Electrocardiography

ELECTROCARDIOGRAPHY
A manual

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The manual covers the most important aspects of theory of the
electrocardiography and formation of the mechanisms of the elect-
rocardiogram, the principles of its interpretation when there is
hypertrophy of the heart chambers, arrhythmias and conduction disorders,
coronary heart disease, and other diseases and pathological conditions.
For undergraduate students of the higher medical institutes, pre-
graduate interns, doctors-interns specializing in therapy and recent
graduate practitioners.

UDC 616.12-073.97(035)

2

PREFACE

Despite the development of costly and complex methods of research in cardiology (such as echocardiography, single photon emission computed tomography or positron emission tomography scan) the electrocardiography remains the most affordable and reliable instrumental method for the diagnosis of a number of diseases of the cardiovascular system. It is the ECG but not the MB-fraction of creatine kinase or echocardiography that dictates the need for rapid and radical thrombolytic therapy. There is no other method that would compete with the ECG in the diagnosis of arrhythmia, which is so prevalent problem in the cardiologic clinic. It should be noted that the diagnosis of hypertrophy of the heart chambers, the diagnosis of myocardial ischemia and other diseases can be confirmed only with the data of the ECG.

HISTORY OF THE METHOD

Although the electrocardiogram as a diagnostic method for heart condition in clinical medicine has been used for over 100 years, but to this day it continues to amaze its contemporaries with its new capacities. For the first time it was an English scientist Augustus D. Waller who registered electromotive force of the heart from the surface of the human body (with the help of a bulky capillary electrometer of Lippmann) in 1887, in fact, almost 100 years after the discovery of "animal electricity" by L. Galvani. However, before clinical application of this technique, it took 15 years. Lippmann capillary electrometers represented the recording of the electromotive force of the heart that was difficult to analyze. Only in 1903 William Einthoven, a Professor of physiology at the University of Leiden received a recording of the electrical currents of the heart in a clinical setting, similar to modern-day performance by means of the string galvanometer of Adler, built on the principle of the apparatus for the reception of transatlantic telegrams. W. Einthoven, the inventor of the technique gave it the name of electrocardiography, and the device that recorded the currents of the heart, was called an electrocardiograph (Fig. 1). In 1907–1908 W. Einthoven created the basics of analysis of the electrocardiogram resulting from outstanding discoveries, which today helps to understand the foundation of this unique method of study of the heart. For the contribution to physiological science of the heart and clinical cardiology W. Einthoven was awarded the Nobel Prize in 1924. This scientist developed the system of standard leads, without which there is no modern electrocardiogram.
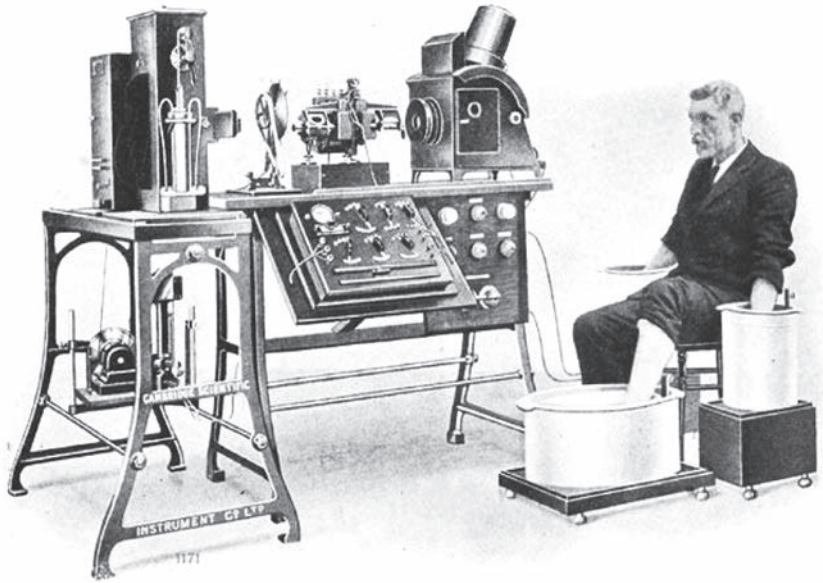
Fig. 1. String galvanometer, invented by W. Einthoven

In 1934, the American scientist F. N. Wilson using the technique of the so-called unipolar leads implemented a system of six chest leads, which greatly expanded the possibilities of using the electrocardiographic method for determining physiological characteristics of a child's heart, and to diagnose diseases of the heart muscle. In 1942, the American scientist E. Goldberger created an original system of reinforced leads from the limbs, which was also included in any standard electrocardiographic studies.

Thus, for 70 years the standard electrocardiogram includes the recording of the electromotive force of the heart in twelve leads, six of which are the leads of the frontal plane: three standard leads according to W. Einthoven (I, II, III) and three unipolar enhanced leads from the limbs by E. Goldberger (aVR, aVL, aVF); six chest leads of the horizontal plane by F. N.Wilson (V1-V6).

Great contributions to the development of clinical electrocardiography were made by the Austrian scientist K. F. Wenckebach and the German scientist W. Mobitz.

In Russia the first work on electrocardiography was published by A. F. Samoilov, the Professor of Kazan University (1908).

ELECTROPHYSIOLOGICAL BASICS
OF ELECTROCARDIOGRAPHY

The function of the heart as an organ responsible for moving blood in the human body includes:

1. Automatism — i. e. the initiation of the pulse, or the ability of the heart to spontaneous diastolic depolarization;

2. Conduction — impulse conduction to the working myocardium;

3. Excitability — ability to excitation;

4. Refractory — i. e., the impossibility of excitation under certain circumstances;

5. Contractibility — a function, which ensures blood flow in the human body.

The electrocardiogram allows us to estimate all the functions of the heart, except for the evaluation of myocardial contractility.

The heart as a tissue structure comprises:

• cells of the working myocardium, contraction of which leads to the ejection of blood from ventricles of the heart; endothelial cells, fibrous tissue;

• connective tissue cells;

• cells of the conduction system (there can be several types: R-cells, carrying out the function of automaticity; cells of Purkinje, forming fibers, which conduct impulse; transitional T-cells, which are located between R-cells and Purkinje cells) (Fig. 2);

• secretory cells, located mainly in the right atrium and produce Na-uretic peptide that is involved in the regulation of acid-base balance, and blood pressure.

In relation to the electrophysiology of the myocardial cells there are typically three alternating conditions: rest or polarization, excitation or depolarization, restoration of the resting potential or repolarization. Each of them is associated with rhythmic recharge of intra- and extracellular environment as a result of the cross-membrane migration of ions K+, Na+, CA++, and CL-. Being strictly regulated, it creates specific ionic basis of transmembrane potential in different phases of the evolution of electric cells (transmembrane potential is measured in millivolt — current between the outer and inner sides of the cellular membranes that are always opposite in sign charge).

Since in practice the recordings of the heart currents are accomplished from the surface of the body only those electrical phenomena that arise on the outside of the membranes of myocardiocytes are accessible. They interest us in the first place.
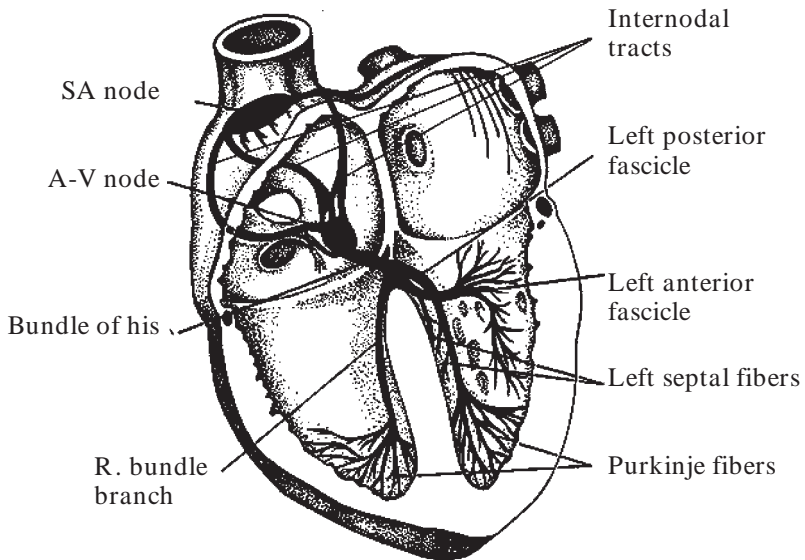
Fig. 2. Conduction system of the heart

At rest all cell membranes are **polarized** so that their outer side, and therefore, the surface of single muscle fibers and myocardium on the whole are **positively** charged, i. e. the potential difference as a precondition for the appearance of current is absent.

Depolarization or activation of the cells under the influence of the electric impulse leads to the recharge of the membranes: the outer side of the excited area (cells, fibers, entire myocardium) acquires a **negative** charge (Fig. 3). Its appearance and rapid spread, accompanied by the neutralization of the positive charge of rest, creates a potential difference and generates an **electromotive force** (EMF) — depolarization current ("minus driving a plus in front of it"). At the end of the depolarization, the potential difference disappears, since the entire surface of the myocardium becomes **electropositive** (Fig. 4).

The essence of **repolarization** is the recovery of the initial potential (rest potential) and readiness for the next excitation, i. e. in restoring the positive charge of the external side of the cell membranes. The gradual substitution of the negative charge re-creates the EMF — this time a **current of repolarization** ("a plus driving minus in front of it").
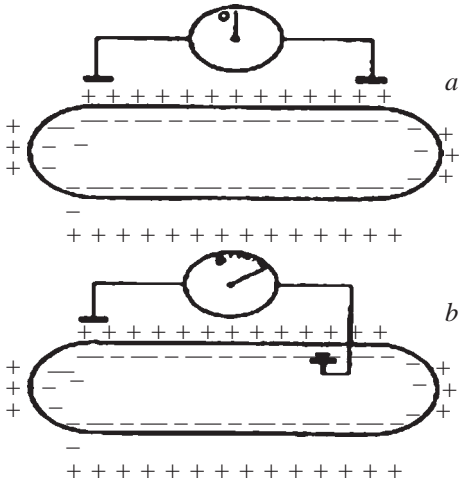
a

b

Fig. 3. Transmembrane cell potential at rest and during depolarization: *a)* cell is polarized, rest state; *b)* depolarization, the cell is "active"
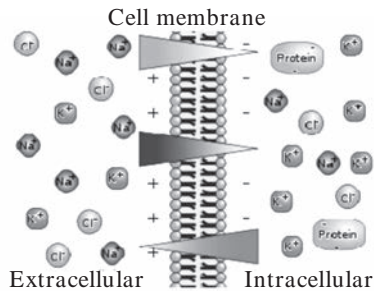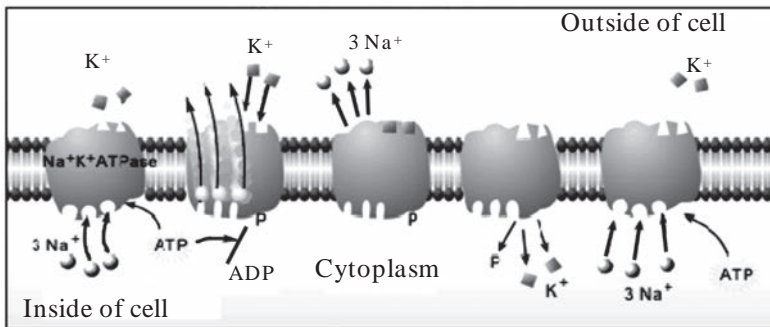
Active Transport System

Fig. 4. The movement of ions across the cell membrane and the formation of the transmembrane potential

Taking into account the laws of physics, de- and repolarization constitute typical examples of the dipole, which implies the coexistence and movement of two equal in magnitude, but opposite sign charges, located at an infinitely small distance from each other.

Under the influence of the excitation impulse countless macrodipoles of single muscle fibers — the basic sources of the EMF in the heart begin to function. Stacking, they form increasingly bigger macrodipoles of the separate fragments of the myocardium, heart chambers and eventually form a single cardiac dipole and the EMF of the heart.

Specialized, the so-called pacemaker cells (PCs) of the cardiac conduction system have the ability to generate electrical impulse. The ability to self-activation known as **automatism**, fundamentally distinguishes them from the contractile cells of the myocardium. The latter, having **excitability,** are activated only under the influence of impulses, coming from the PC.

The highest automatism is peculiar to the sinoatrial node (SA node), which suppressing lower automatic potencies of the downstream PC normally acts as a pacemaker or center of automatism of I order. Downstream PCs in the atria, atrioventricular connection (AV connection) and the ventricles act as passive conductors of excitation. In the physiological sense, they are reserve (emergency) sources of impulse formation, or centers of automatism of II and III order.

Starting in the SA node, the excitation impulse activates first the right, then the left atrium, and after a short delay in the AV connection through the system of His is transmitted to the ventricles. On their territory the interventricular septum gets depolarized first, and its parts facing the left ventricle are the first to receive a negative charge. Consequently, the excitation covers the septum from left to right. Next, the electrical impulse reaches the walls of the ventricles. Their depolarization starts at the inside of the upper subendocardial region, where there are branched terminals of the conduction system — Purkinje fibers and spreads to the epicardium (Fig. 5). Thus, the excitation of the walls of the ventricles occurs in the direction from the inside outwards.

On the whole, there is a general trend of depolarization of the myocardium, from top to bottom and from right to left. After depolarization, the final of which is the contraction of the ventricles, the process of repolarization begins (Fig. 6).

It is clear from the above said that the EMF that occurs in the activation of the heart, is not only characterized by the quantitative value of voltage, but also a direction, i. e. it is a **vector quantity**. It is known from physics that the vector is represented by a straight arrow. If we speak about the
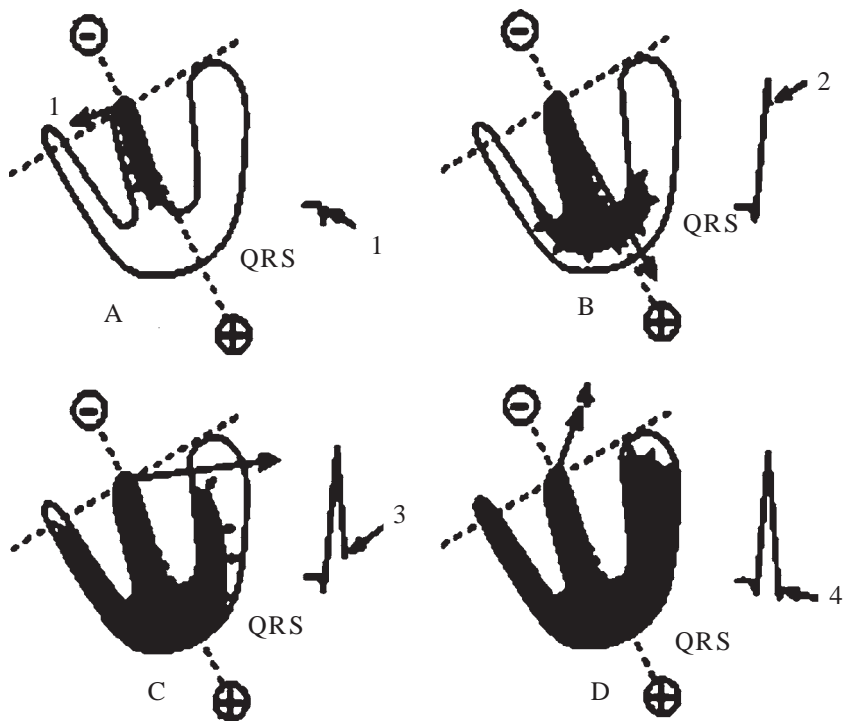
Fig. 5. Phases of depolarization of the ventricle

electric vector, the base of the arrow corresponds to the negative pole of the dipole, and the top — positive. The length of the arrow expresses the quantitative value of the EMF (Fig. 7). In future we will most often operate with the concepts of "current vector" or "EMF vector".

The heart axis indicates the average direction of the depolarization wave. A normal heart axis, the picture shows an example, is between -30 and +90 degrees. In this example, the heart axis is +45 degrees.

Strict sequence of the electrical cycle is recorded on the ECG by a series of waves, which are denoted by the Latin letter P, QR, S, T and U. The waves R and T can be directed both upwards and downwards; the R wave — only upwards and the waves Q and S — only downwards and the first precedes R, and the second follows it (Fig. 8).

Each wave of the ECG, with the exception of the U wave, carries very specific information. The R wave reflects the depolarization of the atria, Q — the interventricular septum, R and S — walls of the ventricles. The
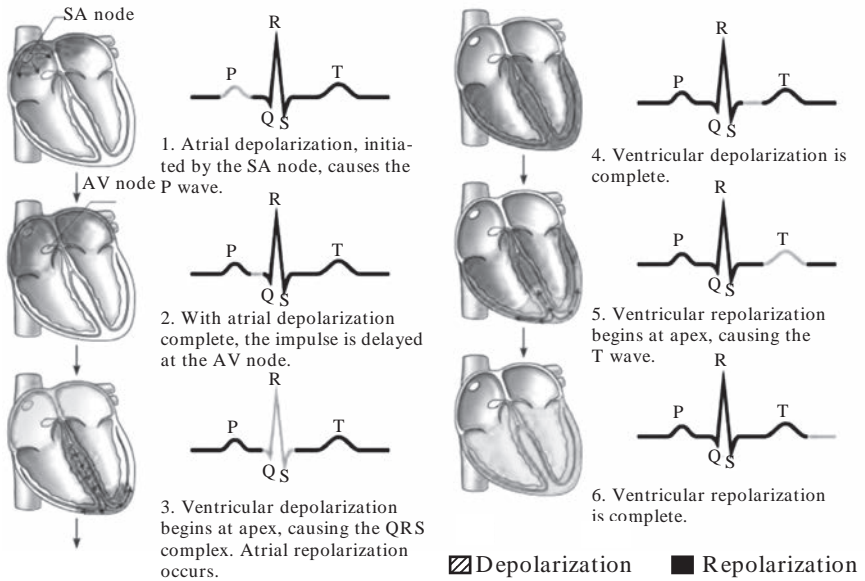
1. Atrial depolarization, initiated by the SA node, causes the P wave.

2. With atrial depolarization complete, the impulse is delayed at the AV node.

3. Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.

4. Ventricular depolarization is complete.

5. Ventricular repolarization begins at apex, causing the T wave.

6. Ventricular repolarization is complete.

🔲 Depolarization   ■ Repolarization

Fig. 6. The process of depolarization and repolarization of the heart chambers
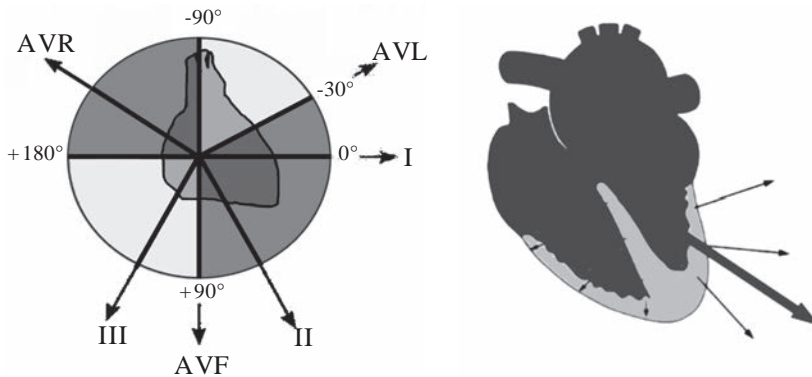
Fig. 7. Formation and the projection of the vector EMF in different planes

10
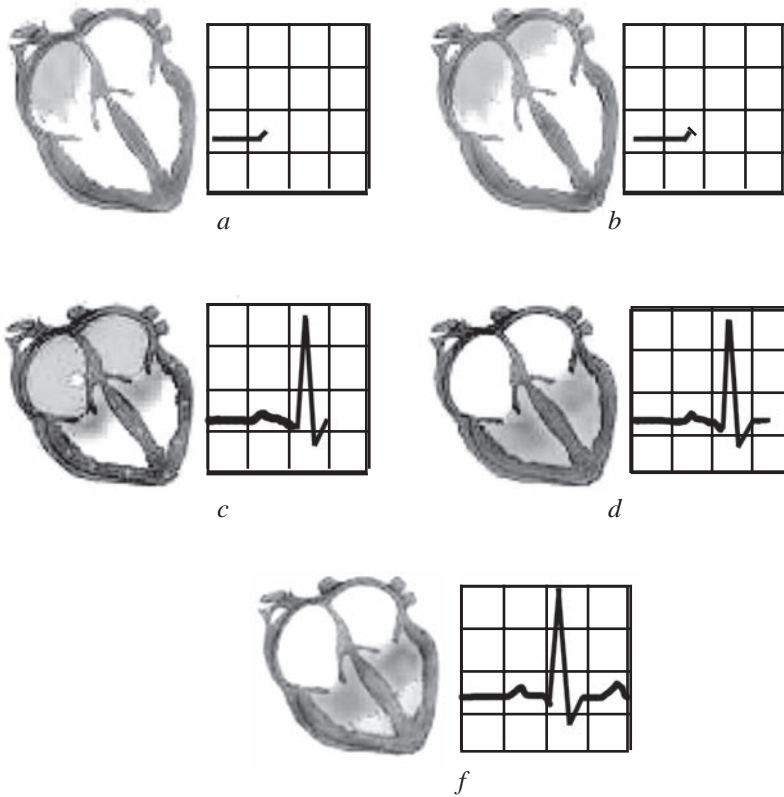
Fig. 8. The formation of the electrocardiographic complexes:
a — depolarization of the atria and formation of the P wave; *b* — formation
of a segment of P(Q)R; *c* — repolarization of the atria; *d* — depolarization
of the ventricles with formation of QRS; *f* — phase of the ventricular repo-
larization, and the formation of the segment S-T

segment of the straight line before the start of the T wave corresponds to
the period of complete ventricular excitation, and the T wave — the phase
of repolarization (repolarization of the atria is not reflected on the ECG
as the time superimposed on the depolarization of the ventricles is the
QRS complex).

Since the repolarization proceeds much more slowly than dep-
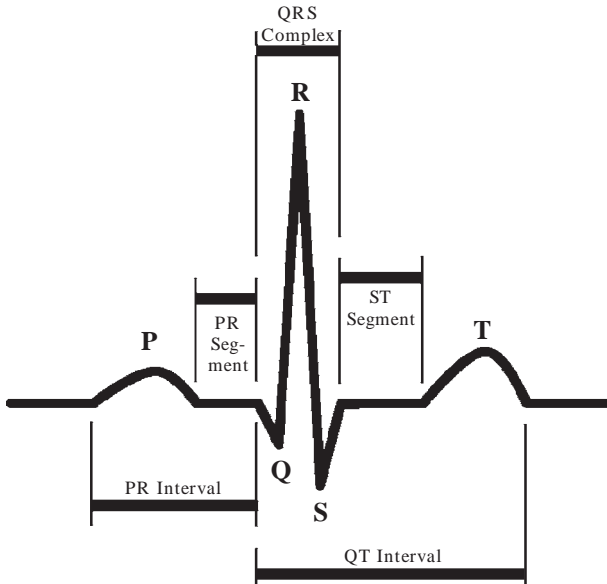olarization, the T wave differs from the narrow and pointed waves Q, R

Fig. 9. Basic components of the ECG

and S by rounded top and greater width. The U wave is rarely seen. Its origin and clinical significance is not clear. A tetrad of the waves Q, R, S and T is called a ventricular complex; there are initial (QRS) and end (ST and T) parts (Fig. 9).

THE PRINCIPLES OF THE LEADS
OF THE HEART CURRENTS
WITH THE POLARITY OF THE BODY

With an assumption, the heart can be regarded as a point-like source of the current — a single cardiac dipole creating an electric field in the environment (body).

The illustration shows a spatial QRS vector QRSsp and a spatial T vector Tsp projected onto the frontal plane to give a frontal plane T axis denoted Tf and a frontal plane QRS axis denoted QRSf. The angle between the frontal plane QRS and T vectors is denoted with 'a' whereas the shaded angle between the spatial QRS and T vectors is denoted with 'b'. QRSf and Tf are derived, in this case, from spatial vectors whereas the frontal plane QRS and T axes are usually derived

from the limb leads of the 12-lead electrocardiogram. However, the illustration shows how there potentially can be large differences between the spatial QRS-T angle and the frontal plane QRS-T angle. The axes are labeled I, aVF, and V2 for ease of linking the frontal plane vectors with the concept of spatial vectors, which are normally derived from XYZ leads.

In the frontal plane spatial depiction of the EMF of the heart or a single heart dipole is a resultant vector of depolarization — the result of an algebraic sum of a plurality of different vectors of the EMF elemental macrodipoles, which are single muscle fiber. Schematically the formation of the resultant vector can be represented in the form of a gradual process. In the course of continuous algebraic addition from the EMF of microdipoles, the more aggregated excitation vector EMF of separate parts of the myocardium, septum, right and left ventricles are formed and finally the resultant depolarization vector — a graphic symbol of a single cardiac dipole (Fig. 10).

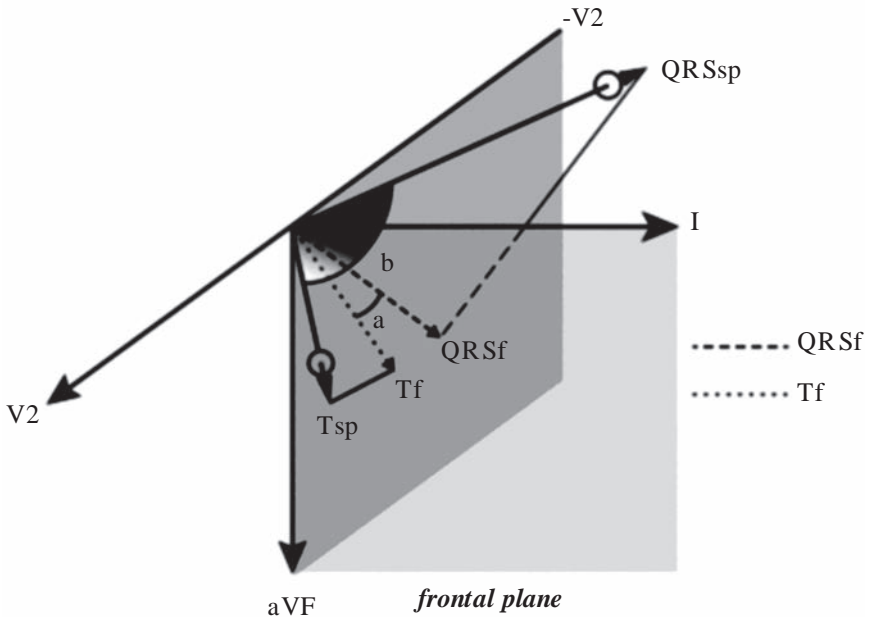The resultant vector of depolarization is called the electric heart axis (EHA).

Fig. 10. Spatial depiction of the EMF in the frontal plane

Normal electrical axis of the heart is approximately at an angle of 60° to a horizontal line drawn through conventional electrical center of the heart. It almost coincides with the direction of the anatomical axis and like it (or after it) can take a more horizontal or more vertical position.

The line of a zero potential divides the electric field of the heart and thus the body becomes negatively and half positively charged. The first is located to the right of the zero line, the second to the left of it.

The ECG can be recorded with a galvanometer by connecting any pair of points of the body, bearing unequal charges. In practice those are used that are convenient to place the electrodes and provide the greatest potential difference. These are the right hand, left hand and left leg.

It is easy to conclude that the right hand is in the field of a negative charge, and the left hand and left foot are in the positive field. This is the natural polarity of the body, which is duplicated by electrodes connected only with the anode (+) or only with the cathode (-) of the recording device depending on the location of their overlap. The theory of the cardiac dipole, taking into account the polarity of the body, is most fully represented by the concept of the equilateral triangle of W. Einthoven. It formed the basis of the first ECG (Fig. 11).

When applying the electrodes on the hands it does not matter whether they are mounted on the wrist, forearm or shoulder. The decisive factor is the polarity of the body at the place of origin of the limbs from the body. By itself, the hand (and foot) only functions as a passive conductor of excitation (like the wire).

EQUIPMENT AND TECHNIQUE OF
THE ECG REGISTRATION

The ECG is recorded using a special instrument, called an electrocardiograph, which converts the electrical signal in mechanical one. The difference in bioelectric potentials that arise in the excitation of the cardiac muscle is seen with electrodes placed on the body of the subject. The electrode arrangement may be different. Special arrangement of electrodes is called leads. The ECG leads are of two- and single-pole. Bipolar leads register difference of potentials between two points of the body, single-pole — reflect the difference of the biopotentials of any part of the body and the potential, constant in magnitude, conditionally assumed to be zero. To create a zero potential, we use the combined F. Wilson (indifferent) electrode formed by connecting the wires (through the additional resistance) of the three limbs (right arm, left arm and left leg), or

Standard Limb & Precordial Leads
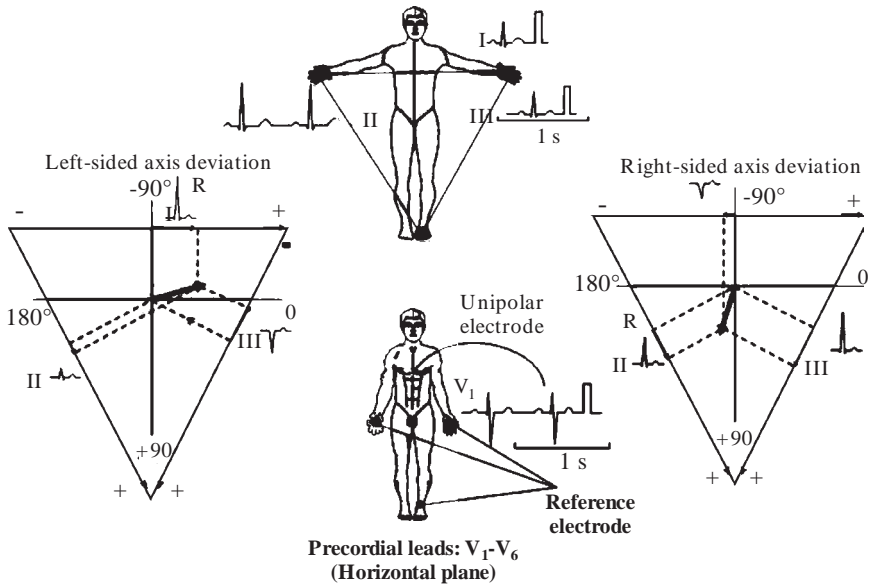Einthovens triangle (frontal plane)

Fig. 11. Formation and direction of the EHA in the frontal plane on the basis of the concept leads after Einthoven

E. Goldberger's combined electrode, from which the ECG is removed (enhanced lead from the extremity).

When registering the ECG, 12 leads are mandatory:

3 standard bipolar leads proposed by W. Einthoven (1898);

3 reinforced unipolar leads from the limb, as proposed by E. Goldberger (1942);

6 thoracic unipolar leads proposed by F. Wilson (1934).

All electrodes are color-coded: when applied to the limbs red one is put on the right arm, yellow — on left arm, green — on left leg, black (ground of the patient) — on the right leg.

To apply the chest electrode a special rubber bulb or other locking electrode fixture is used.

To register a standard bipolar leads (Fig. 12) the electrodes are applied to the limbs with their connection to the electrocardiograph. I lead — right arm (-) and left arm (+); II lead-right arm (-) and left leg (+); III lead — left hand (-) and left leg (+).

15

EINTHOVEN'S TRIANGLE
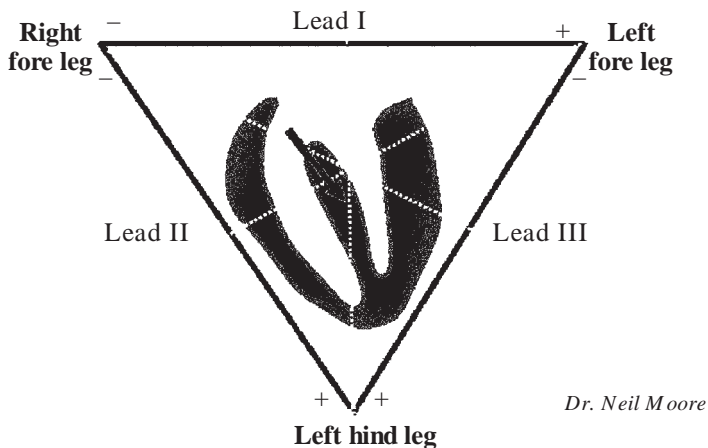
Fig. 12. Standard leads I, II, III

Any lead registers the dynamics of the EMF of all the chambers and walls of the heart simultaneously. But, nevertheless, each lead has its priorities. Finally, active electrode captures most sensitively, completely and accurately the biopotentials of those parts of the myocardium that face it directly. Topical diagnosis of the heart affections is based on this principle.

It should be noted that **I** lead registers more precisely a potential change of the lateral wall of the left ventricle with the exception of its high parts. **II** lead reflects the state of the myocardium of the left ventricle along the longitudinal axis and therefore has no independent diagnostic value. As "a lead — witness" it only confirms the ECG changes that can be detected either in **I** or in **III** lead. **III** lead describes the bioelectric activity of the right ventricle and posterior — lateral (lower) parts of the left ventricle.

Bipolar leads could not meet the needs of the clinic, because too large area of the myocardium remained out of their reach. Clearly there was a problem of perfection of the system of electrode application.

Reinforced unipolar leads of the limbs, denoted by the abbreviation **aV***, were introduced by E. Goldberger in the practice of electrocardiography in the early 40s. Three of them: **aVR** — a lead from the right hand, **aVL** — a lead from the left hand, **aVF** — a lead from the left leg (**a** — the first letter of the English word augmented —

16

amplified; **V** — the universal symbol of the potential from the English "voltage", **R, L** and **F** — initial letter of the English words — right, left and foot).

As bipolar leads, they investigate the EMF of the heart in the frontal plane. A positive electrode mounted on the respective limbs; the function of the negative electrode performs the so-called joint electrode of Goldberger. It joins two other limbs, **making it almost indifferent** (Fig. 13).

AVR lead like the lead **II** "looks through" the whole myocardium in length. Their axes are in the neighborhood, but in **aVR** the resultant vector of the heart EMF, in contrast to II lead, is directed from the active electrode. Taking into account the opposite polarity, the lead **AVR is almost a mirror reflection of the lead II** — the waves have the opposite direction of the isoline on the ECG.

The lead **aVL** reflects fluctuations in the potential of the lateral wall of the left ventricle.

*The lead **aVF**, and **III** lead* equally characterizes the electrical activity of the right ventricle, and lower (lateral-posterior) parts of the left ventricle.

*By analogy with the **aVR**, leads **aVF** and **aVL** are also in constant relationship with standard leads: **aVL** like **I** lead, aVF — **III**.*

AUGMENTED LIMB LEADS (aVR, aVL, aVF)
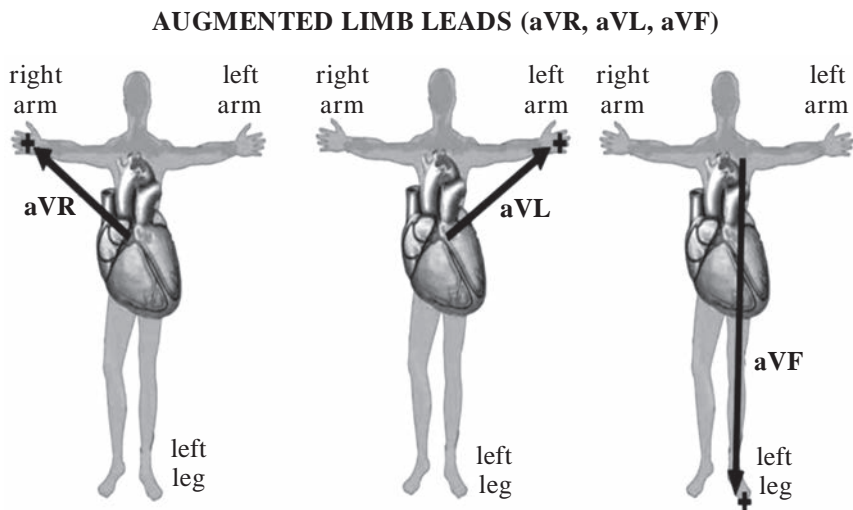
Fig. 13. Enhanced leads from the limb (aVR, aVL, aVF)

It should be noted that the introduction of the unipolar leads from the limbs allowed expansion of the diagnostic capabilities of the ECG. The affection of the high parts of the lateral wall of the left ventricle was not recognized prior to the introduction of the lead aVL into practice, in particular a lateral infarction because this area remained "out of sight" of **I** lead. The lead **aVF** performs the function of a kind of arbiter that allows eliminating the ambiguity of encountered deviations, which are sometimes fixed in **III** lead. It is related to the waves Q and T. If in aVF there is correction or normalization in some cases of the Q wave, in other — T wave, changes in the lead III are not a sign of pathology and may be attributed to constitutional or other noncardiac causes. If the lead **aVF** confirms the changes, which are recorded in **III** lead, their pathological nature is undoubtful.

Chest unipolar leads register difference of biopotentials between the positive active electrode located in certain points on the surface of the chest, and the combined, negative, indifferent electrode of Wilson. Chest electrode is sequentially fixed in the following positions:

V1 — in the fourth intercostal space at the right edge of the sternum;

V2 — in the fourth intercostal space at the left edge of the sternum;

V3 — at the level of the 4th rib on the left parasternal line (between V2 and V4);

V4 — in the 5th intercostal space along the left midclavicular line;

V5 — in the 5th intercostal space at the left anterior axillary line;

V6 — in the 5th intercostal space along the left mid-axillary line (Fig. 14).
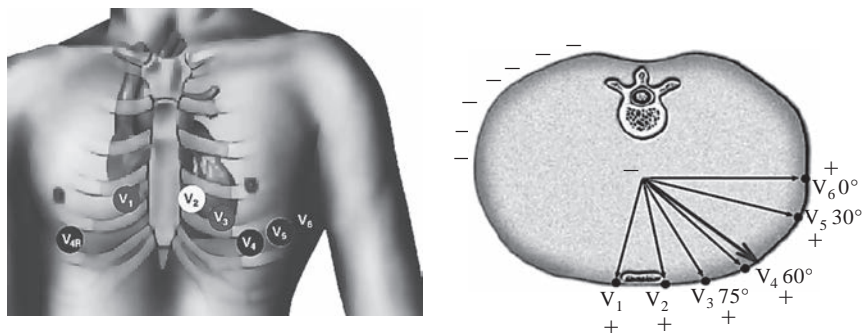

Fig. 14. The application of the electrodes by Wilson and the projection to a horizontal plane

If necessary to diagnose focal changes of the myocardium in the posterior-basal parts of the left ventricle it is also possible to register three more chests, additional leads by applying the active electrode at the level of the 5th intercostals space over the back axillary (V7), left scapular (V8) and left paravertebral lines (V9).

 Serial record of all the leads is carried out by rotation of the handle switch of the leads, mounted in the panel of the electrocardiograph.

For the diagnosis of disorders in the basal parts of the ventricles (ischemia, necrosis) as well as the enlargement of the heart downward to the 6th and even 7th intercostal space chest electrodes can be positioned on the same lines, but for the 1st or 2nd intercostal spaces above or below the conventional levels, with the obligatory indication of the numbers of the intercostal space at the top indicating the lead. For example, $V1^2$, $V2^2$ means that the electrodes are arranged in the first and second positions in the second intercostal space.

In dextrocardia the chest electrodes are placed on the right half of the chest in sequence from V1 to V6 (they are marked V1R-V6R). Accordingly, electrodes are applied to the limbs.

For the diagnosis of right ventricular hypertrophy the leads V3R-V6R can be used, thus obtaining additional ECG information.

In 1938 V. Neb proposed to take three chest bipolar leads: **D** (Dorsalis), **A** (Anterior) and **I** (Inferior). The electrodes for registering the standard leads are used, but with their location on the chest: in the second intercostal space at the right edge of the sternum, the red electrode (right hand), left posterior axillary line at the level of the 5th intercostal space — yellow (left hand) and in the 5th intercostal space along the left midclavicular line — green (left foot). Accordingly I standard lead by the method of Neb registers the lead **D**, respectively II — the lead **A**, respectively III — the lead **I** (Fig. 15, *a*). The application of this technique facilitates the identification of hypertrophy of the ventricles of the heart, disorders of the coronary circulation in the posteriordiaphragm part of the heart, and is useful when carrying out functional tests with physical activity.

The technique of recording four chest leads by Slopack-Partille (S1, S2, S3, S4) that represent the modified lead D is considered more informative for the detection of focal changes in the basal-lateral region of the left ventricle. The electrode from the left hand (yellow) is fixed in the chest position V7, and from the right hand (red) — is moved sequentially along the connecting line between two positions: in the 2nd intercostal space on the left edge of the sternum (S1) and in the 2nd intercostal space on the anterior axillary line (S4). The positions of the electrodes S2 and S3 are situated at an equal distance between the positions
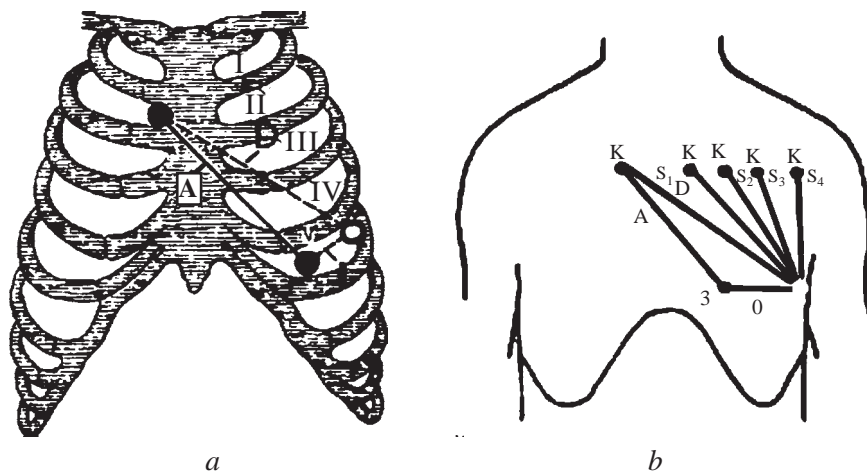
Fig. 15: a) diagram of the leads by Neb; *b)* scheme of the leads by Slopack-Partille and Neb

S1 and S4. When recording these leads the switch on the electrocardiograph is installed in the position 1 (Fig. 15, *b*).

WAVES, INTERVALS AND SEGMENTS
OF THE NORMAL ECG

The ECG recording is performed on the paper with millimeter divisions, both vertically and horizontally that allows us to estimate the amplitude parameters of the main waves (1 mm vertically is equal to 0.1 mV) as well as timing characteristics (width of the wave as well as the duration of segments and intervals). With recording speed of 25 cm/s 1 mm horizontally the duration is equal to 0.04 s, and at 50 cm/s — to 0.02 s. The waves are designated by Latin letters P, Q, R, S, T, U. The waves directed upward are considered positive (+) and downward — negative (-). The voltage (amplitude) of the waves is determined by the displacement from the zero (isoelectric) line and expressed in millimeters or millivolt. The duration (width) of the waves and intervals measured on the isoelectric line (isoline) and is expressed in seconds. The isoelectric line is determined at the level of the electrical diastole, when the heart EMF is zero and corresponds to the segment T-R on the ECG (Fig. 16).

The wave P reflects the excitation (depolarization) of the atria. Normally, the P wave in adults is always positive in I, II, aVF, V2-V6 leads, and the aVR lead is negative. In the leads III, aVL, V, the P wave can be positive, negative, biphasic, flattened (isoelectric). The amplitude (height) of P does not exceed 2–2.5 mm, and the width (duration) — 0.10–0.11 s.

The interval P-Q (from beginning of the P wave to the beginning of the Q wave or in its absence before the beginning of the R wave) represents conduction time of the impulse from the atria to the ventricles via the AV node, His bundle and its branches. Normally, the length of the interval P-Q varies from 0.12 to 0.20 s and depends on the heart rate. The ratio of the duration of the P wave to the length of the segment P-Q (index of Macros) is 1.1 to 1.6.
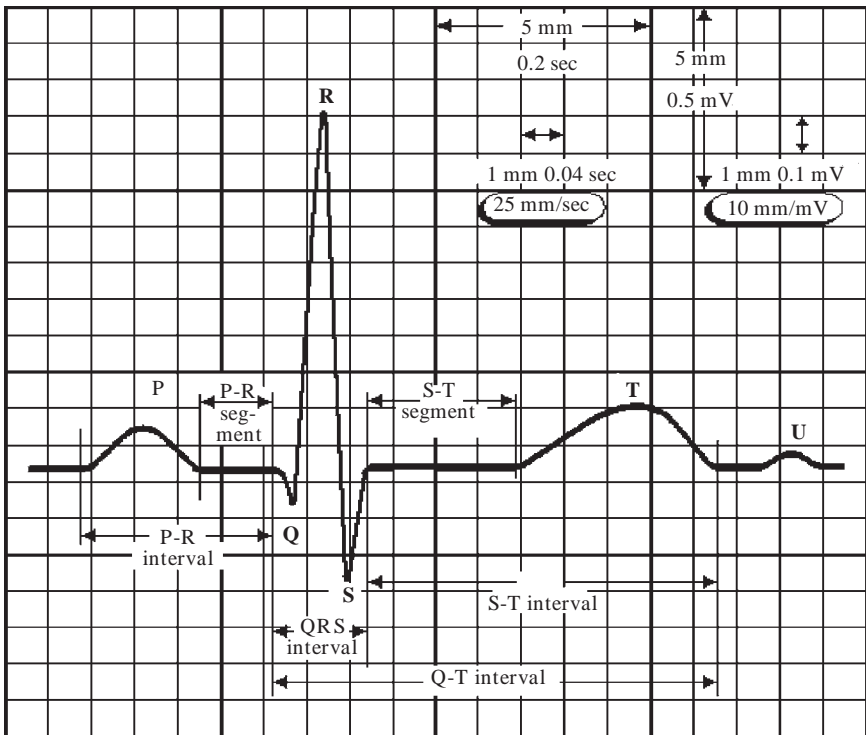
Fig. 16. Basic parameters of the normal ECG

The Q wave reflects excitation of the interventricular septum, its amplitude normally does not exceed 1/4 amplitude of the wave R of the corresponding lead, by width — not more than 0.03 s. The Q wave is the first negative wave of the QRS complex preceding the wave R. The R wave is the first positive wave of the QRS complex. The Q wave, non-permanent wave, may be registered in all three standard leads, but more often in one or two (I and II or II and III). In III standard lead in hypersthenic, obese people it can be deep and wide (more than common value), followed by the small r wave, or even the type QS (the wave of Purdy). However, the depth and duration of the Q wave (as a kind of norm) at III standard lead taken at the height of inspiration, typically decreases (positional Q), and is not changed in pathological cases. In the leads aVR and V1 the deep and wide Q wave or QS are also often recorded. In the leads aVL, aVF, V4-V6 normal or of smaller amplitude and duration waves Q are usually recorded. In the leads V2 and V3 the Q wave is normally absent.

The R wave reflects excitation of the apex and walls of the ventricles, the amplitude ranges from 5 to 26 mm, and the width is 0.03–0.06 s. This is the permanent wave of the QRS complex. However, in III standard lead it may be absent in individuals of hypersthenic constitution as well as in the aVR and V1 leads, where you can also register the small wave r: early (before the Q wave) and late (after the Q wave). Both early and late wave r can rarely be in the lead aVR, in such cases the later wave is denoted by r'. In the chest leads the R wave amplitude gradually increases from V1-2 to V4, and then decreases a little.

The wave S reflects the process of excitation of the base of the ventricles, its width ranges from 0.03 to 0.06 sec, and the depth does not exceed 1/3 of the amplitude of the R wave of the corresponding standard leads and 20 mm in the chest leads (V1), gradually decreasing to V4. In the leads V5, V6 the wave S has low amplitude or it may even be absent. The approximate equality of the wave R and S in the chest leads ("transition zone") is usually in V3 or V4 (Fig. 17). The waves Q, R and S are the initial part of the ventricular complex, equal in duration to 0.06–0.10 s. Transition of the S wave to the ST segment is designated by the connecting point J, which normally should not move more than 1 mm up or down from the isoelectric line.

The segment (interval) ST reflects the state of complete coverage by excitation of the ventricles and is determined from the end of the QRS complex to the beginning of the T wave, its duration is from 0.02 to 0.12 s. It is usually located on the isoelectric line. Normal permitted displacement of the segment up or down from it is not more than 0.5 mm
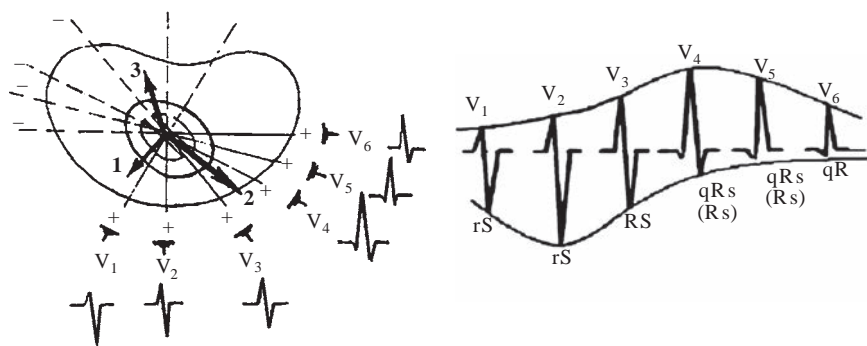
Fig. 17. Changes of the amplitude of the waves R and S in the chest leads (V$_{1-6}$)

in the leads from the limbs and up not more than 2 mm in V1-2 and down — not more than 0.5 mm in the V4-6 leads.

The T wave reflects the rapid process of repolarization (recovery) of the myocardium of the ventricles. The amplitude of the T wave depends on the magnitude of the R wave and is not more than 2/3–1/2 the amplitude of the R wave in standard leads (2–6 mm), and in the chest leads — 15–17 mm. The width of the T wave ranges from 0.16–0.24 s. The normal T wave is always positive in I, II, aVF, V2-6 leads, aVR — negative. In III, aVL and V1 leads the T wave may be positive, negative, biphasic or smoothed (isoelectric) that depends on the location of the heart in the chest. Different polarity of the T wave in III standard lead as a normal variant usually smoothens ("improving") in registration of this lead at the height of inspiration.

The segment (interval) ST and the T wave form a final part of the ventricular complex, describing the end of the process of excitation (depolarization) and during the recovery process (repolarization) of the ventricles of the heart.

The QRST complex (Q-T interval) represents electrical systole of the ventricles corresponds to the period from the beginning of depolarization until the end of ventricular repolarization. Its duration normally takes 0.36–0.44 s and depends on the heart rate and sex of the studied (Table 1).

Sometimes the small, positive wave U in the norm is recorded after the T wave in 0.02–0.04 sec, best of all it is detected in V2-V4 leads. The genesis of this wave is unclear. It is considered that it is a reflection of

23

Table 1

The duration of the Q-T interval at different heart rates

HR, b/min	Q-T, sec	HR, b/min	Q-T, sec	HR, b/min	Q-T, sec
40–41	0.42–0.51	66–67	0.33–0.40	101–104	0.27–0.32
42–44	0.41–0.50	68–69	0.33–0.39	105–106	0.26–0.32
45–46	0.40–0.48	70–71	0.32–0.39	107–113	0.26–0.31
47–48	0.39–0.47	72–75	0.32–0.38	114–121	0.25–0.30
49–51	0.38–0.46	76–79	0.31–0.37	122–130	0.24–0.29
52–53	0.37–0.45	80–83	0.30–0.36	131–133	0.24–0.28
54–55	0.37–0.44	84–88	0.30–0.35	134–139	0.23–0.28
56–58	0.36–0.43	89–90	0.29–0.34	140–145	0.23–0.27
59–61	0.35–0.42	91–94	0.28–0.34	146–150	0.22–0.27
62–63	0.34–0.41	95–97	0.28–0.33	151–160	0.22–0.26
64–65	0.34–0.40	98–100	0.27–0.33		

trace potential in a phase of increased excitability of the myocardium after systole, early diastole.

A horizontal line (interval T-R) is recorded after the T wave or U up to P wave of the next cardiac cycle, reflecting the rest phase (diastole) of the heart muscle.

THE ECG ANALYSIS

The ECG analysis should be started with validation of the correctness of its registration.

Firstly, you should pay attention to the available symbols of each ECG lead and the presence of various interferences. If the interferences are significant, the ECG should be retaken.

Secondly, it is necessary to check up the amplitude of the control millivolt, which should correspond to 10 mm (0.1 mV).

Thirdly, you should evaluate the velocity of the paper during ECG recording. The ECG recording is accepted to carry out in the movement of the paper tape of 50 mm/s, which corresponds to 0.02 to 1 mm. If the speed was different, then this should be noted on the ECG.

When analyzing the ECG it is advisable to use the ECG ruler or a compass, special tables and diagrams.

**Determination of the heart rhythm
and heart rate**

Determination of the heart rhythm and heart rate is usually determined by II standard lead, and if necessary, other leads.

Normal heart rhythm is sinus (pacemaker in the SA node), regular. In II standard lead and in I, aVF, V4-6 leads positive P waves of the same shape in one and the same lead and at the same distance from the QRS complex are recorded.

In pathological cases there may be different options of the nonsinus rhythm: atrial, AV connections, ventricular (idioventricular), migratory, ectopic (replacement) rhythms (see arrhythmia).

Regularity of the cardiac rhythm is assessed by measuring the duration of the intervals R-R between consistently registered cardiac cycles. If the intervals R-R are equal or different from each other by +10% of the average value, the rhythm will be considered regular. In other cases irregular rhythm is diagnosed.

Heart rate (HR) per 1 min is determined by the formula

$$HR = \frac{60}{R\text{-}R},$$

where 60 is the number of seconds in a minute; R-R — duration of the interval in seconds (s).

In the irregular rhythm, you can determine the average heart rate or specify a minimum (for the duration of the maximum R-R interval) and the maximum (for the duration of the smallest R-R interval).

In daily practice, the heart rate is usually determined using a special table, where calculated heart rate is specified for each duration of the R-R interval (see table 2).

Description the serial measurement of the waves and intervals of the ECG:

P, P-Q, Q, QRS, QT, R(S)-T, T, U.

Since QT (duration of the electric ventricular systole) depends on the heart rate and sex of the examined, it is necessary to compare the value obtained with due value (specify in brackets). Due value of the QT can be determined by the formula of Bazett:

$$QTc = \frac{QT}{\sqrt{R\text{-}R}},$$

where QT s (corrected QT) is calculated by dividing the measured QT/ the square root of the R-R interval. The actual duration of QT cannot be

different from the due by more than 15%.

To judge of the electrical systole of the heart one can determine systolic index (L. I. Fogelson, I. A. Chernogorov, 1928). Systolic index (SI) is the percentage ratio of the duration of the electrical systole to the magnitude of the cardiac cycle (SI=QT/R-R x 100). Normal systolic ratio should not exceed 50%, and its deviation from the supposed value should be not more than 5%.

As duration of the electric systole of the heart is normally almost identical to the mechanical one, the significant deviation of the parameters obtained from the due ones is indirect evidence of deficiency of the myocardial activity.

Table 2

The dependence of HR on the duration of the R-R interval

R-R, s	HR min	R-R, s	HR min
1.50	40	0.85	70
1.40	43	0.80	75
1.30	46	0.75	80
1.25	48	0.70	86
1.20	50	0.65	82
1.15	52	0.60	100
1.10	54	0.55	109
1.05	57	0.50	120
1.00	60	0.45	133
0.95	63	0.40	150
0.90	66	0.35	172

Determination of electrical axis of the heart

The electric heart axis (EHA) characterizes the direction of the wave excitation during the entire period of its distribution in the ventricular myocardium. EHA is a projection of the total EMF of the depolarization of the ventricles on the frontal plane. Its direction depends on the position of the heart in the chest. Normal electrical and anatomical axes of the heart have almost the same direction and position, but the electrical axis is located slightly behind from the anatomical, the angle between them varies in the range of 5–15°.

The value of the angle α (EHA) can be determined by geometric construction of the triangle of Eindhoven, knowing the size of waves in any two standard or unipolar leads. For this algebraic sum of the amplitudes of the waves of the QRS complex, for example, in the leads I and III is calculated in millimeters (Fig. 18).

If the angle alpha ranges from +30 to +70°, the direction of the electric axis is denoted as normal, thus the R wave on the ECG will be the highest in the II standard lead (RII>RI>RIII). It is believed that the EHA is directed horizontally when the angle alpha ranges from 0 to +30°. The EHA is
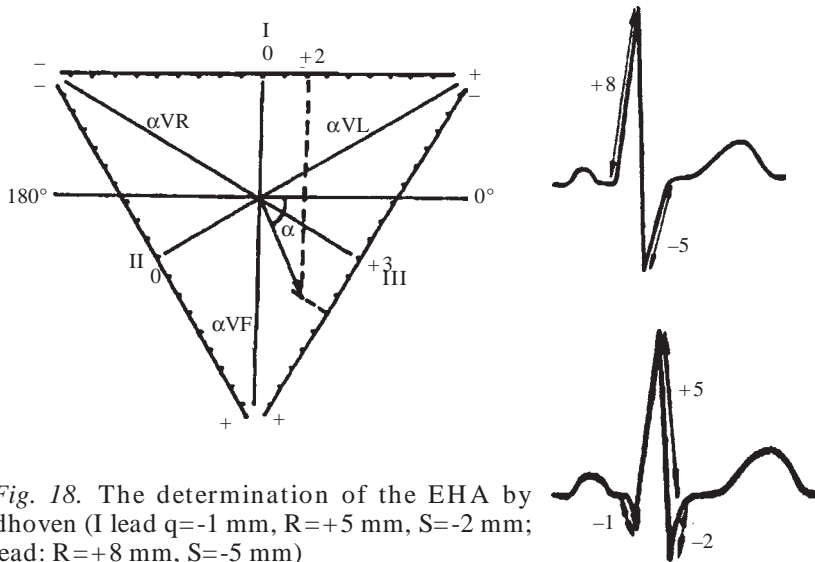
Fig. 18. The determination of the EHA by Eindhoven (I lead q=-1 mm, R=+5 mm, S=-2 mm; III lead: R=+8 mm, S=-5 mm)

deviated to the left at an angle alpha from 0° to -90° and is directed vertically if the angle alpha ranges from +70 to +90° and when the angle α is +90 to +180° it is inclined to the right. In practice, to determine the EHA the visual method (less accurate) is applied defining the direction of the EHA according to the largest amplitude of the R wave in standard leads. If R2>R1>R3 the EHA is in a normal position, if R1>R2>R3, the EHA is deviated to the left, and if R3>R2>R1 — the EHA is deviated to the right (Fig. 19).

Exactly the same as the axis of the QRS complex, it is possible to determine the electrical axis of the waves R and T, i. e. the direction of the total EMF of the depolarization of the atrial and ventricular repolarization. The normal electrical axis of the atria is in the range from 0 to +90°, often between +45 and +50°, and the T wave is between 0 and +90°.

PECULIARITIES OF
THE CHILDREN ECG

General distinctive features of the pediatric ECG compared with the ECG of an adult are:

27

a

Left axis deviation

Left axis

0°

Right axis deviation

Intermediate axis +30°

Right axis +120°

Vertical axis +60°

+90°

Normal range

b
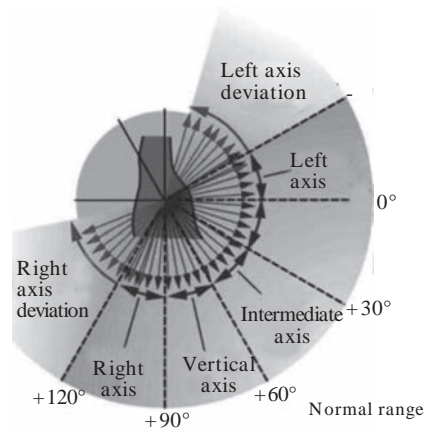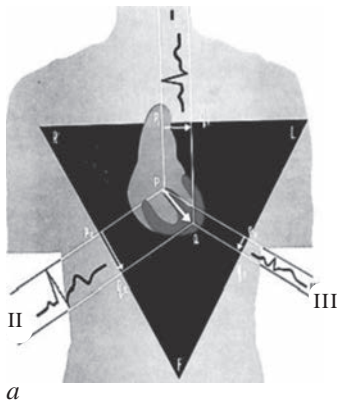
Electrical Axis of the Heart

The electrical axis is the sum total of all electrical currents generated by the ventricular myocardium during depolarization. Analysis of the axis may help to determine the location and extent of cardiac injury, such as ventricular hypertrophy, bundle branch block, or changes in the position of the heart in the chest (from, e.g., pregnancy or ascites).

The direction of the QRS complex in leads I and aVF determines the axis quadrant in relation to the heart.

Clinical Tip: Extreme right axis deviation is also called indeterminate, "no man's land", and "northwest".

c

N type ECG

0–30°
RII=RI>RIII

30–60°
RII>RI>RIII

60–90°
RII>RI=RIII

d

L type ECG

-90°−0°

RI>RII>RIII
(type RISIII)

e
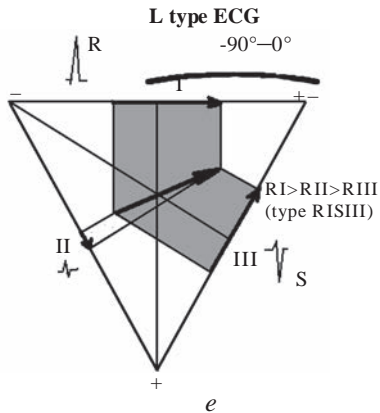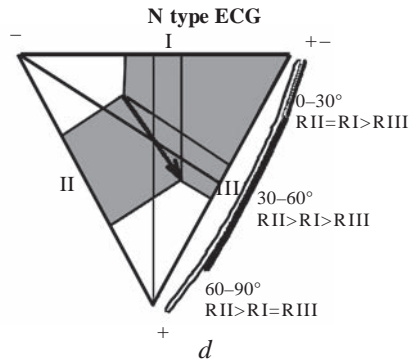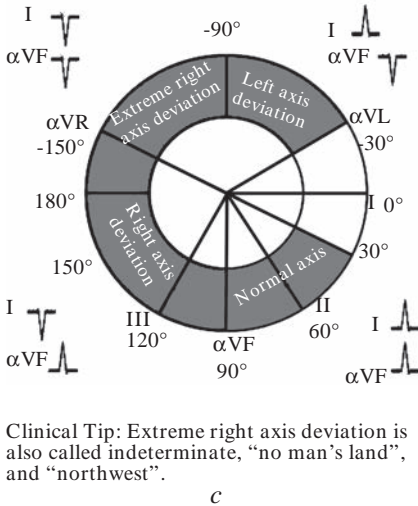
Fig. 19. Determination of the EHA (angle α) with standard leads *(a–f)*

R type ECG

$R III > R II > R I$
(type R IIISI)

$+90–+180°$

f
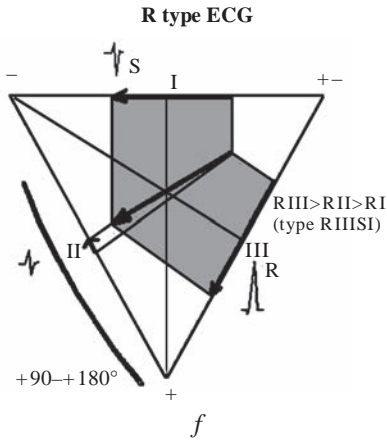
— shorter duration of the waves and intervals of the ECG, as a consequence of the more rapid conduction of excitation in the conduction system and the myocardium because of the smaller absolute size of the heart of a child;

— deviation of the electrical axis of the heart to the right due to the relative predominance of the right heart;

— change in the shape of the QRS complex due to the ratio of the shape and size of the waves, its components:

1) serrations of the waves in the third lead;

2) deep waves Q in II, III and aVF leads;

3) high R waves in V1,V2;

4) the picture of incomplete block of the right His bundle branch in V1;

— negative waves T in the lead III and leads V1-V4;

— sinus (respiratory) arrhythmia.

THE MAIN TRENDS
OF THE ECG CHANGES WITH AGE:

a decrease of the heart rate, stabilization of the rate (becomes regular), "horizontalization" of the heart position, increased duration of the waves, intervals and segments of the ECG, decreasing amplitude of the P wave, increasing amplitude of the R wave, but the reduction in V1,V2, normalization of the wave shape; increasing amplitude and positive direction of the wave T.

ELECTROCARDIOGRAPHIC CONCLUSION

In the ECG — conclusion one should note:

1. Heart rhythm (sinus, nonsinus — what?).
2. The regularity of the heart rhythm (regular, irregular — what?).
3. Heart rate per 1 min.
4. Position of the EHA.
5. The presence of the ECG syndromes:
a) rhythm disturbance and conductivity of the heart;

b) hypertrophy of the atria and ventricles or their acute overload;

c) damage of the myocardium (ischemia, dystrophy, necrosis, scarring).

6. Other infrequent changes of the myocardium.

ELECTROCARDIOGRAPHIC SIGNS OF HYPERTROPHY OF THE HEART CHAMBERS

In typical cases, hypertrophy of the heart is accompanied by expressive changes of the ECG. In hypertrophy of the atria they touch the P wave, and in hypertrophy of the ventricles — the complex QRS-T. A diagnosis of hypertrophy is usually considered, if not the last, but not the first, because it is not the main advantage of the method of electrocardiography. On the contrary, its role in the diagnosis of hypertrophy is relatively small. In this respect, it is markedly inferior to other methods of research, primarily echocardiography. Sometimes even in severe hypertrophy the ECG changes little.

Electrocardiographic signs of hypertrophy of the atria

The R wave is a summation of the excitation of both atria, which occurs at the beginning of excitation of the right and then of the left atrium. Normally its height (amplitude) does not exceed 0.25 mV (2.5 mm) and the duration (width) is not more than 0.11 (Fig. 20).

Hypertrophy of the right atrium

Excitation of the right atrium begins and ends before the left one, so the length (width) of the P wave in hypertrophy of the right atrium is
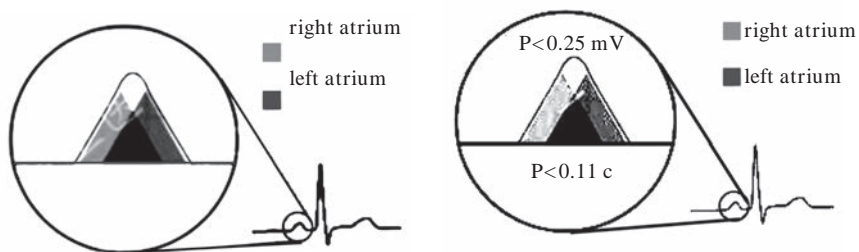
Fig. 20. A scheme of the P wave formation and its normative values
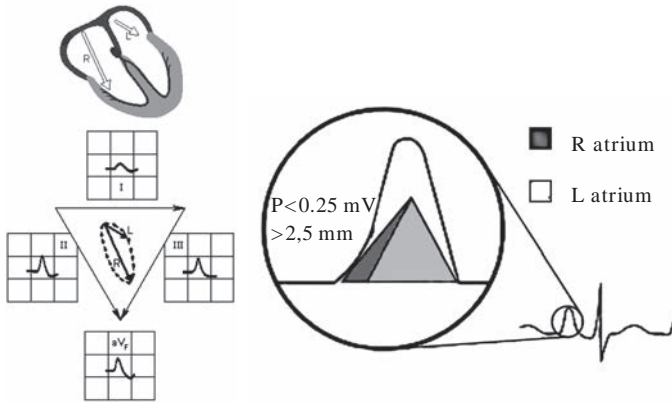
Fig. 21. A scheme of formation of the ECG signs of hypertrophy of the right atrium

normal — up to 0.11 cm or does not exceed 0.12 cm. The R wave becomes high (more than 0.25 mV — 2.5 mm) and pointed in the leads II, III, and aVF — the so-called "R-pulmonale" (Fig. 21, 23, *a*).

Hypertrophy of the left atrium

Hypertrophy of the left atrium, usually caused by its enlargement leads to an increase in the duration of the impulse conduction through the atria. It results in slowing down in its excitation and is accompanied by a significant increase in the duration of the P wave — more than 0.12 s. The wave P becomes bifurcated, "double-humped" — "P-mitrale" — in the leads I, II, aVL and V5-6 (Fig. 22).

Hypertrophy of both atria

Hypertrophy of both atria (Fig. 23, *b*) is most seen in the second lead where the bifurcated P wave of more than 0.12 s duration (due to hypertrophy of the left atrium) is determined with a high and pointed first wave (due to hypertrophy of the right atrium).

Hypertrophy of the ventricles

Hypertrophy of the ventricles differently affects the ECG. In short hypertrophy changes affect only the QRS complex. This form is often

	Lead II	Lead V₁	P wave axis	Condition	P Wave morphology	

Right atrial enlargement

Left atrial enlargement

>2 mm

>1 small box

>120 ms

>1 small box

0°

+75°

+90°

0°

+30°

+90°

Condition	Lead II	Lead VI
Normal sinus rhythm		
Right atrial enlargement (=P pulmonale)	2.5mm	
Left atrial enlargement (=P mitrale)	120 ms	>1 mm²

I

II

L

R

II

V₁

aV_F

R atrium

L atrium

P<0,11 c
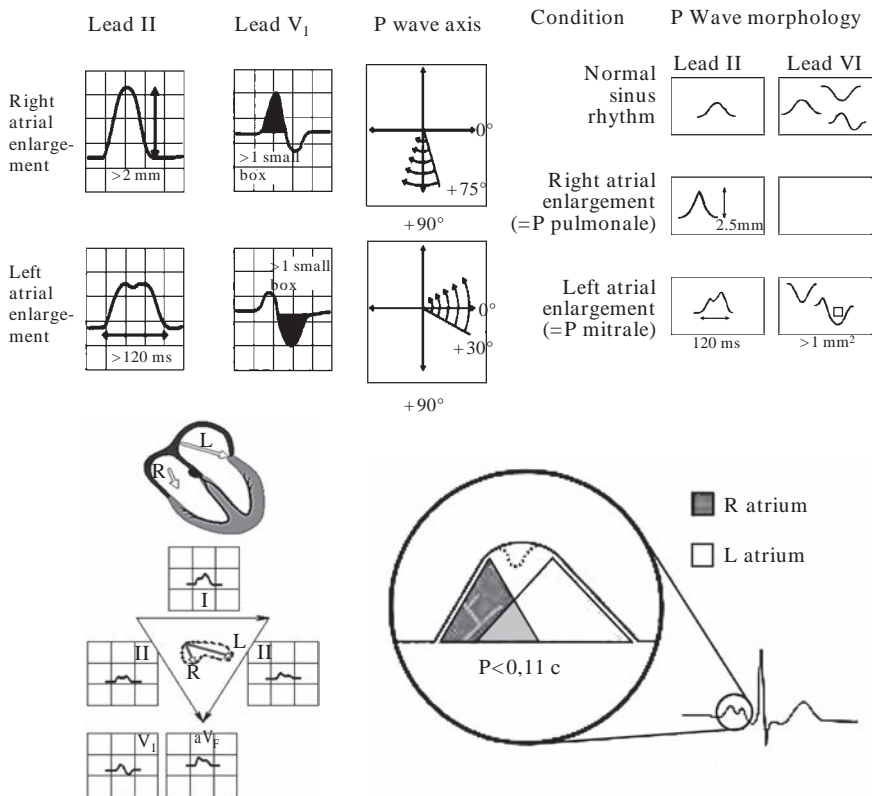
Fig. 22. A scheme of formation of the ECG signs of hypertrophy of the left atrium

called hypertrophy without overload. In significant and long-term hypertrophy the process of repolarization is affected to some extent, which leads to distortion of the final part of the ventricular complex — the segment RS-T and T wave. In this case we speak about hypertrophy with overload or, in new terminology, — hypertrophy with secondary myocardial changes.

The ECG signs of right ventricular hypertrophy

Right ventricular hypertrophy is not always diagnosed by the ECG, as the total heart excitation vector is mainly conditioned by the excitation of a more powerful left ventricle and the EMF of the latter prevails over the
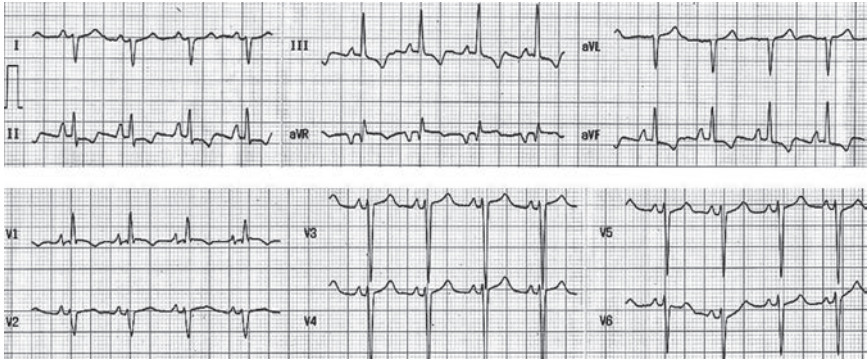
a
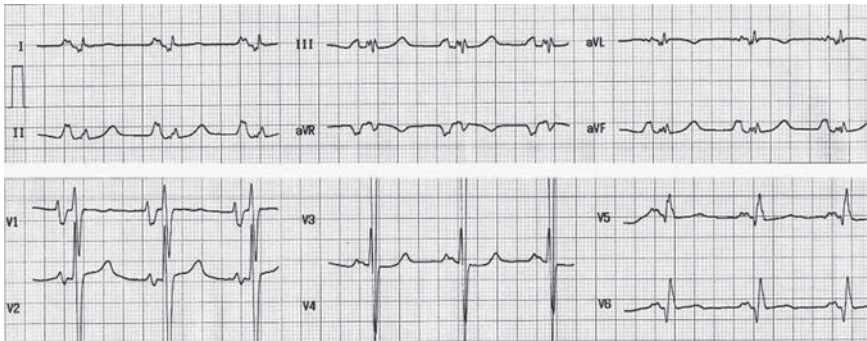
b

Fig. 23. The ECG in hypertrophy of the right *(a)* and both *(b)* atrial fibrillation

potentials of the right ventricle. Significant right ventricular hypertrophy is determined more reliably.

In hypertrophy of the right ventricle (the right ventricle is larger than the left one) the deviation of EHA in the frontal and horizontal planes to the right is registered: a high R wave in leads aVF, III as well as in the lead aVR (R>5 mm); deep S wave in the leads I and aVL; in the leads V1-2 wave R>7 mm with a gradual decrease in the amplitude toward the leads V5-6. The wave S in the chest leads may persist deep enough up to the leads V5-6 (the so-called S-type ECG) (Fig. 24).

In less significant hypertrophy or dilatation of the right ventricle a high R wave in the lead V1 (rSR* complex), and a high R wave in the lead V6

(Fig. 25) may be registered. This is explained by the fact that the excitation of the hypertrophied right ventricle ends later than the left one and final vector of the excitation of the heart is directed to the right in this period.

In hypertrophy of the right ventricle a duration of the QRS complex to 0.12 s and time of internal deviation of the ventricles in the leads V1-2 to 0.04–0.05 (normally up to 0.03 s) may be increased and it is associated with increase of time of coverage of the right ventricle with the excitation.
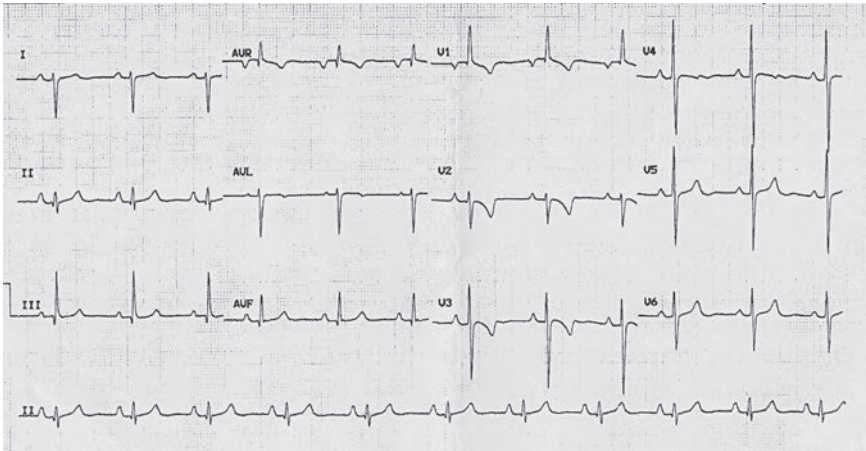
Fig. 24. The ECG in right ventricular hypertrophy without signs of its overloading
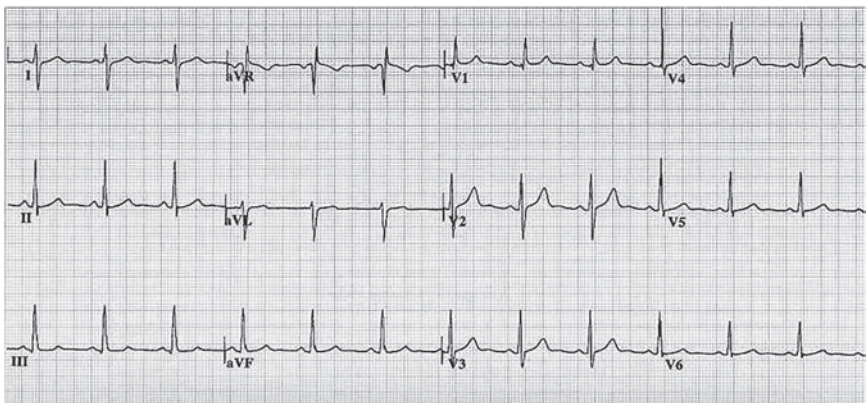
Fig. 25. The ECG in small hypertrophy of the right ventricle

Normally, the ventricles' excitation spreads from the endocardium to the epicardium, while the repolarization is of the opposite direction from the epicardium to the endocardium. Therefore, the direction of EMF depolarization coincides with the direction of EMF of repolarization and on the ECG the ST segment and the T wave usually are concordant to the QRS complex (the T wave and the QRS complex have the same direction). Because of the slowing down of the spread of the excitation in the hypertrophied right ventricle the repolarization is directed from the endocardium to the epicardium and the resulting EMF is opposite to the EMF depolarization direction: in the leads III, aVF, V1-2 we reveal discordant deviation of the ST segment and the biphasic (– +) or negative wave T (Fig. 26).

Left ventricular hypertrophy

In significant left ventricular hypertrophy the deviation of EMF in the frontal plane to the left is recorded: a high R wave in the leads I and aVL and deep S wave in the leads III and aVF. More typical is the deviation of EMF in the horizontal plane to the left (in the lead by Wilson): the R wave in the leads V5-6 is bigger than the R wave in the lead V4, the wave S in the leads V1-2 and sometimes in V3-4 is deep. Often there is no smooth progression of the transition zone in the chest leads: the QRS
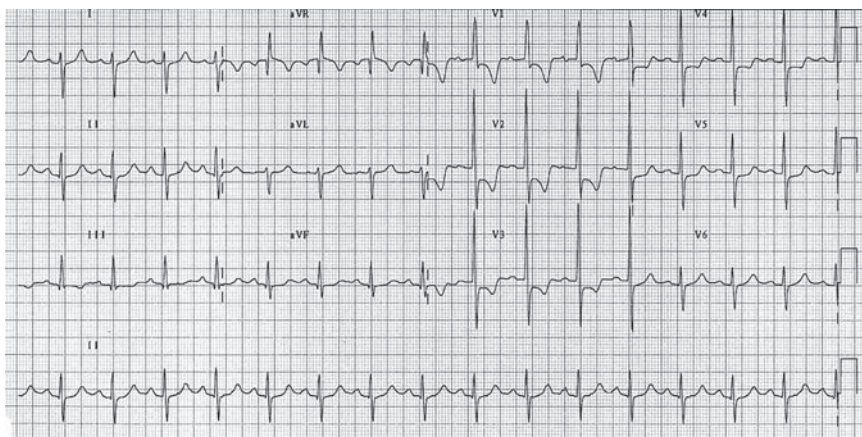
Fig. 26. Severe right ventricular hypertrophy with signs of overloading (depression of the segment ST in the right chest leads V1-V5, negative or biphasic T in V1-V4).

complex type rS in the leads VI or V2 immediately change to a complex type Rs in the leads V3 or V4.

An important sign of left ventricular hypertrophy is the increase in the amplitude of the QRS complex: total sum of the R waves in the lead I and S in the lead III is greater than 2.5 mV (25 mm), the sum of the wave S in the lead V1 (V2) and R in the lead V5 is more than 3.5 mV (the index of the Sokolov–Lyon) (Fig. 27).

These signs are caused by an increase in the left ventricular mass and, therefore, the resultant vector EMF of depolarization.

Hypertrophy of the left ventricle can increase the duration of the QRS complex to 0.11 and 0.12 s and the internal time deviation of the ventricles in the leads V5-6 to 0.06–0.08 s (normally 0.05 s) and it is associated with the increase of time of coverage of the left ventricle by the excitation. Because of slowing down of the excitation spread in the hypertrophied left ventricle and the change in the direction of repolarization in the leads I, aVL, V5-6, discordant displacement of the segment S-T and two-phase (– +) or negative T wave are revealed. (Fig. 28).

Hypertrophy of both ventricles

In a substantial prevalence of EMF in one of the ventricles on the ECG only signs of its hypertrophy are possible, and in a uniform enlargement of both ventricles the ECG may not differ from normal one. However, in some cases signs of hypertrophy of the right and left ventricles are
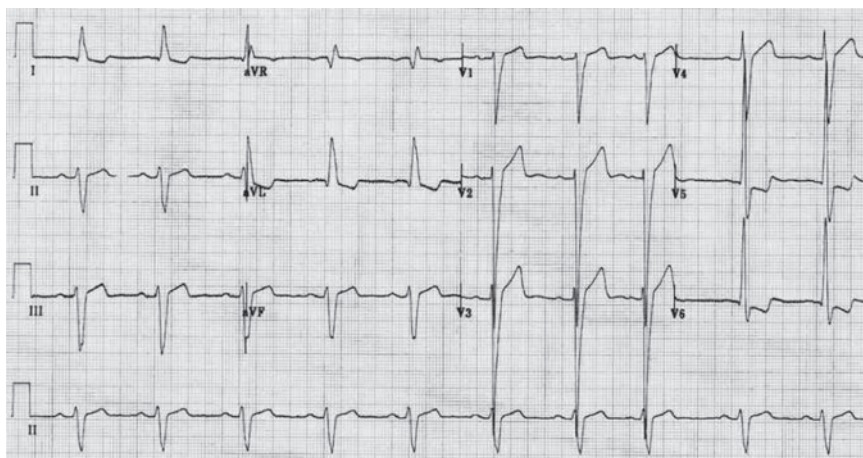
Fig. 27. The ECG in severe left ventricular hypertrophy

determined simultaneously. Options of electrocardiographic pattern of hypertrophy of both ventricles:

1) left ventricular hypertrophy in the chest leads combined with RAD (right axis deviation).

2) hypertrophy of the right ventricle in the chest leads combined with LAD (left axis deviation).

3) signs of hypertrophy of both ventricles in the chest leads — ventricular complex rSR' in the lead V1 and high R wave in the left chest leads (Fig. 29).
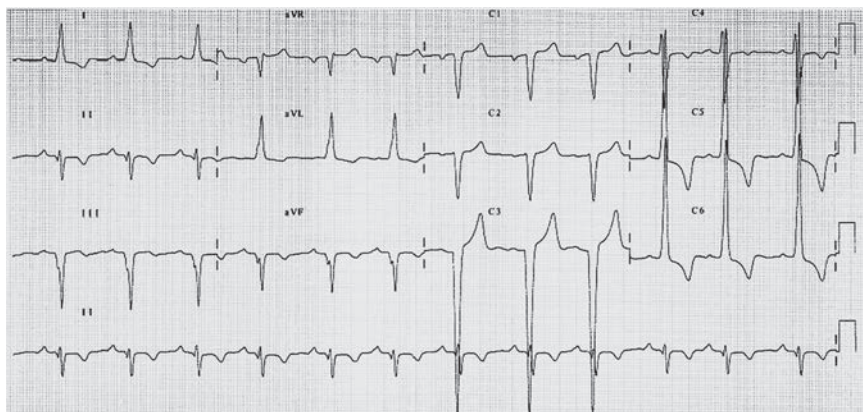
Fig. 28. Increased duration of QRS and discordant displacement of S-T, T in severe left ventricular hypertrophy
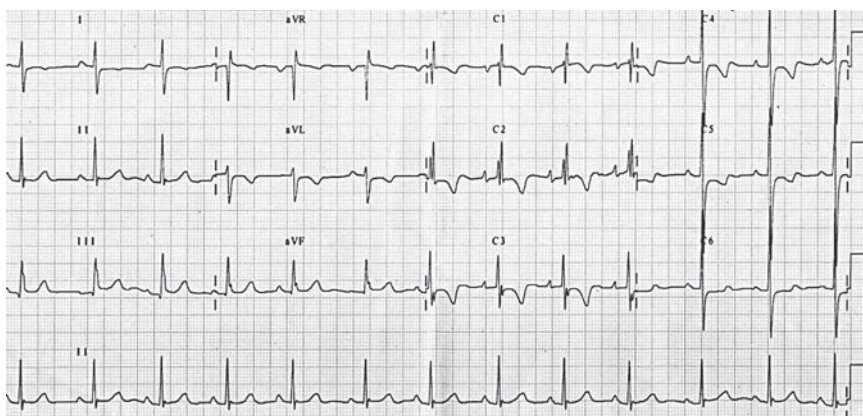
Fig. 29. The ECG in hypertrophy of both ventricles

4) combination of signs of right ventricular hypertrophy in the standard and chest leads with an increase of the amplitude of the wave S in the leads V1-2 (the latter is an indication of left ventricular hypertrophy).

ELECTROCARDIOGRAM
IN RHYTHM DISTURBANCES OF THE HEART

Disturbance of the cardiac rhythm (arrhythmia) is a change of its frequency, the place of formation of the excitation source, or disturbance of its conductivity via the conduction system. Thus, the term arrhythmia does not always mean disturbance of regularity of the heart rate.

Leading electrophysiological mechanisms of cardiac arrhythmias are:

1. Disturbance of the impulse formation.
2. Disturbance of the impulse transmission.
3. Simultaneous disturbance of formation and conduction of impulses.

Disturbance of the impulse formation may occur in the form of strengthening of the "normal automatism", the appearance of the "pathological automatism" and the development of the so-called triggered activity.

A change of the "normal automatism" is a dysfunction of the SA node or the increase in the activity of accessory pacemakers.

"Pathological automaticity" usually refers to the formation of ectopic foci of accessory automatic activity in the myocardium of the atria and ventricles or in partially depolarized cells system of His-Purkinje.

The occurrence of ectopic impulses can also be due to trigger (starting) activity. A necessary condition for the occurrence of the latter is the presence of the previous impulse, which must provoke it.

The given mechanisms responsible for the formation of impulses can be in a number of arrhythmias. The sinus tachy- and bradycardia, respectively is caused by strengthening or inhibition of automaticity of the SA node cells. Examples of arrhythmias caused by ectopic automaticity can be extrasystoles with a variable index of adhesion (including those caused by cardiac glycosides), parasystole, accelerated ectopic rhythms, ventricular arrhythmia of the type "pirouette".

Disturbance of the impulse conduction can be manifested by blocks at different levels of the conduction system of the heart or with arrhythmias caused by the mechanism of reentry of the excitation wave (a circular wave of the excitation, recurrent excitation, reentry, etc.).

The essence of the reentry mechanism consists in repeated or multiple excitation of the myocardium area by the same impulse, making a circular motion (Fig. 30).
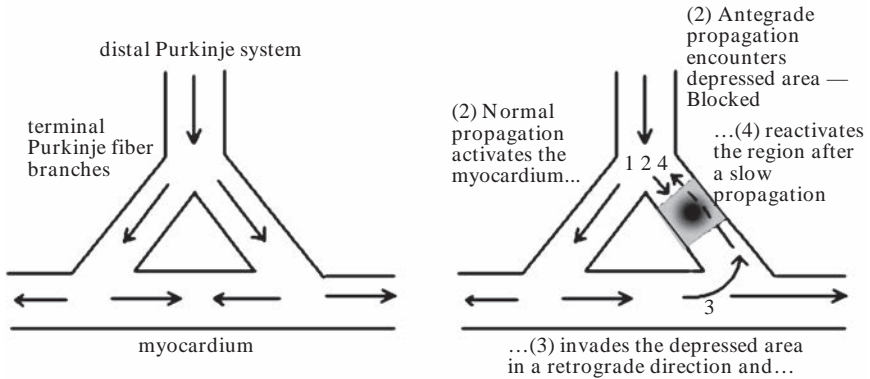
Fig. 30. A scheme of microreentry mechanism in the system of His-Purkinje (red shaded area with a delayed conduction).

To implement this mechanism there should be:

1. The presence of two or more ways of conduction.

2. Unidirectional or bidirectional temporary block of the impulse conduction in one of the paths.

3. Slowdown of the impulse conduction in the adjacent parts of the myocardium, sufficient to ensure that the impulse could pass retrograde the place of the block and re-depolarize the area of the myocardium proximal to the block.

Circulation of the impulse can be carried out by anatomically specific path around the "anatomical barriers" or functional ways — re-entry of the type "leading circle".

In the first case, the impulse almost always moves through the tissues, now completely restored their excitability. Such a long way of recurrent excitation (macroreentry) in practice is relatively rare — in the presence of abnormal additional ways of conduction (WPW syndrome).

In re-entry of the excitation wave according to the type of "leading circle" all the tissues in the path of circulation of the impulse, as a rule, are in the relatively refractory period. Circulation of the impulse is mainly by anastomoses of the branches of the conduction system within the minimum distances (micro re-entry).

The mechanism of reentry is associated with such complex arrhythmias as atrial fibrillation, atrial and ventricular flutter, premature beats, paroxysmal tachycardia. The mechanism of reentry is proven in the WPW syndrome.

The basis for the development of combined disorders of the cardiac rhythm and conduction can be based on a combination of the above described and, perhaps, as yet unknown other mechanisms.

If disorder of the impulse occurs in the sinus node, we speak of nomotopic rhythm disturbances (sinus "tachy-brady" arrhythmia, sinus arrhythmia, failure — arrest of sinus node). In impulse generation in the sinus node we speak of **heterotopic or ectopic rhythm.**

Criteria of arrhythmia:
• Change of the heart rate over 100 or below 50 per minute
• Abnormal rhythm of any origin
• Any nonsinus rhythm
• Disurbance of the impulse conduction in the parts of the cardiac conduction system

Classification of arrhythmia (according to M. S. Kushakovsky and N. B. Zhuravleva in modifications)

1. The disturbance of the **impulse formation**

A. Disturbance of the automatism of the sinus node (nomotopic arrhythmia)
1. Sinus tachycardia
2. Sinus bradycardia
3. Sinus arrhythmia
4. Syndrome sick sinus (SSS)

B. Ectopic (heterotopic) rhythms, due to the predominance of automaticity of the **ectopic centers**
1. Slow (substitutional) slip complexes and rhythms
2. Accelerated ectopic rhythm
3. Migration of the supraventricular pacemaker

C. Ectopic (heterotopic) rhythms, mainly due to the mechanism of re-entry of the excitation wave
1. Premature beats (atrial, AV connections, ventricular)
2. Paroxysmal tachycardia (supraventricular, AV connections, ventricular)
3. Atrial flutter
4. Atrial fibrillation
5. Atrial and ventricular fibrillation

2. Conduction disorders
1. Sinoatrial block
2. Atrioventricular block of I, II and III degree
3. Intraventricular block (block of the His bundle branch)
4. Syndrome of Frederick
5. Ventricular asystole

6. Syndrome of premature ventricular excitation:

a. Syndrome of Wolff-Parkinson-White (WPW)

b. Syndrome of shortened PQ interval (CLC)

3. Combined arrhythmias

1. Parasystole

2. Ectopic beats with exit block

DISTURBANCES OF THE PULSE FORMATION

A. Disturbance of the automatism of the **sinus node (arrhythmia nomotopic)**

Sinus tachycardia

Sinus tachycardia is the increase in the heart rate from 90 to 140–150 per minute while maintaining proper sinus rhythm.

It is based on the greater automaticity of the primary pacemaker — the sinoatrial node. Its causes are endogenous and exogenous influences: physical or emotional stress, emotion, infection and fever, anemia, hypotension, respiratory hypoxemia, acidosis and hypoglycemia, myocardial ischemia, hormonal disorders (hyperthyroidism), medication effects (sympathomimetic). Sinus tachycardia may be the first symptom of heart failure. In sinus tachycardia normal electrical impulses are conducted through the atria and the ventricles.

The ECG signs:

• the P wave of sinus origin (positive in I, II, aVF, V4-6, negative in aVR);

• shortening of the R-R (R-R) intervals in comparison to normal ones;

• the difference between the intervals R-R is not greater than 0.15;

• the correct sequence of the P wave and QRS complex in all cycles;

• the presence of the unchanged QRS complex (Fig. 31).

Sinus bradycardia

Sinus bradycardia is a decrease in the heart rate to 59–40 per minute while maintaining proper sinus rhythm.
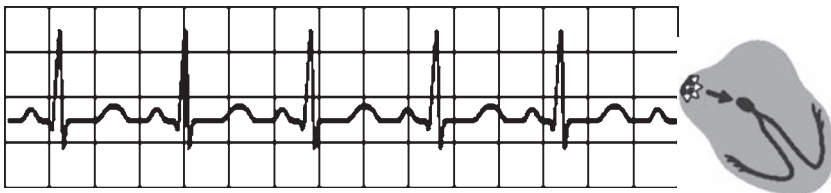
Fig. 31. Sinus tachycardia

It is due to a decrease in automaticity of the sinoatrial node. The main cause of sinus bradycardia is an increase in the vagal tone. Normally it is often found in athletes, however, it may occur in various diseases (myxedema, coronary heart disease, etc.). The ECG in sinus bradycardia is a little different from normal one, except for rarer rhythm.

The ECG signs:

— the P wave of the sinus origin (positive in I, II, aVF, V4-6, negative in aVR);

— lengthening of the R-R intervals in comparison to normal ones;

— the difference between the intervals R-R is not greater than 0.15;

— the correct sequence of the P wave and QRS complex in all cycles;

— the presence of the unchanged QRS complex (Fig. 32).

Sinus arrhythmia

Sinus arrhythmia is irregular sinus rhythm, characterized by periods of gradual acceleration and deceleration of rhythm.

It is due to the irregular impulses in the sinoatrial node, caused by an imbalance of the autonomic nervous system with a distinct predominance of its parasympathetic division. Most common is respiratory sinus arrhythmia, in which heart rate increases on inspiration and decreases on exhalation.

The ECG signs:

• the P wave of the sinus origin (positive in I, II, aVF, V4-6, negative in aVR);

• the difference between the intervals R-R exceeds more than 10% of the average R-R (usually more than 0.12–0.15);

• the correct sequence of the P wave and QRS complex in all cycles;

• the presence of the unchanged QRS complex (Fig. 33).

Syndrome of weakness of the sinus node (SWSN)

The syndrome of weakness of the sinus node is a combination of the ECG signs reflecting structural damage of the sinus node, its inability to function normally as a pacemaker of the heart and (or) to ensure the regular conduction of automated impulses to the atria.

Most often it occurs in diseases of the heart, leading to the development of ischemia, degeneration, necrosis or fibrosis in the region of the sinoatrial node.

The ECG signs (Fig. 34):

— constant sinus bradycardia (see above) with a frequency of less than 45–50 per minute (it is typical when the test with the dosed physical load or after administration of atropine there is no adequate increase of the heart rate);
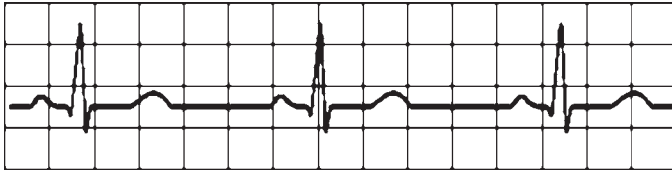
Fig. 32. Sinus bradycardia
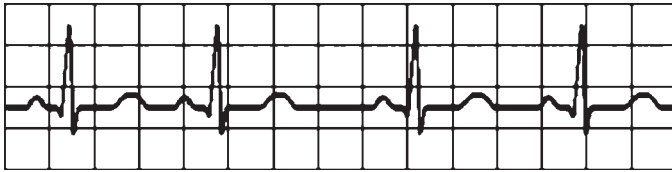
Fig. 33. Sinus arrhythmia
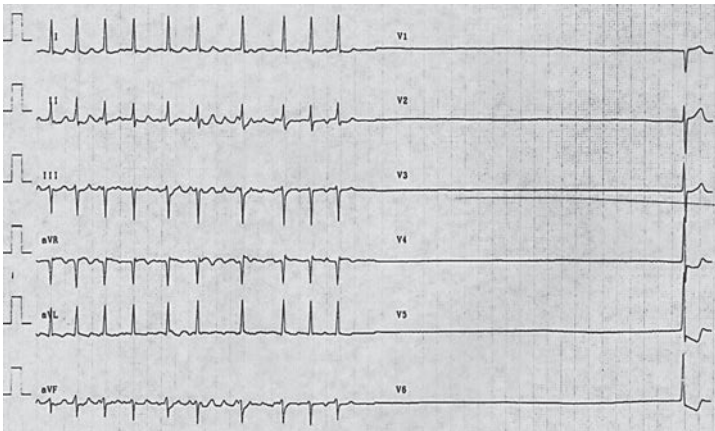
Fig. 34. The ECG in SWSN (the episode of asystole against the background of atrial fibrillation)

— cessation or refusal of the sinoatrial node, long or short-term (sinus pause more than 2–2.5);

— repetitive sinoatrial block;

— repeated alternation of sinus bradycardia (long pauses more than 2.5–3) with paroxysm of atrial fibrillation (flutter) or atrial tachycardia (syndrome of bradycardia-tachycardia).

B. Ectopic (heterotopic) rhythms, due to the predominance of automaticity of ectopic centers

Slow (substitutional) slip complexes and rhythms

Slow (substitutional) slip rhythms is nonsinus ectopic rhythms originating from the atria, AV connection or the ventricles. Being passive, ectopic rhythm as if protects the heart from prolonged periods of asystole associated with inhibition of the basic sinus rhythm. Since the automaticity of ectopic centers of II and III level are much lower than SA node, the heart rate in these ectopic rhythms is usually less than 60 beats per minute, so they are called slow. The reasons can be:

• vagotonia in healthy people;

• organic damage of the sinus node (cardiosclerosis, acute myocardial infarction);

• other factors leading to bradycardia, sinoatrial and atrioventricular blockade.

The ECG-signs of slow slip complexes (Fig. 35):

1. The presence of nonsinus complexes on the ECG of the individual, sources of which are impulses coming from the atria, AV connection or ventricles.

2. The R-R interval preceding the slip ectopic complex is extended, and the next R-R is normal or shortened.

The ECG signs of slow slip rhythms (Fig. 36):

1. The right rhythm with a frequency of 60 or less

2. Signs of the nonsinus (from the atria, the AV connections, ventricular) pacemaker in each complex.

Accelerated ectopic rhythms

Accelerated ectopic beats or non-paroxysmal tachycardia is nonparoxysmal acceleration of the heart rate up to 100-130 per min, caused by relatively frequent ectopic impulses originating from the atria, AV connection or ventricles. Thus, the heart rate in accelerated ectopic rhythms is higher than the slow substitutional rhythms, and lower than in paroxysmal tachycardia.

The origin of accelerated ectopic rhythms is associated with increased automaticity of the **centers of II and III level** (the acceleration of the spontaneous diastolic depolarization of a pacemaker) or trigger activity of the AV connection (the appearance of late delayed post depolarization).

The main causes for the accelerated ectopic rhythms are: digitalis toxicity (the most common cause); acute myocardial infarction; chronic IHD; pulmonary heart.
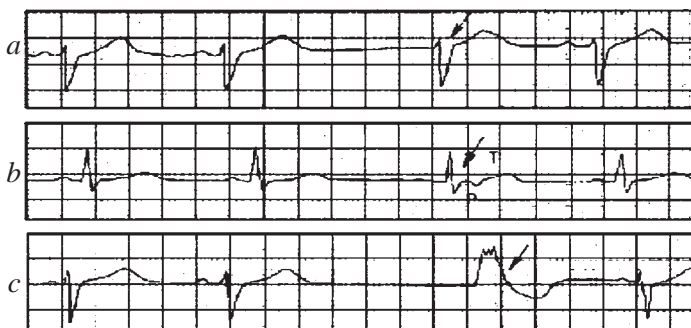
Fig. 35. The ECG during slow slip complexes: *a, b* — slip complex of AV connection; *c* — slip complex of ventricle
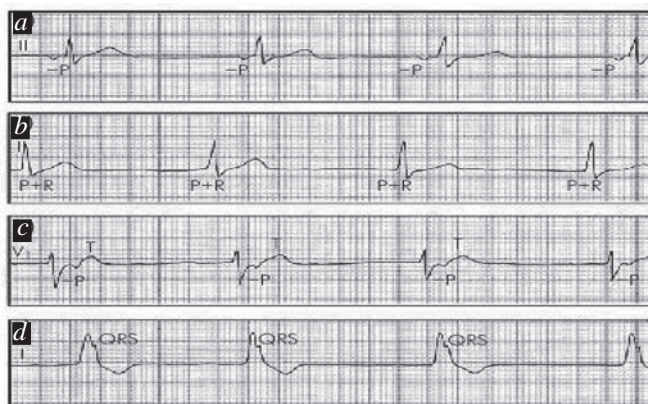
Fig. 36. The ECG in the slow slip rhythms: *a* — atrial rhythm; *b* — the rhythm of the AV connection with a simultaneous excitation of the ventricles and atria; *c* — the rhythm of the AV connection with excitation of the ventricles, pre-excitation of the atria; *d* — ventricular (idioventricular) rhythm

The ECG signs (Fig. 37):

1. Nonparoxysmal gradual acceleration of the heart rate up to 100–130 per min.

2. Correct ventricular rhythm.

3. The presence of characteristics of the nonsinus (atrial, AV connection or ventricular) pacemaker in each complex P-QRS-T.

Migration of a supraventricular pacemaker

Migration of a supraventricular pacemaker is characterized by the gradual, from cycle to cycle, moving of the source of the rhythm from the
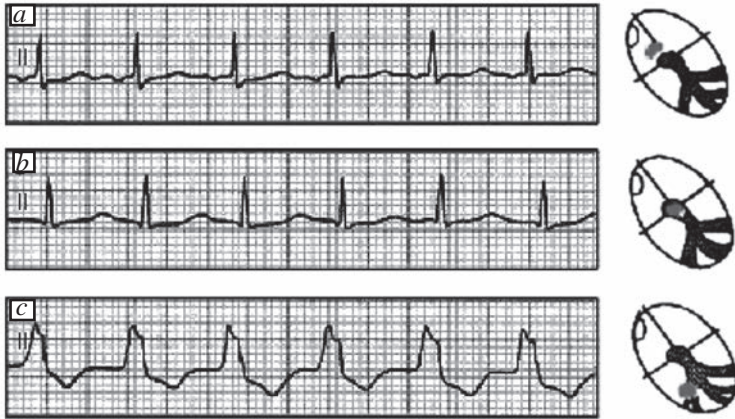
Fig. 37. Accelerated ectopic beats or nonparoxysmal tachycardia:
a — accelerated atrial rhythm; *b* — fast rhythm of the AV connection with a simultaneous excitation of the ventricles and the atria; *c* — accelerated ventricular (idioventricular) rhythm

SA-node to AV-connection and back. Successive contractions of the heart every time are due to impulses coming from different parts of the cardiac conduction system: SA node, from the upper or lower parts of the atria, the AV connection. This migration of the pacemaker can occur in healthy people in the increase of the vagal tone as well as in patients with coronary artery disease, valvular heart disease, myocarditis, and various infectious diseases. The migration of the pacemaker often occurs in the syndrome of weakness of the sinus node (SSS).

The ECG signs (Fig. 38):

1. Gradual, from cycle to cycle, changing of the shape and polarity of the P wave.

2. Change in the length of the P-Q(R) interval depending on the localization of the pacemaker.

3. Mild fluctuations of the duration of the R-R (P-P) intervals.

C. Ectopic (heterotopic) rhythms, mainly due to the mechanism of re-entry of the excitation wave.

The ECG in extrasystoles

Extrasystole is a premature excitation of the heart due to the mechanism of the re-entry wave excitation or increased oscillatory activity of the cell
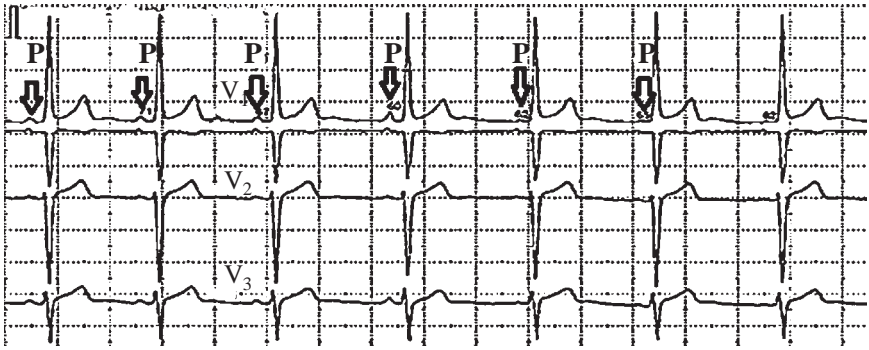
Fig. 38. The ECG during the migration of the supraventricular pacemaker

membranes that occurs in the sinus node, atria, AV connection or different parts of the conduction system of the ventricles.

Before proceeding to the presentation of electrocardiographic criteria of certain forms of arrhythmia, let us briefly examine some general concepts and terms used in the description of extrasystoles.

The interval of adhesion (pre-extrasystole interval) is a distance from the preceding of the next cycle of P-QRST of the basic rhythm to the extrasystole. The compensatory pause is a distance from extrasystole to the next cycle of the P-QRST of the basic rhythm (Fig. 39).

If the sum of the adhesion interval and the compensatory pause is less than the length of two R-R intervals of the main rhythm, it is called an incomplete compensatory pause. In complete compensatory pause, this amount is equal to two intervals of the basic rhythm (see Fig. 39). If extrasystole is wedged between the two main complexes without the postextrasystolic pause, then we speak of interpolated extrasystole (Fig. 40).

Early extrasystoles are extrasytoles, the initial part of which is superimposed on the T wave of the P-QRST cycle of the main rhythm preceding extrasystole or is from the end of the T wave of the complex by not more than 0.04 s (Fig. 41).

Extrasystoles can be single, paired and grouped (up to 4 complexes); monotopic (monomorphic) — originating from a single ectopic source and polytopic (polymorphic), due to the operation of the multiple ectopic foci of the extrasystole formation. In the last case extrasystolic complexes with different intervals of adhesion different from each other in form are registered (Fig. 42 *a, b*).
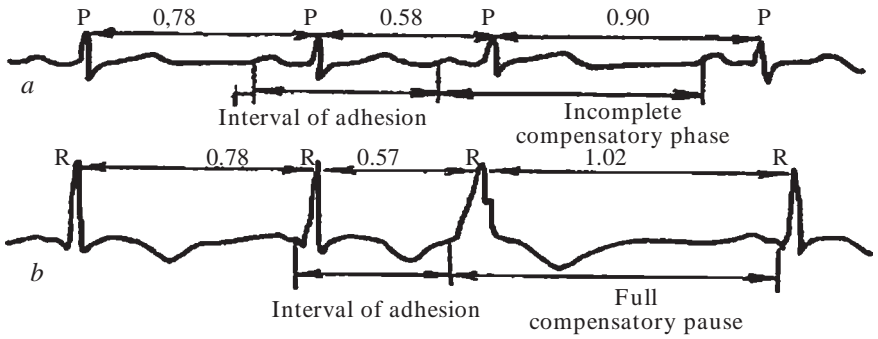
Fig. 39. Measurement of the adhesion interval and the compensatory pause in various extrasystoles
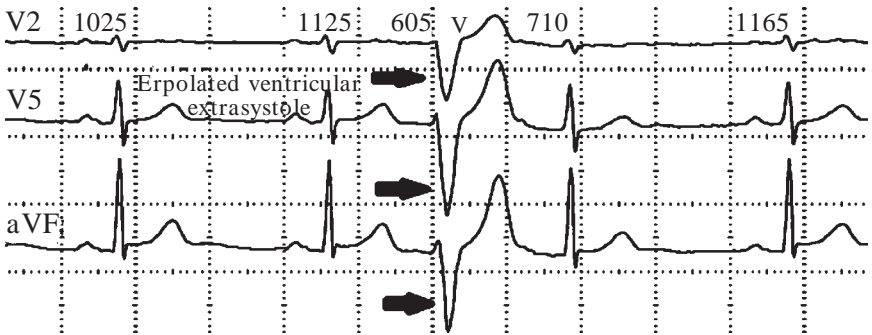
Fig. 40. The ECG with inserted ventricular extrasystole
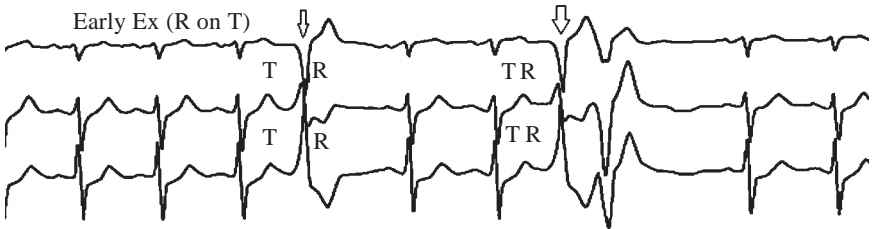
Fig. 41. Early R- and T-ventricular extrasystoles
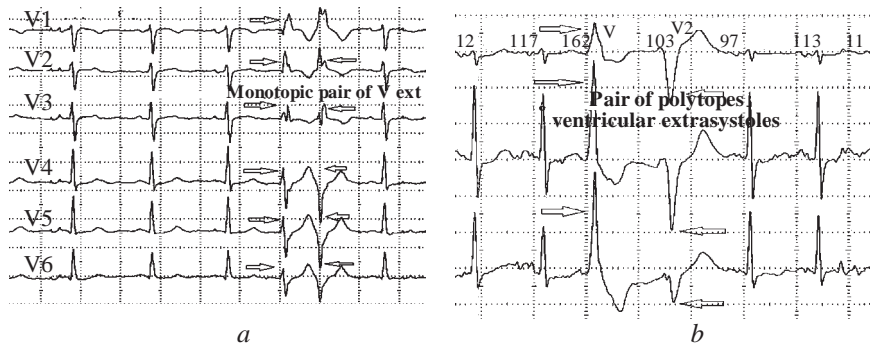
Fig. 42: *a* — monotopic pair; *b* — pair of polytopes
ventricular extrasystoles

Allorrhythmia is a correct alternation of extrasystoles with normal sinus cycles. If the extrasystoles recur after each normal sinus complex, we speak of bigeminy. If one extrasystole occurs in every two cycles of the normal P-QRST, then we speak of trigeminy, etc. (Fig. 43).

According to the place of the ectopic foci origination we distinguish isolated atrial extrasystoles, extrasystole from the AV connections, and ventricular extrasystoles.

Atrial extrasystole

Atrial extrasystole is a premature excitement of the heart, arising under the influence of impulses coming from different parts of the conduction system of the atria.

The ECG signs:

— the premature appearance of the P wave and subsequent QRST complex;

— the distance from the P wave to the QRST complex is from 0.08 to 0.12 s;

— deformation and change of polarity of the P wave beats;

— the presence of unmodified extrasystoles of the ventricular QRST complex;

— incomplete compensatory pause.

In some cases, early atrial extrasystolic impulse is not conducted to the ventricles, as it finds the AV-node in the state of absolute refractoriness. The ECG registers a premature extrasystolic wave, after which there is no QRS complex. In this case we speak of the blocked atrial extrasystole (Fig. 44).
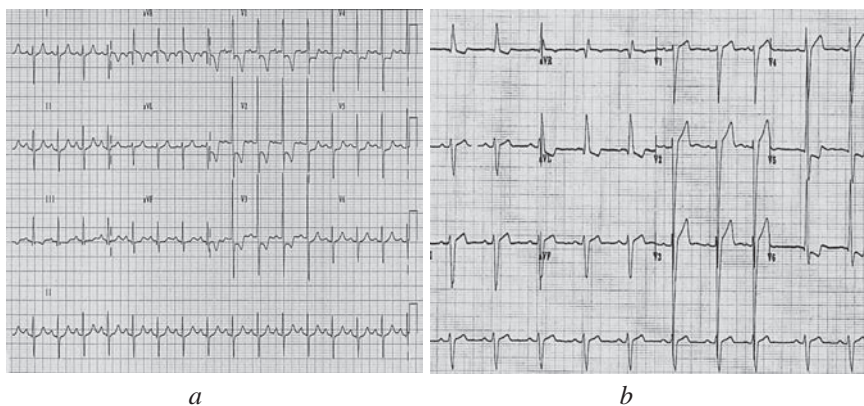
Fig. 43. The ECG of allorhythmia: *a* — supraventricular bigeminy; *b* — ventricular trigeminy
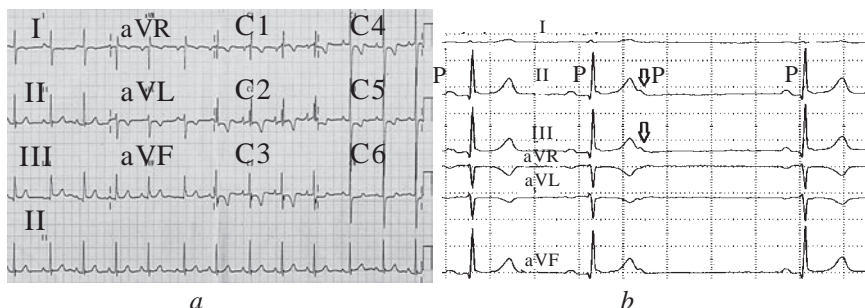
Fig. 44. The ECG with atrial arrhythmia: *a* — typical deformation of the wave "P"; *b* — blocked atrial beat

The extrasystole from the AV-connection

Extrasystole from the AV-connection is the premature excitement of the heart, arising under the influence of impulses originating in the atrioventricular connection. The ectopic impulse arising in the AV-connection extends in two directions: top-down in the conduction system to the ventricles (in this regard, extrasystolic ventricular complex do not differ from that of the ventricular complexes of the sinus origin) and retrograde upward from downward at the AV node and the atria, which lead to the formation of the negative P wave.

50

The ECG signs:

— premature appearance of the unchanged ventricular QRS complex on the ECG;

— the absence of the P wave (with a simultaneous excitation of the atria and ventricles) or negative P wave in the leads II, III and aVF after the QRS complex (if ectopic impulse reaches the ventricles faster than the atria);

— incomplete or complete compensatory pause (Fig. 45).

Ventricular extrasystole

Ventricular extrasystole is a premature excitement of the heart, arising under the influence of impulses coming from different parts of the conduction system of the ventricles.

The ECG signs:

— premature extraordinary appearance of altered ventricular QRS complex on the ECG;

— significant expansion and deformation of the extrasystolic QRS complex;

— the location of the segment S(R)-T and T wave of PHB in discordant direction to the main wave of the QRS complex;

— no P wave prior to ventricular extrasystole;

— the presence of a complete compensatory pause after ventricular extrasystole (Fig. 46).
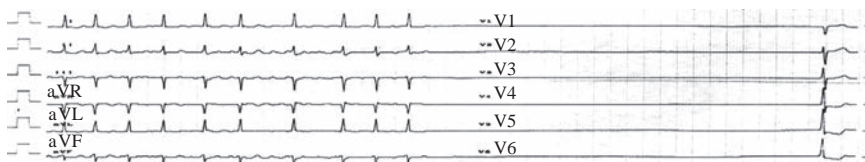
Fig. 45. Option of the ECG with extrasystole from the AV-connection — P wave merged with the QRS complex, the morphology of the QRS complex is not significantly changed

Fig. 46. The ECG example of polytopic (polymorphic) ventricular extrasystole

Using the ECG it is possible to diagnose the localization of the ectopic focus. Generally, there are extrasystoles of the left and right ventricles. The morphology of the complexes QRS in the left ventricular extrasystoles is reminiscent of blockade of the right bundle branch block (RBBB), and the localization of the focus in the right ventricle — the left bundle branch block (LBBB). A topical diagnosis of ventricular extrasystoles is more comfortable to be made in the chest leads (Wilson) (Fig. 47, *b*).

There are also basal and apical ventricular extrasystoles. In basal extrasystoles originating from the base of the heart, extendedly directed upward, the QRS complexes are marked in the right and left chest ECG leads. The upward knee of the R wave thus resembles a **Δ** wave, which gives extrasystolic complexes similarity with the phenomenon of WPW type A. Apical extrasystolic complexes are characterized by a predominant S wave in the right and left chest leads (Fig. 48 *a, b*).

Paroxysmal tachycardia

Paroxysmal tachycardia is à sudden beginning and sudden ending attack of accelerated heart contractions up to 140–250 per minute, while maintaining regular rhythm in most cases. These transient attacks can be unstable with duration of less than 30 s and stable (persistent) with duration of 30 s.

An important feature of paroxysmal tachycardia is sustained correct rhythm and constant rate of heart contractions throughout the paroxysm (except for the first few cycles), which in contrast to sinus tachycardia does not change after physical exertion, emotional stress or after injection of atropine.

Currently, there are two main mechanisms of paroxysmal tachycardia: 1) the mechanism of re-entry of the excitation wave (re-entry); 2) enhanced automaticity of the cells of the conduction system of the heart — ectopic centers of II and III order.

Depending on the localization of the ectopic focus of increased automaticity or the constantly circulating return wave of excitation (re-entry) there are atrial, atrioventricular (supraventricular) and ventricular forms of paroxysmal tachycardia. As in supraventricular paroxysmal tachycardia excitation wave spreads through the ventricles in the usual way, the ventricular complexes in most cases are not changed. The main distinguishing features of atrial and atrioventricular forms of paroxysmal tachycardia**,** detected on the surface electrocardiogram, are different shape and polarity of the P waves as well as their location in relation to the ventricular QRS complex. However, very often the P wave is undetectable
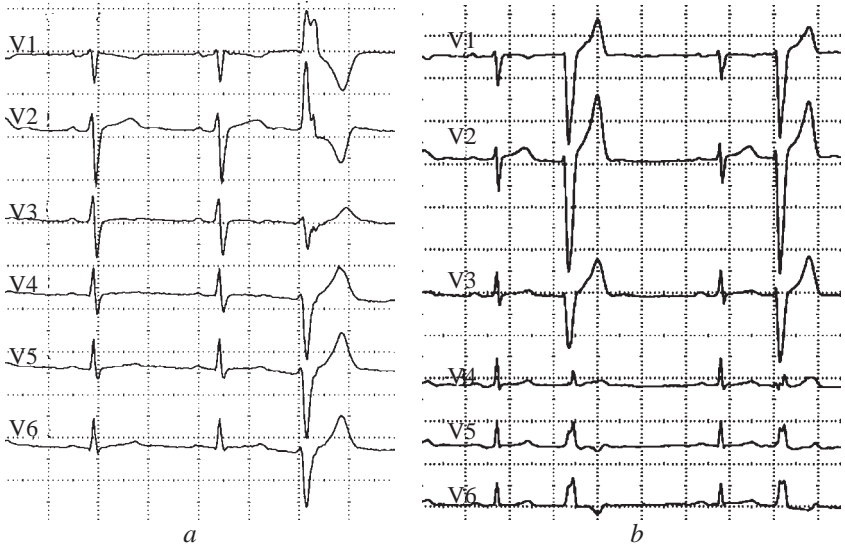
Fig. 47. The ECG of: *a* — left ventricle; *b* — right ventricular extrasystoles
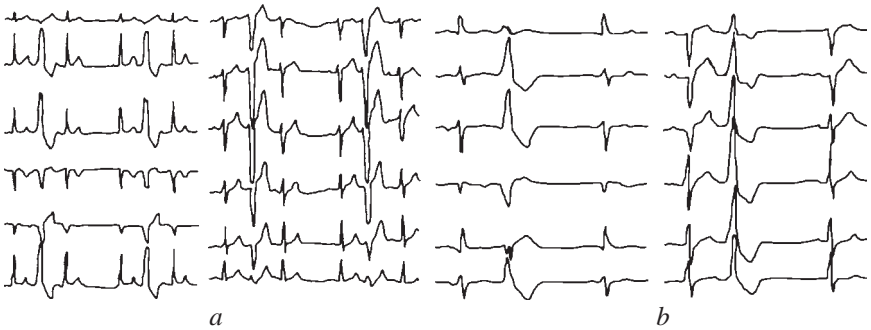
Fig. 48. The ECG in extrasystoles: *a* — basal; *b* — apical

on the ECG recorded at the time of the attack, against the background of pronounced tachycardia. Therefore, in practical electrocardiology atrial and atrioventricular forms of paroxysmal tachycardia are often combined as supraventricular paroxysmal tachycardia, especially because drug treatment of both forms is largely similar (the same drugs are applied).

Supraventricular paroxysmal tachycardia
The ECG signs:
— sudden beginning and sudden ending attack of acceleration of the heart contractions up to 140–250 per minute while maintaining the correct rhythm;
— normal unmodified ventricular QRS complexes similar to QRS complexes, recorded before the attack of paroxysmal tachycardia;
— the absence of the P wave on the ECG, or the presence of it before or after each QRS complex (Fig. 49–51).

Ventricular paroxysmal tachycardia
In ventricular paroxysmal tachycardia the source of the ectopic impulses is the contractile myocardium of the ventricles, bundle of His or Purkinje fibers. Unlike other tachycardia, ventricular tachycardia has worse prognosis due to the tendency to go into ventricular fibrillation, or cause severe circulatory disorders. As a rule, paroxysmal ventricular tachycardia develops against the background of significant organic changes of the heart muscle.

In contrast to paroxysmal supraventricular tachycardia in ventricular tachycardia the course of excitation in the ventricles is severely disturbed: the ectopic impulse initially excites one ventricle, and then behind time it gets to the other ventricle and spread in it in an unusual way. All of these changes resemble those in ventricular arrhythmia, and when there is His bundle branch block (Fig. 52).

An important electrocardiographic sign of ventricular paroxysmal tachycardia is the so-called atrioventricular dissociation, i. e., the complete dissociation in the activity of the atria and ventricles. The ectopic impulses arising in the ventricles are not conducted retrograde to the atria and the atria are excited in the usual way due to the impulses generated in the sinus node. In most cases, the excitation wave is not conducted from the atria to the ventricles as the atrioventricular node is in a state of refractoriness (affected by frequent impulses from the ventricles) (Fig. 53).
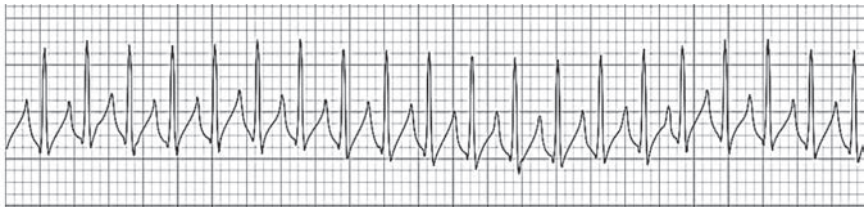
Fig. 49. Paroxysmal supraventricular tachycardia
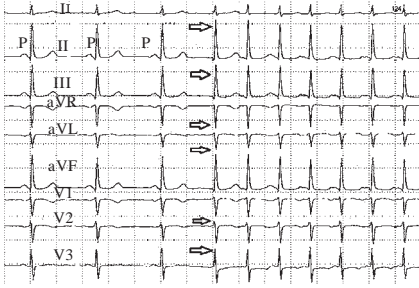
Fig. 50. Beginning of paroxysm of supraventricular tachycardia (indicated by arrows)
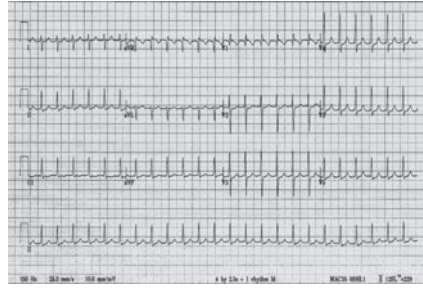
Fig. 51. The ECG in paroxysmal supraventricular tachycardia
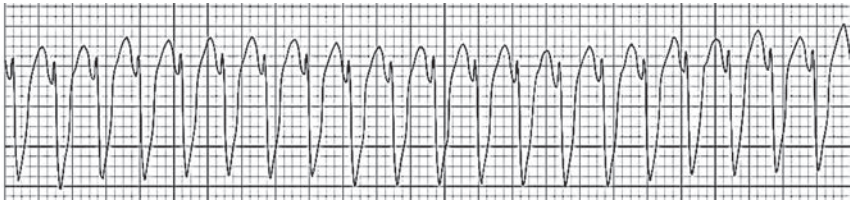
Fig. 52. Morphology of the QRS complex during ventricular tachycardia
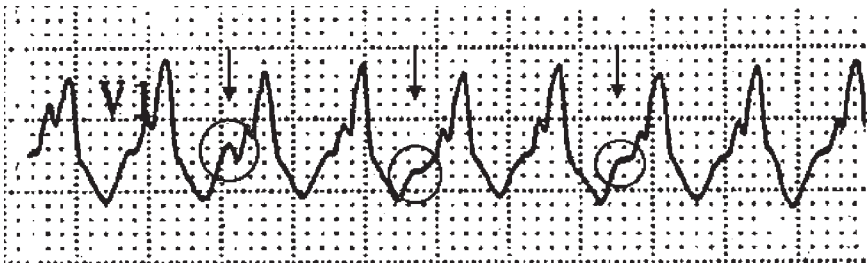
Fig. 53. Example of atrioventricular dissociation with ventricular tachycardia (the P wave is indicated by arrows)

The ECG signs:

— sudden beginning and sudden ending attack of accelerated heart contraction up to 140–250 per minute, while maintaining the correct rhythm in most cases;

— deformation and expansion of the QRS complex more than 0.12 s with a discordant arrangement of the segment RS-T and T wave;

55

Fig. 54. The ECG during ventricular tachycardia

— the presence of atrioventricular dissociation, i. e. the complete separation of frequent rhythm of the ventricles (QRS complex) and the normal rhythm of the atria (P wave) with occasional registration of single unmodified normal QRST complexes of sinus origin ("captured" ventricular contractions) (Fig. 54).

Atrial flutter

Atrial flutter is a significant increase of atrial contractions (up to 250–400) per minute while maintaining regular atrial rhythm. The immediate mechanism, leading to the very frequent excitation of the atria, when atrial flutter is the mechanism of the re-entry wave of excitation, when conditions for long rhythmic circulation of the circular excitation wave are created in the atria (Fig. 55). In contrast to paroxysmal supraventricular tachycardia, when a wave of excitation circulates through the atria with a frequency of 140–250 per minute, in atrial flutter the frequency is higher: 250–400 per minute.

The ECG signs:

— the absence of the P waves on the ECG;

— the presence of frequent — up to 200–400 per minute — regular, similar to each other atrial F waves, which have characteristic saw tooth form (the leads II, III, aVF, V1, V2);

— the presence of normal unmodified ventricular complexes;

— each ventricular complex QRS is preceded by a certain number of atrial complexes (2 : 1, 3 : 1, 4 : 1 etc.) and intervals between QRS are

56

identical in the correct form of atrial flutter (Fig. 56) and in irregular shape (conduction through the AV node changes) the interval between the QRS is different (Fig. 57).

Atrial fibrillation

Fibrillation or atrial fibrillation is a heart rhythm disorder in which frequent (350 to 700) per minute disorderly, chaotic excitation and contraction of separate groups of the muscle fibers of the atria is observed
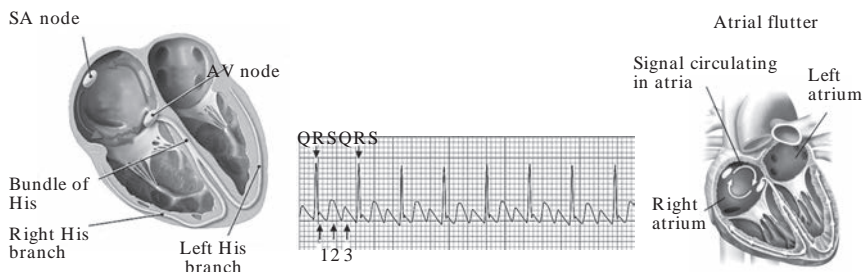
Fig. 55. Mechanism of the AF formation in the right atrium (the circle of re-entry marked in red) with the typical ECG changes (3 : 1 — the number of atrial contractions is 390 and ventricular — 130 min)
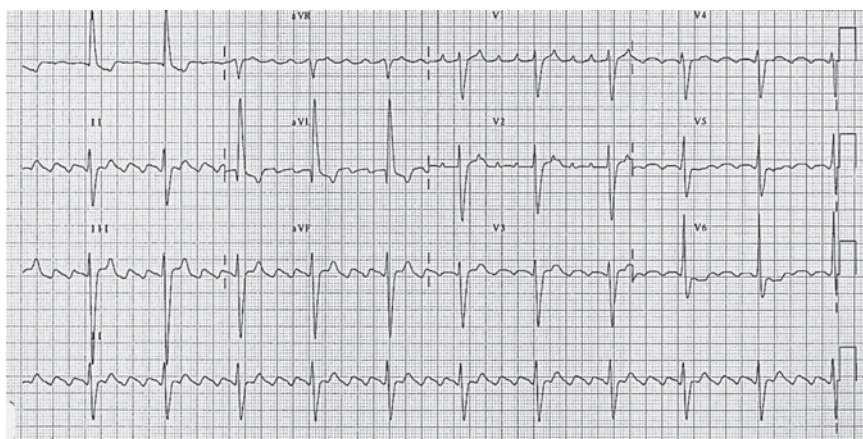
Fig. 56. Correct form of atrial flutter 4 : 1 (contractions of the atria — 260 min, contractions of the ventricles — 65 per min)
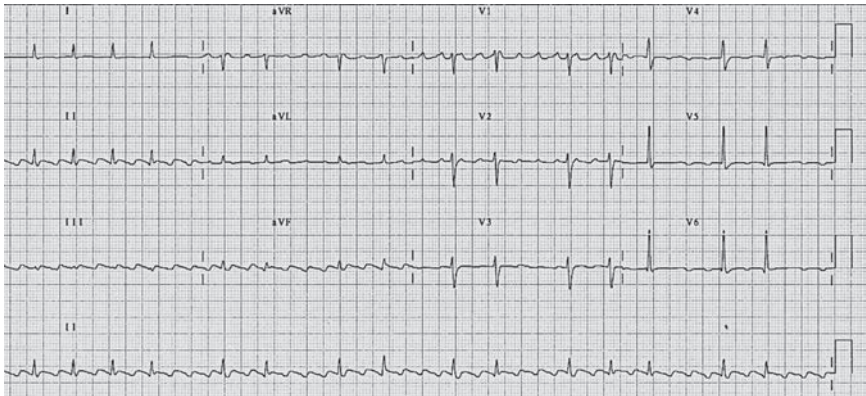
Fig. 57. Atrial flutter in irregular conduction
(QRS intervals are different)

throughout the cardiac cycle. The excitement and contraction of the atria as a single whole is missing.

Depending on the size of waves we distinguish between large and small wave forms of atrial fibrillation. In large wave form the amplitude of the F waves exceeds 0.5 mm, the frequency is 350–450 per minute; they appear relatively more regular. This form of atrial fibrillation is more common in patients with severe hypertrophy of the atria, for example, in mitral stenosis. In small wave form of atrial fibrillation frequency of the F waves reaches 600–700 per minute, their amplitude is less than 0.5 mm. Irregularity of the waves are expressed more sharply than in the first variant. Sometimes the F waves are generally invisible on the ECG in any of the electrocardiographic leads. This form of atrial fibrillation often occurs in older people with cardiosclerosis.

The mechanism of the formation is due to the presence of a focus of automatism at the entry of the pulmonary veins with subsequent re-entry of the impulse of the type micro re-entry in the left atrium or the presence of numerous ways of re-entry in the atria (Fig. 58).

When the heart misfires
Atrial fibrillation occurs the top two chambers of the heart, called the atria, quiver instead of contract, increasing the risk of a stroke.

Rhythm of a healthy heart
1. SA node in right atrium releases an electrical charge, which contracts both atria, goes through the AV node, then contracts the ventricles.

58

2. Charge travels along heart walls, causing a contraction, first in the atrium, then in the ventricle, moving blood through the heart.

3. Right ventricle pumps blood to the lungs, where it is replenished with oxygen; it is returned to the left atrium, then the left ventricle for pumping into the rest of the body.

Atrial fibrillation

1. Erratic charges originate near the pulmonary veins and in the walls of the atria.

2. Charges cause the atrium to quiver; it falls to pump blood fully out of its chamber, allowing pools of blood to form.

3. In time, clots can form in blood pools.

4. Charges also cause ventricles to speed up to 130 or more contractions a minute.

5. Blood clots can travel through the body, eventually blocking a brain artery and causing a stroke.

The ECG signs:

— the absence of the P wave in all leads of the ECG;

— the presence of the "f" waves having different amplitude and shape. The f waves are better recorded in the leads V1, V2, II, III and aVF. Sometimes, instead of the f waves between the ventricular complexes the isoelectric line is recorded;
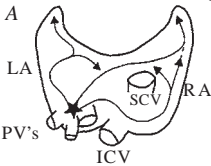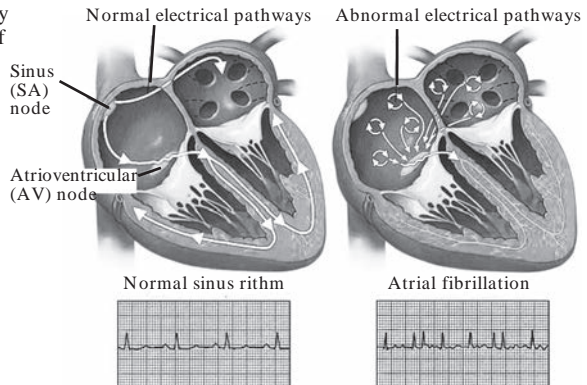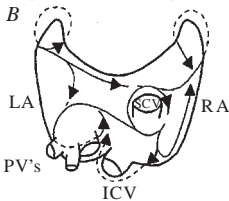
Fig. 58. A mechanism of the formation of atrial fibrillation

— the irregularity of the ventricular complexes QRS (R-R intervals various in duration);

— the presence of the QRS complexes that have, in most cases, the normal unchanged form without deformation and broadening.

There are bradysystolic (the number of ventricular contraction less than 60/min), normosystolic (within 60–90 min) and tachysystolic (heart rate over 90 min) versions of atrial fibrillation (Fig. 59, *a–c*).

Ventricular flutter

Ventricular flutter is frequent (from 150 to 300 per 1 min) rhythmic excitation and contraction of the muscle fibers of the ventricles.

The ECG signs:

— high and wide, of almost equal amplitude, waves passing into each other, in which we cannot distinguish between the P wave, QRS complex, a segment of R(S)-T and T wave.

— short (less than 0.40), of the same or almost the same duration, with the interval between flutter waves (Fig. 60).

Fibrillation of the ventricles

It is chaotic, irregular, frequent (from 250 to 600 per min) excitation and contraction of the separate muscle fibers of the ventricles.

The ECG signs:

— Various amplitude, shape and duration, waves passing into each other, in which we can not distinguish between the P wave, QRS complex, a segment of R(S)-T and T wave.

— Short (less than 0.30) intervals of different duration between the waves of fibrillation.

In the absence of cardiopulmonary resuscitation ventricular fibrillation quickly (within 4–5 min) leads to death (Fig. 61).

CONDUCTION DISORDERS

The transverse blocks (sinoatrial, atrioventricular)

Sinoatrial block

Sinoatrial block is a slowing down of the conduction of the impulses from the sinus node to the atria or blocking them in the area between the sinus node and atrium. There are three degrees of sinoatrial block:

I degree — manifests itself as a delay of the impulse conduction from the sinus node to the atrium. It is not detected on the electrocardiogram.

II degree of SA blocks are of two subcategories just like AV blocks.
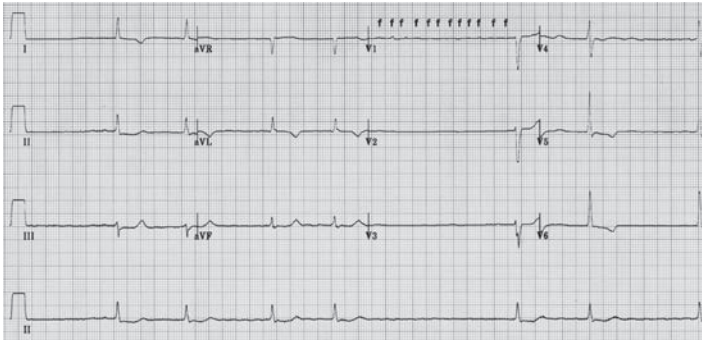
a
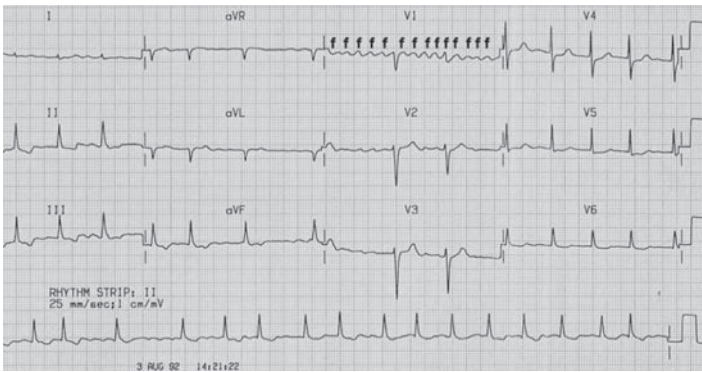
b
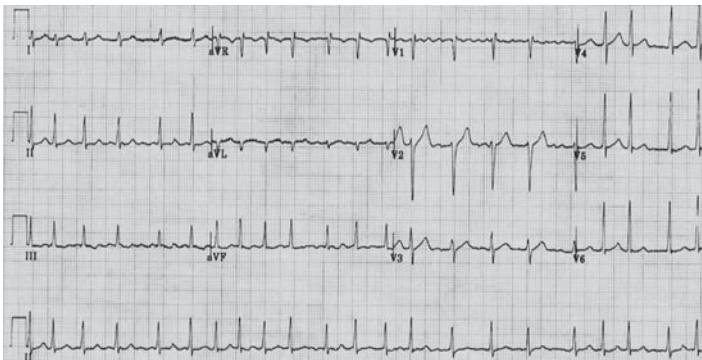
c

Fig. 59. The ECG of bradysystolic*(a)* , normosystolic *(b)* and tachysys-
tolic atrial fibrillation*(c)*
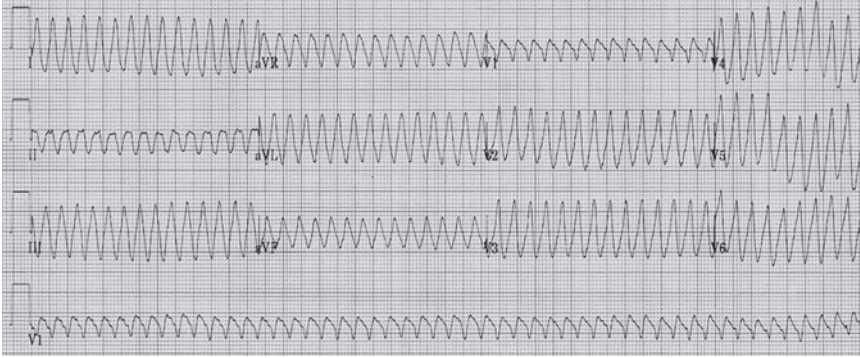
Fig. 60. Flutter of the ventricles (heart rate = 300 per min)
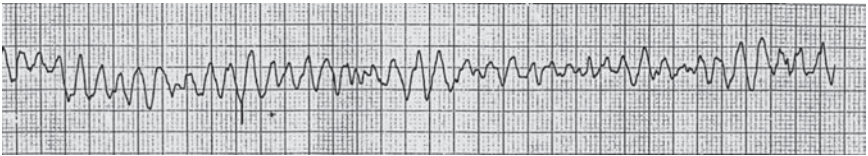
Fig. 61. The ECG during ventricular fibrillation

The first is II degree type I, or Wenckebach block. This rhythm is irregular, and the P-P interval gets progressively smaller, while the P-R interval remains constant, until a QRS segment is dropped. Note that this is quite different from the Wenckebach AV block, in which the P-R interval gets progressively longer, before the dropped QRS segment.

The second degree type II, or sinus exit block, is a regular rhythm that may be normal or slow. It is followed by a pause that is a multiple of the P-P interval. The conduction across the SA node is normal until the time of the pause when it is blocked (Fig. 62).

The third degree sinoatrial block looks very similar to a sinus arrest. However, a sinus arrest is caused by a failure to form impulses. The third degree block is caused by failure to conduct them. The rhythm is irregular and either normal or slow. It is followed by a long pause that is not a multiple of the P-P interval. The pause ends with the P wave, instead of a junctional escape beat the way a sinus arrest would.

Atrioventricular block
Atrioventricular block (AV block) is a partial or total disruption of the electrical impulse conduction from the atria to the ventricles. AV block is

a

b
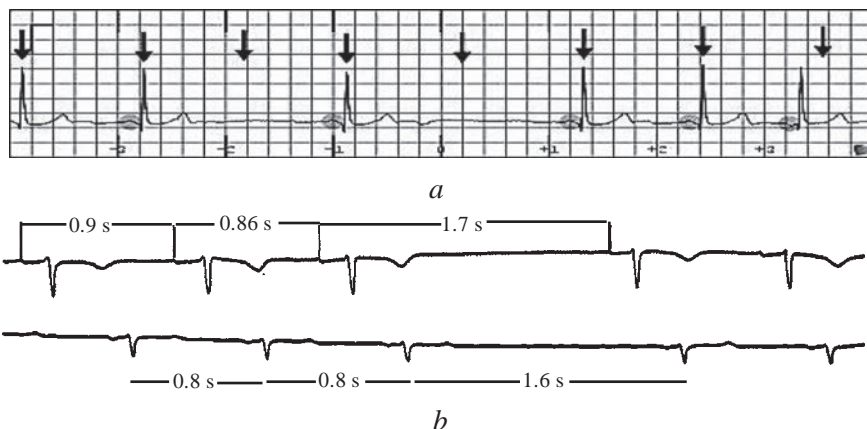
Fig. 62. Sinoatrial block of II degree (type 2). Sinoatrial blockade of II degree: *a* — type 1; *b* — type 2

classified based on several principles. First, taking into account their stability, respectively, AV blockade may be: a) acute, transient; b) intermittent, transient; c) chronic, permanent. Second, we determine the severity or degree of the atrioventricular block. In this regard, there are isolated atrioventricular block of I degree, atrioventricular block II of degree type I and II, and atrioventricular block of III degree (complete).

Atrioventricular block of I degree

It is manifested by slowing down of the impulse conduction from the atria to the ventricles characterized by prolongation of the interval P-Q (R) on the ECG.

The ECG signs:

— heart rate is usually unchanged;

— correct sequence of the P wave and QRS complex in all cycles;

— the interval P-Q (R) is more than 0.22 s;

— the normal shape and duration of the QRS complex (Fig. 63).
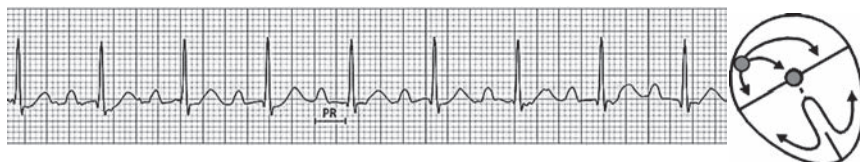
Fig. 63. The ECG with AV block of I degree P-Q (R) = 0.24 s, HR = 76/min

Atrioventricular block of II degree

AV block of II degree is a recurrent cessation of separate impulses from the atria to the ventricles.

There are two main types of atrioventricular block of II degree, type of Mobitz I (with periods of Samoylov–Wenckebach) and the type Mobitz II.

Type of Mobitz I

The ECG signs:

— the same R-R intervals in duration;

— gradual cycle-to-cycle prolongation of the interval P-Q (R) with the subsequent loss of the ventricular QRST complex;

— after the loss of the ventricular complex the normal or lengthened interval P-Q (R) is again recorded on the ECG, then the whole cycle is repeated;

— long pauses equal to the doubled interval R-R.

Periods of gradual increase of the interval P-Q (R) with the subsequent loss of the ventricular complex are called periods of Samoylov-Wenckebach (Fig. 64).

Type of Mobitz II

The ECG signs:

— the same R-R intervals in duration;

— no progressive lengthening of the interval P-Q (R) before blocking of the impulse (stability of the interval P-Q (R);

— loss of separate ventricular complexes;

— long pauses equal to the doubled interval R-R (Fig. 65).
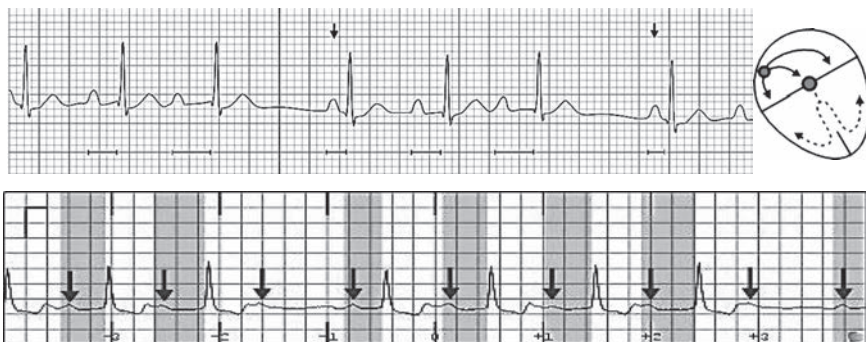
Fig. 64. The ECG of AV block of II degree (Mobitz I) with gradual lengthening of the interval P-Q (R)

a
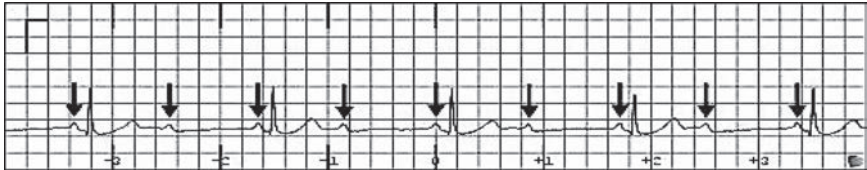
b

Fig. 65. AV block of II degree, type Mobitz II (periodic *(a)* or multiple *(b)* loss of QRS complexes without a preceding lengthening of the interval P-Q (R)

Atrioventricular block of III degree

Atrioventricular block of III degree (complete AV block) is a complete cessation of the impulse conduction from the atria to the ventricles, resulting in excitation of the atria and ventricles and their contraction independently from each other.

The ECG signs:

— the absence of correlation between the P waves and ventricular complexes;

— the intervals P-P and R-R are permanent, but R-R is always greater than P-P;

— the number of ventricular contractions is less than 60 per minute;

— periodic layering of the P waves on the QRS complex and T wave and the deformation of the latter.

If atrioventricular block of I and II degree (type of Mobitz I) can be functional, atrioventricular block of II degree (type of Mobitz II) and III develops against the background of the expressed organic changes of the myocardium and have worse prognosis (Fig. 66).

Syndrome of Frederick

It is a combination of complete atrioventricular block with fibrillation or atrial flutter.

The ECG signs:

— the absence of the P wave before the QRS complexes and the presence of waves of fibrillation (f) or atrial flutter between them;

— widened, deformed QRS complexes;

— the same duration of the intervals R-R (Fig. 67).

Fig. 66. AV block of III degree (complete) — the atria pump in the rhythm with frequency = 96/min, ventricles with frequency = 37/min
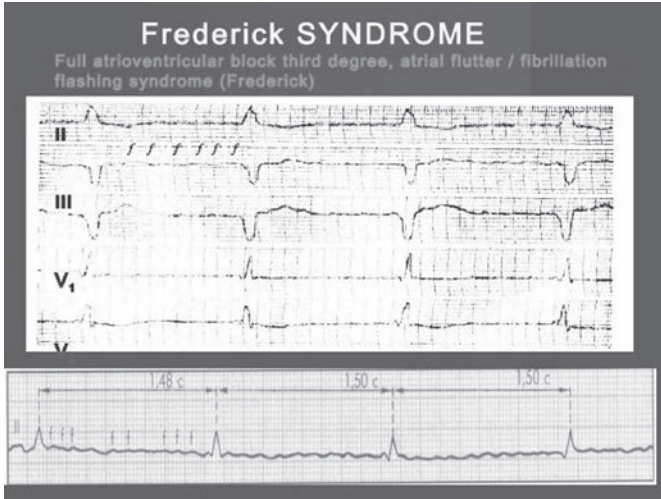
Fig. 67. The ECG with a syndrome of Frederick (ventricular rhythm can be traced with a frequency of contractions 30 per min on the background of the atrial fibrillation)

66

Longitudinal blocks
(His bundle branch block)

A block of His bundle branches and fascicles is a slowing down or complete cessation of the excitation of one, two or three His bundle branches.

The complete cessation of excitation by one or another His bundle branch is evidence of complete block. Partial slowing down of conduction indicates incomplete block of His bundle branch.

Right Bundle Branch Block (complete and incomplete)

The right His bundle branch block (RBBB) is a slowing down or complete cessation of the impulse conduction through the right His bundle branch.

Complete RBBB is cessation of the impulse conduction through the right His branch of bundle.

The ECG signs:

— the presence of complexes QRS of the type rSR" or rsR", with M-shaped form, with R">r in the right chest leads V1, 2;

— the presence of the broadened, often split S wave in the left chest leads (V5, V6) and leads I, aVL;

— increasing of the internal deviation time in the right chest leads (V1, V2) larger than or equal to 0.06 s;

— increased duration of the ventricular complex QRS greater or equal to 0.12 s;

— the presence of depression of the segment S-T and negative or biphasic (–+) asymmetric T wave in the lead V1 (Fig. 68).
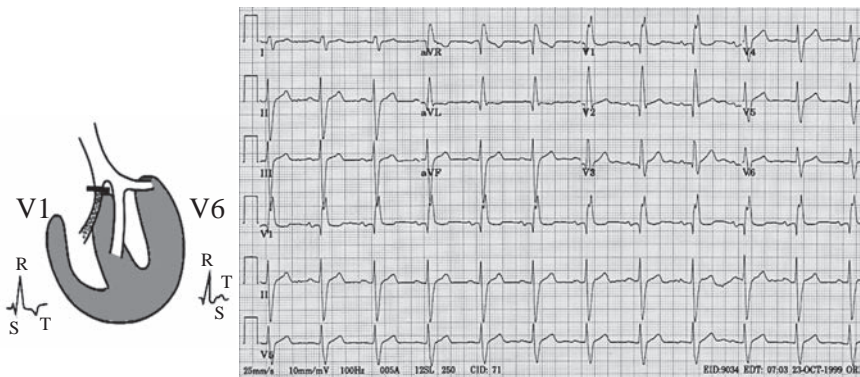
Fig. 68. A diagram and ECG, with complete right bundle branch block

Incomplete right bundle branch block

Incomplete right bundle branch block is a slowing down of the impulse conduction in the right His bundle branch**.** The ECG signs of incomplete RBBB are similar to those of hypertrophy of the right ventricle (see above)

The ECG signs:

— the presence of the QRS complex of the type rSr" or rsR"— "rabbit ear" in the lead V1;

— the presence of the slightly widened wave S in the left chest leads (V5, V6) and in the leads I;

— the internal deflection in the lead V1 of not more than 0.06 s;

— the duration of the ventricular QRS complex less than 0.12 s;

— the segment S-T and T wave in the right chest leads (V1, V2) generally not change (Fig. 69).

Block of the left His bundle branch

The left bundle branch block is a slowing down or complete cessation of the impulse conduction in the left branch of His bundle.

Complete left bundle branch block

Left bundle branch block is the termination of the impulse conduction in the left branch of His bundle.
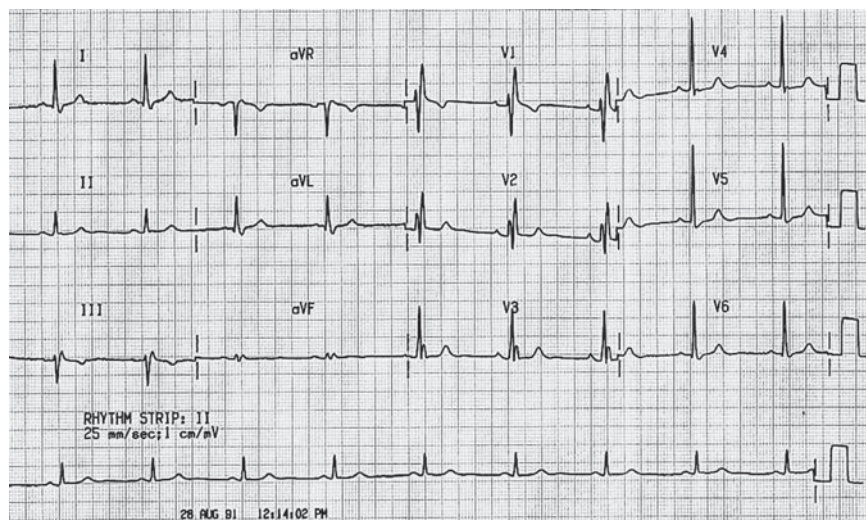
Fig. 69. The ECG with incomplete RBBB (typical changes of the QRS in the leads V1, V2-rSR, the width of the ventricular complex is 0.11 s)

The ECG signs:

— the presence of the widened deformed ventricular complexes of the R type with split or a wide top in the left chest leads (V5, V6), I, aVL;

— the presence of the widened deformed ventricular complexes, having the form of QS or rS with split or wide top of the S wave in the leads V1, V2, III, aVF;

— the internal deviation in the leads V5,6 more than or equal to 0.08 seconds;

— the increase of the total duration of the QRS complex (more than or equal to 0.12 s);

— the presence of discordant in relation to the QRS deviation of the segment S-T and negative or biphasic (–+) asymmetrical T waves in the leads V5,6, I, aVL;

— the absence of q in I, aVL, V5,6 (Fig. 70, 71).
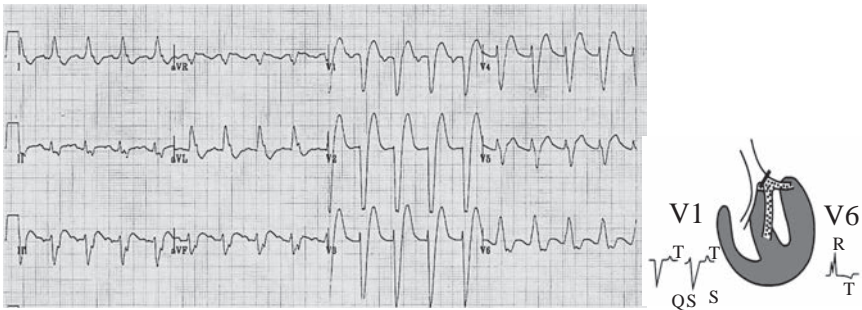
Fig. 70. A diagram and ECG of the complete LBHB
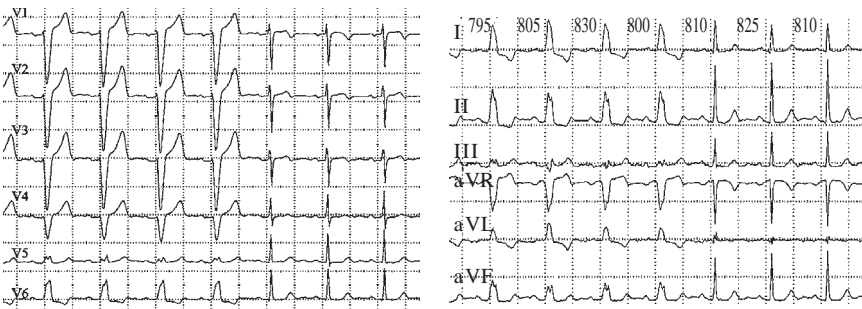
Fig. 71. The ECG demonstration of spontaneous termination of the complete LBBB

Incomplete left His bundle branch block

Incomplete block of the left bundle branch is a slowing down of the impulse conduction in the left His bundle branch.

The ECG signs:

— the presence of the high, widened, sometimes split R waves in the leads I, aVL, V5, 6 (wave q V6 is missing);

— the presence of the widened and deepened complexes such as QS or rS, sometimes with the initial splitting of the S wave in the leads III, aVF, V1, V2;

— the internal deviation in the leads V5,6 0.05–0.08 s;

— the total duration of the QRS complex 0.10–0.11 s;

— no q V5-6.

Due to the fact that the left branch is divided into two branches: anterior and posterior we distinguish block of the anterior and posterior branches of the left bundle branch.

During the block of the anterior branch of the left bundle branch there is impaired conduction of excitation to the anterior wall of the left ventricle. Excitation of the myocardium of the left ventricle proceeds as if in two stages: first excited interventricular septum and the lower portions of the back wall, and then antero-lateral wall of the left ventricle.

The ECG signs:

— sharp deviation of the electrical axis of the heart to the left;

— QRS in the leads I, aVL type qR in III, aVF type rS;

— the total duration of the QRS complex is 0.08–0.011 s (Fig. 72).
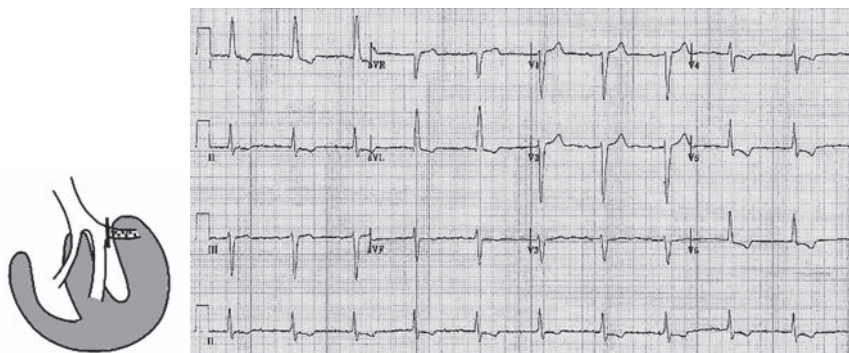
Fig. 72. The ECG in the block of the anterior branch of LBBB (sharp deviation of the axis to the left, QRS in the leads I, aVL type qR, QRS=0.11 s)

In the block of the **left posterior His branch bundle** the sequence of coverage by excitation of the myocardium of the left ventricle is changed. At first the excitation is conducted freely in the left anterior His bundle branch, quickly covers the myocardium of the anterior wall and only after that, through anastomoses of the Purkinje fibers, extends to the myocardium of the low back segments of the left ventricle.

The ECG signs:

— sharp deviation of the electrical axis of the heart to the right;

— the QRS complex in the leads I and aVL rS type, and in the leads III, aVF — type qR;

— QRS complex duration in the range of 0.08–0.11 s (Fig. 73).

Syndrome of premature ventricular excitation (WPW, CLC syndromes)

It results from the simultaneous conduction of the excitation impulse through the main conduction system and additional conduction pathways that bypass the AV node. In the syndrome of Wolff – Parkinson – White (WPW) the impulse is conducted to the ventricles via additional abnormal bundles of Kent, in the syndrome of the shortened interval P-Q(R) — through the bundle of James (syndrome Clerk-Levy-Cristesco or ÑLC syndrome). These syndromes are typically accompanied by a rhythm disturbance in the form of paroxysm of tachycardia with frequency above 150 per min. In addition, there may be distinguished the concept of WPW
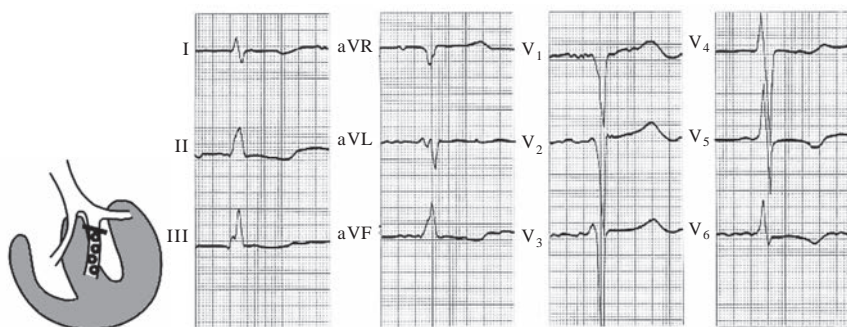
Fig. 73. The ECG in the block of the posterior branches LBBB (a sharp deviation of the heart axis to the right, complex QRS in the leads I and aVL rS type)

phenomenon when the patient has typical ECG changes, but without attacks of tachyarrhythmia.

The ECG signs of WPW syndrome:

1. Shortened (less than 0.12 s) interval P-Q(R).

2. The presence of the Delta waves on an upward or downward bend of the QRS complex.
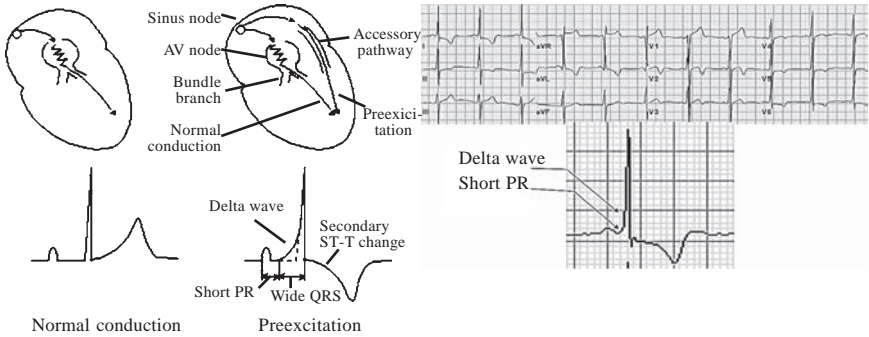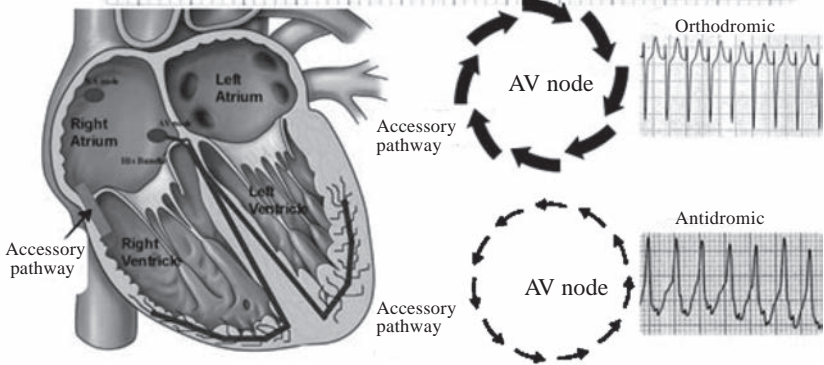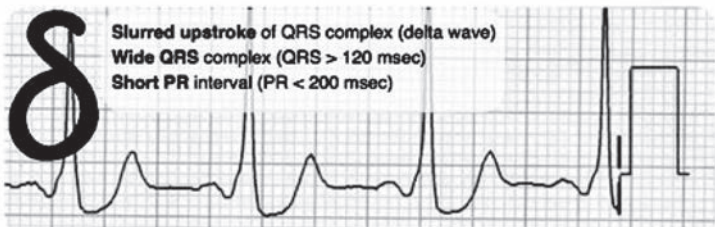
Fig. 74. The scheme of formation of the typical ECG changes and ECG in the WPW syndrome
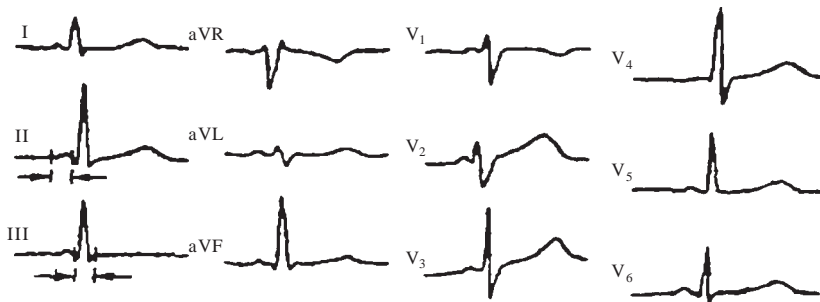
Fig. 75. The ECG in the CLC syndrome

3. Widening (more than 0.11 s) and a small deformation of the QRS complex.

4. Discordant displacement of the segment R(S)-T and T wave (asymmetric biphasic or negative) relative to the main wave of the QRS complex (unstable characteristics) (Fig. 74).

In the CLC syndrome shortening of the interval PQ (R) without the presence of a Delta wave is observed, widening of the QRS complex and changes of repolarization (Fig. 75).

COMBINED ARRHYTHMIAS

Parasystole

Parasystole is a special type of arrhythmia that is characterized by a heterotopic focus, functioning independently of the main pacemaker. Clinically it is manifested in the form of extrasystoles or ectopic tachycardia (paroxysmal or nonparoxysmal). A parasystolic center generates impulses in a certain rhythm. The impulses from the parasystolic center, catching the myocardium outside of the refractory phase, causes its excitation and contraction. The parasystolic center is protected from the penetration of the impulses of the primary (usually sinus) rhythm of the so-called block of the entrance. This heart rhythm disturbance is typical of exit block that prevents the spread of the ectopic impulses from the parasystolic center. The parasystolic center may be localized in: atria; atrioventricular junction; ventricles.

The ECG signs:

— independence of the ectopic complexes on the main rhythm, which is manifested by variability of extrasystolic interval.

— permanence of the shortest inter-ectopic interval or the presence of a common denominator in the distances between extrasystoles. Fluctuations of inter-ectopic intervals, which duration usually does not exceed 130 ms;

— the presence of fusion beats, resulting in the simultaneous occurrence of the impulses of the primary and ectopic pacemaker (Fig. 76, 77).

THE ECG OF THE IMPLANTED ELECTROCARIOSTIMULATOR (ARTIFICIAL PACEMAKER)

Because of the increasing use of permanent electrocardiostimulation we more often see patients with implanted pacemaker of the simplest type (with a given frequency of pulse generation) and complex multiprogram devices (Fig. 78).
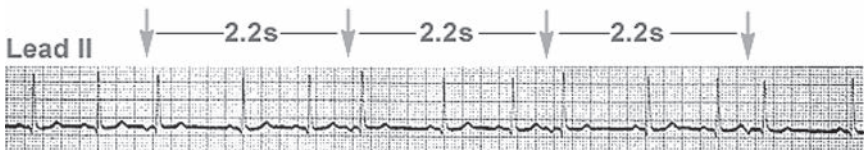
Atrial parasystole (note common interectopic interval of 2.2s)

Fig. 76. Atrial parasystole (additional focus in the atrium, inter-ectopic interval 2.2 s)
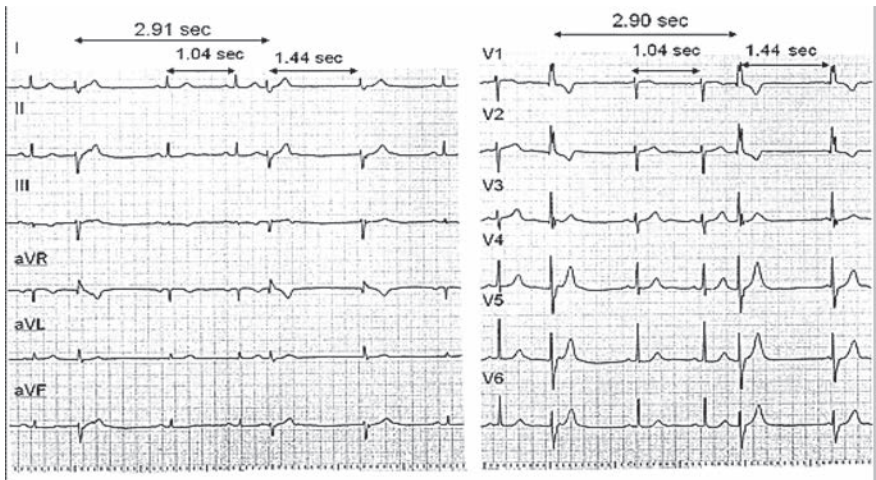
Fig. 77. Parasystole (the pacemaker from the sinus node: the R-R interval 1.04, and from the ventricles: R–R interval=1.44 s)
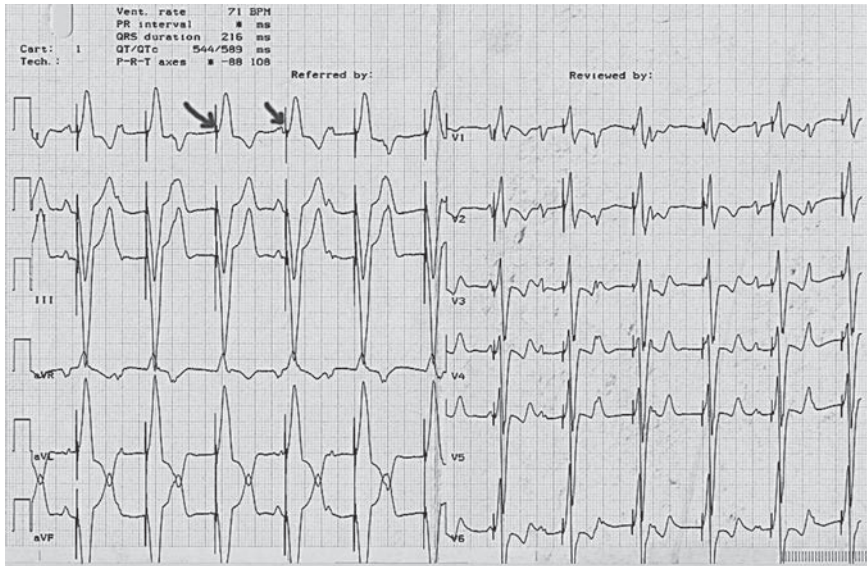
Fig. 78. The ECG of the patient with complete AV block and pacemaker records adhesions of stimulation (arrows), after which widened ventricular complexes QRS are observed

THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF ISCHEMIC HEART DISEASE

The ECG changes of ischemic heart disease (IHD) is caused by a combination of various causes, resulting in hypoxic, anoxic and dysmetabolic processes in the myocardium, insufficient energy supply, leading to damage and death of myofibrils. These pathological changes disturb the bioelectric processes in the myocardium that are detected during electrocardiographic study.

The ECG is the main instrumental method of diagnosis of coronary heart disease. It gives the possibility to distinguish different forms of this disease and to register the clinical findings. It should be noted, however, that many ECG signs of IHD are not strictly specific and may be similar to myocarditis, pericarditis, cardiomyopathy, wounds, tumors of the heart, etc. Therefore, the ECG is just only one of the methods, which in combination with clinical and other examination methods of the patient, ensure the establishment of a reliable diagnosis.

For the clinical interpretation of the key ECG changes in various forms of ischemic heart disease, there is a scheme of Zuckerman (Zuckerman, 1957), according to which the main signs of myocardial ischemia are changes of the T wave, degenerative damages (focal coronarogenic dystrophy) — changes of the ST segment, necrosis (infarction) — changes of the QRS complex. This scheme does not reflect the diversity of the electrocardiographic symptoms of IHD, but allows you to understand the general patterns of the ECG dynamics. You should also consider the duration of the ECG changes. So, short deviation of the ST segment and the deformation of the T wave can be interpreted as a sign of myocardial ischemia. More prolonged dynamics of the same electrocardiographic symptoms should be regarded as evidence of more severe myocardial damage.

Deformation of the T wave and deviation of the ST segment, typical of IHD in damages of the anterior wall of the left ventricle are seen in the leads I, II, aVL, V1-4, the lateral wall -in the leads V5-6, posterior wall — in the leads III and aVF.

Fig. 79 and 80 show the options of configuration of the T wave and the dislocation of the ST segment in ischemic heart disease.

Deformation of the ventricular complex QRS in IHD is indicative of the loss of a substantial part of the contractile myocardium (big focal necrosis, postmyocardial infarction cardiosclerosis). This is indicated by the presence of the pathological Q wave or QS complex formation. It should be noted that these signs are not strictly specific to IHD and can occur in hypertrophy of the ventricles, asymmetric hypertrophic cardiomyopathy (Fig. 81).

Fig. 79. Changes of the T wave in coronary heart disease:

a — normal T wave; *b, c, d* — characterizes intramural ischemia; *e* — the most specific of IHD, describes subepicardial or transmural ischemia; *f* — characterizes the ischemic process on the opposite to electrode wall of the left ventricle (the presence of such wave in the chest leads may indicate ischemia of the posterior wall of the left ventricle) or the upper subendocardial ischemia of the anterior parts of the myocardium

76
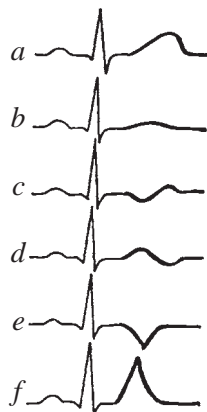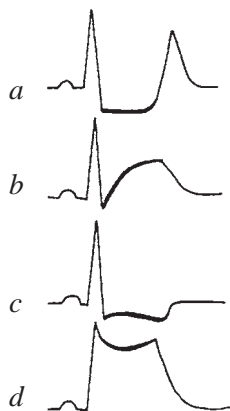
Fig. 80. Variants of changes of the ST segment in ischemic heart disease:

a — specific of ischemic heart disease, characterizes ischemia or subendocardial injury; *b* — less specific of IHD, may occur in healthy people during exercise; *c* — less specific of IHD, may occur in cardiac glycoside intoxication; *d* — specific of ischemic heart disease, is considered as an indicator of "damaged tissue" in the subepicardial region
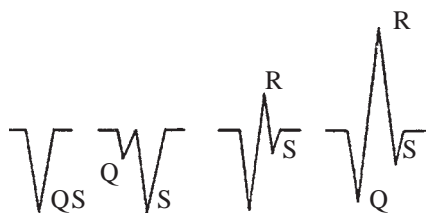
Fig. 81. Variants of deformation of the ventricular complex (QRS) in IHD

ELECTROCARDIOGRAM CHANGES
IN VARIOUS FORMS OF IHD

Diffuse atherosclerotic cardiosclerosis. It is characterized by the following ECG changes (Fig. 82):

— signs of left ventricular hypertrophy, which, however, does not reaches the degree, taking place in hypertension or aortic defects;

— reduction of (in progression of diffuse sclerotic process) voltage of all waves in all ECG leads;

— the instability of the ST segment and T wave due to the increase or reduction of ischemia;

— disturbance of intraatrial conductivity (increase of the P wave duration more than 0.11 s);

— disorders of the atrioventricular and intraventricular conduction (blockades of different levels and different degrees of His bundle branches);

— rhythm disorders, among which extrasystolic arrhythmia, fibrillation and atrial flutter is more common.
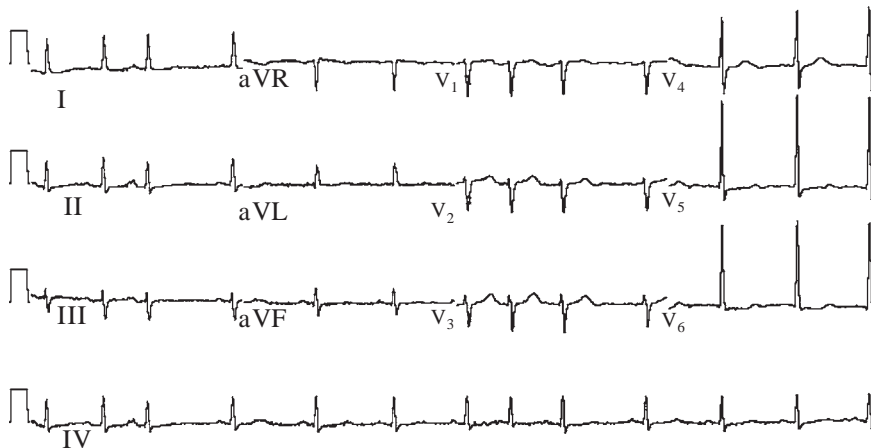
Fig. 82. The ECG of the patient aged 72 with IHD: diffuse cardiosclerosis (atrial fibrillation, signs of hypertrophy of the left ventricle, disturbances of repolarization in the anterior wall of the left ventricle in the leads I, II, aVL, V5-6)

Angina. In patients with IHD during angina attack the ECG changes may be absent or remain undocumented due to their short duration. In other cases, there is inversion or increase in the amplitude of the T wave and dislocation of the ST segment. After the attack these ECG changes usually disappear quickly and sometimes persist for several hours or 1–2 days (Fig. 83, *b*).

Spontaneous angina (Prinzmetal's angina) is a variant of unstable angina and it is characterized by the development of pain, persisting after taking nitroglycerin. The pain usually occurs in the morning, at 4–5 a.m. The occurrence of this form of angina is based on the spasm of the coronary arteries. During the attack there is a marked ST-segment elevation on the ECG, followed by its rapid (within days) return to the isoline (Fig. 84, *b*). Laboratory data are unchanged. In the interictal period a patient tolerates normal physical activity well.

Myocardial infarction. According to the size of the necrotic area large-focal myocardial infarction is distinguished, which is accompanied by changes of the QRS complex, and small-focal one, not accompanied by changes of the QRS complex (in the English literature it is referred to as "myocardial infarction without Q wave " or " non Q infarction").

According to the depth of penetration and location of the necrotic area in the thickness of the ventricular myocardium we distinguish transmural, intramural, subendocardial, subepicardial infarction (Fig. 85).
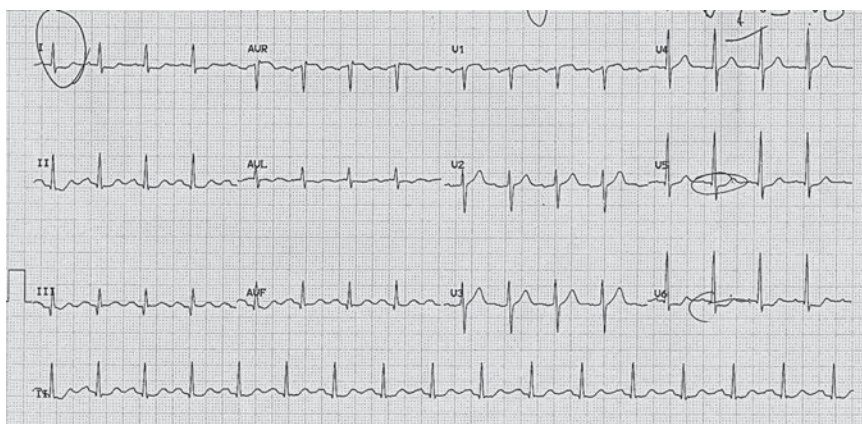
a
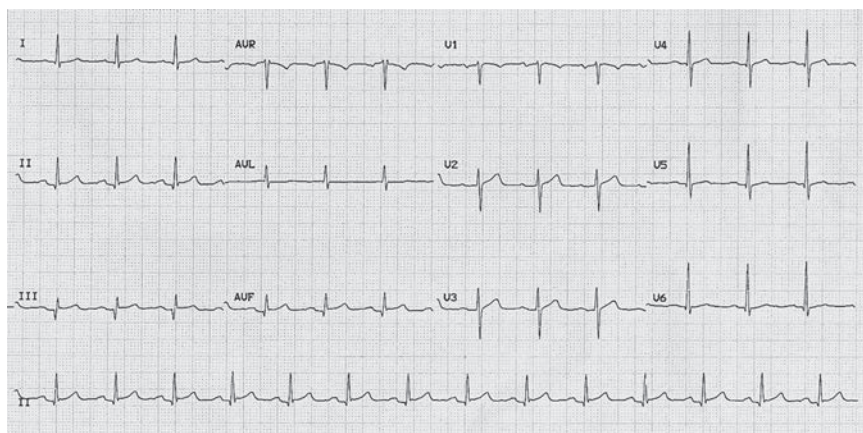
b

Fig. 83: a — the ECG during the ischemic attack (there is a depression of the ST segment in the leads I, II, V5-6, negative T— in aVL); *b* — the ECG after the ischemic attack in patients receiving nitrates (normalization of the ST segment in the leads I, II, V5-6, low-amplitude T in I, aVL, V5-6 remains)

In myocardial infarction the amount of the necrotic tissue and the degree of the ischemic damage decreases towards the periphery of the damage, which is characterized by three sequentially passing each other zones — the area of necrosis (causes changes of the QRS complex), the
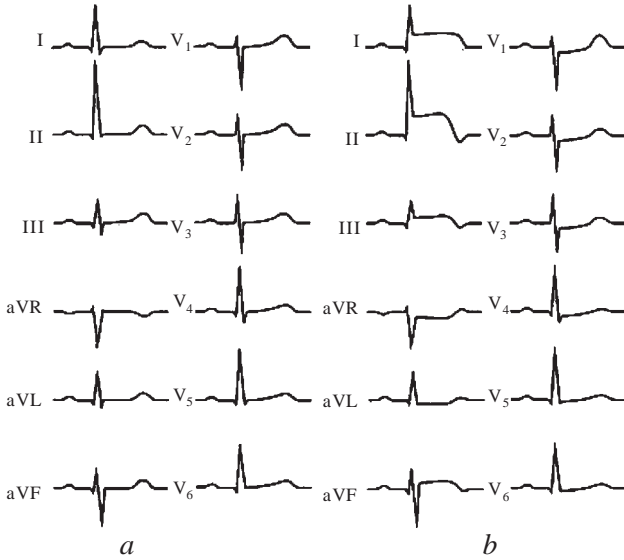
Fig. 84. The electrocardiogram of the patient before *(a)* and during *(b)* Prinzmetal's angina (typical changes — the ST segment elevation)
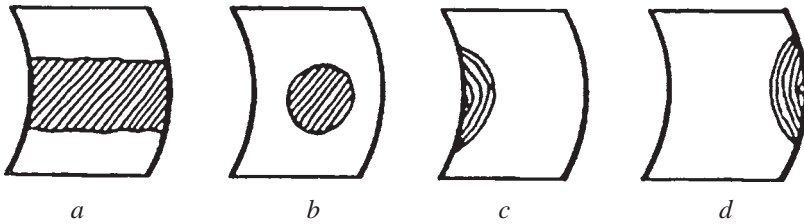
Fig. 85: *a* — transmural; *b* — intramural; *c* — subendocardial; *d* — subepicardial

area of damage (mainly resulting in the dislocation of the segment ST), the area of ischemia (causes changes of the T wave) (Fig. 86).

From the clinical point of view it is accepted to distinguish "Q infarction" (large-focal or transmural), which is characterized by the presence of areas of necrosis and "non Q infarction" where there are only areas of damage and ischemia without necrosis.
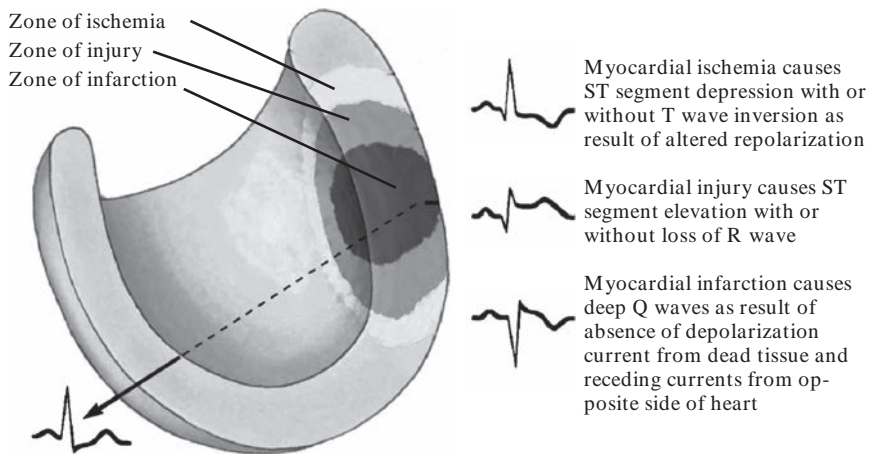
Fig. 86. Main zones in large-focal infarction (area of necrosis, damage, ischemia) and corresponding ECG changes

The ECG in "Q infarction"

In **large-focal myocardial infarction** the necrotizing myocardium completely loses its electrical activity and its EMF drops out of the total EMF of the ventricles. The resulting sum vector of the QRS complex is deviated to the opposite infarcted section side, which is manifested on the ECG in the leads over the damage with increased initial electronegativity (recorded deep and wide, i. e. pathological Q wave) and a decrease of the R wave amplitude. On the contrary, in the leads opposite to the area of infarction (reciprocal leads), the amplitude of the R wave may increase (see Fig. 86).

The Q wave is considered pathological if it is recorded in the leads V1 -3, where it is absent in the normal conditions, if the duration is more than 0.03 s, the amplitude in the leads III and aVF exceeds 25%, and in the leads V5-6 — 15% of the R wave.

Due to the fact that the surrounding necrotic area of dystrophy is not excited and is positively charged during depolarization relative to the healthy tissue, the ST segment in the leads "over the infarction" is shifted upward, and in the leads opposite to infarction — downward in the form of a monophasic curve, merging with the T wave (Fig. 87).

Inversion observed in the evolution of the ECG and then the positive dynamics of the T wave, respectively are due to ischemia of the
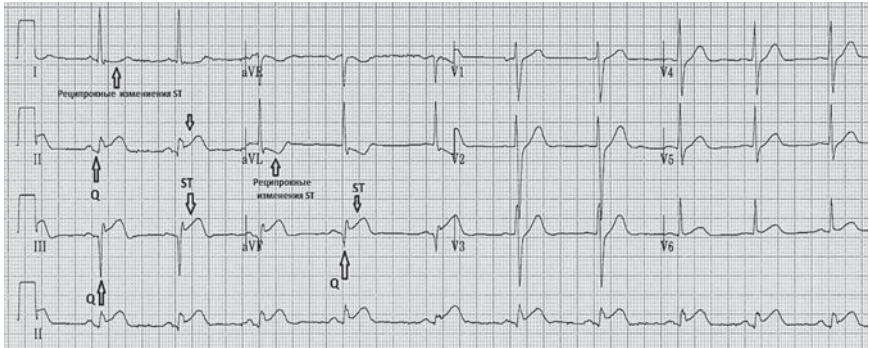
Fig. 87. The ECG of large-focal myocardial infarction (Q infarction): a pathologic Q and ST-segment elevation in the leads II, III, aVF, reciprocal changes of the ST segment in the leads I, aVL

periinfarction area and improvement of the circulation in it (Fig. 87).

Transmural myocardial infarction. It is characterized by the QS complex (instead of the QRS complex) in the leads corresponding to the area of infarction (during the entire period of depolarization of the ventricles, the sum vector is pointed to the opposite area of the necrosis side) with increasing amplitude of the R wave in the leads opposite to the area of infarction (vector excitation, directed toward these leads, does not experience opposition from the vector of the opposite wall of the heart) (Fig. 88).
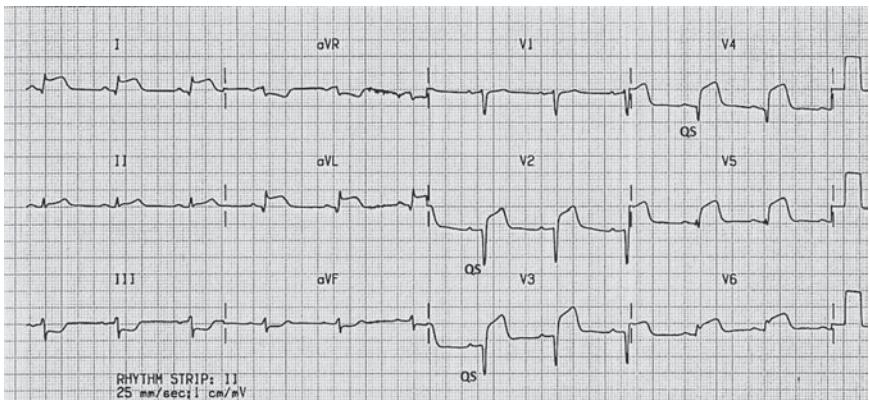
Fig. 88. The electrocardiogram of transmural infarction — the QS complex in the leads V2-4 is recorded

82

The electrocardiogram in small-focal "non Q infarction"

The ECG in small-focal ("non Q infarction") is characterized by depression of the ST segment with the arc, which is convex downwards and long-term (more than 2 weeks) dynamics of these changes. The QRS complex is not changed, as the ECG registered from the chest surface reflects the process of the passage of the excitation wave only on the outside (subepicardial), undamaged layer of the myocardium (Fig. 89).

Topical diagnosis of myocardial infarction

A topical diagnosis of myocardial infarction by the ECG is made with peculiarities of blood supply of the heart. In most cases it is carried out in two coronary arteries (left and right) starting directly from the aorta above the semilunar valves. The left coronary artery (LCA) is a wide but short arterial trunk (left main coronary artery stem, LMCA) of about 11 mm in length and with ramifications. Usually the arterial trunk (LMCA) is divided into two, rarely three or four arteries, the main value of which has a small-focal anterior interventricular branch (left anterior descending coronary artery, LAD) and left circumflex coronary artery (LCx).

The descending coronary artery passes on the anterior interventricular sulcus to the apex of the heart, where it anastomoses with the right coronary artery (RCA). It moves away from its numerous septal branches (septal artery, S1, S2, etc.), supplying the front part of the interventricular septum, and diagonal artery (D1, D2, etc.), branching in the anterior wall of the left ventricle.
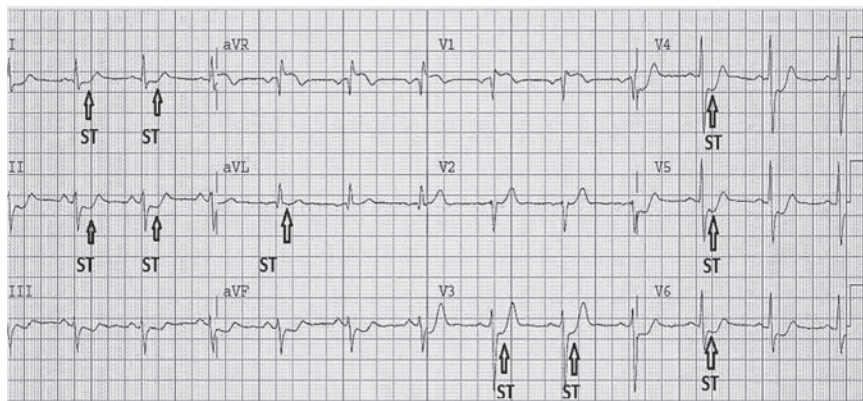
Fig. 89. The ECG in small-focal ("non Q infarction") — typical changes of the ST segment are recorded

The left circumflex coronary artery (LCx), heading for the posterior surface of the left ventricle, gives branches to the anterior and posterior papillary muscles, anterior, lateral, posterior and lower wall of the left ventricle, sinoauricular node. Of great importance is its regional branches supplying the postero-lateral surface of the left ventricle.

At the beginning the right coronary artery (RCA) pass on the outer surface of the right ventricle, and then as the posterior descending artery descends on the posterior surface of the heart to its apex, where it anastomoses with the left descending coronary artery (LAD). It supplies the anterior, lateral, inferior wall of the right ventricle, and the lower and posterior wall of the left ventricle and the lower part of the interventricular septum (Fig. 90).

Taking into account affection of the coronary arteries, as a rule, there are four main types of localization of myocardial infarction of the left ventricle:

1) anterior — in which direct changes are recorded in the leads I, aVL, V2-V4 (Fig. 91);

2) inferior (postero-diaphragmatic)— changes in the leads II, III, Avf (Fig. 92);

3) the lateral — with direct changes in the leads I, aVL, V5-V6 (Fig. 93);

4) the postero-basal (posterior) — in which there are no direct changes in 12 conventional ECG leads, and in the leads V1-V3 reciprocal changes (a high, narrow wave R, depression of the ST segment, sometimes —
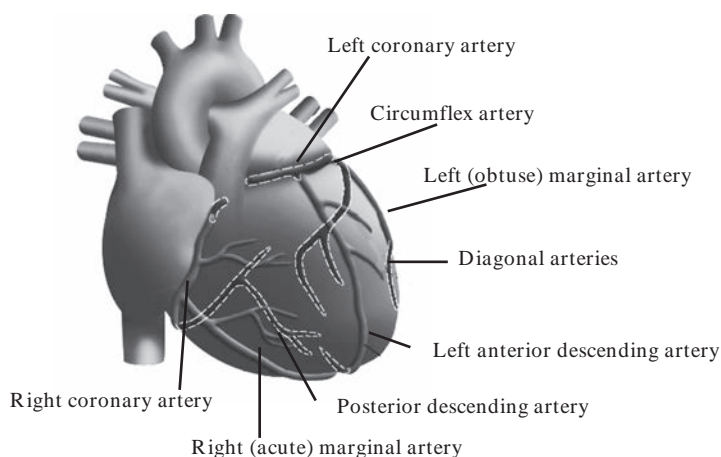
Fig. 90. Coronary arteries of the heart and blood supply
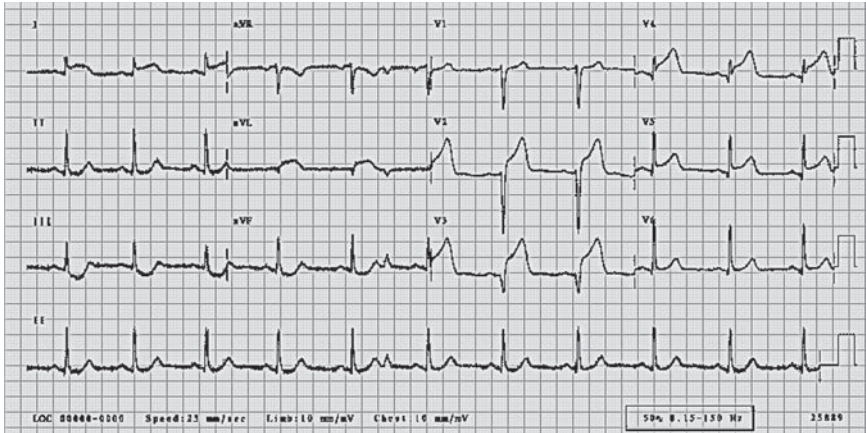
Fig. 91. The ECG of the acute phase of anterior MI (formation of the pathologic Q wave and ST rise in the leads I, aVL, V2-V4)
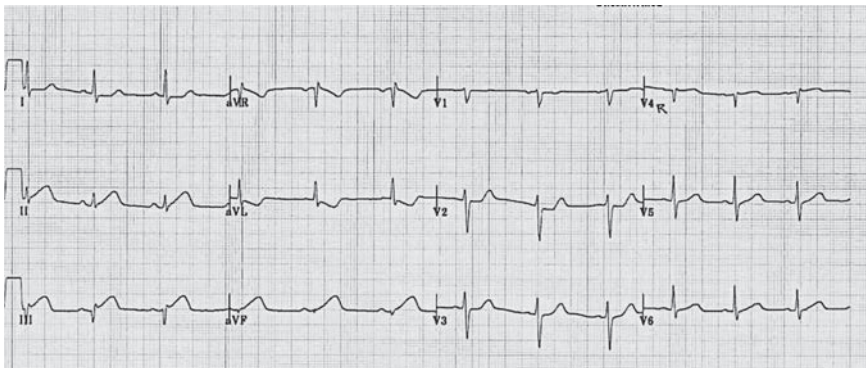
Fig. 92. The ECG in acute inferior (postero-lateral diaphragmatic) myocardial infarction

a high, pointed wave T) are recorded (Fig. 94). Direct changes can only be detected in the additional leads D, V7-V9.

Myocardial infarction of the right ventricle. It is not commonly isolated. Usually it is associated with myocardial infarction of the posterior wall of the left ventricle. The ECG is revealed with difficulty. The following few specific features are seen in the conventional leads: V1-4 ST elevation in the leads AVR. For a diagnosis the right chest leads are used: V3R-4R detects the ST-segment elevation and, in some cases, pathological Q wave (Fig. 95).
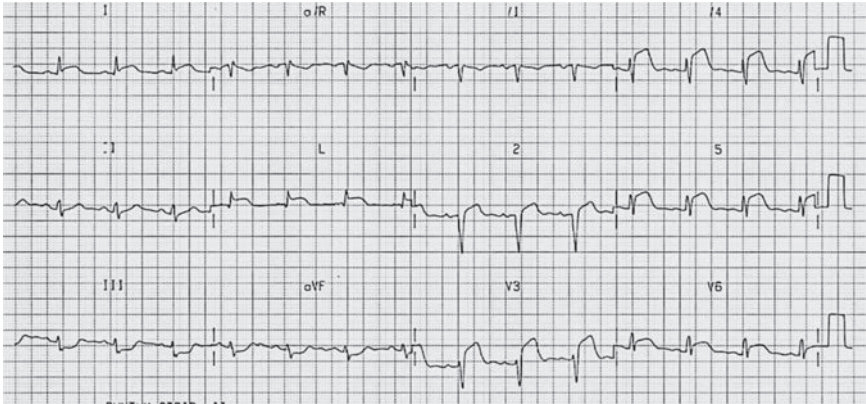
Fig. 93. The ECG in acute lateral MI
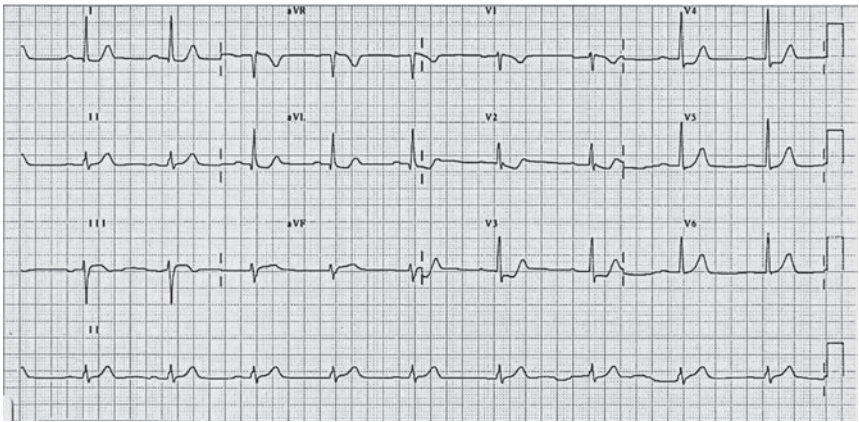
Fig. 94. The ECG in acute posterior-basal (posterior) MI

Topical diagnosis of myocardial infarction on the basis of coronary artery affections is presented in Table 3.

An important criterion of the electrocardiographic diagnosis of myocardial infarction is dynamics of the waves Q, R, T and ST-segment changes. This allows not only to diagnose the stage of myocardial infarction, but also to exclude a number of diseases (hypertrophic cardiomyopathy, aortic defects, etc.) in some cases, there may be infarct-like changes on the ECG changes, but with no ECG changes on re-admission (i. e., there is no criterion of the dynamics).
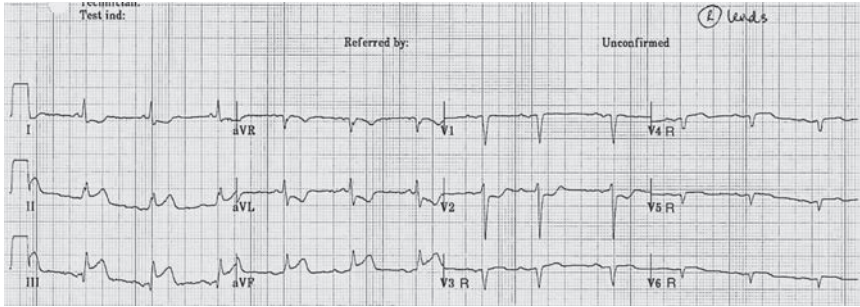
Fig. 95. The ECG of the right ventricle MI (elevation of the ST segment in the leads II, III and right chest leads V3R-V5R with the formation of QS complex)

Table 3

ECG Changes during Myocardial Infarction (MI)

Location of MI	Leads Affected	Vessel Involved	ECG Changes
Anterior wall	V_2 to V_4	· Left Anterior Descending artery (LAD) · Diagonal branch	· Poor R wave progression · ST-segment elevation · T wave inversion
Septal wall	V_1 and V_2	· Left Anterior Descending artery (LAD) · Septal branch	· R wave disappears · ST-segment rises · T wave inverts
Lateral wall	I, aV_L, V_5, V_6	· Left Coronary Artery (LCA) · Circumflex branch	· ST-segment elevation
Inferior wall	II, III, aV_F	· Right coronary Artery (RCA) · Posterior descending branch	· T wave inversion · ST-segment elevation
Posterior wall	V_1 to V_4	· Left Coronary Artery (LCA) · Circumflex branch · Right Coronary Artery (RCA) · Posterior descending branch	· Tall R waves · ST-segment depression · Upright T waves

Wall affected	Leads showing ST segment elevation	Leads showing reciprocal ST segment depression	Suspected affected artery
Septal	V_1, V_2	None	Left Anterior Descending (LAD)
Anterior	V_3, V_4	None	Left Anterior Descending (LAD)
Anteroseptal	V_1, V_2, V_3, V_4	None	Left Anterior Descending (LAD)
Anterolateral	V_3, V_4, V_5, V_6, I, aVL	II, III, aVF	Left Anterior Descending (LAD), Circumflex (LCX), or Obtuse Marginal
Extensive anterior (Sometimes called Antero-septal with Lateral extension)	V_1, V_2, V_3, V_4, V_5, V_6, I, aVL	II, III, aVF	Left main coronary artery (LCA)
Inferior	II, III, aVF	I, aVL	Right Coronary Artery (RCA) or Circumflex (LCX)
Lateral	I, aVL, V_5, V_6	II, III, aVF	Circumflex (LCX) or Obtuse Marginal
Posterior (Usually associated with Inferior or Lateral but can be isolated)	V_7, V_8, V_9	V_1, V_2, V_3, V_4	Posterior Descending (PDA) (branch of the RCA or Circumflex (LCX))
Right ventricular (Usually associated with Inferior)	II, III, aVF, V_1, V_4, R	I, aVL	Right Coronary Artery (RCA)

During the Q myocardial infarction there are the following stages:

— **Ischemic —** the duration of 15–30 minutes from the beginning of a painful attack with the formation of the focus of subendocardial ischemia, which affects the picture on the ECG — the high peaked T wave in the leads over the affected area. In practice it is rarely seen;

— **The acutest —** lasting from 15–30 minutes up to 1–3 hours is characterized by the appearance of the affected focus. On the ECG: the elevated ST segment above the isoline and its fusion with a high T wave in the form of a monophasic curve (the curve of Pardee, "cat back") (Fig. 96 — *1;* 97);

— **Acute** — lasting from 1–3 hours up to 1–3 days — the formation of zones of necrosis, which is reflected in the formation of the pathological Q wave on the ECG and monophasic curve ST-T (in reciprocal leads lower of isoline arc downward). By the end of this phase the ST approaches the isoline, the delimitation of the affection zones and ischemia starts — the first signs of the formation of the coronary T wave (Fig. 96 — *2, 3, 4;* 98);

— **Subacute** — from 1–3 days to 1–3 weeks: the affected zone gradually decreases in size. A pathological Q wave remains on the ECG, but QS complexes may be replaced with Qr or QR. ST is on the isoline. A zone of ischemia is delineated and deep negative isosceles (coronary) T waves are formed (Fig. 96 — *5, 6;* 99);
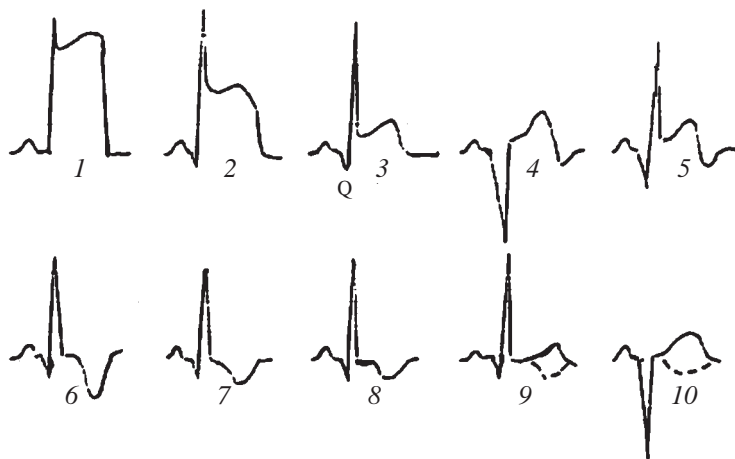
Fig. 96. Changes of the waves and segments of the ECG at different stages of myocardial infarction
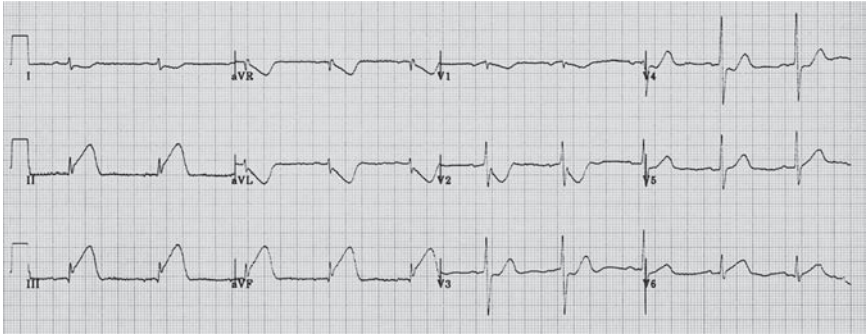
Fig. 97. The acutest stage of MI in the inferior wall (the formation of the monophasic curve ST-T in the leads II,III, aVF, compensation of the segment ST below the isoline in I, aVL, V2-3)
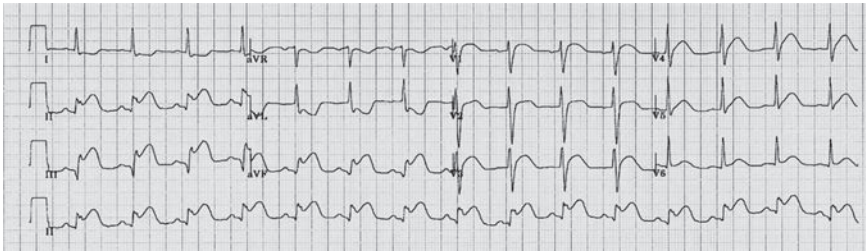
Fig. 98. Acute stage of MI in the inferior wall (appearance of the pathological Q wave, monophasic curve ST-T in the leads II, III, aVF remains)
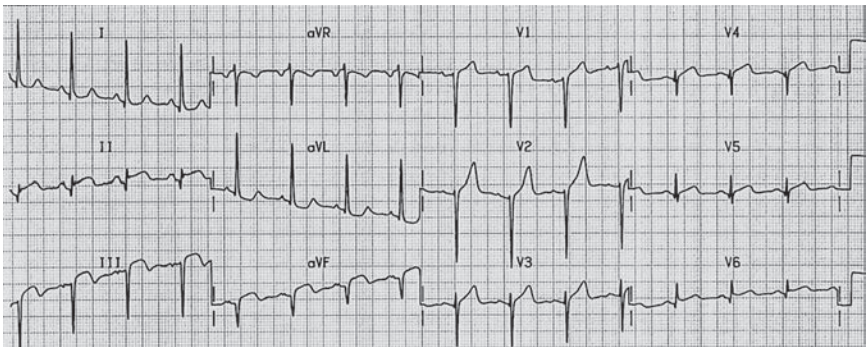
Fig. 99. Subacute stage of MI in the inferior-lateral wall (the appearance of the negative T wave in III, aVF)

90

Fig. 100. Scarring stage of myocardial infarction in the inferior wall of the left ventricle (pathological Q in the leads II, III remains, aVF — the area of the scar, the negative T in these leads — a zone of ischemia)

— **Scarring** — from 1–3 weeks to 1–3 months: the affected area disappears, there remains a scar area and a small area of ischemia around it (or not). the abnormal Q wave remains on the ECG, ST is on the isoline, the coronary T wave is preserved, although by the end of this period it begins to decrease in amplitude, with no isosceles (Fig. 96 — *7, 8;* 100).

The fig. 101 presents the typical ECG changes of anterior *(A)* and inferior *(B)* infarcts in various stages of the disease (*a* — acutest,
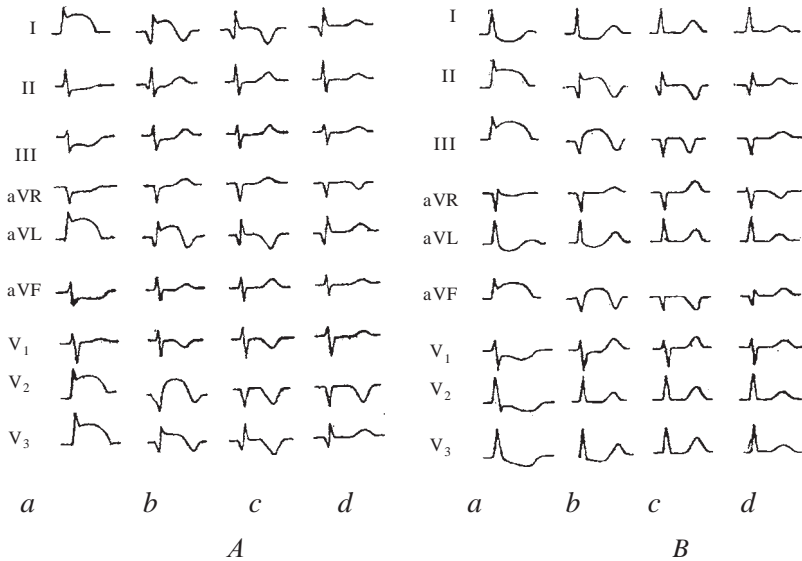
Fig. 101. Dynamics of the ECG changes at different stages of anterior *(A)* and inferior *(B)* Q myocardial infarction

b — acute, *c* — subacute, *d* — scarring). Thus, the electrocardiogram can not only confirm the presence of myocardial infarction, but also assess the depth and breadth of the affection, localization and stage of the process.

THE ECG IN PULMONARY HEART

Basic ECG signs of acute pulmonary heart disease (Fig. 102):

1. The appearance of the wave Q in III and S in I standard leads (sign Q III — S I), causing the QRS complex to take the form of RS in I standard lead and QR in the lead III.

2. The ST-segment elevation in III, aVF, V1 and V2 leads and its discordant lowering in I, aVL, V5 and V6 leads.

3. The appearance of the negative T wave in III, aVF, V1 and V2 leads.

4. Block of the right bundle branch (complete or incomplete).

5. The increase in the amplitude of the P wave in III, II, and aVF leads (the appearance of P-pulmonale).

6. The disappearance of these changes in improvement of the patient's condition (usually 3–5 days).

Basic ECG signs of chronic pulmonary heart (Fig. 103):

1. The presence of P-pulmonale in II, III, aVF, often in V1-V3 leads.

2. Block of the right His branch bundle (complete or incomplete).
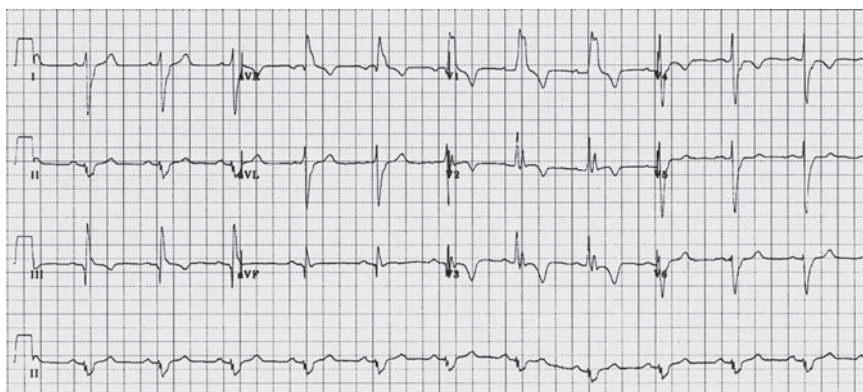
Fig. 102. The ECG in acute pulmonary heart of patients with pulmonary embolism (sign QIII-SI, the ST elevation in III, aVF, V1 and V2, the indication of block of right His bundle branch)

92
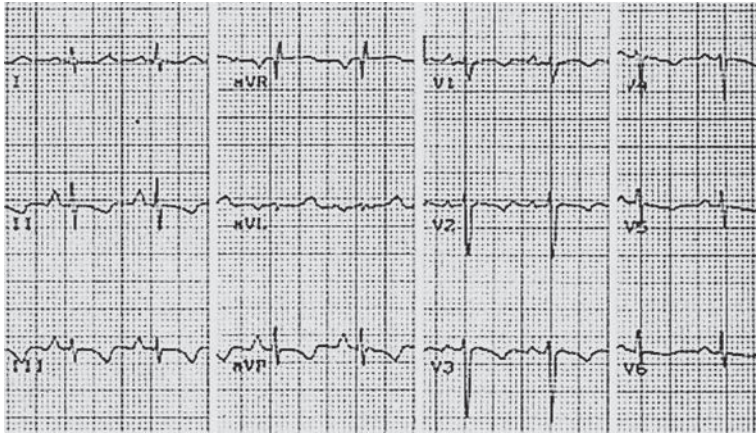
Fig. 103. The ECG in chronic pulmonary heart (signs of hypertrophy of the right chambers, the disturbance of repolarization in II, III, aVF, V1-V3)

3. The combination of block of right bundle branch with the ECG signs of right ventricular hypertrophy of any type (qR, rSR', and especially S).

4. Not infrequently the appearances of the late R wave in aVR lead.

5. Depression of the ST segment, appearance of the negative T waves in II, III, aVF, V1, V2, rarer in V5, V6 leads.

THE ECG CHANGES IN SOME DISEASES

The ECG in noncoronary affections of the heart

Myocarditis

In myocarditis the ECG does not have specific changes. The flattened or inverted T wave is often observed in a number of the leads, less often — a small depression or elevation of the ST segment (Fig. 104). These changes, unlike of IHD, do not undergo dynamics during physical exercise. In myocarditis various disturbances of rhythm and conduction are often observed. The development of sinus tachycardia, atrial fibrillation or flutter, atrioventricular block of I–II degree, intraventricular blockades may be of diagnostic value. In rare cases of severe diffuse myocarditis, for example, Abramov–Fiedler, there are pseudo-infarct changes of the QRS complex with the formation of the pathological Q or QS, but, as a rule, there are no deformation and changes in the dynamics of the final part of the ventricular complex typical of infarction. In diffuse myocarditis and myocarditis
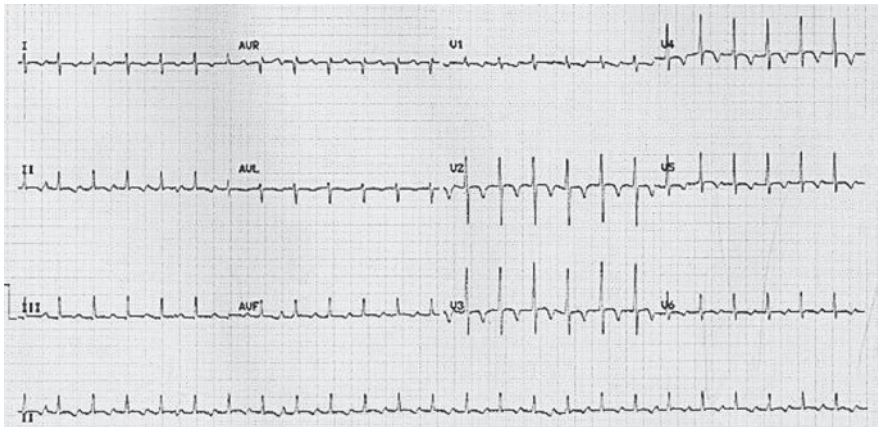
Fig. 104. The ECG of a patient with myocarditis (sinus tachycardia, negative T wave in the standard I, II, aVL and in all chest leads V1-V6)

cardiosclerosis there are sometimes signs of hypertrophy of the atria and ventricles. In small focal inflammatory affection of the myocardium the ECG may be normal.

Pericarditis

In dry pericarditis the inflammatory process in addition to the pericardium involves segments of the myocardium that lie under the epicardium. Therefore, the ECG changes in dry pericarditis resemble changes in subepicardial myocardial infarction. In acute dry pericarditis the ECG changes are of 3 stages. Stage I lasts about a week, the elevation of the ST segment in most leads is observed on the ECG, the shape can be flattened or convex in one direction or another, the T wave remains positive. In contrast to myocardial infarction the pathological Q wave and the compensation of the ST segment is not formed in all leads in the same direction (Fig. 105). In stage II, which lasts 1–2 weeks, the ST segment gradually decreases to the level of the isoline, the T wave becomes negative. Stage III depending on the severity of the disease lasts from several weeks to several months. At the beginning the T waves become deeper and more negative, and then gradually they become less deep and their subsequent normalization is observed. If the inflammatory process is limited to a separate section, the ECG changes do not occur at all, but only in several leads.

In exudative pericarditis the fluid in the pericardial cavity causes as if "short-circuit" of the currents arising in the heart, and fibrinous overlays on the surface of the pericardium reduce their conduction. This results in
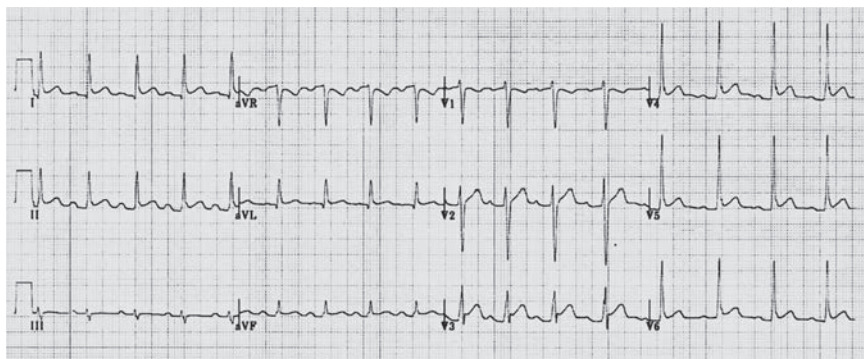
Fig. 105. The ECG in dry pericarditis (sinus tachycardia and typical ST-segment elevation in the leads I, II, aVL, aVF, V2-V5)

a decrease in waves amplitudes on the ECG. The decrease in the amplitude of the ECG waves is observed in accumulation of 300–400 ml of fluid or more in the pericardial cavity. When there is a large amount of effusion seen on the ECG, there is an alternation of the ventricular complexes QRS, i. e. QRS of different amplitude (Fig. 106). Involvement of the atria in inflammation may lead to atrial fibrillation.

Dilated cardiomyopathy (DCM)
The ECG signs (Fig. 107):
— hypertrophy of the left ventricle and the left atrium or the combined enlargement of both atria and ventricles;
— not infrequently presence of the pathological Q wave;
— displacement of the ST segment above or below the isoelectric line;
— changes of the T wave (inversion, reduction of its amplitude);
— disorders of rhythm and conduction: arrhythmia, atrial fibrillation, block of His bundle branches.

Hypertrophic cardiomyopathy (HCM)
The ECG signs (Fig. 108):
— various disturbances of rhythm and conductivity;
— ECG signs of severe left ventricular hypertrophy;
— signs of hypertrophy of the interventricular septum (deep but not wide Q wave in the leads V1-V3, aVL);
— disturbance of repolarization processes of the left ventricle, characterizes its overload — displacement of the ST segment (usually I, II, aVL and left chest leads V4-V6) obliquely downward with a transition to the negative T wave.

Alcoholic cardiomyopathy

The ECG changes in alcoholic cardiomyopathy are detected in patients even in the absence of clinical manifestations of the disease. The most frequently recorded signs are:
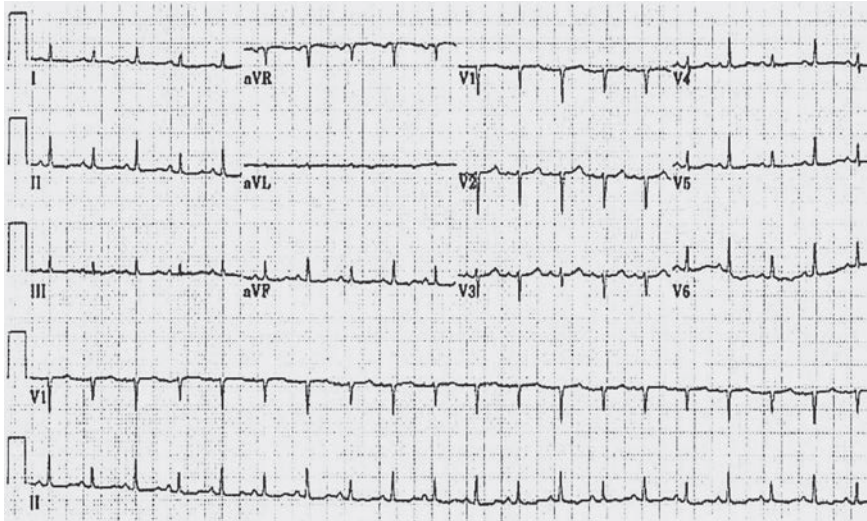
Fig. 106. The ECG in exudative pericarditis (against the background of sinus tachycardia alternation of the QRS complexes and disturbances of repolarization processes is observed in the standard and chest leads)
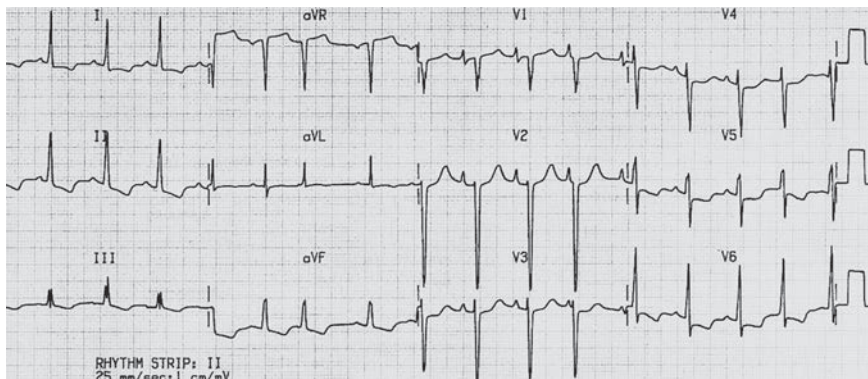
Fig. 107. ECG in DCM (signs of severe hypertrophy of both atria and ventricles and disturbances of repolarization processes)

96
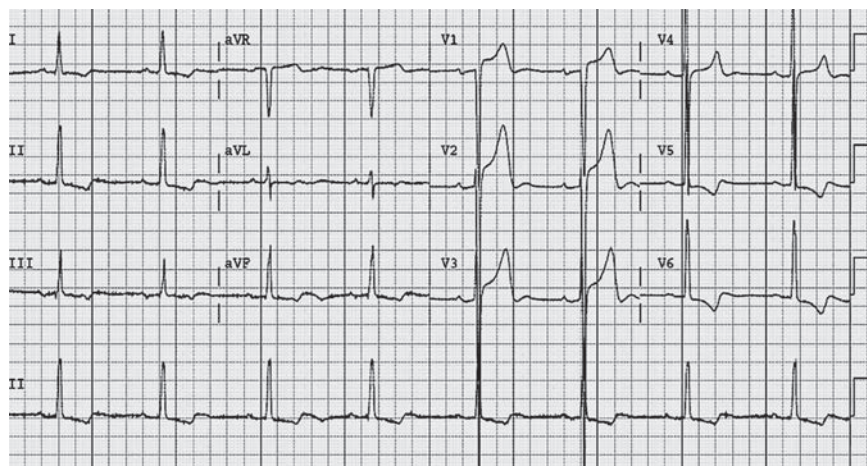
Fig. 108. The ECG in HCM (signs of severe left ventricular hypertrophy with its overload)

— changes of the final part of the ventricular complex: the ST interval compensation downward from the isoline, reducing the T amplitude, its flattening, negativity (which requires a differential diagnosis with IHD). Of great diagnostic value are the circumstances characteristic of alcoholic cardiomyopathy: the appearance of new or strengthening of the existing ECG changes after alcoholic excess and their positive dynamics after cessation of alcohol intake;

— in severe forms — the appearance of the pathological Q wave;

— disorders of rhythm and conduction (sinus tachycardia, extrasystoles, atrial arrhythmia, disturbance of AV-conduction);

— changes of the atrial and ventricular complexes on the ECG that characterize hypertrophy of the heart chambers (Fig. 109).

Damage of the heart in hyperthyroidism (thyrotoxic heart)
The ECG signs:

— arrhythmias: sinus tachycardia, atrial fibrillation or atrial flutter;

— in the initial stages of the disease — increasing of the amplitude of the R and T waves in the standard leads, increasing of the T wave in the chest leads;

— downward displacement of the ST segment in the chest leads and the appearance of the flattened, biphasic, or negative T wave.

Fig. 109. The ECG of the 20-year-old patient with alcoholic cardio-myopathy (sinus tachycardia with a heart rate of 117/min, signs of hypertrophy of the heart chambers and disturbances of repolarization processes)

The electrocardiogram in hypothyroidism

Typical ECG signs of hypothyroidism:

— sinus bradycardia, intraventricular block and elongation of AV-conduction is often diagnosed;

— decrease of the amplitude, flattening or inversion of the T waves in the standard and V3-V6 leads;

— an important ECG feature is a low-voltage curve, characterized by a decrease of the QRS complex amplitude. The greatest decrease is registered in the presence of effusion in the pericardial cavity. There may be a depression of the ST segment. The changes of the T wave and ST segment decrease or disappear along with clinical manifestations in the administration of adequate substitution therapy and remain in elderly patients, suffering from ischemic heart disease (Fig. 110).

ECG in dishormonal and climacteric cardiopathy

During menopause as well as in various hormonal disorders, women are often observed to have the same ECG changes as in ischemic heart disease. They are expressed in the change of the final part of the ventricular complex — ST segment (usually in V1-V4) and T wave.

The development of these abnormalities is not associated with pain in the heart area and do not involve dynamics of biochemical and general blood counts.

Dishormonal or climacteric cardiopathy is characterized by improvement of the ECG after menstruation or during the functional tests with potassium and obsidan (anaprilin). They are widely used in differential diagnosis with IHD (Fig. 111).
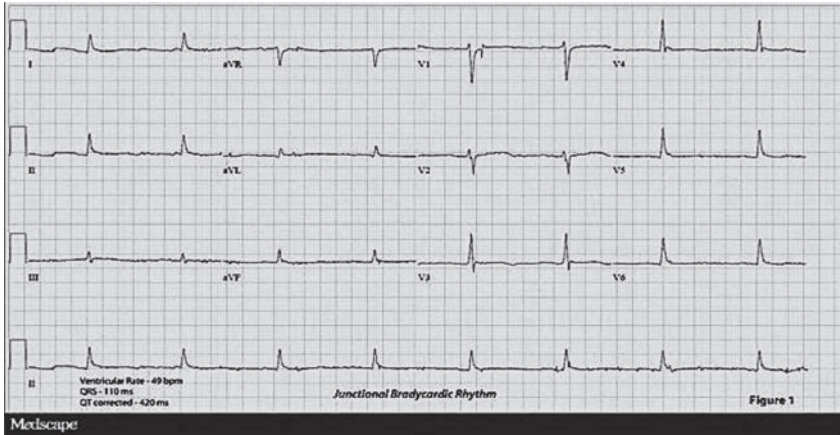
Fig. 110. The ECG in hypothyroidism (sinus bradycardia, decrease in voltage of the T waves and QRS complex in most leads)

THE ECG IN ACQUIRED HEART DEFECTS

The ECG changes in acquired heart defects are signs of hypertrophy, dilatation and overload of the corresponding segments of the heart, and
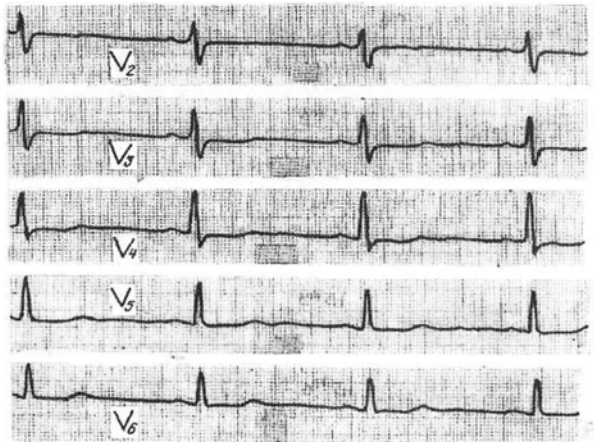
Fig. 111. The ECG (chest leads) of the patient with uterine fibroids and pathological menopause (disorders of repolarization in the biphasic T in the lead V2-V5)

disorders of rhythm and conduction, severity of which depends on degree of the heart defect, combination of defects and associated damage of the cardiovascular system.

Mitral stenosis

Basic ECG signs:

— in the forming defect against the background of preserved sinus rhythm at first there are signs of left atrial hypertrophy (P-mitrale), then right ventricle and later the right atrium (the P wave is distinctly split) (Fig. 112);

— in the development of atrial fibrillation there are signs of right ventricular hypertrophy and large wave form of atrial fibrillation;

— the compensation of the segment S-T below the isoline and negative T in III, aVF, V1-V2 leads;

— possible signs of partial or complete block of the right His bundle branch.

Mitral insufficiency

Depending on the severity of the valve defect and the degree of pressure elevation in the pulmonary circulation, the ECG changes may be different.

If regurgitation of blood in the left atrium is small and does not cause severe overload, the ECG remains normal.

The ECG signs due to volume overload of the left heart chambers:

— when there are moderate defect signs of left atrial hypertrophy (P-mitrale) and the left ventricle. The downward displacement of the ST segment in V5, V6, I, aVL leads is relatively rare at the stage of compensation defect;

— in significant valvular defect and hypertension of the pulmonary circulation — hypertrophy of the left atrium, the left and right ventricles (balanced hypertrophy of both ventricles with a predominance of the left) against the background of atrial fibrillation or flutter (Fig. 113).
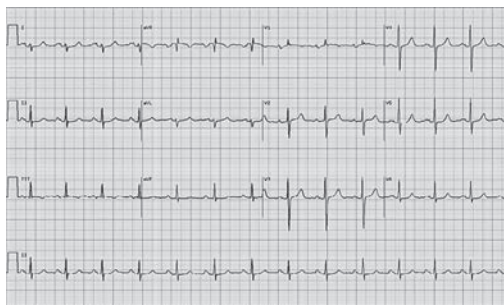
Fig. 112. The ECG of mitral stenosis (against the background of sinus rhythm is "P-mitrale" in the leads I, II, V3-V6 and signs of right ventricular hypertrophy)

100

Aortic stenosis

The ECG signs due to hypertrophy and systolic overload of the left ventricle:

— the criteria of severe left ventricular hypertrophy, usually with manifestations of systolic overload — displacement of the ST segment downwards from the baseline, and the biphasic or negative T wave in V4-V6 (Fig. 114);

— possible development of complete or incomplete left His bundle branch block.

Aortic insufficiency

The ECG signs of this disease are largely similar to those observed in aortic stenosis, but reflect volume overload of the left ventricle:

— the compensated defect signs of severe left ventricular hypertrophy without ST-segment depression and biphasic or negative T wave on the contrary, the T waves in V4, V5, V6 leads are positive, high, pointed;

— in the development of heart failure there are pronounced signs of left ventricular hypertrophy with depression of the ST segment and inversion of the T wave;

— in the "mitralization" of defect, i. e. development of relative mitral insufficiency, signs of left ventricular hypertrophy and, not frequently, signs of hypertrophy of the left atrium (P mitrale) (Fig. 115).

Insufficiency of the tricuspid valve

Basic ECG-signs:
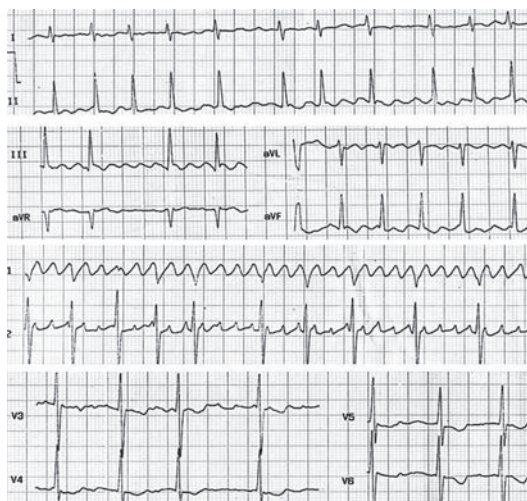
Fig. 113. The ECG of mitral insufficiency (against the background of atrial flutter there are signs of hypertrophy of both ventricles with disturbances of repolarization processes due to their overload)

101
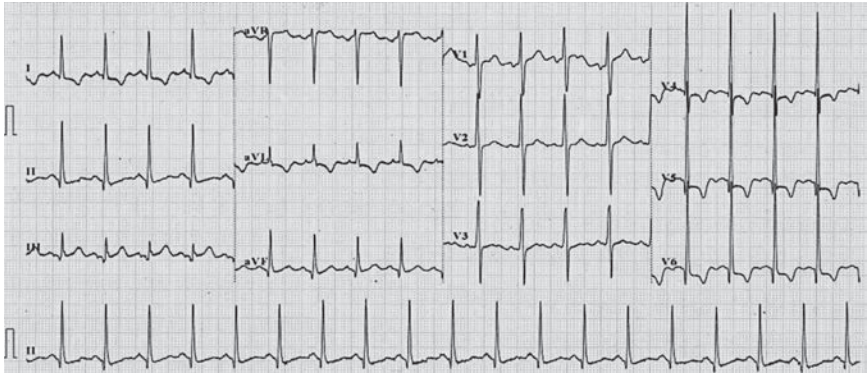
Fig. 114. The ECG of aortic stenosis (against the background of sinus rhythm there are signs of severe hypertrophy and systolic overload of the left ventricle)

— when there are isolated defect signs of right atrial hypertrophy (P-pulmonale) and right ventricular hypertrophy;

— when there is combined mitral-tricuspid disease (mitral stenosis with the development of relative tricuspid insufficiency) signs of right ventricular hypertrophy and combination of both atrial hypertrophy (P-pulmonale and P-mitrale) (Fig. 116).
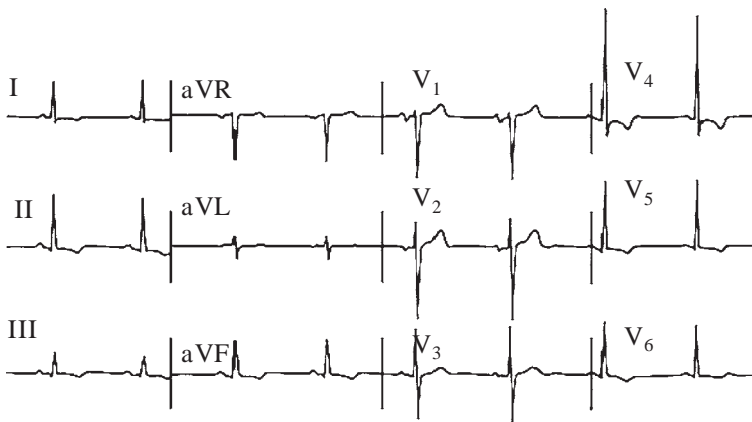
Fig. 115. The ECG of aortic insufficiency (signs of hypertrophy of the left atrium and ventricle with its overload — depression of the ST segment in V4-V4 and negative T in the standard and the left chest leads)

THE ECG IN HYPERTENSION DISEASE

According to the Framingham study, the left ventricular hypertrophy (LVH) is recorded in from 18 to 62% of patients with essential hypertension. The presence of LV hypertrophy is an independent prognostic risk factor, affecting the prognosis of the disease. When examining large groups of HD patients using different ECG criteria, left ventricular hypertrophy is diagnosed in from 18 to 65 %, indicating low sensitivity of this method in detecting LVH. Despite this, the ECG remains a simple, accessible and inexpensive method in the diagnosis of LVH in HD. It must be emphasized that the increase of myocardial mass of the left ventricle (LV remodeling) in HD can be carried out in several variants (Fig. 117): concentric — the myocardial mass increases mainly due to hypertrophy of the left ventricular wall, eccentric — an increase in the myocardial mass occurs due to the enlargement of the LV cavity and mixed (thickening of the LV wall and increase of the cavity).

The ECG changes will depend on the degree of increase of the LV myocardial mass, LV remodelling, and the presence or absence of signs of it overloading (Fig. 118).

The table 4 presents the main ECG criteria in the diagnosis of left ventricular hypertrophy in HD with regard to their sensitivity and specificity. The presence of multiple criteria on the ECG improves the sensitivity of the diagnosis of LV hypertrophy.
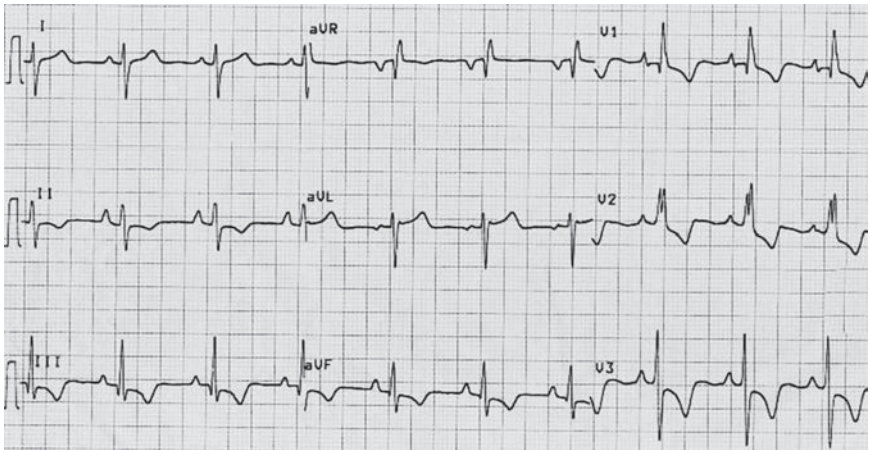
Fig. 116. The ECG in tricuspid insufficiency with signs of hypertrophy and overload of the right chambers of the heart
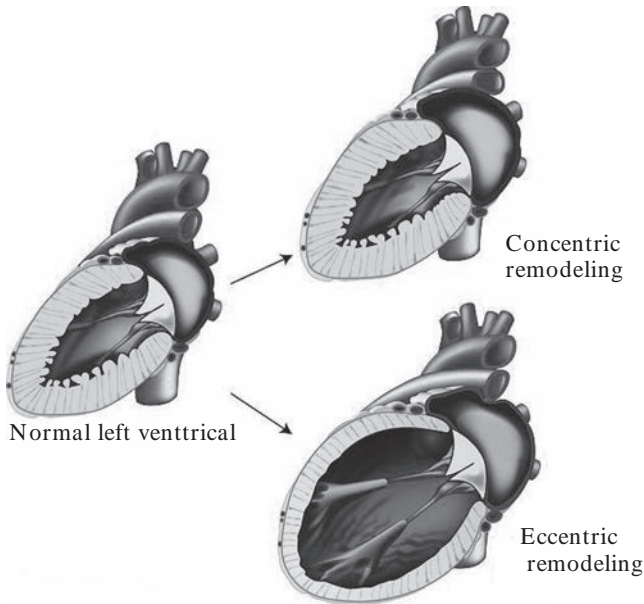
Fig. 117. Options of left ventricular remodelling in HD

THE ECG IN DISTURBANCES OF ELECTROLYTE BALANCE

The greatest influence on the ECG has a content of ions of potassium and calcium.

Hypokalemia. There are typical changes of the final part of the ventricular complex: the horizontal displacement of the ST segment below the isoline, the decrease in the amplitude or inversion of the T wave, increase in the amplitude of the U wave, the elongation of the electrical systole of the ventricles (the interval Q-T). Severe hypokalemia can lead to the development of ventricular arrhythmia of the type "pirouette" (Fig. 119, *a, b*).

Hyperkalemia is accompanied by the high, narrow, pointed T- waves, shortening of the electrical systole of the ventricles, with a tendency to sinus bradycardia and slowing down of atrioventricular and intraventricular conduction (Fig. 119, *c*).

Hypocalcemia is accompanied by lengthening of the electrical systole of the ventricles and shortening of the interval P-Q(R) (Fig. 116, *d*).
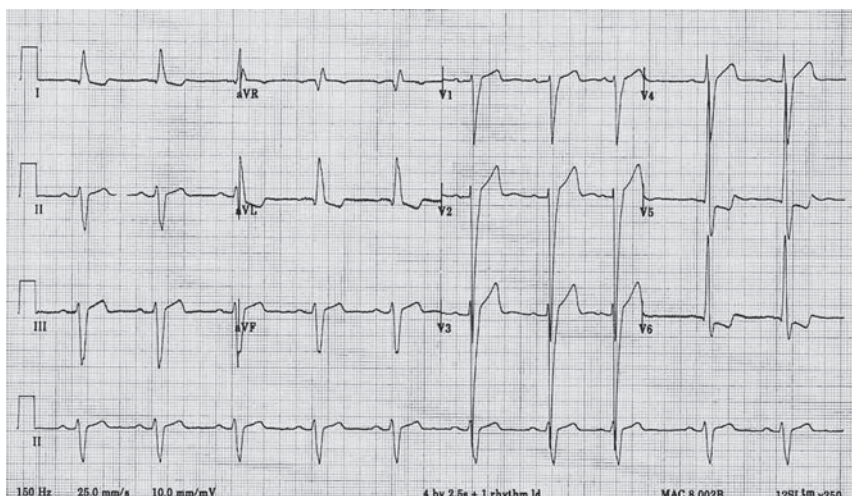
Fig. 118. The ECG in severe LV hypertrophy with signs of its overloading (deviation of the heart axis to the left, R aVL — 14 mm, index of Cornell is 34 mm, index of Sokolov-Lyons — 42 mm)

Hypercalcemia is characterized by shortening of the electrical systole of the ventricles and appearance of the flattened, biphasic, or negative T wave (Fig. 119, *e*).

**Sensitivity and specificity of different ECG criteria
of left ventricular hypertrophy**

Criteria of hypertrophy	Sensitivity, %	Specificity, %
Index of Sokolov – Lyons — sum of the amplitude SV1+R V5 or V6>35 mm	21–52	71–99
Index of Cornell — men: RaVL+SV3>28 mm; women: RaVL+SV3>20 mm	15–42	53–92
R aVL>11 мм	11–35	48–87
Index of Gubner — RI+SIII≥25 mm	9–29	30–59
R V4≤R V5	8–27	30–55

The syndrome of early repolarization of the ventricles

The syndrome of early or premature repolarization of the ventricles (SEPRV) is an electrocardiographic phenomenon with characteristic changes on the ECG in the form of a typical elevation of the place of the ventricular complex transition at the point j (Fig. 120 arrow) in the ST segment above the isoline, as a result of early emergence of the excitation wave in the subepicardial areas of the myocardium.

The syndrome of early ventricular repolarization can simulate the ECG signs of acute coronary syndrome. The prevalence among normal population ranges from 5% to 18%, most frequently recorded among young people. Recent studies have established the prognostic importance of this syndrome for the risk of sudden coronary death, which requires a revision of the therapeutic and pedagogical approaches to the "management" of such persons. A diagnosis of SEPRV is based on characteristic changes

a

b

c
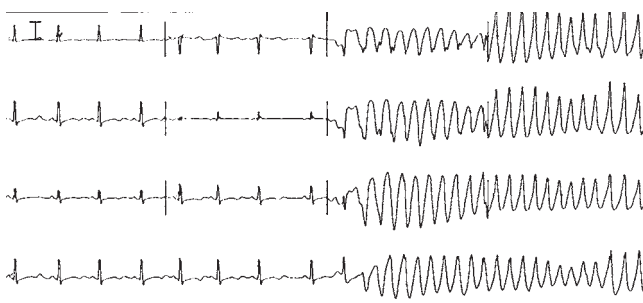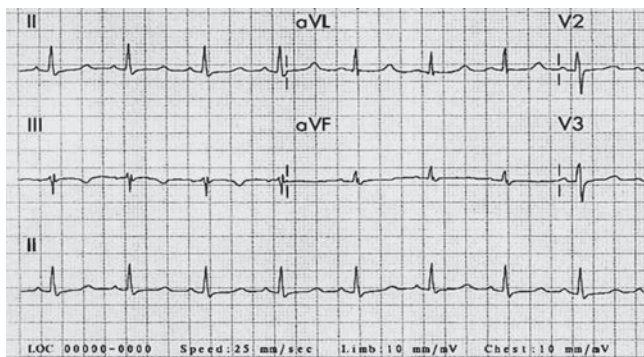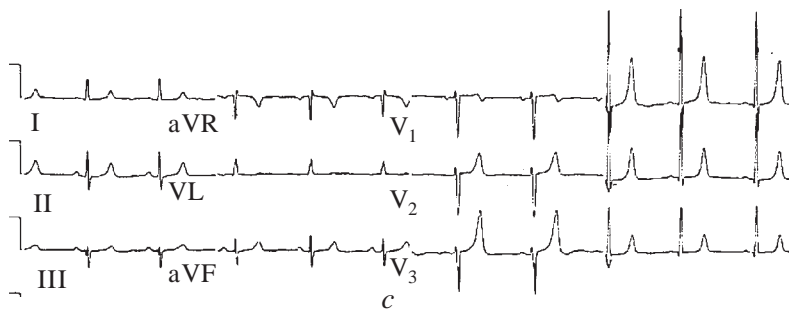
d

e

Fig. 119: a — the ECG in hypokalemia (ST displacement below isoline, inversion of the T wave, increase in amplitude of the U wave, prolongation of the interval Q-T); *b* — the ECG in hypokalemia with the development of an attack of ventricular tachycardia type "pirouette; *c* — the ECG in hyperkalemia (high, narrow and pointed wave T); *d* — ECG in hypocalcemia (prolonged QT to 510 MS), *e* — ECG in hypercalcemia (shortened QT, smooth, negative T)
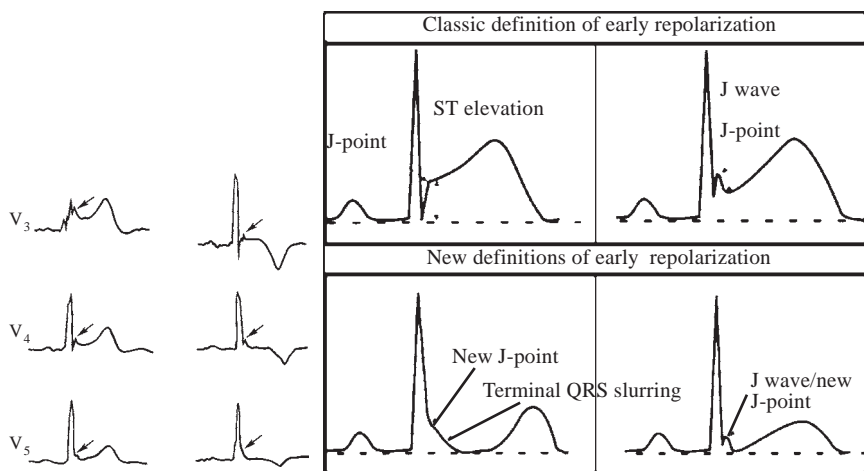
Fig. 120. Registration of the j point and ST-segment elevation in the early repolarization syndrome

of the ECG in the chest leads (Fig. 121). A test with physical exercise and drug test is carried out to exclude other phenomena and for verification of the diagnosis. As a rule, the exercise test in patients with SEPRV eliminates this phenomenon. The specificity of the test is about 40%, the information content is 60%. The test with atropine is generally not used in clinical practice due to possible adverse reactions. When taking potassium (potassium chloride not less than 2 g) there may be a normalization of the final part of the ventricular complex in patients with repolarization disorders. In true SEPRV there is worsening in severity of the ECG criteria.

The ECG diagnosis of Brugada syndrome

Brugada syndrome is genetically determined disorders of the cardiac rhythm characterized by syncope (fainting) state, elevation of the ST segment above the isoelectric line in the right precordial leads (V1-V3), complete or incomplete block of the right His bundle branch and a high risk of developing life-threatening ventricular tachyarrhythmias, usually developing during sleep or at rest. Patients are often discovered supraventricular arrhythmias, atrial fibrillation. The greatest diagnostic importance has the ECG of Brugada type (or type "bull Terrier" — Fig. 122, *a, b*).

108
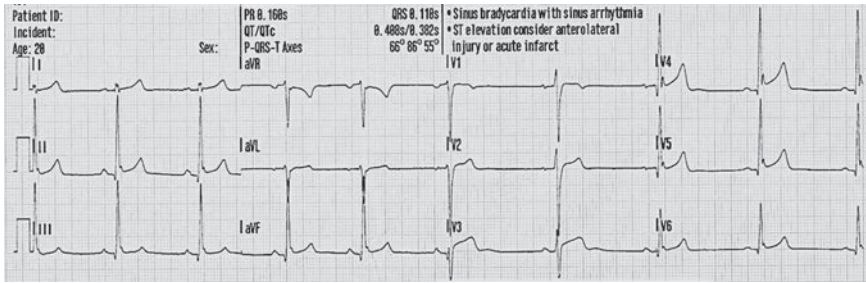
Fig. 121. Typical ECG changes in the syndrome of early repolarization of the ventricles
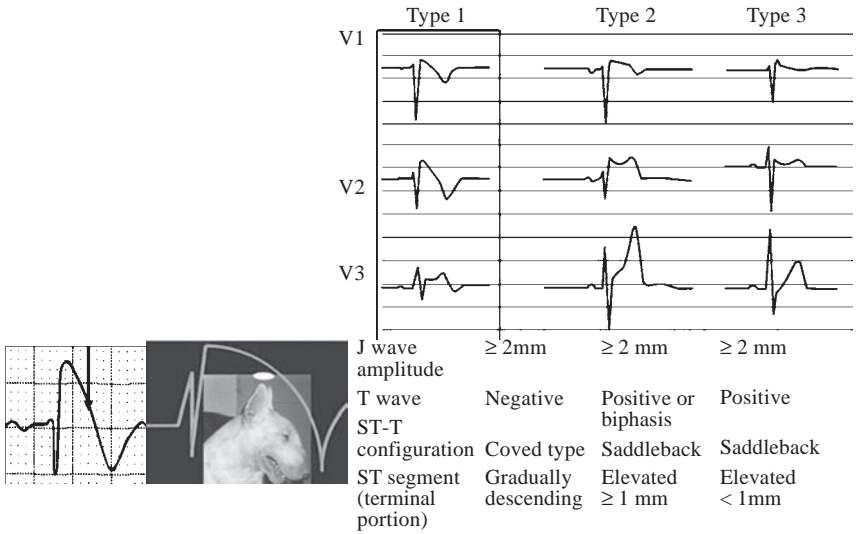
The ECG in the syndrome of digitalis intoxication

In overdose of cardiac glycosides the ECG changes are mainly due to the decrease of the intracellular potassium content in the myocardium, which is manifested by a channel-shaped depression of the ST segment of the type "Dali's moustache" (Fig. 123) and flattening, biphase (– +), and then inversion of the T wave in many leads, the pronounced U wave.

The increased level of the intracellular calcium leads to shortening of the Q-T interval, the U wave is separated from the T wave. In intoxication with cardiac glycosides, as a rule, there are various disturbances of rhythm and conduction. The most common rhythm disturbance associated with the use of cardiac glycosides is ventricular premature beats, often in the form of allorrhythmia (bigeminy, trigeminy, etc.). Not infrequently there are atrial premature beats, atrial fibrillation bradiarrhythmia, atrioventricular block of I and II levels (Table 5, Fig. 124–132).

Table 6 presents features of repolarization disturbances in ischemic diseases and diseases of the heart of nonischemic genesis.

Table 5

**The ST-segment changes in
IHD and disorders of
repolarization of nonischemic genesis**

ECG changes at ischemia	Changes of repolarization of nonischemic genesis	
Depression of ST	Glycoside intoxication	Through shaped depression shaped depression of ST segment Bephasic -/+ T Changes are more prononeced in the left chest leads
	Hypertrophy of LV	Oblique depression of ST segment Convex downword. Signs of left ventricular hypertrophy. Changes of repolarization are more prononeced in the left chest leads
Elevation of ST	Pericarditis	Elevation of ST segment practically in all leads Reciprocal changes are absent Pathological Q in dinamic isn't formed
	Syndrome of early ventricular repolarization	Elevation of ST segment convex upwards with transition in concordanT wave T. Changes have constant character

110

	Type 1	Type 2	Type 3
J wave amplitude	≥ 2mm	≥ 2 mm	≥ 2 mm
T wave	Negative	Positive or biphasis	Positive
ST-T configuration	Coved type	Saddleback	Saddleback
ST segment (terminal portion)	Gradually descending	Elevated ≥ 1 mm	Elevated < 1mm

a
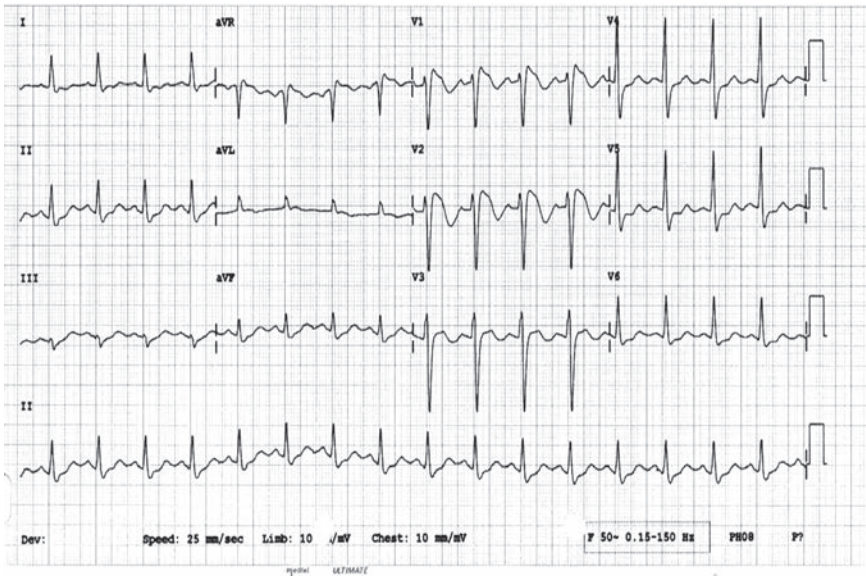
b

Fig. 122: *a* — elevation of the ST segment in V1-V2 type "bull Terrier" in Brugada syndrome; *b* — the ECG in Brugada syndrome

111
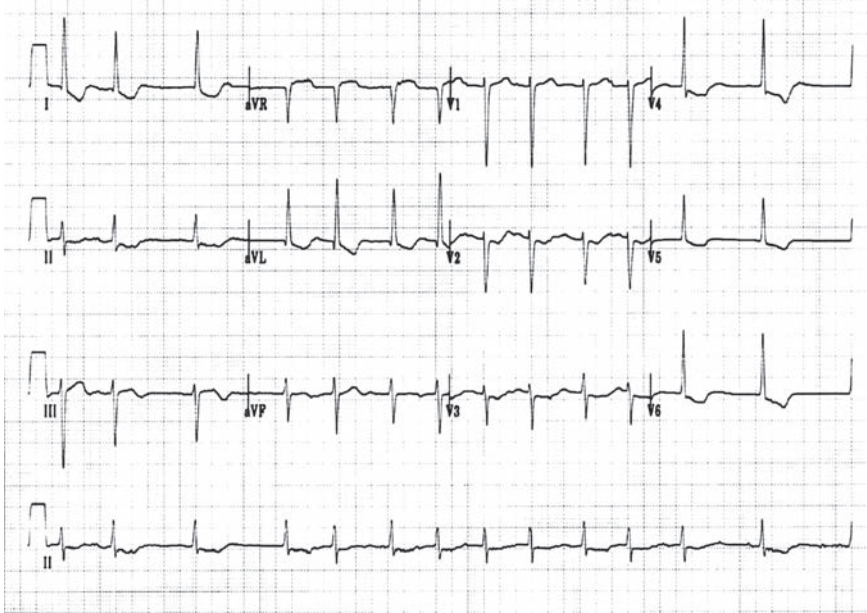
Fig. 123. The ECG in digitalis intoxication (against the background of atrial fibrillation typical channel— shaped displacement of the ST of the type "Dali's moustache" in I, II, aVL, V4-V6 leads is observed)
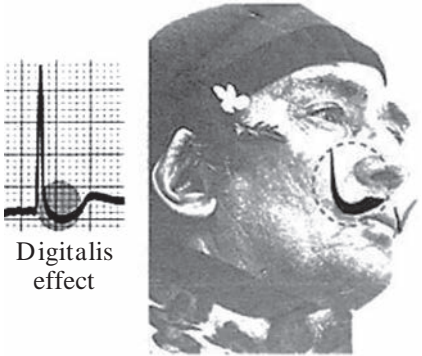
Digitalis
effect

Fig. 124. A channel — shaped ST segment deviation of the type "Dali's moustache" when there is digitalis intoxication
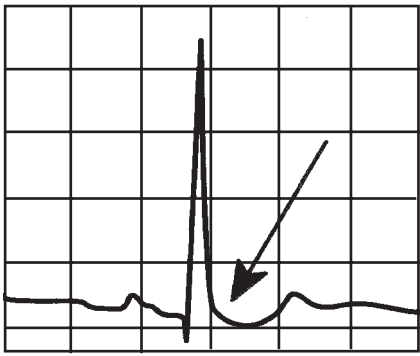
Fig. 125. Through shae deression ST at glycoside intoxication duplex (-+) T

112

Stage I   Stage II
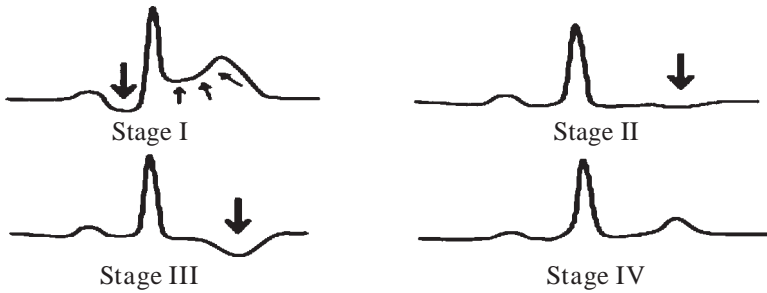
Stage III   Stage IV

Fig. 125. The 4 ECG stages of pericarditis. Stage I is characterized by ST-segment elevation (usually concave [small arrows]) and PR-segment depression (large arrow). Stage II is seen with resolution of the PR- and ST-segment abnormalities; nonspecific T wave abnormalities (diminution or flattening [arrow]). T wave inversion (arrow) is seen in stage III. Stage IV involves normalization of the PR segment, ST segment, and T wave

Normal ECG deflections

R

P   S   T

Q

Atrial depolarization   Ventricular repolarization

a   Ventricular repolarization

| ST segment depression | T wave inversion | ST segment elevation |
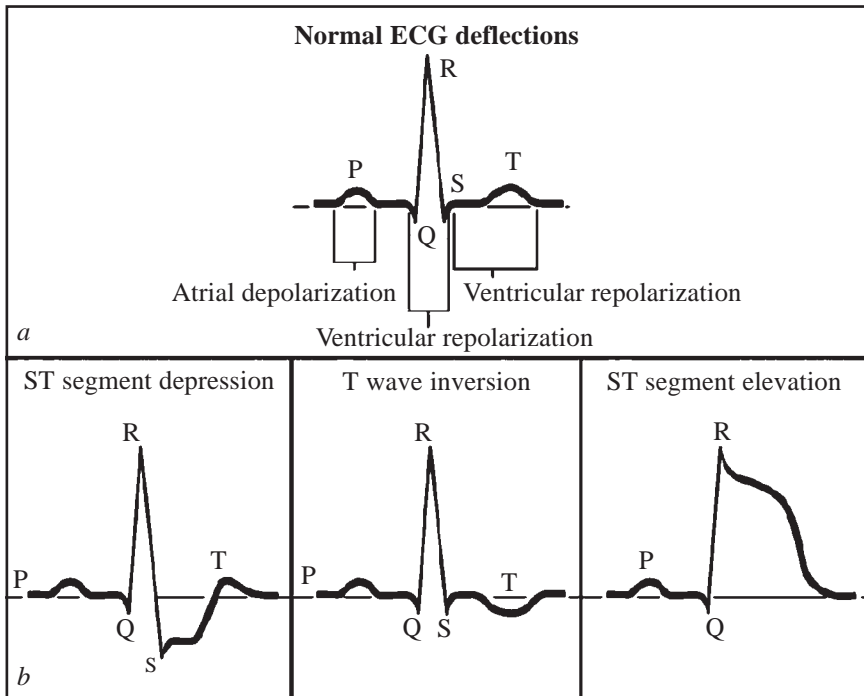
Fig. 126. Electrocardiogram and ischemia: *a* — normal ECG; *b* — electrocardiographic alterations associated with ischemia

113

Common causes of ST segment depression

1. Ischemia
2. "Strain"
3. Digitalis effect
4. Hypokalemia/Hypomagnesemia
5. Rate-related changes
6. Any combination of the above

Fig. 127. Common causes of ST segment depression
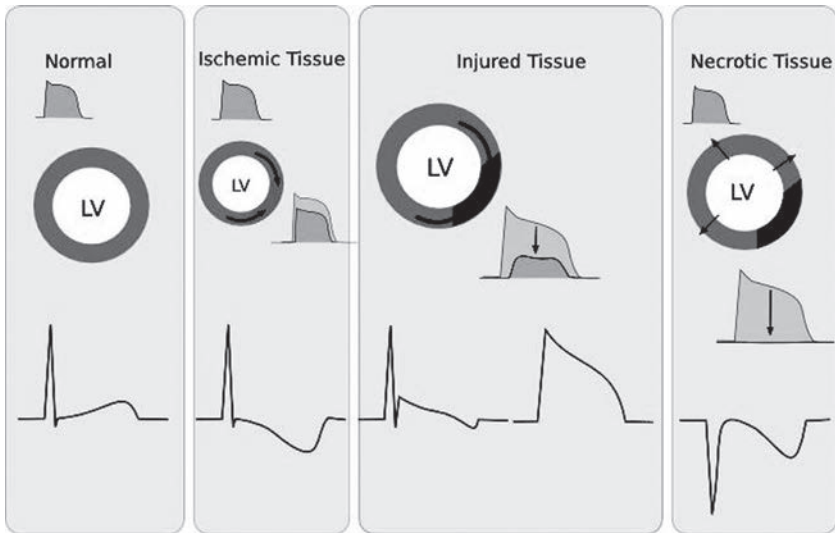
Injury current flows from healthy cardiomyocytes to ischemic cardiomyocytes resulting in ST shift. Necrotic tissue has no electrical activity
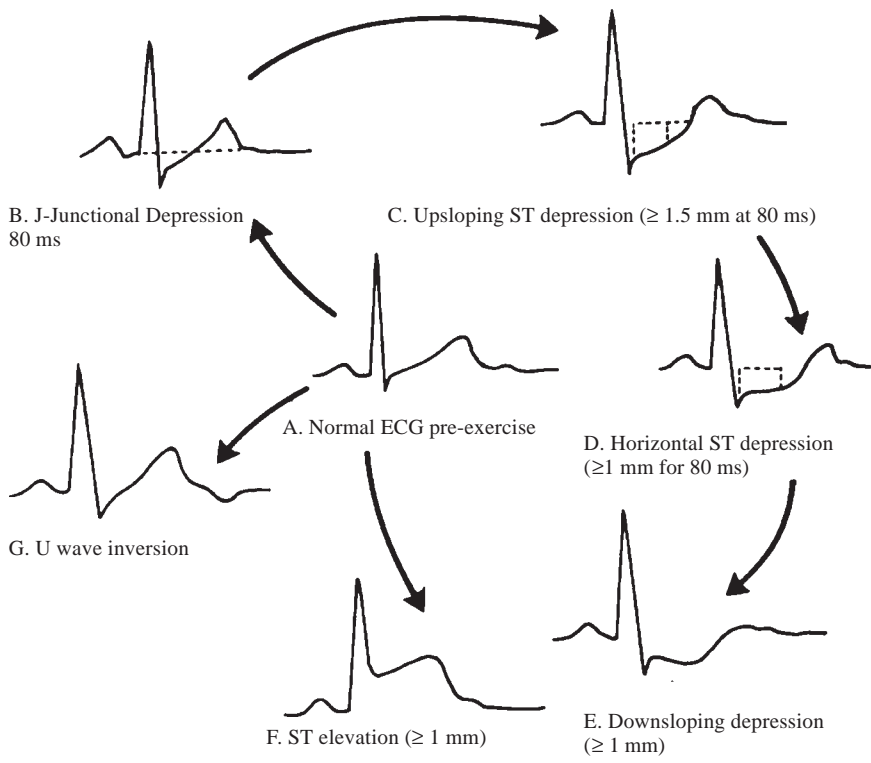
ECG○PEDIA.ORG

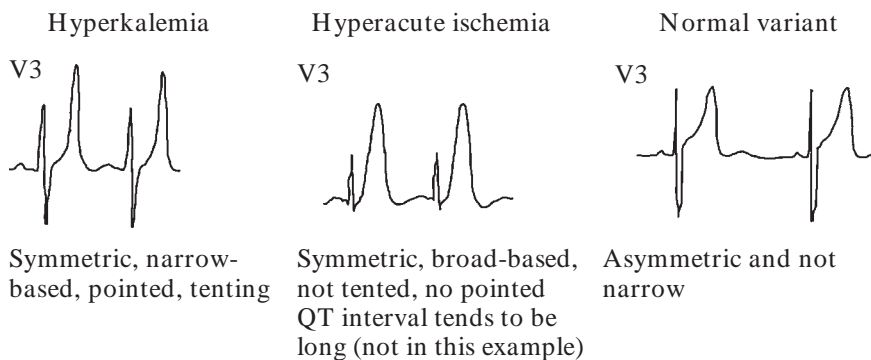*Fig. 128.* Title of the figure

114

Fig. 129. Ischemic ECG

Hyperkalemia	Hyperacute ischemia	Normal variant

Symmetric, narrow-based, pointed, tenting

Symmetric, broad-based, not tented, no pointed QT interval tends to be long (not in this example)

Asymmetric and not narrow

Fig. 130. Different causes of tall T waves

Normal Biphasic Bifid/notched Broad/slow Flat

Nonspecific ST-T wave abnormalities

Hyperkalemia Repolarization variant Ischemia Strain Prolonged QT interval
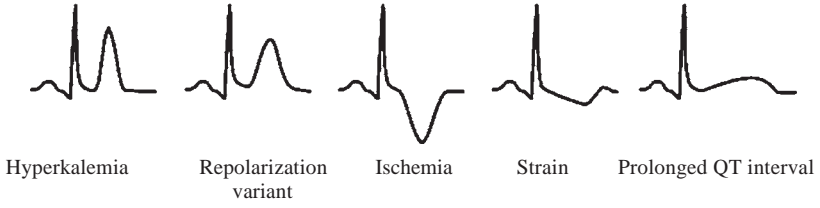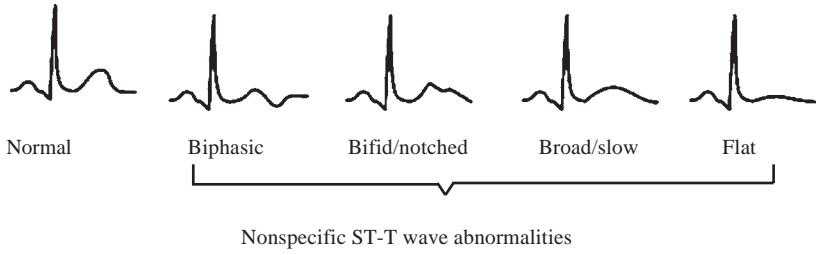
Fig. 131. T wave morphology

Non-STEMI

- ST depression can be seen with acute coronary syndrome, (non-Q non-ST infarction) and can also be seen as a chronic finding in hypertrophy or bundle branch block.
- Drugs such as digitalis can depress the ST segment as well as atrial repolarization waves (Ta waves).
- T wave abnormalities can be seen in over 60 situations, thus the term "nonspecific". A significant inverted T wave with symmetrical shape is typical of ischemia especially in Leads I and V_4-V_6. Inverted T waves can be normal in V_1-V_4, especially in children and woman.

Ischemia (or Non-STEMI) infarction

ST-segment depression or T-wave inversion
- T waves are usually upright in leads with dominant R waves
- Inverted symmetrical T wave

II V_1

Fig. 132. Non-STEMI

ABBREVIATIONS

AV   — atrioventricular
SA   — sinoatrial
EMF   — electromotive force
HCM   — hypertrophic cardiomyopathy
TMP   — transmembrane cell potential
EHA   — electrical axis of the heart
HR   — heart rate
IHD   — Ischemc heart disease
LAD   — descending coronary artery
CLC   — Clerk–Levy–Cristesco
SSS   — syndrome sick sinus
RBBB   — right bundle branch block
LBHB   — left bundle branch block
PVCs   — premature ventricular contractions
LMCA   — left main coronary artery system
LCA   — left coronary artery
LCx   — left circumflex coronary artery
RCA   — right coronary artery
WPW   — Wolf–Parkinson–White syndrome

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CONTENTS

119

У навчально-методичному посібнику йдеться про найбільш важливий аспект теорії електрокардіографії та створення механізму електрокардіограми, принципи її інтерпретації у випадках гіпертрофії серцевих камер, при аритміях, порушеннях провідності, ішемічній хворобі серця та інших патологіях.

Для студентів останніх курсів медичних вищих закладів освіти, переддипломного навчання, лікарів-ординаторів, що спеціалізуються з терапії, і дипломованих практикуючих фахівців.