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In memory of the associate professor of the Department of Propaedeutics of Internal Diseases of the Kazan State Medical University O.V. Bogoyavlenskaya, the initiator of the creation of methodological material on ECG for students of medical universities.

INTRODUCTION

Previous editions of textbooks of this kind have shown that students readily grasp the more difficult topics in electrocardiography when the material is presented from the standpoint of vector theory, in a simple and accessible form, and is not overloaded with unnecessary detail. At the same time, progress in clinical electrocardiography and in experimental electrophysiology makes it necessary to set out current concepts. A physician of any specialty should be able to interpret an electrocardiogram. Electrocardiography is a valuable diagnostic tool, but only when the electrocardiogram is assessed together with a careful history, physical examination of the patient, and observation of both the patient and the serial changes in the electrocardiogram.

The textbook stresses an important principle: the electrocardiogram must be read through the patient.

The textbook has been prepared for students in accordance with the Federal State Educational Standard of Higher Education in specialty 31.05.01 «General Medicine» and with the curriculum for the discipline «Propaedeutics of Internal Diseases». It presents current knowledge of the instrumental methods used to diagnose cardiovascular disease.

The textbook is intended to develop the following competencies in the discipline «Propaedeutics of Internal Diseases»:

- general professional competency GPC-6 — the ability to keep medical records;
- professional competency PC-5 — the ability to collect and analyze a patient's complaints and history, the findings of physical examination and of laboratory, instrumental, pathological and other investigations, in order to recognize a condition or to establish whether or not a disease is present.

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CHAPTER 3. ORIGIN OF THE ELECTROCARDIOGRAM AND METHODS OF ITS REGISTRATION

TRANSMEMBRANE POTENTIAL OF THE CELL

The heart consists of muscle fibers made up of chains of myocardial cells, and it is therefore customary to explain the origin of the cardiac biopotentials using a single muscle cell as an example. As already noted, in 1902 J. Bernstein attributed the origin of the biopotentials to the fact that the cell membrane is semipermeable and changes its resistance to ions in the phases of rest and of excitation. The membrane theory was later substantially extended by A. Hodgkin and A. Huxley (1952), who used microelectrodes to measure potentials directly inside cells.

The membrane does far more than act as a simple semipermeable barrier. It has a complex structure that includes specific proteins and enzyme systems, and it not only alters its permeability to ions sharply but also takes an active part in transporting them. I. Tasaki (1941) showed that the changing permeability of the membrane depends on the ability of its protein molecules to bind either calcium or potassium ions. At rest the proteins are bound to Ca^{2+} and form a structure (a conformation — from Latin *conformatio*, meaning form, structure, arrangement) that is virtually impermeable to the diffusion of ions, except for potassium ions (K^+), to which it remains somewhat permeable. On excitation the membrane proteins bind K^+ and form a new structure, which sharply increases permeability and allows ions to diffuse. The state of the membrane thus determines the ratio of intracellular to extracellular ions. Both the maintenance of the resting potential and the formation of the action potential depend on the differing permeability of the membrane to Na^+, Ca^{2+}, K^+ and Cl^-, which governs the ionic currents that flow across the membrane of the cardiomyocyte. These currents pass through various ion channels and are also driven by pumps and exchange mechanisms.

The interior of a resting cell is electronegative relative to the exterior by about –90 mV. This is true of most cells of the body. Each ion channel is a protein mole-

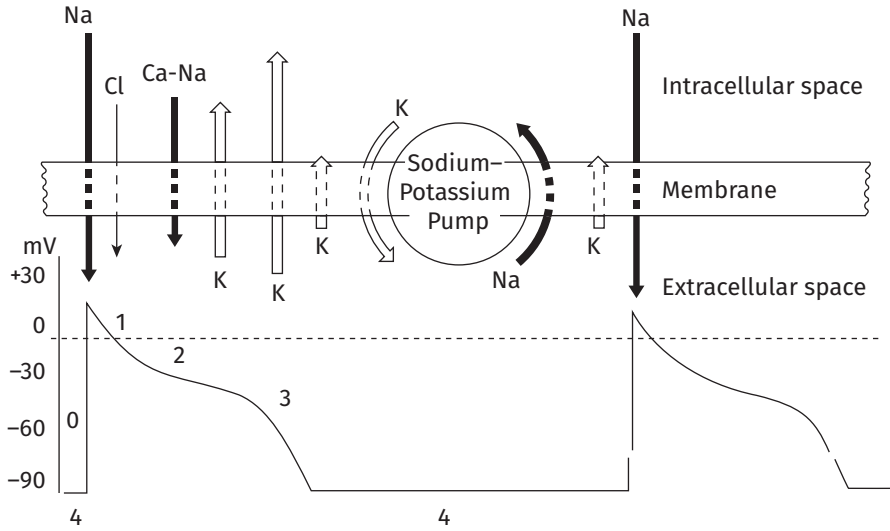


Fig. 3.1. Diagram of the formation of the transmembrane potential and the movement of ionic currents in the cell

cule set in the lipid bilayer of the membrane. Certain stimuli cause conformational changes in the molecule that switch the channel between states — in the simplest case, between rest (closed), activation (open) and inactivation (closed).

At rest the potassium concentration inside the cell is about 30 times that in the extracellular fluid; this ratio is known as the potassium gradient. Sodium, by contrast, is 20 times more concentrated outside the cell than inside. This unequal distribution of ions across the membrane is maintained by the «sodium–potassium pump» (**fig. 3.1**), an energy-dependent mechanism driven by the membrane enzyme Na^+,K^+-ATPase, which sustains the concentration gradients.

VECTOR (DIPOLE) THEORY OF THE ORIGIN OF THE ELECTROCARDIOGRAM

The theory of the origin of the ECG that is now most widely accepted is the vector, or dipole, theory, which originated with **Einthoven** and was given its final form by **Wilson**. Wilson proposed the term *elementary dipole* for the pair of positive (+) and negative (–) charges that arises in each muscle fiber during excitation, at the boundary between the excited and the unexcited regions.

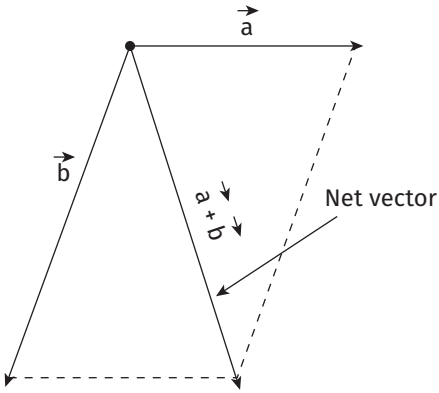


Fig. 3.2. Principle of algebraic addition of vectors directed at an angle to each other

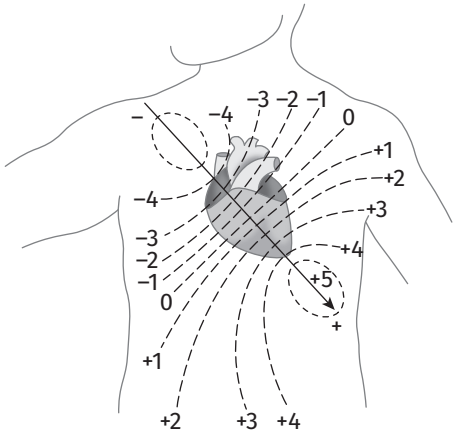


Fig. 3.3. Direction of the resultant dipole (instantaneous vector) of the electromotive force of the heart, with isopotential lines on the surface of the body (after Waller): the figures indicate the values of the potentials

Many dipoles with different orientations arise in the heart at the same time. Their electromotive force (EMF) is a vector quantity: it has both a magnitude and a direction, and the vector always points from the negative charge (–) towards the positive one (+). The electromotive forces of individual dipoles combine by vector addition: if two vectors are represented as the sides of a parallelogram, the resultant is its diagonal (**fig. 3.2**). At every instant the electromotive forces of all the myocardial cells combine into a resultant dipole — the instantaneous vector of the EMF of the heart — which moves progressively from the base of the heart towards the apex, following the wave of depolarization. An electric field forms around the heart (**fig. 3.3**), with negative potentials over its posterior and upper right aspect and positive potentials over its anterior and lower left aspect.

The instantaneous vectors constantly change both their direction and the site at which they arise, but they are conventionally taken to originate from a single point, the electrical center of the heart. For most of the time they point from the base of the heart towards the apex. Their predominant direction determines the resultant vectors of the heart, of which three are distinguished:

- the vector of atrial depolarization;
- the vector of ventricular depolarization;
- the vector of ventricular repolarization.

THE CONCEPT OF THE ELECTRICAL AXIS OF THE HEART

The direction of the resultant vector of ventricular depolarization is termed the electrical axis of the heart (EAH). The EAH is the integral of the initial, middle and terminal vectors of the whole *QRS* period. Its negative (–) pole lies at the base of the heart and its positive (+) pole at the apex. The direction of the EAH is expressed by the angle α, the angle in the frontal plane between the resultant vector of the EMF of the heart and the axis of standard lead I. In healthy subjects the EAH corresponds broadly to the anatomical axis of the heart (**fig. 3.4**).

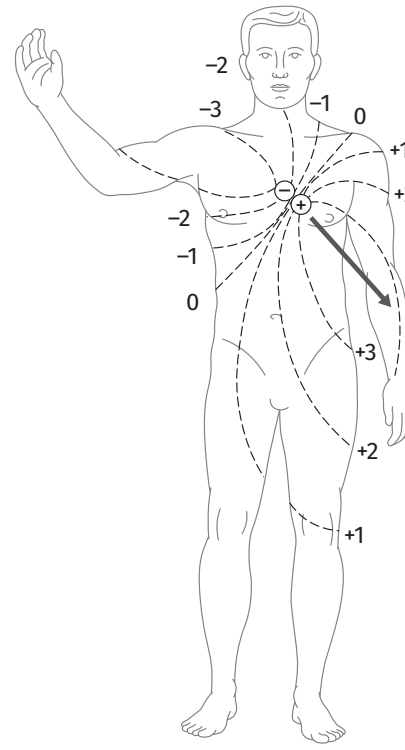


Fig. 3.4. Direction of the resultant vector of ventricular depolarization (Murashko V.V., Strutynsky A.V., 2005)

MECHANISM OF FORMATION OF ELECTROCARDIOGRAM ELEMENTS

The ECG records the changing potential difference generated by the myocardium — that is, it records the passage of the excitation wave through the myocardium over time. The record takes the form of a curve in which waves, segments and intervals are distinguished (**fig. 3.5**). It consists of a baseline, the isoelectric line, and deflections from it, the waves. The stretches of the curve between adjacent waves are called segments; the distances between waves, segments included, are called intervals. Einthoven proposed that the waves be designated by the letters *P*, *Q*, *R*, *S* and *T*.

MECHANISM OF FORMATION OF THE *P* WAVE AND *PQ* SEGMENT

The first wave of the ECG is the *P* wave, which arises from depolarization (activation) of the atria. Although the impulse from the cells of the SA node spreads through the atria in all directions, the resultant EMF is directed downwards and from right to left.

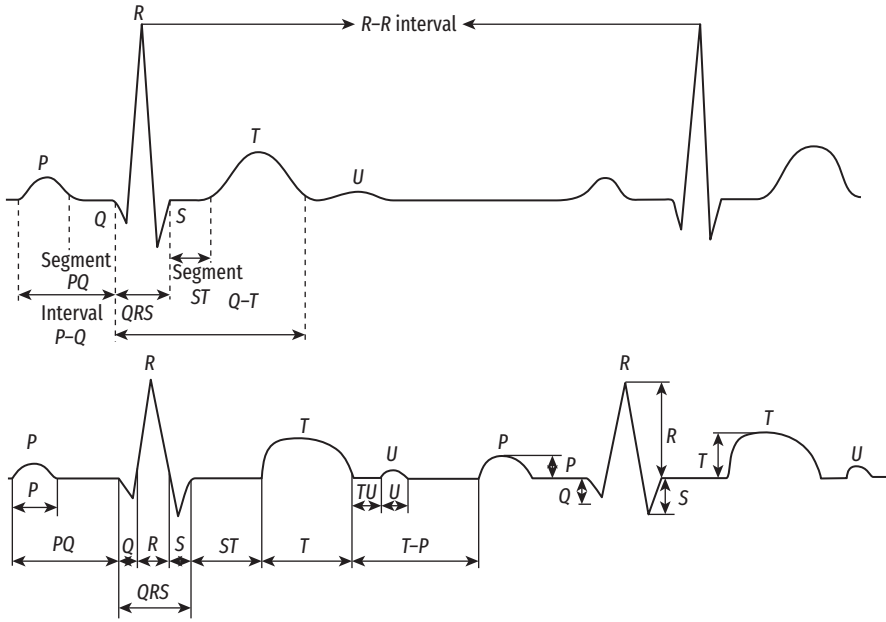


Fig. 3.5. Normal electrocardiogram (diagram)

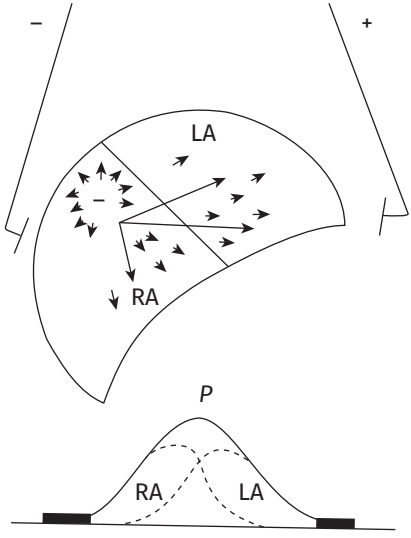


Fig. 3.6. Diagram of the formation of the *P* wave

The depolarization wave reaches the RA first and the left atrium (LA) 0.02–0.03 s later (**fig. 3.6**). The *P* wave is positive in most leads because the resultant vector of atrial depolarization follows the course of the EAH: downwards, from right to left and from back to front. The onset of the *P* wave corresponds to depolarization of the RA; its middle part coincides with the beginning of LA depolarization while the RA is still depolarizing; and its terminal part is produced by the LA alone, the RA by then being fully excited and generating no further potential. Normally the atria are wholly excited within 0.07–0.1 s, and this period does not exceed 0.1 s.

The *PQ* segment (*PQ* segment should not be confused with the *P–Q* interval) follows and lies almost on the zero line. At this point both atria are fully excited, so that there is no potential difference. The depolarizing impulse is passing through the AV node and the bundle of His, but the potential is too small to be recorded on a conventional ECG.

The *QRS* complex follows the *PQ* segment.

MECHANISM OF FORMATION OF THE *QRS* COMPLEX, *ST* SEGMENT, AND *T* WAVE

One point should be made before the elements of the *QRS* complex are described in detail. Excitation of the ventricular myocardium starts in the subendocardial layers, because the Purkinje fibers through which the impulse enters the contractile myocardium lie subendocardially. Excitation spreads through the subendocardial layers so rapidly that all parts of this layer are excited almost simultaneously (because of that, the leads in which subendocardial ischemia is registered do not indicate where the process is situated: subendocardial ischemia is circumferential and diffuse and cannot be localized to particular ventricular walls), and the summed potential of the inner layers is therefore negative during excitation, as is the intracavitary potential. The excitation wave is thought to travel through the ventricular myocardium as a double electrical layer of charges, moving from the subendocardial surface of the heart towards the subepicardial layers and reaching the various anatomical regions of the heart in a definite sequence. Although the depolarization wave appears almost simultaneously along the whole electrical boundary between the inner layers (negative) and the outer layers (positive), it reaches different parts of the surface of the heart at different times, and different parts of the surface layers remain depolarized for different lengths of time (Belenkov Yu.N., Ternovoy S.K., 2007).

The *QRS* complex is also called the initial part of the ventricular complex; the ventricular complex as a whole is designated *QRST*.

The *QRS* complex is formed by three vectors:

- the initial, or septal, vector — the vector of the first 0.02 s;
- the middle, or main ventricular, vector — the vector of the second 0.04 s;
- the terminal, or basal, vector — the vector of the last 0.06 s.

The left half of the interventricular septum (IVS) is excited first, so that it becomes electronegative while the right half remains positively charged. During this period the resultant EMF vector — the initial, or septal, vector — points in the direction opposite to the EAH, that is upwards and from left to right (**fig. 3.7**). In most leads this appears as a small negative *Q* (*q*) wave, whose small amplitude and short duration reflect the brief excitation (0.01–0.03 s) of this part of the myocardium.

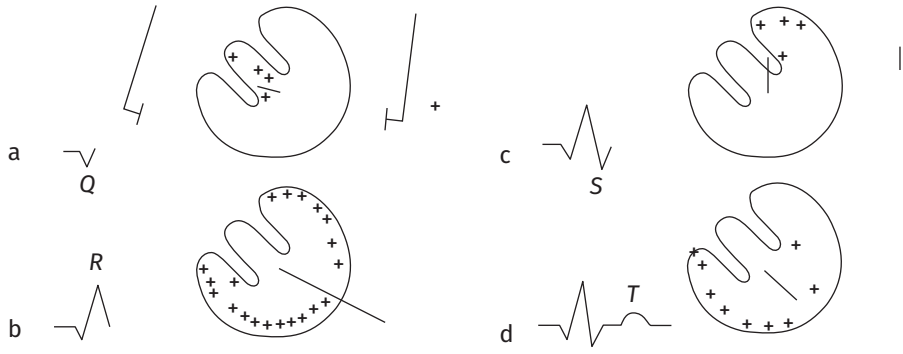


Fig. 3.7. Diagram of the formation of the *QRS* complex, the *ST* segment and the *T* wave: a — excitation (depolarization) of the interventricular septum; b — excitation of the main part of the ventricles, formation of the *R* wave; c — excitation of the posterobasal parts of the ventricles, formation of the *S* wave; d — repolarization of the ventricles, formation of the *T* wave

Excitation then spreads very rapidly to the anterior and lateral walls of both ventricles and to the apex of the heart — the main mass of the myocardium. Because the left ventricle (LV) generates a greater potential than the right, it cancels out the electrical forces of the right ventricle (RV). The resultant EMF vector for this period — the main ventricular vector — therefore follows the course of the EAH: downwards, from right to left and from back to front (**see fig. 3.7**). On the ECG this appears as the second ventricular wave, which in most leads is a tall positive *R* wave. It should be borne in mind that within the ventricular wall excitation spreads from endocardium to epicardium. The *R* wave takes up to 0.06 s to form. The last part of the myocardium to be excited is the base of the ventricles, the posterobasal region; the instantaneous EMF vector — the terminal, or basal, vector — then turns in the direction opposite to the EAH, that is upwards, from left to right and from front to back. On the ECG this appears as a small negative *S* (*s*) wave.

Normally excitation spreads through the ventricles in 0.06–0.1 s, that is in no more than 0.1 s (**fig. 3.8**).

The mechanism described above applies to most ECG leads but not to all. The waves of the *QRS* complex are relative: they depend on the lead — that is, on where the positive (active) electrode is placed and on the direction of the dipole vector. A tall *R* wave in the left chest leads reflects the predominance of the EMF vector of the LV; the same process is reflected in the right chest leads as a deep *S* wave (**fig. 3.9**).

The *ST* segment is recorded after the end of the *S* wave, or of the *R* wave if no *S* wave is present, and extends to the beginning of the *T* wave. The end

of the *QRS* complex and the beginning of the *ST* segment are taken from the *j* point, which corresponds to phase 1 of the action potential, the phase that terminates depolarization. On the ECG this point is defined as the angle

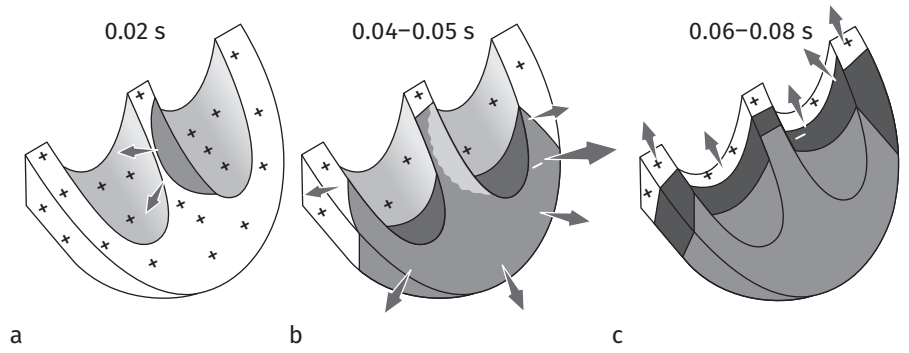


Fig. 3.8. Spread of excitation through the ventricles (Murashko V.V., Strutynsky A.V., 2005): a — excitation (depolarization) of the interventricular septum; b — excitation of the main mass of the ventricles; c — excitation of the basal parts of the ventricles. Parts of the myocardium already excited are shown in gray and those being excited at the moment shown in black

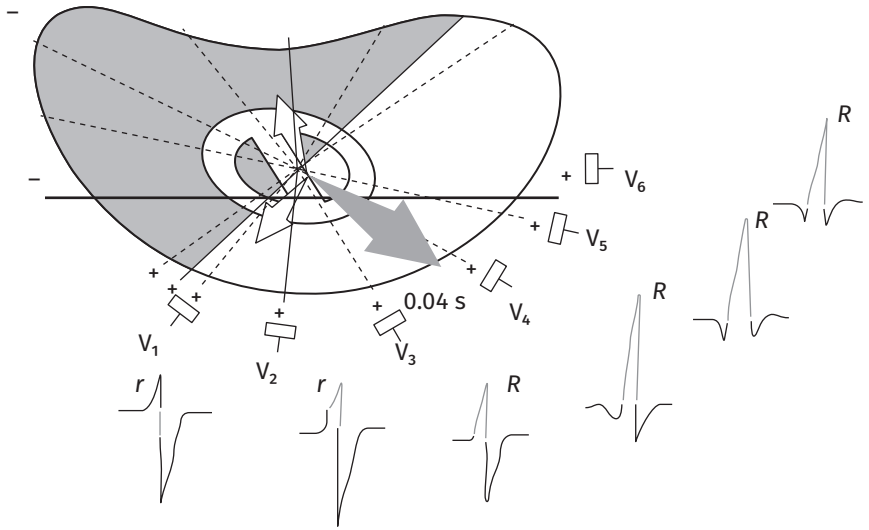


Fig. 3.9. Projection of the middle (main) vector of the *QRS* complex onto the axes of the chest leads (Murashko V.V., Strutynsky A.V., 2005)

between the *S* or *R* wave and the roughly horizontal stretch of the curve that forms the *ST* segment. Normally the *ST* segment lies on the isoelectric line, because during this period the ventricles are wholly excited (completely depolarized) and, as noted above, there is no potential difference. It should be added that the second phase of ventricular repolarization is already taking place during the *ST* segment; but this phase is prolonged, generates very small potentials and is almost entirely cancelled out between the two ventricles, and it therefore does not appear on the ECG and does not disturb the isoelectricity of the *ST* segment.

The *T* wave corresponds to the third and final phase of ventricular repolarization. This phase is rapid and generates a potential difference that the ECG records. According to Wilson the layers of the ventricular myocardium are not physiologically uniform, since they differ in temperature and in intramural pressure, and excitation therefore lasts for different lengths of time in different layers. In the subendocardial layers excitation lasts longer than in the subepicardial layers, so that repolarization begins in the subepicardial layers and proceeds towards the endocardium. A positive potential consequently appears first in the subepicardial region, and the repolarization vector is directed from endocardium to epicardium — from «–» to «+», that is along the course of the EAH. This is why the *T* wave is normally positive. Thus, although the waves of depolarization and of repolarization travel in opposite directions, starting from different parts of the heart muscle, the two vectors point the same way — from «–» to «+» — and the *R* and *T* waves on the ECG are therefore concordant, the *T* wave being directed the same way as the *R* wave. Discordance of these waves is a sign of disease. The *T* wave is smaller than the *R* wave because repolarization is always slower than depolarization and generates a smaller potential.

T–P INTERVAL AND THE *U* WAVE

The *T* wave is followed by an isoelectric line[1] — the *T–P* interval — which corresponds to diastole, when the whole heart is uniformly polarized and, as noted above, there is no potential difference. A small wave, the *U* wave, is sometimes visible after the *T* wave. Most authors regard it as an after-potential and attribute it to the end of ventricular repolarization; others believe that it reflects depolarization of the papillary muscles. A prominent *U* wave ($U \geqslant T$) is of clinical importance, as it may indicate hypokalemia. The amplitude of the *U* wave also increases in bradycardia, after severe physical exertion, in complete atrioventricular block, and with drugs such as cardiac glycosides and antiarrhythmic agents, including procainamide.

[1] An isoelectric line, or isoline, is a stretch of the record during which there is no electrical activity (Orlov V.N., 2004). The isoline is referenced to the *PQ* segment.