LECTURE COMPLEX

Course: "Cardiovascular System in Pathology"
Course code: CSP 3304
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Lecture No. 1

1. Subject: Cardiovascular examination methods: interview, general examination, palpation, and percussion of the heart in patients with cardiovascular pathology. Examination methods for large and peripheral vessels. Cardiac auscultation in normal and abnormal conditions in patients with cardiovascular pathology. Diagnostic value.

2. Purpose: To master the methods of clinical research and semiotics of cardiovascular system lesions.

3. Lecture abstracts: Cardiovascular diseases (CVD) remain one of the leading causes of morbidity and mortality worldwide. Effective diagnosis requires a comprehensive approach that includes clinical methods: interview, general examination, palpation, percussion, auscultation, and assessment of major and peripheral vessels. These methods form the basis for establishing a preliminary diagnosis and determining the need for additional instrumental studies.

Questioning the patient (anamnesis)

The main complaints in cardiovascular diseases:

- Pain in the heart area (angina pectoris, myocardial infarction)
- Dyspnea (in case of circulatory failure)
- Heartbeat (tachycardia, extrasystole)
- Heart palpitations
- Edema (mainly in the lower limbs)
- Fainting, dizziness
- Cyanosis (bluish skin)

Medical history:

- Time of onset of symptoms
- Conditions of occurrence
- The nature and localization of pain
- Relationship of symptoms to physical activity
- Duration and frequency of attacks

Life history:

- Heredity (CVD in relatives)
- Lifestyle: smoking, alcohol, physical activity
- Nutrition, stress
- Comorbidities: diabetes, hypertension, obesity

General examination of the patient. Assessment of the general condition:

- Position (active, passive, orthopnea)
- Skin and mucous membrane color: pallor, cyanosis, acrocyanosis
- Edema
- Pulsations in the neck (jugular veins, carotid pulse)
- Shortness of breath, breathing pattern
- Drumstick-shaped fingers, watch-glass nails
- Pathological pulsation in the heart area

Examination of the cardiac apex and cardiac tremor by palpation. Palpation can detect apical flutter, general palpitations, pulsations in the cardiac region, and thoracic thrill. To locate the cardiac apex, place the palm of your right hand over the heart, with your fingers resting between the third and fourth ribs, oriented toward the axilla. The palm should press on the tip of the heart. Having located the cardiac apex, we should pay attention to its general character. Palpation examines the location of the cardiac apex, its area, strength, height, and elasticity. To do this, use the tips of the three fingers of your right hand, as mentioned above, to precisely locate the point where the cardiac apex is

located. If the cardiac impulse occupies a significant area, we select the outermost, lowest point. Ask the patient to tilt their chest forward; this will make it easier to locate the cardiac impulse.

By the area of the cardiac impulse, we mean the movement of the chest under the influence of the impulse, which under normal conditions has a diameter of 1-2 cm. If it is greater than 2 cm, it is considered disseminated, and less than 2 cm, it is considered limited. A disseminated impulse indicates an increase in the size of the heart, a condition that occurs when a person is exhausted, when the intercostal spaces are wide, when the lower part of the left lung contracts. This occurs in obesity, lung disease, and a low diaphragm.

a limited form of pushing occurs, that is, the heart meets with a smaller volume of the chest.

By the height of the heartbeat, we mean the amplitude of the ribcage's vibrations. The heart's height is divided into two parts: the upper and the lower.

When examined by palpation, the pressure exerted by the end of the heart on the fingers is called the end-of-heart force. This is caused by the contraction of the left ventricle. Like the previous two properties, the force of the impulse depends on the thickness of the chest wall and the proximity of the end of the heart to it. More importantly, it corresponds to the force of left ventricular contraction.

Palpation reveals elasticity in the heart rate, indicating that with hypertrophy, the left ventricular muscle becomes denser and more elastic. When the left ventricle descends sharply, a "dome-shaped" impulse is felt, because the heart is always tightly attached to the chest cavity.

Under normal conditions, the cardiac impulse lies between the fifth rib, 1-2 cm to the right of the midclavicular line. If the patient is lying on their left side, this point may shift 2 cm to the left, and if they are lying on the right side, 1-1.5 cm to the right. When standing upright, such changes should not occur. Non-cardiac factors also influence the displacement of the cardiac impulse. These include upward rib cage movement, changes in chest volume, protrusion of the lungs, etc.

Due to increased abdominal pressure, the thoracic septum is elevated (obesity, pregnancy, etc.). When the heart is flexed, the tip of the heart shifts to the left, rises upward, and lies horizontally. Conversely, when the thoracic septum is lowered (due to decreased abdominal pressure, pulmonary emphysema, asthenic body type, lethargy, visceroptosis), it descends. All this affects the vertical position of the three hearts, which are turned downward to the right.

Pressure can increase in one part of the pulmonary sac, which most often occurs with fluid accumulation, as in pleural effusion, unilateral hydrothorax, or hemothorax, when the heart shifts to the opposite side. At this point, the impulse of the end of the heart also shifts accordingly. When the lungs bulge, they decrease in size, and with obstructive atelectasis (malignant tumors, foreign bodies that have spread from the bronchi into the lungs)

(Lungs), the tip of the heart shifts to the side affected by the pathological processes. Enlargement and thickening of the left ventricle due to heart disease (aortic valve defect, bicuspid valve insufficiency, increased arterial pressure in the systemic circulation, atherosclerosis, cardiosclerosis) shifts the impulse of the end of the heart to the left; due to aortic valve insufficiency, it shifts to the left and downward.

In congenital anomalies - if the abdominal cavity is located on the opposite side (situs vicecrum inversus), the heart lies on the right side, so the cardiac lift is also on the right side.

What's particularly important is that if a large amount of fluid accumulates in the pericardial sac, touching the cardiac tip is not felt at all, and it doesn't correspond to relative closure. When fluid accumulates in the cavity of the left pulmonary sac (exudative pleurisy, hydrothorax, hematothorax), the flutter of the cardiac end is not felt.

When the membrane of the heart attaches to the chest, at this moment in the systole phase, it is observed that the heart moves backward rather than moving forward, calling such a push the negative impulse of the end of the heart.

In addition to the pressure on the tip of the heart, it's also important to pay attention to the cardiac impulse, which describes the function of the right ventricle. This impulse is not observed in healthy people, making it very difficult to detect. The cardiac impulse is determined by palpation of the ventricle (hypertrophy) and during expansion (dilation).

Of great diagnostic value is the definition of the symptom “cat’s purring” (flemissement sateare - cat’s purring), written by French scientists.

The reason it's called this: If you palpate the heart, you'll feel a tingling sensation similar to that felt when stroking a cat's back. This symptom occurs due to narrowing of the mitral valve during diastole, narrowing of its valve during aortic systole, or due to a defect in the pulmonary artery or Battalov's canal in the pulmonary trunk.

Cardiac percussion. Percussion determines the size, position, and configuration of the heart and vascular bundle. The right cardiac dullness and vascular bundle contour is formed from above downwards by the superior vena cava to the upper edge of the third rib, and inferiorly by the right atrium. The left contour is formed superiorly by the left part of the aortic arch, then by the pulmonary trunk, at the level of the third rib by the left atrial appendage, and inferiorly by a narrow strip of the left ventricle. The anterior surface of the heart is formed by the right ventricle. A distinction is made between relative and absolute cardiac dullness. Relative cardiac dullness is the projection of its anterior surface onto the chest and corresponds to the true borders of the heart, while absolute cardiac dullness is the projection of the anterior surface of the heart, not covered by the lungs. Percussion can be performed with the patient in a horizontal or vertical position.

Determining relative cardiac dullness. The heart rate should be moderate. The pleximeter finger should be pressed firmly against the chest wall. The outermost points of the cardiac contour are found, first on the right, then on the left, and finally, from above. First, the lower border of the right lung is determined along the midclavicular line (6th), which is normally located at the level of the 6th rib; the position of the lower border of the lung gives an indication of the level of the diaphragm. Then, the pleximeter finger is moved one intercostal space above the lower border of the right lung and placed parallel to the right border of the heart (normally at the 4th intercostal space).

Percussion is performed by gradually moving the pleximeter finger along the intercostal space, toward the heart, until a dull percussion sound is heard. The right border of the heart is marked along the outer edge of the finger facing the clear percussion sound. Normally, it is located 1 cm lateral to the right sternal border.

To determine the left border, first locate the apical impulse, then place the pleximeter finger lateral to it, parallel to the desired border, and percuss along the intercostal space toward the sternum. If the apical impulse cannot be determined, percussion should be performed in the 5th intercostal space from the anterior axillary line toward the sternum. The left border of relative cardiac dullness is located 1-2 cm medially from the left midclavicular line and coincides with the apical impulse. To determine the upper border, place the pleximeter finger perpendicular to the sternum near its left edge and move it downward until dullness is felt. Normally, the upper border of relative cardiac dullness is located at the 3rd rib. The cardiac diameter is then determined. Normally, it is 11-12 cm. The cardiac configuration is also determined. In pathological conditions, with dilation of the cardiac chambers, a distinction is made between mitral and aortic configurations.

Determining absolute cardiac dullness. For this, soft percussion is used. First, the right border of absolute cardiac dullness is determined. The pleximeter finger is placed on the right border of relative dullness parallel to the sternum and moved medially to the left until a dull sound is heard. The border is marked along the outer edge of the finger facing the clear sound. Normally, this border runs along the left edge of the sternum. To determine the left border of absolute cardiac dullness, the pleximeter finger is placed slightly outward from the border of relative cardiac dullness and moved medially until

a dull sound is heard. Normally, this border is located 1-2 cm medially from the border of relative cardiac dullness. To determine the upper border of absolute cardiac dullness, the pleximeter finger is placed on the upper border of relative cardiac dullness and percussed, moving it downward until a dull sound is heard. Normally, this border is located at the 4th rib. Next, we proceed to determining the borders of the vascular bundle. Using gentle percussion along the second intercostal space, we percuss on the right and left sides, from the midclavicular line toward the sternum. If dullness is noted, mark the outer edge of the finger. The right and left borders of the vascular bundle dullness are normally located along the edges of the sternum; its diameter is 5-6 cm. It should be noted that changes in the boundaries of cardiac dullness can be caused by non-cardiac factors. For example, with a high diaphragm, the heart assumes a horizontal position, leading to an increase in its transverse dimensions. With a low diaphragm, the heart assumes a vertical position, and its diameter decreases accordingly. Fluid or air accumulation in one of the pleural cavities leads to a shift in the boundaries of cardiac dullness toward the healthy side, while in cases of atelectasis, lung contraction, or pleuropericardial adhesions, it shifts toward the diseased side. The area of absolute cardiac dullness decreases sharply or disappears in cases of pulmonary emphysema. An increase in the area of absolute cardiac dullness also occurs with anterior displacement of the heart, for example, by a mediastinal tumor, with fluid accumulation in the pericardium, or with right ventricular dilation. A rightward shift in the boundaries of relative cardiac dullness causes dilation of the right atrium and right ventricle. It should be remembered that a markedly enlarged and hypertrophied right ventricle, displacing the left ventricle, can also shift the boundary of relative cardiac dullness to the left. Aortic dilation leads to an increase in the diameter of the dullness in the second intercostal space.

Pulse Characteristics. Blood Pressure Measurement Method. Pulse is a jerky, periodic oscillation of the walls of peripheral arteries, synchronous with cardiac systole. Pulse is usually determined by palpation, most often using the radial artery. This artery offers ideal conditions for palpation – it can be felt over a large area, is located superficially, just under the skin, and is easily compressed by the radius bone. This allows for easy compression to determine a number of pulse characteristics. It should also be noted that, if necessary, the pulse can be determined on the temporal and carotid arteries, and on the dorsum of the foot and posterior tibial artery in the legs. A few practical tips for measuring the pulse: first, the pulse should be felt with not one, but three fingers (index, middle, and ring), placed along the vessel. This method makes the pulse easier to detect, and its properties can be studied by squeezing with the middle finger and assessing the moment of its disappearance with another finger. Secondly, it's essential to establish a rule: begin checking the pulse on both arms simultaneously. This allows one to rule out (or suspect) a number of conditions (for example, pulseless disease, etc.). If the pulse is uniform on both arms, then further examination continues on one arm.

Determine the pulse rate. Rate is one of the properties with which a physician begins to examine the pulse. In healthy people, the pulse rate normally ranges from 65 to 80 beats per minute (about 70 for men and 80 for women). The pulse can fluctuate between acceleration and deceleration, reflecting the corresponding contractions of the heart, as indicated by the Latin names for these conditions: acceleration (tachycardia) and deceleration (bradycardia). In addition to these terms, there are also purely technical names: a rapid pulse (pulsus frequens) and a slow pulse (pulsus rarus). A rapid pulse, or tachycardia, can be a physiological phenomenon or a symptom of a disease. Physiological tachycardia is observed during physical and mental exertion, while pathological tachycardia is observed in a wide variety of conditions, including endocarditis, myocarditis, thyrotoxicosis, anemia, infectious diseases, and so on. It's important to remember that a one-degree increase in temperature is accompanied by an increase in heart rate of 8-10 beats per minute. From this, we can conclude that a rapid pulse carries general information about some cardiovascular damage. However, an increased

pulse rate can also serve as an important specific diagnostic indicator. For example, it is one of the leading symptoms of myocardial insufficiency, paroxysmal tachycardia, and some other conditions. A slow pulse (less than 60 beats per minute) can also be physiological or pathological. Physiological bradycardia is observed in some completely healthy individuals. This phenomenon can be a sign of vagotonia, but it also occurs in healthy, trained individuals (athletes). Bradycardia can also be a symptom of many different diseases, including cardiac conduction block, decreased thyroid function (myxedema), and increased intracranial pressure. Pulse rhythm is another important indicator. Normally, pulse waves follow at regular intervals—the pulse is rhythmic (p. regularis). In a number of pathological conditions, this regularity is disrupted, resulting in an arrhythmic, irregular pulse (p. irregularis). The two most common types of arrhythmia are extrasystole and atrial fibrillation, which can be detected by palpation. Extrasystole is a feeling of unscheduled contractions, often followed by an unusually long interval (called a compensatory pause). Extrasystole is quite common, sometimes as a harmless neurogenic disorder, while in others it is a sign of serious heart damage (for example, myocardial infarction, myocarditis). With atrial fibrillation, completely irregular pulse beats are palpated (atrial fibrillation is sometimes called complete arrhythmia). Atrial fibrillation is not an independent disease, but a symptom of a number of primary pathological processes (heart defects, most often stenosis of the left venous orifice, hyperthyroidism, atherosclerotic cardiosclerosis, etc.). It is necessary to determine the pulse tension—a property that provides information about the state of the vascular system. Unfortunately, there are no objective criteria for normal tension. A concept of normal pulse tension is developed through practice, palpating the pulse of healthy people. A sharply tense pulse, when it becomes hard, is called the p. durus. This pulse is difficult to compress and may be a sign of high blood pressure (hypertension of various origins) or arterial sclerosis. A decrease in tension and slight compressibility of the pulse may indicate a decrease in blood pressure. In this case, one speaks of a soft pulse – p. mollis. Describe the fullness of the pulse. A distinction is made between a well-filled pulse, or full pulse (p. plenus), and a poorly filled pulse, or empty pulse (p. vacuus). A poorly filled pulse often reflects low blood pressure and is a leading sign of acute vascular insufficiency, along with low blood pressure. In severe cardiovascular insufficiency, a pulse change is observed that combines high tachycardia, very poor fullness, and tension. This pulse is barely palpable and is called a filiform pulse. This peculiar pulse is observed in cases of aortic valve defects.With aortic valve insufficiency, a large volume of blood enters the aorta during systole, which immediately spills back into the ventricle. This results in a high but rapidly falling pulse (p. celler et altus). Conversely, with stenosis of the aortic orifice, filling of the vascular system is impeded, resulting in a slow pulse (p. tardus). Blood pressure is formed primarily due to the hydrodynamic effect of blood on the inner walls of the circulatory system and is maintained by cardiac contractions and complex neurohumoral mechanisms. A distinction is made between arterial and venous blood pressure. Arterial pressure is the pressure of blood in the arterial limb of the vascular system. It is the main part of the circulatory system and ensures all vital functions of the body. Venous pressure is the pressure in the lower limb of the circulatory system, having a comparable value and primarily ensuring the return of blood to the heart.

Blood pressure measurement. Measuring blood pressure in humans became widespread only after the advent of indirect methods, and especially after Riva-Rocci proposed a device (sphygmomanometer) in 1896, the basic design principles of which are preserved in all modern blood pressure measuring devices. Until 1905, only maximum pressure was measured by palpation. 11 Various devices are used to measure blood pressure, including the Riva-Rocci mercury sphygmomanometer, spring-loaded devices, and electronic devices called tonometers. In both cases, the results are expressed in mmHg.

There are certain rules for measuring blood pressure that guarantee the accuracy of the results.

1. The cuff is placed on the patient's bare shoulder, 2-3 cm above the elbow. The cuff should be placed so that there is one finger between it and the shoulder.
2. The arm should be extended, palm up, fingers extended, and muscles relaxed. If the blood pressure is being measured while the patient is sitting, it's best to place a book or rolled-up towel under the elbow.
3. It is necessary to feel the pulse in the elbow area and apply the head of the phonendoscope to this place.
4. Inflate the cuff until the blood pressure in it is 20 mmHg higher than the level at which audible sounds disappear.
5. Open the screw gradually and release it so as to ensure a smooth, continuous movement of the pressure gauge needle along the scale.
6. The appearance of the first sounds corresponds to systolic pressure, and the transition from clear to muffled sounds and their disappearance corresponds to diastolic pressure. When making a final blood pressure assessment, the circumference of the arm should also be taken into account. In thin people, the pressure will be lower than the true value. Therefore, for an arm circumference of 15-30 cm, it is recommended to add 15 mmHg to the systolic pressure, and for a circumference of 45-50 cm, subtract 25 mmHg from the resulting figure. Normal blood pressure values in adults are fairly stable, but depend on age. For people aged 15-50, normal blood pressure falls within the following figures: a maximum of 90-130 and a minimum of 70-79 mmHg. For people over 50, normal blood pressure can be within the range of 140/90 mmHg. In addition to the maximum and minimum, a distinction is also made between pulse pressure - the difference between the minimum and maximum pressure. Normally, pulse pressure is 40-50.

AUSCULTATION OF THE HEART HEART SOUNDS IN NORMAL AND PATHOLOGICAL CONDITIONS.

Mechanism of tone formation I tone — the main component — valvular (oscillations of the atrioventricular valve cusps, both when they slam at the beginning of systole, and when they oscillate in the isometric contraction phase, when these valves are closed); — muscular (ventricular myocardial tension during isometric contraction); — vascular (oscillations of the initial sections of the aorta and pulmonary trunk when they are stretched by blood in the ejection phase); — atrial (contraction of the atria). Duration of the first tone is 0.08-0.12 sec. 20 II tone — occurs due to oscillations that occur at the beginning of diastole when the semilunar cusps of the aortic and pulmonary trunk valves slam. Duration — 0.05-0.08 sec. To correctly evaluate auscultation data, it is necessary to know the projection sites of the valves on the chest wall, as well as where the sounds emanating from a particular valve are best heard. The projection sites of the valves on the anterior chest wall are: mitral - on the left near the sternum in the area of attachment of the 3rd rib; tricuspid - on the sternum, midway between the attachment of the 3rd rib on the left and the cartilage of the 5th rib on the right to the sternum; pulmonary valves - in the middle of the sternum at the level of the 3rd costal cartilages. With such a close location of the valves from each other, listening to the heart in the places of true projection, it is difficult to determine which of them is affected. Sound perception depends not only on the proximity of the valve projection, where sound vibrations originate, but also on their conduction through the blood flow, on the proximity to the chest wall of the part of the heart in which these vibrations are generated. Therefore, the following areas on the chest are distinguished for the best auscultation of sound phenomena associated with the activity of each valve:
1. Mitral valve – the area of the apical impulse (since the vibrations are well conducted by the dense muscle of the left ventricle and the apex of the heart is closest to the anterior chest wall);
2. Tricuspid valve - the lower end of the sternum, at the base of the xiphoid process (right ventricular region);

3. Pulmonary trunk valve – coincides with the true projection of the valve – II intercostal space to the left of the sternum;
4. Aortic valve a) 2nd intercostal space to the left of the sternum (where the aorta comes closest to the anterior chest wall); b) to the left of the sternum, at the attachment site (3rd-4th ribs) (Botkin-Erb point) Rules of auscultation: the heart should be listened to in various positions (if the patient's condition allows) - lying down, standing, after physical activity. Pathology of the mitral valve is easily detected when the patient is lying on the left side, lesions of the aortic valve - 21 are better detected when auscultating the patient in an upright position or on the right side. It is easier to listen to the heart when holding the breath, after a deep inhalation and subsequent deep exhalation (so that breath sounds do not interfere). The order of auscultation (in order of decreasing frequency of valve lesions).
- The mitral valve is at the apex of the heart.
- Aortic valve – II m/r on the right, Botkin-Erb point.
- Pulmonary valve – II m/r on the left. 4. Tricuspid valve – at the base of the xiphoid process of the sternum.

Distinctive features of the first and second heart sounds:
1. Weakening of both tones due to: a) extracardiac causes – obesity, pronounced musculature, pulmonary emphysema, hydrothorax, pericardial effusion, etc.; b) damage to the heart muscle – myocarditis, cardiosclerosis, myocardial infarction, left ventricular aneurysm, myocardial dystrophy.
2. Increased intensity of both tones: asthenic chest, high position of the diaphragm, wrinkling of the lungs, sudden weight loss, after physical exertion, with tachycardia, Graves' disease, some intoxications and cardiac hypertrophy.
3. Weakening of the first heart sound at the apex: a) valvular heart defects (most common) – mitral and aortic valve insufficiency (with these defects, the period of closed valves is absent due to the presence of defects in the valve cusps, the surface and oscillatory movements of the wrinkled cusps are reduced, the muscular component is weakened); b) damage to the heart muscle; c) insufficiency of the tricuspid valve and pulmonary valve (occurs much less frequently).
4. Increased first heart sound at the apex: a) mitral stenosis (due to less filling of the left ventricle with blood during diastole and its faster contraction during systole) – increase and shortening of the first heart sound, “flapping first heart sound”; b) with complete artioventricular block – Strazhesko’s “cannon sound” (with simultaneous contraction of the atria and ventricles).
5. Weakening of the second point on the aorta (in aortic heart defects): a) insufficiency of the aortic valves (due to partially destroyed semilunar cusps, decreased oscillations due to the development of cicatricial compaction), weakening of the second point on the aorta is directly proportional to the degree of insufficiency of the aortic valve; b) narrowing of the aortic orifice (due to decreased pressure in the aorta).
6. Weakening of the second wave on the pulmonary artery – occurs very rarely (with insufficiency of the pulmonary artery valves and narrowing of its orifice).
7. Strengthening (accent) of the 2nd sound. Normally, in adults, the strength of the 2nd sound on the aorta and pulmonary artery is the same during auscultation and comparison (since the pulmonary artery valve is located closer to the chest than the aortic valve, therefore the transmission of sound phenomena from them is equalized). In childhood, the 2nd sound on the pulmonary artery is often stronger than on the aorta, since the pressure in the aorta in children is lower than in adults, and the pulmonary artery is located closer to the chest. The strength of the 2nd sound depends on the force of the blood push against the aortic valve cusps in the pulmonary artery during diastole, and usually goes parallel to the height of blood pressure. a) accentuation of the 2nd sound on the aorta - with increased blood pressure, atherosclerosis, physical exertion, anxiety; b) the emphasis of the second

wave on the pulmonary artery indicates an increase in blood pressure in the pulmonary circulation - both in heart diseases (mitral valve defects, patent ductus arteriosus) and lung diseases (emphysema, pneumosclerosis, pulmonary tuberculosis), with deformation of the chest (kyphoscoliosis, etc.).

8. Splitting and bifurcation of tones – when, instead of one tone, two short tones are heard, following each other within a short period of time. Bifurcation of tones can be physiological (in young people, usually associated with breathing or physical activity, and is constant).

The bifurcation of the first wave is caused by the non-simultaneous closing of the bicuspid and tricuspid valves: under physiological conditions - during exhalation, under pathological conditions - during blockade of one of the legs of the bundle of His, when non-simultaneous contraction of the right and left ventricles of the heart occurs.

Bifurcation of the second heart sound in the pulmonary artery and aorta (due to increased blood pressure in the pulmonary circulation). It may occur "as a temporary phenomenon in children and nervous individuals with unstable autonomic dysfunction on the heart and pulmonary circulation" (N.D. Strazhesko). Pathological bifurcation of the second heart sound is associated with mitral valve disease (primarily stenosis).

Gallop rhythm is a three-part rhythm that occurs in the diastolic phase, with an additional pathological third heart sound. It is heard in cases of severe myocardial damage (myocardial infarction, left ventricular aneurysm, cardiosclerosis). The ratio of murmur to cardiac activity phase is:

1. A systolic murmur is heard after a long pause between the first and second sounds, with the first sound being weakened or even absent. It can be functional or organic. The latter is heard in acquired heart defects (mitral insufficiency, tricuspid insufficiency, aortic stenosis), congenital defects (pulmonary artery stenosis, patent ventricular septum, patent ductus arteriosus, etc.), and pathological processes in the aorta (atherosclerosis, aortic aneurysm).

2. Diastolic murmur is heard during a long pause between the second and first heart sounds; the time of its appearance does not coincide with the apical impulse.

There are three types: 1) protodiastolic (at the beginning of diastole); 2) mesadiastolic (in the middle of diastole); 3) presystolic (at the end of diastole). This murmur is extremely rarely functional. It is heard in the following valvular heart defects: with narrowing of the left atrioventricular orifice, narrowing of the right atrioventricular orifice, with aortic valve insufficiency, with pulmonary valve insufficiency.

MECHANISM OF NOISE GENERATION.

The occurrence of intracardiac murmurs can be explained by the physical laws of fluid flow through the tube. The following factors are important for the development of murmurs in the tube: 1) the measurement of the tube lumen (mainly its narrowing); 2) the velocity of fluid flow; 3) the composition of the fluid. Murmur characteristics. Since heart murmurs can occur during various phases of cardiac function and with damage to various heart valves, it is necessary to determine the following properties during auscultation:

1) in what phase of cardiac activity the noise occurred (in systole, diastole or both phases);
2) the place where it is best to listen to the heart murmur;
3) where it is held;
4) his character;
5) his strength

A clinical examination is a crucial step in diagnosing cardiovascular diseases. A thorough medical history, thorough examination, and cardiac auscultation allow us to identify most pathologies before additional testing is necessary. These skills remain the foundation of practical medicine and are essential for every physician.

4. Illustrative material: presentation
5. Literature: indicated on the last page of the syllabus
6. Test questions:
1) What is angina pectoris?
2) What are the causes of cardiac hump?
3) What is the visible apex impulse formed by and where is it localized?
4) How is relative cardiac dullness determined?
5) What is the definition of absolute cardiac dullness?

Lecture No. 2
1. Subject: Leading clinical syndromes: high blood pressure and coronary heart disease in patients with cardiovascular disease. Main causes, risk factors, clinical presentation, classification, laboratory and instrumental methods for investigating arterial hypertension and coronary heart disease.
2. Objective: To teach students to identify the main symptoms of arterial hypertension, its causes, and coronary heart disease. Explain the mechanism of symptom development and identify typical changes in laboratory and instrumental examinations. To teach students how to collect primary and secondary complaints and a patient's medical history, as well as the methodology for conducting a targeted examination of patients with arterial hypertension and coronary heart disease, and to identify typical symptoms and changes in laboratory and instrumental examinations.
3. Lecture abstracts:
Cardiovascular diseases (CVD) are the leading cause of death worldwide. The most common of these diseases include:

- Arterial hypertension (AH)
- Coronary heart disease (CHD)

These pathologies are closely related: hypertension is one of the main risk factors for the development of coronary heart disease, and their combination aggravates the course of the disease and increases the risk of complications (myocardial infarction, stroke, heart failure).
Arterial hypertension (AH)
Definition: Arterial hypertension is a persistent increase in blood pressure (BP) ≥140/90 mmHg during at least two visits to the doctor.
Etiology (main causes):

- Primary (essential) hypertension—In 90–95% of cases, the cause is not established.
- Secondary (symptomatic)— 5–10%:
  - Kidney diseases (glomerulonephritis, polycystic kidney disease)
  - Endocrine (pheochromocytoma, hyperthyroidism, Itsenko-Cushing syndrome)
  - Coarctation of the aorta
  - Taking medications (oral contraceptives, NSAIDs)

Risk factors:

- Heredity
- Obesity
- Hypodynamia
- Smoking, alcohol
- Salt in the diet
- Stress
- Diabetes mellitus
- Age (>55 years for men, >65 years for women)

Clinical picture:Often asymptomatic in the early stages!

- Headache (especially in the morning)
- Dizziness
- Tinnitus
- Flickering spots before the eyes
- Pain in the heart area
- Decreased performance, irritability

Classification of hypertension (by degree):

Degree	SBP (mmHg)	DBP (mmHg)
I	140–159	90–99
II	160–179	100–109
III	≥180	≥110

By risk:

- Low, moderate, high, very high risk (assessed taking into account risk factors, target organ damage and comorbidities)

Complications:

- Left ventricular hypertrophy
- Stroke
- Myocardial infarction
- Chronic heart failure
- Renal failure
- Aortic aneurysm

Research methods:

Clinical:

- Blood pressure measurement (dynamically)
- Examination, palpation, auscultation of the heart

Laboratory:

- General blood and urine analysis
- Biochemistry: creatinine, urea, glucose, cholesterol, potassium, sodium
- Albuminuria test

Instrumental:

- ECG—detection of LV hypertrophy
- Echocardiography- confirmation of hypertrophy, assessment of cardiac function
- Ambulatory blood pressure monitoring (ABPM)
- Ultrasound of the kidneys and adrenal glands—if secondary hypertension is suspected
- Ophthalmoscopy—assessment of retinal vessels

Coronary heart disease (CHD)

Definition:IHD is a disease caused by a discrepancy between the myocardial oxygen demand and its delivery, most often due to atherosclerosis of the coronary arteries.

The main forms of coronary heart disease:

- Stable angina
- Unstable angina
- Myocardial infarction (with or without ST elevation)
- Post-infarction cardiosclerosis
- Painless ischemia

- Sudden coronary death

Causes of coronary heart disease:

- Coronary artery atherosclerosis (main cause)
- Coronary artery spasm
- Thrombosis, embolism
- Increased blood clotting
- Anemia, tachycardia (exacerbate ischemia)

Risk factors:

- Arterial hypertension
- Hypercholesterolemia
- Smoking
- Obesity
- Diabetes mellitus
- Heredity
- Male, age >50 years
- Stress

Clinical picture (examples):

Angina pectoris:

- Pain behind the breastbone (squeezing, burning)
- Radiation to the left shoulder, arm, lower jaw
- Occurs during exertion and goes away at rest or after nitroglycerin
- Duration of pain: from 1 to 15 minutes

Myocardial infarction:

- Intense, prolonged pain (more than 20 minutes) that is not relieved by nitrates
- Sweating, fear of death
- Shortness of breath, arrhythmia, drop in blood pressure

Canadian Cardiology Society classification of angina:

Class	Characteristic
I	Pain only with heavy loads
II	Minor limitation of normal activity
III	Significant limitation of physical activity
IV	Pain at rest or with minimal exertion

Research methods:

Clinical:

- The nature of pain, provoking factors
- Auscultation (weakening of the first heart sound, noises, gallop rhythm)

Laboratory:

- Cardiac-specific enzymes:
  - Troponin I/T (increase is the main marker of MI)
  - KFK-MV, LDG
- Complete blood count (leukocytosis), biochemistry

Instrumental:

- ECG**:** ST segment changes, T wave, pathological Q
- Holter ECG monitoring
- Echocardiography—hypokinesia of zones, decreased EF
- Bicycle test/treadmill test - with angina pectoris

- Coronary angiography— the "gold standard" of diagnostics

Unifying aspects of the diagnosis of hypertension and coronary heart disease

Criterion	Arterial hypertension	Ischemic heart disease
Symptoms	Headache, tinnitus	Chest pain, shortness of breath, angina
Reasons	Vague (essential), secondary forms	Atherosclerosis, vascular spasm
Diagnostics	Blood pressure, ECG, EchoCG, ABPM	ECG, EchoCG, enzymes, coronary angiography
Treatment	Antihypertensives, diet	Antianginal, thrombolytics
Prevention	Elimination of risk factors	The same + aspirin, statins

Arterial hypertension and coronary heart disease are two of the most common and often interrelated clinical syndromes in cardiology. Early diagnosis, adequate treatment, and prevention can significantly improve the quality and length of life for patients. Physicians must be able to recognize even the "silent" forms of these diseases and be familiar with modern diagnostic methods.

4. Illustrative material: presentation

5. Literature: indicated on the last page of the syllabus

6. Control questions:

1. What blood pressure numbers indicate hypertension?
2. What changes during auscultation can be detected in patients with coronary heart disease?
3. What changes in ECG can be detected in ischemic heart disease?
4. What indicators of arterial hypertension are considered borderline arterial hypertension?
5. What are the main complaints associated with arterial hypertension and coronary heart disease?
6. What is coronary insufficiency syndrome?
7. What are the causes of coronary insufficiency?
8. What forms of coronary insufficiency do you know?
9. What is atherosclerosis?
10. What are acute forms of coronary insufficiency?

Lecture No. 3

1. Subject: Leading clinical syndromes: acute and chronic coronary insufficiency in patients with cardiovascular disease. Pericardial and myocardial diseases. Dyslipidemia. Hypotension. Main causes, risk factors, clinical presentation, classification, laboratory and instrumental methods of examining arterial hypertension and coronary heart disease.

2. Objective: To teach students to identify the main symptoms of acute and chronic coronary insufficiency and valvular disease. Explain the mechanism of symptom development and identify typical changes in laboratory and instrumental examinations. To teach students how to collect primary and secondary complaints and a patient's medical history, as well as methods for conducting targeted examinations of patients with coronary insufficiency and identifying typical symptoms and changes in laboratory and instrumental examinations.

3. Lecture abstracts:

Leading clinical syndromes in patients with cardiovascular diseases:

- Acute and chronic coronary insufficiency
- Diseases of the pericardium and myocardium
- Dyslipidemia

- Arterial hypotension
- Methods for studying arterial hypertension and ischemic heart disease

Acute and chronic coronary insufficiency

Definition:Coronary insufficiency—mismatch between myocardial oxygen demand and its delivery through the coronary arteries.

- Acute coronary insufficiency– sudden and severe disruption of the blood supply to the myocardium.
- **Chronic coronary insufficiency**– a long-term, progressive decrease in myocardial perfusion, most often due to atherosclerosis.

Reasons:

- Coronary artery atherosclerosis
- Thrombosis, spasm, embolism of the coronary arteries
- Anomalies of coronary anatomy
- Myocarditis, vasculitis
- Increased oxygen demand (tachycardia, myocardial hypertrophy)

Risk factors:

- Arterial hypertension
- Smoking
- Diabetes mellitus
- Hypercholesterolemia
- Heredity
- Stress
- Male, age >50 years

Clinical picture:

Acute form (eg myocardial infarction):

- Intense chest pain >20 minutes
- Irradiation to the left arm, neck, lower jaw
- Shortness of breath, sweating, fear of death
- Heart rhythm disturbance, decreased blood pressure

Chronic form (angina):

- Attacks of chest pain during exertion
- They pass with rest or after nitroglycerin
- Tachycardia, shortness of breath during exertion

Diagnostics:

Laboratory methods:

- Troponin I/T – markers of necrosis
- KFK-MV, LDG
- Cholesterol, LDL, HDL, triglycerides

Instrumental methods:

- ECG (ST elevation, abnormal Q wave)
- Echocardiography (hypokinesia, decreased ejection fraction)
- Holter monitoring
- Bicycle ergometry, treadmill test
- Coronary angiography

Diseases of the pericardium and myocardium

Pericarditis

Definition:Inflammation of the pericardium (the outer lining of the heart).

Reasons:

- Viral infections (Coxsackie, influenza)
- Tuberculosis
- Autoimmune diseases (SLE, rheumatism)
- Renal failure (uremic pericarditis)
- Myocardial infarction (post-infarction Dressler syndrome)

Clinic:

- Chest pain that gets worse when breathing in/coughing
- Tachycardia
- Pericardial friction rub
- Signs of tamponade (decreased blood pressure, pulse paradox)

Diagnostics:

- ECG: decreased voltage, saddle-shaped ST
- Echocardiography: effusion, thickening of the pericardium
- X-ray: enlarged heart shadow
- Biochemistry: increased C-reactive protein, leukocytosis

Myocarditis

Definition:

Inflammation of the myocardium, often of viral etiology.

Reasons:

- Viruses (Coxsackie, Epstein-Barr)
- Bacteria, parasites
- Autoimmune reactions
- Toxic effects (alcohol, drugs)

Clinic:

- Weakness, fatigue
- Dyspnea
- Heart palpitations, irregular heartbeat
- Sometimes - chest pain
- Signs of heart failure

Diagnostics:

- ECG: arrhythmias, conduction disturbances
- EchoCG: decreased EF, dilatation
- Increased troponins
- MRI of the heart - inflammation, myocardial edema

Dyslipidemia. Definition:A lipid metabolism disorder characterized by abnormal levels of cholesterol, LDL, HDL, and TG in the blood.

Types of dyslipidemia:

- Primary (hereditary)**:**familial hypercholesterolemia
- Secondary**:**for diabetes, obesity, hypothyroidism, alcoholism

Risk factors:

- Heredity
- Unhealthy diet (excess of animal fats)
- Hypodynamia

- Obesity
- Smoking
- Diabetes mellitus

Diagnostics:

- Lipidogram**:**
  - Total cholesterol
  - LDL (bad)
  - HDL (good)
  - Triglycerides
  - Atherogenic index

Arterial hypotension

Definition:PBP <90 mmHg and/or DBP <60 mmHg

Types:

- Physiological (in athletes, hereditary)
- Pathological:
  - Primary (neurocirculatory)
  - Secondary (with ACS, blood loss, endocrinopathies, infections)

Clinic:

- Dizziness, weakness
- Darkening of the eyes
- Cold extremities
- Fainting
- Tachycardia
- Deterioration of concentration

Diagnostics:

- Blood pressure monitoring
- Orthostatic test
- ECG (tachycardia, arrhythmia)
- Tests: anemia, electrolytes, glucose, hormones

Methods for studying arterial hypertension and coronary heart disease

Laboratory methods:

- OAK, OAM
- Blood biochemistry:
  - Cholesterol, LDL, HDL, TG
  - Creatinine, urea
  - Glucose
  - Troponin, CPK-MB (for ischemic heart disease)

Instrumental methods:

- Blood pressure measurement (repeat, ABPM)
- ECG—identification of signs of hypertrophy, ischemia, arrhythmia
- Echocardiography- structural changes, systolic function
- Holter ECG monitoring
- Load tests— **bicycle ergometry, treadmill**
- Coronary angiography— the "gold standard" for diagnosing coronary heart disease

Clinical syndromes such as coronary insufficiency, myo- and pericarditis, dyslipidemia, and arterial hypotension are the most important manifestations of cardiovascular disease. Their timely diagnosis requires a comprehensive approach, including medical history, physical examination, laboratory

tests, and imaging studies. This ensures effective treatment and the prevention of severe complications such as heart attack, stroke, heart failure, and sudden death.

4. Illustrative material: presentation

5. Literature: indicated on the last page of the syllabus

6. Control questions:

1. What blood pressure numbers correspond to arterial hypertension?
2. What changes during auscultation can be detected in patients with valve dysfunction syndrome?
3. What ECG changes can be detected in pericardial diseases?
4. What indicators of arterial hypertension are considered borderline arterial hypertension?
5. What are the main complaints of valve dysfunction syndrome?
6. What is valve apparatus syndrome?
7. What are the causes of valve damage?
8. What forms of valve apparatus damage do you know?
9. What is atherosclerosis?

Lecture No. 4

1. Subject: Leading clinical syndromes (valvular lesions) in patients with cardiovascular diseases. Arrhythmias. Main causes, risk factors, clinical presentation, classification, laboratory and instrumental methods of investigating arterial hypertension and coronary heart disease. Diagnostic value.

2. Objective: To teach students to identify the main symptoms of valvular lesions and arrhythmia. Explain the mechanism of symptom development and identify typical changes in laboratory and instrumental examinations. To teach students how to collect primary and secondary complaints and a patient's medical history, as well as the methodology for conducting a targeted examination of a patient with valvular lesions, and to identify typical symptoms and changes in laboratory and instrumental examinations.

3. Lecture abstracts:

Heart valve diseases and rhythm disturbances (arrhythmias) are among the most common clinical syndromes in patients with cardiovascular pathologies. They can be either independent conditions or complications of other cardiovascular disorders (hypertension, coronary heart disease, myocarditis, cardiomyopathy, etc.).

Valvular heart defects (valvular apparatus lesions)

Definition:Valvular defects are diseases that involve disruption of the structure and function of the heart valves, leading to stenosis (narrowing) or insufficiency (failure to close) of the valves.

Etiology:

- Rheumatism—a common cause in young people
- Atherosclerosis, calcification- in the elderly
- Infective endocarditis
- Congenital defects
- Cardiomyopathies
- Connective tissue syndromes (Marfan, Ehlers-Danlos)

Most commonly affected are:

- Mitral valve (stenosis and insufficiency)
- Aortic valve (stenosis and insufficiency)
- Less commonly, the tricuspid and pulmonary valves

Classification:

Type of defect	Valve	Consequence
Stenosis	Mitral	Left atrial hypertension, congestion in the pulmonary circulation
Failure	Aortic	Volume overload of the LV, heart failure
Combined	Mitral or aortic	Stenosis + insufficiency

Clinical picture:

Mitral stenosis:

- Shortness of breath, especially with exertion
- Cough with hemoptysis
- Hoarseness (Orner's syndrome)
- Diastolic murmur at the apex

Mitral regurgitation:

- Systolic murmur
- Heart palpitations, fatigue
- Shortness of breath, swelling

Aortic stenosis:

- Chest pain (angina)
- Fainting
- Shortness of breath on exertion
- Systolic murmur in the 2nd intercostal space on the right

Aortic insufficiency:

- Throbbing headache
- "Carotid dance", pronounced pulse
- Diastolic murmur

Diagnostics:

Physical examination:

- Auscultation: noises, additional tones
- Pulse, blood pressure, signs of heart failure

Instrumental methods:

- ECG—signs of hypertrophy, arrhythmia
- Echocardiography with Doppler—main method: visualization of valves, assessment of regurgitation, pressure gradients
- Chest X-ray—cardiomegaly, congestion
- Coronary angiography- before surgery in patients over 40 years of age

Laboratory:

- OAK, OAM - for rheumatism, infections
- RF, ASL-O, C-reactive protein - in rheumatism

Arrhythmias. Definition:Arrhythmias are disturbances in the frequency, rhythm, and excitation sequence of the heart, associated with changes in automatism, excitability, or conductivity.

Classification:

By frequency:

- Bradycardia (<60 bpm)
- Tachycardia(>100 bpm)

By mechanism:

- Disturbances of automaticity (sinus tachycardia or bradycardia)
- Excitability disorders (extrasystole, AF)

- Conduction disturbances (blockade)

By localization:

- Supraventricular (AF, atrial flutter)
- Ventricular (VT, VE)

Reasons:

- coronary heart disease, myocardial infarction
- AG, SN
- Cardiomyopathy, myocarditis
- Electrolyte disturbances (potassium, magnesium)
- Hypothyroidism/hyperthyroidism
- Medicines (antiarrhythmics, glycosides)

Clinical picture:

- Heart palpitations
- Heart palpitations, a feeling of "stopping"
- Dyspnea
- Dizziness, pre-fainting conditions
- Chest pain

Diagnostics:

Physically:

- Changes in pulse rate (rate, rhythm, pulse deficit)
- BP, signs of heart failure

Instrumental methods:

- ECG (main method)
- Holter ECG monitoring
- Transesophageal electrophysiology—for the diagnosis of paroxysms
- Echocardiography- structural changes
- Load test- if ischemic nature is suspected

Laboratory:

- Electrolytes (K^+, Mg^{2+}, Ca^{2+})
- Thyroid hormones
- Troponins (if a heart attack is suspected)

Methods of studying arterial hypertension and ischemic heart disease (and repetition for consolidation)

Laboratory methods:

- OAC, biochemistry
- Lipid profile (total cholesterol, LDL, HDL, TG)
- Glucose, HbA1c
- Troponin, CPK-MB, myoglobin - for ischemic heart disease

Instrumental:

- Blood pressure measurement / ABPM
- ECG / Holter
- Echocardiography—structural changes, PV
- Bicycle ergometry, treadmill test—with coronary heart disease
- Coronary angiography- for high-risk angina
- Ophthalmoscopy— with hypertension

Diagnostic value of clinical methods

1. Anamnesis and questioning:
   - They provide the key to recognizing angina pectoris, arrhythmias, and heart defects
   - Determine the type of pain, its relationship with stress, and previous illnesses
2. Inspection, palpation, percussion, auscultation:
   - Recognition of noises in valve defects
   - Determination of signs of heart failure, hypertension, cardiac hypertrophy
3. ECG and EchoCG:
   - ECG is a method of primary diagnosis of coronary heart disease and arrhythmias
   - Echocardiography is the basis for verification of valve defects
4. Daily monitoring:
   - Detection of hidden arrhythmias
   - Assessment of the daily blood pressure profile

Valvular heart defects and arrhythmias are the leading clinical syndromes in cardiology practice. Their timely recognition requires knowledge of clinical manifestations, risk factors, and the use of modern diagnostic methods. Arterial hypertension and coronary heart disease often underlie these pathologies, and their diagnosis must be comprehensive, incorporating both clinical and instrumental methods.

7. Illustrative material: presentation

8. Literature: indicated on the last page of the syllabus

9. Control questions:

1. What blood pressure numbers correspond to arterial hypertension?
2. What changes during auscultation can be detected in patients with valve dysfunction syndrome?
3. What ECG changes can be detected in pericardial diseases?
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