THE ELECTROCARDIOGRAM, PRINCIPLES AND RECORDING

DEFINITION

The electrocardiogram (ECG or EKG) records the electrical potentials arising within the heart. The potential changes are measured by electrodes placed on the body surface. ECG records only the electrical activity and is not a measure of the mechanical behavior of the heart. Thus three of five major properties of the cardiac muscle (chronotropy, dromotropy, inotropy, bathmotropy and lusitropy) can be evaluated by the ECG:

- **chronotropy**: automaticity, pacemaker activity, the ability to initiate rhythmic electrical impulses;
- **dromotropy**: conductivity, the ability to conduct the action potential in the entire heart;
- **bathmotropy**: excitability (the ability to respond to a stimulus that has an intensity over the threshold) and modifications of the degree of excitability.

THE GENESIS OF THE ECG

BACKGROUND

Cardiac myocytes are excitable cells with polarized membranes. There is an electrical potential difference between the inner and outer side of the membrane. In the resting cell the inside of the cell is more negative than the outside; this is the resting membrane potential (Figure no. 1). The membrane potential arises from the interaction of ion channels and ion pumps embedded in the membrane that maintain different ion concentrations on the intracellular and extracellular sides of the membrane.

![Figure no. 1. Methods for measuring the cell's electrical activity. Upper part: using the intracellular method (measuring the potential difference between an intracellular microelectrode and an extracellular electrode) a negative potential is recorded in the resting cell. Lower part: using the extracellular method (measuring the potential difference between two extracellular electrodes located on opposite sides of the cell) no potential difference is recorded.](image-url)
Depolarization is a change in a cell's membrane potential, making it more positive, or less negative. If the depolarization is large enough (reaches threshold) results in an action potential. Depolarization is caused by an inward current (usually influx of cations).

Repolarization is a change in membrane potential that returns the membrane potential to the resting value. Repolarization is caused by an outward current (usually efflux of cations).

Hyperpolarization is a change in a cell's membrane potential that makes it more negative than the resting membrane potential. Hyperpolarization is caused by an outward current (often an efflux of cations).

**ACTIVATION OF MYOCARDIAL CELLS**

The cardiac myocytes are activated by an electrical signal. In the working heart this electrical signal is an action potential propagating from a neighboring cell. In experiments usually an external current stimulus is used to activate the myocardial cells (Figure no. 2).

Figure no. 2. Applying an external stimulus (arrow) to a myocardial cell will produce local depolarization (the inner face of the membrane will become positive and the outer face negative).

Once initiated, the depolarization will propagate on the cell membrane in every direction (Figure no. 3) and then from cell to cell.

Figure no. 3. Depolarization spreads in every direction on the cell membrane.

**ELECTRIC DIPOLE**

Stimulation of one end of a myocardial cell initiates a wave of depolarization that propagates through the cell. At a certain moment half of the cell will be depolarized, being negatively
charged on the outer surface of the membrane. At the other half of the cell, which is not yet depolarized, the membrane’s outer surface will be positively charged. The opposite charges of the cell surface will form an electric dipole (Figure no. 4). The potential differences arising in the heart (cardiac dipoles) can be represented by electrical vectors.

![Electric dipole diagram](image)

Figure no. 4. If half of the myocardial cell is depolarized, on the surface of the cell an electric dipole is formed, which can be represented with a vector.

**The electric dipole consists of two equal and opposite charges, separated by some (usually small) distance.**

If the electrical field is recorded by extracellular electrodes, a positive potential will be recorded when the vector points toward the positive electrode. If the vector points toward the negative electrode, a negative potential is recorded.

**CARDIAC DIPOLES AND VECTORS**

The vectors which represent electric field point, by convention, to the positive pole (orientation), while the length of the vector indicates the magnitude of the potential difference.

**Vectors are geometric objects that have magnitude (or length) and direction.**

A vector is frequently represented by a line segment with a definite direction, or graphically as an arrow, connecting an initial point with a terminal point. Mathematical operations (e.g. addition, subtraction) can be performed with vectors.
All basic vector operations can be applied to cardiac vectors. Thus cardiac vectors can be added, moved, measured and projected on conventional axes corresponding to the electrocardiographic leads.

Each depolarizing myocardial cell forms a dipole and thus can be represented by a vector called elementary vector. All these vectors can be brought to a single common point: the electrical center of the heart. The sum of all elementary vectors arising in the heart at a certain moment will create an instantaneous vector.

The potential differences generated by the heart change from moment to moment during the cardiac cycle. Once a single myocardial cell is stimulated the depolarization will propagate in every direction throughout the heart, thus a propagating wave of depolarization will be created.

*Each of the moments of the heart cycle can be described by an instantaneous vector with different size and orientation.*

The magnitude and the direction of the electrical forces that are generated by the heart can be recorded as a continuous series of vectors. The terminal points (arrowheads) of the instantaneous vectors describe curving lines (loops) around a central point (electrical center of the heart). The recording of this loop represents the vectorcardiography.

*The projection of this curve as function of time on an axis corresponding to a lead is actually the ECG in that particular lead.*

**IMPULSE PROPAGATION THROUGH THE HEART**

The heartbeat is initiated and controlled by electrical impulses that are generated and conducted by specialized myocardial cells. (Figure no. 5) Activation normally begins in the sinoatrial node (SA node, SAN) located in the right atrium of the heart. Because of its rapid firing rate, the SA node normally serves as the heart's pacemaker.

![Figure no. 5. The conduction system of the heart.](image)
The wave of depolarization initiated by the SA node is propagated through the atrial myocardium first to the right atrium, then the left atrial wall (Figure no. 6).

![Figure no. 6. The main vectors of atrial depolarization, oriented downwards and from left to right. The vector arising in the right atrium points to front, while the vector arising in the left atrium points to the back.](image)

After encountering a delay in the atroventricular node (A-V node, AVN), located in the septum close to the tricuspid valve, the wave of depolarization enters the ventricles through the AV bundle (His bundle). The His bundle bifurcates at the top of the interventricular septum into the right and left bundle branches (Tawara). Impulses transmitted via the bundle branches then enter the Purkinje system, a subendocardial network of rapidly conducting cells that synchronizes ventricular activation (Figure no. 7).

![Figure no. 7. The normal activation sequence of the ventricles. First the upper part of the interventricular septum depolarizes, the arising vector points to the right; then the remaining of the interventricular septum is activated and symmetrically the walls of the ventricles, giving rise to a vector oriented to the left and downwards; finally the latero-basal portion of the left ventricular wall is depolarized, the vector points upward and to the left.](image)

Microscopically, the wave of de/repolarization propagates to adjacent cells via gap junctions located between the cells. The heart is a functional syncytium: electrical impulses propagate freely between cells in every direction.

ECG LEADS

**INTRODUCTION, DEFINITIONS**

The ECG records the potential changes generated by the activity of the heart using electrodes placed on the surface of the body. The placement of electrodes is standardized. The potential change recorded by specifically connected electrodes is called a lead. Each
lead is assigned with an axis and each of the axes has an orientation: by convention the sense of the axis is toward the positive electrode. The projection of cardiac vectors as function of time on the axis corresponding to a lead is the ECG trace recorded in that particular lead. If the orientation of the projected vector corresponds to the orientation of the axis, a positive wave is recorded, if opposite, then a negative wave is recorded. Because the human body is a non-homogeneous conductor, it is necessary to standardize the leads grouped in lead systems. In case of a standard ECG, three lead systems are used: the limb leads, the augmented limb leads, and the chest leads. If necessary, other leads can be recorded using specific electrode locations.

**Limb Leads**

The limb leads use three active electrodes and a grounding electrode. Electrode placement is standard (developed by Einthoven); the electrodes are usually color-coded and labeled. The European color scheme:
- right arm (R): red
- left arm (L): yellow
- left foot (F): green
- right foot: black – the grounding electrode

The limb electrodes can be far down on the limbs avoiding bony prominences or close to the hips/shoulders, but they must be symmetrical.

Figure no. 8. Placement of electrodes for the limb leads

> Willem Einthoven, a Dutch doctor and physiologist, invented the first practical electrocardiogram in 1903 and received the Nobel Prize in Medicine in 1924 for it.

The limb leads are bipolar: they record the potential difference between two electrodes influenced by the dipole.

This lead system is formed by three leads called leads I, II and III (Figure no. 9).
Lead I records the potential difference between the right arm and the left arm:

$$LeadI = VL - VR$$

The axis of lead I is oriented towards the positive electrode, situated on the left arm (L).

Lead II records the potential difference between the right arm and the left foot:

$$LeadII = VF - VR$$

The axis of lead II is oriented towards the positive electrode, situated on the left foot (F).

Lead III records the potential difference between the left arm and the left foot:

$$LeadIII = VF - VL$$

The axis of lead III is oriented towards the positive electrode, situated on the left foot (F).

The three active electrodes are assumed to lay at the corners of an equilateral triangle, called the Einthoven triangle, the heart being at the orthocenter of this triangle. Thus, the sides of the triangle represent the axes of the limb leads.

Applying Kirchhoff’s second law to this electrical circuit the fundamental law of the limb leads is written:

$$LeadII = LeadI + LeadIII$$

**AUGMENTED LIMB LEADS**

The augmented limb leads use the same electrodes in the same position as the limb leads. These leads are unipolar: one of the electrodes (exploring electrode, considered positive) measures the potential generated by the dipole, while the other electrode (indifferent electrode) is not influenced by the dipole, recording a null potential. The indifferent electrode is obtained by Goldberger’s method, connecting the two non-exploring electrodes. The
potentials recorded using this method are smaller than those recorded with the bipolar method, thus supplementary amplification is needed (‘a’ – augmented).

This lead system is formed by three leads called leads aVR, aVL, aVF (Figure no. 10).

The axes of the unipolar limb leads are perpendicular to the axes of the limb leads, pointing towards the exploring electrode.

Applying Kirchhoff’s second law to this electrical circuit the fundamental law of the augmented limb leads is written:

\[ VR + VL + VF = 0 \]

**THE HEXAAXIAL SYSTEM**

The limb leads and the augmented limb leads explore the heart in frontal plane. The axes of these leads form the hexaaxial system. (Figure no. 11)
CHEST (PRECORDIAL) LEADS

The six precordial leads view the heart's electrical activity in the horizontal plane. These leads are unipolar. The exploring electrodes (labeled V1, V2, V3, V4, V5, and V6) are placed directly on the chest. The indifferent electrode is obtained by connecting the electrodes used for limb leads to a central terminal (Wilson) (Figure no. 12).

Figure no. 12. The electrical circuit for recording the chest leads (right) and the placement of precordial electrodes (left). R, L, F – the limb electrodes, WCT – Wilson’s central terminal, MC – midclavicular line, AA – anterior axillary line, MA – midaxillary line, ICS – intercostal space.

The placement of electrodes:
- V1 - fourth intercostal space, just to the right of the sternum;
- V2 - fourth intercostal space, just to the left of the sternum;
- V3 - between leads V2 and V4;
- V4 - fifth intercostal space in the midclavicular line;
- V5 - horizontally even with V4, but in the anterior axillary line;
- V6 - horizontally even with V4 and V5 in the midaxillary line.

The initial point of the axes of chest leads is the electrical center of the heart and the axes are oriented toward the exploring electrode (Figure no. 13).

Figure no. 13. The axes of the six chest leads in horizontal plane.
Leads V1 and V2 view the heart mainly from its right side, thus they are also called right precordial leads. Leads V5 and V6 view the heart mainly from its left side; they are left precordial leads.

Each of the twelve leads record the electrical activity of the heart from a different perspective, which also correlate to different anatomical areas of the heart:

- **anterior wall of the left ventricle**: V1-V4
- **lateral wall of the left ventricle**: D1, aVL, V5-V6
- **inferior wall of the left ventricle**: D2, D3, aVF
- **posterior wall of the left ventricle**: none of the leads, a mirror image appears in leads V1-V2

The standard electrocardiogram contains these twelve leads. If needed, other leads can be recorded, placing the electrodes on the posterior chest wall (V7-V8-V9), on the right side of the chest (V3R-V6R), or using esophageal or intra-cardiac electrodes.

### RECORDING THE ECG

The electrocardiograph is used to record the ECG. The main components of the electrocardiograph are:

- **the signal acquisition system**, which includes the electrodes and the cables. The electrodes are electrical conductors used to make contact with a non-metallic part of an electrical circuit. To record the limb leads four electrodes are used, and six additional electrodes are needed to record the chest leads. The electrodes are placed on the body surface in a standardized way (see Figure no. 8 and Figure no. 12). The cables connect the electrodes to the amplifying system; the connector type depends on the type of compatible electrodes. The cables are usually marked as the electrodes.

- **the amplification and signal filtering system** is used to amplify the recorded small potentials (mV range) and to limit the artifacts. To evaluate calibration, a rectangular 1 mV reference curve is recorded at the beginning of the trace (Figure no. 14).

![Figure no. 14. The reference curve (1 mV = 10 mm) used to evaluate the calibration of the electrocardiograph. A: correct settings; B: over-amplification – small artifact waves may appear and wave amplitudes are higher than real; C: under-amplification – wave amplitudes are lower than real, while small waves may disappear.](image)

- **the signal charting system** displays the ECG trace either on millimeter paper (Figure no. 15) or on a screen. One or more leads can be recorded simultaneously depending on the type of instrument.
RECORDING PROCEDURE

During ECG recording the patient is usually laying back. The room temperature should be between 18-22°C. Place and fix the limb and chest electrodes; attach the cables; use conductor gel if needed. The subject is ready for monitoring.

MORPHOLOGICAL DESCRIPTION OF THE ECG

The morphological analysis describes the elements of the ECG trace recorded during one heart cycle.

ECG PAPER

The ECG is recorded on millimeter paper. The amplitude of the waves is represented on the y-axis, while the x-axis it the axis of time. If standard settings are used, on the y axis 1 mV is represented as 10 mm (1 mm represents 0.1 mV) and on the on the x axis (standard paper speed of 25mm/s) 1 sec is represented as 25 mm (1 mm represents 0.04 seconds) (Figure no. 15).

![Figure no. 15. The ECG paper](image)

THE NORMAL ECG TRACE

The morphological analysis describes the elements of the ECG trace: waves, segments and intervals.

The baseline voltage of the electrocardiogram is known as the isoelectric line. The waves are deflections of the ECG trace from the isoelectric line. Waves are described by their:

- duration (measured in mm or msec/sec);
- amplitude (measured in mm or mV);
- axial orientation (expressed in degrees), and
- shape.

The waves of the ECG trace are: P, Q, R, S (forming the QRS complex), T, and U - the latter inconstantly present (Figure no. 16).

![Figure no. 16. The waves of the normal ECG trace](image)
The segments are parts of the ECG trace situated between two adjacent waves. They are described by their:

- **duration** (measured in mm or msec/sec) and
- **position** to the isoelectric line. If elevated or depressed, the direction and amplitude of the change, as well as the shape of the segment must be specified.

The segments analyzed on the ECG are the **PQ, ST, and TP** segments (Figure no. 17).

![Figure no. 17. The segments of the normal ECG trace](image)

The intervals are parts of the ECG trace situated between two markers. The intervals are described by their:
- **duration** (measured in mm or msec/sec).

The most often analyzed intervals of the ECG trace are the **PQ, QT, and RR** intervals (Figure no. 18).

![Figure no. 18. The intervals of the normal ECG trace](image)

The **P wave** represents the atrial depolarization. The characteristics of the normal P wave:
- **duration**: 0.08-0.10 sec (2-2.5 mm);
- **amplitude**: max. 0.25-0.3 mV (2.5-3 mm);
- **axial orientation**: 30°-60°;
- **shape**: round, in V1 can be biphasic (max. amplitude 1.5 mm).

The **PQ (PR) segment** represents the delay of conduction within the AV junction. The characteristics of the normal PQ segment:
- **duration**: 0.02-0.12 sec;
- **position**: isoelectric.

The **PQ (PR) interval** represents the electrical conduction from the SA node to the ventricles. The duration of the normal PQ segment is 0.12-0.21 sec.

The **QRS complex** represents the ventricular depolarization. The normal activation sequence of the ventricles generates narrow, sharp waves (Figure no. 19).
Figure no. 19. The normal activation sequence of the ventricles. During the depolarization of the upper part of the interventricular septum the Q wave arises; when the remaining of the interventricular septum and symmetrically the walls of the ventricles are activated the R wave arises; finally, when the latero-basal portion of the left ventricular wall is depolarized, the S wave is recorded.

The characteristics of the normal QRS complex:
► duration: below 0.12 sec / 3 mm (measured at the isoelectric line);
► amplitude: 0.5-1.6 mV (5-16 mm) in limb leads and augmented limb leads; in precordial leads the amplitude of the QRS complex is larger, because the ventricles are close to the exploring electrode (Figure no. 20);
► axial orientation: 30°-60°;
► shape: one or more sharp waves, positive or negative.

Figure no. 20. The amplitude of the R and S waves in the horizontal plane. The S wave is larger in the right precordial leads and the R wave is larger in the left precordial leads. The R and S waves are equal in V3 or V4, representing the transitional zone.

The nomenclature of the QRS complex is well established and strict. If the amplitude of the waves is higher than 3 mm (0.3 mV), uppercase letters (Q, R, S) are used to label them (Figure no. 21). If the amplitude of the waves is lower than 3 mm (0.3 mV) the waves are marked with lowercase letters (q, r, s). The first positive wave is the R wave; if there are additional positive waves in a QRS complex they are labeled R’, R”. If the negative deflection between two adjacent positive apices does not cross the isoelectric line, they are nor considered two distinct R waves, but a bifid R wave. The negative wave in front of the R wave is the Q wave, the negative waves that occur after the first positive wave are labeled S, S’. If there is no positive wave, the complex being formed by only a negative wave, this is named QS wave. The QS wave can have a notch on its descendent or ascendant slope.
The ST segment represents the early (plateau) phase of ventricular repolarization. The characteristics of the normal ST segment:

► duration: has no practical importance;
► position: isoelectric, but small ST segment elevations or depressions are not considered pathological (at most 1mm in V1 and V2 and / or at most 2mm in all other leads).

The T wave represents the final ventricular repolarization. The characteristics of the normal T wave:

► duration: 0.12-0.30 sec (no practical importance);
► amplitude: determined relative to the QRS complex, in limb leads approximately 1/3 of the largest R wave;
► axial orientation: 30°-60°; normally T wave is positive in every lead except aVR;
► shape: round and asymmetrical (faster descendent slope).

The QT interval represents the ventricular electrical systole.

► duration: dependent on the heart rate, usually considered normal if it’s under 50% of the RR interval (use charts or formulas to determine the adjusted value); very important for pathology.

The U wave is not always present, is created by ventricular afterdepolarizations. The characteristics of the normal U wave:

► duration: 0.15-0.25 sec,
► amplitude: less than 2 mm,
► shape: round.
ECG ANALYSIS

For physicians, practical understanding of the routine twelve lead ECG and the display on cardiac monitors provides an invaluable diagnostic skill. Every physician is expected to possess a reasonable level of expertise and skill in electrocardiography. As in case of the physical examination, it is desirable to follow a standardized sequence of steps in order to avoid missing subtle abnormalities in the ECG tracing, some of which may have clinical importance.

Several ECG interpretation methods are described, but the systematic approach is always required. Below a seven step ECG interpretation method is presented, covering the following chapters:

1. Rate
2. Rhythm
3. Axis
4. Rhythm disorders (arrhythmias)
5. Conduction disorders (heart blocks)
6. Hypertrophy
7. ECG changes in coronary artery disease

1. RATE

INTRODUCTION

The heart’s dominant (primary) pacemaker is the sinoatrial node (SAN). Thus, the normal, regular rhythm of the heart is the sinus rhythm. The resting heart rate is around 70 bpm (beats/min), but values between 60-100 bpm are considered normal.

Other structures of the conduction system of the heart also have pacemaker activity (automaticity): the atrio-ventricular node can generate excitation at a rate of 50-60 bpm, the His bundle at a rate of 30-40 bpm. The SA node is normally the dominant, driving pacemaker because it has the highest intrinsic rate of spontaneous automaticity. The higher frequency of SA nodal firing suppresses other pacemaker sites (subsidiary pacemakers) by a mechanism called overdrive suppression. Other cells can display automaticity because hypoxic conditions have triggered pacemaker currents.

PHYSIOLOGICAL VARIATIONS

► sinus tachycardia: the SAN paces the heart at a rate greater than 100 bpm; the most common cause is physical exercise;
► sinus bradycardia: the SAN paces the heart at a rate slower than 60 bpm; the most common cause is parasympathetic excess (e.g. at rest in conditioned athletes)

DETERMINATION OF THE HEART RATE

A. Can be calculated precisely:

\[ v = \frac{D}{t} \Rightarrow t = \frac{D}{v} \]

\[ v = \frac{1}{T} = \frac{V}{I_{RR}}, \quad T = \frac{I_{RR}}{V} \]
\[ v = \frac{25 \times 60}{I_{RR}} = \frac{1500}{I_{RR}} \]

where: \( v \) – speed, \( D \) – distance, \( t \) – time, \( V \) – speed of EKG paper (25 mm/s), \( I_{RR} \) – RR interval in mm, \( T \) – duration of the cardiac cycle (s), \( v \) – heart rate.

### B. Quickly, approximately:

Find a QRS complex that is situated on (or close to) a thick line on the millimeter paper. If the next QRS complex is situated on the next thick line, the heart rate is 300 bpm (RR interval duration = 5 mm, \( \frac{1500}{5} = 300 \)). If the QRS complex is situated on the second thick line (RR interval duration = 10 mm), the heart rate is 150 bpm; if the QRS complex is situated on the following thick lines, the heart rate is 100 bpm, 75 bpm, 60 bpm and 50 bpm respectively (Figure no. 37).

![Figure no. 37. Quick approximation of the heart rate.](image)

### 2. Rhythm

#### Introduction, Physiological Variations

The heart rhythm refers to the rhythmicity of the activity of the heart.

The normal (sinus) rhythm indicates:

- the rhythm is normally generated, by the sinoatrial node;
- the excitation propagates within the normal conduction system of the heart;
- the heart rate is within normal range.

The SAN generates impulses at a constant rate, producing heart cycles of equal length. Slight variations can occur with respiration, producing physiological respiratory arrhythmia. **Arrhythmia means abnormality of the rhythm.**

#### Rhythmicity Assessment

To assess the rhythm, first the rhythmicity should be evaluated.

In sinus rhythm impulses are generated at a constant rate, producing cycles of equal length (remember the physiological respiratory arrhythmia!). To determine if the heart cycles are of equal length, measure precisely the RR intervals which should be equal, i.e. duration of RR interval 1 = duration of RR interval 2 = \( \ldots \) = duration of RR interval \( n \).

Using a quick approach: mark on a piece of paper the position of 3-4 QRS complexes and compare it to other parts of the ECG trace. If the marks correspond to the position of the QRS complexes, the heart’s activity is rhythmic (Figure no. 38).
Criteria for sinus rhythm:

► the P wave is present in front of each QRS complex;
► the morphology of the P wave is constant in each cardiac cycle (within the same ECG lead);
► the distance between the P waves is constant (remember physiological respiratory arrhythmia);
► the P waves are positive in leads II and aVF.

If any of these criteria is not fulfilled, arrhythmia is present.

3. Axis

The electrical axis of the heart is the mean direction of the cardiac impulse. The QRS complex that represents ventricular depolarization is used for the determination of the electrical ventricular axis. The term electrical heart axis usually refers to the electrical axis in the frontal plane as measured by the limb leads. The axis of the P and T waves can be defined in the same way as the axis of the QRS complex.

The mean QRS vector

All of the instantaneous QRS vectors inscribed during ventricular activation can be added together to generate the mean QRS vector. Several different methods can be used to find the mean QRS vector, determining its position in the frontal plane. Precise measurements are done by using the hexaaxial system of Einthoven and the calculated amplitude of the waves (amplitude of the QRS complex): pick any two leads, calculate the amplitude of the QRS complex, represent the values in the hexaaxial system and compute the exact angle of the vector (Figure no. 39). This method, although very precise, needs exact calculation and is rarely used.
Figure no. 39. Precise determination of the mean QRS vector (example). Determine the amplitude of the QRS complex in two leads, mark the values in the hexaaxial system, draw perpendicular lines on the axes; the cross point of the two lines will give the orientation and sense of the mean QRS vector.

QUICK DETERMINATION – QUADRANT METHOD:

► Use only two axes: leads I and aVF.
► Decide if the wave (or the complex) is predominantly positive or negative in these leads.
► Mark the axes of leads I and aVF and determine in each case the semicircle in which the axis lays.
► By overlapping the two semicircles one can find the position of the axis with 90° precision (Figure no. 40).

Figure no. 40. Determining the mean QRS axis using the quadrant method (example). If in leads I and aVF the QRS complexes are both positive, the mean QRS axis is between 0-90°, considered normal.

Using this method one can quickly determine if the mean QRS axis is within normal range or deviated (Figure no. 41).

METHOD OF EQUIBIPHASIC WAVES

Find a lead with equibiphasic QRS complex (if there is one). In this lead the forces of the QRS complex in the positive and negative direction are equal. Often this is the lead with the smallest QRS complex. The QRS axis is perpendicular to this lead's orientation (remember that if the direction of the depolarizing wave is perpendicular to the axis of the lead, a biphasic wave is recorded in that lead, see also Figure no. 42).
If there is no lead with equibiphasic wave, there are usually two leads that are nearly equibiphasic, and these are always 30° apart. Find the perpendiculares for each lead and chose an approximate QRS axis within the 30° range.

![Diagram showing determination of mean QRS axis using equibiphasic waves example](image)

Figure no. 42. Determining the mean QRS axis using the method of equibiphasic waves (example). If the equibiphasic QRS complex is in lead aVL, the axis of the mean vector is perpendicular to the axis of lead aVL, i.e. parallel with the axis of lead II. Check the shape of the QRS complex in lead II: if positive, the vector points to the positive direction of the axis of lead II, that is 60°.

4. RHYTHM DISORDERS (ARRHYTHMIAS)

a. Sinus arrhythmia: the SAN generates excitation at irregular intervals. The other criteria of sinus rhythm are fulfilled, the morphology of the ECG trace is otherwise normal (Figure no. 43). This arrhythmia occurs mostly in sick sinus syndrome, caused by coronary disease, and should be differentiated from respiratory arrhythmia, which is a natural variation in heart rate that occurs during a breathing cycle. Respiratory arrhythmia represents subtle changes in the RR interval synchronized with respiration: the RR intervals shorten during inspiration and prolong during expiration (Figure no. 44).

![ECG trace in sinus arrhythmia](image)

Figure no. 43. The ECG trace in sinus arrhythmia.

![ECG trace in respiratory arrhythmia](image)

Figure no. 44. The ECG trace in respiratory arrhythmia.
b. Premature beats (extrasystoles) arise from ectopic pacemakers. The ectopic pacemaker can be situated at the level of:

- the atria;
- the AV junction;
- the ventricles.

Each localization leads to morphologically distinct types of premature beats having different clinical implications.

The non-sinus impulse is early, initiating a heartbeat before the next anticipated sinus beat, and is followed by a compensatory period. Thus, 2xI_{RR} is constant.

The atrial premature beat arises from an irritable focus in one of the atria (ectopic focus). The excitation wave depolarizes the atria prematurely (premature to the next timely sinus beat) and produces a P wave that looks different from a sinus-node generated P wave because the direction in which the atria depolarize is abnormal (abnormal P wave axis). The premature atrial impulse is conducted in a normal fashion via the AV node, the His bundle, and the bundle branches to depolarize the ventricles, thus the QRS complex associated with the atrial premature beat is normal (Figure no. 45).

The ventricular premature beat arises from an irritable focus in the ventricles (ectopic focus). The excitation is not conducted to the rest of the ventricles along the His bundle and bundle branches but along an abnormal pathway in the ventricular myocardium. Thus, conduction of excitation in the ventricles is slow, producing an abnormally wide QRS complex, and a bizarre looking T wave. Because the premature beat is ventricle-generated, there is no P wave before the QRS complex (Figure no. 46). The shape of the ventricular premature beat varies as function of the localization of the ectopic focus, thus mono-focal (arising from a single focus) and multi-focal (arising from different foci) ventricular premature beats exists.

Non-sustained run of premature beats means more than one premature beats following each other, with total duration of less than 30 seconds (Figure no. 47). The origin of the premature beats can be:
► in the atria;
► in the AV node (junction);
► in the ventricles.

The morphology of the waves is the same as in the case of a single premature beat.

![Figure no. 47. The ECG trace in a non-sustained run of premature beats. Note the series of 5 premature beats originating from the same ventricular focus. The rate of the run of premature beats is higher than the sinus rate (adapted from Indian Pacing Electrophysiol. J. 2010;10(8):357-371).]

**Paroxysmal (sudden) tachycardias** arise when an ectopic focus discharges and several premature beats follow each other. The term paroxysmal means the sudden onset and end of the event (or symptoms). The heart rate in this type of tachycardia is usually between 150-250 bpm, and the duration of the run of premature beats is more than 30 seconds (sustained). The ectopic pacemaker can be situated at the level of:
► the atria;
► the AV junction;
► the ventricles.

For the morphological aspects the same rules apply as in the case of a single premature beat.

**Supraventricular paroxysmal tachycardias** arise from a focus situated in the atria or the AV junction. Since during supraventricular paroxysmal tachycardias impulses depolarize the ventricles by passing down the His bundle and bundle branches, the accompanying QRS complexes are normal (Figure no. 48).

![Figure no. 48. The ECG trace in paroxysmal supraventricular tachycardia. Note the high heart rate, the lack of P waves and the normal morphology of the QRS complex and T waves (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).]

**Ventricular paroxysmal tachycardias** (VT) originate in one or several ectopic foci situated in the ventricle. Since these impulses are conducted to the rest of the ventricles via an
abnormal pathway in the ventricular myocardium and not through the His bundle and the bundle branches, the QRS complexes are broader than normal and with abnormal T waves. In case of monomorphic VT (arising from a single focus, i.e. monofocal), consecutive QRS complexes have the same appearance (Figure no. 49). Contrarily, in polymorphic VT (arising from several foci), consecutive QRS complexes can significantly differ. Torsades de pointes ("twisting of the spikes") is a particular form of polymorphic VT, giving a characteristic illusion of a twisting of the QRS complex around the isoelectric baseline.

Figure no. 49. The ECG trace in monomorphic ventricular paroxysmal tachycardia. Note the high rate and the wide QRS complexes, without distinguishable T waves (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).

c. **Fibrillation** defines a rapid, irregular, and unsynchronized contraction of muscle fibers. Based on the origin of the arrhythmia, it can be atrial and ventricular.

*Atrial fibrillation* is the most frequent arrhythmia. This is an irregular rhythm (absolute arrhythmia). In atrial fibrillation there is a disorganized activity, fibrillating (quivering) of the heart muscles of the atria, instead of a coordinated contraction. Because there is no synchronous contraction of the atrial muscle, the ability of the atria to serve as a pump is abolished. The ventricular inflow is lower; thus, the cardiac output is lower (but usually enough). Thus, atrial myocytes depolarize rapidly and randomly at a combined rate that exceeds 400 per minute. Instead of P waves, the quivering of the atria produces fine “f” waves on the ECG baseline. The AV node is constantly 'bombed' by depolarization impulses, but only some of these impulses manage to get through (associated functional AV block). The ventricular rate is usually between 110 and 180 bpm.

The pathomechanism of atrial fibrillation involves reentry circuits and several ectopic foci (zones in the walls of the atria that generate excitation and thus waves of depolarization that propagate until they reach a zone in refractory period). Because impulses that manage to pass through the AV node are conducted down the His bundle and the bundle branches, the ventricles are activated normally and the QRS complexes are normal in width and have the same morphology as in case of sinus rhythm (Figure no. 50).

Figure no. 50. The ECG trace in atrial fibrillation. Note the “f” waves that are irregular in amplitude and frequency, and the irregularly irregular ventricular rhythm (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).
In ventricular fibrillation (VF), ventricular myocytes depolarize rapidly and randomly at a combined rate of at least 400 bpm. Instead of the normal ECG trace, no baseline (i.e. no isoelectric line) can be found, there are no clear and reproducible waves (i.e. no P, Q, R, S, T waves) and is associated with AV dissociation (Figure no. 51). The chaotic depolarization of the ventricles produces no depolarization wave; mechanically, this results in an arrested cardiac pump function. If untreated, ventricular fibrillation results in immediate death.

**Figure no. 51.** The ECG trace in ventricular fibrillation. Ventricular depolarization causes rapid chaotic oscillations in the baseline (adapted from commons.wikimedia.org).

d. Flutter, as an intransitive verb, means "to flap, to vibrate" or "rapid wavering motions". Based on the origin of the arrhythmia it can be atrial and ventricular. The atrial flutter originates in the atrium and is caused by a special form of reentry in which the wave of depolarization moves in a loop in the atrial wall. The atria activates at a rate of about 300 times per minute (250-350 bpm). Each atrial depolarization produces a wave on the ECG trace, giving it a 'saw tooth' appearance in one or more leads ("F" waves). Due to its longer refractory period, the AV node exerts a protective effect on heart rate by blocking atrial impulses in excess of about 180-200 bpm. A 2:1, 3:1, or 4:1 AV dissociation rather than block occurs, yielding a ventricular response rate of 150, 100, or 75 bpm (Figure no. 52). Once atrial flutter impulses pass the AV node, they depolarize the ventricles by passing down the His bundle and bundle branches, thus the accompanying QRS complexes are normal.

**Figure no. 52.** The ECG trace in atrial flutter. Note the "F" waves giving rise to regular saw-tooth-like undulations of the baseline and the variable AV dissociation (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).

5. CONDUCTION DISORDERS (HEART BLOCKS)

In atrioventricular conduction blocks there is a pathological delay in conduction at the AV node. To recognize the main signs of heart block watch the PQ (PR) interval and the P to QRS relationship.

Several degrees of severity exist:
  ► first degree,
  ► second degree, and
  ► third degree atrioventricular block.
In **first degree atrioventricular block** the cardiac rhythm originates in the sinus node, each P wave of atrial depolarization is followed by a QRS complex of ventricular depolarization. The time from the initial depolarization of the atria to the initial depolarization of the ventricles is abnormally delayed. This pathologic delay is reflected in a longer PQ interval (above its upper limit of 210 msec) (Figure no. 53).

![Figure no. 53. ECG trace in first degree AV block. Each P wave is followed by a QRS complex, but the PQ interval is abnormally (and equally) prolonged to 300 msec (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).](image)

In **second degree atrioventricular block** the transmission of the depolarizing impulse from the sinus node through the AV conduction system of the heart is interrupted intermittently. The P wave of atrial depolarization is not always followed by a QRS complex. The PQ (PR) interval is still prolonged.

Two major types of second degree atrioventricular block are described:

- Mobitz I,
- Mobitz II.

**Mobitz I type (with Wenkebach’s phenomenon)** occurs when the PQ interval prolongs from beat to beat up until the drop-out of one QRS complex (Figure no. 54). This indicates increasing delays in AV conduction. The gradually increasing PQ (PR) intervals are called Wenkebach’s intervals (or this gradual increase is called Wenkebach phenomenon).

![Figure no. 54. ECG trace in second degree AV block, Mobitz I type. Note the progressive prolongation of the PQ interval, followed by a blocked P wave (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).](image)

**Mobitz II type second degree AV block** occurs when beats are dropped irregularly without PQ interval prolongation. A non-conducted P wave occurs suddenly without progressive prolongation of the PQ interval (Figure no. 55). The PQ interval can be normal or prolonged, but it is constant. This is a more severe disease, can progress to third degree AV block.

![Figure no. 55. ECG trace of second degree AV block, Mobitz II type. The PQ interval does not increase prior to the dropped beat (adapted from en.ECGpedia.org).](image)
In *third degree (complete) atrioventricular block* all the SA node impulses are blocked, and none is conducted to the ventricles. In the absence of an alternative pacemaker, ventricular contraction comes to a standstill and the patient dies. However, an ectopic pacemaker below the block usually takes over ventricular pacing. The lower pacemaker can be part of the conduction system or an ectopic focus in the ventricular wall. Since the SA node and the ectopic pacemaker pace the atria and ventricles independently, the P waves bear no relationship to the QRS complexes (Figure no. 56). If the block is high in the AV node and the ventricular pacemaker is located lower in the AV junction the QRS complex is normal in width, because the ventricular activation is via the bundle branches. If the block is low in the AV junction, the ventricles are paced by an idioventricular pacemaker, and the QRS complexes will be wide.

**Figure no. 56.** ECG trace in third degree AV block. Note the regular P waves and regular QRS complexes (low ventricular rate), without any relationship between them (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).

**Escape rhythm** means heart rhythm arising from an ectopic focus situated
► in the AV junction or
► in the ventricles.

The escape beat arises when the sinus node fails in its role as a pacemaker or when the sinus impulse fails to be conducted to the ventricles as in complete heart block. The ectopic impulse is always late, appears only after the next anticipated sinus beat fails. If the sinus node failure or heart block is brief, the ectopic focus may generate only a single escape beat. If the sinus node failure or heart block is prolonged, the ectopic focus produces a rhythm of escape beats to assume full pacing function (escape rhythm). The escape mechanism offers protection against total cardiac standstill in the event of sinus node failure or complete heart block.

Compared to single premature beats only the timing is different, for the morphological aspects the same rules apply (Figure no. 57).

**Figure no. 57.** ECG traces of escape rhythms (upper trace: junctional escape rhythm, lower trace: ventricular escape rhythm). Note the ectopic beats arising late, and also the morphological similarities to premature beats.
Bioethical considerations

(adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).

In **bundle branch blocks** the conduction defect is in one of the bundle branches. If the two bundle branches exhibit a block simultaneously, the progress of activation from the atria to the ventricles is completely inhibited; this is regarded as third-degree atrioventricular block. In case of a bundle branch block the activation of one ventricle must await initiation by the opposite ventricle (depolarized through normal conduction). The activation of the ventricle in this case is done entirely on a cell-to-cell basis in the myocardial muscle. In the absence of involvement of the conduction system, there is a much slower activation process. The normal synchrony of right and left ventricular depolarization is lost. The resulting abnormality produces on the ECG trace bizarre shaped QRS-complexes of abnormally long duration.

In **right bundle-branch block (RBBB)** the electrical impulse cannot travel through the right bundle branch to the right ventricle. The depolarization of the right ventricle is done on a cell-to-cell basis after the depolarization of the left ventricle and the septal muscle mass. This progress is slower than that through the conduction system and leads to a wide QRS-complex. Activation of the right ventricle is so much delayed, that it occurs following the activation of the left ventricle.

![Figure no. 58. ECG trace in right bundle branch block. Note the wide terminal R wave in the right precordial leads and the wide S wave in the left precordial leads (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).](image)

The abnormal terminal QRS-vector that is directed to the right ventricle (i.e., rightward and to front) produces on the ECG trace a broad terminal S-wave in lead I, V5-V6 and in lead V1-V2 a notched R-wave or rSR’ shape complex (Figure no. 58). The abnormal spread of depolarization alters the pattern of repolarization; for this reason, secondary T wave changes may occur (T wave inversion).

In **left bundle-branch block (LBBB)** the electrical impulse cannot travel through the left bundle branch to the left ventricle. The depolarization of the left ventricle is done on a cell-to-cell basis after the depolarization of the right ventricle and the septal muscle mass. This progress is slower than that through the conduction system and leads to a wide QRS-complex. Activation of the left ventricle is so much delayed that it occurs following the activation of the right ventricle.

The electric heart vector makes a slower and larger loop to the left, this produces on the ECG trace a broad and tall R-wave, usually in leads I, aVL, V5, or V6 and deep, broad S waves in V1-V2 (Figure no. 59). The abnormal spread of depolarization alters the pattern of
repolarization; for this reason, secondary T wave changes may occur (T wave inversion) in the chest leads.

![Figure no. 59. ECG trace in left bundle branch block. Note the wide S wave in the right precordial leads and the wide notched R wave in the left precordial leads (adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com]).](image)

**PRE-EXCITATION SYNDROMES**

In the most common type of pre-excitation syndrome the passage of activation from the atrium is directly to the ventricular muscle via an abnormal route (the bundle of Kent) which bypasses the AV junction. Thus, part of the ventricular muscle is activated before normal activation reaches it via the conduction system (after a delay in the AV junction). The resulting ECG depends on the specific location of the accessory pathway. This is one of the causes of wide QRS complexes.

On the ECG trace the QRS-complex initially exhibits an early upstroke called the delta wave, the interval from the P-wave to the R spike is normal, but the early ventricular excitation forming the delta wave shortens the PQ interval (Figure no. 60)

![Figure no. 60. ECG trace in the most common pre-excitation syndrome. Note the early upstroke of the QRS complex (delta wave, arrows) and the shortened PQ interval (adapted from en.ECGpedia.org).](image)

**6. HYPERTROPHY**

**INTRODUCTION, DEFINITIONS**

Hypertrophy means the increase in the volume of an organ or tissue due to the enlargement of its component cells. In case of hyperplasia, the cells remain approximately the same size, but increase in number.
Dilation means enlargement of a cardiac chamber.

Atrial and ventricular muscles react to physical stress in the same way as skeletal muscles: the muscles enlarge with increased amount of exercise. The extra tension may arise as a result of increased pressure load or volume load.

These principles and definitions apply to all compartments of the heart.

**ATRIAL ABNORMALITY**

Atrial abnormality means atrial enlargement, atrial dilatation, or atrial hypertrophy. Because these pathologic conditions cannot be distinguished on surface ECG, they are commonly referred to as atrial abnormality. Signs of atrial abnormalities can be found in leads in which the P wave is most prominent: usually lead II, but also leads III, aVF, and V1. Normally, the right atrial depolarization wave precedes that of the left atrium and the sum gives the normal P wave (less than 100 msec wide and less than 2.5 mm high).

The **right atrial abnormality** is a consequence of right atrial overload. The right atrial depolarization lasts longer than normal. The amplitude of the right atrial depolarization vector is unchanged but its maximal value now falls on top of that of the left atrial depolarization vector. The resulting P wave is taller than normal (taller than 2.5 mm), but its width remains within 100 msec (Figure no. 61). The tall P wave found in the frontal leads is called P pulmonale (right atrial hypertrophy most often caused by pulmonary disorders). A tall biphasic P wave in V1 is another sign suggesting atrial hypertrophy. In right atrial hypertrophy, the initial positive portion of the biphasic P wave is larger than the terminal negative portion.

![Figure no. 61. P wave changes in right atrial hypertrophy. Note the tall P wave in frontal leads and the asymmetrical P wave with larger initial positive phase in lead V1.](image)

**Left atrial abnormality** is a consequence of left atrial overload. The left atrial depolarization lasts longer than normal but its amplitude remains unchanged. Therefore, the height of the resultant P wave remains within normal limits, but its duration is longer than 100 msec. A notch near its peak may or may not be present (Figure no. 62). The wide, notched P wave found in the frontal leads is called P mitrale (left atrial hypertrophy most often caused by mitral valve disorders). A biphasic P wave in V1 with its terminal negative deflection more than 40 msec wide and more than 1 mm deep is another ECG sign of left atrial abnormality.

![Figure no. 62. P wave changes in left atrial hypertrophy. Note the wide, notched P wave in frontal leads and the asymmetrical P wave with larger terminal negative phase in lead V1.](image)
VENTRICULAR HYPERTROPHY

In case of normal ventricles the QRS complexes are predominantly negative with small R waves and relatively deep S waves in right precordial leads. In left chest leads, the QRS complexes are predominantly positive, with tall R waves. Leads V3 and V4 reflect a transition between the right and left chest leads (see Figure no. 20). The normal transition zone, where the R wave and S wave are equal, is between V3 and V4. Early transition may appear in V2, while late transition may not appear until V5 or V6.

The right ventricular hypertrophy is a consequence of right ventricular overload. The increase in the muscle mass of the right ventricle produces an increase in the ventricular electrical forces directed to the right ventricle (to the right and front). On the ECG trace in V1 the QRS complexes are positive, with tall R waves, and the S waves are unusually deep in V5-V6 (Figure no. 63). To determine the presence of right ventricular hypertrophy the Sokolov-Lyon index for the right ventricle should be calculated (based on the amplitude of the waves of the QRS complexes in leads V1 and V5). Normally,

$$ RV_1 + SV_5 \leq 10.5 \text{mm} $$

If the index is higher than 10.5 mm, right ventricular hypertrophy is present.

Right ventricular hypertrophy also causes right axis deviation and is often accompanied with signs of right atrial enlargement (resulted from the right atrium having to pump blood into a thick-wall, non-compliant, hypertrophied right ventricle).

![Figure no. 63. ECG trace in right ventricular hypertrophy. Note the right axis deviation; the tall R wave in lead V1 and the deep S wave in lead V5.](image)

The left ventricular hypertrophy is the consequence of left ventricular overload. The increase in the muscle mass of the left ventricle produces an increase in the ventricular electrical forces directed to the left ventricle (to the left and posterior). On the ECG trace, in lead V1 the QRS complexes are negative, with deep S waves, and the R waves are unusually tall in V5-V6 (Figure no. 64). To determine the presence of left ventricular hypertrophy the Sokolov-Lyon index for the left ventricle should be calculated (based on the amplitude of the waves of the QRS complexes in leads V1 and V5). Normally,

$$ SV_1 + RV_5 \leq 35 \text{mm} $$

If the index is higher than 35 mm, left ventricular hypertrophy is present.

Left ventricular hypertrophy also causes left axis deviation due to the overpowering current generated by a hypertrophied left ventricle. It is often accompanied with signs of left atrial enlargement (resulted from the left atrium having to pump blood into a thick-wall, non-compliant, hypertrophied left ventricle). Ventricular repolarization changes manifest as downward sloping of the ST segment and T wave inversion (ventricular strain pattern).
7. ELECTROCARDIOGRAPHIC CHANGES IN CORONARY ARTERY DISEASE

Ischemia occurs if there is a stenosis (abnormal narrowing) of a coronary artery, the transport of oxygen to the cardiac muscle is decreased, causing an oxygen debt in the muscle. Ischemia causes changes in the resting potential and in the repolarization of muscle cells, which is seen as changes in the T-wave (Figure no. 65):
► inversion and/or
► symmetrical and/or
► sharp-tall T waves.

More severe abnormalities in coronary circulation can lead to ECG abnormalities described under the electrocardiographic term of lesion. These abnormalities involve ST segment deviations. The ST segment can be elevated (usually the electrocardiographic mark of acute myocardial infarction) or depressed, by more than 1 mm in leads V1-V2, and/or more than 2 mm in other leads. The electrocardiographic term of lesion is only used for horizontal or descendent ST segment deviations (Figure no. 66).

Complete occlusion of a coronary artery (myocardial infarction), if left untreated, usually leads to necrosis of myocardial cells affected by the lack of oxygen. The electrocardiographic
sign of necrosis is the pathological Q wave. The pathological Q wave has a duration of at least 0.04 sec and an amplitude of at least 1/4 of the adjacent R wave (Figure no. 67).

In untreated (non-revascularized) acute myocardial infarction, caused by complete occlusion of a coronary artery, the ECG depicts five distinct phases, according to the moment of ECG recording following the occlusion of the coronary artery. In the first phase of a myocardial infarction (during the first minutes after coronary artery occlusion) T wave abnormalities (hyperacute, sharp, tall T waves) will be present on the ECG (1). Then, the ECG will show ST segment elevation (2). ST-segment elevation generally occurs with reciprocal ST depression in ECG leads in which the axis is opposite in direction from those with ST elevation. Then, hours to days after the acute phase, pathological Q waves appear (3). Pathologic Q waves typically appear within the first 9 hours of infarction, with a wide interval, ranging from few minutes to 24 hours. Within hours to days, an evolving myocardial infarction will typically demonstrate T-wave inversion (4). Finally, after a peak elevation approximately 1 hour after the onset of chest pain, the ST segment reaches a plateau at about 12 hours, and normalizes completely (goes back to the isoelectric line) within 2 weeks (5). The typical electrocardiographic evolution of an acute myocardial infarction can be strongly influenced by revascularization strategies.

The **signs of infarction** appear only in the leads that view the affected territory of the heart muscle. Different leads view the heart from different angles; the 12 ECG leads can be used to distinguish myocardial infarction occurring in different regions of the heart:

- anterior ventricular wall: V1-V4;
- lateral wall of the left ventricle: I, aVL, V5-V6;
- inferior wall of the left ventricle: II, III, aVF.

Unfortunately none of the leads views the posterior wall of the left ventricle. In case of posterior infarction a 'mirror image' appears in leads V1-V2. To localize the infarction zone other leads can be used beside the standard 12-lead ECG: V7-V8-V9, the electrodes being placed on the posterior chest wall.
8. MISCELLANEOUS

CARDIAC PACING

A patient needs a pacemaker when electrical impulse conduction or formation is dangerously disturbed. The pacemaker rhythm can usually be recognized on the ECG trace, as it shows pacemaker spikes (Figure no. 68): vertical signals that represent the electrical activity of the pacemaker. The position of the pacemaker spikes depend on the site of stimulation: atrial (in front of the P wave), ventricular (in front of the QRS complex), or dual (two pacemaker spikes).

![Figure no. 68. ECG trace in dual chamber pacing. Note the pacemaker spikes both before the P wave and the QRS complex.](image)

ELECTROLYTE DISORDERS

In case of hyperkalemia narrow and tall peaked T waves appear (Figure no. 69, A). Normally it is unusual for T waves to be taller than 5 mm in limb leads and taller than 10 mm in chest leads. As serum potassium concentration continues to rise, the PQ intervals become longer, the P waves loose amplitude and may disappear, and QRS complexes widen (Figure no. 69, B). If the rise in serum potassium continues, the heart arrests in asystole.

![Figure no. 69. ECG trace in hyperkalemia. Note the tall, narrow T waves (A) and the wide QRS complex (B). Adapted from ECG- A Pictorial Primer, by David C Chung [http://www.medicine-on-line.com].](image)

The narrow and tall peaked T wave of hyperkalemia may be confused with the hyper-acute T wave occasionally seen in transmural myocardial infarction! The patient’s presenting history and physical findings would help to differentiate.

In case of hypokalemia the T waves become flattened, together with appearance of a prominent U wave (Figure no. 70); the ST segment may become depressed and the T wave inverted. Unlike in hyperkalemia, these additional changes are not related to the degree of hypokalemia.
When analyzing the ECG trace always correlate the ECG findings with the patient's clinical presentation. If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions. Even with standardized ECG, the normal range variability is high between different patients and a change in the ECG of the same patient has a higher significance than the comparison with the standard ECG.