МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

ОБСЛЕДОВАНИЕ ПАЦИЕНТОВ ПРИ ЗАБОЛЕВАНИЯХ СИСТЕМЫ КРОВООБРАЩЕНИЯ

CARDIOVASCULAR EXAMINATION

Учебно-методическое пособие

Минск БГМУ 2025

УДК 616-005-07(075.8)
ББК 54.10я73
О-25

Рекомендовано Научно-методическим советом университета в качестве учебно-методического пособия 26.06.2024 г., протокол № 18

Авторы: канд. мед. наук, доц. Г. М. Хващевская; канд. мед. наук, доц. М. В. Шолкова; ассист. Т. П. Новикова; ассист. Е. И. Сасинович; ассист. Ю. В. Репина

Рецензенты: канд. мед. наук, зам. гл. врача 11-й городской клинической больницы г. Минска В. В. Груша; 1-я каф. внутренних болезней Белорусского государственного медицинского университета

Обследование пациентов при заболеваниях системы кровообра-
О-25 щения = Cardiovascular examination : учебно-методическое пособие / Г. М. Хващевская, М. В. Шолкова, Т. П. Новикова [и др.]. – Минск : БГМУ, 2025. – 47 с.

ISBN 978-985-21-1813-2.

Подробно изложены основные методы обследования пациентов при заболеваниях системы кровообращения, представлены основные жалобы, осмотр, пальпация, перкуссия и аускультация.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке по дисциплине «Пропедевтика внутренних болезней».

УДК 616-005-07(075.8)
ББК 54.10я73

ISBN 978-985-21-1813-2

EXPLANATORY NOTE

Total duration of classes is 6 hours. Diseases of the cardiovascular system have a leading place in the mortality structure worldwide. The clinical diagnosis in case of cardiovascular pathology is based on a study of: the patient's complaints; present and past medical history (anamnesis morbi and vitae); objective examination data (inspection, palpation, percussion, auscultation); the results of laboratory and instrumental methods. The individual manifestations of the disease and the mechanisms of symptom occurrence also have great value.

The goal of the class is:

1. To master the methodology of interviewing patients with acquired heart defects: basic and additional complaints, collecting anamnesis of the disease and anamnesis of life.
2. To master the technique of general examination and examination of the heart area.
3. To master the technique of palpation of the cardiac and apical impulse, arterial pulse.
4. To get acquainted with percussion as a method of heart examination. To learn the importance of determining relative and absolute cardiac dullness
5. To learn techniques for determining relative and absolute cardiac dullness. Learn how to use the percussion method to determine the configuration of the heart, the diameter of the relative cardiac dullness
6. To get acquainted with auscultation as a method of heart examination.
7. To master the technique of auscultation of the heart. Learn to distinguish between 1 and 2 heart sounds, systolic and diastolic murmurs.

Requirements for the initial level of knowledge. In order fully master the topic, you should repeat from:

– human anatomy: anatomy of the heart and blood vessels;
– normal physiology: physiology of the cardiovascular system;
– general patient care: features of general care for patients with a cardiological profile.

Control questions from related disciplines:

1. The structure of the cardiovascular system.
2. The main functions of the cardiovascular system.
3. Supervision and hygienic care of patients with diseases of the cardiovascular system.

Control questions for the topic of the lesson:

1. List and characterize the main complaints of patients with diseases of the cardiovascular system, indicate their pathogenesis and diagnostic significance.
2. Describe the methods of collecting the anamnesis of the underlying disease and the anamnesis of the patient's life according to their sections.

3. Specify the features of collecting anamnesis of the disease and anamnesis of life in patients with diseases of the cardiovascular system.

4. Assess the general condition of a patient with cardiovascular diseases: position in bed, discoloration of the skin, changes in the color and shape of the nail phalanges, the presence of edema; identify the mechanism of development of the identified symptoms.

5. Describe the importance of examining the area of the heart, blood vessels, and epigastric region.

6. List the general rules and sequence for determining the cardiac and apical impulse.

7. Describe the technique of determining the arterial pulse.

8. List the main characteristics of the arterial pulse, the causes of pulse changes in the radial artery.

9. Name the purpose of palpation of the cardiac and apical impulse.

10. Describe percussion as a method of examining the heart.

11. List the rules for determining relative and absolute cardiac dullness.

12. Indicate what diagnostic capabilities methods for determining relative and absolute cardiac dullness have.

13. Specify the right, left and upper limits of relative cardiac dullness.

14. List the reasons for changing the boundaries of relative cardiac dullness.

15. Name the limits of absolute heart dullness.

16. Describe auscultation as a method of examining the heart and give it a physical justification.

17. Describe the projection of the valves on the surface of the chest.

18. Name the points of auscultation of the heart.

19. Explain the mechanism of occurrence of heart sounds.

20. Name the causes and explain the mechanism of physiological and pathological changes in sound 1 and 2.

21. Specify the causes and mechanism of physiological and pathological splitting and bifurcation of 1 and 2 sounds.

22. Explain the mechanism and name the reason for the appearance of the «quail» rhythm and the «gallop» rhythm.

23. Characterize the heart murmurs.

24. Explain the mechanism of the occurrence of functional murmurs, give them a characteristic and indicate the places of listening.

25. Specify the main organic heart murmurs, explain the mechanism of the occurrence of murmurs, give them a characteristic and indicate the listening places.

26. Specify the difference between functional and organic murmurs.

27. What are the causes of pericardial friction murmur and pleuropericardial murmur?

SUBJECTIVE EXAMINATION METHOD (INTERVIEW)

The main complaints are:

1. Shortness of breath (dyspnea). This concept consists of two Greek words: dys — frustrated, pnein — breathe, that is, painful violation of breathing. Shortness of breath is manifested by violation of the frequency, depth and breathing rhythm. On the features shortness of breath can be subjective and objective. The manifestation of subjective shortness of breath is the feeling of breathing difficulties without objective signs of changes in its frequency and depth. Objective shortness of breath is determined by reliable methods of research and is characterized by changes in the frequency, depth and rhythm of breathing, as well as changes in the duration of the inhalation phase and exhale. Isolated inspiratory (breathing difficulty) and expiratory (shortness of breath) dyspnea, physiological and pathological. Physiological shortness of breath occurs with significant physical activity, hard work or excessive mental excitement.

Pathological shortness of breath occurs in patients with signs of heart failure and can be of varying severity.

When questioning, pay attention to the time of occurrence of shortness of breath (constant or paroxysmal), the connection with physical activity and specify the appearance of shortness of breath at rest. In heart disease, shortness of breath appears first during exercise (walking), and at rest it passes. At the expressed phenomena of heart failure it appears at short walking, conversation, and then disturbs the patient constantly even at rest. Shortness of breath occurs most often in patients with heart defects, and most often — with mitral stenosis.

The cause of shortness of breath is the development of stagnation in the small circle of blood circulation, which leads to a deterioration of gas exchange and a decrease in blood oxygen saturation in the lungs, as well as a slowdown in blood flow in the large circle of blood circulation. These phenomena are developing as a result of reducing the contractile function of the myocardium of the left ventricle and lead to the accumulation of carbon dioxide in the blood and under-oxidized metabolic products, which result to increased excitability of the respiratory center.

Shortness of breath can occur suddenly in the form of acutely developed cardiac asthma. With heart defects during decompensation, especially with mitral stenosis, aortic defects, attacks of suffocation occur more often at night. Such asthma has been called cardiac asthma (asthma cardiale). They may occur at rest, after exercise or emotional stress. The patient feels acute lack of air, there is a bubbling breath and frothy sputum mixed with blood — attack of cardiac asthma.

This condition is extremely disturbing for the patient, he is not sleeping, is sitting in bed, by the morning the attack ends. Patients may experience breathing disorders such as Chain–Stokes, which occurs more often at night during sleep and during the day are not observed.

2. Cough (tussis) — an arbitrary or involuntary sudden sharp forced sonorous exhalation (initially with a closed voice slit, resulting in sharply increased air pressure in the trachea and bronchi). The mechanism of cough in heart disease is associated with stagnation in the small circle of blood circulation. Disorder of blood circulation in the walls of the bronchi leads to the violation of their food (cyanotica bronchitis), increased sensitivity of cough receptors and the emergence of cough. The bronchial lumen is free and the patient's heart coughing is not a reaction of adaptation or protection (there is no physiological expediency in it). Cough dry, attack-shaped, extremely painful for the patient.

3. Hemoptysis (haemarhtoe). Often hemoptysis is observed when coughing as a result of venous fullness in a small circle of blood circulation and is explained by the exit of red blood cells from the bloodstream per diapedesin or rupture of small vessels in the bronchi (scarlet blood in sputum). Microscopic examination of the sputum find cell ser-vices dechnik (histiocytes with trapped blood pigment).

4. Pain (dolor) in the heart. When questioning a patient with complaints of pain in the heart, it is necessary to find out:

1) localization (in the area of the apex of the heart or behind the sternum);
2) irradiation (where the pain spreads);
3) the character of the pain (squeezing, stabbing, pressing);
4) the duration of pain (constantly or occur paroxysmal-different);
5) under what conditions is there pain (physical tension, worry);
6) what are pain (than it is removed, docked).

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 releaved 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 occurs when narrowing of the left atrioventricular orifice associated with compression of the left coronary artery at the site of origin of the enhanced left atrium or rheumatic coronarita.

In the aortic heart defects pain are stenocarditiceski character. The occurrence of pain is associated with insufficient blood filling of coronary vessels as a result of a sharp decrease in diastolic pressure. In case of insufficiency of the aortic valves of syphilitic origin, pain is caused by narrowing of the mouth of the coronary arteries as a result of the phenomena of specific aortitis.

5. Heartbeat (palpitatio cordis). Patients feel strengthened and rapid contractions of the heart. The occurrence of palpitations is related with increased excitability of the nervous apparatus regulating the activity of the heart.

6. Heart failure. Due to a violation of the heart rhythm. Interruptions are accompanied by a sense of fading, cardiac arrest. In the presence of this complaint, the patient is clarified what causes the appearance of interruptions. They appear during exercise or at rest.

7. Edema (oedema). When heart diseases are swelling hydrostate-related as it is caused by obstruction of the venous outflow in a reduction-Institute of cardiac contractility.

The following factors contribute to the occurrence of edema:

– increased hydrostatic pressure in the capillaries and slowing blood flow leads to fluid transudation in the tissue;

– violation of the normal regulation of water-salt balance leads to sodium and water retention;

– with prolonged venous stagnation in a large circle of blood circulation, liver function decreases and albumin production is disrupted, which leads to a drop in the oncotic pressure of blood plasma.

Violation of liver function leads to a decrease in destruction in it antidiuretic hormone and aldosterone. Heart swelling may initially be hidden. Fluid retention in the body can be expressed as a rapid increase in body weight and a decrease in urine excretion. Visible swelling appears first on the lower extremities towards evening (if the patients walking or sitting) or on the sacrum in the supine position. Cardiac edema occurs symmetrically, first on the back surface of the feet, as the progression of heart failure, they extend to the tibia and above. Edema has a cyanotic color, the temperature of the skin over the edema is cold, they are dense to the touch, pass or decrease somewhat at rest (overnight or in a long horizontal position). In the future, the liquid can accumulate in the cavities: in the abdominal — ascites, in the pleural — hydrothorax, in the pericardial cavity — hydropericardium.

8. Disorders of the Central nervous system. In patients with heart defects in the development of decompensation, insomnia, headache, sometimes an excited state, delirium and even the development of psychosis are noted. The mechanism of development of these phenomena is associated with severe venous stagnation in the brain and edema of the meninges.

9. Dyspeptic phenomena. With the development of decompensation there is a pronounced venous stagnation in the vessels of the liver and gastrointestinal tract, which is manifested by a feeling of heaviness and pain in the right hypochondrium and epigastric region, as well as nausea, belching, sometimes vomiting and bloating.

INSPECTION

Examination of the patient should begin with the position that the patient occupies in bed, determining the color of the skin, mucous membranes, identifying edema, examining the heart area, peripheral vascular disease.

The patient's position in bed. Patients with defects in the period of compensation or in the initial stages of decompensation freely move and occupy an active position in bed. With the development of severe chronic heart failure, patients prefer to lie in bed, putting under the head a large number of pillows or a special headrest, which makes it possible to take a more elevated, semi-sitting position, easing the symptoms. When heart asthma attacks appear, patients take a typical forced position (orthopnea), they sit in a chair or in bed, lowering their legs and placing a pillow under their head. Trying to go to bed causes them to have a panic attack. Often they sit for weeks, spending sleepless nights, until they get relief from drug therapy. Forced sitting position the patient takes due to the fact that it disappears or decreases disturbing shortness of breath due to the outflow of blood to the lower extremities and reduce blood stagnation in the small circle of blood circulation, as well as some improvement of the diaphragm excursion.

Inspection of the skin and mucous membranes. Cyanosis may be detected when examined in patients with heart disease. Cyanosis is cyanotic staining of the skin and mucous membranes. The intensity of cyanosis can be different from mild cyanotic color to dramatically you through the world of black-and-blue. Cyanosis on the lips, on the tip of the nose, on the tips of the fingers is called **acrocyanosis**.

Cyanosis occurs in mitral heart disease, especially in mitral stenosis, in which a kind of cyanotic color of the skin of the cheeks is called mitral blush (facies mitralis).

Cyanosis is caused by an increase in the blood content of reduced hemoglobin, the color of which is darker than the color of oxyhemoglobin. In the development of cyanotic color is important venous dilation with venous stasis, which is observed with a decrease in the contractility of the right ventricle.

Increased blood levels of reduced hemoglobin depend on several reasons. There are so-called **central cyanosis**, in which due to lung disease, the process of blood arterialization is disturbed, as a result of which the blood flowing from the lungs is not completely saturated with oxygen. There is also peripheral cyanosis, which occurs as a result of slowing the blood flow and greater oxygen release to the surrounding tissues. The cause of peripheral cyanosis are heart defects in the stage of decompensation. There is also mixed cyanosis, when there is a combination of lung diseases and heart disease. In most heart diseases, cyanosis is mixed.

Pale skin and mucous membranes are usually observed in aortic heart disease. Particularly pronounced pallor is noted in stenosis of the aortic mouth due to the small blood filling of the vascular system during ventricular systole, as well as

reflex spasm of the vessels during diastole. Pallor of the skin is also observed in case of insufficiency of the aortic valves due to insufficient filling of the pre-capillaries with blood during diastole.

Jaundice staining of the skin and mucous membranes. With minor heart defects in the period of pronounced decompensation, a slight icteric sclera, and sometimes skin, can be observed.

The appearance of jaundice is associated with stagnation in the liver or the development of cirrhotic processes in it (cardiac cirrhosis of the liver).

Subcutaneous fat layer. Patients with valvular heart disease may experience a sudden weight loss, combined with the presence of OTE-cov. This symptom is called the cachectic stage of heart disease and occurs in the stage of severe decompensation.

Edema (oedema). With heart defects, edema is a characteristic sign of the development of decompensation. One of the main reasons for the development of edema is a violation of the relationship existing in the capillaries, between hydrostatic and oncotic pressure. If hydrostatic pressure is greater than oncotic comes transudate fluid from the blood into the surrounding tissue and Vice versa.

Normally, in the arterial knee of capillaries, the hydrostatic pressure is approximately 400–450 mm of water column and exceeds the oncotic pressure of plasma, which is 350 mm. This causes the flow of fluid from the blood into the tissues. In the venous knee of the capillaries, the hydrostatic pressure is normally lower than the oncotic plasma pressure and approximately equal to 170 mm of water. art., which leads to the reverse flow of fluid from the tissues into the blood. In mitral defects and insufficiency of the tricuspid valve is the development of chronic right ventricular failure. The outflow of blood from the veins of a large circle of blood circulation to the right ventricle is difficult, which leads to an increase in hydrostatic pressure in the veins and capillaries. As a result, conditions are created for greater flow of fluid from the arterial bed of the capillaries into the tissues, and it is difficult to reverse the outflow of fluid from the tissues the capillary venous system. This leads to the accumulation of fluid in the tissues and the formation of edema. The extension divisions of the venous capillaries, as well as slowing blood flow in them disrupts the nutrition of their walls, cause increased permeability and contribute to the development of edema.

The formation of edema is facilitated by a violation of the excretory function of the kidneys, resulting in a slowdown in their blood flow. Violation of tissue metabolism with the accumulation in the tissues of unoxidized products and sodium chloride also leads to fluid retention in the tissues. A decrease in the oncotic blood pressure, which can be observed in the cachectic stage of heart failure with heart defects, also contributes to the formation of edema. Edema in heart diseases are subject to hydrostatic laws and appear first at the ankles, on the feet, especially in the evening when walking and disappear in the morning, after a night of rest.

Further, the swelling may increase and the definition based on the legs, hips, lower back, genitals, in a chest (hydrothorax), abdomen (ascites), pericardium (hydropericard). Massive common edema is called anasarca.

Cardiac edema under the influence of gravity can change its position: when the position on the back, they focus in the sacrum, when the patient's position on the side are moved mainly the appropriate side. Heart swelling is combined with a bluish color of the skin. Simultaneously with the accumulation of fluid in the subcutaneous tissue develops significant edema in the parenchymal organs: liver, kidneys and gastrointestinal tract.

Edema is detected during examination, as well as palpation by pressing the thumb in the area of the ankles, foot, on the inner surface of os tibiae, sacrum and other parts of the body. When pressed, a fossa appears, which is then gradually smoothed. With edema, the skin looks smooth, shiny and initially soft, and with prolonged swelling becomes dense and difficult to pressure. With an array of governmental edema can occur the bubbles that burst, and of them master-repents liquid. With massive edema of the subcutaneous tissue of the abdomen, ruptures appear, followed by scarring, resembling scars after pregnancy (striae gravidarum).

To judge the reduction or increase of edema, it is necessary to monitor the diuresis and make a systematic weighing of the patient.

Examination of the neck, limbs and peripheral vessels. When examining the neck, sometimes pulsation of the carotid arteries can be detected. This symptom is called «carotid dance» and is characterizing aortic valve insufficiency. It is caused by sharp fluctuations between the maximum and minimum blood pressure. When this Vice can be seen a peculiar phenomenon, which is expressed in shaking the head (a symptom of Musset), which occurs due to a sharp pulsation of the carotid arteries. Pulsation of the carotid arteries is usually combined with a simultaneous sharp pulsation of peripheral vessels (art. subclavia, brachialis, radialis, etc.). Such rapid pulsation of all vessels get name La homo pulsans (pulsating).

On the neck, you can sometimes see the pulsation and swelling of the jugular veins due to difficulty in the outflow of venous blood to the right atrium.

Normally, a healthy person in the supine position may have a swelling of the neck veins, but this swelling disappears completely in vertical position. If the swelling of the veins in the vertical position remains, you can suspect the failure of the right ventricle with heart defects.

The pulsation of the jugular veins, coinciding in time with the ventricular systole (the so-called positive venous pulse), is a symptom of tricuspid valve insufficiency. In these cases, when the vein is pressed with a finger, its pulsation is noted below the place of pressing, which is due to the retrograde blood flow through the not completely closed right atrioventricular valve during the ventricular systole.

Pronounced pulsation in the epigastric region can occur due to the reposition of the enlarged and hypertrophied right ventricle.

Examination of the heart. In patients with heart defects it is possible to detect the deformity of the chest in the form of protrusions in the area of the heart, which is called the heart hump.

Examination of the heart area in healthy people with moderately pronounced subcutaneous fat can reveal a clear rhythmic pulsation due to the impact of the apex of the heart on the front chest wall, which is called the apical push.

The apical impulse is normally located in the fifth intercostal space 1–2 cm inside the left mid-clavicle line and is sometimes visible during examination.

The apical impulse. is clearly visible in thin people with wide intercostals. In fat people with narrow intercostal spaces, it is not visible. There may be displacement of the apical impulse laterally in case of insufficiency of the mitral valve and narrowing of the left atria-ventricular holes when the right ventricle with its hypertrophic status shifts to the left the left ventricle. In the aortic insufficiecy the impetus is shifting not only the left but also downwards. During the inspection the heart is sometimes visible by pronounced diffuse pulsation. It occurs with a significant expansion of the heart, when a large surface of the right ventricle lies directly to the chest (heartbeat).

PALPATION OF THE HEART REGION

Palpation of the heart area allows to confirm the presence or absence of cardiac and apical tremors and the symptom of «cat purring». The cardiac impulse reflects mostly the work of the right one-the heart's ventricle. For palpation of the cardiac impulse (fig. 1) the right hand is positioned so that the palm is above the area absolute cardiac dullness.

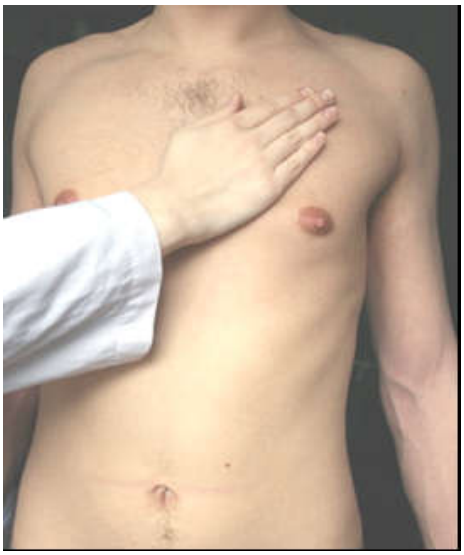


Fig. 1. Palpation of the cardiac impulse

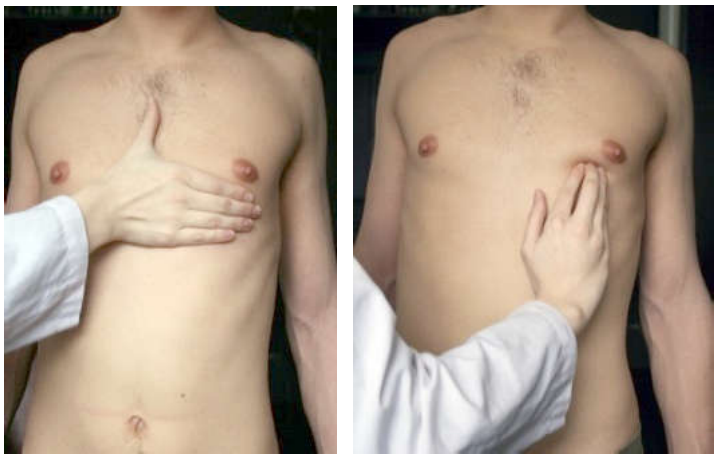
In healthy people the cardiac impulse might not be defined. With hypertrophy and dilatation of the right ventricle, a sharp pulsation appears in the area of absolute dullness of the heart and in the epigastric region, where it can be seen, and is determined palpationally.

To determine the apical impulse, the doctor places the palm of the right hand on the patient's chest in such a way that the fingers cover the area of the apical impulse (fig. 2). The palm of the right hand is placed on the patient's chest (in women, the left breast is previously removed up and to the right) with the base of the hand to the sternum, the thumb is parallel to the sternum, and the remaining fingers go in the direction of the axillary area between the fourth and seventh ribs (fig. 2, *a*). Then the pulp of the end phalanges II–IV fingers, placed perpendicular to the surface of the chest, specify the place of the push, moving them along the intercostals from the outside to the inside to the place where the fingers when pressed with moderate force begin to feel the upward movement of the apex of the heart (fig. 2, *b*). If the apical impulse occupies a significant area, then find its boundaries, determining the leftmost and lowest point of the protruding area, which is considered the location of the apical push. The feeling of the apical impulse can be facilitated by tilting the upper half of the patient's body forward or when the patient takes a deep breath with a delay in breathing — in this position, the heart is more closely adjacent to the front chest wall. When palpation, the localization of the apical impulse, the width or its area, as well as the magnitude, strength and resistance are determined. Normally, the apical impulse is localized in the fifth intercostal space, 1–1.5 cm inside of the left midclavicular line. At position the patient on the left side of the impulse is shifted to the left by 3–4 cm, and on the right side — to the right by 1–1.5 cm. Persistent displacement of the apical impulse may depend on the defeat of the heart or surrounding organs. With an increase in the left ventricle, the apical impulse shifts to the left to the anterior axillary line and simultaneously down to the sixth or seventh intercostal space. With the expansion of the right ventricle, apical impulse can shift to the left, since the left ventricle is pushed aside by the expanded right ventricle to the left side.

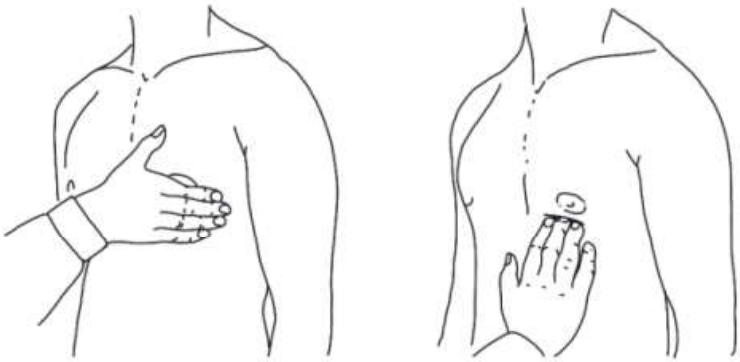
In those cases, when the apical impulse is palpable, determine its width or area. The width of the apical impulse is understood as the area of the concussion of the chest produced by it; normally the area is 1–2 cm^2. In $^1/_3$ of cases, the apical impulse is not palpable, since it is covered by a rib. When the width of the apical impulse occupies a smaller area, it is called limited, if more-time-cast. A limited apical impulse. occurs in cases where the heart is adjacent to the chest with a smaller surface than normal. This can be observed in emphysema, when the lungs cover most of the heart and impulse. it away from the chest.

Next, determine the height or resistance of the apical impulse. This characteristic is determined by the amplitude of oscillations of the chest wall at the apex of the heart. Examiner has to distinguish between high and low apical resistance. This

property of the impulse changes at the same time with his width. Usually, when the large surface of the heart is adjacent to the chest, and also with a faster reduction of the heart, the apical impulse is high. When the heart is pushed back, as well as under some other conditions (obesity, well-developed muscles), the height of the upper impulse decreases.



a



b

Fig. 2. Palpation of the apical impulse

The strength of the apical impulse is measured by the pressure exerted by the apex of the heart on the palpating fingers. The strength of the apical impulse. depends on the thickness of the chest and the proximity of the apex of the heart to the palpating fingers, as well as the strength of the left contraction ventricle's. The increased apical impulse is usually caused by hypertrophies of the left ventricle and an increase in the force of its contraction. At insufficiency of the aortic valves there is a sharp hypertrophy of the left ventricle, and there is a pronounced strong, as they say, «lifting», «dome-shaped» apical impulse. When the pericardium is compared with the anterior wall of the chest, it is possible to observe during ven-

tricular systole not protrusion, but retraction of the chest wall. Such impulse is called negative.

Resistance is determined by palpation of the apical impulse and it allows you to get an idea of the density of the heart muscle itself. The density of the muscle of the left ventricle increases significantly with hypertrophy, and then talk about a resistant apical impulse.

The concept of systolic and diastolic tremor in the heart area. Determination of the symptom of «cat purring». When palpation of the heart area in patients with heart defects can be identified symptom, which is called «cat purring» (fremissement cataire). This symptom is described by French clinicians. The name is due to the feeling (palpation of the heart), which occurs when stroking purring cat. It can appear both during systole and during diastole, and appears when low sounds occur in the heart (about 16 oscillations per second). To identify the «cat purring» you need to put your right hand flat on all points, where it is customary to listen to the heart. The sensation of this symptom on the heart's apex during diastole, and more often at the end is called pre-systolic or diastolic jitter and is characteristic of mitral stenosis. The tremor of the chest over the aorta during systole is called systolic tremor and occurs when stenosis of the aortic mouth. Chest tremor over the pulmonary artery occurs when stenosis of the pulmonary artery or non-infection of the Botallov's duct.

PERCUSSION OF THE HEART

Percussion of the heart is performed to determine the boundaries of relative and absolute cardiac dullness, heart configuration, the size of the heart diameter and vascular bundle.

The principle of percussion is based on the fact that the lungs surrounding the heart give a clear pulmonary (loud) sound when percussion, and the heart as a dense muscular organ — a quiet dull sound. This allows you to determine the boundaries of the heart by percussion. The anterior surface of the heart is partially covered with lungs and when percussion over this area there is a blunt percussion sound. This area is called relative cardiac dullness. Percussion of the boundaries of relative cardiac dullness makes it possible to reveal the true size of the heart and their projection on the chest. The area of the heart that is not covered with lungs will give absolutely dull percussion sound. The definition of its boundaries is called absolute cardiac dullness and is formed by the right ventricle.

The position of the patient. Percussion of the heart is carried out in the vertical position of the patient, the hands are down («at the sides»), with normal breathing. In the severe condition of the patient, percussion is carried out in a hori-

zontal position, in this position the size of the heart dullness will be 15–20 % more than in the vertical due to the lower standing of the diaphragm.

Doctor's position. When percussion of the heart, the doctor is standing in such a way that it is convenient for him to put his finger-plessimeter correctly and apply a percussion blow.

Percussion order. When percussion of the heart is done, use indirect percussion (percussion is applied with a finger on the finger). Finger-plessimeter tightly applied to the chest and have parallel to the expected border. In determining the boundaries of relative cardiac dullness percussion should be of medium strength (quiet percussion) (fig. 3). To identify the boundaries of absolute cardiac dullness, the method of the quietest percussion is used. Percussion is carried out from a clear pulmonary sound towards a blunt one, the border is noted from the side of a clear pulmonary sound (along the outer edge of the finger-plessimeter facing the organ giving a louder percussion sound).

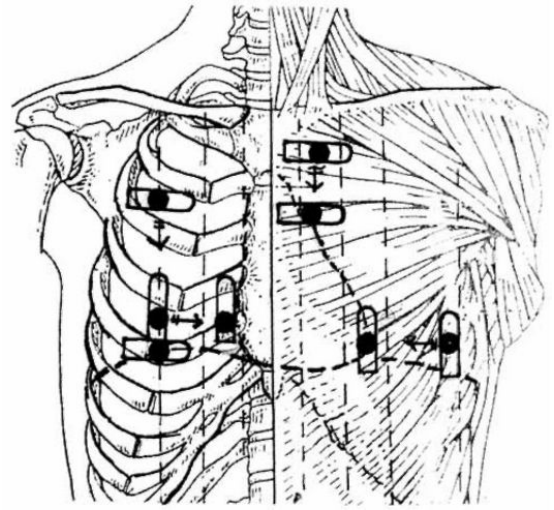
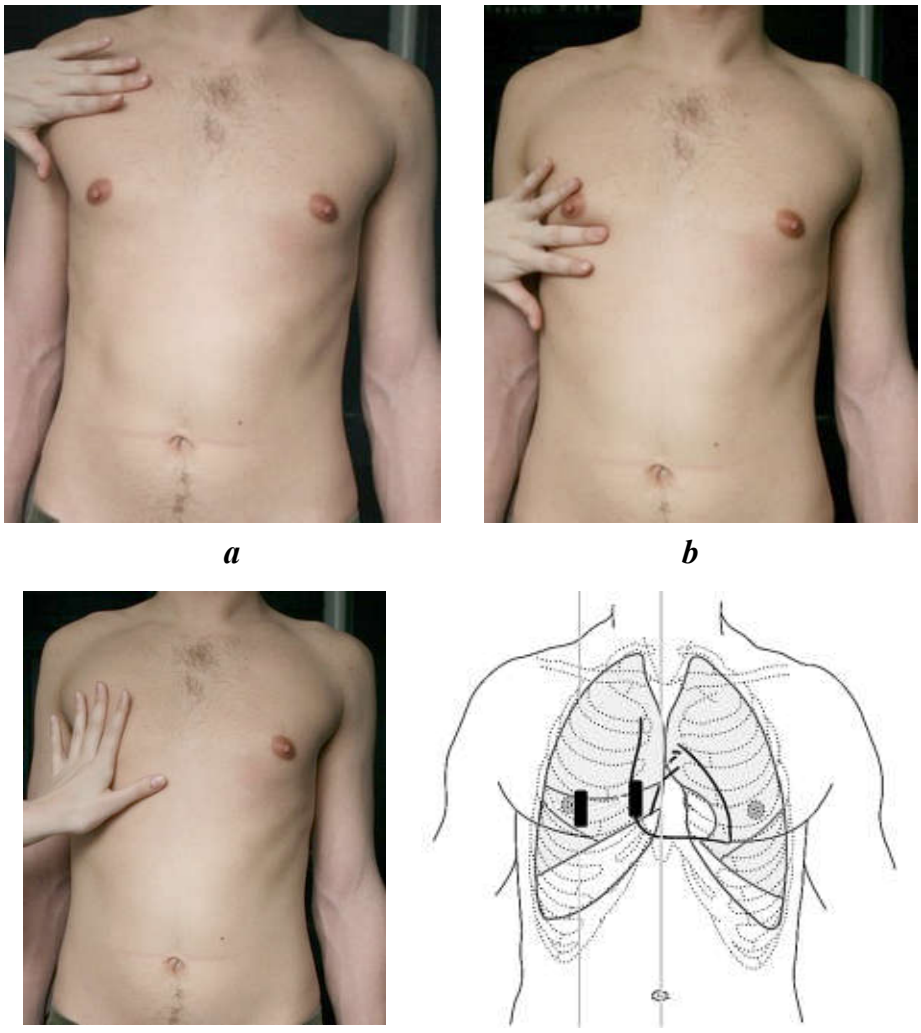


Fig. 3. The initial position of the plessimeter finger and the direction of its movement during percussion of the boundaries of relative cardiac dullness

Determination of the boundaries of relative cardiac dullness

Before percussion of the boundaries of relative cardiac dullness, it is necessary to determine the lower border of the right lung or the upper border of absolute hepatic dullness along the right midclavicular line (fig. 4, *a*). This is done in order to determine the height of the diaphragm, as it affects the size of the boundaries of relative cardiac dullness. With a high standing diaphragm, the heart takes a more horizontal position (the so-called lying heart) and in this case the size of the relative cardiac dullness will be somewhat larger than normal. When the diaphragm is low, the heart takes a more vertical position, and the size of the boundaries of relative cardiac dullness becomes smaller.

Finger-plessimeter put in the second intercostal space, right midclavicular line parallel to the ribs (fig. 4, *a*) and percutated top to bottom moving percussion sounds from the clear lung to dullness, mark the border from the clear pulmonary sound. The lower border of the lungs is normally at the level of the sixth rib (fifth intercostal space) (fig. 4, *b*). After that, the finger-plessimeter is moved one intercostal space up (from the fifth to the fourth) and placed parallel to the right border of the heart (fig. 4, *c*). Percussion is carried out from the right midclavicular line towards the sternum, strikes of medium force (the method of quiet percussion). In the transition from percussion to clear pulmonary blunting the border mark on the outer edge of the finger from the clear pulmonary sound. Normally, in a healthy person, the right border of relative cardiac dullness is 3–4 cm to the right of the anterior median line in the fourth intercostal space.



a ***b*** ***c***

Fig. 4. Determination of the right border of relative cardiac dullness (*a*, *b*, *c*)

Before determining the left border of relative cardiac dullness (fig. 5), it is necessary to find the apical impulse, which coincides with the left border of the relative dullness of the heart and is formed by the left ventricle. If apical impulse palpation is not defined, then the definition of the left border of the relative cardiac dullness are produced in the fifth intercostal space starting from the mid-axillary line. In this case, the finger-plessimeter is placed parallel to the expected left border and percussion is carried out from left to right until the transition of a clear pulmonary sound into dull. The method of quiet percussion is used. The border is marked on the outer edge of the finger from the clear pulmonary sound. Normally, in a healthy person, the left border of relative cardiac dullness is in the fifth intercostal space 8–9 cm to the left of the anterior median line.

The finger-plessimeter is placed near the left edge of the sternum (fig. 6) parallel to the ribs in the first intercostal space and percussion down by the method of quiet percussion until the transition of the percussion sound from the clear pulmonary to dull, the border is marked on the upper edge of the finger, facing the clear pulmonary sound. Normally, the upper limit of the relative dullness of the heart is located on the upper edge of the third rib and is formed by the cone of the pulmonary artery and the ear of the left atrium.

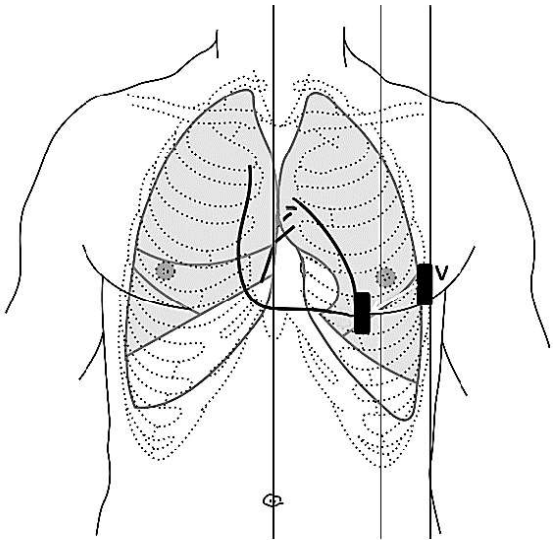


Fig. 5. Determination of the left border of relative cardiac dullness

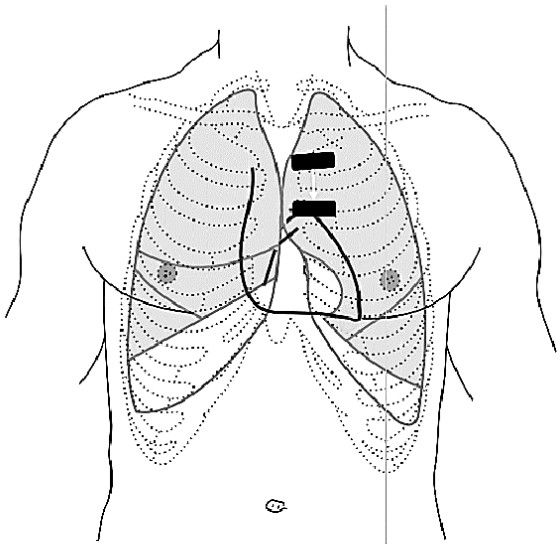


Fig. 6. Determination of the upper limit of relative cardiac dullness

The definition of the boundaries of the vascular bundle

The boundaries of the vascular bundle are determined percutaneously in the second intercostal space to the right and left of the sternum (fig. 7). The finger-plessimeter is placed vertically along the mid-clavicle line so that its middle phalanx lies in the second intercostal space. Using quiet percussion beats, percussion at this

level towards the edge of the sternum, holding the finger-plessimeter in an upright position and shifting it after each pair of strokes at 1 cm to detect the border of the transition of clear pulmonary sound in blunt.

Normally, the width of the vascular bundle is 5–6 cm.

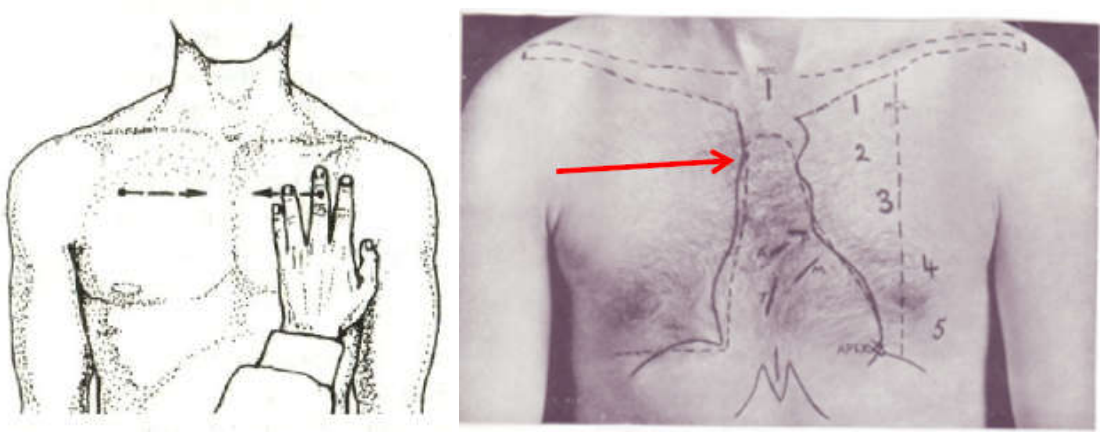


Fig. 7. Determination of the boundaries of the vascular bundle

The determination of the diameter of the relative dullness of the heart (or measurement of the transverse dimensions of the heart) is carried out on the basis of establishing the boundaries of the relative dullness of the heart. The diameter of the relative dullness of the heart is the sum of the values of the right (normally 3–4 cm) and left (normally 8–9 cm) boundaries of the relative dullness of the heart in relation to the anterior median line (fig. 8).

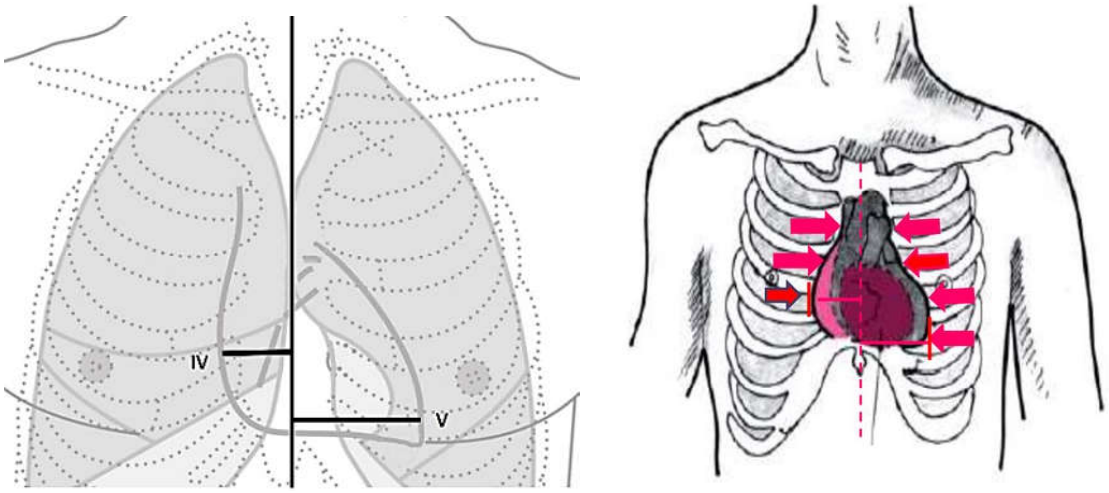


Fig. 8. Determination of the diameter of relative cardiac dullness and configuration of the heart by intercostal space

Normally, the diameter of the heart is 11–13 cm, to determine this value, it is necessary to measure the distance in the 4th intercostal space to the right of the marked border of the relative dullness of the heart to the anterior median line. After that, the distance from the mark of the left border of the relative dullness of the heart to the anterior median line is measured in the 5th intercostal space on the left. The boundaries of relative and absolute cardiac dullness are shown in fig. 9.

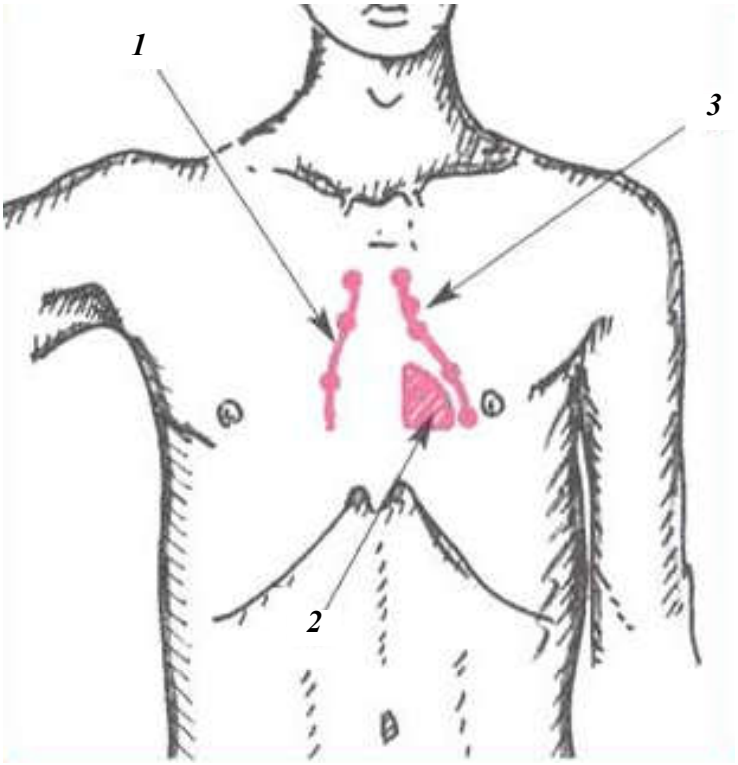


Fig. 9. Boundaries of cardiac dullness:
1, *3* — relative; *2* — absolute

The types of configurations of the heart

The configuration of the heart can be normal (table, fig .10) (unchanged), and in diseases of the heart — mitral, aortic and triangular (or trapezoidal) (fig. 11).

Limits of relative cardiac dullness (relative to the anterior median line)

Intercostal space	Right	Left
2	2.5–3 cm	2.5–3 cm
3	3–4 cm	4–5 cm
4	3–4 cm	–
5	–	8–9 cm

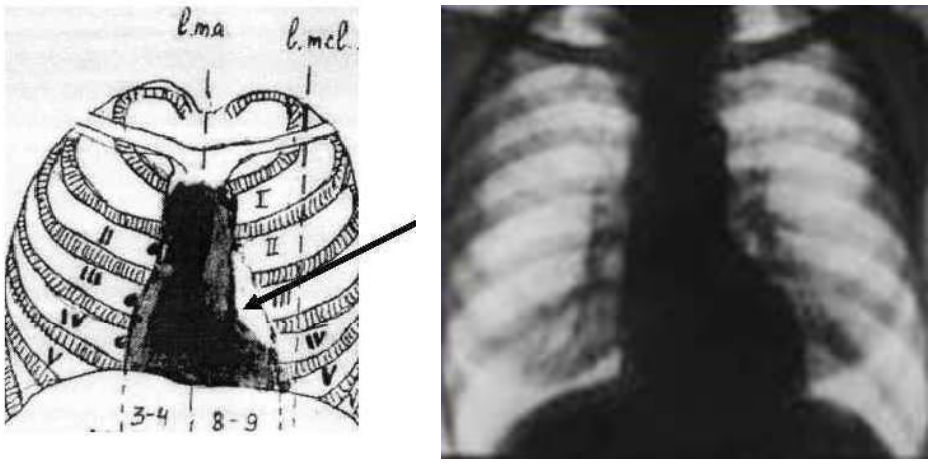
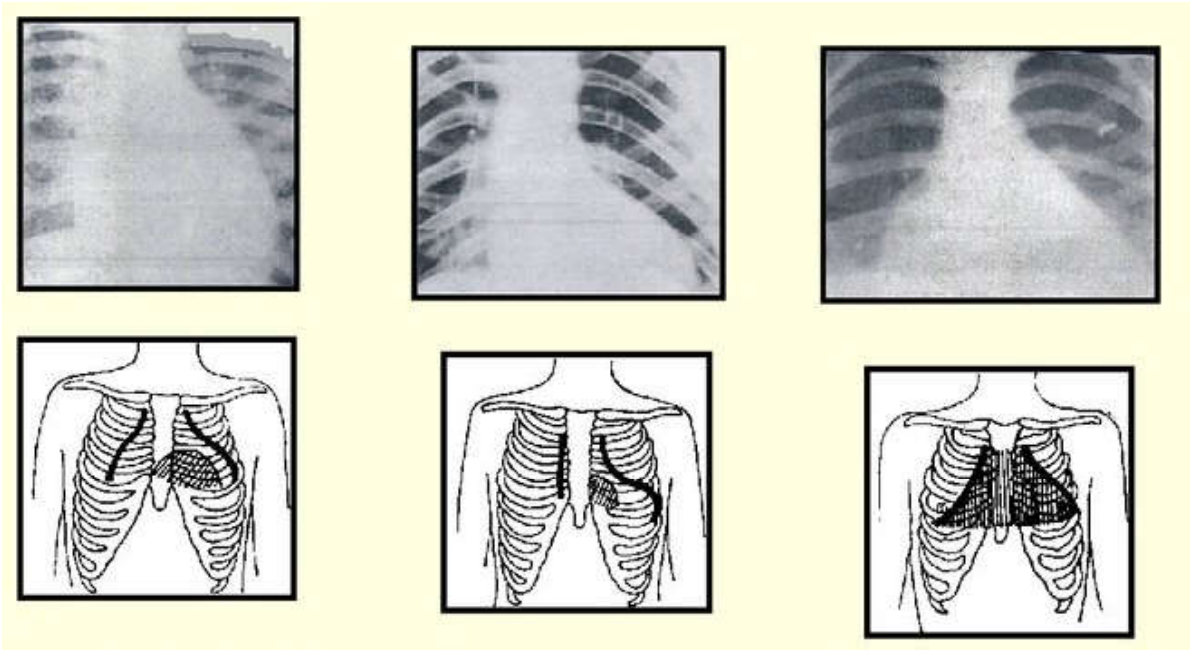


Fig. 10. Normal configuration of the heart



mitral aortic trapezoidal

Fig. 11. The configuration of the heart mitral, aortic and triangular (or trapezoidal)

Normally, the left contour of the heart between the vascular bundle and the left ventricle has a blunt angle — this is the normal configuration of the heart.

In pathological conditions, the ways of «inflow» and «outflow» of the right and left ventricles increase, which leads to the formation of a pathological configuration of the heart. Mitral configuration of the heart is in mitral heart disease. In such cases, hypertrophy of the left ventricular inflow pathway occurs, the angle along the left contour of the heart disappears between the vascular bundle

and the left ventricle due to an increase in the left atrium. There is a shift in the outer and right boundaries of the relative dullness of the heart. The waist of the heart is smoothed.

The aortic configuration of the heart takes in lesions of the aorta or its valves, as well as hypertension. In this configuration, there is hypertrophy of the outflow pathways, the boundaries of the relative dullness of the heart to the left due to an increase in the left ventricle, the angle along the left contour between the vascular bundle and the left ventricle approaches the right corner. The heart takes the form of a «boot» or «sitting duck».

Triangular (or trapezoidal) configuration of the heart acquires due to the accumulation of a large amount of fluid in the pericardial cavity. As a result, the boundaries of relative dullness are shifted in both directions. The heart in this case resembles a «roof with a chimney».

Determination of the boundaries of absolute dullness of the heart

Absolute dullness of the heart is a part (fig. 9) the anterior surface of the heart, which is directly adjacent to the chest, is not covered by the lungs and is formed by the right ventricle. Percussion in this area reveals a dull percussion sound. The absolute dullness is determined by the method of the quietest percussion. First, it is necessary to determine the boundaries of the relative cardiac dullness of the heart, and then determine the right, left and upper limits of the absolute dullness of the heart.

When determining the right border of the absolute dullness of the heart percussion begin from the right border of relative dullness and causing silent strokes, move the finger-plessimeter inwards before the advent of the absolutely dull sound. Having on this place a finger-plessimeter, do a mark on its external edge turned to border of relative dullness. Normally, the right border of the absolute dullness of the heart passes along the left edge of the sternum (left sternal line) in the fourth intercostal space.

When determining the left border of the absolute dullness of the heart, percussion leads from the left border of the relative dullness inside to the appearance of a dull sound. Normal left border of absolute dullness is 1–2 cm medially from the borders of relative dullness of heart.

To determine the upper edge of the absolute dullness of the heart, the finger-plessimeter is placed on the upper limit of the relative dullness of the heart and percutate, moving it downwards until a dull sound appears. Normally, the upper edge of absolute dullness of the heart is located on the fourth rib.

AUSCULTATION OF THE HEART

Patient position. Auscultation of the heart is carried out in various positions of the patient — vertical, horizontal, lying on the left side. Since the sound phenomena that occur with the pathology of various heart valves depend on the positioning of the valve to the surface of the chest and on the pathological blood flow through it. For example, protodiastolic murmur, with aortic valve insufficiency, is better heard in a vertical position of the patient, and systolic murmur, with mitral valve insufficiency, in a horizontal position.

Auscultation of the heart is performed in different phases of the breathing act. The heart must be auscultated while holding the breath to eliminate sound effects from the lungs, which may complicate the assessment of auscultation data from the heart. The patient is asked to inhale and then exhale and then hold his breath. While holding your breath, listen to the heart. Since holding your breath cannot be long, the procedure must be repeated.

Auscultation of the heart is performed after mild physical activity, for example, after repeated squats or other physical exercise. This makes it possible to more clearly identify sound phenomena from the heart, due to increased heart contractions and accelerated blood flow.

Doctor's position. When auscultating the heart, the doctor is located on the right side of the patient. In this case, his position should be such that he can freely and correctly apply the stethoscope to the places where the heart is heard.

True projection of the heart valves on the anterior surface of the chest.

The valve openings of the heart are located at the base of the heart and their projection onto the anterior wall of the chest is at a very close distance from each other). The projection of the mitral valve corresponds to the place of attachment of the third rib to the sternum on the left, the aortic valves are located behind the sternum at the level of the third costal cartilages, the pulmonary valves are in the second intercostal space on the left at the edge of the sternum, the tricuspid valve is located behind the sternum in the middle of the line connecting the places of attachment of the cartilages of the third rib on the left and the fifth rib on the right. Such proximity of the valve openings to each other makes it difficult to listen to the heart in the places of the true projection of the valves. Therefore, the places of auscultation were clarified where sound phenomena are best carried out from each of these valves separately.

The procedure for auscultation of the heart

Auscultation of the heart is carried out in the following order (fig. 12–17).

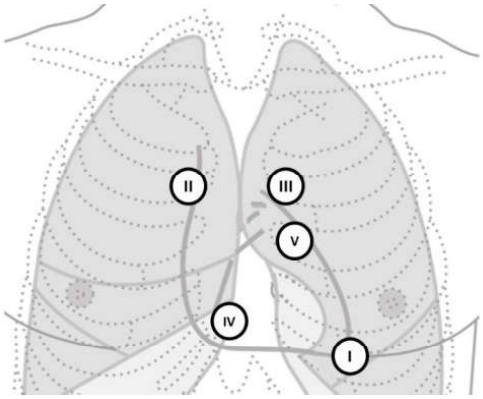


Fig. 12. The auscultation scheme of the heart

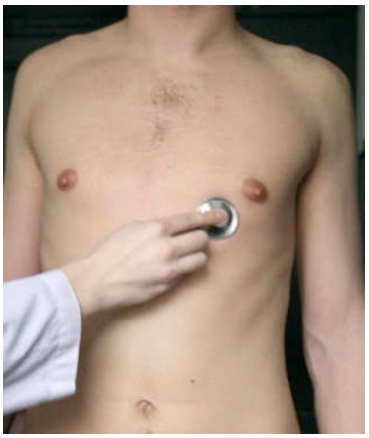


Fig. 13. The first point of auscultation is the apex of the heart, listening to the mitral valve

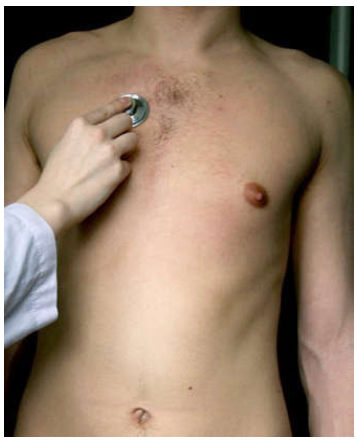


Fig. 14. The second point of auscultation is the second intercostal space to the right of the sternum, the aortic valve is heard

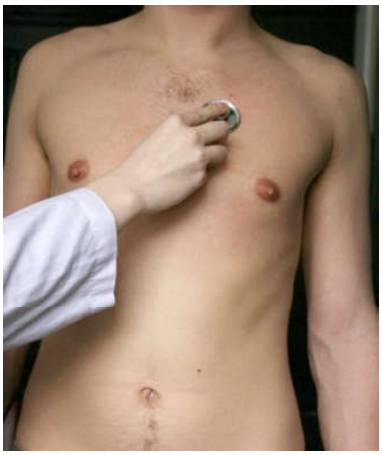


Fig. 15. The third point of auscultation is the second intercostal space to the left of the sternum, listening to the pulmonary valve

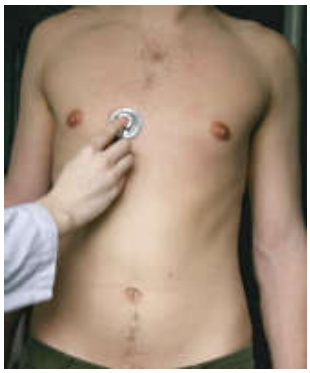


Fig. 16. The fourth point of auscultation is at the base of the xiphoid process to the right of the sternum, the tricuspid valve is heard

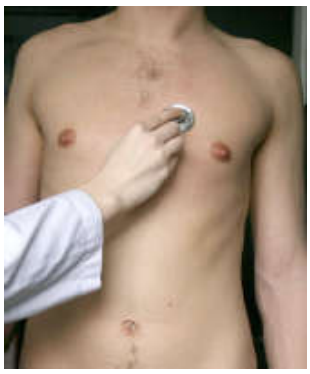


Fig. 17. The fifth point of auscultation is the place of attachment of the third — fourth rib to the sternum on the left, an additional point for listening to the aortic valve. Received the name — Botkin-Erb point

This auscultating sequence is determined by the frequency of damage to the heart valves. With the development of acquired heart defects, damage to the mitral valve is most often detected, and then to the aortic valve.

Auscultation of the heart in healthy people

Normally, two sounds are heard at all 5 points of auscultation: S1-1 sound, occurring during ventricular systole, is called systolic, and S2-2 sound, occurring during ventricular diastole, is called diastolic.

Sometimes, in addition to the first and second sounds, adolescents and young adults may hear the third and fourth sounds during diastole.

The mechanism of heart sounds 1st sound is formed as a result of the summation of sound phenomena occurring in the heart at the beginning of systole. It consists of four components:

a) **the valve component** is the main one, caused by vibrations of the atrio-ventricular valve leaflets in the phase of isometric contraction. The magnitude of oscillations of the atrioventricular valves is influenced by the speed of contraction

of the ventricles; the faster they contract, the faster the intraventricular pressure increases and the more sonorous the 1 sound. An additional role is played by the position of the atrioventricular valve leaflets at the beginning of systole, which depends on the blood filling of the ventricles: the less the ventricles are filled with blood in diastole, the wider the valve leaflets are open, and the greater the amplitude of their oscillations during systole;

b) **muscle component** — is formed during the period of isometric tension and is caused by vibrations of the ventricular myocardium;

c) **vascular component** — associated with fluctuations in the initial segments of the aorta and pulmonary trunk when they are stretched by blood during the expulsion period;

d) **atrial component** — caused by fluctuations during atrial contraction. 1 sound begins with this component, since atrial systole precedes ventricular systole. Normally, vibrations formed by atrial systole merge with sound vibrations caused by ventricular systole and are perceived as one sound.

The 2nd sound occurs during diastole and consists of two components:

a) **valve component** — closure of the semilunar valves of the aorta and pulmonary trunk;

b) **vascular component** — vibrations of the walls of the aorta and pulmonary trunk.

The S3 **3th sound**. The mechanism of occurrence of the third sound is associated with vibrations of the walls of the ventricles during their rapid filling with blood at the beginning of diastole. The duration of the sound is 0.03–0.06 s.

The S4 **4th sound** precedes the 1st sound and occurs at the end of diastole. The mechanism of occurrence of this sound is associated with the rapid filling of the ventricles with blood due to contraction of the atria. An image of normal heart sounds is shown in Figure 1 Difference between 1st and 2nd heart sounds.

Both sounds differ from each other based on the following characteristics:

– the 1st sound at the apex of the heart is stronger, louder and longer than 11 sound; this is explained by the fact that sound phenomena from the mitral valve, vibrations and tension of the valves, which participate in the formation of the 1st sound, are best transmitted to the apex of the heart, while the 11th sound occurs far from the apex of the heart and is weaker transmitted to this area;

– the 1st sound is heard after a long pause;

– the 1st sound coincides with the apical impulse and pulse in the carotid artery;

– the 2nd sound, on the contrary, is heard more strongly than the 1st — in the second intercostal space on the right (aorta) and on the left at the edge of the sternum (pulmonary artery), since sound phenomena from the semilunar valves are better transmitted here, when they close, the 2th sound is formed.

The normal sound image diagram is shown in fig. 18.

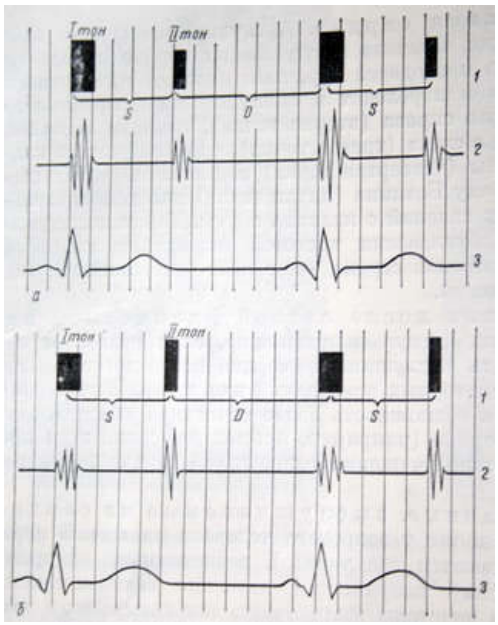


Fig. 18. Image of normal heart sounds (diagram). Above is the top of the heart, below is the base of the heart:

1 — graphic image of heart sounds; *2* — FKG; *3* — ECG; *S* — systole; *D* — diastole

Changes in heart sounds. Changes in heart sounds can manifest themselves in the form of a weakening or strengthening of the sonority of both sounds or each of them separately, a change in their duration, as well as splitting and bifurcation of sounds and the appearance of additional sounds.

Weakening of both heart sounds. It may depend both on reasons outside the heart, which create worse conditions for sound phenomena from the heart, and on diseases of the heart itself.

Extracardiac causes that cause a weakening of both heart sounds can be: obesity, pronounced muscles, chest edema, enlarged mammary glands, emphysema, left-sided exudative pleurisy, hydrothorax, pneumothorax and pronounced effusion pericarditis, when large particles accumulate in the cavity of the cardiac membrane. amount of liquid.

When the heart itself is damaged, weakening of both heart sounds most often indicates a decrease in the contractility of the myocardium of the left and right ventricles (non-rheumatic myocarditis, atherosclerotic cardiosclerosis, myocardial infarction, left ventricular aneurysm and other lesions of the heart muscle). Weakened sounds are often given the name muffled, dull sounds. A graphical representation of the weakening and strengthening of sounds is presented in fig. 19.

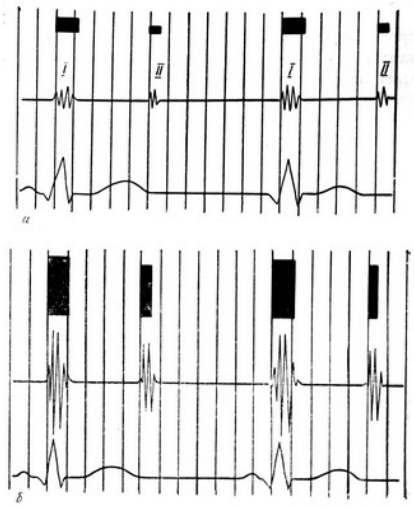


Fig. 19. Image of changes in heart sounds (diagram). Weakening of heart sounds (above). Increased heart sounds (bottom)

Strengthening both heart sounds. Increased heart sounds are observed after physical exertion, with tachycardia, Graves' disease. This is due to an increase in the influence of the sympathetic nervous system on the heart. An increase in both sounds can be heard in asthenics, with a thin chest, with a high diaphragm, wrinkling of the lungs, sudden weight loss, and thickening of the edges of the lungs close to the heart.

Weakening of 1 sound at the top. More often occurs with valvular heart defects — insufficiency of the mitral and aortic valves.

With mitral valve insufficiency due to a decrease in the surface and oscillatory movements of its wrinkled valves, a weakening of the valve component of the 1st sound occurs. With this defect, in addition, there is no period of closed valves due to the presence of a defect in the valve leaflets, which leads to the reverse flow of part of the blood from the left ventricle into the left atrium and weakening of the muscle component involved in the formation of 1 sound. It should be noted that the degree of weakening of the 1st sound with this defect is directly proportional to the degree of mitral valve insufficiency, i. e., the more pronounced its insufficiency, the weaker the 1st sound is heard. This is very important for assessing the degree of valve damage in a given heart defect. The same can be said for aortic valve insufficiency. The weakening of the 1st sound in this heart defect is also due to the absence of a period of closed valves of the left atrioventricular orifice, due to the development of relative mitral valve insufficiency.

Weakening of the 1st sound can be observed with lesions of the heart muscle (myocarditis, cardiosclerosis), as well as with insufficiency of the tricuspid valve and pulmonary valve. However, these defects are much less common. A graphical representation of the weakening and strengthening of 1 sound is presented in fig. 20.

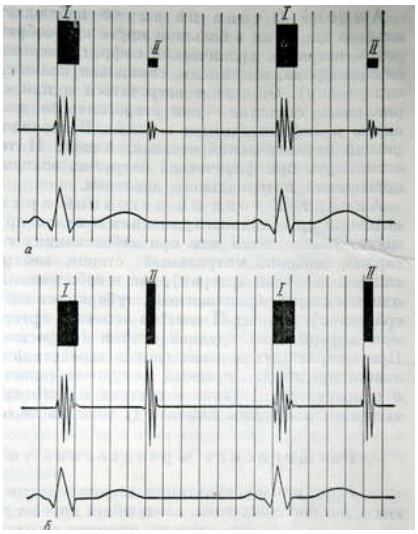


Fig. 20. Changes in the 1st heart sound at the apex (diagram). Weakening (top), strengthening (bottom)

Strengthening 1 sound at the top. Typically, 1 sound becomes louder when there is less filling of the ventricle with blood during diastole and a faster contraction during systole. These conditions are especially pronounced with mitral stenosis. With this defect, due to the narrowing of the left atrioventricular orifice, blood passes during diastole from the left atrium into the left ventricle with difficulty, and its filling at the beginning of systole is less than normal. This causes rapid contraction of the left ventricle and leads to increased and shortened sound at the apex of the heart. Such a loud, shortened 1st sound at the apex of the heart with mitral stenosis is called the «flapping sound» and is a characteristic sign for this defect.

When the right atrioventricular orifice is narrowed, an increase in the first sound in the lower third of the sternum (the fourth point of auscultation) may also be observed, but this type of defect is rare.

With atrial fibrillation, ventricular extrasystoles, when the filling of the ventricles with blood during diastole is insufficient, an increase in the 1st sound may also be noted at the apex of the heart. The 1st sound is especially loud during complete atrioventricular heart block, when contraction of the atria and ventricles occurs simultaneously. This loud sound was called the «gun sound» and was first described by N. D. Strazhesko.

Weakening of the 11th sound on the aorta. It can be observed with aortic heart defects: aortic valve insufficiency and narrowing of the aortic mouth (fig. 21). In case of aortic valve insufficiency, the weakening of the 11th sound is partly due to the destruction of the semilunar valves, as well as a decrease in their vibrations due to the development of scar compaction in them. The weakening of the 11th sound in the aorta with this defect is directly proportional to the degree of

aortic valve insufficiency. With pronounced aortic valve insufficiency, the 11th sound may not even be heard at all. Weakening of the 11th sound in the aorta when the aortic orifice narrows occurs due to a decrease in pressure in it. Changes in sound 11 above the aorta are graphically presented in fig. 21.

Weakening of the 11th sound on the pulmonary artery. It can occur with very rare valvular heart defects: pulmonary valve insufficiency and narrowing of the orifice of the pulmonary trunk. The reasons for the weakening of the 11th sound in these cases are the same conditions as for aortic defects.

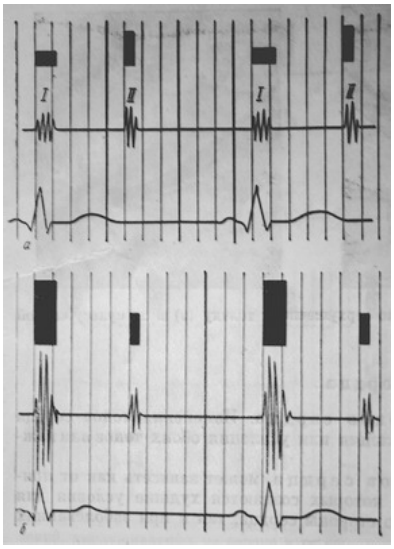


Fig. 21. Change in 2 sound over the aorta (diagram). Weakening (top), strengthening (bottom)

Gain (accent) 11 sounds. Normally, in an adult, when listening and comparing the 11th sound in the aorta and pulmonary artery, it is noted that their strength is the same. This is explained by the fact that the pulmonary artery valve is located closer to the chest than the aortic valve, due to which the transmission of sound phenomena from them is equalized.

In children and young people, the 11th sound on the pulmonary artery is often stronger than on the aorta. This is explained by the fact that the pressure in the aorta in children is lower than in adults, and the pulmonary artery is located closer to the chest.

Under some conditions, the strength of the 11th sound in the aorta and pulmonary artery may be unequal. In cases where the 11th sound on the aorta is stronger than on the pulmonary artery, they speak of an emphasis (intensification) of the 11th sound on the aorta (fig. 21); if the 11th sound is stronger on the pulmonary artery, they speak of its emphasis on the pulmonary artery. The strength of the 11th sound depends on the force of the push of blood against the leaflets of the aortic valve or pulmonary artery during diastole and usually runs parallel to the height of blood pressure.

Accent of the 11th sound on the aorta. It is observed with increased blood pressure in the systemic circulation (arterial hypertension, acute and chronic glomerulonephritis, pyelonephritis, other symptomatic hypertension). It can also occur in the absence of increased blood pressure — with atherosclerosis of the semilunar leaflets of the aortic valve and syphilitic aortitis. In the latter case, it takes on a sharp metallic tint. An emphasis on the 11th sound on the aorta can also occur during physical activity, anxiety, or due to a temporary increase in blood pressure.

Emphasis of the 11th sound on the pulmonary artery. Indicates an increase in blood pressure in the pulmonary circulation, which can be observed both in heart diseases (mitral heart defects, especially mitral stenosis, patent ductus, pulmonary artery sclerosis) and lung diseases (pulmonary emphysema, pneumosclerosis, bronchiectasis, pulmonary tuberculosis, extensive pleural adhesions). The emphasis of the 11th sound on the pulmonary artery can also occur with chest deformities (kyphoscoliosis). With mitral heart defects, due to difficulty in the outflow of blood from the left atrium to the left ventricle, blood stagnation appears in the lungs and pressure in the pulmonary circulation system increases — this causes the appearance of an emphasis on the 11th sound on the pulmonary artery.

Splitting and splitting sounds. If, during auscultation of the heart, instead of one of the sounds, 2 short sounds are heard, following each other after a short period of time, this phenomenon is usually called split sound. Such a split can be noted in both the 1st and 11th sounds. If the distance between these two sounds is very small and there is no complete impression of their splitting, then they speak of splitting the sounds.

The splitting of both 1st and 11th sounds can be either physiological, i.e. found in completely healthy people, and pathological, observed in some heart diseases.

Physiological double sounds are more common in young people and are usually associated with the act of breathing or physical activity and are not permanent.

Split 1 sound. Depends on the non-simultaneous closure of the bicuspid and tricuspid valves.

Under physiological conditions, this phenomenon can sometimes occur during exhalation, when, due to increased pressure in the chest, blood flows more forcefully into the left atrium and thereby slows down the closure of the mitral valve. This leads to the fact that sound phenomena from the atrioventricular valves are perceived separately. In pathological cases, bifurcation of the 1st sound can occur when one of the bundle branches is blocked, when non-simultaneous contraction of the right and left ventricles of the heart occurs. Some authors call this bifurcation the systolic gallop rhythm.

Bifurcation of the 11th sound on the pulmonary artery It is explained by the non-simultaneous closure of the pulmonary artery and aortic valves due to increased blood pressure in the pulmonary circulation. A bifurcation of the 11th sound in the pulmonary artery can sometimes occur under physiological conditions, during deep inhalation or exhalation, when a temporary change in pressure in the pulmonary vessels may occur.

According to N.D. Strazhesko, it can occur «as a temporary phenomenon in children and nervous subjects with an unstable influence of the autonomic nervous system on the heart and pulmonary circulation». In most cases, bifurcation of the 11th sound in the pulmonary artery is observed mainly in mitral stenosis. When the left atrioventricular orifice is narrowed, blood flow from the left atrium to the left ventricle is obstructed, which leads to an increase in pressure in the left atrium and, reflexively, due to spasm of the pulmonary arterioles, an increase in pressure in the pulmonary circulation occurs (Kitaev reflex). As a result, the systole of the right ventricle is prolonged, which leads to non-simultaneous closure of the aortic and pulmonary valves.

Quail rhythm. The quail rhythm or three-part rhythm is most often heard with stenosis of the left atrioventricular orifice and consists of a loud (popping) first sound, a second sound, and the opening sound of the mitral valve.

An amplified first sound (clapping) is heard at the apex of the heart. This is due to the fact that during diastole the left ventricle is not filled with blood sufficiently and contracts quite quickly. Due to insufficient filling of the left ventricle with blood, the mitral valve leaflets at the time of contraction of the left ventricle are at a greater distance from the left atrioventricular orifice, and their movement with a greater amplitude produces a stronger jerky sound. The first popping sound will be heard only in the absence of gross deformations of the valves (no fibrosis or calcification of the valve).

At the apex, after the second sound, an additional sound is heard — the sound of the mitral valve opening (opening click). This phenomenon is associated with a sharp movement of the mitral valve leaflets at the beginning of heart diastole (protodiastole). Under normal conditions, the mitral valve leaflets open silently, 0.075 seconds after the onset of diastole. The opening sound of the mitral valve is heard as a result of the fact that the valve leaflets are sclerotic and fused together, they cannot completely move away to the walls of the ventricle and therefore, when a stream of blood flows from the left atrium, sound vibrations arise that form this additional sound. It usually occurs after the second sound in 0.03–0.11 seconds; the shorter the interval between the second sound and the sound of the mitral valve opening, the higher the atrioventricular pressure gradient and the more pronounced the stenosis. The opening sound does not disappear with atrial fibrillation. In some cases, the opening sound is better heard in the fourth intercostal space to the left of the sternum.

The clapping first sound, combined with the second sound and the opening sound of the mitral valve, creates a special mitral melody of a three-part rhythm at the apex of the heart — the rhythm of a quail (since it resembles the cry of a quail). The quail rhythm is best heard at the apex of the heart.

Gallop rhythm. A gallop rhythm or a three-part rhythm indicates severe myocardial dysfunction. V.P. Obraztsov gave it a figurative definition: «gallop is a cry from the heart for help». The rhythm of the gallop resembles the sound characteristics of the gallop of a galloping horse. According to the observations of V.P. Obraztsov, the additional sound that occurs during the gallop rhythm is reproduced more like a push, like a weak shaking of the chest, and therefore he suggested listening to it directly with the ear.

The gallop rhythm is heard in the area of the apex of the heart or in the third–fourth intercostal space on the left, preferably with the patient positioned on the left side and is not carried out anywhere.

It is conventionally proposed to distinguish protodiastolic, mesodiastolic and presystolic gallop.

The protodiastolic rhythm of the gallop consists of a weakened first sound (due to weakening of the muscular component, and sometimes the valve), a second sound and a pathological third sound, formed during the phase of rapid filling of the ventricles with blood, the myocardium of which expands faster than normal due to the loss of his sound. The vibrations of the ventricular wall that arise in this case create an additional third sound, which is separated from the second sound by an average of 0.12–0.15 seconds. The additional pathological third sound included in the gallop rhythm is an enhanced physiological third sound that occurs during diastole. Currently, protodiastole and the subsequent phase of isometric contraction of the ventricles means that period of diastole when the atrioventricular valves, aortic valve and pulmonary valve are closed and blood does not flow from the atria into the ventricles and therefore no additional sound can occur during this period. It occurs in the subsequent phase of rapid filling of the ventricles with blood, and therefore V.P. Obraztsov proposed calling this gallop rhythm diastolic.

The presystolic gallop rhythm is a three-part rhythm in which the first sound is weakened, followed by the second sound and an additional fourth sound is heard. The occurrence of a pathological fourth sound is due to the fact that when the hypertrophied left atrium contracts, a stream of blood under pressure enters the left ventricle, the sound of the myocardium, which is significantly reduced due to degenerative-inflammatory processes. A decrease in the sound of the ventricular myocardium leads to a sharp stretching of the wall during the flow of blood into them. Presystolic gallop is better heard in atrioventricular conduction disorders, when atrial systole is separated from ventricular systole by a longer period of time than normal. The fourth sound appears 0.08–0.14 seconds from the beginning of the P wave and has a frequency response of over 70 Hz.

The mesodiastolic gallop rhythm or summation gallop rhythm is a three-part rhythm in which the first sound is weakened, the second sound is unchanged, and the third and fourth merge into one. It is heard with tachycardia and prolongation of the P-Q interval; in the presence of these two factors, the third and fourth sounds can overlap each other. An additional sound with this gallop rhythm is heard in the middle of diastole.

HEART MURMUR

During auscultation of the heart, in addition to sounds, sound phenomena can be heard, which are called heart murmurs. Heart murmurs, depending on the causes of their occurrence, are divided into extracardiac (extracardial) and intracardiac (intracardial), organic and functional, systolic and diastolic.

Extracardiac murmur

Extracardiac murmurs include pericardial friction rub and pleuropericardial murmur.

Pericardial friction rub. For the first time, a pericardial friction rub during myocardial infarction was described by V. M. Kernig. The mechanism of occurrence of this murmur is associated with the deposition of fibrin on the pericardial sheets, during inflammatory processes in it, as a result of which they become rough and uneven. The murmur is usually heard during dry pericarditis, and sometimes during effusion, when the amount of fluid in the pericardium is insignificant. This can be observed with uremia, sometimes on days 10–14 of myocardial infarction (Dressler syndrome).

Characteristics of pericardial friction murmur:

– the murmur is heard during systole and diastole, that is, in both phases of cardiac activity;

– the murmur is very gentle in nature or, on the contrary, very rough, reminiscent of the scratching or crunching of snow underfoot, the rustling of paper;

– the murmur is heard in the area of absolute cardiac dullness (at the left edge of the sternum in the 3–4 intercostal spaces) and is not carried anywhere;

– the murmur is not constant, it may disappear and then appear after a while;

– the murmur is better heard when the body is tilted forward and when breathing is held.

Pleuropericardial friction rub. This murmur occurs when the pleural area, which is adjacent to the heart, is involved in the inflammatory process and resembles a pericardial friction murmur.

Characteristics of pleuropericardial murmur:

– the murmur is better heard on the left side of the relative cardiac dullness;

– murmur occurs during the act of breathing;

– the murmur is heard during inhalation and exhalation and is not heard while holding the breath.

Intracardial murmur

Functional murmur. Functional intracardiac murmurs occur when the heart valves are unchanged and can be observed with an increase in blood flow velocity or a decrease in blood viscosity. The causes of functional murmur are acceleration and disruption of innervation. With a constant width of the lumen of the bloodstream, murmur occurs due to an increase in the speed of blood flow during thyrotoxicosis, fever, nervous excitement. Blood viscosity decreases, for example, with anemia, and this helps to increase the speed of blood flow and can cause murmur.

Functional murmurs are:

– often systolic and heard at the apex of the heart and above the pulmonary artery, less often diastolic. In case of aortic valve insufficiency, a functional presystolic murmur (Flint murmur) can be heard at the apex of the heart, associated with the formation of functional stenosis of the left atrioventricular orifice due to the elevation of the anterior leaflet of the mitral valve by the blood flow returning from the aorta. With mitral stenosis, the pressure in the pulmonary circle increases and a functional diastolic Graham-Still murmur is detected above the pulmonary artery, caused by a sharp expansion of the mouth of the pulmonary artery;

– unstable, can appear and disappear in different body positions, after physical activity, in different phases of breathing;

– short-lived, rarely occupying the entire systole;

– soft and blowing in nature;

– limited and not carried out far from the place of origin;

– the boundaries of relative cardiac dullness do not change;

– there is no history of acute rheumatic fever;

– functional murmurs are not accompanied by other signs of valve damage (changes in heart sounds, hypertrophy of the atrial or ventricular myocardium).

Organic murmur. Intracardiac organic murmurs occur in the heart itself, and are most often caused by deformation of the cusps of the atrioventricular valves, or the valves of the aorta and pulmonary trunk, shortening of the chordae tendineae, narrowing of the atrioventricular orifices, aortic orifice and pulmonary trunk. Damage to the valves can occur individually or in combination. Organic murmur occurs in two ways; when blood passes through a narrow opening or as a result of reverse blood flow.

The occurrence of intracardiac murmurs can be explained by the physical laws of fluid flow through the tube.

For murmur to occur in the tube, certain conditions must be met:

– change in the lumen of the tube — narrowing or expansion;
– change in fluid flow speed;
– change in the composition of the liquid.

If you pass a liquid at a certain speed through a tube with the same cross-section along its entire length, then the flow of liquid through it will pass silently. When the lumen of the tube narrows in a limited area and liquid passes through it at the same speed as in the first case, now before and after the narrowing, vortex movements will arise in the tube, which will cause murmur in this place. An example of this is the following: if you place a phonendoscope on the femoral artery without squeezing it, the doctor will not hear the blood flow through the vessel; if you lightly compress the vessel with a phonendoscope, you can listen to sound phenomena. It has been established that the greater the narrowing of the tube, the stronger the murmur above it will be heard, but with a very pronounced degree of narrowing of the tube, the murmur above it may not be heard. Sometimes in patients with mitral stenosis, the left atrioventricular orifice is so narrow that the diastolic murmur may not be heard with this defect. This defect is called aphonic or silent heart defect.

The occurrence of murmur can be associated with the expansion of the tube in a limited area, and then when liquid passes at the same speed from the narrow part to the expanded part, vortex movements arise, which create conditions for murmur to occur. Proof of this can be the formation of murmur over the aneurysm of the aorta and other vessels.

Murmur can occur when the speed of fluid flow changes; the higher the speed, the stronger the murmur and vice versa. With a sharp decrease in the speed of blood flow, murmur may not form, despite the presence of a narrowing of the tube lumen.

Based on the time of appearance of the murmur, depending on the phase of the heart, systolic and diastolic murmurs are distinguished. During auscultation it is necessary to determine:

– the ratio of murmur to the phase of cardiac activity (ventricular systole or diastole);
– properties of murmur, its nature, strength, duration;
– localization of murmur (place of best listening);
– direction of murmur transmission (irradiation).

Systolic murmur occurs during systole, when blood moves from one part of the heart to another or from the ventricles to large vessels (fig. 22). Systolic murmur is heard with stenosis of the aortic mouth or pulmonary trunk, since with these defects, during the expulsion of blood from the ventricles, obstruction in the form

of a narrowing of the vessel. Systolic murmur occurs when the mitral and tricuspid valves are insufficient, as a result of the fact that blood from the ventricles flows not only into the aorta and pulmonary trunk, but also back into the atria through incompletely covered atrioventricular openings.

Systolic murmur appears after a long pause, along with the first sound, coincides with the apical impulse and the pulse of the carotid artery, sometimes the first sound may be weakened or even absent.

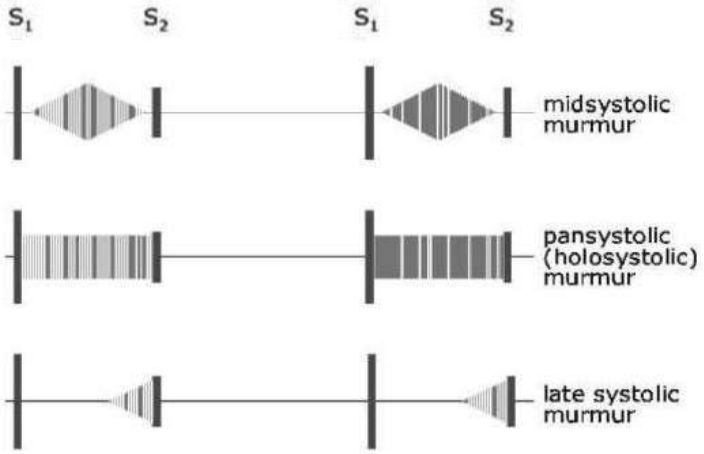


Fig. 22. Main types of systolic murmurs

Diastolic murmur is heard when there is narrowing of the left or right atrioventricular orifice, since during diastole there is a narrowing in the path of blood movement from the atria to the ventricles (fig. 23). When the left atrioventricular orifice is narrowed, the murmur occurs in the middle of diastole (mesodiastolic) or at its end (presystolic).

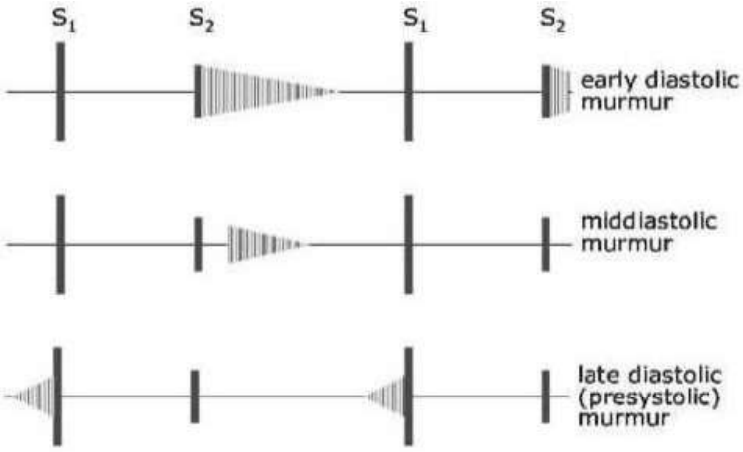


Fig. 23. Main types of diastolic murmurs

Diastolic murmur is detected during a long pause between the second and first sounds and the time of its appearance does not coincide with the apex beat. Diastolic murmur also occurs with aortic valve insufficiency in protodiastole and gradually decreases towards its end.

The nature of the murmurs can be: soft, blowing, rough, scraping, sawing. With organic damage to the valvular apparatus of the heart, the murmur is usually rough, but sometimes it can be soft. The murmur can be musical with shortening of the papillary muscles, perforation and other lesions of the valve leaflets. The strength of the murmur depends on the degree of narrowing of the hole and on the speed of blood flow through it. Diastolic murmur with mitral stenosis is not strong, since it occurs during contraction of the left atrium — the weakest part of the heart. The systolic murmur during aortic stenosis is sharp and strong, as it is formed during the period of blood expulsion by the hypertrophied left ventricle, which sharply accelerates the blood flow through the narrowed opening.

Best places to listen to heart murmurs. In order to distinguish murmurs caused by damage to various valves, it is necessary to clearly understand the places where the heart valve apparatus can be heard. If, when listening to the heart at 5 points, a systolic murmur is heard with the epicenter at the apex of the heart, then this indicates mitral valve insufficiency. In case of tricuspid valve insufficiency, the systolic murmur is best heard at the fourth point of auscultation — the lower third of the sternum at the xiphoid process. Systolic murmur in the second intercostal space to the right of the sternum or at the Botkin-Erb point indicates a narrowing of the aortic mouth or pathological processes in the aorta itself (atherosclerosis, syphilitic aortitis, aortic aneurysm or narrowing of the aortic isthmus). With pulmonary stenosis, a systolic murmur is heard in the second intercostal space to the left of the sternum.

Diastolic murmur with an epicenter at the apex indicates stenosis of the left atrioventricular orifice. The same murmur in the second intercostal space on the right or at the fifth point of auscultation is detected with aortic valve insufficiency. With pulmonary valve insufficiency, diastolic murmur is heard in the second intercostal space to the left of the sternum, and with narrowing of the right atrioventricular orifice — in the lower third of the sternum (fourth point of auscultation).

In case of valvular heart defects, in addition to differences in the properties of murmur, one should pay attention to heart sounds, which can change and are of great importance in assessing the degree of damage to the heart valves.

Conductivity of murmur. With valvular lesions, murmurs are usually carried out along the blood flow from the place where they arise, or through the dense myocardial muscle during its contraction. With stenosis of the aortic mouth, systolic murmur propagates through the bloodstream and can be heard over the carotid and subclavian arteries, over the area of the clavicle and thoracic spine. In case of mitral valve insufficiency, the systolic murmur is initially conducted upward

towards the third intercostal space on the left (left atrium), and in case of decompensation of the defect and a sharp expansion of the left ventricle, it is heard in the left axillary region. In case of aortic valve insufficiency, the diastolic murmur is conducted along the blood flow down to the left ventricle and is best heard at the Botkin-Erb point. With mitral stenosis, a diastolic murmur is heard at the apex of the heart in a limited area and is not carried anywhere.

Depending on the patient's position, the sound characteristics of the murmur change (it can be amplified or heard weaker). Therefore, it is necessary to listen to patients with valve defects in various positions (vertical, horizontal, lying on the left side, with the torso tilted forward), sometimes after light physical exercise, which increases the speed of blood flow through the affected valves and helps identify the murmur.

Systolic murmurs with insufficiency of the mitral and tricuspid valves, as well as with narrowing of the aortic orifice and pulmonary trunk, are better heard in a horizontal position of the patient, since better conditions are created for accelerating blood flow through these openings.

Diastolic murmurs due to insufficiency of the aortic and pulmonary valves during auscultation are better heard in an upright position of the patient. With mitral stenosis, the diastolic murmur is better heard with the patient in the left lateral position.

PULSE EXAMINATION

The pulse is the vibration of the walls of the peripheral arteries that occurs simultaneously with the systole of the left ventricle. Blood, which is rhythmically pumped into the aorta by the left ventricle, creates fluctuations in blood pressure and leads to elastic stretching and collapse of the arterial walls. The pulse is examined by palpation, since the amplitude of vascular dilation is small and it is not possible to detect it with the eye. Palpation of the pulse is most often carried out on the radial artery (fig. 24), which is located superficially directly under the skin between the styloid process of the radius and the tendon of the internal radial muscle. The topographical features of the radial artery allow the vessel to be pressed against the bone, which makes it easier to determine the properties of the pulse. Determination and examination of the pulse can also be done in the carotid, temporal and other arteries.

Pulse examination on the radial artery is carried out simultaneously in both arms. The patient's left and right hands are grasped in the area of the wrist joint, respectively with the right and left hands, so that the first finger is located on the back of the forearm on the ulnar side, and the index and middle fingers feel the radial artery, slightly pressing it against the radius (fig. 24). The patient's arms are

at the level of the heart in a dorsiflexion position. Palpation of the pulse on both hands is carried out due to the fact that the pulse can be different in filling (pulsus differens) and in the speed of propagation of the pulse wave. Pulsus differens can be observed with mitral stenosis, when a sharply enlarged left atrium compresses the left subclavian artery, Popov-Savelyev's symptom. A different pulse can be detected when there is a narrowing of the lumen or an anomaly in the location of one of the radial, brachial, or subclavian arteries, as well as when the subclavian artery is compressed by an aortic aneurysm, tumor, or enlarged lymph nodes.

If there is no difference in pulse, the study is then carried out on one arm.

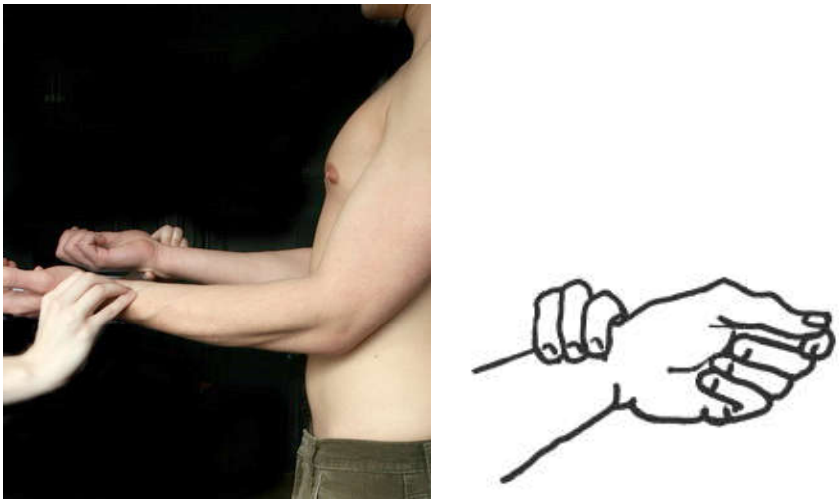


Fig. 24. Method of determining pulse

Pulse properties

Pulse rate. Normally, the pulse rate is 60–90 beats per minute, it is rhythmic, that is, pulse waves follow each other at regular intervals. The rhythmic pulse is counted for 15–30 seconds, the result obtained is multiplied by 4 or doubled. With an arrhythmic pulse, the pulse waves are counted for a minute and at the same time the number of heart contractions is counted by the apex beat of the heart. This is done in order to identify pulse deficiency (pulsus deficiens), when the number of heart contractions exceeds the number of pulse waves.

Based on frequency, there are frequent pulses (pulsus frequens) and rare pulses (pulsus rarus). A frequent pulse, more than 90 beats per minute, occurs with organic lesions of the heart or with impaired innervation of the heart. An example of diseases in which a rapid pulse is observed are thyrotoxicosis, circulatory failure, heavy bleeding, etc., and an increase in heart rate is possible during physical activity, with excitement and an increase in body temperature. When body temperature rises by 1 degree, the pulse increases by 8 to 10 beats. A rare pulse, less than 60 beats per minute, occurs when the innervation of the heart by the vagus

nerve predominates, as well as when the functional ability of the sinus node to produce impulses decreases. A rare pulse can occur in completely healthy vagotonic people and well-trained athletes. A decrease in pulse rate is observed with myxedema, overdose of digitalis drugs, brain diseases, with a critical decrease in temperature, in high-fever patients, with complete transverse heart block.

Pulse rhythm. This characteristic of the pulse is determined by the work of the left ventricle of the heart. The pulse can be regular (pulsus regularis), pulse waves follow each other at equal intervals, and irregular, arrhythmic (pulsus irregularis), the time intervals between pulse waves are different. By palpation, several types of arrhythmic pulse can be detected. With atrial fibrillation, contraction of the left ventricle occurs randomly, and pulse waves follow each other at various intervals. With extrasystole there will be additional pulse waves followed by a compensatory pause. Respiratory arrhythmia is accompanied by an increase in heart rate during inhalation and a slowdown during exhalation. With adhesive as well as effusion pericarditis, during inspiration there may be a complete disappearance of pulse waves; such a pulse is called paradoxical (pulsus paradoxus, pulsus respiratione intermittens).

Pulse voltage. This property of the pulse is determined by the force with which the artery must be pressed so that pulse waves are not detected below the point of compression. A hard, tense pulse (pulsus durus) will occur with aortic valve insufficiency, arterial hypertension and sclerotic changes in the vascular wall. A soft, easily compressible pulse (pulsus mollis) is detected when vascular sound decreases during bleeding, decreased blood pressure, collapse and shock.

Pulse filling. This value is determined by the amount of blood ejected into the aorta by the left ventricle. By squeezing the radial artery with different forces with your fingers, you get a feeling of the volume of its filling during systole and diastole of the left ventricle of the heart. In terms of filling, the pulse can be full (pulsus plenus) or empty (pulsus vacuus, pulsus inanis). In healthy people, as well as during physical activity, the pulse is well filled and full. The pulse is poorly filled and empty during bleeding, collapse, or shock.

Pulse value. This characteristic of the pulse is associated with filling and tension. It depends on the degree of expansion of the artery during systole and on its collapse during diastole. The degree of expansion and collapse of the artery depends on the pulse value, blood pressure and the ability of the arterial wall to elastically expand. With an increase in stroke volume of blood, large fluctuations in pressure in the artery, and also with a decrease in the sound of the arterial wall, the magnitude of pulse waves increases. Such a pulse is called large (pulsus magnus). On a sphygmogram, a large pulse is characterized by a high amplitude of pulse oscillations, which is why it is called a high pulse (pulsus altus). When the filling and tension of the pulse is reduced, they speak of a small pulse (pulsus parvus). A barely palpable, small and soft pulse is called threadlike (pulsus filiformis).

In a healthy person, the pulse is rhythmic and the magnitude of the pulse waves is the same, the pulse is uniform (pulsus alqualis), with disturbances of the heart rhythm the pulse is uneven (pulsus inaequalis), sometimes with a rhythmic pulse an alternation of large and small pulse waves is determined — an intermittent pulse (pulsus alternans). The occurrence of an intermittent pulse is associated with the alternation of heart contractions of different strengths in severe myocardial damage.

Pulse shape. The shape of the pulse is determined by the rate of change in pressure in the arterial system during systole and diastole and is graphically presented on a sphygmogram (fig. 25).

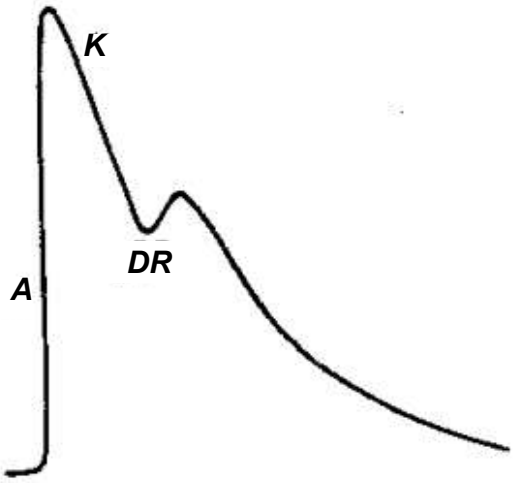


Fig. 25. Graphic recording of pulse fluctuations in blood pressure in the artery:
A — anacrotic; *K* — catacrota; *DR* — dicrotic rise

With aortic valve insufficiency, a large volume of blood is released into the aorta during systole and the pressure in it quickly increases, and in diastole, when some of the blood returns, it quickly falls, which leads to rapid expansion and collapse of the artery. Such a pulse is called fast (pulsus celer), or jumping (pulsus saliens). On a sphygmogram, a fast pulse is characterized by a steeper than normal rise in anacrotic and an equally sharp decrease in catacrotic. With aortic valve insufficiency, the pulse is not only fast, but also high and large (pulsus celer et altus, et magnus). When the aortic mouth is narrowed, the pulse is usually slow (pulsus tardus), associated with a slow increase in pressure in the arterial system and its small fluctuations during the cardiac cycle. The size of the pulse waves with this defect decreases, so the pulse will not only be slow, but also small and rare (pulsus tardus et parvus, et rarus).

In addition to the listed basic properties of the pulse, there may be other changes. Rarely during the period of a decrease in the pulse wave is a second additional wave detected. It is associated with an increase in the dicrotic wave, which is normally not palpable, but is determined only by the sphygmogram.

When the sound of the peripheral arteries decreases (fever, infectious diseases), the dicrotic wave increases and is determined by palpation. There is also a paradoxical pulse (pulsus paradoxus). A feature of this pulse is the decrease in pulse waves during inhalation. Paradoxical pulse is observed when the pericardial layers fusion due to compression of large veins and a decrease in blood supply to the heart during inspiration.

Features of the vascular wall. Features of the vascular wall are determined by sliding movements of the fingers along the vessel. An increase in the sound of the vascular wall is observed with arterial hypertension, and thickening of the artery wall is observed with atherosclerosis. A decrease in the sound of the vascular wall is detected during collapse, sepsis, and infectious diseases.

TEST CONTROL

1. Specify the main complaints of diseases of the cardiovascular system:

a) cough and abdominal pain;
b) cough and palpitations;
c) rashes on the lower extremities;
d) shortness of breath and chest pain, swelling;
e) cough and chest pain.

2. Specify the mechanism of cough in heart defects:

a) congestion in the small circle of blood circulation;
b) congestion in the large circle of blood circulation;
c) infection of the upper respiratory tract;
d) decreased contractile function of the myocardium;
e) increased cardiac output.

3. Specify the mechanism of shortness of breath in heart defects:

a) stagnation in the small circle of blood circulation;
b) stagnation in the large circle of blood circulation;
c) infection of the upper respiratory tract;
d) decreased contractile function of the myocardium;
e) increased cardiac output.

4. Specify the mechanism of occurrence of edema in heart diseases:

a) stagnant phenomena in the small circle of blood circulation;
b) stagnant phenomena in the large circle of blood circulation;
c) infection of the upper respiratory tract;
d) decreased contractile function of the myocardium;
e) increased cardiac output.

5. Specify the mechanism of pain in angina pectoris:

a) congestion in the small circle of blood circulation;
b) inflammatory process in the coronary arteries;
c) atherosclerosis of the coronary arteries;
d) decreased contractile function of the myocardium;
e) increased cardiac output.

6. The impact of the tip of the left ventricle on the chest wall is called a _____ push .

7. Periodic jerky vibrations of the artery wall caused by the release of blood from the heart during its contraction are called_____ the pulse.

8. The difference between systolic and diastolic pressure is called _____ pressure.

9. The «cat purring» at the top of the heart is called _____ trembling.

10. «Cat purring» over the aorta of the heart is called _____ trembling.

11. With percussion, the diameter of the relative dullness of the heart is determined:

a) in the 2nd intercostal space on the left and right;
b) in the 3rd intercostal space to the right and left of the sternum;
c) in the 4th intercostal space on the right and 5th intercostal space on the left of the sternum;
d) in the 4th intercostal space to the right and left of the sternum;
e) in the 5th intercostal space to the right and left of the sternum.

12. The vascular bundle is percutorially determined:

a) in the 2nd intercostal space to the right and left of the sternum;
b) in the 3rd intercostal space to the right and left of the sternum;
c) in the 5th intercostal space to the right and left of the sternum;
d) in the 4th intercostal space to the right and left of the sternum;
e) only in the 2nd intercostal space to the right of the sternum.

13. Specify the correct sequence for determining the boundaries of relative cardiac dullness:

a) right, left, upper;
b) upper, left, right;
c) left, upper, right;
d) right, upper, left;
e) left, upper, right.

14. In a healthy person, the right border of relative cardiac dullness is percutorially determined:

a) 1 cm to the right of the anterior median line in the 4th intercostal;
b) 3–4 cm to the right of the anterior median line in the 4th intercostal;
c) 2–3 cm to the right of the right edge of the chest.

15. In a healthy person, the left border of relative cardiac dullness is percutorially determined:

a) 1 cm to the right of the anterior median line in the 4th intercostal;
b) 8–9 cm to the left of the anterior median line in the 5th intercostal;
c) 2–3 cm to the right of the right edge of the sternum in the 4th intercostal;
d) 1–1.5 cm to the right from the right edge of the sternum in the 3rd intercostal;
e) 8–9 cm to the left of the anterior median line in the 4th intercostal space.

16. The systolic sound of the heart, consisting of 4 components, is called:

a) the first; c) the third;
b) the second; d) the fourth.

17. The diastolic sound of the heart, consisting of 2 components, is called:

a) the first; c) the third;
b) the second; d) the fourth.

18. What sounds does the protodiastolic gallop consist:

a) 1, 2, 3; c) 1, 2, 4;
b) 1, 2; d) 1, 2, 3, 4.

19. What sounds does the presystolic gallop consist:

a) 1, 2, 3; b) 1, 2;
c) 1, 2, 4; d) 1, 2, 3, 4.

20. What are the auscultative signs of systolic murmur

a) is listened to in the diastole of the cardiac cycle;
b) the time coincides with the pulse on the radial artery;
c) it is listened to in the systole and coincides with the pulse on the carotid artery;
d) it is listened to in the systole and does not coincide with the pulse on the carotid artery;
e) it is listened to in the diastole and does not coincide with the pulse on the carotid artery.

The answers: 1 — d; **2** — a; **3** — a; **4** — b; **5** — b; **6** — apical; **7** — arterial; **8** — pulse; **9** — diastolic; **10** — systolic; **11** — c; **12** — a; **13** — a; **14** — b; **15** — b; **16** — a; **17** — b; **18** — a; **19** — a; **20** — c.

REFERENCES

Basic

1. *Пронько, Т. П.* Пропедевтика внутренних болезней = Propedeutics of internal diseases : учеб. пособие. – Минск : Адукацыя і выхаванне, 2020. – 472 с.

2. *Kovalyova, O. M.* Propedeutics of internal medicine. Part 1. Diagnostics : textbook for English learning students of higher medical schools. – Vinnytsia : Nova Knyha, 2017. – 424 p.

3. *Kovalyova, O. M.* Propedeutics of internal medicine. Part 2. Syndromes and diseases : textbook for English learning students of higher medical schools. – Vinnytsia : Nova Knyha, 2017. – 264 p.

Additional

4. *Bickley, Lynn S.* Bates' guide to physical examination & history taking : guest ed. R. M. Hoffman. – Philadelphia [etc.] : Wolters Kluwer, 2017. – 1034 p.

5. *Немцов, Л. М.* Общая пропедевтика внутренних болезней : курс лекций (на английском языке) / Л. М. Немцов. – 2-е изд. – Витебск : ВГМУ, 2016. – 175 с.

6. *Special* propedeutics of internal diseases : lecture course / Vitebsk State Medical University, Dep. of Propedeutics of Internal Diseases ; comp. by L. M. Nemtsov. – 2nd ed. – Vitebsk : VSMU, 2016. – 318 p.

7. *Арсентьева, И. Л.* Учебная история болезни = Educational case history : практикум для студентов по специальности «Лечебное дело» / И. Л. Арсентьева, Э. А. Доценко. – Минск : БГМУ, 2021. – 40 с.

8. *Пропедевтика* внутренних болезней = Propaedeutics of Internal Diseases : контрольные вопросы к текущим и итоговым занятиям / И. Л. Арсентьева [и др.]. – Минск : БГМУ, 2018. – 40 с.

9. *Физикальные* методы исследования = Physical examination methods : практикум / Э. А. Доценко [и др.]. – Минск : БГМУ, 2021. – 104 с.

10. *Дополнительные* методы исследования в клинике внутренних болезней : практикум = Diagnostic methods in the internal medicine : workbook : учеб.-метод. пособие / Э. А. Доценко [и др.]. – 2-е изд., испр. – Минск : БГМУ, 2022. – 159 с.

CONTENT

Учебное издание

Хващевская Галина Михайловна
Шолкова Мария Владимировна
Новикова Татьяна Петровна и др.

ОБСЛЕДОВАНИЕ ПАЦИЕНТОВ ПРИ ЗАБОЛЕВАНИЯХ СИСТЕМЫ КРОВООБРАЩЕНИЯ

CARDIOVASCULAR EXAMINATION

Учебно-методическое пособие

На английском языке

Ответственный за выпуск Э. А. Доценко
Компьютерная вёрстка А. В. Янушкевич

Подписано в печать 02.04.25. Формат 60×84/16. Бумага писчая «PROJECTA Special».
Ризография. Гарнитура «Times».
Усл. печ. л. 2,79. Уч.-изд. л. 2,64. Тираж 70 экз. Заказ 223.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 24.11.2023.
Ул. Ленинградская, 6, 220006, Минск.