Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

ANATOMICAL AND PHYSIOLOGICAL ASPECTS OF THE CARDIOVASCULAR SYSTEM

Methodical recommendations
for self-preparation of 2nd year students of the School of medicine
in the discipline «Anatomical and physiological aspects
of the cardiovascular system»

Kharkiv – 2023

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Methodical recommendations for students in course «Anatomical and physiological aspects of the cardiovascular system» are developed in accordance with the current programs in physiology for students of medical faculties of universities. The manual is designed for students in preparation for the course «Anatomical and physiological aspects of the cardiovascular system». Each topic contains a list of practical skills and control questions. The topics are illustrated with drawings and diagrams that facilitate the perception of the material and promote its better assimilation. The materials allow students to form a correct understanding of the laws of the human body. For medical students.

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CONTENT

INTRODUCTION

Human anatomy and Physiology is the science of structure of biological organisms and life processes, the activity of individual organs and a living organism. The subject of these two sciences are the parameters of the living organism, for physiology, these are the functions of the body, their relationships, regulation and adaptation to the environment, for anatomy, these are the morphological features of the individual as a whole and in the process of evolution during individual development. This discipline is designed to combine the existing knowledge of human anatomy and physiology, to give the highest understanding of the existence of biological matter, to form the foundation for medical tactics in the medical practice of a future doctor.

Specific goals for students:

- Analyze information about the main indicators characterizing the normal functional state of the organism as a whole;
- Determine the relationship between the mechanisms of functioning of organs and the relationship of their structural components;
- Explain the mechanisms of functioning of organs and draw conclusions about the norm and pathology;
- Assess the state of health of people of different ages and in different conditions;
- Apply laboratory and instrumental research methods to assess the state of health;
- Anticipate changes in the activities of organs and systems under the influence of various environmental factors;
- To identify the leading mechanisms for ensuring the integrative activity of the organism;
- Demonstrate mastery of the moral and ethical principles of attitude towards a living person and the body as an object of study.

Topics of practical classes

№	The topic of the practical lesson	Hours
1	Introduction to the anatomical and physiological aspects of the cardiovascular system. Anatomical and physiological properties of the heart. Physiological properties of the myocardium. Mechanisms of automation. The action potential of typical myocardial cells and its phase. Conduction and speed of excitation.	5
2	Anatomical and physiological aspects and dynamics of cardiac excitation. ECG. ECG lead. ECG structure. Myocardial contractility. Pumping function of the heart. Cardiac cycle. Atrial systole. Ventricular systole.	5
3	Anatomical and physiological aspects of cardiac output and factors influencing its size. Methods for determining MVB. Mechanical work and heart tones. Regulation of heart activity. Interaction of intra- and extracardiac nervous systems. Reflex and humoral regulation of heart activity.	5
4	Anatomical and physiological aspects of blood vessels and their role in blood circulation. Basic patterns of blood flow. Vascular tone. The movement of blood in the vessels. Blood pressure in systemic vessels. Blood pressure measurement.	5
5	Anatomical and physiological aspects of systemic circulation regulation. Central mechanisms. Central regulation of blood circulation. Reflexes from the carotid sinuses. Reflexes from the aortic arch. Local mechanisms of blood circulation regulation.	5
6	Anatomical and physiological aspects of regional blood circulation. Circulation in the coronary vessels. Pulmonary circulation. Large circle of blood circulation. Lymphatic system. Age features of blood circulation.	5
	Total	30

Tasks for independent work

№	Topic	Hours
1.	Preparation for passing test tasks as components of USQE and USMLE RX on the topic: "Introduction to the physiological anatomy of the cardiovascular system. Anatomical and physiological properties of the heart. Physiological properties of the myocardium. Mechanisms of automation. The action potential of typical myocardial cells and its phase. Conduction and speed of excitation.	15
2.	Preparation for passing test tasks as components of USQE and USMLE RX on the topic: "Anatomical and physiological aspects and dynamics of cardiac excitation. ECG. ECG lead. ECG structure. Myocardial contractility. Pumping function of the heart. Cardiac cycle. Atrial systole. Ventricular systole.	15
3.	Preparation for passing test tasks as components of USQE and USMLE RX on the topic: "Anatomical and physiological aspects of cardiac output and factors influencing its size. Methods for determining MVB. Mechanical work and heart tones. Regulation of heart activity. Interaction of intra- and extracardiac nervous systems. Reflex and humoral regulation of heart activity ".	15
4.	Preparation for passing test tasks as components of USQE and USMLE RX on the topic: "Anatomical and physiological aspects of blood vessels and their role in blood circulation. Basic patterns of blood flow. Vascular tone. The movement of blood in the vessels. Blood pressure in systemic vessels. Blood pressure measurement ".	15
5.	Preparation for passing test tasks as components of USQE and USMLE RX on the topic: "Anatomical and physiological aspects of systemic circulation regulation. Central mechanisms. Central regulation of blood circulation. Reflexes from the carotid sinuses. Reflexes from the aortic arch. Local mechanisms of blood circulation regulation ".	15
6.	Preparation for passing test tasks as components of USQE and USMLE RX on the topic: "Anatomical and physiological aspects of regional circulation. Circulation in the coronary vessels. Pulmonary circulation. Large circle of blood circulation. Lymphatic system. Age features of blood circulation ".	15
	Total:	90

Lesson 1.

Introduction to the anatomical and physiological aspects of the cardiovascular system.

The heart (cor) is a hollow muscular organ divided inside into 4 cavities: the right and left atrium and the right and left ventricles. Each atrium connects to the corresponding ventricle of the atrial-ventricular opening (right and left), each of which is closed by flap valves. The atria take blood from the veins and push it into the ventricles; and the ventricles release blood into the arteries. The size of the heart of a healthy person correlates with the size of his body. The length of the heart of an adult varies from 10 to 15 cm, the width of the heart at the base is 8 - 11 cm, front size 6-8.5 cm. The heart mass in women is on average 250 g, in men - 300 g. In an adult, its volume is on average for men - 780 cm3, for women - 560 cm3. The shape of the heart is not the same in different people and is associated with age, gender, physique, physical activity and other factors. It usually resembles a slightly flattened cone, the position of which depends on the shape of the chest, age and respiratory movements of the person. When you exhale, the diaphragm rises, so the heart is located more horizontally, and when inhaling - more vertically.

In the heart, the posterior-upper dilated part is distinguished - the base of the heart (basis cordis) and the anterior-lower part - the apex of the heart (apex cordis). The base of the heart is formed by the atria, turned upwards back and to the right. The apex is the lowest and most protruding to the left pointed end of the heart (formed by the left ventricle).

Also in the heart emit surfaces:

1. sterno-rib (facies sternocostalis) - anterior;

2. diaphragmatica (facies diaphragmatica) - lower;

3 pulmonary (facies pulmonalis) - lateral (right and left)

On the outer surface of the heart, 3 furrows are distinguished, which are visual boundaries that separate the atria from the ventricles, as well as the ventricles themselves from each other.

Furrows of the heart:

1. The coronary sulcus (sulcus coronarius) - transversely located, separates the atrium from the ventricles. On the anterior surface of the heart, it is almost invisible, because it is interrupted by the pulmonary trunk and the ascending part

of the aorta. This furrow is most pronounced on the diaphragmatic surface of the heart.

2. *Anterior interventricular sulcus (heart) (sulcus interventricularis* anterior) - divides the anterior surface of the heart into a wider right part, corresponding to the right ventricle, and a smaller left one belonging to the left ventricle.

3. Posterior interventricular sulcus (heart) (sulcus interventricularis posterior) - located on the back of the heart. It begins at the confluence of the coronary sinus into the right atrium, goes down and reaches the top of the heart, where it connects to the anterior interventricular sulcus with the help of a notch of the apex of the heart (incisura apicis cordis). In the coronary and interventricular grooves pass the blood vessels that feed the serce - the coronary arteries and veins. The anterior-upper protruding part of each atrium is called the atrial ear (auricula cordis). They are curved hollow outgrowths, on the inner surface of which are located the comby muscles. The ears cause the suction function of the heart, are an additional reservoir and biological shock absorber for the blood and entering the atrium.

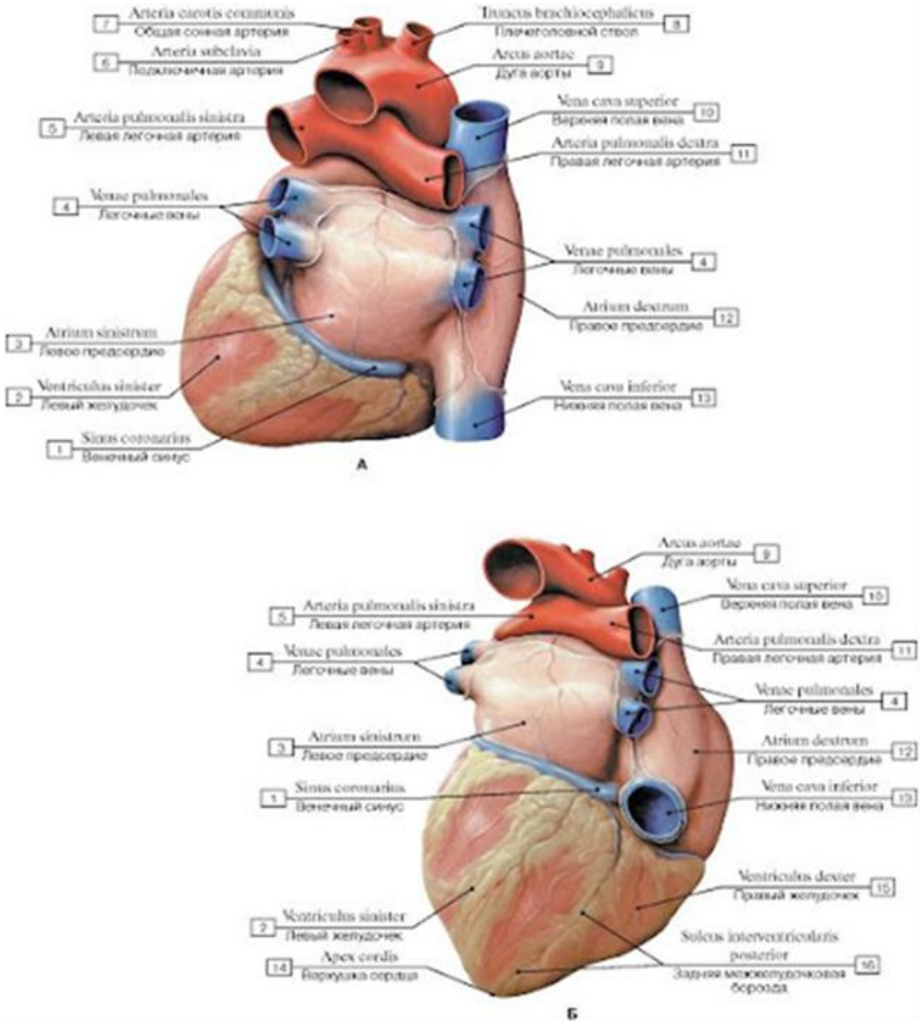


Fig. 1. Anatomy of the heart.

ANATOMY OF HEART CHAMBERS

The right atrium (atrium dextrum) has a shape close to the cuboid. The lower border of the atrium is the coronary sulcus Formation of the right atrium:

1. Right ear (auricula dextra). On its inner surface and the adjacent part of the anterior wall of the atrium there are several rollers corresponding to the comb muscles (mm. pectinati).

2. Atrial atrial (septum interatriale) - separates the right atrium from the left.

3. Right atrial-ventricular opening (ostium atrio-venticulare dextrum) - connects the right atrium and the right ventricle.

4. In the right atrium there is a hole of the superior vena cava, (ostium venae cavae superioris), and a hole of the inferior vena cava, (o. venae cavae inferioris). The inner surface of the walls of the right atrium is smooth, there are two folds on it

5. The flap of the inferior vena cava (Eustachius flap) (valvula venae cavae inferioris) - located at the confluence of the inferior vena cava

6. The latch of the coronary sinus (valvula sinus coronarii) - located at the confluence of the coronary sinus.

7. Oval fossa (fossa ovalis) - located on the atrial tumor. It is surrounded by a slightly protruding edge. In its place in the prenatal period was an oval hole (foramen ovale),with which the atria were connected.

The left atrium (atrium sinistrum) has the shape of a cube. The lower border of the left atrium is the coronary sulcus. Formation of the left atrium:

1. Holes of the pulmonary veins (usually in the amount of 4) - two on each side;

2. Left ear (auricula sinistra). It contains the comby muscles, which, unlike the right ear, do not pass to the wall of the atrium itself.

3. The left atrial-ventricular opening (ostium atrio-ventriculare sinistrum) through which the left atrium communicates with the left ventricle.

Right ventricle (ventriculus dexter). The shape resembles a triangular pyramid with a top facing downwards. Formation of the right ventricle:

1. Interventricular recurrent (septum interventriculare) - separates the right ventricle from the left. It consists of 2 parts: larger - muscular, and smaller - membranous, located in the uppermost section, closer to the atria.

2. Right atrial-ventricular opening through which venous blood from the right atrium enters the right ventricle.

3. The opening of the pulmonary trunk (ostium trunci pulmonalis), through which, when the right ventricle is reduced, venous blood is pushed into the pulmonary trunk and then into the pulmonary arteries.

4. The right atrial-ventricular opening has a single, right atrial-ventricular (tricuspid) valve (valva atrio-ventricularis dextra) located near the right atrial-ventricular opening. The valve consists of three valves (front, rear and intersected). Sometimes there may be additional sash.

5. Tendon chords (strings) in the amount of 10 to 12 pieces begin from the ventricular surface of the valve valves and are attached by opposite ends to the papillary muscles.

6. The three juice-like muscles (musculi papillares) are located on the inner surface of the anterior, posterior and intersected wall of the right ventricle. These are, respectively, the anterior, posterior, and peretinal mastoid muscles. Part of the tendon chords begins from the fleshy translations (trabeculae) of the interventricular retention. Chords (strings) are attached simultaneously to the free edges of two adjacent valves, as well as to their surface facing the ventricular cavity. These muscles, together with tendon chords, hold the valves and, when contracting (systole) of the ventricle, prevent the atrial valves from turning into the atrial cavity and thereby prevent the return of blood from the ventricles to the atrium.

7. Fleshy translations (trabeculae) (trabeculae sarneae) - muscle valikas (crossbars) on the walls between the papillary muscles inside the ventricle.

8. Arterial cone (conus arteriosus) – anterior and upper part of the right ventricle, which continues further into the pulmonary trunk. In the area of the arterial cone, the walls of the right ventricle are smooth.

9. The valve of the pulmonary trunk (valva trunci pulmonalis) consists of three crescent flaps (left, right and anterior - valva semilunares), freely passing blood from the ventricle into the pulmonary trunk.

10. Crescent damper nodulus (nodulus valvulae semilunaris) - thickening in the middle of the free edge of each of these flaps. Nodules contribute to a tighter closure of the semi-lunar flaps when closing the valve.

11. The sinus (sinus) of the pulmonary trunk is a small pocket located between the wall of the pulmonary trunk and thereaping of the semi-lunar flaps.

With the contraction of the right atrium, blood enters the right ventricle, heading towards its top along the lower wall. When the ventricle contracts, blood is pushed into the pulmonary trunk, passing from the apex of the ventricle to its base through the opening of the pulmonary trunk, in the area of which is the valve of the same name. The convex lower surface of the flaps is facing the cavity of the right ventricle, and the concave - into the lumen of the pulmonary trunk. In the middle of the free edge of each of these flaps there is a thickening. When the muscles of the ventricle contract, the crescent dampers are pressed by the blood flow to the wall of the pulmonary trunk and do not interfere with the passage of blood from the ventricle. When the muscles of the ventricle relax, the pressure in its cavity drops, and in the pulmonary trunk the pressure is high. The reverse flow of blood is impossible because blood fills the sinuses (sinuses) and opens the flaps. In contact with the edges, the flaps close the hole and prevent the return flow of blood.

The left ventricle (ventriculus sinister) has the shape of a cone. Its walls are 2-3 times thicker than the walls of the right ventricle. This is due to a greater load. His muscles push blood into the vessels of the great circle of blood circulation.

Anatomical structures of the left ventricle:

1. Left atrial-ventricular opening (ostium atrioventriculare sinistrum) - serves to communicate with the left atrium.

2. Left atrial-ventricular valve (vulva atrioventricularis sinistra). It has only two flaps, it is called a bicuspid, or mitral valve. The anterior sash (cuspis anterior) of this valve begins near the interventricular thrash; posterior sash (cuspis posterior), begins on the posterior lateral side.On the inner surface of the left ventricle, like the right one, there are endocardially covered muscle strands –

3. Fleshy trabeculae (translations).

4. Two mastoid muscles (anterior and posterior).

5. Tendon chords (strings) attached to the valves of the left atrial-ventricular valve.

6. Entrance to the aortic opening (ostium aortae) – contained in the upper part of the ventricle.

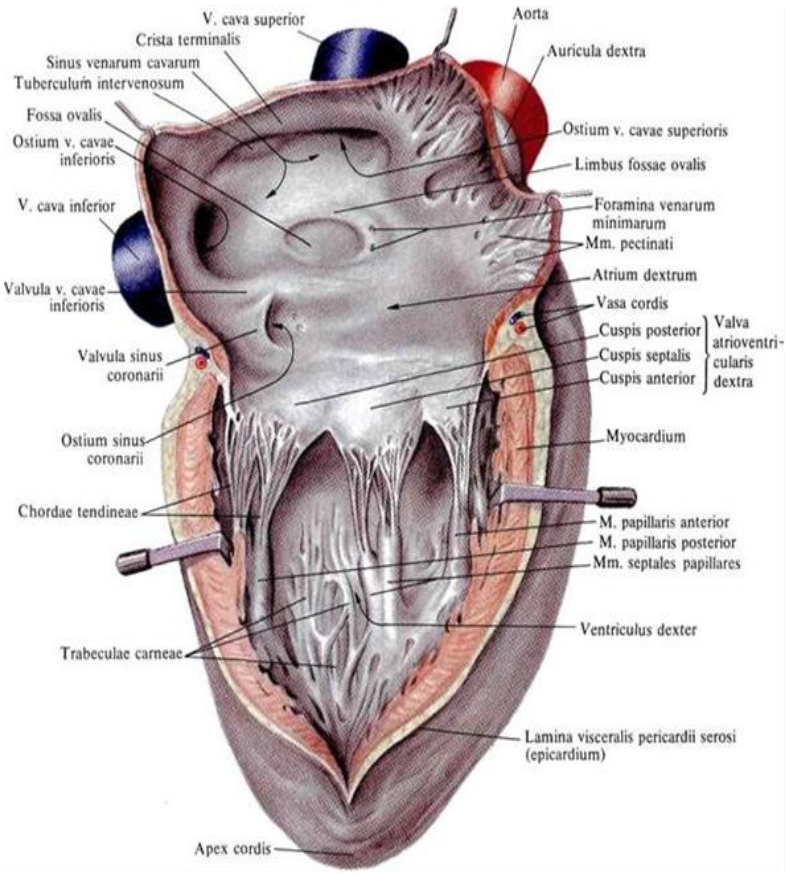


Fig. 2. Heart. Heart chambers.

7. The aortic valve (valva aortae), consists of 3 semi-lunar flaps - right, left and posterior (valvulae semilunares dextrae, sinistra et posterior). Aortic flaps have the same structure as the dampers of the pulmonary trunk. However, the aortic valve flaps are thicker, and the nodules of the crescent flaps located in the middle of their free edges are larger than those of the valve of the pulmonary trunk.

8. The interventricular membrane (septum interventricularis) consists of a larger muscular part (lower part) and a smaller one - the membranous part (its upper part), which is formed only by fibrous tissue covered on both sides of the endocardium.

Physiological properties of the myocardium

The physiological properties of myocardial cells include: automation, excitability, conductivity and contractility (Fig. 3). Excitability, conductivity and contractility – these properties have all muscle fibers, both skeletal and smooth. Myocardium belongs to the striated muscles, but its specialized cells of the conductive system (atypical, or pacemaker) also have such a property as automation.

Automation is the ability of atypical pacemaker cells of the heart to spontaneous, rhythmic depolarization of the membrane, which leads to the generation of action potentials, myocardial contraction. All fibers of the conductive system of the heart have automatia: sinoatrial (CA) node, internodular atrioventricular (AB) node, His bundle and its legs, Purkinje fibers. But only the CA node is the driver of the rhythm, because it provides the generation of action potentials (PD) with a frequency that causes the frequency of contraction of the heart as a pump under normal physiological conditions (Fig. 4). Other structures of the cardiac conductive system belong to the latent drivers of the heart rhythm, because only under certain conditions can they act as rhythm drivers.

Fig. 3. Physiological properties of myocardial cells.

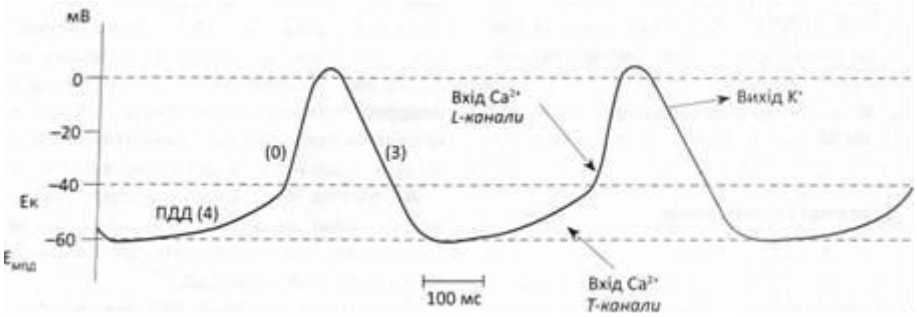


Fig. 4. Generation of AP in cells of the sinus-atrial node.

Atypical cells of the SA node, located at the mouth of the confluence of hollow veins into the right atrium, have a membrane resting potential smaller than other excitatory cells, its value is -60 mV. However, it is not stable and therefore it is called – the maximum diastolic potential (MDP), which, decreasing, generates phases of the action potential (AP) of the cells of the SA node.

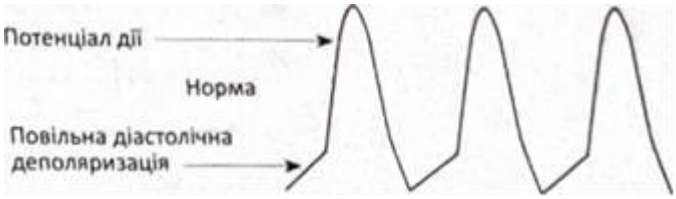
The first phase is spontaneous slow diastolic depolarization (PDD), or prepotential (4); It is this phase that causes automation. Its mechanism is

associated with the ionic permeability of the membranes of pacemaker cells. At the end of diastole, the yield of K+ ions from cells decreases sharply. The rest potential returns to its original level - -60 mV. Ca2+ T-channels are opened (from the English transient – temporary), through which the input of Ca2+ ions increases, which leads to the development of cell depolarization, which reaches a critical level (-40 mV). The role of Na+ ions in the development of PDD cells of SA- and AV nodes is insignificant, as evidenced by the absence of a phase of rapid depolarization, characteristic of the myocardium of the atria and ventricles, and other structures of the conductive system.

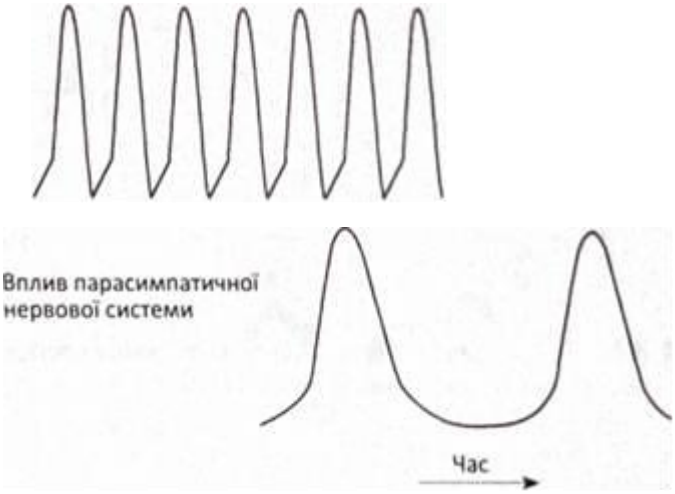
The second phase is the phase of rapid depolarization (0), which reaches the 0-potential or has a slight reversion due to the entry of calcium ions through Ca2+ L-channels (from the English long-lasting – long-term).

The third phase – the repolarization phase (3) – is caused by an increase in the yield of K* ions from the cell, which leads to the restoration of the potential to the original level of -60 mV.

The frequency of PD generation by SA node cells, called a pacemaker, depends on: 1) the duration of the PDD phase (4); 2) the value of the threshold of depolarization; 3) amplitudes of the rest potential. For example, when stimulating the fibers of the sympathetic nervous system, norepinephrine is released, which binds to the β-adrenergic receptors of the cells of the SA-node, as a result of which the level of cAMP increases in them, Ca2+ channels open, the input of Ca2+ ions accelerates, which leads to a decrease in the duration of traffic rules and an increase in the frequency oftencials of action, heart contractions. Conversely, when stimulating the vagus nerve, the isolated acetylcholine reacts with the M-cholinergic receptors of the SA-node cells and, with the participation of the subunits of the protein G, opens the K+ channels and, accordingly, enhances the yield of K+ ions, which causes hyperpolarization of pacemaker cells, prolongation of the duration of PDD, decrease in the number of AP and heart rate (Fig. 5). The rhythm of the heart, due to the frequency of excitations of the pacemaker cells of the SA node of the automata), is called the sinus rhythm, which is on average 75 per 1 min. It is the largest compared to the latent rhythm drivers present in the cardiac conduction system. There is an automatic gradient. If the cells of the SA node generate AP with a frequency of 75 in 1 minute, then the cells of the atrioventricular node (AV node) – 50-55, the His bundle – 40-50, the Purkinje fibers – 30-20 pulses in 1 minute, the rest – even less, that is, the SA node dictates its own rhythm to all the below-located automation nodes. In case of damage to the SA node or violation of the conduction of impulses through it, latent (ectopic) rhythm drivers (for example, AV node) become basic and impose their own frequency of contractions on the heart.



Influence of the sympathetic nervous system

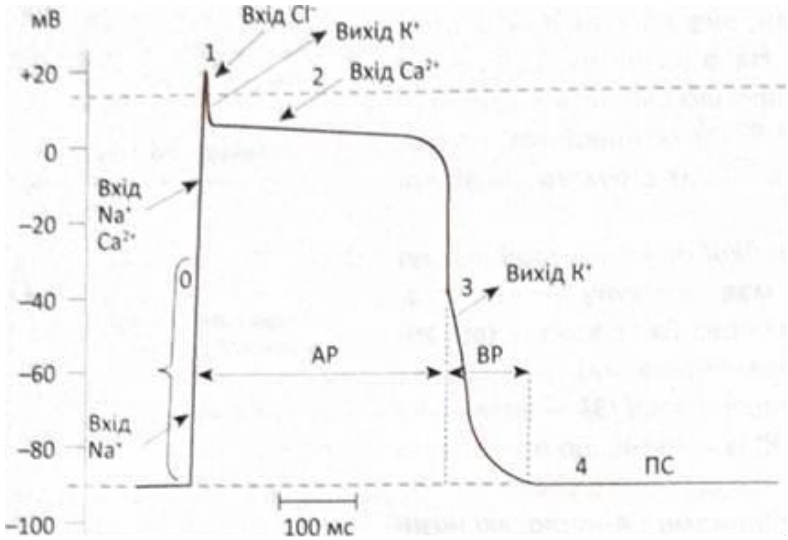


Rice. 5. Changes in the membrane potential of the SA node under the influence of the extracardial nervous system.

Excitability is the ability of atrial and ventricular myocardial cells to generate action potentials (AP) when exposed to irritation. The heart responds to single threshold and supra-threshold irritations with maximum contractions, that is, it acts according to the law "all or nothing". During the development of AP, the cardiomyocyte membrane loses the ability to respond to other stimuli, becomes unexcited – refractory (Fig. 6). There are 2 periods of refractoriness: Absolute refractory period (AR) – there is no excitability, cells are not able to generate AP under the action of a stimulus. This period occurs immediately after the beginning of the phase of rapid depolarization and lasts almost until the completion of the plateau (270 ms) and is due to sodium inactivation and an increase in the input Ca2+ and output K+ currents. The relative refractory period (RP) is the period when only a large force of irritation causes the generation of AP, but it is characterized by a lower rate of development and amplitude, because not all sodium channels have left the state of inactivation. Its duration is 30 ms.

The resting potential of atrial and ventricular myocardial cells is stable and is -90 mV, approaching the equilibrium diffusion potassium potential. The action potential is 120 mV. It is long-lasting: up to 100 ms – in the atrial myocardium,

and up to 350 ms – in the myocardium of the ventricles. The critical level of depolarization is closeto 70 mV.



Rice. 6. Development of the action potential of typical ventricular myocardial cells and periods of their refractoriness: AR – absolute, RP – relative.

The action potential consists of the following phases:

■ The phase of rapid depolarization (phase 0) and completion arise due to the entry of Na+ ions through fast sodium channels in which the potential-dependent activation gate opens, as happens in skeletal muscles and nerve fibers, followed by their inactivation. The amplitude of AP reaches the diffusion equilibrium sodium potential. A certain contribution to the development of the upper part of this phase is made by Ca2+ ions.

■ The phase of rapid initial repolarization (phase 1) is due to sodium inactivation, the entry of SI ions into cells and the beginning of the release of K ions

■ The plateau phase (phase 2) is a consequence of the entry of Ca2+ ions through open slow calcium channels. Slow calcium channels become active even when the membrane potential in phase 0 of depolarization decreases to -30 mV. -40 mV, but long-term calcium conductivity increases significantly during the plateau, maintaining a membrane potential level of about 0.

■ The phase of final repolarization (phase 3) is due to the rapid release of K+ ions from the cells and the closure of Ca2+ channels. due to this, the membrane resting potential (RP) begins to recover.

■ RP (phase 4) - complete restoration of the resting potential to the value of the diffusion equilibrium potassium potential (-90 mV).

Conductivity, sequence and speed of excitation

The excitations that occur in the SA node quickly spread through the specialized conductive bundles of Bachmann, Wenkebach, Tore la and muscle cells that connect through the atruventricular node to the ventricles.

Conductivity characterizes the stages and time of excitation transmission from one excitatory myocardial structure to another. During depolarization, AP quickly spreads to neighboring cells that connect through intercellular contact structures (nexuses) with low resistance and greater conductivity. The speed of excitation depends on the strength of local electric currents, due to the amplitude of the AP, the threshold of depolarization, the resistance value of the contact structures.

In fig. 7. The sequence and speed of excitation by the structures of the heart are given Three internode bundles (anterior, middle, posterior) of the Purkinje fiber type depart, which carry excitation to the AV node located in the back wall of the right atrium next to the tricuspid valve. Under normal conditions, it is possible to transmit excitation to the AV node directly by the muscle fibers of the atrium. The rate of atrial excitation is about 0.3-1 m/s. Due to the fact that the atrial-ventricular valves are surrounded by fibrous tissue, the spread of AP from the atria to the ventricles is possible only through the AV node. The rate of excitation in the AV node is reduced to 0.02-0.15 m/s due to an increase in resistance to electric current in thin atypical fibers and a low pd development rate due to slow Ca2' current. There is an atrioventricular delay (about 0.13 s), which makes it possible to complete the atrial systole. Conducting excitation in the AV node is normally only one-sided – from the atria to the ventricles, which prevents the reverse spread of impulses (AP), which would lead to a heart rhythm disturbance.

The rate of excitation by the His bundle, its legs and Purkinje fibers increases to 1-1.8 m/s, because these structures have a significant density of sodium channels, a fast input current that rapidly propagates in and between cells and ensures rapid AP conduction to ventricular myocardial cells. Thick Purkinje fibers permeate the myocardium from inside to outside by 2/3, and excitation in them spreads through the ventricular myocardium towards the epicardium with a sutureof 1.9-3.1 m/s.

Blockade of pulses from the SA node to the departments of automatism located below. More often than others, there is a blockade of the impulse between the atrium and the ventricle – AV blockade. With a complete blockade – the atriumcontracts in its rhythm (75 beats / min).

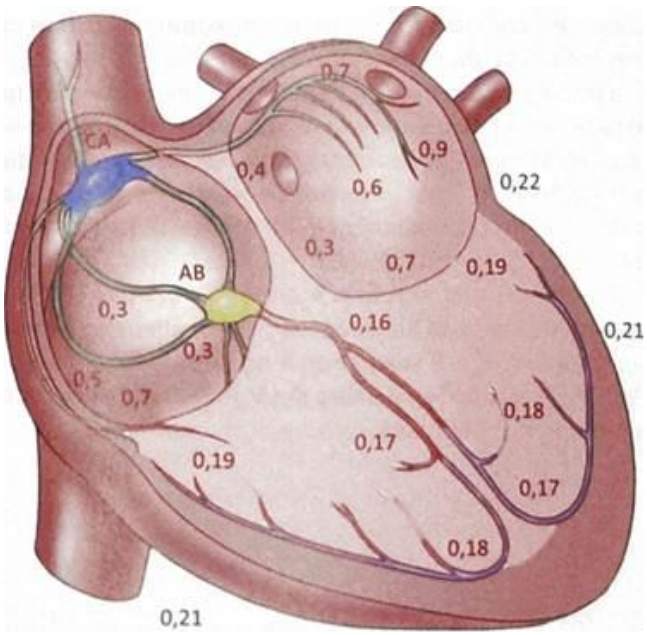


Fig. 7. The rate of propagation of excitation in the structures of the heart.

The given numbers are the time interval (in fractions of a second) that passes from the moment of occurrence of AP in the SA node to its appearance in a certain area of the atrial myocardium, and from the moment of occurrence of AP in the AV node to its occurrence in the myocardium of the ventricles, the ventricles – in another (40-50 beats / min), due to the pacemaker activity of the bundle of His. At the time of the occurrence of a complete blockade, asystole occurs, which can lead to impaired blood supply to the brain, seizures, loss of consciousness (Morgany–Adams–Stokes syndrome). Sometimes a person has an additional conductive path between the atrium and the ventricle – a bundle of Kent bypassing the AV node, then ventricular contractions occur earlier, because there is no AV delay, and are complete.

Lesson 2.

Anatomical and physiological aspects and dynamics of heart excitation. ECG. ECG lead. ECG structure

An electrocardiogram (ECG) is a record of changes in total electrical potentials (depolarization and repolarization) from the surface of the body that occur in the heart during the cardiac cycle. The ECG has been used for almost 100 years and was described by the German physiologist V. Einthoven, for which the Nobel Prize was awarded.

The propagation of the wave of depolarization and repolarization of the action potential is the movement of a double layer of charges located at the border of the excited and not yet excited areas of the myocardium. These charges are the same in size and opposite in sign and are at a very short distance from each other and are denoted by a dipole. The negative (-) pole of the dipole is turned towards the excited area of the myocardial fiber, and the positive (+) is turned towards the unexcited. The dipole is an elementary electromotive force (EEF), which is a vector quantity. The direction of the vector of any field goes from its negative pole to the positive (Fig. 8).

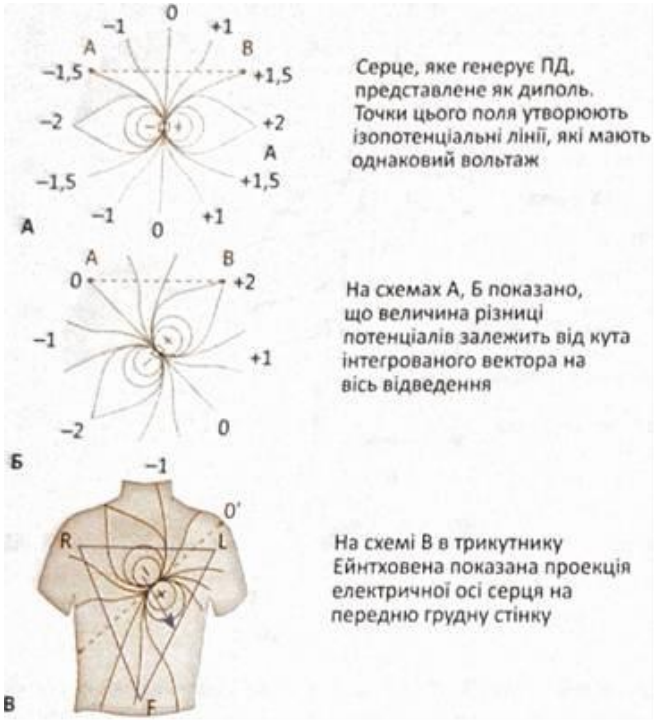


Fig. 8. The mechanism of ECG.

When excited in the heart at the same time there are many vectors that have different sizes and directions. The total vector is written on the ECG, which is defined as the algebraic sum of all the vectors that make up it. Thus, when registering an ECG, the voltage that appears between two electrodes on the surface of the body depends on the values of the total EEF vector, the resistivity of the body tissues and the orientation of the vector relative to the discharge electrodes. The voltage is proportional to the cosine of the angle between the axis of the dipole and the axis of the lead. It will be the highest, provided that the axes are parallel to each other and equal to zero. The amplitude of the ECG teeth is inversely proportional to the square of the distance from the electrode to the heart as an EEF source. This means that the farther the electrode is from the current source, the smaller the amplitude of the ECG teeth. Nevertheless, further distance (more than 12 cm) almost does not affect the amplitude of the teeth.

ECG diversion

ECG registration is carried out from the surface of the skin of the human body, as a rule. The leads are bipolar and unipolar. Einthoven bipolar standard leads record the dynamics of changes in the potential difference between two electrodes (+ and -) of human limbs. The lead lines connecting the points of the located electrodes on the limbs form an isosceles triangle called the Einthoven triangle. In the center of it there is a heartsingle dipole. Perependiculars lowered from the center of the triangle divide each lead line into 2 parts – positive and negative. If the momentary vector of the heart is projected onto the positive part of the withdrawal line, a positive tooth (+) is recorded, directed upwards, if the negative one is a negative tooth (-) directed downwards.

Goldberger's unipolar amplified leads – aVR, aVL, aVF – are written from the active electrode, which is always connected to the positive pole (+) of the electrocardiograph, and two electrodes combined into one, which are used in the registration of standard leads.

Wilson's Thoracic unipolar leads record a potential difference at 6 points (V1-V6) of the anterior thoracic wall. The first electrode is superimposed on one of the 6 points V. The second (indifferent) is three electrodes joined together, superimposed on both arms and left leg (Fig. 9).

Point V1 - 4 intercostal space to the right of the sternum; V2 - 4 intercostal space to the left of the sternum; V3 - 5 intercostal space to the left of the sternum; V4 - 5 intercostal space along the middle clavicular line; V5 - 5 intercostal space to the left of V4; V6 - 5 intercostal space along the middle axillary line.

ECG structure

The electrocardiogram normally has teeth, intervals and segments, which are shown in the ECG diagram recorded in the II standard lead (Fig. 10). Teeth are usually denoted by letters of the Latin alphabet. The direction of the teeth depends on the projection of the instantaneous mean vector of the heart: if its projection occurs on the positive side of the withdrawal line, then such a tooth is positive, if it is negative – then the tooth is negative, if its projection is perpendicular to the withdrawal line – then it is equal to 0 in this lead.

Intervals are the distance between the teeth on the ECG, they characterize the speed of propagation of the processes of depolarization or repolarization from one structure of the heart to another.

Segments are ECG sections that are located on the isoline, which means there is no potential difference.

Wave P – reflects the depolarization of the atrial myocardium; normally, it is positive in standard leads (I, II, III), because the projection of the vector in Einthoven's triangle is carried out on the positive side of the withdrawal lines and is negative - in the aVR lead. The amplitude of the tooth P is 0.2 mV, the duration is 0.11 s.

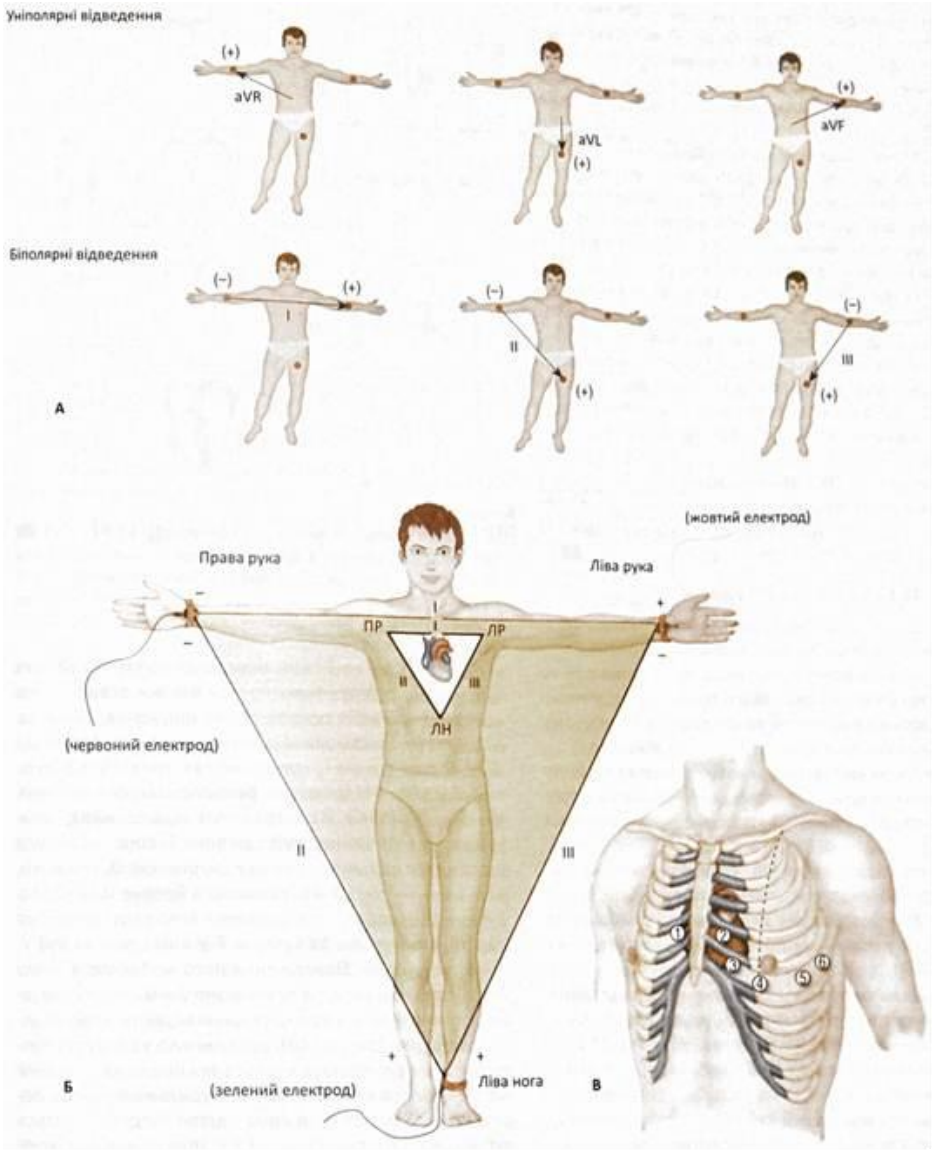


Fig. 9. The structure of the ECG (adapted by S. Fox).

A - unipolar and bipolar leads, B - Einthoven's triangle, C - December leads

The ventricular complex QRS reflects the depolarization of the ventricles.

The wave Q – the initial prong of this complex, is always negative and reflects the beginning of depolarization of the interventricular septum, where the structures of the conductive system of the ventricles are located. Its amplitude is less than 1/4 of the prong R, the duration is 0.04 s.

The wave R is the main vector of the complex, and it is also the main vector of the heart; reflects further depolarization, which extends from the interventricular septum to the myocardium of the right and left ventricles; He is always positive in all leads. Its amplitude is 1.4-1.6 mV, duration – 0.04 s.

Wave S – the final vector of depolarization of both ventricles, is negative, at this time the instantaneous vector is directed towards the base of the left ventricle, where there is still a polarized area of the myocardium. Its amplitude varies widely, the duration is 0.06 s.

Wave T – reflects ventricular repolarization, it is positive in standard leads, negative in aVR lead and some others. The amplitude in standard leads is 0.5-0.6 mV, in thoracic leads – 1.5-1.7 mV, its duration is 0.16-1.20 s.

The U wave is a small deviation up from the isoline, which is recorded after the T tooth in the leads V2 and V3 in some people. Its origin is unknown. Normally, its amplitude is approximately 2 mm. It has a certain diagnostic value in violation of the activity of the heart.

Excitation (Fig. 11), which occurred in the SA node, within 0.1 s spreads along the right and upper parts of the left atrium. The integral atrial vector is directed downwards and left and is displayed by the wave P. In the next 0.08-0.1 s, excitation spreads through the conductive system to the ventricles, the integral vector has a slight amplitude and is not recorded on the ECG. The excitation that has passed to the working myocardium of the ventricles, extends to different parts of the subendocardial layer towards the epicardium, causes the QRS complex, which is characterized by three vectors. The septum (Q vector), which occurs in the first 0.03 s, causes excitation of the left surface of the interventricular septum, directed upwards (to the base of the heart), the arrow shows the direction and magnitude of the integral vector to the right and forward. The main one, which occurs in the next 0.03-0.04 s, forms a wave R, which reflects the depolarization of the ventricles from the endocardium to the epicardium and is oriented down and to the left. Last (the next 0.035 s) the area of the right ventricle at the base of the pulmonary trunk is excited. The final integral vector of complex S reflects the

depolarization of the ventricular base in the next 0.03-0.04 s and is directed upwards, backwards, right or left. During the period of ventricular repolarization, reflecting the T wave and lasting 0.16-0.22 s, the integral vector is directed to the left, slightly down and forward.

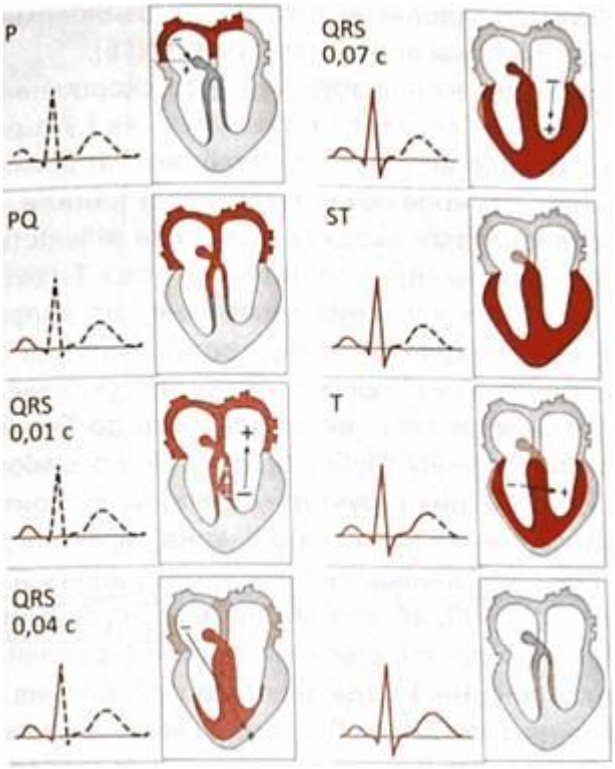


Fig. 11. Dynamics of excitation propagation and direction of the integral vector of the heart in the process of ECG development.

PQ interval – the distance on the ECG from the beginning of the wave P to the beginning of the wave Q, in the absence of the latter – to the beginning of the wave R, reflects the time from the beginning of atrial depolarization to the beginning of depolarization of the interventricular septum, which characterizes the rate of excitation by the atria, AV node, His bundle and its branches. Its duration is 0.1-0.21 s.

QRS interval – the distance on the ECG from the beginning of the wave Q to the end of the wave S, reflects the time of spread of depolarization by the ventricles of the heart. Its duration is 0.06-0.1 s.

The QT interval – the distance from the beginning of the wave Q to the end of the wave T, reflects the time of depolarization and repolarization of the ventricles and characterizes the speed of these processes. Its duration is 0.35–0.40 s.

The RR interval – the distance on the ECG between the tops of the teeth R, characterizes the duration of the cardiac cycle. Its duration is 0.8 s.

PQ segment – the distance on the isoline from the end of the wave P to the beginning of the wave Q, characterizes the duration of the delay in excitation in the AV node.

ST segment – the distance on the isoline from the end of the wave S to the beginning of the wave T, characterizes the time of complete depolarization of the ventricles.

Registration method. Before registering an ECG, it is necessary to wipe the skin at the diversion points with 96% ethyl alcohol or 20% soap solution, lubricate with electrode paste or put a pad of gauze moistened with isotonic sodium chloride solution under the electrode. Adjust the calibration signal so that the deviation of the pen by 10 mm corresponds to 1 mV. Turn on the movement of the instrument tape and record the calibration signal. The examinee should relax, lie quietly during the recording, breathe superficially. It is necessary to record several cardiac cycles in each of the leads and proceed to the ECG analysis. To do this, in all leads should be marked teeth and pay attention to their direction.

Simplified ECG analysis algorithm. Check the recording of the calibration signal. It should be equal to 1 mV (10 mm).

1. Determination of the driver of heart rhythm, frequency of contraction, rhythm.

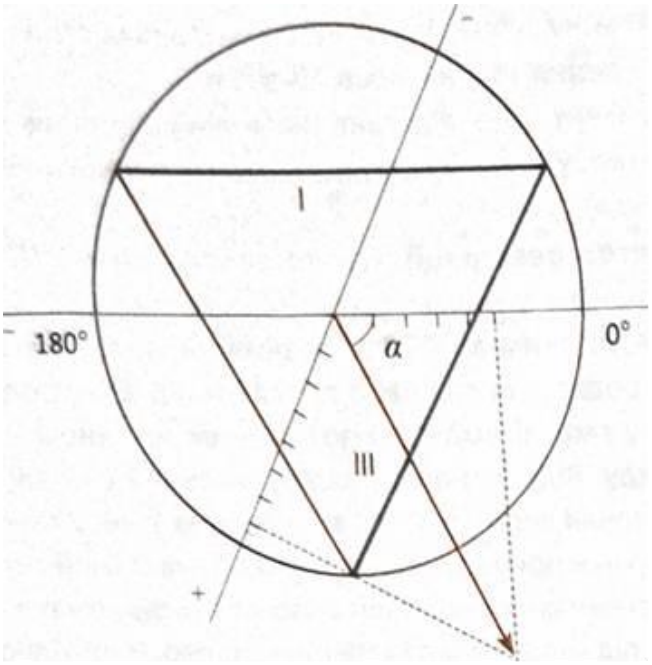
To establish the driver of the heart rhythm, it is necessary to trace in the standard leads the sequence of positive atrial wave P and ventricular complexes QRST, which are recorded when the driver of the rhythm is the sinoatrial node. To characterize the rhythm of pulse generation by the driver of the heart rhythm, it is necessary to determine the duration of several consecutive RR intervals and compare them with each other. The correct rhythm is considered when the recorded duration of the RR intervals differs from the average by no more than 10 %.

2. Determination of the intervals and duration of the teeth.

Measure the duration of PQ intervals. QRS, QT, RR and the duration of the P and T wave are compared with the proper values to draw conclusions about the rate of excitation propagation in the heart. The speed of movement of the electrocardiograph tape is set at 50 mm/s, so the movement of the tape by 1 mm is 0.02 s. To measure the duration of the teeth, the number of millimeters is calculated from the beginning to the end of the tooth and this number is multiplied by 0.02 s; The same is done in determining the appropriate intervals.

3. Determination of the direction of the electrical axis of the heart.

The electric axis of the heart characterizes the direction of depolarization of the ventricles of the heart and is the middle resulting vector QRS. Its position is determined by the magnitude of the angle a, formed by line I of the standard lead and the axis. The following axis position options are distinguished: 1) normal, when the angle a is from +30 to +69°: 2) vertical - the angle a is 70-90°; 3) horizontal - angle a ranges from 0 to +29°; 4) the deviation of the axis on the right - the angle A is from +91 to +180°; 5) the deviation of the axis on the left - the angle A is from 0 to - 90°. Normally, the electric axis of the heart lies in the sector from 0 to 90°, only occasionally going beyond these limits. As a rule, the electric axis coincides with the anatomical one.



Rice. 12. The electric axis of the heart.

In fig. 12 shows the Einthoven triangle, inscribed in the circle of determination of the angle a, characterizing the position of the electrical axis of the heart. The magnitude of the angle α can be determined graphically. To do this, draw a diagram of an isosceles triangle, the sides of which are lines of standard leads. This is the Einthoven triangle. Enter this triangle in a circle. Through the center draw lines parallel to the lines I and III leads. According to the ECG, find the algebraic sum of the teeth of the QRS complex in the I and III leads and set aside the segments corresponding to the found sums (taking into account the sign) on the lines drawn through the center. At the ends of the segments, hold perpendiculars to the lines I and III of the leads. From the center of the circle to the point of intersection of the perpendiculars, draw a line that will correspond to the position of the electrical axis of the heart. Using a protractor, determine the magnitude of the angle a located between the electric axis of the heart and the line I of the lead in the Einthoven triangle.

Myocardial contractility. Pumping function of the heart. Heart cycle. Atrial systole. Ventricular systole.

The heartis pratz rhythmically according to the pump principle (Fig. 13). The contraction of the heart (systole) alternates with its relaxation (diastole). Contractions and relaxations of the atria and ventricles under normal conditions are strictly mutually consistent and constitute a single cycle of the heart. The resting heart rate is individual and ranges from 60 to 80 beats per minute. With a heart rate of 75 per 1 minute, the duration of the cycle is 0.8 seconds.

Before starting work, the heart is in the common diastole of the atria and ventricles (general pause of the heart). At this time, the crescent valves are closed, atrioventricular open. Blood freely fills the cavities of the atria and ventricles. Their pressure is 0 mm mm Hg. Art..

The beginning of the heart is atrial systole. During this phase, which lasts 0.1 seconds, due to the reduction of muscle fibers, the pressure in the atrial cavities increases. In the right atrium, the pressure rises to 5-8 mm Hg., in the left to 8-15 mm Hg., which leads to the pushing of blood into the ventricles through open atrioventricular openings. The ventricles at this moment are relaxed (ventricular diastole), the flaps of the atrioventricular valves hang down and the blood freelyblows from the atria into the ventricles. The reverse flow of blood from the atria into the veins becomes impossible due to the reduction of muscle fibers located near the venous openings. Then atrial diastole is carried out, lasting 0.7 seconds. At the end of atrial systole, ventricular systole begins, lasting about 0.3 - 0.33 seconds. It is divided into two periods, respectively, consisting of phases. At the time of ventricular systole, the atria are already relaxed. Both ventricles contract simultaneously. The voltage period is extended until the opening of the crescent valves. To do this, it is necessary that the level of pressure in the ventricles becomes higher than in the great vessels. Diastolic pressure in the aorta is 70-80 mm Hg., in the pulmonary trunk 10-15 mmHg.

The voltage period lasts 0.08 seconds. It begins with the phase of asynchronous contraction (0.05 sec), when not all areas of the myocardium are covered by the contractile process and at this moment there is still no increase in pressure in the ventricular cavities.

In the isometric contraction phase (0.03 sec), the contractile process covers the bulk of the myocardium. The pressure in the ventricular cavities begins to increase significantly, reaching 15 - 20 mm Hg. Art. Century. in the right and 70 -

90 mm Hg.. in the left. Due to the increase in intraventricular pressure, the atrioventricular valves quickly choke, the crescent valves are also closed, so the ventricular cavity is closed and the volume of blood in it remains constant. As a result, the tension of muscle fibers increases without changing their length (isometric tension).

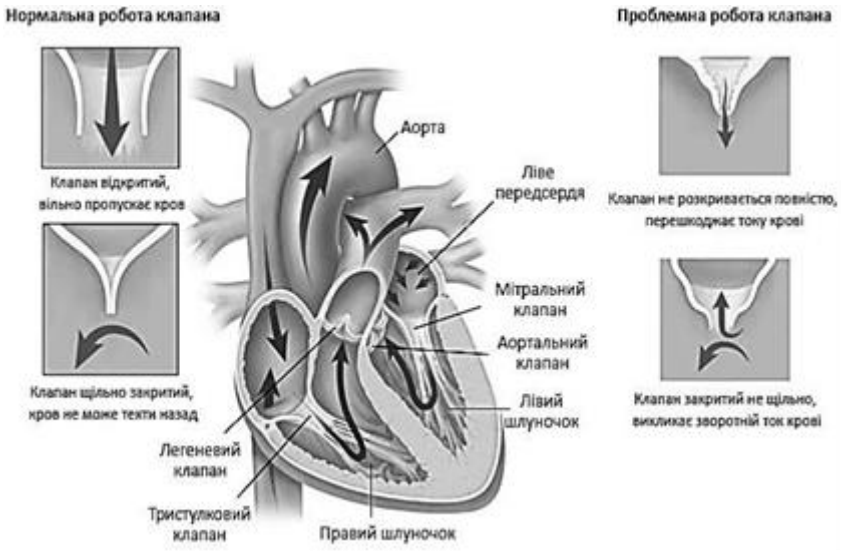


Fig. 13. Heart valves and direction of blood flow.

The period of expulsion of blood begins with the opening of the valves of the aorta and pulmonary trunk and lasts 0.25 seconds. This period consists of the phases of rapid (0.12 sec) and slow (0.13 sec) expulsion of blood. The opening of the aortic valves occurs when the pressure in the cavity of the left ventricle reaches 80 mm Hg., valves of the pulmonary trunk - 15 mm Hg.. in the cavity of the right ventricle. Myocardial contractions contribute to a further rise in pressure in the ventricular cavities in accordance with the right to 30 mm Hg., left - up to 120 mm Hg.. As a result of this increase in pressure, the blood is pushed into the aorta and pulmonary trunk very quickly (phase of rapid expulsion).

During the phase of rapid expulsion, most of the systolic volume of blood enters from the heart. As the vessels fill with blood, the pressure in them increases. The gravitating pressure between the ventricles and the vessels gradually decreases, the blood is poured out slowly - the phase of slow expulsion of blood begins. At the end of the expulsion of blood, ventricular diastole begins, lasting 0.47-0.5 seconds. It consists of a period of relaxation and a period of filling the ventricles. The time from the beginning of relaxation to the choking of the crescent valves by the return flow of blood is called the protodiastolic period. It lasts 0.05 seconds. With closed atrioventricular and crescent valves, the ventricles continue to relax until their pressure becomes lower than in the atria - a period of isometric relaxation begins (0.08 seconds). At this time, the peri-heartsare completely filled

with blood. When the pressure in the ventricles becomes slightly less than in the atria, the atriventricular valves open and the filling period begins. First, there is a rapid passive filling of the ventricles for 0.05 seconds (with atrial diastole), then - slow filling for 0.25 seconds.

A rest period of 0.4 seconds is enough for the heart to fully restore its performance. With an increase in heart rate, the duration of the heart cycle is reduced mainly due to the rest period. At the end of the slow filling phase, atrial systole occurs (0.1 sec). The atrium is pushed into the ventricles by an additional volume of blood, after which the heart cycle repeats again.

The amount of blood ejected by the ventricles with each contraction is called systolic, or shock volume (SV). The size of the systolic volume depends on gender, age, functional state of the organism. In a calm state, the systolic volume is 65-70 ml in men, in women 50-60 ml. The systolic volume of both ventricles is about the same. There should also be the same minute volume of blood circulation (CO), which is also called cardiac output. The value of the minute volume of blood circulation can be found by the formula SV x heart rate = CO. At rest, CO is 4.5-5.0 l. During exercise, the minute volume of blood circulation can increase to 20-30 liters.

Lesson 3.

Anatomical and physiological aspects of cardiac release of blood and factors affecting its magnitude.

The expulsion of blood from the ventricles of the heart into the aorta and pulmonary artery is carried out during systole due to the pressure gradient between the left ventricle and the aorta, the right ventricle and the pulmonary artery, which leads to the opening of the crescent valves. Cardiac output depends on contractility, preloading and post-loadingof the ventricular myocardium (Fig. 14). The amount of blood emitted by the heart in one contraction is called systolic (SV) or strock volume and is determined by the value of the minute volume (MV) and heart rate (HR): **SV=MV/HR**. In an adult, it is when itis in the range of 50-70 ml.

An integral indicator of the function of the heart as a pump is the minute volume of blood (MVB), which is due to SV and HR: MVB = **SV • HR**. At rest, in a person weighing 70 kg, its value is about 5 l / min with a consumption of 0.3 liters of oxygen in 1 minute. With maximum physical exertion, MVB can increase to 25-40 l/min.

Methods for determining of minute volume of blood flow

In animal experiments or on vessels accessible during operations, people use an electromagnetic or ultrasonic flowmeter, which registers the volume of flowing blood, its systolic and diastolic waves. The direct Fick method is also used, which makes it possible to calculate the minute of volume of blood flow by the arterio-venous difference in oxygen and the volume of oxygen that a person consumes in 1 minute. For example, blood flowing in the lungs absorbs 200 ml of oxygen in 1 minute. Arterial blood from the lungs is introduced into the left half of the heart and is released into the aorta, and from it into the arteries, the oxygen content in it is, respectively, 200 ml in 1 liter of blood. In the venous blood located in the right atrium or pulmonary artery, 160 ml of oxygen is retained by in blood. So, the arterio-venous difference is 40 ml of oxygen. If we divide 200 ml/min by 40 ml/min, we get 5 l, reflecting the value of minute volume of blood flow:

MVB (l/min) = O2, absorbed by the lungs (ml/min) / arterio-venous difference O2 (ml/L of blood).

The method is simple to calculate, but technically it is difficult to perform it in a general clinical institution, because it requires catheterization of the pulmonary artery or right atrium, arterial blood sampling.

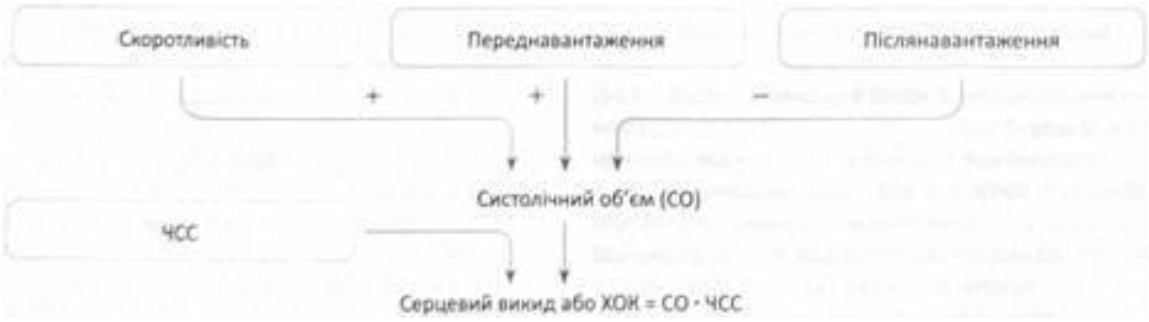


Fig. 14. The effect of preload and post-load on the amount of cardiac output. Sign "+" - increase of CO, sign "-" - its decrease.

Simpler and bloodless is the rheographic method based on the registration of the electrical resistance of a tissue or organ through which a weak high-frequency electric current is transmitted. According to the Kubichek formula, the milk yield(systolic) volume of blood is calculated:

$$УО = p \cdot l^2 / Zo^2 \cdot A_{диф} \cdot T\ (мл),$$

where: p – resistivity of blood (135 Ohm-cm);

I – the distance between the discharge electrodes (cm);

Zo – complete resistance of the fabric between the electrodes;

$A_{диф}$ – amplitude of the first derived rheogram;

T – duration of the exile period (c).

The breeding method has been widely used. A certain amount of dye or radioactive isotope is injected into the brachial vein, after which the average concentration of the substance in arterial blood is determined. Calculate HOC by the formula:

$$XOK = 60 \cdot J / C \cdot T,$$

where: J – amount of injected substance;

C – the average concentration of the substance, calculated by the dilution curve;

T – duration of the first wave of circulation;

60 – 1 min.

The method of thermodilution (the introduction of a cooled isotonic solution of sodium chloride) allows you to repeatedly determine the minute volume of blood flow in the same person for a short time.

Mechanical work and heart tones

A change in body position is accompanied by significant changes in the work of the heart. That is why the normal values of cardiac output in humans are given for standard conditions – the horizontal position of the body. The transfer of the body from a horizontal position to a vertical one causes the deposition of blood in the veins of the lower half of the body. As a result, there will be less return of blood to the right cavities of the heart, and then to the left, and therefore a decrease in systolic volume. A decrease in systolic blood volume when a person is transferred to a vertical position is observed naturally, and this decrease is 30-40% to the size of the systolic volume in a horizontal position.

A decrease in the venous return of blood to the heart is not by itself a direct cause of a decrease in systolic volume, since the reserve volume of blood is quite sufficient to ensure cardiac output. To do this, you only need to increase the power of the heartbeat. This is the relative lack of power of the heartbeat and is the direct cause of the decrease in systolic blood volume. At the same time, the duration of intraventricular pressure drops.

The transition from the horizontal position of the body to the vertical (orthostasis) leads to a change in hydrostatic pressure in the vascular system. The action of gravity makes it difficult for blood to return to the heart from the veins, even in healthy individuals, with relaxed leg muscles, an additional 300 to 800 ml of blood is retained. As a result, venous return and, accordingly, the shock volume of the heart decreases. As a result, impulses from the mechanoreceptors of the aorta, carotid sinus, pulmonary artery trunk fall, which leads to a narrowing of the resistive and capacitive vessels and an increase in heart rate by no more than 20 beats / min. Systolic blood pressure decreases briefly (in the first 1–2 minutes) and returns to its original value, and diastolic blood pressure rises by no more than 10 mmHg. The movement of blood into the vessels during short-term standing and especially when walking is normally prevented by active tension and contraction of the muscles of the legs, which reduces the capacity of the veins.

A decrease in systolic volume under orthostatic effects is usually accompanied by a compensatory acceleration of heart contractions. Due to this, minute volume of blood flow is reduced slightly. With a high quality of regulation of the circulatory system, a change in body position from horizontal to vertical is not accompanied by any unpleasant sensations.

In case of insufficient compensatory reactions to orthostatic load, orthostatic circulatory disorders develop, especially dangerous for the brain. Subjectively, this is manifested by thuggery, "darkening" in the eyes, even loss of consciousness is possible. With prolonged orthostasis, due to high hydrostatic pressure, there is an excessive filtration of the liquid part of the blood in the capillaries, which leads to some hemoconcentration, a decrease in the volume of circulating blood, the occurrence of swelling of the feet.

When moving from a vertical to a horizontal position (clinostasis), a decrease in heart rate is observed, which reaches its original value on average in 20 s. Subsequently, clinostatic exposure leads to a decrease in heart rate below baseline by 4–6 per minute. During just 10 minutes of clinostasis, a decrease below baseline diastolic blood pressure is mainly observed. These hemodynamic reactions are due to an increase in impulses from the mechanoreceptors of the aorta, carotid sinus, trunk of the pulmonary artery.

During the activity of the heart, sound phenomena arise, called heart tones. In healthy people, during auscultation of the heart, two tones are well heard - the I tone that occurs during systole is systolic, and the II tone that occurs during diastole is diastolic (Fig. 15).

At the heart of heart tones are oscillatory movements of various structures of the heart: valves, muscles, vascular wall. Like any oscillation, heart tones are

characterized by parameters such as intensity /amplitude/, frequency/number of oscillations of 1 sec/ and duration. Currently, most authors believe that it is possible to distinguish 6 normal heart tones. At the same time, I and II tones are always heard, III and IV tones are not determined in all people /more often recorded on the phonocardiogram/, V and VI are detected only on the phonocardiogram. The latter are investigated very rarely. Therefore, we will consider only the mechanism of origin of I-IV tones and the place of their listening.

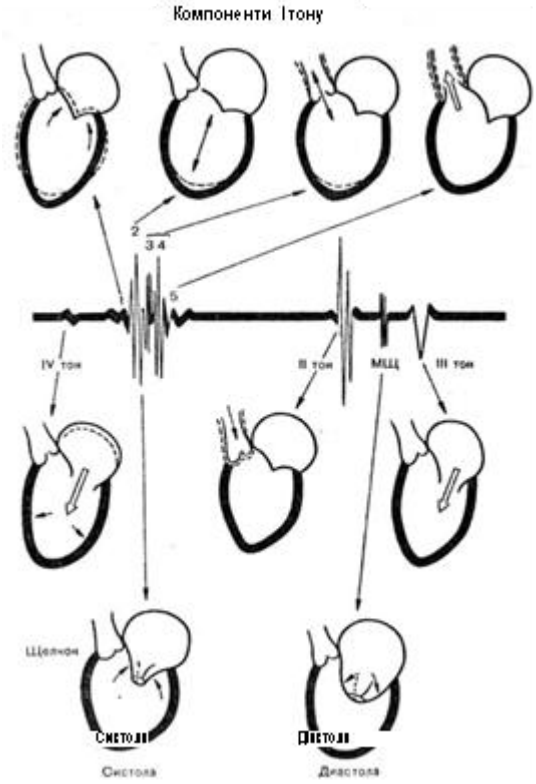


Fig. 15. Formation of heart tones.

The first tone is heard as a short, rather intense sound above the heart, but it is optimally expressed in the area of the apex of the heart. 3 1 tone begins systole cardiac activity. The main component is the valve component. It is caused by oscillation of the valves of the atrio-ventricular valves and tendon filaments in the phase of isometric contraction. The second component – muscular – occurs as a result of fluctuations associated with ventricular myocardial tension during isometric contraction. The third component – vascular – oscillations of the initial parts of the aorta and pulmonary artery, the opening of the crescent valves in the phase of rapid expulsion. The fourth component – the atrial – arises as a result of fluctuations associated with the contraction of the atria. In auscultation, the first tone begins with this component, since the vibrations caused by the atrial systole merge with the sound vibrations caused by the ventricular systole and are auscultatively perceived as one tone.

Under physiological conditions, there may be an increase or decrease in the volume of the first tone. Increased volume tone can be observed in people with a thin chest wall, with physical exertion, nervous excitement, tilting the body forward. The weakening of the volume of the first tone is associated with a thickening of the chest wall due to the deposition of a significant amount of fatty tissue, expressed by the development of the pectoral muscles.

In addition to changing the volume of the psrsh tone, its physiological bifurcation can be observed (instead of one heart tone, two consecutive short tones are heard). This is due to the asynchronous activity of the ventricles, namely the delay in the end of the systole of the right ventricle.

The second tone is optimally heard in the second intercostal space on the left (above the pulmonary artery) and on the right (above the aorta) from the sternum during diastole. It is formed due to oscillations arising at the beginning of diastole when the crescent valves of the aorta and pulmonary artery are closed, by the flow of blood that hits them. This is the first, valve component.

The second component is vascular due to fluctuations in the walls of the aorta and pulmonary artery.

The third tone can be heard sometimes in children, or in people with a thin chest. It is caused by the rapid filling of the ventricles with blood during the rapid filling phase.

Functional noise.

They arise due to a violation of the laminarity of blood flow in the cavities of the heart and blood vessels. These noises can be heard either between the first and second tones, that is, during a systolic pause, or between the second and subsequent first tone, that is, during a diastolic pause. In the first case, the noise is called systolic, and in the second - diastolic.

Heart murmurs are also divided into functional and organic. The latter are caused by changes in the structure of the valve apparatus (valve insufficiency), or a decrease in the diameter (stenosis) of the openings.

Functional heart murmurs occur when the ratio of the size of the cavities and valve openings of the heart changes with the formation of so-called relative stenosis or valve insufficiency. This is observed in childhood and young age. Functional noises, with extremely rare exceptions, are systolic and are most often heard over the pulmonary artery. This is explained by the fact that in childhood the difference between the lumen of the right ventricle and the lumen of the pulmonary artery is greater than the corresponding difference between the left ventricle and

the aorta. Also, the pulmonary artery in children is easily compressed by deep exhalation due to an increase in intrathoracic pressure.

Functional noises are characterized by significant lability: they either appear or disappear, arising at one position of the subject's body, they can disappear at the second. Their appearance is associated with mental arousal, or with physical exertion. With a deep breath, they either weaken sharply or disappear altogether, at the end of the breath, on the contrary, they appear or intensify.

Regulation of heart activity. Interaction of intra- and extracardial nerve systems. Reflective and humoral regulation of heart activity

The activity of the heart can be regulated by local myogenic, nervous and humoral mechanisms.

Myogenic mechanisms are associated with the physiological properties of heart structures. The study of the dependence of the force of contractions of the heart on the stretching of its chambers showed that the strength of each heart contraction depends on the volume of venous blood flow and is determined by the final diastolic length of myocardial fibers. As a result, the Frank-Starling law was formulated, according to which, the more the heart stretches into the diastole phase, the more it will contract during the systoles.

There are also myogenic mechanisms, for the implementation of which the degree of end-diastolic stretching of myocardial fibers does not matter. Thus, the dependence of the force of contraction of the heart on the pressure in the aorta (Anrep effect) was revealed. An increase in pressure in the aorta initially causes a decrease in the systotic volume ofthe heart and an increase in the residual final diastolic volume, as a result of which there is an increase in the force of contraction of the heart and cardiac output stabilizes at a new level of contraction force (increase in systolic blood volume).

Chronoinotropy belongs to myogenic mechanisms, that is, the dependence of the contraction force of the heart on the frequency of its activity (Bowdich's ladder). In response to an increase in rhythm, the human heart reacts by increasing the strength of contractions, and, conversely, with a decrease in rhythm, the force of contractions decreases. The basis of this phenomenon is an increase or decrease in the intracellular concentration of Ca2+ and a change in the number of actin-myosin complexes.

Nervous regulation of the heart is quite complex and its implementation is carried out by intracardial and extracardial mechanisms.

Intracardiac nervous regulation is provided by the peculiarities of the functioning of cardiomyocytes and the myocardium as a whole. So, in particular, cardiomyocytes are able to synthesize various proteins when they are destroyed during the performance of a contractile function. The fastest decay of energy-rich compounds of ATP and glycogen occurs during systole and corresponds to the QRS electrocardiogram complex. Resynthesis and restoration of the level of these substances has time to fully realize during diastole.

Cardiomyocytes are able to selectively adsorb from circulating blood and accumulate substances in the cytoplasm that support their bioenergy and compounds that increase the cells' oxygen demand. For example, adsorption from the blood of catecholamines (adrenaline, norepinephrine), which occurs due to intracellular mechanisms, provides an increase in cardiac circulation.

Inserted discs - nexuses provide a compound of myofibrils, the transition of excitation from cell to cell. Also, due to the nexuses, cardiomyocytes interact with connective tissue cells that make up the myocardial stroma and are sources of high-molecular organic compounds necessary to perform the function and maintain the cell structure. The presence of nexuses allows the myocardium to respond to excitation as a functional syncytium.

Peripheral reflexes are formed in the heart, the arc of which closes in the intramural ganglia of the myocardium. After heart transplantation and degeneration of nerve elements of extracardial origin, it is the intraorgan nervous system that is preserved and functioning in the heart, organized according to the reflex principle. It has a receptor apparatus on myocardial fibers and coronary vessels (receptors that perceive active tension and passive wall stretching), afferent neurons, interneurons and efferent neurons, the axons of which innervate the myocardium and smooth muscles of the coronary vessels. Intracardiac reflexes provide a change in the contractile activity of various parts of the heart. For example, an increase in the stretching of the myocardium of the right atrium (with an increase in blood flow to the heart) causes an increase in the contractile activity of the myocardium of the left ventricle. That is, the reduction of not only the part of the heart that stretches the blood, but also other departments is enhanced in order to "free up space" for the blood and accelerate its release into the arterial system. These reactions occur with the help of intracardiac peripheral reflexes (G.I. Kositsky). This response is observed against the background of low initial filling of the heart and relatively low pressure in the aorta and coronary vessels. With overflow of the chambers of the heart, aorta and coronary vessels, stretching of the atria inhibits

the contractile activity of the myocardium of the ventricles. As a result, cardiac output decreases and pressure in the aorta and coronary vessels decreases.

The intracardial nervous system is not completely autonomous, it is included in a complex hierarchy of nervous mechanisms that regulate the activity of the heart. Under the conditions of a whole organism, peripheral intracardial reflexes interact closely with reflexes that close at the level of other parts of the autonomic nervous system.

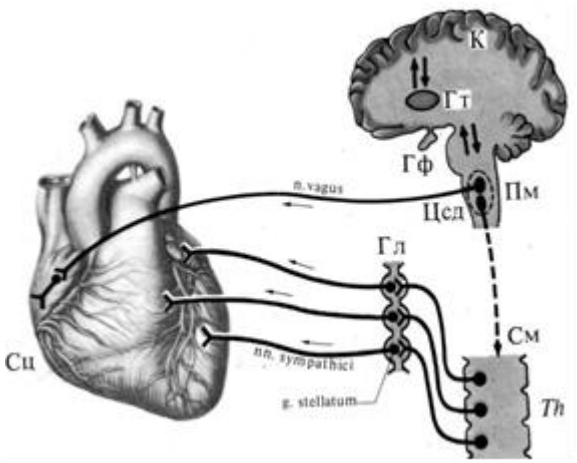


Fig.16. Inervation of the heart.

Extracardial nervous regulation is provided by special mechanisms in which such parts of the brain as the cortex, the stem part of the brain, the medulla oblongata, the spinal cord are involved - which transmit their influences along the fibers of the vagus and sympathetic nerves that innervate the heart (Fig. 16). The work of the heart will ultimately depend on their functional activity.

Effects of the vagus nerve. The nuclei of the vagus nerves are located in the medulla oblongata, from which the axons of the cells go to the heart. The fibers of the right vagus nerve are distributed mainly in the right atrium, innervate the myocardium, coronary vessels and especially densely penetrate the sinus node. The fibers of the left vagus nerve transmit their effects to the atrial-ventricular node. Therefore, with the enhanced work of the right vagus nerve, the heart rate (chronotropic action) mainly changes, the left - atrioventricular conduction (dromotropic action) and the amplitude of contraction (inotropic action) (Fig. 17).

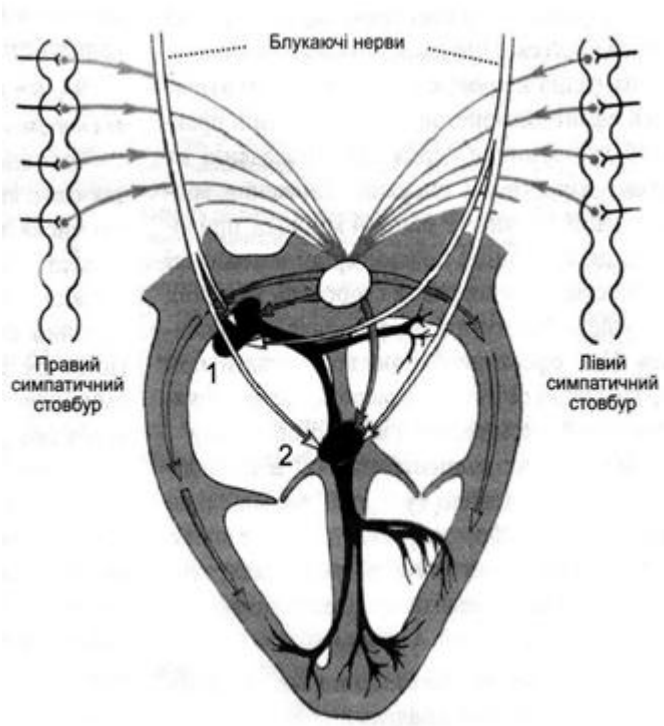
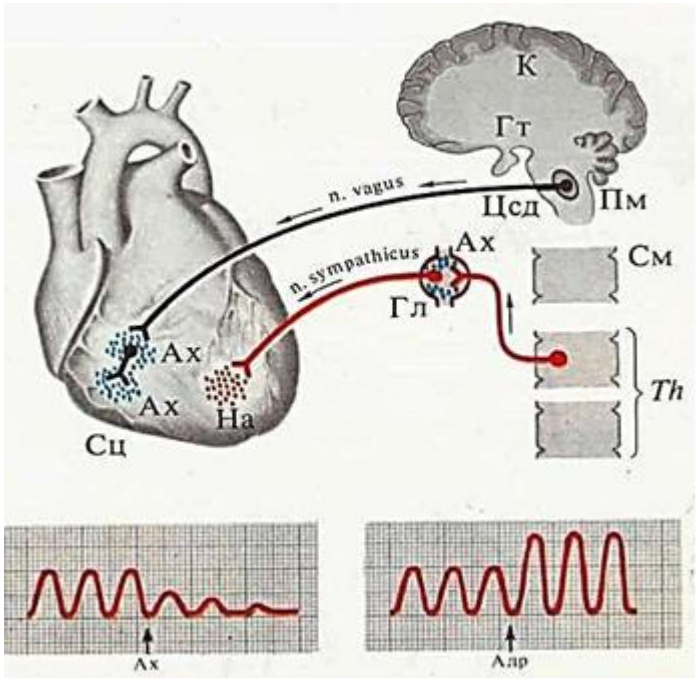


Fig. 17. The influence of the ***wicked nerve on the work of the heart.***

Irritation of the vagus nerves leads to negative changes in the work of the heart. In particular, there is a negative bathmotropic effect (a decrease in the excitability of the heart muscle), a negative dromotropic effect (decrease in the speed of conduct), a negative chronotropic effect (slowing down of the heart rhythm), a negative tonotropic effect (decrease in myocardial tone), a negative inotropic effect (decrease in the amplitude of contractions). With severe irritation of the vagus nerve, a complete cessation of cardiac activity may occur.

Effects of sympathetic nerves. The influence of sympathetic nerves is the exact opposite and is manifested in an increase in the excitability of the heart - a positive bathmotropic effect, an improvement in the conduct of arousal in the heart - a positive dromotropic effect, an increase in the frequency of heart contractions - a positive chronotropic effect, an increase in heart tone - a positive tonotropic effect, an increase in contractions of the atria and ventricles - a positive inotropic effect. When sympathetic fibers are irritated, spontaneous depolarization of rhythm driver cells in diastole is accelerated, which leads to an acceleration of heart contractions. Theamplitude of the action potential is also increased.

The basis of the effects caused by the stimulation of the peripheral ends of the autonomic nerves is the release of biologically active substances (mediators): acetylcholine - in the parasympathetic region and noradreof enalin - in the sympathetic (Fig. 18).



Rice. 18. Mediators of the heart nerves and their effects.

Acetylcholine increases the permeability of membranes to K+ ions and thereby causes hyperpolarization, stimulates the formation of cGMP, which leads to inactivation of Ca2+ channels. As a result of these effects, there is a decrease in the rate of depolarization, a reduction in the duration of the action potential, a decrease in the force of contraction. Norepinephrine, stimulating β-adrenergic receptors, increases the concentration of adenylate cyclase, cAMP, which leads to the activation of Ca2+ channels, the accumulation of intracellular Ca2+ and an increase in heart activity.

The central parts that regulate the function of the heart (and blood vessels) include neurons located in the medulla oblongata, hypothalamus, and the cerebral cortex. At the bottom of the fourth ventricle in the dorsolateral (pressor) section there are neurons, the excitation of which through sympathetic nerves is accompanied by increased heart activity and expansion of the coronary vessels (Fig. 26). Excitation of the ventromedial (depressor) section causes the opposite effect. In the hypothalamus there are areas whose irritation leads to a change in cardiac activity. Changes in rhythm, myocardial contraction forces can also be caused by irritation of certain parts of the limbic system and cerebral cortex.

The reflexes that regulate the activity of the heart can also include a whole group of reactions associated with the work of various receptive zones - for example, from receptors for stretching the mouth of hollow veins - the Bainbridge reflex: with an increase in blood pressure in the hollow veins, a reflex decrease in the tone of the vagus nerve and an increase in the tone of the sympathetic one

occurs, which leads to an increase in the work of the heart. Vagus reflexes, which manifest as a slowdown in cardiac activity, include reflexes from peritoneal receptors (Holtz reflex) and from eyeball receptors (Danini-Ashner ocular-cardiac reflex).

The humoral mechanism of heart regulation is carried out by biologically active substances released into the blood from the endocrine glands, as well as the ionic composition of the intercellular fluid. In the atria around the myofibrils are specialized cells that are similar to thyroid cells or the adenohypophysis. They form biologically active compounds (products of arachidonic acid, catecholamines, digitalis-like factors) and hormones – atrial sodium-uretic peptide and renin-angiotensin compounds. These biologically active substances are released by stretching the atria, a steady increase in pressure in the aorta, and an increase in the tone of n. vagus. Atrial hormones are involved in the regulation of myocardial contractile activity, minute circulation. Adrenaline through the adenylate cyclase system provides myocardial energy during the breakdown of intracellular glycogen to form glucose, increases the permeability of cell membranes to Ca2+ ions. Glucagon provides a positive inotropic effect by stimulating adenylate cyclase. Thyroxine increases heart rate and increases the sensitivity of the heart to sympathetic effects. Corticosteroids, serotonin, angiotensin - increase the strength of myocardial contractions. Ca2+ ions increase excitability and conductivity of muscle fibers, activating phosphorylase and providing a combination of excitation and contraction. Reduce the action of the heart muscle - acetylcholine, K+ , HCO- , H +, individual prostaglandins**.**

Lesson 4.

Anatomical and physiological aspects of blood vessels and their role in blood circulation. The basic law of the severity of blood movement.

Blood vessels are elastic tubes with which blood is transported to all organs and tissues, and then collected again to the heart. The study of blood vessels, along with lymphatic vessels, is engaged in the section of medicine – angiology. Blood vessels form: a) the macrocirculatory bed – these are arteries and veins with which blood moves from the heart to the organs and returns to the heart; b) microcirculating bed – includes capillaries, arterioles and venules located in organs that provide metabolism between blood and tissues.

Arteries are the blood vessels with which blood moves from the heart to organs and tissues. The walls of the arteries have three layers:

• the outer layer is built of loose connective tissue, nerves pass in it, regulating the expansion and narrowing of blood vessels;

• the middle layer consists of a smooth muscle membrane and elastic fibers (due to the contraction or relaxation of the muscles, the lumen of blood vessels can change, regulating the flow of blood, and elastic fibers give the vessels elasticity);

• inner layer – formed by a special connective tissue, the cells of which have very smooth membranes that do not interfere with the movement of blood.

Depending on the diameter of the arteries, they also change the structure of the wall, so there are three types of arteries: elastic (for example, aorta, pulmonary trunk), muscular (arteries of organs) and mixed, or muscular-elastic (for example, carotid artery) type.

Capillaries are the smallest blood vessels that connect arteries and veins and provide metabolism between blood and tissue fluid. their diameter is about 1 micron, the total surface of allx capillaries of the body is 6300 m^2 . The walls consist of one layer of squamous epithelial cells – the endothelium. The endothelium is the inner layer of flat, elongated cells with uneven wavy edges, which lined the capillaries, as well as all other vessels and the heart. Endotheliocytes produce a number of physiologically active substances. Among them is nitric oxide, which causes relaxation of smooth myocytes, thereby causing the expansion of blood vessels. In organs, capillaries provide microcirculation of blood and form a network, but can also form loops (for example, in the papillae of the skin), as well as glomeruli (for example, in kidney nephrons). Different organs have different levels of development of the capillary grid. For example, in the skin by 1 mm^2 there are 40 capillaries, and in the muscles – about 1000. Significant development of the capillary network has the gray matter of the central nervous system, endocrine glands, skeletal muscles, heart, adipose tissue.

Veins are blood vessels with which blood moves from organs and tissues to the heart. They have the same wall structure as arteries, but are thinner and less elastic. In the middle and some large veins there are crescent valves that ensure the flow of blood in only one direction. Veins are muscular (hollow) and muscleless (retina, bones). The movement of blood through the veins to the heart contributes to the absorption effect of the heart, stretching of the vena cava in the chest cavity during inhalation of air, the presence of a valve apparatus.

The movement of blood through the vessels obeys the laws based on the laws of hydrodynamics. The section of hydrodynamics that studies the causes, conditions and mechanisms of blood movement in the cardiovascular system is called *hemodynamics*.

The factors determining the blood flow through the vessels include: pressure, *resistance and speed*. The force that creates the initial pressure in the vascular system is the heart. In a middle-aged person, with each contraction of the heart, 60-70 ml of blood (systolic volume) is pushed into the vascular system, which is 4-5 liters per minute (minute volume of blood circulation). The pressure difference at the beginning and end of the great circle of blood circulation (120-0 mmHg contributes to the movement of blood through the vessels), Interferes with movement – vascular resistance, which depends on the length of the vessel, blood viscosity and vascular radius.

Pressure in different parts of the vascular system. In the systemic circle of hemodynamics, due to the alternation of contraction and relaxation of the left ventricle, the pressure in the aorta is pulsating in nature, varying from about 80 mmHg. in diastole (diastolic blood pressure - ATD) up to about 120 mmHg in systole (systolic blood pressure - ATS). As the blood moves to the right atrium, the pressure in the vascular system decreases to zero in the hollow veins. The pressure in the capillaries of the great circle varies from 35 mmHg. at the arterial end up to 10 mm. Hg at the venous end, with the exception of the glomerular capillaries in the kidneys. At the same time, in the capillaries there are practically no pulse fluctuations in blood flow, and in arterioles they are minimally expressed. In the vessels of the small circle, the blood pressure reaches the highest values in the pulmonary artery (ATS = 25 mmHg, ATD = 10 mmHg), and the average blood pressure in the pulmonary capillaries is only 7 mmHg, which is almost impossible to filter the fluid into the alveoli. Blood from the capillaries enters the venules. The smooth muscles of these vessels can block their lumen. In these cases, a lot of blood can accumulate in the organs. The increase in the amount of blood due to the overlapping of venules is especially noticeable in organs such as the liver, lungs, spleen, skin and abdominal vessels. These organs are called the blood depot. Thus, the abdominal vessels can hold about 45% of all blood. The reserve blood of these organs, if necessary, enters the bloodstream with the help of reflex and humoral mechanisms of regulation. Most veins have valves that direct the movement of blood, from the capillaries to the heart. Especially a lot of these valves in the veins of the limbs, where they are well developed.

The movement of blood through the veins is provided by three factors:

1. A significant number of veins are located between the muscles, mainly skeletal. When contracting, they press on the veins and squeeze out the blood, ensuring its movement, rushing to the heart with valves.

2.The second factor is due to the difference in blood pressure. In the venules and middle veins it is larger than in the mouths of the vena cava, where it is even negative.

3. Blood moves through the veins due to the suction action of the heart and chest. This in turn is caused by negative pressure in the pleural cavity and a decrease in pressure in the chest cavity in the inhalation phase.

Blood moves along a closed bloodstream from the place where its pressure is greater (aortic arch) to the mouth of the vena cava, where it is zero. The blood pressure in the vessels decreases as it moves away from the heart, because their general lumen increases due to branching into small vessels. The bloodstream is narrowest at the level of the aorta and the widest at the level of capillaries. The lumen of the aorta is 500 times narrower than the total lumen of the capillaries. Part of the blood pressure is spent on the friction of blood particles between themselves and the walls of blood vessels, as well as on the stretching of their elastic fibers. The elasticity of arterial blood vessels creates a feature of blood movement, giving the blood flow a pulsating character. This applies only to arterial vessels. At the time of systole, the speed of blood flow in them increases, and during diastole, blood slows down in the veins evenly.

Blood circulation in the vascular system obeys three basic principles (A.Gyton, 2010):

1) The degree of blood supply to eachtissue of the body is always consistent with its metabolic needs. The range of changes in the blood supply of individual organs and tkanin with their active functioning of the bath reaches 20-30 times the level of p o in relation to its knowledge at rest. At the same time, the ace heart can maximize the pain of sewing minute volume of blood flow only 5-6 times. This means that there are mechanisms for the redistribution of bloodat the local level from active tissues to less active ones. Obviously, the state of metabolism (degree of hypoxia, hypercapnia, intermediate metabolites of metabolism) affects the tone of blood flow distribution vessels, causing their vasodilation or vasoconstriction. This basic mechanism, if necessary, is complemented by refle and humoral influences from the central nervous system and the endocrine system.

2) Minute volume of blood flow is controlled by the sum of all local blood flow in the tissues. The blood returning to the heart from the tissues forms a venous turn, which automatically through the Frank-Starling law leads to its release into a small circle and return to systemic hemodynamics. With insufficienteffectiveness of this mechanism, the central nervous system and the endocrine system are connected to the regulation of minute volume of blood flow.

3) Blood pressure is regulated regardless of the regulation of local blood flow intissues ax and depending on the regulation of minute volume of blood flow. The cardiovascular system has a very sensitive sensory apparatus to control the

level of systemic blood pressure. These are baroreceptors of the aortic arch, carotid sinus, vascular mechanoreceptors.

Monitoring of systemic blood pressure immediately includes mechanisms for its normalization in case of deviation from optimal values, especially in the case of hypotension. For example, with a decrease in average blood pressure below 60 mm. Hg.. Baroreceptor reflexes enhance the discharge function of the heart, constrict arterioles in most regions of the body, and return deposited blood from the veins due to their vasoconstriction. Hormonal mechanisms (RAAS) are activated and the excretory function of the kidneys changes in the direction of fluid retention in the body.

Under physiological conditions, in almost all parts of the vascular system, a laminar (layer-by-layer) type of blood flow is observed. With this type of flow, the blood moves in cylindrical layers, the axis of which coincides with the axis of the vessel. Blood particles in each layer move relative to the others parallel to the axis of the vessel. At the same time, the layer that is directly adjacent to the wall of the vessel sticks to it and remains motionless, another slides along this layer, the next one along it, etc. As a result, the axial flow consists almost entirely of red blood cells, which form a compact movable cylinder covered with a plasma shell. Under certain conditions, the laminar flow turns into turbulent. A turbulent flow is characterized by the presence of turbulences in which blood particles move not only parallel to the axis of the vessel, but also perpendicular to it. These turbulences significantly increase the internal friction of the blood and the flow profile is significantly flattened.

The main indicators of hemodynamics are: volumetric speed of blood flow, linear speed of blood movement, blood circulation rate, pressure in different parts of the vascular system.

The volumetric speed of blood flow is characterized by the amount of blood passing through the cross section of any part of the circulatory system per unit of time. Due to the continuity of blood flow through the cardiovascular system, the volume of blood flowing through each section of the vascular bed per unit of time is the same. This means that in 1 minute, for example, through the aorta, or through the pulmonary artery, or through the total cross section of the capillaries the same volume of blood flows, equal to minute volume of blood flow.

In different organs, it is different (for example, in the vessels of the brain about 750 ml / min, kidneys - 1200 ml / min) and varies depending on the functional state of the organ. The volumetric speed of blood can increase or decrease in any particular organ depending on its activity.

The linear velocity of blood flow is characterized by the length of the circulatory system, which blood flows per unit of time with laminar blood flow. It is expressed in m/sec. It is inversely proportional to the lumen of blood vessels. The linear velocity is different in some parts of the vascular bed - in the aorta - 0.20 m / s, arteries - 0.3-0.5 m / s, in capillaries - 0.03 cm / s, in the veins - 1-5 cm / s. The erythrocyte passes the capillary in 1 s, and 1 mm3 of blood — in 4-6 hours. All this contributes to a fast metabolism. In the veins, the blood flow rate gradually increases to 33 cm/s as they combine into two hollow veins. In these veins, the speed of blood flow depends on the respiratory movements of the chest. During inhalation, it accelerates, and slows down during exhalation. In the middle veins, blood flows at a rate of 6-14 cm / s. Thus, the speed of blood flow in the veins is about 2 times less than in the arteries, which in the body are almost 2 times less than the veins. The time during which a piece of blood passes a certain part of the circulatory system is called the time of blood flow, it is determined using neutral colors or pharmacological substances that are administered intravenously.

In a similar way, with the help of paints, you can determine the time of the full flow of blood in two circles of blood circulation. The speed of the blood cycle reflects the time during which the formed elements of the blood pass a large and small circle of blood circulation. On average, it is 14-20 seconds. An indicator that the blood has passed two circles of blood circulation is the appearance of paint in a vein, symmetrical to the one into which the paint was injected.

Other methods are also used to determine the time of blood flow: plethysmography, ultrasound and the method of labeled atoms using isotopes, in particular sodium.

Arteries are considered vessels responsible for transporting blood to arterioles and pushing blood towards the heart in the diastole phase when the heart does not generate high pressure. This functionis facilitated by the peculiarity of the structure of their walls, namely, the presence of a powerful elastic layer, which accumulates part of the kinetic energy of the blood when stretching the wall in the systole and removesthis energy when pushing bath of blood in diastole. The described process is characterized by the formation in the artery of systolic (maximum pressure) in systole during the release of blood from the ventricles and its reductionto diastolic (minimal) pressure immediately before the subsequent release of blood. The range of oscillations (norm) of systolic pressure in healthy people is 100-140 mmHg, and diastolic pressure is 60-90 mmHg. Usually, in women, pressure indicators of 5-10 mmHg. lower than in men.

The difference between systolic and diastolic pressure is called pulse pressure. It is proportional to the volume of blood ejected by the heart with each systole. The larger the systolic volume, the shorter the period of expulsion and the

stiffer the wall of the arteries, the greater the magnitude of the pulse pressure. The rigidity of the vascular wall increases significantly with arteriosclerosis, as well as in all people – with age. Therefore, in this category of persons, while maintaining the normal discharge function of the heart, a higher pulse pressure is recorded.

As you move through the vessels, the pressure changes. If in the aorta it is equal to 120-130 mmHg, in the arteries 100-120, arterioles - 40-80, capillaries - 20-40, veins - 10-5-0 mmHg.

In addition to these types of pressure, there is also the so-called average blood pressure. It is an equivalent fluctuation of blood pressure in different phases of the heart cycle, that is, the average size of the pressure without pulse fluctuations.

To measure blood pressure in clinical practice, the auscultative Korotkov method is used most closely and when lad, which is called a sphygmomanometer. A cuff with a built-in pressure sensor is applied to the patient's shoulder and air is injected into it until a pressure exceeding systolic pressure (usually more than 180 mmHg) is reached. After that, slow decompression of air in the cuff and using a stethoscope is carried out, superimposed over the projection of a.radialis in the elbow bend, listening to specific sound phenomena – Korotkov tones. The appearance of tones corresponds to the moment of equalization of the pressure in the cuff with systolic pressure, and their disappearance – the moment of equalization of air pressure with diastolic pressure, when the cuff no longer interferes with the free flow of blood. The mechanism of occurrence of Korotkov tones is associated with the formation of turbulent blood flow in the area of the vessel narrowed by the cuff.

Blood flow in arterioles and mechanisms of its regulation.

Arterioles perform 2 main functions in the vascular system:

1. Theyare responsible for the regional redistribution of blood flow between metabolically active and inactive tissues and organs at a certain level of systemic blood pressure;

2. They largely form an adequate level of systemic blood pressure due to the effect on the overall peripheral resistance. The implementation of these functions is subordinated to the main morphological personality of their wall – the presence of a powerful circular smooth muscle layer, which can change its tone under the action of various regulatory mechanisms. Even without the influence of these regulatory factors, the smooth muscles of the arterioles have a background level of contraction, which is called basal tone. It is formed due to spontaneous myogenic activity of smooth muscle cells and background sympathetic stimulation.

Regulatory stimuli alter this tone by regulating the concentration of calcium ions in the cytosol of muscle fibers. The result of this regulation is either vasoconstriction (with an increase in the concentration of Ca + 2), or vasodilation (with a decrease in Ca + 2). All mechanisms that control vasoconstriction and vasodilation can be divided into 3 main groups:

1. Mehanisms of local control;

2. Withexternal neurogenic regulation of arteriole tone;

3. Gormonal control of arteriole tone.

Local control mechanisms operate independently of reflector and humoral control and coordinate local blood flow with the metabolic needs of the region.

Manifestations of the action of these mechanisms are working hyperemia, reactive hyperemia, myogenic autoregulatory reactions and local response to tissue damage.

Working hyperemia is the result of vasodilation due to hypoxia and accumulation of vasoactive agents in the extracellular space that sharpens the vascular wall of arterioles. These factors include: carbon dioxide secreted by cells as the final product of substrate oxidation; hydrogen ions secreted by intermediate metabolic products (for example, lactic acid in skeletal muscles); adenosine is a decay product of ATP, which is intensively used in various cellular processes; potassium ions that accumulate with frequent repetition of the AP repolarization in actively functioning cells; osmotically active substances (ions, low molecular weight peptides, glucose, etc.), which accumulate with active metabolism; ecosanoids (prostaglandins, prostacyclins).

Physical factors that can induce working hyperemia include local warming of tissues. This method is often used in the clinic to stimulate blood circulation in pathologically changed areas of the body (compresses). If there is a need to limit blood flow in such areas, then, on the contrary, cold is applied to them (for example, bags of ice in acute travmah).

Reactive hyperemia is a sharp increase in blood flow that occurs after the cessation of prolonged vascular occlusion (for example, a blood clot). The mechanism of this phenomenon is similar to that described in the working hyperemia, but it has a more pronounced character due to the high degree of local hypoxia and a higher concentration of vasoactive agents. This type of hyperemia can be observed at home, if a certain time (1-2 minutes) Squeeze your own finger at its base. At first it will turn pale, but after stopping the compression it will be bright red for the next few minutes. A local reaction to damage is manifested by vasodilation in the damaged area and is an element of the inflammatory reaction. It

is realized by histamine, ecosanoids, the kallikrein-kinin system and other mediators secreted by blood cells, endothelium and tissues, or converted from their precursors in the blood plasma. The mechanisms of inflammatory reactions are discussed in detail in the course of pathophysiology.

Local changes in blood flow are possible not only in the direction of its increase, but also in the direction of restriction with a low level of metabolism. In this case, vasodilator substances are practically not released into the extracellular space, the tone of the smooth muscles increases and relative vasoconstriction occurs.

External neurogenic control of smooth muscle tone artheriol is provided by sympathetic post-ganlionary neurons. These neurons innervate arterioles in almost all regions of the body and realize their effect through the stimulation of α1-adrenergic receptors with norepinephrine. The result is increased vasoconstriction and limited blood flow through the region. However, the opposite reaction is also possible - vasodilation when inhibiting the activity of the sympathetic link of the ANS and removing the contribution of sympathetic impulses to the basal tone of the smooth muscles of the arterioles. Such a reaction, for example, is observed in the microvasculature of the skin with increasing body temperature. When cooling the body in the skin, on the contrary, vasoconstriction and restriction of blood flow are observed.

External hormonal control is realized with the participation of hormones such as adrenaline, angiotensin-2, and ntidiuretic hormone (vasopresyn) and atrial natriuretic hormone. The influence of these hormones complements the local and nervous regulation of blood flow in arterioles in the inte-res to ensure an optimal response for the whole organism to the stimulus that caused the secretion of this hormone.

Lesson 5.

Anatomical and physiological aspects of regulation of systemic circulation. Central mechanisms. Central regulation of blood circulation.

Regulation of systemic circulation ensures the adaptation of the minute volume of blood to the metabolic needs of the body, primarily transportation of oxygen and nutrients to the cells. The regulated parameter in the circulatory regulation circuit is the value of the systemic blood pressure (AR), the changes of which are signaled by baroreceptors (BR), located mainly in the main reflexogenic zones – the carotid sinus and aorta. The executive structures on which the minute

volume of blood directly depends are the heart as a pump, the systemic vessels (their lumen and volume) and the amount of circulating blood that ensure the venous return of blood to the heart and, accordingly, cardiac output.

Regulation of systemic blood pressure is carried out along the contour of the negative feedback relationship. The most important mechanisms of regulation are *nervous and humoral*, which develop over time in stages and are divided into the following in duration:

1. Rapid (immediate) regulation is a nervous regulation that is carried out reflexively, mainly with the participation of baroreceptors and chemoreceptors of blood vessels, and leads to a change in blood pressure due to pressor or depressor reflexes. Its duration is 20-30 s. For example, during physical exertion, stress, brain ischemia, blood pressure rises sharply. The first to perceive the deviation of pressure from the normal baroreceptors of the sinocarotid and the aortic zones and react very quickly (seconds) to them. Information about the increase in pressure along the Goering nerve enters the medulla oblongata and stimulates the depressor center and inhibits the pressor. As a result, there is an expansion of peripheral vessels (veins and arterioles), the frequency and strength of heart contractions decreases, which leads to a drop in cardiac output and general peripheral vascular resistance, as a result of which a decrease in blood pressure develops. And, conversely, with a decrease in blood pressure, an opposite reaction occurs, aimed at increasing it. For the ability of the baroreceptor system to withstand both an increase and a drop in blood pressure, it is called the buffer system.

2. Intermediate regulation is a neurohumoral regulation that is carried out with the participation of nerve centers and hormonal factors, lasting minutes. An example of it could be the restoration of blood pressure during bleeding. Blood loss leads to a decrease in circulating blood volume (CBV) and a drop in blood pressure. Baroreceptors of the synocarotide and aortic zones, which have received information about a decrease in blood pressure, send it not only to the pressor center of the medulla oblongata, but also to the hypothalamus, namely to the supraoptic and paraventricular nuclei that produce vasopressin. After 3-8 minutes, the secretion of vasopressin, with a loss of 20% of blood, increases 40-50 times. Vasopressin causes: a) vascular spasm, which leads to an increase in blood pressure; b) significantly increases the reabsorption of water in the nephrons of the kidney; c) increases the transition of water from the intercellular space to the capillaries. As a result, CBV increases, blood pressure rises.

3. Slow (pushed back) regulation is a humoral regulation involving the renin-angiotensin system, which leads to the formation of angiotensin II, which constricts blood vessels and stimulates the secretion of aldosterone over the kidneys by the cortex, due to which the reabsorption of sodium ions in the kidneys

increases, and after them - water, resulting in an increase in CBV, the return of blood pressure to normal values. Its duration is hours, days.

To prevent the accumulation of a large concentration of sodium ions in tissues and the potential development of salt hypertension, atrial natriuretic atriopeptide (ANP) is produced, the main effect of which is to reduce CBV and increase excretion in the urine of Na+ and, accordingly, reduce the processes of depolarization of the smooth muscles of the vascular wall, normalize their tone and blood pressure.

In order to respond to constant changes in the requirements of the organism, its organs and tissues, the circulatory system must continuously change the mode of its functioning, adapt it to the needs that have arisen. The main role in these processes is played bythe nervous and humoral systems.

In most structures of the central nervous system there are clusters of neurons, the stimulation of which causes certain changes in the function of the circulatory system. One of them is represented in the medulla oblongata, which rigidly controls the activityof the cardiovascular system. The role of the medulla oblongata in the regulation of blood circulation in 1871. V. F. Ovsyannikov in the laboratory of K. Ludwig found that successive frontal sections of the medulla oblongata led to a significant decrease in blood pressure (up to 40-50 mmHg). Based on the facts obtained, it was concluded that in the medulla oblongata in the area of the bottom of the IV ventricle there are structures responsible for lowering blood pressure. Further studies have established, that on the dorsal part of the medulla oblongata (the bottom of the IV ventricle and 3-4 mm below) there are areas whose stimulation led to a slight decrease or increase in blood pressure. At that time, these areas were considered as depressor and pressor zones of the vasomotor center, but it was not possible to establish a clear connection between them, both in localization and in the direction of the response. Only in the second half of XX century. the role and significance of the ventral part of the medulla oblongata, as an integral part of the hemodynamic (vasomotor) center, was determined. On the ventral lateral surface of the brainstem, close to its surface, they found clusters of neurons that formed three small areas – zones M, S and L, each with an area of 2 mm ^{2}, which lie above the soft meninges and enter the brain thickness by 200-600 microns. They are highly sensitive to chemical (glutamate, glycine, H2CO3) and electrical irritation and are actively involved in the regulation of blood pressure and respiration (Fig. 19).

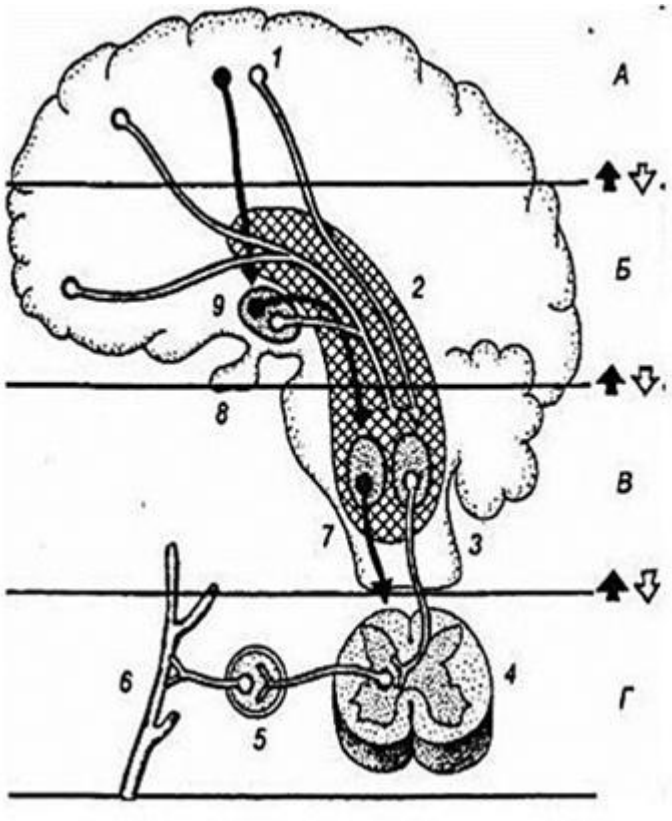


Fig. 19. Localization of the structures of the vasomotor (***vasomotor) center.***

A-cortical representation of the vascular-motor center;

B-centrihypothalamus: anterior hypothalamus (depressor zones), posterior hypothalamus (pressure zones);

B-bulbar centers (vasodilator, vasoconstrictor);

G-centers of the spinal cord (lateral horns).

1 - cerebral cortex, 2 - reticular formation, 3 - vasomotor center, 4 - spinal cord, 5 - sympathetic ganglia, 6 - blood and blood vessels, 7 - medulla oblongata, 8 - pituitary gland, 9- hypothalamus

Regulation of systemic circulation

In the rostral (anterior) ventrolateral zone (RV/13) of the brainstem (zones S and M) there are clusters of sympathetic neurons (having access to the spinal cord and synthesizing and secreting adrenaline), electrical or chemical (glutamate) irritation of which causes narrowing of blood vessels and an increase in blood pressure. After the destruction of these zones or the application of the inhibitory mediator – GABA – to them, the pressure decreases sharply and for a long time.

In the caudal (posterior) ventrolateral zone (CVLZ) (zone L) there are clusters of neurons (with no outlets to the spinal cord), electrical or chemical (glutamate) irritation of which leads to a decrease in blood pressure. their destruction or GABA application causes an increase in blood pressure. Thus, neurons of the rostral and caudal ventral zones of the medulla oblongata are

integral parts of the vasomotor center. The former are sympathetic, their arousal increases blood pressure; the latter, on the contrary, lower blood pressure by inhibiting theinhibition of sympathoactive neurons. Reflex regulation of blood circulation of reflexes that close in the vasomotor center of the medulla oblongata and are accompanied by changes in blood pressure and heart function include reflexes from the reflex zones of the cardiovascular system (synocarotide, aortic, vessels of the pulmonary circulation – pulmonary arteries and hollow veins), called their own. Reflexes that arise from others divided into thebody are called conjugated. Own reflexes arise from the baro- and chemoreceptors of reflexogenic zones due to a sharp increase or decrease in blood pressure in them, or a change in the concentration of chemicals in the blood.

The most studied mechanism for regulating blood pressure are baroreceptor reflexes. Baroreceptors are receptors whose irritation occurs during the stretching of the walls of blood vessels and the heart. The main role in the regulation of blood pressure is played by baroreceptors of the carotid sinus and aortic arch. Receptors of the synocarotid zone are contained in the bifurcation wall of the common carotid artery on the inner and outer branches, the aorta – in the outer layer of the wall of its arc. They are represented by cone-shaped twisted endings of myelin nerve fibers. Afferent fibers from carotid receptors pass through the thin fibers of the Gering's nerve to the tongue-pharyngeal nerve and along the lonely tract to the medulla oblongata. Their maximum impulse activity occurs when blood pressure changes in the range of 50-180 mm Hg.. Lower or higher blood pressures do not affect changes in impulse activity.

The sensory fibers from the aortic arch go along the depressor nerve (X pair of cranial nerves), flow into the bundle of the lonely tract and end in the medulla oblongata. Sensory fibers coming from the synocarotide zone and the aortic arch are called buffer nerves. Sensory information from the baroreceptors of the carotid sinus enters both the pressor and depressor centers. From the aortic arch, through the sensory nucleus of the lonely tract, information enters only the depressor center, and from the chemoreceptors of the same zones – to the pressor center (Fig.20).

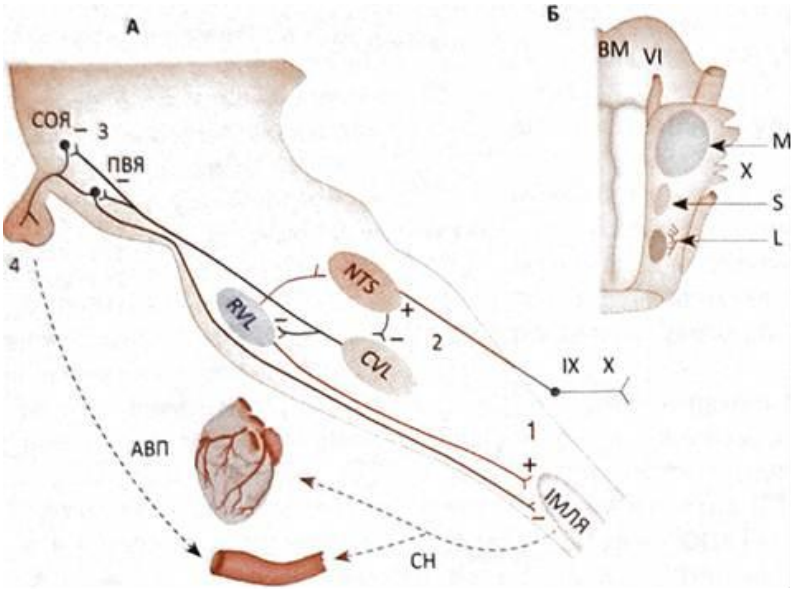


Fig. 20. Structures of the vasomotor cen tru, their localization and connections.

A: 1 - spinal cord; 2 - medulla oblongata; 3 - hypothalamus 4 - pituitary gland. IX, X - afferent fibers from the baroreceptors of the carotid sinus and aorta; CH - sympathetic innervation of blood vessels and the heart; AVP - arginine-vasopressin; CVL - caudal ventrolateral zone; RVL - rostral ventrolateral zone; NTS - the nucleus of the lonely tract (the sensory nucleus of the vagus nerve); PVA - paraventricular nucleus; SOYA - supraoptic nucleus, "+" - excitatory effect,- inhibitory effect, IMLA - intermediolateral nucleus of the spinal cord, B; M. S and L – electro- and chemosensitive zones of the ventral surface of the medulla oblongata; VM - varolii bridge; X - 10th pair of c.n.

Reflexes from carotid sinuses. Reflexes from the aortic arch. Local mechanisms of circulatory regulation

The depressor synocarotide reflex develops with an increase in blood pressure in the synocarotide zone, its reflexand arc is constructed as follows (Fig. 21).

Information on the activation (stretching) of baroreceptors along the Goering nerve (increased amount of AP) through the negative feedback channel enters the caudal (caudal) ventrolateral part of the medulla oblongata and, with the help of a glutamate mediator, excites the sensitive nuclei of the vagus nerve – n. tractus solitarius (NTS). From these, information enters the caudal ventrolateral L-zone, in which it is in contact with the inhibitory interneuron, which transmits impulses (AP) to the rostral ventrolateral region of the medulla oblongata, where, with the help of a GABA mediator, it inhibits the neurons of the sympathetic nervous system, which carry information to the lateral horns of the spinal cord, and from

there to the vessels and the heart. As a result of inhibition of the influence of the sympathetic nervous system, its tonic effect on the vessels and the heart is significantly reduced, as a result of which the vessels dilate, the work of the heart decreases and, as a result, blood pressure drops. In parallel, information on the sensitive fibers of n. tractus solitarius (NTS) is sent to the motor nucleus of the vagus nerve – n. ambiguus (caudal ventrolateral zone), and from it to the heart, which reacts with a negative chronoinotropic effect – a decrease in cardiac release of blood into the vessels, which also leads to a decrease in blood pressure.

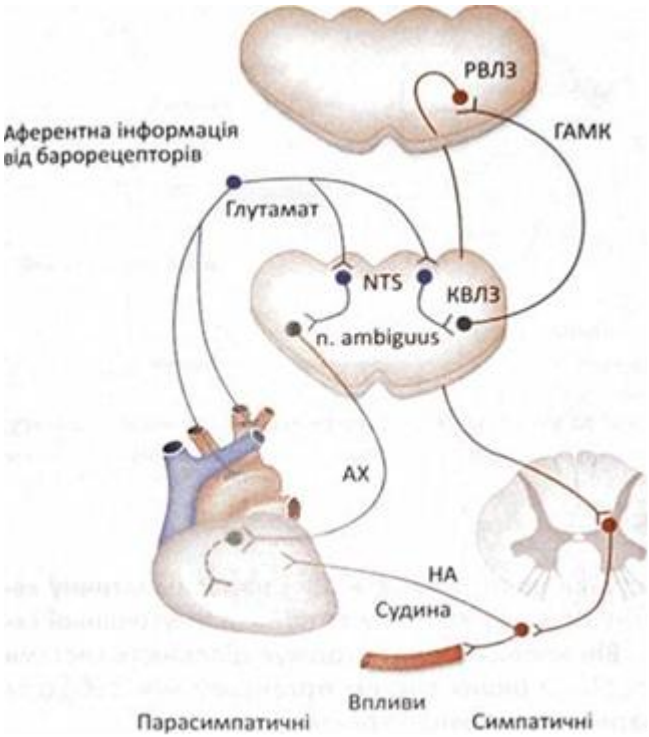


Fig. 21. Reflex arc of the depressor synocarotide reflex.

RVLZ - rostral ventrolateral zone; CVLZ - caudal ventrolateral zone; NTS - the sensory nucleus of the vagus nerve; n.ambiguus – motor nucleus of the vagus nerve; GABA - gamma-aminobutyric acid; AH - acetylcholine; NA – norepinephrine

The pressor synocarotide reflex develops with a decrease in blood pressure in the synocarotide zone (wrinkling of baroreceptors). A decrease in information from receptors in hypotension (the amount of AP in the Goering nerve drops significantly) through the negative communication channel is transmitted to the sensitive nuclei of NTS, and from them to the rostral ventrolateral part (sympathetic-activating tonic zones), which dramatically increases its activity, which leads to an increase in the frequency of impulses in sympathetic fibers, narrowing of blood vessels and an increase in blood pressure. In parallel, information is sent to the heart and enhances its work, which leads to an increase in

the release of blood into the vessels, as a result of which blood pressure also rises (Fig. 22).

Fig. 21. The contour of regulation of systemic blood pressure (Ra) with the participation of the pressor synocarotide reflex, which arose in response to hypotension in the carotid zone. Ra is an *adjustable parameter, an upward arrow is an increase, a down arrow is a decrease. "+" sign – excitation, 0 – intact*

Reflexes from the aortic arch

With an increase in blood pressure in the aortic arch, baroreceptors are excited, information (AP) from which flows through the negative feedback channel – the depressor (parasympathetic) nerve (X pair of c.n.) to the caudal region of the ventrolateral region. By stimulating it, it causes the activation of the inhibitory insertion neion, which blocks the sympathetic-activating neurons of the rostral ventrolateral region. As a result, the frequency of impulses going along the sympathetic nerve fibers to the vessels decreases sharply or stops, they expand, blood pressure drops. At the same time, the motor nuclei of the vagus nerve located in the caudal ventrolateral region are excited. Information from them on the vagus nerves is sent to the heart and causes a decrease in blood output, which also helps to lower blood pressure. Thus, the vasomotor center is under constant influence from the baroreceptors and, thanks to them, controls the levelof ar terial pressure in the body (Fig. 23).

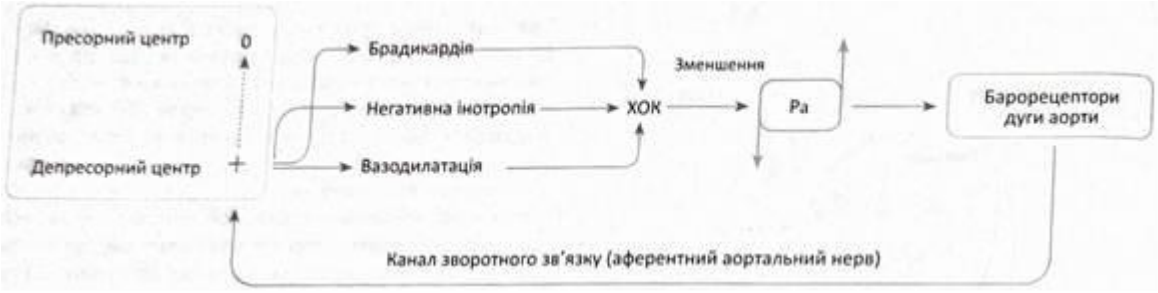


Fig. 23. The contour of regulation of systemic blood pressure (Pa) involving the depressor aortic reflex that arose in response to hypertension in the aortic arch. *Pa - adjustable parameter; up arrow - increase; down arrow - decrease. The "+" sign is excitation. 0 – intact*

Reflexes from chemoreceptors of the synocarotide and aortic reflexogenic zones occur when deviation from the norm of regulated parameters of the organism; an increase in arterial blood of Rso2, hydrogen ions [H+], a decrease in the carotid sinus of Ro2 to 60 mm Hg.. The chemoreceptors of the carotid sinus are also excited under the influence of cyanides, nicotine and other compounds that come to them with blood. Excitation of chemoreceptors leads to stimulation of both the pressor sympathoactive M and S lobes of the rostral ventrolateral zone of the medulla oblongata, as a result of which the vessels narrow, and to the caudal depressor L-zone, which leads to a decrease in heart rate, a decrease in cardiac output. However, the effects associated with vasoconstriction predominate over the decrease in minute volume of blood flow, which leads to an increase in systemic blood pressure and, as a result, an acceleration of the linear speed of blood movement, an acceleration of carbon dioxide excretion and an increase in oxygen tension in arterial blood aimed at maintaining the body's gas homeostasis. Under natural conditions, these reactions are influenced by the respiratory system, as well as the direct effect of the above factors on the vessels.

Lesson 6.

Anatomical and physiological aspects of regional blood circulation. Blood circulation in the coronary vessels.

Both coronary arteries depart from the sinuses behind the two valves of the aortic valve at the root of the aorta. In 50% of people, the blood flow is greater in the right coronary artery, in 20% - in the left, in 30% - the same in both vessels. The right coronary artery provides blood to the right ventricle and back wall of the leftventricle, the left–mainly anterior and lateral walls of the left ventricle (Fig. 24).

Passing outside under the epicardium, the arteries give numerous branches deep into the heart muscle, which penetrate to the endocardium. Under the endocardium are subendocardial arterial plexuses, which partially supply the myocardium with blood during systole. Between the small coronary arteries with a diameter of 20-250 microns there is a large number of collateral anastomoses. Venous blood flows to the chambers of the heart through the venous sinus, tebesium veins, arteriosinusoidal and arterioluminal vessels.

The volume of blood flowing through the coronary vessels is about 5% of cardiac output (250 ml / min) or 50-80 ml of blood / 100 g of heart tissue in 1 minute.

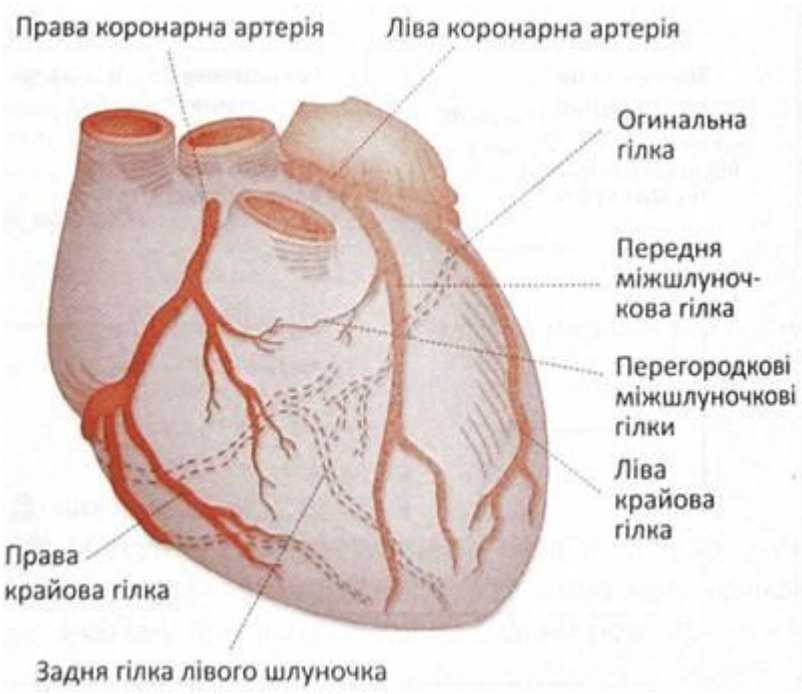


Fig. 24. Coronary vessels of the human heart.

The specificity of myocardial metabolism, the high intensity of oxidative processes in it, the wide amplitude of changes in its functions put forward special requirements for coronary circulation. The use of 02 by the heart already under normal conditions is higher than in other organs. So, if the heart absorbs 10.1 ml / 100 g • min, then the brain – 3.3; kidneys – 4.2; liver – 4.4 ml/100 g•min. At the same time, the blood supply to the myocardium is much less than the above-mentioned tissues. So, if the myocardial blood flow is 50 ml / 100 g•min, then the brain is 65; kidneys – 420; liver – 150 ml/100 g•min. Under these conditions, the oxygen demand of the heart is provided by higher extraction of O2 from the blood – 140 ml / l of blood (in comparison - the brain is only 25 ml / l). It is known that the work of the heart can increase by 5-8 times and this requires high oxygen supply, which is achieved by enhancing coronary blood flow and its regulatory mechanisms.

The main energy substrate for the heart are free fatty acids, glucose and lactic acid. The heart can include in its energy metabolism underoxidized metabolic products from other organs, including skeletal muscles. Therefore, the main danger to the heart in violation of its blood supply is not the lack of energy substrates, but oxygen deficiency.

Coronary blood flow during the cardiac cycle

Due to the specifics of the heart (systole – diastole), the coronary blood flow undergoes significant fluctuations. During systole, the coronary vessels are compressed and the blood flow sharply decreases or stops. In diastole, during the isometric relaxation of the myocardium, when the aortic pressure is still high, it reaches its maximum level. According to the literature and our data, the blood flow in systole is 55-85% less than in diastole (Fig. 10.53). Blood pressure in the coronary arteries at rest is about 25-30 mmHg.

Despite a sharp decrease in coronary blood flow in systole, heart metabolism is maintained at a high level. This is achieved by: 1) a high rate of blood flow and a slight stretching of blood vessels, which at this moment provides an increased volume of blood; 2) thick capillary grid – each cell has its own capillary; 3) a very short distance from the capillary to the cardiomyocyte; 4) phase oscillations of venous outflow – fast in diastole and slow in systole; 5) high extraction 02 from the blood; 6) the function of the vessels of Tebezia – Viennese.

Regulation of coronary blood flow.

Myogenic regulation.

Coronary vessels are characterized by high basal myogenic tone, which ensures a certain independence of the coronary circulation with a change in systemic blood pressure in the range of 70-160 mm Hg. So, with an increase in pressure in the coronary vessels, there is a contraction of smooth muscles, with a decrease – the smooth muscles of the vessel wall relax.

Nervous regulation.

At the beginning of studies of coronary blood flow, it was quite difficult to prove its nervous regulation. First, because for a long time they could not detect the presence of nerve endings on the coronary vessels. Secondly, because under the influence of nervous stimulation, the work of the heart changes, which can affect the functions of blood vessels. However, the presence of adrenergic and cholinergic endings was established, the stabilization of the heart made it possible to confirm the direct effect of the sympathetic and parasympathetic nervous systems on the coronary vessels.

Stimulation of the sympathetic nervous system (irritation of the stellate cervical ganglion) and the parasympathetic nervous system (irritation of the vagus nerve) causes the expansion of the coronary vessels, an increase in coronary blood flow.

Reflex effects from the coronary vessels. Coronary vessels are extremely richly equipped with nerves in the form of clusters, tree branches of diffusion endings, highly sensitive to changes in blood pressure. Thus, with an increase in pressure in the left artery, especially in the area of its bifurcation, a systemic depressor reaction and bradycardia develop, blocked by weightsby otomy. With a decrease in coronary pressure, a pressure reaction and tachycardia are observed, which is associated with an increase in the activity of the sympathetic nervous system. These data allow us to conclude that 8 coronary vessels have mechanoreceptors that react to changes in blood pressure.

Chemoreceptors are also found in the coronary vessels, which react to the introduction of chemicals, such as veratrin, which leads to bradycardia and systemic hypotension. With the restriction of coronary blood flow or pinching of the coronary vessel, ECG changes and a significant pain reaction develop, which is associated with a sharp excitation of the sympathetic nervous system and its afferent fibers. Crossing them significantly weakens or eliminates pain.

Humoral regulation.

The mediator of the parasympathetic nervous system – acetylcholine leads to a distinct dilatation of the coronary vessels, an increase in coronary blood flow. Its effect is inhibited by the M-cholinergic receptor blocker – atropine. The mediator of the sympathetic nervous system – norepinephrine – also causes the expansion of the coronary vessels, an increase in coronary circulation, and this effect is associated with β receptors of the coronary vessels. Blockade of their β-blocker – inderal – prevents dilator effect and increased blood flow. It is known that in the coronary vessels there are α- and β-adrenergic receptors, that is, two points of application of catecholamines. At the beginning of the coronary vessels, α-adrenergic receptors are placed, their number is small. β-adrenergic receptors are located in the distal section of small diameter vessels in large quantities, so the resulting reaction is dilatation. The constriction of coronary vessels under the influence of catecholamines is observed against the background of blockade of β-adrenergic receptors, when they only interact with a-adrenergic receptors. The blockade of their α-adrenergic blocker – regitin prevents a constrictor effect. A similar effect – narrowing of the coronary vessels under the influence of the sympatho-adrenal system – is observed quite often in old animals, in which a decrease in the number of β-adrenergic receptors in the vessels of the heart is detected.

The hormones insulin, thyroxine cause the expansion of the coronary vessels; narrowing – adrenaline, vasopressin, angiotensin and others.

Metabolic regulation.

Heavy physical work is accompanied by a sharp increase in cardiac output (5-8 times) and coronary blood flow (4.5 times), which leads to a sharp increase in metabolism in the heart – oxygen absorption increases three times (from 30 to 90 ml / min). O2 deficiency causes ATP to "degrade" on ADP, then on AMP and adenosine (blocks Ca2+- channels), which penetrates the membranes of cardiomyocytes into the intercellular space and increases local coronary blood flow. The same effect, in addition to adenosine, is caused by CO2, H+ and K+ ions, nitric oxide, bradykinin and prostaglandins, histamine and lactic acid.

The main factors affecting the value of coronary blood flow are physical – the amount of blood pressure in the aorta and the frequency of ventricular contractions, metabolic – vasodilation occurs due to the metabolite – adenosine. Nervous regulation of coronary vascular tone is less important.

Pulmonary circulation.

The small, or pulmonary, circle of blood circulation begins in the right ventricle of the heart, from where the pulmonary trunk comes from, which is divided into the right and left pulmonary arteries, and the latter branch out in the lungs into arteries transitioning into capillaries. In capillary nets that braid the alveoli, blood gives off carbon dioxide and is enriched with oxygen. Oxygen-enriched arterial blood comes from the capillaries into the veins, which, having merged into four pulmonary veins (two on each side), flow into the left atrium, where the small (pulmonary) circle of blood circulation ends.

The most important feature of the organization of blood supply to the lungs is its two-component nature, since the lungs receive blood from the vessels of the pulmonary circulation and bronchial vessels of the great circle of blood circulation. The functional significance of the vascular system of the pulmonary circulation is to ensure the gas exchange function of the lungs, while the bronchial vessels satisfy their own circulatory-metabolic needs of the lung tissue.

The pulmonary artery and its branches with a diameter of more than 1 mm are vessels of an elastic type, which contributes to a significant smoothing of the pulsation of blood entering the lungs during the systole of the right ventricle. Smaller arteries (with a diameter of 1 mm to 100 microns) belong to the arteries of the muscular type. They determine the amount of hydrodynamic resistance in the pulmonary circulation. In the smallest arteries (with a diameter of less than 100 microns) and in arterioles, the content of smooth muscle cells (SMC) progressively decreases and in arterioles with a diameter of less than 45 microns they are completely absent. Since muscleless arterioles are closely related to the alveolar parenchyma, the intensity of the blood supply to the lungs directly depends on the intensity of ventilation of the alveoli.

The capillaries of the lungs form a very dense network on the surface of the alveoli and at the same time there are several capillaries on one alveoli. Due to the fact that the walls of the alveoli and capillaries are in close contact, forming, as it were, a single alveolar-capillary membrane, the most favorable conditions are created for effective ventilation-perfusion relationships. Under conditions of functional rest in humans, capillary blood is in contact with alveolar air for about 0.75 s. During physical work, the duration of contact is shortened more than twice and averages 0.35 s. As a result of capillary fusion, muscleless postcapillary venules characteristic of the pulmonary vascular system are formed, transforming into muscle-type venules and further into pulmonary veins. A feature of the vessels of the venous region are their thin walls and a weak severity of SMC. The structural features of the pulmonary vessels, in particular the arteries, determine the greater extensibility of the vascular bed, which creates conditions for lower resistance (about 10 times less than in the system of the large circle of blood circulation), and therefore lower blood pressure.

In this regard, the system of the pulmonary circulation belongs to the area of low pressure. The pressure in the pulmonary artery averages 15-25 mm Hg., and in the veins - 6-8 mm Hg. The pressure gradient is approximately 9-17 mm Hg., that is, much less than in the large circle of blood circulation. Despite this, an increase in systemic blood pressure or a significant increase in blood flow (with active physical work of a person) does not significantly affect the transmural pressure in the pulmonary vessels due to their greater elasticity. The great elasticity of the pulmonary vessels determines another important functional feature of this region, which is the ability to deposit blood and thereby protect the lung tissue from edema with an increase in the minute volume of blood flow.

The minute volume of blood in the lungs corresponds to the minute volume of blood in the large circle of blood circulation and in conditions of functional rest averages 5 l / min. With active physical work, this figure can increase to 25 l / min.

The distribution of blood flow in the lungs is characterized by uneven blood supply to its upper and lower parts, because the blood in their vessels is under double pressure – hemodynamic and gravitational. In an upright position, the upper parts of the human lungs are above the level of the heart, and the basal parts are at the same level or lower, which contributes to the equalization of blood pressure in the upper parts of the lungs with atmospheric pressure in the alveoli. This leads to a weakening of the perfusion of the tops of the lungs, inadequate gas exchange. In the lower regions of the lungs, the alveolar pressure is lower than in the vessels, as a result of which their blood supply increases significantly. High ventilation and relatively high alveolar Ro2 and low blood supply to the tops of the lungs contribute to the development of tuberculosis in these areas.

The intensity of the blood supply to the lungs depends on cyclical changes in pleural and alveolar pressure in different phases of the respiratory cycle. During inhalation, when the pleural and alveolar pressure decreases, there is a passive expansion of large extrapulmonary and intrapulmonary vessels, the resistance of the vascular bed is further reduced and the blood supply to the lungs during the inhalation phase increases.

Regulation of pulmonary circulation.

Local regulation of pulmonary circulation is mainly represented by metabolic factors, the leading role among which belongs to pO2 and pCO2. With a decrease in pO2 and / or an increase in pCO2, local vasoconstriction of pulmonary vessels occurs. So, a feature of the local regulation of the blood supply to the lungs is the correspondence of the intensity of local blood flow to the level of ventilation of this part of the lung tissue.

Nervous regulation of pulmonary circulation is carried out mainly by sympathetic vasoconstrictor fibers. The pulmonary circulation system is distinguished by a functional connection with the central regulation of systemic hemodynamics in the large circulation. All reflexes of self-regulation of blood circulation from the baro-and chemoreceptors of the carotid (carotid) sinus are accompanied by active changes in pulmonary blood flow. In turn, the vessels of the pulmonary circulation are a powerful reflexogenic zone that causes reflex changes in the cardiovascular system.

The humoral regulation of pulmonary circulation is largely due to the influence of biologically active substances such as angiotensin, serotonin, histamine, prostaglandins, which cause mainly a vasoconstrictor effect in the lungs and increase blood pressure in the pulmonary arteries. The activity of other humoral factors (adrenaline, norepinephrine, acetylcholine) in the system of regulation of pulmonary blood flow is less pronounced.

Cerebral circulation.

The blood supply to the brain occurs through two internal carotid and two vertebral arteries; together under the hypothalamus form the circle of Willisius, which gives rise to the lateral branches that carry blood to all parts of the brain. The internal carotid arteries are the most important in the blood supply of the brain in humans. Short-term pinching of them leads to loss of consciousness. Venous outflow is carried out into the internal jugular vein through the deep veins and sinus cavities of the dura mater. The capillaries of the brain are much less permeable to proteins, peptides, ions, that is, they perform the function of the blood-brain barrier. This property of capillaries allows you to maintain the

composition of extracellular fluid (ECF) relatively constant, which is important for neuronal function.

Normal cerebral blood flow with a brain mass of 1500 g is 750 ml / min, or 13% of the value of minute volume of blood flow. In gray matter, cerebral blood flow is 69 ml/100 g/min, in white matter – 28 ml/100 g/min. The greatest blood supply is observed in the frontal and premotor areas. The right half of the brain supplies more blood than the left. During mental work, the blood circulation of the brain can increase by 100 – 150 ml / min, during rest – decreases. A sharp increase in blood circulation (by 50% or more) is observed with mental arousal and convulsions.

The amount of cerebral circulation is affected by cerebrospinal fluid (CSF), which in a volume of 150 ml fills the ventricular cavity and subarachnoid space. Its composition is almost the same as that of extracellular fluid (ECF). About 500 ml of CSF is formed daily, of which 350 ml is adsorbed by arachnoid villi into the veins of the venous sinuses. With an increase in intracranial pressure of more than 33 mm Hg.. (norm 0 mmHg) Cerebral blood flow is significantly weakened. Any changes in venous pressure affect the pressure of the CSF.

Due to the fact that brain tissue and cerebrospinal fluid cannot be compressed, blood volume and CSF must be unchanged.

These interactions help compensate for changes in blood pressure at head level. An example would be the impact of acceleration. If the body moves upwards, then the blood goes to the legs, and blood pressure at the head level decreases. In parallel, venous and intracranial pressure decreases, and cerebral blood flow remains within the normal range. Conversely, when the body moves downwards, blood pressure in the head rises. Along with it, intracranial pressure increases, which prevents damage to blood vessels. Despite significant changes in cerebral circulation from the nervous and physical activity of the body, the total blood flow remains constant. The factors involved in the regulation of blood circulation in the brain are presented in Fig. 25.

Innervation of cerebral vessels is carried out by postganglionic sympathetic neurons (mediators – norepinephrine, neuropeptide Y) and postganglionic cholinergic neurons (mediators – acetylcholine, VIP, other peptides). Afferent nerves that transmit information to the vessels are part of the trigeminal nerve. Stretching the cerebral vessels or touching them is accompanied by pain.

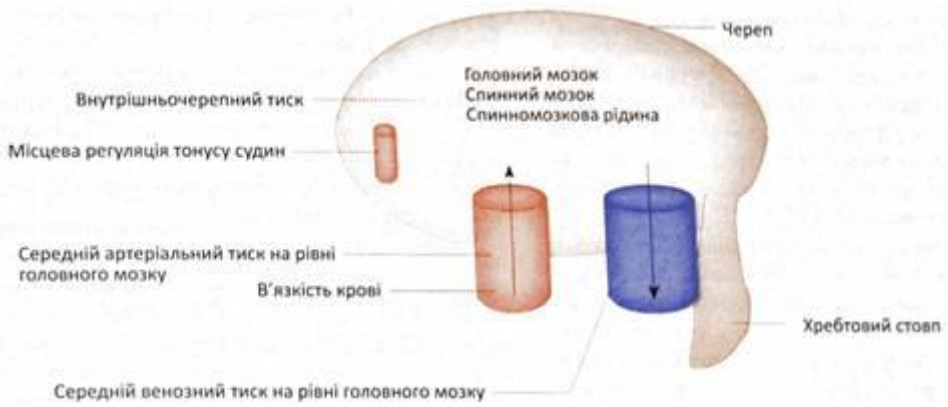


Fig. 25. Factors affecting cerebral circulation.

Regulation of cerebral circulation

Myogenic self-regulation. It is carried out by its own smooth muscles of arterial vessels and is effective when blood pressure fluctuates in the range of 80-180 mm Hg.. With an increase in blood pressure in the willisium and pial vessels – they narrow, with a decrease – expand. These effects do not disappear after cutting the sympathetic and parasympathetic nerves that go to the cerebral vessels and are not blocked by adrenergic and anticholinergic blockers.

Neurogenic effects. They can be traced in vessels with a diameter of 25-30 microns. their effect is manifested in the case of changes in the blood supply to the brain, the initial tone of blood vessels and the tension of O2 and CO2 in the blood.

Humoral regulation is provided by a direct effect on the smooth muscles of the cerebral vessels of vasoactive substances. Vasoconstrictors include catecholamines, angiotensin II, serotonin, prostaglandin F, bradykinin (only in humans); vasodilators – acetylcholine, histamine, nitric oxide, adenosine, GABA, acting through specific receptors.

Metabolic regulation. Related to brain function and metabolism. An essential metabolic regulator is an increase in the CO2 voltage in arterial blood and an increase in the concentration of H+ ions in the perivascular environment, which leads to a drop in pH (acidosis) in the cerebrospinal fluid. For every millimeter of a graduated change in CO2 voltage, the value of cerebral blood flow varies by 6 %. An increase in CO2 in the blood (hypercapnia) causes the expansion of cerebral vessels, a decrease (hypocapnia) – their narrowing.

A local increase in the functional activity of neurons causes an increase in the intercellular environment of adenosine and especially K+ ions, which lead to a significant expansion of the cerebral vessels and an increase in its blood flow. Given that the level of K+ ions changes in a fraction of a second from the moment the neuron's function increases, the described mechanism is an urgent contour of

the regulation of cerebral circulation. The slow circuit is associated with the accumulation of H+ ions and acidosis.

Large circle of blood circulation.

The large, or bodily, circle of blood circulation (Fig. 26) serves to deliver nutrients and oxygen to all organs and tissues of the body. It begins in the left ventricle of the heart, where arterial blood flows from the left atrium. The aorta emanates from the left ventricle, from which the arteries depart, going to all organs and tissues of the body and branching out in their thickness up to the arterioles and capillaries — the latter pass into the venules and further into the veins. Through the walls of the capillaries there is a metabolism and gas exchange between blood and tissues of the body. Arterial blood flowing in the capillaries gives up nutrients and oxygen and receives metabolic products and carbon dioxide. The veins merge into two large trunks — the upper and lower vena cava, which flow into the right atrium, where the large circle of blood circulation ends.

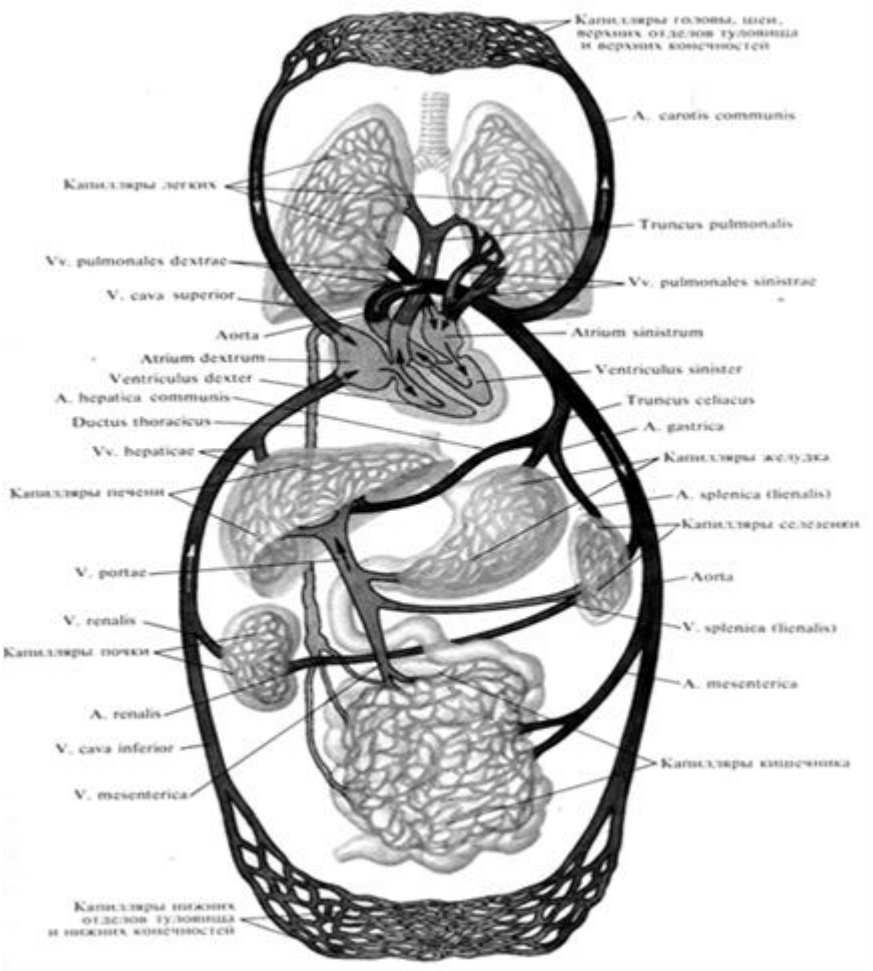


Fig.25. Scheme of the large and small circle of blood circulation.

Physiology of the lymphatic system. Age features of blood circulation

In humans, there is another group of vessels that forms the lymphatic system, which transports the lymph — a clear yellowish liquid. Lymphatic system – is a part of the immune system, which is morphologically and functionally combined with the circulatory system and is represented by a set of lymphatic capillaries, blood vessels, trunks, ducts and lymph nodes that provide drainage of organs, removal of decay products, microbial bodies and other particles, as well as lymphopoietic and protective functions.

Lymph is an analogue of blood plasma, but differs in protein content and contains cellular elements (lymphocytes, monocytes, plasma cells), saturated with enzymes (diastase, lipase) and bactericidal substances, it is a physiological transudate and a product of organ activity. Its formation occurs in the intercellular spaces by exiting part of the liquid composition of the blood, through the walls of capillaries within this tissue. During the day, about 2 liters of lymph are formed.

There are primary organs of the lymphatic system (bone marrow, thymus) and secondary (spleen, pharyngeal lymphoepithelial ring, lymph nodes, lymphoid clusters).

Functions of the lymphatic system:

-drainage – removes excess water from the tissues with crystalloids dissolved in it;

-transport (conductor) – carries out the absorption and transportation of colloidal substances, proteins, drops of fat;

-protective – lymphocytes and antibodies are formed in the organs of the lymphatic system, foreign substances are retained in the lymph nodes;

-hematopoietic – produces blood cells (lymphocytes);

-support homeostasis of the body; metastasis.

The structure of the organization of the lymphatic system: lymphatic capillaries – lymphocapillary reticulum – lymphatic vessels – lymph nodes – lymphatic plexuses – lymphatic trunks – lymphatic ducts.

Lymphatic vessels have valves that prevent retrograde lymph flow and have a rosary. Vessels carrying lymph from the skin, subcutaneous tissue, lie outside the superficial fascia and are called superficial (epifascial). In the area of the joints, they usuallyfind on the bending surfaces.

Deep (subfascial) lymphatic vessels collect lymph from muscles, joints and other organs, accompany deep blood vessels and are part of the neurovascular

bundles. The lymphatic vessels extending from the lymph nodes rush to the following lymph nodes of this or that group, or to the duct that collects lymph from this part of the body. On the way of lymph flow from the organs lies from 1 to 10 lymph nodes. The greatest number of them is located along the lymphatic vessels carrying lymph from the small and large intestines, kidneys, stomach, lungs.

The lymphatic system serves to return fluid from the intercellular space to the circulatory system and therefore the lymphatic arteries do not exist.

The main trunks are: jugular, subclavian, bronchomediastinal, lumbar, intestinal.

The lymphatic vessels of the lower limb accompany the arteries and veins, are interrupted in the popliteal and inguinal lymph nodes, from where the lymph flows into the right and left lumbar trunks. The fusion of the right and left lumbar trunks is a lymph collector and is called the "milk tank", occurs at the level of the XII thoracic-II lumbar vertebrae. After this fusion, the thoracic duct is formed, which collects the lymph from the left half of the head, neck, chest cavity, left upper limb, abdominal cavity, pelvic cavity, lower extremities; accepts the left jugular, bronchomediastinal and subclavian trunks; flows into the left venous corner. The right lymphatic duct exists in 25 % and collects lymph from the right half of the head, neck, chest cavity, right upper limb; is formed from the fusion of the right jugular, broncho-mediastinal and subclavian trunks; flows into the right venous corner.

The lymph node is predominantly oval in shape. Lymph nodes lie in groups in the path of lymphatic vessels that go from organs and tissues to the lymphatic trunks and lymphatic ducts. The node has a gate through which arteries and nerves enter, and veins and external lymphatic vessels exit. From the outside, the node is covered with a connective tissue capsule, from which capsular trabeculae depart into the parenchyma. In the area of the gate, the capsule thickens, forming a portal thickening, from which the portal trabeculae depart into the parenchyma, between which there is a reticular stroma (lymphoid tissue lies in its loops). The parenchyma is divided into cortical and medulla. In the cortical are lymphoid nodules surrounded by lymphoid tissue. On the border with the medulla is the thymus-dependent zone, which contains T-lymphocytes. The brain substance forms the B-dependent zone.

Differences between lymph and blood plasma:

1. Lymph, which was collected on an empty stomach or after eating low-fat foods, is transparent and contains less protein than blood plasma (4 times).

2. Emulsified fats are absorbed from the intestines into the lymph, which causes its milky color, 6-8 hours after ingestion of fatty foods.

3. It has less viscosity and low relative density.

The composition of lymph includes: proteins, mineral salts, forming elements (leukocytes), hemoglobin, glucose. Among leukocytes, lymphocytes are predominantly found (up to 90%), monocytes account for 5%, eosinophils 2%. Normally, red blood cells are absent, but in some pathological conditions (radiation exposure, injuries, etc.) due to an increase in the permeability of the vascular wall or a violation of its integrity, red bodies can enter the lymph from the blood. Depending on the functions and metabolic processes, the composition of lymph in different organs may differ. Thus, in the liver tissue, lymph is characterized by an increased amount of protein, and lymph flowing from the endocrine glands contains hormones.

The process of lymph formation is characterized by the transition of water and substances dissolved in it from the bloodstream to the tissue, and then to the lymphatic vessels. Capillaries have a semi-permeable vascular wall with ultramicroscopic pores through which filtration is carried out. In different organs, the pores have a different size. The greatest permeability is observed in the liver, so about half of the lymph volume is formed here. Water, dissolved salts, glucose, oxygen easily pass into tissue fluid. This is due to increased intravascular pressure(hydrostatic). High molecular weight substances (plasma proteins) are not able to penetrate the capillary wall, they maintain oncotic pressure and retain water in the channel. The difference between hydrostatic and oncotic pressure gives filtration pressure, which ensures the transition of water into the tissue fluid. Part of it goes back into the bloodstream, and part becomes lymph.

The movement of lymph is carried out through the following processes:

1. Rhythmic contractions of the lymphatic vessels (about 10 per minute). Due to the presence of valves, current is possible only in one direction.

2. Sympathetic inervation of the walls of the lymphatic vessels, by spasming and relaxing their certain areas.

3.Facilitates the movement of intrathoracic pressure, which during inhalation becomes negative, the volume of the chest increases, which contributes to the expansion of the thoracic duct.

4. Walking, flexion and extensor movements of the limbs. During the day, theflow returns to 3 liters of lymph in the bloodstream.

Age features of blood circulation

The heart and large vessels begin to form at 2 weeks of embryo life. From the end of the 2nd month of the prenatal period, placental blood circulation is established. Blood saturated with oxygen and nutrients comes from the mother to the fetus through the umbilical vein, which departs from the placenta and is contained in the umbilical cord along with two umbilical arteries. After passing through the umbilical ring of the fetus, the umbilical vein is divided. Several of its branches go to the liver, and the main part of it continues in the form of a venous (arancial) duct flowing into the inferior vena cava, into which venous blood flows from the lower half of the body.

The blood of the vena cava, relatively rich in oxygen, enters the right atrium, then from it through the oval opening into the left atrium, left ventricle and aorta, and from it to all organs and tissues. A small amount of venous blood from the pulmonary veins from the lungs that do not function enters the left atrium, which does not significantly affect the gas composition of the blood of the left atrium. In addition to the inferior vena cava, the upper vena cava flows into the right atrium, which carries venous blood from the upper half of the body. From the right atrium, blood enters the right ventricle and then into the pulmonary artery. Only a small part of this blood enters the pulmonary circulation and returns through the pulmonary veins to left atrium. Most of the blood through the open arterial duct from the pulmonary artery is discharged into the final part of the aortic arch, being almost completely in the descending aorta. Venous blood collects in the umbilical arteries, which rise along the back surface of the anterior abdominal wall, passes through the umbilical ring and through the umbilical cord reaches the placenta, where gas exchange takes place.

So the features of the blood circulation of the fetus are:

a) placental type of nutrition, in which blood oxygen saturation occurs in the placenta, and the flow of mixed (venous and arte) blood to the tissues of the fetus;

b) the presence of connections between the small and large circles of blood circulation (oval opening, open arterial duct);

c) a high level of pressure in the pulmonary circulation, compared with a high level due to increased resistance to blood flow in the vessels of the lungs that do not function;

d) the intensity of the germinal blood tract (arancial duct, umbilical vein, umbilical arteries, etc.).

From the moment of birth after the first breath of the child, the small circle of blood circulation begins to function, there is a desolation of the umbilical vein, which laterturns into a round ligament of the liver, umbilical arteries, from which

the external vesicular-umbilical ligaments are formed. In the first hours after birth, the pressure in the left atrium increases significantly, which ensures the cessation of blood exchange between the atria due to the latch of the oval opening, which is pressed against the edge of the oval fossa. At the 2nd week after birth, the adhesion of the flap with the edge of the oval hole begins. Anatomically, the oval openingbecomes at the end of 5-7 months of life. From the first minutes of life, the venous duct starts, and its complete obliteration ends at the end of the 8th week of life. With the first cry comes a spasm of the arterial duct, but it continues to function for another 8 hours. Anatomically obliteration of the arterial duct ends at 6-8 weeks of the postnatal period.

In the embryonic period, the main structures of the heart are formed early, so at the 3rd week it is already functioning under the influence of its own rhythm of the sinus node. Inervation of the sinus node is observed even in the prenatal period and is carried out initially by adrenergic structures; Cholinergic inervation develops significantly differently - a few months after birth. Fetal heart rate depends on the automatism of the rhythm driver. For newborns, the intramural type of location of the conductive system is characteristic - in the thickness of the muscular part of the interventricular septum. The full development of the histological structures of the conductive system, which is represented by atypical muscle fibers capable of periodic self-excitation, ends by 13-15 years.

Heart rate in a fetus is 140 - in 1 minute, in a newborn - 140-160 in 1 minute, in 1 year - about 120 in 1 minute, in 2-4 years - 105for 1 minute, in 5-7 years - 90-100 in 1 minute, in 14 years - 75 in 1 minute.

In the fetus, through the oval atrial opening and arterial duct, both ventricles of the heart are connected into a functional organ that expels blood into the general circulatory system. Therefore, in the fetus and infant, the walls of both ventricles are almost the same thickness. The right ventricle of the heart has a slightly larger mass (38.6% of the total mass of the heart, and the left 29.9%), since it accounts for a greater load due to overcoming the resistance exerted by the narrow arterial duct. After birth, due to the inclusion of the pulmonary circulation and the distribution of both circles, the resistance in the large circle rises sharply and the wall of the left stomach gradually hypertrophies. Over the entire period of childhood, the mass of the left ventricle increases by 2.5 times, and the right only 0.5 times. It is important to note that the ratio of heart mass to body weight is growing unevenly. At the age of 5-6 months, there is a minimum heart mass relative to body weight (0.38%), at the same time ina newborn 0.8%, and in an adult - 0.4%. In boys, the minimum heart weight is observed at 8 years old (0.44%), and in girls - at 12 years old (0.48%). It is during these periods that one

should be careful about physical and psycho-emotional stress, since various complications from the heart are possible.

Myocardium of newborns has signs of embryonic structure and is an undifferentiated symplast with rich vascularization. It is loose, the muscle fibers are thin, the myofibrils are poorly separated and contain a large number of oval nuclei, there is no twitchiness. The connective tissue of the myocardium is weakly expressed, there are few elastic elements. They appear at the age of 7. The nervous regulation of the heart is not the same, which causes quite frequent dysfunctions in the form of embryocardia, extrasystomy, respiratory artemia. The development of the myocardium usually ends before the beginning of puberty.

In newborns, the minute and shock volume of the heart is much smaller than adults (SV in newborns is 340-490 ml, in 1 year - 1250 ml, in 6-9 years old - 2500 ml, in 14-15 years old - 4300 ml, in adults - 3100-5200 ml). SV in newborns - 2.5 ml, in 1 year - 10.5 ml, in 6-9 years - 23-25 ml, in 14-15 years - 59 ml, in adults - 60-90 ml).

It is important that the relative shock volume of the heart (per 1 kg of body weight) in children is not less, and the relative minute volume of the heart is even greater than in adults. The physiological essence of this is that due to the high relative SV, the need of body tissues for oxygen and nutrients is ensured.

With age, the minute volume of the heart increases more slowly than the SV asa result of the decrease with age of heart rate.

Due to the greater SV in newborns and young children, a decrease in the duration of the cardiac cycle is noted. In newborns, the duration of the heart cycle is 0.4-0.5 s. With age, it lengthens and is 0.7 s. at the age of 10, and in adults - 0.77-0.8s. The duration of the cardiac cycle increases due to ventricular diastole, which in adults (0.48s.), is about 2 times longer than in infants (0.23s).

Blood circulation in newborns is twice as fast as in adults. The full circulation of blood in newborns is 12s., in 3-year-old children - 15s., in adults - 22s. This provides better blood supply to the organs of the child's body.

The value of blood pressure in children is lower than in adults. This is due to the lower contractility of the heart, the relatively larger lumen of the arteries and the insufficient development of elastic fibers of the arteries.

Nervous regulation of the heart is carried out through the superficial and deep plexuses formed by the fibers of the vagus nerve and cervical sympathetic nodes, which are in contact with the ganglia of the sinus and atrial-ventricular nodes in the wall of the right atrium. The fibers of the right vagus nerve innervate mainly the right atrium and especially generously the sinoatrial node. The left

vagus nerve reaches the atrioventricular node and the working myocardium. Sympathetic postganglionic fibers innervate both the conductive system and the working myocardium of all departments. The development and myelination of the vagus nerve ends in 3-4 years. Until this age, cardiac activity is regulated mainly by the sympathetic nervous system, with which, in part, physiological tachycardia in newborns and children of thefirst years of life is associated. Under the influence of the vagus nerve, not only decreases heart rate, but sinus arrhythmia (such as respiratory) and individual "vagus impulses" may appear - a sharp lengthening of the intervals between heartbeats. In addition, the receptor effect is carried out by interoreceptors of both the heart (baroreceptors, chemoreceptors, cardio-cardiac reflexes) and other internal organs, which changes heart rate under the influence of physiological factors.

There are central mechanisms for regulating the work of the heart: the main centers that regulate the work of the heart and blood vessels are contained mainly in the medulla oblongata.

No less difficult is the humoral regulation of the heart. In the heart, especially in its atria, biologically active compounds (digitalis-like factors, catecholamines, arachidonic acid products) and hormones are formed, in particular, atrial natriuretic and renin-angiotensin compounds. Both hormones are involved in the regulationof myocardial oral function, minute volume of blood flow. The vast majority of regulatory effects on the functional state of the heart are associated with membrane mechanisms of the conductive system and cardiomyocytes. Membranes are primarily responsible for the penetration of ions. With a decrease in the content of Ca ++ in the blood, excitability decreases and the contractility of the heart decreases. With a decrease in the level of K + increases heart rate. A double increase in the content of K+ in the blood can lead to cardiac arrest. This effect is used in clinical practice for cardiac arrest during its surgeries. A decrease in the content of Na+ in the blood can lead to cardiac arrest. The basis of this effect is the violation of the grading ntu of the transmembrane transport Na+ and Ca+ and the combination of excitability with contractility. A slight increase in the level of Na+, thanks to Na+ - Ca+ - the exchanger will lead to an increase in myocardial contractility.

A number of real hormones (adrenaline, norepinephrine, insulin, etc.) and tissue (angiotels II, histamine, serotonin, and others) also stimulate heart function, acting on the corresponding receptors, activating calcium channels.

During physical activity, impulses from muscle proprioceptors, vascular chemoreceptors contribute to the excitation of vasoconstrictor neurons. When muscle work in the regulation of blood flow involves the adrenal system of the adrenal glands, as well as vasopressin, thyroxine, renin, atrial natriuretic hormone.

Serious age-related changes are occupied by the circulatory system. Senile heart is characterized by sclerotic changes in the myocardium, focal atrophy and dystrophy of muscle fibers, division (expansion) of cavities. Myocardial contractility decreases, blood shock volume, minute volume of blood flow and cardiac output decrease. The speed of blood flow slows down. A decrease in cardiac output is not accompanied by a corresponding decrease in the work performed by the left ventricle. The ability of the heart to adapt to physical exertion decreases. Prerequisites are created for the rapid development of heart failure under stressful conditions.

The functions of the conductive system of the heart change. The automatism of the sinoatrial node decreases, additional foci of excitation arise, the transmission of impulses in individual links of the conductive system slows down, a tendency to arrhythmias appears: sinus bradycardia, extrasystoles, atrial fibrillation.

Reducing the function of automatism of the sinus node and, as a result, bradycardia is protective in nature, protecting the heart from overstrain. In people of senile age, tachycardia on the background of physical activity quickly leads to a discrepancy between the blood supply to the heart through the coronary vessels and a sharp increase in metabolic processes in the heart, which creates conditions for ischemia.

The main cause of atrophic and dystrophic changes in the heart is hypoxia, which develops due to sclerosis of the coronary vessels and the discrepancy between the mass of the myocardium and the volume of its microcirculatory bed.

In large arterial trunks, the processes of sclerotic compaction of intima, atrophy of the muscle layer, and a decrease in the elasticity of the vascular walls develop. Peripheral vascular resistance increases, blood pressure increases, while venous pressure decreases due to loss of vein tone and expansion of the venous bed.

Under these conditions, the activity of the cardiovascular sieve becomes less effective, especially in physical exertion, when cardiac output increases and peripheral resistance remains high. This leads to compensatory left ventricular hypertrophy and an increase in myocardial mass. In all organs, the density of functioning capillaries decreases, their fragility increases and permeability decreases. The metabolism and gases between the blood and tissues are difficult. The slowdown of transcapillary diffusion is explained by the thickening of the basement membrane, the accumulation of insoluble collagen, and a decrease in the diameter of the pores.

Age-related changes in the heart and blood vessels are favorable conditions for the development of pathology of the circulatory system: coronary heart disease, hypertension, heart failure.

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АНАТОМО-ФІЗІОЛОГІЧНІ АСПЕКТИ СЕРЦЕВО-СУДИННОЇ СИСТЕМИ

Методичні рекомендації для самостійної роботи студентів
2-го курсу навчання медичного факультету з дисципліни
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(Англ. мовою)

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