulmonary congestion accompanied by transient distal bronchial obstruction.

An important pathogenetic mechanism of the attacks of cardiac asthma is a sudden increase in blood in-flow to the right heart at insufficient contractile function of the left ventricle. At night at complete relaxation of the muscles venous congestion occurring in the muscles is more pronounced in-patients with blood circulation disorders. In a wakeful state and at restless sleep, frequent muscular motions provide even in-flow of blood to the right atrium. In patients with heart diseases accidental movement during sleep after a long physical rest causes increase of blood in-flow to the right heart and increase of the blood ejection to the pulmonary circulation; the left ventricle cannot respond by the corresponding increase of systolic blood volume. Besides, at night in patients with hidden fluid retention the fluid moves from the tissues to the vessels, which increases the volume of circulating blood and venous congestion in the lungs due to left ventricle weakness.

In sudden increase of congestion in the lungs, the respiratory center is irritated by reflex thus producing dyspnea.

On waking up powerful respiratory movements promote venous blood in-flow to the right heart, which causes further increase of pulmonary congestion and stimulation of the respiratory center. This vicious circle can be destroyed by morphine administration, which decreases sensitivity of the respiratory center. Congestion phenomena in the pulmonary vessels are accompanied by vagus reflexes causing bronchospasm.

Increased pressure in the pulmonary capillaries is the cause of serous fluid transudation to the pulmonary alveoli, exit of erythrocytes from the capillaries; movement of air froths the fluid, which enters the bronchi and trachea and can cause asphyxia.

Increase of pressure in the pulmonary capillaries is the most important factor of pulmonary edema development: elevation of the pressure in the pulmonary capillaries above the level of oncotic pressure of the blood proteins (25 mm Hg) causes fluid transudation from the pulmonary capillaries to the alveoli. But the correlation between the pressure value in the pulmonary capillaries and pulmonary edema does not always exist. A role is played by other factors, which increase vascular wall permeability, sensitivity of the respiratory center.

Blood congestion in the pulmonary system causes cough. It frequently occurs in decubitus and awakes the patient. But it may develop at daytime, on physical load or after meals, occurs in attacks lasting from several minutes to more than an hour, frequently appears with dyspnea, accompanying the attack of cardiac asthma or pulmonary edema. The cough is dry at first then it is accompanied by some amount of mucous viscous discharge sometimes with traces of blood.

The cough may be a sign of trachea or bronchi compression with aortic arch aneurysm, in this case it becomes barking, loud, coarse. Similar cough appears at compression of the left recurrent nerve and bronchi with dilated pulmonary artery, enlarged left atrium in mitral stenosis. Hemoptysis can be a sign of lung infarction resulting from embolism of the branches of the pulmonary artery or their thrombosis. The amount of blood coughed up can be large (haemoptoe) and blood can be mixed to the sputum (haemoptysis). Sometimes the cause of hemoptysis is rupture of varicose bronchial veins and collaterals between the bronchial and pulmonary veins in the submucous membrane of the bronchi.

Discharge of the foamy pink sometimes bloody sputum is typical for pulmonary edema.

Hemoptysis can be associated with atherosclerosis of the intrapulmonary branches of the pulmonary artery or bronchial arteries, with primary atherosclerosis of the pulmonary artery, pulmonary hypertension in congenital heart defects.

Syncope can be a frequent sign of aortic stenosis. Loss of consciousness may develop on physical exercise or at rest, may last for 15-30 minutes, be accompanied by epileptiform convolutions and not infrequently may have fetal outcome. Syncope in aortic stenosis results from cerebral ischemia due to the fact that with this defect the left ventricle cannot quickly promote minute blood volume at increased requirements in blood supply.

Syncope sometimes develops in marked mitral stenosis, chiefly at paroxysm of atrial flatter and presence of a free thrombus in the left atrium, which, when the body has been bent down and then taken a vertical position, closes the mitral opening causing arrest of blood circulation and sometimes death.

The pathogenesis of syncope is similar in tumors in the left atrium. Syncope is less frequent in incompetence of semilunar valves of the aorta, aortic aneurysm, exudative pericarditis, cor pulmonale.

Syncope may develop during angina attack and acute myocardial infarction (syncope anginosa). Syncope is caused by ventricular flatter or heart arrest. In myocardial infarction, brain ischemia and syncope can be caused by vaso-vagal reflex due to intensive pain, sudden reduction of the arterial pressure (Betzold-larish reflex), paroxysmal tachycardia, ventricular flatter, complete atrioventricular blockade, rupture of the heart wall or interventricular septum, papillary muscles, severe heart failure. Syncope may be the only clinical sign of myocardial infarction.

Loss of consciousness with rapid fatal outcome may develop in acute cor pulmonale caused by pulmonary artery embolism.

Syncope and deep loss of consciousness are important signs of Morgagni-Adams-Stokes syndrome resulting from temporary blockade of cerebral circulation at arrest of the ventricles activity, extreme deceleration or acceleration of heart contractions, weakness of the sinus node.

Loss of consciousness can be preceded by feeling of feeling of sand in the eyes, sharp weakness, dizziness, and torpor occurring at sharp movements or without an evident cause. The patient has the time to sit or lie down, lean upon the surrounding objects. The attack frequently develops without precursors, the syncope lasts from several seconds to several minutes. The loss of consciousness is accompanied by widened pupils, absence of reaction to light, convolutions, biting of the tongue, involuntary urination, defecation, foam from the mouth.

A frequent cause of a sudden deceleration of the heartbeat is complete atrioventricular blockade, especially the so-called preautomatic pause, interval between the arrest in conduction of impulses from the atria and work of ventricular pace-makers. The intensity of the attack is directly proportional to the duration of this pause. If brain anoxia lasts for 6-8 seconds, darkness in the eyes and dizziness develop, if 8—10 seconds, it results in syncope, in 15 seconds, spasms and paralysis, 30 seconds, Cheyne-Stokes breathing, 4-5 minutes, death.

Loss of consciousness is typical for patients with the frequency of ventricular automatism <10 per minute. Sudden pronounced sinus bradycardia can also be accompanied by syncope.

High incidence of ventricle contraction in paroxysmal tachycardia, flatter and fibrillation of ventricles result in less filling of the ventricles with the blood, reduction of the cardiac output, brain ischemia and loss of consciousness, especially in the persons with coronary artery disease, valvular heart defects, cardiomyopathy.

Coarse voice and aphonia may result from compression of the left recurrent nerve with aortic aneurysm, dilated pulmonary artery in mitral stenosis (the left recurrent nerve goes between the left branch of the pulmonary artery and aorta).

Combination of mitral stenosis and paralysis of the recurrent nerves is called Ortner syndrome. Dysphagia can be a sign of esophagus compression with enlarged left atrium in mitral stenosis, aortic aneurysm, congenital right or double aortic arch, with right subclavicular artery as well as in exudative pericarditis.

Constant complaints of the patients with heart diseases are headaches, sleeplessness, dizziness, and rapid fatigue.

Headache in the occipital area appears frequently in the morning in hypertensive patients and those with atherosclerosis of the brain vessels. Headache is a frequent sign of aortic heart defects. This sign suggests disturbances of cerebral circulation. Sleep disorders are frequently caused by blood congestion in the pulmonary system and may appear earlier than the patients faints or feels dyspnea.

Dizziness (vertigo*)* may occur in atherosclerosis of the vessels supplying the brain and vestibular apparatus, blood congestion in the brain vessels as well as be the consequence of vagotonic vascular reactions.

Nosebleed (epistaxis*)* may develop in arterial hypertension, rheumatism, bacterial endocarditis.

Punctate hemorrhages (petechid) and large subcutaneous hemorrhages (ecchymoses) also appear in bacterial endocarditis, chronic heart failure, atherosclerosis.

Thirst is a frequent sign of fluid retention in patients with developing cardiac failure.

Thirst may be accompanied by *edema*. Cardiac edemas appear on the feet and legs, are bilateral, at first they are felt as tight footwear, then the patients notice imprints of laces, socks, footwear on the legs, later they notice edema of the lower legs, dorsal surfaces of the feet and lower legs. The edema appears in the evening at first and subsides by the morning, later it becomes constant and rises cranially, usually up to the umbilicus, but may involve higher regions in advanced stages of cardiac failure.

Development of cardiac edemas is associated with decreased secretion of atrial natriuretic factor, which is accompanied by retention of sodium and water as well as increased hydrostatic capillary pressure. Secondary hyperaldosteronism due to blood congestion in the liver and cardiac cirrhosis and increased production of antidiuretic hypophyseal hormone, ACTH, and glycocorticoids also play a role.

Edema is a frequent sign of other diseases (those of kidneys, vessels, and thyroid gland, tumors) that is why this complaint advocates for thorough history taking.

The heart percussion is the most important objective method of examination of the cardiovascular system allows determining its size, shape as well as the size of the vascular band.

Auscultation of the heart is an objective method of examination, which consists in listening to and evaluation of acoustic phenomena developing when the heart works. Auscultation is the most important objective method of examination of the cardiovascular system, diagnostic hypothesis about the diseases is based on it.

Theme 9. **The Pulse and Arterial Pressure Study.**

Places of pulse investigation. Investigation of arterial and venouse pulse. Investigation of the pulse on the radial artery. Pulse properties: frequency, rhythm, tension, shape, value, causes of the changes of the pulse properties, pulse different, the state of the vascular wall. Pulse deficiency. Dicrotic pulse. Arterial pressure and factors, which determine it. Systolic, diastolic, mean, pulse pressure. Methods of arterial pressure measurement. Technique of the blood pressure measurement. Phases of acoustic phenomena according to Korotkoff. Normal arterial pressure. Diagnostic significance of arterial pressure.

The contents: The vascular system consists of a number of separate tubes connected parallel and serially. A continuous blood flow in the system is due to elastic properties of the aorta and large arteries. During cardiac systole, the kinetic energy is used to stretch and fill the arterial system with the blood. The kinetic energy produced by the heart turns into the energy of elastic tension of the arterial walls. When the systole ends, the extended walls of the arteries tend to collapse and push the blood to the capillaries thus providing blood flow during diastole. The pressure in the aorta is directly proportional to the blood volume pushed out by the heart to the arteries and the peripheral resistance. The resistance of the vessels depends on a number of factors: the length of the vessels, viscosity of the blood in them, the radius of the tubes. Blood viscosity is not constant. The smaller is the vessel diameter, the lower is viscosity of the blood in them. Main resistance to the blood flow develops in the arterioles, containing a thick layer of smooth circular muscles in their walls. According to I.M. Sechenov, the arterioles are the faucets of the cardiovascular system. They play a dual role: they participate in maintenance of the blood pressure necessary for the organism and regulate the local blood flow. In a working organ, the tone of the arterioles decreases, which provides increased blood in-flow. To prevent reduction of the total pressure in the other (not working) organs, the tone of the arterioles increases.

As the blood is ejected by the heart in separate portions, the blood flow in the arteries is pulsating. In the moment of ejection the pulse wave is present in the aorta, the wave of increased pressure and extension of the walls, which propagates from the aorta to the arteries and capillaries where it dies. Periodic, simultaneous with the work of the heart vibrations of the arterial walls are called arterial pulse. Besides, capillary and venous pulses are distinguished. The study of the arterial pressure is most important. The pulse wave can be seen and felt. But the amplitude of the wall motion is small that is why palpation is the main method of arterial pulse study.

The most frequent place to study the pulse is the radial artery as it is located superficially under the skin between the styloid process and the tendon of the inner radial muscle. The topography of the radial artery allows to press the vessel to the bone, which facilitates the study of the pulse. The hand of the patient is held with the physician's right hand in the area of the radioulnar joint, the thumb of the physician should be on the elbow side, the fingers on the radial side. After the artery is felt it is pressed with the point and middle fingers. When the wave passes the artery, the physician feels dilation of the artery, that is the pulse. First, it is necessary to study the properties of the radial artery. The fingers of the physician should glide along the artery in transverse and longitudinal direction. The normal sensation is that of thin, soft, even, elastic, pulsating tube. The artery is soft but elastic. If it is soft but not elastic, this suggests decreased vascular tone, which is present in fever, shock, collapse. If it is hard and elastic, this suggests increased tone of the arterial muscles observed in increased blood pressure. If the artery is hard and not elastic (rigid), this suggests development of connective tissue or calcification of the vessel, a sign of atherosclerosis. This vessel looks curly. In significant atherosclerosis, separate hard regions with calcific deposits can be felt (atherosclerotic beads). In addition to the radial arteries, the properties of the

wall and pulsation can be studies on the temporal, carotid, brachial, femur, popliteal, tibial arteries and the sole of the foot. In narrowed isthmus (coarctation), the volume of the pulse waves decreases considerably on the lower extremities, while on the carotid arteries and those of upper extremities it remains normal or increased. In obliterating arteritis of the large vessels originating from the aorta arch (Takayasu disease, absence of the pulse) pulsation of the carotid, axillary, brachial, radial arteries disappears or decreases. Reduction or absence of pulsation on the plantar arteries is the sign of endoarteritis (in young patients), obliterating atherosclerosis (in the middle or elderly age), thrombosis of the vessels.

Then it is necessary to determine whether the pulses are equal on the both hands. Normally they are equal. If the pulses are unequal, this is called *pulsus differens.* Pulsus differens in observed in anomalies of the radial artery (it goes to the back side of the hand and the usual place is occupied by its branch), in pathological changes: aortic arch aneurysm, mediastinal tumors, narrowing of the left atrioventricular orifice when enlarged left atrium presses the subclavicular artery and the pulse of the left hand, especially in the left decubitus, decreases (Popov-Saveliev sign), when a tumor or enlarged lymph nodes compress the artery, when the lumina of the large vessels are compressed with scars.

After comparison of the pulse on the both hands, it is necessary to study the properties the pulse on one hand. If the pulse is different on the both hands, it is studied on the hand where it is more intensive.

The following properties are to be determined. **Pulse rate**, the number of pulse beats per minute. In healthy individuals pulse rate is 60-80 beats per minute. Rapid pulse (pulsus frequens, may be due to physiological conditions. In women the pulse rate is 7-8 beats more than in man, the pulse accelerates with physical work, excitement, during digestion, on breathing in, in persons over 60, in some diseases (it increases by 8-10 beats per each degree of the body temperature, in thyrotoxicosis, anemia, acute and chronic diseases of the heart, endocarditis, myocarditis, pericarditis, cardiac failure, after taking some drugs and poisons, such as alcohol, atropine, caffeine, adrenaline). In typhoid fever at fever of 40 degrees the pulse may be 76-80 per minute (relative bradycardia), in tuberculous meningitis due to excitation of the vagus nerve under the influence of increased intracranial pressure bradycardia may be observed.

Pulse rate disorders: a) rapid; b) rare. In healthy subjects a rare pulse (pulsus rarus, <60) is not frequent, chiefly observed in sleep. Pulse deceleration is observed in the following pathological conditions: complete atrioventricular blockage, stenosis of the aorta orifice, cachexia, hanger, jaundice, cerebral hemorrhage, brain tumors, fracture of the skull, myxedema.

Rhythm of the pulse, the beats follow with equal intervals and are equal, i.e. regular pulse (pulsus regularis). In disturbances of the heart function, this regularity changes, it becomes arrhythmical, irregular, an irregular pulse (pulsus irregularis). Three types of arrhythmias are observed: extrasystole (extraordinary heart contractions), the interval between this and the following contraction can be unusually long (compensatory pause), ciliary arrhythmia (disordered pulse waves are palpated), paroxysmal tachycardia (very frequent pulsation which is difficult to count, appearing and disappearing suddenly).

If the pulse is arrhythmical, it is necessary to determine if the number of the pulse waves corresponds to the number of the heart contractions. In frequent arrhythmical contractions of the heart, separate systoles of the left ventricle may be so weak that the blood is not ejected to the aorta, or the amount of the blood is so small that the pulse wave does not reach the periphery. The difference between the number of the heart contractions and pulse waves per one minute is termed *pulse deficiency*, the pulse is called *a deficiency pulse* (pulsus deficiens). The more is the deficiency, the more unfavorable is its effect on the blood supply of the organs and tissues. Pulse rhythm disorders: a) extrasystole; b) bigeminal pulse; c) ciliary arrhythmia.

Pulse tension is the pressure of the blood exercised on the wall of the artery. It is determined by the force, which should be exercised to compress the artery completely in order to arrest the blood flow in it. This property of the pulse gives the information about the state of the vascular system and the arterial pressure. In healthy persons the pulse tension is satisfactory. In a tense pulse, the force of compression to arrest the pulse wave should be great *(pulsus durus),* this is a sign of hypertension of various origin or arterial sclerosis. Reduction of tension, *soft pulse (pulsus mollis)* suggests decreased arterial pressure (reduction of the heart contractile function, shock, collapse, blood loss).

Pulse filling is the amount of blood in the vessel. This property is most difficult to determine, namely according to the maximum and minimum volume of the vessel (how the diameter of the vessel changes in the period of dilation and collapse). To do this, proximal fingers on the radial artery should press the vessel gradually, the distal finger determines its maximum filling. In healthy persons the pulse is satisfactory. In reduction of the volume of circulating blood (blood loss, shock, collapse), disturbances of contractile function of the heart, the pulse filling decreases, *pulsus vacuus,* in increased volume of the circulating blood, blood filling increases, *full (strong) pulse* (pulsus plenus). Pulse filling and tension give similar information.

Pulse value is a collective concept, uniting such properties as filling and tension. It depends of the degree of the artery widening during systole and its collapse during diastole. In healthy persons the pulse is sufficient. With the increase of the stroke blood volume, great fluctuations of the arterial pressure as well as decreased tone of the arterial wall, the value increases, *pulsus magnus;* in insufficiency of the aortic valve, thyrotoxicosis, fever, the tone of the aorta wall decreases. Reduction of the stroke volume, increased tone of the arterial wall reduces the number of pulse waves, *small pulse (pulsus parvus).* This is observed in stenosis of the aorta opening, mitral stenosis, tachycardia, heart failure; in shock, massive blood loss the pulse is poorly felt, *thready pulse (pulsus filiformis).*

The shape or rate of the pulse is the rate of dilation and the following contraction of the artery. This property depends of the rate of the pressure changes in the arterial system during systole and diastole. In aortic valve incompetence, *an abrupt pulse (pulsus celer)* or *a bouncing pulse (pulsus silens)* as well as *pulsus altus:* the stroke blood volume and systolic blood pressure are increased, during diastole the pressure drops quickly as the blood returns from the aorta to the left ventricle can be present.

Abrupt pulse is also observed in thyrotoxicosis, nervous excitement.

Slow pulse (pulsus tardus) is opposite to an *abrupt pulse.* This is associated with slow increase of the blood pressure in the arterial system and its small fluctuations during a cardiac cycle. This is observed in stenosis of the aorta opening. Due to reduction of the pulse waves it is not only slow but also small (pulsus parvus). Pulse shape disorders: a) bouncing pulsus magnus; c) slow small pulse.

Dicrotic pulse (pulsus dicroticus) is a second additional wave after reduction of a normal pulse wave. In healthy subjects it is not palpated but registered on sphygmogram. A dicrotic pulse is present in reduced tone of the peripheral arteries (fever, infections, severe pneumonia).

An alternating pulse (pulsus alterans) is alterations of large and small pulse waves when the pulse is rhythmical (severe affection of the myocardium, i.e. myocarditis, cardiomyopathy.

A paradoxical pulse (pulsus paradoxus) is reduction of the pulse waves during breathing in (in adhesion of the pericardium layers due to compression of the large veins and reduction of the heart filling during expiration.

The study of the arterial pulse allows to evaluate the contractile function of the heart, the amount of the ejected blood, the properties of the arterial wall, arterial pressure, in some cases it suggests the affections of the aorta valves, increase of the body temperature, the state of the nervous system.

In addition to arterial, capillary and venous pulses are also distinguished. *Capillary pulse* is observed in insufficiency of the aortic valve, sometimes in thyrotoxic goiter. It is determined in the following way: it necessary to press the tip of the nail until a white spit appears in the center. It will widen and narrow with each pulse beat. Similarly hyperemic spot produced by rubbing the skin (e.g. on the forehead) may widen and narrow. The pulse is termed capillary, which is not accurate, it depends on the pulse fluctuations or arteriole blood filling.

In physiological conditions, a slight *pulsation of the jugular veins* can be observed. A normal vein pulse is called negative. In pathological conditions examination of the jugular veins demonstrates a wave of synchronous with the carotid artery pulsation. This is a so-called positive venous pulse. This pulse is present in insufficiency of the tricuspid valve. Overfilled hypertrophic right atrium does not manage the load and dilates. Congestion is transmitted to the veins, which loose the tone of the circulatory bundle. Elevation of the venous wave on the jugular veins coincides with contractions of the right ventricle. Besides tricuspid valve insufficiency, positive venous pulse is observed in ciliary arrhythmia, atnoventricular (nodular) rhythm, when the atria and ventricles contract simultaneously. To answer the question if the pulsation of the jugular veins is caused by a reverse blood flow, it is necessary to press the

jugular vein preliminary emptying it. If the jugular veins quickly are filled with the blood in spite of the compression, the blood comes from the right ventricle.

Arterial pressure is the stress exerted by the blood on the walls of the vessels (lateral pressure) and the column of the blood from the site of the pressure to periphery (end pressure). The values of the arterial pressure depend on many factors: the force with which the left ventricle contracts, the amount of the blood entering the circulation per a unit of time, tension of the walls of the arteries, the amount of the blood out-flow through the capillaries, blood viscosity, heart rate, extracardiac factors (the state of the nervous system).

Constant arterial pressure is due to two factors: blood in-flow to the arterial system (pumping function of the heart) and the tone of the arterioles. The values of the arterial pressure drop from the center to the periphery, especially at the level of arterioles.

During left ventricle systole, blood pressure in the arteries is highest, this is systolic or maximal blood pressure. During diastole it is the lowest, minimum or diastolic pressure. The difference between maximum and minimum pressure is termed pulse pressure.

Maximum or systolic pressure consists of stroke and lateral pressure. Stroke pressure reflects kinetic energy of the moving blood. This is the pressure created by an obstacle before the blood flow. Lateral (true systolic) pressure is that exerted to the lateral wall of the artery during ventricular systole. The value of systolic pressure is determined by the following factors: the force of left ventricle contraction, the volume of the ejected blood, the state of the arteries. Minimum or diastolic pressure is the smallest value of the blood pressure in the artery by the end of diastole. Its value depends on the arteriole tone. The higher is precapillary system resistance, the high is minimum pressure. Blood in-flow to the peripheral arteries is influenced by the state of elasticity of the large vessels. Elastic vessels carry the blood more easily and smooth the pulses with systolic and diastolic pressure. The vessels with decreased elasticity (rigid) create more pronounced elevations of systolic and more pronounced drops of diastolic pressure. The blood viscosity also influences the out-flow. In increased viscosity blood resistance increases. The pulse rate also influences the blood pressure. The slower is the pulse, the higher is systolic pressure and the lower is diastolic pressure. And vice versa, the quicker is the pulse, lower is maximum pressure and the higher is minimal pressure. This can be explained by the following: during prolonged diastoles the blood has more time for outflow, than in shortened systoles. Besides, during prolonged diastoles the ventricles fill better, their contractile energy restores more perfectly resulting in increased amount of blood ejected into the aorta.

Systolic pressure is unstable, it ranges during a day influenced by the environmental factors, the cortex of the brain, subcortical centers. Diastolic pressure is relatively stable and to a larger degree reflects the state of intravascular pressure. Constant movement of the blood from the arterial system to the venous one is influenced by the mean pressure. Mean or dynamic pressure is a constant value, a physiological constant, it is less influenced by the factors, which change systolic and diastolic pressure.

Investigation technique. Arterial pressure can be measured with a direct and indirect methods. Direct measurement is performed with artery puncture. This is mainly used in cardiosurgery. Three methods are used for indirect measurement: auscultation, palpation, oscillographic. The most practical is an auscultation method proposed by N. S. Korotkoff in 1905. It allows measuring both systolic and diastolic pressure. The measurement is done using a sphygmomanometer (mercury, Riva-Rocci apparatus, spring, electronic). The pressure is usually measured on the brachial artery. The cuff is wrapped and fastened around the bare upper arm of the patient. The cuff should be tightened to allow only one finger between it and the patient's skin. The edge of the cuff with the rubber tube should face downward. The zero level of the apparatus, the artery and the patient's heart should be at the same level. The patient's arm should rest comfortably with the palm upright and the muscles relaxed. Than the valve of the apparatus is turned off and the cuff is inflated with air until the pressure in it exceeds the 30 mm the level when pulsation of the brachial and radial artery is not felt. After that the valve is turned on and the air is allowed to escape slowly from the cuff. At the moment the pressure in the cuff becomes a little lower than systolic pressure, the first slight pulsations of the radial artery will appear (palpation method of measurement systolic blood pressure). Diastolic pressure cannot be determined using this method.

The most frequently used is Korotkoffs method, which allows determining both minimal and maximal pressure. With this method, when the pressure in the cuff is a little lower than systolic pressure, sounds simultaneous with the heartbeat are heard with a phonendoscope over the brachial artery. When the sound appears, the values noticed correspond to systolic pressure. N. S. Korotkoff described four phases of sound phenomena, which are heard during blood pressure measurement over the vessel. Phase 1 is appearance of the sounds over the artery, that is first portions of the blood entering the vessel under the place of narrowing causing vibrations in the relaxed wall of the empty vessel. While the pressure in the cuff is dropping, more blood can pass the narrow area thus turbulent blood movement appears above the narrowing, the sound resembles murmurs (phase 2). Gradually, more blood enters the vessel increasing the vibration of its wall and the sound increases (phase 3, low sounds). When the pressure in the cuff equals diastolic pressure, the obstacle to the blood flow disappears, the vibrations decrease sharply. This moment is characterized by evident weakening and disappearing sounds (phase 4) and corresponds to diastolic pressure.

Oscillography allows to register both systolic, mean, diastolic pressure as a curve, oscillogram. The pressure fluctuations are registered on a paper band with an arterial oscillograph. Systolic pressure is pictured as low-amplitude waves. The highest oscillations correspond to the dynamic or mean pressure. The last wave corresponds to the level of diastolic pressure. Normal oscillogram: Mx - maximal pressure, Mn - minimal pressure, My - mean pressure.

Arterial pressure in healthy people is subjected to physiological changes depending on physical load, emotions, body position, meals, and other factors. It is lower in the morning, on an empty stomach, at rest, in conditions of basal metabolism. This pressure is called basal.

Arterial pressure is measured in millimeters mercury. Normal systolic pressure ranges within 100-140 mm Hg (13-18 kPa), diastolic pressure 60-90 mm Hg (8-11 kPa). Z.M. Volynsky and co-authors suggested a mathematical correlation between arterial pressure and the age. According to them, "ideal" value of the blood pressure is calculated using the formula: systolic pressure = 102+(0.6 x age), diastolic pressure = 63 + (0.4 x age).

Increased arterial pressure is called hypertension. This can be primary and secondary. Secondary hypertension is a sign of some disease, e.g. renal hypertension (glomerulonephritis, pyelonephritis, amyloidosis), endocrine arterial hypertension (hyperthyroidism, tumor of the adrenal cortex, Conn's syndrome, pheochromocytoma, tumor of the anterior lobe of the hypophysis), hemodynamically mediated arterial hypertension (aorta atherosclerosis, aorta coarctation, insufficiency of the aortic valve), CNS affection. In primary hypertension (essential hypertension) increase of the arterial pressure is the main sign of the disease. Essential hypertension is responsible for 80% of arterial hypertension, the rest 20% are secondary arterial hypertension, of them 14% are caused by the diseases of the kidneys or their vessels.

Main signs of increased systolic arterial pressure are a) increased cardiac output and increased blood inflow to the arterial system during ventricular systole; b) reduction of aortic wall elasticity (increase in density, rigidity), e.g. in atherosclerosis. The main cause of increased diastolic arterial pressure is increased tone (spasm) of the arteries resulting in increased peripheral resistance.

In *systolic hypertension,* increased maximum arterial pressure is noted in atherosclerosis of ascending portion of the arch or thoracic aorta, in young persons when hypertension develops.

High diastolic and slightly increased systolic pressure are due to reduction of contractile heart function in arterial hypertension.

Arterial hypotension is decreased arterial pressure observed in shock, collapse, profuse hemorrhage, myocardial infarction, intoxication (atropine, chloralhydrate), adrenal insufficiency, acute infections.

Vascular insufficiency (collapse, syncope) results from rapid drop in the vascular tone. As the result, large portions or the blood are deposited in the vessels of the abdominal cavity, the volume of the circulating blood, blood in-flow to the hear and cardiac output decrease, systolic and diastolic arterial pressure drop. This is accompanied by temporary brain hypoxia, blood supply to other organs decreases. The patient suddenly develops weakness, tinnitus, darkness in the eyes, nausea, palpitation, dyspnea, sometimes loss of consciousness, paleness of the skin, cold perspiration, thready pulse. The cause of acute

vascular insufficiency may be blood loss, influence of toxins, reflexes (fear, pain) causing disturbances of central and vegetative nervous system.

The changes of the blood pressure on other than brachial artery (especially lower extremities) are important for diagnosis of some diseases. In physiological conditions systolic pressure in the arteries of the legs is 10-20 mm Hg higher than that of the arms. For example, in aorta coarctation (congenital narrowing of the aorta isthmus), marked reduction of the pressure in the femur arteries and increased in the brachial is observed. To measure the pressure in the femur artery, the cuff is applied on the thigh of the patient in a prone position, the popliteal artery is heard in the popliteal fossae. Sometimes it is necessary to measure the pressure on the both arms and legs.

Theme 10. **Visual Examination and Palpation of the Precardial Area. Determination of the Borders of Relative and Absolute Heart Dullness. Determination of the Vascular Band.**

Visual examination and palpation of the precardial area. Visibl pulsation in the precardial area. Cardiac humb. Technique of apical thrust investigation, mechanism of formation, location. Causes of displacement. Height, width, resistance of the apical thrust. Negative apical thrust. Cardiac thrust. Epigastric pulsation: pulsation of the abdominal aorta and the liver. Pulsation of the aneurysms of ascending aorta and the arch of aorta. Pulsation of the dilation of the trunk of the pulmonary artery. “Cat’s purring”. Mechanism of formation. Determination of the borders of relative and absolute heart dullness (right, upper, left). Determination of the vascular band.

The contents: The precordial area is the area of the anterior surface of the chest to which the heart and large vessels are projected. The borders of the precordial area are as follows: to the right - right medioclavicular line, top - clavicles, to the left - anterior axillary line, bottom - right and left costal arches.

During the examination the studied area should be well lightened.

At norm, the vessels of the skin on the chest are not seen or are slightly noticeable. Sometimes widened small veins of the skin forming a band of several centimeters (so-called Stokes's band) can be seen. These changes do not have any diagnostic significance and can be observed in healthy individuals. Branching curling veins in the area of the manubrium of sternum can be a sign of aortic aneurysm, mediastinal tumor, insufficiency of the right heart, adhesive pericarditis. Dilated pulsating arteries are characteristic for aorta coarctation.

Stable diffuse outpouching of the precordial area, or cardiac hump (*gibbus cardiacus*), suggests heart enlargement, which develops in childhood or early youth in rheumatics or congenital heart defect. Temporal diffuse outpouching of the precordial area, dilation and filling of the intercostal spaces occur in exudative pericarditis in persons with an elastic chest. Outpouching in the epigastric area can develop in marked accumulation of fluid in the pericardial sac (Auenbrugger's sign).

Limited pulsating outpouching of the sternal region and neighboring intercostal spaces can be seen in aneurysms of ascending aorta (on the right), its root (on the left), arch (manubrium of sternum). Palpation of this outpouching reveals systolic pulsation seen on visual examination.

Pulsation to the left of the sternum in the 2nd and 3rd intercostal spaces can develop due to hypertrophy of the right ventricle, dilation of the trunk of the pulmonary artery.

Pulsation in the cardiac area associated with left ventricle systole is called apical thrust, or apical beat (ictus apicalis). This is a conventional term as apical thrust does not correspond to the physical position of the heart apex. The pulsation involves the areas of the left ventricle located cranially and medially the anatomical heart apex. Apical thrust does not coincide with the left border of the heart and is located more internally. The mechanism of apical thrust is rather complicated.

The heart muscle does not have a constant immobile point of support necessary for contraction. The only more or less fixed areas of the myocardium are the places of origin of the large vessels and the heart apex. Immobility of the apex is due to a complicated interlace of the muscular fibers forming a node (vortex cordis) and due to anterior turning of the apex during which it presses to the chest and becomes a fixed point of support allowing to start blood ejection. With the onset of diastole the apex goes back without loosing the touch with the chest.

The possibility to see and feel the apical thrust depends on many factors: age, position of the body, the position of the diaphragm, the shape of the chest, the state of the lung tissue, the type of cardiac activity. In adults the apical thrust is not seen and not palpated (thick chest, covered with a rib). The apical thrust can be felt when the patients stands, lies on the left side, after holding the breath at the height of forced expiration, after physical load.

Technique of apical thrust investigation. The palmar surface of the right hand is placed on the precordial area between the presternal and anterior axillary lines and 3rd - 4th ribs.

First, location of the thrust is determined with the whole palm, then it is placed perpendicular to the ribs; the end phalanges of the fingers are used to determine the characteristics of the apical thrust: location, area, height, resistance. The lowest left point of the outpouching, which is felt by a non-lateral surface of the finger, is considered the location of the apical thrust. The place where the apical thrust is felt surely is decisive for determining the size of the thrust. This place is located a little cranially and medially the anatomical apex of the heart inner the left heart border.

In adult males and in the majority of women apical thrust can be palpated in a supine position in the 5th intercostal space 1-1.5 cm inner the medioclavicular line. In left lateral decubitus the apical thrust moves 2 cm to the left. If, on turning to the left side, the location the thrust remains unchanged, this may indicate adhesions between the layers of the pericardium.

In deep breathing in the apical thrust moves downwards and inwards and can disappear as it is covered by extended lungs. On deep breathing out the apical thrust moves upward to the left.

In a vertical position of the heart (long chest, asthenic constitution) the apical thrust is low: behind the 4th rib 2 cm inward the medioclavicular line.

In hypersthenics and in children the apical thrust moves to the 4th intercostal space and can be palpated near the medioclavicular line. In elderly individuals apical thrust is frequently palpated in the 6th intercostal space in the result of a low position of the diaphragm.

Apical thrust can displace to the left and upward in flatulence, pregnancy, ascites, enlarged liver and spleen. Apical thrust moves to the left behind the medioclavicular line in right hydrothorax, pneumothorax, cirrhosis, atelectasis of the left lung, pleuropericardial adhesions on the left side.

Apical thrust displaces to the right when the volume of the right lung decreases or the pressure in the left pleural cavity increases. The location of the apical thrust depends on the size of the heart.

At widening of the left ventricle, apical thrust displaces downwards to the left to the anterior axillary line and the 6th intercostal space. In hypertrophy of the left ventricle without dilation, location of the apical thrust does not change.

When the right ventricle dilates, apical thrust can move to the left. The left ventricle is displaced to the right and the heart apex is formed by the right ventricle.

The area of the apical thrust is the area of the outpouching. Normally this is about 2 cm^2. An enlarged area is called generalized thrust, a decreased one limited.

Generalized apical thrust is observed on breathing out, in tumors of the posterior mediastinum, in shrinkage of the lungs, a high position of the diaphragm, when the anterior borders of the lungs depart from the anterior surface of the heart resulting in increase of the area. Generalized apical thrust up to diffuse systolic thrill of the cardiac area to the left of the sternum (cardiac thrust) can be seen in children, asthenics, at excitation, on physical overstrain, in fever, hyperthyroidism.

When the above causes are absent, enlargement of the thrust area is the consequence of the heart enlargement (not only the left ventricle) as enlargement of any portion of the heart increases the intrathoracic pressure, the borders of the lungs collapse opening a large surface of the heart.

Limited apical thrust is observed when the heart is displaced backward from the chest in emphysema, a low position of the diaphragm, exudative pericarditis, hydro- and pneumothorax, in hypersthenics, thick chest. It is possible that apical thrust will not be determined.

Height of the apical thrust is the amplitude of movement of the outpouching, that is the distance to which this area displaces forward. If this distance increases, apical thrust is termed high, if decreases - low. High apical thrust is observed in the same conditions as the generalized thrust. The causes are the same. High apical thrust is also called "elevating".

Resistance (force) of apical thrust is resistance, which is produced by the cardiac muscle to the pressure of the palpating fingers. Resistance is determined by density, thickness of the cardiac muscle, and force with which the heart hits the chest. Resistant apical thrust is a sign of hypertrophy of the left ventricle muscle. If the left ventricle is dilated, hypertrophic apical thrust becomes resistant and high. The palpating hand feels a dense, elastic thick dome, the so-called dome-shaped apical thrust is frequently present in mitral valve incompetence. Thrust of the left ventricle can be that forceful that causes shaking of the whole cardiac region.

Hypertrophy and dilation of the right ventricle can cause systolic pulsation to the left of the sternum between the 3rd and 5th ribs (*cardiac thrust*). In this case the apical thrust is not palpable, sometimes it is produced by the hypertrophic right ventricle. In marked enlargement of the both ventricles, shaking of the whole cardiac region can occur.

In hypertrophy and dilation of the right ventricle as well as in drooping (low position) of the diaphragm, pulsation may develop in the epigastric area.

Epigastric pulsation can also be produced by the abdominal aorta or liver.

Pulsation of the right ventricle is seen directly under the xiphoid process, increases on breathing in, drawing in is more prominent that sticking out.

Pulsation of the abdominal aorta is noted in slim individuals with thin weak abdominal muscles, in enteroptosis, dilation of the aorta, aortic aneurysm. Pulsation of the abdominal aorta is located in the middle of the line connecting the xiphoid process with the umbilicus, is directed forward. The aorta is frequently palpable, its pulsation decreases in breathing in. The signs of aneurysm are limited pulsating formation in the abdominal cavity above the umbilicus or in the left hypochondrium.

Pulsation of the liver may be true and "transmitted". True pulsation consists of increase and reduction of the liver volume in case of tricuspid valve incompetence due to systolic regurgitation of blood from the right ventricle to the right atrium, which causes increased pressure in the vena cava and hepatic veins during systole and enlargement of the liver in all directions. "Transmitted" pulsation is caused by the beat of the right ventricle or abdominal aorta, the liver moves forward.

Other pulsations of the cardiac region. Aneurysm of the heart can cause pulsation in a limited area cranially the heart apex in the 4th intercostal space.

In marked mitral insufficiency with dilation of the left atrium pulsation along the medioclavicular line between the 3rd and 4th ribs may be observed (the left atrium displaces to the right half of the chest).

In tricuspid valve insufficiency along the whole lower portion of the chest on the right to the 4th rib there is forceful systolic pulsation transmitted from the liver pulsating synchronously with the systole of the right ventricle.

Aortic pulsation can occur in the 2nd intercostal space to the right at the border of the sternum in shrinkage of the border of the right lung, in aneurysm of the ascending aorta or its arch, in insufficiency of the aortic valve. In aneurysm pulsation is expansive, is felt on the bottom of the fossa, is directed upward (the patient lifts the shoulders and bends the head forward).

Pulsation of the pulmonary artery is felt in the 2nd intercostal space to the left of the sternum in shrinkage of the border of the left lung and dilation of the pulmonary artery (mitral stenosis). The beat is felt during diastole.

Atrial pulsation is sometimes seen and felt in the area of the heart base. This pulsation is associated with diastolic filling of the atria coinciding with the systole of ventricles and is observed at shrinkage of the lungs, in considerable dilation of the atria in insufficiency of the mitral valve or tricuspid valve (pulsation is caused by blood regurgitation).

Systolic drawing in of the cardiac region can be revealed in the area of the heart apex as well as in the whole cardiac region. Frequently this drawing in is observed in asthenics in the area of the apical thrust, if the thrust is covered by the rib and reduction of the heart volume during systole may be associated with sucking effect near the areas where the heart adjoins the cardiac region. In these cases it is necessary to investigate the apical thrust in left and right decubitus. If the drawing in remains in place, this is the sign of pathology and can be attributed to marked enlargement of the right ventricle, sudden reduction of the heart volume (tricuspid valve insufficiency), adhesive pericarditis.

The heart immured in dense cicatrix adhesions cannot turn forward during systole and contracting it will involve the chest (negative apical thrust).

During diastole diastolic outpouching of the chest is seen (“diastolic rebound”). Similar phenomena can be observed in insufficiency of the tricuspid valve.

An important visual and palpation diastolic phenomenon is shaking of the chest during diastolic filling of the ventricles at reduced tone of the myocardium (cardiac failure).

Palpation of the precordial area can elicit a special phenomenon, shaking, vibration of the chest resembling the feeling which can be felt when you put your hand on the chest of a cat (“cat’s purring”). This phenomenon occurs when the blood passes a narrowed opening forming turbulent currents causing vibration of the heart muscle transmitted to the cardiac region.

“Cat’s purring” is better felt with a palm put to the cardiac region.

In mitral stenosis, “cat’s purring” is reveled in the area of the heart apex, sometimes in left decubitus, during diastole before beginning of systole (presystolic) or during the whole diastole. Sometimes such presystolic shaking occurs in aortic valve incompetence (the flow of blood regurgitation lifts the anterior cusp of the mitral valve narrowing the mitral opening and causes oscillations).

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“Cat’s purring” can develop in considerable enlargement of the left ventricle (relative narrowing of the left venous orifice). In the right 2nd intercostal space and in the area of the manubrium of sternum systolic thrill is formed in aortic stenosis, it is revealed during forced expiration and after physical load. In defects of the interventricular septum, systolic “cat’s purring” is felt in the middle of the sternum near its left edge. In the 2nd and 3rd intercostal spaces to the left of the sternum, systolic thrill is a sign of pulmonary artery stenosis, continuous systolodiastolic thrill in this area can be felt in patent arterial duct.

In dry pericarditis, pericardial rub can be felt as scratching sensations on the left edge of the sternum and on the heart base. They increase at pressure on the cardiac region, increase in a sitting position and on bending forward, can be continuous or consist of separate beats, do not coincide with separate phases of the cycle and disappear in exudate accumulation or adhesion formation.

The heart percussion allows determining its size, shape as well as the size of the vascular band. The heart is spherical and only a small area of its anterior surface adjoins the chest.

The normal outlines of the heart are presented by the superior vena cava in the 2nd and 3rd intercostal spaces, the right atrium in the 4th intercostal space. Its left border is the left potion of the aortic arch and further the trunk of the pulmonary artery in the 2nd intercostal space. At the level of the 3rd rib

is limited by the auricle of the left atrium, its lower border is presented by a narrow band of the left ventricle at 4-5th ribs.

The heart is a dense air-free organ. Percussion over the heart produces a dull sound. A smaller portion of the anterior surface of the heart adjoins the chest, its larger portion is covered with the borders of the lungs. The area of the heart not covered by the lungs, which produces the dull sound on percussion, is termed absolute heart dullness. It corresponds to the position of the right ventricle, which forms the anterior border of the heart. The portion of the heart covered with the lungs produces a deadened sound termed relative dullness. This is the projection of the anterior heart surface on the chest and corresponds to the true heart borders. To diagnose cardiac diseases topographic percussion is used, its purpose to determine the borders of relative and absolute dullness of the heart. Percussion of the heart can be done both in a sitting and upright position of the patient. It should be remembered that the size of the heart dullness in an upright position is smaller than in a lying position. This is associated with the heart mobility and shifting of the diaphragm with the change in the position.

Before percussion it is necessary to determine the level of the diaphragm as the changes in the position of the diaphragm influence the location of the heart in the chest and the borders of the heart dullness. With this purpose, the location of the lower border of the right lung (normally in the 6th intercostal space) is determined along the right medioclavicular line. Percussion is started from the 1st intercostal space along the intercostal spaces from a clear to dull sound.

The position of the diaphragm corresponds to the lower border of the lungs, that in the 6th intercostal space. After it has been determined, the right, upper and, at last, left borders of relative cardiac dullness should be determined. The lower border of the heart cannot be determined as it borders with the liver producing similar dull sound. To determine **the borders of relative dullness**, percussion with a medium force is used. After determining the position of the diaphragm the plessimeter finger is shifted one intercostal space upper (or two ribs, in the 4th intercostal space) placing it parallel to the right border of the heart. Making taps using medium force, the plessimeter finger is moved along the intercostal space to the heart until deadened sound appears. The right border of the heart is noted along the external border of the piessimeter pointing to the clear sound. *The right border* is formed by the right atrium and is normally in the 4th intercostal space 0.5 cm outer (to the right) the right sternal line or along the right edge of the sternum. To determine the upper border of the heart the plessimeter finger is placed in the first intercostal space 1 cm outer the left sternal line (not on the left medioclavicular line) and is shifted downwards making taps using medium force until a deadened sound appears. The upper border of the heart is noted along the upper (external) border of the plessimeter finger (closer to clear percussion sound). *The upper border* of relative heart dullness is formed by the auricle of the left atrium and the trunk of the pulmonary artery and is normally located in the 3rd intercostal space. The left border of the relative dullness is determined in the same intercostal space, where apical thrust is located, usually in the 5th intercostal space.

Percussion is performed in the 5th intercostal space from the left anterior axillary line towards the sternum. The plessimeter finger is placed parallel the supposed border, the percussion is accomplished from a clear until a deadened sound. *The left border* is marked along the outer border of the plessimeter finger facing the clear sound. Normally, it is formed by the left ventricle and is in the 5th intercostal space 1-2 cm inner the left medioclavicular line and coincides with the apical thrust.

The 4th and 5th intercostal spaces, in which the borders of relative heart dullness are determined, are the uttermost points of the outer outline of he heart. Changes in the heart borders in these spaces are also accompanied by the changes in other spaces. That is why percussion is usually performed in the 4th intercostal space for the right border, in the 5th intercostal space for the left border.

Then **the borders of absolute heart dullness** (the area of the heart which is not covered with the lungs and adjoins the breastbone) are determined. Light percussion is used for this purpose. The percussion of this area produces a dull sound. There are two methods, which allow determining the borders of absolute heart dullness. The first one uses light percussion done from the relative dullness until a dull sound appears. The border of absolute dullness is marked along the outer edge of the plessimeter finger facing the deadened sound. To determine the right border of absolute heart dullness after determining the border of relative dullness in the 4th intercostal space, the plessimeter finger is placed

parallel the sternum and then is moved inward until dull sound appears. The border of the absolute dullness is marked along the outer edge of the finger facing the border of relative dullness. Normally *the right border* of absolute heart dullness is noted along the left edge of the breastbone. Actually the right border of absolute heart dullness is located in the middle of the sternum along the inner border of the right lung. Accurate determining of this border is hindered by the breastbone, which also vibrates on percussion. To determine the upper border of absolute heart dullness, the plessimeter finger is placed in the 3rd intercostal space parallel the ribs, the percussion is performed downward the intercostal spaces until a dull sound appears. *The upper border* is marked along the outer edge of the plessimeter finger pointing upward. This is normally located in the 4th intercostal space. To determine the left border of absolute heart dullness percussion is done along the 5th intercostal space from the border of relative heart dullness until a dull sound appears. *The left borde*r is marked along the outer edge of the plessimeter finger facing the left border of relative heart dullness. This is normally 1-2 cm inner the left border of relative dullness. The lower border of absolute dullness is not determined as it coincides with liver dullness. The whole area of absolute dullness is formed by the anterior surface of the right ventricle.

It is sometimes difficult to differentiate absolute and relative heart dullness if the percussion is performed from the lungs to the heart. In this case the other method is applied: percussion from relative to absolute heart dullness. The plessimeter finger is placed in the center of heart dullness, slight percussion is accomplished from a dull to deadened sound. Transition from a dull sound to a deadened one corresponds to the borders of relative heart dullness. This method requires very light percussion: the plessimeter finger placed on the investigated surface should the flexed in the first interphalangeal joint. Very light taps are done with the right-hand finger on the place of the flexion. The obtained sound is supplemented with a tactile sensation: some rigidity is noted in the area of dullness. Determining the absolute heart dullness is technically easier than relative.

To have a clear picture of the outlines of the absolute and relative heart dullness, percussion should be performed in several intercostal spaces on the left and right. If all the obtained points are joined with a line, the outlines of the dullness will be obtained. To receive more accurate information about the area of relative heart dullness, the transverse section of the relative heart dullness is measured. This is the distance between the uttermost right heart pint (usually in the 4th intercostal space, about 3.5-4 cm) and from the uttermost left point (in the 5th intercostal space, 8-9 cm) to the median body line. The sum of these two values is the large transverse section of relative heart dullness, which normally equals 11.5-13 cm. In women this is 0.5 cm less than in men. The borders of the vascular band are determined in the second intercostal space using silent percussion. First, the plessimeter finger is placed in the 2nd intercostal space on the medioclavicular line parallel the sternum. In the process of percussion it is moved to the sternum until a deadened sound appears. The right border of the vascular bundle is marked on the outer edge of the plessimeter closer to the clear pulmonary sound. After the plessimeter finger is located on the left of the 2nd intercostal space along the medioclavicular line and is moved to the sternum until dullness appears. The left border of the vascular bundle is marked at the outer edge of the plessimeter. The transverse section of the vascular bundle (the distance between the right and left borders) normally equals 5-6 cm. Enlargement of this distance is observed in tumors of the middle mediastinum, which displace the heart to the anterior thoracic wall. Increased dullness in the 2nd right intercostal space is observed in dilation of aneurysm of the ascending aorta (arterial hypertension, atherosclerosis), on the right in dilation of the pulmonary artery (high pressure in the pulmonary artery), or dilation of descending aorta (aortic hypertension, atherosclerosis of the aorta). As the heart is spherical, percussion helps to determine only the changes in the transverse heart section. The changes in the anteroposterior section of the heart cannot be distinguished by percussion. But the changes in the heart sizes develop simultaneously therefore percussion changes in the transverse size allow to suggest the changes in the whole heart. The changes in the heart dullness may depend on three factors: the position of the diaphragm, changes in the lungs and pleura, the size of the heart.

The area of relative or absolute dullness may also change due to physiological factors. Physiological enlargement of absolute heart dullness is observed in deep breathing out, pregnancy, high position of the diaphragm resulting from fat accumulation in the abdominal cavity. In deep breathing in the area of absolute dullness decreased. In asthenics with low position of the diaphragm absolute heart

dullness decreases ("hanging heart"). Physiological increase of the area of relative heart dullness is noted in all cases of elevated diaphragm, a reverse state is observed in drooping of diaphragm. Dilation of absolute heart dullness is noted in shrinkage of anterior heart borders, tumor of the posterior mediastinum. A seeming enlargement of absolute heart dullness is observed in inflammatory consolidation of the anterior border of the lungs, exudative pleurisy as dullness over the lungs merge with heart dullness. Reduction of the area of absolute heart dullness is observed in pneumothorax, accumulation of air in the pericardium (pneumopericardium), pulmonary emphysema, subcutaneous emphysema. In considerable pulmonary emphysema absolute dullness may disappear, relative dullness also decreases, it can be revealed using only loud percussion. Increase of the area of relative heart dullness is observed in widening of the heart or its chambers. Hypertrophy of the myocardium only does not change the percussion size of the relative heart dullness, it enlarges due to dilation of separate hear chambers.

In enlargement of the left ventricle the left border of the relative heart dullness shifts to the left (mitral insufficiency, aortic insufficiency, aortic stenosis, arterial hypertension). In aortic heart defects, arterial hypertension the outline of the heart is termed aortic. The angle (waist of the heart) between the vascular bundle, the auricle of the left atrium and left ventricle is almost right (normally obtuse). The outlines of relative heart dullness resemble a sitting duck or a boot. Enlargement of the right ventricle is accompanied by displacement of the right border of relative heart dullness to the right, later to the left due to displacement of the left ventricle, which is observed in mitral stenosis, congenital heart defects, cor pulmonale (chronic bronchitis, pulmonary emphysema). But in cor pulmonale the shift of the border cannot be determined in the result of pulmonary emphysema. Dilation of the left atrium, if moderate, is not revealed by percussion as it takes place in the posterior regions. Only in considerable dilation its upper border shifts upward due to the auricle of the left atrium. In the area of the waist an outpouching is formed. This outline of the heart is termed mitral as it is usually observed in defects of the bicuspid valve (stenosis and insufficiency). Enlargement of the heart borders in all directions can be present in myocarditis, cardiomyopathy, effusion pericarditis. When fluid accumulates in the pericardial cavity the dullness acquires a typical trapezoid shape, or "roof with a chimney" appearance, the "roof" is formed by the dullness of the exudate, the "chimney" the dullness from the vascular bundle.

Theme 11. Auscultation of the Heart. Normal Heart melody. Changes of the Heart Sound: Doubl and Split Sound, Gallop Rhythm, Mitral Snap.

Rules of the heart auscultation. Basic points of auscultation. Places of valve projection and areas of auscultation. Mechanism of sound formation. Changes of the sound: the increase and decrease of the sound. Doubl and split sound. Pathological sound: gallop rhythm (protodiastolic, systolic, presystolic), mitral snap. Immediate, appliances (stethoscope, phonendoscope) methods of heart auscultation.

The contents: Auscultation of the heart is an objective method of examination, which consists in listening to and evaluation of acoustic phenomena developing when the heart works.

Auscultation is the most important objective method of examination of the cardiovascular system, diagnostic hypothesis about the diseases is based on it.

There are two methods of heart auscultation: direct (immediate) performed when the physician's ear is applied to the precordial area of the patient and mediate, which uses special appliances (stethoscope, phonendoscope). At present, immediate auscultation is used in addition to mediate one when the third heart sound is listened to.

Rigid stethoscopes are no longer used, variable binaural flexible stethoscopes with a funnel-shaped and flat tips (a flat one is provided with a membrane) are available. Low-pitch acoustic vibrations (20-200 Hz) are better heard with a stethoscope, high-pitched sounds (>200 Hz) with a phonendoscope, the membrane of which can filter away low-pitched vibrations (diastolic sound resulting from insufficiency of the aortic valve).

The olives, which are put in the external auditory meatus, must close it tightly, the funnel should be densely pressed to the surface of the precordial area.

Rules of heart auscultation. The chest should be bare, the areas with thick hair on the chest should be moistened with water to reduce the friction of the hair. The room should be warm and silent. The

funnel of the stethoscope is densely pressed to the chest, but is should not compress the tissues. The patient is asked not to breathe deeply. Auscultation is performed at quiet respiration, when the patients does not breathe, at the height of the expiration and inspiration, in a lying and upright position, on the left side, and after dosed physical load.

The physician should have a healthy hearing, be able to concentrate, evaluate the loudness, duration, timber of the sounds, which are produced by the work of the heart. He should know the location of various auscultation phenomena, how they are induced or increased, how they are related to different phases of the cardiac cycle. Auscultation of the heart is performed in definite regions of the precordial area (main auscultation points), their choice is determined by better conduction of the acoustic phenomena related to the functioning of various heart structures: muscular strain, stroke of the blood at the cusps of the valves, stretching of the walls of the large vessels, as well as conversion of laminate blood flow to turbulent. The consequence of listening is determined by the incidence of the diseases of the respective structures: most frequent are the affections of the left ventricle, mitral valve, aortic valve, right ventricle, tricuspid valve and pulmonary artery valve. When necessary, other regions can be listened to, e.g. the place of sound conduction, but this is done after the standard auscultation.

Point 1 - the area of apical thrust, in case it is absent, left border of relative cardiac dullness should be listened to. The acoustic phenomena which occur at the beginning of systole at tension of the muscular structures of the left ventricle, the stroke of the blood in the phase of isometric strain with closed cusps of the mitral valve and their vibration (first sound) are better heard in this point.

Point 2 - 2nd intercostal space to the right near the edge of the breastbone. The acoustic phenomena which occur at the beginning of diastole as a result of the stroke of the blood moving in the direction of the lowest pressure with closed cusps of the aortic valve are heard here (second sound).

Point 3 - 2nd intercostal space on the left edge of the breastbone. The phenomena which are evoked by the stroke of the blood at the closed cusps of the pulmonary artery and their vibrations are hard here (second sound).

Point 4 - lower edge of the sternum in the place of xiphoid process origin. The acoustic phenomena evoked at the beginning of systole at strained muscles of the right ventricle, stroke of the blood moving in the direction of the lowest pressure at the closed cusps of the tricuspid valve and their vibrations.

Point 5 (Botkin-Erb) is listened to in two positions of the stethoscope: in the middle of the breastbone at the level of the 3rd costal cartilages and in the 3rd intercostal space at the left edge of the breastbone. These positions arc associated with the projection of the aortic valve on the precordial area. It is the place where acoustic phenomena developing at the beginning of diastole, especially when the cusps of the aortic valves are not closed (diastolic sound of blood regurgitation), are well heard.

The projection of the cusps of the pulmonary artery coincides with point 3 (2nd intercostal space at the edge of the sternum).

The projection of the mitral and tricuspid valves are at a distance from the place of auscultation: the projection of the mitral valve is located in the place of origin of the 3rd costal cartilage from the left edge of the sternum; the projection of the tricuspid valve coincides with the middle of the direct line joining the places of origin of the 3rd and 5th costal cartilages from the sternum.

Thus, the projections of the heart valves are localized nearby, which hampers evaluation of the heart sounds.

The farthest from the place of listening is the projection of the mitral valve, besides point 1 changes its location in the precordial area depending on the location of the apical thrust and the left border of relative heart dullness. In the place of projection the mitral valve is located deep under the right ventricle and the tricuspid valve. At the beginning of systole the acoustic phenomena associated with the mitral valve are conducted along the strained muscle of the left ventricle to the heart apex which in this period turns forward and densely adjoins the precordial area separated by a thin layer of the lung tissue.

Similarly, the acoustic phenomena associated with the tricuspid valve are conducted along the strained muscle of the right ventricle to point 4.

The acoustic phenomena associated with the aortic valve propagate along the blood flow to point 2.

In healthy adults the heart melody consists of two sounds (first and second), this is divided by two pauses (systolic and diastolic).

Heart sounds are short acoustic phenomena resulting from tissue vibration, which occur at vibrations of strained muscles of the ventricles and valve cusps. The first sound (systolic) is heard at the beginning of systole, the second heart sound (diastolic) is heard at the beginning of diastole.

In children and teen-agers accessory sounds (third and fourth) are sometimes heard.

Mechanism of sound formation. **The first sound** consists of three components: muscular, valvular, vascular. The *muscular component* is formed during asynchronous contraction (contraction of papillary muscles and the neighboring muscles of the ventricles) and at the beginning of isometric strain as a result of vibration of these muscular structures. Some authors believe that vibrations of atrial muscles at their contraction also take part in formation of this component and distinguish the atrial component of the first sound. But distinguishing the atrial component of the first sound seems groundless, as atrial systole is related to the end of ventricular diastole (so-called pre-systole) and ends by the beginning of asynchronous contraction: the first sound is a systolic phenomenon. The muscular component of the first sound is presented by low-frequency and law-amplitude vibrations.

Valvular component of the first sound is formed at stroke of the blood at closed cusps of the atrioventricular valves in the phase of isometric strain. The blood moves in the direction of the lowest pressure of the ventricular walls to the atria and strikes the cusps of the atrioventricular valves, making them to do vibration movements.

The vibrations, which compose the ventricular component of the first sound, have the highest frequency and amplitude when compared with the muscular component.

The vascular component of the first sound is associated with vibrations of the walls of the aorta and pulmonary artery at the beginning of blood ejection from the ventricles. This component consists of low-frequency and low-amplitude vibrations.

The second sound also consists of two components. The first component, *cusp*, occurs when the blood strikes the closed cusps of the aorta and pulmonary artery while moving in the direction of the lowest pressure (to the ventricles). This component consists of high-frequency and high-amplitude vibrations. The second component, *vascular*, is formed when the walls of the vessels vibrate during the period of reverse movement from the vascular valves. The vascular component is presented by low-frequency and low-amplitude vibrations.

A physiological *third sound* is heard in children, teen-agers, and young individuals (above 30) with relatively thin elastic chest. This accessory sound occurs at the end of the first third of the diastole (the period of fast filling of the ventricles with blood), when the ventricles turn slightly forward and strike the precordial area. The third physiological sound, low-amplitude, low-frequency, appears immediately after the patient has changed an upright position to the lying one, on the left side, during breathing out, it increases after physical load, emotions, excitement, and other factors causing tachycardia. The heart apex is the point where the third sound is best heard.

The first and second sounds are heard over the whole precordial area, but the cardiac melody differs in different auscultation points: the first sound is better heard in points 1 and 4, the second one in points 2, 3, 5. There are other features, which allow to distinguish the first and second heart sounds: the first sound is longer, the frequency of vibrations is lower. The first sound is heard at the beginning of a short pause (systole), it coincides with the apical thrust and pulsation of the carotid arteries. But these features do not always help to distinguish the first and second heart sounds. Thus, accelerated and arrhythmic activity of the heart is accompanied by shortened diastole, its duration becomes equal to that of systole. The loudness of sounds in all points becomes similar and the cardiac melody resembles the sounds of the fetal heart, so called embryocardia, or pendulum-like rhythm, when the first and second sounds cannot be differentiated.

When blood pressure drops considerably, the second heart sound can disappear, the cardiac melody consists only of the first sound, and as hypotension is accompanied by tachycardia, the cardiac melody resembles that in rabbits and is termed kiniclocardia or rabbit’s rhythm.

Difficulties in differentiation of the sounds occur when their loudness changes or the sounds split with appearance of accessory sounds.

In this cases graphic registration of the cardiac melody (phonocardiography) is necessary. This allows to register low- and high-frequency acoustic phenomena, which are not heard with the ear. For

instance, in children and young people the fourth heart sound can be heard in addition to the third one. It results from acceleration of the blood flow through the atrioventricular opening during atrial systole.

Simultaneous registration of phonocardiogram, electrocardiogram, phlebogram, carotid pulse and intracardiac pressure (polycardiography) has facilitated specifying the mechanisms of various auscultation phenomena.

Changes of cardiac melody. The changes of cardiac melody can be associated with the changes in the sound loudness, timber, and number.

The loudness of the first sound is mainly determined by the cusp component and depends on the following: the position of the cusps of the atrioventricular valves immediately before the onset of ventricular systole, their anatomical structure, kinetic energy of blood movement during isometric tension.

Kinetic energy is determined using a well-known formula $E=mv^2/2$, that is directly proportional to the mass of the blood in the ventricles, the mass of the muscular fibers and squared speed of the blood movement and the rate of transition of the muscular fiber from relaxed to strained state.

Thus, the greater is the mass of the blood in the ventricles and the mass of working muscular fibers, the weaker is the first heart sound and vice versa, the less blood is in the ventricles and the mass of the muscular fibers, the louder is the first heart sound.

An important factor influencing the loudness of the first sound is location of the atrioventricular valves before ventricular systole: the deeper are opened cusps of the valves, the higher is the amplitude in the phase of isometric strain and the louder is the first sound.

The location of atrioventricular valves is determined by the blood volume in the ventricles: if more blood enters the ventricles during diastole, the valves rise to the surface, their cusps get closer, the amplitude in the period of isometric strain decreases and the first sound weakens. Thus, weakening of the first sound occurs in hypertrophy of the left ventricle (arterial hypertension, aortic heart defects, hypertrophic cardiomyopathy), weakening of the first sound depends on the degree of the hypertrophy.

Weakening of the first sound is observed when isometric strain is absent when the cusps of the atrioventricular valves do not close due to shrinkage, deformity, widening of the atrioventricular opening in dilation of the ventricles), the blood at the beginning of systole passes through the cleft between the cusps to the atria, which decreases the force of beat, determining the cusp component of the first sound. The degree of weakening of the first heart sound reflects the degree of the defect.

In mitral valve incompetence, both absolute (heart defect) and relative (dilation of the left ventricle in prolonged arterial hypertension, dilation cardiomyopathy), the first sound weakens over the apex.

In organic tricuspid valve incompetence (heart defect), relative tricuspid valve incompetence at dilation of the right ventricle (mitral stenosis, cor pulmonale, congenital defects), the first heart sound is weak in point 4.

Increase of the first heart sound has the highest diagnostical importance in narrowing of the left and right atrioventricular orifice and is explained by insufficient filling with blood of the left ventricle during diastole.

In mitral and tricuspid stenosis, the fused cusps of the atrioventricular valves form a kind of membrane, which sags deeply during diastole to the ventricular cavity due to high pressure in the atria and low in the ventricles. In the period of isomeric tension, the blood moves at a high rate in the direction of the atria as its amount in the ventricular cavities is lower than in healthy subjects, kinetic energy of the stroke increases and the first sound becomes more forceful. The closed valves move along longer distance due to insufficiently filled ventricles, sag in a dome-like manner in the cavity, and vibrate with high frequency. The first sound acquires a special timber, resembling a flopping sail, and is called Hopping.

A flopping first sound suggests that the valve has preserved some elasticity. In calcification of the valve, insufficient mobility, rigidity of the cusps, arrhythmia this change of the timber may disappear.

Increase of the first sound is heard in other conditions, accompanied by decreased filling of the ventricles with blood (extrasystole, hyperthyrosis, fever).

Loudness of the second sound depends mainly on the valve component and is determined by the kinetic energy of the blood moving in the direction of the valves at the beginning of diastole under the influence of difference in pressure in the large vessels and the respective valves. Besides, elasticity of the

vascular wall, the valves of the aorta, and the pulmonary artery play a role: the less elastic are the vessels receiving the blood, the lower is the degree of extension, and the higher is the pressure in the respective vessel, the louder is the second sound. The second heart sound is heard in points 2, 3, 5.

In points 2 and 5 the loudness of the second sound is influenced by the difference of pressure in the aorta and the left ventricle and elasticity of the aortic walls.

In point 3 the loudness of the second sound is determined by the difference in pressure in the pulmonary artery and the right ventricle and elasticity of the pulmonary artery walls.

In healthy subjects the loudness of the second sound in points 2 and 3 is similar, as the influence of higher pressure in the aorta is balanced by the fact that the pulmonary artery is closer to the precordial area. In children, teen-agers, and young individuals the forward turn of the heart ventricle is more prominent than in adults, the aorta is deeper, the second sound over the pulmonary artery is louder than that over the aorta. In elderly, the second sound over the aorta can increase due to rotation of the heart with the left ventricle forward. If, in an adult, the loudness of the second sound increases in points 2 and 3, when compared with the symmetrical point, this phenomenon is called accent of the second heart sound. This appears at increased arterial pressure in the systemic circulation, in atherosclerosis of the aorta, syphilitic aortitis, rigidity, or calcification of the semilunar valve.

With marked changes of the aortic wall and its valve, the second sound gets a special timber, a bell shade (clangor or bell second sound).

Accent of the second sound over the pulmonary artery is the sign of increased blood pressure in the pulmonary system resulting from mitral heart defects, especially mitral stenosis, insufficiency of contractile function of the left ventricle, chronic diseases of the lungs (emphysema, chronic obstructive bronchitis), in patent arterial duct, idiopathic sclerosis of the pulmonary artery (Ayerza's disease).

If the second sound is increased in points 2 and 3, it is necessary to consider over which point the second sound is accentuated. It is recommended to move the stethoscope outwards to the left and to the right at equal distance. If the second sound is still heard on one side and disappeared on the other, the accentuated is the sound over the valve where it is heard.

Weakening of the second sound is usually caused by stenosis of the aorta orifice (point 2), stenosis of the pulmonary artery orifice (point 3), insufficiency of the respective semilunar valves. In narrowing of the vascular openings less blood is pumped to the vessels during systole, which is accompanied by reduction of the force of stroke on the closed cusps of the valves at the beginning of diastole and results in weakening of the second sound.

In insufficiency of the valves the cusps do not close, the blood goes back (regurgitates) from the vessels to the ventricles, the force of the stroke and the amplitude of vibration of the valves decrease, the second sound weakens or disappears. Insufficiency of the valves of the pulmonary artery is chiefly observed in considerable dilation of the cavity of the left ventricle (mitral stenosis).

Stenosis of the opening of the pulmonary artery is a congenital heart defect.

Relative narrowing of the aorta orifice occurs in marked hypertrophy and dilation of the left ventricle.

Intensification in the both sounds is frequently associated with extracardiac factors (thin flat chest, shrunken lungs, infiltrations of the lung borders, high position of the diaphragm) as well as shortening of diastole and decreased filling of the ventricles with blood at increased heart rhythm (tachycardia at physical exercise, emotions, fever, anemia, hyperthyroidism.

Weakening of the both sounds can be associated with extracardiac causes: thick outpouching chest, pulmonary emphysema, left hydrothorax, low position of the diaphragm. As a rule, the both sounds weaken and are poorly heard (surditas cordis — deafness of the heart) in affection of the cardiac muscle (myocarditis, myocardial infarction, cardiosclerosis, cardiomyopathy), in acute and chronic heart failure, sharp drop of the blood pressure (collapse), hydropericarditis, fibrinous pericarditis.

Changes in the number of heart sounds. This phenomenon can occur at either splitting of the heart sounds or at appearing accessory sounds (extrasounds).

Split and dual heart sounds are produced by nonsimultaneous contraction and relaxation of the ventricles and can be ether physiological or pathological.

At splitting, two consequent sounds are heard without a pause; dual sounds are divided by a pause.

Dual and split heart sounds thus vary in the time interval between the both parts of the sound. Splitting of the first sound can be heard in all healthy individuals medially the heart apex in the 5th intercostal space to the left of the sternum, near the xiphoid process, in the 4th intercostal space near the parasternal line. This phenomenon is frequently heard by the end of breathing in and at the beginning of breathing out, in standing position, at physical exercise. Physiological splitting of the first sound is associated with the fact that the mitral valve closes earlier than the tricuspid valve.

Dual first sound is heard over a large area with its maximum between the heart apex and the breastbone. It does not depend on external factors and is associated with hypertrophy of the left ventricle, blockade of the pedicles of the bundle of His, chiefly the right one, ventricular extrasystole, antesystole (WPW syndrome), in tachysystolic form of atrial flatter.

Split and dual second sound is observed in healthy individuals and is heard in the second and third intercostal spaces to the left of the sternum in a supine position at deep breathing in. This phenomenon disappears with holding the breath on expiration. Delay in the closure of the valves of the pulmonary artery at the height of the inspiration, which is the cause of split and dual second sound, is associated with greater in-flow of the blood to the right ventricle during inspiration due to increase of negative intrathoracic pressure in the dilating chest. The blood is held in the dilated vessels of the lungs, the amount of the blood entering the left atrium and later the left ventricle decreases, the systole ends earlier than during breathing out.

Emptying of the right ventricle during inspiration is decelerated due to hold-up of the blood in the dilated vessels of the lungs. The pulmonary component of the second sound delays.

Unstable physiological splitting of the second sound results from the influence of two opposite factors: on inspiration simultaneously with prolongation of the right ventricle systole, its diastole shortens due to reduction in the pressure in the pulmonary system. On expiration simultaneously with shortening of the right ventricle systole, increased pressure in the pulmonary system facilitates its prolongation. The duration of the interval between the aortic and pulmonary components of the second sound depends on a number of factors: nervous system influence, changes of pressure in various departments of the heart and vessels, depth of respiration, elasticity of the chest and pulmonary vessels.

Pathological dual second sound in the point of listening to the pulmonary artery is frequently heard in mitral stenosis: narrowing of the left atrioventricular opening results in less blood in the ventricle, that is why its systole ends and diastole begins earlier than usually. Due to blood congestion in the pulmonary system the right ventricle systole ends and diastole begins later.

The pulmonary component of the second sound delays and is accentuated. This dual sound is not associated with the phases of respiration, it is constant.

Dual second sound is frequently observed in defects of interatrial septum due to constant enlargement of diastolic filling of the right ventricle not depending on the phases of respiration.

In pulmonary artery stenosis systolic phase of ejection from the right ventricle elongates, the closure of the valves delays. The pulmonary component of the second sound is weakened.

Arterial hypertension in the general and pulmonary system is not accompanied by dual second sound as it does not cause considerable prolongation of ventricular ejection periods but increases the duration of the phase of isometric tension of the ventricles.

In stenosis of the aortic orifice pronounced delay in the aortic valve closure produces a dual second sound, its aortic component follows the pulmonary one and is weak.

Mitral valve incompetence with marked regurgitation of the blood to the left atrium and defect of the interventricular septum with great volume of the blood pumped to the left ventricle, shortening of systole of the left ventricle cause early closure of the aortic valve and dual second sound, which can be masked by a loud systolic murmur.

Split and dual second sound is observed in disorders of the intraventricular conductivity.

Thus, blockade of the right pedicle of the bundle of His increases insimultaneous work of the heart ventricles and is accompanied by splitting of the second sound, which increases on breathing in and decreases on breathing out. Association of the dual sound and the phase of respiration disappears when left ventricle insufficiency develops.

In blockade of the left pedicel of the bundle of His physiological asynchrony of the ventricles can balance the affected conductivity. Marked delay of the closure of the valve of the aorta in blockade of the left pedicle of the bundle of His can cause marked dual second sound, the sequence of the closure of the semilunar valves is reverse when compared with the normal conditions and the dual sounds become paradoxical (decrease at breathing in and appears or decrease at breathing out).

Quail's rhythm. This is a three-part rhythm resembling dual second sound, is a pathognomonic sign of mitral stenosis.

Quail's rhythm results from appearance in the early diastole an accessory sound associated with anatomical changes of the mitral valve. This short abrupt, snapping or knocking sound follows the second sound like a loud echo, frequently more intensive that the second sound and is called opening snap. This term is not accurate, as it does not reflect the mechanism of appearance of the accessory sounds in mitral stenosis. The research done during surgery on the mitral valve have shown that opening snap appears at tension or sagging of the mitral membrane when it protrudes to the cavity of the left ventricle at the onset of diastole. In healthy individuals the mitral valve opens silently, its cusps are freely pushed aside to the walls of the ventricle with the blood, they do not strain or vibrate. Fused dense cusps of the mitral valve in heart defects cannot open freely, before opening the valve sharply protrudes into the cavity of the left ventricle with a specific snapping sound. This accessory sound is better heard in the 4th and 5th intercostal spaces between the left edge of the sternum and the heart apex, sometimes at the left edge of the sternum in these spaces, in the axillar and even under the angle of the left scapula and above the projection of the mitral valve, and increases on breathing out. The degree of mitral stenosis is in reverse correlation with the interval between the snap and the second sound. This interval allows to judge about the pressure in the left ventricle, because its increase facilitates faster opening of the valve. The accessory mitral sound is heard in 4/5 of the cases of mitral stenosis. Its appearance is prevented by an advanced deformity, rigidity of the valve with shortening of the tendinous fibers, considerable insufficiency of the mitral and aortic valves, high blood pressure in the pulmonary system.

This phenomenon is especially important in those cases of mitral stenosis when a diastolic murmur is not heard and diastolic thrill is not determined in the heart apex.

Gallop rhythm is a three-part rhythm, in which the sounds are separated by approximately equal pauses and repeat regularly resembling the sound of a galloping horse. This rhythm is heard with the heart rate of 100 beats per minute, when the rate is <70 or >120 beats per minute this rhythm disappears.

Gallop rhythm results from formation of accessory diastolic sounds: at the end of diastole - presystolic fourth sound, in the beginning - protodiastolic third sound.

Gallop sounds occur as a result of vibration of the ventricular walls when the blood quickly fills it in at decreased myocardial tone due to the lesion. At the beginning of diastole this vibration develops as a result of quick filling of the ventricles with the blood under the influence of pressure difference in the atria and ventricles. At the end of ventricular diastole blood pumping accelerates due to atrial systole, which ejects the blood to the ventricles. When the atrial systole is absent (ciliary arrhythmia, atrial arrest) presystolic gallop sound cannot appear.

It is frequently difficult to differentiate presystolic and protodiastolic gallop rhythm, especially in tachycardia or elongation of the interval between the contraction of the ventricles and atria, when the contraction of the atria coincides with the protodiastolic period of quick filling in of the ventricles. This rhythm is called summary.

Left ventricle gallop, heard in the area of the heart apex or somewhat cranially and medially, and gallop of the right ventricle, heard over the lower portion of the sternum in the region of the xiphoid process and to the left, can also be distinguished. More frequent is presystolic left-ventricle gallop, which is heard, felt, and even seen medially and cranially the heart apex, approximately in the 4th interspace near the left medioclavicular line, especially in the left decubitus, after slight exercise, sometimes it is heard over the whole precordial area. It sometimes seems that gallop sound merge with the first sound resembling split first sound.

Protodiastolic gallop is heard over the heart apex or in the region between the apex and the sternum, better in a lying than in a sitting position, more distinctly on the left side and during breathing out. It

disappears on pressing the precordial area with the stethoscope. The accessory third sound can mimic dual second sound.

In patients with atrial fibrillation only protodiastolic gallop may be heard, in atrial flutter contractions of the atria may promote blood flow to the ventricles, which is sufficient enough to cause vibrations of their walls.

Protodiastolic gallop is a more serious prognostic sign than presystolic.

It is frequently difficult to distinguish gallop rhythm. It should be remembered that gallop rhythm occurs in persons with myocardium affection and signs of cardiac failure: dyspnea, edema, attacks of cardiac asthma, heart enlargement, tachycardia, arterial hypertension.

Physiological third and fourth sounds develop mainly in children and young persons with normal heart borders, with unchanged first and second sounds without the signs of cardiac failure. Physiological split and dual sounds are not constant phenomena, they depend on the position of the patient and phases of respiration, better heard in the upright position and are not accompanied by tactile sensations.

Dual second sound in mitral stenosis is heard over the pulmonary artery, the second sound is increased.

The sound of the mitral valve opening is characterized by a clear timber, sonority, abruptness, it is closer to the second sound than a pathological sound. The first sound is increased, squelching.

But it should be noted that the most reliable way of distinguishing gallop rhythm is constant training of auscultation skills, attentive thorough listening to the patient.

Neither description of this phenomenon can characterize the whole complex of auditory, tactile, visual sensations occurring in gallop rhythm.

Any gallop rhythm suggests cardiac failure.

Left-ventricle gallop rhythm is observed in coronary artery disease, frequently in myocardial infarction, stable arterial hypertension, aortic valve incompetence, cardiomyopathy, acute myocarditis, and severe anemia.

Right-ventricle gallop rhythm develops in chronic and acute cor pulmonale, congenital heart defects. Other signs of right-ventricle insufficiency are also present: enlarged liver, edema, relative incompetence of the tricuspid valve.

Differentiation of gallop rhythm as to the ventricle in which it develops or its place in the cardiac cycle is not decisive as this rhythm always suggests a serious disturbance of the contractile function of the myocardium, this is "a call for help" (V.P. Obraztsov) and signals the necessity of the respective treatment. The prognosis in gallop rhythm is always serious, but sometimes gallop rhythm disappears at rest or after an adequate treatment and appears only on exertion. It is known that gallop rhythm can disappear shortly before the death in progressing cardiac failure.

Three-part rhythm may be associated with an accessory 5th sound during the systole, so-called systolic click. It is better heard over the heart apex, sometimes over the whole precordial area and can be close to the first sound (protosystolic click), in the middle of diastole (mesodiastolic click) and closer to the second sound (telesystolic click). The cause of this sound is prolapse of the mitral valve: sagging of a cusp to the cavity of the left atrium. Mitral valve prolapse can be associated with the changes in the length of the tendinous chords, the structure and function of the papillary muscles. The fifth sound decreases on breathing in and in the upright position.

The accessory protodiastolic sound can develop in constrictive pericarditis due to vibration of the calcified myocardium within the period of quick filling of the ventricles. Pericardium sound is loud, abrupt, clicking, heard over the whole precordial area, in the supraclavicular area, jugular fossa, it is better hard over the heart apex and on the left of the xiphoid process.

An accessory diastolic sound appears in significant reduction of diastolic extension of the cardiac muscle lacking elasticity (elastofibrosis, amyloidosis). The developing vibrations are called myocardium sound.

In healthy asthenics with a funnel-shaped or rachitic chest a special crisping sound accompanying systolic and sometimes diastolic or both sounds is sometimes heard. This is termed sternal crisp. It is heard at the left edge of the lower third of the sternum, near the xiphoid process. This phenomenon is better heard when the patient bends forward. In a lying position it disappears.

Sternal crisp is attributed to the movement of junctions of the costal cartilages and the sternum and the xiphoid process when the heart turns forward during systole.

Pericardial splash is a special sound resembling gurgling fluid heard simultaneously with the heart activity. It develops in hydropneumopericardium: the contracting heart shakes the fluid in the pericardial sac containing some gas.

In left pneumothorax a special knocking sound can be heard during systole, it results of collision of the heart with the left cupola of the diaphragm over the extended stomach and large intestine or chest wall. This sound is especially distinct in left decubitus.

Theme 12. **Auscultation of the Heart. Heart Murmur.**

Mechanism of formation and classification of the heart murmur (extracardiac, intracardiac, organic, functional, systolic, diastolic, murmur of regurgitation, ejection, filling). Rules of the heart auscultation. Determination of the place of heart auscultation in the patient. Elicit the presence of murmur. Determination of the relation to the cardiac cycle. Determination of the place of optimum auscultation. Determination of the loudness, timber, character of the murmur. Determination of the murmur radiation. Kukoverov's sign, Udinzev's sign. Discrimination of the organic and functional murmur. Extracardiac murmur. Pericardial friction rub. Pleuropericardial murmur. Cardiopulmonal murmur. Venous murmur in the jugular vein. Doubl sound and murmur on the femoral artery. Method, causes, mechanism of formation.

The contents: Heart murmurs are prolonged acoustic phenomena produced by the working heart. They are divided into two groups: extracardiac and intracardiac.

Extracardiac murmurs are pericardial friction rub and cardiopulmonary murmurs. *Intracardiac murmurs* develop inside the heart and large vessels and can be explained by the following mechanisms:

1. Laminar blood flow turns into turbulent.
2. Formation of turbulent movements in the blood.
3. Forceful stream of the blood produced by pressure. Turning of laminate flow into turbulent directly depends on the blood flow velocity, blood viscosity and the lumina of the heart cavities and the vessels. Critical acceleration of the blood flow may occur on physical and emotional overstrain, fever, hyperthyroidism, narrowing of the openings between the heart cavities, vessels, atriovenous anastomoses. Turbulent blood flow appears when the blood viscosity is low (anemia). In sudden dilation or narrowing of a limited portion of the blood vessel the blood flow produces a sticking action on the surrounding tissues. In this case the vibration of the walls of the heart and vessels depends on the blood flow velocity, the ratio of the transverse sections of the heart cavities to that of the vessels, the tissue elasticity and is heard as a murmur.

Vibration of the walls of the vessels and the heart also causes a stroke of the blood flow in aortic and mitral valve incompetence.

Narrowing of the blood flow itself without a sudden dilation of the channel behind the place of the narrowing does not produce a murmur. In quick blood flow the murmur is louder, in slow this is weaker. Thus, a murmur can be absent both in slight and considerable narrowings. To produce murmurs a definite velocity of the blood flow should correspond to each degree of narrowing. Murmurs are not heard in a healthy heart, as the blood passes the natural narrowings (from the wide atrium through the atrioventricular opening to the wide ventricle) gradually at a normal rate. But turbulent flow producing murmurs can occur even in a healthy heart. Depending on the cause of the murmur, the latter are divided into organic (valvular), functional and organ-functional (muscular). A valvular murmur develop due to considerable anatomical changes in the hart valves, muscular ones due to disturbances of the valve function without anatomical changes (heart cavity dilation, affection of the papillary muscles). This classification does not include the murmur occurring due to congenital defects of the heart and large vessels.

Functional murmurs are observed in 20-70 % of children and young individuals (under 20). Functional murmurs are not constant, they are changeable, depend on the position of the body and the phase of respiration. They may develop on physical overstrain and disappear at rest, they increase on deep

breathing, most distinctly by the end expiration. This is almost always a systolic murmur over the pulmonary artery and the heart apex, as a rule it is low, short, blowing high-pitched.

A cardiopulmonary murmur occurs in the lungs, but is caused by heart contraction and regulated by the cardiac rhythm, that is why it seems to be cardiac. This murmur is attributed to compression of the lungs with the heart during diastolic dilation, the air is pushed from the lungs. It may also be due to aspiration of the air to the areas of the lungs neighboring with the heart during systole or pushing out the air from the lungs as a result of the heart beat during systole. A cardiopulmonary murmur develops during systole, sometimes in diastole, it is short and resemble peep or a whipping sound. On pressing with the stethoscope and holding the breath it disappears. It is frequently heard over the heart apex and the sternum in the 3rd and 4th intercostal spaces and in the area of the pulmonary artery.

Superficial short scratching sounds associated with the friction of the normal layers of the pericardium at increased pressure at them are sometimes heard on increased cardiac activity along the left edge of the sternum during systole and diastole.

An organic (valvular) and organ-functional murmur occurs when the following factors are present:

1) Changes in the lumen of the heart cavities or disturbances of the ratio of the lumen of two neighboring portions of the heart to that of the large vessels.

2) The respective blood flow velocity.

Narrowing of the channel of the blood flow can occur in dysfunction of the valves caused by anatomical changes, in widening of the heart cavities and large vessels, as a result the blood passes through a relatively narrow opening when compared with the enlarged chambers, when pathological openings are present (e.g. in the interventricular and interatrial septa).

An organic murmur develops in anatomical disturbances of the valve structure. If, due to anatomical disorders (fusion of the cusps), the valve cannot open completely, venous or arterial opening of the heart narrows. If the cusps of the valve do not close due to their fibrous thickening, shortening, destruction, or perforation, the blood goes back to the heart chamber from which it has been ejected, this defect is called incompetence {insufficientia valvuarum), the backward blood flow is called regurgitation (regurgitatio).

If incomplete closure of the unchanged valves occurs due to considerable enlargement of the heart or affection of the papillary muscles, this causes relative valvular incompetence. One of the openings can be narrower than usually when compared with the enlarged cavity (relative narrowing of the opening). The murmurs developing in this case are called muscular or organ-functional.

Valvular murmurs heard in the period of the orifice opening are the sign of the orifice narrowing. The murmurs, developing when the orifice must be closed, suggest incompetence of the respective valve.

The murmur occurring during systole (systolic) is the sign of atrioventricular valve incompetence or narrowing of the arterial orifice. The murmur developing during diastole (diastolic) is the sign of the venous orifice narrowing or incompetence of the semilunar valves.

The following properties of the murmur have diagnostic significance:

- association of the murmur with the phase of the cardiac cycle (systolic, diastolic);
- the area where it is heard best (heart apex, 2nd intercostal space to the right and left of the sternum, basis of the xiphoid process);
- direction of the murmur radiation (axillary area, cervical vessels, subscapular area);
- loudness, duration of the murmur;
- the timber of the murmur (blowing, sawing, scratching, musical);
- relation of the murmur and the sound after which it is heard (merges with the sound, separated from it);
- if the murmur increases or decreases during the pause; - influence of the position, physical exercise, phases of respiration.

It is not usually difficult to determine the relation of the murmur and the phases of the cardiac cycle. In tachycardia or ciliary arrhythmia, it is necessary to compare the time of the murmur development with pulsation of the carotid arteries or with the apical thrust. It is more difficult to determine if the murmur is heard at the beginning (proto-), middle (meso-) or at the end (tele-) of systole or diastole, or if it is heard during the whole pause (holosystolic, holodiastolic).

The area, where the murmur is best heard, allows to decide on the location of the lesion: the murmur over the apex is the signs of mitral valve affection, in the 2nd intercostal space On the right that of aortic valves, in the 2nd interspace on the left that of valves of the pulmonary artery, in the base of the xiphoid process - tricuspid valve. The murmur radiates along the blood flow causing it, the range of propagation is related to the loudness of the murmur. A systolic murmur in narrow aortic opening has the largest area of radiation: carotid arteries, heart apex, epigastric, interscapular regions. A diastolic murmur in the aortic valve incompetence radiates to the heart apex and the left edge of the sternum. A systolic murmur over the heart apex in mitral valve incompetence radiates to the left axillar and under the angle of the left scapular.

The loudness of a murmur ranges from poorly heard to those heard at distance (distance murmur). Six degrees of loudness are distinguished (Levine): 1- the murmur is heard only on concentrated auscultation, 2 - weak, 3 - medium, 4 - loud, 5 - very loud, 6 - distant.

But it is necessary to remember that in inconsiderable narrowing or in very large narrowing of the orifice, the murmur is not heard. In cardiac failure the loudness of the murmur decreases.

The loudness of the murmur is influenced by numerous extracardiac factors: thickness of the chest wall, the degree of coverage of the anterior surface of the heart with emphysemically widened lungs, distention of the stomach and intestines with gases.

The changes of the murmur loudness at prolonged observation of the patient can be of diagnostic and prognostic significance. For instance, if in the patient with mitral stenosis the murmur increases at first and later decreases, this may be the sign of stenosis progression, as organic anatomical affection of the valve cannot decrease. In contrast, in the patient with pronounced heart failure the murmur appears or increases during treatment, it means that the contractile function of the myocardium has improved and the rate of the blood flow has increased.

As to the pathogenesis, the murmurs are divided into ejection, regurgitation, filling murmurs.

Ejection murmurs develop as a result of the blood flow acceleration when passing the narrowed orifice at contraction of the heart muscle.

A systolic ejection murmur is characteristic for stenosis of the aorta, pulmonary artery, in increased stroke volume when the valves are intact. This murmur begins after a pause separating the murmur from the first sound, its duration is directly proportional to the degree of stenosis. The murmur gains its maximum in mesosystole, then decreases and dies before the appearance of the second sound separated from it by a pause. It is often accompanied by a "cat's purring". A systolic ejection murmur associated with increased circulation begins immediately after the first sound, it is short and reaches its maximum during protosystole.

A presystolic ejection murmur in mitral and tricuspid stenosis appears during telediastole resulting form atrium contraction. This special rumbling murmur increases by the moment the first sound merges with it and is accompanied by a cat's purring.

A systolic regurgitation murmur in mitral or tricuspid valve insufficiency is long, frequently pansystolic, merges with the first sound and decreases by the second sound appearance.

A diastolic regurgitation murmur in incompetence of the semilunar valves of the aorta and pulmonary artery is joined with the second sound, its loudness increases before telediastole, it is, as a rule, blowing and low.

A diastolic filling murmur in atrioventicular stenosis is separated with a pause from the second sound, it follows the snap produced by opening mitral and tricuspid valves, it is long, decreasing.

A systolic murmur at the heart apex is frequently observed causing the problems for diagnosis.

Organic incompetence of the mitral valve is a rare cause of this murmur. In typical cases it merges with a weakened first sound, is of moderate loudness, is long (not less than 2/3 of systole), can be blowing, noisy, whistling, sometimes rough and musical. It is better heard directly at the heart apex or a little cranially, radiates to the left axillar and to the angle of the left scapula, is frequently heard in the 3rd-4th interspaces at the left edge of the sternum, it increases in left decubitus and in a lying position. A mesosystolic decreasing murmur frequently appearing after systolic click is characteristic for mitral prolapse.

This murmur can be muscular (organ-functional) at considerable widening of the left ventricle.

A low short inconstant systolic murmur, which does not radiate to the axillar, can be muscular (organ functional) in acute myocardial infarction (affection of the papillary muscle of the mitral valve), in myocarditis, it can also be functional (physiological). Aortic and tricuspid systolic murmurs frequently radiate to the region of the heart apex.

A systolic murmur in point 3 in the area of pulmonary artery auscultation is frequently observed in healthy children and young persons and is functional. It is usually low, blowing, does not merge, does not radiate, appears or increases on physical overstrain, in a lying position, especially by the end of deep breathing out, is frequently accompanied by a split or dual, increased second sound.

This murmur accompanied by a dual second sound can be the sign of the interatrial septum defect.

A systolic murmur in pulmonary artery stenosis is loud, long, rough, sometimes musical, is accompanied by "a cat's purring", is well heard in an upright position, radiates to the interscapular region and the left supraspinal fossa, and does not radiate to the vessels of the neck.

This murmur can occur on compression of the pulmonary artery with pleural exudation, enlarged lymph nodes of the mediastinum, widening of the pulmonary artery resulting from increased pressure in the pulmonary system, in hypothyrosis. The murmur from point 2 radiates to this area in aortic stenosis.

A systolic murmur in the area of the aorta is also a frequent acoustic phenomenon. If this is an organic murmur of aortic stenosis, it is loud, long, sowing, scratching, deadens the first sound; the second sound is not heard as well. It radiates to the carotid arteries, median portion of the right clavicle, the whole precordial area, epigastric area, right supraspinal fossa. The murmur increases first, then decreases. The murmur is heard in a sitting position, when the body is bent forward, on deep breathing out, after physical load, is accompanied by "a cat's purring". Similar murmur can be observed in aorta atherosclerosis, syphilitic aortitis, arterial hypertension, aortic valve insufficiency.

Sometimes a systolic murmur over the aorta is heard in healthy persons, it. is low, blowing, does not radiate, changes with respiration and the position of the patient, is not accompanied by "a cat's purring" (functional).

But in organic stenosis of the aorta a systolic murmur can be low or is not heard (severe stenosis) when the stenosis is accompanied by mitral stenosis or cardiac failure. A systolic murmur in the area the tricuspid wave (point 4) frequently results from radiation from the mitral or aortic orifices. Less frequently this murmur appears in relative or organic incompetence of the tricuspid valve.

Lower pressure in the right heart is obviously responsible for poor hearing of the systolic murmur in affection of the tricuspid valve.

In relative tricuspid valve incompetence, a systolic murmur is heard over the lower border of the sternum or at its left edge at the level of the 4th-5th intercostal spaces. It is low, soft, low-pitched, frequently poorly heard, prolonged, increases on deep inspiration and disappears on expiration, can radiate to the heart apex. Sometimes, when the signs of tricuspid insufficiency are well pronounced (pulsation of the jugular veins, liver), a systolic murmur is not heard.

Organic tricuspid valve incompetence is rare. In this defect the murmur radiates from point 4 along the edge of the sternum cranially, to the epigastric area and right axillar. Its properties range from loud, rough to low, tender, blowing even in one patient, this is accompanied by the a cat's purring in the 4l -5l intercostal spaces on the right of the sternum, sometimes along the right parasternal line at this level. The murmur can increase when pressure is exercised on the enlarged liver or abdomen.

A systolic murmur with the epicenter in the 3rd-4th interspaces at the edge of the sternum is the sign of interventricular septum defect. This murmur is loud, overlaps the first sound, is heard during the whole systole, is accompanied by a cat's purring, has a specific timber as appears because the blood goes from the left ventricle to the right one under pressure. It radiates to the whole precordial area, to the brachial arteries, the arteries of the neck, interscapular space, under the shoulder blades to the elbow, increases in a lying position and does not change with breathing. A diastolic murmur over the heart apex can be local, autochthonous or can radiate from the heart base, more frequently from the aorta in aortic valve incompetence.

In mitral stenosis a diastolic murmur over the apex has a specific timber and is low-pitched, this is described as grumbling, rolling, roaring. But it can often be low and better heard using a stethoscope with a funnel-like tip without a membrane. Sometimes it is so short that does not resemble a murmur and only

with the patient in left decubitus it becomes long enough to be diagnosed. The murmur is sometimes not heard due to its low frequency, but a cat's purring is present. It is more frequently heard for a long time, sometimes during the whole diastole. It is better heard during those phases of diastole when the velocity of the blood flow is maximal: in the 1/3 of diastole when pressure gradient between the atrium filled with blood and relaxed ventricle is maximal. As the ventricles is being filled with the blood, the blood flow velocity and the murmur intensity decrease up to complete disappearance (protomesodiastolic filling murmur). Then, at the end of ventricular diastole, the atrium contracts, the blood flow accelerates, the murmur increases, This is pre-systolic ejection murmur and is frequently accompanied by a cat's purring, increases in left decubitus and after a physical load. This murmur is characterized by a special "roaring" timber, increases by the moment the first sound appears and merges with it.

A presystolic murmur suggests sufficient contractile function of the atria, it disappears on dilation and development of ciliary arrhythmia.

A diastolic murmur in mitral stenosis is heard over a limited area, frequently in the area of the heart apex, sometimes medially or laterally, to the axillar and midaxillary line. It can frequently be heard at the lower angle of the left scapula. In a supine position and in left decubitus after physical load this murmur increases and sometimes disappears after several cardiac cycles following the physical exercise.

A diastolic filling murmur is characterized by a pause between the second sound and the murmur, which occurs as a result of delay in opening of the changed mitral valve.

A muscular diastolic murmur caused by relative narrowing of the left atrioventricular orifice can sometimes be heard in patients with an enlarged left ventricle accompanied by shortened blood flow through the mitral orifice caused increased blood pressure in the pulmonary veins (patent arterial duct, a large defect of the interventricular septum, severe anemia, insufficiency of the mitral valve with considerable blood regurgitation, essential hypertension, coronary artery disease).

In aortic valve incompetence, a low short presystolic murmur described by A. Flint is heard over the heart apex. This murmur is not accompanied by snapping first sound and mitral click, a cat's purring, does not increase in left decubitus. This murmur is produced by relative narrowing of the left atrioventricular opening due to considerable dilation of the left ventricle. It is believed that relative narrowing of the left atrioventricular orifice moves the anterior cusp of the mitral valve towards the atrioventricular opening when the blood coming back from the aorta to the left ventricle. In the both cases the murmur is organ-functional.

At the heart base in points 2, 3, 5, one can hear diastolic murmurs associated with incompetence of the semilunar valves, more frequently of the aorta and less frequently of the pulmonary artery. They are difficult to discriminate: their epicenters are close to each other, sometimes in the same place, their acoustic differences are minimal. The pathogenesis of the murmur should be established basing on the entity of the clinical signs.

A diastolic regurgitation murmur in aortic valve insufficiency is better heard in point 5 over the middle of the sternum or to the left at the level of the 3rd-4th interspaces, sometimes over the xiphoid process, less frequently in point 2 in the 2nd intercostal space on the right of the sternum. A right-Bided murmur is considered to be the sign of significant dilation of the ascending aorta in syphilitic mesarteritis, traumatic injuries of the aortic valves, bacterial endocarditis, dissection aortic aneurysm. Sometimes this murmur is heard only over the heart apex.

It frequently radiates to the area between the heart apex and the sternum, to the heart apex and the left axillar as well as cranially to the vessels of the neck and supraspinous fossae. This murmur begins at the very beginning of diastole, merges with the second sound filling the most part of diastole, gradually decreasing to its end, but it can also be very short. The murmur is low, tender, blowing, high-pitched, better heard with a phonendoscope provided with a membrane or with an ear densely pressed to the precordial area. It sounds like going from the depth, therefore experience and skill of the physician are necessary to hear it. It is sometimes better heard in an upright position, when the body is bent forward with the hands on the head, the respiration should be held on the height of expiration.

A musical loud, sometimes squeaky, diastolic murmur is heard in rapture of the valve cusp (trauma, bacterial endocarditis), in a congenial defect (bicuspid aortic valve).

The diastolic murmur of incompetence of the semilunar valves of the pulmonary artery is usually muscular (organ-functional) and results from increased pressure in the pulmonary system in mitral stenosis, Graham-Still's murmur. It is produced due to a considerable dilation of the pulmonary artery and the right arterial opening, which cannot be closed by the unchanged semilunar valve.

Less frequently, the cause of the pulmonary artery dilation can be chronic cor pulmonale, Ayerza's disease, high defect of the interventricular septum, defect of the interatrial septum, patent arterial duct. An organic affection of the semilunar valves of the pulmonary artery is rare and is usually a congenital defect. A diastolic murmur in the area of the tricuspid valve auscultation (point 4) is the sign of organic narrowing of the right venous opening, it can be heard in the 5th intercostal space at the edge of the sternum. This murmur is short, low, increases on inspiration (Karvallo's sign).

A continuous murmur is that heard during both systole and diastole because the blood flows in the same direction during the both phases of the cardiac cycle. This murmur is heard as one, rhythmically increased in a definite phase sound.

An arteriovenous murmur, occurring when the blood enters the vein from the artery, and a venous turbulent murmur, occurring when the blood enters the heart from the periphery, are distinguished. The turbulent venous murmur is heard over the jugular vein, cranially the collarbone between the sternum and the clavicular portion of the sternoclaviculopapillary muscle. This resembles a continuous rumble, buzz, a cat's purring, is accompanied by palpatory sensations of systodiastolic rumbling of the neck near the jugular vein. The murmur increases on inspiration and during diastole, when the head is turned to the side opposite the place of auscultation, in an upright position, better on the right than on the left side. This murmur is heard in severe anemia. In arteriovenous aneurysms, a continuous murmur is heard over the place of the aneurysm, it radiates from the periphery to the center, increases during systole and is accompanied by a cat's purring.

The murmur in patent arterial duct is better heard in the 2nd-3rd interspaces parasternally on the left, radiates to the 4th intercostal space, to the heart apex, as well as the left subclavicular area, interscapular area and is sometimes accompanied by a cat's purring. The diastolic portion of the murmur is usually weaker than the systolic one. This is a continuous, loud, blowing sound resembling working machinery, it increases when the patient is lying, on deep inspiration, physical load.

During the initial 2-3 years of the life only a systolic murmur resembling a functional one can be heard. This murmur is observed in pulmonary hypertension. Only diastolic component of the murmur can be heard in relative incompetence of the pulmonary artery valves.

A continuous murmur can be heard in congenital arteriovenous pulmonary aneurysms, defects of the aortopulmonary septum and rupture of Valsalva sinus aneurysm resulting in disposal of the blood to the right atrium or ventricle.

Extracardiac murmurs are those associated with the heart rhythm but originating outside the heart cavities. These are pericardial friction rub and pleuropericardial friction rub.

Pericardial friction rub occurs when the surface of the pericardium is rough or uneven (fibrinous pericarditis, tumor-like formations).

Pericardial friction rub is heard in the both phases of the cardiac cycle, especially at the beginning of systole and beginning of diastole, it resembles a double murmur in aortic defect. Sometimes it can be only systolic of diastolic or consists of three parts: presystolic associated with the work of the atria, systolic and protodiastolic. If four short murmurs are heard, a sound of a working locomotive appears.

Pericardial friction rub is heard over various areas of the precordial area, more frequently at the heart base at the left edge of the sternum in the 4th-5th intercostal spaces, the zone, to which it radiates, is limited. It is frequently low, tender, superficial but sometimes can be loud, scratching, crispy, resembles crispy snow, shuffling of silk, squeak of the sole of new shoes, is accompanied by tactile sensations and is heard at a distance. In contrast to pleura friction rub, it is associated with the work of the heart, not with respiration. But this murmur is sometimes heard only at forced expiration, sometimes only on deep inspiration. Pericardial friction rub is louder when the patient is sitting, the body is bent forward, when pressure with the funnel of the stethoscope is exercised, at Valsalva maneuver.

Pericardial friction rub is labile, in some cases this is constant during several weeks or even months.

With fluid accumulation in the pericardial cavity it weakens and disappears.

A pleuropericardial murmur occurs in inflammation of the mediastinal, costal, pulmonary pleura adhering to the external layer of the pericardium. This is heard at the left edge of the sternum, is associated both with heart contraction and with respiration: on deep respiration both pleura friction rub and pericardium friction rub are heard if the patients holds the breath.

A peritoneopericardial murmur occurs in serofibrinous perihepatitis due to the heart movement along the rough surface of the liver and diaphragm and is heard near the xiphoid process.

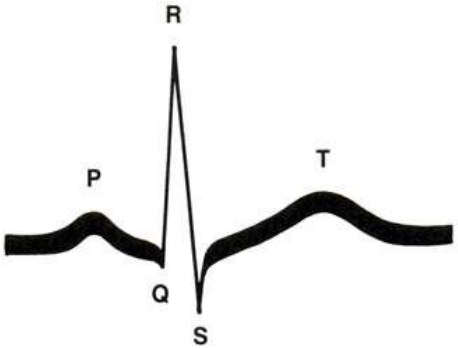
Theme 13. **Electrocardiographical Methods of Examination the Functions of the Heart. Methods of the Registration and Detailed Study of ECG. ECG Signs of Hypertrophia of the Auricle and Ventricle.**

Clinical and diagnostic importance of ECG. Biophysical and physiological basis of ECG. Structure and function of pacemakers of heart rhythm and conduction system. Method of ECG registration: Standard leads; Unipolar leads from Extremities and Sternal leads. Basic elements of ECG: importance of duration and amplitude of waves, duration of intervals and segments in normal. Methods of **ECG** study: ECG signs of the right and left ventricular hypertrophia.

The electrocardiogram presents a visible record of the heart's electrical activity by means of a stylus that traces the activity on a continuously moving strip of special paper.

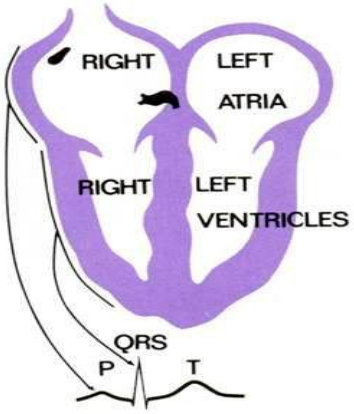
Normal ECG. All beats appear as a similar pattern, equally spaced, and have three major units: P wave, QRS complex, and T wave.

Normal single heartbeat



Normal single heartbeat. Each beat manifests as five major waves: P, Q, R, S, and T. The Q, R, and S all represent the same part of the heart (ventricles). They are usually referred to as a unit: the QRS complex.

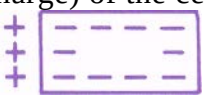
The heart in relation to the ECG



The heart in relation to the ECG. Each wave represents transmission of an electrical impulse through the heart muscle (depolarization), which causes the muscle to contract and thus eject blood. The P wave reflects the impulse going through the atria. The QRS complex reflects the impulse

going through the ventricles. The T wave is produced by the electrical recovery (repolarization) of the ventricles.

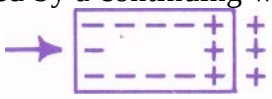
Depolarization. As the electrical impulse moves across the cells of the myocardium, the polarity (negative or positive electrical charge) of the cells is changed.

The resting cell has a negative charge: 

The electrical impulse carries a positive charge into the cells, changing the polarity:

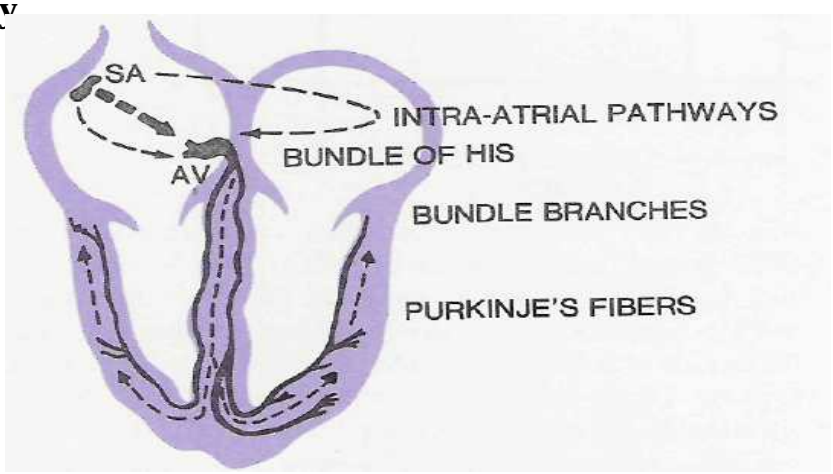
This is called depolarization: 

It is followed by a continuing wave of repolarization that restores the cell to its original charge:



The cell is then ready to receive another stimulus.

Normal electrical pathway



Normal electrical pathway. The impulse is conducted to the muscle cells by way of specialized tissue that has automaticity and conductivity. All myocardial tissue has these properties, but they are developed to a greater degree in the conduction system.

The electrical impulse originates in the sinoatrial (SA) node - the normal physiological pacemaker - located near the top of the right atrium. The impulse spreads through intra-atrial pathways to the atrioventricular (AV) node located at the junction of the atria and ventricles. After a brief delay, the impulse continues through the bundle of His, the right and left bundle branches, and Purkinje fibers, and finally activates the ventricular muscle cells. Both the SA and AV nodes are innervated by the sympathetic system, which increases the heart rate, and by the parasympathetic system (vagus nerve), which slows the rate.

The SA node normally discharges impulses at a rate of 60-90 times per minute, AV junctional tissue at 40-60, and Purkinje fibers at 20-40. The pacemaker firing at the fastest controls the heartbeat. The presence of multiple pacemakers provides a reserve or backup system against cardiac arrest.

ECG paper. To understand the significance of each wave and interval, we need to know the significance of the small and large blocks on the ECG paper. The paper moves through the ECG machine at the rate of 1 inch per second (standard setting). One small block represents 0.02 second on the horizontal line

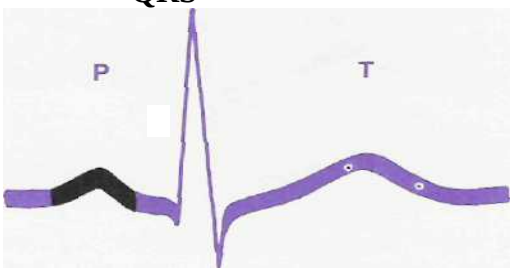
and 1 mm on the vertical line. Since a large block is five small blocks wide and five high, each large block represents 0.10 second (horizontal) and 5 mm (vertical). 10 mm = 1 millivolt of electrical activity.

Now that we know these basic measurements and are familiar with the relation of the ECG waves to the heart anatomy, let's discuss the significance of each wave and interval:

The **P wave** is the first upward deflection and represents the atrial depolarization. Enlargement of the P wave might occur in such conditions as mitral stenosis or chronic obstructive pulmonary disease, which would cause atrial hypertrophy. The P wave is usually considered enlarged if it is more than 3 mm high and 0.10 second wide.

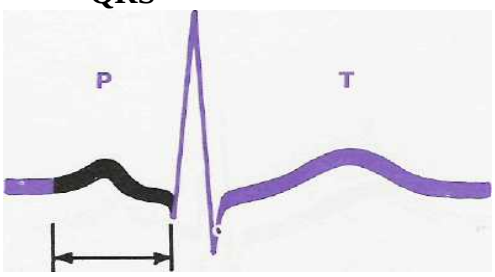
QRS

P wave



The **PR interval** extends from the beginning of the P wave to the onset of the QRS. It represents conduction of the impulse through the atria and the AV node. The PR interval is abnormally lengthened when the impulse is forced to travel at a slower rate, which can occur in arteriosclerosis, inflammation, insufficient oxygen supply, or scarring from rheumatic heart disease. It can also occur as an effect of depressant drugs or digitalis. The normal PR interval is 0.12-0.20 second wide .

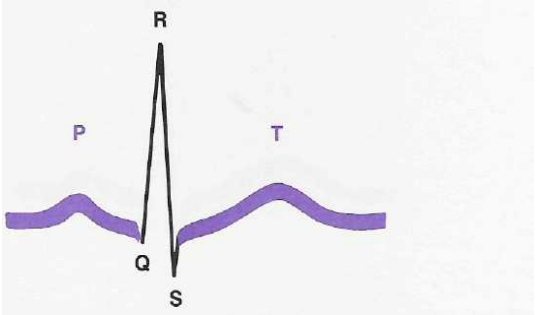
PR interval **QRS**



PR INTERVAL

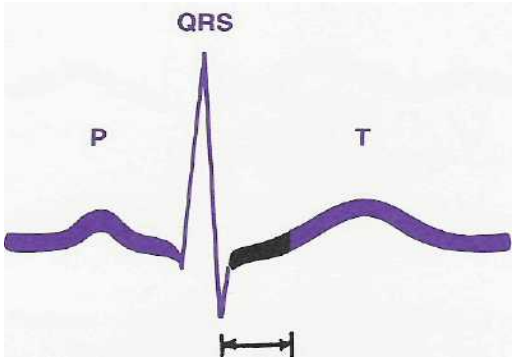
The **QRS complex** consists of three deflections: Q wave, the downstroke before the R; R wave, the first upward deflection; and S wave, the downstroke following the R wave. Not every QRS complex shows a discrete Q, R, and S wave, but the configuration is still referred to as the QRS complex to denote a ventricular impulse. An enlarged Q wave (Its amplitude is small and does not normally exceed one-fourth amplitude of the R wave; the length of the Q wave does not exceed 0.03 second. The Q wave may be absent on an ECG.) may indicate a myocardial infarction. A vertically enlarged R wave usually indicates enlarged ventricles. The normal duration of the QRS is 0.06 – 0.10 second wide.

QRS complex



The **ST segment** begins at the end of the S wave (the point where the line turns right) and ends at the beginning of the T wave. It is elevated in an acute myocardial infarction or muscle injury. It is depressed when the heart muscle isn't getting a sufficient supply of oxygen - for example, during an episode of angina or coronary insufficiency. It may sag as an effect of digitalis. ST changes are usually transient.

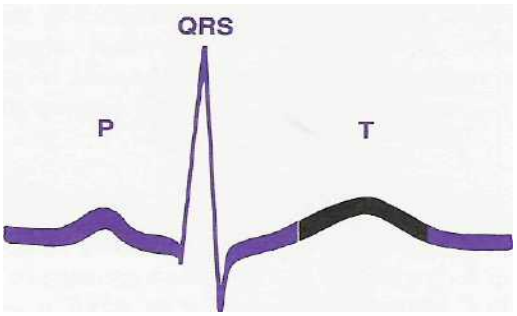
ST segment



ST SEGMENT

The **T wave** represents electrical recovery of the ventricular contraction. (The electrons are in the process of moving back into the normal resting position.) The T wave is flat or inverted in response to ischemia, position change, food intake, or certain drugs. It may be elevated when the serum potassium is elevated. The normal T wave is no more than 10 mm high in the precordial (chest) leads and 5 mm high in the remaining leads.

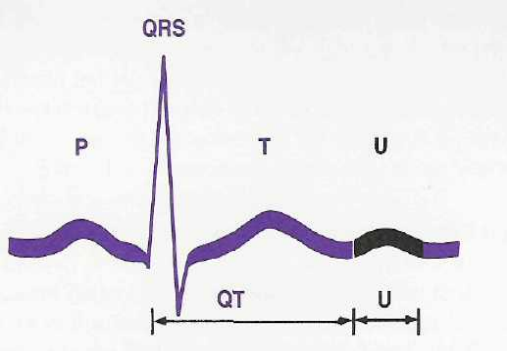
T wave



The U wave is a small upward deflection following the T wave. It is seldom present, but may occur when the serum potassium level is low.

The **QT interval** represents the time from the beginning of the Q wave (downward deflection following the P wave) through the QRS and the T wave. It includes the time until the T wave is completed (goes back to the baseline). The time of this interval should be less than one-half of the R-R interval (from the peak of one R wave to the peak of the next R wave). If the QT time is prolonged, it presents an extended opportunity for stray irritable impulses to excite the heart tissue and trigger dangerous ventricular rhythms. After the T wave is completed the tissue is repolarized and at rest, ready to respond normally. Impulses that arrive during the T wave find the ventricular tissue incompletely recovered and vulnerable to an erratic response. The Q-T interval in women is longer than in men (at the same leart rate). For example, at the rate of 60-80 beats per minute, the length if the Q-T interval in men is 0.32-0.37 second and in women: 0.35-0.40 second.

U wave, QT interval



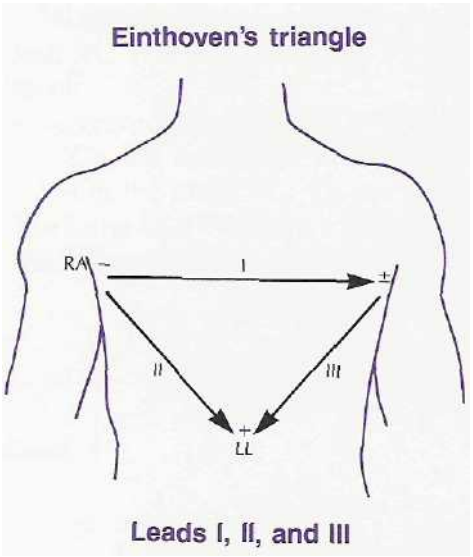
THE ECG LEADS

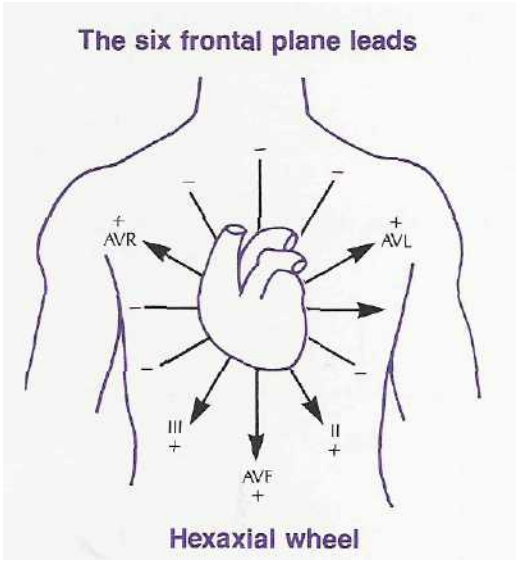
ECG leads are formed by placing electrodes at specific places on the body and amplifying and recording the electrical activity that occurs along this pathway. A vector is a force of a known magnitude and direction. The recordings may be said to display certain vectors or electrical forces traveling in the direction between the leads.

The first three bipolar leads used by ECG pioneer Dr. Einthoven were leads I, II, and III recording the electrical activity along the pathway from a negative electrode toward a relatively positive electrical pole.

Each lead records the same electrical impulse on the ECG but from a different position in relation to the heart. Each can be read separately, or they can be read in combination.

The limb leads show the current flow from one area of the body toward another. Lead I shows electrical activity from the right arm to the left arm, lead II from right arm to left leg, and lead III from left arm to left leg. The right-leg position is not displayed as part of the flow of current through the heart, as it is used for grounding the system. In the 12-lead ECG, the leads are placed on the limbs. For continuous monitoring, these leads are positioned on the chest in a smaller configuration.





Whenever electrical activity flows from a negative pole toward a positive pole, it causes an upward deflection on the ECG tracing. The lead that will show the tallest upward deflections is the one that is parallel to the actual forces in the body. Lead II is frequently used for monitoring as it is usually parallel to the direction of the electrical activity in the heart, and shows an easily visible P wave. Illustrations in this book are taken from lead II unless otherwise labeled.

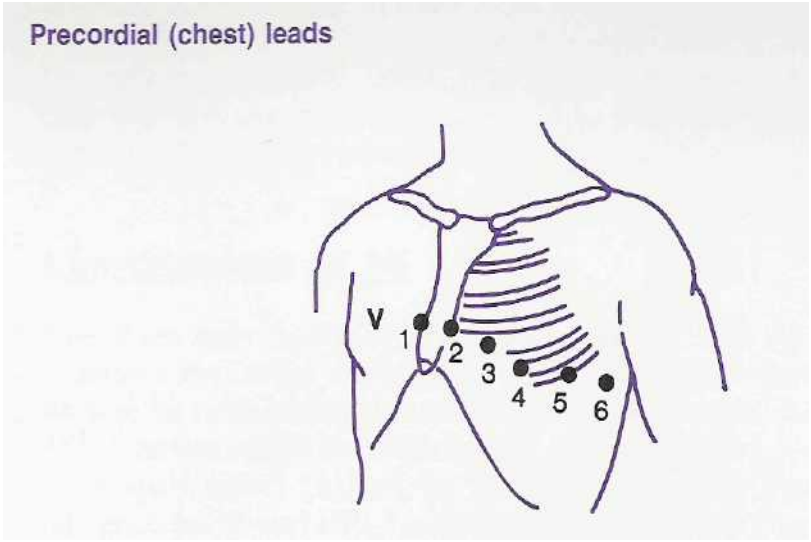
If more lead placements are used, specific areas of heart dysfunction can be identified. Therefore the positive or exploring electrode is positioned at other sites and by using combinations of other electrodes for the neutral or negative pole, unipolar lead sites are formed.

The abbreviation AVR stands for augmented vector right, an added point of reference for diagnosis. AVL and AVF represent the left-side and left-foot positions, respectively.

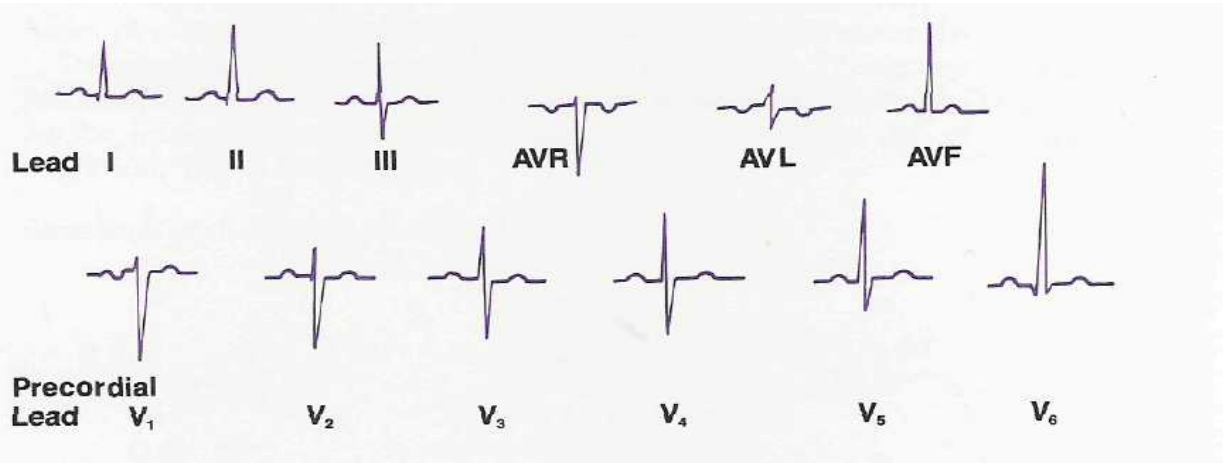
The combination of all six leads on the frontal plane gives us a full circle of references to evaluate cardiac function. This is called the hexaxial wheel.

In addition to the frontal plane, a horizontal plane of reference can be used for more precise location of problems.

The **precordial leads** provide points of reference across the chest wall as illustrated. They differentiate right-sided and left-heart events.



The electrode is placed successively at 6 positions: V1 - right sternal border, the 4th intercostal space; V2 - left sternal border, the 4th interspace; V3 - left parasternal line, between the 4th and 5th interspace; V4 - left midclavicular line, the 5th interspace; V5 - left anterior axillary line, the 5th interspace; V6 - left midaxillary line, the 5th interspace. Unipolar chest leads proposed by Wilson are more popular now. The chest electrode, which is attached to the positive pole of the electrocardiograph is only active; the electrodes leading from the limbs are united and connected to the negative terminal of the apparatus. With this connection, the total potential difference recorded from the limbs is practically zero.



Modified chest lead I is a monitoring lead that simulates precordial lead V1. This lead will help to differentiate right from left bundle branch block.

To record a routine ECG, 12 leads are used:

On the limbs: I, II, III, AVR, AVL, AVF
On the chest: V1, V2, V3, V4, V5, V6

The same beat can have a different configuration when seen from a different lead. These are the normal variations in the 12-lead ECG.

The most important leads to remember in relation to the anatomy of the heart are:

I, AVL	Anterior heart
II, III, AVF	Inferior heart
V1, V2	Right side of heart
V3, V4	Transition between right and left sides of heart
V5, V6	Left side of heart

The area of pathology shown on the ECG can be localized by analyzing tracings from different leads. For example: If an infarct shows up on leads II, III, and AVF only, it is located in the inferior aspect of the heart.

EHA DEFINITION

The hypothetic line connecting the two electrodes taking part in the formation of the electrocardiographic lead is called the axis of the lead. The axis of the three standard leads in a frontal plane make an equal sides Einthoven triangle as if moving through both the hands and the left leg used for choosing the direction of the heart electric axis and the α angle. The heart vector, a scheme arrow showing the direction of the heart moving power (the length of which characterises the quality of this power, starts in the center of the heart). To find the direction of this electric heart axis (EHA) one must find the algebra sum of R and S waves of the QRS complex in the I and III leads and mark this figure on the axis of the corresponding lead, then draw a perpendicular from every point appeared and draw a vector to the place of their crossing. This will be direction of the electric heart axis.

Electric heart axis (EHA) – is an average direction of the electric heart moving power (EHMP) during the whole depolarization (the projection of the EHMP on the thorax and it coincides with the anatomic heart axis.

The EHA can be determined by the R and S waves in standard leads.

- EHA isn't inclined: RII = RI + RIII;
- EHA is inclined to the left: RI SIII;
- EHA is inclined to the right: SI RIII.

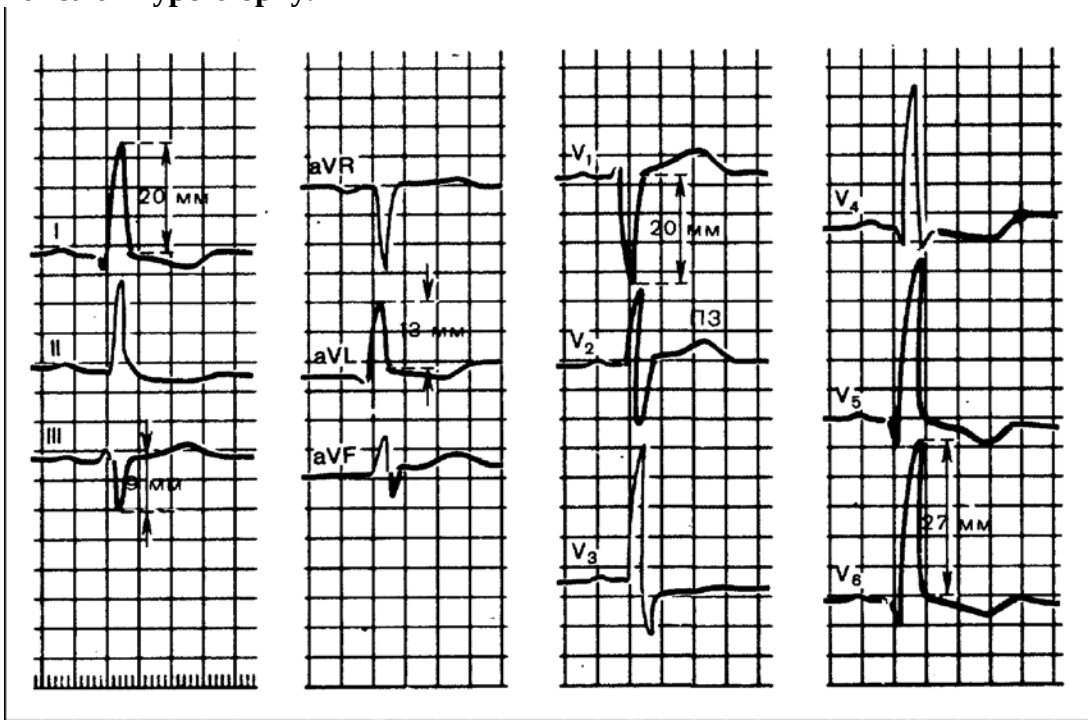
The angle between EHA and the first horizontal lead is called angle α.

- EHA isn't inclined: $0° < \alpha < +90°$
- EHA is in inclined to the left: $-30° < \alpha < 0°$

EHA is in inclined to the right: $+90° < \alpha < +120°$.

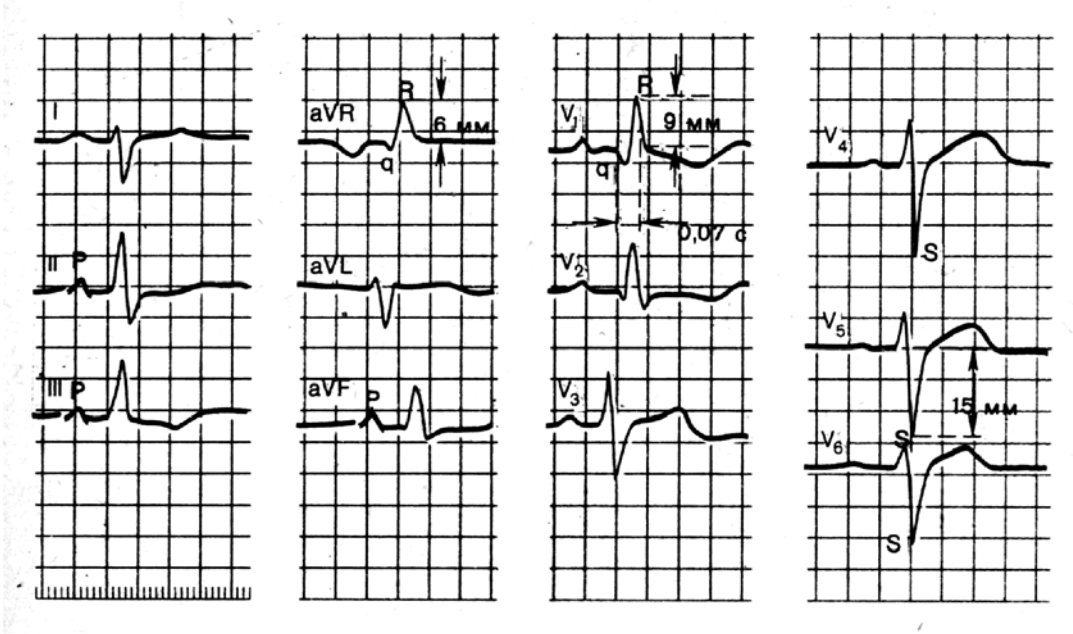
VENTRICULAR HYPERTROPHY

Left ventricular hypertrophy.



1. The position of the electrical heart axis is inclined to the left: RI SIII;
2. The tall R wave in I, AVL and V5,V6;
3. The deep S wave in III, AVF, V1,V2;
4. S-T depression and T wave inversion in leads I, AVL and V5,V6.

Right-ventricular hypertrophy.

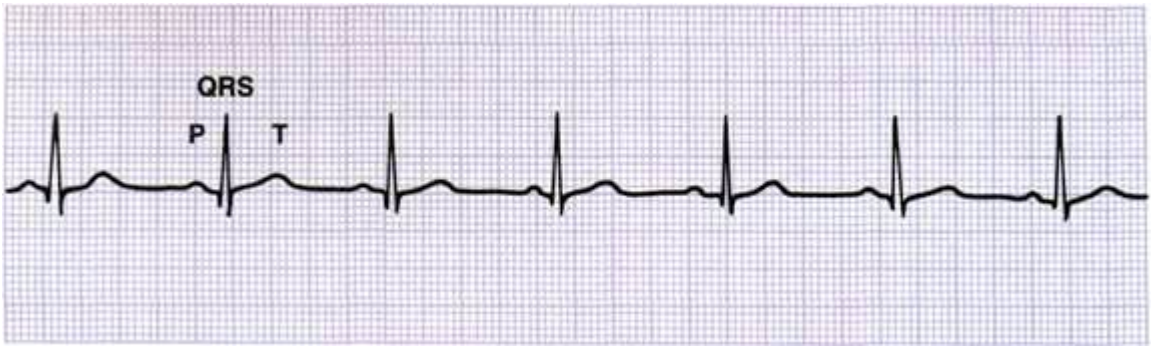


1. The position of the electrical heart axis is inclined to the right: SI RIII.
2. The tall R wave in II, III, AVF and V1, V2;
3. The deep S wave in I, AVL, V5, V6;
4. S-T depression and T wave inversion in leads II, III, AVF and V1, V2.

Theme 14. **Electrocardiographical Examination of the Patients with Disorders of Functions of Automatism, Excitation.**

The main structures, which provide the function of the Heart automatism. ECG signs of infringements of automatism: sinus tachycardy, sinus bradycardy, sinus arrhythmia, and syndrom of weakness of sinus node. Types of extrasystoles. ECG signs of sinus, auricle, auriclaventricular and ventricular extrasystoles. Differentiation of the right and left ventricular extrasystoles. Classification of ventricular extrasystoles. Types of allorhythmies.

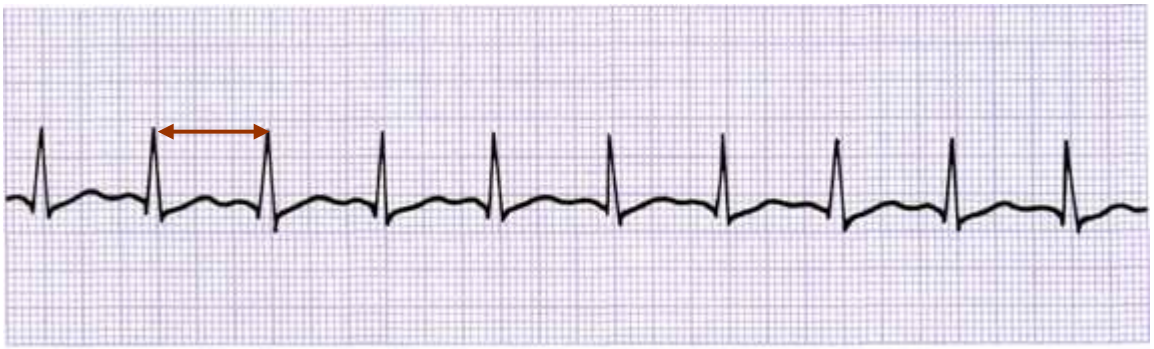
NORMAL SINUS RHYTHM



If all findings are normal, **normal sinus rhythm** is present.

Rhythm. Regularity may be determined using calipers or any device that can be marked to show a fixed interval for comparison.

Determining the cardiac rate



3 large block

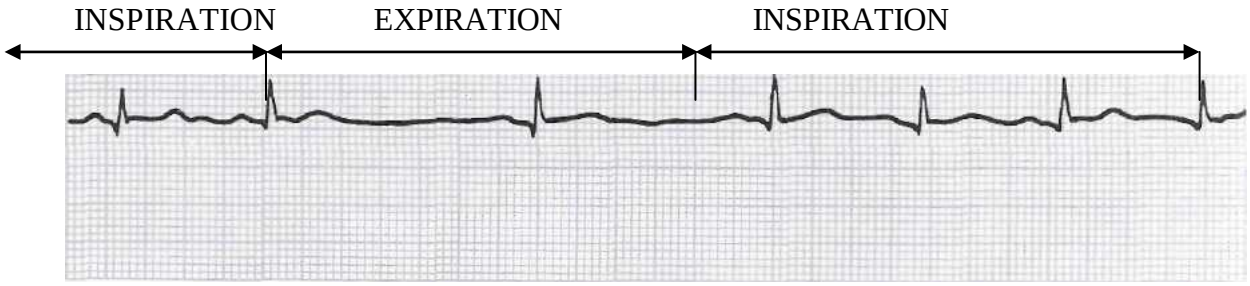
Alternative methods of **determining the cardiac rate.** To that end, duration of one cardiac cycle (the R-R interval) and the number of such cycles in one- minute length should be determined. For example, if one cycle lasts 0.3 second, there will be 200 such cycles in a minute (60 : 0.3 = 200). If the cardiac rhythm is irregular, the length of five or ten R-R intervals is determined, the mean R-R interval found, and the cardiac rate is finally determined as for regular cardiac rhythm. Lengths of the maximum and minimum R-R intervals are given in parentheses.

SINUS ARRHYTHMIAS

Pathway of sinus rhythms. Sinus arrhythmia, sinus tachycardia, and sinus bradycardia originate in the SA node. The path of their electrical impulses is exactly the same as that of normal sinus rhythm. Because of this, the P wave, the PR interval, and the QRS complex are of normal configuration. The difference lies in the regularity and rate of the impulses.

Sinus arrhythmia. All complexes are normal, but the heart rhythm is irregular. The rate increases with inspiration, decreases with expiration. This irregularity is common in children and may occur in adults in relation to certain respiratory patterns. It does not decrease cardiac output and does not lead to more serious arrhythmias.

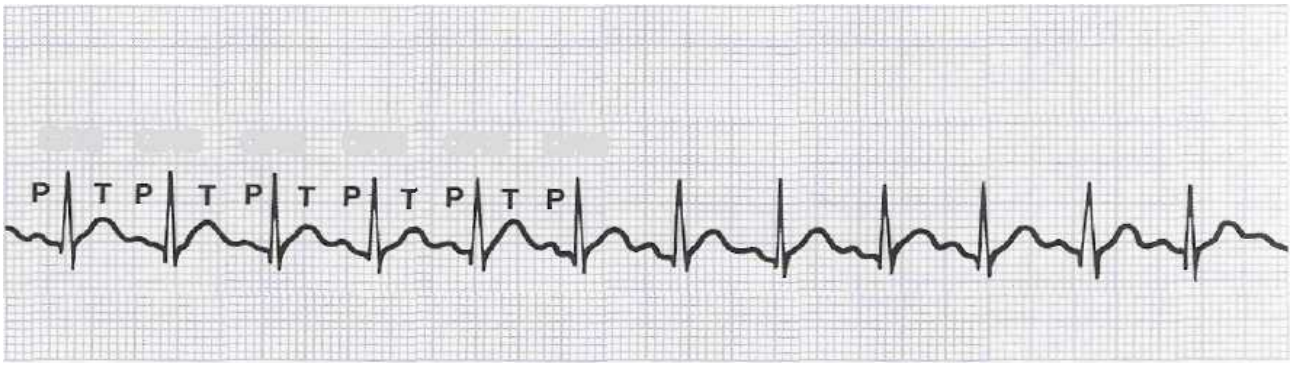
Sinus arrhythmia



Sinus tachycardia. All complexes are normal, but the heart rate is more than 100. (It seldom exceeds 160.) Excessive sympathetic nerve stimulation causes the increased rate. Common causes are physical activity, anxiety, and fever. An increased rate may also be a compensatory response to decreased cardiac outpul.

Complications such as congestive heart failure, pulmonary embolism, cardiogenic shock, or bleeding may show sinus tachycardia as an early symptom. Look for the problem. Treatment is directed to the physiologic cause.

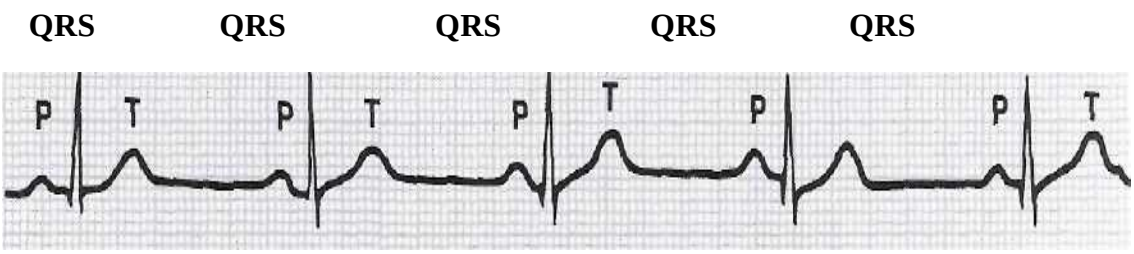
Sinus tachycardia



Sinus bradycardia. This arrhythmia is defined as a heart rate below 60 while all complexes remain normal. Sinus bradycardia is seen as an expected manifestation in well-trained athletes. It may occur in patients on digitalis, propranolol, morphine, and pressor amines for treatment of low blood pressure. A significant slowing may cause a decrease in cardiac output that can lead to cerebral or coronary insufficiency. An additional hazard is that bradycardia may permit ectopic pacemaker foci to take over, causing serious arrhythmias. Sinus bradycardia may be beneficial in a person at rest but if seen during stress, it could indicate inability of the heart to compensate.

The decision to treat sinus bradycardia is based on an evaluation of the patient's clinical picture. If drug therapy is indicated, IV atropine is used to inhibit the vagus (heart-slowing) nerve, thus speeding up the heart rate. If the patient does not respond, use of an artificial pacemaker may be necessary.

Sinus bradycardia



Format for analyzing arrhythmias

ECG characteristic	Summary of normal findings
Rhythm	Regular (distance between QRS complexes varies by no more than three small squares)
Rate	60-90 beats per minute
P wave	Present and upright (in leads I, II, AVF, V2 - V6) All shaped alike
PR interval	P wave precedes QRS 0.12 - 0.20 sec. Time interval is the same for all beats
QRS complex	Present All shaped alike 0.10 sec.

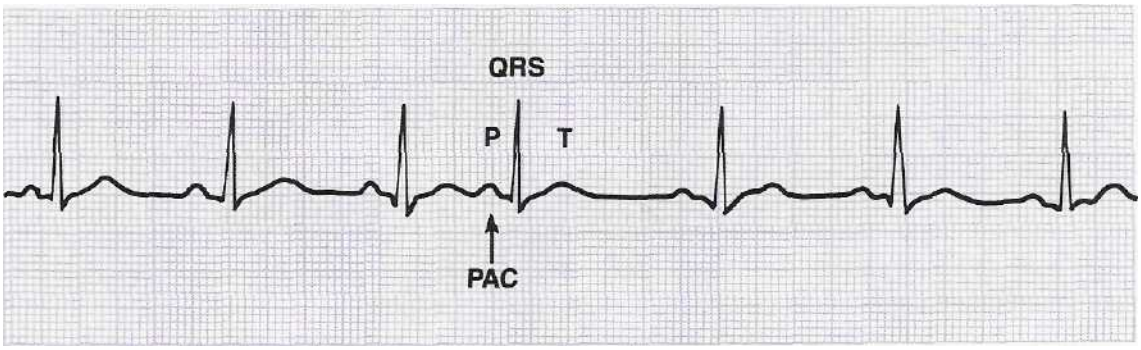
PREMATURE CONTRACTIONS OR ECTOPIC BEATS

Portions of atrial tissue may become excitable and initiate impulses. These ectopic foci will control the heartbeat if they occur at a rate faster than impulses from the SA node.

Premature atrial contraction (PAC) is a beat initiated by an ectopic atrial focus that appears early in the cycle (before the next expected sinus beat). Since the impulse arises from a site other than the sinus node, the shape of the P wave and the length of the PR interval may be different. The premature P wave is sometimes difficult to distinguish when it is superimposed on the preceding T wave.

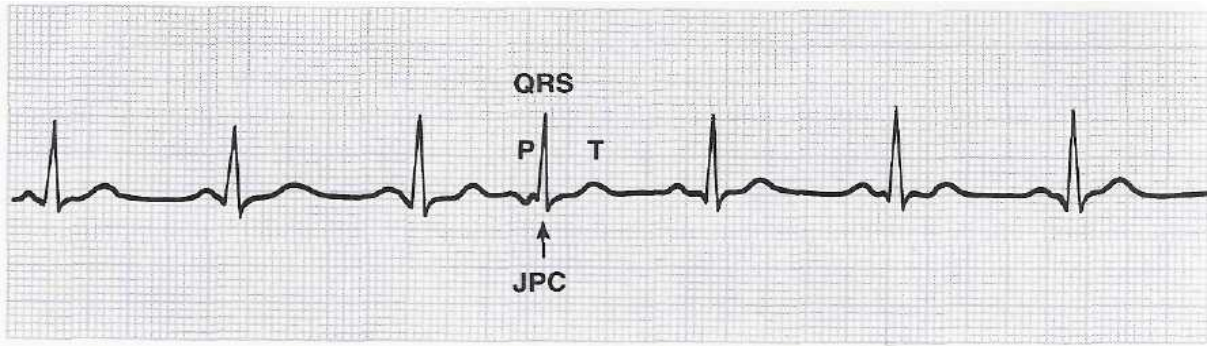
The PAC is usually conducted through the ventricular pathway in the normal manner not affecting the shape of the QRS. A pause will follow the beat, and the SA node will start a new cycle of sinus beats.

ECG for PAC



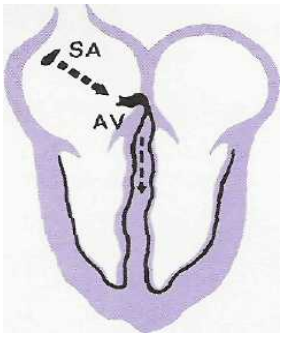
A junctional premature contraction (JPC) is an ectopic beat that arises from tissue in the junctional area and appears before the next expected sinus beat. The P will be inverted before or after the QRS, or it may be completely hidden in the QRS. The inverted P will always be very close to the QRS. A JPC can be distinguished from a PAC by the appearance of the P wave. Infrequent JPCs do not require treatment. Frequent JPCs indicate tissue irritability and may be treated with a myocardial depressant.

Junctional premature contraction

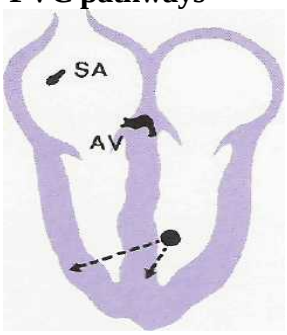


Premature ventricular contractions (PVCs) or ventricular premature beats (VPBs) occur in most myocardial infarction patients and are the most common and easily recognized rhythm disturbances seen on the ECG. They are also seen in normal persons, and may be caused by smoking, coffee, and alcohol. When pathological, they are seen most often in patients with ischemic or arteriosclerotic heart diseases.

Normal pathway

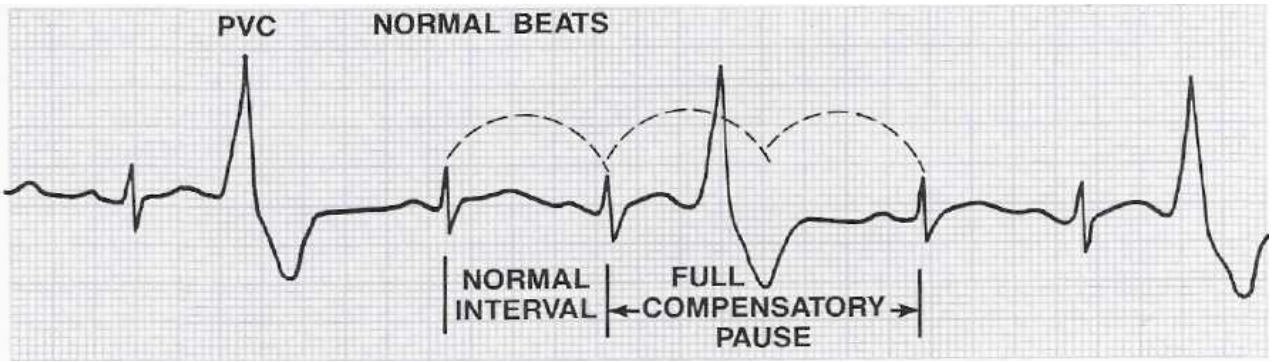


PVC pathways



Normal and PVC pathways. As the name denotes, PVCs originate in the ventricles below the AV node. Because the PVCs do not follow the normal conduction path in the ventricles, they show a bizarre QRS configuration on the ECG.

ECG for PVCs



ECG for PVCs. Notice that the PVCs come early in the cycle (premature) and are wider than the normal beat. PVCs can be identified because they:

1. Usually occur early in the cycle.
2. Are not usually preceded by a P wave.
3. Have a wide and distorted QRS.

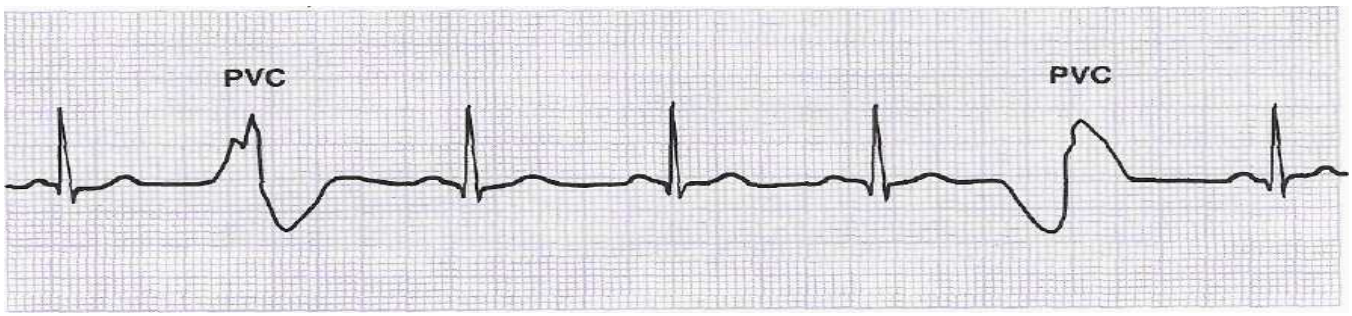
4. Have a large looping ST segment opposite in direction to that of the QRS.
5. Are usually followed by a full compensatory pause. (The interval between the R waves before and after the PVC is twice that of the normal R-R interval.)

Terms used to describe PVCs

Unifocal PVCs. Those that originate from the same site and therefore have the same configuration.

Multifocal PVCs. Those that originate from different sites and have different shapes.

ECG for multifocal PVCs

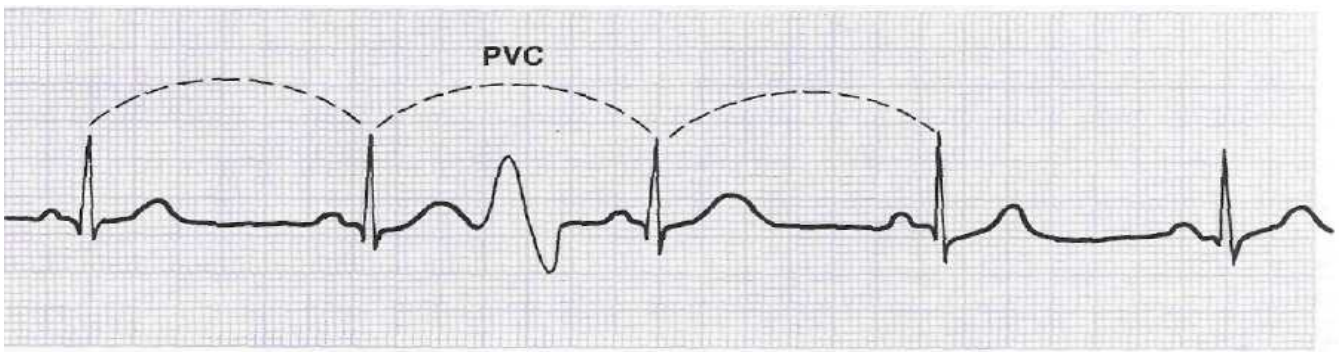


Bigeminy. Paired or coupled rhythm; a repeating pattern of two beats, with PVCs and normal beats alternating.

Trigeminy. Rhythm with a repeating pattern of three beats; the ratio of PVCs to normal beats is 2:1 or 1:2.

Interpolated PVCs. Those that fall between two normal beats without interrupting the rhythm. These PVCs are not followed by compensatory pauses.

ECG for interpolated PVC



For the patient with an infarct, PVCs are usually given vigorous treatment because they can precipitate ventricular fibrillation by occurring on a T wave. They are especially dangerous when they:
1. Occur more frequently than one in 10 beats.
2. Occur in groups of two or three.
3. Are multifocal: Several ventricular sites are irritable.

Occur on or near the T wave. At this time (the vulnerable period) in the cycle, the conduction tissue is partially repolarized and may respond in an erratic manner. Some cells respond immediately and others later, causing intermittent depolarization and triggering ventricular fibrillation.

ATRIAL FLUTTER AND FIBRILLATION

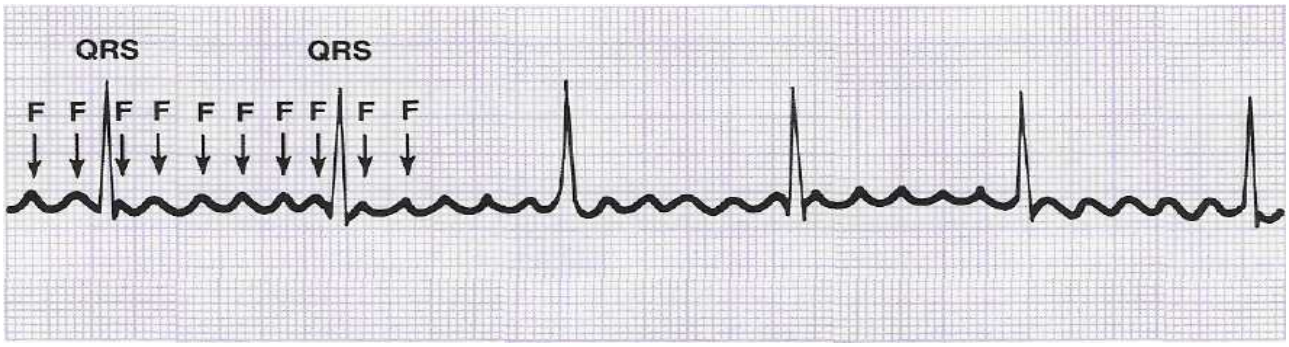
Atrial flutter is a rapid, regular firing of an irritable ectopic focus in the atrium. It is probably due to a re-entry mechanism, it usually occurs in a pathologic (arteriosclerotic or rheumatic) heart, in contrast to PAT, which may be associated with a normal heart.

The flutter ("F") waves take on a saw-toothed appearance because they are coming from a focus other than the sinus node at a very rapid rate. The atrial rate is between 250 and 350 impulses per minute. Not all of the impulses are conducted, so the ventricular rate is usually slower. The ventricular response may be regular or irregular.

Note these differences to identify types of supraventricular tachycardia:

1. The atrial rate in sinus tachycardia goes up gradually to 150/minute.
2. The atrial rate in PAT is 140-250/minute and starts abruptly.
3. The atrial rate in atrial flutter is 250-350/minute.

ECG for atrial flutter



ECG for atrial flutter. The arrows indicate the F waves that are coming from the fast ectopic focus in the atrium. Notice that not every wave stimulates a QRS complex. Since the abnormality is above the AV node, the QRS complexes that appear are normal in configuration.

Because the impulses are coming so rapidly, the AV node cannot accept and conduct each one, and therefore some degree of blockage occurs at the node. For example: If the atrial rate is 300, the ventricular rate (same as the pulse rate) may be 150. The block is thus said to be 2:1, since there are two atrial impulses per one ventricular response. In the diagram, five or six F waves precede each QRS.

Treatment of atrial flutter is indicated if the ventricular rate is sufficiently rapid to be potentially dangerous to the patient.

A fast cardiac rate is relatively ineffectual and may lead to congestive heart failure. The quickest way to slow a very fast flutter is by elective cardioversion. By using low voltage, depolarization of all heart tissue is accomplished with the electrical energy of the defibrillator paddles. The discharge is synchronized with the QRS to avoid any stimulus occurring during the vulnerable period, the T wave. Cardioversion breaks the re-entry cycle and permits the sinus node to gain control.

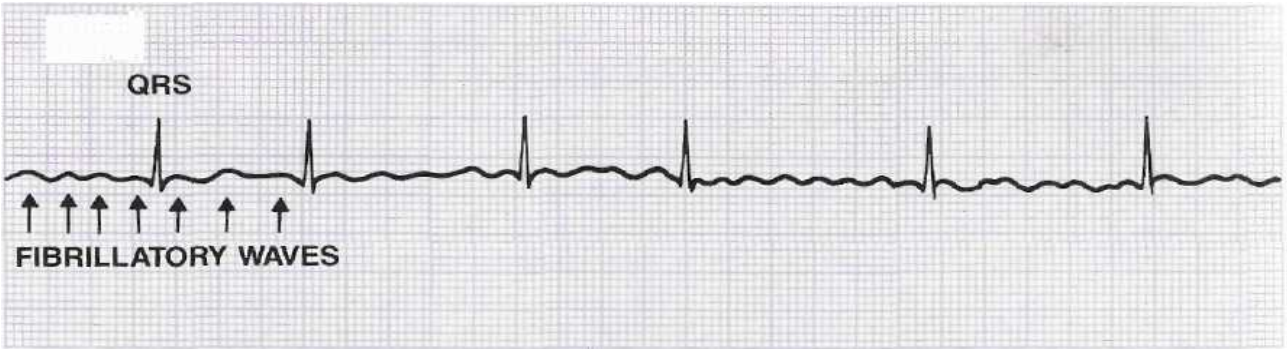
Atrial fibrillation is a very fast atrial rate arising from many ectopic foci. There is an irregular ventricular response, normal P waves are replaced by irregular rapid waves, and the total atrial configuration may resemble a wavy baseline or almost straight line.

These waves (often called fibrillatory or "f" waves) assume different shapes because different parts of the atrial tissue are depolarized in a variable, uncoordinated way. This occurs in enlarged atrial chambers often impaired by arteriosclerotic heart disease or scar tissue from surgery or infections such as rheumatic fever. Impulses rebound at various times from this uneven depolarization, producing a quivering action instead of an organized atrial contraction. A significant volume of blood which would have been contributed toward ventricular filling by the atrial “kick”/contraction is lost. The atrial component accounts for 15 to 30 per cent of the cardiac output.

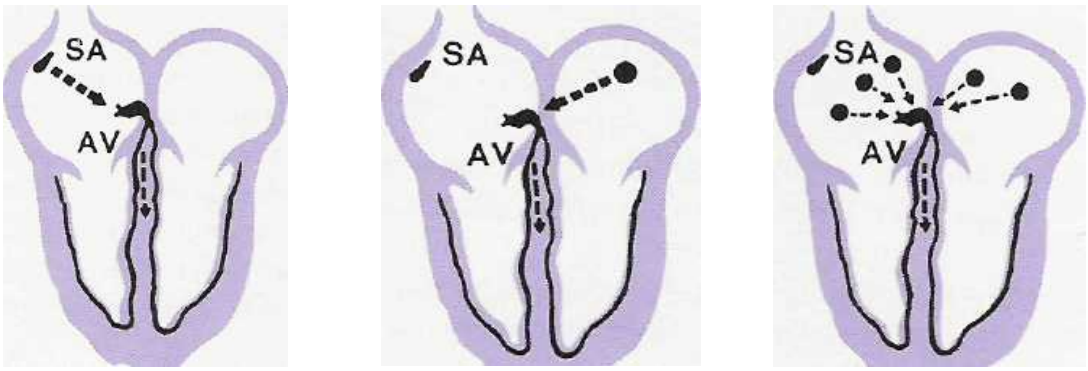
Normal pathway **Atrial fibrillation pathway**

Since P waves in atrial fibrillation are not clearly discernible, the atrial rate cannot be measured but is much faster than the ventricular rate. With no definite P waves, no PR interval can be determined. The ventricular rate may be fast or slow but will be irregular. If it becomes regular, this usually indicates a junctional pacemaker is controlling the ventricles. This is a form of AV dissociation which may result from digitalis toxicity.

ECG for atrial fibrillation



Summary of electrical pathways and ECGs in atrial arrhythmias



Normal pathway **PAC, PAT, and flutter pathway.** **Atrial fibrillation pathway.**

Only one ectopic focus is present. Many ectopic foci are present.

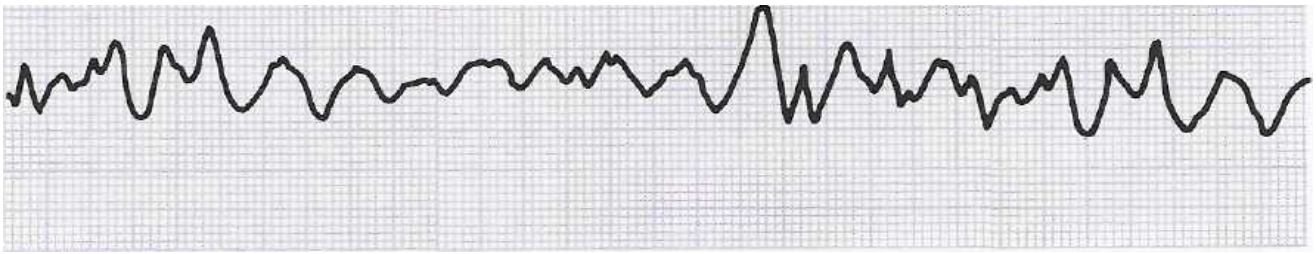
VENTRICULAR FIBRILLATION

Ventricular fibrillation. It is extremely important to be cognizant of this rhythm; therapy should be instituted immediately. If the arrhythmia is not terminated, the patient will die within minutes.

Normal and ventricular fibrillation pathways. In the fibrillating heart, it can be considered that numerous ectopic foci in the ventricles are firing erratically. Thus, there is no effective contraction of the cardiac musculature, and the patient has no pulse.

Normal pathway **Ventricular fibrillation pathways**

ECG for ventricular fibrillation

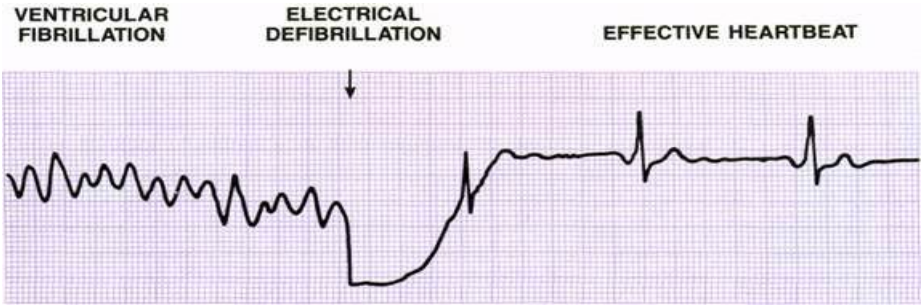


ECG for ventricular fibrillation. Notice the complete distortion and irregularity of the complexes. Since similar distortion may also be caused by the movement of the patient or the monitor wires, it is important to rule out these possibilities. If the patient is alert, or if not alert but has a pulse, the rhythm is not ventricular fibrillation.

ECG of a successful defibrillation. Clearly shown is the point at which the electrical shock stopped the fibrillating heart. After a moment, the heart began to beat in normal fashion.

Patients who have frequent episodes of ventricular tachycardia and fibrillation not responsive to drug therapy are possible candidates for an automatic implantable cardioverter-defibrillator. AICD is a new electronic device that can monitor cardiac rhythm, detect ventricular tachycardia or fibrillation, and discharge a shock. The use of this device requires careful patient evaluation, extensive emotional support, and close monitoring for complications.

ECG of a successful defibrillation



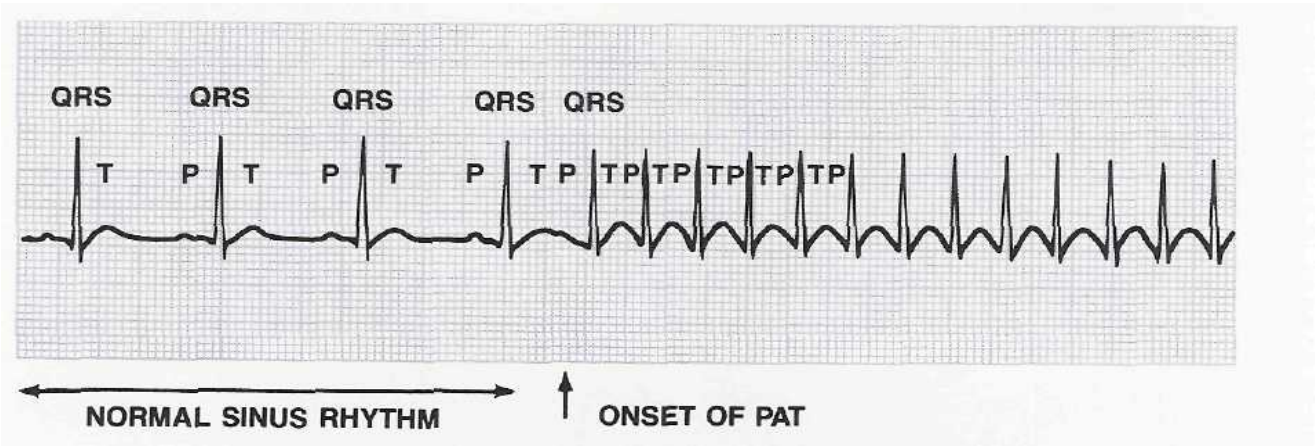
Treatment of ventricular fibrillation. If the patient is unresponsive and pulseless, call for help and begin cardiopulmonary resuscitation (CPR). At the onset of witnessed fibrillation, a precordial thump may be effective. If not, continue CPR and defibrillate as soon as possible starting with 200 joules. Defibrillation depolarizes all of the myocardial cells simultaneously and allows the SA node to resume normal conduction. Be sure the synchronizer, which is used only for elective cardioversion, is turned off during defibrillation.

Good oxygenation of the patient with intubation and/or assisted respiration is vital to successful response and correction of acidosis. The use of sodium bicarbonate should be guided by blood gas results.

PAROXYSMAL TACHYCARDIA

Paroxysmal atrial tachycardia (PAT) is an abrupt episode of tachycardia with the heart rate usually between 140 and 250 beats per minute, averaging about 170. The pacemaker site is an ectopic atrial focus. As with a PAC, the P wave may be abnormally shaped, or not seen because it is buried in the preceding T wave. The QRS appears normal. PAT may be seen in young adults with normal hearts or in individuals with organic disease. The patient frequently complains of a sudden pounding or fluttering in the chest associated with weakness or breathlessness.

ECG for PAT



The fast rate stresses the heart and increases its need for oxygen. Tachycardia may also diminish cardiac output because of shortened ventricular filling time. The heart is beating so rapidly that the ventricle does not have time to fill completely. Therefore, each beat pumps out less blood. In a relatively asymptomatic and stable patient, sedation and calming measures may be helpful. If PAT persists, the usual treatment is stimulation of the vagus nerve, which slows the heart rate. The physician may accomplish this by carotid sinus massage. Since this can produce dangerous slowing or cardiac arrest, the patient should be monitored, resuscitation equipment should be readily available and an IV line established.

Other measures that stimulate the vagus nerve include: vomiting, stimulating the anal sphincter with a rectal thermometer or tube, and applying pressure to the eyeball. These measures are not suggested as therapy but explain some cardiac responses to such activities. A cough or Valsalva maneuver will increase intrathoracic pressure, decrease venous return, raise the blood pressure, and possibly slow the pulse. A Valsalva maneuver consists of tightening abdominal muscles while holding the breath. This is similar to the activity of bowel elimination and causes indirect vagal stimulation.

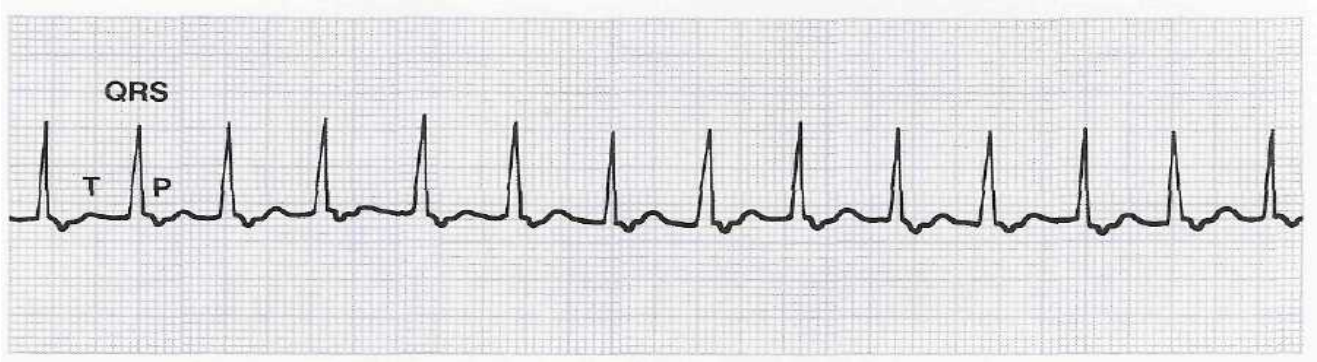
When the heart continues to beat rapidly for a period of time, or if the patient becomes symptomatic, a synchronized electrical shock (cardioversion) can be used for immediate conversion.

Junctional tachycardia is a junctional rhythm with a rate of 100 – 180 beats per minute.

This arrhythmia may be difficult to distinguish from sinus or atrial tachycardia. Whenever the origin of the tachycardia cannot be determined, and the QRS complex is of normal configuration, the term **supraventricular tachycardia** is used (often called SVT).

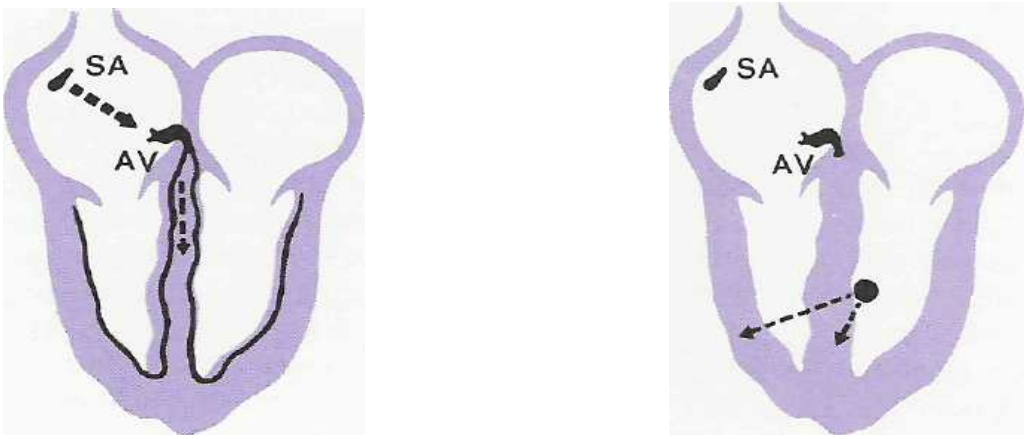
As with other fast-rate arrhythmias, this inefficient rhythm puts stress on the heart and may cause angina, congestive heart failure, or other dangerous conditions. Treatment would be similar to that used for atrial tachycardias. Digitalis toxicity may be a possible cause.

Junctional tachycardia



Ventricular tachycardia. This dreaded complication of a myocardial infarction may be defined as a series of multiple (three or more), consecutive PVCs occurring at a rate usually between 150 and 200 per minute. Ventricular tachycardia is very dangerous because it leads to reduced cardiac output and, many times, to ventricular fibrillation.

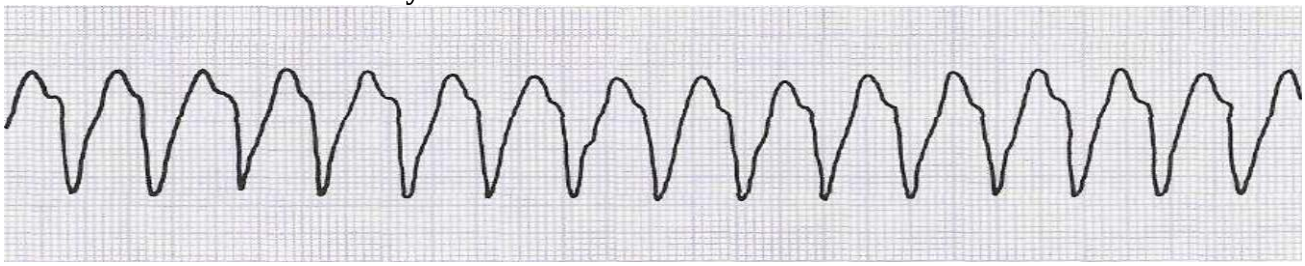
Normal pathway **Ventricular tachycardia pathway**



Normal and ventricular tachycardia pathways. These are the same sketches used to illustrate the PVC pathways since ventricular tachycardia can be considered as a series of PVCs. Like the PVCs, the tachycardia shows a bizarre configuration on the ECG.

ECG for ventricular tachycardia. Notice that the rate is fast and that the QRS is wide. If the ventricular rate is not too fast, independent P waves may sometimes be visible in the QRS complex. (P waves are not seen in this example).

ECG for ventricular tachycardia



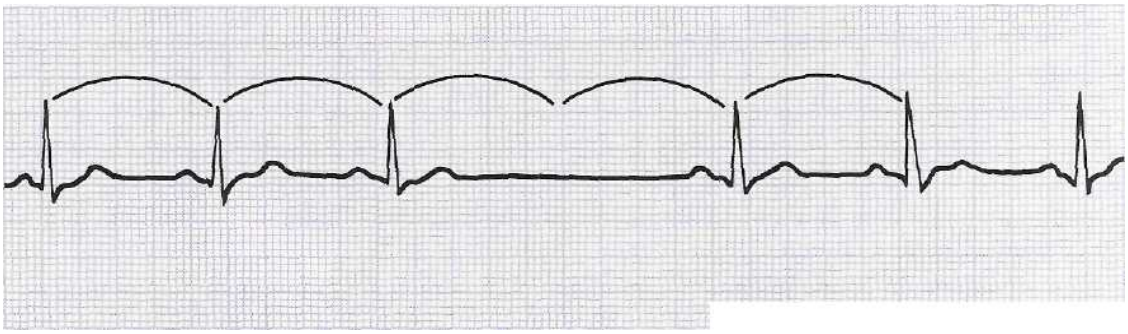
***Theme 15*. ECG Examination of the Patients with Infringement of a Conductive Function. Basis of Realization of Electroimpulse Therapy.**

Transmission of the impulse at any part of the heart conduction system. ECG- properties of sinoatrial and intraatrial block. Classification and ECG-properties of atrioventricular block. Attacks Morgagni-Adams-Stokes, courses, and clinical signs. Intraventricular block: right and left bundle branch block. Indications to realization and rules of performance of electroimpulse therapy.

Sinus block occurs when a beat is not transmitted out of the SA node. No R QRS, or T is present at the cycle interval for one or more beats.

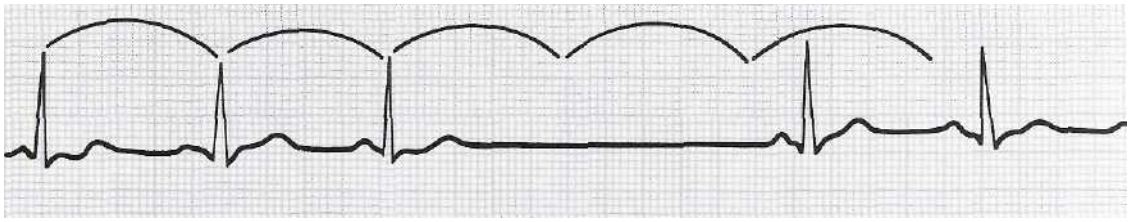
Sinus block

Sinus arrest ***occurs when the SA node fails to send out an impulse for a period of time. This interval between beats is not a multiple of the heartbeat cycle length.***



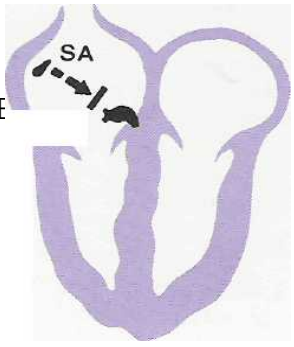
If either event produces symptoms of hemodynamic insufficiency, treatment (atropine or an artificial pacemaker) is indicated.

Sinus arrest



AV blocks. In this condition the AV node is diseased and has difficulty conducting the P waves into the ventricles. The most common causes are arteriosclerosis and myocardial infarction. Digitalis toxicity may also produce such blocks.

BLOCK OF AV NODE

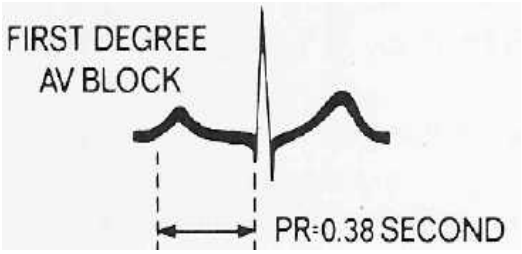


AV block. Scarring, inflammation, or edema prevents or slows transmission of the electrical impulse by the AV node. The degree of block varies from very slight to complete, and is classified as first, second, or third degree block.

First degree AV block. Because the tissue around the AV node is abnormal, the impulse takes longer to traverse the area.

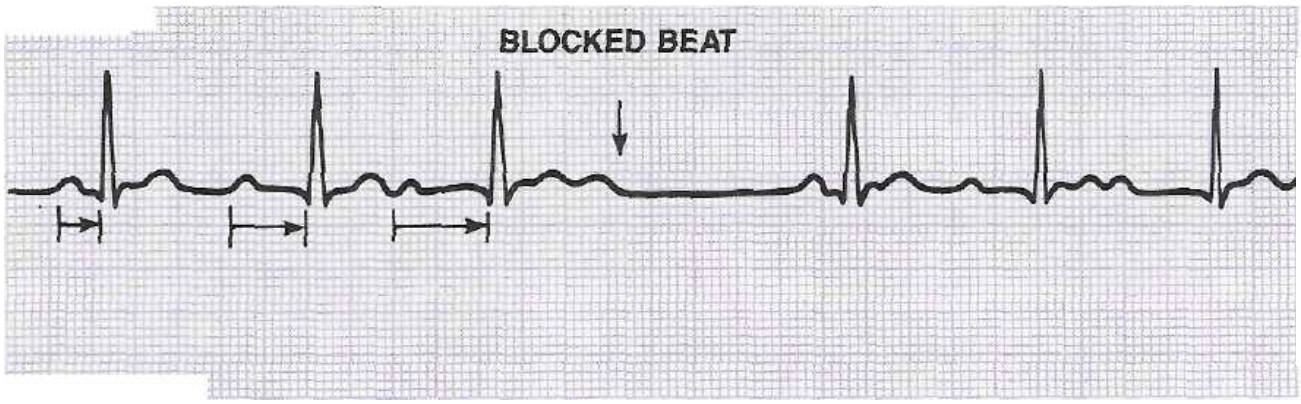
This is reflected by an increased length in the PR interval on the ECG. (The PR interval represents the impulse going through the atrium and the area of the AV node). In contrast to second and third degree blocks, all P waves in first degree block penetrate the ventricles to form QRS complexes. In normal conduction, the PR interval is not over 0.20 second. A PR interval such as the one shown (0.38 second) is an indication of first degree block.

First degree block does not diminish cardiac output. However, it is an indicator of possible damage to junctional tissue or of drug effect, especially from digitalis. Careful observation is indicated for possible progression to higher degrees of block.



Second degree AV block exists when some of the P waves are conducted to the ventricles and others are blocked at the AV node. This condition is divided into two classifications: Mobitz I (Wenckebach) and Mobitz II, or Type I and Type II.

Mobitz I (Wenckebach) second degree block



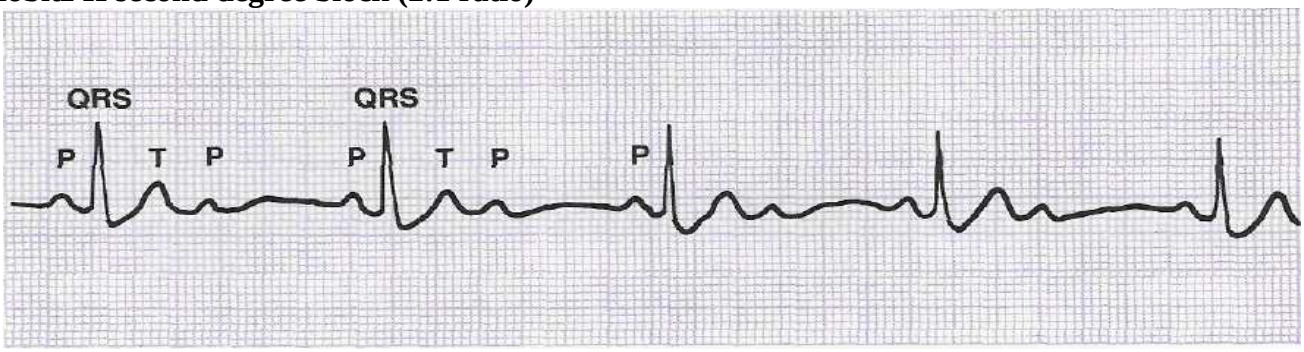
Mobitz I (Wenckebach). The ischemic or drug-affected AV node requires a progressively longer interval of time to transmit each beat until a beat fails to be conducted. When the next impulse arrives, the rested AV node is able to transmit the beat in a shorter time, but the PR interval again lengthens, and the cycle repeats.

On the ECG, this results in a progressive lengthening of the PR interval until a beat is blocked (P not followed by a QRS), and then the cycle repeats. The rhythm (R-R interval) is irregular, and there are more P waves than QRS complexes.

Wenckebach block may be caused by digitalis or MI, especially one involving the inferior wall. It is generally transient and reversible.

In Mobitz II, some beats are conducted and others are not. Conducted beats have a consistent PR interval. In blocked beats, there is a P wave not followed by a QRS complex. One type of pattern shows a specific ratio of blocked beats such as 2:1, 3:1, or 4:1. In such cases, the R-R interval will be regular.

Mobitz II second degree block (2:1 ratio)

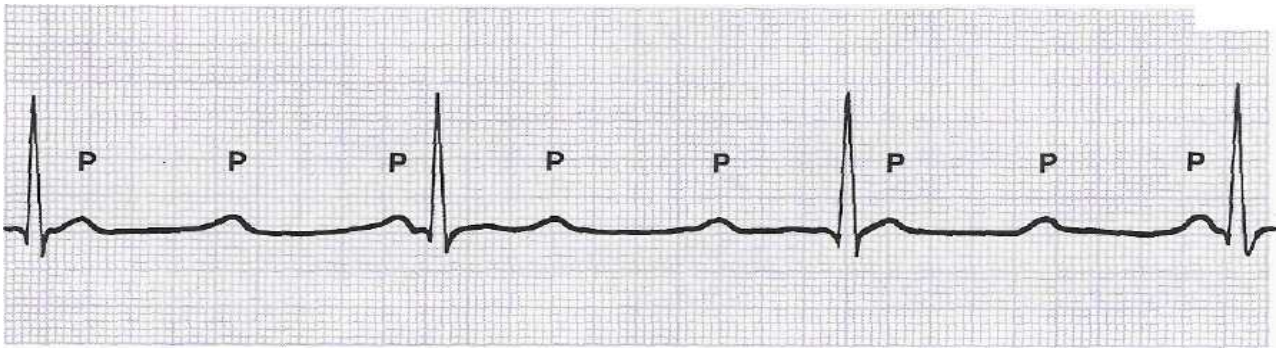


Third degree AV block is also called complete heart block. In this arrhythmia, no atrial impulses (P waves) activate the ventricles. The QRS originates from a junctional or ventricular pacemaker site. Therefore, the P waves and QRS complexes occur independently.

Both the P waves and QRS complexes occur regularly, but there is no relationship between them. The PR interval varies and some P waves may be partly obscured by QRS complexes.

Complete heart block may be caused by inflammation, scarring, myocardial infarction, or drugs such as digitalis. The pulse rate is usually slow because of the inherent rate of junctional or ventricular pacemaker sites. Since these secondary pacemakers are not dependable, the treatment of choice is an artificial pacemaker.

Third degree AV block



***Theme 16*. Questioning and Inspection of the Patients with the Disorders of Digestive System. Inspection and Surface Tentative Palpation of Abdomen.**

Questioning and detail complaints of the patient with the disorders of digestive system. The specific character of anamnesis morbi and vitae is important during inquiry of these patients. Visual examination of the abdomen (shape of the abdomen, symmetrical shape of the both halves, umbilicus, participation of the abdomen in the act of respiration, the state of the skin, development of the subcutaneous veins). Topography of the anterior abdominal wall. Objective, technique of superficial palpation of the abdomen (abdominal wall resistance, muscular strain, tenderness, signs of peritoneum irritation, hernias (umbilical, inguinal, linea alba). Determine presence of ascites (inspection of the abdomen, the sign of fluid fluctuation, abdomen percussion).

The contents: Patients with the disorders of digestive system complain of poor appetite, perverted taste, regurgitation, heartburn, nausea, vomiting, epigastric pain, haematemesis and the feeling of overfilled stomach after meals. Determining the specific character of each symptom is important during inquiry of the patient.

Deranged (poor or increased) appetite occurs in infectious diseases, metabolic disorders, etc. Poor appetite or its complete absence (anorexia) is usually characteristic of gastric cancer. Appetite often increases in peptic ulcer, especially in duodenal ulcer. Appetite is perverted in pregnant women and in persons suffering from achlorhydria.

Bitter **belching** indicates intensive degradation of proteins. Belching is characteristic of stenosed pylorus with great distention of the stomach and significant congestion in it. Acid regurgitation is usually associated with hypersecretion of gastric juice and occurs mostly during pain attacks in ulcer.

Pyrosis is otherwise known as **heartburn**, i.e. burning pain in the epigastric and retrosternal region. Heartburn arises in gastro-esophageal reflux, mostly the presence of gastric hyperacidity in various diseases of the alimentary tract (e.g. peptic ulcer or cholecystitis), hiatus hernia, and sometimes in pregnancy. Heartburn in healthy subjects can be due to hypersensitivity to some foods.

Nausea, the reflectory act associated with irritation of the vagus nerve, is an indefinite feeling of sickness and sensation of compression in the epigastrium. Nausea is often attended by pallidness of the skin, general weakness, giddiness, sweating, and salivation; fall in the arterial pressure, cold in the limbs, and sometimes-semisyncopal state. Nausea often (but not necessarily) precedes vomiting. Nausea may develop without any connection with diseases of the stomach, e.g. in toxaemia of pregnancy, renal failure, deranged cerebral circulation, and sometimes in healthy people. Some diseases of the stomach are attended by nausea, e.g. acute and chronic gastritis or cancer of the stomach.

Vomiting (emesis) occurs due to stimulation of the vomiting centre. This is a complicated reflex through the esophagus, larynx and the mouth (sometimes through the nose as well). Vomiting is an important symptom of many diseases of the stomach. Vomiting of gastric etiology is caused by stimulation of receptors in the gastric mucosa by inflammatory processes (acute or chronic gastritis). If the patient complains of vomiting, the physician should inquire about the time when the vomiting occurred, possible connections with meals, association with pain, the amount and character of the vomited material. Hyperacid vomiting in the morning indicates nocturnal hypersecretion of the stomach. Vomiting occurring 10-15 minutes after meals suggests ulcer or cancer of the cardial part of the stomach, or acute gastritis. If vomiting occurs 2-3 hours after meals (during intense digestion) it may indicate ulcer or cancer of the stomach body. In the presence of ulcer of the pylorus or duodenum, vomiting occurs 4-6 hours after meals.

Pain is the leading symptom in diseases of the stomach. Epigastric pain may develop in diseases of abdominal organs (sometimes of organs located outside the abdomen) by the viscero-visceral reflex (acute appendicitis, myocardial infarction, affection of the diaphragmatic pleura, etc). In order to locate correctly the source of pain, the physician should ask the patient (1) to show exactly the site of pain; (2) to characterize the pain which may be periodical or paroxysmal (at certain time of the day); permanent or seasonal (in spring or autumn); (3) to describe the connection (if any) between pain and meals, the quality of food and its consistency; (4) to indicate possible radiation of pain (into the back, shoulder blade, behind the sternum, left hypochondrium); (5) to describe conditions under which pain lessens (after vomiting, after

taking food or baking soda, after applying hot-water bottle or taking spasmolytics); (6) to describe possible connections between pain and physical strain or strong emotions. Intensity and character of pain are also important diagnostically. The pain may be dull, stabbing, cutting, etc.

Haemorrhage is a very important symptom. It can be manifested by vomiting of blood (haematemesis) or by black tarry stools (melaena). Gastric haemorrhage is usually manifested by the presence of blood in the vomitus. The colour of the vomitus depends on the time during which the blood is present in the stomach. If the blood was in the stomach for a long time, the blood reacts with hydrochloric acid of the gastric juice to form haematin hydrochloride. The vomitus looks like coffee grounds. If haemorrhage is profuse (damage to a large vessel) the vomitus contains much scarlet (unaltered) blood. Haematemesis occurs in peptic ulcer, cancer, and polyps, in erosive gastritis, rarely in sarcoma, tuberculosis and syphilis of the stomach, and in varicosity of the esophageal veins. Tarry stools are not an obligatory sign of gastric haemorrhage.

When collecting **anamnesis**, the patient should be asked about his nutrition. It is important to establish if meals are regular because taking food at random is an important factor in the etiology of gastric diseases. Food quality is as important as its amount taken during one meal. Mastication of food matters as well. Conditions of rest and work, and possible occupational hazards should be established. Abuse of alcohol and smoking are important factors in the etiology of gastric diseases. It is very important to find out if the patient's condition has undergone some changes during recent time (e.g. loss of weight, anemia, blood vomiting, or tarry stools). Gastro-intestinal diseases of the past, surgical intervention on the abdominal organs, long medication with preparations irritating the stomach mucosa (acetylsalycilic acid, sodium salycilate, steroid hormones, potassium chloride, etc.) are also very important.

Physical Examination. During general inspection of the patient the physician may assess poor nutrition of the patient (cachexia), which is characteristic of stomach cancer and untreated benign pyloric stenosis. Pale skin is observed after gastric haemorrhage. Patients with uncomplicated peptic ulcer look practically healthy.

Next stage is inspection of the abdomen. Physical examination of the abdominal organs is performed in two positions: lying and upright.

The abdominal cavity is divided into several areas. Two parallel horizontal lines, one of which joins the costal arches, the other upper axes of the iliac bones, divide the abdomen into three portions: upper, epigastric region; median, mesogastric region; lower, hypogastric. Two parallel vertical lines, going through the external edges of the abdominal rectal muscle, divide the abdomen into the right and left hypochondriac and epigastric regions, mesogastric region is divided into left and right iliac regions and umbilical region, which is located between them; the hypogastric region is divided into left and right inguinal regions and suprapubic region located between them.

The abdomen is examined in the following way: the shape of the abdomen, symmetrical shape of the both halves, umbilicus, the integument of the abdominal wall, participation of the abdomen in the act of respiration.

Shape of the abdomen is determined by the constitution. In pathologic conditions, the shape of the abdomen may change: first, it may enlarge or diminish, the halves being symmetrical; second, it may be pulled in or stuck out in some regions. General enlargement of the abdomen is seen in pregnant. Pathological enlargement of the abdomen is observed in ascites, obesity, flatulence (accumulation of gases in the intestine). Asymmetrical sticking out, when only one area is prominent, is observed at enlargement of separate organs, e.g. liver, spleen, in tumors from these organs and other abdominal organs (stomach, pancreas, intestine). In case of general retraction, the abdomen is boat-shaped. This is observed in alimentary dystrophy, chronic dysentery, narrowing of the pylorus, cancer cachexia, prolonged diarrhea and vomiting. Retraction of separate areas of abdomen is rare. Thus, retraction in the epigastric area may be seen in gastroptosis or splanchnoptosis.

The state of the abdominal integument. The normal skin of the abdomen is pale rose, smooth, dull. It is necessary to pay attention to the rashes, hernias, development of the subcutaneous veins, separate pulsations, scars, and pigmentations.

Doing inspection it is necessary to pay attention to the movement of the anterior abdominal wall which may be associated with stomach peristalsis, intestine, pulsation of the aorta, right ventricle, liver as well as respiratory excursions.

Palpation of the abdomen. Palpation is the main method of physical examination in diagnosis of diseases of the abdominal organs. Surface and deep palpation are used.

Surface tentative palpation. The purpose of surface palpation is to determine the presence of tenderness of the whole abdominal wall or its separate portions, the tone of the abdominal muscles, presence of large tumors and enlargement of the abdominal organs, presence of tumors in the abdominal wall, edema of the abdominal wall, presence of hernias.

The physician assumes his position by the bedside as described above and places his right hand flat on the abdomen of the patient (the fingers may be slightly flexed) to examine carefully and gradually the entire abdomen without trying to penetrate the deep parts of the abdomen. By this examination the physician should establish the strain of the prelum, its tenderness, and location of the painful site. The left inguinal area should be examined first, provided the patient does not complain of pain in this region. Palpation is then continued by examining symmetrical points of the abdomen on its left and right sides to end in the epigastric region. If the patient complains of pain in the left inguinal area, the sequence of palpation should be so changed that the least painful site on the anterior abdomen should first be examined. The physician should simultaneously assess the condition of the abdominal skin and subcutaneous connective tissue, the strain of the abdominal wall, the zones of superficial and deeper painful areas to locate them accurately. Hernial separation of muscles and protrusions, and also other anatomical changes should be revealed, if any. Resistance and marked strain of muscles of the abdominal wall are usually palpated over the organ affected by inflammation, especially so if the peritoneum is involved. In the presence of acute inflammation of the peritoneum (local inflammation included, e.g. in appendicitis, cholecystitis, and the like), local pressure causes strong pain but it becomes even more severe when the pressure is released Shchetkin-Blumberg symptom). In the presence of pronounced enlargement of the parenchymatous organs, in strained abdomen or intestinal loops, and also in the presence of large tumors, even surface palpation can give much diagnostic information.

The sign of fluid fluctuation helps to recognize ascites. The palm of the left hand is placed on the lateral surface of the right half of the abdomen, short pushes are made by the finders of the right hand along the lateral surface of the abdomen. When large volumes of free fluid are present in the abdominal cavity, the left hand feels clearly its pushes simultaneously with the movements of the right hand fingers.

***Theme 17*. Deep Sliding Methodical Palpation of the Abdominal Organs.**

The role of Ukrainian scientists in creation of the method about palpation of the abdominal organs. The topography of the abdominal organs. Sequence of palpation of the abdominal organs according to Obraztsov and Strazhesko. Normal signs of intestine (sigmoid colon, cecum, the ascending and descending colons, transverse colon). Methods of definition of the bottom border of a stomach (palpation of the greater curvature of the stomach, percussion of the inferior border of the stomach, stethacoustic palpation, by splashing sound). The rules of palpation of the pyloric portion. Percussion: determining the size and borders of the liver according to Obraztsov and Kurlov. The causes of enlargement and diminutive the size of the liver. Technique of deep sliding methodical palpation of the liver. Characteristics of palpated of the lower border of the liver in normal and pathological. Technique of percussion of spleen and main causes of enlargement of its. The rules of palpation of the spleen. Diagnostic significance of Pasternatsky′s sign. Technique of the kidneys palpation in supine and upright positions.

The contents: Percussion is used to determine the borders, size and configuration of the liver. The superior and inferior borders of the liver are outlined. Two superior borders of liver dullness are distinguished: relative dullness, which is the true upper border of the liver, and the absolute dullness, i.e. the upper border of that part of the anterior surface of the liver which is directly adjacent to the chest and is not covered by the lungs. But in practice only absolute dullness is determined, because the upper border of the liver is covered by the lung and the percussion sphere dose not reach it. The upper border of absolute dullness corresponds to the lower border of the right lung. Deep palpation of the abdomen as an

objective methods of investigation of the abdominal organs was suggested by professor V.P. Obraztsov and improved by proffers N.D. Strazhesko and V.H. Vasilenko.

The purpose of deep palpation is to study the topography of intestine, stomach, liver, spleen, kidneys, to determine the size, shape. Location of these organs, their motility, tenderness, consistence, as well as the properties of the wall and the character of the content (for the hollow organs) and palpate the tumors in the abdominal cavity.

The palpation technique includes the following four steps. *First*: proper positioning of the physician's hands. The right hand is placed flat on the anterior abdominal wall, perpendicular to the axis of the examined part or the edge of the examined organ. *Second*: formation of a skin fold to facilitate further movements of the examining hand. *Third*: moving the hand inside the abdomen. Deep palpation is when the fingers are moved gradually, with each expiration, into the abdomen when the abdominal wall is relaxed. The examining hand thus reaches the posterior wall of the abdomen or the underlying organ. *Fourth*: sliding movement of the fingertips in the direction perpendicular to the transverse axis of the examined organ. The organ is pressed against the posterior wall and the examining fingers continue moving over the examined intestine or the stomach curvature. Depending on the position of the organ, the sliding movement should be either from inside, in the outward direction (the sigmoid, caecum) or in the downward direction (the stomach, transverse colon); the movements should then be more oblique in accordance with the deviation of the organ from the horizontal or vertical course. The examining hand should always move together with the skin and not over its surface.

By palpating the intestine, the physician establishes its localization, mobility, tenderness, consistency, diameter, the condition of the surface, the absence or presence of rumbling sounds during palpation.

The **sigmoid** is palpated from top right to medial left, downward and laterally, perpendicularly to the axis of the intestine which runs obliquely in the left iliac space at the border of median and the outer third of the linea umbilico-iliacae. Palpation is carried out by four fingers, placed together and slightly flexed, or by the ulnar edge of the right little finger. The fingers are immersed medially of the expected position of the intestine and as soon as the posterior wall of the abdomen is reached, the fingers slide along the intestine in the given direction, i.e. laterally and downward. The intestine is pressed against the posterior wall and first slides along it (to the extent allowed by the mesenteric length) but later it slips from under the examining fingers. The sigmoid can be palpated by the described technique in 90-95 per cent of cases. Normally the sigmoid can be palpated over the length of 20-25 cm as a smooth firm cylinder, its thickness being that of a thumb or an index finger; the sigmoid is painless to palpation, it does not produce rumbling sounds, its peristalsis is rather flaccid and infrequent. The sigmoid can be displaced 3-5 cm to either side.

The **caecum** is palpated by the same technique, except that the direction is different. Since the caecum is situated at the border of the median and lateral third of the umbilico-iliac line (5 cm by the iliac spine), the palpation is carried along this line or parallel to it. A normal caecum can be palpated in 80-85 per cent of cases as a moderately strained cylinder (widening to the round bottom), 2-3 cm in diameter; when pressed upon, it rumbles. Palpation is painless. It reveals a certain passive mobility of the caecum (to 2-3 cm).

The **ascending** and **descending colons** are palpated by two hands. The left hand is placed under the left and then the right lumbar side, while the fingers of the right hand press on the anterior wall of the abdominal cavity until the examiner feels his right and left hands meet. The examining fingers then slide laterally, perpendicularly to the axis of the intestine.

The **transverse colon** is palpated by four fingers of the right hand held together and slightly flexed. Bimanual palpation can also be used. Since the position of the transverse colon is unstable, it is useful first to determine the lower border of the stomach, and only then to search for the colon some 2-3 cm below this border. The right hand (or both hands) is placed on the sides of the linea alba and the skin is moved slightly upwards. The examining hand is then immersed gradually during relaxation of the prelum at expiration until the posterior wall of the abdomen is felt. Once the posterior wall is reached, the examining hand should slide down to feel the intestine: this is an arching (transverse) cylinder of moderate density (2-2.5 cm thick), easily movable up and down, painless and silent. If the intestine is impalpable in this region, the same technique should be used to examine the lower and lateral regions, the position of the palpating hands being changed accordingly. Normal transverse colon can be palpated in 60-70 per cent of cases.

Methods of definition of the bottom border of a **stomach**.

Deep palpation of the stomach. The examiner pulls up the skin on the abdomen and presses carefully the anterior wall of the abdomen to penetrate the depth until the examining fingers reach the posterior wall. When pressed against the posterior wall of the abdomen, the stomach slips from under the examining fingers. The greater curvature and the pylorus can best of all be examined by this method. The greater curvature can be examined by deep sliding palpation in 50-60 per cent and the pylorus in 20-25 per cent of healthy subjects. The greater curvature is found to either side of the median line, 2-3 cm above the nave.

Percussion is used to determine the inferior border of the stomach. Provided professional skill is high, the inferior border of the stomach can be outlined by light percussion by, differentiating between gastric and intestinal tympany.

Better results are obtained on direct percussion with one finger according to Obraztsov (technique of *percutory palpation*). It consists in determining the location of the lower border of the stomach according to a splashing sound. A splashing sound is produced by quick tapping with the finger of the right hand on the abdominal wall, the tapping finger should do gathering movements downwards and inwards. A splashing sound appears at the moment of tapping. The fingers gradually move down beginning from the epigastric area. The place, where the splashing sound disappears, corresponds to the border of the stomach. In healthy moderately nourished men the border is 3-4 cm above the umbilicus along the median line, in women 1-2 cm below.

Auscultation of the stomach is used together with palpation of the stomach to outline its inferior border. *Stethacoustic palpation* is performed as follows: a stethoscope is placed beneath the left costal arch, below the Traube's face. The examiner rubs the abdominal wall overlying the stomach by the finger and gradually moves the finger away from the stethoscope bell. As during as the finger rubs the skin overlying the stomach, the physician hears the friction, but when the finger moves outside the stomach borders, the found disappears. This method is very simple but the findings are sometimes inaccurate.

Palpation of the pyloric portion: the palpating fingers are placed in the triangle formed by the lower border of he liver, median line, and transverse line going 3-4 cm above the umbilicus in the area of the right rectal muscle and are moved on the left of this space downward and to the right. A thin moderately movable cylinder is felt, its density varies (from dense to not detected). Sliding along this cylinder may produce borborygmus.

Determination of the lower border of absolute liver dullness (according to Obraztsov and Strazhesko) should be begun from the right part of the abdomen along the right anterior axillary line with the patient in the horizontal position. The plessimeter finger is placed parallel to the expected inferior border of the liver. As the plessimeter finger is then moved upwards, tympany is followed by absolute dullness. The point of disappearance of tympany is marked in each pitied line (right midclavicular, right parasternal, and anterior median line).

When determining the left border of liver dullness, the plessimeter finger is placed perpendicularly to the edge of the left costal arch, at the level of the 8-9th ribs, and percussion is carried out to the right, directly liver the edge of the costal arch, to the point where tympany changes to dullness.

Normally the inferior border of absolute dullness of a lying patient with normosthenic chest passes at the 10th rib in the right anterior axillary line, it the superior edge of the right arch in the midclavicular line, 2 cm below the interior edge of the right costal arch in the right parasternal line, and 5-6 cm away from the inferior edge of the xiphoid process (at the border of the upper third of the distance from the base of the xiphoid process to the navel) in the anterior median line; on the left the border does not extend beyond the left parasternal line.

The size of the liver can be determined according to M.G. Kurlov. First, the percussion is done downward the right medioclavicular line up to the liver dullness. This place is marked on the skin. After that percussion is done along the same line from the level of the umbilicus upwards until a dull sound appears, this point is also marked on the skin with ink. The distance between the points (normally 9±1-2 cm) is the size of the right lobe of the liver. The third point is marked at the base of the xiphoid process along the median line. Then percussion is done upward from the umbilicus up to the point of dullness,

which is also marked on the skin. The distance between the points is the size of the liver in its middle portion (approximately 8±1-2 cm). At last, percussion is done from point 3 along the left costal arch until a tympanic sound appears, the fifth point is also marked. The distance between these points is 1±1-2 cm (the length of the left half of the liver).

Palpation on the liver. The palm of the left finger presses the right costal arch, the palm of the right finger is placed flat on the right side of the abdomen the fingers are parallel to the direction of the lower border of the liver below its supposed level which can be detected by percussion as well as by sliding of the palpating fingers from the lower edge of the costal arch downwards until the consistence becomes soft. The tips of the fingers penetrate 1-2 cm in depth, them without removing the fingers, the patient is asked to breathe deeply and slowly. The edge of the liver, making respiratory excursions, surrounds the palpating fingers, this allows to have an idea about its properties. In 80% of healthy persons the edge of the liver is rounded, thin, elastic, insensitive, and is felt on the costal arch along the medioclavicular line.

The enlargement of the liver often results from various pathological processes (hepatitis, cirrhosis, cancer, echinococcus, blood congestion in heart failure).

Percussion of the spleen allows determining the size of the organ. Only the lower 2/3 of the organ adjacent to the chest wall can be percussed. Low percussion is used, it produces a dull sound over the spleen. As the spleen borders on air-containing organs, even minute increase of percussion force involves them in the percussion sphere and a tympanic sound is added to the dull percussion sound. Percussion is better performed in a vertical position on in right decubitus along the middle axillary line. The patient is asked to breathe normally. Dullness, corresponding to the location of the spleen, occupies the area of the 9-10th ribs; it does not cross the anterior axillary line. The long axis of the spleen is 5-6 cm, the width of the spleen dullness is 4-8 cm.

Spleen palpation is done in supine and right diagonal position. The patient is supine. The palm of the right hand presses the left costal arch, the left palm is flat on the upper portion of the left half of the abdomen below the left hypochondrium. The fingers are slightly flexed and directed parallel the costal arch. The palpating fingers penetrate 1-2 cm in depth, then the patient is asked to breathe slowly and deeply, the fingers remain still. If the border of the spleen is detected, it suggests its enlargement. Moderate enlargement is observed in acute infections, syphilis, cirrhosis of the liver, bacterial endocarditis, lymphogranulomatosis, hemolytic jaundice.

The kidneys should be palpated in the lying and standing position. When the patient is in the horizontal position this kidneys are better palpated because the strain of the prelum is absent. But the movable kidney can be palpated in the standing patient because it hangs by gravity and is displaced downward by the pressure of the low diaphragm.

During palpation of the patient in the lying position his legs, should be stretched and the head placed on a low pillow; the prelum is relaxed and the arms are free placed on the chest. The physician should assume his position by the right side of the patient with, his left hand under the patient’s loin, slightly below the12ribs so that the finger tips are near the spinal column. During palpation of the left kidney, the physician's hand should be moved further, beyond the vertebral column, to reach the left part of the lumbar region. The right hand should be placed on the abdomen, slightly below the corresponding costal arch, perpendicularly to it and somewhat outwardly of the rectus abdominis muscles. The patient is asked to relax the abdominal muscles as much as possible and breathe deeply and regularly. The physician's right hand should press deeper with each expiration to reach the posterior abdominal wall, while the left hand presses the lumbar region to meet the fingers of the right hand. When the examining hands are as close to each other as possible, the patient should be asked to breathe deeply by "the abdomen" without straining the prelum. The lower pole of the kidney (if it is slightly descended or enlarged) descends still further to reach the fingers of the right hand. As the physician feels the passing kidney, he presses it slightly toward the posterior abdominal wall and makes his fingers slide over the anterior surface of the kidney bypassing its lower pole. If ptosis of the kidney is considerable, both poles and the entire anterior surface of the kidney can be palpated. The physician should assess the shape, size, surface (smooth or tuberous), tenderness, mobility, and consistency of the kidneys. Bimanual palpation of the kidney can also be done with the patient lying on his side. In healthy persons the kidneys are bean-

shaped, the borders are rounded, they are dense, elastic. Enlarged kidneys are observed in hydronephrosis, tuberculosis, and tumors.

A method for examination of the kidneys is tapping. The physician places his left hand on the patient's loin and using his right hand taps with a moderate force on the right hand overlying the kidney region on the loin. If the patient feels pain, the symptom is positive (Pasternatsky's symptom). This symptom is also positive in nephrolithiasis, inflammation of the pelvis, paranephritis.

Literature

1. Internal diseases an introductory course. - Vasilenko V., Grebenev A. - M.: Mir Publishers, 1990. - 647 p.
2. Propedeutics to internal medicine. Part 1.-Vinnytsya: NOVA KNYHA, 2006.-424 p.
3. Propedeutics to internal medicine. Part 2.-Vinnytsya: NOVA KNYHA, 2007.-264 p.
4. Introduction to the course of internal diseases. Book 1. Diagnosis: [Textbook/Zh.D. Semidotskaya, O.S. Bilchenko, et al.].-Kharkiv: KSMU, 2005. -312p.
5. Case report: Manual for the 2 – 3 year students of medical universities (Zaporozhye State Medical University). Zaporozhye, 2006.
6. Electrocardiography for II-III years students of the medical faculty: Manual for II-III-year students of the medical universities (Zaporozhye State Medical University). Zaporozhye, 2006.
7. Clinical tasks in the propedeutics of internal diseases for II-III years students of the medical faculty: Manual for II-III - year students of the medical universities (Zaporozhye State Medical University). Zaporozhye, 2006.
8. The general care of the patients in the therapeutic hospital: Manual for students of the medical universities (Zaporozhye State Medical University). Zaporozhye, 2003.