GENERAL PROPEDEUTICS OF INTERNAL DISEASES

LECTURE COURSE

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VITEBSK STATE MEDICAL UNIVERSITY
DEPARTMENT OF PROPEDEUTICS OF INTERNAL DISEASES

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Курс лекций «Общая пропедевтика внутренних болезней» составлен в соответствии с типовой учебной программой по пропедевтике внутренних болезней, утвержденной Министерством Здравоохранения Республики Беларусь в 1997 г., регистрационный № 08-14/5906, и рабочей учебной программой по пропедевтике внутренних болезней для студентов лечебно-профилактического факультета, утвержденной ВГМУ 29.08.2003 г. по специальности «Лечебное дело».

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CONTENT

The lecture course of general propedeutics of internal diseases is designed basically for students of the faculty for the training of foreign citizens through the mediation of the English language during the course of studies. The lectures can be useful to teachers of therapeutic departments of medical universities while preparing for their classes with foreign students, to senior students, post-graduates and clinical residents.

The lectures correspond to the basic educational thematic parts of general propedeutics of internal diseases, according to Standard Educational Program of internal diseases propedeutics approved by Ministry of Public Health of Republic of Belarus in 1997 (registration number 08-14/5906) and the syllabus of internal diseases propedeutics for students of medical faculty approved by Vitebsk state medical university in 2003.

The reference sources of the lectures are selected with regard for the modern level of internal medicine development. We ask to send all critical remarks and wishes to the department of propedeutics of internal diseases of Vitebsk state medical university.

The subject and problems of propedeutics of internal diseases. General plan of inspection of a patient

Subjective examination (inquiry) of the patient and its role in establishment of the diagnosis. Objective examination of the patient. General inspection (survey)

The subject and problems of propedeutics of internal diseases

The term "internal diseases" came into use in the 19th century to stand alongside with the then-popular term "therapy". Internal Medicine is a nonsurgical medical specialty concerned with diseases of internal organs in adults.

The development of new complicated methods of diagnosis and treatment requires specialized skills and training on the part of the physician and has led to the formation of separate branches of medicine such as cardiology, gastroenterology, endocrinology, and hematology. This by no means indicates the division of the concept of "internal medicine" into "daughter subjects".

The subject of propedeutics of internal diseases is introduction to therapy and diagnosis of the most common diseases of internal organs. Propedeutics of internal diseases includes 2 main parts: general propeudeutics (basic principles and methods of diagnosis of internal diseases) and special (particular) propeudeutics (basis of diagnosis of most common diseases of internal organs).

Diagnosis in medicine is the determination of the nature of a disease. Modern diagnosis combines the taking of the patient's health history, a physical examination, and laboratory and radiological examinations.

All symptoms (signs of diseases) are divided into subjective and objective symptoms. S*ubjective symptoms such as p*ain or nausea are 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.

Examination consists of two main parts - *subjective (interview, or inquiry, taking of anamnesis) and objective examination.*

Subjective examination (inquiry)

The founder of the Russian therapeutic school, Matvey Mudrov (1776-1831), assumed that disease is a result of exposure of a man to unfavourable effects of the environment. He was the first who interrogated the patient in order to substantiate the anamnestic method. He developed a planned clinical examination, and recording case histories. In the field of general therapy he followed the principle of

individual approach and claimed that the patient should be treated rather than his disease.

Grigory Zakharyin (1829-1897) worked out in detail the anamnestic method of diagnosis which helped establish individual diagnosis (in addition to the physical examination of the patient) in the presence of not only morphological but also functional changes in various organs. An outstanding French practitioner of medicine Huchard wrote: "Zakharyin's school used observation, accurate anamnesis, and knowledge of etiology, which where raised to the level of an art".

The examination begins with an *interview (inquiry, taking of anamnesis).* 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).

General scheme of inquiry is represented in the following kind:

1. Inquiry of the patient about complaints, about his sensations, experiences (*Present complaints*).

2. Inquiry about the present disease, about its beginning and the subsequent course to the present day, i.e. day of research of the patient, an anamnesis of disease (*Anamnesis morbi, or History of present illness*).

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.).

3. Inquiry about previous life of the patient, an *anamnesis of life (Anamnesis vitae, or Life history, or Past history*). 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 of the anamnesis begins with the *general biographical information.* Unfavourable *labour conditions* and *industrial hazards* are important. For example, some harmful dusts may cause bronchial asthma and chronic diseases of the bronchi and lungs.

Social history (SH) includes information about housing and living conditions, flat (house), heating, house, running water, sewerage, etc.

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 provokes heart diseases.

Family history (FH), or hereditary history. Health of the parents, sisters or brothers is often informative. If some of the family have 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.

Allergy anamnesis is very important. Some patients (and even healthy subjects) often develop a pathologically heightened (or an inverted) response of the immune system (allergy), and this factor is essential in the pathogenesis of certain diseases of internal organs. It is necessary therefore to collect an allergy anamnesis, that is determine whether the patient or his relatives had allergic reactions to various foods, because strawberry, eggs, canned crabs, and other foods may frequently act as allergen. Some medicinal preparations, perfumes, pollen also do. Allergic reactions are quite varied: from vasomotor rhinitis, nettle rash or Quincke's edema to anaphylactic shock.

Others parts of life history are obstetrical (gynecological) history, social history (SH), urological history, pharmacologic history, surgical history, epidemiological history.

4. Inquiry about the general state of the patient, about the major departures of his organism at present and in the terms immediately previous to disease (*Status functionalis, review of systems, or general anamnesis*).

This interrogation is performed according to the following schema:

The general state of the patient: delicacy, malaise, loss of weight, fever, edemas.

State of respiratory system: respiration by a nose, shortness of breath, coughing, dyspnea, pains in the thorax.

State of circulatory (cardiovascular) system: breathlessness, palpitation, pains in range of heart.

State of an alimentary system: appetite, swallowing, eructation, a heartburn, vomiting, meteorism, pains, stool (defecation).

State of excretory system: a diaphoresis (sweating), diuresis, pains at urination.

State of nervous system: a headache, giddiness, sleeplessness, vision, audition, sense of smell (olfaction), taste.

Objective examination

Another stage of examination is *objective examination* of the patient's condition at the present time (*status praesens*). *Objective examination* consists of two parts: *(1) physical examination,* and *(2) laboratory and instrumental examination.* Objective study of the patient (status praesens***)*** gives information on the condition of the entire body and the state of the internal organs. This examination includes various diagnostic procedures (inspection, measuring temperature, percussion, auscultation, palpation, laboratory tests, X-ray examination, electrocardiography, endoscopy, 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.

Physical Examination

After listening to the patient's complaints and becoming acquainted with the history of the present disease, social and housing conditions, and the family history, the physician should proceed to do a physical examination of the patient. Physical examination includes: (1) general inspection (survey) with assessment of the general condition (general state of patient), (2) examination of the major departures of patient’s organism - special (local) inspection, palpation, percussion and auscultation of respiratory system, blood circulation organs, alimentary system, kidneys and urinary tract, etc.

General Inspection

Inspection technique. The patient should be examined in the daytime, because electric light will mask any yellow colouring 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.

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 upright (vertical) or supine (dorsal) position. The examination should be performed 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 angioma which are characteristic of cirrhosis of the liver).

The entire body is first inspected in order to reveal general symptoms. Next, separate parts of the body should be examined: the head, face, neck, trunk, limbs, skin, subcutaneous fat, edemas, bones, joints, visible mucosa, and the hair cover. General survey includes necessarily the examination (inspection and palpation) of thyroid gland and lymphatic nodes.

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

Consciousness (*mental state*)

It can be *clear or deranged* (clouded) consciousness.

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 in 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 derange 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 hemorrhage). The face is red, breathing is slow, deep, and noisy, pulse is full and rare.

Hypoglycemic 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.

Uremic 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, and 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).

Posture (position) of the patient.

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

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, and the body slips down from the pillows to the foot end of the bed.

Forced (compelled) posture is often assumed by the patient to relieve or remove pain, cough, dyspnea. For example, the sitting position relieves orthopnea: dyspnea becomes less aggravating in cases with circulatory insufficiency. The relief that the patient feels is associated with the decreased volume of circulating blood in 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 membranes 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 is also erecting (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 tumours 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.

Habitus

The concept of habitus includes *the body-build,* i.e. *constitution,* height, and body weight.

Constitution 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 adopted by M. Chernorutsky differentiates between the following three main constitutional types: asthenic, hypersthenic, and normosthenic.

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.

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, and 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; hemoglobin 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 makeup of the body and is intermediate between the asthenic and hypersthenic constitutions.

General state (general condition) of a patient can be estimated in the following degrees of assessment: grave (serious) condition of the patient, state of moderate severity (moderately grave condition), satisfactory state of the patient. The general condition of the patient can be only partly evaluated from the information given by the patient (psychic condition, asthenia, loss of weight, elevated temperature). State of patient's health and general condition of the patient are not the same.

Assessment of the general condition of patient is based on such objective criteria as the state of consciousness, a position of the patient, bearing, gait, a look, a state of a feeding, body temperature, parameters of activity of cardiovascular system (pulse, heart rate, BP), a respiratory organs (rate and character of respiration), functions of excretory system (especially daily urination).

Scheme of a case history

According to the plan of general examination the common schema of a case history includes such sections:

1. Passport data - surname, name, patronymic; sex, age, the home address, the place of employment, the diagnosis at entering a hospital, the clinical diagnosis.
2. Inquiry:

a) Present complaints of the patient;
b) Anamnesis morbi, or history of present illness;
c) Anamnesis vitae, or life history, or past history;
d) Status functionalis, review of systems, or general anamnesis;

3. Objective examination of the patient's condition at the present time (status praesens):

a) general survey, survey of the head and the neck;
b) respiratory system (survey, palpation, percussion of the chest, auscultation of the lungs);
c) circulatory system (survey; palpation, percussion and auscultation of the heart and the large blood vessels);
d) system of digestion (examination of an oral cavity, examination of an abdomen in vertical and horizontal position - survey, auscultation, percussion and palpation of the abdomen).
e) genitourinary system **(**survey of lumbar region and external genetalia, percussion and palpation of kidneys and urinary bladder, ureteric points).

f) nervous system
4. Substantiation of the provisional diagnosis
5. Plan of additional examination and treatment of the patient
6. Results of laboratory and tool researches
7. Conclusions of consulting physician
8. The clinical diagnosis and its substantiation
9. Diaries.

A diary of a case history is filled every day and reflects dynamics of patients' state during the running day and efficacy of prescribed medical actions. The diary of the serious patient is filled 2-4 times day on hours with a precise statement of all medical actions and their results, description of new symptoms and substantiation of new prescriptions. Body temperature, pulse rate, respirations rate, stool and diuresis are marked daily in the diary.

10. Epicrisis

The case history routinely is ended by an epicrisis. The epicrisis is a briefly described summary of the basic complaints of the patient, the history of his disease, objective data, basic laboratory and instrumental studies, the diagnostic resume, course of the disease during the observation, the treatment and its results, the further recommendations in attitude of the treatment and regimen, and a job placement.

Examination of patients with diseases of respiratory system: Subjective examination of patients with diseases of respiratory system. General survey. Static and dynamic survey of the chest. Palpation of the chest, definition of vocal fremitus (voice tremor)

Subjective examination (inquiry) of patients with diseases of respiratory system

Complaints

The main complaints typical for the respiratory system are dyspnea, cough, bloody expectorations, and pain in the chest.

Dyspnea (*short breathlessness*) can be subjective, objective, or subjective and objective simultaneously. By subjective dyspnea is understood the subjective feeling of difficult or laboured breathing. Objective dyspnea is determined by objective examination and is characterized by changes in the respiration rate, depth, or rhythm, and also the duration of the inspiration or expiration. Diseases of the respiratory system are often accompanied by mixed (i.e. subjective and objective)

dyspnea. It is often associated with rapid breathing (tachypnea). These symptoms occur in pneumonia, bronchogenic cancer, and in tuberculosis. Cases with purely subjective dyspnea (in hysteria, thoracic radiculitis) or purely objective dyspnea (in pulmonary emphysema or pleural obliteration) occur less frequently. Dyspnea is possible with both normal and slow rate of breathing (bradypnea). Three types of dyspnea are differentiated by the prevalent breathing phase: inspiratory dyspnea, expiratory dyspnea and mixed dyspnea when both expiration and inspiration become difficult.

Dyspnea may be physiological (caused by heavy exercise) and pathological (associated with pathology of the respiratory organs, diseases of the cardiovascular and hemopoietic systems, and poisoning). Dyspnea associated with respiratory pathology may be of various etiology. It can be caused by obstruction of the respiratory ducts, contraction of the respiratory surface of the lungs due to their compression by liquid or air accumulated in the pleural cavity, decreased pneumatization of the lung in pneumonia, atelectasis, infarction or decreased elasticity of the lungs. These conditions are associated with decreased total (vital) lung capacity and ventilation, which causes increased carbon dioxide content of blood, and acidosis of tissues due to accumulation in them of incompletely oxidized metabolites (lactic acid, etc.).

Cough may indicate the presence of lung disease, but cough per se is not useful for the differential diagnosis. Cough may be dry, without sputum, and moist, during which various amounts of sputum of different quality are expectorated. The presence of sputum accompanying the cough often suggests airway disease and may be seen in asthma, chronic bronchitis, or bronchiectasis. 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.

Hemoptysis is expectoration of blood with sputum during cough. Hemoptysis can originate from disease of the airways, the pulmonary parenchyma, or the vasculature. Diseases of the airways can be inflammatory (acute or chronic bronchitis, bronchiectasis, or cystic fibrosis) or neoplastic (bronchogenic carcinoma or bronchial carcinoid tumors). Parenchymal diseases causing hemoptysis may be either localized (pneumonia, lung abscess, tuberculosis, or infection with *Aspergillus)* or diffuse (Goodpasture's syndrome, idiopathic pulmonary hemosiderosis). Vascular diseases potentially associated with hemoptysis include pulmonary thromboembolic disease and pulmonary arteriovenous malformations.

Chest pain caused by diseases of the respiratory system usually

originates from involvement of the parietal pleura. As a result, the pain is accentuated by respiratory motion and is often referred to as *pleuritic*. Common examples include primary pleural disorders, such as neoplasm or inflammatory disorders involving the pleura, or pulmonary parenchymal disorders that extend to the pleural surface, such as pneumonia or pulmonary infarction. *Pain* in the chest is classified by its location, origin, character, intensity, duration, and irradiation, by its connection with the respiratory movements, cough, and the posture. Pain may arise during the development of a pathological condition in the thoracic wall, the pleura, the heart, and the aorta, and in diseases of the abdominal organs (by irradiation).

Pain may develop in injury of the skin (trauma, erysipelas, herpes zoster, etc.), muscles (trauma, myositis), intervertebral nerves (thoracic radiculitis in spondylarthrosis), ribs and costal pleura (metastases of the tumour, fractured bones, periostitis).

Anamnesis

When questioning a patient the physician should determine the time the disease began. Acute onset is a characteristic of acute pneumonia, especially acute lobar pneumonia. Pleurisy begins more gradually. A non-manifest onset and a prolonged course are the 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.

Information about risk factors for lung disease should be explicitly explored to assure a complete basis of historic data. A history of current and past smoking, especially of cigarettes, should be sought from all patients. The smoking history should include the number of years of smoking, the intensity (i.e., number of packs per day), and, if the patient no longer smokes, the interval since smoking cessation. The risk of lung cancer falls progressively with the interval following discontinuation of smoking, and loss of lung function above the expected age-related decline ceases with the discontinuation of smoking. The patient may have been exposed to other inhaled agents associated with lung disease, which act either via direct toxicity or through immune mechanisms. Important agents include the inorganic dusts associated with pneumoconiosis (especially asbestos and silica dusts) and organic antigens associated with hypersensitivity pneumonitis (especially antigens from molds and animal proteins). Bronchial asthma, which is more common in women than men, is often exacerbated by exposure to environmental allergens (dust mites, pet dander, or cockroach allergens in the home or allergens in the outdoor environment

such as pollen and ragweed) or may be caused by occupational exposures (diisocyanates).

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 helps the physician establish connections between the present disease and diseases of the past history.

Family history is important for evaluating diseases that have a genetic component. These include disorders such as cystic fibrosis, α-antitrypsin deficiency, and asthma.

Survey of patients with diseases of respiratory system

General survey

General survey has crucial importance for assessment of the general state of the patient. It is performed by studying a state of his consciousness and position. In connection with a hypoxia of a brain in respiratory failure all kinds of disordered consciousness can be observed: sopor, stupor, hypoxemic coma, hallucinations.

The forced lateral recumbent (edgewise) position (lateral decubitus) is accepted by patients in pneumonia, tuberculosis, exudative and dry pleurisy, pulmonary abscess or gangrene, bronchiectases.

The forced sitting position is connected mainly to dyspnea (in pneumothorax, an attack of bronchial asthma, emphysema, stenosis of a larynx). In sharp degrees of dyspnea the patients put arms on knees, on edges of a bed, a seat of a chair or the handle of an armchair fixing thus a shoulder girdle and starting auxiliary respiratory muscles.

The characteristic face is observed in acute stage of pneumonia: it is a little reddened and edematous (facies febrilis), restless, with suffering expression, with running over at coughing (in view of its tenderness) a grimace, with motility of wings of a nose (owing to a short breathlessness), with typical blisters of herpes on lips of the mouth and wings of a nose.

In presence of respiratory failure, central cyanosis is observed in various degrees of blueness from moderate cyanosis of the face up to diffuse cyanosis with crimson shade owing to a hyperglobulinemia.

Typical changes of fingers of arms are observed in prolonged suppurative processes in lungs (abscesses and gangrene), emphysema, tumours of a mediastinum, a bronchoectatic disease. Clubbing of the

fingers is enlargement of the terminal digital phalanxes with loss of the nail bed angle. In the typical cases distal phalanxes are represented *drumstick (clubbed, Hippocratic) fingers*. Nails become convex and get looking alike watch glass.

Inspection of the chest

Examination of the chest should be done according to a definite plan. The general configuration of the chest should first be estimated (position of the clavicles, supra- and subclavicular fossae, shoulder blades) during *static survey*; the next step (*dynamic survey*) 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.

Static survey of the chest

Static survey estimates the shape of the chest at quiet respiration. 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

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 moderately 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.

Hypersthenic chest in persons with hypersthenic constitution has the shape of 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.

Asthenic chest in persons with asthenic constitution is elongated, narrow (both the anteroposterior and transverse diameters 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

Emphysematous (barrel-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 dyspnea 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.

Paralytic chest resembles the asthenic chest. It is found in emaciated patients, in general asthenia and constitutional underdevelopment; it often occurs in grave chronic diseases, more commonly in pulmonary tuberculosis and pneumosclerosis, in which fibrous tissue contracts 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.

Rachitic chest (keeled or pigeon chest). It is characterized by a markedly greater anteroposterior 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.

Funnel chest can occur in normosthenic, hypersthenic or asthenic subjects; it has a funnel-shaped depression in the lower 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.

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.

Spine deformities

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. Four types of spine deformities are distinguished: (1) lateral curvature of the spine, called scoliosis; (2) excessive-forward and backward curvature of the spine (gibbus and kyphosis, respectively); (3) forward curvature of the spine, generally in the lumbar region (lordosis); (4) combination of the lateral and forward curvature of the spine (kyphoscoliosis).

Scoliosis is the most frequently occurring deformity of the spine. It mostly develops in schoolchildren 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.

Asymmetry of the chest

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 tumour 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 fossa on one side often depends on the diminution of the apex of a fibrosis-affected lung.

Dynamic survey of the chest

Respiratory movements of the chest should be examined during *dynamic survey of the chest.* 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 et minor, etc.) are actively involved in the respiratory movements in pathological conditions associated with difficult breathing.

The type, frequency, depth and rhythm of respiration can be determined by carefully observing the chest and the 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 of the abdominal wall. During expiration the muscles are relaxed, the diaphragm rises, and the abdominal wall returns to the initial position. This type of respiration is also called diaphragmatic and is mostly 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 in such cases is sometimes carried out only by the intercostal muscles.

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 (1) 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; (2) 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, tumour of mediastinum), in obturation or compression of the main bronchus by a tumour, 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; (3) 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 tumour metastasis into the ribs), in a sharp increase in the intra-abdominal 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 tumour, meningitis, cerebral hemorrhage, 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 uremia, 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 900 ml (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 breathing is, on the contrary, 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 accompanied by decompensated acidosis (diabetic hyperglycemic-hyperketonemic coma, uremic 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 cerebral edema. Derangement of the respiratory function can cause disorders 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 (apnea). This respiration is known as *periodic respiration*.

Biot's respiration is characterized by rhythmic but deep respiratory movements which alternate (in 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 pause with subsequent deepening of the respiratory movement, followed by slowing down. This type of arrhythmic dyspnea can probably be regarded as the early stages of the same pathological processes which responsible for Cheyne-Stokes respiration.

Palpation of the chest

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 (resistance), vocal fremitus, 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.

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 (careful) 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.

Resistance (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. The chest of a healthy person may be compressed on 2-3 cm under the moderate pressure in both the anteroposterior and lateral directions. In the presence of pleurisy with effusion, or pleural tumour, 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 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 *(fremitus vocalis s. 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. Fremitus vocalis is not determined at the region of the heart below the third rib on left parasternal and midclavicular lines.

Vocal fremitus is 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 fremitus 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 fremitus becomes unequal over symmetrical parts of the chest.

Vocal fremitus 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, accumulation of air or fluid in the pleural cavity, etc. Vocal fremitus is also intensified in the presence in the pulmonary tissue of an air cavity communicated with the bronchus.

Vocal fremitus- becomes weaker (1) 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; (2) in complete obstruction of the bronchial lumen by a tumour which interferes with normal conduction of sound waves to the chest wall; (3) in asthenic emaciated patients (with weak voice); (4) in significant thickening of the chest wall in obesity.

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.

Percussion of lungs

Percussion

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 percussing 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.

Mediate percussion is done by tapping with a *plexor* (hammer) on a *pleximeter* placed on the body, or by a finger on another finger. 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, *loudness, sound volume*) 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, and 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 (box sound).* 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.

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 (quite), and lightest (quitest, 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. Loud 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).

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 (see above).
6. In topographic percussion, the finger-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.
7. Comparative percussion should be carried out on exactly symmetrical parts of the body.

Percussion of lungs

Depending on the object of examination, various methods of percussion are used to examine the lungs. The examination begins with comparative percussion.

Comparative percussion of lungs

Comparative percussion is performed to a comparison of the percussion sound (resonance) on the symmetrical points of the appropriate topographical lines of the chest (Table 1).

The 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 3th rib along the parasternalis line and to the level of 4th rib along the

medioclavicular line (further the percussion is continued only on the right side of the chest to hepatic dullness). 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.

Table 1

Topographical (vertical) lines of the chest

Topographical lines	Position
Lin. mediana anterior (anterior/front median line/midline)	on the middle of the breast bone
Lin. sternalis dextra et sinistra (sternal right [left] line)	the right and left edges of the breast bone
Lin.parasternalis dextr. et sin. (parasternal right [left] line)	exactly in the middle between the media-clavicular and sternal lines
Lin. medioclavicularis dextr. et sin. (media-clavicular/midclavicular right [left] line, mammary line)	from the middle of the clavicle and perpendicularly downwards
Lin. axillaris anterior et posterior dextr. et sin. (anterior/posterior axillary right [left] line)	accordingly on the anterior and posterior edges of axillary space
Lin. axillaris media dextr. et sin. (mid-axillary right [left] line)	downwards from the middle of axillary space
Lin. scapularis dextr. et sin. (scapular right [left] line)	on the inferior scapular angle
Lin. paravertebralis dextr. et sin. (paravertebral right [left] line)	on the middle between the posterior median and scapular lines
Lin. mediana posterior (posterior/back median line/midline)	on the spinous processes of spinal /vertebral column vertebrae

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 (1) 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 appropriate

side of the shoulder girdle; (2) in the second and third interspace on the left, because of the closer location of the heart; (3) over the upper lobes of the lung (compared with the lower lobes) because of the varying thickness of pneumatic pulmonary tissue; (4) 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 the fact 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 (“air bladder”) 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 (1) pneumosclerosis, fibrous-focal tuberculosis, (2) pleural adhesion or obliteration of the pleural cavity which interferes with normal distention of the lung during inspiration; the difference in the percussion sound will be more pronounced at the inspiration level and weaker during the expiration; (3) lobular and especially confluent pneumonia, in which pulmonary tissue alternates with consolidations; (4) considerable edema of the lungs, especially in the inferio-lateral regions due to insufficient contractility of the left ventricle; (5) compression of the pulmonary tissue by the pleural fluid (compression atelectasis) above the fluid level; (6) complete obstruction of the large bronchus with a tumour and gradual resorption of air from the lungs below the closure of the lumen (obstructive atelectasis). Clear pulmonary sounds become shorter and higher (i.e. duller) in the mentioned pathological conditions. If these 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 over the atelectatic zone becomes dull with a tympanic tone. This sound can also be heard during percussion of a 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: (a) acute lobar pneumonia at the consolidation stage, when the alveoli are filled with the inflammatory exudate containing fibrin; (b) formation in the lung of a large cavity, which is filled with the inflammatory fluid (sputum, pus, echinococcous cysts, etc.), or heterogeneous airless tissue (tumour); (c) 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 gives a soft short and high sound which is called dull or, by analogy with the percussion sounds of

airless organs and tissues (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 in the lung increases in emphysema. The percussion sound in lung emphysema is louder than 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 is 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, tuberculosis 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 (6—8 cm in diameter) will be tympanic, resembling 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).

Topographic percussion of lungs

Topographic percussion is used for delimitation (1) the upper borders of the lungs or the upper level of their apices and their width (Kroenig's area); (2) the lower borders of the lungs, and (3) variation mobility of the lower border of the lung.

The topographic percussion is always performed from a clear note in the direction of dull sound. Percussion begins from the level of II-d intercostal space to reach a dull note. The border of a lung is marked on that side of a finger-plexímeter which is reversed to more clear sound. The upper edge of the plexímeter lying in intercostal space will correspond to the inferior edge of the superposed rib which is considered to be the lower border of a 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 plexímeter finger is placed parallel to the clavicle and percussion is effected from the middle upwards and slightly medially along the edge of m. scalenus med. to dullness. The upper level of the apices in healthy persons is 3-4 cm above the clavicles. 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 plexímeter finger is placed over the supraspinous fossa parallel to the scapular spine and stroked from the middle. The plexímeter 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 4 to 7 cm, at the right side it is lesser on 1-1.5 cm. The anterior border of the trapezius muscle divides the Kroenig's area into its anterior field which extends to the clavicle, and the posterior one that widens toward the supraspinous fossa. Light (quiet) percussion is used to determine the width of the lung apex. The position of a physician is in back and some right of a patient. 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's area widens accordingly. The presence of connective tissue in the lung apex (which usually develops during inflammation 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's area decreases.

To outline the *lower borders of the lungs* their percussion is carried out in the downward direction along conventional vertical topographical lines (Table 2). 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 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 costal interspace (with the patient in the lying or upright position).

When percussing on a line parasternalis dextra the more precise determination of position of border of the lung is performed by means of immediate percussion according to Obraztsov. The immediate percussion is carried out on two superposed ribs above the dullness. The upper rib lies obviously above a pulmonary tissue and serves as the control, it generates clear pulmonary sound. Next the second rib lied directly above a dull sound

is percussing. If at percussion of this rib it is obtained the same sound as above overlying control rib, it means the border lays on the inferior edge of this rib. If above the lower rib the sound will be a little dulled hence the liver lies under this rib, and the border of the lung will be on the upper edge of the rib that most often happens.

Table 2

Normal Lower Border of the Lungs

Topographical lines	Position of lower border of the lungs
Lin. parasternalis dextr.	the upper edge of 6-th rib
Lin. medioclavicularis dextr.	the inferior edge of 6-th rib
Lin. axillaris anterior dextra et sinistra	the inferior edge of 7-th ribs
Lin. axillaris media dextr. et sin.	the inferior edge of 8-th ribs
Lin. axillaris posterior dextr. et sin.	the inferior edge of 9-th ribs
Lin. scapularis dextr. et sin.	the inferior edge of 10 ribs
Lin. paravertebralis dextr. et sin.	the level of the spinous process of the 11-th thoracic vertebra

The examination of the lateral surface of the chest is performed from the axillary fossa (armpit). 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).

The lower borders of the lungs in normosthenic persons usually occur as given in Table 2. 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 inactivation of the other lung (pleurisy with effusion, hydrothorax, pneumothorax, hemiparesis of the diaphragm).

The elevation of the lower border of the lungs is usually unilateral and occurs in (1) restriction of the lung due to development of connective tissue (pneumosclerosis); (2) complete obstruction of the lower-lobe bronchus by a tumour which causes gradual collapse of the lung, atelectasis; (3) accumulation of fluid or air in the pleural cavity which displace the lung upwards and medially toward the root; (4) 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, *active 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 three lines on the right side (midclavicular, axillary, and scapular lines) and two lines on the left side (midaxillary and scapular lines). The normal mobility of the lower border of the lungs is described by the figures given in Table 3. Mobility of the lower border of the left lung by the midclavicular line cannot be determined because of the interference of the heart.

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. Further the patient is 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. Next the patient is then asked to maximum air from the lungs and to keep breath again. The percussion is now continued in the downward direction from starting point until the clear vesicular resonance disappears. 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.

Table 3

Active respiratory mobility of the lower border of normal lungs

Topographical lines	Active respiratory mobility(sm)
Lin. medioclavicularis dextr.	4-6
Lin. axillaris media dextr. et sin.	6-8
Lin. scapularis dextr. et sin.	4-6

Respiratory mobility of the lower border of the lungs is diminished in inflammatory infiltration or congestive plethora of the lungs, decreased elasticity of the pulmonary 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.

Objective examination of respiratory organs:

Auscultation of lungs, its techniques and diagnostic value. Examination of lung ventilation

Auscultation

Auscultation (L *auscultare* to listen) means listening to sounds inside the body. Auscultation is *immediate (direct)* when the examiner presses his ear 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, Russia included. The first papers devoted to auscultation methods were published in Russia in 1824.

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). The modern stethoscope was invented by the American physician George Philip Cammann.

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

Respiratory act, cardiac contractions, movements in the stomach and the intestine produce vibrations in the surrounding tissues. Some of these vibrations reach the surface of the body and can thus be heard directly by the physician's ear or by using a phonendoscope. Both *direct and indirect auscultation* is used in practical medicine. *Immediate or direct auscultation* is more effective (heart sounds and slight bronchial respiration are better heard by direct auscultation) because the sounds are not distorted and are taken from over a larger surface (the area covered by the physician's ear is larger than that of the stethoscope chest piece, or bell). Immediate auscultation is unpractical for auscultation of the supraclavicular fossa and armpits and sometimes for hygienic considerations. *Mediate (instrumental) auscultation* ensures better localization and differentiation of the sounds of various etiology on a small area (e.g. in auscultation of the heart), although the sounds themselves are slightly distorted by resonance. Sounds are usually more distinct with mediate auscultation.

During mediate auscultation with a solid stethoscope, vibrations are transmitted not only by the air inside the instrument but also through the solid part of the stethoscope and the temporal bone of the examiner (bone conduction). A simple stethoscope manufactured from wood, metal or plastics consists of a tube with a bell which is pressed against the chest wall, the other end of the stethoscope bearing a concave plate for the examiner's ear. Binaural stethoscopes are now widely used. These consist of two rubber or plastic tubes ending with self-retaining ear pieces connected to a single chest piece. The binaural stethoscope is more convenient, especially for auscultation of children and seriously ill patients. Phonendoscopes differ from simple stethoscopes in that they have a membrane covering the bell. Stethoscopes with electrical sound amplification were designed. They, however, were declined by most physicians because of difficulties in differentiation and interpretation of sounds which can be achieved by

experience. Amplifiers that are now available do not ensure uniform amplification of all frequencies and this distorts the sounds.

A stethoscope is a closed acoustic system where air serves as a transmitting medium for sounds. Therefore, if the tube is clogged, or communicates with ambient air, auscultation becomes impossible. The skin against which the bell of the stethoscope is pressed acts as a membrane whose acoustic properties change under pressure: if the pressure on the skin increases higher frequencies are better transmitted, and vice versa. Excess pressure on the bell damps vibration of the underlying tissues. A large bell better transmits lower frequencies.

The human ear perceives vibrations in the range from 16-20 to 20000 per second, i.e. from 16 to 20000 Hz; variations in frequency are differentiated better than in the sound intensity. The highest sensitivity of the ear is to sounds of 2000 Hz. The sensitivity decreases sharply with decreasing frequency. For example, it decreases to 50 per cent at 1000 Hz and to 0.9 per cent at 100 Hz. It should also be remembered that a weak sound is perceived with difficulty after a loud sound.

Auscultation techniques

Special rules should be followed during auscultation paying particular attention to conditions in which it is carried out. The first requirement is silence in the room and the absence of any extraneous sounds that might mask the sounds heard by the physician. The ambient temperature should provide comfort for the undressed patient. During auscultation the patient is either sits or stands upright. If the patient is in grave condition he may remain lying in bed. During auscultation of the lungs of a lying patient, his chest is first auscultated on one side and then the patient is turned to the other side and auscultation is continued.

The skin to which the bell of the phonendoscope is pressed should be hairless because hair produces additional friction which interferes with differentiation and interpretation of the sounds. When using a stethoscope its bell should be pressed firmly and uniformly to the patient's skin but the pressure should be moderate since excess pressure damps vibration of the skin to diminish the intensity of the sounds. The bell of the stethoscope should be held by the thumb and the forefinger. The posture of the patient should be varied in order to ensure better conditions for auscultation of each particular organ. The respiration of the patient should be regulated by the physician and in some cases the patient is asked to cough (e.g. rales in the lungs may disappear or change their properties after expectoration).

Many various stethoscopes and phonendoscopes are now produced by the medical industry but they differ mostly in design. It is important that the physician should use an instrument to which he got accustomed. An experienced physician will always feel it difficult to differentiate and interpret sounds if a new stethoscope is used for some reasons. This explains

the necessity of sufficient theoretical knowledge on the part of the physician so that he might correctly interpret the heard sounds. Hence it is the necessity of constant training in auscultation. Only the permanent use of this diagnostic technique will make it a useful tool of diagnosis.

Auscultation of the lungs

Auscultation of the lungs should be performed 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.

Respiratory murmurs (breath sounds) during various phases of respiration are first compared during auscultation of the lungs as well as their character, length, and intensity (loudness). Then these sounds are compared with the respiratory murmurs at the symmetrical points of the other half of the chest (comparative auscultation). Attention should be paid to the *main respiratory sounds*, such as *vesicular (alveolar) breathing* which is heard over the pulmonary tissues, and *bronchial (laryngotracheal) breathing* which is heard over the larynx, trachea, and large bronchi.

In the presence of pathology in the airways, in the alveolar tissue or in the pleura, *adventitious sounds such as rales, crepitation, and pleural friction* are heard in addition to the main breath sounds during inspiration and expiration. These adventitious sounds should be examined only after the character of the main sounds has been established. Normal breathing sounds should be better auscultated with the nasal breathing (with the patient's mouth closed) while adventitious sounds are better heard with deep respiration through the open mouth.

Vesicular breathing (vesicular/ alveolar respiration)

Respiratory sounds known as vesicular respiration arise due to vibration of the elastic elements of the alveolar walls during their filling with air in inspiration. The alveoli are filled with air in sequence. Therefore, the summation of the great number of sounds produced during vibration of the alveolar walls gives a long soft (blowing) noise that can be heard during the entire inspiration phase, its intensity gradually increasing. This sound can be simulated by pronouncing the sound "T" during inspiration, or by drawing tea from a saucer. Alveolar walls still vibrate at the initial expiration phase to give 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.

Normal vesicular breathing is better heard over the anterior surface of the chest, below the 2nd rib, laterally of the parasternal line, and also in the axillary regions and below the scapular angle, i.e. at points where the largest masses of the pulmonary tissue are located. Vesicular breathing is heard worse at the apices of the lungs and their lowermost parts, where the masses of the pulmonary tissue are less abundant. 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 respiration

Vesicular breathing can vary, i.e. it may be louder or softer for both physiological and pathological reasons.

Physiological weakening of vesicular respiration occurs in patients with thicker chest wall due to excessively developed muscles or subcutaneous fat. *Physiological intensification* of *vesicular respiration* may be observed in patients with underdeveloped muscles or subcutaneous fat. Intensified vesicular breathing is characteristic of children with a thin chest wall, good elasticity of the alveoli and the 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 larger vesicles incapable of 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 over 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 tumour). Air admission to the alveoli can be decreased in patients with a markedly weakened inspiration phase (as a result of inflammation of the respiratory muscles, intercostal 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 membranes 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 respiration can be heard during expiration or 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 respiration during which the inspiration and expiration phases are intensified, is called *harsh (or coarse, rough) respiration.* 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 or 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 uneven unfolding of the alveoli. Interrupted breathing indicates pathology in fine bronchi and is more frequently heard at the apices of the lungs during their tuberculosis infiltration.

Bronchial breathing/respiration (laryngotracheal respiration)

Respiratory sounds known as bronchial or tubular breathing arise in the larynx and the 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 the 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 (by the site of its generation).

Bronchial breathing is well heard in physiological cases over the larynx, the trachea, and 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 parts 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 (or in addition to the vesicular breathing) 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 duration of the pulmonary tissue when the alveoli are filled with effusion (acute lobar pneumonia, tuberculosis, etc.), with blood (lung infarction), or due to compression of the alveoli by air or fluids accumulated in the 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 inflated pulmonary tissue by connective tissue (pneumosclerosis, carnification of the lung lobe, which sometimes occurs in acute lobar pneumonia, etc.).

Depending on degree of induration, its size and location in the lung, pathological bronchial breathing may have different intensity and pitch. If induration is large and superficial, loud bronchial breathing is heard as if near the ear. Bronchial breathing can be heard in acute lobar pneumonia at its

second stadium (affection of the entire lobe of the lung). Especially soft and low sounds are heard inpatients with compression atelectasis.

Pathological bronchial breathing 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 respiration to the surface of the chest wall, the more so that sound is intensified in the resonant cavity at the moment of air passage from narrow bronchus the air is set in vortex motion.

Depending the origin there are three *types of pathological bronchial respiration:*

1) *Infiltrative type* arises in consolidation of a pulmonary tissue (II stages of acute lobar pneumonia, infarct of lungs, tuberculosis),

2*) Cavitary type* is auscultated above superficially posed smooth-bore lumen of the big diameter connected with a bronchus (an abscess, a tubercular cavern, bronchiectasias with an appreciable distention of bronchi),

3) *Atelectatic type* - it is observed in compression atelectasis (exsudative pleurisy of 1,5-3 litres), is auscultated at a column on high bound of a dullness where is compressiated lung, rarely passes for lin. axillaries anterior.

Amphoric respiration arises in the presence of a smooth-wall cavity (not less than 5-6 cm in diameter) communicated with a large bronchus. Sounds of this kind can be produced by blowing over the mouth of an empty glass or clay jar. This altered bronchial breathing is there called a*mphoric (Gk amphoeus* jar*).*

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

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

Bronchovesicular or *mixed respiration* is 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.

Adventitious sounds (additional respiratory sounds)

Adventitious sounds are rales, crepitation, and pleural friction.

Rales arise in pathology of the trachea, bronchi, or if a cavern is formed in the affected lung. Rales are classified as dry (rhonchi) and moist rales.

Dry rales, or 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 focal (in tuberculosis or tumour of the bronchus). Dry rales can be due to (1) spasms of smooth muscles of the bronchi during attacks of 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 and narrows its lumen; (4) formation of fibrous 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 air 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 rales (high-pitched and whistling sounds ,or ronchi sibilantes) and sonorous rales (low-pitched and sonoring sounds, or ronchi sonori).* 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 caliber and large caliber bronchi or when viscous sputum is accumulated in their lumen.

Propagation and loudness of dry rales depend on the size of the affected area in the bronchial tree, on the depth of location of the affected bronchi, and the force of the respiratory movements. When the walls of a medium size and large bronchi are affected to a limited extent, rhonchi are insignificant and soft. Diffuse inflammation of the bronchi or bronchospasm arising during attacks of bronchial asthma is attended by both high-pitched sibilant and low-pitched sonorous rales which vary in tone and loudness. These rhonchi can be heard at a distance during expiration. If rhonchi are due to accumulation of viscous sputum in the bronchi, during deep breathing (or immediately after coughing) they can be either intensified or weakened, or else disappear altogether for a short time.

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 caliber of bronchi where rales are generated, they are classified *as fine, medium* and *coarse (large) bubbling rales*. Fine bubbling rales are generated in fine bronchi and are percepted by the ear as short multiple sounds. Rales originating in the finest bronchi and bronchioles are similar to crepitation from which they should be differentiated (see below). Medium bubbling rales are produced in bronchi of a medium size and coarse bubbling 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 calibers, 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,* and *non-consonating* or *bubbling rales*. *Consonating moist rales* are heard in the presence of liquid secretions in the bronchi surrounded by airless (consolidated) pulmonary tissue or in lung cavities with smooth walls surrounded by consolidated pulmonary tissue. The cavity itself 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 tuberculosis 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 sounds produced by collapsing air bubbles in the bronchi are dampened by the "air cushion" of the lungs as they are conducted to the chest surface.

Crepitation

As distinct from rales, crepitation originates in the alveoli. Some authors erroneously classify these sounds as crepitant and subcrepitant rales. Crepitation is a slight crackling sound that can be imitated by rubbing a lock of hair. The main condition for generation of crepitation is accumulation of a small amount of liquid secretion in the alveoli. During expiration, the alveoli stick together, while during inspiration the alveolar walls are 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, e.g. at the first (initial) and third (final) stages of acute lobar pneumonia, when the alveoli contain small amounts of inflammatory exudate, in infiltrative pulmonary tuberculosis, lung infarction, and finally in congestions that develop due to insufficient contractile function of the left-ventricular myocardium or in marked stenosis of the left venous orifice of the heart. Crepitation can be heard in the inferolateral portions of the lungs of aged persons during first deep inspirations, especially so if the patient was in the recumbent position before auscultation. The same temporary crepitation can be heard in compressive atelectasis. During pneumonia, crepitation is heard over longer periods and disappears when a large amount of inflammatory secretion is accumulated in the alveoli or after its complete resolution.

By its acoustic properties, crepitation can often resemble moist fine rales that are produced in fine bronchi or bronchioles filled with liquid secretion. Differentiation of moist rales from crepitation is of great diagnostic importance. Persistent crepitation may indicate pneumonia while fine non-consonating rales suggest bronchitis.

Differential-diagnostic signs of these rales and crepitation are as follows: moist fine rales are heard during both inspiration and expiration; they can be intensified or disappear after coughing, while crepitation can only be heard at the height of inspiration; it does not change after coughing.

Pleural friction sounds (murmur)

In physiological conditions visceral and parietal layers of the pleura are constantly "lubricated" by pleural fluid and are therefore smooth. Their friction during breathing is 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 rough; moreover, cicatrices, commissures, and bands are formed between pleural layers at the focus of inflammation. Tuberculosis and cancer are also responsible for the friction sounds.

Pleural friction sounds are heard during both inspiration and expiration. The sounds are differentiated by intensity, or loudness, length, and site over which they are heard. During early dry pleurisy the sounds are soft and 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 massive deposits on the pleural surfaces. This friction (to be more exact, vibrations of the chest) can be even identified by palpation of the chest.

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 tuberculosis 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.

If the inflammatory focus is localized in the pleura adjacent to the heart, *pleuropericardial friction* sound may be heard during both inspiration and expiration, and also during cardiac systole and diastole. As distinct from cardiac murmurs, this noise is best heard at the height of a deep inspiration because at that time the pleural surfaces come in closer contact with the pericardium.

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 pressed 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, while pleural friction sound is heard during both inspiration and expiration; (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 pleural 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.

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

Bronchophony

This is the voice conduction by the larynx to the chest, as determined by auscultation. But as distinct from vocal fremitus, the words containing sounds “V” 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 (like exaggerated vocal fremitus) suggests consolidation of the pulmonary tissue (which better conducts sound waves) 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.

Examination of lung ventilation

The indices of lung ventilation are not constant and depend not only on the pathological conditions of the lungs or bronchi, but also on the patient's constitution, physical fitness, height, weight, sex, and age. The data obtained during examination of the patient are therefore assessed by comparing them with the data that might be expected from a person with the given physical properties. These data are calculated by special nomograms and formulas that have been compiled from basal metabolism indices.

Measuring respiratory capacity

Various indices are used to characterize lung ventilation. The so-called volumes of the lungs are most popular but they are not accurate enough.

1. The *respiratory volume* (RV) is the volume of air inspired and expired during normal breathing. It is 500 ml on the average varying from 300 to 900 ml. Of this volume, about 150 ml is the physiological dead-space volume of air (PDSV) which is present in the larynx, trachea, and bronchi, but which does not participate in art air to warm and moisten it, which makes residual air physiologically important.

2. The *expiratory reserve volume* (ERV) (1500-2000 ml). This is the volume of air which can be expired by maximum effort after completion of a normal expiration.

3. The *inspiratory reserve volume* (IRV) (1500-2000 ml). This is the volume of air that can be inspired after a normal inspiration.

4. The *vital capacity* (VC) is found by summation of the IRV and ERV and the respiratory volume (3700 ml on the average). This is the greatest volume of air that can be expired from the lungs after a maximum inspiration. The vital capacity of the lungs can be calculated by multiplying the tabulated (optimal) volume of basal metabolism by an empirically found factor 2.3. The deviation from the expected (optimum) vital capacity calculated by this method should not exceed ± 15 per cent.

5. The *residual air volume* (RAV) (1000-1500 ml) is the air that remains in the lungs after maximum expiration.

6. The *total lung capacity* (TLC) is the sum of the RV, ERV and IRV, and RAV. It is about 5000-6000 ml.

Respiratory volumes can be used to assess possible compensation of respiratory insufficiency by increasing respiratory depth at the expense of expiration and inhalation and residual volume.

Normal respiratory volume is about 15 per cent of the vital lung capacity; expiratory and inspiratory air volumes are 42-43 per cent (inspiratory air usually slightly exceeds expiratory air volume); residual air is about 33 per cent of the vital capacity of the lungs. The VC slightly decreases in patients with obstructive hypoventilation, while expiratory and residual air volumes increase at the expense of decreased inspiratory air. RAV (especially the RAV: TLC ratio) increases in some cases to 50 per cent of the TLC (in lung emphysema, bronchial asthma, to a lesser degree in aged persons). VC in patients with hypoventilation also decreases because of the decreased IRV, while the RAV changes only insignificantly.

Spirography gives more reliable information on respiratory volumes. A spirograph can be used not only to measure various respiratory volumes but also some additional ventilation characteristics of intensity of lung ventilation such as the respiratory volume, minute volume, maximum ventilation of the lungs, and the volume of forced expiration.

Intensity of lung ventilation

1. The *minute volume* (MV) is calculated by multiplying the respiratory volume by respiratory rate; it is about 5000 ml on the average. More accurately the MV can be determined by a Douglas bag or using a spirograph.

2. The *maximum lung ventilation* (MLV) is the amount of air that can be handled by the lungs by maximum effort of the respiratory system. It is determined by spirometry during deepest breathing at a rate of 50 r/min; normal ventilation is 80-200 1/min. According to Dembo, the predicted value of the maximum ventilation is the vital capacity of the lungs multiplied by 35 (MLV = VC x 35).

3. The *respiratory reserve* (RR) is determined by the formula RR = MLV - MV. In norm the RR exceeds the MV by at least 15-20 times. In healthy persons the RR is 85 per cent of the MLV, while in patients with respiratory insufficiency it decreases to 60 per cent or lower. This value shows the reserves of a healthy person by which he ensures adequate ventilation under considerable loads, or of a patient with respiratory insufficiency by which he may compensate for significant insufficiency by increasing the minute respiratory volume.

All these tests help study lung ventilation and its reserves, which are important when heavy work is done or there are respiratory diseases.

Mechanics of the respiratory act

The study of this mechanics is necessary for determining changes in the inspiration to expiration ratio, the respiratory efforts at various respiratory phases, and other indices.

1. The *forced expiratory vital capacity* (FEVC). According to Votchal-Tiffeneau this is determined like the vital capacity except that the forced expiration should be performed as fast as possible. The FEVC is 8-11 per cent (100-300 ml) lower than the VC in healthy persons, mainly due to the increased resistance of fine bronchi to the passage of air. When this resistance increases due to bronchitis, bronchospasm, emphysema, etc., the difference may be as great as 1500 ml and more. The volume of forced expiration per minute is also determined. In healthy persons it is more than 75.0 per cent of the VC (average 82.7%).

2. The *forced inspiratory vital capacity* (FIVC) is determined during forced inspiration at a maximum speed. It does not change in emphysema non-aggravated by bronchitis but decreases in obstructed patency of the airways.

3. *Pneumotachymetry and peakflowmetry* are the technique used for measuring peak velocities of air streams in forced inspiration and expiration and is intended to determine the condition of bronchial patency.

Types of disordered lung ventilation

Depending on the cause and mechanism of developing respiratory insufficiency, three types of disordered lung ventilation are distinguished: obstructive, restrictive and mixed (combined).

The obstructive type is characterized by difficult passage of air through the bronchi (because of bronchitis, bronchospasm, contraction or compression of the trachea or large bronchi, e.g. by a tumour, etc.). Spirography shows marked decrease in the MLV and FEVC, the VC being decreased insignificantly. Obstruction of the air passage increases the load on the respiratory muscles. The ability of the respiratory apparatus to perform additional functional load decreases (fast inspiration, and especially expiration, and also rapid breathing become impossible).

Airflow obstruction is usually determined by forced expiratory spirometry - the recording of exhaled volume against time during a maximal expiration. Normally, a full forced expiration takes between 3 and 4 sec, but when airflow is obstructed, it takes up to 15 or even 20 sec and may be limited by breath-holding time. The normal forced expiratory volume in the first second of expiration (FEV1) is easily measured and accurately predicted on the basis of age, sex, and height. The ratio of FEV1 to forced vital capacity (FEV1/FVC, or *index of Tiffeneau*) normally exceeds 0.75 (75%), in bronchial obstruction FEV1/FVC <0.7 (70%).

The restrictive type of ventilation disorder occurs in limited ability of the lungs to expand and to collapse, i.e. in pneumosclerosis, hydro- and

pneumothorax, massive pleural adhesions, kyphoscoliosis, ossification of the costal cartilages, limited mobility of the ribs, etc. These conditions are in the first instance attended by a limited depth of the maximum possible inspiration. In other words, the vital capacity of the lungs (VC) decreases together with the maximum lung ventilation (MLV), but the dynamics of the respiratory act is not affected: no obstacles to the rate of normal breathing (and whenever necessary, to significant acceleration of respiration) are imposed.

The mixed, or combined type includes the signs of the two previous disorders, often with prevalence of one of them; this type of disorder occurs in long-standing diseases of the lungs and the heart.

Examination of patients with diseases of circulatory system:

Subjective examination. Objective examination of circulatory system: survey and palpation of region of the heart and large vessels. Measuring arterial (blood) pressure

Inquiry

Complaints

Patients with diseases of the heart usually complain of *dyspnea,* i.e. distressing feeling of air deficit. Dyspnea is a sign of the developing circulatory insufficiency, the degree of dyspnea being a measure of this insufficiency. When questioning the patient, it is therefore necessary to find out the conditions under which dyspnea develops. At the initial stages of heart failure, dyspnea develops only during exercise, such as ascending the stairs or a hill, or during fast walk. Further, it arises at mildly increased physical activity, during talking, after meals, or during normal walk. In advanced heart failure, dyspnea is observed even at rest. Cardiac dyspnea is caused by some factors which stimulate the respiratory centre.

Attacks of asphyxia, which are known as *cardiac asthma,* should be differentiated from dyspnea. An attack of cardiac asthma usually arises suddenly, at rest, or soon after a physical or emotional stress, sometimes during night sleep. It may develop in the presence of dyspnea. In paroxysmal attacks of cardiac asthma, the patient would usually complain of acute lack of air; respiration becomes stertorous, the sputum is foamy with traces of blood.

Patients often complain of *palpitation.* They feel accelerated and intensified heart contractions. Palpitation is determined by the increased excitability of the patient's nerve apparatus that controls heart activity. Palpitation is a sign of affection of the heart muscle in cardiac diseases such as myocarditis, myocardial infarction, congenital heart diseases, etc., it may

arise as a reflex in diseases of some other organs, in fever, anemia, neurosis, hyperthyroidism, and after administration of some medicinal preparations (atropine sulphate, etc.). Palpitation may also occur in healthy persons under heavy physical load, during running, emotional stress, smoking or coffee abuse. Patients with serious heart diseases may feel palpitation constantly, or it may arise in attacks of paroxysmal tachycardia.

Some patients complain of *intermissions (escaped beats)* which are due to disorders in the cardiac rhythm. Intermissions are described by the patient as the feeling of sinking, stoppage of the heart. Questioning the patient is aimed a determining the circumstances under which intermissions develop. They may arise at rest and physical exercises; they may be intensified in special postures of the patient, etc.

Pain in the heart region is an important and informative sign. The character of pain is different in various diseases of the heart. The physician should determine (by questioning) the location of the pain, the cause or condition under which it develops (exercise, emotional stress, walking, attack of pain at rest, during night sleep), the character of pain (acute, boring, piercing, a feeling of heaviness or retrosternal pressure, small boring pain in the region of the apex), duration and radiation of pain, conditions under which the pain abates. Pain often develops due to acute insufficiency of the coronary circulation, which results in myocardial ischemia. This pain syndrome is called *stenocardia or angina pectoris.* In angina pectoris pain is retrosternal or slightly to the left of the sternum; it most commonly radiates to the region under the left scapula, the neck, and the left arm. The pain is usually associated with exercise, emotional stress, and is abated by nitroglycerin. Angina pectoris pain occurs mostly in patients with coronary atherosclerosis but it may arise in inflammatory diseases of the vessels, e.g. rheumatic vasculitis, syphilitic mesaortitis, periarteritis nodosa, and also in aortal heart diseases and grave anemia.

Pain is especially intense in myocardial infarction and, unlike in angina pectoris, it persists for a few hours, and sometimes for several days. It does not abate after vasodilatory preparations are given. Pain in dissecting aneurysm of the aorta is piercing (like in myocardial infarction). Unlike in myocardial infarction, pain radiates usually to the spinal column, and moves gradually along the course of the aorta. Myocarditis is characterized by intermittent and pressing pain; it is dull, mild, and is intensified during exercise. Pain in pericarditis is located at the middle of the sternum or throughout the entire cardiac region; the pain is stabbing or shooting, and is intensified during movements, cough, even under the pressure of a stethoscope; the pain may persist for several days or arise in attacks. Permanent pain behind the manubrium sterni that does not depend on exercise or emotional stress (the so-called aortalgia) occurs in aortitis. Stabbing pain at the heart apex that arises in emotional stress or fatigue is

characteristic of cardioneurosis. It should be remembered that pain in the cardiac region may arise due to affections of intercostal muscles, nerves, pleura, or the-adjacent organs (diaphragmatic hernia, cholecystitis, ulcer, gastric cancer).

Patients with heart diseases often complain of *cough* which is due to congestion in the lesser circulation. The cough is usually dry; sometimes a small amount of sputum is coughed up. Dry cough is also observed in aortal aneurysm because of the stimulation of the vagus nerve. *Hemoptysis* in grave heart diseases is mostly due to congestion in the lesser circulation and rupture of fine bronchial vessels (e.g. during coughing). Hemoptysis mostly occurs in patients with mitral heart disease. It may occur in embolism of the pulmonary artery. When the aneurysm opens into the respiratory ducts, profuse bleeding occurs.

Venous congestion in the greater circulation occurs in severe heart diseases with circulatory insufficiency. The patients would complain of *edema,* which first develops only in the evening and resolves during the night sleep. Edema occurs mostly in the malleolus region and on the dorsal side of the foot; shins are then affected. In graver cases when fluid is accumulated in the abdominal cavity (ascites) the patient would complain of heaviness in the abdomen and its enlargement. Heaviness most commonly develops in the right hypochondrium due to congestion and enlargement of the liver. In rapidly developing congestion, pain is felt in this region due to distention of the liver capsule. Patients may complain also of poor appetite, nausea, vomiting, and swelling of the abdomen. These symptoms are associated with disordered blood circulation in the abdominal organs. The renal function is upset for the same reason and diuresis decreases.

Patients with cardiovascular pathology often have dysfunction of the central nervous system, which is manifested by *weakness, rapid fatigue, decreased work capacity, increased excitability, and deranged sleep*. Complaints of *headache, nausea, noise* in the ears or the head are not infrequent in essential hypertension patients.

Some heart diseases (myocarditis, endocarditis, etc.) are attended by *elevated (usually subfebrile) temperature;* sometimes *high fever* may occur. The patient should be asked about the time of the day when the temperature usually rises, how long it persists and if this rise is accompanied by chills, profuse sweating, etc.

History of present disease

The time of the onset of the disease and its first symptoms should be determined such as pain, palpitation, dyspnea, elevation of the arterial pressure, the character and intensity of these symptoms, connection with infections and other diseases of the past, cooling, and physical overloads. The character of development of the primary symptoms is important. It is also necessary to find out if any treatment was given and its effect, if any. If there

were exacerbations of the disease, their course and causes should be established.

Anamnesis

Special attention should be paid to various possible causes of the present heart disease. Information should be carefully collected concerning diseases of the past, especially such diseases as *rheumatism, frequent tonsillitis, diphtheria, syphilis,* which would normally provoke cardiovascular pathology. It is important to know the *unfavourable living and working conditions,* chronic exposure to cold and high humidity, *nervous and psychic overstrain, hypodynamia, overeating,* occupational hazards, *smoking and alcohol abuse* and other *harmful habits.* It is also important to ask the patient about cardiovascular diseases that occurred in his relatives, because familial predisposition to some heart diseases is possible. It is necessary to *inquire women* about *past pregnancies and labor,* the onset of menopause because sometimes symptoms of cardiovascular pathology develop in them during this period.

General inspection

The general appearance of the patient, his posture in bed, colour of the skin and visible mucosa, the presence or absence of edema, the shape of the distal finger phalanges (drum-stick fingers) and of abdomen should be assessed. Patients with a marked *dyspnea* usually assume a *half-sitting position;* if dyspnea is grave, the patient assumes a forced position; he sits in bed with the legs on the floor *(orthopnea).* Greater portion of blood is retained in the vessels of the lower extremities in this position to decrease the volume of the circulating blood and congestion in the lesser circulation. Lung ventilation is thus improved. Furthermore, the diaphragm descends in the orthopnoeic position; if ascites is present, the pressure of the ascitic fluid on the diaphragm is lessened to facilitate respiratory excursions of the lungs and to improve gas exchange.

Patients with exudative pericarditis choose to sit in bed slightly leaning forward. Patients with enlargement of the heart lie on the right side because they feel discomfort when lying on the left side (the dilated heart more tightly presses the anterior wall of the chest).

Cyanotic skin is a common sign of heart diseases. In patients with circulatory disorders, cyanosis is more pronounced in parts of the body that are farther remoted from the heart, i.e. the fingers and toes, the tip of the nose, the lips, and the ear lobes. This phenomenon is known as *acrocyanosis.* It depends on the increased content of reduced hemoglobin in the venous blood because of excessive oxygen absorption by tissues in slow blood circulation. In other cases, cyanosis becomes central in conditions of oxygen hunger of blood due to its insufficient arterialization in the pulmonary bed. The degree of cyanosis varies from a slightly detectable blue tinge to the dark

blue colour. Cyanosis is especially pronounced in patients with congenital heart diseases and arteriovenous shunting. It should be remembered that cyanosis can arise in poisoning by chemicals or drugs that form methemoglobin and sulphmethemoglobin.

The colour of the skin is important for diagnosis of some heart diseases. *Mitral stenosis* can be diagnosed by the violet-red colour of the patient's cheeks, mildly cyanotic colour of the lips, nose, and extremities ("facies mitralis").

The skin and visible mucosa of patients with aortal heart diseases are usually pale. Cyanosis in combination with paleness (pallid cyanosis) is characteristic of stenosis of the orifice of the pulmonary trunk or thrombosis of the pulmonary artery. Icteric colour of the sclera and skin is characteristic of grave circulatory insufficiency. The skin of patients with persisting septic endocarditis has a peculiar colour resembling that of coffee with milk *(colour " coffee with milk ").*

Edema frequently attends heart diseases. If the patient stays out of bed, edema is localized mainly in the malleolus, the dorsal side of the feet, and the shins, where a pressure of fingers leaves slowly leveling impressions. If the patient lies in bed, edema is localized in the sacrolumbar region. If edema is significant, it may extend onto the entire body while the ascitic fluid accumulates in various cavities of the body, such as the pleural cavity (hydrothorax), abdominal cavity (ascites), or in the pericardium (hydropericardium). Generalized edema is called *anasarca.* The edematous skin, especially the skin of the extremities, is pallid, smooth, and tense. In persistent edema, the skin becomes rigid, its elasticity is lost, and the skin acquires a brown tinge due to diapedesis of erythrocytes from the congested vessels. Linear ruptures may develop in the subcutaneous fat of the abdomen in pronounced edema, which resemble the scars of pregnancy. In order to assess objectively the degree of edema, the patient should be weighed regularly and the amount of liquid taken and excreted should be strictly recorded.

Local edema sometimes develops in cardiovascular pathology. When the superior vena cava is compressed, for example in exudative pericarditis or aneurysm of the aortal arch, the face, neck, and the shoulder girdle can be affected by edema (“the collar of Stokes”). Edema of the affected extremity is complicates thrombophlebitis; ascites develops during thrombosis of the portal vein or the hepatic veins.

The shape of the nails and distal phalanges of the fingers is informative. *Drum-stick (Hippocratic) fingers* are characteristic of subacute septic endocarditis and some congenital heart diseases.

Inspection of the heart region and peripheral vessels

Cardiac "humpback" can be seen during inspection of the precordium. This is bulging of the area over the heart, the degree of protrusion depending on the enlargement and hypertrophy of the heart (provided these defects develop in childhood when the chest is liable to changes). General protrusion of the cardiac region and leveling of the costal interspaces are observed in massive effusive pericarditis. The cardiac humpback should be differentiated from deformation of the chest caused by changes in the bones, e.g. in rickets.

In patients with underdeveloped subcutaneous fat and asthenic body build, a limited rhythmic pulsation *{the apex beat)* can be seen in the fifth interspace, medially of the midclavicular line. This is caused by the perssure of the heart apex against the chest wall. In cardiac pathology, the apex beat may produce a stronger pulsation. If precordial depression is found instead of prominence, the apex beat is said to be *negative.* It occurs in adhesive pericarditis because of adhesion of the parietal and visceral layers of the pericardium.

The *false negative (pseudonegative) apex beat* can be observed at asthenics, in a case if the range of an apical beat settles down opposite to a rib. It is a systolic intrusion of a thorax wall to the right and higher of a place of the routine localization of an apex beat that it is inaccurately possible to accept for a negative apical beat.

Pulsation is sometimes observed to the left of the sternal line over a vast area extending to the epigastric region. This is the so-called *cardiac beat*. It is due to contractions of the enlarged right ventricle; a synchronous pulsation can also be seen in the upper epigastric region (below the xiphoid process).

Pulsation in the region of the heart base is sometimes observed. Pulsation of the aorta can be felt in the second costal interspace to the right of the sternum; it appears either during its strong dilation (aneurysm of the ascending part and of the arch of the aorta; aortic valve incompetence), or in sclerotic affection of the overlying right lung. In rare cases, the aneurysm of the ascending aorta can destroy the ribs and the sternum. Elastic pulsating tumour is then seen. Pulsation in the second and third costal interspace that can be seen by an unaided eye is caused by dilatation of the pulmonary trunk. It occurs in patients with mitral stenosis, marked hypertension in the lesser circulation, patent ductus arteriosus with massive discharge of the blood from the aorta to the pulmonary trunk, and in primary pulmonary hypertension. Pulsation occurring lower, in the third and fourth interspace to the left of the sternum, can be due to the aneurysm of the heart in post-infarction patients.

Inspection of the vessels is very important for assessing the cardiovascular system. Swollen and tortuous arteries, especially temporal arteries, are found in patients with essential hypertension and atherosclerosis; this is the result of their elongation and sclerotic changes. Pulsation of the

carotids can only be observed in healthy persons; this pulsation is synchronous with the apex beat. In pathological conditions, mainly in aortic valve incompetence, pronounced pulsation of the carotid arteries can be observed (“carotid shudder”). Synchronously with pulsation of the carotid arteries, the head of the patient may rhythmically move. This is de *Mussel's sign.* Pulsation of arteries, e.g. subclavian, brachial, radial and other arteries can also be observed.

Even arterioles may pulsate (the so-called *capillary pulse).* In order to reveal the capillary pulse, the finger nail should be slightly pressed in order to form a small white spot: the margin between the red and blanched part will be seen to ebb and flow with each pulse beat. Similar pulsation can be seen on hyperaemic skin, e.g. of the forehead, after rubbing it. The name "capillary pulse" is not quite correct because it mostly depends on pulse variations in the arterioles. Capillary pulse can be found in patients with aortic valve failure and sometimes in thyrotoxic goitre.

During *inspection of the veins* the physician can observe their overfilling and dilation. This picture is found in general venous congestion and also in local disorders of blood outflow from the veins. The general venous congestion is caused by affection of the right heart and also by diseases that increase intrathoracic pressure and interfere with the outflow of venous blood through the venae cavae. The neck veins are dilated and become swollen. Local congestion is caused by compression of the vein from the outside (tumour, scars, etc.), or by its thrombosis. Local venous stasis is characterized by dilation of collaterals, while edema is formed at the site where blood outflows through the corresponding vein. In conditions of difficult outflow of blood through the superior vena cava, there are dilated veins of the head, neck, upper extremities, and the anterior surface of the trunk. In conditions of difficult blood outflow through the inferior vena cava, the veins of the lower extremities and lateral surfaces of the abdominal wall are dilated. The blood flow in this case is directed into the system of the superior vena cava, i.e. upwards. If the blood outflow through the portal vein is difficult, the collaterals, connecting the system of the portal vein with the vena cava, become arranged round the umbilicus to form the *caput Medusae* and the blood is directed through the dilated superficial veins to the system of the superior and inferior vena cava. In order to determine the direction of the blood flow in dilated veins, a length of a thick vein is pressed by the finger (after the blood is displaced from it). As the vein becomes filled, the direction of the blood flow can easily be determined: when it is directed downward, the vein portion lying above the compressed site is filled, it is directed upward when the part below this point is filled.

Jugular veins can be seen pulsating on the neck. Blood flow in the jugular vein is slowed down during atrial systole and accelerated during ventricular systole. The neck veins somewhat swell when the blood flow

slows down, and collapse when the blood flow is accelerated. It follows that the veins collapse during systolic dilation of the arteries. This is the so-called *negative venous pulse.* It is hardly noticeable in healthy persons and becomes more evident when the veins are filled with blood due to congestion. Pulsation of the jugular veins caused by pulsation of the carotid arteries can be mistaken for the venous pulse. It should therefore be remembered that pulsation of the carotid artery can be seen medially of the sternocleidomastoid muscle, while pulsation of the vein laterally of this muscle. Moreover, if the vein is pressed by a finger along its course, the transmitted vibrations of the peripheral portion of the vein become more visible, whereas pulsation of this portion discontinues in genuine venous pulse. Distinct pulsation of the neck vessels in the presence of a slow pulse on the radial artery is caused by venous and not by arterial pulsation. The *positive venous pulse* presents in a failure of the three-cuspidate valve because of the backward wave of a blood from a right ventricle in a dextral auricle at a ventricular systole detains outflow a blood from veins in auricles, and veins swell simultaneously with appearance of arterial pulse wave.

Palpation of the heart

Palpation of the heart helps to reveal more accurately the *apex beat*, the presence of the *cardiac beat*, to find other pulsations, or detect *"cat's purr" symptom.*

Palpation of the apex beat

In order to determine the apex beat, the palm of the right hand is placed on the patient's chest. (The left mammary gland in women is first moved upward and to the right.) The base of the hand should be rested on the sternum, while the fingers should be directed toward the axillary region, between the 3rd and 4th ribs. The distal phalanges of three fingers should be flexed to form a right angle to the surface of the chest, and moved slowly along the interspaces toward the sternum until the moderately pressing fingers feel the movement of the heart apex. If the apex beat is felt over a considerable area, its localization are outlined by locating the extreme left and lower point of the protruding area, which is considered to be the point of the apex beat. The apex beat can be better detected if the patient slightly leans forward, or by palpation during a deep expiration: in this position the heart is pressed closer to the chest wall. If the apex beat is palpable, its *properties* are determined: *localization; area; height; resistance.*

Localization of the apex beat. A normal apex beat is found in the fifth interspace, 1—1.5 cm toward the sternum from the left midclavicular line. When the patient lies on his left side, the beat is displaced 3-4 cm to the left, and - 1.5 cm to the right when the patient lies on the right side. Stable displacement of the apex beat may depend on the changes in the heart itself or the adjacent organs. For example, if the left ventricle is enlarged, the apex

beat is displaced to the left to the axillary line, and downwards to the 6th and 7th interspace. If the right ventricle is dilated, the apex beat may be displaced to the left as well because the left ventricle is moved to the left by the distended right ventricle. In cases with abnormal congenital heart position, e.g. in dextrocardia, the apex beat is felt in the fifth costal interspace, 1 -1.5 cm toward the sternum from the right midclavicular line.

The position of the apex beat depends also on the diaphragm. Increased Pressure in the abdominal cavity (in pregnancy, ascites, meteorism, tumours) displaces the apex beat upward and to the left because the heart is not only lifted but also turned to the left to assume a horizontal position. If the diaphragm is low (after childbirth, wasting, visceroptosis), the apex beat is displaced downward and slightly to the right to assume the more vertical position.

Area (width) of the apex beat. The width of an apex beat is defined after a finding its localization. The 3-rd finger of the right arm is placed at the point of localisation of the apex beat, and 2-nd finger is displaced further the extreme right point of the pulsating area. The distance between two marks corresponds to width of the apex beat. Normal width of the apex beat is 1-2 sm. Considering that the apex beat in norm is localized in one intercostal space, and distance between two ribs equally 1 sm it is possible to calculate the area of an apex beat, having multiplied its width by 1 sm. In norm the area of the apex beat 1-2 $см^2$. If the area is smaller than 1 $см^2$, the apex beat is restricted. If the area exceeds more than 2 $см^2$, the apex beat is considered diffused.

In the presence of effusion or gas in the right pleural cavity, the apex beat is displaced to the left accordingly. Pleuropericardial adhesions and sclerotic affection of the lungs due to growth of connective tissue in them displace the heart to the involved side. In patients with left-sided pleurisy with effusion and in accumulation of the fluid in the pericardial region, the apex beat disappears. In about one third of cases the apex is impalpable (covered by the rib).

The most frequent and important diagnostic cause of diffuse apex beat is enlargement of the heart, especially of the left ventricle. The width of the apex beat may increase also due to a closer contact of the heart apex to the chest wall in patients with thin thoracic wall, wide interspaces, sclerotic affection of the lower border of the left lung, displacement of the heart anteriorly by a growing tumour of the mediastinum, etc. The area of the apex beat decreases in patients with developed or edematous subcutaneous fat tissue, narrow interspaces, emphysema of the lungs, and low diaphragm.

Height of the apex beat is the amplitude of vibration of the chest wall at the apex beat area.

For definition height of the apex beat the 3-rd finger is displaced perpendicularly to the chest wall at the point of a maximal pulsation. If the

finger feels a pressing of mild tissues of the distal phalanx, it is moderate height of the apex beat. In case of distal phalanx deflection synchronously with a pulsation it is a high apex beat. At a weak sensation of a pulsation without pressing of mild tissues of a phalanx it is a low apex beat.

Normally it is moderate height of the apex beat. At a pathology, and sometimes and in norm, apex beat can become high or low. This property of the apex beat usually varies with the width. Moreover, the height of the apex beat depends on the contractile strength of the heart. When a person is excited, performs exercises, or has fever, or thyrotoxicosis, the height of the apex beat increases due to the increased contractions of the heart.

Resistance of the apex beat is estimated by resistance that is felt by palpating finger at attempt to prevent pulsations of the apex beat. For this purpose a physician presses with 3-th finger of the right arm perpendicularly to the chest wall in a place of a maximal pulsation. Normal resistance of the apex beat is moderate. If expressed resistance at attempt to prevent pulsations with a pressing of palpating finger it is a resistant apex beat.

Like the former two properties, the resistance of the apex beat depends on thickness of the chest wall and the distance from the heart apex to the examining fingers; but it depends mostly on the strength of contractions of the left ventricle and density of the heart muscle. Density of the left ventricular muscle considerably increases with its hypertrophy to cause resistant apex beat. Hypertrophy of the left ventricle is characterized by diffuse, high, and resistant apex beat. In pronounced hypertrophy of the left ventricle attended by its dilation, the apex of the heart becomes tapered and can be felt by the palpating fingers as a dense and firm dome.

Extra-apical pulsations

Aortic pulsation is not palpable in healthy subjects (except in asthenic persons with wide costal interspaces). Palpation can be used to detect pulsation of the aorta during its distension. If the ascending part of the aorta is dilated, pulsation can be felt to the right of the sternum, and if the aortic arch is dilated, the pulsation can be felt in the region of the sternal manubrium. Aneurysm or pronounced dilation of the aortic arch is characterized by pulsation in the jugular fossa (retrosternal pulsation). Thinning and usuras of the ribs or the sternum can be caused by the pressure of the dilated aorta.

Epigastric pulsation, i.e. visible protrusion and retraction of the epigastric area, is synchronous with the heart work, and may depend not only on hypertrophy of the right ventricle, but on the pulsation of the abdominal aorta and the liver. Epigastric pulsation due to hypertrophy of the right ventricle is usually felt under the xiphoid process and becomes especially vivid during deep inspiration, whereas pulsation caused by the abdominal aorta is slightly lower and becomes less marked during deep inspiration.

Intact abdominal aorta can pulsate in asthenic patients with a flaccid abdominal wall.

Pulsation of the liver can be detected by palpation. True and transmitted pulsations of the liver are distinguished. *The true liver pulsation* is the so-called positive venous pulsation; it may be seen in patients with tricuspid valve incompetence. During systole, the blood flows back from the right atrium to the inferior vena cava and hepatic veins. The liver therefore swells rhythmically with each heart contraction. *The transmitted liver pulsation* depends on the impulses transmitted by the contracting heart. Each systolic contraction displaces the entire mass of the liver in one direction.

The symptom of a cat's purr, i.e. low vibrating murmur, resembles purring of a cat. It is of great value in the diagnosis of heart diseases. This sign depends on the same causes that are responsible for the murmur arising in stenosed valve orifices. In order to determine the thrill, the palpating hand should be placed flat on the points where the heart is normally auscultated. Cat's purr palpated over the heart apex during diastolic contraction is characteristic of mitral stenosis, and thrills felt over the aorta during systole indicate stenosed aortic orifice.

Palpation of the arterial pulse

Palpation of the arterial pulse is the main method of examination of pulse. As a rule, pulse is studied first on the radial artery, since it is superficial and runs immediately under the skin and can thus be readily felt between the styloid process of the radial bone and the tendon of the internal radial muscle. The patient's hand is grasped by the examiner so that the thumb of the right hand is placed on the dorsal side of the arm (near the radiocarpal joint) while the other fingers remain on the frontal side of the arm. As soon as the artery is found, it is pressed against the underlying bone. The pulse wave is felt by the examining fingers as a dilation of the artery. The pulse may be different on different arms, and therefore it should first be palpated simultaneously on both radial arteries.

Palpation of arterial wall allows define the following *properties of pulse: -s*imilarity (uniformity) of pulse on both arms;

- rhythm of pulse, frequency (pulse rate),
- condition of the vascular wall,
- filling (volume) of pulse,
- strain (pressure) of pulse,
- size of pulse,
- deficiency of pulse.

Palpation of pulse begins with research of pulse on both arms. In norm pulse is identical on both arms. If pulse is identical on both arms following researches of its characteristics is performed on one hand. At presence of

various pulse on both hand its characteristics is examined on that side where they are expressed better.

After definition of *similarity (uniformity) of pulse* on both hands, a physician determines *rhythmicity of pulse*. For definition of rhythmicity of pulse 2, 3, 4 fingers of a palpating arm are positioned on a radial artery, and the big finger on a forward surface of a forearm from the back side. If pulsations follow one after another through identical intervals pulse is rhythmical (*pulsus regularis, s. rhythmicus*). When the cardiac rhythm is upset, pulse waves follow at irregular intervals (*pulsus irregularis, s. arrhythmicus*). Some pulse waves may be missing or they may appear prematurely, which is characteristic of extrasystole and also complete arrhythmia (fibrillation), in which pulse waves follow one another at irregular intervals.

In normal conditions, pulse is rhythmic and the pulse wave uniform. Such a pulse is called *uniform (pulsus equalis*). In cardiac rhythm disorders, when the heart contracts at irregular intervals, the pulse wave becomes *non-uniform*, and this pulse is called *unequal (pulsus inequalis*).

For definition of *pulse rate* a physician puts three fingers of a palpating arm (2-, 3-d, 4-th) on radial arteria and counts number of pulse strokes for 15 s or 30 s and received number multiply accordingly on 4 or on 2 (at rhythmical pulse). At arrhythmic pulse rate is counted inventory within 1 minute. A normal frequency of pulse rate is 60-90 in 1 minute.

A pulse rate more than 90 for 1 minute pulse refers to frequent pulse (*pulsus frequens*). Decrease of a pulse rate less than 60 for 1 minute refers to infrequent pulse (*pulsus rarus*).

The *condition of the vascular wall* should be assessed simultaneously. To that end, the artery is pressed by the index and middle fingers of the left hand slightly above the point where the pulse is examined by the right hand. When the vessel stops pulsating under the fingers of the right hand, the arterial wall is felt. A normal artery is a thin elastic tube. In norm an arterial wall is mild, elastic, smooth, and flat. In some diseases, for example, in atherosclerosis, the arteries change, their walls become firm, and the course more tortuous. If calcification is considerable, the artery walls are rough, tortuous tubes, sometimes with bead-like thickenings.

For definition *filling (volume) of pulse* three fingers of a palpating arm (the 2-, 3-d, 4-th) are placed on a. radialis. In the beginning the 2-nd finger squeezes a. radialis up to the arrest of a reversed current of a blood from vessels of a hand, and then the 4-th finger squeezes out a blood from the vessel and squeeze it up to the arrest of transit of pulse wave. The 3-rd finger freely lies on an empty arteria. The 4-th finger is raised with pulse wave. Pulse wave passing under the 3-rd finger raises it and hits about the 2-nd finger. Filling of pulse is estimated on a degree of arising of the 3-rd finger.

Normal pulse is of satisfactory filling. It is distinguished pulse of good filling - complete pulse (*pulsus plenus*), and bad filling - empty pulse (*pulsus vacuus*). Complete pulse may be in sportsmen during sports meets, at exercise stresses. Pulse volume shows the artery filling with blood, which in turn depends on the amount of blood that is ejected during systole into the arterial system and which produces variations in the artery volume. Pulse volume depends on the stroke volume, on the total amount of circulating blood, and its distribution in the body. If the stroke volume is normal and the artery is sufficiently filled with blood, the pulse is said to be full (pulsus plenus). In abnormal circulation and blood loss, the pulse volume decreases (pulsus vacuus).

Strain of pulse, or pulse pressure, is determined by the force that should be applied to the pulsating artery to compress it completely. For definition of strain of pulse the 2-, 3-d, and 4-th fingers of a palpating arm squeeze an artery up to the arrest in it pulsations. This property of pulse depends on the magnitude of the systolic arterial pressure. If arterial pressure is normal, the artery can be compressed by a moderate pressure. A normal pulse is therefore of moderate tension, or of satisfactory strain. The higher the pressure, the more difficult it is to compress the artery; such a pulse is called *pulsus durus* (hard or high-tension pulse). If the arterial pressure is small, the artery is easy to compress, and the pulse is soft (*pulsus mollis*).

Pulse size. The pulse size implies its filling and tension. It depends on the expansion of the artery during systole and on its collapse during diastole. These in turn depend on the pulse volume, fluctuation of the arterial pressure during both systole and diastole, and distensibility of the arterial wall.

Pulse wave increases with increasing stroke volume, great fluctuations in the arterial pressure, and also with decreasing tone of the arterial wall. This pulse is called *high pulse (pulsus altus) or pulsus magnus*. Large-volume or high pulse is characteristic of aortic valve incompetence in thyrotoxicosis, when the pulse wave increases due to the high difference between systolic and diastolic arterial pressure. Such a pulse may develop in fever in connection with decreased tone of the arterial wall.

Pulse wave decreases with decreasing stroke volume and amplitude of pressure fluctuations during systole and diastole and with increasing tone of the arterial wall. The pulse becomes *small pulse (pulsus parvus).* Pulse is small when the amount of blood discharged into the arterial system is small, and the rate of its discharge is low. This is observed in stenosis of the aortic orifice or of the left venous orifice, and also in tachycardia and acute cardiac failure. The pulse wave may be quite insignificant (barely perceptible) in shock, acute cardiac failure and massive loss of blood. This pulse is called *thready pulse (pulsus filiformis).*

In rare cases (in rhythmic pulse), high and low pulse waves are alternating. This is *alternating pulse (pulsus alternans).* It is believed that this

pulse is due to alternation of heart contractions that vary in force. It usually occurs in severe myocardial affection.

Deficiency of pulse is a disharmony between a heart rate and a number of pulse waves on periphery. Deficiency of pulse is defined by a palpatory-auscultative method. For deficiency of pulse the heart rate does not corresponds with the pulse rate on radial artery.

There are two methods for definition of deficiency of pulse:

1. It is performed by one physician. The doctor simultaneously positions the bell of the stethoscope on the point of an apex beat for counting of heart rate, and other loose arm - on the radial artery for scoring the pulse rate during 1 minute. 2. It is performed by two physicians. In this case the first physician listens to heart and counts heart rate within 1 minute, and the second physician counts the pulse rate on the radial artery during the same time. Then among the number of cardiac beats is subtracted the pulse rate and received the deficiency of pulse.

Presence of deficiency of pulse is typical in atrial fibrillation.

Measuring arterial pressure (taking blood pressure)

The pressure of the blood in the arterial system varies rhythmically, attaining its maximum during systole and lowering during diastole. This is explained as follows; when blood is ejected during systole it meets resistance of the arterial walls and of the blood contained in the arterial system; the pressure in the arteries thus increases to cause distention of the arterial walls. During diastole the arterial pressure falls and remains at a certain level due to the elastic contraction of the arterial walls and resistance of the arterioles, owing to which the blood flow into the arterioles, capillaries, and veins continues. It follows therefore that arterial pressure is proportional to the amount of blood ejected by the heart into the aorta (i. e. the stroke volume) and the peripheral resistance.

Arterial pressure is expressed in millimeters of mercury column. The normal systolic (maximal) pressure varies from 100 to 140 mm Hg and diastolic (minimal) from 60 to 90 mm Hg, The difference between systolic and diastolic pressure is called the pulse pressure (normally it is 40-50 mm Hg). Arterial pressure can be measured by a direct or indirect method. In the direct method, the needle or a cannula is introduced directly into the artery and connected to a pressure gauge. This method is mostly used in heart surgery.

Three techniques exist to take blood pressure indirectly. These are auscultatory, palpatory, and oscillographic. The auscultatory method is commonly used in medical practice. The method was proposed by N. Korotkoff in 1905 and is used to measure systolic and diastolic blood pressure. A sphygmomanometer is used to read pressure. It consists of a mercury or a spring manometer which is connected to a cuff and rubber bulb

used to inflate the cuff through a connecting tube. A valve on the bulb is used to admit air into the cuff and the manometer, and to hold pressure at the needed level. A more accurate instrument is a Riva-Rocci mercury manometer. This is a mercury container communicated with a thin vertical glass tube attached to a scale graduated in millimeters from 0 to 300.

The pressure in the brachial artery is usually measured. To that end the arm of the patient is freed from tight clothes and a cuff is attached snugly and evenly onto the arm (a finger-breadth between the cuff and the skin). The inlet socket of the cuff should be directed downward, 2-3 cm above the antecubital fossa. The arm should be placed comfortably on a level surface, the palm up; the muscles of the arm should be relaxed. The phonendoscope bell is placed over the brachial artery in the antecubital space, the valve on the bulb is closed, and air is pumped into the cuff and the manometer. The pressure of the air in the cuff that compresses the artery corresponds to the mercury level as read off the instrument scale. Air is pumped into the cuff until pressure inside it is 30 mm above the level at which the brachial or radial artery stops pulsating. The valve is then opened slowly to release air from the cuff. Using the phonendoscope, the brachial artery is auscultated and the readings of the manometer followed. When the pressure in the cuff drops slightly below systolic, tones synchronous with the heart beats are heard. The manometer readings at the moment when the tones are first heard are taken as the *systolic pressure*. This value is usually recorded to an accuracy of 5 mmHg (e.g. 135, 130, 125mmHg, etc.). When pressure inside the cuff equals *diastolic pressure*, and the blood flow is no longer obstructed, the pulsation of the vessel suddenly decreases. This moment is characterized by a marked weakening and disappearance of the tones.

In normal condition an arterial pressure changes in rather appreciable limens -from 100/70 - up to 140/90 mm Hg. In healthy female the inferior border of a normal arterial pressure (BP) compounds 90/60 mm Hg.

According to classification of levels the BP (WHO/ISH, 1999) the folowing categories of the normal BP:

Optimal BP - less than 120/80 mmHg,

Normal BP - less than 130/85 mmHg,

High normal BP - 130/85 -139/89 mm Hg.

Arterial pressure of healthy subjects varies physiologically within a certain limit depending on physical exertion or emotional strain, the posture, time of meals, and other factors. The lowest pressure is normally observed at rest, before breakfast, in the morning, i.e. in conditions under which basal metabolism occurs. This pressure is therefore called *basal.* When pressure is taken for the first time, it may appear slightly higher than actual which is explained by the patient's response to the procedure. It is therefore recommended that pressure be taken several times at a run without taking off the cuff but only deflating it completely. The last and the least value should

be considered the closest to the true pressure. A transient increase in the arterial pressure may occur during heavy exercise (especially in persons who are unaccustomed to it), in excitation after taking alcohol, strong tea or coffee, in heavy smoking or during attacks of intense pain.

Many diseases are attended by changes in arterial pressure. Elevation of systolic pressure over 140 mm and of diastolic over 90 mm Hg is called *arterial hypertension. A* drop in the systolic pressure below 100 mm and of diastolic below 60 mm Hg is known as *arterial hypotension.* Longstanding elevation of arterial pressure occurs in essential hypertension, many renal diseases (glomerulonephritis, vascular nephrosclerosis), in certain endocrinological diseases, and heart diseases, etc. Systolic pressure alone is sometimes elevated, whereas diastolic pressure remains normal or decreased. This causes a marked increase in the pulse pressure. This condition occurs in aortic incompetence, thyrotoxicosis, less markedly in anemia of any etiology and atherosclerotic affections of the vessels.

Percussion of heart

Conception of cardiac (heart) dullness

Percussion is used to determine sizes, position and configuration (shape) of a heart and its vascular bundle.

The right contour of dullness of the heart and the vascular bundle is formed (from top to bottom) by the superior vena cava to the upper edge of the 3rd rib and by the right atrium at the bottom. The left contour is formed by the left part of the aortic arch at the top, then by the pulmonary trunk, by the auricle of the left atrium at the level of the 3rd rib and downward by a narrow strip of the left ventricle. The anterior surface of the heart is formed by the right ventricle. Being an airless organ, the heart gives a dull percussion sound. But since it is partly covered on its sides by the lungs, dullness is dual in its character, i.e. it is relative (deep) and absolute (superficial). *The relative cardiac dullness* is the projection of heart anterior surface onto the chest. It corresponds to the true borders of the heart. The relative cardiac *dullness is* covered by the lungs. T*he absolute dullness of heart* corresponds to the anterior surface of the heart that is not covered by the lungs.

Rules of percussion of heart:

1. Percussion is performed in most cases in a vertical position of the patient, with the arms lowered downwards, at impossibility of keeping of this rule it is possible to confine percussion in a horizontal position. It should, however, be remembered that the area of cardiac dullness in the vertical position is smaller than in the horizontal. This is due to mobility of the heart and the displacement of the diaphragm as the patient changes his

posture.

2. The doctor can sit or stand to the right of the patient at the time of percussion.

3. Respiration of the patient should be superficial.

4. The finger-pleximeter (3-rd finger of the left arm) must be densely applied to intercostals spaces to avoid lateral distribution of vibrations along the ribs.

5. Percussion is conducted from a clear sound to dulled or dull depending on the purpose of percussion (that is from lungs to heart).

6. The revealed border of the heart dullness is marked on outside edge of the finger-pleximeter inverted to a louder percussion sound.

7. The strength of percussion stroke depends on the purpose of percussion: at delimitation of relative dullness of heart the medium (quiet, or light) percussion is used, at delimitation of absolute dullness of heart - the quietest percussion.

8. The sequence of percussion:

- Delimitation of relative dullness of heart,
- Definition of a configuration of heart
- Definition of transverse length of relative cardiac dullness,
- Definition of size of heart vascular bundle,
- Delimitation of absolute dullness of heart.

Delimitation of relative dullness of heart

It is distinguished right, left and upper borders of relative dullness of the heart. Determining the borders of relative cardiac dullness, interspaces should be percussed in order to avoid lateral distribution of vibrations along the ribs. The percussion stroke should be of medium strength. The pleximeter-finger should be tightly pressed against the chest so that the percussion vibration might penetrate deeper regions.

In the beginning *the right border of relative dullness* of the heart is determined. Since the border of cardiac dullness depends on the position of the diaphragm, the lower border of the right lung is first determined in the midclavicular line; its normal position is at the level of the 6-th rib. The position of the lower border of the lung indicates the level of the diaphragm. The various height of position of diaphragm can be reflected in the dimensions of heart and by that on a position of heart in thorax. For this purpose the finger- pleximeter is applied at the level of 2-d intercostals space on midclavicular line, and percussion is performed strictly on intercostals spaces downwards by quiet percussion before change of a clear pulmonary sound on a dull sound. The mark is made on the edge of the finger-pleximeter inverted to a side of a clear pulmonary sound.

Further the right border of relative dullness of the heart can be defined immediately. The pleximeter-finger is moved on two interspace

above the lower border of the right lung and placed parallelly to the right border of the heart being determined (normally, in the 4-th costal interspace). Percussion is continued by moving the pleximeter-finger gradually along the interspace toward the heart until the percussion sound dulls. The right border of the heart is marked by the external edge of the finger directed toward a clear resonant sound. Its normal position is 1 cm laterally of the right edge of the sternum (Table 4). In case of a change of height of standing of a diaphragm the rules of percussion for definition of the right border of relative dullness are not variated.

In order to *definition of the right contour of the heart* the finger - pleximeter is located in the 3-d and 2-d intercostals spaces at the level of midclavicular line parallel to a sternum (parallel to a finding border of heart in this intercostals space). Percussion with medium strength is continued by moving the pleximeter-finger gradually along the interspace toward the heart until the percussion sound dulls. Further the points received at a percussion in the 4-th, 3-d, 2-d intercostals space are connected among themselves to representation of a right contour of heart. The right contour of heart is formed at the 2-d to 3-d intercostals spaces by superior vena cava and ascending aortic arch, and at the 4-th intercostals space by right auricle.

Table 4

Normal position of relative heart dullness

Border/ Countour	Position	Anatomical structure
Right - 4-th interspace	1 cm laterally of the right edge of the sternum	right atrium
Right – 2-d and 3-d interspaces	0.5 - 1cm laterally of the right edge of the sternum	superior vena cava and an ascending aortic arch
Left – 5-th interspace	1-1.5 cm medially of the left midclavicular line	left ventricle
Left – 4-th interspace	more medially than in 5-th interspace	left ventricle
Left – 3-th interspace	on the middle between midclavicular and parasternal lines	left auricle
Left – 2-th interspace	0.5 - 1cm laterally of the left edge of the sternum	left part of an aorta arch and a pulmonary trunk
Superior	on the upper edge of 3-d rib at the left parasternal line	cone of a pulmonary artery and left auricle

The left border of the relative cardiac dullness is determined in the interspace, where the apex beat is present Therefore the apex beat is

first determined by palpation, and the pleximeter-finger is then placed laterally of this point, parallel to the sought border, and the interspace is percussed toward the sternum. If the apex beat cannot be determined, the heart should be percussed in the 5-th interspace from the anterior axillary line toward the sternum. The normal left border of relative cardiac dullness is located 1-2 cm medially of left midclavicular line; it coincides with the apex beat.

Definition of the left contour of the heart begins with definition of localization of the apex beat. Further the left border of relative dullness of the heart is determined (in norm it settles down in the 5-th intercostal space on 1 sm medially from midclavicular line). Next the pleximeter-finger is raised on one intercostals space above, the pleximeter-finger position in the 4-th intercostals space is parallel to sternum at the level of anterior axillary line, and percussion is performed before change of a clear pulmonary sound on a dulled sound. The point is marked from the side of a clear note. Percussion in the 3-d intercostal space is performed by the same rules. Later the left border of heart vascular bundle in the 2-d intercostals is defined by percussion from midclavicular line to sternum before change of a clear pulmonary note on a dulled sound. The points received by means of percussion in the 5-th, 4-th, 3-d, 2-d intercostals spaces are connected and represents about the left contour of heart.

The left contour of the heart is formed in the 2-d intercostals space by the left part of the aorta arch and the pulmonary trunk, at the 3-d intercostals space - the left auricle, and lower - left ventricle.

The superior border of relative cardiac dullness is determined on a left parasternal line (1-2 cm to the left of left sternal line). To that end the pleximeter-finger is placed at the 1 -t intercostals space perpendicularly to the sternum, and then moved downward until dullness appears. The normal superior border of the relative cardiac dullness is located in the 3-d intercostals space.

For more accurate determination of the superior border the immediate percussion (Obraztcov method) is performed on two overlying ribs above a dulled sound (first – the 2-d control rib, then – the 3-d test rib). If the percussion by ribs yields an identical note, the border is placed on the inferior edge of the lower (the 3-d) rib. If the dull percussion sound is found above the lower rib, the superior border is defined on the upper edge of this rib.

The normal superior border of relative cardiac dullness is located at the level of the upper edge of the 3-d rib and is formed by a cone of a pulmonary artery and the left auricle.

The enlargement of relative dullness of the heart is observed under the following conditions:

- elevated position of diaphragm (in hypersthenic constitution,

meteorism ascites, pregnancy);

- in hypertrophy and dilatation of the right auricle and the right ventricle (in stenosis and incompetence of tricuspid valve, stenosis of ostium of the pulmonary artery, sclerosis of the pulmonary artery, development of the pulmonary heart, mitral stenosis) the borders of heart are displaced to the right;

- as a result of the hypertrophy and dilatation of the left ventricle (in arterial hypertension, stenosis of ostium of aorta, incompetence of the aortal valves, aneurysm of the left ventricle) the borders of heart are displaced to the left;

- as a result of the hypertrophy of the left auricle (mitral stenosis and incompetence of the mitral valve) the borders of heart are displaced upwards;

- as a result of combined heart valves diseases the enlarging of the dimensions of heart is observed in all directions.

The restriction of the relative dullness of heart is observed:

- as a result of phrenoptosis (descent position of a diaphragm in asthenic constitution, at the general enteroptosis);

- as a result of pulmonary pathology (pulmonary emphysema).

Determination of a configuration of heart

The shape of the heart can be determined by percussion of *the borders of the vascular bundle* in the 2-th intercostal space on the right and left, and of relative cardiac dullness in the 4-th or 3-rd interspace on the right, and in the 5-th, 4-th, or 3-rd interspaces on the left. The plexímeter-finger is moved parallel to the borders of expected dullness and the elicited points of dullness are marked on the patient's skin. The points are connected later by a line to mark the contours of the relative cardiac dullness. Normally, an obtuse angle is formed by the lines of the left heart contour between the vascular bundle and the left ventricle. The narrowing of contours of relative cardiac dullness is normally placed at the 3-d intercostal space and named «*waist of heart*». The heart is of normal configuration in such cases. In pathological conditions, when the chambers of the heart are dilated, mitral and aortal configurations are distinguished.

The angle formed by the vascular bundle and the left contour of heart becomes more significant when the left ventricle is enlarged. Since it is more pronounced in aortic incompetence and aortic stenosis, this configuration of heart is known as *"aortic configuration "*. The left atrium is enlarged and the pressure in the pulmonary artery increases in mitral incompetence and mitral stenosis. In this connection *«waist of heart»* becomes smooth. This configuration of the heart is known as *"mitral configuration"*.

Percussion shows considerable enlargement of the cardiac dullness in all directions in pericarditis with effusion. Absolute and relative dullness are almost indistinguishable. The area of dullness resembles a trapezium or a triangle. This configuration of the heart is known as "*trapezoidal configuration"*.

"Spherical configuration", or "cor bovinum", is characterized by the enlargement of heart in all directions as a result of combined heart valves diseases, congenital heart disease, dilated cardiomyopathy, diffuse cardiosclerosis.

Determination of transverse length of relative cardiac dullness

Once the area of relative cardiac dullness of the heart has been established, its *transverse length is measured by a measuring tape,* from the extreme points of the relative dullness to the anterior median line. The normal distance from the right border of relative cardiac dullness (usually in the 4-th intercostals space) to the anterior median line is 3 or 4 cm, while the distance from the left border of relative cardiac dullness (usually in the 5-th intercostals space) to the same line is 8 or 9 cm. The sum of these lengths is the transverse length of relative cardiac dullness (normally 11-13 cm).

Determination of size (width) of a vascular bundle

The vascular bundle of heart is formed: on the right - by cava vein and an ascending part of an aortic arch, on the left -by a pulmonary artery and a part of an aortic arch. The vascular bundle of heart can be determined by percussion of *the borders of relative heart dullness* in the 2-nd intercostal space on the right and left. The *borders of the vascular bundle* are determined by quiet (light) percussion in the second intercostal space, to the right and left from the midclavicular line, toward the sternum. When the percussion sound dulls, a mark should be made by the outer edge of the finger. The right and left borders of vascular dullness are normally found along the edges of the sternum; the transverse length of dullness is 5—6 cm.

Delimitation of absolute (superficial) cardiac dullness

The part of anterior wall of the right ventricle heart is not covered normally by the lungs. Percussion of the anterior wall of heart not covered by the lungs area produces the dull sound and reveals the *absolute cardiac dullness* of the heart. To determine absolute dullness of the heart, *the quietest (lightest) percussion* strokes are needed. *The right border of absolute cardiac dullness* is first elicited. The pleximeter-finger is placed on the right border of relative (deep) cardiac dullness, parallel to the sternum, and then moved medially, to the left, to dullness (change a dulled note on dull). The border is marked by the outer edge of the finger directed toward resonance. In normal subjects this border passes along the left edge of the sternum (Table 5).

Table 5

Normal position of absolute heart dullness

Border	Position	Anatomical structure
Right - 4-th interspace	at the left edge of the sternum	right ventricle
Left – 5-th interspace	1.5-2 cm medially of the left midclavicular line	right ventricle
Superior	on the lower edge of 4-d rib at the left parasternal line	right ventricle

For detection *of the left border of absolute cardiac dullness,* the plexímeter-finger is placed slightly outside the border of relative cardiac dullness, and then moved medially to dullness. The left border of absolute cardiac dullness is normally 1.5-2 cm medially of the left midclavicular line.

For detection of *the superior border of absolute cardiac dullness,* the plexímeter-finger is placed on the superior border of relative cardiac dullness and then moved downward to dullness. The superior border of absolute cardiac dullness is normally at the level of the 4th rib.

For more accurate determination of superior border the immediate percussion (Obraztcov method) is performed on two overlying ribs above a dulled sound (first – the 3-d control rib, then - the 4-th test rib). If the percussion by ribs yields an identical note, the border is placed on the inferior edge of a lower (the 4-th) rib. If more dulled percussion sound is found above a lower rib, the superior border is defined on the upper edge of the 4-th rib. In norm the superior border of absolute dullness of heart settles down at a level of the inferior edge of the 4-th rib.

The *area of cardiac dullness* can be modified by extracardiac factors. At high position of the diaphragm, the heart assumes a horizontal position and its transverse dimensions thus increase. When the diaphragm is low, the heart assumes the vertical position and its transverse diameter is thus diminished. Accumulation of liquid or air in one pleural cavity displaces cardiac dullness toward the healthy side; in atelectasis and pneumosclerosis, or in the presence of pleuropericardial adhesion the borders of cardiac dullness are displaced to the affected side. The area of absolute cardiac dullness markedly diminishes or disappears in pulmonary emphysema, while it increases in pneumosclerosis. The area of absolute dullness is also enlarged in the anterior displacement of the heart (e.g. by a mediastinal tumour, due to accumulation of fluid in the pericardium, or in dilatation of the right ventricle). The borders of relative dullness are displaced in the presence of enlarged heart chambers. Displacement to the

right is due to dilatation of the right atrium and the right ventricle. If the left atrium or the conus of the pulmonary trunk is enlarged, the area of relative dullness is displaced upwards. Dilatation of the left ventricle displaces the area of relative dullness to the left. It should be remembered that a markedly enlarged and hypertrophied right ventricle displaces the left ventricle and can also displace the border of relative dullness to the left. Aortic dilatation increases the dullness area in the second interspace.

Auscultation of heart: heart sounds

Anatomical and physiological bases of auscultation of the heart

The sounds produced by a working heart are called *heart sounds*. Two sounds can be well heard in a healthy subject; the first sound, which is produced during systole and the second sound, which occurs during diastole.

A mechanism by which the heart sounds are produced connects with the phases of the cardiac cycle. The heart contraction begins with the systole of the atria, which is followed by contraction of the ventricles. During the early systole the following phases are distinguished: (1) *asynchronous contraction;* the myocardium is involved only partly and the intraventricular pressure does not increase; (2) *isometric contraction;* it begins when the main mass of the myocardium is involved; atrioventricular valves are closed during this phase and the intraventricular pressure markedly increases; (3) *ejection phase;* the intraventricular pressure increases to level with that in the main vessels; the semilunar valves open. As soon as the blood has been ejected, the ventricles relax (diastole) and the semilunar valves close. The ventricles continue relaxing after the closure of the atrioventricular and semilunar valves until the pressure in them is lower than in the atria *(isometric relaxation phase).* The atrioventricular valves then open to admit blood into the ventricles. Since the difference between pressures in the atria and the ventricles is great during the early diastole, the ventricles are quickly filled *(ventricle rapid filling phase).* The blood flow then slows down *(flow filling phase).* Atrial systole begins, and the cardiac cycle is repeated.

The first sound is produced by several factors. One of them is the valve component, i.e. vibrations of the cusps of the atrioventricular valves during the isometric contraction phase, when the valves are closed. The second component is muscular, and is due to the myocardial isometric contraction. The intensity of myocardial and valves vibrations depends on the rate of ventricular contractions: the higher the rate of their contractions and the faster the intraventricular pressure grows, the greater is the intensity of these vibrations. The first heart sound will thus be more resonant. The third component of the first heart sound is the vascular one. This is due to vibrations of the nearest portions of the aorta and the pulmonary trunk caused

by their distention with the blood during the ejection phase.

The *second sound* is generated by vibrations arising at the early diastole when the semilunar cusps of the aortic valve and the pulmonary trunk are shut (the valve component) and by vibration of the walls at the point of origination of these vessels (the vascular component).

Both sounds can be heard over the entire precordium but their strength changes depending on the proximity of the valves involved in the formation of the first or second sound. Therefore, in order to assess correctly the findings of auscultation, it is necessary to know the sites where the valves project on the chest wall (auscultatory valve areas) and also areas where the sounds produced by a valve can be better heard.

Projections of the valves on the anterior chest wall are very close to one another:

- mitral valves projects to the left of the sternum at the 3-rd costosternal joint;

- tricuspid valve - on the sternum midway between the 3-rd left and 5-th right costosternal joints;

-valves of the pulmonary trunk are projected in the 2-nd intercostal space, to the left of the sternum;

-aortic valves are projected in the middle of the sternum at the level of the 3-rd costosternal joint.

Since all heart valves are projected on a small area of the chest, it is difficult to decide which of them is damaged if the valves are auscultated at sites of their actual projections. Perception of sounds generated in the heart depends on the distance from the valve to its projection on the chest wall and on sound conduction by the course of the blood flow. It is therefore possible to find certain sites on the chest where sounds of each valve can be better heard.

The *auscultatory areas (points)* are as follows:

(1) area of the apex beat - for the mitral valves because the vibrations are well transmitted by the firm muscle of the left ventricle and the cardiac apex is at the nearest distance to the anterior chest wall during systole;

(2) lower part of the sternum near its junction with the xiphoid process (the right-ventricular area) - for the tricuspid valves;

(3) valves of the pulmonary trunk are best heard at its anatomical projection onto the chest, i.e. in the second intercostal space, to the left of the sternum;

(4) aortic valves are best heard in the second intercostal space, to the right of the sternum where the aorta is the nearest to the anterior chest wall;

(5) heart sounds which are associated with the contractions of aortic and mitral valves or which develop during its affections can be heard to the left of the sternum at the 3-rd and 4-th costosternal joints (the so-called fifth listening post at the *Botkin-Erb point*).

Rules of auscultation of heart

- The heart is usually auscultated by a stethoscope or a phonendoscope, but direct (immediate) auscultation is also used.
- The condition of the patient permitting the heart sounds should be heard in various postures of the patient: erect, recumbent, after exercise (e.g. after repeated squatting).
- Sounds associated with the mitral valve pathology are well heard when the patient lies on his left side, since the heart apex is at its nearest position to the chest wall; aortic valve defects are best heard when the patient is in the upright posture or when he lies on his right side.
- The heart sounds are better heard if the patient is asked to inhale deeply and then exhale deeply and keep breath for short periods of time so that the respiratory sounds should not interfere with auscultation of the heart.
- The valve sounds should be heard in decreasing order of their affection frequency. The mitral valve should be heard first (at the heart apex); next follows the aortic valve (in the second intercostal space to the right of the sternum), the pulmonary valve (in the second intercostal space, to the left of the sternum), tricuspid valve (at the base of the xiphoid process), and finally the aortic and mitral valve again at the Botkin-Erb point. If any deviations from normal sounds have been revealed at these points, the entire heart area should be auscultated thoroughly.

Heart sounds

Normal heart sounds

The first sound is produced during systole, after a long pause. It is best heard at the heart apex since the systolic tension of the left ventricle is more pronounced than that of the right ventricle. The first sound is longer and louder than the second heart sound. The *second sound* is generated during diastole, after a short pause, and is best heard at the heart base because it is produced by the closure of the semilunar cusps of the aortic and pulmonary trunk valves. As distinct from the first sound, the second sound is shorter and higher. The tone of the heart sounds may change in pathology, and in order to differentiate between the first and second sounds it should be remembered that the first sound coincides in time with the apex beat (if the latter can be palpated) or with the pulse of the aorta and the carotid artery. Sometimes the third and the fourth sounds can be heard, especially in children and in thin youths.

The *third sound* is caused by vibrations generated during quick passive filling of the ventricles with the blood from the atria during diastole of the heart; it arises in 0.15-1.12 s from the beginning of the second sound.

The fourth sound is heard at the end of ventricular diastole and is produced by atrial contractions during quick filling of the ventricles with

blood.

The third and fourth sounds are low-pitch and soft and are therefore hardly heard in normal subjects. But they are clearly seen on a phonocardiogram. These sounds are better heard in immediate (direct) auscultation. The presence of the third and fourth sounds in the middle-aged usually indicates severe affection of the heart muscle.

Changes in the heart sounds

The heart sounds may increase or decrease their intensity, the tone, or length; they may be split or reduplicated, or adventitious sound may appear.

Intensity of the heart sounds may depend on conditions of the sound wave transmission, i.e. on the extracardiac causes. If subcutaneous fat or muscles of the chest are overdeveloped, or there are lung emphysema, liquid in the left pleural cavity, and some other affections that separate the heart from the anterior chest wall, the intensity of the heart sounds decreases. If conditions for sound transmission are improved (thin chest wall, the lung edges are sclerosed, the heart is pressed against the anterior chest wall by a growing tumour in the posterior mediastinum, etc.), the intensity of the heart sounds increases. The sounds can also be increased by the resonance in large empty cavities filled with air (a large cavern in the lung, large gastric air-bubble). The intensity of the heart sounds also depends on the composition of the blood flowing through the heart: if the blood viscosity decreases (in anemia) the intensity increases.

Intensity of the heart sounds can decrease in decreased myocardial contractility in patients with myocarditis, myocardial dystrophy, cardiosclerosis, collapse, and accumulation of fluid in the pericardial cavity.

Both heart sounds can be increased due to the effect of the sympathetic nervous system on the heart. It occurs in physical and emotional strain, during exercise, and in patients with exophthalmic goitre.

Changes of only one of heart sounds are very important diagnostically.

Intensity of the first heart sound diminishes in the mitral and aortic valve insufficiency. The cusps of the affected mitral valve fail to close completely the left atrioventricular orifice during systole. Part of the blood is thus regurgitated to the left atrium. The pressure of the blood is below norm against the ventricular walls and the cusps of the mitral valve, and the valvular and muscular components of the first heart sound markedly diminish. The period of closed valves is absent also during systole in the aortic valve insufficiency. It means that the valves and muscle components of the first heart sound will also diminish significantly.

In tricuspid and pulmonary valve failure, the diminution of the first heart sound will be better heard at the base of the xiphoid process due to the diminution of the muscular and valves components of the right ventricle.

The first sound can be diminished at the heart apex in stenotic aortal

orifice because systolic tension of the myocardium grows slowly when the blood flow from the left ventricle is obstructed and it is overfilled with blood; the amplitude of the sound vibrations decreases. In diffuse affections of the myocardium (due to dystrophy, cardiosclerosis or myocarditis), the first heart sound only may be diminished because its muscular component also diminishes in these cases.

The first sound increases at the heart apex if the left ventricle is not adequately filled with blood during diastole. The first sound often becomes louder in stenosis of the left atrioventricular orifice, when less than normal amount of blood is discharged from atrium to the ventricle during diastole. The muscle of the left ventricle is therefore less distended by the blood by the start of systole; it is more relaxed and therefore contracts more rapidly and energetically to intensify the first sound. The first sound increases in stenosed right atrioventricular orifice at the base of the xiphoid process. This sound is also intensified during extrasystole (premature contraction of the heart) due to inadequate diastolic filling of the ventricles.

The variation of the first sound at the heart base is not important because this sound is transmitted here from its best auscultative area, i.e. from the cardiac apex area.

The second heart sound is heard over the base. In normal cases the intensity of this sound over the aorta is the same as over the pulmonary trunk. Although the blood pressure in the aorta is higher and the cusps of its valve are closed with a greater force than those of the pulmonary valve, the sound produced by the closing aortic valve is perceived by the examiner as being of the same intensity as the sound of the pulmonary valve, because of the deeper location of the aortic valve.

The second sound over the aorta *is diminished* in aortic valve affections because either the cusps of the valve are destroyed or their vibrating power decreases due to developing cicatrices. Moreover, the thrust of the blood discharged at early diastole from the aorta to the cusps of the aortic valve is weaker than in normal persons because part of the blood is regurgitated to the ventricle through an incompletely closed aortic orifice. The second sound can be inaudible over the aorta if the aortic valve is much destroyed. The second sound diminishes over the aorta in cases of marked hypotension; the second sound diminishes over the pulmonary trunk in cases of aortic valve incompetence (in very rare cases) and in decreased pressure in the lesser circulation.

The *second sound may increase (accent)* either over the aorta or over the pulmonary trunk. If the sound is more intense over the aorta, it is said to be accentuated over the aorta, and if it is stronger over the pulmonary trunk, accentuation of the second sound over pulmonary artery is meant.

The aortic second sound is accentuated when the blood pressure in aorta increases (in essential hypertension, during heavy exercise, in psychic

excitation), because during early diastole, the aortic valve cusps are closed with a greater force due to increased blood pressure in the aorta. The tone of the second heart sound over the aorta often varies. For example, in patients with sclerotic aortic valve, the second sound over the aorta acquires a metallic character which, however, can be heard in normal arterial pressure as well.

The accentuated second sound over the pulmonary artery occurs when pressure in the lesser circulation is elevated or when the vessels of the lesser circulation are overfilled with blood (e.g. in mitral heart diseases), deranged circulation in the lungs and stenosed pulmonary artery (in lung emphysema or pneumosclerosis).

Reduplication of the heart sounds may be revealed by auscultation. Two short sounds which quickly follow one another are heard instead of one. Reduplication of the sounds occurs in asynchronous work of the left and right chambers of the heart. Asynchronous closure of the atrioventricular valves splits the first sound while asynchronous closure of the semilunar valves causes reduplication of the second heart sound. If the two short sounds follow one another at a short interval, they are not perceived as two separate sounds, the sound is said to be split. Both physiological and pathological splitting of the heart sounds is possible. Physiological *reduplication* or *splitting of the first sound* is due to asynchronous closure of the atrioventricular valves, e.g. during very deep expiration, when the blood is ejected into the left atrium with a greater force to prevent the closure of the mitral valves; the valvular component of the left ventricle is therefore split and is perceived as a separate sound.

Pathological reduplication of the first sound can occur in impaired intraventricular conduction (through the His bundle) as a result of delayed systole of one of the ventricles.

The *second sound is reduplicated* more frequently than the first heart sound. Reduplication occurs due to asynchronous closure of the valve of the aorta and pulmonary trunk because of the different length of contractions of the left and the right ventricles. The length of the ventricular systole depends on the volume of the ejected blood and the pressure in that vessel (aorta or the pulmonary artery) into which the blood is expelled. When the amount of blood in the left ventricle decreases and the pressure in the aorta is low, systole of the left ventricle ends sooner and the aortic valve cusps will close earlier than the cusps of the valve of the pulmonary trunk. The second heart sound can therefore be duplicated in cases with diminished or increased filling of one of the ventricles or when pressure in the aorta or the pulmonary artery changes. Physiological reduplication of the second sound is mostly connected with various respiratory phases: the filling of the right and left ventricles differs during inspiration and expiration and the length of their systole changes accordingly, as well as the time of closure of the valve of the

aorta and pulmonary trunk. The amount of blood flowing to the left ventricle decreases during inspiration because part of blood is retained in the distended vessels of the lungs. The left-ventricular systolic blood volume decreases during inspiration, its systole ends earlier, and the aortic valve therefore closes earlier as well. At the same time, the stroke volume of the right ventricle increases, its systole prolongs, the pulmonary valve closure is delayed and the second sound is thus doubled.

Pathological reduplication of the second sound can be due to delayed closure of the aortic valve in persons suffering from essential hypertension, or if the closure of the pulmonary valve is delayed at increased pressure in the lesser circulation (e.g. in mitral stenosis or emphysema of the lungs), delayed contraction of one of the ventricles in patients with bundle-branch block.

True reduplication of the heart sounds should be differentiated from apparent doubling which is connected with the appearance of adventitious sounds. The *mitral valve opening sound* is an example. This sound is heard at the heart apex of patients with mitral stenosis. The sound is heard 0.07-0.13 s following the second sound, during diastole. In normal conditions, the cusps of the atrioventricular valves open noiselessly; they are freely forced back by the blood flow ejected from the atria to the ventricles. In mitral stenosis, the cusps of the sclerosed valve adhere to each other by their edges and cannot freely move to the walls of the ventricle. Therefore, blood thrusts against the valve as it passes from the atrium to generate sound vibrations that are responsible for the appearance of adventitious sounds.

The mitral valve opening sound follows soon after the second heart sound to give it the character of reduplication. This sound is best heard at the heart apex rather than at the heart base; it is characterized by stability and is combined with other auscultative signs of mitral stenosis. The mitral valve opening sound is heard together with a loud (snapping) first sound characteristic of mitral stenosis, and the second sound, to form a specific triple rhythm - *"rhythm of quail"*.

An *extrapericardial sound* can occur in pericardial adhesion. It originates during diastole, 0.08-0.14s after the second sound, and is generated by the vibrating pericardium during the rapid dilatation of the ventricles at the beginning of diastole. The extra sound in adhesions in the pericardium can also arise during systole, between the first and the second heart sounds. This short and loud sound is also known as the systolic click.

Changes in heart sounds in heart diseases can be due to intensified physiological third or fourth sound. In normal subjects these sounds are better revealed in graphic recording (phonocardiography). But if the ventricular myocardium is much weakened, these sounds can be revealed by auscultation. Intensification of one of these sounds gives a three-sound rhythm, known as *the gallop rhythm* (because it resembles the galloping of a

horse). The sounds of the gallop rhythm are usually soft and low, always attended by a thrust, for which reason they are best heard on direct auscultation; the gallop rhythm can also be heard in auscultation with a phonendoscope, but the patient should lie on the left side after a mild exercise. Protodiastolic (at the beginning of diastole), mesodiastolic (in the middle of diastole), and presystolic (at the end of diastole) gallop rhythms are distinguished by the time of appearance of the extra sound in diastole. Gallop rhythm is also classified as ventricular or atrial, according to its origin.

Protodiastolic gallop rhythm arises in considerably diminished tone of the ventricular myocardium. The ventricles distend quickly during their filling with blood at the beginning of diastole and the vibrations thus generated are audible as an extra sound. The sound appears 0.12 - 0.2 s after the second heart sound and is an increased physiological third sound.

Presystolic gallop rhythm arises in intensification of the physiological fourth sound, which is due to the diminished tone of the ventricular myocardium and a stronger atrial contraction. Intensified contraction of the overfilled atrium increases blood ejection into the ventricle, while a diminished tone of the ventricular myocardium causes quicker distention of its walls. The presystolic gallop rhythm is better detected in delayed atrioventricular conduction, when atrial systole is separated from the ventricular systole by a longer than normal period.

Both the third and the fourth heart sounds can intensify significantly in grave myocardial affection, but in tachycardia they sum up to give a *mesodiastolic or summation gallop rhythm.* Gallop rhythm is an important sign of myocardial weakness, and it has a great diagnostic and prognostic value. It develops in severe heart affections in patients with myocardial infarction, essential hypertension, myocarditis, chronic nephritis, decompensated heart diseases.

A. pronounced acceleration of the cardiac rhythm makes the diastolic pause shorter so that it becomes almost as short as the systolic one. If the heart sounds heard at the cardiac apex are similar in intensity, a peculiar auscultative picture resembles the tic-tac or fetal rhythm, known also as *embryocardial* or *pendulum rhythm.* It occurs in severe cardiac failure, attacks of paroxysmal tachycardia, high fever, etc.

Auscultation of heart: cardiac murmurs

Cardiac murmurs

Physical and hemodynamic bases of originating of cardiac murmurs

In addition to the normal heart sounds, abnormal sounds known as murmurs may be heard. Cardiac murmurs may be both *endo- and exocardiac*.

Endocardiac murmurs occur most frequently. These may occur in anatomical changes of the structure of the heart (*organic murmurs*) or in dysfunction of the intact valves (*functional murmurs*). Functional murmurs may be heard with increased rate of blood flow or decreased blood viscosity. The mechanism of endocardiac murmurs can be easier understood if one remembers the laws of physics concerning the flow of liquids in tubes. If a tube has a point where its otherwise even lumen is narrowed, the passing liquid produces noise. This noise is associated with turbulent flow of liquid above the narrowed portion of the tube, which causes vibration of the tube. The intensity of noise depends on two factors, viz., the liquid velocity and the extent of narrowing. The higher the velocity of the liquid, the more intense is the noise; when the liquid velocity decreases, the noise lessens or disappears. As to the extent of tube narrowing, its influence on noise intensity is directly proportional only within a certain range. If the lumen is narrowed to a very high degree, noise may weaken or even disappear. Liquid is also set in vortex movement when it passes a narrow portion of the tube and enters its wider part again. The same reasons account for the murmurs that arise in the heart. If the passage is narrowed or on the contrary widened, blood is set in turbulent flow which generates murmurs. If the vascular lumen remains unchanged, murmurs may be produced by the changes in the blood flow rate, as is the case with thyrotoxicosis, fever, or nervous excitation. Decreased blood viscosity (e.g. in anemia) increases the flow rate of blood and can also be the cause of murmurs. The most frequent cause of endocardiac murmur is various heart defects.

According to the time of appearance, murmurs are classified as systolic and diastolic (Table 6). *Systolic murmur* occurs in cases when, during systole, blood moves from one chamber of the heart to another or from the heart to the main vessels and meets an obstacle. Systolic murmur is heard in the stenotic orifice of the aorta or the pulmonary trunk because blood ejected from the ventricles meets a narrowed vessel (ejection murmur). Systolic murmur is also heard in cases with mitral and tricuspid incompetence (regurgitation murmur). Generation of systolic murmur is explained by regurgitation of blood which is not completely expelled into the aorta and pulmonary trunk during the ventricular systole, but is partly returned to the atrium through an incompletely closed mitral or tricuspid orifice. Since this partly closed orifice is actually a narrow slit, murmur is generated as blood passes through it.

Diastolic murmur occurs if blood meets a narrowed passage during diastole (ejection murmur). This murmur is heard in a stenosed left or right atrioventricular orifice, since blood meets a narrow passage in its flow from the atria into the ventricles. Diastolic murmur also occurs in aortic or pulmonary valve incompetence. Murmur is generated when blood flows back from the vessels into the ventricles through a slit formed by incomplete

closure of the cusps of the affected valve (regurgitation murmur).

Table 6

Classification of cardiac murmurs

Criterion of classification	Classification groups
Cause	Endocardiac and exocardiac murmurs (pleuropericardial/ cardiopulmonary and pericardial friction murmurs)
Changes of the structure of the heart	Organic and functional murmurs
Time of appearance	- Systolic murmurs - Diastolic murmurs (protodiastolic mesodiastolic, presystolic murmurs)
Relation to course of blood flow	Ejection and regurgitation (regurgitant) murmurs
Amplitude of the murmur	High and low amplitude murmurs
Oscillation frequency of the murmur noise	High-pitched and low-pitched murmurs
Character of the murmur noise	Faint (weak), soft, blowing, coarse, rough, grating or grazing sounds; musical murmurs
Changes of the intensity of the noise with the phase of the heart activity.	Decrescendo (decreasing), crescendo (increasing, growing), and crescendo-decrescendo (diamond-shaped) murmurs

Properties of murmurs

The following *characteristics* of *murmurs* should be determined during auscultation:

(1) the relation of the murmur to the phase of the heart activity (to systole or diastole);

(2) the features, character, strength, and length of murmur;

(3) localization of the murmur, i.e. the area where this murmur is heard best;

(4) direction of transmission (radiation).

The *relation of murmurs to systole or diastole* is determined by the same signs that are used to differentiate between the first and the second heart sounds. Systolic murmur appears with the first heart sound, during a short pause of the heart; it is synchronous with the apex beat and the carotid pulse. Diastolic murmur follows the second sound, during the long pause of the heart. Three types of diastolic murmurs are distinguished: (1) *protodiastolic murmur* which arises at the very beginning of diastole, immediately after the second heart sound; (2) *mesodiastolic murmur* which is

heard soon after the second heart sound; *(3) presystolic murmur* which appears at the end of diastole. Murmurs may be *pansystolic and pandiastolic murmurs* which arise at the very beginning of systole (or diastole) and longs to the end of systole (or diastole).

Character of murmur. By their character, murmurs may be soft, blowing, or on the contrary rough, grating or grazing sounds; musical murmurs can also be heard. By duration, heart murmurs are classified as short and long, and by their intensity as soft and loud.

*The intensity of the nois*e may change with the phase of the heart activity. Murmurs may become weaker (*decrescendo*) or louder (*crescendo*), and *crescendo-decrescendo (diamond-shaped)*. Decrescendo murmurs occur more frequently. This can be explained as follows: as blood begins flowing from one heart chamber to another or from the heart to the main vessel, the difference in pressures in two chambers is high and the blood flow rate is therefore high as well. But as the blood is expelled, the pressure inside the chamber from which the blood is ejected gradually decreases, the blood flow rate slows down, and the noise intensity decreases. Presystolic murmur has an increasing character and occurs mostly in stenosis of the anterior left atrioventricular orifice, at the very end of ventricular diastole, when atrial systole begins to increase the blood outflow from the left atrium to the left ventricle. Systolic crescendo-decrescendo (diamond-shaped) murmur presents in aortic stenosis.

Location of the murmur corresponds to the best listening post of that particular valve where this murmur is generated. In certain cases, however, murmurs are better heard at a distance from the point where they are generated, provided their transmission is good. Murmurs are well transmitted in the direction of the blood flow. They are better heard in areas where the heart is close to the chest wall and where it is not covered by the lungs.

Systolic murmur due to mitral valve incompetence is best heard at the heart apex. It can be transmitted by the firm muscle of the left ventricle to the axillary area or by the course of the backward blood flow from the left ventricle to the left atrium, i.e. into the second and third interspace, to the left of the sternum.

Diastolic murmur generated in a narrowed left atrioventricular orifice is usually heard over a limited area at the heart apex.

Systolic murmur due to stenosed aortic orifice is heard in the second interspace, to the right of the sternum. As a rule, it is well transmitted by the course of the blood flow onto the carotid arteries. Since this heart defect is characterized by a rough and loud sound, it can be determined in auscultation over the entire heart region and be transmitted to the interscapular space.

Diastolic murmur due to aortic valve incompetence is better heard not over the aortic valve but rather at the Botkin-Erb point, where it is transmitted by the back flow of blood from the aorta to the left ventricle.

Systolic murmur associated with tricuspid insufficiency is best heard at the base of the xiphoid process, since the right ventricle is the closest to the chest wall at this point, from which the sound can be transmitted upwards and to the right, in the direction of the right atrium. In the rare heart disease associated with stenosis of the right atrioventricular orifice, the diastolic murmur is heard over a limited area at the base of the xiphoid process.

It should be remembered that *murmurs are best heard in certain postures of the patient*. Systolic murmurs associated with incompetence of atrioventricular valves or with stenosis of the aortic or pulmonic orifice, are best heard with the patient in the recumbent posture because the blood flow from the ventricles is facilitated and the blood-flow rate increases. Diastolic murmurs arising due to stenosis of the atrioventricular orifice or incompetence of the aortic valve and the valve of pulmonary trunk are better heard in the upright position, since the blood flow to the ventricles from the atria or from the vessels (in insufficiency of the corresponding valves) is thus facilitated and the blood-flow rate increases.

Differentiation of murmurs

If several murmurs are heard simultaneously over different valves, it is necessary to determine the affected valves and the character of their affections. Systolic and diastolic murmurs over one valve indicate its composite affection, i.e. incompetence of the valve and stenosis of the orifice. If systolic murmur is heard over one valve and diastolic murmur over the other, a combined affection of two valves can be diagnosed.

It is more difficult to decide whether one or two valves are affected if murmurs can be heard at various listening points during one and the same phase of heart activity. The character of the murmur should then be analyzed. If a soft and blowing murmur is heard over one valve and rough and grating over the other, the murmurs are generated by two different affected valves. By moving the stethoscope bell along the line connecting the two valves, the changes in the murmur intensity should be followed. If at some point of the line the murmur disappears or weakens markedly, and then again becomes louder, it will in most cases indicate affection of two valves. If the murmur decreases or increases as the stethoscope bell moves in the direction of the second valve, it usually indicates affection of only one valve. But this is not an indisputable sign because the degree of valve affection may differ too, and an independent, though less loud, murmur will then be heard over milder stenotic affection. The character of murmur transmission helps differentiation. For example, systolic murmur occurring in mitral valve incompetence is transmitted into the axillary region. It may be heard also over the aortic valve but it will not be transmitted onto the carotid arteries (as distinct from systolic murmur associated with stenosis of the aortic orifice).

During auscultation of the heart, it is necessary to differentiate between functional and organic, and between endocardial and exocardial murmurs.

The following *properties of functional murmurs* help differentiate them from *organic murmurs:*

(1) in most cases of functional murmurs are systolic;

(2) functional murmurs are not permanent and may arise and disappear when the person changes his posture, after exercise and during various respiratory phases;

(3) they are mostly heard over the pulmonary trunk and less frequently over the heart apex;

(4) functional murmurs are transient and are rarely heard during the entire systole; these are soft and blowing sounds;

(5) the murmurs are normally heard over a limited area and are not transmitted to long distances from their source;

(6) functional murmurs are not accompanied by other signs of valve affections (e.g. enlargement of the heart chambers or changes in the heart sounds).

Systolic apex murmur

Systolic murmur can be heard at the point of apex beat, which is the main sign of mitral incompetence. It arises during systole when the stream of blood passes a narrow slit leading from the left ventricle to the left atrium. The systolic murmur is synchronous with the first sound. It can be transmitted by the firm muscle of the left ventricle to the axillary area or by the course of the backward blood flow from the left ventricle to the left atrium, i.e. into the second and third interspace, to the left of the sternum. Auscultation findings are confirmed and verified by phonocardiography. The amplitude of the first sound is decreased on a PCG taken at the heart apex; systolic murmur occupies the entire pause between the first and second heart sounds.

Diastolic apex murmur

Diastolic murmur is characteristic of mitral stenosis because the passage from the left atrium to the ventricle during diastole is narrowed. This murmur can be heard to follow the mitral valve opening sound (protodiastolic murmur) because the velocity of the blood flow in early diastole is higher due to the pressure difference in the atrium and the ventricle. The murmur disappears when the pressures equalize. If stenosis is not pronounced, the murmur can be heard only at the end of diastole, immediately before systole proper (presystolic murmur); it arises during acceleration of the blood flow at the end of ventricular diastole because of the early atria systole. Diastolic murmur can be heard in mitral stenosis during the entire diastole. It increases before systole and joins the first snapping sound.

Murmurs of functional etiology can also be heard in aortic incompetence at the heart apex. If the left ventricle is markedly dilated, relative mitral incompetence develops and systolic murmur can be heard at the heart apex. Diastolic murmur (presystolic, or Austin Flint, murmur) can

sometimes be heard. It arises due to an intense regurgitation of the blood that moves aside the mitral valve cusp to account for functional mitral stenosis.

Systolic murmur at the second intercostal space by the right edge of a sternum

Rough systolic murmur over the aorta is characteristic of aorta stenosis. This murmur is generated by the blood flow through the narrowed orifice. It is conducted by the blood onto the carotids and can sometimes be heard in the interscapular space. The phonocardiogram shows the specific changes in the heart sounds: diminished amplitudes of the first sound at the heart apex and of the second sound over the aorta. Systolic murmur over the aorta is typical; its oscillations are recorded in the form of specific diamond-shaped figures.

Diastolic murmur at the second intercostal space by the right edge of a sternum

Diastolic murmur heard over the aorta and at the Botkin-Erb listening point is characteristic of aortic incompetence. This is a low blowing protodiastolic murmur which weakens by the end of diastole as the blood pressure in the aorta drops and the blood-flow rate decreases. The described changes in the sounds and murmurs are clearly visible on PCG in aortic incompetence. The amplitude of the heart sounds and diastolic murmur are decreased on the PCG taken over the aorta.

Systolic murmur at the second intercostal space by the left edge of a breast bone is characteristic of pulmonary artery valves stenosis. The mechanism formation of this murmur is similar to systolic murmur in aortic stenosis.

Diastolic murmur at the second intercostal space by the right edge by the breast bone is characteristic of pulmonary artery valves incompetence. The mechanism formation of this murmur is similar to diastolic murmur in aortic incompetence.

Systolic murmur above the xiphoid process can be heard in tricuspid valve incompetence at the same listening point and also at the 3rd and 4th interspaces, to the right of the sternum; this murmur increases when the patient keeps his breath at the height of inspiration.

Diastolic murmur above the xiphoid process is characteristic of tricuspid stenosis. The mechanism formation of this murmur is similar to diastolic murmur in mitral stenosis.

Extracardial murmurs

Although synchronous with the heart work, they arise outside the heart. These are pericardial and pleuropericardial friction sounds.

Pericardial friction murmurs are connected with the changes in the

visceral and parietal pericardial layers in which fibrin is deposited (in pericarditis), cancer nodes develop, etc. The mechanism by which pericardial friction sounds are generated is similar to that of the pleural friction sounds, except that they depend not on- the respiratory movements but on the movements of the heart during systole and diastole. Pericardial friction murmurs vary. Sometimes they resemble pleural friction or the crisping sounds of snow, and sometimes they are very soft, as if produced by rattling of paper or scratching.

The following signs can be used for *differentiation between pericardial friction sounds and intracardiac sounds*:

(1) there is no complete synchronism of pericardial friction sounds with systole and diastole; friction sounds are often continuous, their intensity increasing during systole or diastole;

(2) friction sounds can be heard for short periods during various phases of the heart work, either during systole or during diastole;

(3) pericardial friction sounds are not permanent and can reappear at intervals;

(4) friction sounds are heard at sites other than the best auscultative points; they are best heard in the areas of absolute cardiac dullness, at the heart base, at the left edge of the sternum in the 3rd and 4th intercostal spaces; their localization is inconstant and migrates even during the course of one day;

(5) friction sounds are very poorly transmitted from the site of their generation;

(6) the sounds are heard nearer the examiner's ear than endocardial murmurs;

(7) friction sounds are intensified if the stethoscope is pressed tighter to the chest and when the patient leans forward, because the pericardium layers come in closer contact with one another.

Pleuropericardial friction murmurs arise in inflammation of the pleura adjacent to the heart and are the result of friction of the pleural layers (synchronous with the heart work). As distinct from pericardial friction sounds, pleuropericardial friction is always heard at the left side of relative cardiac dullness. It usually combines with pleural friction sound and changes its intensity during the respiratory phases: the sound increases during deep inspiration when the lung edge comes in a closer contact with the heart and decreases markedly during expiration, when the lung edge collapses.

Electrocardiography examination:

Algorithm of interpreting ECG. ECG signs of hypertrophy of heart chambers. ECG in IHD (ischemic heart disease)

Electrophysiological bases of ECG (electrocardiography)

Electrocardiography is a method of graphic recording of electric currents generated in the working heart. Contractions of the heart are preceded by its excitation during which physicochemical properties of cell membranes change along with changes in the ionic composition of the intercellular and intracellular fluid, which is accompanied by generation of electric current.

Electrophysiological functions of heart:

-*Automaticity* – function of pacemaker cells to produce spontaneously the action potential (transient depolarization);

-*Conduction* - capability to impulse propagation through cardiac tissues;

-*Excitability* – capability to become excited under the influence of impulses;

-*Refractoriness* is a property of cardiac cells that defines the period of recovery that cells require before they can be reexcited by a stimulus;

-*Contractility* – capability of myocardium to contract in response to excitement.

Cardiac conduction system

The depolarization stimulus for the normal heartbeat originates in the sinoatrial (SA) *node* or *sinus node*, a collection of *pacemaker* cells. These cells fire spontaneously; that is, they exhibit *automaticity*. *Pacemaker cells exhibit automaticity in all departments of conduction system:* I- sinus node (SA), II - AV junction (and atrial fibres) and AV node and His-bundle), III- His-bundle branches, Purkinje fibers.

The first phase of cardiac electrical activation is the spread of the depolarization wave through the right and left atria, followed by atrial contraction. Next, the impulse stimulates pacemaker and specialized conduction tissues in the atrioventricular (AV) nodal and His-bundle areas; together, these two regions constitute the AV junction. The bundle of His bifurcates into two main branches, the right and left bundles, which rapidly transmit depolarization wavefronts to the right and left ventricular myocardium by way of Purkinje fibers. The main left bundle bifurcates into two primary subdivisions, a left anterior fascicle and a left posterior fascicle. The depolarization wave fronts then spread through the ventricular wall, from endocardium to epicardium, triggering ventricular contraction. Ventricular depolarization can be divided into two major phases, each represented by a vector. The first phase denotes depolarization of the ventricular septum, beginning on the left side and spreading to the right. Simultaneous

depolarization of the left and right ventricles (LV and RV) constitutes the second phase.

Recording ECG

Twelve-lead ECG recording has gained wide use: three standard leads (classical), six chest, and three augmented unipolar limb leads (Table 7). Special leads are also used in some cases.

The six frontal plane and six horizontal plane leads provide a three-dimensional representation of cardiac electrical activity. The frontal plane leads - standard and augmented leads**.** The horizontal plane leads – chest leads.

Table 7

Basic leads of ECG

Leads	Position of an electrode	Projection of heart chambers
Standard leads I	Right arm - left arm	Anterior wall of left ventricle
II	Right arm - left foot	-
III	Left foot - left arm	Posterior wall of left ventricle and right ventricle
Augmented leads aVL	Left arm	Anterior wall of left ventricle
aVR	Right arm	-
aVF	Left foot	Posterior wall of left ventricle and right ventricle
Chest leads V1	right sternal border- the 4th intercostal space	Anterior wall of right and left ventricle
V2	left sternal border- the 4th interspace	Anterior part of interventricular septum
V3	between V2 and V4	Anterior wall of left ventricle to apex
V4	left midclavicular line – the 5th interspace	Apex of left ventricle
V5	left anterior axillary line – the 5th interspace	Side wall of left ventricle
V6	left midaxillary line – the 5th interspace	Side wall of left ventricle

Normal ECG

Basic ECG waves and intervals

During diastole the heart does not generate current and an electrocardiograph records a straight line which is called isoelectrical. Action current is represented by a specific curve. An ECG of a healthy subject has the following elements: (1) positive waves *P, R,* and *T,* negative waves *Q* and S; the positive wave *U* is accidental; (2) *P-Q, S-T, T-P,* and *R-R* intervals; (3) *QRS* and *QRST* complexes (Table 8, Fig. Suppl. 2-3). Each of these elements characterizes the time and sequence of excitation of various parts of the myocardium.

Generation of ECG waves and intervals: P – depolarization of atriums, QRS - depolarization of ventricles, ST,T,U - repolarization of ventricles. The QRS-T cycle corresponds to different phases of the ventricular action potential.

Table 8

Waves and intervals of normal ECG

Waves Intervals	Width (s)	Height (mm)	Other characteristics
P	≤0.10	≤2-2.5	(+) I, II, aVF, (-) aVR may be (±) III, aVL, V_{1-2}
PQ	0.12—0.20	-	isoline
QRS	0.06—0.10	≥5 all leads; ≥ 10 chest leads	Q(<0.04 s, ≤2 mm) except aVR, V_{1-2} ; transition zone (R wave = S wave) between V_2 –V_4
ST	-	-	isoline (as rule); may be - (+)1 or (-) 0.5 mm in aVL, aVF. aVR; oblique (+) 3 mm in V_{1-6}
T	-	≤1/4-1/2 R	(+) I, II, V_3—V_6; (-) aVR, V_1; may be(±) III, aVL, aVF, V_{1-2}
QT	0.30—0.46 correlates HR	-	QT = K/√RR (<0.46-0.47 s, K=0.37 for men and 0.39 for women)

In normal conditions, the cardiac cycle begins with excitation of the atria *(P* wave on an ECG). The ascending portion of the *P* wave is mainly due to excitation of the right atrium, while the descending one of the left atrium. The wave is small, and its normal amplitude does not exceed 1-2 mm; the length is 0.08-0.1 s. The *P* wave is followed by a straight line lasting to Q wave; if this wave is small, the line extends to the *R* wave. This is the *P-Q* interval. It extends from the beginning of the *P* wave to the beginning of the *Q* (or *R)* wave and corresponds to the time from the beginning of atrial excitation to the beginning of ventricular excitation, i.e. includes the time of

pulse propagation in the atria and its physiological delay in the atrioventricular node. The normal length of the *P-Q* interval is 0.12-0.18 s (to 0.20 s).

Excitation of the ventricles corresponds to the *QRS* complex. Its waves vary in size and are different in various leads. The length of the *QRS* complex (measured from the beginning of the *Q* wave to the end of the 5 wave) is 0.06—0.1 s. This is the time of intraventricular conduction. The first wave of this complex is the negative *Q* wave. It corresponds to excitation of the interventricular septum. Its amplitude is small and does not normally exceed ^{1}A amplitude of the *R* wave; the length of the *Q* wave does not exceed 0.03 s. The Q wave may be absent on an ECG. The *R* wave corresponds to almost complete excitation of both ventricles. It is the highest wave of the ventricular complex; its amplitude varies from 5 to 15 mm. The negative 5 wave is recorded in full excitation of the ventricles; usually it is not high, actually not exceeding 6 mm (2.5 mm on the average). Sometimes the 5 wave is very small. At the moment of complete depolarization of the myocardium, the potential difference is absent and the ECG is therefore a straight line (the *S-T* interval.) The length of this interval varies greatly depending on the cardiac rhythm; the *S-T* interval may be displaced from the isoelectric line to not more than 1 mm.

The *T* wave corresponds to the repolarization of the ventricular myocardium. The *T* wave is normally asymmetrical: the gradual ascent converts into a rounded summit, which is followed by an abrupt descent. Its amplitude varies from 2.5 to 6 mm, the length from 0.12 to 0.16 s. A small positive *U* wave sometimes follows the *T* wave in 0.02—0.04 s. Its amplitude exceeds 1 mm in rare cases: the length is 0.09—0.16 s. The origin of the *U* wave is disputed.

The *Q-T* interval *(QRST* complex) shows the time of excitation and recovery of the ventricular myocardium i.e. it corresponds to their electrical system. It extends from the beginning of the *Q* wave (or the *R* wave, if the *Q* wave is absent) to the end of the *T* wave. Its length depends on the rate of cardiac contractions: in accelerated heart rhythm the *Q-T* interval shortens. The *Q-T* interval in women is longer than in men (at the same heart rate). For example, at the rate of 60-80 beats per minute, the length of the *Q-T* interval in men is 0.32-0.37 s and in women—0.35-0.40 s.

The *T-P* interval (from the end of the *T* to the beginning of the P wave) corresponds to the electrical diastole of the heart. It is located on the isoelectric line because all action currents are absent at this moment. Its length depends on the cardiac rhythm: the faster the heart rate the shorter the *T-P* interval.

The R-R interval is a distance between the summits of two neighbouring *R* waves. It corresponds to the time of one cardiac cycle, whose length depends on the cardiac rhythm as well.

Interpretation of ECG

In the beginning of interpretation of ECG technical conditions of tape recording must be defined (voltage of the ECG and speed of a tape). The ECG graph paper records the time (interval) between cardiac electrical events along the horizontal axis and their amplitude (voltage) along the vertical axis (Fig. Suppl. 4). . It is important for correct estimation of heart rate, amplitude and duration of ECG waves and intervals (Fig. Suppl. 4).

The sequence of ECG analysis:

1. *Voltage of the ECG* is estimated in compliance *with* standard size of 1 mv =10 mm. To that end, the amplitude of *R* waves is measured in standard leads. Normal amplitude is 5- 15 mm. If the amplitude of the highest *R* wave does not exceed 5 mm in standard leads, or the sum of amplitudes of these waves in all three leads is less than 15 mm, the ECG voltage is considered decreased.

2. *Speed of tape*. If speed of tape is 50 mm/min – 0.02 s in 1 mm of tape (width of QRS=3-4 mm). If speed of tape 25 mm/min – 0.04 s in 1 mm of tape (width of QRS=1-2 mm).

3. *Regularity and pacemaker of the cardiac rhythm.*

Since the sinoatrial node is the pacemaker of a normal heart, and the excitation of the ventricles is preceded by excitation of the atria, the *P* wave should come before the ventricular complex. The R-R intervals should be equal. Its fluctuations normally do not exceed 0.1 s. Greater variations in the length of the *R-R* intervals indicate disordered cardiac rhythm.

Sinus rhythm signs - P-wave positive in II standard lead and corresponds (previous) to complexes QRS.

4. *Heart rate (HR)*

$$\frac{\text{60 (seconds in 1 minutes)}}{\text{[R-R] (in seconds, i.e. to - in divisions x 0.02)}}$$

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 0.8 s, there will be 75 such cycles in a minute (60 : 0.8 = 75). 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. Other variant of HR determination - multiply in 20 times (×20) the number of R-R intervals during 3 seconds (Fig. Suppl. 5).

5. *Analysis of myocardial conduction depending duration of:*

- P wave (≤0.10-0.12 s)– intraatrial conduction;
- PQ interval (0.12-0.20 s) – conduction in atrioventricular node;
- QRS complex (0.06-0.10 s) - intraventricular conduction.

6. *The position of the electrical axis of heart* changes with changes of the position of the heart in the chest. The relation between the electrical axis and the magnitude of the *QRS* complexes in standard leads is described by the so-called Einthoven triangle. The electrical axis of the heart is determined by the shape of ventricular complexes in standard leads:

- normal electrical axis - RII> RI> RIII;
- horisontal electrical axis (levogram)- RI> RII> RIII;
- vertical electrical axis (dextrogram) - RIII> RII> RI.

7. Analysis of waves and intervals. The length and size of ECG elements *(P* wave, *R-Q* interval and *QRST* complexes) are then determined in those leads where the waves are the largest (usually in lead II). Moreover, the direction of the *P* and *T* waves is determined (they can be positive and negative). Smaller and split waves can be present as well. Additional waves can appear. The shape of the ventricular complex in all leads is thoroughly examined, character of the *S-T* interval is noted (see Table 9-2). The length of the *QRST* complex (Q-T interval) depends on rate: the higher the rate, the shorter the interval.

The ECG of healthy persons depend on their age and constitution, on the posture at the moment of taking an ECG (sitting, lying), on the preceding exercise, etc. ECG may change during deep breathing (the position of the heart in the chest is changed during deep inspiration and expiration), in increased tone of the sympathetic and parasympathetic nervous systems and in some other conditions.

It is difficult to overestimate the clinical importance of electrocardiography. It is used to reveal disorders of heart activity, enlargement of heart chambers and to diagnose coronary circulatory disorders.

ECG in hypertrophy of atriums

Hypertrophy of auricles is determined by changes of P-wave.

Left atrium hypertrophy is detected by “P mitrale”: wide (>0.1 s), splitted P-wave in I, II, AVL, left chest leads (V_{5-6}); flat or negative P in III, biphasic or negative (>1 mm) P V_1 (Fig. Suppl. 6). Left atrium hypertrophy is typical in mitral valves diseases (mitral stenosis and mitral incompetence).

Right atrium hypertrophy is detected by “P pulmonale”: high (>2.5 mm) acute P in II, III, AVF and right chest leads (V_{1-2}). Right atrium hypertrophy is typical in chronic pulmonary diseases (pulmonary heart) and tricuspid valves incompetence.

ECG in hypertrophy of ventricles

Hypertrophy of ventricles is determined mainly by changes of ventricular complex QRS (Fig. Suppl. 7). Ventricular hypertrophy causes the following changes in ECG:

(I) the position of the electrical axis is changed:

in left-ventricular hypertrophy - levogram,

in right-ventricular hypertrophy dextogram;

(2) the amplitude of the ventricular complex and its length increase,

(3) changed terminal part of the ventricular complex of the ECG because of repolarization abnormalities:

ST-segment depression and T-wave inversion in leads with a prominent R wave;

(4) in left-ventricular hypertrophy -

amplitude of the S wave increases in V_{1-2};

amplitude of the *R* wave increases >20-25 mm in V_{4-6}

sum of amplitude $S_{V1} + R_{V5}$ (or R_{V6})≥35 mm;

(5) In right-ventricular hypertrophy the changes in the S and *R* waves are the reverse:

high R wave ≥ 7 mm appears in V_{1-2}, deep S wave in V_{4-6} .

ECG in ischemic heart disease (IHD)

Acute ischemia of myocardium causes a current of injury of myocardium (Fig. Suppl. 8):

Subendocardial ischemia - the resultant ST vector directs toward the inner layer of the affected ventricle. Overlying leads therefore will record ST depression.

Transmural or epicardial ischemia - the ST vector is usually shifted in the direction of the outer (epicardial) layers, producing ST elevations and sometimes, in the earliest stages of ischemia, tall, positive so-called hyperacute T waves over the ischemic zone.

ECG in myocardial infarction

Electrocardiographic examination is especially important. It establishes the presence of myocardial infarction and also some important details of the process such as localization, depth of the process, and the size of the affected area.

Three zones of myocardial damage in acute myocardial infarction can be detected by ECG: necrotic zone, ischemic myocardium injury zone, and zone of ischemia.

Myocardial necrosis is detected by pathological Q-wave:

Pathological Q-wave is characterized by width≥0.04 s (in V_{4-6} >0.025 s), depth>2 mm or>1/4 R-wave (in V_{4-6} >15%R) (Fig. Suppl. 8).

Ischemic myocardium injury is detected by ST-interval:

-Transmural or epicardial injury - convexing elevation ST with transmission in T-wave (Fig. Suppl. 9a-b);

- Subendocardial injury – horizontal or concaving depression ST.

Ischemia of myocardium is detected by T-wave:

- Subendocardial ischemia – symmetrical acute high T-wave in overlying leads (>6 mm in standard and augmented leads, >8-10 mm in chest leads) (Fig. Suppl. 10);

- Transmural or epicardial ischemia - symmetrical acute deep T-wave.

The *S-T* segment and *T* wave change during the first hours of the disease (Table 9). The descending limb of the *R* wave transforms into the *S-T* segment without reaching the isoelectric line. The *S-T* segment rises above the isoelectric line to form a convexing arch and to coincide with the *T* wave. A monophase curve is thus formed. These changes usually persist for 3—5 days. Then the *S-T* segment gradually lowers to the isoelectric line while the *T* wave becomes negative and deep.

Table 9

Stages of myocardial infarction

Stage	Changes of ventricular complex	Duration of changes
Initial	S-T elevation in form a convex arch coincide with the T wave (monophase curve)	Hours –days from the beginning of infarct
Acute	Deep Q, small R-wave, negative T – wave begin be differentiated	3—5 (7) days – to 1-3 weeks
Subacute	Deep Q, S-T on isolectric line, negative ischemic (symmetrical) T	2-6 weeks from beginning of infarct
Reduction (cicatrization)	Penetrating and widened Q, negative T	Can remain persistant

A deep *Q* wave appears, the *R* wave becomes low or disappears at all. The *QS* wave is then formed, whose appearance is characteristic of transmural infarction.

Depending on localization of infarction, changes in the ventricular complex are observed in the corresponding leads (Table 10, Fig. Suppl. 11-12). The initial shape of ECG can be restored during cicatrization, or the changes may remain for the rest of life.

Table 10

Localization of myocardial infarction

Pathological changes of ventricular complex (Q-wave)	Localization of myocardial infarction
V1-4, I, aVL	Anterior wall of LV
V1-2	Anterior part of interventricular septum
I, aVL, V5-6	Lateral wall of LV
II, III , aVF	Posterior (inferior diaphragmatic) wall of LV
High R V1-2	Posterior (superior basal) wall of LV

Variability of ECG patterns with acute myocardial ischemia:

- *Non–infarction subendocardial ischemia* – transient ST depressions;
- *Non–infarction transmural ischemia* - transient ST elevation or paradoxical T-wave normalization, some times followed by T-waves inversions;
- *Non–Q-wave (non-ST elevation) infarction* - ST depressions or T-inversions without Q-wave*;*
- *Q-wave – infarction* - Q-wave with hyperacute T-waves/ST-elevations followed by T-waves inversions.

Exercise stress ECG testing:

Because the diagnosis of angina pectoris is usually primarily based on the patient's history, exercise testing in a patient with typical symptoms is generally used to determine functional and ECG response to graded stress (for exercise stress testing using radionuclide imaging; for exercise testing in asymptomatic persons to determine fitness for exercise programs, see below).

The patient exercises to a predetermined goal (e.g., 80 to 90% of maximal heart rate, which can be approximated as 220 less the age in years), unless distressing cardiovascular symptoms (dyspnea, reduced endurance, fatigue, hypotension, or chest pain) supervene. The ischemic ECG response during or after exercise is characterized by a flat or downward-sloping ST segment depression>0.1 millivolts (1 mm on the ECG when properly calibrated) lasting > 0.08 sec.

ECG diagnosis of sinus node automatism disorders, disordered myocardial conduction and excitability

Cardiac arrhythmia

Arrhythmias are deviations from the normal rhythm of the heart. Cardiac arrhythmias are common in many organic and functional disorders of circulatory system.

Causes of cardiac arrhythmias include:

(1) Affected automaticity of the sinus node;

(2) Foci of increased activity in the myocardium can generate impulses to initiate heart contractions apart sinus node (ectopic arrhythmia);

(3) Disorders of cardiac conduction system, local conduction disorder (re-entry mechanism);

(4) Combined changes in several functions of the heart such as automaticity, excitability, conduction or contractility.

Re-entry mechanism according to up-to-date view of point is a most common cause of cardiac arrhythmia. *Re-entry mechanism* means a pathological circuit of impulse because of:

(1) electrophysiological inhomogeneity (i.e., differences in conduction and/or refractoriness) in two or more regions of the heart connected with each other to form a potentially closed loop;

(2) unidirectional block in one pathway;

(3) slow conduction over an alternative pathway, allowing time for the initially blocked pathway to recover excitability;

(4) reexcitation of the initially blocked pathway to complete a loop of activation.

Classification of cardiac rhythm disorders (arrhythmias):

1. Arrhythmias associated with altered automaticity of the sino-atrial node (sinus arrhythmia).
2. Ectopic (heterotopic) arrhythmias.
3. Arrhythmias due to disordered myocardial conduction (heart blocks).
4. Combined cardiac rhythm disorders.

Sinus arrhythmia

Sinus arrhythmia -arrhythmias associated with altered automaticity of the sinus (sinoatrial) node.

When automaticity of the sino-atrial node is upset, the rate of impulse generation may either accelerate (sinus tachycardia) or slow down (sinus bradycardia), or the sequence of impulses may be changed with their generation at irregular intervals (sinus arrhythmia).

Normal sinus rhythm characteristics (Fig. Suppl. 13):

- HR (heart rate) equals 60-80 in min, regular rhythm (differences between minimal and maximal R-R intervals is not more than 15%);
- P wave - positive in I, II, aVF, P wave – negative in aVR, PQ $\geq$0.12 s.

Sinus tachycardia

Sinus tachycardia is directly connected with effects of biologically active substances which increase excitability of the sinoatrial node. This phenomenon may also depend on the change in the tone of the vegetative nervous system. It develops with intensified effect of the sympathetic nervous system. The rate of cardiac contractions in sinus tachycardia usually varies from 90 to 120 and sometimes to 150-160 per min. Sinus tachycardia develops during meals, physical exertion and emotional stress. At elevated body temperature, the heart rate increases by 8-10 per min per each degree over 37 °C. Sinus tachycardia is a frequent symptom of myocarditis, heart defects, and other diseases. It develops by reflex mechanism in heart failure and in response to the increased pressure in the orifices of venae cavae. Tachycardia often develops in neurosis, anemia, hypotension, and in many infectious diseases and toxicosis; it can be provoked by some pharmacological preparations (adrenaline, caffeine, atropine sulphate, etc.), and in thyrotoxicosis.

The clinical signs of sinus tachycardia are heart palpitation and accelerated pulse.

ECG signs of sinus tachycardia (Fig. Suppl. 14)*:*

- acceleration of heart rate from 90-100 up to 160-180 in one minute;
- P-wave of the normal form precedes complex QRS;
- regular accelerated rhythm (all intervals R-R are identical).

Sinus bradycardia

Sinus bradycardia is connected with slowed excitation of the sino-atrial node, which in turn depends mostly on the increased influence of the parasympathetic nervous system on the heart (or decreased influence of the sympathetic nervous system). Automaticity of the sino-atrial node decreases in sclerotic affections of the myocardium and in the cold. The cardiac rate in sinus bradycardia decreases to 50-40 (in rare cases to 30) beats per min, Bradycardia may occur in well-trained athletes. It is not permanent and the heart rhythm is accelerated during exercise as distinct from pathological bradycardia in atrioventricular block when bradycardia persists during and after exercise. If automaticity of the sino-atrial node sharply decreases (sick-sinus syndrome), the second- or third-order centres may function as the pacemaker, i.e. ectopic arrhythmias develop (see below).

Sinus bradycardia occurs in increased intracranial pressure (tumour and edema of the brain, meningitis, cerebral hemorrhage), in myxedema, typhoid fever, jaundice, starvation, lead and nicotine poisoning, and due to effect of quinine and digitalis preparations. It may develop by reflex during stimulation of baroreceptors of the carotid sinus and the aortic arch in essential hypertension, and can be provoked by pressure on the eye-ball (Dagnini-Aschner reflex), or by irritation of receptors of the peritoneum and the internal organs.

Mild bradycardia is not attended by any subjective disorders, nor does it produce any effect on the circulation. Marked bradycardia (under 40 beats per min) may cause nausea and loss of consciousness due to cerebral anemia. Objective examination reveals slow pulse.

ECG signs of sinus bradycardia (Fig. Suppl. 15)*:*

- decrease of a heart rate less than 60 in one minute;
- P-wave has a normal form;
- regular infrequent rhythm.

Sinus (respiratory) arrhythmia

Sinus arrhythmia characterized by irregular generation of impulses is due to variations in the tone of the vagus. It would commonly be associated with respiratory phases *(respiratory arrhythmia):* the cardiac rhythm accelerates during inspiration and slows down during expiration. Sinus arrhythmia is observed in children and adolescents (juvenile arrhythmia), in

patients convalescing from infectious diseases, and in certain diseases of the central nervous system. It can be a sign of pathology in rare cases when arrhythmia is not connected with respiration or when it develops in the aged during normal respiration.

Clinically sinus arrhythmia is not attended by any subjective disorders.

ECG signs of sinus arrhythmia

- different intervals R-R;
- P always precedes complex QRS;
- cardiac rhythm accelerates during inspiration and slows down during expiration.

Ectopic arrhythmias

Additional (heterotopic or ectopic) foci of excitation can arise at any site of the conduction system (in the atria, ventricles, atrioventricular region). They can cause premature contraction of the heart before termination of the normal diastolic pause. This premature contraction is called extrasystole, and the disorder of the cardiac rhythm is called *extrasystolic arrhythmia*. If the activity of the ectopic focus is very high, it can become a temporary pacemaker, and all impulses governing the heart will during this time be emitted from this focus. The cardiac rhythm is then markedly accelerated. The condition is known as *paroxysmal tachycardia*. Ectopic arrhythmias are often due to increased excitability of the myocardium. The phenomenon known as re-entry can be another mechanism of ectopic arrhythmia. If an impulse meets an obstacle in the pathway of its conduction (local conduction disorder), the excitation wave can return from this obstacle to excite the myocardium.

Extrasystolic arrhythmia (Ectopic beats, Extrasystoles)

Extrasystoles are premature cardiac beats resulting from an abnormal electrical focus or re-entry mechanism in the atria, AV (atrio-ventricular) junction and ventricles (Fig. Suppl. 16).

Extrasystole usually develops during normal contractions of the heart governed by the sino-atrial node (nomotopic contractions). Ectopic foci of excitation can arise at any site of the conduction system. Usually excitations arise in the ventricles, less frequently in the atria, the atrioventricular node, and in the sino-atrial node (sinus extrasystole). A nomotopic contraction of the heart that follows extrasystole occurs in a longer (than normal) interval of time. This can be explained as follows. During the atrial extrasystole, excitation from the ectopic focus is transmitted to the sino-atrial node to "discharge" it, as it were. The next impulse arises in the sino-atrial node only in a lapse of time that is required to "discharge" the node and to form a new impulse.

In ventricular extrasystole, the time between the extrasystolic contraction and subsequent nomotopic contraction is even longer. The impulse from the heterotopic focus, located in the ventricles, propagates only over the ventricular myocardium; it would not be usually propagated to the atria via Aschoff-Tawara (AV) node. The impulse occurs in normal time in the sino-atrial node but it is not transmitted to the ventricles because they are refractory after the extrasystolic excitation. The next impulse from the sino-atrial node will only excite and contract the atria and the ventricles. A long "compensatory" pause therefore follows the ventricular extrasystole which lasts till the next nomotopic contraction.

Extrasystolic arrhythmia is quite common. *Functional extrasystole* may occur in practically healthy individuals as a result of overexcitation of certain sites of the conduction system due to the action of the extracardiac nervous system in heavy smokers and in persons abusing strong tea or coffee; it can occur by reflex in diseases of the abdominal organs, or it may be due to hormonal disorders (thyrotoxicosis, menopause), various intoxications, disorders of electrolyte metabolism, etc. *Organic extrasystole* often attends various cardiovascular pathological conditions due to inflammatory or dystrophic affections of the myocardium or its deficient blood supply.

Patients with extrasystole can feel their heart missing a beat (escape beat) and a subsequent strong stroke. Auscultation of the heart reveals its premature contraction with a specific loud first sound (due to a small diastolic filling of the ventricles). Extrasystole can be easily revealed by feeling the pulse: a premature weaker pulse wave and a subsequent long pause are characteristic. If extrasystole follows immediately a regular contraction, the left ventricle may be filled with blood very poorly and the pressure inside it may be so small that the aortic valve would not open during the extrasystolic contraction and the blood will not be ejected into the aorta. The pulse wave on the radial artery will not be then detectable (missing pulse).

ECG characteristics of all extrasystoles:

(1) premature appearance of the cardiac complex;

(2) elongated pause between the extrasystolic and subsequent normal contraction;

(3) Compensatory pause - the sum of pre-extrasystolic and post-extrasystolic intervals. Complete compensatory pause equals to 2 normal R-R intervals. Incomplete compensatory pause is lesser than 2 normal R-R intervals.

If excitability of the myocardium is high, several (rather than one) ectopic foci may exist. Extrasystoles generated in various heart chambers and having different configuration then appear on the ECG *(polytopic extrasystole).*

Wherever an ectopic focus may arise, its impulses may alternate in a certain order with the normal impulses of the sino-atrial node. This phenomenon is known as *allorhythmia.* Extrasystole may alternate with each sinus impulse (*bigeminy*), or it may follow two normal impulses (*trigeminy*), or three normal impulses (*quadrigeminy*). If the heterotopic focus is even more active, a normal contraction may be followed by several extrasystoles at a run (*group extrasystole*), which sometimes precedes an attack of paroxysmal tachycardia.

Classification of extrasystolic arrhythmia

1. According to the origin: functional and organic extrasystoles.

2. According to the site of origin: -atrial, atrioventricular (nodal) (common name of supraventricular); - ventricular (left- and right-ventricular).

3. According to quantity of ectopic beats:

-single, group, paired, frequent extrasystoles;

-allorhythmia - alternaton of extrasystoles with sinus beats - bigemeny (1:2), trigeminy (1:3), quadrigeminy (1:4).

4. According to quantity of ectopic foci:

- monotopic and polytopic (polymorphic) extrasystoles**.**

Atrial extrasystole

Excitation of the atria only changes in atrial extrasystole because the impulse is generated not in the sino-atrial node, and the ventricles are excited by the usual way.

ECG signs of atrial extrasystole (Fig. Suppl. 17)*:*

(1) premature appearance of the cardiac complex;

(2) *P* wave - normal shape or slightly disfigured and superimposed on the preceding *T* wave;

(3) normal shape of QRS;

(4) slight elongation of the diastolic pause *(T-P* interval) following the extrasystolic contraction (incomplete compensatory pause.

Atrioventricular (nodal) extrasystole (premature atrioventricular junctional complex)

In atrioventricular (nodal) extrasystole the excitation of the atria differs from normal more substantially than in atrial extrasystole. The AV node impulse is transmitted to the atria retrogradely, from bottom to top. The ventricles are excited in nodal extrasystole in the usual way.

ECG signs of atrioventricular extrasystole (Fig. Suppl. 18)*:*

(1) premature appearance of the cardiac complex;

(2) negative *P* wave because of the retrograde atrial excitation (may be absence of *P* wave);

(3) the position of the *P* wave with respect to the ventricular complex depends on the rate of propagation of the excitation wave onto the atria and the ventricles;

(4) shape of QRS is normal or slightly deformed;

(5) incomplete compensatory pause.

Ventricular extrasystole (ventricular ectopic beat)

Heart excitation order changes sharply in *ventricular extrasystole*. *T*he ventricular impulse is not usually transmitted retrogradely through the AV node and the atria are not therefore excited. Second, the ventricles are not excited synchronously (as in normal cases), but one after another, i.e. that ventricle is excited first where the ectopic focus is located. The time of excitation of the ventricles is therefore longer and the *QRS* complex wider. The ventricular extrasystole is followed by a long (full) compensatory pause (except in interpolated extrasystoles): the atria are only excited by the sinus impulse that follows the extrasystole because the ventricles are refractory at this moment. The *P* wave corresponding to the atrial excitation is "lost" in the disfigured extrasystolic ventricular complex. Only next (second to the extrasystole) sinus impulse excites both the atria and the ventricles, while the EGG shows a normal cardiac complex.

ECG signs of ventricular extrasystole (Fig. Suppl. 19):

(1) premature appearance of the ventricular complex;

(2) absence of the *P w*ave;

(3) deformation of *QRS* complex due to increased voltage and length;

(4) the shape and the height of the *T* wave changes, its direction is opposite to the maximum wave of the *QRS* complex (*T* wave is negative if the *R* wave is high, and positive if the *S* wave is deep;

(5) complete compensatory pause.

Sometimes it is possible to determine in which particular ventricle the ectopic focus is located. This can be done from the configuration of the ventricular complex in various ECG leads. Left-ventricular extrasystole is characterized by a high *R* wave in III standard lead and the deep S wave in I lead. In right-ventricular extrasystole, the extrasystolic complex is characterized by a high *R* wave in I lead, and a deep *S* wave in the III.

Chest leads are very important for the topic diagnosis of ventricular extrasystole. Left-ventricular extrasystoles are characterized by the appearance of the extrasystolic complex with a high *R* wave in the right chest leads and a broad or deep S wave in the left chest leads. In right-ventricular extrasystole, on the contrary, the deep S wave is recorded in the right chest leads, and a high *S* wave in the left chest leads.

Paroxysmal tachycardia (PT)

Paroxysmal tachycardia is a sudden acceleration of the regular cardiac rhythm with more than three complexes (to 140-250 beats per min) resulting

from an abnormal ectopic impulses or re-entry mechanism in the atria, AV junction and ventricles.

At attack of paroxysmal tachycardia may last from several seconds to a few days and terminate just as unexpectedly as it begins. During an attack, all impulses arise from a heterotopic focus because its high activity inhibits the activity of the sino-atrial node. Paroxysmal tachycardia (like extrasystole) may occur in subjects with increased nervous excitability, in the absence of pronounced affections of the heart muscle, but it arises more likely in the presence of a severe heart disease (e.g. myocardial infarction, heart defects or cardiosclerosis).

During an attack of paroxysmal tachycardia, the patient feels strong palpitation, discomfort in the chest, and weakness. The skin turns pale, and if attack persists, cyanosis develops. Paroxysmal tachycardia is characterized by swelling and pulsation of the neck veins, because during accelerated pulse (to 180-200 per min) the atria begin contracting before the ventricular systole ends. The blood is ejected back to the veins from the atria to cause pulsation of the jugular veins. Auscultation of the heart during an attack of paroxysmal tachycardia reveals decreased diastolic pause, whose length nears that of the systolic one, and the heart rhythm becomes fetal (pendulum). The first sound increases due to insufficient ventricular diastolic filling. The pulse is rhythmic, very fast, and small. Arterial pressure may fall. If an attack persists (especially if it develops in the presence of a heart disease) symptoms of cardiac insufficiency develop.

Like in extrasystole, the heterotopic focus in paroxysmal tachycardia may be located in the atria, the atrioventricular node, and the ventricles. It is possible to locate the focus only by electrocardiography: a series of extrasystoles follow on an ECG at regular intervals and at a very fast rate

Classification according to the site of origin:

- atrial, atrioventricular (nodal) (common name of supraventricular) PT; - ventricular PT.

Supraventricular paroxysmal tachycardia characteristics (Fig. Suppl. 20):

-Sudden acceleration of the regular cardiac rhythm to 140-250 per min and sudden deceleration of HR;

- QRS shape is not changed as rule (or slightly deformed);

-P wave – disfigured (or biphase, negative) prior to QRS in atrial PT or follows QRS in AV nodal PT, or is not differed.

Ventricular paroxysmal tachycardia is characterized (Fig. Suppl. 21):

-Sudden acceleration of the regular cardiac rhythm to 140-220 per min and sudden deceleration of HR;

-QRS shape is changed and broadened (>0.12 s); ventricular complexes are similar to those in ventricular extrasystoles;

-P waves not correspond to ventricular complexes QRS (atrioventricular dissociation) or are not differed on ECG.

Torsade de pointes is a one of the most serious variants of ventricular PT characterized by a continuously changing vector of QRS, may be as predictor of ventricular flutter, ventricular fibrillation and cardiac arrest.

Ventricular flutter and fibrillation

Ventricular flutter usually appears as a sinusoidal wave with a rate between 150 and 300 per minute. *Ventricular fibrillation is* a rapid irregular ventricular rhythm due to multiple re-entrant activities associated with essentially zero cardiac output. It is a variant of cardiac arrest (Fig. Suppl. 23). The absence of adequate ventricular systole and contraction of separate ventricular muscles cause pronounced disorders in the hemodynamic and rapidly lead to death. Ventricular fibrillation occurs in grave affections of the myocardium (diffuse myocardial infarction, etc.).

The patient loses consciousness, becomes pallid, the pulse and arterial pressure become indeterminable. The ECG shows abnormal complexes on which separate waves are distinguished with difficulty

Ciliary arrhythmia

Ciliary arrhythmia includes two variants: a*trial fibrillation and atrial flutter*. According to course of ciliary arrhythmia is divided on paroxysmal and persistent forms of ciliary arrhythmia.

Atrial fibrillation

Atrial fibrillation is rapid irregular atrial rhythm due to multiple reentrant wavelets. Atrial fibrillation otherwise known as *complete or absolute arrhythmia*. It arises in cases with suddenly increased excitation of the myocardium and simultaneous conduction disorders. The sino-atrial node fails to function as the pacemaker and many ectopic excitation foci (to 600-800 per min) arise in the atrial myocardium, which becomes only possible with a marked shortening of the refractory period. Since conduction of these impulses is difficult, each of them only excites and causes contraction of separate muscular fibres rather than the entire atrium. As a result, minor contractions develop in the atrium (atrial fibrillation) instead of adequate atrial systole.

Only part of the impulses is transmitted to the ventricles through the AV node. Since conduction of atrial impulses is irregular, the ventricles contract at irregular intervals to cause complete arrhythmia of the pulse.

Depending on the conductability of the AV node and according to rate of ventricular contractions three forms of atrial fibrillation are distinguished:

- normasystolic form - 60-100 per min;
- tachysystolic form ->100 per min;
- bradysystolic form - <60 per min.

Fibrillation is characteristic of mitral heart diseases (especially of mitral stenosis), coronary atherosclerosis, thyrotoxicosis, etc. Fibrillation may occur as a permanent symptom or in attacks of tachyarrhythmia. Clinically fibrillation (bradyarrhythmia) may cause no subjective symptoms. Tachyarrhythmia is usually characterized by palpitation. Examination of the heart reveals complete irregularity of the heart contractions. Variations in the length of diastole account for variations in ventricular filling and hence in the intensity of the heart sounds. The pulse is also arrhythmic, pulse waves vary in height (irregular pulse), and pulse deficit often develops in frequent heart contractions.

ECG characteristics of atrial fibrillation (Fig. Suppl. 24):

(1) *P* wave disappears,

(2) multiple small irregular f waves,

(3) QRS ventricular complexes follow are irregular, their shape does not change.

Atrial flutter

Atrial flutter is a rapid regular atrial rhythm due to a constant well-defined macro-reentrant circuit in the right atrium. Atrial flutter is the upset cardiac rhythm, which nears in its pathogenesis to fibrillation. As distinct from fibrillation, the number of impulses arising in fluttering atria does not usually exceed 250-300 per min, and their conduction through the AV node is usually rhythmic. As a rule, not all atrial impulses are conducted to the ventricles. Each other, third or fourth impulse, is only conducted to the ventricles since partial (incomplete) atrioventricular block develops simultaneously. Conduction of the AV node sometimes constantly changes: each other impulse is now conducted; then the rhythm changes to conduction of each third impulse, and the ventricles contract arrhythmically. Like fibrillation, atrial flutter occurs in mitral defects, coronary atherosclerosis, and thyrotoxicosis; flutter sometimes develops in poisoning with quinine or digitalis.

Patients with accelerated heart rate (high conduction of the AV node) complain of palpitation. Examination reveals tachycardia that does not depend on the posture of the patient, exercise or psychic strain, since the sino-atrial node does not function as the pacemaker in atrial flutter (being governed by extracardial nerves). Heart contractions are arrhythmic in patients with varying conduction of the AV node.

ECG characteristics of atrial flutter (Fig. Suppl. 25):

(1) high F waves instead of the normal atrial *P* waves;

(2) The number of F waves preceding each ventricular complex depends on the AV conduction;

(3) QRS complexes follow at regular intervals.

Conduction disorders (heart blocks)

Heart blocks are delayed conduction or complete absence of conduction in some department of cardiac conduction system.

Transmission of the impulse may be blocked at any part of the heart's conduction system. Block may develop in inflammatory, dystrophic, and sclerotic affections of the myocardium (e.g. rheumatic and diphtheritic myocarditis or cardiosclerosis). The conduction system may be affected by granulomas, cicatrices, toxins, etc. Conduction is often impaired in disordered coronary circulation, especially in myocardial infarction (the interventricular septum is involved). Block may be persistent and intermittent. Persistent block is usually connected with anatomic changes in the conduction system, whereas intermittent block depends largely on the functional condition of the atrioventricular node and the His bundle and is often connected with increased influence of the parasyrnpathetic nervous system; atropine sulphate is an effective means that restores conduction.

Clinical and ECG signs of block depend on its location.

The following *types of heart blocks* are distinguished:

- *sino-atrial block* - impairment of conduction between sinus node and atria;
- *intra-atrial block* - impairment of conduction through the atrial myocardium;
- *atrioventricular block* - impairment of conduction between atria to the ventricles;
- *intraventricular block (His bundle branch block)* - impairment of conduction through the His bundle and its branches.

Sino-atrial block

Sino-atrial block is characterized by periodic missing of the heart beat and pulse beat.

ECG signs of sino-atrial block (Fig. Suppl. 26):

-periodic missing of the heart complex (PQRST) in the presence of a regular sinus rhythm;

-the length of diastole doubles.

Intra-atrial block

Intra-atrial block can only be detected electrocardiographically because clinical signs are absent.

ECG signs of intra-atrial block (Fig. Suppl. 27):

- P waves are broadened $\geq$0.11 s and splitted;
- P wave in the V_1 lead has two phases.

Atrioventricular block

Atrioventricular block is most important clinically. It is classified into three degrees by gravity.

I degree of AV block

This block cannot be detected clinically, except that splitting of the first sound may sometimes be detected by auscultation (splitting of the atrial component) (Fig. Suppl. 28).

I degree of AV block can only be revealed electrocardiographically:

- increased *P-Q* interval (> 0.21 s to 0.3-0.4 s and more) without missing QRS;
- regular heart rhythm.

II degree of AV block with Samoilov-Wenckebach periods (Mobitz-1 type)

Conduction of the AV node and His bundle is impaired: each impulse transmitted from the atria to the ventricles increases and the P-Q interval on the ECG becomes longer with each successive beat. A moment arrives at which one impulse does not reach the ventricles and they do not contract, hence the missing QRS complex on an ECG. During a long diastole, which now follows, the conduction power of the atrioventricular system is restored, and next impulses will again be transmitted, but their gradual slowing down will be noted again; the length of the-P-Q interval will again increase in each successive complex. The length of diastole which follows the P wave is called the *Samoilov- Wenckebach period*. This type of block is characterized clinically by periodically missing ventricular contractions, and hence missing pulse beats, which correspond to the Samoilov-Wenckebach period.

ECG signs of II degree of AV block with Samoilov-Wenckebach periods (Mobitz-1 type) (Fig. Suppl. 29):

- gradual elongation PQ (Samoilov-Wenckebach periods);
- periodically missing ventricular contractions.

II degree of AV block (Mobitz-2 type)

The second-degree atrioventricular block can be characterized by a worse conduction. The *P-Q* interval remains constant, but only each second, third, or *(*less frequently) fourth impulse is transmitted to the ventricles. The number of *P* waves on the ECG is therefore larger than of ventricular complexes. This is known as incomplete heart block with a 2:1, 3:1, etc. ratio. Considerable deceleration of the ventricular rhythm and slow pulse are characteristic, especially in 2:1 block. If each third or fourth beat is missing, the pulse is irregular and resembles trigeminy or quadrigeminy with early extrasystoles and pulse deficit. If the heart rhythm slows down significantly, the patient may complain of giddiness, everything going black before his eyes, and transient loss of consciousness due to hypoxia of the brain.

ECG signs of II degree of AV block *(*Mobitz-2 type) (Fig. Suppl. 30):

- periodic missing QRS without gradual elongation PQ (2:1, 3:1);
- PQ can be normal or a little bit prolonged.

III degree of AV block (complete heart block)

There is no electrical communication between the atria and the ventricles, and sinus node becomes the only pacemaker for the atria. The ventricles contract by their own automaticity in the centres of the second or third order (about 30-40 per min).

ECG signs of complete heart block:

1) atrial *P* waves and ventricular complexes QRS are recorded independently of each other;

2) the number of QRS is usually much smaller than the number of atrial P waves;

3) the shape of the ventricular complex does not change if the pacemaker arises from the AV node or His bundle;

4) with lower location of the pacemaker in the conduction system, the *QRST* complexes are altered.

The heart rate in persistent complete heart block may be sufficiently high (40- 50 beats/min) but the patient may be unaware of the disease for a long time. Examination of such patients reveals slow, rhythmic, and full pulse. The heart sounds are dulled but a loud first sound ("pistol-shot" sound according to Strazhesko) may be heard periodically. It occurs due to coincidence of the atrial and ventricular contractions. If the ventricular rhythm slows down significantly (to 20 beats/min and less), or the heart misses a beat when incomplete heart block converts into a complete one, i.e. when the impulses from the atria are not conducted to the ventricles, while their automaticity has not yet developed, attacks (the Morgagni-Adams-Stokes syndrome) may occur due to disordered blood supply, mainly to the central nervous system. During an attack the patient loses consciousness, falls, general epileptiform convulsions develop, the respiration becomes deep, the skin pallid, the pulse very slow or even impalpable. When the ventricular automaticity restores, the patient regains his consciousness and all other signs of the syndrome disappear. If automaticity is not restored for a time, fatal outcome is possible.

His bundle branch blocks

Intraventricular block usually develops as the right or left bundle-branch block. The left limb of the His bundle ramifies almost immediately to give left anterior and left posterior branches. Only one branch can therefore be blocked. Block of the right limb may be combined with block of the branches of the left limb. In complete block of either of the limbs, the impulse from the sino-atrial node is normally conducted through the AV node and the main part of the His bundle to meet an obstacle to its conduction in that ventricle whose branch is affected. The ventricle with the intact branch is therefore first excited and excitation is transmitted to the

ventricle with the affected branch. The ventricles are thus excited slowly and in an unusual way.

ECG signs of His bundle branch blocks:

(1) *P* wave does not change;

(2) ventricles contract rhythmically by the impulse from the sinus node;

(3) *QRS* complexes are markedly altered and widened ≥ 0.12-0.18 s and resemble complexes in ventricular extrasystole;

(4) The shape of the ventricular complexes depends on the particular bundle branch which is blocked.

ECG in left bundle branch block is characterized (Fig. Suppl. 32):

-wide and deformed QRS has the form of qR in I, II, V_{5-6}; rS in III, aVF, V_{1-2} (the shape of the ventricular complexes resembles that of right ventricular extrasystoles);

-disconcordance of ST, T and the main wave of QRS;

- levogram.

ECG in right bundle branch block is characterized (Fig. Suppl. 33)*:*

- wide QRS in III, V_{1-2} has the form of rsR, rSR, RsR´(similar to "M") (the shape of the ventricular complexes resembles that of left-ventricular extrasystoles);
- wide S in I, aVL,V_{5-6};
- negative ST and T in V_{1-2};
- dextrogram.

Examination of patients with diseases of esophagus, stomach and intestines:

Subjective examination of patients. Objective examination of patients: survey, percussion and auscultation of abdomen

Subjective investigation in diseases of digestive system

Complaints of patients with diseases of a digestive system depend on the organ having the pathological changes. Pains and dyspeptic symptoms are the most often complaints at diseases of the digestive system. It is necessary to distinguish, however, pains from a heavy feeling, strains, distension or pressure in an abdomen, which patients quite often combine in one concept of "pain".

Detailed elaboration of a pain syndrome includes: localization of a pain with statement of the following questions: character of sensation; periodicity or a persistence of it; connection with reception of nutrition (or defecation, physical and emotional activity) and its quality; presence or absence of

irradiation; a position of the patient at the moment of an attack; how pains are revealed (a vomiting, medicines, a heater).

Dyspepsia means literally «disorders of digestion». Dyspeptic symptoms connect with affections of digestion and/or passage of nutrition in definite parts of alimentary tract.

Subjective investigation in diseases of esophagus

Complaints

Dysphagia (difficult passage of food via the esophagus) is the most frequent symptom of esophageal pathology. The patient feels difficulty in swallowing (mostly solid food); the food bolus sometimes stops in the esophagus and the patient feels pain and esophageal distention, sensation of a lump in a throat or thorax, sense of pressure in range of an esophagus or difficulty of transit of nutrition.

Dysphagia can be due to organic or functional narrowing of the esophagus. Organic stenosis develops gradually and progresses in cancer, and cicatricial stenosis of the esophagus. Solid food first passes with difficulty, then the patient feels difficulty in swallowing soft, and then liquid food. When cancer tumour disintegrates, patency of the esophagus may be restored almost completely. Dysphagia develops immediately in the presence of a foreign body or if the esophagus is burnt. Dysphagia may also develop due to compression from outside by an aortic aneurysm or mediastinal tumour.

Functional narrowing of the esophagus is explained by muscular spasms caused by reflex disorders of innervation of the esophageal muscles, or by neurosis. As distinct from organic dysphagia, functional dysphagia more often occurs in paroxysms when food passes the esophagus. Sometimes solid food passes more readily than liquid.

Pain occurs in acute inflammation of the esophageal mucosa (esophagitis) and in burns. The patient usually feels pain by the course of the entire esophagus, both with and without swallowing; pain may radiate into the interscapular region.

Patients with achalasia of the cardia (cardiospasm) may have spontaneous attacks of pain, usually during night. Pain is quite severe; it radiates into the back, upwards by the esophagus, into the neck, the jaws, and continues for minutes and even hours. In the presence of hiatus hernia and gastroesophageal reflux, pain may radiate into the left side of the chest and simulate heart diseases.

Esophageal vomiting occurs in considerable narrowing of the esophagus. Food is accumulated over the constricted point, in the wider portion of the esophagus, and is expelled by antiperistaltic contractions of the muscles, Esophageal vomiting differs from gastric vomiting in the following: it occurs without nausea and is preceded by the feeling of food retained behind the sternum; the vomitus includes unaltered (non-digested) food

which contains neither hydrochloric acid (gastric juice) nor pepsin; the vomitus containing food that has been taken long time ago has foul odour; taken food can be retained for long periods in the presence of esophageal diverticulum or degrading cancer.

Regurgitation is the return of swallowed food into the mouth due to esophageal obstruction. Regurgitation sometimes occurs in neuropathic patients in whom it becomes a habitual symptom or a result of cardiospasm.

Hypersalivation occurs in esophagitis, cicatricial narrowing of the esophagus or in cancerous stenosis as a result of the esophago-salivary reflex.

A foul breath may be due to a cancer tumour of the esophagus or congestion and decomposition of food in cardiospasm.

Heartburn (pyrosis) is a specific burning sensation behind the sternum associated with regurgitation of gastric contents into the inferior portion of the esophagus. This is the cause of the so-called reflux esophagitis.

Hemorrhage can be due to ulcer of the esophagus, injury to the esophagus by a foreign body, degradation of a tumour, bleeding of dilated esophageal veins (which occurs in congestion of blood in the portal vein system), and also bleeding of the mucosa due to small lacerations of the vessels in the esophagogastric junction in straining and vomiting (Mallory-Weiss syndrome).

History of present disease

In organic affections of the esophagus, the disease has a progressive course. Functional diseases (cardiospasm) are characterized by exacerbations connected with psychogenic factors which are followed by remissions. From the anamnesis of the patient's life it can be established whether the patient had past burns of the esophagus, since acid or alkali burns are frequent causes of cicatricial changes in the esophagus. Syphilis is an important disease of the past history since the patient's complaints of dysphagia can sometimes be associated with syphilitic aortitis (compression of the esophagus by a dilated aorta). Development of traction diverticula in the esophagus may be due to bronchoadenitis in the past (tuberculous bronchoadenitis included). Pulsion diverticula arise due to esophagospasm.

Subjective investigation in diseases of stomach and duodenum

Complaints

Patients with diseases of the stomach complain of poor appetite, perverted taste, regurgitation, heartburn, nausea, vomiting, epigastric pain, and hematemesis. Regurgitation, heartburn, nausea, vomiting, and the feeling of overfilled stomach after meals are the group of the so-called dyspeptic complaints. These symptoms may be observed in diseases of some other organs and systems. 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. This symptom is often an early sign of cancer. Appetite often increases in peptic ulcer, especially in duodenal ulcer. Loss of appetite should be differentiated from cases when the patient abstains from food for fear of pain (*citophobia*). This condition often occurs in subjects with gastric ulcer, though their appetite is increased.

Perverted appetite that sometimes occurs in patients is characterized by the desire to eat inedible materials such as charcoal, chalk, kerosine, etc.

Appetite is perverted in pregnant women and in persons suffering from achlorhydria. Some patients with cancer of the stomach or some other organs often feel aversion to meat. The developmental mechanism of appetite is connected with excitation of the food centre (according to Pavlov). Excitation or inhibition of this centre depends on impulses arriving from the cerebral cortex, on the condition of the vegetative centres (excitation of the vomiting centre causes loss of appetite), and on reflex effects from the alimentary organs. The multitude of factors that act on the food centre accounts for the high variation in appetite.

Taste may be perverted due to the presence of unpleasant taste in the mouth and partial loss of taste in an individual. It can often be associated with some pathology in the mouth, e.g. caries or chronic tonsillitis. A coated tongue can be another cause of unpleasant taste in the mouth.

Regurgitation usually implies two phenomena: a sudden and sometimes loud uprise of wind from the stomach or esophagus (eructation), and the return of swallowed food into the mouth (sometimes together with air). Regurgitation depends on contraction of the esophageal muscles with the open cardia. Regurgitation may be due to air swallowing (*aerophagy*). It is heard at a distance and occurs in psychoneurosis. In the presence of motor dysfunction of the stomach, fermentation and putrefaction of food with increased formation of gas occur in the stomach (the phenomenon otherwise absent in norm). In abnormal fermentation in the stomach, the eructated air is either odourless or smells of bitter oil, which is due to the presence of butyric, lactic and other organic acids that are produced during fermentation in the stomach. In the presence of abnormal putrefaction, the belched air has the odour of rotten eggs (hydrogen sulphide). 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. But it can also occur in normal or insufficient secretion of the stomach in the presence of insufficiency of the cardia (when the stomach contents are regurgitated into the esophagus). Bitter regurgitation occurs in cases with belching up of bile

into the stomach from the duodenum, and also in hyperchlorhydria; bitterness depends on the bitter taste of peptone.

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

Nausea is a reflectory act associated with irritation of the vagus nerve, indefinite feeling of sickness and sensation of compression in the epigastrium. Nausea is often attended by pallidness of the skin, general akness, giddiness, sweating, salivation, fall in the arterial pressure, cold the limbs, and sometimes semisyncopal state. Nausea often (but not necessarily) precedes vomiting. The mechanism of nausea is not known. Its frequent association with vomiting suggests that it might be the early sign of stimulation of the vomiting centre. The leading role in the development of nausea is given to the nervous system and also the tone of the stomach, the duodenum, and the small intestine. Nausea may develop without any connection with diseases of the stomach, e.g. in toxemia of pregnancy, renal failure, deranged cerebral circulation, and sometimes in healthy people in the presence of foul odour (or in remembrance of something unpleasant). Some diseases of the stomach are attended by nausea, e.g. acute and chronic gastritis or cancer of the stomach. Nausea associated with gastric pathology usually occurs after meals, especially after taking some pungent food. Nausea often develops in secretory insufficiency 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 may be caused by ingestion of spoiled food, by seasickness, or irritation arising inside the body (diseases of the gastro-intestinal tract, liver, kidneys, etc.). In most cases vomiting is preceded by nausea and sometimes hypersalivation. Factors causing the vomiting reflex are quite varied. This can be explained by the numerous connections that exist between the vomiting centre (located in the medulla oblongata, in the inferior part of the floor of the 4-th ventricle) and all bodily systems. Depending on a particular causative factor, the following can be differentiated: (1) nervous (central) vomiting; (2) vomiting of visceral etiology (peripheral or reflex); (3) hematogenic and toxic vomiting.

Vomiting is an important symptom of many diseases of the stomach, it can be regarded as the symptom of a particular disease only in the sense of other signs characteristic of this disease. Vomiting of gastric etiology is caused by stimulation of receptors in the gastric mucosa by inflammatory processes (acute or chronic gastritis), in ingestion of strong acids or alkalis, or food acting on the gastric receptors by chemical (spoiled) or physical

(overeating or excessively cold food) routes. Vomiting can be caused by difficult evacuation of the stomach due to spasms or stenosed pylorus. If patient complains of vomiting, the physician should inquire the time when the vomiting occurred, possible connections with meals, association with pain, the amount and character of the vomited material. Morning vomiting (on a fasting stomach) with expulsion of much mucus is characteristic of chronic gastritis, especially in alcoholics, Hyperacid vomiting in the morning indicates nocturnal hypersecretion of the stomach. Vomiting occurring 10-15 minutes after meals suggests ulcer or cancer of the cordial 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. Expulsion of food taken a day or two before is characteristic of pyloric stenosis. Patients with peptic ulcer often vomit at the height of pain thus removing it, which is typical of the disease. The odour of the vomit is usually acid, but it can often be fetid (putrefactive processes in the stomach); the odour may be even fecal (in the presence of a fecal fistula between the stomach and the transverse colon).

The vomited material may have acid reaction (due to the presence of hydrochloric acid, in hyperchlorhydria), neutral (in achylia), or alkaline (in the presence of ammonia compounds, in pyloric stenosis, hypofunction of renal function, and also in regurgitation of the duodenal contents into the stomach). Vomitus may contain materials of great diagnostic importance, e.g. blood, mucus (in chronic gastritis), ample bile (narrowing of the duodenum, gastric achylia), and fecal matter. Vomiting may attend acute gastritis, exacerbation of chronic gastritis, gastric neurosis, peptic ulcer, spasm and organic stenosis of the pylorus, and cancer of the stomach.

Pain is the leading symptom in diseases of the stomach. Epigastric pain is not obligatory connected with diseases of the stomach. It should be remembered that the epigastrium is the "site of encounter" of all kinds of pain. Epigastric pain may be due to diseases of the liver, pancreas, and due to hernia of the linea alba. Epigastric pain may develop in diseases of other 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 (weight lifting, traffic jolting, etc.), or strong emotions. Intensity and character of pain are also important diagnostically. The pain may be dull, stabbing, cutting, etc. Pain in hollow organs with smooth muscles (e.g. stomach) is provoked by spasms (spastic pain), distension of the organ (distensional pain), and by its motor dysfuncion.

Paroxysmal, periodical epigastric pain is due to the spasm of the pyloric muscles. It arises under the influence of strong impulses arriving from the vagus nerve centre in cerebral cortex dysfunction. The spasm of the pylorus is stimulated by the hyperacidity of gastric juice due to hyperstimulation of the vagus.

Depending on the time of paroxysmal pain (after meals), it may be *early pain* (occurring 30-40 min after meals), *late pain* (90-120 min after meals), *nocturnal pain*, and *hunger pain* (which is abated after taking food). If pain occurs after meals stimulating secretion of gastric juice (bitter, pungent, spicy or smoked foods), this indicates the leading role of hypersecretion in its etiology. The pain then localizes in the epigastrium, radiates to the back, and is rather intense; it is abated after vomiting and taking alkali or foods that decrease acidity of gastric juice, and also after taking antispastic preparations and applying hot-water bottle (which removes spasms).

A seasonal character of pain, i.e. development of periodic pain during spring and autumn, is characteristic of peptic ulcer, especially if the process is localized in the peripyloric region. Permanent boring pain is usually caused by stimulation of the nerve elements in the mucous and submucous layer of the stomach; the pain is usually intensified after meals and is characteristic of exacerbation of chronic gastritis or cancer of the stomach.

Perigastritis (chronic inflammation of the peritoneum overlying the stomach and its adhesion to the neighbouring organs) is manifested by pain developing immediately after taking much food (irrespective of its quality). The full stomach distends to stimulate nerve fibres in the adhesions. In the presence of perigastritis and adhesions between the stomach and the adjacent organs, pain may be caused by any physical strain and when the patient changes his posture.

Gastric hemorrhage is a very important symptom. It can be manifested by vomiting of blood (hematemesis) or by black tarry stools (*melena*). Gastric hemorrhage 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 hematin hydrochloride. The vomitus looks like *coffee grounds*. If hemorrhage is profuse (damage to a large vessel) the vomitus contains much scarlet (unaltered) blood. Hematemesis 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 hemorrhage.

Anamnesis

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). Gastrointestinal diseases of the past, surgical intervention on the abdominal organs, long medication with preparations irritating the stomach mucosa (acetylsalicylic acid, sodium salycilate, steroid hormones, potassium chloride, etc.) are also very important.

Subjective examination in diseases of intestines

Complaints

The main complaints with intestinal diseases are pain, meteorism (inflation of the abdomen), motor dysfunction of the intestine (constipation and diarrhea), and intestinal hemorrhage.

Pain. If the patient complains of pain in the abdomen, the following should be established: location of pain, its radiation, intensity, character, duration, and means by which it is lessened. The general signs by which intestinal pain may be differentiated from gastric one are: (1) absence of regular dependence of pain on food taking; the only exception is inflammation in the transverse colon (*transversitis*): pain develops immediately after meals; the pathogenesis of this pain is connected with reflex peristaltic contractions of the transverse colon when food enters the stomach; (2) close association of pain with defecation: pain occurs before, during, and (rarely) after defecation; (3) pain relief after defecation or passage of gas.

Pain may be boring and spasmodic (intestinal colic). Colicky pain is characterized by short repeated attacks which arise and disappear quite of a sudden. Pain may very quickly change its location, the main site being round the navel. Sometimes pain may arise in other areas of the abdomen. Boring pain is sometimes permanent; it intensifies during cough, especially if the mesenterium or peritoneum is involved. Pain is characteristic of inflammatory diseases of the intestine. As inflammation extends onto the peritoneum, pain is attended by a pronounced muscular defence.

Exact location of the source of pain is very important. Pain in the right iliac region occurs in appendicitis, tuberculosis, cancer, or inflammation of the cecum (*typhlitis*). Acute pain in the left lower abdomen occurs in intestinal obstruction and inflammation of the sigmoid (*sigmoiditis*). Pain in the umbilical region occurs in inflammation of small intestine (*enteritis*) and inflammation or cancer of the colon. Pain in the perineal region, and especially during defecation (with the presence of blood in feces), is characteristic of the rectum diseases (proctitis, cancer). Pain in intestinal pathology may radiate into the chest; pain associated with affection of the spleen angle of the descending large intestine radiates into the left side of the chest (it is sometimes mistaken for pain attacks of angina pectoris); colics of appendicitic origin radiate into the right leg.

In acute affection of the left portions of the large intestine (dysentery), pain radiates into the sacral area. Thermal procedures, spasmolytics, passage of gas, and emptying of the bowels can relieve pain or remove it completely.

Intestinal pain is caused by obstruction of intestinal patency and upset motor function. Intestinal pain is mostly caused by spasms (spasmodic contraction of smooth muscles; hence *spastic pain*), or by distension of the intestine by gases. Both mechanisms often become involved.

Spastic pain can be due to various causes. Individual predisposition to spastic contractions in general (vegetoneurosis) may be as important as irritation originating in the intestine proper, e.g. in enteritis, colitis, intestinal tumour, poisoning with arsenic or lead, and also in diseases of the central nervous system (posterior spinal sclerosis).

Pain arising due to intestinal distension by gases, and associated with tension and irritation of the mesentery, differs from spastic pain (1) by the absence of periodicity; it is long-standing and gradually lessens in prolonged inflation; and (2) by exact localization. In intestinal obstruction (complete or partial) colicky pain is combined with almost permanent pain in the abdomen. It is characterized by exact and permanent location (the umbilical region and large intestine). The pain intensifies with intestinal peristalsis.

Appendicular colic first localizes round the navel and the epigastrium but in several hours (or even on the next day) it descends to the right iliac region where it intensifies gradually. Sometimes the pain arises straight in the right iliac region. *Rectal colic, or tenesmus*, is also known. It occurs in frequent and painful tenesmus to defecate and is associated with spasmodic contractions of the intestine and the sphincter ani. Only clots of mucus are sometimes expressed instead of actual defecation. Tenesmus occurs in dysentery and other inflammatory or ulcerous diseases, and in cancer of the rectum. Pain associated with defecation depends on many factors. Pain preceding defecation is associated with the disease of the descending colon or sigmoid colon. Pain during defecation is characteristic of hemorrhoids, anal fissures, and cancer.

Meteorism. The patient feels flatulence, inflation, and boring distension of the abdomen. The causes of meteorism are (1) excessive gas formation in the intestine due to ingestion of vegetable cellular tissue and easily fermented food (peas, beans, cabbage, etc.); (2) intestinal motor dysfunction due to decreased tone of the intestinal wall or intestinal obstruction; (3) lowered absorbability of gases by the intestinal wall, the process of gas formation being normal; (4) *aerophagia*, i.e. excess swallowing of air, with its subsequent propulsion to the stomach and the intestine; (5) hysterical meteorism: the abdomen is rapidly inflated to the size of the abdomen of a pregnant woman at her last weeks; this nervous mechanism is very complicated.

When inquiring the patient, the physician should ask about the character of his nutrition and the site of abdomen inflation (the entire abdomen or only its limited part may be inflated). If inflation is local, it is necessary to ask the patient whether or not inflation occurs always at one and the same area. In intestinal obstruction, the patient feels rumbling sounds inside the abdomen, feels movement of liquid in the intestine, and intense peristaltic movements above the point of obstruction.

Diarrhea. Frequent and liquid stool is a common sign of intestinal pathology. Diarrhea occurs in acute and chronic intestinal infections (enteritis, enterocolitis, sigmoiditis, proctitis), in various exogenous intoxications (poisoning with arsenic or mercury), endogenous intoxications (uremia, diabetes, gout), in endocrine disorders (adrenal dysfunction, thyrotoxicosis), and in hypersensitivity to some foods (allergy).

The mechanism of diarrhea is very complicated. Different pathogenic factors may prevail in various pathological conditions. Accelerated movement of the liquefied food in the intestine due to peristalsis is among them. Almost undigested food can thus be evacuated. Another factor is disordered absorptive function of the intestine. Affection of the intestinal wall, disordered mechanisms regulating absorption, purgatives and upset water metabolism produce a marked change in the absorption process and are the cause of diarrhea.

The third cause of liquid stools is inflammation of the intestine. Large quantities of inflammatory secretion stimulating the intestinal receptors are released into the lumen of the intestine to intensify its peristalsis and to impair its absorptive function.

Paradoxical diarrhea occurs in prolonged constipation due to mechanical irritation of the intestinal wall by hard fecal masses.

Upset equilibrium between the fermentative and putrefactive flora of the intestine is another important factor in the etiology of diarrhea.

Diarrhea occurring in organic affections of the large intestine is mostly of the inflammatory character. It is not copious, nor does it produce strong negative effect on the patient's general condition (as compared with

affections of the small intestine which is attended by profuse diarrhea associated with deranged motor and absorption function of the intestine). The pronounced disorder in digestion causes some metabolic disorders in the patient (impaired absorption of proteins, iron, vitamins, and electrolytes).

Obstipation (constipation). This is obstinate constipation during which feces are long retained in the intestine (for more than 48 hours). But the duration of constipation is only relative, because in many cases it is not the result of pathology but of the living conditions and nutrition. If vegetable food dominates in the diet, the subject may defecate two or three times a day. Stools become rarer if the diet is rich in meat. A radical change in nutrition can remove constipation. Limited mobility of the subject, hunger, and irregular defecations (during the day) may prolong pauses between defecation. The main factor determining defecation is the condition of intestinal motor function. Bowel contents are retained in the large intestine and the rectum during constipation

Organic and functional constipation is differentiated. *Organic constipation* is usually associated with mechanical obstruction, such as narrowing of the intestinal lumen due to a tumour, scar, adhesion, and also abnormalities in the intestine (megacolon, dolichosigmoid, megasigmoid, diverticulosis).

Functional constipation is subdivided into: (1) alimentary constipation; it occurs due to ingestion of easily assimilable foods, which leave small residue and normally stimulate peristalsis of the intestine by irritating its nervous receptors; (2) neurogenic constipation due to dysfunction of the intramural nervous apparatus or vagus nerve; these are the so-called dyskinetic constipation, caused by the reflex action on the intestinal motor function of another affected organ (cholecystitis, adnexitis, prostatitis, etc.), or by organic affections of the central nervous system (tumours of the brain, encephalitis, posterior spinal sclerosis); (3) constipation associated with inflammatory affections, mainly of the large intestine (dysentery); (4) toxic constipation occurring in exogenous poisoning with lead, morphine, or cocaine; (5) constipation of endocrine etiology, occurring in thyroid or pituitary hypofunction; (6) constipation caused by lack of physical exercise; (7) constipation caused by flaccidity of the prelum.

Intestinal hemorrhage often occurs in ulcerous affections of the alimentary system. It develops in the presence of tumour, protozoal and helminthic invasions, acute infections (typhoid fever, bacillary dysentery), in thrombosis of mesenteric vessels, ulcerous non-specific colitis, etc.

Anamnesis

The patient should be inquired thoroughly about his nutrition from his early childhood till the onset of the disease (especially directly before the disease), about poisonings in the past history and hypersensitivity to some feeds. It is necessary to find out if the patient's meals are regular, if the food

is varied, and if the patient smokes or drinks alcohol. Information on the past diseases of the intestine and also on pathology of other organs is sometimes decisive for establishing the cause of the present affection.

Some functional disorders of the intestine can be associated with occupation (lead or arsenic poisoning, constipation due to frequent suppression of tenesmus to defecate).

Objective examination of patients in diseases of digestive system

General survey of patients in diseases of digestive system

The general condition and state of consciousness of the patient are first assessed.

The general inspection of the patient with dysphagia may suggest an organic affection of the esophagus if the patient is extremely asthenic (*cachexia*). During general inspection of the patient with stomach diseases the physician may assess poor nutrition of the patient (cachexia) which is characteristic of stomach cancer and untreated benign pyloric stenosis. Patients with uncomplicated peptic ulcer look practically healthy. Severe prolonged affection of the absorptive function causes grave cachexia.

Pale skin is observed after gastric and intestinal hemorrhage, and in anemia. Edema is possible in loss of protein with simultaneous retention in the body of water and salt. Inspection of the skin reveals its dryness and pallidness; the mucosa is pale due to insufficient absorption of iron and anemization of the patient. Insufficient absorption of vitamins results in development of fissures of the lips, the skin becomes rough, and perleche develops.

Facies Hippocratica (first described by Hippocrates) is associated with collapse in grave diseases of the abdominal organs (diffuse peritonitis, intestinal obstructionб, 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.

Survey of oral cavity

Next stage is inspection of the mouth. 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 pyorrhea, acute leukemia, 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.

The absence of many teeth accounts for inadequate disintegration and mastication of food in the mouth, while the presence of carious teeth favours penetration of microbial flora into the stomach.

The tongue is not the "mirror of the stomach" as it was formerly believed. Nevertheless in some diseases its appearance is informative: clean and moist tongue is characteristic of uncomplicated peptic ulcer, while the tongue coated with a foul smelling white-grey material is characteristic of acute gastritis; a dry tongue indicates a severe abdominal pathology or acute pancreatitis; a tongue with atrophied papillae suggests cancer of the stomach, atrophic gastritis with pronounced gastric secretory hypofunction, or vitamin B deficiency. The glassy tongue is characteristic of gastric cancer, pellagra, sprue, and ariboflavinosis.

The tongue in intestinal diseases often becomes crimson (cardinal tongue) in vitamin PP deficiency (pellagra), its papillae are smoothed down. The gums may be loose and bleeding.

Disordered movement of the tongue may indicate nervous affections, grave infections and poisoning.

Survey of abdomen

Inspection of the abdomen should be done with the patient in vertical or lying position. Research of an abdomen in a vertical position begins with survey. Thus the doctor sits on a chair, and the patient faces the doctor, the person to him, completely having naked the abdomen.

For exact delimitation of localization of the signs revealed by objective inspection, abdomen conditionally part on some regions. Two horizontal lines (the first line bridges the tenth ribs, the second - the top edges of ileac bones) divide a front abdominal wall part on three departments, locating one under another: *epi-, meso-and hypogastric regions*. Two collateral vertical lines conducted on outside edges of rectus abdominis muscles divide epigastric region into two *subcostal (hypogastric) regions* (*right and left*) and (in more narrow sense) *epigastric region* posed in the middle; mesogastric - on two *lateral flancs (flanks)* and on *umbilical region*; hypogastric region - on two *inguinal (ileac) regions* locating on each side and *suprapubic region*.

At the beginning of survey the form of the abdomen is defined. In the healthy person the form of the abdomen substantially depends on his constitution.

The general outlines of the abdomen should be inspected. The abdomen can be of a normal shape with slightly protruding suprapubic region; it can be enlarged due to excess subcutaneous fat, and inflated in the presence of meteorism or ascites. Regularity of the abdomen shape should be assessed. An enlarged liver may protrude in the upper abdomen; an enlarged

uterus causes protrusion of the lower abdomen. Inspection of the abdomen may give information about the contours and peristalsis of the stomach if the patient is cachectic. In pathological cases (pyloric stenosis) peristalsis can be easily seen (ridges raising the abdominal wall). If a physician rubs or taps on the epigastric region peristalsis becomes more distinct. Sometimes, in neglected cases, the abdominal wall can be protruded by tumour.

The patient is asked to breathe "with his abdomen" to assess the mobility of the abdominal wall. The patient is unable to take a deep breath in the presence of pain, e.g. in an attack of acute appendicitis or cholecystitis. Divarication of the rectus abdominis muscles can be revealed if the patient raises his head. Regular application of hot-water bottle leaves its traces on the abdomen; these, together with postoperative scars, often help the physician to interpret correctly the present patient's complaints. Antiperistaltic movements in the epigastrium or by the course of the intestine can give a hint on the presence of an obstacle to propulsion of food masses in the intestine.

If an abdomen is inflated, the causes should be established. These may be obesity, accumulation of liquid, or meteorism. Slight distension of the abdomen may be due to a tumour, encapsulated fluid, or meteorism associated with intestinal stenosis. The latter suggestion is confirmed by visible peristalsis over the constricted portion of the intestine where the flatulence is observed.

Character and localization of postoperative scars enable rather precisely to establish the organ on which operation has been made. Survey of an abdomen in a vertical position comes to an end with survey of a white line, inguinal and femoral canals where find out the hernias producing strong pains in an abdomen. For detection of hernias it is necessary palpate hernial rings by the index finger which dilating promotes formation of hernias. The outside inguinal ring routinely loosely passes the index finger, intrinsic inguinal ring - only its tip. In a vertical position of the patient it is possible to distinguish a separation of recti abdominis muscles by a palpation of a white line of an abdomen.

During research the patient should lay on the back with completely naked abdomen on a bad with a low pillow, the extended legs and hand are posed along the trunk. The doctor should sit by the right side from a patient on a chair which level is close to the level of bed, having face-to-face contact with a patient.

At the time of survey in horizontal position it is paid attention first of all to those changes which have taken place at the moment of a postural change of a body of the patient. In a horizontal position hernias are seen approximately routinely absent.

The abdomen can be enlarged significantly due to accumulation of free fluid *(ascites).* This occurs in liver cirrhosis concurrent with portal hypertension. The abdomen may be enlarged due to pronounced hepato- or

splenomegaly. When the patient with ascites stands erect, his abdomen becomes pendulous due to the downward flow of fluid; in the lying position the abdomen is flattened (*"frog belly").* The navel often becomes protruded in ascites when the patient stands erect. It is due to increased infra-abdominal pressure. This sign can be used to differentiate between enlargement of the abdomen in ascites (also large intraabdominal tumours) and pronounced obesity (the navel is retracted).

Percussion of abdomen

Percussion of the abdomen is only relatively informative. Percussion of the anterior abdominal wall at points of projection of the intestine gives tympany of various characters which depends on the uneven distribution of gaseous, liquid or solid intestinal contents.

Percussion of abdomen in vertical position of the patient is used for revealing free fluid in an abdominal cavity and definitions of its level. By percussion on midline and lateral flanks from top to down, it is possible to differentiate the tympanic sound above intestines and the dull sound lower than fluid level.

In horizontal position of the patient percussion of the abdomen is performed from umbilicus on midline to epigastrium and hypogastrium, and from umbilicus - to flanks in lateral directions. With the purpose of differentiation dull sounds originated from free fluid and contents of intestines the physician can repeat percussion from the umbilicus to flanks in lateral directions in position of the patient on the side of body. At presence of ascites the level of dull sound is changed in this position of patient.

By means of *percussionary palpation method* the *symptom of fluctuation of fluid* also is defined a presence of an ascites. For this purpose the palmar surface of the left arm is put on a right half of abdomen in region with detection of dullness. The right arm impacts one-digital percussion mild strikes on the left half of the abdomen according to V.P. Obraztcov. At presence of loose fluid in the abdominal cavity in a significant amount the palm of the left arm clearly accepts fluctuation - jerky fluctuations of fluid. For the prevention of transfer of oscillating motions on the anterior abdominal wall it is possible to put the edge of the arm or the book along the white line of the abdomen. If the patient cannot eat the full meal (the capacity of the stomach gradually decreases), it is necessary to determine the Traube's space, which can be markedly decreased. The presence of these two symptoms requires an X-ray examination to exclude cancer of the stomach. Short strokes of the hammer or the flexed fingers on the epigastrium (Mendel sign) are used to determine involvement of the parietal peritoneum: pain indicates affection of the peritoneum.

Auscultation of abdomen

Auscultation of abdomen in vertical position of the patient it is performed for definition of a *friction murmur of a peritoneum* in the right and left hypogastrium in perihepatitis and episplenitis. The importance of auscultation for diagnosis of diseases of the liver and gall bladder is only relative. In only rare cases peritoneal friction can be heard over the liver and the gall bladder (in perihepatitis or pericholecystitis). This sound resembles pleural friction, and is a dangerous sign. It indicates deep extension of inflammation onto all walls of the gall bladder and possible perforation.

Auscultation of esophagus. Listening to epigastric range below xiphoid process or above it, at swallowing fluids by the healthy person it is possible to hear two murmurs: the first - at once after swallowing, and 6-9 seconds later the second - connected to transit of fluid through cardia. Delay or absence of the second murmur specifies an interrupting arised in the inferior third of an esophagus, in a cardiac department of a stomach.

Auscultation of abdomen in horisontal position

Auscultation of peristalsis intestinal tones gives information about the motor function of the intestine. During gastric digestion and movement of the chyme along the small intestine, long periodic rumbling can be heard. Rhythmic intestinal murmurs can be heard 2-3 per minute 5—7 hours after meals. The peristalsis intestinal tones are listened in the cecum (right inguinal range), in the small intestine (above the point of Porges – 2 sm from umbilicus in upper and left direction) and in sigmoid (left inguinal range). In mechanical obstruction of the intestine, its peristalsis is resonant (in large waves). Peristalsis disappears in paralytic obstruction of the intestine; the abdomen is absolutely "silent" in perforation of the ulcer with secondary paralysis of the intestine; peritoneal friction can be heard in patients with fibrinous peritonitis during respiratory movements.

Auscultation of stomach. Splashing sound (succussion) can be heard if the patient is lying on his back, while the examiner pushes the anterior wall of the peritoneum with four flexed fingers of the apt hand. The other hand of the physician should fix the muscles of the abdominal prelim against the sterna edge. The thrust of the hand is transmitted through the stomach wall to the liquid and air contained inside it to cause a readily audible splashing sound which is inaudible outside the inferior borders of the stomach. This technique for outlining the inferior border of the stomach is effective in cases where the stomach border formed by the greater curvature is at the normal level or lowered. Succession gives information about the evacuator function of the stomach: the splashing sounds in healthy subjects can only be heard after meals. Splashing sounds heard 7-8 hours after meals suggest evacuator dysfunction of the stomach (mostly in pyloric stenos is) or its pronounced hypersecretion (*gastrosuccorrhea*). Splashing sounds heard to the right of the

median line of the abdomen indicate dilatation of the prepyloric part of the stomach (Vasilenko's symptom).

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

Auscultation of abdominal aorta is performed on midline 5-7 sm above umbilicus. Systolic murmurs can be listened in abdominal aorta aneurysm, atherosclerosis of abdominal aorta and its branches.

Palpation of abdomen:

Surface tentative palpation and deep sliding palpation of abdomen (according to Obraztsov and Strazhesko)

Palpation of abdomen: history of the method

Palpation is the main method of physical examination in diagnosis of diseases of the abdominal organs. This method was first appreciated by French physicians (Glenard) in 80-years of XIX century. Later the Russian internists (Obraztsov, Strazhesko, Gausmann and others) further developed this useful method.

Glenard proposed palpation of the abdomen and believed that this method should systematically be used for clinical examination of the abdominal cavity. He maintained that palpation can be used to examine not only the abdominal organs but also various portions of the intestine. Having established that the cecum, transverse colon, sigmoid, and the colon proper can sometimes be palpated, he believed erroneously that their palpability indicated their pathology.

Independently of Glenard, Obraztsov developed methods for palpation of the gastro-intestinal tract and proved that some parts of the stomach and the intestine can be palpated in the absence of any pathology. He gave a detailed description of physical properties of each part of the abdominal organs in normal conditions. He substantiated thus usefulness of palpation in clinical practice along with other physical methods of examination; secondly he stimulated the study of the topographic relationships in the abdominal cavity before X-rays were discovered; and thirdly his teaching made it possible to compare the physical properties of organs and their topographic

relations in health with those in pathology, which has become an important tool in the diagnosis of diseases of the abdominal cavity.

Later Obraztsov and his disciples developed in detail palpation techniques for examination of the abdominal cavity; they studied the organs and their separate parts that can be palpated under various conditions, and also gave a detailed description of normal palpatory signs of organs and their changes in various pathological conditions. They have proved finally the importance of palpation as an invaluable method of examination of the abdominal organs. It should however be emphasized that it is very difficult to master properly the palpation techniques for diagnostic purposes. It requires much experience and training. The palpation method described below has been proposed by Obraztsov and Strazhesko.

The common rules of the surface and the deep palpation

It is necessary that the abdominal cavity should be accessible to palpation, i.e. that its muscles of the anterior abdominal wall (prelum) be relaxed, and that the examiner should not provoke their straining by his manipulations. The patient should relax in his bed. (The bed should not be too soft.) His legs should be stretched and the arms flexed on the chest. The patient's breathing should not be deep; his head should rest against a small firm pillow. This position ensures relaxation of the abdominal muscles. The physician takes his place by the right side of the bed, facing the patient. The chair should be firm and level with the patient's bed. The ambient temperature should be comfortable for the patient, and the hands of the doctor should be warm and dry, nails must be short. A palpation is performed only after an auscultation and a percussion of the abdominal cavity.

The examining movements should be careful and gentle so as not to hurt the patient. Touching the abdomen roughly with cold hands will cause reflex contraction of the prelum to interfere with palpation of the abdomen. The patient with distended abdomen should first be given laxative or enema to empty the bowels. These are the conditions for palpation of the patient in the recumbent position. But some organs or their parts can only be palpated when they hang by gravity with the patient in the vertical position. Thus the left lobe of the liver, the lesser curvature of the stomach, the spleen, the kidneys, the cecum, or tumours can become palpable. The epigastrium and the lateral parts of the abdominal cavity should also be palpated with the patient in the vertical position.

Surface and deep palpation are used. *The surface palpation* examines condition of the anterior abdominal wall. The d*eep palpation* is used to establish normal topographic relations between the abdominal organs and their normal physical condition; the other object is to detect any possible pathology that changes the morphological condition of the organs and their topographic relations responsible for their dysfunction, to locate the defect,

and to determine its nature. In other words, the deep palpation gives information on the topography of the abdominal cavity (topographic palpation).

The surface tentative palpation of abdomen

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. The surface tentative palpation of an abdomen is performed in a direction against a course of a wrist-watch, i.e. after the left inguinal range palpation is continued on left flank from below upwards up to the left hypochondrium, then epigastric range, right hypochondrium and right flank from top to down up to right inguinal range. After that a right arm is placed in epigastric range to a surface tentative palpation of median zone from a xiphoid process down to the suprapubic area.

It is also a procedure of a surface tentative palpation of symmetrically areas of an abdomen. In this case after of the left inguinal area 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 surface tentative palpation of an abdomen reveals a presence of morbidity, a resistance of a forward abdominal wall or its muscle strain, to probe the inspissations formed in a wall, hernias, tumours, to distinguish puffiness of a skin from augmentation of a hypodermic fatty tissue. For an establishment of morbidity before a palpation it is necessary to warn the patient that he has told when at him the pain sensation will be maximal, will appear and stop. Pay attention also to a look of the patient.

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. 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 purulent 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 tumours, even surface palpation can give much diagnostic information. But only deep systematic palpation can give full information about the condition of the abdominal cavity and its organs, as well as their topography.

The deep sliding methodical topographic palpation (according to Obraztsov and Strazhesko)

When starting deep palpation the examiner should always be aware of the anatomical relations in the abdominal cavity, the shape and physical properties of the organs, their supporting structures and possible deviations in topographical relations that may depend on the constitution of the patient, his special condition, nutrition, relaxation of the abdominal muscles, etc.

Obraztsov used the double-checking principle in his examinations. For example, in order to make sure that a given section of the intestine is actually ileum terminale it is necessary to locate the cecum; to determine the size of the stomach, the palpatory findings are checked by percussion and percussive palpation of the stomach. Respiratory excursions of the organs should be taken into consideration during palpation according to a strictly predetermined plan, beginning with more readily accessible parts.

The rules and techniques of deep palpation of abdomen

The success of a deep sliding palpation of an abdomen depends on strict observation of the rules, a convenient and easy position of the patient and the doctor, correct respiration of the first and a position and state of arms of the second, rational palpation tactics of investigator and the conforming readiness to a palpation of researched patient.

Necessary condition is the maximal relaxation of muscles, especially front abdominal wall. The optimal for palpation of abdomen is the diaphragmatic respiration at which during an inspiration muscles of a abdominal wall exert a little, and during an expiration - are as much as possible relaxed. The deep sliding palpation of an abdomen provides necessity of a palpation of members of the abdominal cavity for fixed sequence and good knowledge of clinical topographical anatomy.

The following sequence of deep palpation is recommended: the left ileum area – the sigmoid and the descending colon, the right ileum area – the cecum with the terminal end of the ileum and the ascending colon, further the epigastric and paraumbilical regions - the stomach with its parts (greater curvature and pylorus) and the transverse colon; the following stage - palpation of the liver, the spleen and kidneys.

The deep sliding palpation is performed only after the surface tentative palpation of an abdomen. The posture of the patient and the physician should

be the same as in surface palpation. Palpation should be carried out by the right hand. In some cases the other hand should be placed on the examining hand to increase pressure. Palpation can also be bimanual (palpation with both hands simultaneously). If only one hand is used, the other hand presses the prelum laterally to the palpated zone in order to lessen or overcome resistance of the abdominal wall and hence to promote relaxation of the prelum in the palpated zone. The other hand can be used to move the palpated organ closer to the examining hand or in order to perform bimanual palpation.

The deep sliding 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 parallel 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 performed when the fingers are moved gradually with each of expirations, 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, cecum) 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, and diameter, the condition of the surface (smooth, tubercular), the absence or presence of rumbling sounds during palpation. All these signs indicate the presence or absence of pathology.

Palpation of sigmoid

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 the median and the outer third of the linea umbilico-iliacae. Palpation is performed by four fingers, placed together and slightly flexed. The fingers are placed 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. The sigmoid is only impalpable in excess inflation of the abdomen and in obese patients. If the sigmoid is not found where it belongs, it may be displaced to some other location because of long mesenterium which accounts for the high sigmoid mobility. It is then usually displaced closer to the navel and to the right. The sigmoid can usually be found by deep palpation of the infraumbilical and suprapubic areas. 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.

Palpation of cecum

The *cecum* is palpated by the same technique, except that the direction is different. Since the cecum 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. Palpation is used not only to locate the cecum but also a certain part of the ascending colon (10-12 cm of its length), i.e. the part of the large intestine which is known in the clinic as typhlon. A normal cecum 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 cecum (to 2—3 cm). The lower-edge of the cecum is 0.5 cm above the biiliac in man and 1—1.5 cm below it in women.

Palpation of the terminal end of the ileum

Further *the terminal end of the ileum* can be palpated in the depth of the right iliac space as a soft, easily peristalting and passively mobile cylinder, the thickness of the little finger (or a pencil); it slips out from under the examining fingers and rumbles distinctly. Fingers of palpating arm are installed at the border external and medium third of linea biliaca under the angle 15-20º. The terminal end of an ileum is palpated as the sleek dense cylinder in diameter of 0,5-1,0 sm in case of reduction of a muscle layer of an intestine, or as an impressed thin-walled mild tubule which palpation is accompanied by a rumble in case of a release phenomenon of a musculation of an intestine and its fluid contents. The palpated part of a small bowel is routinely moderately mobile (up to 5-7 sm) and tolerant. Quite often during a palpation it is possible to establish transferring of an intestine from the weakened state in spasm condition when it as though «plays» near at hand.

The cecum and the terminal part of the ileum are palpated by four fingers of the right hand; the fingers should be held together and slightly flexed. If the prelum is tense, the muscles in the palpation zone can be relaxed by pressing the umbilical area with the radial edge of the left hand.

Palpation of the ascending and descending colons

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 (Vasilenko).

The *descending colon* is palpated immediately after palpation of the sigmoid. The fingers of the right arm are installed in the left flank paralelly to midline on 3-5 sm above the position of sigmoid. The descending colon is similar to sigmoid, the difference consists in relatively slight mobility.

The *ascending colon palpation* follows the palpation of the cecum. The fingers of the right arm are installed in the right flank paralelly to midline on 3-5 sm above the position of cecum.

Palpation of the stomach

The stomach should be palpated in both the vertical and horizontal position of the patient because the lesser curvature of the stomach and its high standing tumours are impalpable in the lying position. First palpation should be superficial and tentative. Its aim is to establish tenderness of the epigastrium, irritation of the peritoneum (Shchetkin-Blumberg symptom), divarication of the abdominal muscles, the presence of hernia of the linea alba, tension in the abdominal wall in the region of the stomach, and the presence of muscular defence (*defense musculaire*).

The deep palpation of the stomach should be carried out according to Obraztsov and Strazhesko. In connection with feature of its location, character of a surface and a consistence of various departments the stomach entirely almost is never palpated. More often the big curvature of a stomach and its pylorus are palpated.

The deep palpation of the greater curvature of the stomach begins at epigastric range from a xiphoid process downwards on 3-5 sm. 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. If the first attempt of a palpation appeared unsuccessful, i.e. the sensation of sliding was not, it is necessary to repeat all over again, having established tips of fingers of a right arm is lower on 3-5 sm. And so the palpating arm is displaced down while the greater curvature will not be palpated, down to suprapubic range.

In absence of the stomach pathology the greater curvature of the stomach is posed at men on 3-4 sm above a level of a navel, at the woman of 1-2 sm are higher than a navel or at its level. The surface of a stomach is smooth, and the big curvature is represented as elastic, thin, smooth fold. Palpation of a stomach is painlessly in healthy men.

The shape of the stomach and the size of the examined part can thus be assessed. 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 lesser curvature can be palpated in gastroptosis. It appears to palpating fingers as a ridge on the back bone and by its sides. In cases with gastroptosis, the greater curvature can descend below the navel. Correctness of determination can be confirmed if the position of the ridge coincides with that of the lower border of the stomach as determined by other techniques (by percussion, by the splashing sound or stethacoustic palpation).

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.

Splashing sound (succussion) can be heard if the patient is lying on his back, while the examiner pushes the anterior wall of the peritoneum with four flexed fingers of the apt hand. The other hand of the physician should fix the muscles of the abdominal prelum against the sternal edge. This technique is useful for outlining of the inferior border of the stomach.

Stethacoustic palpation (s. auscultative percussion, or auscultative affricsion) *of the stomach* is helpful when used together with palpation of the stomach to outline its inferior border.

Palpation of the pylorus

The pylorus is located in the triangle formed by the lower edge of the liver to the right of the median line, by the median line of the body, and the transverse line drawn 3-4 cm above the navel, in the region of the right rectus abdominis muslce. Since the position of the pylorus is oblique (upwards to the right) the palpating movements should be perpendicular to this direction, i.e. from left downwards to the right. The pylorus is identified by palpation as a band (tense or relaxed). When the pylorus is manipulated by the fingers, a soft rumbling sound can be heard. When contracted spastically (pylorospasm) the pylorus remains firm for a long time. Sometimes the pylorus is mistaken for cancer infiltration.

Palpation of the stomach can reveal tumours of the pylorus, of the greater curvature, and of the anterior wall. Tumours of the lesser curvature can be diagnosed with the patient in the upright position. Tumours of the cardial part of the stomach are inaccessible to palpation. Exact information on their location gives X-ray examination.

Palpation of the transverse colon

The delimitation of the greater curvature of the stomach always should precede a palpation of transverse colon.

The *transverse colon* is palpated bimanually by four fingers of the right and left hand held together and slightly flexed. Since the position of the transverse colon is unstable, it is useful first to determine the lower border of the stomach by percussive palpation (after Obraztsov) or by palpation of greater curvature of a stomach, and only then to search for the colon some 2—3 cm below this border. The both hands are placed on the sides of the linea alba and the skin are moved slightly upwards. Then the fingers are plunged gradually during relaxation of the prelum at expiration until the posterior wall of the abdomen is felt. Once the posterior wall is reached, the arms 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.

Having examined transverse colon in median region, it is necessary to palpate this intestine to the right and to the left outside as far as it is possible. In some cases, following a course of transverse colon, it is possible to reach up to hepatic (more often) or splenic (less often) curvature of the transverse colon.

In addition to the mentioned portions of the intestine, the horizontal parts of the duodenum and the curvature of the colon can in rare cases be palpated; an occasional loop of the small intestine that may happen in the iliac cavity can also be palpated. But the small intestine is usually impalpable because of its deep location, high mobility, and thin walls.

Examination of patients with pathology of the liver and biliary tracts:

Subjective and physical examination

Subjective examination (inquiry)

Complaints

Patients with disorders of the hepatobiliary system usually complain of abdominal pain, dyspepsia, skin itching, jaundice, enlargement of the abdomen, and fever.

Pain is localized in the right hypochondrium and sometimes in the epigastrium and differs depending on the cause. Pain may be persistent and dull, or it may be severe and occur in attacks. Persistent pain is usually boring, or the patient feels pressure, heaviness, or distension in the right hypochondrium. Pain may radiate to the right shoulder, scapula, and in the interscapular space (in chronic cholecystitis, perihepatitis and

pericholecystitis, i.e. when the process extends onto the peritoneum overlying the liver and the gall bladder, and also in rapid and considerable enlargement of the liver which causes distension of Glisson's capsule). This radiation of pain is quite characteristic of many diseases of the liver and gall bladder, because the right phrenic nerve, innervating the capsule in the region of the falciform and the coronary ligaments of the liver and the extrahepatic bile ducts, originates in the same segments of the spinal cord where the nerves of the neck and shoulder originate as well. Pain usually becomes more severe in deep breathing; in adhesion of the liver or the gall bladder to the neighboring organs, pain is also intensified when the patient changes his posture, and sometimes during walking.

Attacks of pain (biliary or hepatic colics) develop suddenly and soon become quite severe and unbearable. The pain is first localized in the right hypochondrium but then spreads over the entire abdomen to radiate upwards, to the right, and posteriorly. An attack of pain may continue from several hours to a few days during which pain may subside and then intensify again; the attack ends as suddenly as it arises; or pain may lessen gradually. Attacks of pain occur mostly in cholelithiasis. They are provoked by jolting (as in riding) or by fatty food. Pain attacks occur also in hypermotoric dyskinesia of the gall bladder and bile ducts. Pain usually develops quite unexpectedly due to spastic contractions of muscles of the gall bladder and large bile ducts caused by irritation of their mucosa by a stone, and due to comparatively rapid distension of the gallbladder in congestion of bile (e.g. due to obstruction of the common bile duct by a stone). Warmth applied to the liver (provided the attack is not attended by considerable fever) and also administration of cholino- and myospasmolytics (atropine sulphate, papaverine hydrochloride, etc.) remove pain characteristic of the colic. An attack of hepatic colic can be attended by subfebrility (fever develops with pain and subsides with alleviation of pain), which is followed by a slight transient subicteric colour of the sclera or pronounced jaundice in obstruction of the common bile duct by a stone.

Pain developing in dyskinesia of the bile ducts is associated with upset coordination between contractions of the gall bladder and of the Oddi sphincter under the effect of increased tone of the vagus nerve. As a result, bile congests in the ducts, and the gall bladder is no longer emptied. This causes its convulsive contraction. Dyskinetic pain is characterized by the absence of signs of inflammation (leucocytosis, ESR, etc.).

Dyspeptic complaints include decreased appetite, often bitter taste in the mouth, eructation, nausea, vomiting, distension of the abdomen and rumbling, constipations or diarrhea. These complaints are characteristic not only of diseases of the hepatobiliary system but also of other parts of the digestive system. Causes of these symptoms in diseases of the liver and bile ducts are explained by deranged secretion of bile (and hence impaired

digestion of fats in the intestine) and derangement of the detoxicating functions of the liver.

Fever occurs in acute inflammatory affection of the gall bladder and bile ducts, in abscess and cancer of the liver, in hepatitis, and active cirrhosis.

Skin itching attends hepatic or obstructive jaundice. It can develop without jaundice, as an early forerunner of the liver disease. Itching is caused by accumulation in the blood of bile acids which are otherwise excreted together with bile, or by stimulation of sensitive nerve endings in the skin. Itching is usually persistent and is a great annoyance to patients during night sleep (to cause insomnia). Severe itching causes scratching of the skin with its subsequent infection.

Icteric colouration of the skin and the visible mucosa (*jaundice*) is due to accumulation of bile pigments in the blood and tissues. Jaundice may develop unnoticeably to the patient and only the surrounding people may pay attention to the icteric colouration of the sclera and then the skin. In other cases jaundice can occur all of a sudden, following an attack of hepatic colics (in obstruction of the common bile duct by a stone in cholelithiasis). Jaundice may persist for months or even years, only slightly changing in intensity (chronic hepatitis and cirrhosis of the liver, benign bilirubinemia).

Jaundice can develop with severe itching of the skin, skin hemorrhages and hemorrhages of the nose and the gastro-intestinal tract.

Jaundice occurs in many diseases of the liver, bile ducts, blood, and also diseases of other organs and systems, to which bilirubin metabolic disorders are secondary. Some clinical symptoms attending jaundice indicate to a certain degree of its type and origin. Accurate diagnosis of various types of jaundice is possible with special laboratory studies.

True jaundice can develop due to the following three main causes: (1) excessive decomposition of erythrocytes and increased secretion of bilirubin (hemolytic jaundice); (2) impaired capture of unbound bilirubin by the liver cells and its inadequate combination with glucuronic acid (parenchymatous jaundice); (3) obstacles to excretion of bilirubin with bile into the intestine and reabsorption of bound bilirubin in the blood (obstructive jaundice).

Hemolytic (hematogenous) jaundice develops as a result of excessive destruction of erythrocytes in the cells of the reticulohistiocytic system (spleen, liver, bone marrow). The amount of unbound bilirubin formed from hemoglobin is so great that it exceeds the excretory liver capacity to account for its accumulation in the blood and development of jaundice. Hemolytic jaundice is the main symptom of hemolytic anemia. It can also be a symptom of other diseases, such as B_{12}-(folic)-deficiency anemia, malaria, protracted septic endocarditis, and other diseases.

The skin of a patient with hemolytic jaundice is lemon-yellow. Skin itching is absent. The amount of unbound bilirubin in the blood is moderately increased (50—200 per cent); the van den Bergh test for bilirubin is indirect.

Bilirubin is absent from the urine but the urine is still coloured rather intensely by the markedly increased (5—10 times) stercobilinogen and (partly) urobilinogen. Feces are intense dark due to the presence of considerable amount of stercobilinogen.

Parenchymatous (hepatocellular, hepatic) jaundice develops due to the damage of the parenchyma cells (hepatocytes). These cells can capture bilirubin of the blood and bind it with glucuronic acid (the natural detoxicating function of the liver). The natural process of bilirubin excretion in the bile in the form of bilirubin glucuronide (bound bilirubin) is thus impaired. The content of free and bound bilirubin in the blood serum thus increases 4—10 times. In rare cases the increase may be even greater: free bilirubin increases due to hepatocyte dysfunction and bound bilirubin content increases as a result of back diffusion of bilirubin glucuronide from biliary into blood capillaires in dystrophy of the liver cells. Bound bilirubin appears in the urine (bilirubin glucuronide is water soluble and easily passes via the capillary membranes as distinct from free bilirubin). Bile acids are also present in urine, but their content gradually increases. Excretion of stercobilinogen with feces also decreases because amount of bilirubin excreted by the liver into the intestine decreases, but feces are rarely completely discoloured.

This type of jaundice is mainly determined by infection (virus hepatitis or Botkin's disease, leptospirosis) and toxic affections of the liver (poisoning with mushrooms, phosphorus, arsenic and other chemical substances, medicinal preparations included). But parenchymatous jaundice can develop also in liver cirrhosis.

The skin of patients with this jaundice is typically yellow with a reddish tint. Skin itching is less frequent than in obstructive jaundice because the synthesis of bile acids by the affected liver cells is upset. Symptoms of pronounced hepatic insufficiency may develop in severe course of the disease.

There exists a group of congenital pigmentary hepatoses in which the liver is not affected pathologically; the functional tests are negative, while the process of bilirubin conjugation with glucuronic acid is upset at some of these stages (Gilbert syndrome). This condition is attended by a permanent or intermittent jaundice, which is sometimes pronounced and develops from infancy.

Obstructive (mechanical, surgical) jaundice develops due to partial or complete obstruction of the common bile duct. This occurs mostly due to compression of the duct from the outside, by a growing tumour (usually cancer of the head of the pancreas, cancer of the major duodenal papilla, etc.), or due to obstruction by a stone. Bile congestion above the point of obstruction develops and this elevates pressure inside bile passages in continuing bile excretion. As a result, the interlobular bile capillaries become

distended and bile diffuses into the liver cells (where dystrophic processes develop) and passes into the lymph and the blood. Moreover, due to increased pressure inside fine bile capillaries, communications are formed at the periphery of the lobules between the capillaries and the lymph spaces, through which bile enters the blood vessels.

Skin and mucosa of patients with obstructive jaundice are yellow. Later, as bilirubin is oxidized to biliverdin, the skin and mucosa turn green and dark-olive. The bound bilirubin content in the blood with direct van der Bergh test is as high as 250-340 mmol/1 or 15-20 mg/100 ml, and more. In protracted jaundice associated with liver dysfunction, free bilirubin content increases as well. Bound bilirubin can be found in the urine (the presence of bile pigments is determined by urinalysis) to give it brown colour and bright-yellow foaming. Feces are colourless either periodically (in incomplete obstruction, usually by a stone), or for lengthy periods of time (in compression of the bile duct by a tumour). Jaundice in-creases progressively in such cases; the skin and mucosa gradually turn greenish-brown; cachexia of the patient increases. In complete obstruction of the bile ducts, feces become colourless (acholic); their colour is clayish and grey-white; stercobilin is absent from feces.

Bound bilirubin and also bile acids produced by the hepatocytes in ample quantity (cholemia) are delivered to the blood in this type of jaundice. Some symptoms associated with toxicosis develop: pronounced skin itching, which intensifies by night, and bradycardia (bile acids increase the tone of the vagus nerve by reflex). The nervous system is also affected: the patient develops rapid fatigue, general weakness, adynamia, irritability, headache, and insomnia. If it is impossible to remove the cause of impatency of the common bile duct (stones or a tumour) the liver is gradually affected to add symptoms of hepatic insufficiency.

Enlargement of the abdomen (sometimes rapid) can be due to accumulation of ascitic fluid in the abdominal cavity (in obstructed blood outflow from the intestine via the portal vein), in considerable meteorism (due to deranged digestion in the intestine in upset bile excretory function), or in pronounced hepato- or splenomegaly.

Many chronic diseases of the liver and biliary tracts are attended by *general weakness, non-motivated fatigue, and decreased work capacity.*

History of the present disease

When collecting anamnesis, it is necessary to find out if the patient had in his past history jaundice or acute diseases of the liver or the gall bladder (Botkin's disease, acute cholecystitis, cholangitis), attacks of hepatic colics, enlargement of the liver or the spleen, which might be an early symptom of the present disease (chronic hepatitis, liver cirrhosis, chronic cholecystitis, cholangitis, cholelithiasis).

Life history of patient

When inquiring the patient it is necessary to establish factors that might be important for the etiology of the present disease of the liver or bile ducts: liking for fat and meat foods, exposure to chemical and vegetable poisons (alcohol, carbon tetrachloride, compounds of phosphorus, copper, lead, arsenic, dichloroethane, etc.), poisoning with mushrooms containing strong hepatotropic poisons (e.g. helvellic acid, amanitotoxin, etc.), some infectious diseases (Botkin's disease, lambliosis, typhoid fever, malaria, syphilis, etc.), diseases of the gastro-intestinal tract (gastritis, colitis), and diabetes mellitus. Familial predisposition is also important in the development of some liver diseases (e.g. congenital benign hyperbilirubinemia) and diseases of the gallbladder (cholelithiasis).

Physical Examination

General inspection (survey)

The general condition of the patient is first assessed. In the presence of marked functional hepatic insufficiency of various etiology (liver cirrhosis, cancer, prolonged obstructive jaundice, etc.), the patients's condition can be grave because of pronounced poisoning (hepatic coma). The patient's condition may be grave in acute inflammatory diseases of the liver (abscess), gallbladder (acute cholecystitis), or bile ducts (acute cholangitis). But in many chronic diseases of the liver and the bile ducts, the general condition of the patient may remain satisfactory for long periods of time. Patients with hepatic colics are restless, they toss in bed, try to find (without success) a position in which the pain might be relieved. Hepatic coma is characterized by deranged consciousness in the form of pronounced euphoria or inhibition to complete loss of consciousness.

The general appearance (*habitus*) of the patient usually does not change. At the same time, hypersthenic constitution with predisposition to obesity is often characteristic of patients with cholelithiasis. Quite the reverse, significant wasting (to cachexia) occurs in cirrhosis or malignant tumour of the liver or the bile ducts. If the disease of the liver begins in childhood or adolescence, the patient may look infantile.

An important diagnostic sign *is jaundice* of varying intensity. In order to assess correctly the colour of the skin, the patient should be inspected in daylight or in the light of the luminescent lamp. A subicteric symptom is jaundice of the sclera, the lower surface of the tongue, and the soft palate; next coloured are the palms, soles, and finally the entire skin. Inspection of the sclera helps differentiate between true (bilirubinogenic) and exogenic jaundice. Prolonged use of quinacrine, ethacridine lactate (rivanol), carotin (carrots), excess tangerines and oranges, exposure to trinitrotoluene and picric acid can cause slight jaundice of the skin (*false jaundice*) but the sclera

is not coloured in such cases. Hepatic jaundice is usually attended by itching and scratching of the skin.

Icteric skin can be of various hues. The skin is orange-yellow (rubinicterus) due to accumulation of bilirubin in the skin; it is usually characteristic of the early stages of the disease. Lemon-yellow colour of the skin (flavinicterus) is characteristic of hemolytic jaundice. Greenish-yellow colour (verdinicterus) is due to accumulation of biliverdin (the product of gradual oxidation of bilirubin); it is mostly due to obstructive jaundice. In long-standing mechanical jaundice the skin becomes dark bronzy (melasicterus).

In certain cases the skin becomes pallid due to anemization (hemorrhage from varicose esophageal or hemorrhoidal veins in portal cirrhosis); the skin may be greyish ("dirty") in patients with some hepatic diseases. Greyish-brown or brown skin is characteristic of hemochromatosis (bronzed diabetes or pigmentary cirrhosis of the liver), the disease associated with primary or secondary excessive absorption of iron in the intestine and accumulation of hemosiderin in various organs and tissues (in the first instance in the liver and the pancreas). Local hyperpigmentation of the skin in the right hypochondrium can be due to frequent application of a hot-water bottle, which indicates persistent pain in this region (in chronic diseases of the gallbladder).

Inspection of the skin (especially in obstructive and less frequently in parenchymatous jaundice) can reveal *scratches* due to severe itching. The scratches are often infected and purulent. Jaundice of this type can be attended by hemorrhagic diathesis - *petechial eruption* and hemorrhage into the skin (*ecchymosis*).

In patients with cirrhosis of the liver associated with disordered cholesterol metabolism, cholesterol is deposited intracutaneously in the form of yellow plaques *(xanthomatosis)* which are often located on the eyelids *(xanthelasma)* and less frequently on the hands, elbows and soles *(xanthomas).* Xanthomatosis occurs also in other diseases attended by cholesterol metabolic defect (atherosclerosis, diabetes mellitus, essential hyperlipemia, etc.).

An important symptom for diagnosis of chronic diseases of the liver is *spider angiomata.* These are slightly elevated pulsating angiomata with fine vessels radiating from the centre. Their size varies from that of a pin head to 0.5-1 cm in diameter. The angiomata are often found on the neck, face, shoulders, hands, and the back; less frequently they are practically absent from the lower part of the body. The spider angiomata may disappear with improvement of the liver function. In addition to these angiomata, patients with chronic diseases of the liver may have specifically coloured palms and soles - *liver palms*; symmetrical reddening is especially characteristic in the thenar and hypothenar region. When pressed, the reddened site becomes pale

but when the pressure is removed, the redness is quickly restored. The mechanism of development of the spider angiomata and liver palms is believed to be connected with the grave hepatic dysfunction of the liver during which estrogens are destroyed incompletely and therefore act as a vasodilatory agent on the skin vessels.

Excess estrogens in the blood are also associated with other symptoms that may be revealed on inspection. Patients with chronic diseases of the liver have a glassy crimson tongue *(raspberry tongue).* Uni- or bilateral enlargement of the mammary glands often occurs in men (gynecomastia) along with defective growth of hair on the chin, chest, and the abdomen. Hair growth is decreased in the armpits and on the pubis in women. When the, hepatic condition improves, the hair growth is restored. *Drum (Hippocratic) fingers*, sometimes with white nails, occur in patients with chronic diseases of the liver. It is believed to depend on excess estrogens and serotonin in the blood.

A greenish-brown *Kayser-Fleischer ring* round the outer edge of the cornea is characteristic of the Konovalov-Wilson disease (congenital disease characterized by decreased synthesis in the liver of ceruloplasmin, the copper transport protein, and its increased deposition in tissues).

Inspection of the mouth can repeal *angular stomatitis* (inflammation of the mucosa and skin in the mouth angles) characteristic of group B hypovitaminosis occurring amongst patients with chronic liver diseases.

Inspection of the abdomen

Inspection of the abdomen should be done with the patient in vertical and horizontal position.

Important diagnostic symptoms can be found during inspection of the abdomen. The abdomen may be enlarged significantly due to accumulation of free fluid *(ascites).* This occurs in liver cirrhosis concurrent with portal hypertension. The abdomen may be enlarged due to pronounced hepato- or splenomegaly. When the patient with ascites stands erect, his abdomen becomes pendulous due to the downward flow of fluid; in the lying position the abdomen is flattened (“frog belly”). The navel often becomes protruded in ascites when the patient stands erect. It is due to increased infra-abdominal pressure. This sign can be used to differentiate between enlargement of the abdomen in ascites (also large intraabdominal tumours) and pronounced obesity (the navel is retracted).

Inspection of the abdomen can reveal another important symptom of portal hypertension, the presence *of dilated venous network on the anterior abdominal wall*. This network is formed by anastomoses of the portal and both vena cava systems. The superior vena cava and portal vein are anastomosed above the navel, while the portal and inferior vena cava are anastomosed below the navel; cavocaval anastomoses are located on the sides

of the abdomen. Anastomoses may develop in obstructed blood flow in the inferior vena cava (thrombosis, compression, etc.). Dilated, swollen and twisted venous collaterals, found round the navel and radiating from it, form the so-called *Medusa head*. These symptoms are characteristic of the portal hypertension syndrome occurring in cirrhosis of the liver, thrombosis and compression of the portal vein. Establishing the direction of the blood flow in the collaterals helps determine the type of anastomosis and hence locate the vessel where the blood flow is obstructed in the system of the portal vein or the inferior vena cava. To that end a small area of the dilated venous branch is pressed between two fingers (with an attempt to empty this area from the blood). In a short lapse of time the pressure of the upper finger is released. If blood fills the vessel to the level of the other finger, the blood flows from the portal vein system to the inferior vena cava (i.e. in the downward direction). If the vessel remains empty, the blood flows upwards, i.e. from the vena cava inferior-system to the superior vena cava.

In patients with cachexia and pronounced enlargement of the liver, the right hypochondrium and epigastrium are protruded. If the abdominal wall is thin, the protruded surface is uneven and tuberous (in tumours or cysts of the liver). Only significantly enlarged gallbladder can be responsible for protrusion of the abdomen, especially in cachectic patients (in hydrops of the gallbladder, cancer of the common bile duct, or cancer of the pancreas head which compresses the common bile duct). The left hypochondrium is protruded in cases with considerably enlarged spleen attending cirrhosis of the liver (*hepatolienal syndrome*).

Percussion of abdomen

Percussion of abdomen in a vertical position of the patient is used for revealing a free fluid in the abdominal cavity and definitions of its level. By percussion on midline and lateral flanks from top to down, it is possible to differentiate the tympanic sound above intestines and the dull sound lower than fluid level.

In a horizontal position of the patient percussion of an abdomen is performed on midline from umbilicus to epigastrium and hypogastrium, and in lateral directions from umbilicus to flanks. With the purpose of differentiation a dull sound originated from free fluid and contents of intestines the physician can repeat percussion in lateral directions from umbilicus to flanks in position on one side of the patient’s body. In the presence of ascites the level of dull sound is changed in this position of patient.

With the help of *percussionary palpation method* the symptom of fluctuation of fluid is defined also the presence of an ascites. For this purpose the palmar surface of the left arm is put on the right half of abdomen in region of dullness. The right arm one-digital percussion mild impacts strike on the left half of abdomen according to V.P. Obraztcov. In the presence of a significant amount of loose fluid in an abdominal cavity the palm of the left arm clearly accepts fluctuation - jerky fluctuations of fluid. For a prevention of transfer of oscillating motions on the front abdominal wall it is possible to put a rib of an arm or the book along white line of the abdomen.

Percussion and palpation of liver and lien

Percussion of liver

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. Practically, absolute dullness is determined only because a position of the superior border varies depending on the size and configuration of the chest, the height of the right cupula of diaphragm, and also because the upper edge of the liver is deeply hidden behind the lungs. Finally, the liver usually becomes enlarged in the downward direction. This is determined by the position of its inferior edge.

Liver (as the dense organ) produces a percussion dull sound; right lung adjoining above - a clear pulmonary sound; stomach and intestine, adjoining below - a tympanic sound. As the right pulmonary inferior edge locates into space between anterior chest wall and liver, filling a costal-diaphragmatic sinus, the high border of the dulled sound coincides with true edge of a liver, and appearance of a dull sound corresponds to its part which is not covered with edge of lung. The border between a dulled and dull sound is designated as a high border of absolute hepatic dullness.

The upper border of a liver determined by percussion is always below the true anatomical border. The quiet percussion is applied to revealing of a high border of absolute hepatic dullness.

Percussion of the liver is performed according to the general rules of topographic percussion, i.e. a position of the pleximeter-finger should be collateral to the border which is necessary for determining, percussion from a clear sound to dull, border designate from the side of a clear sound. Quiet percussion is used to determine the absolute liver dullness. The direction of percussion is from top to bottom, along the vertical lines, like in determining the borders of the right lung. The border is detected by contrast between the

clear pulmonary resonance and liver dullness. The found border for each vertical line is marked on the skin by dots by the upper edge of the pleximeter-finger.

Superior border of absolute hepatic dullness is determined on parasternalis, midclavicular, right anterior axillary lines by percussion on intercostal spaces. On the parasternalis line a position of the border is specified by percussion on two overlying ribs above the dullness. Having received different percussion sound above them, a physician marks the border on the upper edge of the subjacent rib from them (routinely the 6-th).

In norm the superior border of absolute hepatic dullness passes on right parasternalis line at the level of the upper edge of the 6-th rib, on the midclavicular line - at the level of inferior edge of the 6-th rib, on anterior axillary line - at the level of inferior edge of the 7-th rib. The superior bound of relative dullness of a liver is posed on one rib above absolute dullness of the liver. The superior border of the liver can be determined posteriorly, but normally the determination ends by percussion in the three mentioned lines.

Delimitation of the *inferior border of absolute hepatic dullness* is difficult because of the presence of hollow organs in the vicinity of the liver. The stomach and the intestine give high tympanic sound that masks the liver dullness. The lightest (quietest) percussion should therefore be used.

The inferior border of absolute dullness of a liver is defined on anterior axillary, midclavicular, parasternalis right lines, anterior midline and parasternalis left lines. Determination of the *inferior border of absolute dullness* (according to Obraztsov and Strazhesko) should begin from the right part of the abdomen along the right anterior axillary line with the patient in the horizontal position. The pleximeter-finger is placed parallel to the expected inferior border of the liver, some distance away from it, so that tympany might first be heard (at the umbilical level or slightly below the navel). As the pleximeter-finger is then moved upwards, tympany is followed by absolute dullness. The point of disappearance of tympany is marked in each vertical line on the inferior edge of the pleximeter-finger.

When determining the left border of liver dullness, the pleximeter-finger is placed perpendicularly to the edge of the left costal arch, at the level of the 8-9-th ribs, and percussion is carried out to the right, directly over the edge of the costal arch, to the point where tympany changes to dullness (in the region of Traube's space).

Normally the inferior border of absolute dullness of a lying patient with normosthenic chest passes at the level of upper edge of 10-th rib in the right anterior axillary line, at the inferior 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 3-6 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) on the anterior median line; on the left parasternalis line - at the level of the inferior edge of a costal arch.

The lower margin of the liver in norm can be very depending on the shape of the chest and constitution of the patient, but it has only effect on the position in the anterior median line. The lower margin of the liver in a hypersthenic chest is slightly above the mentioned level, while in an asthenic chest below it, approximately midway between the base of the xiphoid process and the navel. If the patient is in the upright posture, the lower margin of the liver descends 1-1.5 cm. If the liver is enlarged, its lower margin is measured in centimeters from the costal arch and the xiphoid process.

Percussion gives information about the vertical dimensions of the area of liver dullness. The distance between the superior and inferior borders of absolute dullness is measured on the three vertical lines. The distance between the superior and inferior borders of absolute dullness of a liver compounds the *height of absolute hepatic dullness*. This distance in the right anterior axillary line is normally 10-12 cm, in the right midclavicular line - 9—11 cm, and in the right parasternal line - 8—10 m. The increase of the height of absolute hepatic dullness has relation mainly to enlargement of the right lobe of liver.

It is difficult to determine liver dullness on the back because it is masked by dullness of the thick layer of lumbar muscles, the kidneys, and the pancreas. In some cases, a 4—6 cm wide band of liver dullness can be determined. This precludes erroneous diagnosis of liver enlargement in cases where the liver descends below the right costal arch, or where it is turned anteriorly round its axis; dullness then becomes narrower. Definition of height of hepatic dullness enables to distinguish the enlarged liver from its shift which is observed at low standing a diaphragm owing to pulmonary emphysema or the general enteroptosis.

Outlining the liver by percussion is diagnostically important. But ascending or descending of the superior margin of the liver is usually associated with extrahepatic changes (high or low diaphragm, sub-diaphragmatic abscess, pneumothorax, or pleurisy with effusion). The superior margin of the liver can ascend only in echynococcosis or cancer of the liver. Elevation of the inferior margin indicates diminution of the liver; it can also occur in meteorism and ascites which displace the liver upwards. The lower border usually descends when the liver is enlarged (due to hepatitis, cirrhosis, cancer, echynococcosis, blood congestion associated with heart failure, etc.). But it can sometimes be explained by low position of the diaphragm. Systematic observation of the liver borders and changes in the liver dullness gives information on changes in its size during the disease.

Examination of the size of liver according to M.G. Kurlov method

The size determination of a liver according to M.G. Kurlov's method is widely used in clinical practice. According to this method five points are marked, and three dimensions are taken. The first and second points of Kurlov correspond to superior and inferior borders of absolute liver dullness on the right midclavicular line, the first dimension of liver according to M.G. Kurlov is measured between them (in norm of 9 sm).

The third point of Kurlov is marked at intersection of the anterior midline and the perpendicular line installed from the first point of Kurlov (the level of superior border of absolute liver dullness on the right midclavicular line). This point conventionally corresponds to the superior border of a liver on the anterior midline. The fourth point according to Kurlov is marked on the inferior border of a liver on the anterior midline. The second dimension of a liver according to M.G. Kurlov is measured between the superior and inferior borders of the liver on the anterior midline (in norm 8 sm).

The fifth point according to M.G. Kurlov corresponds to the left border of liver dullness. The pleximeter-finger is placed perpendicularly to the edge of the left costal arch, at the level of the 8-9-th ribs between the left anterior axillary and midclavicular lines, and quiet percussion is performed to the right, directly over the edge of the costal arch, to the point where tympany changes to dullness (in the region of Traube's space). The third dimension of liver according to M.G. Kurlov is measured between the third and fifth point of Kurlov (superior border on anterior midline and left border of liver dullness on a costal arch - in norm 7 sm). An increase of the second and third dimensions testifies a pathological process of the left hepatic lobe, an increase of the first dimension – of right hepatic lobe.

Percussion of gallbladder

The gallbladder cannot be determined by percussion as a rule. But if its enlargement is pronounced, it can be determined by very light percussion.

Percussion is used not only to determine the borders of the liver and the gallbladder (topographic percussion) but also to assess their condition: careful percussion of the area overlying an enlarged liver or the gall bladder causes painful sensations in the presence of inflammation (hepatitis, cholecystitis, pericholecystitis, etc.). Succussion on the right costal arch also causes pain in diseases of the liver and the bile ducts, especially in cholelithiasis (Ortner's symptom).

Palpation of liver

Surface palpation in diseases of the liver can reveal a tender zone in the right hypochondrium and epigastrium. Especially severe local pain (caused even by a slight touch on the anterior abdominal wall in the zone overlying the gallbladder) is observed in acute cholecystitis and biliary colic. In chronic

cholecystitis slight or moderate tenderness is only revealed at the point of projection of the gall bladder fundus onto the anterior abdominal wall. In healthy subjects this point is found immediately below the right costal arch by the lateral edge of the right rectus abdominis muscle.

Palpation of a liver purposes detection of the inferior edge, definition of its localization, form, lineament, consistence, character of surface and tenderness. Percussion of hepatic inferior borders on all lines foreruns always to palpation of the liver.

The liver is palpated by the Obraztsov and Strazhesko method. As the lower edge of the liver descends to meet the examining fingers during a deep inspiration it slides over the fingers and thus becomes detectable. It should be remembered that the respiratory mobility of the liver is the highest compared with that of the other abdominal organs because the liver is the closest to the diaphragm. It follows therefore that during palpation of the liver, the active role belongs to its respiratory mobility rather than to the palpating fingers (as is the case with palpation of the intestine).

Position of the patient. The patient should lay horizontally with slightly raised head and the stretched legs. The hands routinely settle down along a trunk or are crossed on a chest with the purpose of restriction of mobility of a chest in the sides on an inspiration. It promotes increase of diaphragm motility according to a liver in the upper-inferior direction that is important for a palpation of a lower edge of a liver.

The patient should stand or lie during palpation of the liver and the gall bladder. But in certain cases the liver can be easier palpated if the patient lies on his left side: the liver hangs by gravity from under the hypochondrium and its inferio-anterior edge can thus be better palpated.

Position of the doctor. The examiner sits by the right side, facing the patient. He places four fingers of his left hand on the right costal arch of the patient chest and uses his left thumb to press on the costal arch to move the liver closer to the palpating fingers of the right hand and to prevent expansion of the chest during inspiration. It stimulates greater excursions of the right cupula of diaphragm. The palm of the right hand is placed flat on the abdomen below the costal arch between the right parasternalis and midclavicular lines. The slightly flexed fingers press lightly on the abdominal wall.

Procedure of palpation of the liver. The patient is asked to take a deep breath; the liver descends to touch the palpating fingers and then slides to bypass them. The examiner's hand remains motionless. The procedure is repeated several times. The position of the liver margin varies depending on conditions. It is therefore necessary first to determine the lower margin of the liver by percussion before positioning the palpating fingers.

Common rules should be followed during palpation of the liver and the gall bladder. Special attention should be paid to the antero-inferior margin of

the liver whose properties (outlines, form, tenderness, consistency) are indicative of the condition of the liver, its position, and configuration. In many cases (especially if the liver is enlarged or lowered) the liver can be palpated not only from the left hypochondrium to the right hypochondrium, but its superio-anterior surface becomes palpable as well.

The four moments of deep sliding palpation must be taken into account for palpation of the liver:

The first moment is the position of arms. The right arm is placed at the region of right hypochondrium on the right parasternalis line with slightly bent fingers whose tips should be 3-5 sm lower than the percussionaly found inferior border of the liver. The left arm covers the inferior department of the right half of chest so that the big finger is placed on the anterior surface of the right costal arch while other fingers (2-5-th fingers) settled down behind. Thus we aspire to confine motility of the chest during an inspiration and to strengthen motion of the diaphragm from top to bottom.

The second and third moments (formation of the artificial pouch according to V.P. Obraztsov) are united and performed during the one expiration. For this purpose it is necessary to make a superficial motion to dislocate a skin fold downwards and to plunge tips of fingers of the right arm in depth of the abdominal cavity during the one expiration when there is a maximal release of the anterior abdominal wall muscles, and the liver follows the diaphragm.

The fourth moment is palpation of the inferior edge of a liver. After dipping a palpating arm in abdomen and formation of the artificial pouch the patient is asked to take a deep breath. The liver descends to touch the palpating fingers and then slides to bypass them.

If by time of the inspiration the perception of hepatic edge was not possible, palpation of the liver should be repeated. The tips of fingers of the right arm must be transferred 1-2 sm upwards. If repeated result is negative the research is retried again and again, positioning tips of fingers each time higher and higher. Unsuccessful finally palpation of a liver is considered in that case when the right arm reaches the edge of the costal arch. In this case palpation of the liver is recommended to be repeated from the very beginning. The tips of fingers of the right arm must be transferred 2-3 sm lower than their initial situation.

The lower edge of a normal liver is usually palpated between the right parasternal and midclavicular line; the liver is impalpable to the right of the midclavicular line because it is located behind the costal arch; the liver is hardly palpable to the left of the line because of the abdominal muscles. An enlarged or consolidated liver can be palpated in all lines. It is easily to perform a palpation on the right parasternalis line as here the inferior edge of a liver settles down in standard conditions on 2 sm of below costal arch. On a right midclavicular line it is as a rule at a level of a costal arch.

According to Obraztsov, normal liver can be palpated in 88 per cent of cases. Physical properties of the liver can be determined by palpating its lower edge (it can be soft, firm, rough, sharp, rounded, tender, etc.). The margin of an unaffected liver palpated at the height of a deep inspiration is 1—2 cm below the costal arch. It is soft, sharp or slightly rounded under the form, readily bending, smooth and insensitive.

The liver of patients with pronounced distension of the abdomen should be examined with the empty stomach to facilitate palpation. In accumulation of much fluid in the abdominal cavity (ascites) the liver is not always palpable if the patient is lying. The patient should then be examined in the erect position, or he may lie on his left side. If the amount of fluid in the abdomen is very large, it should be released by paracentesis.

Expressed accumulation of fluid in an abdominal cavity (ascites) often very much complicates carrying out of a palpation of a liver on V.P. Obraztsov. In accumulation of much fluid in the abdominal cavity, ballotment should be used to palpate the liver. To that end the right hand (two or four flexed fingers) should be placed on the lower right part of the abdomen, perpendicularly to the expected lower edge of the liver. The abdominal wall is given a sharp tap from the palpating fingers which move upward to meet the firm object, the liver, which is first tossed to the deeper parts of the abdominal cavity but is then returned back to strike the fingers (a sign " floating ice ").

Palpation is painful if the liver is inflamed and the affection extends onto the liver capsule; the liver is also tender when it is distended (e.g. in blood congestion due to heart failure). The liver of a healthy subject (if it is accessible to palpation) is soft; it becomes firmer in hepatitis, hepatosis, and cardiac congestion. The liver is especially firm in cirrhosis. Its edge becomes sharp and the surface smooth or covered with small tubercles. The liver is also firm in the presence of tumour and multiple metastases of cancer. Its surface then becomes covered with rough tubercles (surface metastases) and the lower margin is rough. The liver is firm in amyloidosis. Comparatively small tumours and echinococcosis can sometimes be palpated. Protrusion of the lower margin of an enlarged liver is assessed with respect to the costal arch in the right anterior axillary line, right midclavicular line, right parasternal line, anterior median line, and left parasternal line. Palpation verifies the findings obtained by percussion of the liver.

The gallbladder cannot be palpated in healthy subjects because of its soft consistency and the insignificant protrusion. But if the gallbladder is enlarged (hydrops, stones in the bladder, cancer, etc.) it becomes palpable. The position of the patient for palpation of the gallbladder is the same as in palpation of the liver. After the margin of the liver has been found, the gall bladder should be palpated at the lateral edge of the right rectus abdominis muscle. The palpation technique is the same as that for palpation of the liver.

The gallbladder can easier be found by moving the palpating fingers in the direction perpendicular to the axis of the gallbladder. The bladder is felt like a pear of variable size, firmness and tenderness depending on the character of pathology in the gallbladder proper or the surrounding organs (e.g. the gallbladder is enlarged, soft, and elastic in tumour-obstructed bile duct: Courvoisier-Terrier sign; the bladder is firm and tuberous in the presence of newgrowths in its wall, in overfilling with stones, in inflammation of the wall, etc.). An enlarged gallbladder is mobile during respiration (it performs lateral pendulum-like movements). The gallbladder loses its mobility in inflammation of the overlying peritoneum (pericholecystitis). In the presence of cholecystitis and cholelithiasis, the palpation is difficult because of sharp pain and reflectory rigidity of the muscles of the anterior abdominal wall.

The described technique of palpation of the liver and the gallbladder is simple and effective. There are others palpation techniques. But most of them differ only in the position of the examining hands (Glenard, Mathieu, Chaufard, Chiray) or the position of the physician himself with respect to the patient during examination. But none of these techniques used for palpation of the liver and the gallbladder can give significant advantages over others. Success of palpation depends not on the variety of techniques but on the experience of the examiner.

Percussion of spleen

Since the spleen is surrounded by hollow organs (the stomach, the intestine), which give loud tympany during percussion, it is impossible to determine accurately its borders by percussion. The percussion of a lien in view of its small size and the close surrounding with gassy organs (lung, a stomach and an intestine) is inconvenient. The lien is placed in norm under the left dome of a diaphragm in the lateral part of the left hypochondrium, adjoining the chest wall between the 9- and -11-th ribs. The longitudinal axis of the spleen passes in an oblique, anteroposterior direction, parallel to the 10-th rib.

During percussion the patient lies usually on his right side with a little bit bent left leg and the left arm stretched forward, more rarely the patient stands upright. Quiet percussion should be used with transition from clear resonance to dullness. Obraztsov's percussion is recommended. Percussion of the superior and the inferior borders of the lien is performed first, the anterior and posterior borders of the lien are percussed second.

For delimitation of the *superior border of lien* the finger- pleximeter is placed parallel to the ribs at the 3-d or 4-th intercostal space on the left medium axillary line. Percussion is conducted from top to bottom before appearance of the dulled sound. The border is marked on the edge of the finger - pleximeter from the side of a clear sound.

Delimitation of the *inferior border of lien* is performed also on the left medium axillary line. The finger- pleximeter is positioned below the inferior edge of the left costal arch. Percussion is conducted upwards the spleen dullness, marking the border from the side of a tympanic note.

For delimitation of the *anterior border of lien* it is necessary to continue mentally its superior and inferior borders in the line of umbilicus. In the interspace between them the finger - pleximeter is positioned parallel to the required border. Starting from the umbilicus a quiet percussion is proceeded on the 10-th intercostals space. The required border of lien is marked on the side of a tympanic sound.

For delimitation of the *posterior border of lien* it is necessary to find the 10-th rib corresponding to its longitudinal axis and to place a finger - pleximeter on these lines parallel to the required border (i.e. upright) in the space between the posterior axillary and scapular lines. Percussion is performed immediately on the 10-th rib before appearance of a dulled sound. The posterior border of lien is marked from the side of a tympanic sound.

Normally the superior border of the splenic dullness corresponds to the lower edge of IX rib, inferior border - to the lower edge of XI ribs. The anterior border of the splenic dullness is on 1-2 sm outside of anterior axillary line, the posterior border – on the posterior axillary line.

The measurement of the lines bridging the superior and inferior, anterior and posterior borders of splenic dullness gives conception about size of lien. Its width is 4—6 cm, its length is 6-8 sm.

Palpation of spleen

In norm the lien is not palpated, as there is placed deeply in a left hypochondrium. Its inferior pole does not reach the lower edge of the left costal arch on 3-4 sm. That is why any case of a successful palpation of a lien testifies its enlargement or splenoptosis (descent of lien). The percussion distinguishes the enlargement or ptosis of lien and adds to a palpation in recognition of various changes of this organ. Palpation of a spleen is in principle precisely the same, as well as a liver. The essence of it consists in reception of tactile perception of edge of a lien at its shift together with a diaphragm downwards during the deep inspiration.

The spleen should be palpated with the patient in the recumbent position or on his right side. In the former case the patient should lie on a low pillow, the arms and the legs being stretched. If the patient lies on his right side, his head should be slightly down, the left elbow bent and resting freely on the chest; the right leg should be stretched and the left knee bent and drawn up to the chest. The prelum is relaxed to a maximum. In this position, the spleen is displaced anteriorly to facilitate its palpation even if it is slightly enlarged. The physician sits on the right side of the patient and faces him.

The first moment of palpation is the position of arms. The left hand of the physician is placed on the left part of the patient's chest, between the 7-th and 10-th ribs in the axillary lines and slightly pressed on the chest to limit its respiratory movements. The physician's right hand is placed on the anterolateral surface of the patient's abdominal wall so that the tips of the 2-5-th fingers are positioned opposite 10-th ribs 3-5 sm below the left costal arch, and the back of the arm is in range of an umbilicus. The position of the distal phalanxes depends on localization of the anterior border of the lien preliminarily found by means of percussion. If it is below the level of the costal arch the palpating fingers should be displaced in the direction of the umbilicus 3-5 sm downwards from the anterior border of the lien.

The second and third moments (the formation of an artificial pouch according to V.P. Obraztsov) are united and performed during the one expiration. For this purpose it is necessary to make a superficial motion to dislocate the skin fold downwards to umbilicus and to plunge the tips of fingers of the right arm in depth of the abdominal cavity when a maximal release of the anterior abdominal wall and the lien departs up after a diaphragm.

The fourth moment is the palpation of the lien. After dipping the palpating arm in depth the abdomen and building the artificial pouch according to V.P. Obraztsov the patient is asked to make a deep inspiration. If the spleen is palpable (and provided the palpation is performed correctly), it is displaced during the inspiration by the descending diaphragm to come in contact with the palpating fingers of the right hand and to slip over them. This manipulation should be repeated several times in order to examine the entire palpable edge of the spleen. If in time of the inspiration the perception of the edge of the lien is not possible received, palpation of the lien is retried. The fingers of the right arm must be transferred 1-2 sm upwards in the direction of the edge of the left costal arch. Palpation of the lien is performed until the distal phalanxes of the right arm feel the lien or the edge of the left costal arch. In the latter case it is considered that palpation of the lien is not possible and, hence, the lien is not enlarged. If the lien is determined by palpation at the inferior edge of the left costal arch there is a 1.5 (and more) increase of the lien.

The size, shape, character of the surface, sensitivity, consistence, mobility, and configuration of the anterior edge of the spleen should be determined by palpation.

The *four degrees of enlarged lien* are distinguished:

I degree - lien protrudes from under the left costal arch not more than the width of one patient’s finger; II degree - lien reaches the middle of distance between the umbilicus and the left costal arch; III degree - lien reaches the midline of the abdomen, i.e. occupies only the left half of the

abdomen; IV degree - lien reaches to the right half of the abdominal cavity and the pelvic cavity.

The characteristic peculiarity of lien is one or several notches (incisures) on the anterior edge of the spleen can be palpated if its enlargement is considerable. The notches are used to identify the spleen (to differentiate it from other organs, e.g. from the left kidney, tumors originated from the left kidney, splendid curvature of a transverse colon and caudal part of pancreas).

A normal spleen is impalpable. It can only be palpated in rare cases of extreme splenoptosis, and more frequently in enlargement of the organ. The anterior surface of the enlarged spleen emerges from under the costal arch and also becomes palpable.

The spleen is enlarged in some acute and chronic infectious diseases (typhus, viral hepatitis, sepsis, malaria, etc.), in liver cirrhosis, thrombosis or compression of the splenic vein, and also in many diseases of the hemopoietic system (hemolytic anemia, thrombocytopenic purpura, acute and chronic leucosis). Considerable enlargement of the spleen is called *splenomegaly*. The greatest enlargement of the spleen is observed at the terminal stage of chronic myeloleucosis: it often occupies the entire left part of the abdomen, while its lower pole is found in the small pelvis.

The spleen is not firm in acute infectious diseases; it is especially soft (the consistency of dough) in sepsis. In chronic infectious diseases, liver cirrhosis, and leucosis the spleen is firm, especially in amyloidosis.

In most diseases the spleen is insensitive to palpation. It becomes tender in infarction, perisplenitis, and in distension of the capsule, due to the rapid enlargement, e.g. in venous blood congestion due to thrombosis of the splenic vein. The spleen surface is usually smooth; the edges and the surface are irregular in perisplenitis and old infarctions (depressions in the surface). In syphilitic gummas, echinococcosis, cysts and very rare tumours of the spleen its surface is tuberous.

The spleen is normally quite mobile, but the mobility becomes limited in perisplenitis. A markedly enlarged spleen remains motionless during respiration but it can however be displaced by the palpating fingers.

Subjective and objective examination of patients with diseases of kidneys and urinary tract

Inquiry (Subjective examination)

Complaints

Patients with diseases of the kidneys complain most commonly of pain in the lumbar region, disordered urination, edema, headache, and dizziness.

They may also complain of deranged vision, pain in the heart, dyspnea, absence of appetite, nausea, vomiting, and elevated body temperature. But diseases of the kidneys may also proceed without any symptoms of renal or general clinical insufficiency.

If the patient complains *of pain*, its location should first of all be determined. Pain of renal origin localizes frequently in the lumbar region. If the ureters are affected, the pain is felt by their course. If the bladder is involved, pain is suprapubical. Radiation of pain into the perineal region is characteristic of an attack of nephrolithiasis.

The character of pain should then be determined. It is necessary to remember that the renal tissue is devoid of pain receptors. The pain is felt when the capsule or the pelvis is distended. Dull and boring pain in the lumbar region occurs in acute glomerulonephritis, abscess of the perirenal cellular tissue, in heart decompensation ("congestive kidney"), in chronic pyelonephritis (usually unilateral) and less frequently in chronic glomerulonephritis. Pain arises due to distension of the renal capsule because of the inflammatory or congestive swelling of the renal tissue. Sharp and suddenly developing pain on one side of the loin can be due to the renal infarction. The pain persists for several hours or days and then subsides gradually. The pain is rather severe in acute pyelonephritis: inflammatory edema of the ureter interferes with the normal urine outflow from the pelvis and thus causes its distension. The pain is usually permanent. Some patients complain of attacks of severe piercing pain in the lumbar region or by the course of the ureter. The pain increases periodically and then subsides, i.e. has the character of *renal colic*. Obstruction of the ureter by a calculus or its bending (movable kidney) is the most common cause of this pain, which is usually attended by spasmodic contraction of the ureter, retention of the urine in the pelvis, and hence its distension. The spasmodic contractions and distension of the pelvis account for the pain. Pain in renal colic is usually unilateral. It radiates into the corresponding hypochondrium and most frequently by the course of the ureter to the bladder and to the urethra. This radiation of pain is explained by the presence of nerve fibres (carrying the impulses from kidneys, ureters, genital organs and the corresponding skin zones) in the immediate vicinity of the relevant segments of the spinal cord (D_X-D_{XII} and L_I-L_{II}). This facilitates propagation of the excitation. Patients with renal colic (like those with colic of other etiology) are restless; they toss in bed. Patients with severe pain of other etiology would usually lie quiet in their beds (movements may intensify the pain).

The conditions promoting pain should be established. For example, pain in nephrolithiasis can be provoked by taking much liquid, jolting motion, or the like; pain is provoked by urination in cystitis. Difficult and painful urination is observed in *stranguria*. Patients with urethritis feel a burning pain in the urethra during or after urination.

It is necessary also to establish the agent that lessens or removes the pain. For example, atropine sulphate, hot water-bottle or warm bath helps in renal colic. Since these remedies only help in spasmodic pain by removing spasms of the smooth muscles, their efficacy in renal colic confirms the leading role of the ureter contraction in the pathogenesis of this pain. Pain of the renal colic-type in patients with movable kidney may lessen with changing posture: urine outflow improves with displacement of the kidney. Pain slightly lessens in patients with acute paranephritis if an ice pack is placed on the lumbar region and if the patient is given analgesics.

Many renal diseases are attended by *deranged urination:* changes in the daily volume of excreted urine and in the circadian rhythm of urination.

Secretion of urine during a certain period of time is called *diuresis.* Diuresis can be positive (the amount of urine excreted exceeds the volume of liquid taken) or negative (the reverse ratio). Negative diuresis is observed in cases of liquid retention in the body or its excess excretion through the skin, by the lungs (e.g. in dry and hot weather). Positive diuresis occurs in resolution of edema, after administration of diuretics, and in some other cases. Deranged excretion of urine is called *dysuria.*

Increased amount of excreted urine (over 2 1 a day) is called *polyuria.* It can be of renal and extrarenal etiology. Polyuria is observed in persons who take much liquid, during resolution of edema (cardiac or renal), and after taking diuretics. Long-standing polyuria with a high relative density of urine is characteristic of diabetes mellitus. In this case polyuria arises due to a deranged resorption of water in renal tubules because of increased osmotic pressure of the urine rich in glucose. Polyuria occurs in diabetes incipidus because of insufficient supply of antidiuretic hormone secreted into blood by the posterior pituitary.

Polyuria also occurs in the absence of sensitivity of the tubules to the ADH, in affected interstice of the renal medulla of various nature, in hypokaliaemia, and hypo- and hypercalcaemia.

Persistent polyuria with low specific gravity of urine (hyposthenuria) is usually a symptom of a severe renal disease, e.g. chronic nephritis, chronic pyelonephritis, renal arteriolosclerosis, etc. Polyuria in such cases indicates the presence of a neglected disease with renal insufficiency and decreased reabsorption in renal tubules.

Decreased amount of excreted urine (less than 500 ml a day) is called *oliguria.* It can be not connected directly with renal affections (*extrarenal oliguria*). For example, it can be due to limited intake of liquid, during staying in a hot and dry room, in excessive sweating, intense vomiting, profuse diarrhea, and during decompensation in cardiac patients. But in certain cases oliguria is the result of diseases of the kidneys and the urinary ducts (*renal oliguria*), such as acute nephritis, acute dystrophy of the kidneys in poisoning with corrosive sublimate, etc.

A complete absence of urine secretion and excretion is called *anuria*. Anuria persisting for several days threatens with possible development of uremia and fatal outcome. Anuria may be caused by the deranged secretion of urine by the kidneys (*secretory anuria*) which occurs in severe form of acute nephritis, nephronecrosis (poisoning with sublimate or other nephrotoxic substances), transfusion of incompatible blood, and also some general diseases and conditions such as severe heart failure, shock, or profuse blood loss.

In certain cases the secretion of urine is normal but its excretion is obstructed mechanically (obstruction of the ureters or the urethra by a calculus, inflammatory edema of the mucosa, proliferation of a malignant tumour). This is called *excretory anuria*. It is usually attended by strong pain in the loin and the ureters due to distension of the renal pelvis and the ureters. Excretory anuria is often attended by renal colic.

Renal (secretory) anuria can be of reflex origin, e.g. in severe pain (contusion, fractures of the extremities, etc). Anuria should be differentiated from *ischuria*, when the urine is retained in the bladder and the patient is unable to evacuate it. This occurs in compression or other affection of the spinal cord, and in loss of consciousness.

Pollakiuria (frequent micturition) is observed in certain cases. A healthy person urinates from 4 to 7 times a day. The amount of excreted urine during one micturition is from 200 to 300 ml (1000-2000 ml a day). But frequency of micturition may vary within wider range under certain conditions: it may decrease in limited intake of liquid, after eating much salted food, in excessive sweating, in fever, and the like, or the frequency may increase (polyuria) if the person takes much liquid, in getting cold, and the like circumstances. Frequent desire to urinate with excretion of scanty quantity of urine is the sign of cystitis. A healthy person urinates 4-7 times during the day time; a desire to urinate during night sleep does not arise more than once. In the presence of pollakiuria the patient feels the desire to urinate during both day and night. In the presence of chronic renal insufficiency and if the kidneys are unable to control the amount and concentration of excreted urine in accordance with the amount of liquid taken, physical exertion, the ambient temperature, or other factors important for the liquid balance in the body, the patient urinates at about equal intervals with evacuation of about equal portions of urine. This condition is called *isuria.*

Under certain pathological conditions, the frequency of urination is normal during the day time but increases during night. The amount of urine excreted during night often exceeds the amount of daily urine *(nycturia).* Nocturnal enuresis (nycturia) and oliguria during day time occur in cardiac decompensation and are explained by a better renal function at night, i.e. at rest (cardiac nycturia). Nycturia may concur with polyuria in renal dysfunction, at the final stage of chronic glomerulonephritis, chronic pyelitis,

vascular nephrosclerosis, and other chronic renal diseases (renal nycturia). In the presence of isuria and nycturia of renal origin, which arise due to the loss by the kidneys of their concentrating ability, the specific gravity of the urine is monotonous. The condition is known as *isosthenuria.* The specific gravity of urine is usually decreased *(hyposthenuria).* The specific gravity of urine varies from 1.009 to 1.011, i.e. approaches the specific gravity of primary urine (plasma ultrafiltrate) in patients with pronounced nephrosclerosis, which is the final stage of many chronic renal diseases.

Some diseases of the bladder and the urethra are attended by difficult and painful urination. The patient would complain of change in the colour of the urine, its cloudiness, and traces of blood.

Edema is observed in acute and chronic diffuse glomerulonephritis, nephrotic syndrome, amyloidosis, and acute renal excretory dysfunction (anuria). It is important to ask the patient about the site that was the first to be attacked by edema, the sequence of edema spreading, and the rate of intensification of this phenomenon.

Headache, *dizziness*, and *heart pain* may result from kidney affections. These symptoms occur in those renal diseases which are attended by considerable increase of arterial blood pressure, e.g. in acute and chronic glomerulonephritis or vascular nephrosclerosis. A pronounced and persistent increase in the arterial pressure can be among the causes of deranged vision (neuroretinitis).

Patients with diseases of the kidneys can complain of weakness, indisposition, impaired memory and work capacity and deranged sleep. Vision may be deranged along with skin itching and unpleasant breath. Dyspeptic disorders sometimes include loss of appetite, dryness and unpleasant taste in the mouth, nausea, vomiting, and diarrhea. All these phenomena are associated with retention in the body of protein decomposition products due to renal insufficiency which develops at the final stage of many chronic renal diseases, and sometimes in acute diseases attended by retention of urine during several days.

Fever is the common symptom of infectious inflammatory affections of the kidneys, the urinary ducts and perirenal cellular tissue.

History of the present disease

When questioning the patient, it is necessary to establish the connection of the present disease with previous infections (tonsillitis, scarlet fever, otitis, acute respiratory diseases). This sequence is especially characteristic of acute glomerulonephritis. But it is sometimes difficult to establish the time of onset of the disease because some chronic affections of the kidneys and the urinary ducts can for a long time be latent. Moreover, when questioning the patient, it is necessary to find out if he had deranged hearing or vision in his childhood that might be suggestive of congenital renal pathology.

Special attention should be given to the presence in the patient's past history of diseases of the kidneys and the urinary ducts (acute nephritis, pyelitis, cystitis) or symptoms that might suggest them (dysuria, hematuria, edema, arterial hypertension, attacks of pain in the abdomen or loin resembling renal colics), since these symptoms can be connected with the present renal pathology. In certain cases the cause and the time of onset of grave kidney affections (necronephrosis) can be established by revealing industrial or domestic poisoning, intentional (or by mistake) taking of some poisons (corrosive sublimate, preparations of bismuth, phosphorus, silver, large doses of sulpha preparations, or of some antibiotics, e.g. aminoglycosides, expired tetracyclines, phosphorus compounds), transfusion of incompatible blood, etc. Amidopyrin, phenacetin, barbiturates, camphor, and some other medicines can cause allergic changes in the kidneys.

The patient must be asked about the character of the disease course: it may be gradual (arteriolosclerosis, chronic diffuse glomerulonephritis, amyloidosis of the kidneys), or with periodical exacerbations (chronic pyelonephritis, chronic diffuse glomerulonephritis). It is necessary to establish the cause of exacerbations, their frequency, clinical signs, the character of therapy given and its efficacy, the causes inducing the patient to seek medical help.

Anamnesis (Past history)

Special attention should be given to the factors that might provoke the present disease or have effect on its further course. For example, a common factor promoting development of acute and chronic nephritis and pyelonephritis is chilling and cooling (poor housing or working conditions, draughts, work in the open, acute cooling of the body before the disease). Spreading of genital infection onto the urinary system can be the cause of pyelonephritis. It is necessary to establish the presence or absence in the past of tuberculosis of the lungs or other organs. This helps establish the tuberculous nature of the present disease of the kidneys.

It is necessary to establish if the patient has some other diseases that might cause affections of the kidneys (collagenosis, diabetes mellitus, certain diseases of the blood, etc.). Various chronic purulent diseases (osteomyelitis, bronchiectasis) can be the cause of amyloidosis of the kidneys. Occupations associated with walking, riding, weight lifting, etc., can have their effect on the course of nephrolithiasis and provoke attacks of renal colic. Some abnormalities of the kidneys, nephrolithiasis, amyloidosis, etc., can be inherited. It is also necessary to record thoroughly the information on past operations on the kidneys or the urinary ducts.

When examining women, it is important to remember that pregnancy can aggravate some chronic diseases of the kidneys and be the cause of the so-called nephropathy of pregnancy (toxemia of late pregnancy).

Physical Examination

Inspection

Inspection of the patient should give the physician the idea of the gravity of the patient's condition. Very grave condition with loss of consciousness may be due to severe affections of the kidneys attended by renal insufficiency and uremic coma; the condition may be satisfactory or of moderate gravity (in milder cases). It is necessary to pay attention to the patient's posture in bed: active (at initial stages of many diseases of the kidneys), passive (in uremic coma), or forced (in paranephritis; the patient may lie on his side with the leg flexed, bringing the knee to the abdomen on the affected side). In the presence of renal colic the patient is restless, tosses in bed, groans or even cries from pain. Convulsions are observed in the presence of uremic coma, renal eclampsia, and nephropathy of pregnancy (toxemia of late pregnancy with involvement of the kidneys).

Edema is characteristic of acute and chronic glomerulonephritis, nephrotic syndrome, and amyloidosis of the kidneys. The appearance of the patient with edema of the renal origin is quite specific. The face is pallid, swollen, with edematous eyelids and narrowed eye-slits (*facies nephritica*). In patients with more pronounced signs of pathology, edema affects the upper and lower extremities and the trunk (anasarca).

The colour of the patient's skin is also important. Edematous skin in chronic nephritis is pallid due to the spasm of skin arterioles, and anemia which attends this disease. The skin is wax-pallid in amyloidosis and lipoid nephrosis. It should be remembered that in cardiac edema (as distinct from renal edema) the skin is more or less cyanotic.

When inspecting a patient with chronic nephritis, it is possible to observe scratches on the skin and coated dry tongue; an unpleasant odour of ammonia can be felt from the mouth and skin of the patient (*factor uremicus*). All these signs characterize chronic renal insufficiency (uremia).

Inspection of the abdomen and the loin does not usually reveal any noticeable changes. But in the presence of paranephritis, it is possible to notice swelling on the affected side of the loin. In rare cases, an especially large tumour of the kidney may be manifested by protrusion of the abdominal wall. Distended bladder can be protruded over the pubic bone in thin persons. The distension can be due to overfilling of the bladder, for example, due to retention of urine in adenoma or cancer of the prostate.

Percussion of kidneys

It is impossible to percuss the kidneys in a healthy subject because they are covered anteriorly by the intestinal loops which give tympany. Dullness can only be determined in the presence of very marked enlargement of the kidneys.

A much more informative method for examination of the kidneys is *tapping*. The physician places his left hand on the patient's loin and using his right hand (palm edge or fingers) 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, paranephritis, inflammation of the pelvis, and also in myositis and radiculitis. This decreases the diagnostic value of Pasternatsky's symptom.

Percussion of urinary bladder

The finger-pleximeter is placed horizontally, i.e. collaterally to a pubis, on anterior abdominal wall at a level of umbilicus or slightly below it, and a quiet percussion is performed from top to down on anterior midline in the direction of pubis. If urinary bladder is full of urine, there is dullness on percussion above a pubis at percussion. If it is empty the tympanic note down to a pubis in a vertical and horizontal position of the patient is determined.

Palpation of kidneys

The posterior location of the kidneys, and also the absence of anterior approach to them due to the interference of the costal arch, makes palpation of the kidneys difficult. Relaxation of the prelum and pronounced cachexia can be attended by certain ptosis of the kidneys and make them accessible to palpation even in healthy subjects. But the results of palpation can only be reliable in considerable enlargement of the kidneys (at least 1.5-2 times, e.g. due to formation of a cyst or a tumour), or their displacement by a tumour, or in cases with a floating kidney. Bilateral enlargement of the kidneys is observed in polycystosis.

Palpation of kidneys becomes possible in enlarged volume of kidneys or at their ptosis. Palpation of kidneys should be effected in vertical (according to S.P. Botkin) and horizontal (according to V.P. Obraztsov) positions of patient. Palpation of kidney in vertical position of the patient is especially valuable for revealing nephroptosis, it is especial in case of its small shift. In a lying position a kidney is reverted, and any deep respiratory motions of a diaphragm are not capable to shift it downwards to be available for palpation.

It is necessary to remember that the kidneys can move about in the range of 2-3 cm in the proximal and distal directions when the subject changes his position from horizontal to vertical, and also during respiratory movements of the diaphragm. Passive movements of the kidneys transmitted from the diaphragm during inspiration and expiration should be taken into consideration during palpation: the Obraztsov-Strazhesko palpation method should be used. The patient should be palpated in the lying or standing position.

When the patient is in the horizontal position, his 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 freely placed on the chest.

The first moment of palpation is the position of arms. Palpation of kidneys in a horizontal position of researched patient, as well as in a vertical position, is performed by two arms, i.e. bimanually. The physician should hold a position by the right side of the patient with his left hand under the patient's loin, slightly below the 12-th rib so that the finger tips are placed 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. It is connected with a little bit slanting position of kidneys which inferior poles are located far from a vertebrae column, rather than superior poles.

The second moment is the formation of artificial skin bunch by superficial movement of a palpating arm upward.

The third moment is the gradual dipping the tips of fingers of the right arm into an abdomen on an expiration when a maximal abdominal wall muscles relaxation. It is possible routinely to reach a posterior abdominal wall for 2-3 expirations. 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 of expirations to reach the posterior abdominal wall, while the left hand presses the lumbar region to meet the fingers of the right hand.

The fourth moment is the palpation of the kidney. 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 contrast to other organs, an enlarged or ptosed kidney can be examined by ballottement (Guyon's sign): the right hand feels the kidney

while the fingers of the left hand strike rapidly the lumbar region in the angle between the costal arch and the longissimus thoracic muscles: the fingers of the right hand feel vibration of the kidney. In deranged urine outflow through the ureter and in pronounced distension of the renal pelvis by the accumulated urine or pus, liquid fluctuation can be felt during palpation of the kidney.

If the physician palpates some formation where he expects to find a kidney, he must check reliably if this is actually a kidney because it is easy to mistake for the kidney an overfilled and firm part of the large intestine, tumor of perirenal cellular tissue (lipoma, fibroma), an enlarged right lobe of the liver, the gall bladder (during palpation of the right kidney), or an enlarged or displaced spleen (during palpation of the left kidney). The kidney is a bean-shaped body with a smooth surface, slipping upwards from under the palpating fingers and returning to normal position, tossed up by ballottement and giving tympany during percussion over the kidney (by overlying intestinal loops). Protein and erythrocytes sometimes appear in the urine after palpation of the kidney. But all these signs are of only relative importance. For example, if a malignant tumour develops, the kidney may lose its mobility due to proliferation of the surrounding tissues; its surface becomes irregular and the consistency more firm; if the tumour is large, the kidney moves apart the intestinal loops and percussion gives dullness. But the kidney can nevertheless be identified by the mentioned signs by differentiating it from the neighbouring organs and other formations.

Palpation of the kidneys in the standing patient was proposed by S.P. Botkin. During palpation the patient stands facing the physician who sits on a chair. The prelum muscles should be relaxed and the trunk slightly inclined forward.

Palpation can be used to diagnose ptosis of the kidneys. Three *degrees of nephroptosis* can be distinguished (A.A. Shelagurov, 1964):

I degree – a palpated kidney (*ren palpabilis*), its inferior pole can be palpated only. Mobility of kidneys is small;

II degree – a movable kidney (*ren mobilis*), the entire kidney can be palpated in the second degree; it is easily displaced, not translocated for a white line of an abdomen.

III degree - a wandering (vage) kidney (*ren migrans*), the kidney freely moves about in all directions to pass beyond the vertebral column in the side of the other kidney, and to sink downwards at a considerable distance.

Palpation of urinary bladder

Palpation of the urinary bladder in absence of its pathology and its overflow yields a negative result. Palpation of urinary bladder is performed from top to bottom on midline under all rules of deep sliding methodical palpation according to V.P. Obraztsov. If it contains much urine, especially

in persons with thin abdominal wall, the urinary bladder can be palpated over the pubic bone as an elastic fluctuating formation. If the bladder is markedly distended, its superior border reaches the umbilicus. In the long-term ischuria the urinary bladder is determined at a palpation as a rounded elastic body in suprapubic range of abdomen.

Palpation of ureteric points

Tenderness at palpation along the course of ureter and sensitive loin (sensitive to pressure exerted in the angle between the 12-th rib and the longissimus thoracic muscles) is of certain diagnostic importance. Palpation of the anterior surface of abdomen and lumbar range in some cases enables to determine presence of the pain points connected to an affection of kidneys and urinary tract.

Three pairs of *anterior ureteric points*: (1) s*ubcostal point* - at the anterior end of 10-th rib; it corresponds to renal pelvis; (2) s*uperior ureteric point* - at the edge of the rectus abdominis muscle at the level of the umbilicus; it corresponds to superior third of ureter; (3) *medium ureteric point* - at the intersection of the biiliac line and the vertical line passing the pubic tubercle; it corresponds to medium third of ureter.

Two pairs of *posterior ureteric points*: (1) c*ostovertebral point* - in the angle formed with the inferior edge of 12-th rib and a columna vertebralis; (2) *costolumbar point* – at the intersection of lumbar muscle and 12-th rib.

Pressure in these points in norm routinely painless becomes sharply responsive at a pyelonephritis, a paranephritis, a nephrolithiasis, a tumor and tuberculosis of kidneys.

Auscultation of renal arterias

Auscultation in diagnostics of kidneys disease is used for recognition of pathology of renal arterias. Systolic murmurs of renal artery stenosis can be auscultated in lumbar range in a costovertebral angle and on anterior abdominal wall in points placed 5 sm above umbilicus and near 4-5 sm aside.

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Electrocardiography examination

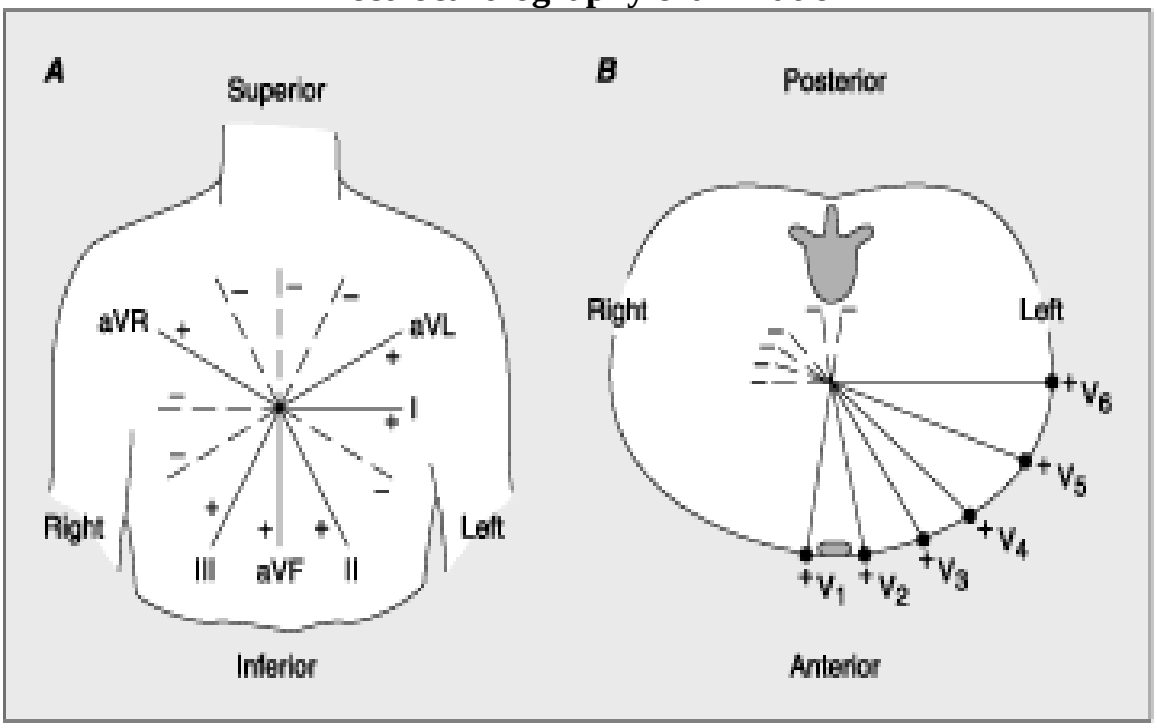


Fig. Suppl. 1.The six frontal plane and six horizontal plane leads provide a three-dimensional representation of cardiac electrical activity. The frontal plane leads – standard (I, II, III) and augmented leads (aVR, aVF, aVL)**.** The horizontal plane leads – chest leads (V_{1-6}).

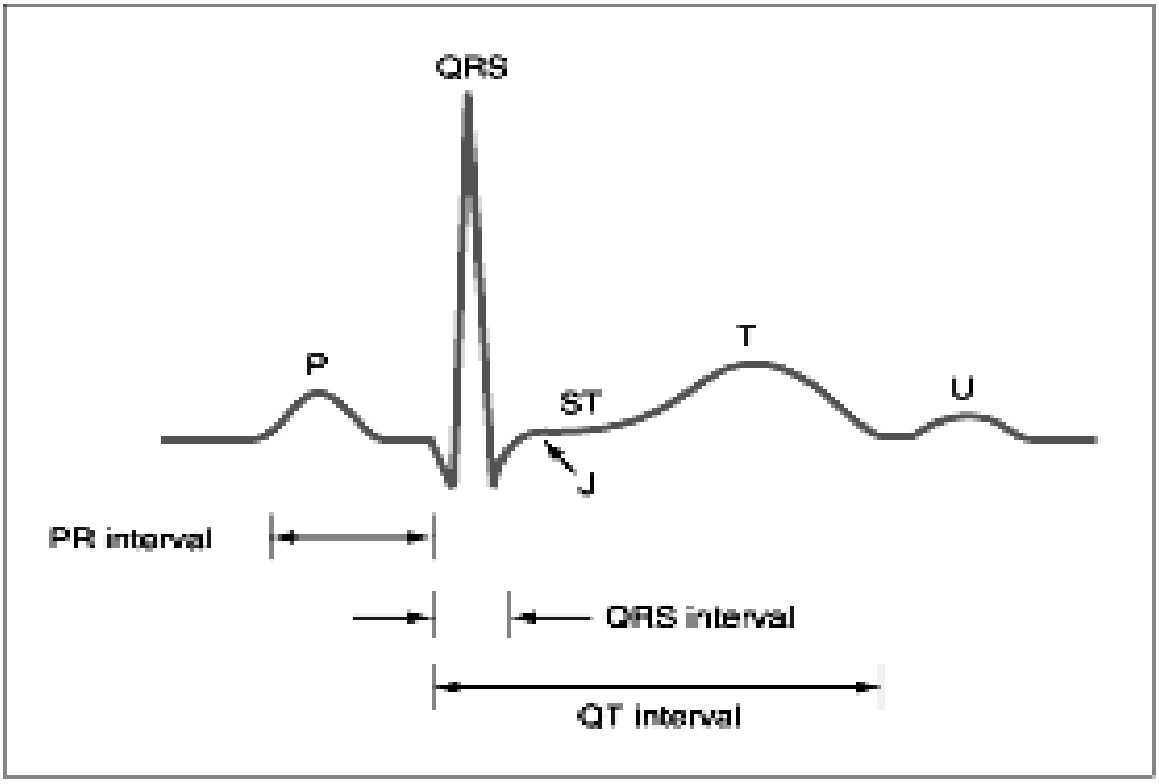


Fig. Suppl. 2. Basic waves and intervals of normal ECG.

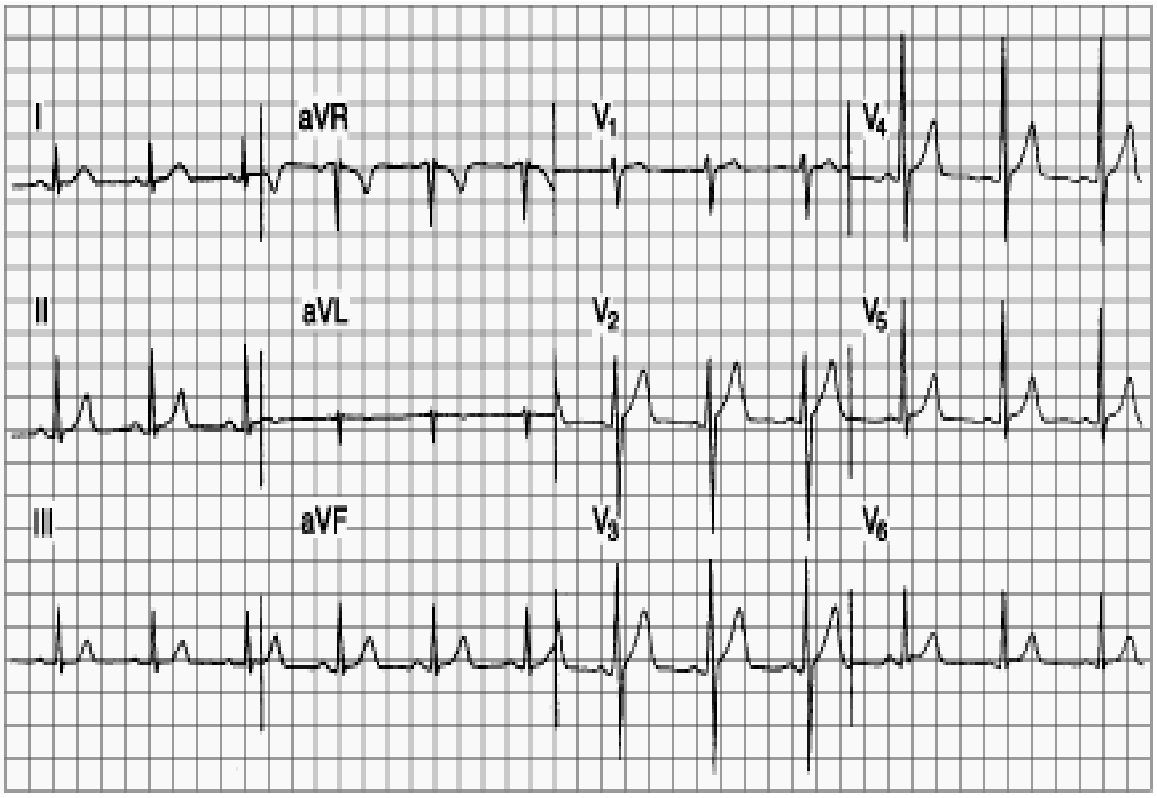


Fig. Suppl. 3. Normal ECG of the healthy subject. Sinus rhythm is present with a heart rate of 75 per minute. PR interval is 0.16 s; QRS interval (duration) is 0.08 s; QT interval is 0.36 s; the mean QRS axis is about +70°, transition zone V3.

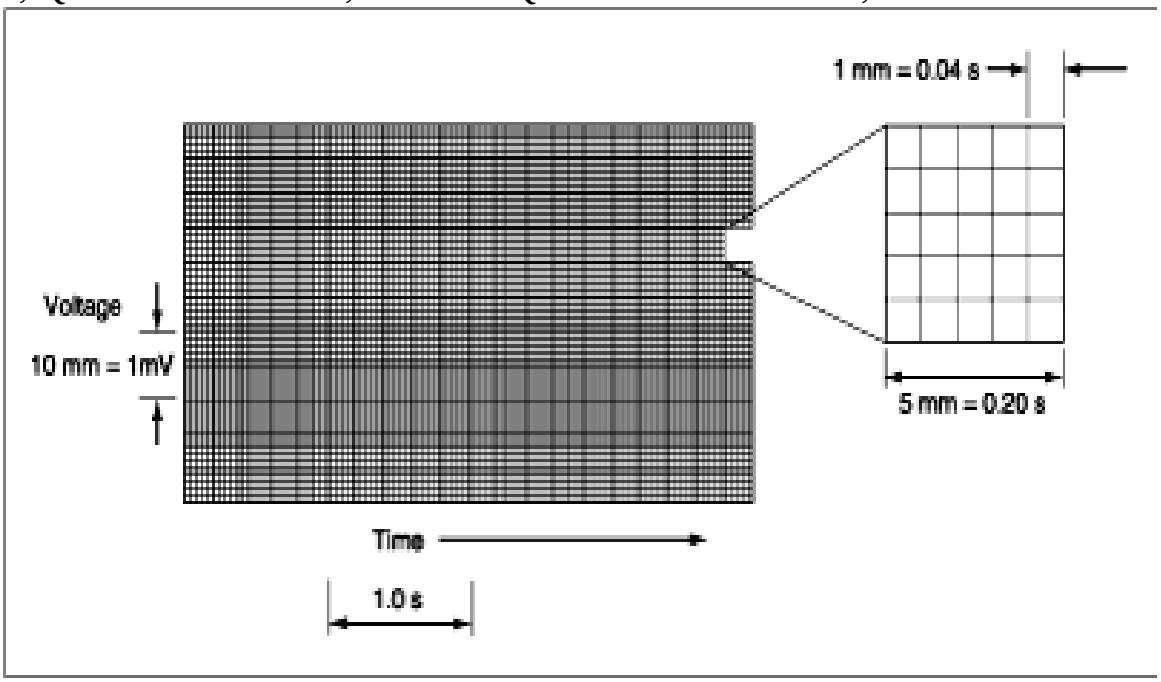


Fig. Suppl. 4. The ECG graph paper records the time (interval) between cardiac electrical events along the horizontal axis and their amplitude (voltage) along the vertical axis.

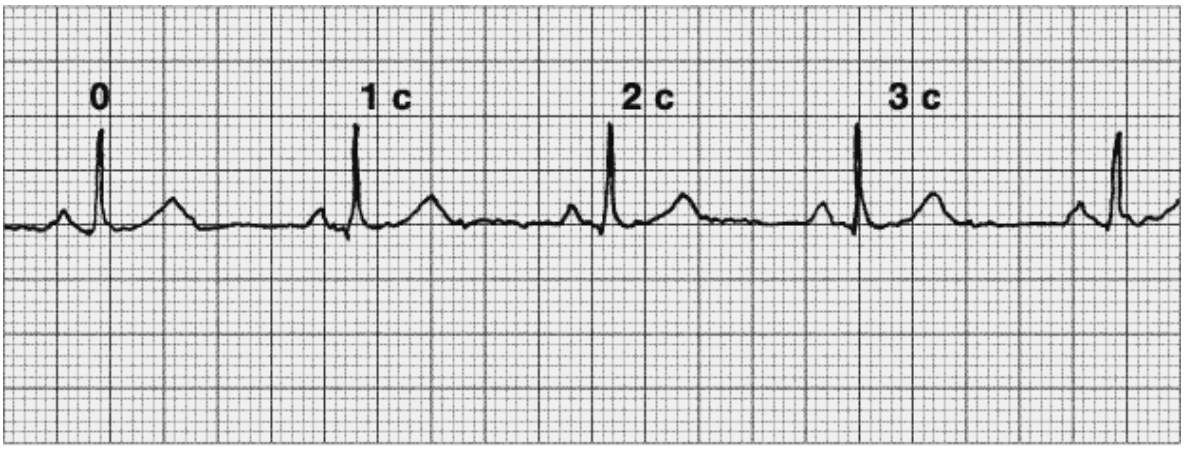


Fig. Suppl. 5. HR determination - multiply in 20 times (×20) the number of R-R intervals during 3 seconds.

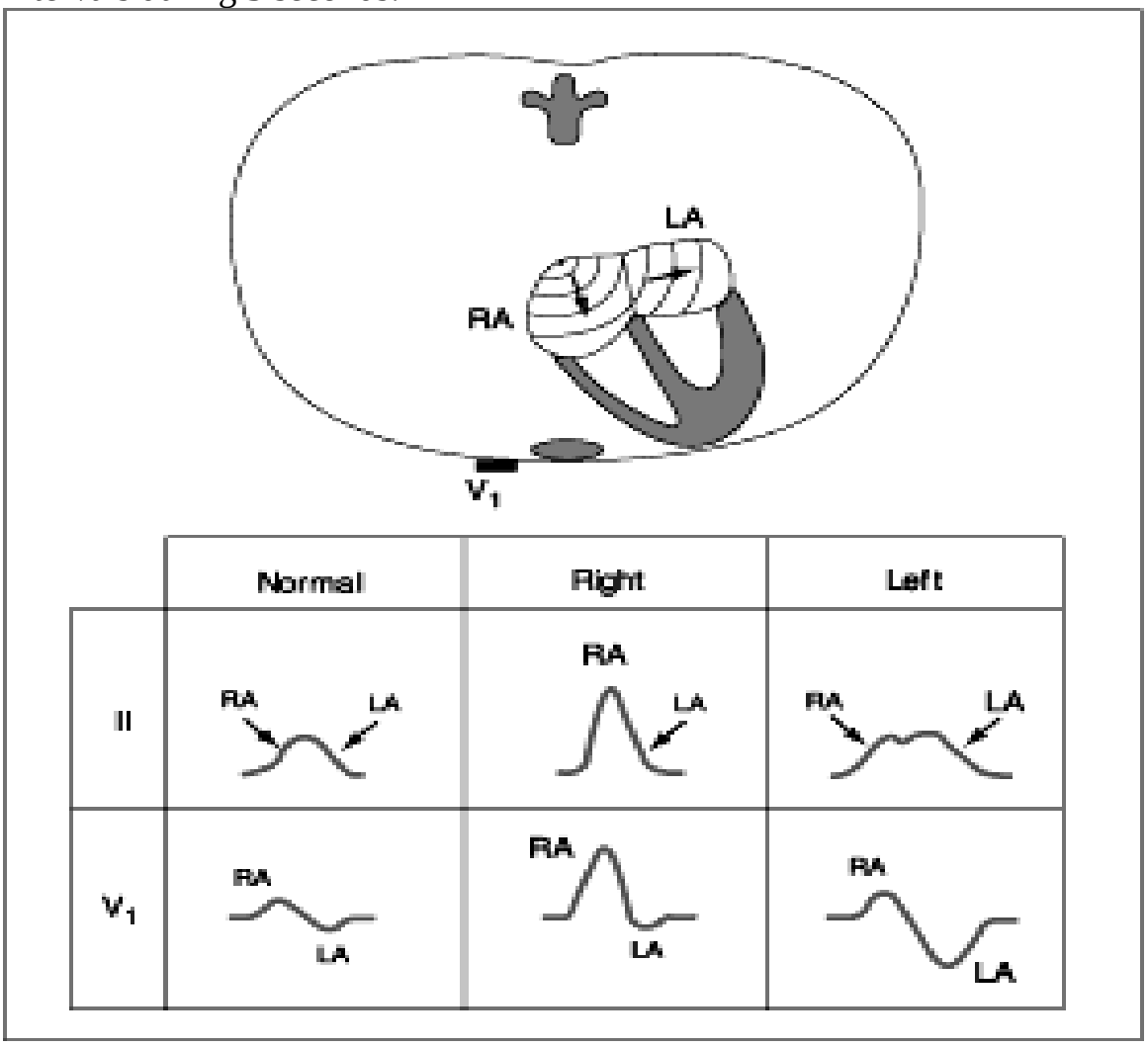


Fig. Suppl. 6. ECG in hypertrophy of atriums.
Right atrium hypertrophy is detected by “P pulmonale”: high (>2.5 mm) acute P in II, III, AVF and right chest leads (V_{1-2}).
Left atrium hypertrophy is detected by “P mitrale”: wide (>0.1 s), splitted P-wave in I, II, AVL, V_{5-6}; flat or negative P in III, biphasic or negative P (>1 mm) V_1.

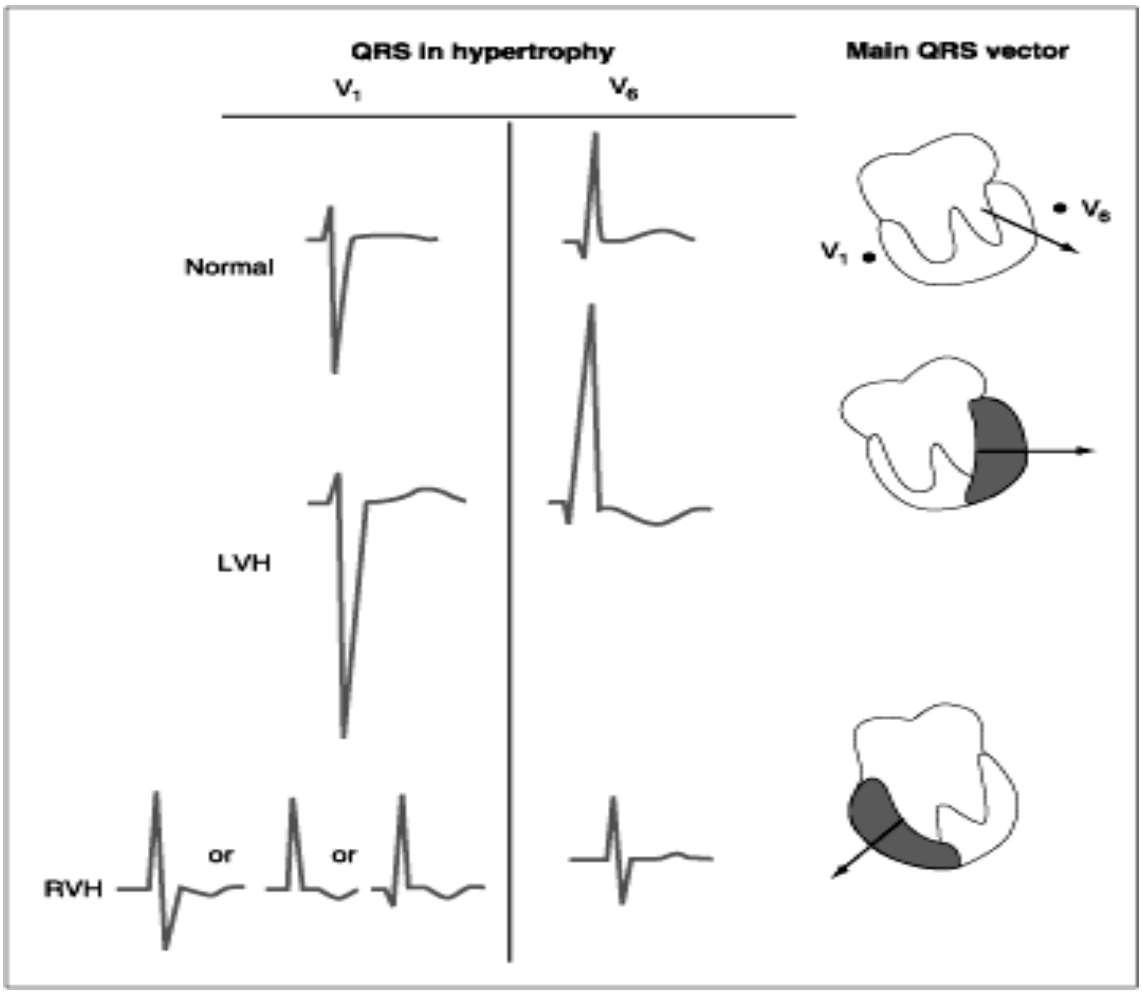


Fig. Suppl. 7. ECG in hypertrophy of ventricles.

Left-ventricular hypertrophy (LVH) - amplitude of the S increases in V_{1-2}; amplitude of the *R* increases >20-25 mm in V_{4-6}.

Right-ventricular hypertrophy (RVH) the changes in the S and *R* waves are the reverse - high R wave ≥ 7 mm in V_{1-2}, deep S wave in V_{4-6} .

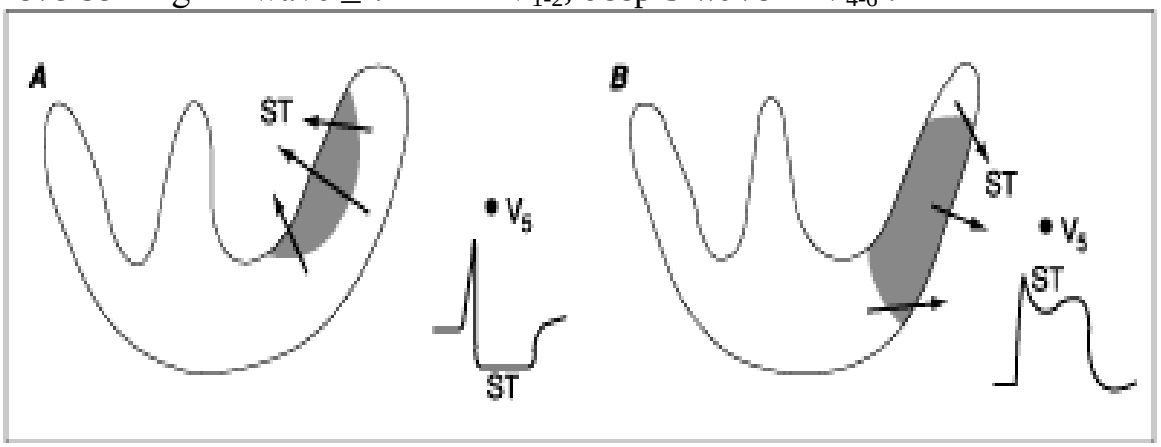


Fig. Suppl. 8. Acute ischemia causes a current of injury of myocardium. Subendocardial injury (*A*) - the resultant ST vector directs toward the inner layer of the affected ventricle, overlying leads therefore will record ST depression. Transmural or epicardial injury *(B)* - ST elevations and sometimes tall, positive, hyperacute T waves over the ischemic zone.

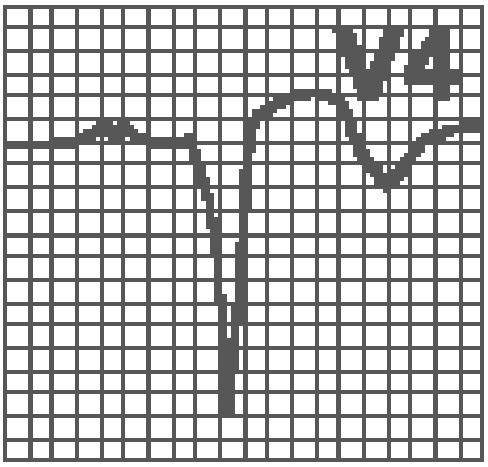


Fig. Suppl. 8. Myocardial necrosis is detected by the pathological Q-wave - - width≥0.04 s (in V4-6 >0.025 s), depth>2 mm or>1/4 R-wave (in V_{4-6} >15%R).

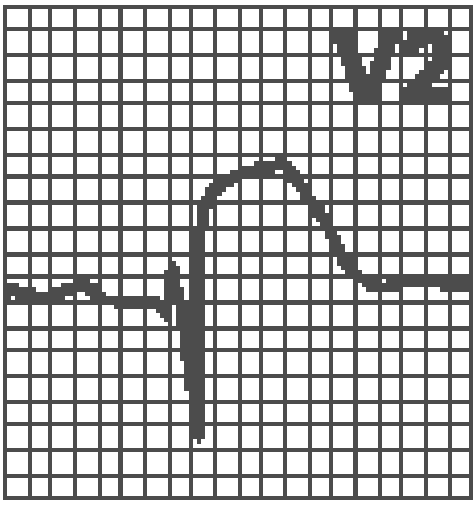
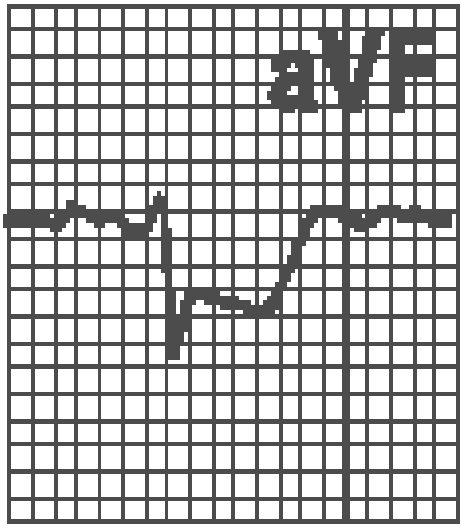


Fig. Suppl. 9a. Ischemic myocardium injury is detected by ST-interval. Transmural or epicardial injury - convexing elevation ST with transmission in T-wave.



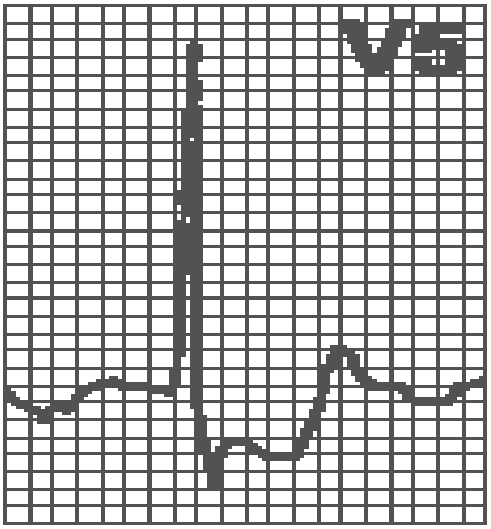


Fig. Suppl. 9b. Subendocardial injury – horisontal or concaving depression ST.

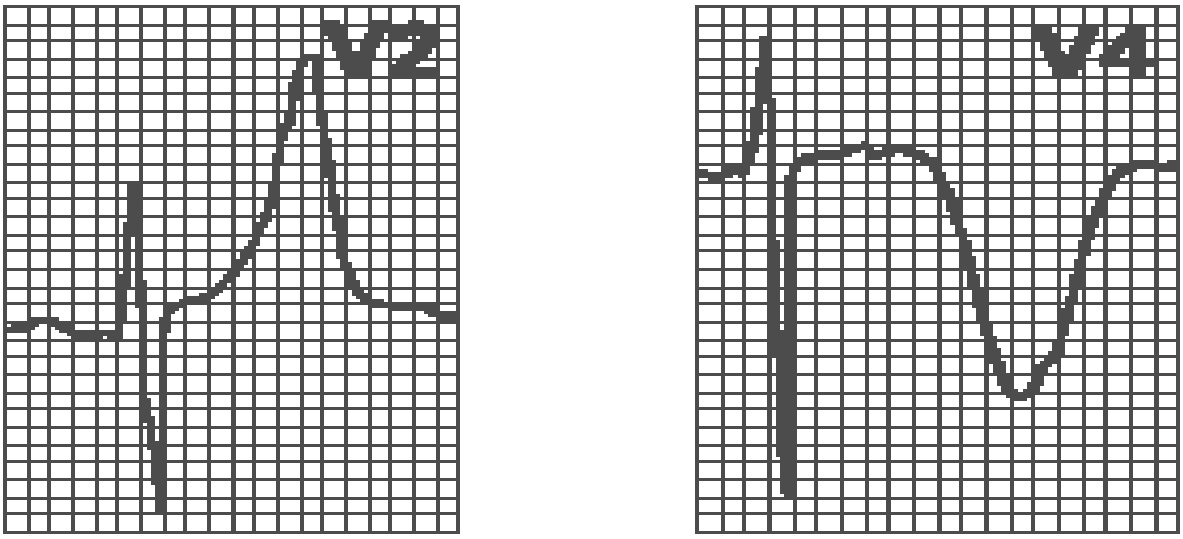


Fig. Suppl. 10. Ischemia of myocardium is detected by T-wave.
Subendocardial ischemia - symmetrical acute high T-wave in overlying leads (>6 mm in standard and augmented leads , >8-10 mm in chest leads)
Transmural or epicardial ischemia symmetrical acute deep T-wave.

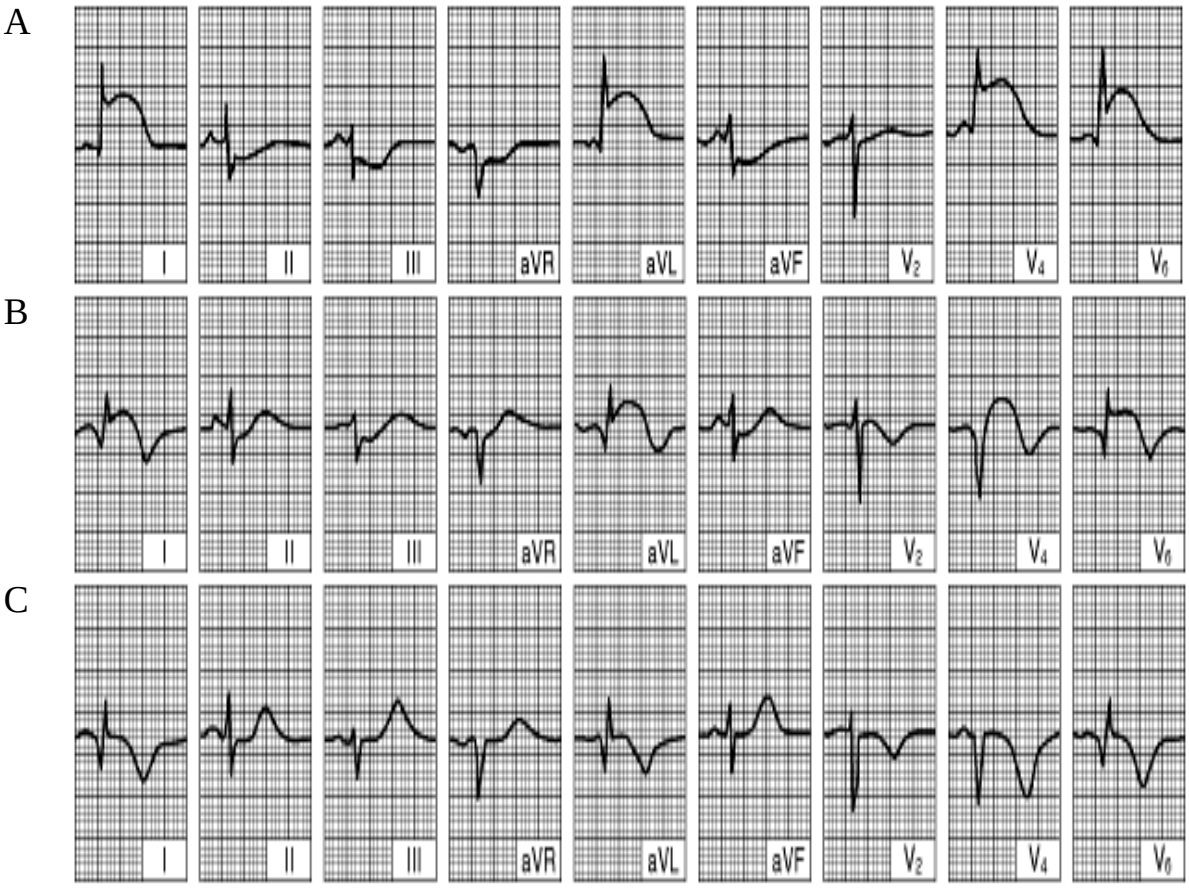


Fig. Suppl. 11. Acute anterior left ventricular myocardial infarction
(A) Superacute period (1-2 hours), (B) acute period (24 hours), (C) subacute period (>1 week).

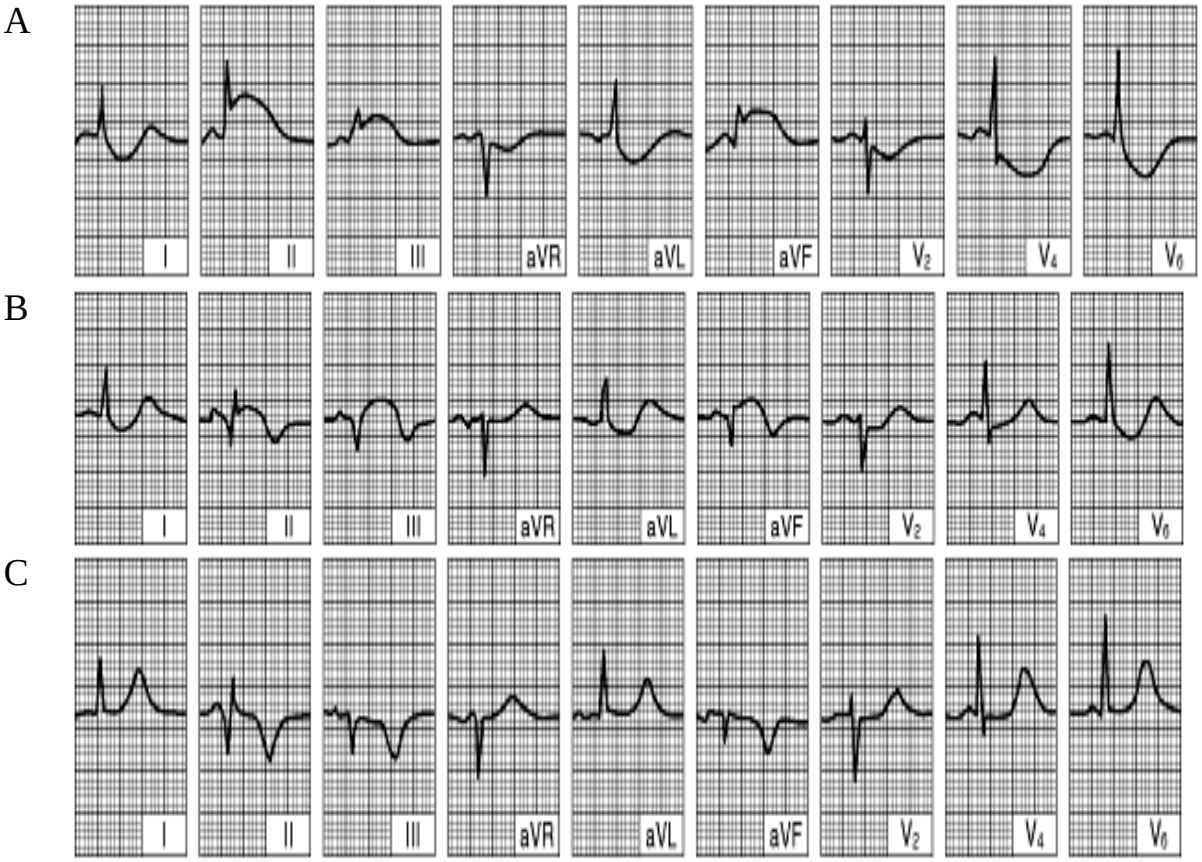


Fig. Suppl. 12. (A) Superacute period (1-2 hours), (B) acute period (24 hours), (C) subacute period (>1 week).

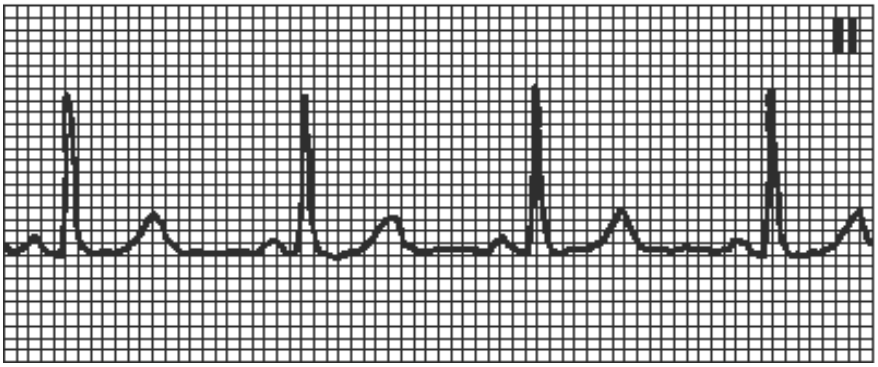


Fig. Suppl. 13. Normal sinus rhythm.
HR=60-80 in min, P(+) I, II, aVF, (-) aVR, PQ ≥0,12s

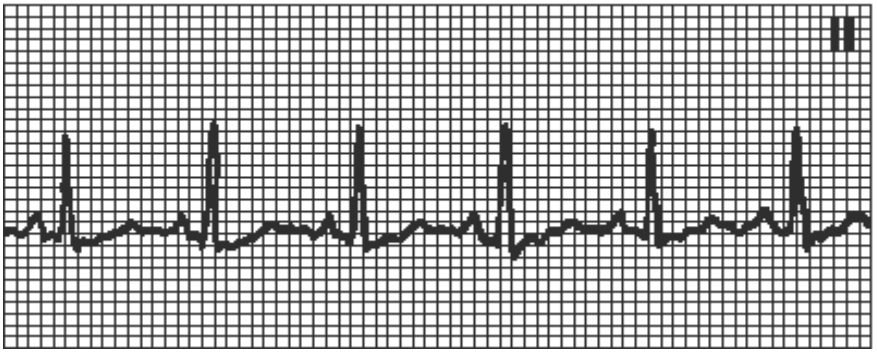


Fig. Suppl. 14. Sinus tachycardia.
HR>90 in min, regular rhythm.

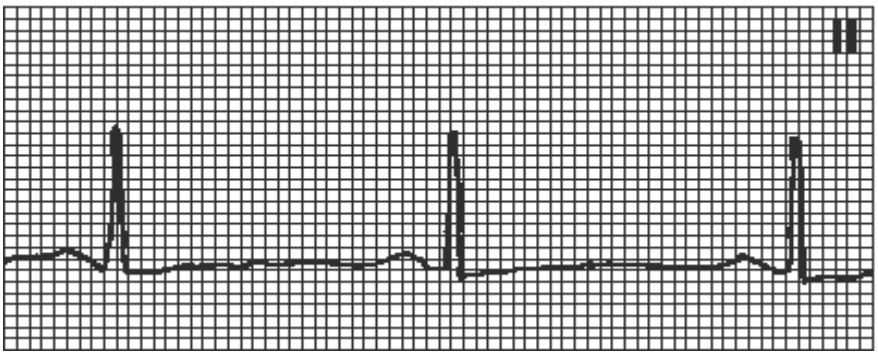


Fig. Suppl. 15. Sinus bradycardia.
HR<60 in min, regular rhythm.

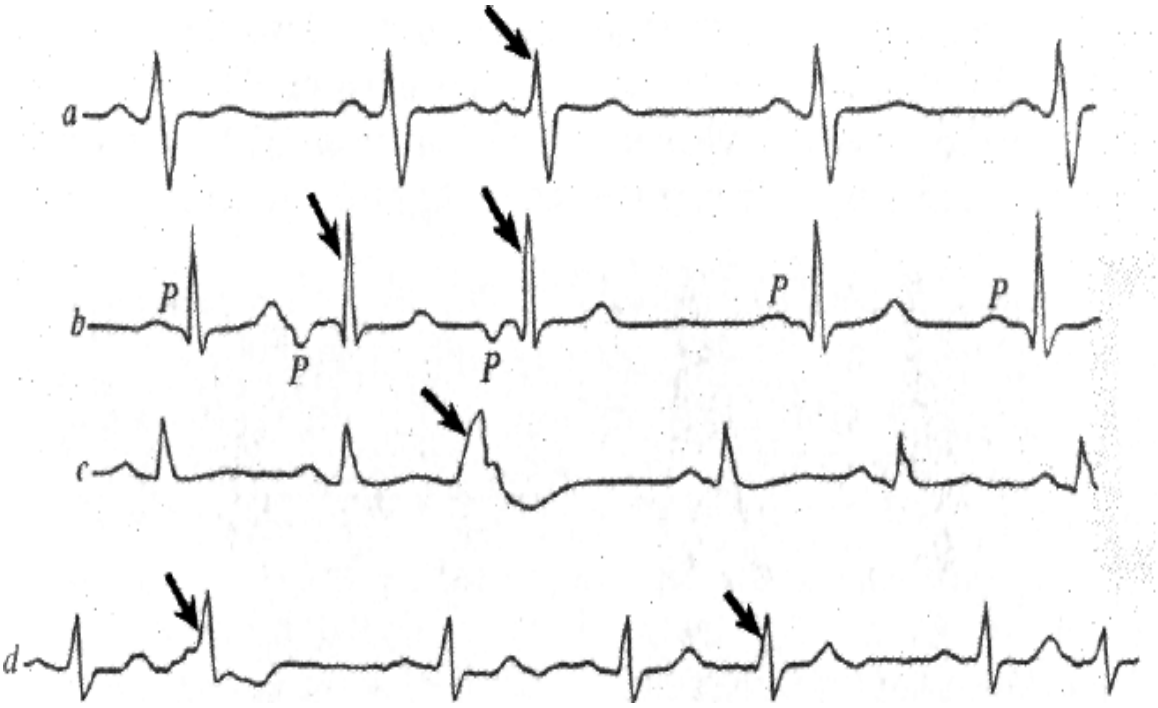


Fig. Suppl. 16. Extrasystolic arrhythmia:
a – atrial extrasystole, b - AV-junction extrasystole, c – ventricular extrasystole ,d - polytopic extrasystole.

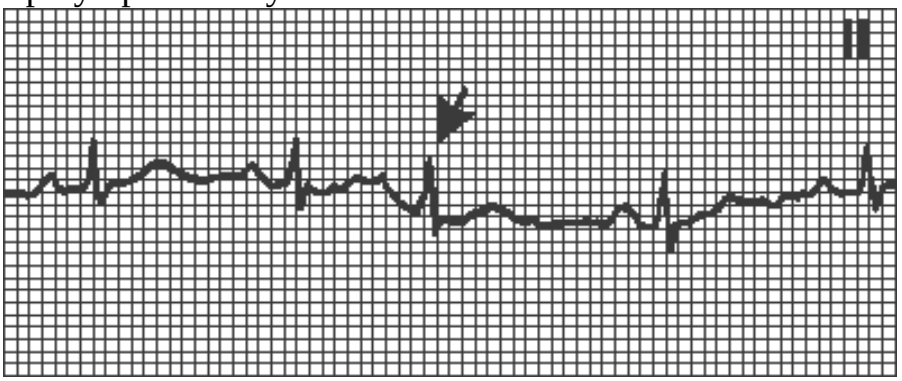


Fig. Suppl. 17. Atrial extrasystole: (1) premature appearance of the cardiac complex, (2) *P* wave - normal shape or slightly disfigured and superimposed on the preceding *T* wave; (3) normal shape of QRS (4) incomplete compensatory pause.

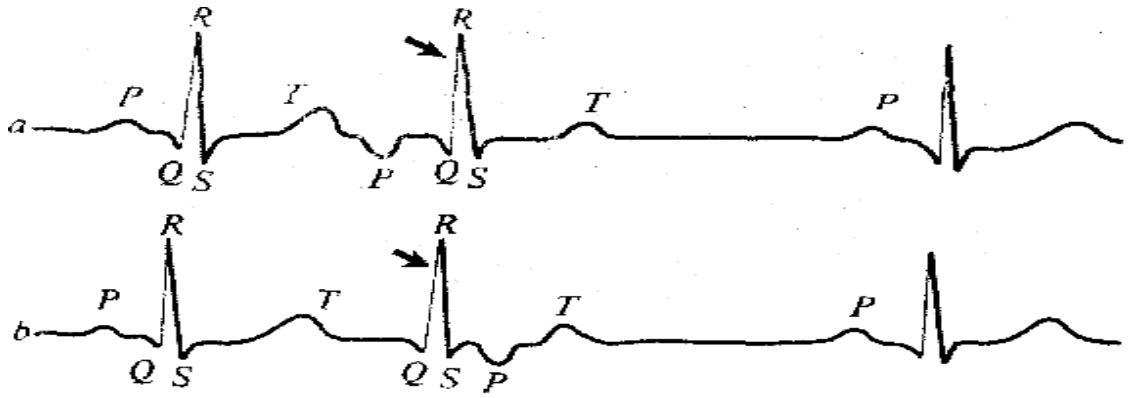


Fig. Suppl. 18. Atrioventricular (nodal) extrasystole:
(1) premature appearance of the cardiac complex; (2) negative *P* wave because of the retrograde atrial excitation; (3) the position of the *P* wave with respect to the ventricular complex depends on the rate of propagation of the excitation wave onto the atria and the ventricles;(4) shape of QRS is normal or slightly deformed; (5) incomplete compensatory pause

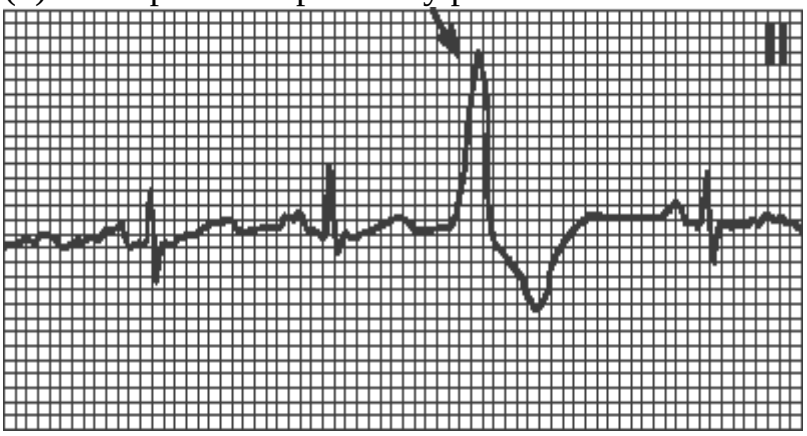


Fig. Suppl. 19. Ventricular extrasystole:
(1) premature appearance of the ventricular complex; (2) absence of the *P wave*; (3) deformation of the *QRS* complex due to its increased voltage and length; (4) the shape and the height of the *T* wave changes, its direction is opposite to the maximum wave of the *QRS* complex; (5) complete compensatory pause

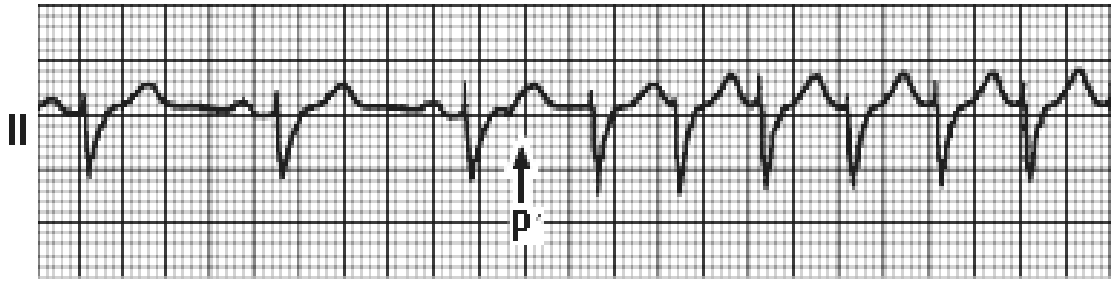


Fig. Suppl. 20. Initiation of the atrioventricular nodal paroxysmal tachycardia (reentry tachycardia - abnormal P wave and the atrioventricular nodal delay (long PR)

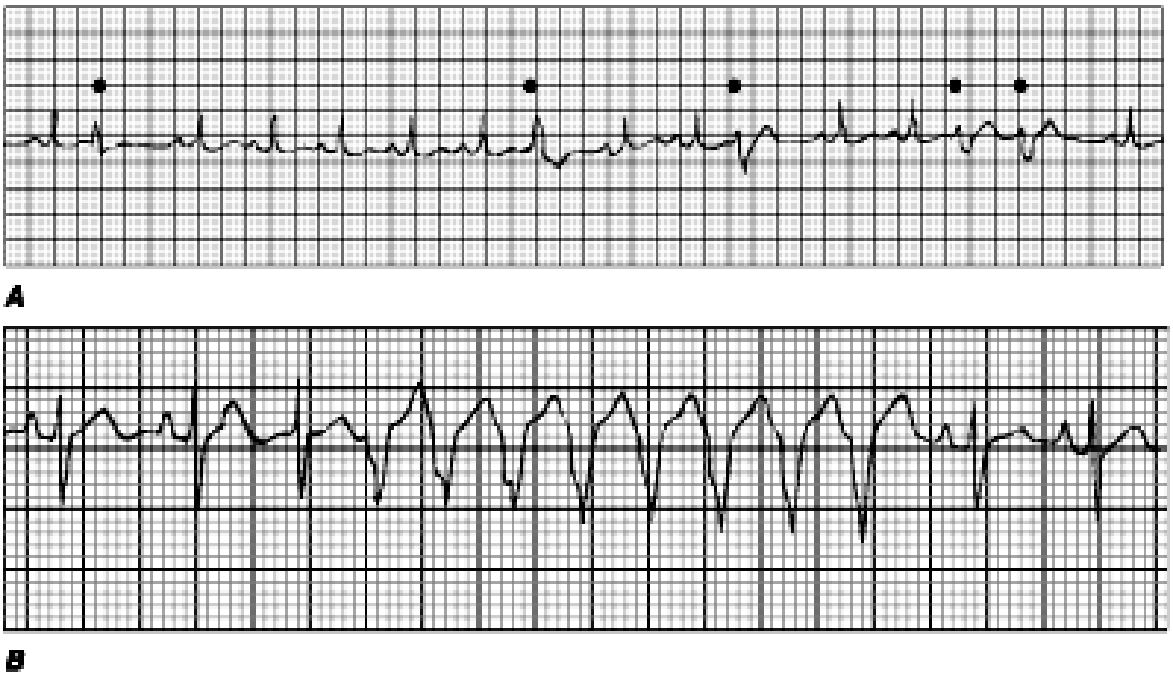


Fig. Suppl. 21. Ventricular paroxysmal tachycardia: A- Frequent ventricular extrasystoles precede PT; B- Episode of ventricular PT (at the same patient).

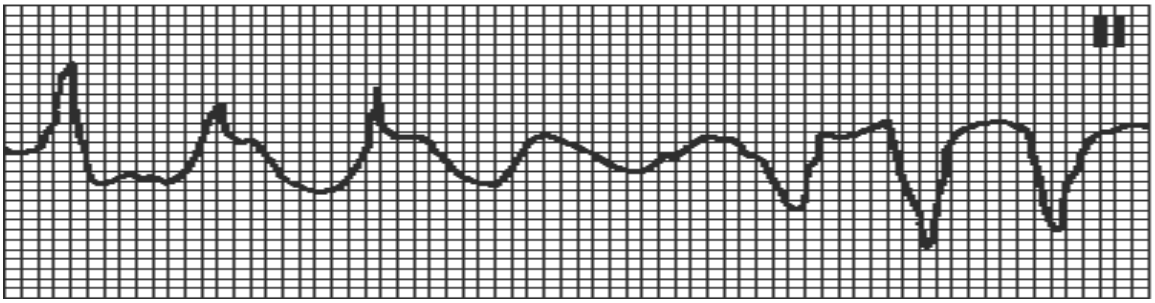


Fig. Suppl. 22. Torsade de pointes:
Ventricular PT characterized by the continuously changing QRS, may be as predictor of ventricular fibrillation and cardiac arrest.

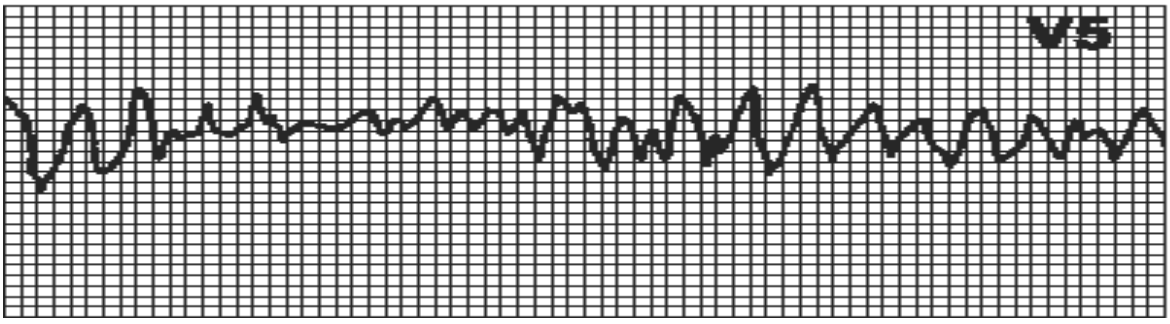


Fig. Suppl. 23. Ventricular fibrillation:
A rapid irregular ventricular rhythm due to multiple reentrant activity associated with essentially zero cardiac output.

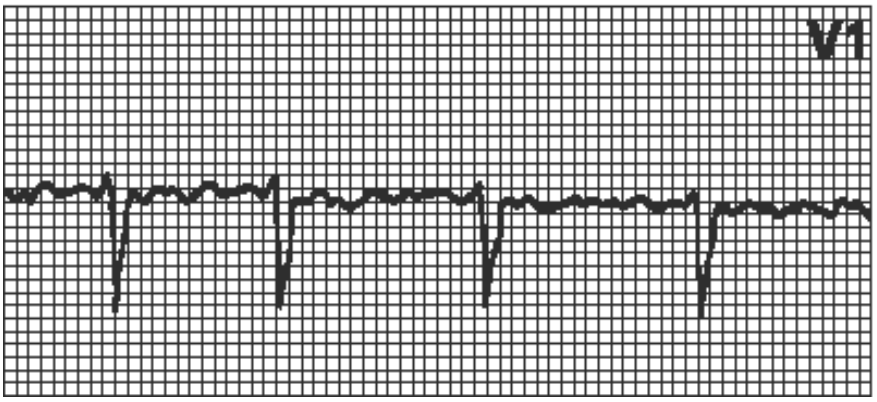


Fig. Suppl. 24. Atrial fibrillation:
(1) *P* wave disappears; (2) multiple small irregular f waves; (3) QRS ventricular complexes follow are irregular, their shape does not change.

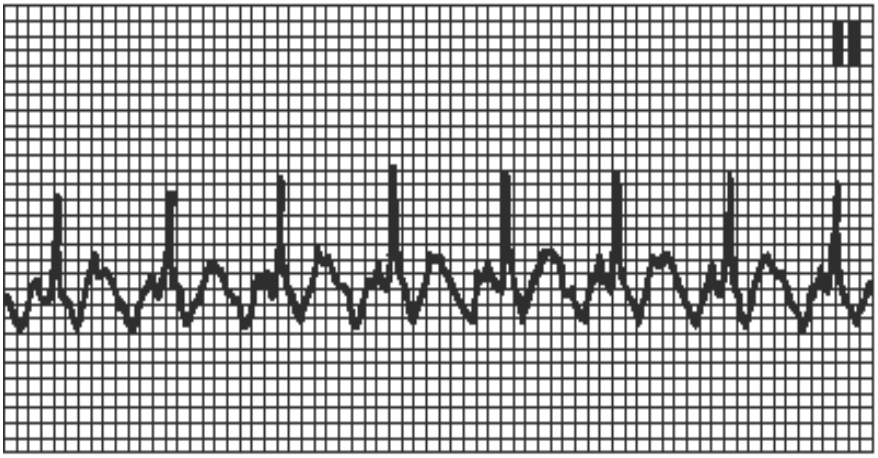


Fig. Suppl. 25. Atrial flutter:
(1) high F waves instead of the normal atrial *P* waves; (2) the number of F waves preceding each ventricular complex depends on the AV conduction; (3) QRS complexes follow at regular intervals.

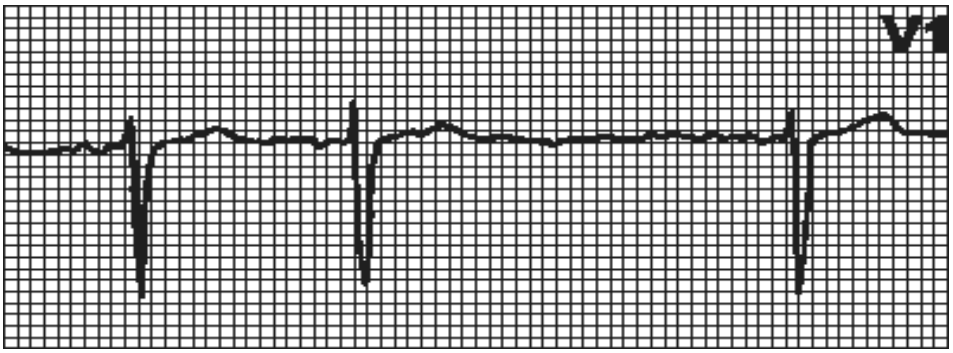


Fig. Suppl. 26. Sino-atrial block:
(1) periodic missing of the heart complex (PQRST) in the presence of a regular sinus rhythm; (2) the length of diastole doubles.

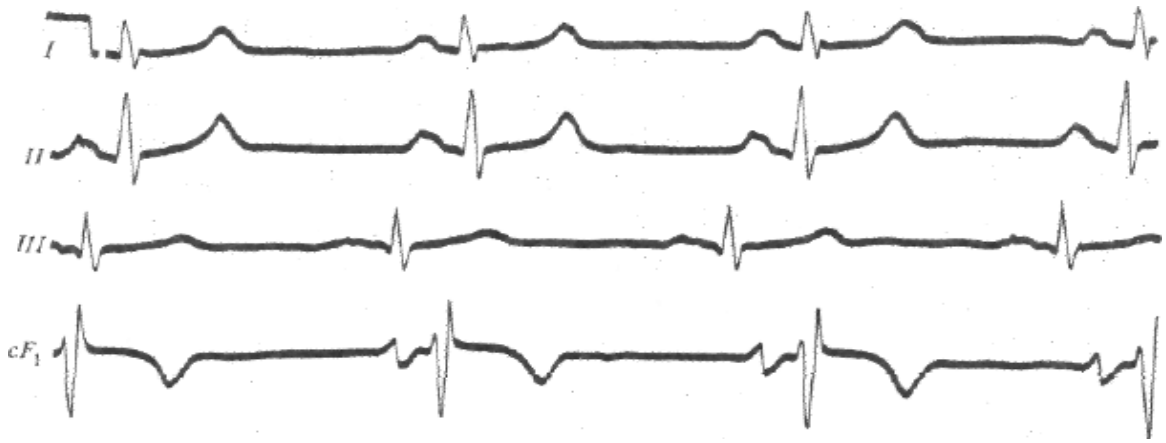


Fig. Suppl. 27. Intra-atrial block:
(1) P waves are broadened ≥0.11 s and splitted;
(2) P wave in the V_1 lead has two phases.

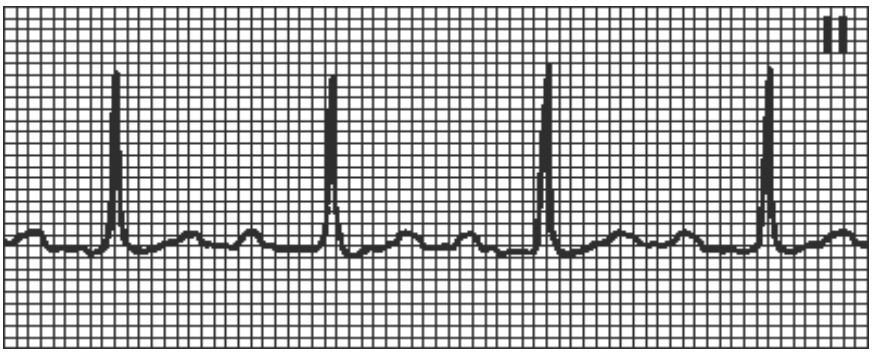


Fig. Suppl. 28. I degree of atrioventricular block:
(1) increased *P-Q* interval (> 0.21 s to 0.3-0.4 s and more) without missing QRS;
(2) regular heart rhythm.

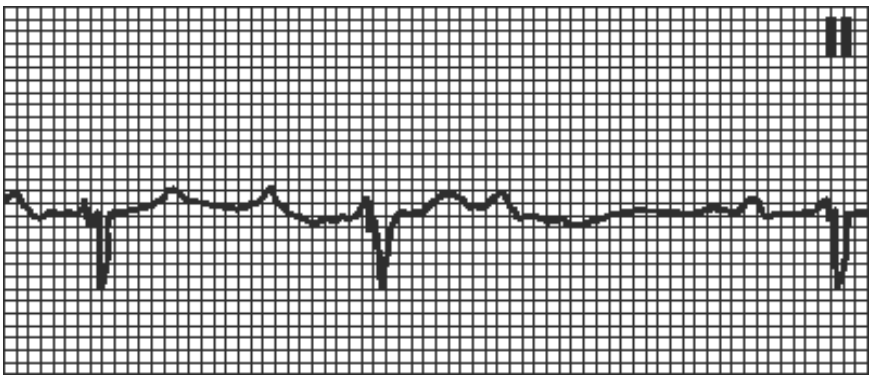


Fig. Suppl. 29. II degree of atrioventricular block with Samoilov-Wenckebach periods (Mobitz-1 type):
(2) gradual elongation PQ (Samoilov-Wenckebach periods);
(3) periodically missing ventricular contractions

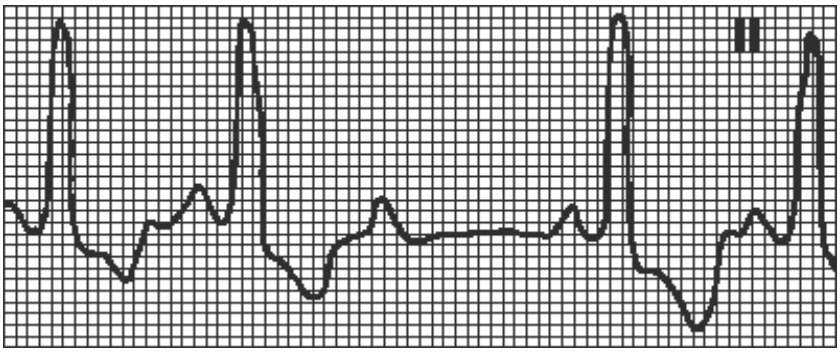


Fig. Suppl. 30. II degree of atrioventricular block (Mobitz-2 type):
(1) periodic missing QRS without gradual elongation PQ (2:1);
(2) PQ can be normal or a little bit prolonged

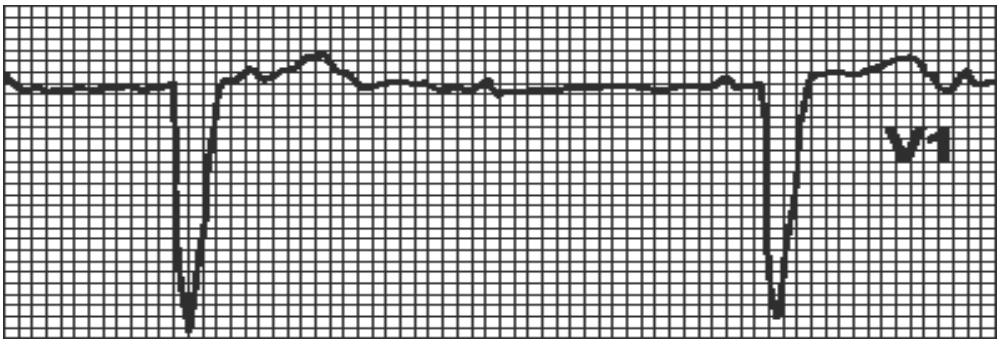


Fig. Suppl. 31. III degree of atrioventricular block (complete heart block):
(1) atrial *P* waves and ventricular complexes QRS are recorded independently of each other;
(2) the number of QRS is usually much smaller than the number of atrial P waves;
(3) the shape of the ventricular complex does not change if the pacemaker arises from the AV node or His bundle;
(4) with lower location of the pacemaker in the conduction system, the *QRST* complexes are altered.

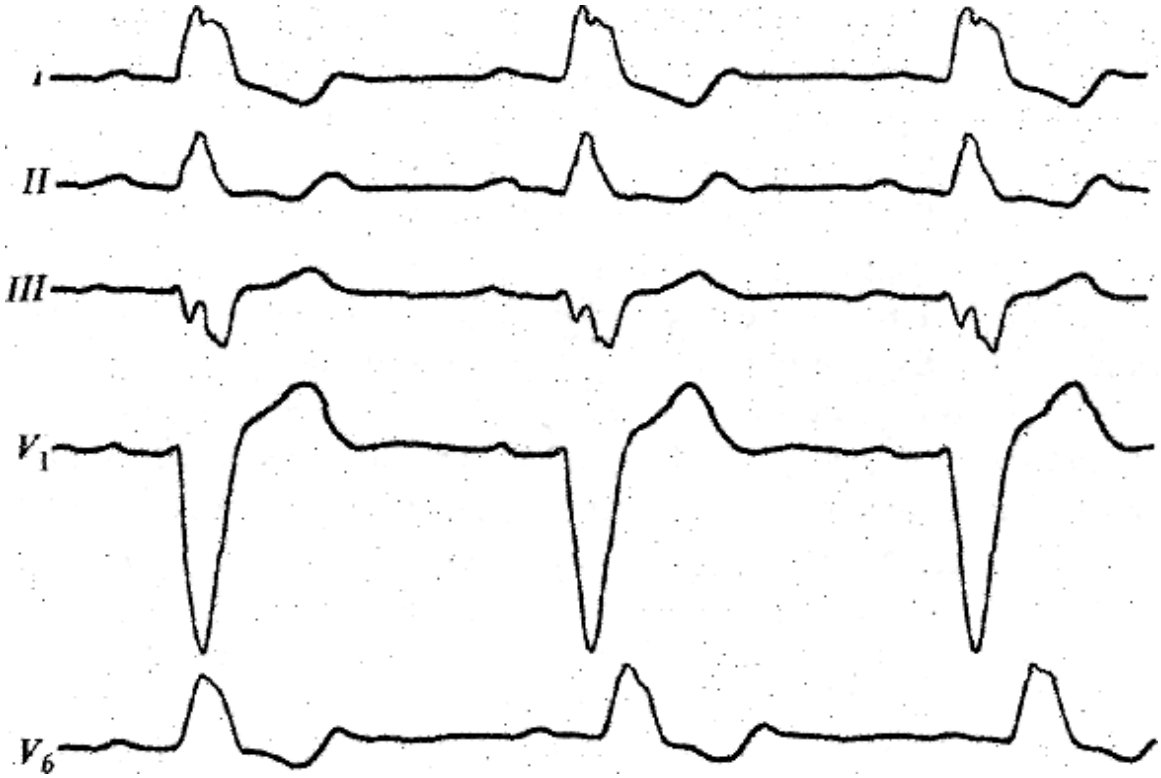


Fig. Suppl. 32. Left bundle branch block:
(1) wide and deformed QRS has the form of qR in I,II, V_{5-6}; rS in III, aVF, V_{1-2} ;
(2) disconcordance of ST,T and the main wave of QRS; (3) levogram

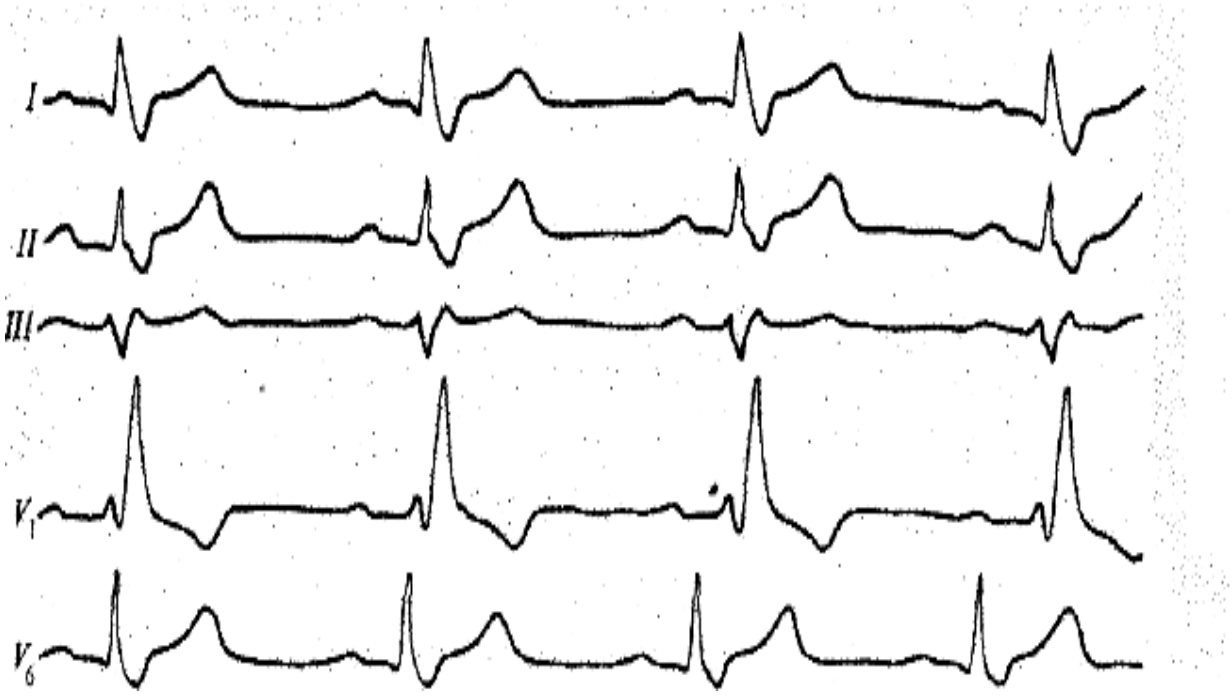


Fig. Suppl. 33. Right bundle branch block:
(1) wide QRS III, V_{1-2} has the form of rsR, rSR, RsR´(similar to “M”);
(2) wide S I, aVL,V_{5-6}; (3) negative ST and T in V_{1-2}; (4) dextrogram

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