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Federal State Budgetary Educational Institution of Higher Education "North Ossetian State Medical Academy" of the Ministry of Health of the Russian Federation

(FGBOU VO SOGMA of the Ministry of Health of the Russian Federation)

Department of Internal Medicine No. 1

METHODOLOGICAL MATERIALS on Propaedeutics of internal diseases

FUNDAMENTALS OF SEMIOTICS OF DISEASES OF THE CIRCULATORY SYSTEM

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Methodological materials are intended for teaching students of 2-3 courses (4-6 semesters) of the Medical Faculty of the Federal State Budgetary Educational Institution of the Ministry of Health of the Russian Federation the discipline " Propaedeutics of internal diseases»

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INTRODUCTION

The textbook "Fundamentals of semiotics of diseases of the circulatory system" offers material that allows you to master the basic principles of conducting a clinical examination of patients with diseases of the cardiovascular system,including the subjective part: collecting passport data, complaints with their details, anamnesis of the disease and life. Elucidation of risk factors that play a significant role in the development of cardiovascular pathology. The symptoms characteristic of cardiovascular pathology, which can be detected during a general examination of the patient, are described in detail. The technique and methodology of palpation, percussion, auscultation of the heart, pulse research, as well as the clinical interpretation of the data obtained in normal and in various pathological processes are explained.

The manual contributes to the formation of students ' professional competencies (skills) for conducting questioning, general examination, chest examination, palpation, percussion and auscultation of the heart, pulse research in accordance with the requirements of the third generation of the Federal State Educational Standard.

In the course of training, students under the guidance of a teacher need to have theoretical knowledge and practical skills, without which it is impossible to formulate the main clinical syndromes in cardiology, and in the future, to make a diagnosis.

For better assimilation, the manual is illustrated with a large amount of visual material. At the end of each topic, the student is asked to answer questions and test tasks to conduct self-control of the learned material.

Test tasks.

1. The presence of paroxysmal pain in the heart area associated with physical activity is characteristic of:

a) angina pectoris;

b) dry pleurisy;

c) myocarditis;

d) pericarditis;

e) mitral heart disease.

2. Patients with heart failure with stagnation in a large circle of blood circulation have pain:

a) Behind the sternum.

b) In the region of the apex of the heart.

c) In the right hypochondrium.

d) In the left hypochondrium

3. What is not a risk factor for atherosclerosis?

a) Lack of body weight.

b) Diabetes mellitus.

c) Hyperlipidemia.

d) Arterial hypertension.

e) Smoking.

4. A negative apical push is determined when:

a) Adhesive pericarditis.

b) Exudative pericarditis.

c) Myocarditis.

d) Endocarditis.

5. Fingers in the form of "drumsticks" are in patients with:

a) Prolonged septic endocarditis.

b) In some congenital heart defects.

c) In case of circulatory insufficiency.

d) Angina pectoris.

e) Hypertension.

6. In what disease of the cardiovascular system is characterized by pallor of the skin and mucous membranes:

a) Mitral defects.

b) Aortic malformations.

c) Angina pectoris.

d) Myocardial infarction.

e) Hypertension.

f) Heart failure.

1. The face of Corvisar is:

a) Puffy face, yellowish-pale with a cyanotic tinge, mouth constantly half-open, lips cyanotic, eyes close together and dull.

b) Hyperemia of the skin, bright eyes, excited expression.

c) With a lumpy-knotty thickening of the skin under the eyes and above the eyebrows and an enlarged nose.

d)An amymic person.

e) With an increase in prominent parts (nose, chin, cheekbones).

e) An intensely red, moon-shaped, glossy face with the development of a beard and mustache in women.

2. The most common hemoptysis observed in severe heart diseases is observed in patients with:

a) Mitral heart disease.

b) Aortic heart disease.

c) Coronary heart disease.

d) Hypertension.

3. Peripheral cardiac cyanosis is characterized by:

a) diffuse character, gray tint, "warm" cyanosis;

b) distal localization (acrocyanosis), " cold cyanosis;

c) localization in the face, neck;

d) the most frequent localization on the feet, hands and shins;

e) combination with signs of lung diseases.

4. What conditions can cause chest pain?

a) Pleural lesion.

b) Pathology of the chest itself — intercostal neuralgia.

c) Violation of the integrity of the bone base (fractures, tumor metastases).

d) Diseases of the hematopoietic organs (leukemia).

e) Diseases of other organs (heart).

Topic 2: Palpation, percussion of the heart. Defining the boundaries of relative and absolute cardiac dullness.

THE PURPOSE OF THE LESSON: To teach students to methodically correctly conduct palpation and percussion of the heart. Teach the technique of palpation and percussion of the heart, the ability to interpret the results of the physical examination of the patient.

Tasks: teach

1. Perform palpation of the heart area.

2. Interpret the symptoms obtained by palpation of the heart.

3. Perform a percussion of the heart to determine the relative cardiac dullness.

4. Determine the absolute cardiac dullness, the diameter of the heart, the width of the vascular bundle, the contour of the heart.

5. Correctly interpret changes in the boundaries of relative and absolute cardiac dullness, the width of the vascular bundle.

The student should know:

1. How to perform palpation of the heart area, what symptoms can be determined in diseases of the cardiovascular system?

2. How to find the limits of relative cardiac dullness and what they correspond to in the norm.

3. How to interpret changes in the boundaries of relative cardiac dullness in pathology, and what diseases of the cardiovascular system correspond to these changes.

4. How to find the limits of absolute cardiac dullness and what they correspond to in the norm.

5. How to interpret changes in the boundaries of absolute cardiac dullness in pathology, and what diseases of the cardiovascular system and other systems correspond to these changes.

6. How to find the boundaries of the diameter of the heart, the width of the vascular bundle and what they correspond to in the norm.

7. How to interpret changes in the diameter of the heart, the width of the vascular bundle in pathology, and what diseases of the cardiovascular system correspond to these changes.

The student must be able to:

1. Palpate the heart area.

2. Percutorily determine the boundaries of relative and absolute cardiac dullness.

3. Percutorily determine the boundaries, the diameter of the heart, the width of the vascular bundle, the configuration of the heart.

2. Interpret the data obtained during palpation and percussion of the heart.

3.Make a fragment of the medical history.

The student must own:

1. The technique of palpation of the heart area.

2. The technique of quiet and quiet percussion to determine the boundaries of relative and absolute heart dullness.

3. Percussion technique for determining the diameter of the heart, the width of the vascular bundle.

4. The skills of determining the configuration of the heart.

Questions for monitoring the initial level of knowledge:

1. What structures form the left contour of the heart?

2. What structures form the right contour of the heart?

3. Who first proposed heart percussion?

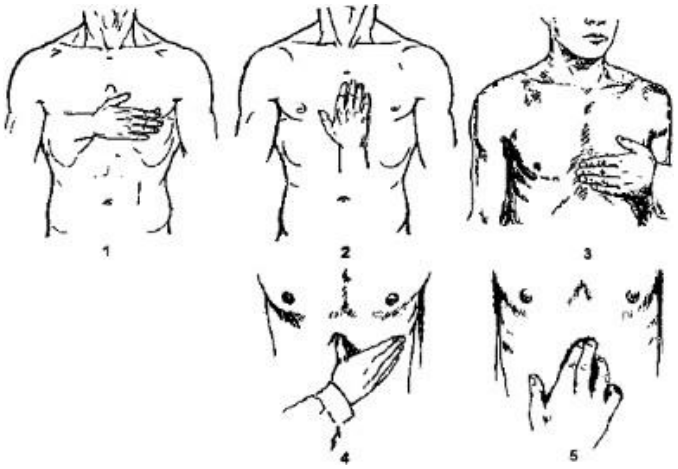
4. What laws of physics are used in conducting percussion?

5. Heart percussion technique?

INFORMATION BLOCK.

Palpation.

Palpation of the heart area allows you to more accurately characterize the apical push, to identify the presence of a heart push, to clarify the visible pulsation or detect them, to identify the tremor of the chest - a symptom of "cat purring" (Fig.1).



To determine the apical push, place the palm of the right hand on the chest of the subject (in women, the left breast is first drawn up and to the right) with the base of the hand to the sternum, and with the fingers to the axillary region, between the IV and VII ribs. Then the" pads " of the end phalanges of the three bent fingers, placed perpendicular to the surface of the chest, specify the place of the push, moving them along the intercostals from outside to inside to the place where the fingers, when pressed with moderate force, begin to feel the lifting movements of the apex of the heart (Fig. 2).

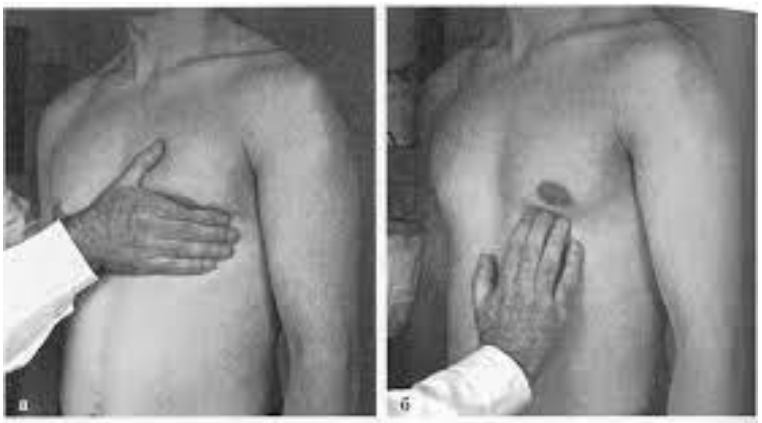


Рис. 3.50. Положение руки врача при пальпации верхушечного толчка: а – ориентировочное выявление пульсации; б – определение локализации, силы и площади верхушечного толчка

In the norm, the pericarp is located in the fifth intercostal space, 1-1. 5 cm inside of the left mid-clavicular line. When the patient is on the left side, the push is shifted to the left by 3-4 cm, and on the right side-to the right by 1-1.5 cm.

Persistent displacements of the apical push may depend on changes in the heart itself or the surrounding organs (Figure 3): верх

the apical push shifts to the left to the axillary line and simultaneously down to the sixth and seventh intercostal spaces with an increase in the left ventricle;

the apical push also shifts to the left when the right ventricle expands, since the left ventricle is pushed to the left by the expanded right ventricle, but there is no downward shift;

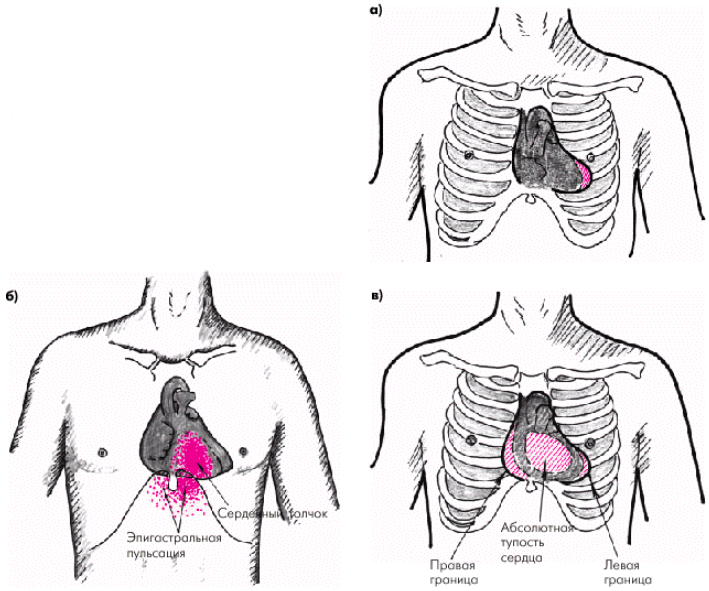
the apical push is shifted to the left in the presence of effusion or gas in the right pleural cavity;

the apical push is displaced when pleuropericardial adhesions and lung shrinkage due to the growth of connective tissue in them (pneumosclerosis) pull the heart in the affected direction;

the apical shock may disappear with left-sided exudative pleurisy or fluid accumulation in the pericardial cavity;

the apical push is displaced with the displacement of the heart itself by the volume formation in the lungs or mediastinum;

the apical push is also displaced, with the displacement of the heart by the raised diaphragm as a result of the accumulation of fluid or gas in the abdominal cavity



PIC. 3. Results of palpation and percussion of the heart in patients with CHF. a-displacement of the apical push and the left border of the heart with LV dilatation; b — increased and diffused cardiac push and epigastric pulsation with hypertrophy and dilatation of the pancreas; c — displacement of the right border of the heart with dilatation of the pancreas

Normally, the apical push may not be felt if it is closed by a rib. In the same cases, when the apical push is palpated, its following properties are determined (Fig. 4)

Width (or area). The width of the apical shock is the area of the part of the chest that is shaken by the impact of the apex of the heart; normally it is 1-2 cm2. If the width of the apical push is more than 2 cm2, then it is called spilled, if less-limited. The width of the apical push can also increase when the lower edge of the left lung is wrinkled, the heart is displaced anteriorly by a mediastinal tumor, etc. A decrease in the width of the apical push is observed in obesity, emphysema of the lungs, low standing of the diaphragm.

Remember! An increase in the area of the apical shock of more than 3 cm2 in diameter is a sign of significant dilatation of the left ventricle.

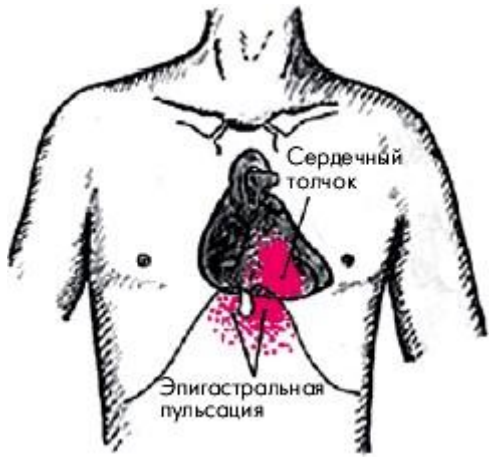
2. Height. The height of the apical push is the magnitude of the amplitude of the oscillation of the chest wall in the region of the apex of the heart. There is a high and low apical push. As the height increases, so does its width, and vice versa. The height of the apical push depends on the strength of the heart contraction. With physical exertion, excitement, fever, thyrotoxicosis, when the heart contractions increase, the height of the apical push increases.

3. The force of the apical push. It is measured by the pressure exerted by the tip of the heart on the palpating fingers. Like the first two properties, the force of the push depends on the thickness of the chest and the proximity of the top of the heart to the palpating fingers, but, to a greater extent, on the strength of the contraction of the left ventricle. An enhanced apical push is observed in left ventricular hypertrophy, and in concentric hypertrophy, the force of the push can increase even without increasing its width.

4. The resistance of the apical push, determined by palpation, allows you to get an idea of the density of the heart muscle itself. The density of the left ventricular muscle increases significantly with its hypertrophy, and then there is talk of a resistant apical push.

Remember! Left ventricular hypertrophy is characterized by a diffuse, high, amplified, resistant apical shock. With a sharp hypertrophy of the left ventricle, combined with its expansion, the tip of the heart becomes cone - shaped and is felt by the hand in the form of a dense elastic dome-a dome-shaped push.

The cardiac shock is detected to the left of the sternum, sometimes extending to the epigastric region (Fig. 5).



Remember! The cardiac shock is caused by hypertrophy and dilatation of the right ventricle.Normally, the heartbeat is not palpable. With severe emphysema of the lungs, it is not visible and is not palpable.

Other types of pulsation in the area of the heart and in the vicinity of it. In healthy people, aortic pulsation is not determined, with the rare exception of persons with asthenic physique, who have wide intercostal spaces.

Пуль Pulsation in the second intercostal space to the right of the sternum is detected when the ascending part of the aorta expands.

Pulsation in the jugular fossa is determined by an aneurysm or a significant expansion of the aortic arch (retrosternal or retrosternal pulsation).

Эп Epigastric pulsation, i.e. visible elevation and retraction of the epigastric region. Synchronous with the activity of the heart, epigastric pulsation may depend not only on the hypertrophy of the right ventricle, but also on the pulsation of the abdominal aorta and liver. Epigastric pulsation caused by right ventricular hypertrophy is usually detected under the xiphoid process and becomes more pronounced with deep inspiration, while pulsation caused by the abdominal aorta is localized slightly lower and becomes less pronounced with deep inspiration. Pulsation of the unchanged abdominal aorta is detected in emaciated patients with a relaxed abdominal wall.

Дрож Chest tremor, or the "cat purr" symptom, which is the feeling you get when stroking a purring cat, is of great importance for the diagnosis of heart defects. This symptom is due to the same reasons as the formation of noise in stenosis of the valve openings.

Remember! To detect the symptom of "cat purring", you need to put your hand flat on the points where it is customary to listen to the heart. "Cat purr", defined above the apex of the heart during diastole, is characteristic of mitral stenosis (diastolic, presystolic tremor), above the aorta during systole - for stenosis of the aortic mouth (systolic tremor).

Percussion.

The percussion method can determine the projection area of the heart and its individual chambers on the anterior chest wall, as well as the position and configuration of the heart and vascular bundle. When percussion occurs in the area of the heart covered by the lungs, a blunted percussive sound is formed. This zone is called the zone of relative dullness of the heart. When percussion is performed over a part of the heart that is not covered by the lungs, an absolutely different sound is detected. This zone is called the zone of absolute dullness of the heart .

The right contour of the relative dullness of the heart and vascular bundle is formed from above by the superior vena cava (up to the upper edge of the III rib), from below by the right atrium;

The left contour is formed by the left part of the aortic arch, the pulmonary trunk, at the level of the third rib - the ear of the left atrium, and from below—a narrow band of the left ventricle.

The anterior surface of the heart is formed by the right ventricle.

Remember! The relative dullness of the heart is the projection of its anterior surface on the chest and corresponds to the true boundaries of the heart, the absolute-the anterior surface of the heart, not covered by the lungs.

Percussion can be performed in the horizontal and vertical positions of the patient: it should be taken into account that the size of the cardiac dullness in the vertical position is less than in the horizontal position. This is due to the mobility of the heart and the displacement of the diaphragm when changing position (Fig. 6).

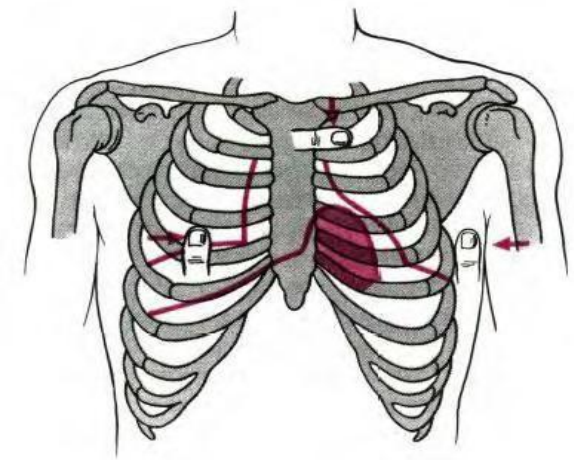


Рис. 40. Положение пальца-плессиметра при определении правой, левой и верхней границ относительной сердечной тупости.

When determining the boundaries of relative dullness, you need to percute along the intercostals. The percussion stroke should be of medium strength – the percussion technique is quiet. It is necessary to ensure that the plessimeter finger is firmly pressed against the chest wall (to achieve a deeper spread of strokes and a clearer definition of the boundary between relative and absolute cardiac dullness) (Fig.7).

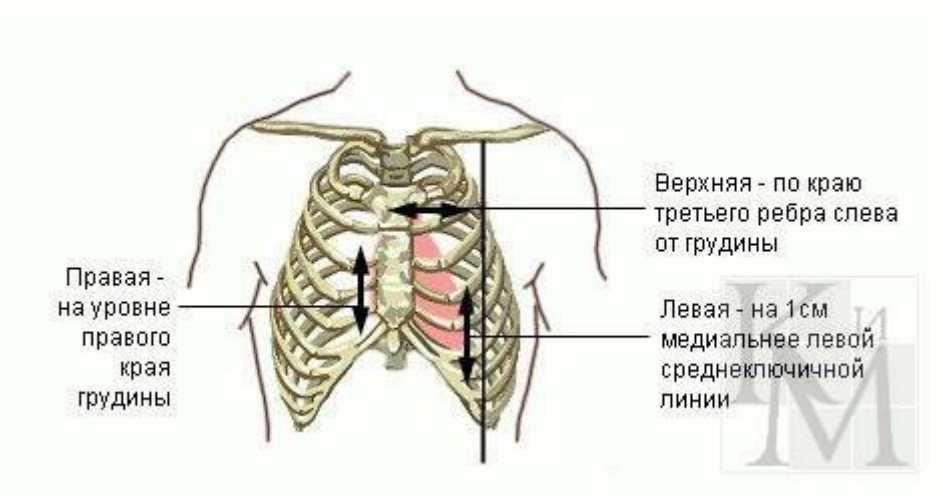


Fig. 7 Limits of relative cardiac dullness.

To find the right border of relative cardiac dullness, it is necessary: first, to determine the lower border of the right lung by the mid-clavicular line, which is normally located at the level of the VI rib (the position of the lower border of the lung gives an idea of the level of standing of the diaphragm). They are lifted into the fifth intercostal space, then the plessimeter finger is transferred to one intercostal space above the lower border of the right lung and placed parallel to the determined right border of the heart (normally in the fourth intercostal space). Percute by gradually moving the plessimeter finger along the intercostal space, towards the sternum, until a blunted percussive sound appears. Mark the right border of the relative dullness of the heart along the outer edge of the finger, facing the clearer percussion sound. Normally, the right border of relative cardiac dullness is located 1 cm outside of the right edge of the sternum or along the edge of the sternum.

To find the left border of relative cardiac dullness, it is necessary: first, palpationally find the apical push, then the finger-plessimeter is placed outside of it parallel to the desired border and percutated along the V intercostal space towards the sternum. If the apical push cannot be determined (which may be normal), percussion should be performed in the fifth intercostal space from the anterior axillary line towards the sternum. The left border of the relative cardiac dullness of the heart is located 1-2 cm inside of the left midclavicular line and coincides with the apical push.

To find the upper limit of relative cardiac dullness, it is necessary to: step 1 cm to the left of the left sternal line. To do this, the plessimeter finger is placed perpendicular to the sternum near its left edge and moved downwards until the percussion sound is dulled. Normally, the upper limit of relative cardiac dullness is located on the III rib. (Fig. 40)

To find the border of the vascular bundle, it is necessary to: percute to the right and left along the second intercostal space from the mid-clavicular line towards the sternum when the percussion sound is dulled.

a mark on the outer edge of the finger.Normally, the width of the vascular bundle is equal to the width of the sternum: its diameter is 5-6 cm (Fig. 8).

Having established the boundaries of the relative dullness of the heart, a centimeter tape can measure the diameter of the heart, for this purpose, determine the distance from the extreme points of the boundaries of the relative dullness to the anterior median line. Normally, the distance from the right border of relative dullness, usually located in the fourth intercostal space, to the anterior median line is 3-4 cm, and the distance from the left border of relative dullness of the heart, usually located in the fifth intercostal space, to the same line is 8-9 cm. These values in total form the diameter of the relative dullness of the heart, normally it is 11-13 cm.

An idea of the configuration of the heart can be obtained by determining percutorily the boundaries of the vascular bundle in the second intercostal space on the right and left, and the relative dullness of the heart in the fourth and third intercostal spaces on the right and in the fifth, fourth and third intercostal spaces on the left. To do this, the plessimeter finger is moved parallel to the boundaries of the expected dullness and the points are marked on the patient's skin (the boundary of the emerging dullness of the percussion sound). Connecting these points, mark the contours of the relative dullness of the heart. Normally, there is an obtuse angle along the left contour of the heart between the vascular bundle and the left ventricle. In these cases, they talk about the normal configuration of the heart (Fig. 9, 10)

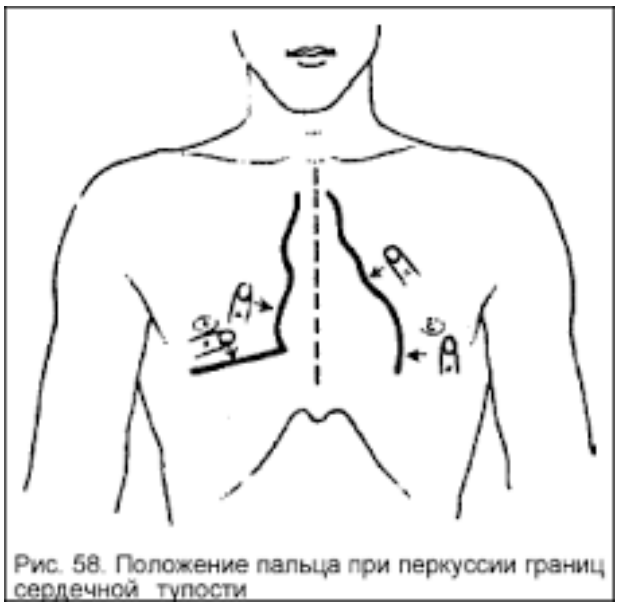
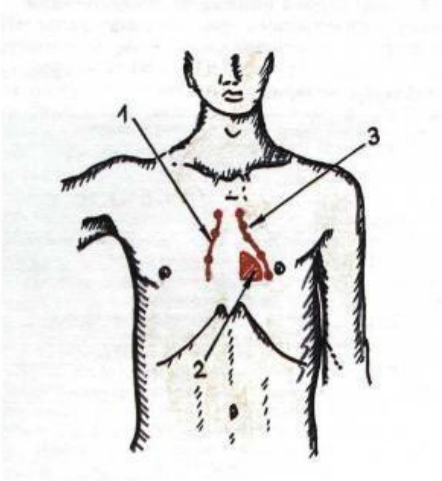


Рис. 58. Положение пальца при перкуссии границ сердечной тупости

Нормальная конфигурация сердца.



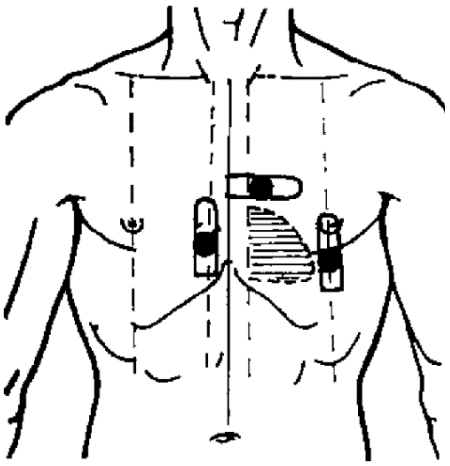
1 — контуры относительной тупости;
2 — абсолютная тупость;
3 — талия сердца.

- Когда угол между сосудистым пучком и левым желудочком тупой
- Этот угол называется сердечной талией

In pathological conditions, with the expansion of the heart, the mitral and aortic configurations are distinguished.

Determining the limits of absolute dullness of the heart.

The anterior wall of the heart, uncovered by the lungs, corresponds to the area of its absolute dullness. Therefore, when percussion of this part of the heart is marked by a dull sound. To determine the absolute dullness of the heart, the quietest percussion is used (Fig. 11)



To find the right border of absolute cardiac dullness, it is necessary: place the plessimeter finger along the right border of the relative dullness of the heart parallel to the sternum and continue percussion, moving it inside in the direction of the left edge of the sternum, until a dull sound appears. The border is marked on the outer edge of the finger, facing a clearer (blunted) sound, normally it passes along the left edge of the sternum.

To find the left border of absolute cardiac dullness, it is necessary to: place the plessimeter finger slightly outside of the border of relative dullness and percute in the direction of the sternum until a dull sound appears. The left border of absolute dullness is normally located 1-2 cm inside of the border of relative dullness of the heart.

To find the upper limit of absolute cardiac dullness, it is necessary to: place the plessimeter on the upper limit of the relative dullness of the heart and percussion, moving it down until a dull sound appears. The upper limit of absolute dullness of the heart is normally located on the IV rib.

Changes in the boundaries of the dullness of the heart.

Changes in the boundaries of relative cardiac dullness can be caused by non-cardiac causes. Thus, when the diaphragm is high, the heart takes a horizontal position, which leads to an increase in its transverse dimensions. When standing low, the diaphragm occupies a vertical position and, accordingly, its vertical size becomes smaller. The accumulation of fluid or air in one of the pleural cavities leads to a shift of the boundaries of the dullness of the heart to the healthy side, with atelectasis or lung shrinkage, pleuropericardial adhesions-to the painful side. The area of absolute dullness of the heart abruptly decreases or disappears with emphysema of the lungs, while with wrinkling of the lungs it increases. The area of absolute dullness also increases when the heart is displaced anteriorly, for example, by a mediastinal tumor, when fluid accumulates in the pericardium, and when the right ventricle is dilated. In the case of dilatation of the heart cavities, the boundaries of relative dullness are shifted.

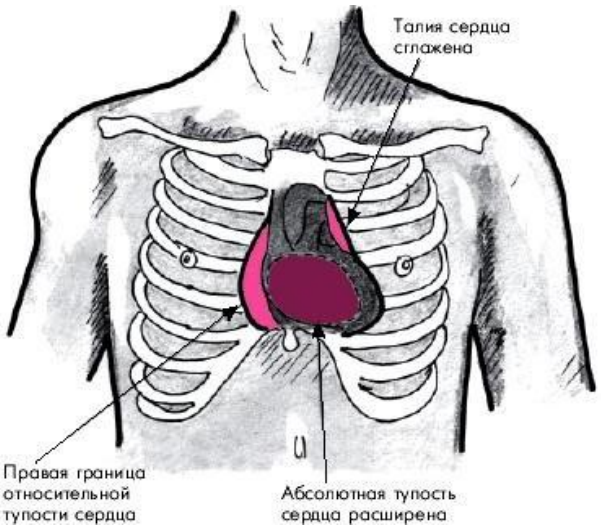
Remember!

The shift of the boundaries of relative dullness to the right is caused by the expansion of the right atrium and right ventricle. For example, in mitral stenosis (Figure 12).

 With an increase in the left atrium, the cone of the pulmonary trunk, the relative dullness shifts upward.

The shift of the border of relative dullness to the left occurs when the left ventricle dilates. For example, in the case of aortic valve insufficiency (Fig. 13

A sharply enlarged and hypertrophied right ventricle, pushing the left, can also shift the border of relative dullness of the heart to the left.



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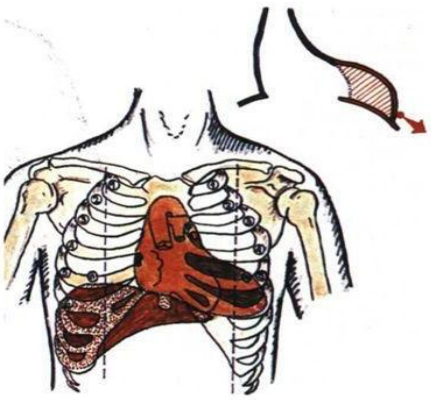


- Изменение границ сердца влево (ЛЖ) аортальная конфигурация сердца.

The expansion of the aorta leads to an increase in the diameter of the relative dullness in the second intercostal space and the heart takes on an aortic configuration.

CONTROL QUESTIONS:

1. How to perform palpation of the heart area?

2. What can be determined by palpation of the heart area?

3.What is determined by percussion?

4. Technique for determining the relative dullness of the heart.

5. How to determine the absolute dullness of the heart?

6. How to determine the boundaries of the vascular bundle, the diameter of the heart?

7. Why can the boundaries of relative cardiac dullness change?

8. How and in what pathological processes do the boundaries of absolute cardiac dullness change?

Test tasks.

1. The diameter of relative cardiac dullness is:

a) The distance from the right border of relative cardiac dullness of the lobar border of relative cardiac dullness.

b) The distance from the right border of relative cardiac dullness to the middle of the chest and from the middle of the sternum to the left border of relative cardiac dullness.c) the distance from the upper limit of relative cardiac dullness to the lobar limit of relative cardiac dullness.

2. The mitral configuration of the heart is characterized by:

a) a pronounced cardiac waist;

b) smoothed heart waist;

c) constant heart waist.

3. The left contour of the heart is formed by:

a) the aorta, the left atrium, the left ventricle;

b) the trunk of the pulmonary artery, the ear of the left atrium, the left ventricle;

c) aorta, inferior vena cava, left ventricle;

d) the trunk of the pulmonary artery, the right atrium, the right ventricle.

4. The vascular bundle is defined:

a) in the 2nd intercostal space;

b) at the level of 2 edges;

c) at the level of 3 edges;

d) in the 3rd intercostal space.

5. Absolute cardiac dullness is formed:

a) the left atrium;

b) the right atrium;

c) the left ventricle;

d) the right ventricle.

6. An increase in the upper limit of relative cardiac dullness is observed when:

a) left ventricular hypertrophy;

b) hypertrophy of the left atrium;

c) hypertrophy of the right atrium and right ventricle;

d) hypertrophy of the right atrium.

7. The left border of relative cardiac dullness is:

a) 1 cm from the left edge of the sternum;

b) 1.5-2 cm inside of the left mid-clavicular line;

c) along the left mid-clavicular line;

d) 1-1. 5 cm outside of the left mid-clavicular line.

8. The heartbeat is caused by contractions:

a) The left ventricle.

b) The right ventricle.

c) The left atrium.

d)The right atrium.

9. The place of projection of the tricuspid valve on the anterior chest wall is located:

but)To the left of the sternum in the attachment area of 111 ribs.

b) In the middle of the distance between the place of attachment to the sternum of the cartilage 111 of the rib on the left and the cartilage at the rib on the right.

c) In the second intercostal space to the left of the sternum.

d) In the middle of the sternum at the level of the third costal cartilage.

10. Choose the most correct interpretation of the palpation data - a spilled high (dome-shaped) apical push in the VI intercostal space 2 cm outwards from the midclavicular line:

1. hypertrophy of the left ventricle without its pronounced dilatation;

2. left ventricular hypertrophy and dilatation;

3. hypertrophy and dilatation of the right ventricle;

4. fusion of the pericardial leaves (adhesive pericarditis);

5. postinfarction aneurysm of the anterior wall of the left ventricle

Topic 3: Auscultation of the heart. Basic heart tones, heart noises. Heart tones and noises.

THE PURPOSE OF THE LESSON: To teach students how to methodically correctly perform auscultation of the heart, to evaluate the heart tones at the points of auscultation. Acquire the skill of detecting heart murmurs and their interpretation.

Tasks: teach

1. Perform auscultation of the heart.

2. Distinguish the first tone from the second at the points of auscultation.

3. Be able to interpret changes in heart tones.

4. Determine auscultatively heart murmurs.

5. Be able to interpret noises at various points of auscultation.

6. Evaluate the melody of the heart.

The student should know:

1. Rules of auscultation of the heart.

2. The procedure for listening to auscultation points.

3. Listening points and the true projection of the valves on the chest.

4. Auscultative characteristics of heart tones in a healthy person. The mechanism of occurrence of the four heart tones.

5. The reasons for the strengthening and weakening of the I and I heart tones.

6. Additional heart tones. The concept of the rhythm of the "gallop" and the rhythm of the "quail".

7. Classification of heart murmurs.

8. Causes and conditions of functional heart murmurs.

9. Causes and conditions of organic noises.

10. Distinguishing features of functional and organic noise.

11. Interpretation of the detected changes in the auscultation of the heart.

The student must be able to:

1. Perform auscultation of the heart.

2. Distinguish the heart tones at the auscultation points.

3. Identify changes in heart tones.

4. Determine additional heart tones, in pathological processes.

5. Detect heart murmurs.

6. Identify three-part rhythms.

7. Characterize the listened heart murmurs.

8. Interpret the data obtained during auscultation.

9.Make a fragment of the medical history.

The student must own:

1. The technique of auscultation of the heart.

2. The method of determining the I and II heart tones at the auscultation points.

3. The method of determining the additional tones of the heart.

4. The method of determining the heart murmurs at the points of auscultation.

5. The ability to interpret the received auscultative symptoms

Questions for monitoring the initial level of knowledge:

1. The rules of auscultation of the heart?

2. What are the places of projection of the valves on the anterior chest wall?

3. What are the points of auscultation of the heart, and the order of their listening?

4. What is the mechanism of occurrence of the main heart tones?

5. What are organic heart noises and how are they formed?

6. What are functional heart murmurs and how are they formed?

INFORMATION BLOCK.

Auscultation of the heart.

Auscultation of the heart is the most important part of the physical examination of the patient. During auscultation, heart tones are evaluated and heart murmurs are detected. Auscultation should be performed primarily in the patient's supine position, in some cases supplemented by listening in the position on the left side, on the stomach, standing or sitting, while holding the breath on the inhale or exhale, after physical exertion. All these techniques allow you to detect a number of symptoms that are of important diagnostic significance. The room where the auscultation is performed should be quiet and warm.

During the activity of the heart, sound phenomena occur, which are called heart tones. In healthy people, two tones are well heard during auscultation of the heart: the I tone that occurs during systole-systolic, and the II tone that occurs during diastole-diastolic.

For a better understanding of the mechanism of formation of heart tones, let us recall the phase structure of the cardiac cycle. The contraction of the heart begins with the atrial systole, followed by the contraction of the ventricles. During ventricular systole secrete

1) the phase of asynchronous contraction, when not all parts of the myocardium are still present

they are covered by the contractile process and the intraventricular pressure does not increase;

2) the phase of isovolumic contraction, which occurs when the contractile process covers the main mass of the myocardium; in this phase, the atrioventricular valves close and the intraventricular pressure increases significantly;

3) the ejection phase, when when the intraventricular pressure rises above the pressure level in the aorta and pulmonary trunk, the valves of the aorta and pulmonary trunk (semilunar) open.

At the end of the expulsion, the relaxation of the ventricles begins — the period of diastole:

1) during diastole, the semilunar valves close-protodiastolic period;

2) when the atrioventricular (atrioventricular) and semilunar valves are closed, the ventricles continue to relax until the pressure in them becomes lower than in the atria (the phase of isovolumic relaxation);

3) after that, the atrioventricular valves open and the blood begins to flow into the ventricles. Since at the beginning of diastole, the pressure difference in the atria and ventricles is large, the ventricles fill quickly - the phase of rapid filling;

4) then the blood flow slows down - the slow filling phase

5) After this, atrial systoles begin, and the cardiac cycle repeats.

I tone is formed from several components (Fig. 42; 1, 2). The main one is the valvular component, i.e., the oscillations of the atrioventricular valves in the phase of isovolumic contraction. The frequency of atrioventricular valve oscillations is affected by the rate of ventricular contraction: the faster they contract, the faster the intraventricular pressure increases and the louder the I tone. An additional role is played by the position of the atrioventricular valve flaps to the beginning of the systole, which depends on the blood filling of the ventricles: the less blood-filled the ventricles in the diastole, the wider the valve flaps are open and the greater the amplitude of their oscillations during systole.

The second component is the muscle component, which also occurs during the period of isovolumic tension, simultaneously with the valvular one, and is caused by fluctuations in the myocardium of the same ventricles.

The third component, the vascular component, is associated with fluctuations in the initial segments of the aorta and pulmonary trunk when they are stretched by blood during the period of expulsion.

The fourth component is the atrial component, and the fluctuations associated with atrial contraction play a role in its origin. With this component, the I tone begins, since the atrial systole precedes the ventricular systole. Normally, the vibrations caused by the atrial systole merge with the sound vibrations caused by the ventricular systole and are perceived as one tone.

The greatest importance in the occurrence of the I tone is given to the tension of the atrioventricular valves, consisting of elastic tissue, at the moment when they are already closed. The closing of the flaps of the valves (the flaps are closed, causing sound vibrations) is given less importance; it takes part only in the formation of the initial phase of the I tone. In the formation of the I tone, the fluctuations of the myocardium take part in the contraction of the ventricles in the process of their tension. The last two components are less important because the vascular component is associated with fluctuations in the initial parts of the aorta and pulmonary trunk when they are stretched by blood, the atrial component - with atrial contraction.

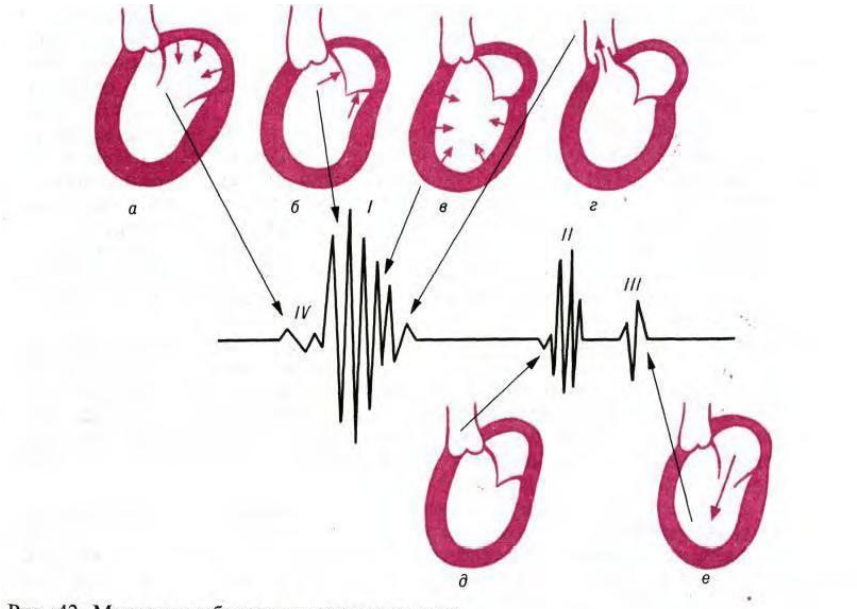


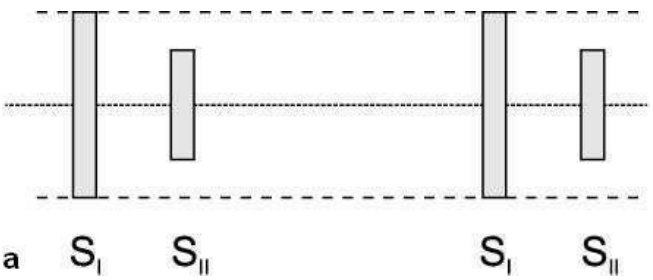
Рис. 42. Механизм образования тонов сердца.
а—г — механизм образования I тона (*а* — предсердный компонент, иногда воспринимается как самостоятельный IV тон; *б* — клапанный компонент; *в* — мышечный компонент; *г* — сосудистый компонент); *д* — механизм образования II тона; *е* — механизм образования III тона.

The second tone is formed due to vibrations that occur at the beginning of diastole when the semilunar valves of the aorta and pulmonary trunk are closed(the valvular component), and vibrations of the walls of these vessels (the vascular component).

Differences between the I and II heart tones.

Under normal conditions, it is relatively easy to distinguish between the I and II heart tones, since a relatively short systolic pause is determined between them (Fig. 1,2). The pause between the II and I heart tones during diastole is much longer. Difficulties in identifying tones can occur when the heart rate increases. At the same time, it should be borne in mind that the I tone corresponds to an apical push or an easily determined pulsation of the carotid artery.

The second tone and its components associated with the closing of the semilunar flaps of the aortic valves and the pulmonary trunk are always better heard in the second intercostal space on the left and right at the edge of the sternum. The I tone, which is primarily associated with the tension of the mitral valve flaps, is evaluated during auscultation at the top of the heart, as well as at the lower edge of the sternum (Fig.1,3) .



I and II heart tones: differences in the tones at the top (a) and above the base (b) of the heart. At the top of the heart, the I tone is better heard, at the base of the heart - II. SI-I heart tone, SII-II heart tone.

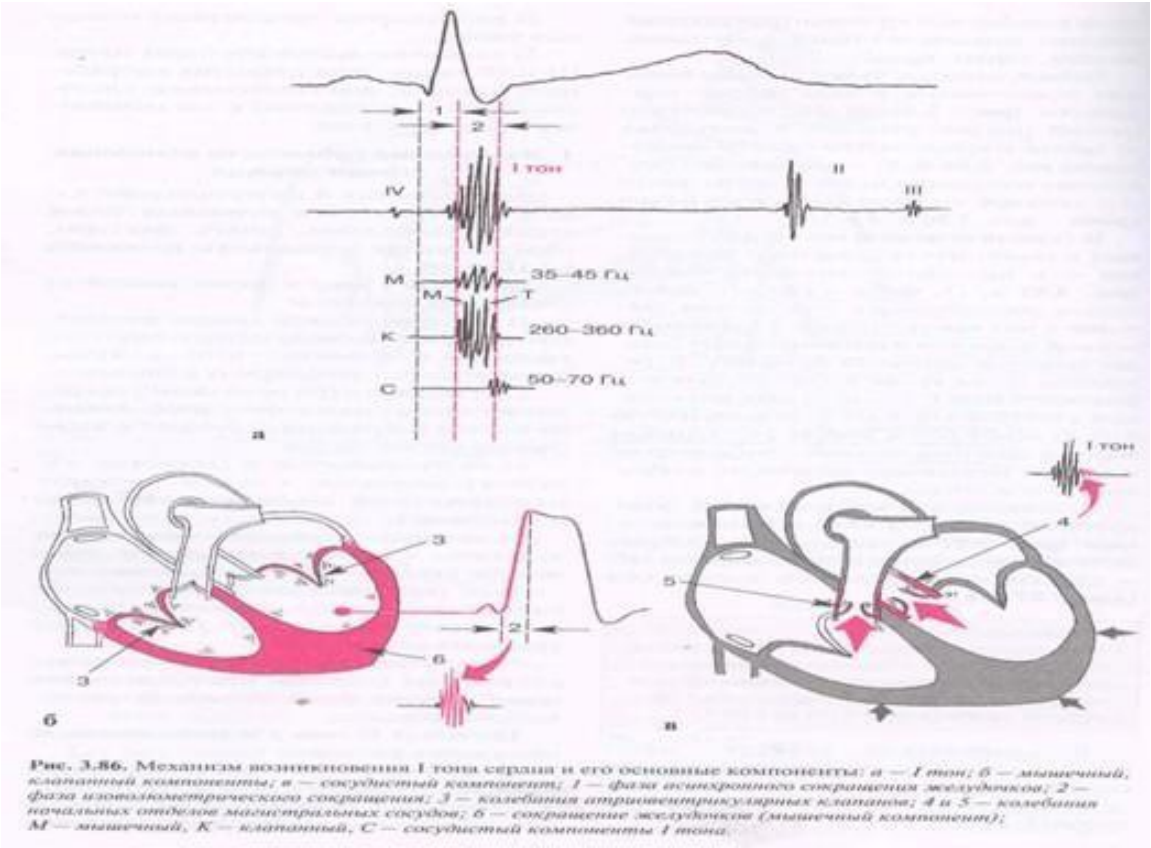
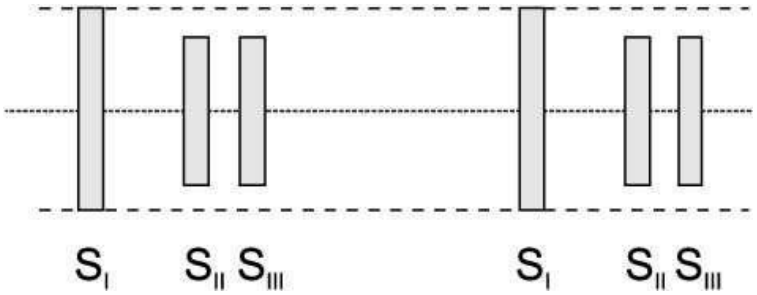


Рис. 3.86. Механизм возникновения I тона сердца и его основные компоненты: *а — I тон; б — мышечный, клапанный компоненты; в — сосудистый компонент; 1 — фаза асинхронного сокращения желудочков; 2 — фаза изоволюметрического сокращения; 3 — колебания атриовентрикулярных клапанов; 4 и 5 — колебания начальных отделов магистральных сосудов; 6 — сокращение желудочков (мышечный компонент); М — мышечный, К — клапанный, С — сосудистый компоненты I тона.*

The third tone of the heart is listened to after the second tone (after 0.15 seconds; Figure 4.5). It is caused by fluctuations in the ventricular myocardium during rapid passive filling with blood (from the atria) at the beginning of diastole. Normally, the third tone can be heard in children, adults up to 35-40 years old, as well as in the third trimester of pregnancy. This tone is low, hollow (so it is desirable to use a stethoscope), and is best heard at the top of the heart in the patient's lying position on the left side.



The pathological III tone (sounds like a physiological one) takes part in the formation of the protodiastolic rhythm of the gallop. The appearance of tone III in persons over 40 years of age is always a pathological sign. The most common causes are the following conditions:

a decrease in the contractility of the myocardium (III) is characteristic of chronic heart failure);

volume overload of the ventricles - for example, in case of mitral or tricuspid valve insufficiency.

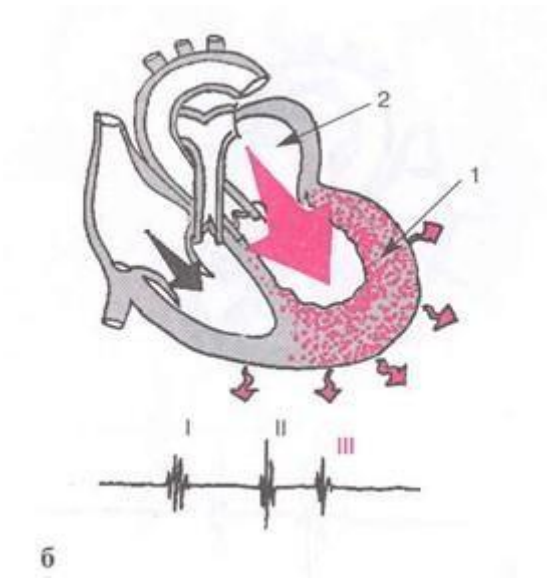


Fig. 5. The mechanism of formation of the third tone.

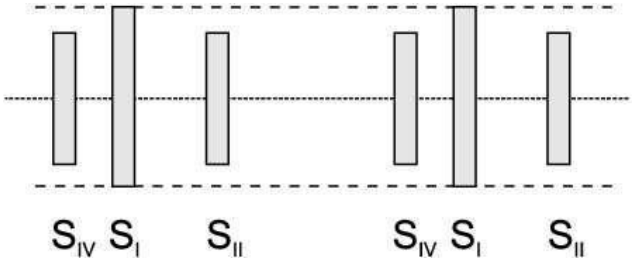
The IV tone of the heart occurs immediately before the I tone - at the end of the ventricular diastole (Fig. 6, 7) and is associated with their rapid filling due to atrial contractions (the ventricles have increased resistance to the blood filling them). It is low, deaf, so it is also better to listen to it with a stethoscope. The IV tone against the background of a relatively rare heart rhythm can be heard in the elderly without much heart changes, as well as in healthy, trained individuals.Most often, the following conditions lead to a pathological IV tone:

Hypertension.

Aortic stenosis.

Cardiomyopathy.

The delay in conducting the excitation from the atria to the ventricles makes the IV tone more audible. The IV tone is the basis of the presystolic rhythm of the gallop.



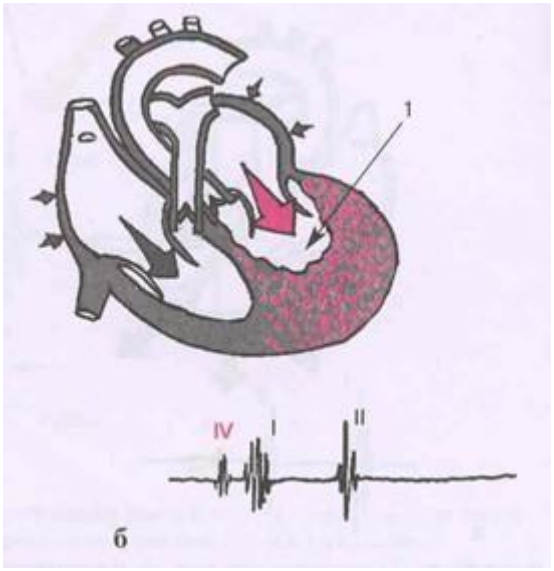


Fig. 7 The mechanism of formation of the fourth tone.

The first two tones can be heard over the entire area of the heart, but their sonority will vary depending on the proximity of the valves involved in the formation of either the I or II tones. Therefore, in order to correctly assess the auscultation data, it is necessary to know the locations of the projection of the valves on the chest wall and the points at which the sound phenomena coming from a particular valve are better listened to.

Each valve opening corresponds to a specific listening area. These areas (auscultation points) do not exactly coincide with the places of projection of the valves on the anterior chest wall. Sounds that occur in the valve openings are conducted in the direction of the blood flow. This allows you to find certain points on the chest where the sound phenomena associated with the activity of each valve are most well listened to. The following points of the best listening of the heart valves are established (Fig. 8).

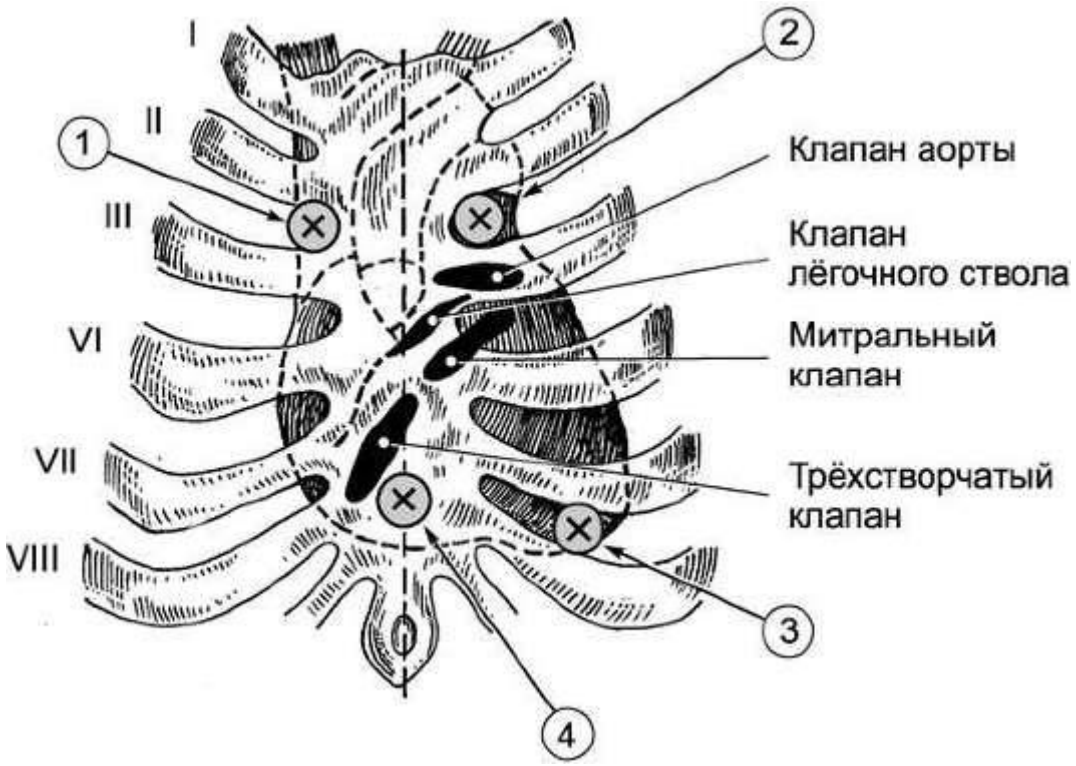


Fig. 8. Listening points of the heart valves and their projection on the anterior chest wall. 1 - the point of listening to the aortic valve (the second intercostal space to the right of the sternum); 2 - the point of listening to the pulmonary trunk valve (the second intercostal space to the left of the sternum); 3 - the point of listening to the mitral valve (the apex of the heart, usually the fifth intercostal space 1-1.5 cm inside the left midclavicular line); 4-the point of listening to the tricuspid valve (the lower third of the sternum). (From: Myasnikov A. L. Propaedeutics of internal diseases. Moscow: Medgiz, 1956; with changes.)

The 1st point of auscultation - the area of the apical push-for the mitral valve (since the vibrations are well conducted by the dense muscle of the left ventricle, and the apex of the heart during systole is closest to the anterior chest wall);

2nd auscultation point - second intercostal space on the right at the edge of the sternum-the aortic valve is best heard in the second intercostal space to the right of the sternum, where the aorta is closest to the anterior chest wall;

The 3rd auscultation point - the second intercostal space on the left at the edge of the sternum-is the valve of the pulmonary trunk, for this valve the place of best listening coincides with its true projection;

4th auscultation point - lower part of the sternum (base of the xiphoid process) - tricuspid valve (right ventricle area);

The 5th auscultation point - (Botkin-Erb point) - is the third intercostal space on the left near the sternum; auscultation of this area allows you to more clearly listen to the diastolic noise that appears when the aortic valve is insufficient.

As can be seen from the figure, the places of projection of the valves on the anterior chest wall are very close to each other (Fig. 8). The projection of the left atrioventricular (mitral) valve is located to the left of the sternum in the area of attachment of the III rib, the right atrioventricular (tricuspid) valve is on the sternum, at the middle of the distance between the place of attachment to the sternum of the cartilage of the III rib on the left and the cartilage of the V rib on the right. The valve of the pulmonary trunk is projected in the second intercostal space to the left of the sternum, the aortic valve-in the middle of the sternum at the level of the cartilage of the III ribs. Listening to the heart in the places of the true projection of the valves at such a close location from each other does not allow us to determine which of the valves is affected.

Remember! During auscultation of the heart, the valves should be listened to in order of increasing frequency of their damage. First, the mitral valve at the apex of the heart is listened to, then the aortic valve in the second intercostal space to the right of the sternum, then the pulmonary trunk valve in the second intercostal space to the left of the sternum, the tricuspid valve at the base of the xiphoid process of the sternum, and finally the aortic valve again at the Botkin-Erb point.

Changes in heart tones.

Changes in the tones of the heart can be expressed in a weakening or strengthening of the sonority of one or both tones, in a change in their timbre, duration, in the appearance of a split or splitting of tones, the appearance of additional tones.The sonority of heart tones may depend on the conditions of sound vibrations, i.e., on extra-cardiac causes. In the case of excessive development of subcutaneous fat or chest muscles, with emphysema of the lungs, accumulation of fluid in the left pleural cavity and other processes that move the heart away from the anterior chest wall, the sonority of the tones weakens.

When the conditions for the conduction of sound vibrations improve (thin chest, wrinkling of the edges of the lungs, the approach of the heart to the anterior chest wall due to the development of a tumor in the posterior mediastinum, etc.), the sonority of the heart tones increases. The tones of the heart are enhanced by resonance when large air cavities are located near it (a large pulmonary cavity, a large gas bubble of the stomach).

The sonority of the tones also depends on the composition of the blood flowing through the heart: with a decrease in the viscosity of the blood, as is observed in anemia, the sonority of the tones increases.

In the diagnosis of heart diseases, it is of great importance to identify changes in the tones caused by the defeat of the heart itself. The weakening of both tones can be observed with a decrease in the contractility of the heart muscle in patients with myocarditis, myocardial dystrophy, acute myocardial infarction, cardiosclerosis, fluid accumulation in the pericardial cavity.

The amplification of both tones occurs due to the increased influence of the sympathetic nervous system on the heart. This is noted in cases of heavy physical work, anxiety, and severe respiratory illness.

It is especially important in the diagnosis of heart diseases to change one of the tones. The weakening or strengthening of the I tone can be discussed when listening only at the top of the heart:

Attenuation of the I tone (Fig.) at the apex of the heart, there is an insufficiency of the mitral and aortic valves. In case of mitral valve insufficiency during systole, the valve flaps do not completely cover the left atrio-ventricular opening. This allows some of the blood to return back to the left atrium. The amount of blood pressure on the walls of the ventricle and the leaves of the mitral valve will not reach the value that is observed in the norm, so the valvular and muscular components of the I tone are significantly weakened. In case of aortic valve insufficiency during the systole period, there is also no period of closed valves, therefore, the valvular and muscular components of the I tone will also significantly weaken. With insufficiency of the tricuspid valve and the pulmonary trunk valve, the weakening of the I tone will be better detected at the base of the xiphoid process of the sternum due to the weakening of the valvular and muscular components of the right ventricle with these defects.

The weakening of the I tone at the apex of the heart can be detected with a narrowing of the aortic mouth, since with difficulty emptying the left ventricle and its overflow, the systolic tension of the myocardium increases slowly, the amplitude of sound vibrations decreases.

weakening of the I tone at the apex of the heart in diffuse myocardial lesions (due to dystrophy, cardiosclerosis, myocarditis), there may be a weakening of not both tones, but only I, since in these cases its muscle component also weakens.

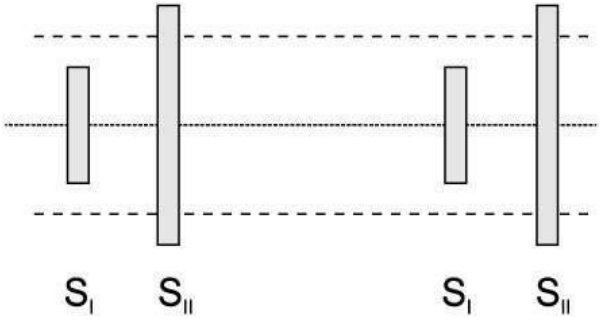


Fig. 9. Attenuation of the I tone. SI - I heart tone, SII-II heart tone. The explanation is in the text.

An increase in the I tone at the apex of the heart (Fig.10) is observed with a decrease in the blood filling of the left ventricle during diastole. Often, an increase in the I tone is noted when the left atrioventricular orifice is narrowed, when during diastole, less blood enters the ventricle from the atrium than normal. Therefore, by the beginning of systole, the left ventricular muscle is less stretched, more relaxed, which allows it to contract faster, causing an increase in the I tone, then the I tone is defined as" flapping " (typical for mitral stenosis).

In stenosis of the right atrioventricular foramen, the I-tone amplification is heard at the base of the xiphoid process of the sternum.

an increase in the I tone can occur in tachycardia after physical exertion(physiological increase in the I tone), in extrasystole (premature contraction of the heart) due to a small diastolic filling of the ventricles;

An increase in the I-tone may occur in anemia, hyperthyroidism (the increase is associated with an increase in cardiac output).

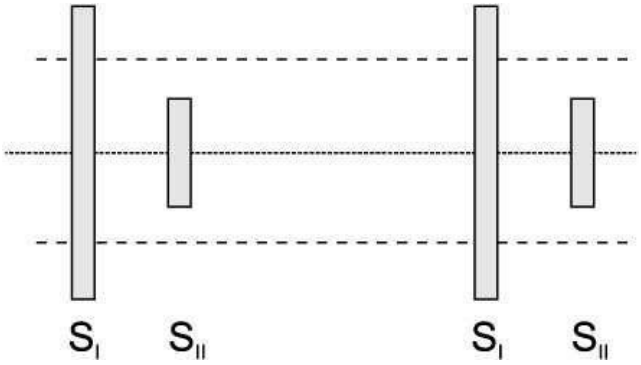


Figure 10 . I-tone gain. SI - I heart tone, SII-II heart tone. The explanation is in the text.

The weakening or strengthening of the second tone can only be said when listening to it on the basis of the heart (in the second intercostal space). If the second tone consists of two components when listening on the basis of the heart, we can talk about its bifurcation.

The weakening of the II tone above the aorta is observed with a significant decrease in arterial pressure and a decrease in their blood supply;

the weakening of the II tone above the aorta is possible when calcium salts are deposited in the aortic valve flaps, which leads to a decrease in the mobility of the valves and a violation of their closure in atherosclerosis;

the weakening of the II tone above the aorta is observed in the case of aortic valve insufficiency, since there is either a destruction of the valve flaps, or a decrease in their ability to oscillate due to scarring. In addition, the push of blood rushing at the beginning of diastole from the aorta to the aortic valve leaves is weaker than normal, since some of the blood returns to the stomach through the aortic opening that is not completely covered. II the tone above the aorta may not be heard at all if the aortic valve is significantly destroyed, for example, in bacterial endocarditis;

the weakening of the II ton over the pulmonary artery is possible with a large thickness of the thoracic cell, as well as with stenosis of the pulmonary artery trunk;

the weakening of the II tone above the pulmonary trunk appears in the insufficiency of the pulmonary trunk valve (an extremely rare heart defect) and with a decrease in pressure in the small circle of blood circulation;

The second tone increases with an increase in blood pressure in large vessels - the aorta or pulmonary artery, while it is said that the second tone is accentuated on one or the other vessel, respectively.

In this case, the second tone, for example, to the right of the sternum, is listened to as essentially more intense than on the left, and vice versa (i.e., to determine the accent of the second tone, you need to compare its volume in the second intercostal space to the right and left of the sternum). The accent of the second tone is explained by a faster slamming the corresponding valves and a louder sound perceived during auscultation. the accent of the second tone above the aortic valve is most often detected in arterial hypertension (this type of accent of the second tone is listened to most often), as well as in pronounced sclerotic changes of the aorta with a decrease in the elasticity of its walls; The accent of the second tone over the pulmonary artery is determined when the pressure in it increases in patients with mitral defects and pulmonary heart.The weakening of both tones can be observed with a pronounced diffuse lesion of the ventricular myocardium and a decrease in their contractility, for example, in myocarditis,

myocardiodystrophies, coronary heart disease, cardiomyopathies. Split heart tones. The bifurcation of the heart tones is spoken of when their main components are captured separately (in this case, instead of two short tones, quickly following each other, are heard in the same tone.for a friend). Bifurcation of tones occurs when the sound components that make up the tone occur at different times, i.e. it depends on the asynchronism in the activity of the right and left halves of the heart: non - simultaneous closure of the atrioventricular valves leads to a bifurcation of the I tone, non - simultaneous closure of the semilunar valves leads to a bifurcation of the II tone.If the two parts of a bifurcated tone are separated by such a short interval that they are not perceived as two separate tones, they are talking about splitting the tone. Split tonesit can be physiological and pathological (Fig. 11).

Fig. 11 .. Physiological bifurcation of the second tone. SI - I heart tone, SII-II heart tone.

The explanation is in the text.

A slight bifurcation of the second tone, always heard at the base of the heart, i.e. in the second intercostal space, can also occur in physiological conditions (Fig. 11). With a deep breath, due to an increase in blood flow to the right heart, the duration of the systole of the right ventricle may be slightly longer than the left, so the pulmonary artery is listened to splitting of the second tone, with the second component associated with the closure of the pulmonary artery valve, and the first - with the closure of the aortic valve.

This physiological cleavage of tone II is better heard in younger individuals.

Above the aorta, the splitting of the second tone is not heard, since the pulmonary component of thesecond tone is too weak to be carried to the listening area of the aortic

valve.Usually, a split of the second tone is stated. It may be associated with non-

temporary closure of the aortic and pulmonary artery valves, which is due to the

different duration of contraction of the left and right ventricles accordingly, changes in the large and small circle of blood circulation. When the pressure increases, for example, in the pulmonary artery, the second component of the second tone is associated with a later closure of the pulmonary artery valve. In addition, bifurcation of the second tone may be associated with increased blood flow in the small or large circulatory circle.

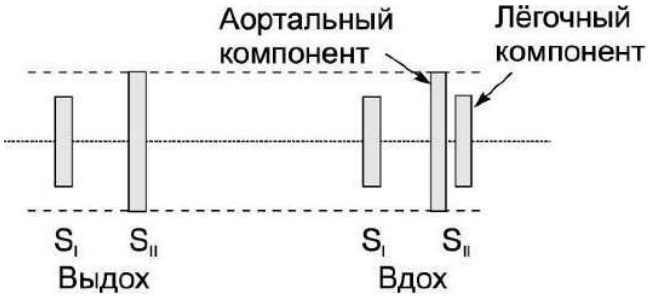
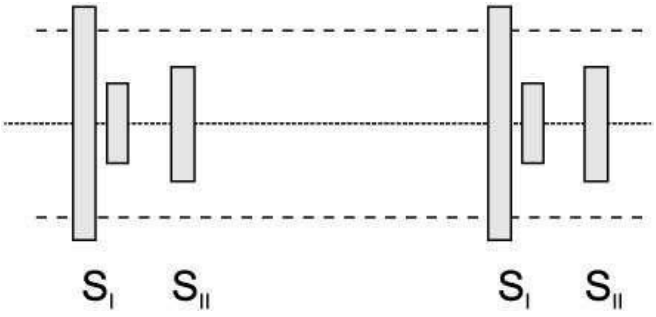


Fig. 12. Fixed bifurcation of the second tone. SI - I heart tone, SII-II heart tone. Explanation in the text.

In pulmonary hypertension in patients with chronic lung diseases, the division of tone II is less pronounced and distinct, since the right ventricle (although it works against increased pressure in the lungs) is usually hypertrophied, and therefore its systole does not lengthen.

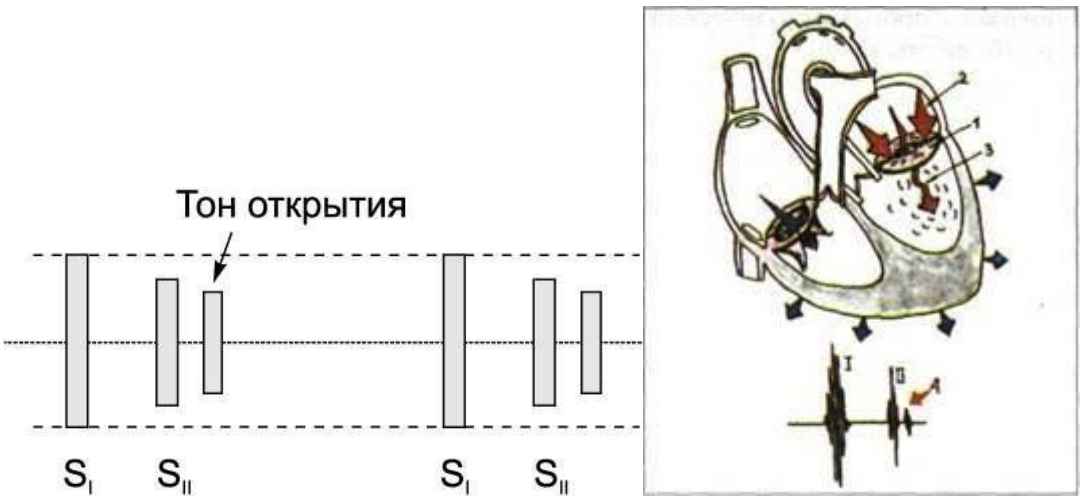
Bifurcation of the I tone (Fig. 13) can be heard normally along the left edge of the sternum (the tricuspid component of the I tone is heard). This cleavage is sometimes heard at the top of the heart (additionally). Pathological splitting of the I tone is observed in violation of intraventricular conduction along the legs of the Gis bundle, which leads to a delay in the systole of one of the ventricles.



ADDITIONAL HEART TONES.

The tone of the opening of the mitral valve. The opening of the mitral valve usually occurs silently, at the beginning of diastole. When the mitral valve flaps are fused in patients with mitral stenosis, their opening at the beginning of diastole is limited, so the blood flow causes fluctuations in these flaps, which are perceived as an additional tone-the tone of the opening of the mitral valve (Fig. 14, 15). It is quite high and has a "slit" character, which helps to distinguish it from the III tone. This tone can be heard shortly after the second tone, but only in the upper part of the heart (slightly medial to it and along the left edge of the lower third of the sternum), indicating its connection with mitral valve oscillations. It is most optimal to use a stethoscope with a membrane. The opening tone is best heard in the upper part of the heart, not at the base, it is characterized by constancy and is combined with other auscultative signs of mitral stenosis. The tone of the opening of the mitral valve, heard together with a loud (pop) I

tone, characteristic of mitral stenosis, and II tone form a kind of three-part rhythm, called "quail rhythm", because it resembles the cry of a quail.



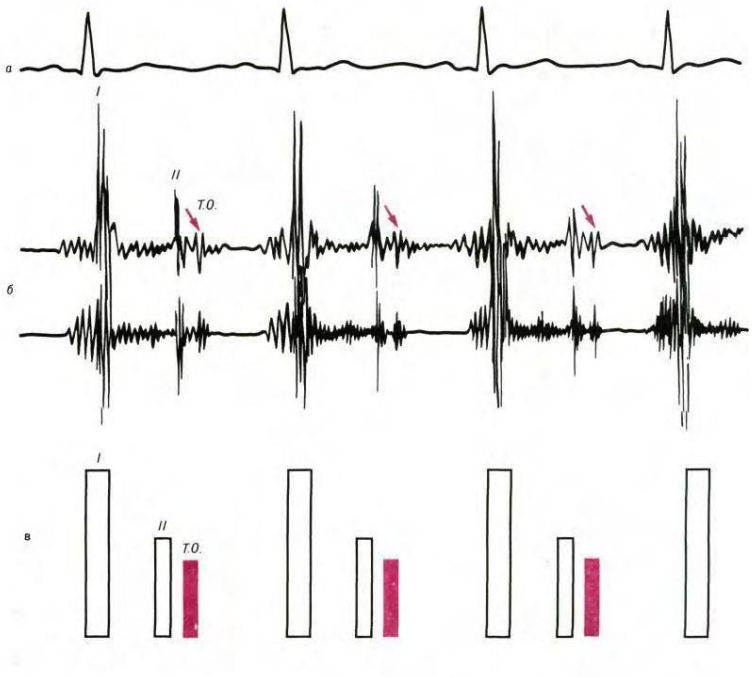


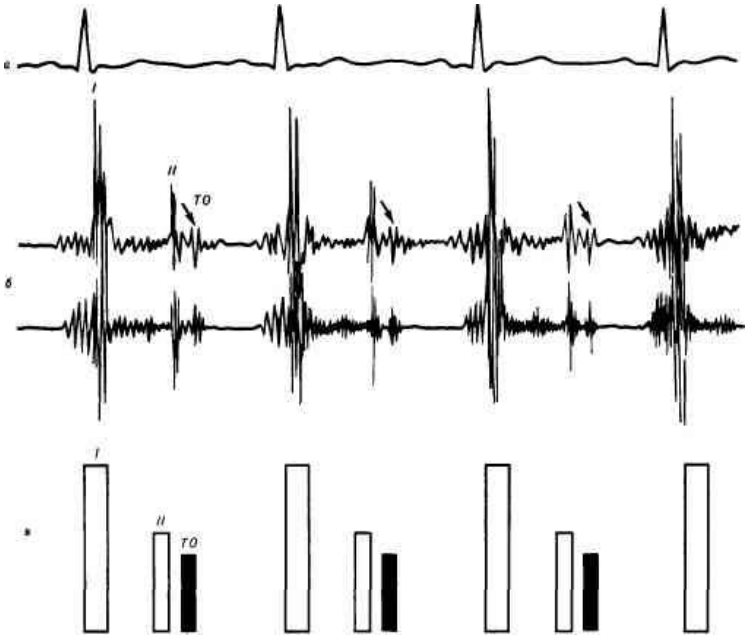
Рис. 43. Изменение тонов при митральном стенозе («ритм перепела»).
а—ЭКГ; *б*—ФКГ; *в*—схема изменения тонов; *Т. О.*—тон открытия митрального клапана.

A similar tone of opening of the tricuspid valve is heard at the lower part of the sternum, but quite rarely.

Tones of exorcism.Systolic ejection tones are heard immediately after the first heart tone (Fig. 16); they occur in connection with fluctuations in the aortic valve or the pulmonary artery valve, so they are better heard in the second intercostal space on the left or right at the edge of the sternum. The exile tones are relatively high, have a sharp "clicking" character, and are better listened to with a stethoscope with a membrane. Their occurrence is also associated with the appearance of fluctuations in the walls of large vessels, especially when they expand.

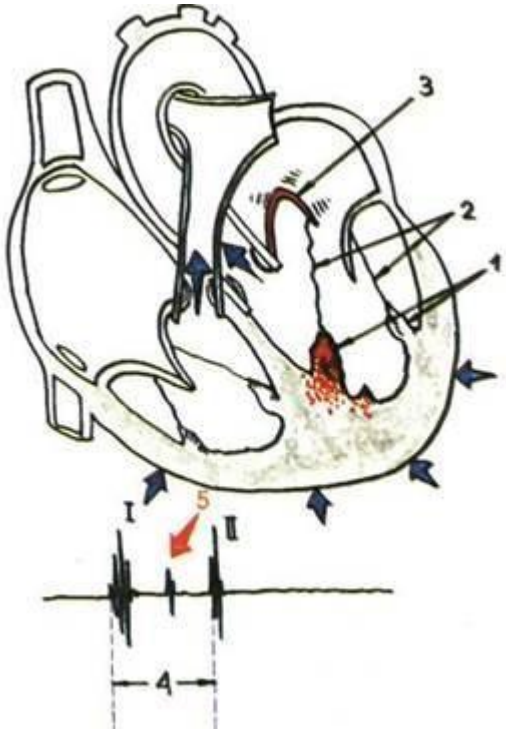
With pericardial fusion, an additional pericardial tone may occur. It appears during diastole 0.08-0.14 s after the second tone and is associated with pericardial fluctuations with rapid ventricular expansion at the beginning of diastole (Fig.17). An additional tone during

pericardial fusion can also occur during systole between the first and SECOND heart tones. This loud short tone is also called a systolic click.



17. When the pericardium is fused, an additional pericardial tone may occur.

A systolic click can also occur with mitral valve prolapse (bulging or protruding of the mitral valve leaf into the left atrial cavity during left ventricular systole) (Figure 18). Mitral valve prolapse occurs when the diastolic volume of the left ventricular cavity decreases, or when the papillary muscles are affected, or when the tendon filaments are elongated, which disrupt the movement of the valve leaf.



The origin of heart murmurs.

In auscultation of the heart, in some cases, in addition to tones, sound phenomena called heart noises are heard. These are the sound vibrations that occur in the heart during the turbulent flow of blood. The occurrence of blood flow turbulence is possible in the following situations:

1) The valve flaps are soldered together, and therefore they cannot be fully opened. In this case, they talk about stenosis - narrowing of the valve opening.

2) A decrease in the area of the valve flaps or expansion of the valve opening, which leads to incomplete closure of the valve opening and backflow (regurgitation) of blood through the narrowed (partially open) space.

3) The presence of abnormal openings in the heart, for example, in the interventricular or atrial septum.

4) Another factor involved in the occurrence of noise is the speed of the blood flow: the faster the blood flows, the louder the noise, and vice versa.

In all these situations, there are "swirls" of the blood flow and fluctuations of the valves, the fibrous threads of the valves, and individual sections of the myocardium, which spread and are heard on the surface of the chest.

The noises are divided:

1) at the place of origin – intracardial or extracardial noises occurring inside the heart itself and outside it-extracardial or extra-cardiac noises associated with changes in the pericardium and the pleura in contact with it (pericardial friction noise, pleuropericardial noise). Intracardial noises are most common.

2) due to the occurrence of organic (may occur with anatomical changes in the structure of the valves or the heart itself) and functional (appear when the function of unchanged valves is impaired);

3) according to the time of the appearance of noise during systole or diastole, systolic and diastolic noises are distinguished.

The pericardial friction noise (Fig. 19)occurs during inflammatory changes in its leaves (acute pericarditis, constrictive pericarditis). It can be tricomponent, coinciding with the phases of cardiac contraction: ventricular systole, ventricular diastole, and atrial systole. Pericardial friction noise is associated with changes in the visceral and parietal pericardial sheets, when fibrin is deposited on them (in pericarditis), cancer metastases appear, etc. The mechanism of formation of pericardial friction noise is similar to the mechanism of occurrence of pleural friction noise, only instead of respiratory movements, the movement of the heart during systole and diastole plays a role in its appearance.

The noise of pericardial friction can be of different sonority, sometimes it is similar to the noise of pleural friction, resembles the crunch of snow, sometimes a very quiet noise is heard, similar to the rustle of paper or resembling scratching. The noise of pericardial friction differs from intracardiac noises

in the following signs:

1) does not always exactly coincide with systole and diastole, it is often heard continuously, only becoming stronger during systole or diastole;

2) for a short time, it can be heard in different phases of cardiac activity: then during systole, then during diastole;

3) fickle, can disappear and reappear;

4) does not coincide in localization with the points of the best listening of the valves; it is most well listened to in the area of absolute dullness of the heart, at its base, at the left edge of the sternum in the third or fourth intercostal space; its localization is not constant and can change even within one day;

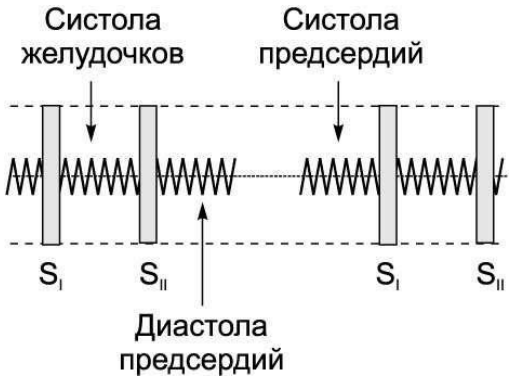
5) very poorly conducted from the place of its formation;

6) feels closer to the investigator's ear than intracardiac noises;

7) increases when the stethoscope is pressed against the chest and when the patient's torso is tilted forward, since the pericardial leaves touch more closely;

8) the noise intensity can be different;

9) at a high volume, it is heard regardless of the patient's position, while a weak noise should be heard in the patient's vertical position (when the body is tilted forward and the stethoscope is pressed).



19. Pericardial friction noise. SI - I heart tone, SII-II heart tone. Explanation in the text.

Pleuropericardial friction noise occurs when the pleura is inflamed, directly adjacent to the heart, due to the friction of the pleural leaves, synchronous with the activity of the heart. In contrast to the noise of pericardial friction, it is heard along the left edge of relative cardiac dullness; it is usually combined with the noise of pleural friction and changes its intensity in different phases of breathing: it increases with a deep breath, when the edge of the lung is in closer contact with the heart, and sharply weakens on exhalation, when the edge of the lung decreases.

Organic (associated with anatomical changes in the structure of the valves or heart) and functional (with unchanged valves) intracardiac heart murmurs. Functional noises differ from organic noises in a number of ways:

in most cases, they are systolic;

noises are not constant, they can occur and disappear at different positions of the body, after physical exertion, in different phases of breathing;

functional noises can be observed when the blood flow rate increases or the blood viscosity decreases;

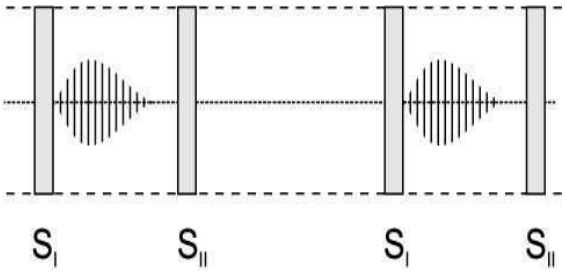
most often they are heard over the pulmonary trunk, less often-over the top of the heart;

the noises are short-lived, rarely occupy the entire systole, by nature soft, blowing and quiet (no more than 2-3 points). Scratching and other rough noises are not considered functional;

functional noises are not accompanied by other signs of damage to the valves (an increase in the heart, changes in tones, etc.). they occur even in healthy people (more often in children and young people).

The most common cause of organic noise is congenital or acquired heart defects, or changes in the anatomical structure of the heart.

Systolic murmur occurs when, during systole, the blood, moving from one part of the heart to another or from the heart to large vessels, meets a narrowing in its path. Systolic murmur is heard in stenosis of the aortic mouth or pulmonary trunk, since with these defects, during the expulsion of blood from the ventricles, an obstacle occurs in the path of blood flow — narrowing of the vessel (systolic murmur of expulsion), as well as as a result of accelerating blood flow through the same, but unchanged holes. The noise usually increases towards the middle of the systole, then decreases and stops shortly before the second tone (there is always a pause between the noise and the second tone; the absence of a pause indicates a pansystolic noise; Fig. 20). The noise may be preceded by a systolic tone of ejection.



20. Mesosystolic noise (ejection noise). SI - I heart tone, SII-II heart tone. The explanation is in the text.

Systolic murmur is also heard in cases of mitral and tricuspid valve insufficiency. Its occurrence is explained by the fact that during systole, the jelly-daughter blood enters not only the aorta and the pulmonary trunk, but also back into the atrium through the not fully covered mitral (or tricuspid opening), i.e. through a narrow slit (systolic noise of regurgitation).

Late systolic murmurs occur in the second half of the systole. Such noises are observed primarily in mitral valve prolapse. In this condition, there is an elongation (or rupture) of the chords, which leads to the occurrence of prolapse of the mitral valve flaps and mitral insufficiency with the return (regurgitation) of blood to the left atrium. The prolapse itself is manifested by a systolic click in the middle of the systole and mitral insufficiency with systolic noise occurring after the systolic click.

Diastolic murmur occurs when there is a narrowing in the blood flow pathway and appears in the diastole phase. It is heard when the left or right atrioventricular orifice is narrowed, since with these defects, blood flows from the atria to the ventricles through the existing narrowing during diastole. Diastolic murmur also occurs when the aortic valve or the pulmonary trunk is insufficient due to the reverse blood flow from the vessels to the ventricles through the gap formed when the valves of the altered valve are not fully closed. Diastolic murmurs are relatively rarely functional; In particular, they occur in anemia in patients with renal insufficiency and are heard most often on the basis of the heart in the second intercostal space on the left near the edge of the sternum.

GENERAL CHARACTERISTICS OF HEART MURMURS.

Noises in the heart are heard not only in the areas of the projection of the valves (Fig. 21; see also Fig. 6-7), but also in a larger area of the heart area. Usually, the noises are well conducted by the blood flow. Thus, when the aortic mouth is narrowed, the systolic murmur also spreads to large vessels, for example, the neck. In case of aortic valve insufficiency, diastolic noise can be detected not only in the second intercostal space on the right, but also on the left in the third intercostal space at the left edge of the sternum, in the so-called fifth point (Botkin-Erb point). With mitral valve insufficiency, systolic murmur can spread to the left axillary region.

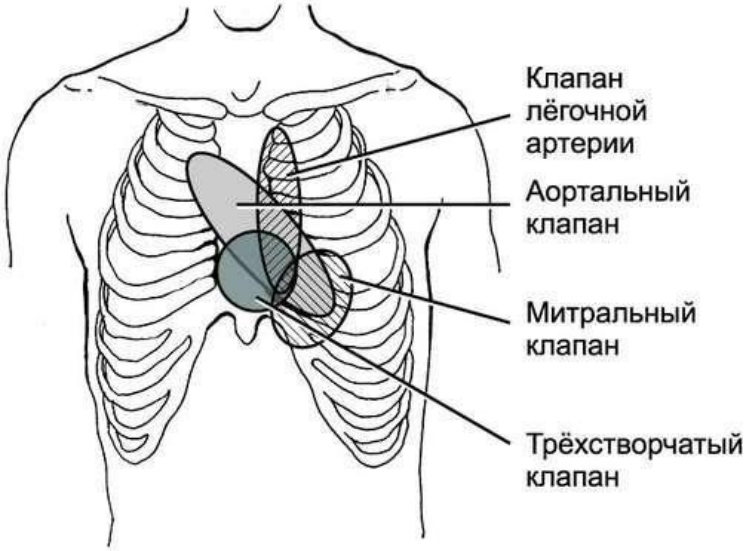
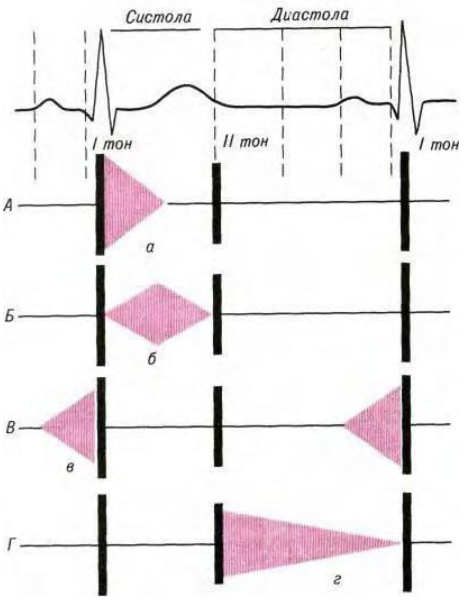


Fig. 21. Areas of noise propagation depending on the affected valve. The explanation is in the text.

If there is a narrowing or sharp widening of the bloodstream in the path of the blood flow, a turbulent blood flow occurs, causing fluctuations that are perceived as noise. In the absence of changes in the width of the lumen of the bloodstream, noise can occur due to an increase in the speed of blood flow, as is observed in thyrotoxicosis, fever, nervous excitement. A decrease in the viscosity of the blood (for example, in anemia) contributes to an increase in the speed of blood flow and can also cause noise. Features in the structure of the valvular apparatus of the heart (such as an abnormal arrangement of chords in the cavities of the heart or their excessive length) contribute to the appearance of functional noises. These changes, usually detected by echocardiography, are not combined with any other pathological signs, and noises are heard in practically healthy people.

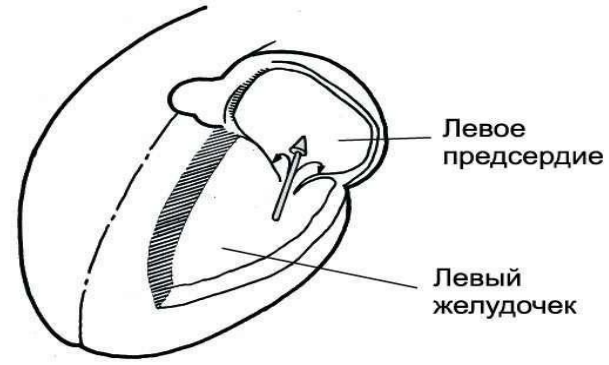
During auscultation, the following noise characteristics must be determined:

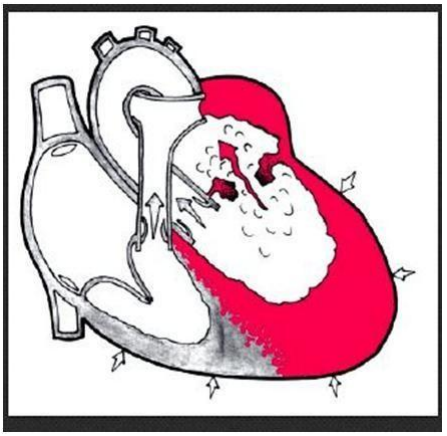
1) the relation to the phase of cardiac activity (to systole or diastole). The ratio of noise to systole or diastole is determined by the same characteristics that distinguish the I and II tones. The systolic murmur appears together with the I tone during a short pause of the heart; it coincides with the apical shock and the pulse of the carotid artery. Diastolic murmur occurs after the second tone during a long pause of the heart. There are three types of diastolic noise:a) early protodiastolic, occurring at the very beginning of the diastole, immediately after the second tone b) mesodiastolic, heard somewhat later than the second tone c) late diastolic or presystolic, appearing at the end of the diastole (fig. 45)



a) early diastolic (protodiastolic noises) occur immediately after the second tone and stop before the first tone. Usually these noises appear when blood regurgitates through the aortic valve and the pulmonary trunk valve;
b) mesodiastolic noise occurs a short time after the second tone. By the beginning of the first tone, it either subsides or passes into a late diastolic murmur, which usually begins in the middle of the diastole and continues until the appearance of the First tone (due to increased blood flow in the last phase of the diastole due to left atrial systole). Both types of noise occur when there is a turbulent flow of blood through the atrioventricular openings. The most typical example of such noises is mitral stenosis
c) diastolic Flint (Austin-Flint) noise (mesodiastolic or late systolic). It is heard in cases of severe aortic valve insufficiency at the apex of the heart (similar to diastolic murmur in mitral stenosis) in the absence of organic changes in the mitral valve. It is caused by the relative stenosis of the mitral valve in the diastole-the result of the displacement of the anterior leaf of the mitral valve to the posterior leaf by a stream of blood flowing back.
2) the properties (timbre, duration, intensity), the properties of noise are very different. According to the timbre, the noises can be soft, blowing or, conversely, rough, scratching, sawing; sometimes musical noises are heard. According to the duration, there are short and long noises, according to the volume - quiet and loud. In this case, the change in the volume, or intensity, of noise during a certain phase of cardiac activity is noted. The noise intensity can gradually decrease (decreasing noise) or increase (increasing noise). Decreasing noises are heard more often. This is explained by the following: at the beginning of the transition of blood from one part of the heart to another or from the heart to a large vessel, the pressure difference in both parts is large, so there is a high speed of blood flow. As the blood is expelled, the pressure in the part where the blood comes from gradually decreases, the speed of blood flow slows down and the intensity of the noise decreases. The increasing character has a presystolic noise, heard most often with a narrowing of the left atrioventricular opening at the very end of the ventricular diastole. At this point, atrial systole begins, which increases the rate of blood flow from the left atrium to the left ventricle.
The noise volume is evaluated on a 6-point scale:

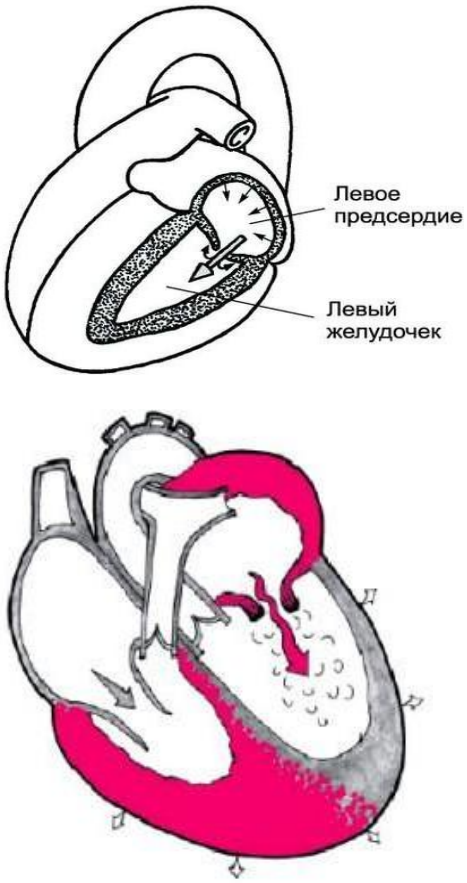
1 point - barely audible noise (at maximum hearing strain), not heard in all positions and sometimes disappearing.
2 points - a louder noise, heard constantly immediately after applying the stethoscope to the chest.
3 points - even louder noise, but without shaking the chest wall.
4 points - a loud noise, usually with a tremor of the chest wall, also felt by the hand (in the form of a "cat purr") placed on the chest.
5 points - a very loud noise, heard not only over the heart area, but also at any point in the chest; it can be heard even if the stethoscope does not fit tightly to the chest wall.
6 points - a very loud noise heard from the surface of the body outside the chest, for example, from the shoulder, or when the stethoscope does not touch the chest wall.
3) localization, i.e. the place of the best listening and the direction of conducting (irradiation).
The localization of the noise corresponds to the best listening location of the valve in which the noise was formed; only in some cases, the noise is better listened to at a distance from the place of origin, provided that it is well conducted. The sounds are well conducted in the direction of the blood flow; they are better heard in the area where the heart is closer to the chest and where it is not covered by the lungs.
Systolic murmur in mitral valve insufficiency is best heard at the apex of the heart; it can be carried along the dense muscle of the left ventricle to the axillary region or along the reverse blood flow from the left ventricle to the left atrium-to the second and third intercostals to the left of the sternum (Figure 22).



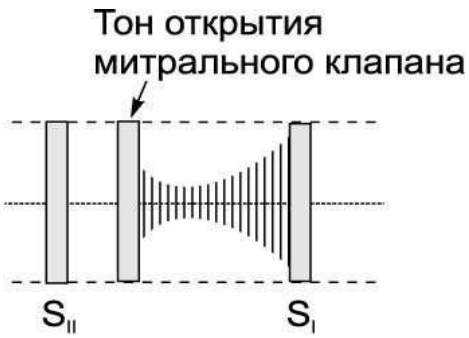


22. Blood regurgitation in mitral insufficiency. The arrow indicates the flow of regurgitation from the left ventricle to the left atrium during ventricular systole.

Diastolic murmur with narrowing of the left atrioventricular orifice is usually heard in a limited area in the region of the apex of the heart and practically does not radiate. (Figure 23)
The noise is usually low-tonal, so it is necessary to use a stethoscope without a membrane. The noise is better heard in the position of the patient on the left side on the exhalation. The average physical activity also allows you to amplify the noise (Figure 24).

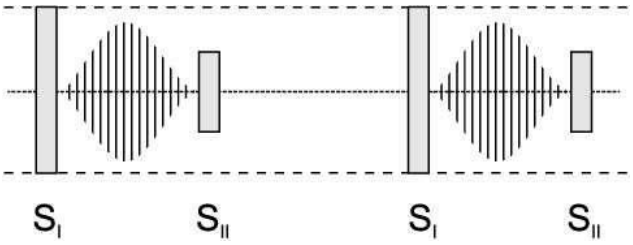


23. Hemodynamics in mitral stenosis. Noise (diastolic) occurs during left atrial systole when blood passes through the narrowed opening of the mitral valve.

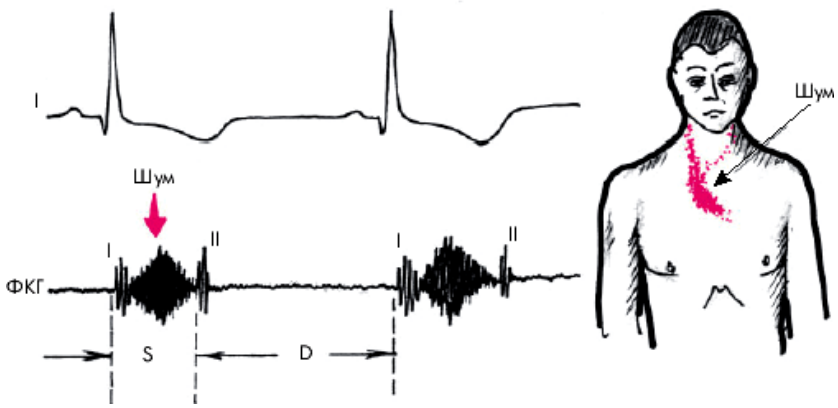


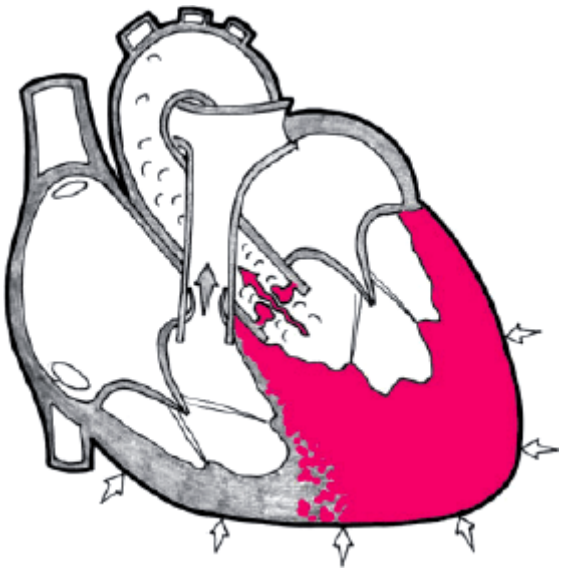
24. Noise in mitral stenosis: the mesodiastolic component smoothly passes into the presystolic one. SI - I heart tone, SII-II heart tone. The explanation is in the text.

Systolic noise in stenosis of the aortic mouth is heard in the second intercostal space to the right of the sternum when the patient leans forward (Fig. 25, 26, 27). Since this defect is characterized by a rough and loud (sawing, scraping) noise, it can be determined during auscultation over the entire area of the heart and carried out in the interscapular space. If the aortic stenosis is pronounced, and the contractile function of the left ventricle is preserved, usually rough in its timbre, loud noise, accompanied by systolic tremor. It is also performed on the carotid arteries, sometimes along the left edge of the sternum to the top. In the event of heart failure, the noise can significantly decrease and become softer in timbre. Sometimes it can be clearly heard at the top of the heart, where it can be even louder than at the base of the heart.



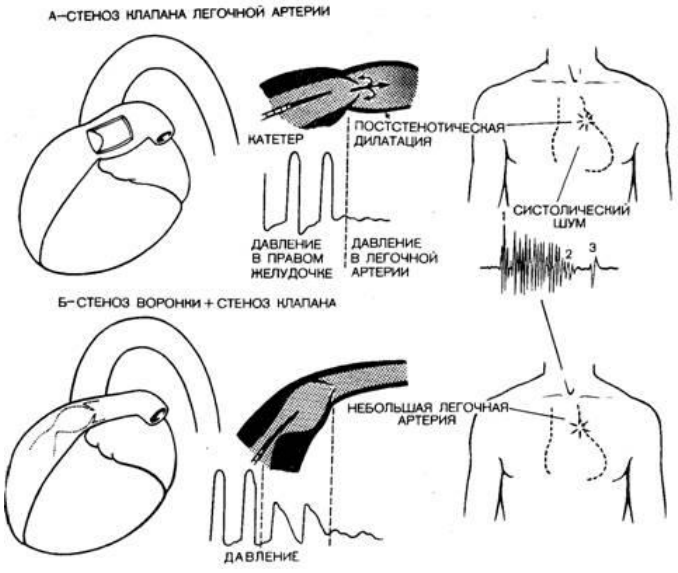
25. Mesosystolic ejection noise in aortic stenosis. SI - I heart tone, III heart tone. The explanation is in the text.





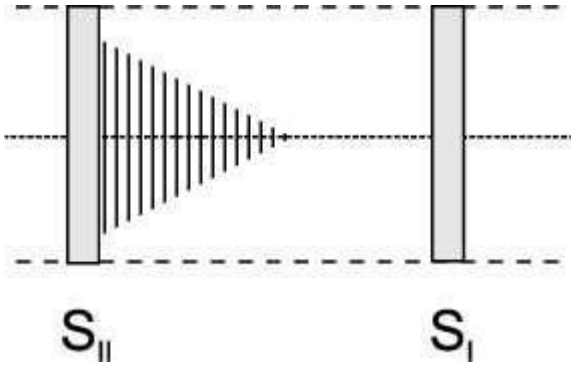
27. Hemodynamic disorders in aortic stenosis.

In pulmonary artery stenosis, the systolic ejection noise is similar to that in aortic stenosis, but it is better heard in the second and third interstices on the left (Fig. 28). The noise is carried out on the left shoulder and neck.



Diastolic noise in aortic valve insufficiency, often blowing protodiastolic noise (Fig. 29, 30) of varying intensity is better heard in the second intercostal space on the right and in the fifth point (Botkin-Erb point), where it is carried out in the course of the reverse blood flow from the aorta to the left ventricle. With a weak diastolic noise, it can sometimes be heard only when holding the breath on the exhalation, in the patient's position with a forward tilt. Since the noise

is highly tonal, it is necessary to use a phonendoscope (with a membrane), pressing it tightly against the chest wall.



29. Early diastolic murmur in aortic insufficiency. SI - I heart tone, SII-II heart tone. The explanation is in the text.

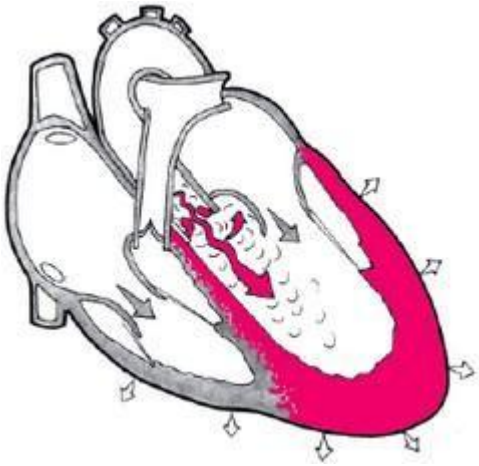
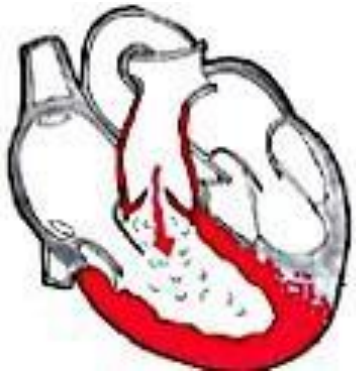


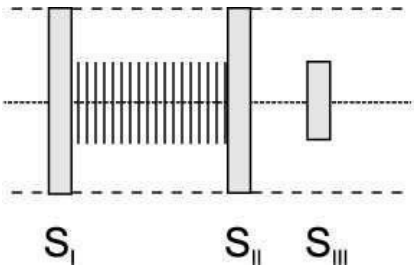
Fig. 30 Hemodynamic disorders in aortic valve insufficiency.

In case of insufficiency of the pulmonary artery valve (Fig.31), which occurs due to the expansion of the fibrous ring of the pulmonary trunk valve with severe pulmonary hypertension, diastolic noise is heard in the second intercostal space to the left of the sternum.

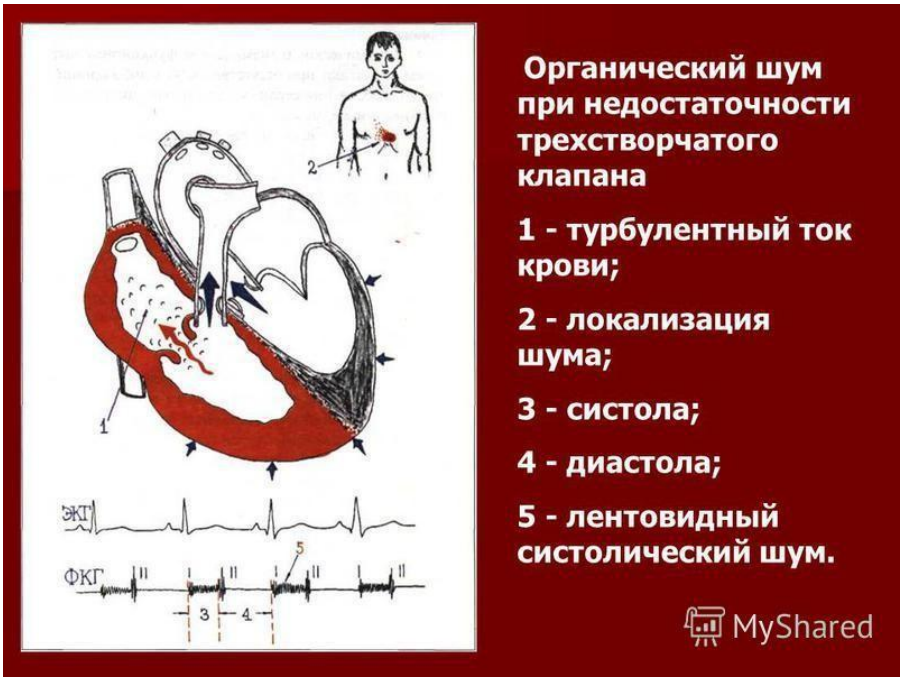


31 Hemodynamic disorders in pulmonary artery valve insufficiency.

Systolic murmur in the case of insufficiency of the right atrioventricular (tricuspid) valve is usually diastolic (Fig. 32, 33), it is better heard above the right ventricle of the heart at the left edge of the sternum in the fourth intercostal space (the lower part of the left edge of the sternum). This noise is not characterized by radiation to the axillary region, but unlike mitral insufficiency, the intensity of the noise increases slightly during inspiration. From here, it can be carried up and to the right, towards the right atrium.



32. Pansystolic noise in tricuspid valve insufficiency. SI - I heart tone, SII-II heart tone. The explanation is in the text.



With a rare defect-narrowing of the right atrioventricular orifice-diastolic noise is heard in a limited area at the base of the xiphoid process of the sternum (Fig. 34)

34 Hemodynamic disorders in right atrioventricular stenosis.

It is necessary to take into account the position of the patient when listening to noises, in which position the patient is better listened to noise.Systolic symptoms associated with insufficiency of the atrioventricular valves or with narrowing of the mouths of the main vessels are better listened to in the supine position, as this facilitates the flow of blood from the ventricles and increases the speed of blood flow. Diastolic noises that occur with narrowing of the atrioventricular openings or with insufficiency of the aortic valve or pulmonary trunk are easier to listen to in an upright position of the patient, since this facilitates blood flow to the ventricles from the atria or from the vessels (with insufficiency of the valves of the corresponding vessels) and increases its speed.

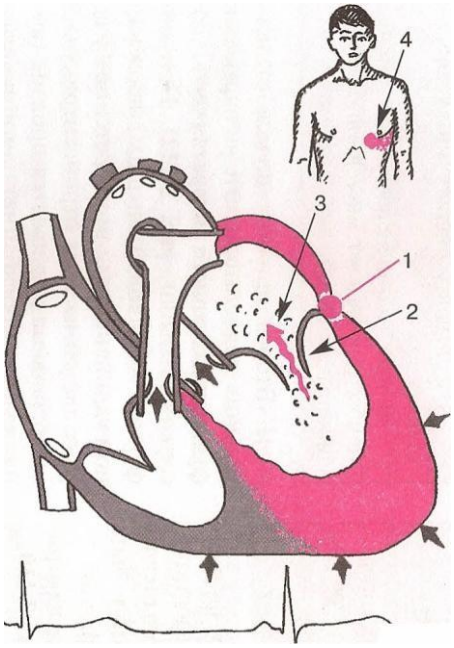
A special group is represented by functional noises of relative valve insufficiency or relative stenosis of valve openings. They can be caused by three groups of reasons:

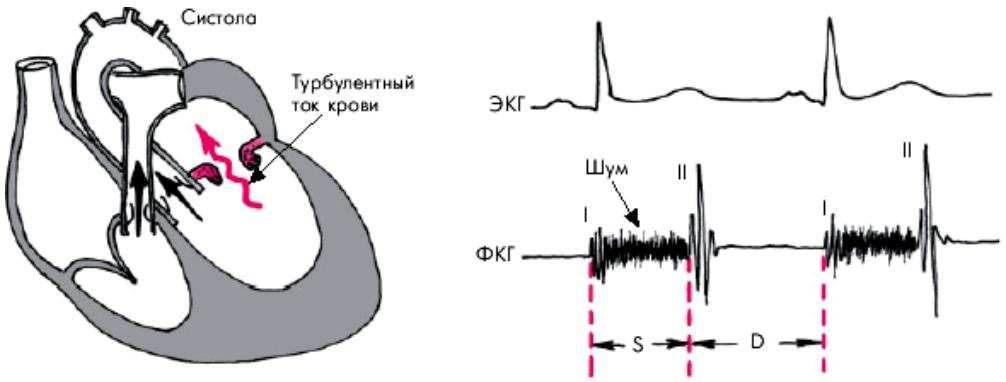
1. The expansion of the fibrous ring of the atrioventricular valves with pronounced ventricular dilatation leads to incomplete closure of the anatomically unchanged atrioventricular valve flaps (2) and the development of relative insufficiency of these valves with a turbulent flow of blood from the ventricle to the atrium. The characteristic of the relative insufficiency of the mitral (Fig.35, 36) and tricuspid valves (Fig.37 A) in these cases is similar to that of the corresponding organic defects (see above).

35. Functional systolic noise of relative insufficiency of the mitral valve caused by the expansion of the fibrous ring.

1-expansion of the fibrous ring; 2 - loose closure of anatomically unchanged valve flaps;

3-turbulent blood flow; 2-systolic noise.





37. A. Functional systolic noise of relative insufficiency of the tricuspid valve caused by the expansion of the fibrous ring. The designations are the same as in Figure 35.

Figure 37B. Functional systolic noise of relative insufficiency in mitral valve prolapse.

A. B 1-papillary muscle;

2-prolapse of one of the flaps

mitral valve;

3-turbulent blood flow

(regurgitation)from LV to LL;

4-systolic click;

5-meso and post-mortem

systole; 6-systole;

. 7-chord.

Remember: Relative mitral valve insufficiency caused by the expansion of the fibrous ring can develop with dilatation of the left ventricle in patients with:

a) arterial hypertension of any origin;

b) with aortic heart defects, mainly in the stage of decompensation (the so-called mitralization of the aortic defect);

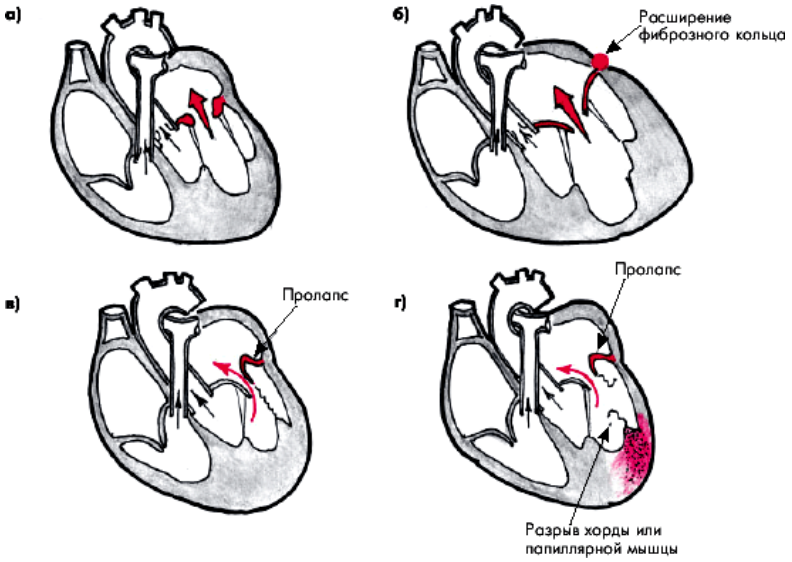
c) with heart failure of any origin (due to myogenic dilatation of the left ventricle).

Relative insufficiency of the tricuspid valve, due to the expansion of the fibrous ring, can develop in patients with significant dilatation of the right ventricle:

a) in the late stages of mitral stenosis;

b) with decompensated pulmonary heart (due to myogenic dilatation of the right ventricle).

2. The second reason for the noise of relative insufficiency is a violation of the function of the valvular apparatus (chords and papillary muscles, Fig. 37 B, 38). In case of papillary muscle infarction, congenital elongation or acquired rupture of one of the chords of the atrioventricular valves, prolapse (protrusion, deflection) of one of the flaps of the atrial cavity develops during ventricular systole. This leads to incomplete closure of the valves and the development of relative insufficiency of the atrioventricular valve. In this case, a short systolic noise is heard (more often meso - or late systolic), usually with 1 tone preserved.

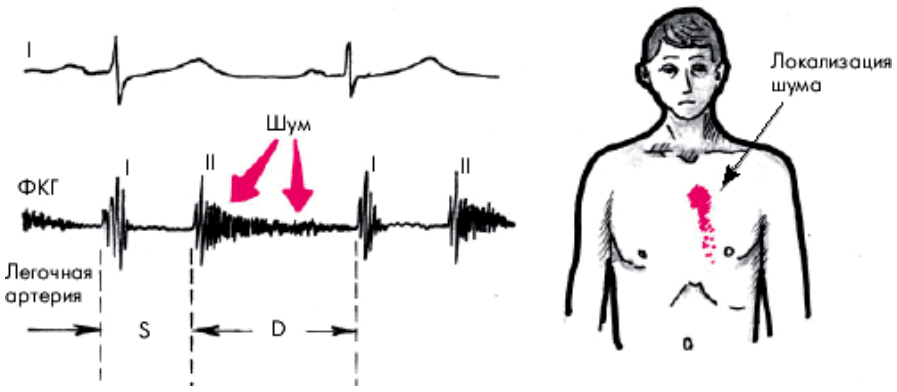


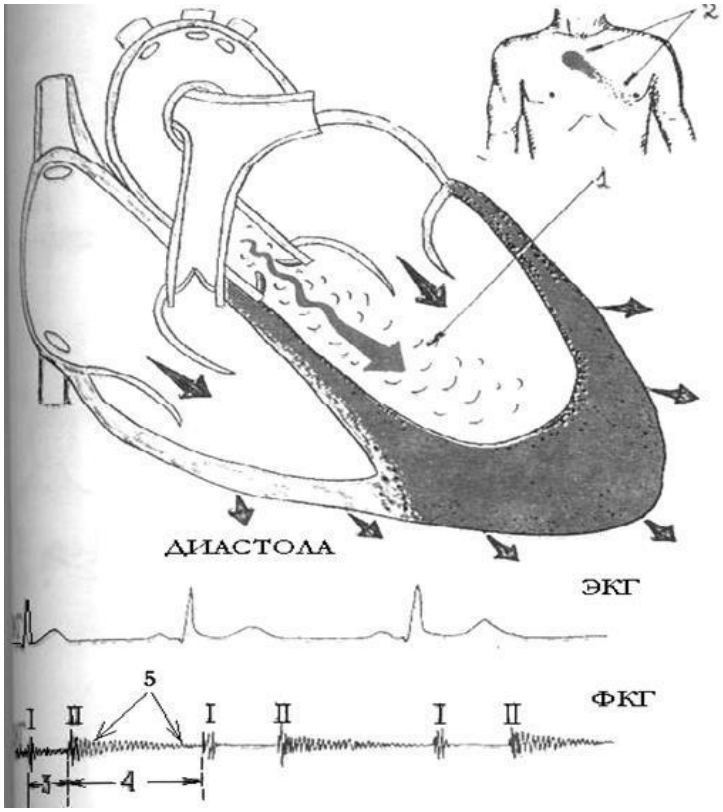
3. The third cause of functional noises of relative insufficiency of the valves or relative stenosis of the valve openings is hemodynamic displacement of the valve flaps, dilation of the aorta or pulmonary artery, and some others.

Graham-Still noise (Fig. 39, 40) is a functional diastolic noise caused by a pulmonary artery valve insufficiency that occurs with prolonged pressure increase(1) in the pulmonary artery(for example, in patients with mitral stenosis, primary pulmonary hypertension, and pulmonary heart). In the second intercostal space to the left of the sternum and along the left edge of the sternum, a quiet, decreasing diastolic noise (4) is heard, starting immediately with the SECOND tone.

Flint noise (Fig. 41) is a presystolic noise of relative (functional) stenosis of the left atrioventricular orifice, which sometimes (rarely) occurs in patients with organic aortic valve insufficiency due to the lifting of the mitral valve flaps (2) by a strong stream of blood regurgitating during diastole from the aorta to the LV (1). This results in obstructed blood flow from the left atrium to the left ventricle (3) during the active ventricular filling phase (4). At the same time, at the apex of the heart, in addition to the wired organic diastolic noise of aortic insufficiency (5), there is also a presystolic noise amplification (6) - noise

Flint. This noise is usually a gentle blowing, starting immediately without a pause after the second heart tone.





The presence of Austin Flint noise indicates the presence of significant (at least moderate) aortic insufficiency. Austin Flint is an American physician who lived in the nineteenth century (Figure 43)

Coombs noise (Fig. 44, 45 A)• is a functional mesodiastolic noise caused by relative stenosis of the left atrioventricular orifice, which occurs in patients with severe organic mitral valve insufficiency with significant dilatation of the left ventricle (LV) and left atrium (LP) and lack of expansion of the fibrous ring of the valve (2). In this case, the heart (LV and LP) resembles an hourglass in shape with a relatively narrow bridge in the area of the left atrioventricular foramen (2). At the time of emptying the LP in the phase of rapid filling, this opening becomes relatively narrow for a short time for the increased volume of blood in the LP (1) and there is a relative stenosis of the left atrioventricular opening with a turbulent flow of blood from the LP to the LV (3). At the top of the heart, in addition to the organic systolic noise of mitral insufficiency (6), you can listen to a short and quiet mesodiastolic noise due to functional mitral stenosis (7, Fig. 44. Fig. 45 A)

Functional systolic noise.Functional systolic murmur of relative stenosis of the mouth of the aorta, with organic insufficiency of the aortic valve (Fig. 45. B). Noise occurs due to a significant increase in stroke volume of blood pumped into the aorta by the left ventricle in the time of exile (1) for which the normal-Noe

unmodified opening of the aortic valve (2) is relatively narrow - formed relative (functional) stenosis of the mouth of the aorta with the turbulent flow of blood

from the left ventricle to the aorta (3). On the aorta and at the Botkin-Erb point, in addition to the organic diastolic noise of aortic insufficiency (6), during the period of expulsion (4), a functional systolic noise of relative stenosis of the aortic mouth is heard, which is

carried out on the vessels of the neck.

Differential diagnosis of heart murmurs.

If several noises are heard simultaneously over different valves, it is necessary to decide how many valves are affected and what the nature of this lesion is. The presence of systolic and diastolic noises over one of the valves indicates a combined lesion, i.e., the existence of both valve insufficiency and stenosis of the opening. In cases where a systolic murmur is heard over one of the valves and a diastolic murmur is heard over the other, there is usually a combined lesion of the two valves.

It is more difficult to decide whether one valve is affected or two if a noise is heard at different points in the same phase of cardiac activity. In this case, you need to pay attention to the nature of the noise. If a soft, blowing noise is heard in the area of one valve, and a rough, scratching noise is heard over the other, then we are talking about different noises over the two affected valves. Moving the stethoscope along the line connecting the valves over which the noise is heard, also note the change in its volume. If in any place the noise is interrupted or abruptly weakens, and then increases again, most often there is a lesion of two valves; the attenuation or

amplification of the noise as it approaches the second valve is usually indicative of damage to one valve. However, this can not be considered an absolute sign, since the degree of damage to the valves can be different, and then with a lower degree of narrowing, an independent noise will be heard, but less loud.

Helps to distinguish between noise and the nature of their conduct. For example, a systolic murmur in the left atrioventricular (mitral) valve insufficiency is transmitted to the axillary region; it can also be heard over the aorta, but this murmur will not be transmitted to the carotid arteries, unlike a systolic murmur associated with aortic stenosis.

By combining various techniques in the process of physical examination and monitoring the changes in the sonority of the noise, it is possible to determine its origin with a certain degree of probability. For example:

Noises originating from the right parts of the heart can be differentiated from others by increasing their sonority on the inhale and weakening on the exhale.

Noises in mitral regurgitation and DMJP are amplified when the hand is clenched into a fist.

With a Valsalva test (straining with the glottis closed), due to an increase in intra-thoracic pressure, venous flow to the heart and, accordingly, blood pressure decreases, which can lead to increased noise in obstructive hypertrophic cardiomyopathy (muscular subaortic stenosis) and a decrease in noise associated with aortic stenosis and mitral insufficiency.

Noise in hypertrophic cardiomyopathy increases during the Valsalva test and when standing up from a squat position; when squatting on the cor-points, the noise decreases.

With a rapid transition from the supine position to the standing position, the venous flow to the heart decreases, which also leads to a weakening of noise in the stenosis of the aortic valve and the trunk of the pulmonary artery.

With the introduction of nitroglycerin, blood pressure decreases due to systemic vasodilation, cardiac output increases, which increases the ejection noises in stenosis of the aortic valve or the pulmonary trunk valve and obstructive hypertrophic cardiomyopathy.

TECHNIQUES THAT FACILITATE AUSCULTATION OF THE HEART.

REMEMBER!

1. Auscultation of the heart should be performed in the patient's standing and lying position. In addition, useful information can be obtained by listening to the patient in a position on the left side and in an inclined position (Fig. 46)

2. The position of the patient on the left side facilitates the perception of auscultative phenomena associated with the mitral valve and the left ventricle, since with this turn of the trunk, the left ventricle approaches the anterior chest wall. In the left-side position, the third and fourth heart tones are better listened to (with a stethoscope without a membrane), as well as noises on the mitral valve, especially diastolic noise in mitral stenosis.

3. The position of the patient with a slight inclination of the trunk forward (while the patient should completely exhale and hold his breath) facilitates the detection of noises in the pathology of the aortic valve. In this case, it is best to use a phonendo-osprey (with a membrane).

4. It is mandatory to listen to all five auscultation points (see Figure 6-6). It is necessary to clearly assess the features of the I and II heart tones (loudness, cleavage), the accent of the II tone over the aortic and pulmonary artery valves, identify additional tones (III and IV), detect and characterize systolic and diastolic noises.

5. When auscultating the heart, it is very important to observe a strict sequence. You should not try to immediately evaluate all possible auscultative phenomena - the conclusion about their presence or absence should be made only after a purposeful search.

CONTROL QUESTIONS:

1. How many heart tones occur during cardiac activity

2. What is the phase structure of the cardiac cycle

3. How is the first tone formed?

4. How is the second tone formed?

5. How is the third tone formed?

6. How is the fourth tone formed?

7. Where can I listen to the heart tones and what is their auscultative difference?

8. Where are the auscultation points located?

9. When and in what pathological processes does the first tone change?

10. When and in what pathological processes does the second tone change?

11. What are additional heart tones and when can they be listened to?

12. How are heart murmurs divided?

13. What is related to intracardiac murmurs?

14. In what pathological processes can I listen to systolic noise?

15. In what pathological processes can you listen to diastolic noise?

16. Give the main characteristics of heart murmurs.

17. How to differentiate the heart murmurs heard during auscultation?

18. How is the Graham-Still noise formed?

19. Why is the Austin – Flint noise heard in aortic insufficiency?

20. In what situation is the Coombs noise heard?

21. When there is a functional systolic noise of relative stenosis

the mouth of the aorta?

22. How can heart murmurs be differentiated?

23. What techniques can facilitate auscultation of the heart?

Test tasks.

1. The strengthening of the 2nd tone on the pulmonary artery is characteristic of:

a) a decrease in the contractility of the myocardium;

b) increasing the contractility of the myocardium;

c) increased pressure in the large circulatory system;

d) increased pressure in the small circle of blood circulation.

2. Where the 3-fold valve is listened to:

a) xiphoid process;

b) 2 intercostal space to the right of the sternum;

c) 2 intercostal space to the left of the sternum;

d) the tip of the heart;

e) the Botkin - Erb point.

3. The noise of pericardial friction is characterized by the following signs:

a) Constant, coincides with the points of auscultation of the heart.

b) Unstable, does not coincide with the points of auscultation of the heart, is well listened to in the zone of absolute dullness of the heart.

c) It is not listened to when holding the breath, it is listened to in both phases of the cardiac cycle.

4. Where is the systolic murmur performed in aortic stenosis?

a) on the aorta;

b) to the Botkin-Erb point;

c) on the vessels of the neck;

d) on the xiphoid process;

e) in the axillary area.

5. In what phase of cardiac activity is the IV tone heard?

a) during atrial systole;

b) at the beginning of ventricular systole (stress phase);

c) in the phase of expelling blood from the ventricles;

d) at the very beginning of diastole (protodiastolic period);

e) in the phase of rapid filling of the ventricles.

6. The reasons for the bifurcation of the second tone (the appearance of a three-part rhythm) are

a) mitral stenosis;

b) insufficiency of the double-leaf valve;

c) reduction of pressure in the aorta and pulmonary artery;

d)aortic stenosis;

e) insufficiency of the aortic valves.

7. What is correct about the second tone based on the heart?

a) Normally, in an adult, the volume of the second tone above the aortic valve and the pulmonary valve is the same.

b) The accent of the second tone above the aorta occurs in arterial hypertension.

c) The weakening of the II tone above the aorta is observed in atherosclerosis of the aortic valves.

d) Increased tone II over the pulmonary artery occurs in pulmonary hypertension.

e) The weakening of the II tone above the pulmonary artery corresponds to the stenosis of the aortic mouth.

8. Bifurcation of the first tone is observed when:

but)In case of violation of intraventricular conduction.

b) Hypertension.

c) Angina pectoris.

d) Myocarditis.

9. What statement about the I and II tones is incorrect?

a) The I tone at the top of the heart is stronger, louder, longer than the II, follows after a long pause.

b) the second tone follows after a short pause, it is better listened to on the basis of the heart.

c) The I tone coincides with the apical push.

d) The apical push coincides with the second tone.

e) The pulsation of the carotid artery coincides with the I tone.

10. Name the next noise. In a patient with aortic valve insufficiency, a presystolic increase in diastolic noise is determined:

1. Flint noise

2. " the noise of the top»

3. coombs noise

4. The Graham-Still noise

5. functional noise of relative mitral valve insufficiency.

Topic 4: Pulse research.

THE PURPOSE OF THE LESSON: To teach students to conduct a study of the pulse of patients with diseases of the circulatory system. Teach the technique of palpation and auscultation of the arteries and veins, the ability to interpret the results obtained.

Tasks: teach

1. Perform an examination of the vascular system.

2. Perform palpation of the arteries.

3. Give a description of the arterial pulse.

4. Perform auscultation of the arteries and veins.

5. Interpret the tones and noises heard over the various vessels.

6. Methods of investigation of the microcirculatory bed.

The student should know:

1. Physiology of the microcirculatory bed.

2. The procedure for palpation of the arteries.

3. The main characteristics of the pulse.

4. Changes in the characteristics of the pulse in pathological conditions.

5. The technique of studying the pulse on the radial and carotid arteries.

6. The technique of auscultation of the arteries and veins.

7. Areas of listening to systolic noises above large arteries.

8. The method of studying the capillary bed.

The student must be able to:

11. Palpate the vessels.

12. Determine the properties of the pulse (rhythm, frequency, calculate the pulse deficit in arrhythmias).

13. Conduct a study of the pulse on the carotid arteries.

14. Perform auscultation of the arteries and veins.

15. Interpret the tones and noises heard on the major arteries.

16. Interpret the results of capillaroscopy.

The student must own:

6. The technique of palpation of blood vessels.

7. The method of determining the properties of the pulse on the radial artery.

8. The method of determining the tones and noises on the arteries and veins.

9. The ability to interpret the symptoms obtained during palpation and auscultation of large vessels.

Questions for monitoring the initial level of knowledge:

1. From what type of vessels is the human vascular system formed?

2. What are the divisions of the vascular system of the great circle of blood circulation?

3. When did you start conducting a pulse study?

4. What methods of pulse research exist?

5. How do you technically conduct an arterial pulse study?

6. How to calculate the arterial pulse?

7. What characteristics should be given to the arterial pulse?

8. How to properly inspect the vessels?

9. How to properly perform auscultation of the vessels?.

INFORMATION BLOCK.

Vascular examination.

The movement of blood through the vessels obeys the laws of hemodynamics. The driving force of blood flow is the pressure gradient between the arterial and venous regions of the vascular system. In an adult healthy person, the blood flow in the vessels is mainly laminar, with the central axial blood flow, consisting mainly of shaped elements, having the maximum speed, and the peripheral layers, directly adjacent to the vessel wall and consisting of plasma, having the minimum speed

Turbulent blood flow normally occurs in the places of branching and natural narrowing, and bends of the aorta and large arteries (Fig. 1. b).

The average speed of blood flow in different vascular areas depends on the degree of pressure at the beginning and end of the vessel and the amount of vascular resistance, which, in turn, is determined by the viscosity of the blood and the cross-section of the vessel. The greater the pressure gradient and/or the lower the blood viscosity and the total cross-section of the vessels in a given area, the higher the linear velocity of blood flow. In the aorta, it is maximum (50-70 cm/s), in the arteries and arterioles, the total transverse lumen and vascular resistance of which is many times greater, the average linear velocity is greater (20-40 cm/s in the arteries and about 0.5 cm/s in the arterioles). Capillaries have the largest total area of the lumen. Here, the speed of blood movement does not exceed 0.05 cm/s.

Remember!The total time of passage of" particles " of blood through the large and small circles of blood circulation is normally about 23 seconds.

So, the level of blood pressure in the terminal arteries and arterioles decreases by about 1.5-2 times compared to the pressure in the aorta, and the pulsating blood flow is gradually replaced by a continuous one.

Remember!The ability of arterioles to significantly change their lumen is the main mechanism that regulates the volume velocity of blood flow in various vascular regions and the distribution of blood to different organs.

Approximately 84% of the total blood volume is concentrated in the large circle of blood circulation; and about 16% - in the small circle and the heart (Fig.)

Blood vessels and the heart make up a single human system, so when identifying pathological symptoms from the peripheral vessels (both arteries and veins), it is necessary to remember their connection with the pathology of the heart. To assess the function of the cardiovascular system, it is possible to determine the characteristics of the peripheral pulse and arterial pressure, as well as using special instrumental methods of their study. However, it should be remembered that the detection of pathological changes in the peripheral arteries or veins is of independent importance in the diagnosis of diseases.

Arterial pulse.

Pulse (pulsus) is the rhythmic fluctuations of the artery wall caused by changes in its blood supply as a result of heart contractions, the release of blood into the arterial system and changes in pressure in it during systole and diastole. The propagation of the pulse wave is associated with the ability of the arterial walls to stretch and fall elastically, as mentioned above.

The examination of the peripheral arteries usually begins with an examination, during which it is possible to detect a visible pulsation, for example, of the carotid arteries in the neck. However, the main clinical method for assessing the condition of the arteries is the palpatory determination of the peripheral arterial pulse, performed when examining the carotid, brachial, radial, femoral, popliteal and foot arteries.

Most often, the radial artery is palpated, since it is located directly under the skin and is well felt between the styloid process of the radial braid and the tendon of the internal radial muscle. During palpation, the hands of the subject are covered in the area of the wrist joint so that the first finger is located on the back of the forearm, and the rest-on its front surface (Fig. 3). The study simultaneously on both hands is due to the fact that the pulse value on them may not be the same due to the different degree of dilation of the arterial vessels (Fig.4).

Different (unequal) pulse (pulsusdifferens) is observed when the passage is narrowed or an abnormality of the location of one of the radial, brachial or subclavian arteries, or when the subclavian artery is compressed by an aortic aneurysm, tumor, or enlarged lymph nodes. In mitral stenosis, the pulse may also be uneven, since the sharply enlarged left atrium compresses the subclavian artery, resulting in reduced blood flow and filling of the pulse on the left (Savelyev-Popov symptom).

After comparing the value of the pulse on both hands, you should proceed to the study of its properties on one hand (if the pulse is different on both hands - on the one on which the value is greater). Give an assessment of the properties of the pulse.

The rhythm of the pulse is determined by the work of the left ventricle of the heart. Normally, it is correct (regular, rhythmic, pulsusregularis), and may be incorrect (irregular, arrhythmic,

pulsusirregularis). The second is observed in atrial fibrillation and occurs as a result of random fluctuations in the arterial wall due to irregular heart contractions.

Sometimes, against the background of a normal rhythm, additional weak pulse waves are felt, followed by an extended pause (compensatory pause). This is the so-called extrasystole(extraordinary contraction of the heart). In some cases, it occurs so quickly after the main contraction of the heart that its cavities do not have time to fill with blood and it contracts idly — blood does not enter the aorta, and therefore, there is no pulse wave. When palpating the pulse, this is perceived as its loss. Extrasystoles can occur after each normal heart contraction (bigeminia), after two (trigeminia), after three (quadrigeminia) contractions, etc. This correct alternation of normal and additional contractions is called allorhythmia.

In addition, it is possible to periodically lose the pulse without extrasystolic (extraordinary) reduction. It is observed in incomplete atrioventricular blockade. These are the so — called Samoylov-Wenckebach periods. The pulse rate on the inhale and exhale may be different (increases on the inhale, slows down on the exhale). Such a respiratory arrhythmia can also be observed in healthy people, more often in young people.

In case of adhesive and effusive pericarditis (clumping of pericardial leaves or accumulation of exudate between them), the pulse waves almost completely disappear during inspiration. This pulse is called paradoxical.

The pulse rate normally corresponds to the heart rate and is on average 60-90 beats per minute. The pulse count is usually carried out within 10 seconds and multiplied by 4, or 1 minute (for arrhythmias, it is mandatory!), or half a minute. In the latter case, the result is doubled.

With tachycardia (the number of heartbeats is more than 90 per minute), there is a frequent pulse (pulsusfrequens) This happens with fever, thyrotoxicosis, myocarditis, and heart failure.

In the case of bradycardia (the number of heartbeats is less than 60 per minute), a rare pulse (pulsusrarus) is observed. An extremely rare pulse (40 beats per minute or less) occurs when the atrioventricular node is completely blocked.

In some cases, for example, in atrial fibrillation, some extrasystoles, the amount of blood released into the aorta by the left ventricle is so small that the individual pulse waves do not reach the periphery. The difference between the number of heartbeats and pulse waves is called a pulse deficit, and the pulse itself is deficient (pulsusdeficiens).

Remember! In atrial fibrillation, the number of heartbeats is always greater than the number of pulse waves. To detect a pulse deficit, it is necessary to count the number of heart contractions during auscultation and pulse waves during palpation of the pulse during a minute. However, since the number of heartbeats in arrhythmias (for example, in atrial fibrillation) may be different at different times, to more accurately determine the magnitude of the pulse deficit, the number of heartbeats and pulse beats should be counted at the same minute. This is done by two researchers.

The pulse tension is determined by the force that must be applied to the patient to completely compress the pulsating artery. This property of the pulse depends on the value of systolic blood pressure. At normal pressure, the pulse is of moderate tension. The higher the pressure, the more

difficult it is to compress the artery; this pulse is called a tense, or hard (pulsusdurus), which is characteristic of hypertension and sclerotic changes in the vascular wall. At low blood pressure, the artery contracts easily — the pulse is soft (pulsusmollis) and indicates a reduced vascular wall tone, which may be due to hypotension(a decrease in blood pressure), bleeding, etc.

Pulse filling reflects the filling of the examined artery with blood, which in turn is caused by the amount of blood that is released into the systole into the arterial system and causes the artery to oscillate. It can be good full (pulsusplenus) and bad empty (pulsusvacuus). Poor filling is caused by the same reasons as a soft pulse.

The magnitude of the pulse, i.e. the magnitude of the pulse shock, is a concept that combines its properties such as filling and tension. It depends on the degree of expansion of the artery during systole and on its decline at the time of diastole. This, in turn, depends on the filling of the pulse, the magnitude of the fluctuation of blood pressure in the systole and diastole, and the ability of the arterial wall to expand elastically. With an increase in the shock volume of blood, a large fluctuation in the pressure in the artery, as well as with a decrease in the tone of the arterial wall, the value of pulse waves increases. Such a pulse is called a large one (pulsusmagnus). On the sphygmogram, a large pulse is characterized by a high amplitude of pulse oscillations, which is why it is also called a high pulse (pulsusaltus). A large, or high, pulse is observed with aortic valve insufficiency, with thyrotoxicosis, when the value of pulse waves increases due to the large difference between systolic and diastolic blood pressure; it can appear with fever due to a decrease in the tone of the arterial wall.

A decrease in the shock volume, a small amplitude of pressure fluctuations in the systole and diastole, an increase in the tone of the artery wall lead to a decrease in the magnitude of pulse waves-the pulse becomes small (pulsusparvus). A small pulse is observed with a small or slow flow of blood into the arterial system: with narrowing of the aortic mouth or left venous opening, tachycardia, acute heart failure. Sometimes, in shock, acute heart failure, or massive blood loss, the magnitude of the pulse waves can be so insignificant that they are barely detected, and the pulse is called a filamentous pulse (pulsusfiliformis) (Fig. 47 a, b, c).

The barely palpable small soft pulse is called a filiform pulse (pulsusfili-formis).It is noted with significant blood loss, acute heart and vascular insufficiency.

In the case of severe myocardial lesions, it is possible to alternate large and small pulse waves due to the weakness of the contractile ability of the heart. Then they talk about an intermittent (alternating) pulse.

The shape of the pulse depends on the rate of change in pressure in the arterial system during systole and diastole. If the pulse wave rises quickly and falls quickly, then the amplitude of the oscillation of the vascular wall is always large. Such a pulse was called fast, jumping, fast, high (pulsusceler). It is characteristic of insufficiency of the aortic valve. The opposite of a fast slow pulse (pulsus-tardus), when the pulse wave slowly rises and slowly falls. A slow pulse is characteristic of narrowing of the aortic mouth, as it makes it difficult to expel blood from the left ventricle, and the pressure in the aorta increases slowly. The magnitude of the pulse waves decreases with this defect, so the pulse will be not only slow, but also small (pulsustardusetparvus).

If, after the pulse expansion of the radial artery, a second small expansion of it is felt (the second weak pulse wave), then they speak of a dicrotic pulse (pulsusdicroticus). It is observed when the tone of the arteries decreases, which happens with fever, infectious diseases.

In addition to these properties of the arterial pulse, there are other changes in it. Sometimes, during the period of the pulse wave decrease, a second additional wave is detected. It is associated with an increase in the dicrotic wave, which is normally not palpable and is only determined on the sphygmogram. With a decrease in the tone of the peripheral arteries (fever, infectious diseases), the dicrotic wave ages and is detected by palpation. This pulse is called dicrotic (pulsusdicroticus).

There is also a paradoxical pulse (pulsusparadoxus). Its features are the reduction of pulse waves during inspiration. It appears when the pericardial leaves are fused due to compression of large veins and a decrease in blood supply to the heart during inspiration.

After completing the study of the pulse on the radial artery, it is studied on other vessels: temporal, carotid, femoral, popliteal arteries, arteries of the back of the foot, etc. It is especially necessary to study the pulse on various arteries if their damage is suspected (with obliterating endarteritis, atherosclerosis, vascular thrombosis). The femoral artery is well felt in the groin area, easier when the hip is straightened with a slight outward turn. The pulse of the popliteal artery is felt in the popliteal fossa in the position of the patient lying on his stomach. The posterior tibial artery is palpated in the condylar groove behind the inner ankle; the arteries of the back of the foot are palpated on the back of the foot, in the proximal part of the first interplatarsal space. The determination of the pulse of the last two arteries is of great importance in the diagnosis of obliterating endarteritis.

Remember! The study of the pulse of the carotid arteries should be carried out carefully, one by one, starting with a slight pressure on the arterial wall, because of the risk of a carotid reflex, which can lead to a sharp slowdown in cardiac activity until it stops and a significant decrease in blood pressure. Clinically, this is manifested by dizziness, fainting, convulsions (carotid sinus syndrome).

Some diseases of the cardiovascular system are characterized by different values of the pulse in the upper and lower extremities. With narrowing of the isthmus of the aorta (aortic co-arctation), the magnitude of pulse waves in the lower extremities significantly decreases, while in the carotid arteries, the arteries of the upper extremities, it remains normal or even increases.

In Takayasu's disease (absence of pulse disease), in which there is obliterating arteritis of large vessels extending from the aortic arch, first of all, the pulsation of the carotid, axillary, brachial and radial arteries disappears or decreases.

Auscultation of the arteries.

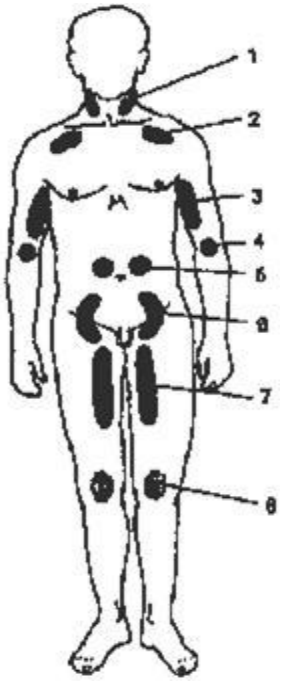
Usually, they listen to medium-sized vessels — the carotid, subclavian, femoral, popliteal arteries and the aorta. The examined artery is first palpated, then a phonendoscope is placed, trying not to squeeze the vessel to avoid the occurrence of stenotic noise.

Remember!Above the arteries, you can sometimes listen to both tones and noises that may occur in the arteries themselves or are carried to them from the heart valves and aorta. The conducted tones and noises are heard only on the arteries close to the heart-carotid, subclavian.

In healthy individuals, two tones can be heard on the carotid and subclavian arteries. The first tone is caused by the tension of the arterial wall when it expands during the passage of the pulse wave, the second-is carried out on these arteries from the aortic valve.

On the femoral artery, one tone is normally heard, due to the tension of the wall. In aortic insufficiency, the first tone is louder due to the passage of a large pulse wave. On the femoral artery with this defect, you can listen to two tones (double Traube tone) - due to fluctuations of the vascular wall in the systole and diastole.

Figure 5 shows the areas of listening to systolic noises over large arteries.



The noises heard above the arteries are more often referred to as systolic. The carotid and subclavian arteries are usually well affected by systolic murmur caused by aortic stenosis. In the same vessels, systolic noise may occur, associated with a decrease in blood viscosity and an increase in blood flow rate (with famine, anemia, and basal disease). Systolic murmur sometimes appears with narrowing or aneurysmal dilation of large vessels.

If the aortic valve is insufficient, squeezing the femoral artery with a stethoscope, you can listen to a double Vinogradov — Durosier noise above it. The first of them — stenotic noise-is caused by the flow of blood

through a narrowed stethoscope vessel. The origin of the second noise is still unclear; it is explained by the acceleration of reverse blood flow towards the heart during diastole.

Listening to the abdominal aorta along the midline of the abdomen from the xiphoid process of the sternum to the navel can reveal systolic and systolic-diastolic sounds that are caused by

stenosis or aneurysmal expansion of this department. Systolic murmur under the xiphoid process of the sternum may occur when the ventral artery is narrowed or compressed. With narrowing of the renal arteries, systolic noise is heard along the outer edge of the rectus abdominis muscles at 2.5-5.0 cm above the navel; the same noise can sometimes be heard in the lumbar region.

Auscultation of the veins.

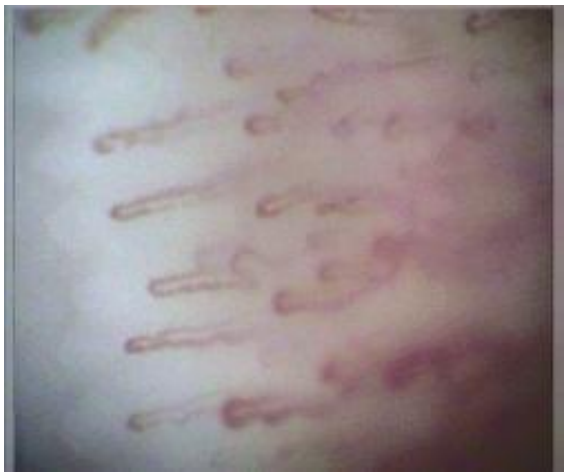
In healthy people, neither tones nor noises are usually heard above the veins. Diagnostic significance is auscultation of the jugular veins, over which the so-called gyroscope noise appears in anemia. This continuous blowing or buzzing noise is associated with an acceleration of blood flow with reduced blood viscosity in patients with anemia. It is better heard on the right jugular vein and is amplified when the head is turned in the opposite direction.

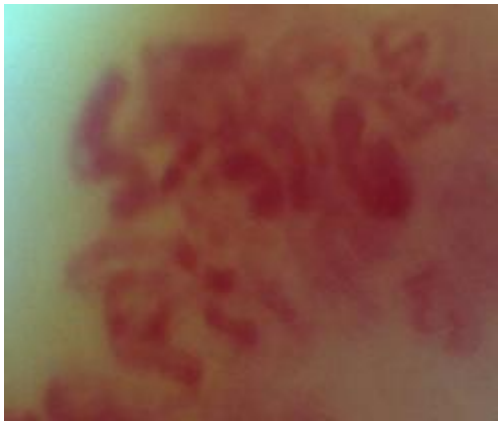
Study of capillaries.

The study of capillaries is carried out by examination, by capillary scopy and conjunctival biomicroscopy.

Capillaroscopy is a method of studying the capillaries of the intact surface of the epithelial integument (skin, mucous membrane). For capillaroscopy, you can use a small magnification of the microscope in normal daylight diffused light or use special capillaroscopes. In addition to capillaroscopy, there is a method of capillarography, which consists in photographing the capillaroscopic picture with the help of special microphotos

When capillaroscopy is most often studied the state of the capillaries of the edge of the nail bed of the IV finger. To "enlighten" the skin before the study, a drop of liquid cedar or peach oil is applied to the studied area (Fig. 7). Under normal conditions, the capillaries are visible as elongated light red loops on a yellowish-pink background. The arterial knee of the capillaries is narrower and shorter than the venous one; the passing part of the loop is usually rounded. Occasionally there are loops, twisted in the form of an eight. The number of capillaries is 16-20 in the field of view. The blood flow in them is almost indistinguishable, it occurs continuously, and in the arterial knee faster than in the venous one





Conjunctival biomicroscopy-the study of the capillaries of the eyeball using a microscope and special illumination. This study allows us to assess the state of blood flow, to identify changes in capillaries: edema and deformation of their walls, microaneurysms, etc.

The study of capillaries is used in the diagnosis of a number of diseases (atherosclerosis, hypertension, Takayasu disease, etc.).The capillaroscopic picture changes with vascular spasm, congestion, and diabetes mellitus.

CONTROL QUESTIONS:

1. Name the features of the microcirculatory bed.

2. What are the physiological features of the arterial and venous vascular systems?

3. Define the pulse.

4. What are the main properties of the pulse do you know?

5. How does the pulse rhythm change depending on the pathological processes?

6. How will the pulse rate change depending on the pathological processes?

7. What is the filling of the pulse and how does it change under different conditions?

8. What is the pulse voltage and how does it change under different conditions?

9. Under what conditions and how is the pulse deficit calculated.

10. How to perform auscultation of arteries and veins.

11. What symptoms can be detected during vascular auscultation?

12. What is capillaroscopy and why is it performed?

Test tasks.

1. Indicate the most characteristic changes in the arterial pulse in the following clinical situation: compression of large arterial trunks by an aortic aneurysm, mediastinal tumor, sharply enlarged left atrium:

1) pulsus dificiens

2) pulsus filiformis

3) pulsus differens

4) pulsus plenus

5) pulsus durus.

2. The pulse of the tardus, parvus is characteristic of:

a) tricuspid valve insufficiency;

b) mitral valve insufficiency;

c) aortic valve insufficiency;

d) stenosis of the aortic mouth;

3. In bradycardia, the pulse is called:

a) Pulsus filiformis;

b) Pulsus parvus;

c) Pulsus freguens;

d) Pulsus celler et altus;

e)+Pulsus rarus

e)Pulsus tardus et parvus;

g)Pulsus regularis;

h) Pulsus irregularis.

4. Pulsusfiliformis is noted when:

a) shock;

b) massive blood loss;

c) mitral valve insufficiency;

d) aortic valve insufficiency;

e) aortic stenosis;

f) tachycardia;

g)acute heart failure.

5. Which statement regarding pulsusdifferens is incorrect?

a) This is a delay in the pulse on one of the hands or its different value.

b) It is observed in mitral stenosis.

c) Occurs as a result of mechanical compression of the subclavian artery.

d) It is observed with an aneurysm of the aortic arch.

e) It does not depend on the heart activity and the state of the vascular system.

6. Listening to the arteries is carried out in the following areas:

a) the carotid artery is listened to above the clavicle;

b) subclavian-at the inner edge of the sternocleidomastoid muscle;

c) femoral-under the pupart ligament;

d) renal - in the parotid region on the left and right;

e) popliteal — in the popliteal fossa.

7. What clinical symptoms depend on the rapid rise and fall of diastolic pressure in the aorta?

a) double Traube and Durosier noise in peripheral vessels;

b) enhanced spilled apical push;

c) diastolic murmur;

d) capillary pulse;

e) high and rapid pulse.

8. In the diagnosis of obliterating endarteritis, the study of the pulse is of great importance:

a) on the back surface of the foot;

b) in the popliteal fossa;

c) in the groin area;

d) on the temporal artery;

e) on the carotid artery.

9. Atrial fibrillation is characterized by:

a) Pulse deficit.

b) Leaping, highpulsuscelleretaltus.

c) Slow sluggish, low-filling pulsustardusetparvus.

d) Tense solid pulsusdurus.

d)Little Softpulsusfiliformis.

10. Select the correct statements.

a) Pulse deficit is defined as the difference between heart rate and pulse, it is characteristic of atrial fibrillation.

b) Hopping, high pulsuscelleretaltus is characteristic of aortic valve insufficiency.

c) Slow sluggish, low-filling pulsustardusetparvus is characteristic of aortic stenosis.

d) A tense solid pulsusdurus corresponds to a low blood pressure.

e) Small soft pulsusfiliformis corresponds to high blood pressure.

Answers to test tasks.

TOPIC 1. 1 a. 2 b. 3a. 4 a. 5 a, b. 6 b. 7 a. 8 a. 9 b, d. 10 a, b, c, d.

TOPIC 2.1 b. 2 b. 3 b. 4 a. 5 b. 6 b. 7 b. 8 b. 9 b. 10 2.

TOPIC 3.1 g. 2 a. 3 b. 4 c. 5 a. 6 a. 7 a, b, g. 8. a. 9. g. 10 1.

TOPIC 4.1 3. 2 g. 3 d. 4 a, b, w. 5 d. 6 c, d. 7 a, d. 8 a. 9 a. 10 a, b, c.

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