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

Department of Internal Diseases No. 4

Methodological guidelines for conducting practical classes

with students of the 6th year of the Faculty of Medicine on the topic:

"Instrumental methods of research of diseases of the cardiovascular system"

Vladikavkaz 2022

3. Instrumental methods of research of the cardiovascular system.

Electrocardiography

The structure and functions of the conducting system of the heart.

The rhythmic work of the heart is provided by a conducting system, the feature of which is the ability to self-regulate, i.e. adjust to the required heart rate, depending on the volume of blood flowing to the heart or the body's need for it.

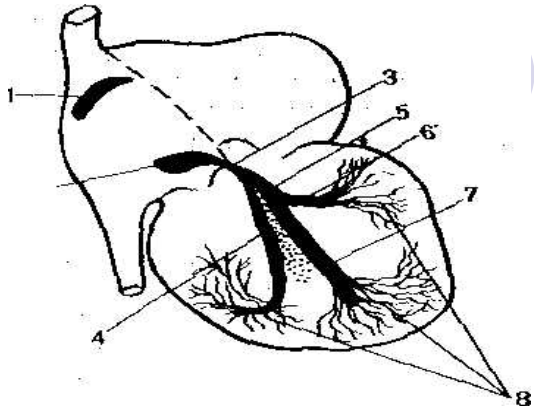


Схема строения проводящей системы сердца.

I — синусовый узел;
2 — атриовентрняулярный узел;
3 — пучок Гиса;
4— правая ножка пучка Гиса;
5 — общий ствол левой ножки *пучка* Гиса;
6 — передняя ветвь левой ножки;
7 —задняя вещь левой ножки;
8 —конечные разветвления ножек пучка Гиса - волокна Пуркинье.

The main dominant driver of the rhythm of the 1st order (pacemaker) – the sinoatrial (CA) node - is responsible for the function of automatism (the ability to autonomously generate impulses using p-cells (pacemaker). The length of the CA-node is 9-15 mm. The bathroom is located at the junction of the superior vena cava and the right atrium. The CA node generates pulses at a speed of 60-80 per minute. The CA node controls and regulates the heart rate in accordance with the activity of the body, the time of day and many other factors affecting a person

From the sinus node, the pulse spreads to the right and left atrium through 3 interstitial tracts: the anterior interventricular tract (Bachmann), the middle (Wenckebach) and the posterior (Torrel). The speed of the pulses is 1 m per second, i.e. the AV node is reached in 0.04-0.05 seconds.

First, the right atrium is activated, then the left atrium after 0.02□, so the P tooth is double-humped with a notch between it's teeth of 0.02□. When the conduction along this bundle is disrupted, atrial dissociation occurs.

The atrioventricular (AV) node is the center of automatism of the 2nd order. Located in the right atrium, in the lower part of the LV, slightly above the inner flap of the tricuspid valve, length — 5mm.

Functions:

1) When the AV node conducts an impulse from the atria to the ventricles, a delay of 0.04 s occurs. This ensures relaxation of the ventricles and filling them with blood during atrial contraction. The duration is 0.08 -0.12 sec.

2) filtration function or sorting of atrial excitation waves – not all impulses from the atrium pass to the ventricles,

The AV node (AV connection) is divided into 3 zones: 1) upper – AN (atrium nodus) 2) middle zone - (Nodus), 3 – lower (Nodus His). In the upper zone, the common path of the pulse is divided into 2 channels: alpha and beta, and then a common distal path. Each channel can conduct pulses in the ante- and retrograde direction. They differ in the speed of conduction and refractoriness: α-channel conducts pulses slower and has a short refractory period, β –faster and has a long refractory period. Channels can function in different directions – closed circuit – AV dissociation – re–entry mechanism, which underlies reciprocal tachycardia. This phenomenon is often observed in children.

There are no rhythm driver cells in the AV node itself, but P cells are focused in the lower part of the AV node, thanks to which a replacement rhythm and extrasystoles arise. P-cells are also located in the atria around the valves and, if the sinus node does not function, the atrial and AV centers of automatism take over its function and produce pulses with a frequency of 40-60 pulses in 1 min. This is a protection against asystole.

The lower part of the AV node passes into the trunk of the Gis bundle, it is divided into 2 legs - right and left. The left leg is divided into 2 or 3 main branches: the left anterior, the left posterior, some people have a third branch of LDL –the median or septum, lying in the same bed with the left anterior branch. The branches have a cable structure, i.e. each branch conducts impulses to a strictly defined segment at a speed of 3-4 m / sec.The right branch conducts impulses to the right ventricle, the left anterior one – to the anterior wall, the high lateral sections and the anterior papillary muscle of the left ventricle. The left posterior – to the posterior wall and the lower lateral walls of the left ventricle, as well as to the posterior papillary muscle, the median – to the MVP. The speed of the pulse along the left leg is greater than on the right, which allows the more powerful left ventricle to contract simultaneously with the right one. Purkinje fibers provide contact with the

ventricular myocardium. The pulse duration according to the Gis - Purkinje system is 0.035- 0.055 sec. I.e., in total, the interval PQ is 0.12- 0.20 sec.

In Gis bundles, Purkinje fibers, there are also P - cells that can generate pulses with a frequency of 30-40 in 1 min, they are centers of automatism of the 3rd order (idioventricular or ventricular rhythm).

Normally, the sinus node, due to the phenomenon of super-frequency suppression, oppresses the underlying centers of automatism.

Electrophysiological basics of ECG

The electrical processes in the myocardium are based on the movement of potassium, sodium, calcium, and chlorine ions through the membrane of the myocardial cell.

Phases of the cardiac cycle:

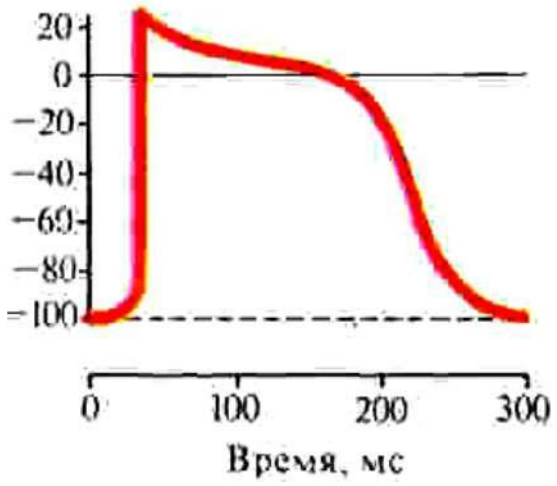
0-depolarization period

1,2,3 –the period of repolarization (1-fast or early, 2-slow, 3-late, 4-diastolic period).

Возбуждение - это процесс, в результате которого возникает **потенциал действия (ПД)**

Фазы ПД:

1. Деполяризация
2. Начальная быстрая реполяризация
3. Плато (медленная реполяризация)
4. Быстрая реполяризация - конечная



1.The muscle cell membrane separates two solutions that contain almost the same amount of ions of different chemical composition. Over 90% of the ions located outside the membrane are positively charged sodium ions and negatively charged chlorine ions. Inside the cell there are mainly potassium ions (positive ions), and the negative ions are heterogeneous organic, mainly protein, molecules. The concentration of sodium ions is almost 10 times greater outside the cell, and the concentration of potassium ions is almost 30 times greater inside the cell.

2. The concentration gradient of sodium ions promotes their flow into the cell, potassium ions – their diffusion from the cell to the outside. Concentration gradients of ions are caused by the active activity of ion pumps of the membrane. As a result, a potential difference of the order of 60-90 mV occurs on the membrane at rest, and positively charged ions predominate outside the cell membrane at rest, and negatively charged ions predominate on the inside of the cell membrane.

3. Ions move against concentration gradients due to the functioning of the so–called sodium pump - a special enzyme system that consumes a minimum amount of energy.

4. The cell membrane at rest is not permeable to sodium ions. However, when the membrane is irritated, its permeability to ions increases.
5. First, the current of sodium ions coincides with the concentration gradient, and sodium ions penetrate through the membrane into the cell. Penetrating into the cell, sodium introduces positive charges. This continues until equality of concentrations of sodium ions outside and inside the cell is achieved. The current of sodium ions into the cell coincides with the process of its excitation, or depolarization (phase 0).

.**6.** The flow of sodium ions from the extracellular fluid into the cell during the depolarization process leads to the fact that the outer side of the cell becomes negatively charged with respect to the non-excited areas of the muscle fiber. On the contrary, positive charges prevail inside the cell. As a result, the depolarization process spreads along the muscle fiber. As the excitation wave propagates in the muscle fiber, the permeability of the membrane also changes.

7. During depolarization, there is also an ionic calcium flow into the cell and calcium output from intracellular depots. The calcium ion triggers the mechanism of electromechanical coupling, providing the activity of contractile proteins.

8. The process of returning ions to their original position is called repolarization. Repolarization corresponds to phases 1-3 and therefore takes up almost the entire duration of the action potential. Since the cell is refractory to the next depolarization until the end of the repolarization process, the time period from the end of phase 0 to the final part of phase 3 is called the refractory period of the cell. Thus, the duration of the action potential determines the duration of the refractory period; if the duration of the action potential changes, the refractory period will also change. Repolarization of cardiac cells is a complex process that has not yet been fully studied. Repolarization begins quickly (phase 1), but is almost immediately interrupted by the plateau phase (phase 2), which is inherent only in cardiac cells (there is no plateau, for example, in nerve cells). Phase 2 depends on the functioning of "slow" calcium channels, through which positively charged calcium ions slowly enter the cell, suspending repolarization and lengthening the action potential. The most important ionic shift during repolarization is the outflow of positively charged potassium ions, which returns the action potential to the initial state of negative polarization. At least six different potassium "currents" have been identified; they function at different times of the action potential and are modulated by several factors (including the magnitude of the potential, calcium ions, muscarinic receptors, acetylcholine and adenosine triphosphate) under different circumstances.

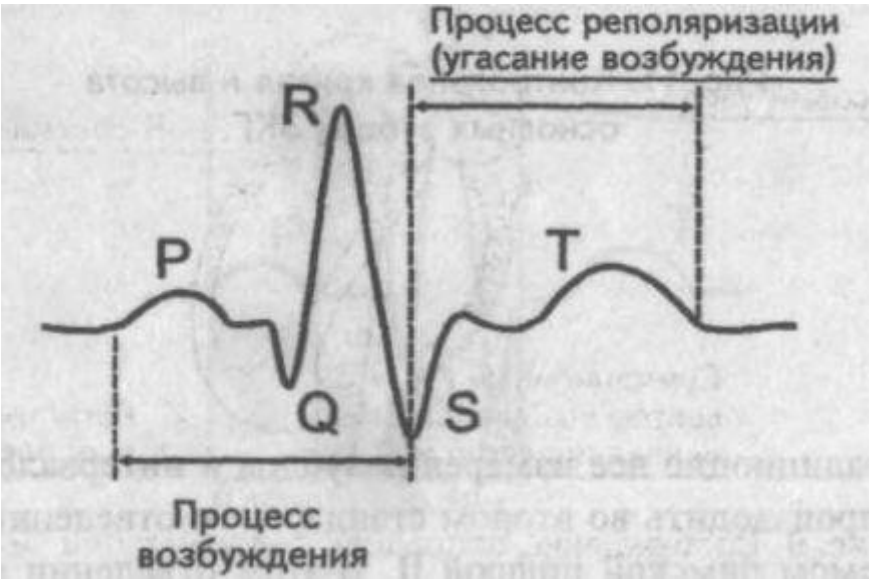
During the cardiac cycle, the processes of depolarization (excitation followed by contraction) and repolarization (return to the initial state of cells) are repeated.

In the myocardium there are groups of cells whose resting potential is higher than the resting potential of other fibers, and gradually increases, reaching the threshold potential level. As a result, spontaneous (spontaneous) polarization occurs. The function of automatism is possessed by cells of the sinus node, some groups of cells in both atria, some cells in the area of the atrioventricular node, cells of the Gis bundle and its legs and Purkinje fibers. The cells that perform the contractile work of the heart do not show the ability to spontaneously depolarize.

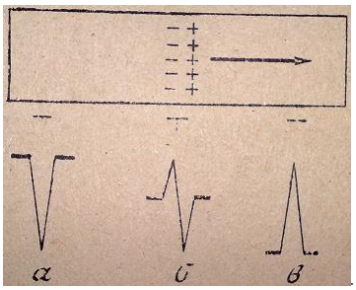
Thus, as a result of the electrical activity of the heart, an electric current arises, which spreads from the heart through the surrounding tissues, reaching the skin. Due to the electrical conductivity of the body tissues, electrodes placed on certain areas of the skin with different potentials can perceive this electric current and transmit it to an electrocardiograph. The electrocardiograph consists of an input device, a biopotential amplifier and a recording device.

The electrocardiograph converts the electrical signals received from each lead into a series of teeth that correspond to the depolarization and repolarization of the heart muscle, i.e.

An ECG is a graphical recording of the biopotentials of the heart using various lead systems, each lead registers a potential difference between two points.



The shape of the ECG complex of an individual muscle fiber depends on the location of the active electrode in relation to the depolarization front. If the electrode is located on a muscle fiber so that the excitation front moves away from it, i.e. it faces the negative side of the dipole, then a downward deviation occurs on the ECG.



Изменения формы ЭКГ в зависимости от места расположения активного электрода по отношению к фронту деполяризации мышечного волокна: а — электрод обращен к отрицательному полюсу диполя; б — электрод вначале обращен к положительному полюсу диполя, а после прохождения волны возбуждения — к отрицательному; в - электрод обращен к положительному полюсу диполя.

If the electrode is positioned so that the depolarization front approaches it, i.e. it faces the positive pole of the dipole, an upward deviation is recorded on the ECG. If the electrode is positioned so that the depolarization front initially approaches it, then passes the location of the electrode and continues to move away (for example,

in the middle of a muscle fiber), then an upward deviation is recorded on the ECG, and then a downward deviation is recorded.

At each moment of excitation of the heart, many dipoles of different magnitude and polarity, i.e. moment vectors, arise in it. The moment vectors in different muscle fibers act in relation to each other not in parallel, but at some angle or in the opposite direction. The resultant of the moment vectors formed in all the muscle fibers of the heart at the moment is the resulting moment vector.

During the propagation of excitation in the ventricular myocardium, each resulting moment vector is directed from the endocardium to the epicardium. The process of gradual coverage of the excitation of the heart muscle is accompanied by the sequential appearance of differently directed resultant moment vectors emanating from one common point of the dipole center.

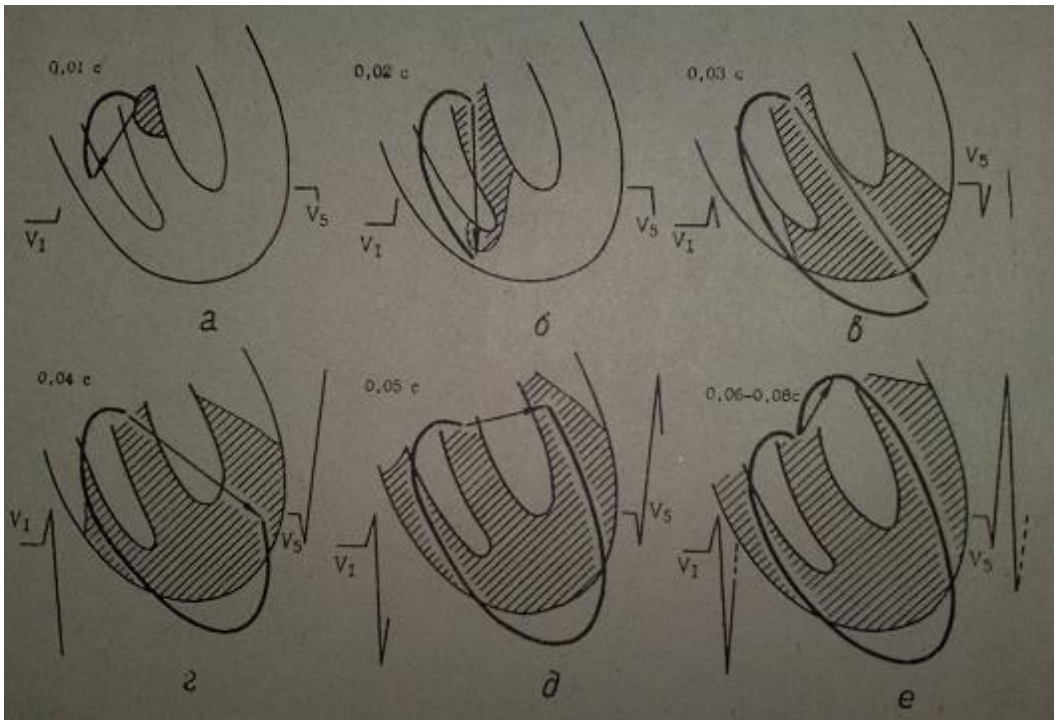


Рис. Схемы последовательности распространения процесса возбуждения по миокарду желудочков и образования результирующих моментных векторов сердца, формирующих петлю векторкардиограммы, и последовательности формирования комплекса в правых и левых грудных отведениях.

Standard ECG leads.

Currently, 12 ECG leads are most widely used in clinical practice: 3 standard, 3 reinforced single-pole leads from the extremities and 6 thoracic leads.

The concept of "leads" means the registration of an ECG when electrodes are applied to certain areas of the body that have a difference in potentials.

The mechanism of electrocardiography was discovered in 1913 by the outstanding Dutch physiologist Willem Einthoven. The standard leads proposed by Einthoven fix the potential difference between 2 points located on the extremities. To record these leads, an electrode with a red marking is applied to the right arm, with a yellow one - to the left arm, green - to the left leg, the fourth electrode is black, grounding - on the right leg. These electrodes are connected in pairs to an electrocardiograph and two-pole standard leads are recorded. Denoted by Roman numerals:

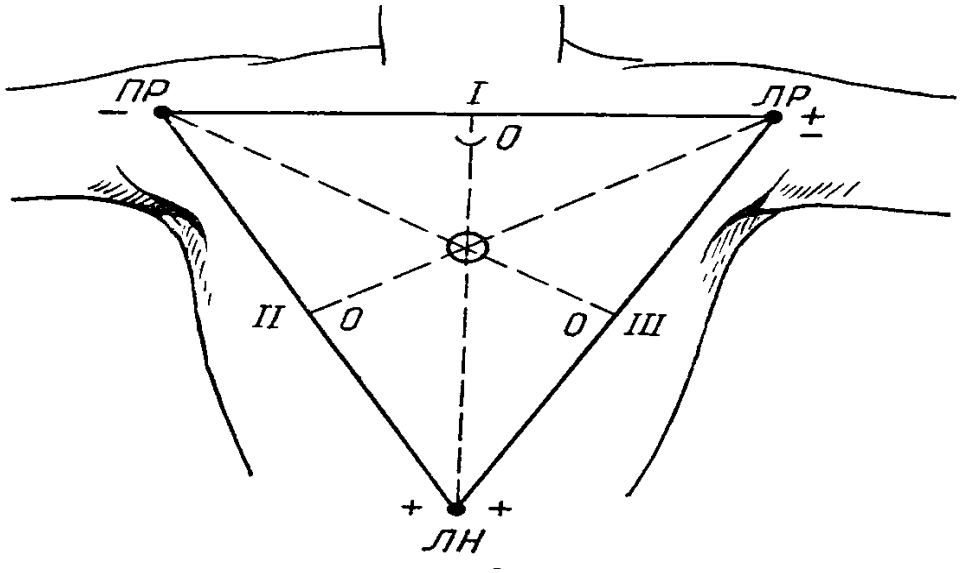
I lead - left hand (+) and right hand (-).

II lead – left leg (+) and right arm (-).

III abduction – left leg +) and left arm (-).

To record an ECG in standard leads, three recording electrodes are used, superimposed on the limbs. The red electrode is attached to the right arm, the yellow electrode to the left; the green electrode is fixed on the left leg. The fourth electrode, black, performs the role of grounding the patient and is superimposed on the right leg.

Three standard leads form an equilateral Einthoven triangle, the vertices of which are the right hand, the left hand and the left leg.



In the center of the triangle is the electrical center of the heart or a single cardiac dipole. The perpendiculars drawn from the center of the heart to the axis of each standard lead divide each axis into 2 equal parts: the positive one facing the positive (+) electrode and the negative one (-) facing the negative electrode. The line connecting the 2 electrodes involved in the formation of the lead is called the lead axis. The sides of the Einthoven triangle are the axes of standard leads.

Enhanced limb leads (proposed by Goldberg in 1942): They register the potential difference between one of the limbs on which the active positive electrode of this lead is installed (right arm, left arm or left leg) and the average potential of the other two limbs. These leads are designated as follows: aVR, aVL, aVF. The designations of reinforced leads from the limbs come from the first letters of English words: a - augmented (enhanced), V - voltage (potential), R - right (right), L - left (left), F - foot (leg).

avR- increased withdrawal from the right hand.

avL- enhanced withdrawal from the left hand.

avF-increased withdrawal from the right leg.

The axes of reinforced leads from the extremities are obtained by connecting the center of the heart with one of the vertices of the Einthoven triangle. Single-pole leads serve to confirm the changes found in standard leads. So aVR is a mirror image of the I lead, aVL repeats the changes of the I lead, aVF repeats III. In addition, they help to determine the electrical position of the heart.

Unipolar thoracic leads – proposed by Wilson in 1932, unipolar, are installed on the surface of the chest and are indicated by the capital Latin letter V (potential, voltage) with the addition of the position number of the active positive electrode, indicated by Arabic numerals:

Отв.V1- 4 intercostal space on the right edge of the sternum

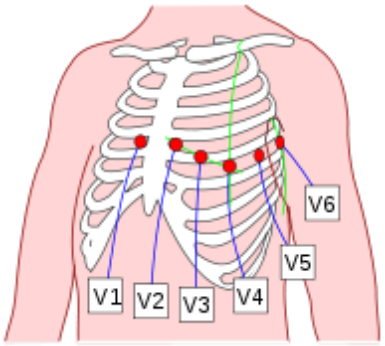
Отв. V2- 4 intercostal space on the left edge of the sternum.

Отв.V3- between the second and fourth position.

Отв.V4- в 5 intercostal space along the midclavicular line.

Отв.V5- в 5 intercostal space along the anterior axillary line.

Отв.V6- 5 intercostal space along the middle axillary line.



Thoracic leads register changes in EMF in the horizontal plane.

ECG recording technique

Application of electrodes: 4 plate electrodes with the help of rubber bands or plastic clips (clothespins) are applied to the limbs (see above), and one or more electrodes are placed on the chest. To improve the contact of the electrodes with the skin and reduce induced currents, the skin is degreased with alcohol, and the electrodes are coated with a special conductive paste. In the presence of a 6-channel electrocardiograph, 6 electrodes are simultaneously applied to the chest: a red wire is attached to the V1 electrode. V2- yellow, V3- green, V4- brown, V5- black, V6- purple or blue.

ECG recording is performed in the patient's supine position, which allows for maximum muscle relaxation, is carried out with calm breathing, in a warm room, away from possible sources of electrical interference. Use a tape pulling speed of 25 or 50 mm/sec. The patient should be stripped to the waist, the lower legs should be freed from clothing. It is advisable to record 2 hours after eating or on an empty stomach. The calibration of the electrocardiograph channel is set to 10 mm or 1 mV. It is necessary to register at least 6-10 cardiac cycles, and in the presence of arrhythmia significantly more - on a long tape (usually-II lead).

Normal ECG

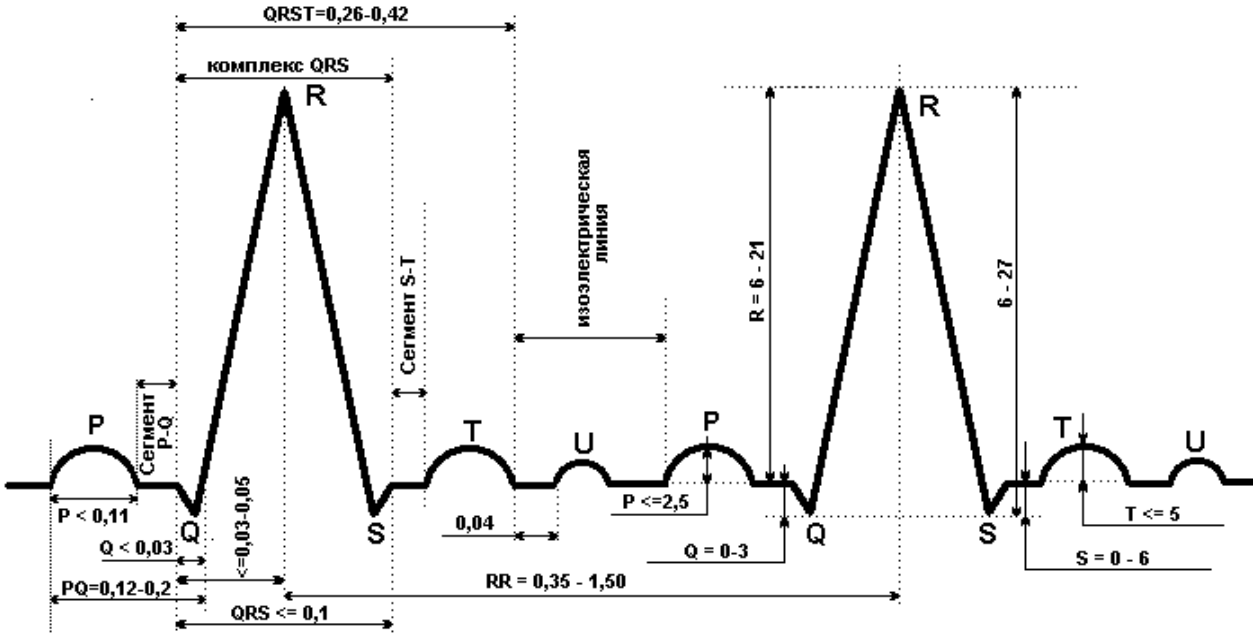
A normal ECG is represented by a number of teeth, segments and intervals, indicated by Latin letters.

ECG teeth are usually depicted in large Latin letters. The teeth directed upwards are considered to be positive, downwards-negative. All teeth are characterized by a

height (amplitude) in mm, and the time duration of the intervals and ECG teeth is measured in seconds or milliseconds. If the amplitude of the tooth is more than 5 mm, it is indicated by a capital letter, if less than 5 mm, then a small letter.

At the speed of paper movement-50 mm \ sec, 1 mm-0.02 sec, 5 m or one large cell = 0.1 sec. At a speed of 25 mm- 1 mm-0.04 sec, 5mm or 1 large cell = 0.2 sec.

Z. R is always positive, z. Q and S are always negative, z. R, T and U can be both positive and negative.



Atrial depolarization is recorded on the ECG in the form of the first wave P, if an electric pulse, as expected, is formed in the CA node. Normally it has the form of a semi-oval. Amplitude up to 2.5 mm, width-0.1 sec. First, the right atrium is activated, after 0.02 seconds.- left. At the normal position of the electric axis z.P is positive in I-III standard rel.(maximum in II standard rel.), avF.

Repolarization of the atria is barely noticeable, as it is hidden by powerful forces created by subsequent depolarization of the ventricles.

The PQ interval is the time of conduction from the CA node, along the atrioventricular junction to the ventricular myocardium, measured from the beginning of z. P before the beginning of z. Q, duration 0.12-0.20 sec, with bradycardia up to 0.22 sec

The QRS complex reflects ventricular depolarization, the total duration is 0.08-0.1 sec, with bradycardia - wider - up to 0.11 sec. z. Q- depolarization of the LVP,

width - 0.03 sec, amplitude - 2 mm. This tooth is unstable, there is always no right thoracic leads. z.R- depolarization of the apex, anterior, posterior, side walls, height in standard leads is 5-10mm, in thoracic leads -from 8 to 25mm. Normally, it is maximum in the II standard lead, in V4-5-6. The voltage is determined by the amplitude of the wave R: R I + RII + RIII > 15 mm (or R = 5 mm in standard, 8 mm –in chest). The S wave is the depolarization of the base of the heart. There may be no standard leads, the largest in V2 is 20-22mm. In V3 – the transition zone, R= S.

Segment ST- from the end of p.S. to the beginning of z. T.These are 1-2 phases of repolarization, normally in standard leads it is allowed to rise above the isoline up to 1 mm, depression-up to 0.5 mm. In avL, avF- on the isoline. In V1-2- lifting from 1.5-3.5 mm. In the left thoracic, there may be a slight depression up to 1 mm.

The T-wave is the 3rd phase of ventricular repolarization, equal to 1/8 of the amplitude of the ZR. Where the maximum ZR is, there will be a maximum wave.(V4). T is always positive in I,II,avL, avF, in V1- negative or two-phase. Juvenile negative z.T in V1-V2-V3, the deepest -in V1 (in children).

The state of the ST segment and the T wave is a marker of metabolic processes in the myocardium.

The U-wave presumably reflects the repolarization of the distal fibers of the conducting system. Increases with hypokalemia.

The QT interval is the electrical systole of the ventricles, measured from the beginning of the z to the end of the z. Its duration depends on the frequency of the rhythm. Its elongation by 10% of the norm is considered a pathology (with electrol. violations, taking antiarrhythmic drugs, ischemia) and is a marker of life-threatening arrhythmias. The congenital syndrome of prolonged QT (Jewel and Lange-Nielsen) is described: deaf-mute, syncopal states, QT prolongation on ECG. The prognosis is unfavorable, because at the time of syncope, paroxysmal tachycardia develops with transformation into ventricular fibrillation. Congenital syndrome, prolongation of Q-T (Romano-Ward) – syncope and prolongation of QT on ECG without deafness.

The R-R interval is used to measure the duration of the cardiac cycle (between the vertices of neighboring QRS complexes). Fluctuations of no more than 10% are allowed, if more – sinus arrhythmia, occurs in children in 95% of cases.

Criteria of sinus rhythm:

1**. in front of each QRS complex there should be a prong P, the same size and shape. At the same time, z.P is positive in leads I, II, avF, V5-V6, otp. in avR, two-phase in V1, positive in V2 -V3.**

2. The correct or regular rhythm is determined if the duration of the R-R intervals is the same and the difference between them is no more than 10% or 0.15 seconds, in other cases the rhythm is determined as incorrect.

Heart rate is calculated according to tables, rulers, or, according to the formula: 60 / RR in seconds.

Electrical axis of the heart

Electrical axis of the heart: the average direction of EMF during the entire period of depolarization. The position of the electrical axis of the heart is quantitatively expressed by the alpha angle, which is formed by the electrical axis of the heart and the I axis of the lead.

The nature of the location of the heart in the chest, and, accordingly, the main direction of its electrical axis, is largely determined by the features of the physique, as well as the state of the Gis bundle and the ventricular muscle. In children, persons with asthenic physique, the location of the heart is vertical. In people with a hypersthenic constitution, as well as with a high standing of the diaphragm (pregnancy, flatulence, ascites) - horizontal, with a deviation of the tip to the left. More significant rotations of the EOS around the anterior-posterior axis both to the right (more than +90°) and to the left (less than 0°) are usually caused by pathological changes in the heart muscle.

The definition of EOS is carried out according to the tables. To do this, compare the algebraic sum of the teeth R and S in I and III standard leads.

An easier way to approximate the direction of the EOS is to find the branch from the extremities in which the highest prong is R (without a prong S or with a minimum prong S). If the maximum tooth R in the I lead is the horizontal position of the EOS, if in the II lead it is the normal position, if in the aVF it is vertical.

There are the following options for the position of the electric axis:

1. Normal position- angle α=+30 до +69˚. RII >R I >RIII.

2. Horizontal axis- angle α =от 0до 29˚. R I >RII>RIII; R avf ≥s avf SIII>rIII.
3. Deviation to the left: angle α = от 0 до - 90˚. R I >RII>RIII; SIII≥rIII.

S avf > r avf.

4. Vertical axis: угол α =+70 - +90˚. RII>RIII>RI; SI=RI.
5. Deviation to the right: angle α = > + 90˚. RIII>RII >RI; S I> r I.

A moderate deviation of the electrical axis of the heart to the left or right in the absence of other ECG changes is not in itself a sign of pathological changes in the myocardium. Thus, a moderate deviation of the axis to the left may be due to the horizontal location of the heart in the chest and occasionally occurs in people with hypersthenic physique, obesity and other conditions that lead to the elevation of the diaphragm dome. Moderate deviation of the electrical axis of the heart to the right can, as a rule, be observed in children and adolescents, and sometimes in adults, especially asthenic physique.

In pathological conditions, hypertrophy of the left ventricle, developing in patients with GB, aortic and mitral (insufficiency) heart defects, some congenital malformations (open arterial (Botall) duct, moderate DMZHP), kidney diseases with hypertension is accompanied by a deviation of the electrical axis of the heart to the left. Hypertrophy of the right ventricle leads to a deviation of the electrical axis of the heart to the right in patients with chronic lung diseases with a pulmonary heart, with mitral stenosis, tricuspid valve insufficiency, with CHD with overload of the right parts of the heart.

ECG-characteristics of hypertrophy

Hypertrophy of the heart is a compensatory adaptive reaction of the myocardium, expressed in an increase in the mass of the heart muscle. Hypertrophy develops in response to increased stress in the presence of acquired or congenital heart defects or with increased pressure in the small or large circulatory circle.

The electrocardiographic concept of ventricular cavity enlargement includes hypertrophy of the wall, as well as dilation of the cavity as such, or a combination of these disorders. From the point of view of anatomy, the term "ventricular hypertrophy" refers to an increase in the size of the fibers and mass of the myocardium, whereas dilation is an increase in the cavity as such.

Electrocardiographic changes in this case are caused by: an increase in the electrical activity of the hypertrophied part of the heart; a slowdown in the conduction of an electric pulse through it; ischemic, dystrophic and sclerotic changes in the altered heart muscle.

Common signs:

1.with hypertrophy of a particular department of the heart, the EMF of this department increases, the total vector of the heart lengthens, and the amplitude of the teeth increases.

2. In addition, the electric axis deviates towards the hypertrophied department, but unlike leg blockades, the deviation is insignificant. Asthenics with an initially vertical axis may have a normal electric axis.

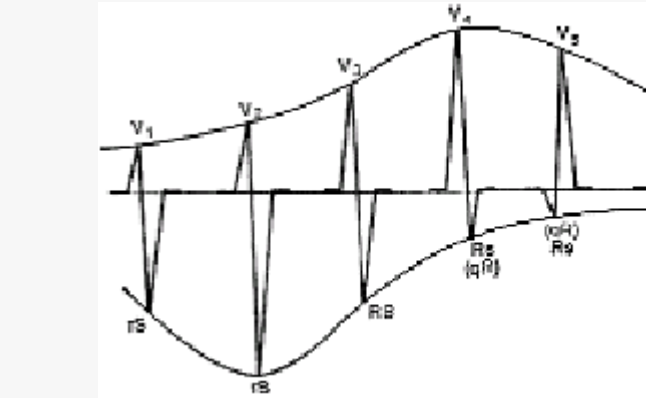
3.The broadening of the teeth reflecting the excitation of the hypertrophied part, but the broadening, unlike the blockades of the legs, is insignificant (due to myocardial dystrophy). If it was, n/a 0.06 sec, it will become 0.08.

4.sometimes the shape of the teeth changes, reflecting the excitation of this department. n/a for GPJ.

5. A violation of the processes of repolarization in the myocardium is characteristic→ the final part of the ventricular complex changes (while the total vectors of de- and repolarization have different directions) → obliquely descending depression of the ST with an asymmetric inverted tooth T. In opposite leads -the rise of the ST segment.

In the area of hypertrophied myocardium, repolarization proceeds much slower, not only because of the greater muscle mass, but mainly due to the lag in the growth of capillaries from the growth of hypertrophied muscles.

The asynchronism of repolarization leads to the displacement of the RS-T segment from the isoline and the inversion of the T wave. When analyzing the ECG, the transition zone in the thoracic leads should be taken into account.



The transition zone is determined by a lead in which the K and S teeth, i.e. their amplitude on both sides of the isoelectric line, are equal

A slight or moderate increase in the heart may not lead to changes in the ECG. In general, the sensitivity of the ECG method in the diagnosis of hypertrophy of the right and left ventricles is low. Echocardiography is a sensitive, reliable, non-invasive and easily reproducible method for determining dilatation of the heart, especially. the left ventricle, the thickness of their walls and the increase in mass.

Electrocardiographic signs of hypertrophy (overload) of the left atrium (P-mitrale).

Enlargement of the left atrium is a consequence of mitral regurgitation in congenital, acquired (due to rheumocarditis or infectious endocarditis), relative mitral insufficiency or mitral stenosis. Characterized by:

- an increase in the total duration (width) of the prong P of more than 0.10 s;
- widened double-humped prong P in leads I, aVL, V5-V6;
- the presence of a pronounced negative phase of the P wave in the V1 lead (more than 0.04 s in duration and more than 1 mm in depth).

Electrocardiographic signs of hypertrophy (overload) of the right atrium (P-pulmonale).

It develops with pulmonary pathology, as well as with a chronic pulmonary heart, with congenital heart defects. Characterized by:

- a high-amplitude prong P with a pointed tip in leads II, III, aVF, this feature is mandatory in leads V1 or V2;
- the duration of the prong P, not exceeding 0.10 s.

ECG-signs of left ventricular hypertrophy:

Causes: aortic stenosis, aortic insufficiency, aortic coarctation, GB, coronary artery disease, cardiomyopathy. LV hypertrophy is characteristic of athletes.

Since the ECG normally reflects the activity of only the left ventricle, electrocardiographic signs of left ventricular overload emphasize (exaggerate) the norm. Where there is normally a high prong R (in lead V4, whose position coincides with the left border of the heart), it becomes even higher; where there is normally a deep prong S (in lead V2), it becomes even deeper.

ECG signs of LVH are considered in the leads: I, II, aVL, V5,6.

- Sokolov-Lyon index: the sum of the amplitudes of the wave R in the lead V5 or V6 (where more) and S in the lead V1 or V2 (where more) more than 35 mm, more than 45 mm in young people.
- Cornell voltage index, gender-specific: Ravl + Sv3 > 28 mm for men, > 20 mm for women
    - R in V6 is larger than R in V5, larger than R in V4 and larger than 25 mm.
    - The displacement of the electric axis to the left.
    - RI" 15 mm, RavL≥11 mm.
    - The transition zone shifts to the right thoracic leads

ECG-signs of right ventricular hypertrophy:

Reasons: in children with CHD, in adults with mitral valve stenosis, with lung diseases (chronic pulmonary heart). In some cases (PE or acute decompensation in a patient with bronchopulmonary disease), acute dilation of the right ventricle may occur without hypertrophy of the walls, which is an exceptionally rare phenomenon in the left ventricle.

Electrocardiographic signs of overload (hypertrophy) of the right ventricle appear when its mass increases 2-3 times.

General ECG signs of right ventricular hypertrophy are considered in leads III, II, aVF V1,2.

- the high prong R in VI-V2 (R`>7 mm) when RV1>=SV1. In leads V5,V6, a deep S-wave appears.
- **with pronounced hypertrophy of the right ventricle, the ECG in V1,V2 has the form qR, with pronounced - rSR`, or rSR`, or rR`, with moderate - RS, Rs.**
- deviation of the electrical axis of the heart to the right
- RS-T segment decrease and T wave inversion are observed in III,II, aVF, V1- V2.

• The transition zone shifts to the left thoracic leads There may be an S-type ECG in thoracic leads, in which a pronounced S wave is observed from V1 to V6. The ECG has the form S, RS, or Rs. S-type is combined with the electrical axis of the spike SI-SII-SIII, it is more common in patients with pulmonary emphysema, pulmonary heart, mitral stenosis, pulmonary hypertension.

ECG diagnostics of intraventricular blockages

Intraventricular blockages: this is the slowing down or complete cessation of excitation along one, two or three branches of the Gis bundle.

ECG signs of blockage of the right leg of P. Gis:

1 - broadening of the QRS complex >0.12 sec.

2.QRS V1-V2 и III -avf типа - rSR’ или rsR’.

3.In the same leads, ST is shifted downward with a bulge upward, z.T is negative.

4.In the left thoracic leads (V5-V6); I and avl - wide, jagged z.S, ST –slightly raised and z.T +.

With an incomplete blockade of the PNPG, the holding on the right leg is preserved, but somewhat slower. ECG signs –QRS -0.09 sec.-0.11 sec. QRS V1 of type rSr’-rSR' , and in I rel. and in V6 –expanded z.S

ECG signs of blockage of the left leg of P. Gis: (2 branches of the left leg);

1. broadening of the QRS complex >0.12 sec.
2. deviation of the electrical axis of the heart to the left (non-permanent sign.).
3. in leads I, avl, V5-V6 – high, widened, M-shaped prong R, z.q, V5-V6 – absent. In these leads, ST is shifted downwards with a bulge upwards, z.T is negative
4. in leads III, avf, V1, V2 – an expanded and deepened complex of type QS or rS.

ECG-diagnostics of rhythm and conduction disorders.

The term "cardiac arrhythmias" is understood as arrhythmias and heart blockages. Arrhythmias are a violation of the frequency, regularity and sequence of heart contractions. Disturbances of excitation cause the development of heart blockades.

All arrhythmias are the result of changes in the basic functions of the heart: automatism, excitability and conduction. They develop when the formation of the action potential of the cell is disrupted and the rate of its conduction changes as a result of changes in potassium, sodium and calcium channels. Violation of the activity of potassium, sodium and calcium channels depends on sympathetic activity, the level of acetylcholine, muscarinic M2 receptors, ATP.

Clinical and electrocardiographic classification of arrhythmias

For practical use, the clinical and electrocardiographic classification of cardiac arrhythmias is convenient (According to M.S. Kushakovsky and N.B. Zhuravleva, modified by G.E. Roitberg and A.V. Strutynsky):

- 1.Violation of impulse formation.
  - Violations of the automatism of the CA node (nomotopic arrhythmias):
    - Sinus tachycardia.
    - Sinus bradycardia.
    - Sinus arrhythmia.
  - Sinus node weakness syndrome.

    2.Ectopic (heterotopic) rhythms caused by the predominance of automatism of ectopic centers:
    - Slow (substitute) slipping complexes and rhythms:
      - Atrial complexes and rhythms.
      - Complexes and rhythms from the AV connection.
      - Ventricular complexes and rhythms.
    - Accelerated ectopic rhythms (non-paroxysmal tachycardia):
      - Atrial ectopic rhythms.
      - Ectopic rhythms from the AV connection.
      - Ventricular ectopic rhythms.
    - Migration of the supraventricular pacemaker.

    3.Ectopic (heterotopic) rhythms, mainly due to the mechanism of re-entry of the excitation wave:
    - Extrasystoles:
      - Atrial extrasystole.
      - Extrasystoles from the AV connection.
      - Ventricular extrasystole.

- Paroxysmal tachycardia:
  - Atrial paroxysmal tachycardia.
  - Paroxysmal tachycardia from the AV connection.
  - Ventricular paroxysmal tachycardia.
- Atrial flutter.
- Atrial fibrillation.
- Fluttering and flickering (fibrillation) of the ventricles.

I.violations of the function of automatism:

Sinus tachycardia.

An increase in heart rate from 90 to 150 per minute while maintaining the correct sinus rhythm.On the ECG - heart rate >90-100 per minute, the rhythm is sinus, correct, shortening of the intervals PQ (at least 0.12") and RR.

Sinus bradycardia.

Reduction of heart rate to 60-40 per minute while maintaining a normal sinus rhythm. If less than 30-CA-blockade, etc.

On an ECG, the heart rate is less than 60 per minute, the rhythm is correct sinus, an increase in the duration of the intervals PQ (up to 0.22") and RR.

Sinus arrhythmia

Irregular sinus rhythm with periods of increased and slower heart rate, while the difference between RR intervals exceeds 0.15.

On the ECG, the rhythm is sinus, the RR intervals are different (fluctuations of more than 0.15").

II. Disorders of the excitability function**:**

Extrasystole (Ex).

Ex -this is a premature excitation of the heart or any part of it.

ECG. 1. premature extraordinary appearance on the ECG of the ventricular complex.

2. The distance from the preceding normal P-QRS complex to the extrasystole is the coupling interval or the preectopic interval. The distance from Ex to the next P-QRS cycle is called the compensatory pause –the post-extrasystolic interval. The compensatory pause can be complete (equal to two R-R intervals) and incomplete (less than two R-R intervals).

3**.** According to the place of occurrence of an extraordinary pulse, extrasystoles are distinguished: atrial and from AV connections (supraventricular - unchanged ventricular complex). P is deformed, negative, or absent, incomplete compensatory pause) and ventricular (the ventricular complex is deformed and widened, the T wave and the ST segment are discordant, the P wave is absent, complete compensatory pause)

.

Paroxysmal tachycardia (PT).

This is a sudden onset and sudden termination of an attack of increased heart rate from 120 to 250 per minute, while maintaining in most cases the correct regular rhythm. In accordance with the localization of the pathological focus , PT is isolated:

1. atrial
2. atrioventricular
3. ventricular.

On an ECG: 3 or more consecutive extrasystolic complexes are taken for PT. Heart rate up to 140-250.

With atrial PT –per minute, the P-wave is deformed, located in front of the QRS, the QRS complex is supraventricular (narrow), the P-R and R-R intervals are short, the same in duration. In AV-nodular tachycardia, the P–waves are negative, located behind the QRS complex, or absent. In ventricular PT, deformation and expansion of the QRS complex is more than 0.12', with a

discordant location of the ST segment and z.T. Unchanged positive z are recorded. P in its rhythm -70-90 per minute.

Atrial fibrillation.

This is frequent (from 350 to 700 in 1 min.), disorderly, chaotic excitation and contraction of individual groups of muscle fibers from ectopic atrial foci, while there is no effective atrial contraction.

ECG signs: absence of atrial fibrillation waves-f are recorded in all leads instead of atrial fibrillation throughout the entire cardiac cycle. Irregular ventricular rhythm (different R-R intervals). Ventricular complexes –QRS –have a normal appearance. There are tachy- (more than 100 sokr. in min.), norm- (60-100) and bradysystolic forms (less than 60).

Atrial flutter.

This is frequent -200-300 in 1 min., rhythmic excitation of the atria, fibrillation is less common, fluttering waves also occur –(characteristic "sawtooth" pattern), regular; if the ratio of f and QRS is stable- the correct form is 2:1, 3:1, etc.

Flutter and ventricular fibrillation.

These are random ineffective contractions of individual bundles of myocardial fibers with a high frequency.

Flutter – regular waves – "saw", there is no isoline, 180-250-per minute, ventricular complexes are not differentiated.

Fibrillation is a high frequency of ventricular contractions, more than 300 per minute, completely different waves, different duration and amplitude, the absence of differentiated ECG elements.

III. Impaired conduction function (sinoatrial, atrial, atrioventricular, intraventricular blockades).

The slowing down or complete cessation of the electrical impulse through any part of the conducting system has been called a heart block.

Sinoatrial blockade is a violation of the electrical impulse from the sinus node to the atria.

ECG. The 1st and 3rd degree of blockade cannot be established by this method. The blockade of the 1st degree is recognized only by special electrophysiological methods. With a block of the 2nd degree, separate cardiac cycles (z.R and QRS) periodically fall out. At the time of such falls, a long pause is recorded on the ECG, exceeding the usual b –R –R intervals by 2-3-4 times, if 2-3 or 4 cycles in a row fall out.

Atrioventricular blockades: - this is a violation of the conduction of the pulse from the atria to the ventricles at the A-V node level.

There are 3 degrees of AV blockade:

AV block of the 1st degree–slowing of atrioventricular conduction, on ECG - prolongation of the PQ interval of more than 0.2 seconds..

AV block of the 2nd degree – some impulses are not carried out from the atria to the ventricles and part of the ventricular complexes falls out.. If the PQ interval gradually lengthens before the loss of the QRS complex, such a blockade is called an AV blockade of the 2nd degree of type 1 with Samoilov-Wenkebach periods, (Mobitz 1). In other cases, the ventricular complex periodically falls out, but the PQ interval is stable-AV block of the 2nd degree of the 2nd type (Mobitz 2).

AV-blockade of the 3rd degree –complete-the pulse from the atria is not carried out to the ventricles, they function independently of each other – z.P in its rhythm, more frequent than QRS complexes (the frequency of ventricular contractions is less than 60 per minute). The atria are excited from the sinus node, and the ventricles from the atrioventricular node or ectopic foci of automatism of the II or III order. Severe

bradycardia with ineffective hemodynamics may develop. The ECG shows complete dissociation between the P teeth and the QRS complexes.

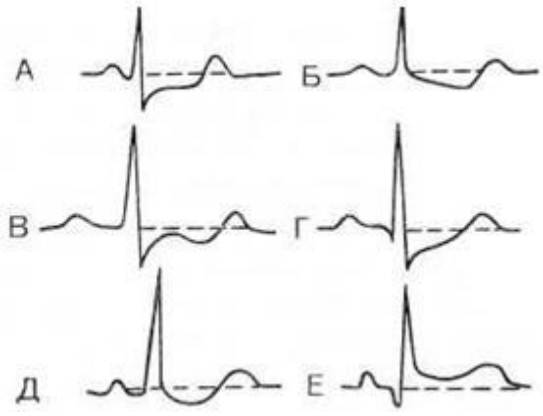
ECG diagnosis of coronary heart disease (angina pectoris and myocardial infarction).

The emerging ECG changes are nonspecific for chronic coronary heart disease and chronic coronary insufficiency and are also observed in various diseases.

Therefore, clear objective electrocardiographic criteria for chronic coronary heart disease are absent in most cases and the diagnosis is made on the basis of a complex of clinical and electrocardiographic data using various functional tests.
It should be borne in mind that in 30-85% of patients with known chronic ischemic heart disease, the ECG may be completely normal. Naturally, the diagnosis is significantly helped by a history of myocardial infarction or focal myocardial changes. Diagnosis is also facilitated when the patient complains of typical, especially frequent angina attacks. During myocardial ischemia, ECG records changes in the final part of the ventricular complex — the ST segment and the T wave.

Types of ST segment displacement observed in chronic coronary heart disease. A – horizontal; B – oblique descending; B – with an arc facing the bulge upwards; D – oblique ascending; D – trough–shaped; E - the rise of the ST segment

.



For chronic coronary heart disease, the first two types of ST segment displacement are most specific, i.e. horizontal and oblique descending. ST segment changes characteristic of coronary heart disease are more often observed in thoracic leads V4 –V6, as well as in leads II, III, aVF, I and aVL.

Tests with physical activity (bicycle ergometry, treadmill test).

During the exercise test, the patient performs an increasing load on a treadmill or bicycle ergometer, while heart rate and ECG are constantly recorded, blood pressure is monitored at regular intervals (1-3 minutes).

The main indications for carrying out load tests:

- differential diagnosis of coronary heart disease and its individual forms;
- determination of individual tolerance to FN in patients with an established diagnosis of coronary heart disease and clarification of FC angina pectoris;
- evaluation of the effectiveness of therapeutic, including surgical and rehabilitation measures;
- examination of the ability to work of patients with CVD;
- estimation of the forecast;
- evaluation of the effectiveness of antianginal drugs.

Absolute contraindications to the FN test are the acute stage of MI (within 2-7 days from the start), unstable angina pectoris, cerebrovascular accident, acute thrombophlebitis, pulmonary embolism, HF III-IV FC according to the classification of the New York Heart Association (NYHA), severe pulmonary insufficiency, fever. It is impractical to perform a diagnostic test for tachyarrhythmias, complete blockage of the left leg of the Gis bundle, high degrees of sinoatrial and atriovenocular blockages.

The FN test is performed before the development of an angina attack, the appearance of signs of myocardial ischemia on the ECG, the achievement of the target heart rate, the development of severe fatigue, making it impossible to prolong the FN, and the patient's refusal to conduct the test. The FN test should be stopped when:

- the development of a typical angina attack;
- the appearance of life-threatening cardiac arrhythmias: frequent, or polytopic, or volleyed ventricular extrasystole, paroxysmal tachycardia or paroxysmal atrial fibrillation;
- the occurrence of severe shortness of breath (the number of breaths more than 30 per minute) or an attack of suffocation;
- the development of conduction disorders – blockade of the legs of the Gis beam, atrioventricular blockade of 2 degrees or more;
- ischemic displacement of the ST segment upwards > 1 mm in any of the leads except V1–2, where the rise is considered to be 2 mm or more, or down from the isoelectric line > 1 mm and lasting 80 ms from point J, slow downward descent of the ST segment at point J +80 ms > 2 mm (rapid downward descent ST is not accepted as ischemic);
- lifting SAD > 220 mmHg, DAD > 110 mmHg, lowering SAD by 20 mmHg.;
- the appearance of neurological symptoms – dizziness, impaired coordination of movements, severe headache;
- the occurrence of intense pain in the legs;
- the development of severe fatigue of the patient, his refusal to further perform the test;
- as a precautionary measure by a doctor's decision;
- reaching 75% of the maximum age heart rate.

If the above criteria are met, the sensitivity of the FN sample for detecting patients with anatomically significant coronary artery lesion, i.e. narrowing > 50%, is 65-80%, and the specificity is 65-75%. Patients with positive test results are more likely to have severe damage to several coronary arteries. A test with FN is considered positive in terms of the diagnosis of coronary heart disease if it reproduces typical pain or tightness in the chest for the patient and there are changes characteristic of ischemia

on the ECG. Pain does not always accompany a decrease in the ST segment, the test is considered positive if the decrease appears without pain, or if a typical angina attack develops without a decrease in the ST segment.

Given the great importance of textual information, it is necessary in all cases (in the absence of contraindications) to strive to perform stress tests in patients with SS.

ECG diagnosis of MI.

Electrocardiography is one of the main methods of diagnosing myocardial infarction, but a normal ECG does not exclude the presence of this disease. ECG changes depend on a number of factors-

а) reversibility of the lesion (ischemia or myocardial infarction);

б) the duration of the disease (myocardial infarction or postinfarction cardiosclerosis);

в) the depth of the lesion (transmural or subendocardial myocardial infarction);

г) localization (anterior or inferior myocardial infarction), concomitant disorders (myocardial hypertrophy, conduction disorders).

With the development of myocardial infarction, there are violations of repolarization processes (changes in the ST segment and T wave), as well as depolarization processes (changes in the QRS complex). Extensive myocardial necrosis leads to a decrease in the amplitude of the R wave and the appearance of pathological Q waves.

Numerous studies comparing autopsy and ECG data have shown that transmural infarcts can occur without pathological Q waves, and vice versa, subendocardial (nontransmural) infarcts can occur with pathological Q waves. Therefore, it is more correct to divide myocardial infarcts into infarcts with and without pathological Q waves.

Reliable electrocardiographic criteria for myocardial infarction:

-the appearance of new Q teeth with a width of more than 30 ms and a depth of more than 2 mm in at least two leads from the following: a) leads II, III or aVF; b) leads V1 - V6; c) leads I and aVL.

-A newly appeared rise or depression of the ST segment more than 1 mm 20ms after the point J* in two adjacent leads.

-Complete blockade of the left leg of the Gis bundle in the presence of an appropriate clinical picture.

ECG is the most important method of diagnosing MI, allowing:

1) identify myocardial infarction

2) to establish the localization of MI, its depth and prevalence

3) diagnose complications of MI (arrhythmias, formation of a heart aneurysm, etc.)

According to Bailey, an ECG in MI is formed under the influence of three zones formed in the infarction area: 1. necrosis zone. 2. the zone of pronounced dystrophy, which in the ECG is called the damage zone. 3. ischemia zone.

Ischemia develops primarily in the subendocardial parts of the myocardium, which are supplied with blood worse than subepicardial, because the endocardial vessels experience greater pressure from the blood contained in the ventricles, so ischemia develops earlier in the subendocardial layers.

ECG-a manifestation of subendocardial ischemia will be a high, pointed ZT in the thoracic leads or leads from the extremities – this is the acute stage of MI.

Ischemia usually cannot last long (max.30 min.-1 hour): either the metabolism in the myocardium is restored or the focus of ischemia begins to spread and reaches the subepicardium, which is manifested by a negative z.T on the ECG.

The next stage is subendocardial damage, based on histological changes in myocardial cells, manifested by ST depression. All these changes in the myocardium M.B. are reversible. In case of subepicardial or transmural damage, we register an increase in ST on the ECG.

In myocardial infarction, myocardiocytes die, intracellular potassium ions leave the dead cell, accumulate under the epicardium, forming "electric currents of damage" in the necrosis zone, the vector of which is directed outward. These damage currents significantly alter the repolarization processes (ST and T) in the necrosis zone, which is displayed on the ECG tape. Recording electrodes located both above the infarction area and the opposite one record these damage currents, but each in its own way.

Рис. 91. Токи повреждения

With a continuing violation of coronary circulation, muscle fibers die and necrosis of muscle fibers develops. The damaged zone is not excited, because it does not produce electricity, as a result, the z.R < or disappears, a wide and deep z.Q or Q-S is formed instead. The greater the depth of necrosis, the wider the pathological z.Q. We call a pathological tooth Q such a tooth Q, the width of which exceeds 0.03 s. The genesis of the normal Q wave is the excitation of the interventricular septum, and the time of its excitation does not exceed 0.03".

Рис. 90. Возбуждение при инфаркте миокарда

Thus, we have 3 main ECG signs of MI: pathological z.Q, displacement of S-T upward from the isoline and negative z.T. On the opposite walls -reciprocal changes– (discordant displacement of the S-T segment below the isoline in leads opposite to the infarction area).

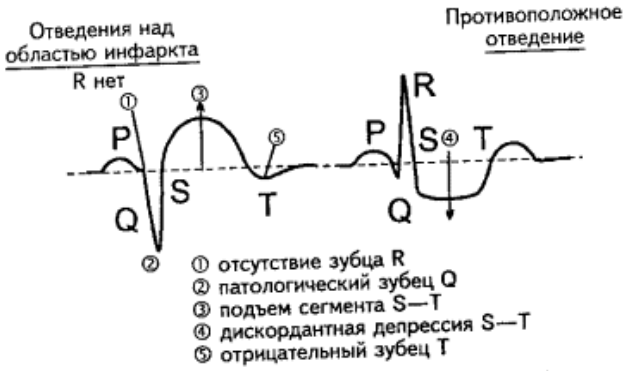


Рис. 92. ЭКГ признаки инфаркта миокарда

In myocardial infarction with a Q wave, there are four stages: acute, acute, subacute and scarring.

- acute - characterized by the development of a "monophasic curve" (pronounced ST segment elevation, at the same time ST segment depression may be observed in reciprocal leads, the appearance of which indicates the vastness of myocardial infarction).
- acute - the "monophase curve" persists on the ECG and a pathological Q wave appears, the amplitude of the R wave decreases or it disappears completely (the QS wave is formed).
- subacute - the ST segment returns to the isoline, and pathological Q or QS teeth, low-amplitude R teeth and negative T teeth remain.
- scarring — in the cicatricial stage, signs of myocardial infarction may persist on the ECG in the form of pathological Q teeth, low-amplitude R teeth, negative T teeth. In a number of patients, signs of the acute or acute stage of myocardial

infarction ("monophasic curve") may persist on the ECG, which may indicate an aneurysm.

IM without a Q prong, more often small-focal:

1.Intramural (necrosis zone in the thickness of the heart muscle,) - in the acute stage, for a short time (several hours, less often-days), S-T is shifted upwards from the isoline, in the second half of 1 day a negative T appears, which is often the only sign of MI on the first ECG. The QRS complex has not been changed - there is no z.Q and a decrease in z.R. In the future, the usual dynamics of z.T occurs. Negative z.T is recorded on the ECG for more than 2 weeks (4-6), often remains negative and in the scar stage.

2. Subendocardial MI, as a rule, is a common process, subendocardial necrosis can permeate half of the myocardium. The main change in the ECG is depression of the ST segment by 2 or more mm in 2 or more leads, at the end of 1-2 days a negative T is added. Within 1-2 weeks, the ST segment approaches the isoline, a deep negative z.T. QRS remains unchanged, there is no pathological z.Q.

Topical diagnosis of myocardial infarction.

Infarction zone	Leads in which pathological Q teeth are detected
Перегородочная	V,-V_2
Передняя	V3-V4
Переднесептальная	V1-V3
Боковая	I, aVL,V_4 -V_6
Нижнебоковая	II, III, aVF, I, aVL, V_4 - V_6
Переднебоковая	I, aVL,V, -V_6
Нижняя	II, III, aVF
Задняя	R/S>1 в отведениях V1, V2
Правый желудочек	V_4R,V_5R*

* - V_4R , V5R - записывать ЭКГ, расположив электроды на правой половине грудной клетки, зеркально расположению электродов V_4 и Vs. (рис. 2-8)

ECG for some heart diseases (myocarditis, heart defects, pulmonary heart, dysmetabolic processes in the myocardium).

Myocarditis causes the following ECG changes (one or more):

- various conduction disorders, including elongation of the ventricular electrical systole (QT), atrioventricular blockades of I or II degree, intraventricular conduction disorders and blockages of the legs of the Gis bundle;
- various cardiac arrhythmias, such as sinus tachycardia, ectopic rhythms, atrial fibrillation, extrasystole, etc..;
- ECG changes observed in acute fibrinous or exudative pericarditis.

In myocarditis of any etiology, changes in the terminal part of the ventricular complex, a downward shift of the ST interval and the appearance of a reduced, smoothed or negative T wave can be detected. These changes are determined in the thoracic or (and) in standard leads.

It is possible to reduce the voltage of the teeth of the QRS complex, its serration. Sometimes there are blockages of the legs of the Gis bundle and branches of the left leg, as well as various combinations of them. With rheumatic myocarditis, various disorders of atrioventricular conduction most often appear first.

With pulmonary embolism , the following electrocardiographic signs of acute overload of the right parts of the heart usually appear:

1. There is a deviation of the electrical axis of the heart to the right or a tendency to such an arrangement of the electrical axis of the heart. For example, before the acute situation, the electrical axis of the heart was located horizontally. With the development of pulmonary embolism, the electrical axis may become normal or vertical.
2. "Ppulmonale" appears, indicating the development of overload of the right atrium with high pointed teeth PII, III, aVF.
3. The amplitude of the R teeth increases in the II, III and aVF leads.
4. The electrical axis of the heart type SI–SII–SIII is determined.

Signs of hypertrophy or overload of the right ventricle in the thoracic leads are revealed:

- an increase in the amplitude or the appearance of a high R wave in the right thoracic leads V1, V2, V3. ECG in leads V1, V2, V3. R may look like R, Rs, qR, and sometimes even QR. In rare cases, the QS complex is registered in these leads. This sometimes makes it necessary to make a differential diagnosis with myocardial infarction of the anteropeptal region;

- registration of a pronounced RV5, V6 prong;

- development of signs of complete or incomplete blockade of the right leg of the Gis beam with registration of ECG type rsR in V1, V2 and with the appearance of a widened tooth SV5, V6;

- reduction of the amplitude of the Rv5, v6 prong with a decrease in the R/S ratio in the V4–V6 leads;

- increase in the activation time of the right ventricle in leads V1, V2;

- the rise or fall of the ST segment in V1- V2. The rise of the STV1, V2 segment is noted more often with massive embolism of the vessels of the small circle, the bulge of the ST segment is turned upward. ST segment depression is usually observed with a moderate increase in pressure in the pulmonary artery system;

- ST segment reduction in V4-V6;

- the appearance of a negative T-wave in leads V1–V3. Sometimes, with pulmonary embolism, a negative T wave is noted not only in leads V1–V3, but from V1 to. V6, which is caused by a deterioration in the nutrition of the left ventricle;

- registration of the late R wave in the aVR lead;

- increase in the amplitude of the P-wave in leads V1 – V5;

- shift of the transition zone to the left;

- the development of sinus tachycardia, and sometimes other rhythm disorders: atrial flicker and flutter, paroxysmal tachycardia, ectopic rhythm from the atrioventricular junction, atrioventricular conduction disorders, etc..

These ECG changes occur in only 15 to 40% of cases and are more often observed when the pulmonary artery lumen is blocked by half or more.

Electrocardiography can help in the diagnosis of acquired heart defects [Makolkin V. I., 1978]. Electrocardiographic diagnosis of heart defects is based on determining the position of the electrical axis of the heart, identifying signs of

hypertrophy and overload of various parts of the heart. Information about the nature and topic of rhythm disturbances obtained during electrocardiographic examination can also be used in solving the problem of damage to the valvular apparatus of the heart.

It should be borne in mind that the vertical location of the electrical axis of the heart or its deviation to the right in combination with electrocardiographic signs of right ventricular hypertrophy most often occur with mitral defects with a predominance of left venous stenosis or mitral tricuspid defects, less often – with combined mitral aortic defects.

The horizontal location of the electrical axis of the heart or its deviation to the left in combination with electrocardiographic signs of left ventricular hypertrophy are characteristic of aortic defects, mitral insufficiency, as well as for combined mitral defects with predominance of mitral valve insufficiency. Such an electrocardiographic picture can also be observed with combined mitral aortic heart defects. In mitral malformations, a frequent electrocardiographic finding is hypertrophy of the left atrium.

Electrocardiographic signs of hypertrophy of both ventricles in heart defects are usually caused by a combined mitral defect or a combined lesion of the mitral and aortic valves.

Atrial fibrillation is most specific for mitral stenosis. The coarse-waved form of flicker distinguishes it from atrial fibrillation of another etiology. Electrocardiographic picture of diastolic overload of the left ventricle with deep qV5,V6 is very characteristic of aortic valve insufficiency. With stenosis of the aortic mouth, electrocardiographic signs of systolic overload of the left ventricle with a small qV5, V6 are often noted. Blockade of the left leg of the Gis bundle more often indicates stenosis of the aortic mouth, less often – insufficiency of the aortic valves and does not occur with isolated mitral heart disease.

Dysmetabolic myocardial disorders.

During menopause or in the pre-menopausal period, as well as with various hormonal disorders, women often experience the same ECG changes as with chronic coronary heart disease [Vorobyev A. I. et al., 1980]. These changes concern the final part of the ventricular complex – the ST segment and the T wave.

Significantly more often, pathology is observed on the part of the T-wave: a high positive, reduced, smoothed, two-phase or negative T-wave in one or more often in several leads. Changes in the T-wave are observed mainly in the thoracic leads, more in the right ones.
They can occur acutely – in the pre- or menstrual period. Their appearance is not associated with pain in the heart area. Pathological ECG changes are not accompanied by the dynamics of biochemical and general blood parameters and an increase in body temperature characteristic of acute coronary circulatory disorders with the development of focal changes in the myocardium.
Pathological ECG changes can also occur against the background of pronounced menopause and are characterized by stability, without changing in dynamics. For menopausal or dishormonal cardiopathy, it is specific to improve the ECG during a functional test with potassium and obsidan.

Daily monitoring of ECG and blood pressure

Daily (Holter) ECG monitoring is a method by which daily monitoring of the work of the heart is carried out.
To do this, a wearable portable recorder is used, which makes a round-the-clock recording of an electrocardiogram and transfers information about the work of the heart per day to a computer.

Summarizing twenty-five years of research experience in the field of recording electrical phenomena and the possibility of transmitting electroencephalograms by radio, Norman J. Holter created and introduced in 1961 a new method for recording ECG.

Over the following years, we witnessed the improvement of this method of examination, which, unlike the standard ECG method, is called daily, outpatient or Holter.

Over the years, the 40-kilogram radio transmission recorder, which was fixed on the patient's back, was replaced with a magnetic tape recorder, which weighed about 2 kg at first, and now less than 0.5 kg.

Technical improvement has led to improved recording quality, minimized artifacts associated with the patient's physical activity. Now it is possible to register 12 leads, automatic analysis.

The improvement of the equipment made it possible to increase the number of recorded and analyzed parameters. Currently, along with the analysis of cardiac arrhythmias, it has become possible to quantify the displacement of the ST segment, assess the function of the pacemaker (EX) and the cyclic variability of the heart rhythm, determined automatically in the form of various temporal and spectral parameters, record averaged ECG signals with high gain and automatic measurement of the duration of the QT interval.

As a rule, modern XM apparatuses use ECG registration in 1-12 modified thoracic leads, imitating (but not identical!) chest leads V1 (CM1) – V5 (CM5).

All patients with XM are recommended to keep a diary of activity with a record of the symptoms that arise during the study

- Physical and emotional stress
- Taking medications
- Eating
- Travel by transport/ driving
- Rest
- Sleep
- Unpleasant sensations (palpitations, "interruptions" in the work of the heart, chest pain, etc.)

-
- Indications for daily ECG monitoring
- Analysis of the presence and nature of cardiac arrhythmias and conduction
- Evaluation of the effectiveness of antiarrhythmic therapy
- Detection of ischemic episodes
- Evaluation of the pacemaker
- Assessment of heart rate variability

Daily blood pressure monitoring (SMAD).

A single definition of blood pressure provides information only for a single moment in time and does not always reflect the real clinical picture. Since the 70s of the 20th century, daily blood pressure monitoring (SMAD) has been widely used, providing additional information about the blood pressure level outside the doctor's office (in the most natural conditions). In the PAMELA study [2010], the incidence of white coat hypertension was 9-12% in the general population, and 36% among patients with grade I hypertension, if the level of average daily blood pressure<135/85 mmHg was used as its criterion. Automatic wearable devices for SMAD reproduce the algorithm of auscultative or oscillometric measurement methods. Most devices measure blood pressure using the oscillometric method. However, the auscultative method is preferable when monitoring patients with increased motor activity.

Indicators of the daily blood pressure profile according to the SMAD data

1. The normal degree of nocturnal decrease in blood pressure "dippers" is a decrease in nocturnal blood pressure of 10-20%
2. Insufficient degree of decrease in blood pressure "nondippers" - decrease in night blood pressure 0-10%
3. Increased degree of nocturnal decrease in blood pressure "overdippers" - more than 20%

4. Steady increase in nighttime blood pressure "nightpickers" - the pressure does not decrease.

SMAD methodology

- Installing the device;

-Mandatory control measurements;

-Oral instruction of the patient;

-Input of the received data into the computer with their subsequent processing using statistical and graphical methods;

-Analysis of results.

Echocardiography and Dopplerography.

Echocardiography is a method of ultrasound diagnostics aimed at studying morphological and functional changes of the heart and its valve apparatus. It is based on the capture of ultrasound signals reflected from the structures of the heart.

A sensor containing a piezoelectric ceramic crystal capable of transforming electrical energy into mechanical energy (sound) and vice versa acts both as a sound source and a receiver of reflected waves. There are three types of echocardiographic studies: M-echocardiography, two-dimensional echocardiography and Doppler study. With M-echocardiography, one sensor emits sound with a frequency of 100F—2000 pulses per 1 second along one axis. This type of echocardiography allows you to get a high-quality image in time. By changing the direction of the beam, you can scan the heart from the ventricles to the aorta and the left atrium. With two-dimensional echocardiography, by directing an ultrasonic beam along an arc of 90 ° with a frequency of about 30 times per 1 second, an image is obtained in two planes. Using various sensor location points, it

is possible to obtain a high-quality spatial image that allows analyzing the movements of heart structures in real time.

With the help of Doppler echocardiography, it is possible to determine the speed of blood flow and its turbulence. When the sound collides with moving red blood cells, the frequency of the reflected signal changes. The magnitude of this change (Doppler shift) indicates the blood flow velocity (V), which can be calculated taking into account the following characteristics of the sound beam:

$$V = \frac{C \cdot (\text{допплеровский сдвиг})}{(2 \cdot \text{излучаемая частота}) \cdot \cos\Theta},$$

где C — the speed of sound in tissues, Q — the angle between the Doppler beam and the middle axis of the blood flow.

The upward shift direction (an increase in the frequency of the reflected sound) indicates that the blood flow is directed to the sensor; the downward shift direction is from the sensor. When blood passes through the stenosed valve openings, its velocity increases, which can also be recorded using Doppler echocardiography. Then using the modified Bernoulli equation, it is possible to calculate the transvalvular pressure gradient (P): P=4V2. The registration of signals in certain small areas makes it possible to determine the spatial localization of turbulence characteristic of stenosis, valve insufficiency or blood bypass. The combination of Doppler studies with imaging methods allows you to calculate cardiac output. Unfortunately, not all patients can have echocardiography performed successfully. The penetration of sound into tissues can be difficult for many elderly people suffering from obesity and emphysema.

Нормальные величины размеров отдельных структур на М-эхокардиограмме

Измеряемый параметр	Размер, *см*	
	пределы колебаний	среднее значение
Полость правого желудочка в конце диастолы	0,9—2,6	1,7
Полость левого предсердия (в период систолы желудочков)	1,9—4	2,9

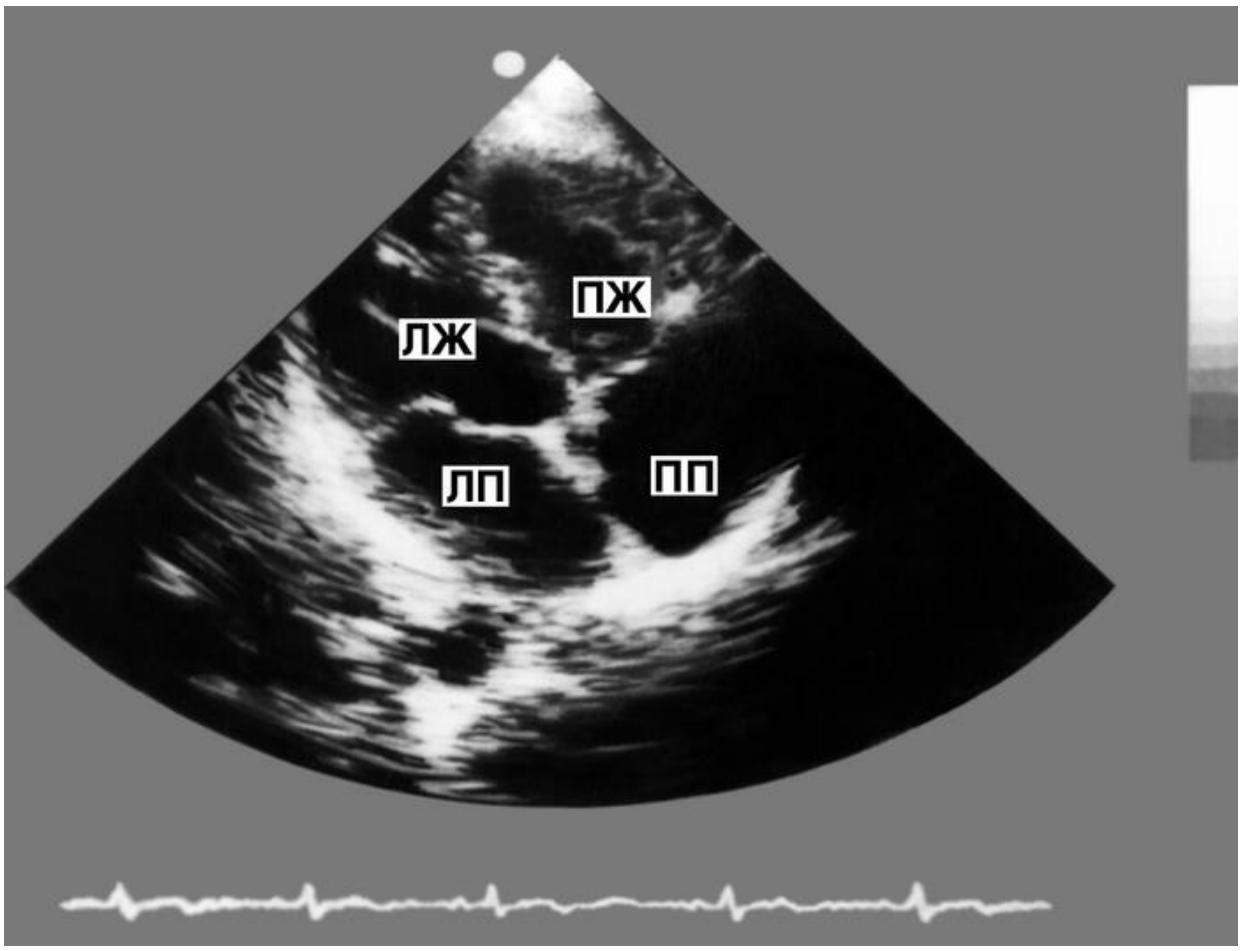
Полость левого желудочка в конце диастолы	3,5—5,7	4,7
Толщина задней стенки желудочка в конце диастолы	0,6—1,1	0,9
Амплитуда систолического движения задней стенки левого желудочка	0,9—1,4	1,2
Толщина межжелудочковой перегородки в конце диастолы	0,6—1,1	0,9
Амплитуда систолического движения межжелудочковой перегородки на уровне средней трети	0,3—0,8	0,5
на уровне верхушки сердца	0,5—1,2	0,7
Диаметр устья аорты	2,0—3,7	2,7
Сепарация створок аортального клапана	1,5—2,5	1,9

Evaluation of the contractile function of the left ventricular myocardium using echocardiography is based mainly on measuring the shock volume of the heart by the difference between the final diastolic and final systolic ventricular volumes (they are calculated using special formulas from the results of measuring the ventricular cavity), determining the ejection fraction (the ratio of shock volume to the final diastolic ventricular volume) and the rate of circular fiber shortening myocardium. The latter indicator most closely characterizes the actual contractility of the myocardium. The central hemodynamic parameters obtained by echocardiography should be interpreted with great caution in patients with the following diseases: acute myocardial infarction, postinfarction cardiosclerosis, congenital heart defects accompanied by shunts from right to left and from left to right, acquired heart defects, especially mitral and aortic.

An important characteristic of the contractile function of the myocardium is given by the study of the movements of the interventricular septum and ventricular walls; an increase in function is manifested by hyperkinesia, and a decrease is manifested by asinergia of contraction and hypokinesia of the walls of various prevalence, including in certain areas of the studied myocardium.

The ejection fraction is the ratio of UO to BWW. In most cases, it is calculated by the formula: FV = (KDO - CSR)/KDO x 100 (%), where PV is the ejection

fraction, KDO is the final diastolic volume, CSR is the final systolic volume. The normal LVF value is 55-75%.



Двухмерная эхокардиограмма из верхушечного доступа: Видны полости всех четырех камер сердца в поперечном сечении.

Radiographic examination of the heart.

The heart is most often examined in a straight (frontal) projection, in the first (right) and second (left) oblique positions and in lateral projections. A direct projection makes it possible to determine the contour of the heart. On the left, it consists of 4 arches: the uppermost is the aortic arch, then the pulmonary artery, the auricle of the left atrium and the left ventricle. On the right, the contour of the heart is formed by the arc of the aorta and the right atrium. Examination of the heart in the frontal projection allows you to determine its three positions: an oblique position, in which the angle of inclination of the heart is 43-48 °, a transverse (recumbent heart) with an angle of inclination of 49-56 ° and a median (vertical) with an angle of inclination of 35-42 °.

In the first oblique position, i.e. with the patient's right shoulder forward, the retrocardium space is well projected, i.e. the space between the shadow of the heart and blood vessels on the one hand, and the spinal column on the other. In this position, in diseases, an expansion of the ascending aorta and an increase in the left atrium can be detected.

In the left oblique position (left shoulder forward), all parts of the aorta and the so-called aortic window are visible. Changes in the arch and descending aorta, as well as enlargements of the right and left ventricles of the heart are clearly visible here. The shape of the heart changes significantly with its various diseases and lesions of large vessels. Especially large changes are observed in heart defects.

Normal picture of the heart and large vessels in the X-ray image
The contours of the vascular-cardiac shadow in the anterior position are outlined in the form of a peculiar figure, the upper-right contour of which in the area of the shadow of the vascular bundle corresponds to the superior vena cava adjacent to the ascending aorta; the lower right arc corresponds to the contour of the right atrium; further inside is the right ventricle, which, with this ray projection, does not participate in the drawing of the contour of the heart; occasionally detected as a small shadow in the corner between the right contour of the cardiac shadow and the diaphragm of the inferior vena cava. Three contour bends are formed on the left: the upper one corresponds to the aortic arch and its descending part, the next one corresponds mainly to the pulmonary artery and partly to the left atrium (auricle) and the lower one corresponds to the left ventricle. Some authors distinguish four bends or arcs on the left, corresponding to the aorta, pulmonary artery, auricle of the left atrium and left ventricle.

The shadow of the heart gives a very distinct image, but its lower contour can only rarely be differentiated from the dense shadow of the liver. With a deep breath, the lower border is visible for a slightly longer length due to the lowering of the diaphragm, and with it the liver.

The heart shadow is depicted in the form of an irregular elliptical figure, resembling most of all a chicken egg, (placed so that its narrow end faces left and down and the longitudinal diameter with a horizontal line is an angle approximately equal to 35-45 °. With a high standing of the diaphragm, which is often observed in hypersthenics, the specified angle decreases to 25-30 °, and the heart assumes a more transverse position, i.e. its longitudinal diameter approaches the horizontal line. On the contrary, in asthenics, the diaphragm is relatively low, and the tip of the heart occupies a correspondingly lower position, which is why

the angle of inclination of the heart reaches 60 ° or more. In pronounced asthenics, the heart, being relatively small in size ("small heart"), seems to hang on vessels ("hanging heart", "drip heart") and is characterized by significant mobility during changes in body position ("mobile heart").

Instrumental examination of vessels

1. Ultrasound examination of the arteries

Ultrasound technologies, being highly informative and publicly available, provide the doctor with a wide range of information about the early preclinical manifestations of atherosclerosis of the arteries. Ultrasound Dopplerography \ UZDG\ – allows you to determine the presence of pulsation, segmental blood pressure, volumetric minute blood flow, blood flow rate, assesses the condition of the vascular wall, the zones of collateral and main circulation on the segments of the limb.

Duplex ultrasound scanning with Dopplerography – significantly complements the ultrasound with a graphic image of occlusion or stenosis zones with an assessment of the characteristics of changes in the patency of the vascular bed, measuring the extent of the occluding substrate with an assessment of its density, an assessment of the diameters and condition of the vascular wall. In the presence of fistulas - allows you to estimate its size and volume of overflows.. Ultrasound of the vessels allows you to determine the zones of occlusion and stenosis of the main arteries.

In clinical and scientific practice, methods for determining the thickness of the intima-media complex of the carotid artery (TIM), detecting atherosclerotic plaques (ACB) in the carotid arteries and determining the ankle pressure index (LEAD) are most in demand.

Thickening of the intima-media complex according to duplex scanning of vessels.

During ultrasound examination in a healthy person, the intima-media complex is a two-layer structure with a hyperechoic layer adjacent to the lumen and a hypo-echogenic layer. Measuring the layers of intimacy and media separately with the

help of modern instrumental technologies is impossible. When the intima-media complex thickens, the differentiation of layers disappears in its image, heterogeneity and surface roughness appear. In order to obtain reliable TIM measurement results, a number of provisions must be observed. Numerous studies have shown an increase in TIM with age. In healthy individuals under 30 years of age, the TIM is 0.52± 0.04 mm, from 30 to 40 years – 0.56±0.02 mm, from 40 to 50 years – 0.60± 0.04 mm, older than 50 years – 0.67± 0.03 mm.

TIM values greater than or equal to 75 percentiles for their gender-age group are determined to be significantly high and predict an increase in the risk of developing CVD in a patient of this gender and age. Values between 75 and 25 percentiles are average and do not change the risk assessment of CVD according to the Framingham scale. Values less than or equal to 25 percentiles correspond to a low risk of developing CVD.

The important role of detecting atherosclerotic plaques in the carotid arteries is due to the fact that their presence is associated with a higher cardiovascular risk compared to a diffuse increase in TIM in the carotid arteries. Perhaps this fact is explained by the fact that, according to JD Spence et al., the ACB area increases 2.4 times faster than the TIM increases.

When examining the carotid arteries, the structure protruding into the lumen of the artery is described as an atherosclerotic plaque if its height is 0.5 mm or 50%

One of the methods of diagnosis and assessment of the severity of atherosclerosis of the arteries of the lower extremities is the measurement of regional arterial pressure in the arteries of the lower extremities with an assessment of the LPID, which is calculated as the ratio of pressure on the ankle and pressure on the shoulder, determined by ultrasound. In a healthy person, the LDL ranges from 0.9 to 1.3. With a decrease in this parameter of less than 0.9, the presence of hemodynamically significant pathology of the arteries of the lower extremities should be assumed. In recent years, there have been reports in the literature that the

ankle-shoulder pressure index of less than 0.9 is an independent factor predicting the development of CVD (unstable angina, non-fatal heart attack, "cardiac" death).

2. MRI with contrast is the most informative of the available non–invasive methods of investigation. Allows you to assess the nature of changes in the vascular wall, the degree of narrowing, the extent of occlusion, minute blood flow, volumetric blood filling of the limb, gives a three-dimensional image from different angles, etc.д.

3. Angiography remains the main method of lifetime diagnostics of morphological changes in blood vessels, is widely used to diagnose diseases of all organs and systems, allows a comprehensive assessment of blood supply to several organs. It is always recommended when preparing for operations on the cardiovascular system and in doubtful cases when other diagnostic methods do not allow an accurate diagnosis or for differential diagnosis.

Coronary angiography (CAG) - x-ray method of imaging of coronary arteries with selective administration of a contrast agent at the mouth of the coronary artery. The purpose of CAG is to accurately determine the anatomy of the arteries of the heart, down to the smallest branches, as well as to identify pathological changes. The information obtained during the study includes: establishing the anatomical type of blood supply, the length and diameter of the coronary arteries, assessing the degree of their narrowing, identifying X-ray morphological features of narrowing (type of atherosclerotic plaque, presence of parietal thrombosis or plaque rupture, calcium deposits, artery spasm in the affected segments), evaluation of coronary blood flow. In addition, the study determines the presence and severity of collateral blood supply.

Although serious consequences may occur during diagnostic CAH, their risk is low. According to the Society of Cardiac Angiography and Invasive Interventions of the USA, the incidence of complications during diagnostic CAH does not exceed 2%.

More severe complications occur more often in patients with an initially high risk. The most unfavorable factor that increases the risk of complications is the instability of the clinical condition. The frequency of complications of CAH is higher when conducting an emergency study.

Angiographic diagnostics can be combined with manipulations performed after angiography – balloon dilation of the arteries – angioplasty, closed commissurotomy, stenting of the arteries, intracoronary thrombolysis and other intravascular procedures.

ультразвуковой метод исследования, позволяющий получить двухмерное изображение сосудов с возможностью оценки состояния сосудистой стенки, характера и скорости кровотока по ним. Проще говоря, дуплексное сканирование позволяет диагносту увидеть исследуемые сосуды, оценить места их сужений, закупорок или, напротив, расширенные участки, а также определить наличие в них тромбов, атеросклеротических бляшек, нарушений кровотока.