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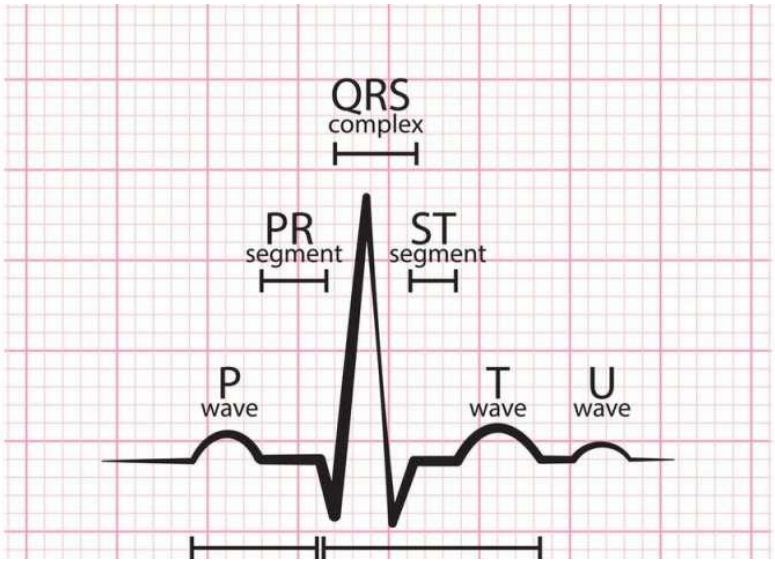
КАФЕДРА ФУНДАМЕНТАЛЬНЫХ ОСНОВ КЛИНИЧЕСКОЙ МЕДИЦИНЫ

ОСНОВЫ НОРМАЛЬНОЙ ЭЛЕКТРОКАРДИОГРАММЫ

Учебно-методическое пособие

THE BASICS OF NORMAL ECG

Manual



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Учебно-методическое пособие составлено в соответствии с Федеральным государственным образовательным стандартом высшего образования по направлению подготовки 31.05.01 «Лечебное дело», типовой и рабочей программами по дисциплине «Пропедевтика внутренних болезней». В учебно-методическом пособии подробно освещается содержание занятий по нормальной электрокардиографии, изложен теоретический материал, приводятся контрольные вопросы и задания. Пособие предназначено для иностранных студентов медицинских вузов.

CONTENTS

The physical basis of electrocardiography. performing the electrocardiographic test. electrocardiographic leads. analyzing and interpreting the electrocardiogram. the normal electrocardiogram

EDUCATIONAL MATERIAL

THE PHYSICAL BASIS OF ELECTROCARDIOGRAPHY. PERFORMING THE ELECTROCARDIOGRAPHIC TEST. ELECTROCARDIOGRAPHIC LEADS. ANALYZING AND INTERPRETING THE ELECTROCARDIOGRAM. THE NORMAL ELECTROCARDIOGRAM

Goal:

To get a notion about physiological basis of the Electrocardiogram (ECG) and the principles of the performing the electrocardiographic test, to master skills.

Knowledge objectives:

To know the physiological basis of the Electrocardiogram, the principles of the performing the electrocardiographic test, the characteristic features of the ECG-leads (3 standard limb leads, 3 augmented limb leads, 6 unipolar chest (precordial) leads), the ECG-wave morphology, the term "electrical axis" and the angle α, approach to ECG-analysis.

Skill objectives:

To develop practical skills in performing the electrocardiographic test and analyzing the normal electrocardiogram.

EDUCATIONAL MATERIAL

The heart is comprised of muscle (myocardium) that is rhythmically driven to contract and hence drive the circulation of blood throughout the body. Before every normal heartbeat, or systole, a wave of electrical current passes through the entire heart, which triggers myocardial contraction. The pattern of electrical propagation is not random, but spreads over the structure of the heart in a coordinated pattern which leads to an effective, coordinated systole. This results in a measurable change in potential difference on the body surface of the subject. The resultant amplified (and filtered) signal is known as an electrocardiogram (ECG, or sometimes EKG).

A broad number of factors affect the ECG, including abnormalities of cardiac conducting fibers, metabolic abnormalities (including a lack of oxygen, or ischemia)

of the myocardium, and macroscopic abnormalities of the normal geometry of the heart. ECG analysis is a routine part of any complete medical evaluation, due to the heart's essential role in human health and disease, and the relative ease of recording and analyzing the ECG in a noninvasive manner.

Understanding the basis of a normal ECG requires appreciation of four phenomena: the electrophysiology of a single cell, how the wave of electrical current propagates through myocardium, the physiology of the specific structures of the heart through which the electrical wave travels, and last how that leads to a measurable signal on the surface of the body, producing the normal ECG.

1. Cellular processes that underline the ECG

Each mechanical heartbeat is triggered by an action potential which originates from a rhythmic pacemaker within the heart and is conducted rapidly throughout the organ to produce a coordinated contraction (Fig. 1). As with other electrically active tissues (e.g., nerves and skeletal muscle), the myocardial cell at rest has a typical transmembrane potential, Vm, of about −80 to −90 mV with respect to surrounding extracellular fluid. The cell membrane controls permeability to a number of ions, including sodium, potassium, calcium, and chloride. These ions pass across the membrane through specific ion channels that can open (become activated) and close (become inactivated). These channels are therefore said to be gated channels and their opening and closing can occur in response to voltage changes (voltage gated channels) or through the activation of receptors (receptor gated channels) (Fig. 2).

Before the action potential is propagated, it must be initiated by pacemakers, cardiac cells that possess the property of automaticity. That is, they have the ability to spontaneously depolarize, and so function as pacemaker cells for the rest of the heart. Such cells are found in the sino-atrial node (SA node), in the atrio-ventricular node (AV node) and in certain specialized conduction systems within the atria and ventricles.

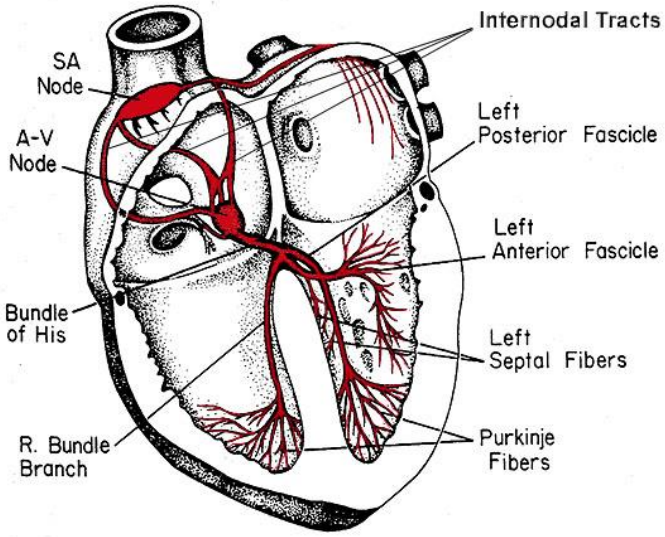

Fig. 1. Conduction system of heart

An action potential, once initiated in a cardiac cell, will propagate along the cell membrane until the entire cell is depolarized. Myocardial cells have the unique property of transmitting action potentials from one cell to adjacent cells by means of direct current spread (without electrochemical synapses).

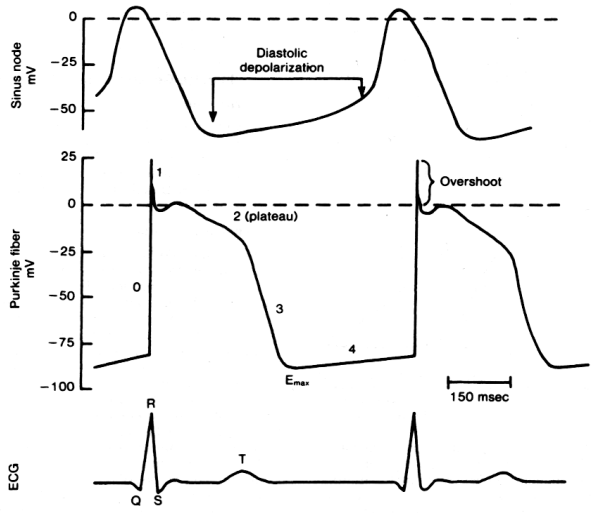

Fig. 2. Transmembrane potentials from the sinus node (upper panel) and a Purkinje fiber (middle panel); relationship to the ECG (the lower panel)

2. The physical basis of electrocardiography

As a result of the electrical activity of the cells, current flows within the body and potential differences are established on the surface of the skin, which can be measured using suitable equipment (ECG-apparatus). The graphical recording of these body surface potentials as a function of time produces the electrocardiogram. The simplest mathematical model for relating the cardiac generator to the body surface potentials is the single dipole model (Fig. 3). This simple model is extremely useful in providing a framework for the study of clinical electrocardiography and vectorcardiography, though of course much more complex treatments have been developed. The descriptions in this chapter are therefore simplifications to aid the understanding of the surface potential signal that manifests as an ECG.

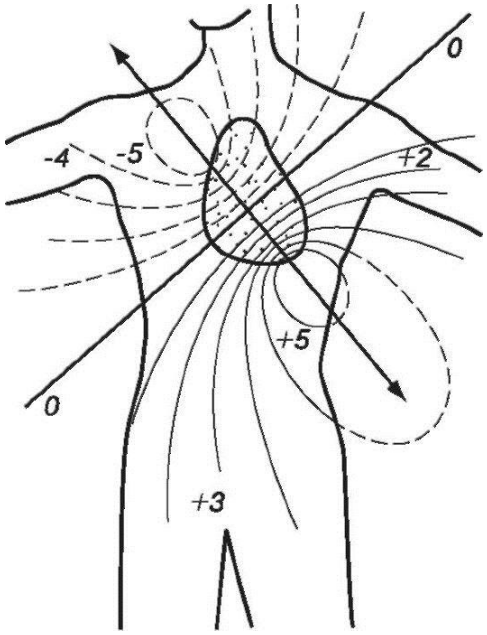

Fig. 3. Dipole model

3. Performing the electrocardiographic test

Steps to Perform an Electrocardiogram:

1.Ask your patient to remove electrical equipment such as mobile phones or watches that may interfere with the signal when performing the electrocardiogram.

2. The patient must also remove metallic objects, such as chains or bracelets, from the ECG electrode area.

3. Ask your patient to undress from the waist up and expose their ankles, in order to place correctly the electrocardiogram electrodes.

4. Make the patient lay down near the electrocardiograph.

5. Clean and disinfect with an alcohol solution those areas in which electrocardiogram electrodes are to be placed to ensure good attachment to the skin and a better EKG signal.

6. Place the EKG electrodes on the thorax, wrists and ankles. It is important to rigorously place the electrodes to ensure a correct reading of the electrocardiogram.

7. Advise your patient not to move nor speak, and to breath normally as the EKG is being carried out.

8. Record the electrocardiogram.

9. Check the electrocardiogram before removing the electrodes. Make sure every lead is well seen and that there are not many artefacts present. If in doubt, consult a doctor. Repeat the EKG procedure, if necessary.

10. Important: Always label the EKG tracing with the patient's full name, time and date.

11. Clean the electrodes from any remaining gel. Once your patient is dressed up, you can tell them when they will be informed of the electrocardiogram report and, if possible, who will be the doctor to do so

4. *ECG electrodes placement*

Limb electrodes:

The four Limb Electrodes are located on the patient's limbs. They are usually distinguishable by colour (Read Tips for placing EKG Electrodes) (Fig. 4)

RA: Right Arm.

LA: Left Arm.

LL: Left Leg.

RL: Right Leg (Ground).

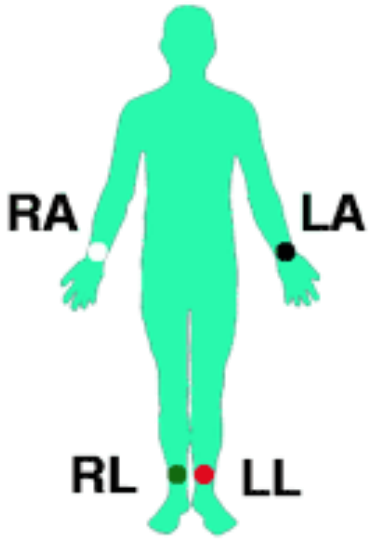

Fig. 4. The location of the limb electrodes

Precordial (chest) electrodes

Electrocardiogram Precordial Electrodes

The six precordial electrodes are located on the precordial region (Fig. 5).

V1: In the fourth intercostal space, just to the right of the sternum.

V2: In the fourth intercostal space, just to the left of the sternum.

V3: On a line midway between electrodes V2 and V4.

V4: In the fifth intercostal space, in the mid-clavicular line.

V5: At the same level as electrode V4, in the left anterior axillary line.

V6: At the same level as electrodes V4 and V5, in the left midaxillary line.

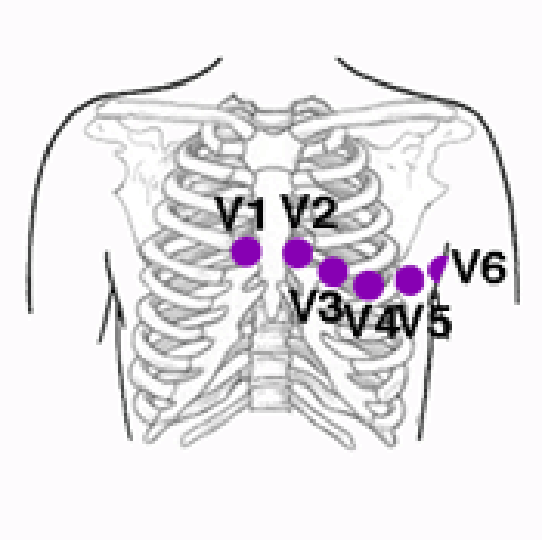

Fig. 5. The location of chest (precordial) electrodes

5. *ECG leads*

On the Electrocardiogram, leads are the tracing of the electrical potential difference between two points, either two electrodes (bipolar leads) or a virtual point and an electrode (unipolar leads).

It is important to know leads should not be analysed separately but as a whole, as each lead is a different point of view of the same electrical stimulus.

Depending on the electrical plane of the heart they register, we will group leads into limb leads (frontal plane) or precordial leads (horizontal plane).

Limb leads:

It is the name given to the electrocardiogram leads that are obtained from the electrodes placed on the limbs.

This leads provide electrocardiographic data from the frontal plane (not from the potentials which are directed forward or backward).

Limb leads are grouped into bipolar leads —also known as classic or Einthoven leads— and augmented unipolar leads.

Electrocardiogram Standard Bipolar Leads

Limb Leads and Einthoven's Triangle (Fig. 6).

They are the classic electrocardiogram leads as described by Einthoven. They register the potential difference between two electrodes situated in different limbs.

Lead I: Potential difference between right arm and left arm. Vector oriented to 0º

Lead II: Potential difference between right arm and left leg. Vector oriented to 60º.

Lead III: Potential difference between left arm and left leg. Vector oriented to 120º.

Triangle and Einthoven's Law: The three bipolar leads form what is called the Einthoven's Triangle (named so after the inventor of the Electrocardiogram). These leads maintain a mathematical proportion explained by the Einthoven's Law, which says: II = I + III.

This law is of great value when Interpreting an Electrocardiogram. It allows us to determine whether the limb electrodes are correctly placed; if the position of any electrode is altered, this law would not hold, thus allowing us to realise the ECG is not correctly done (See How to perform an ECG).

Unipolar Leads

On the Electrocardiogram, the unipolar limb leads register the potential difference between a theoretical null point at the centre of the Einthoven's triangle and the electrode of each extremity, thus allowing us to know the absolute potential in that electrode.

These leads were initially named VR, VL and VF. The V stands for Vector and R, L and F stand for Right, Left and Foot. Afterwards a lowercase a was added, which stands for augmented (present time unipolar leads are augmented with regard to the initial ones).

aVR: Right arm absolute potential, vector oriented at -150º.

aVL: Left arm absolute potential, vector oriented at -30º.

aVF: Left leg absolute potential, vector oriented at 90º.

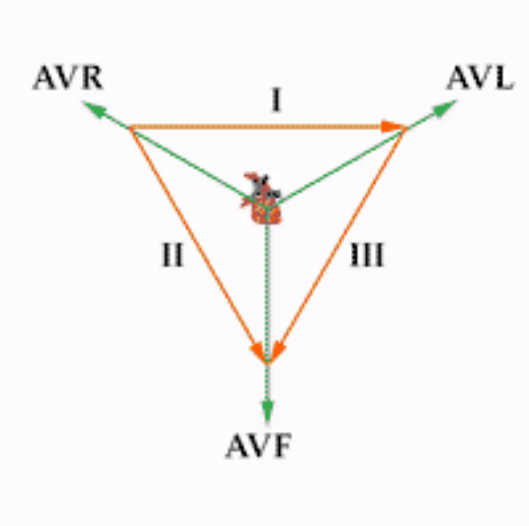

Fig. 6. Einthoven's Triangle

Precordial Leads (Chest Leads)

Chest Leads

There are six precordial leads. They are designated by a capital V and a number from 1 to 6 (Fig. 5).

They are unipolar leads, they register the absolute potential of the point where the electrode of the same name is placed.

They are the leads best suited for pinning down Left Ventricle abnormalities, especially of the anterior and posterior walls.

On a normal electrocardiogram, QRS are predominantly negative in V1 and V2 leads and predominantly positive in V4 to V6 (Rs pattern).

Precordial Leads

V1: This chest lead registers potentials from the atria, part of the septum and the right ventricle anterior wall. The QRS complex is formed by a small R wave (septum depolarization) followed by a deep S wave (ventricles activation), see QRS Morphology.

V2: The electrode for this precordial lead rests over the right ventricle wall. Therefore, the R wave is slightly bigger than in V1, followed by a deep S wave.

V3: It is the transitional lead between the electrocardiogram left and right potentials, as the electrode rests over the interventricular septum. The R and S waves are almost identical (Biphasic QRS).

V4: The electrode for this chest lead rests over the left ventricle apex, where the walls are thicker. Therefore, it presents a tall R wave followed by a small S wave (right ventricle depolarization).

V5 y V6: These electrocardiogram leads are situated over the Left Ventricle myocardium, which has thinner walls than in V4. Therefore, the R wave is not as tall as in V4, preceded by a small q wave (septum depolarization).

6. Genesis of the normal ECG

P WAVE

The normal atrial depolarization vector is oriented downward and toward the subject's left, reflecting the spread of depolarization from the sinus node to the right and then the left atrial myocardium (Fig. 7). Since this vector points toward the positive pole of lead II and to the negative pole of lead aVR, the normal P wave will be positive in lead II and negative in lead aVR. By contrast, activation of the atria from an ectopic pacemaker in the lower part of either atrium or in the AV junction region may produce retrograde P waves (negative in lead II, positive in lead aVR).

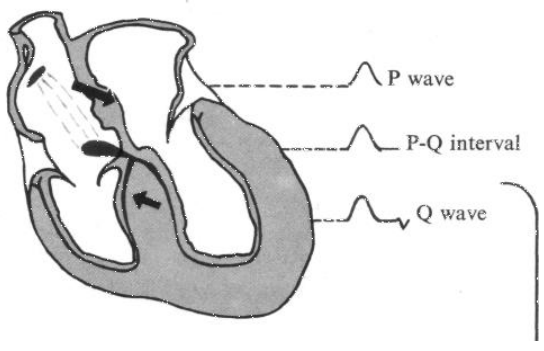

Fig. 7. The ECG deflections (P and Q)

QRS COMPLEX

Normal ventricular depolarization proceeds as a rapid, continuous spread of activation wavefronts. This complex process can be divided into two major, sequential phases, and each phase can be represented by a mean vector. The first phase is depolarization of the interventricular septum from the left to the right (Fig. 7).

P-wave: excitation of the atrial myocardium, initiated by the impulse discharge from the sino-atrial node.

PQ-interval: passage of the impulse through the atrio-ventricular node (slow) and a-v bundle (rapid).

Q wave: excitation of the interventricular septum from the Left bundle branch, fractionally before the rest of the ventricular myocardium.

The second phase results from the simultaneous depolarization of the main mass of the right and left ventricles; it is normally dominated by the more massive left ventricle, so that vector 2 points leftward and posteriorly (Fig. 8).

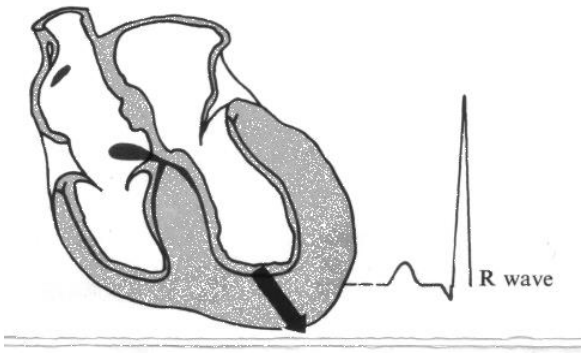

Fig. 8. The ECG deflections (R wave).

Synchronous excitation of the ma¬jority of both ventricles, via respective bundle branches; wave travels outwards through ventricular walls, average direc¬tion being mainly influenced by preponderant LV mass.

S wave deflects when excitation of remote portion of RV wall occurs, fractionally later than main ventricular excitation (Fig.9).

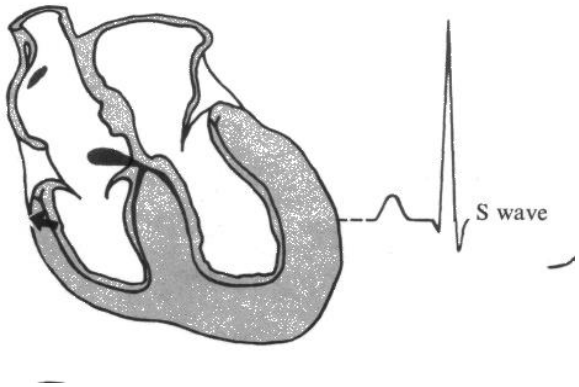

Fig. 9. The ECG deflections (S wave)

Therefore, a right precordial lead (V1) will record this biphasic depolarization process with a small positive deflection (septal r-wave) followed by a larger negative deflection (S wave). A left precordial lead, e.g. V6, will record the same sequence with a small negative deflection (septal q wave) followed by a relatively tall positive deflection (R wave). Intermediate leads show a relative increase in R-wave amplitude (normal R-wave progression) and a decrease in S-wave amplitude progressing across the chest from right to left. The precordial lead where the R and S waves are of approximately equal amplitude is referred to as the transition zone (usually V3 or V4).

Interval during which the ventricular myocardium remains in “excited” (depolarised) state is called as an ST interval (Fig. 10).

T WAVE AND U WAVE

Spread of electrical recovery wave through ventricular walls is slower than excitation. Normally, the mean T-wave vector is oriented roughly concordant with the mean QRS vector. Since depolarization and repolarization are electrically opposite processes, this normal QRS-T-wave vector concordance indicates that repolarization must normally proceed in the reverse direction from depolarization (i.e., from the epicardium to the endocardium or from the cardiac apex to the base).

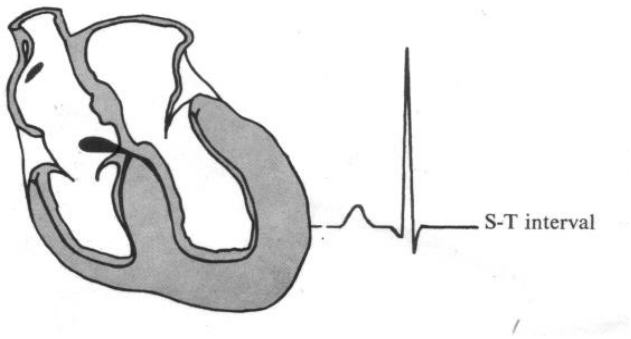

Fig. 10. ST interval.

The normal U-wave is a small, rounded deflection that follows the T-wave and usually has the same polarity as the T wave. An abnormal increase in the U-wave amplitude is most commonly due to drugs (e.g., quinidine, procainamide, disopyramide) or hypokalemia. Very prominent U-waves are a marker of increased susceptibility to the torsades de pointes type of ventricular tachycardia.

Diastolic interval, during which the myocardium remains in the resting (repolarised) state until sinoatrial discharge initiates the next cardiac cycle.

REMEMBER:

1. P-wave – atrial depolarization.
2. PQ(R)-interval – depolarization of atriae & AV-node.
3. QRS – ventricular depolarization.
4. ST-segment – electrical quiescence.
5. U-wave – part of ventriculae repolarization, corresponds to period of time when ventricles are most vulnerable to developing serious ventricular arrhythmias.

7. Characteristics of normal ECG

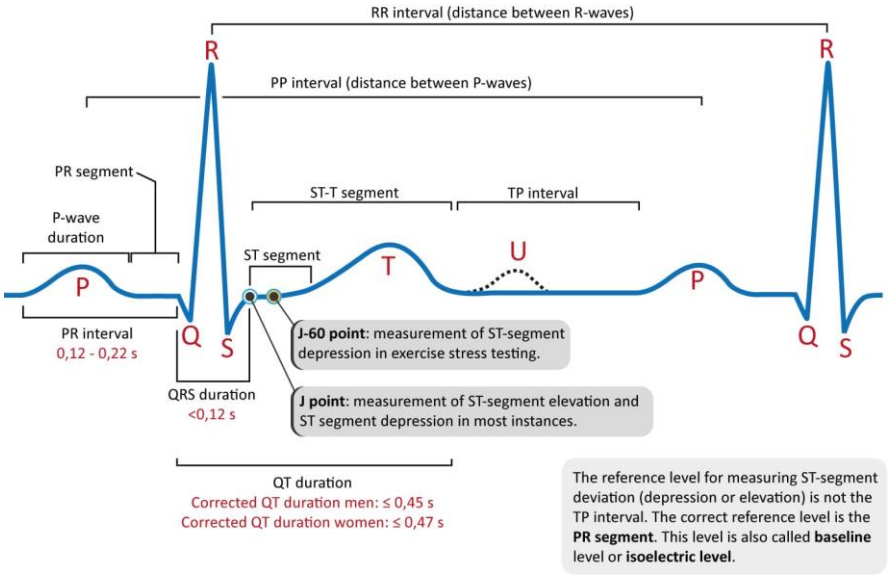

Fig. 11. Normal ECG waves

P-wave:

Duration 0,06 - 0,10 sec; amplitude 0,5 – 2,5 mm (Fig. 11).

P-wave is recorded after the impulse leaves the sinus node and corresponds to the time of atria excitation.

The wave PII > PI > PIII and PII, III, aVF < TII, III, aVF in the norm.

PI, PII, PV2-V6 - always positive.

PIII, PV1 – can be positive, two-phase or negative.

PaVR - always negative.

PQ(R)-interval:

Duration 0,12 - 0,20 (0,21) sec.

PQ interval includes P-wave and PQ(R)-segment and reflects the time of the excitation spreading on the atria and AV-junction, i.e. it characterizes the duration of AV impulse passage. PQ-interval tends to increase with a patient's age; in bradycardia PQ can make up to 0,21 sec. In tachycardia PQ interval shortens (PQ normal values are advised to determine by special tables).

QRS complex:

Duration 0,06 - 0,10 sec.

QRS-complex is recorded during excitation spreading along the myocardium of the ventricles.

Q-wave:

Duration < 0,03 sec, amplitude Q < 1/4 R (of the same lead).

R-wave: in standard and augmented leads R-wave amplitude depends upon electrical axis location; in the chest leads R-wave amplitude mustn't exceed 25 mm. Normally the amplitude of QRS-complex must exceed 5 mm in one of standard leads and 8 mm in one of the chest leads at least.

S-wave:

Duration SI < 0,04 sec, amplitude SI = 0 - 4 mm.

S in not obligatory wave, by the amplitude usually makes up ⅕– ¼ from the R-wave.

ST-segment:

Normally it is located on the isoline. It can be wavy, oblique ascending or oblique descending, but only in 1 mm limits.

N.B.! If 1 mm dislocation of ST-segment appears in dynamics, it is necessary to exclude the pathological ST depression or elevation.

ST-segment reflects the period when both ventricles are excited.

T-wave:

Duration 0,1 - 0,25 sec, amplitude TI, II - 3 - 5 mm.

T-wave reflects processes of ventricular repolarization. The largest (by its amplitude) T-wave is naturally recorded in the lead where there is the largest (by its amplitude) R-wave, and TI>T III. Normally TI, II, V2-6 is always positive, TIII, aVL, V1 can be smooth, negative or two-phase.

QT-interval:

Duration 0,35 - 0,44 sec.

QT-interval increase is a risk factor of sudden death from the ventricular fibrillation. Dispersion increase of QT-interval is a predictor of the development of fatal arrhythmia and sudden death.

U-wave:

Amplitude 1 - 2 mm, inconstant wave. The clinical importance has a marked U-wave (U ≥ T), which can point at hypokalemia. The U-wave amplitude also increases in bradycardia, physical exertion, complete heart block, use of cardiac glycosides, quinidine, novocainamide, etc.

Measurement of heart rate (HR):

Precise calculation of the heart rate requires measurement of cycle length. This is the duration between any two analogous deflections of successive complexes (R peak to R peak is often the most convenient). Measure RR-interval in sec (at 50 mm/sec paper speed, each small box = 0,02 sec, each large box = 5 small boxes = 0,1 sec). Divide 60 (the number of sec per minute) by RR:

HR=60/RR.

Determining the electrical axis (EA) of the heart:

By concept, the electrical axis (EA), or let's call it by its proper name, the QRS axis, is nothing else but the direction of the total vector of the ventriclular depolarization.

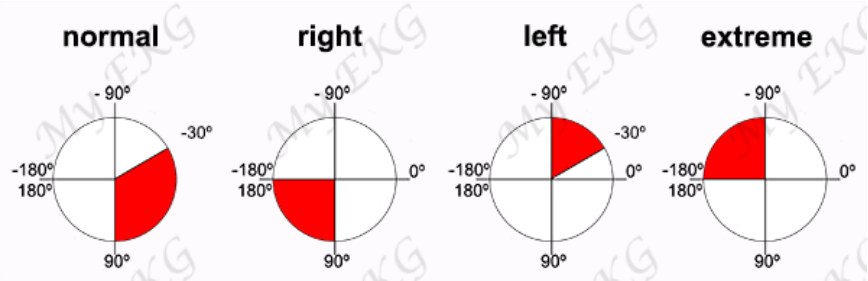

Fig. 12. Determining the electrical axis (EA) of the heart

Find the lead which defines the highest R-wave in the minimal S-wave or without S-wave – EA is located in this area. E.g.:

RImax - horizontal electrical axis position,

RaVLmax - left electrical axis deviation,

RIImax - normal electrical axis position,

RaVFmax - vertical electrical axis position,

RIIImax - right electrical axis deviation (Fig. 12).

8. Approach to ECG analysis

Algorithm of ECG- analysis:

1. Rate and rhythm.
2. Electrical axis.
3. P-wave morphology (size, shape, duration).
4. PQ(R) interval.
5. QRS morphology (size, shape, duration).
6. ST-segment, T-wave, U-wave changes.
7. QT interval.
8. Comparison with the previous tracings, if exist

CONTROL QUESTION

1. Electrocardiography. Physiological basis of the electrocardiogram. Genesis of the normal ECG.
2. The characteristic features of the ECG-leads (standard limb leads, augmented limb leads, unipolar precordial (chest) leads). Eintchovens triangle from the point of the genesis of the standard leads.
3. The ECG-wave morphology. The main elements of the ECG in the norm. Duration and amplitude of the waves, characteristic features of PQ- and QT- intervals, measurement of heart rate.
4. The notion “electrical axis”. Definition, electrical axis positions, method of electrical axis determination.
5. Approach to ECG analysis: consequence of the main steps, ECG-protocol filling in

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