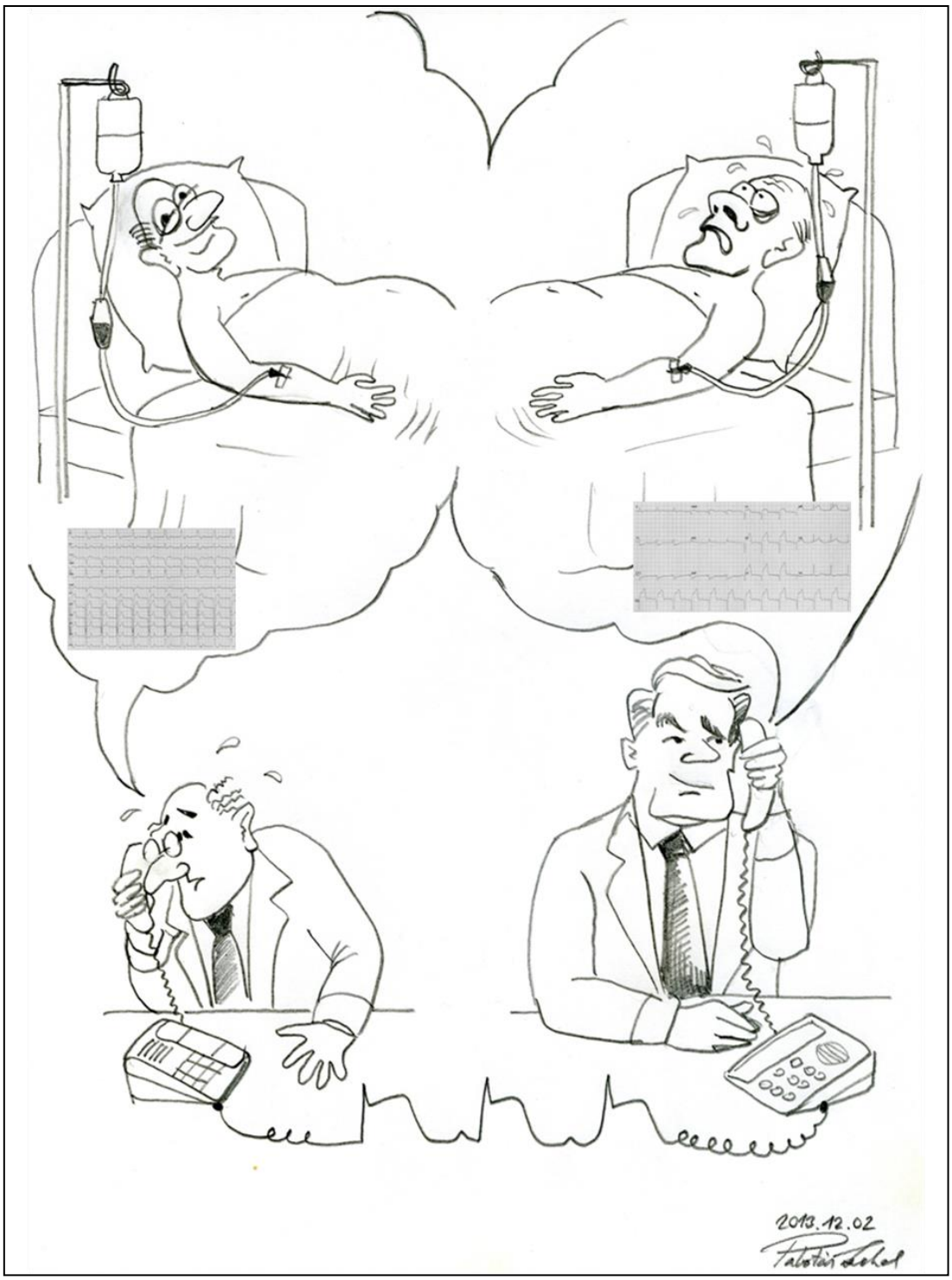
ECG BASICS



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INTRODUCTION

By writing these lecture notes, our goal was to provide an overview on the aspects of ECG interpretation. There are numerous excellent books in Hungarian and English, which contain brilliant illustrations and may be considered for us as governing. Our objective was not to fill in a gap in the literature, but to organize the educational viewpoints in clinical physiology. We would like to provide a summary, which may make it easier to understand how individual waves are originating and which may be flipped through again later if you are facing some problems during the interpretation.

An ECG tracing carries much more information than we would think, but its significance is merely underestimated due to its cheap nature and simple execution. I hope that by the time its readers complete these lecture notes, they will realize that a lot of diseases can be diagnosed even on the basis of medical history, physical examination and ECG or they can help decide on the next diagnostic step to be taken during differential diagnosis.

The lecture notes are recommended not only for medical students, but also colleagues performing ECG interpretations frequently may also find novelties while reading the 'fine print' sections. In each chapter, we tried to subdivide the information to be learned by students as well as the knowledge that may appear to be less important in a way that the latter one was written in 'fine print'. These sections in 'fine print' assist in the understanding of the subdivision of arrhytmias and may also be important from differential diagnostic aspects in a more precise ECG evaluation.

We would like to emphasize that your opinion created during the ECG interpretation should be worded with precise terms that carry the same meaning for all healthcare professionals. By using the accurate description, it can be avoided that, during a medical consultation (eg. on the phone), one of the consulting parties would say that the patient has a 'very ugly' ECG tracing. In addition, we would like to stress that it is essential to differentiate between signals and noises. We often encounter with the problem that a myopotential originating from muscle movements (eg. tremor) is erroneously considered to be a wave of cardiac origin, which may frequently be misleading in regard to the basic rhythm. Therefore, ECG tracings with noise will also be presented in the last chapter.

The artistic visualization of the graphic charts in the book originates from Lehel Palotás, a cardiac surgeon colleague. In particular, it should be noted that, in contrast to the books where 'sterile' ECG tracings generated by a computer and being free of any noises can be seen, we tried to demonstrate vivid and real-life ECG recordings. One could think that searching for ECG tracings that look like 'textbook cases' is easy because it is them that occur most frequently. However, it is not like this; a search has to be made for a 'good' recording, through which a specific phenomenon can be demonstrated. Sándor Nagy was a great help in the scanning process, who tried his best effort to make the available materials presentable. As a consequence of these, the quality of some of the individual recordings is substandard. The pool of the more than 150 ECG recordings is largely the result of my own collection or it was made available for us by Ildikó Beke, a cardiologist colleague. I would like to thank Ildikó Beke and Áron Gábor Fülöp for reading over these lecture notes as well as for their critical evaluation. I hope that I can donate for many colleagues the joy of comprehension with these notes. I wish you a successful learning!

László Balogh M.D.

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1. CHAPTER

ORIGIN OF THE ECG WAVES

For a number of points, we would like to rely on the knowledge you managed to gain during your studies in physiology, but for certain points we need to repeat ourselves. These include origin of the action potential (AP) and description of the ion channel function being responsible for the individual phases of AP. For the heart, two basic types of action potential (AP) morphology can be differentiated:

1.1. Slow action potential of nodal tissues

Slow action potentials are not characterized by a resting membrane potential. After reaching the maximum diastolic potential, a hyperpolarization-activated, non-specific cation current (If – 'funny') generates spontaneous diastolic depolarization, which is mainly the result of a process determined by a permanent Na^+ inflow and later by a Ca^{2+} inflow through voltage-dependent T-type calcium channels, and a continuous decrease of K^+ conductance is also participating in its development. After reaching the threshold potential, faster Na^+ and Ca^{2+} channel-dependent depolarization is observable. As K^+ conductance increases, there is a decrease in the steepness of the upslope of the action potential (AP), then K^+ channel-dependent repolarization occurs. The downslope of the AP is generated by closure of the Ca^{2+} channels and opening of the K^+ channels. Two factors are responsible for the frequency of impulse formation:

a. maximum level of the diastolic potential and;

b. steepness of the slope of spontaneous diastolic depolarization.

Such action potentials are characteristic for the sinoatrial (SA) node and the atrioventricular (AV) node; however, certain subordinate group of cells (mainly under pathological circumstances) are also capable of having spontaneous impulse formation.

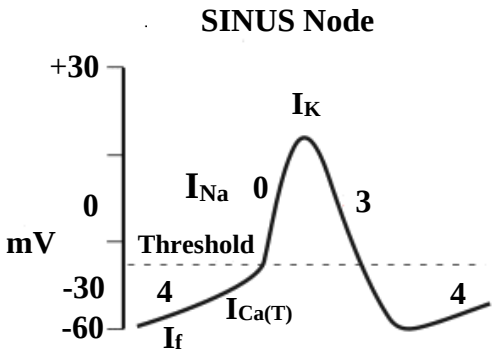


Figure 1/1.

Phases of the action potential morphology characteristic for nodal tissues and ion channels determining these. 4-spontaneous diastolic depolarization, 0-fast depolarization, 3-repolarization.

1.2. Fast action potential of the myocardium and cardiac conduction system,

the phases of which are as follows:

The resting membrane potential is followed by a fast depolarization phase, which only lasts for 1-2 msec and is caused by activation of the voltage-dependent, rapidly activating and inactivating Na^+ channels (dual-gating: activation and inactivation) as well as by Na^+ influx into the cells, thereby creating the rapid upslope of the AP curve (**phase 0**).

This is followed by the early or fast repolarization (**phase 1**), which is the consequence of a depolarization-activated I_{to} (transient outward current), i.e. K^+ ($I_{to}1$) and Cl^- ($I_{to}2$), outflow.

The next phase is the plateau (**phase 2**), during which Ca^{2+} ions flow into the cells through depolarization-activated and slowly inactivating, voltage-dependent Ca^{2+} channels, ($I_{Ca}L$), which process plays an important role in generating electromechanical coupling, that is initiation of the contraction. L-type Ca^{2+} channels, similar to Na^+ channels, are also characterized by dual gating with slow inactivation.

A small proportion of the opened sodium channels becomes inactivated only slowly or not at all, therefore the ion current through them takes part in the plateau phase. This Na^+ window current (I_{Na}) is more pronounced in the Purkinje fibers and weaker in the atrial and ventricular musculature. This provides an explanation for the fact that the majority of sodium channel blocking agents significantly shortens the duration of the action potential of the Purkinje fibers. The I_{K1} channel (see below) closes at a voltage above -20 mV, thereby contributing to the maintenance of the plateau phase

The $3Na^+/1Ca^{2+}$ exchanger is also working during the plateau phase.

The late or fast repolarization (**phase 3**) is achieved by K^+ ions flowing out of the cells through voltage-dependent (single-gated) K^+ channels (I_K), whereas closure of the Ca^{2+} channels occurs at the same time. It is this K^+ channel that determines most the AP duration (i.e. the higher the plateau, the more Ca^{2+} gets into the cells and the sooner they are activated). It has three forms: I_{Kr}, I_{Ks} (ventricular), I_{Kur} (atrial); r = rapid, s = slow, ur = ultra rapid.

Finally, the cell regains its resting potential (**phase 4**), which is maintained by the reactivation during repolarization of the inwardly rectifying K^+ channel (I_{K1}) having been closed during the depolarization phase as well as by operation of the $3Na^+/2K^+$ ion pump. I_{K1} channels are playing a role in maintaining the resting potential. This current is extremely weak in nodal tissues, and this explains the fact why the resting potential of these cells is less negative.

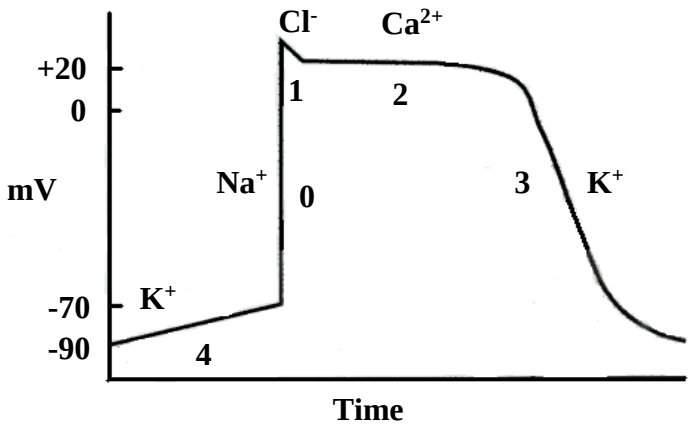


Figure 1/2.

Action potential morphology of ventricular cardiomyocytes and the most important ion currents generating it.

1.3. Explanation and significance of the morphological differences in the action potentials in various areas of the heart

Out of the two essential action potential (AP) morphologies, several other forms of AP curves can be registered in various groups of cells in the heart. This is explained by the fact that each group of cells carries different ion channels on the surface of cells or there is a difference in channel density, that is the number of ion channels, in a given area. Let's see some examples. The AP of the SA node and AV node is of slow-type, corresponding to what has been described above. Atrial muscle cells hardly have any plateau phase at all, since the upslope of their AP graph is generated by a Na^+ and Ca^{2+} conductance having lower velocities, while the downslope is created by their cessation as well as activation of the K^+ current (I_{Kur}). The movement of Ca^{2+} ions is necessary for the contractility of cells. The layers of the ventricular musculature cannot be regarded as homogenous either in regard to AP and the distribution of ion channels. The most important difference is in the Ito current generating early rapid repolarisation, which is present in the epicardial, midmyocardial and Purkinje cells, but is absent in the endocardial layer. Longitudinal heterogeneity of APs (epicardial < endocardial < midmyocardial) is also due to this.

It should be noted that epicardial APs have a shorter duration than endocardial APs, and this fact will play a role in understanding the phenomenon of T wave concordance.

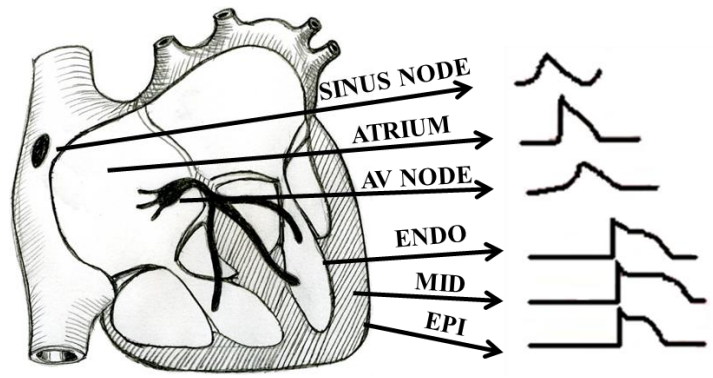


Figure 1/3.

Morphological heterogeneity of action potentials that can be registered in various points of the heart.

1.4. Some practical aspects of the operation of ion channels

Being aware of the operation of ion channels is important for several reasons. They take part in the development of cardiac arrhythmias as well as their operation is influenced by many physiological (e.g. physical exertion, autonomous reflexes) and pathological (e.g. ischemia, disturbances in ion balance, wall tension) circumstances. Let's review some more important examples of these.

During physical exercise, more Ca^{2+} will get into the cells in response to the activation of the sympathetic nervous system (β-receptor stimulation), because the function of I_{CaL} is increased by the elevation of intracellular cAMP, therefore I_K is activated earlier and repolarization occurs sooner. This provides an explanation for AP shortening in response to β stimulation. The impulse-generating rate of the sinoatrial node largely depends on the activation status of the autonomic nervous system and normal automaticity, i.e. heart rate, changes depending on this.

In case of increased sympathetic activity, in response to activation of the I_f current, there is an increased slope of spontaneous diastolic depolarization and the heart beating is accelerating (tachycardia), whereas in response to increased parasympathetic activity, the

activation of specific acetylcholin-sensitive K^+ channels (I_{KACh}) decreases the time of onset of diastolic depolarization, thereby slowing the heart beating (bradycardia).

In ischemia, activation of specific ATP-sensitive K^+ channels (I_{KATP}) is observable, which substantially alters the repolarization pattern of the ischemic zone and slows its conduction velocity. In such a case, heterogeneity of the action potential being present even normally is increasing further, and the conduction and repolarization as well as the refractoriness of cells (effective refractory period, ERP) becomes inhomogenous, which forms one of the most important electrophysiological basis for arrhythmias induced by ischemia and other pathogenetic factors.

Genetic disorders of ion channels, the so-called 'channelopathies" are subject to intensive research. Numerous channel mutations and familial ion channel diseases have been described, which have predisposition to the occurance of malignant arrhythmias as a common feature. For example, Na^+ channel mutation – Brugada syndrome, K^+ channel mutation – long QT syndromes (Romano-Ward and Jerwell-Lange Nielsen syndrome).

Antiarrhythmic drug treatment leaves nearly none of the ion channels 'unaffected'; each of them is influenced selectively or non-selectively by one of the antiarrhythmic medications being in use. This is why these drugs not only have an antiarrhythmic, but also proarrhythmic (i.e. generating arrhythmias) effect. In order to predict or, possibly prevent, the development of side effects, first we need to know which ion channels, and in which direction (stimulation or inhibition) are affected by these drugs. Therefore, to influence arrhythmias medically is not a simple task most of the time.

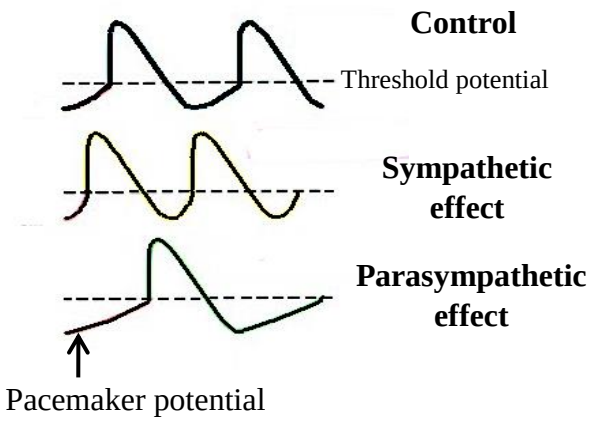


Figure 1/4.

The effect on heart rate of the autonomic nervous system is determined by the slope of spontaneous diastolic depolarization.

1.5. Origin of the ECG tracing

After discussing the operation of ion channels, let's now have a look at how we get from the channels to an ECG tracing drawn on graph paper. There are many factors that affect the shape of the tracing in ways that are still not fully understood; however, there is a generally accepted theory, which may explain the origin of the waves, i.e. the *vector concept*. Myocardial cells are connected to one another through gap junctions forming a syncitium, i.e. a conduction unit.

End-to-end connections of cardiomyocytes contain more gap junctions than side-to-side connections, therefore resulting in a faster impulse conduction in the former direction (corresponding to the long axis of the cell). Conduction properties of the myocardium are not identical (isotropic) in all directions, so conduction is different in different directions, i.e. anisotropic.

Electric potentials induced by the ion channels are adding up and generate a *dipole vector*, which points to the direction where the impulse is propagating. The cumulation of these dipole vectors creates the so-called *depolarization wavefront*, which propagates from a specific point in space (e.g. SA node) toward another point (e.g. cardiac apex).

Propagation of the depolarization wavefront is characterized by non-concentric spherical symmetry due to the anisotropic conduction.

Consequently, summation of the tiny cellular dipole vectors induces a large electrical vector, the direction of which is normally identical to that of the anatomic axis of the heart. The generated electrical signals are transmitted by the human body as a volume conductor to every point of the body and these become conductible with the help of electrodes placed onto the skin. *By performing temporal visualization of the electric potential differences between electrodes, you receive the so-called ECG lead*. An ECG lead is therefore the visualization of the projection of the depolarization vector by electrodes. In addition, the graphs drawn onto the paper are the temporal visualization of the vector displacements in the specific direction, i.e. projection of the potential difference between two points over time. Thus, the projection of potential differences takes place along a line, in a single dimension and over time. The size of the vector sum is determined by the number of dipole vectors pointing toward that direction, that is the amplitude of the ECG curve is proportional to the number of activating cardiac muscle cells. A vector can be described with its direction (direction of the depolarization) and size (number of activating cells) accurately. *Electrocardiography (ECG) is the graphic visualization over time of the electric potential differences originating during operation of the heart*. Therefore, individual ECG leads may be perceived as sensors that observe the same phenomenon, i.e. propagation of the depolarization front, from different directions in space. Our job is to learn that from which direction a given lead 'looks down' onto the three-dimensional depolarization vector of the heart and to learn to make conclusions from the signs seen in each ECG abnormality about the basic phenomenon causing them (e.g. a change in electrical axis – an alteration of the direction and sequence of depolarization).

By convention, the current (e.g. depolarization) heading *toward the electrode* will result in a *positive* (upward) deflection on the ECG, while the current *moving away from the electrode* will result in a *negative* (downward) deflection. By being aware of this rule, several ECG phenomena can be explained and this is why we find it very important to be emphasized.

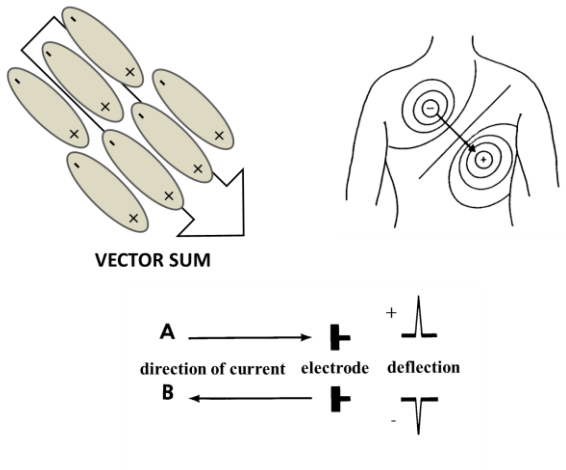


Figure 1/5.

Vector concept of the electric field propagation induced by the heart. An ECG curve is the change in the propagation direction and size of the electric field over time.

ECG leads: they are potential differences detected by the electrodes and compared to one another or a reference point.

Unipolar: the potential changes of our studied electrode (exploring electrode) are compared to a reference point (0 potential or indifferent electrode), which is created by the attachment of two electrodes at a high electrical resistance. These include the Goldberger's unipolar leads and the augmented leads, i.e. aVR, aVL and aVF, made from the former leads. For aVR, the electrodes of the left arm and left leg are attached to each other at a high resistance and the potential changes of the right arm (being a recording electrode in this case) are compared to this as a reference (zero) point (indifferent electrode) or, in other words, the electrical activity of the heart is observed from the right shoulder. Since the depolarization propagates from the sinus node toward the cardiac apex, so these currents will be moving away from the right shoulder and all waves in aVR will therefore have a negative deflection. Based on similar considerations, aVL looks onto the heart and its electrical forces from the lateral direction, while aVF does from the inferior direction. Unipolar leads also include chest leads, i.e. V1-6-(9) and VD1-3 located dorsally. The amplitudes recorded with the unipolar leads show marked differences depending upon the distance of the electrodes from the heart.

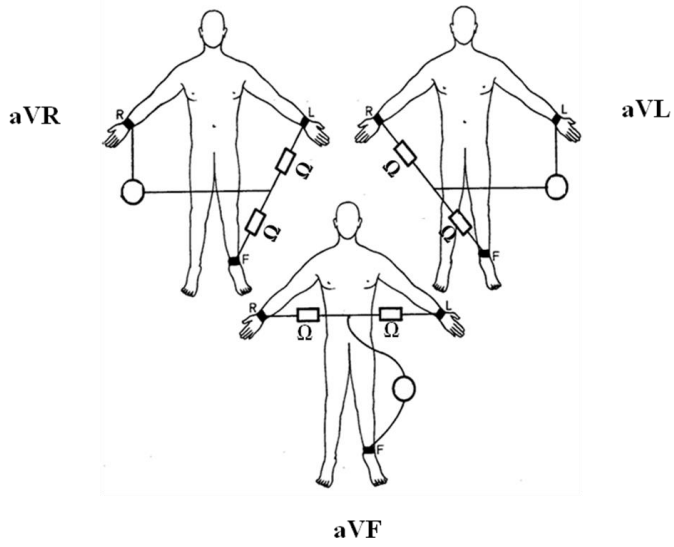


Figure 1/6.

Origin of the unipolar leads.

Bipolar: the potential difference between two electrodes is recorded, i.e. they are compared to each other. Typical examples include Einthoven's bipolar leads, where electrodes of the left arm, right arm and right leg form a virtual triangle, thereby creating the standard leads I, II and III. Imagining the heart into the center of the triangle, its depolarization vector will be projected onto the three sides of the triangle (onto the leads) and, from the three projections, taking into consideration the rules of vector editing and being in possession of the angles, one might draw conclusions on the most recent direction of the depolarization vector of the heart. The amplitudes recorded with the bipolar leads are independent on the distance of the electrodes from the heart.

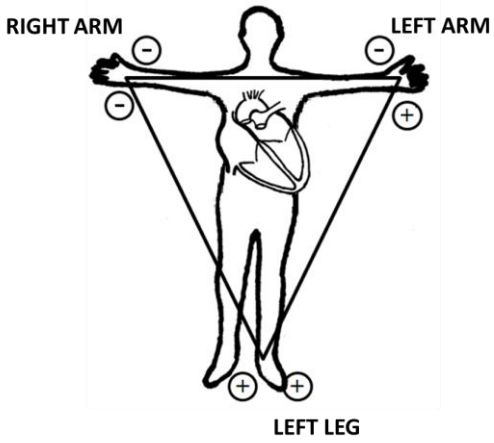


Figure 1/7.

Einthoven's triangle and the origin of bipolar leads.

Each lead projects the spatial electrical vector of the heart in specific planes. The following groups of leads correspond with the three major planes:

- **Frontal:** Einthoven limb leads (synonym: standard leads) (I, II, III) and Goldberger augmented limb leads (aVR, aVL, aVF) study the electrical vector along the frontal plane.
- **Horizontal:** Wilson's chest leads (precordial V1-6 and dorsal V7-9).
- **Sagittal:** dorsal leads (VD1-3).

Lead	Frontal	Horizontal	Sagittal
Bipolar	I, II, III	-	-
Unipolar	aVR, aVL, aVF	V1-9	VD1-3

Table 1/1.

In order to get a correct recording and leads, you must be aware of the rules of accurate electrode placement and the location of electrodes.

By convention, limb leads are color coded as follows:

Right arm - **red**, left arm – **yellow**, left leg - **green**, right leg - **black**.

Location of the chest electrodes:

V1: 4th intercostal space, right parasternal area;

V2: 4th intercostal space, left parasternal area;

V3: halfway between leads V2 and V4;

V4: left-sided 5th intercostal space in the midclavicular line;

V5: in the plane of lead V4 in the anterior axillary line;

V6: in the plane of leads V4-5 in the midaxillary line;

(**V7-8-9**: in the plane of leads V4-6 in the posterior axillary line, scapular line and paravertebrally (i.e. near the spine) on the left side, respectively).

Location of the dorsal electrodes (paravertebrally on the left side):

VD1: at the level of the 3rd thoracic vertebra (T3);

VD2: at the level of the 9th thoracic vertebra (T9);

VD3: at a distance of about 5 cm below VD2.

In addition, so-called right ventricular leads are also distinguishable, which are placed corresponding to the chest leads in a symmetrical fashion:

V1R = V2;

V2R = V1;

V3-6R: to be placed onto corresponding points of the right side of the chest symmetrically.

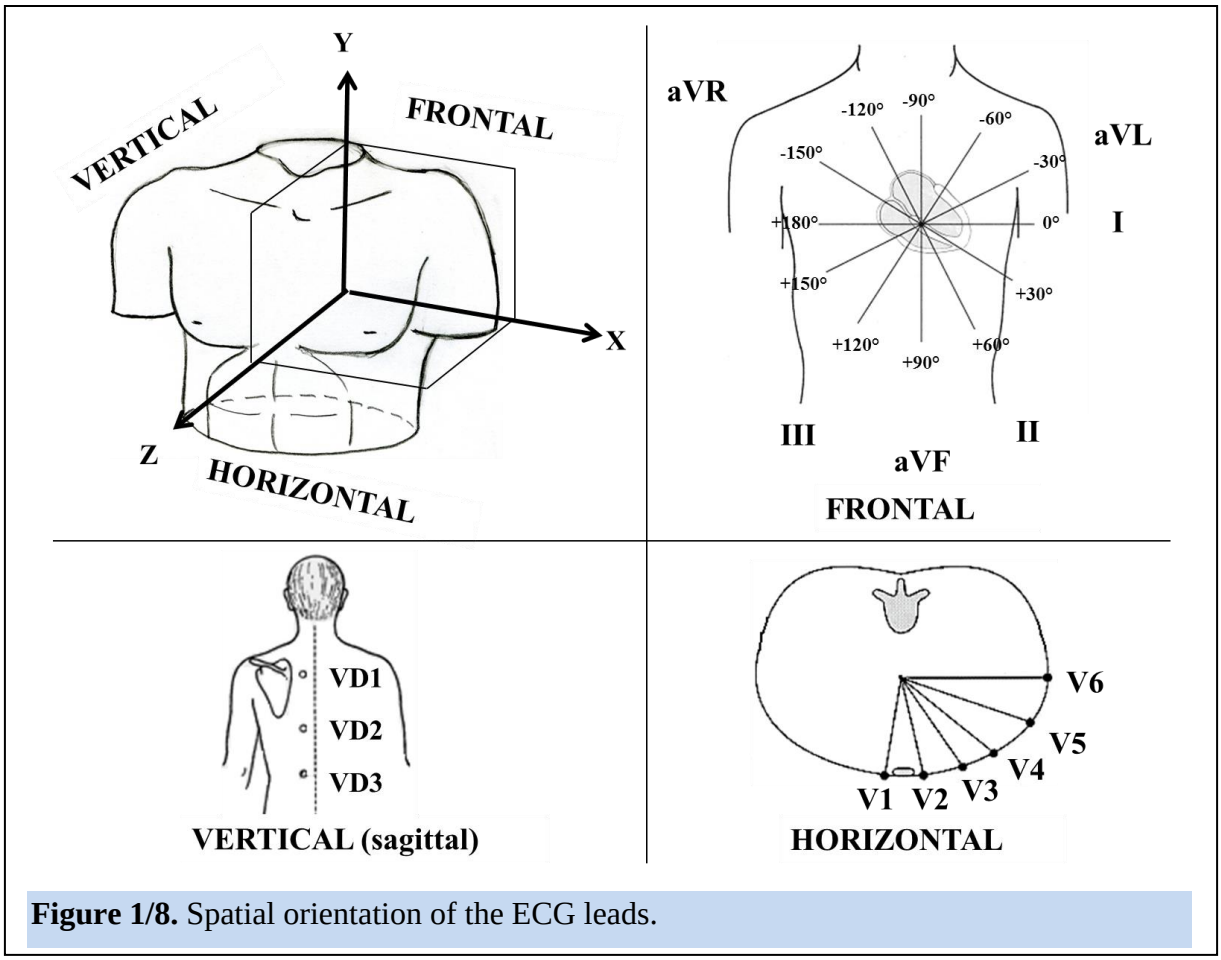


Figure 1/8. Spatial orientation of the ECG leads.

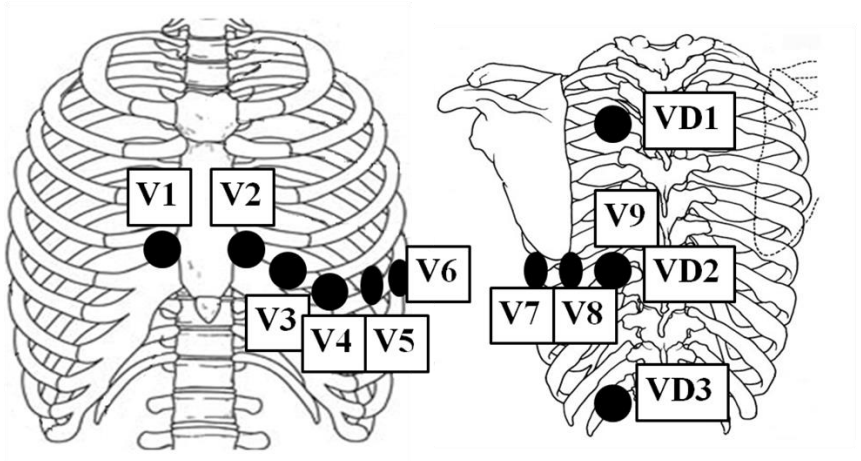


Figure 1/9.

Location of the chest and dorsal leads.

1.6. Normal sequence of the electrical activation of the myocardium:

1.6.1. Depolarization

Normally, it is the sinus node located in the upper portion of the right atrium, at the influx area of the superior vena cava that is activated. However, it has no sign on the surface ECG because it is composed of too few cells for its potential to be represented. Spontaneous diastolic depolarization is initiated in several centers simultaneously, however, since it is the sinus node where the slope of spontaneous diastolic depolarization is the steepest, therefore it is here that the threshold potential is reached within the shortest time and the subordinate centers undergo overdrive-suppression by the sinus node. The activation pattern of the right atrium is directed to forwards, leftwards and downwards, then the left atrium becomes excitated with a small delay, forming a vector directed to backwards, leftwards and downwards. The joint vector of right and left atrial activation/depolarization generates the P wave.

The interval from the beginning of the P wave to the beginning of the QRS complex is called PQ (or PR) interval; Ht is this that represents atrioventricular conduction (AV conduction time).

AV conduction time can be subdivided even further with signals recorded with intracardiac electrodes, i.e. with the so-called His bundle electrogram. By placing the electrode near the bundle of His, it becomes possible to record the potential of the bundle of His (H). Thus, the PQ interval is divided into two parts by the H potential: AH lasts from the beginning of the P wave to the H potential and HV from the H potential to the beginning of ventricular activation (QRS).

The atrial activation front gets to the bundle of His through the AV node, then the bundle of His branches off into bundle branches. The left bundle branch diverges into an anterior and posterior fascicle before the impulses would propagate to the Purkinje fibers. The right bundle branch is in direct connection with the Purkinje fibers. Virtually, the QRS complex is the sum of the depolarization vectors of the ventricular myocytes as well as the temporal visualization of their change. The impulse reaching the ventricle first activates the interventricular septum, namely from left to right. *Left-to-right activation of the interventricular septum* will play an extremely important role in the understanding of the development of left bundle branch block and that of the presence of physiological q waves. After the interventricular septum, the free walls of the ventricles begin to become activated. It

is the *septal activation* that determines the shape of the *first 1/3 of the QRS complex* or, if you prefer, the Q wave. However, it is not advisable to call the first 1/3 of the QRS complex a Q wave, because this is not always a negative deflection and a Q wave is defined as the first negative deflection not preceded by a positive deflection. Thus, one would rather use the expression *first 1/3 of the QRS complex*. The *middle third of the QRS complex* - which is not always an R wave either based on the former explanation, since an R wave is defined as the positive wave within the QRS complex – represents the activation of the apicoseptal region of the left ventricle and that of the *free wall of the ventricles*. The *last third of the QRS complex* – which is not always an S wave because an S wave is defined as the negative deflection following a positive wave – is the activation of the *posterobasal region of the left ventricle* and that of left ventricular papillary muscles.

It is not only the knowledge of the sequence of consecutive activation of the heart walls that is important, but also the activation sequence of the layers of the cardiac wall. Since Purkinje fibers are located subendocardially, therefore areas being adjacent to the endocardium are activated first and the epicardial layer becomes depolarized only later. Thus, the *wall of the ventricles* is depolarized *from the endocardium to the epicardium.* The time, during which ventricular activation from the endocardium to the epicardium is completed, is the so-called ventricular activation time (VAT = intrinsicoid deflection = R wave peak time) and represents the time from the onset of the QRS complex until the peak of the R wave. The normal value of VAT is ≤ 0.03 sec measured in leads V1-2 (thinner right ventricle) and ≤ 0.05 sec measured in V5-6 (thicker left ventricle). Obviously, thickening of the ventricular walls and abnormal impulse conduction may prolong these values. Thus, the activation sequence is the following:

SINOATRIAL NODE → RIGHT ATRIUM and LEFT ATRIUM → AV NODE → BUNDLE OF HIS → BUNDLE BRANCHES → INTERVENTRICULAR SEPTUM (from left to right) and PURKINJE FIBERS → VENTRICULAR ENDOCARDIUM → VENTRICULAR EPICARDIUM

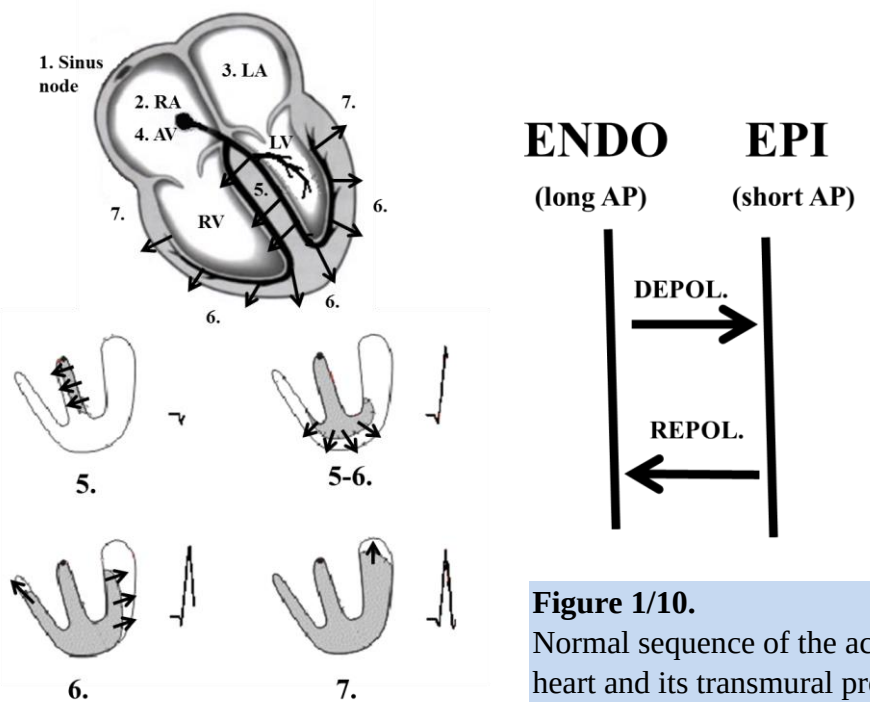


Figure 1/10.
Normal sequence of the activation of the heart and its transmural propagation.

1.6.2. Repolarization

Atrial repolarization is mostly obscured by the QRS complex on the ECG after the P wave, so it cannot be seen most of the time. Ventricular repolarization has a slow and a rapid component. Slow ventricular repolarization appears on the ECG as the ST segment, which corresponds on the action potential graph with the plateau phase or phase 2. Rapid ventricular repolarization is responsible for creating the T wave on the ECG, which corresponds with phase 3 of the action potential, that is fast repolarization. Since epicardial cells have the shortest AP, therefore the direction of repolarization, contrary to the direction of depolarization, points from the epicardium to the endocardium. The direction of intramural propagation of the depolarization and repolarization are contradictory, however, these waves generally have the same polarity on the ECG, i.e. they are concordant.

Thus, the polarity of the repolarization vector is identical to that of the QRS complex (positive T wave in case of a positive QRS complex and negative T wave in case of a negative QRS complex). If the polarity of the repolarization vector is contrary to that of the QRS complex, it is referred to as discordance of the QRS complex – ST segment – T wave. The explanation for concordance is that the direction of depolarization and repolarization currents regarding intramural motion is opposite, but these currents are generated by ion movements through the cell membrane that have just the opposite direction. During depolarization, inflow of positive charges into the cardiomyocytes is taking place and outflow of positive charges from those occurs during repolarization.

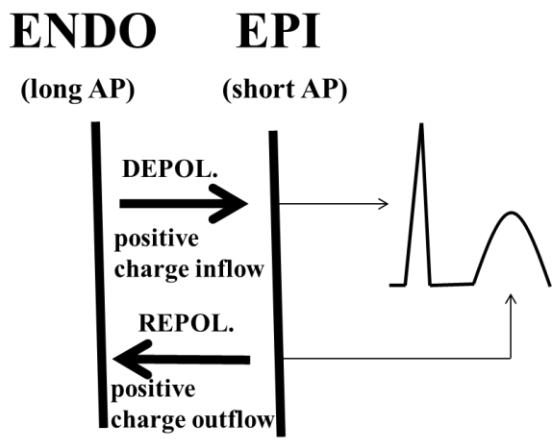


Figure 1/11.

Explanation for QRS and T wave concordance.

Repolarization also has a late component, the so-called late ventricular repolarization, which is represented on the ECG as the U wave and corresponds with the repolarization of midmyocardial (M cells) layer having the longest AP. The repolarization sequence in the ventricle is as follows:

VENTRICULAR EPICARDIUM → VENTRICULAR ENDOCARDIUM → MIDMYOCARDIAL LAYER

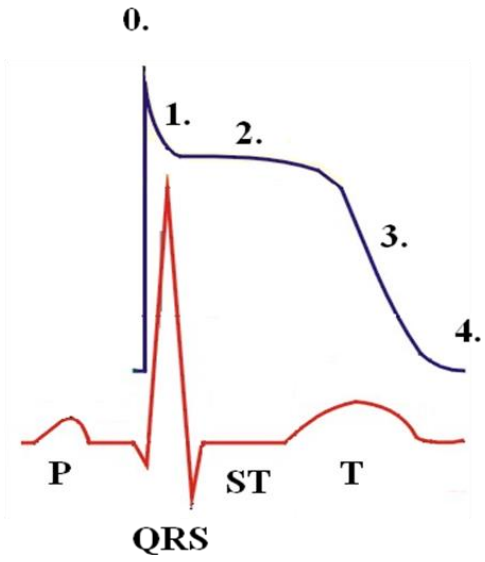


Figure 1/12.
Identical points of the ventricular action potential and ECG graph.

1.7. Conduction properties of particular areas of the heart

The conduction velocity of the myocardium and that of individual components of the cardiac conduction system is largely different, which fundamentally depends on the density of gap junctions in the longitudinal direction. The conduction velocity of the atria is 1 m/sec, however, conduction significantly slows down in the AV node, with the conduction velocity being 0.05 m/sec in its upper region and appr. 0.2 m/sec in its lower region. The slowing of AV node conduction has an important role because it does not allow supraventricular impulses to pass on above a certain rate (decremental conduction property), as if it would be 'filtering' them. In addition, atrioventricular delay plays a role in the synchronization of mechanical systoles. It is due to this that ventricular contraction begins after completion of the entire atrial systole and they do not occur simultaneously. The maximum rate of impulses conducted through the AV node is about 180-200 bpm, but it largely depends on the current status of the autonomic nervous system (increased sympathetic or parasympathetic activity) and some medications also have an influence. The impulse is conducted in the bundle of His, bundle branches and Purkinje fibers at a speed of about 4 m/sec, and the conduction velocity of the ventricular musculature is about 0.3 m/sec. The difference in the conduction velocity of almost ten times between the cardiac conduction system and ventricular cardiomyocytes provides an explanation why the QRS complex will be narrow when the impulse is transmitted through the normal cardiac conduction system, and why the QRS complex broadens if the impulse is not running down on its designated pathway, that is it uses the myocardium and not the cardiac conduction system (see the chapter on wide QRS complexes for details).

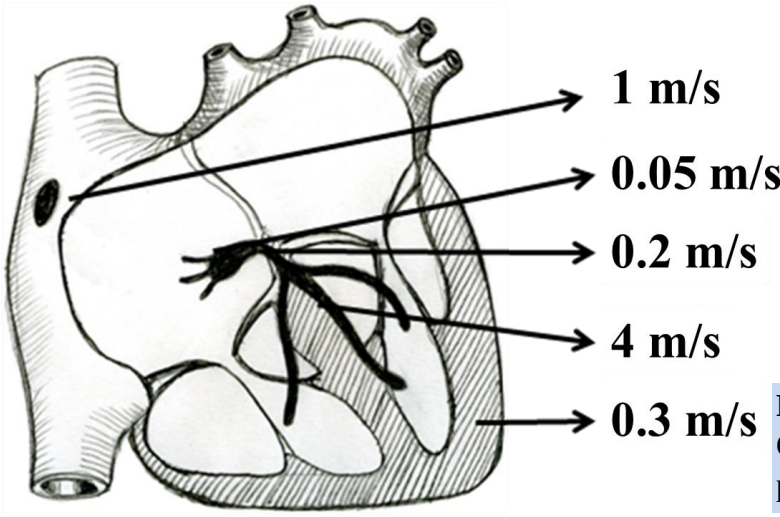


Figure 1/13. Conduction velocity of particular areas of the heart

1.8. Characteristics of individual ECG waves

In order to be able to speak about the duration and amplitude of individual waves, first we need to clarify the definition of some basic concepts such as chart speed or the dimensions of small and large squares on the ECG graph paper. During recording of the leads of the standard 12-lead ECG (I, II, III, aVR, aVL, aVF, V1-6), the chart speed of the graph paper in the ECG instrument is 25 mm/sec, which must always be checked to avoid false interpretation. The chart speed is automatically indicated on the recording. In the majority of ECG instruments, one might choose a chart speed of 10 or even 50 mm/sec, while in the electrophysiological laboratory work is performed at a speed of 100 mm/sec.

For a standard chart speed of 25 mm/sec, 1 mm corresponds with 0.04 seconds, i.e. 40 milliseconds, and one large square corresponds with 0.2 seconds, i.e. 200 milliseconds. In terms of the amplitude of deflections, 1 mm represents 0.1 mV. One small square represents 0.04 second on the „x" axis of the graph paper and 0.1 mV on the „y" axis.

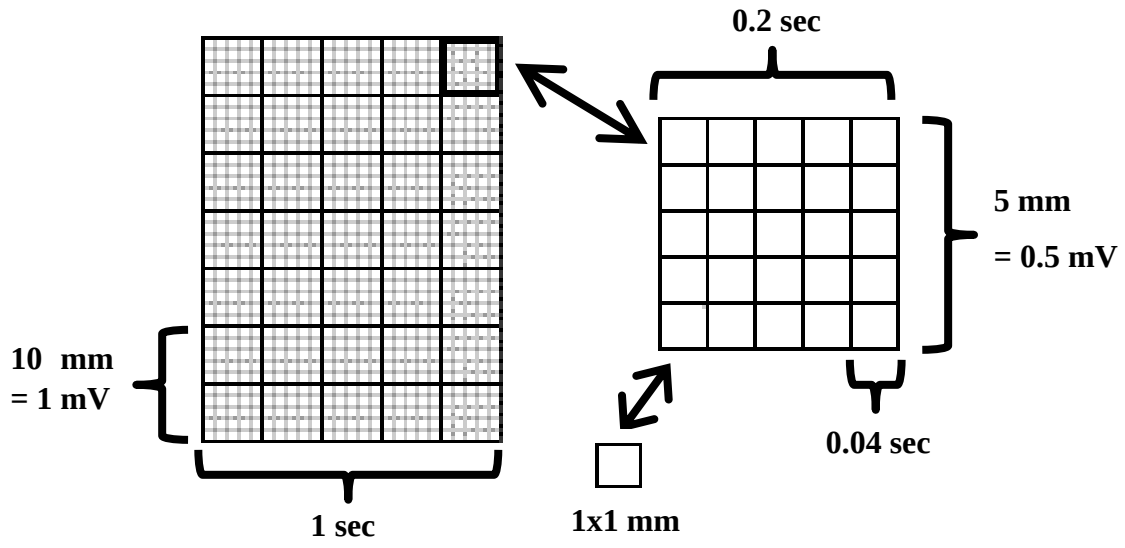


Figure 1/14. Typical voltage and time values measured on the graph paper at a paper speed of 25 mm/sec.

P wave:

Since the SA node is located in the right atrium, activation of the right atrium therefore occurs a bit earlier than that of the left atrium, however, the right and left atrial activation cannot be differentiated clearly in the majority of leads. Lead V1 is an exception, in which the P wave has a biphasic pattern, that is the initial wave will be positive (right atrium), while the terminal deflection will be negative (left atrium). This separation becomes visible due to the special location of the right and left atrium. The right atrium is situated 'over' the right ventricle and its activation forms a vector pointing forwards, leftwards and downwards, whereas the left atrium is located rather 'behind' the left ventricle and its activation is represented by a vector pointing backwards, leftwards and downwards. Given the position of lead V1, right atrial depolarization moves toward this lead and left atrial depolarization moves away from it, thereby creating an initially positive, then negative deflection, i.e. a biphasic P wave. During normal activity of the sinus node, P waves will be positive in all leads except aVR, where they will be negative and V1, where they will be biphasic for reasons mentioned above. The P wave negativity in lead aVR is explained by the fact that aVR 'looks' onto the atrial depolarization 'from the right shoulder', so the vector of atrial deplarization, which is initiated from the SA node being located in the upper pole of the right atrium, is moving away from lead aVR at any moment. For the rest of the leads, the projection of the vector of atrial depolarization demonstrates approaching to some degree, so the vector is directed toward the given lead.

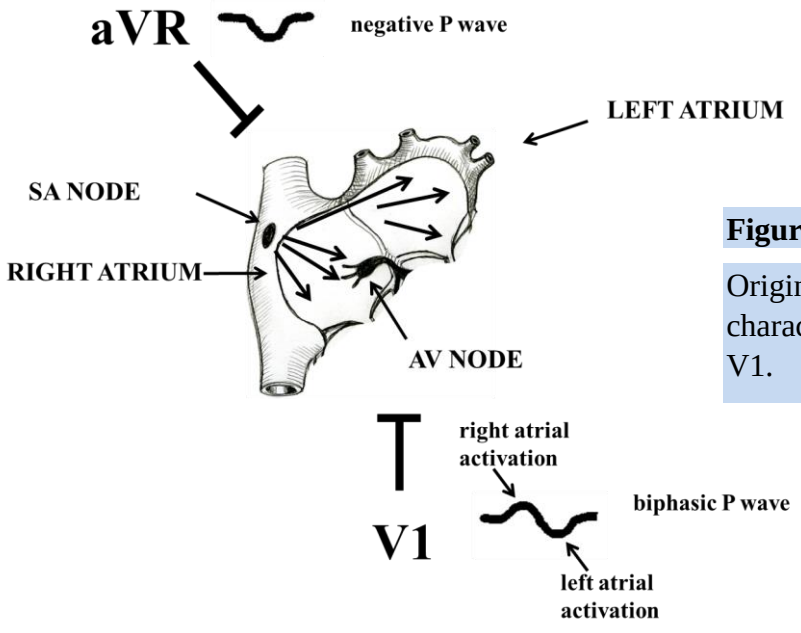


Figure 1/15

Origin of P waves and their characteristics in leads aVR and V1.

Thus, we are already able to answer one of the most important questions of ECG interpretation, namely, where the impulse depolarizing the ventricles is originating from. If the origin is in the sinus node, so the P wave morphology is identical to what has been described above, we might say that the basic rhythm is sinus rhythm. In a search for P waves, one should use lead II, but if it is not unequivocal or the recording is noisy, one should check lead V1 and then all the other leads.

The amplitude of P waves is ≤ 0.25 mV and their duration is ≤ 0.1 second. For an easier memorization, one might simply say that a P wave is not higher and not longer than the height and length of 2.5 small squares, respectively (if the chart speed is 25 mm/sec).

When analyzing P waves, you should observe their shape, polarity, amplitude and length in each lead as well as the frequency of their appearance and their relation to the QRS complexes.

The normal findings are sinus rhythm (and you might add: normal P wave morphology).

It rarely occurs that, in case of a regular rhythm, no P waves are seen or the P waves occur independent on the QRS complexes, but the 12-lead surface ECG does not provide complete information on the temporal occurrance of P waves. In such a case, recording of the so-called Lewis lead may be useful, during which the bipolar electrode of the right arm is put to the left-sided 2nd intercostal space parasternally and the bipolar electrode of the left arm is placed to the left-sided 4th intercostal space parasternally. The performed recording (lead I) clearly shows the P waves and their relation to the QRS complexes is easier to evaluate.

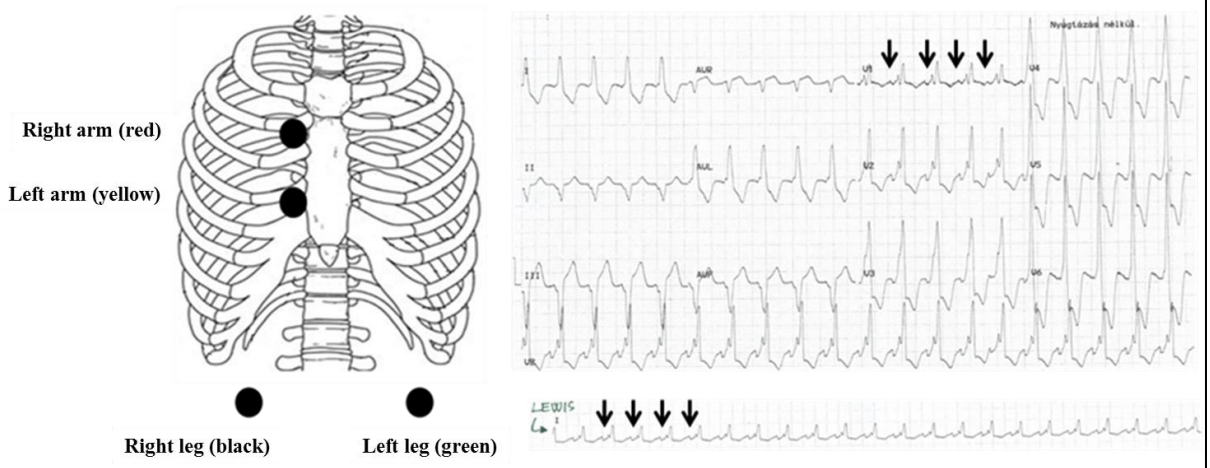


Figure 1/16. Lewis leads for better identification of the P waves (the right arm electrode placed to 2R2, the left arm electrode applied to 4R2). The ECG demonstrates atrial tachycardia with 1:1 AV conduction, for which the presence of P waves may be suspected in leads V1-2 (arrows), but the Lewis leads make their presence evident.

PQ (or PR) interval = (atrioventricular) conduction time:

The time from the beginning of P waves to the onset of the QRS complex normally ranges from 0.12-0.2 seconds, that is the length of 3-5 small squares (at a paper speed of 25 mm/sec). For its measurement, it is advisable to choose the lead where the onset of the P wave and QRS complex can be defined accurately. By convention, the conduction time can be divided into two components, one of which is the P wave itself and the other one is the interval between the end of the P wave and the QRS complex, which is usually isoelectric. The PQ interval can be divided into two segments (AH and HV) with the help of the His potential recorded by an intracardiac electrode as described above. The physiological delay of the impulse conduction in the AV junction has an important haemodynamic role because perfect contraction of the atria and their emptying toward the ventricles are time-consuming and, in addition, electrical phenomena are followed by the mechanical phenomena with a small delay (i.e. electromechanical coupling). If there were no delay, the atria and ventricles

would possibly contract simultaneously and the atria could not forward the blood toward the ventricles through a closed atrioventricular valve.

The normal findings are normal AV conduction time (or it may be provided numerically, e.g. AV conduction time: 0.16 sec).

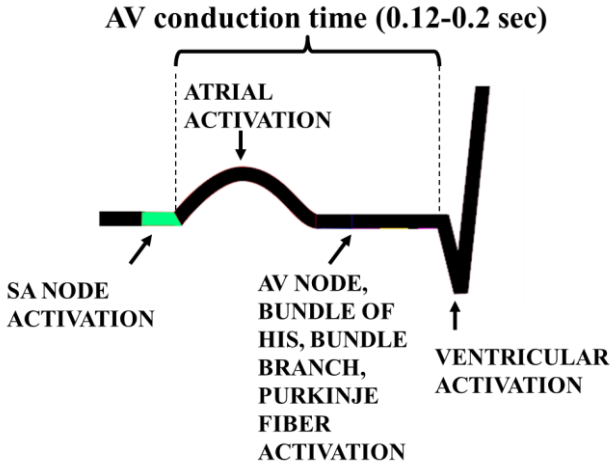


Figure 1/17.

Representation on the surface ECG of the structures getting into excitation from activation of the SA node up to ventricular activation.

QRS complexes:

It is the ECG sign representing ventricular depolarization (phase 0 of AP). Let's start our discussion with the particular QRS patterns and their nomenclature. QRS complexes are not always composed of Q, R and S waves, so we need to define what we mean under these waves: Q wave: the first negative wave not preceded by a positive wave;

R wave: the positive wave;

S wave: the negative wave occurring after an R wave.

It can be concluded from the definition that there can be only one Q wave in a QRS complex, while there can be even several from an R and S wave. In this case, the second R wave is called R' (comma) and the second S wave is called S'(comma). In addition, it also has a significance whether the name of the waves is written in normal or capital letters. The relatively small deflections compared to the entire amplitude of the QRS are written in small letters (e.g. q, r, s) and the dominant or large deflections in capital letters (e.g. Q, R, S). To decide upon if a deflection is 'small' or 'large' is relative and it is no use applying rules for that.

Figure 1/18. QRS waveform nomenclature.

It was described earlier that a QRS complex can be divided into three major parts. Its initial third represents the depolarization of the interventricular septum from left to right. Accordingly, the depolarization is moving away from the leads looking onto the heart from the lateral direction (i.e. I, aVL, V5-6), therefore resulting in small, so-called non-pathological q waves. The characteristics of q waves being visible also normally include that they are narrow (≤ 0.03 sec) and their amplitude does not exceed 25% of that of the consecutive R wave. It is extremely important to distinguish these physiological small q waves from the pathological Q waves, which will be described in the chapter „ECG signs of ischemia".

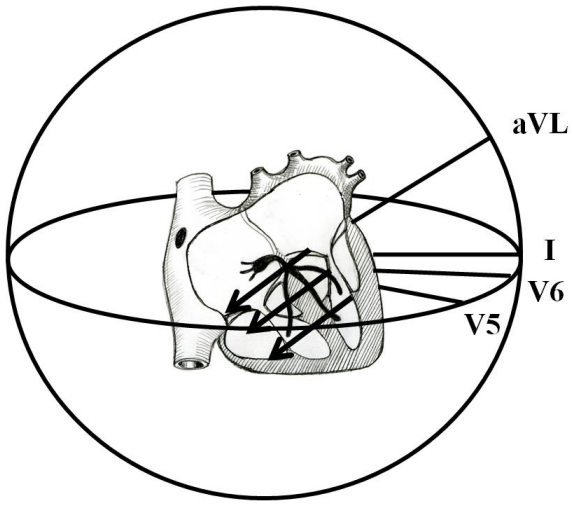


Figure 1/19.

The left-to-right activation front moving away from I, aVL and V5-6 results in small q waves in these leads.

The other aspect that must be taken into consideration during QRS analysis is the QRS width, which normally ranges from 0.06 to 0.1 sec (1.5-2.5 'small squares'). The thinness of the QRS complex is due to the fact that the impulse conducted by the bundle branches and Purkinje fibers, each having fast conduction properties, is getting to the vast majority of the myocardium very rapidly, thereby making simultaneous activation of the left and right ventricle as well as that of the walls opposite to each other possible. An important factor in ensuring optimal ventricular emptying (ejection) is the synchronous contraction of opposite walls and to ensure a contraction wave from the cardiac apex to the base of the heart. If the QRS width reaches or exceeds 0.12 sec (3 'small squares'), it is referred to as a wide QRS complex. In such a case, certain parts of the ventricle are activated in a delayed fashion and the asynchronous contraction of opposite walls impairs the efficiency of the ejection. One might see that there is an intermediate zone (between 0.1 and 0.12 sec), about which as a 'grey zone' only little is mentioned. At this time, the duration of the QRS complex is no longer in the normal range, but it cannot be regarded as wide either. The width of the QRS complex cannot be measured in each lead with full accuracy, so it is recommended to perform measurements where it can be determined as accurately as possible where the QRS complex begins and ends, i.e. in the lead demonstrating the widest QRS complex.

The third viewpoint during the analysis of a QRS complex is its amplitude, which is varying lead by lead, generally ranging between 5 and 20 mm (0.5-2 mV); values below this are called low deflections (*low voltage*) and those above this are called high deflections (*high*

voltage). The amplitude is different in each lead and reference ranges also vary depending on age.

The fourth aspect of the analysis is to determine the position of the so-called *transition zone*. Moving from V1 to V6 in the precordial leads, the amplitude of the R wave progressively increases (r→R) and the amplitude of the S wave gradually decreases in parallel (S→s). The point where the amplitude of the R and S wave is identical is called the transition zone. R wave transition normally occurs in leads V3-4. If it occurs earlier (in leads V1-2), it is referred to as early transition and, if later (in leads V5-6), then as late transition. The position of the transition zone largely depends on the position of the heart within the chest and it may dynamically change during inspiration and expiration, while following the movements of the diaphragm. Positional changes should be imagined in a way as if one would have a look from leftwards and downwards into the direction of the cardiac apex and rotation would occur around the axis that passes through the apex. Clockwise rotation can be observed in inspiration (i.e. the right ventricle gets forward) and in right ventricular hypertrophy, and early transition is seen on the ECG in this case. Counterclockwise rotation may be typical in expiration (the left ventricle gets forward) and in left ventricular hypertrophy and late transition is detectable on the ECG in such a case.

In inspiration and expiration, rotation is observable not only around the anatomical axis of the heart, but the heart is also rotating in planes perpendicular to this. In inspiration, the heart will have a vertical position and the cardiac apex moves forwards, whereas in expiration, it will have a horizontal position and the apex moves backwards.

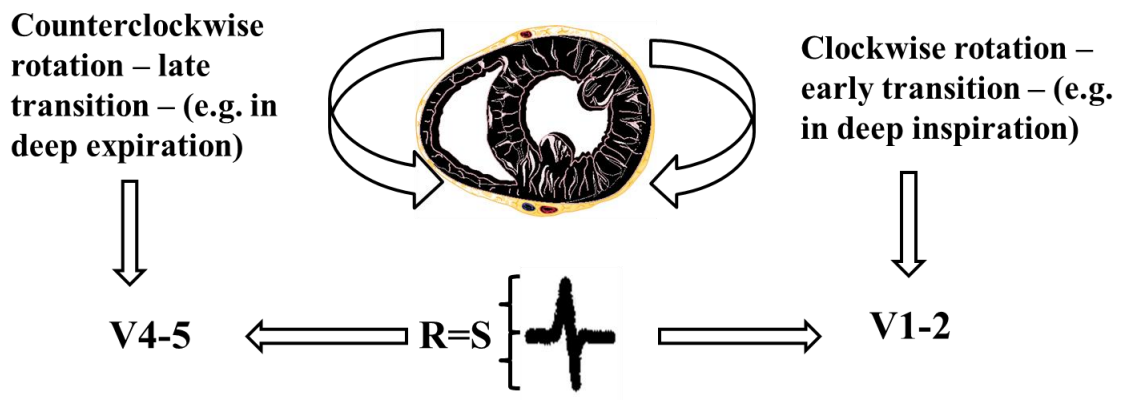


Figure 1/20. Shifting of the transition zone in the precordial leads.

Thus, one must answer four questions when analyzing the QRS complexes:

- Are there any pathological Q waves?
- How wide is the QRS?
- How much is the amplitude of the QRS?
- What is the R wave progression like?

The normal findings are the following: normal ventricular conduction, the QRS complexes are narrow, the transition zone is in lead V3 or V4 (it is not recommended to use the term 'normal QRS complexes').

ST segment:

The ST segment corresponds with the plateau phase, i.e. slow repolarization, of the action potential (phase 2 of AP). It is the segment from the end of the QRS (J point – junction) up to the beginning of the T wave. This segment is normally aligned with the isoelectric line, which by convention lasts from the end of the T wave until the P wave (TP segment) and it is this segment to which horizontal deviation of the examined ST segment is compared to. It is a frequently committed mistake that the ST segment is not compared to the TP segment, but to that lasting from the end of the P wave to the beginning of the QRS complex. This may cause problems because this latter segment may also deviate upwards or downwards in certain diseases (e.g. atrial infarction, pericarditis), so the ST segment is compared to a wrong reference segment and one might therefore reach false conclusions.

If the ST segment is situated above the isoelectric line, it is referred to as ST segment elevation, which, if exceeding *2 mm* (0.2 mV) *in leads V1-3, 1 mm* (0.1 mV) *in the limb leads and in leads V4-6 and 0.5 mm in leads V7-9, VD1-3 and V3-6R*, is regarded as marked, i.e. significant, ST segment elevation. It is referred to as ST segment depression when the ST segment is situated below the isoelectric line by at least *1 mm* (0.1 mV) in the limb leads and left-sided precordial leads and exceeds *0.5 mm* (0.05 mV) in the right-sided precordial or the dorsal leads. An ST segment depression below 1 mm may generally be described as 'trivial' (insignificant) and that of, or exceeding, 1 mm is given in millimeters.

ST segment	Elevation	Depression
Limb leads and V4-6	≥ 1 mm	≥ 1 mm
V1-3	≥ 2 mm	≥ 0.5 mm
V7-9 and VD1-3	≥ 0.5 mm	≥ 0.5 mm

Table 1/2.

If the ST segment is not completely horizontal, which occurs frequently, certain points of this are situated above, while others below, the isoelectric line. In such a case, the intersection of the ST segment and an imaginary vertical line that is located, depending on the heart rate, at 60-80 msec (1.5-2 small squares) from the end of the QRS (i.e. the J point), is compared to the isoelectric line. The various abnormal ST segment patterns will be described in the chapter on ischemia.

<u>The normal findings</u> are isoelectric ST segments.

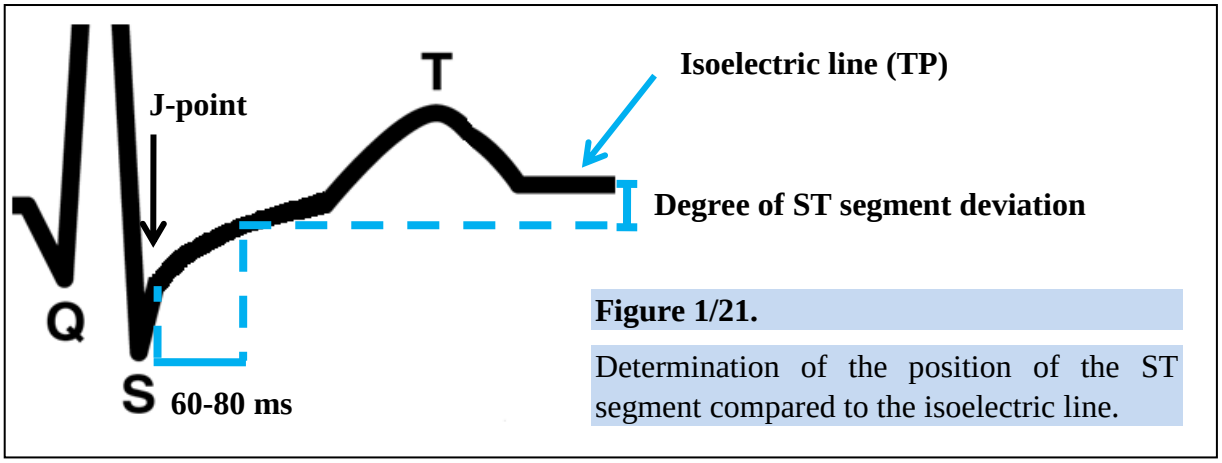


Figure 1/21.

Determination of the position of the ST segment compared to the isoelectric line.

T wave:

It represents the fast ventricular repolarization (phase 3 of AP) on the ECG. This wave typically falls into the same direction (concordant) with the largest deflection, i.e. the dominant vector, of the QRS complex. This means that if the positive deflection is the dominant one in the QRS complex, the T wave will be positive and, if the QRS complex is negative, the T wave will also be negative. The explanation for the T wave concordance with the QRS should be sought in the difference in the duration of endocardial and epicardial action potentials and in the direction of depolarization and repolarization, see above for the explanation. Of course, there are exceptions to that rule even on ECG recordings made from entirely healthy subjects, e.g. the QRS is negative in lead(s) V1-(2), while the T wave might be positive; lead III is just the opposite (positive QRS, negative T wave); and T waves might often be isoelectric even in several leads. However, T waves are usually positive in leads I, II, aVL, aVF, V2-6 and always negative in lead aVR normally. (Actually, all deflections (P, QRS, T) are negative in lead aVR because events in the heart (e.g. atrial and ventricular depolarization and repolarization, respectively) are moving away from the 'right shoulder'.)

Lead	T wave polarity
I, II, aVL, aVF, V2-6	+
aVR	-
III, V1-2	+/(-)

Table 1/3. T wave polarity in individual leads.

T waves are generally not larger than 2/3 of the preceding QRS or the line adjusted to the downslope of the T wave intersects the QRS complex. The expression 'generally', which is almost everywhere used in relation to T waves, indicates that there are no general rules for their description and not even for the disctinction between normal and abnormal patterns in each case. The reason for that is that T wave morphology is influenced by both plasma concentrations of ions, the current tone of the autonomic nervous system and, even the state of mind.

For example, it has been described that severe T wave abnormalities may develop during mourning or under greater psychic stress. It is also an interesting phenomenon that some factors predisposing to arrhythmias may cause alternating variations of the consecutive T wave amplitudes in the order of μVs, which is unnoticeable for the human eye and can only be recorded with certain special procedures (e.g. signal averaging). The microvolt T wave alternans is considered to be an important predictor of arrhythmias.

Tall and peaked T waves (>5 mm in the limb leads, >15 mm in the precordial leads) can be seen in the following cases: 1. increased sympathetic activity (autonomic dysbalance); 2. hyperkalemia and 3. hyperacute coronary artery occlusion (hyperacute T waves)

Negative or biphasic T waves are observable in the following instances:

1. In ischemia ('coronary T waves', i.e. ischemia-induced T wave inversion /TWI/, Wellens' syndrome – biphasic, i.e. positive-negative).
2. In hypokalemia (biphasic, i.e. positive-negative).
3. Extensive T wave negativity is frequently observable in left ventricular hypertrophy, hypertrophic cardiomyopathy and bundle branch block.

4. In cerebrovascular catastrophes (e.g. severe subarachnoid hemorrhage, stroke), the shape of the T waves may change significantly and giant, bizarre-looking and wide-based negative T waves develop, in addition, QT prolongation may also occur and macroscopic T wave alternans may also be observable sometimes.
5. T wave negativity in the right-sided precordial leads is normal in childhood, however, it may occur that this juvenile pattern still persists in adulthood, resulting in differential diagnostic problems without the presence of any other structural heart disease.
6. In pulmonary embolism, the negative T waves appearing in leads V1-3 are an important and sensitive marker in diagnosing the disease.
7. It is also in arrhythmogenic right ventricular dysplasia (ARVD) that negative T waves in leads V1-3 can be observed.
8. Sustained ST segment and T wave abnormalities are frequently observable after myocarditis.

The normal findings are normal ventricular repolarization (if you wish to describe the ST segment as well as the T and U waves together).

U waves:

According to old ideas, U waves might represent the repolarization of Purkinje fibers or papillary muscles or posterobasal ventricular segment. However, based on current ideas, U waves are dominantly generated by the repolarization of midmyocardial cells (M cells), because it is the layer of cells that has the longest action potential duration. The direction of U waves is identical with that of the T waves and their duration does not exceed 0.22 sec. They are regarded as abnormal when being negative (most often in the precordial leads), because in this case they indicate a stenosis of the left anterior descending branch (LAD) with an accuracy greater than 90%. This is a rare phenomenon, but it is a very sensitive and specific marker of an LAD stenosis. Prominens U waves are visible, for example, in hypokalemia and bradycardia or they may also be induced by left ventricular hypertrophy and overdosing of digitalis preparations. U waves are not always seen on the surface ECG, however, if visible, they appear in leads V2-4. Their appearance may cause differential diagnostic problems. Namely, their shape may be similar to that of P waves and can therefore be confused with 2:1 AV block (see later). U waves are generally flatter and more elongated than P waves.

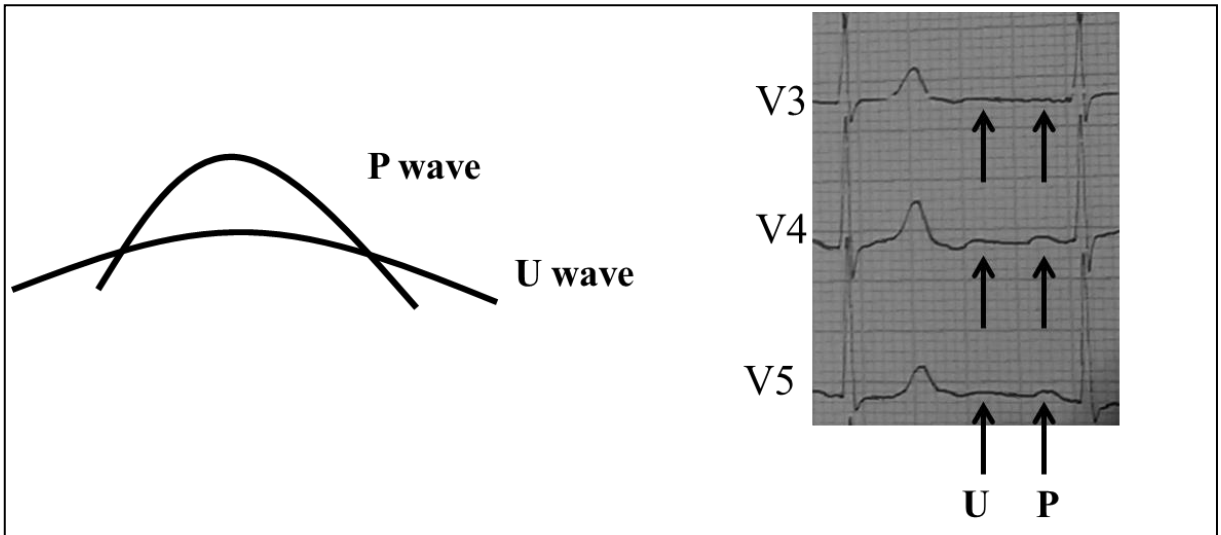


Figure 1/22. The shape of P and U waves is generally different, however, you can see on the left-sided recording that they can be similar sometimes.

In the ECG report, you generally do not mention U waves unless you observe abnormal findings.

QT interval:

The interval from the beginning of the QRS complex to the end of the repolarization (i.e. the T wave) corresponds with the overall duration of action potentials, therefore it is also called electrical systole. Its duration largely depends on the heart rate (see above for an explanation), so its normal value is adjusted for the given heart rate. This is called corrected QT interval or QTc. QTc is calculated by dividing the measured QT interval by the square root of the RR interval measured in seconds (Bazett's formula).

$$QTc = \frac{QT}{\sqrt{RR}}$$

The normal value of QTc is 0.35-0.43 sec (only values greater than 0.5 sec have clinical relevance). Generally, the QT interval is reported only if it is abnormal. Its shortening and prolongation may both predict arrhythmias. A short QT interval is seen only rarely (short QT syndrome, hypercalcemia), while a long QT interval more frequently. The latter one may be caused by congenital defects of K+ channels (long QT syndrome), ion balance disorders (e.g. hypokalemia, hypocalcemia) and several drugs (a number of antiarrhythmic drugs as well as those applied in completely other therapeutic areas). U waves are usually not included in the QT interval if they are clearly distinguishable from the T waves.

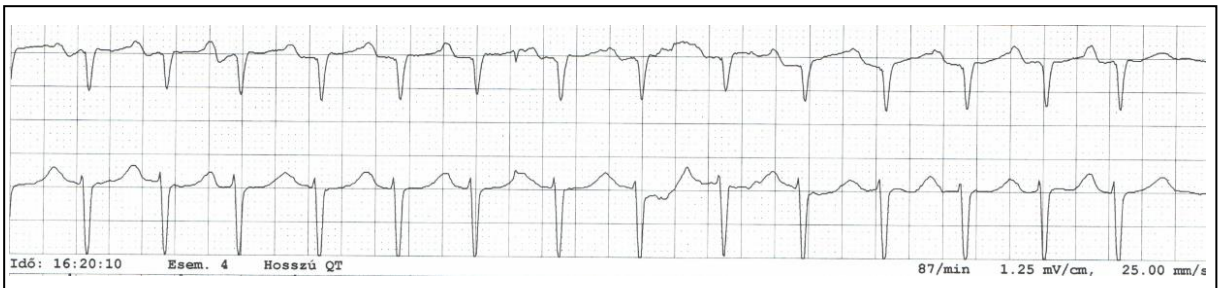


Figure 1/23. Long QT interval.

The action potentials in various areas of the heart are different in duration, thus the QT interval may be different by 10-30 msec as measured in the different leads; this phenomenon is called QT dispersion. QT dispersion mostly depends on the duration of repolarization; that is if repolarization in various areas of the heart has a largely different duration, it is referred to as inhomogenous repolarization, which can be expressed numerically with the QT dispersion. Inhomogenous repolarization (increased QT dispersion) forms a major pathogenetic basis for the development of various arrhythmias.

TP segment:

It is the isoelectric line serving as the reference segment and representing the resting potential (phase 4 of AP).

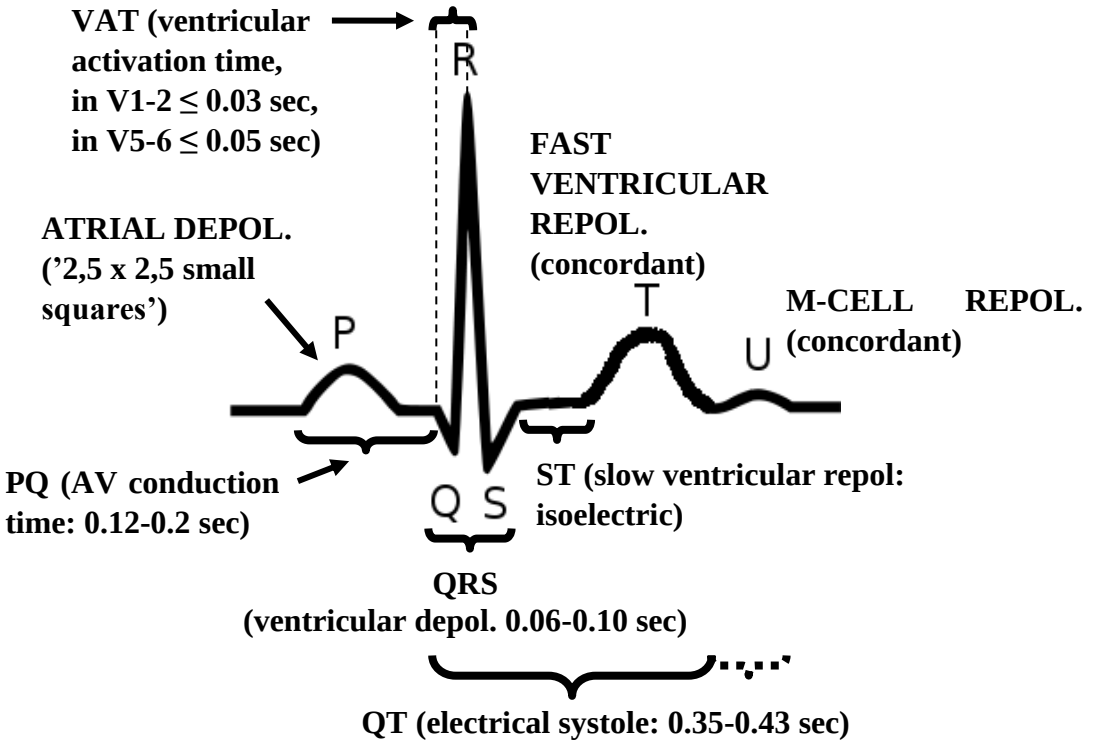


Figure 1/24. ECG waves, their reference ranges and normal description.

1.9. Aspects and steps of ECG interpretation

For practiced ECG interpreters, it is often not problematic to immediately recognize an abnormality and assess the situation within seconds; nevertheless, they also follow these interpretation aspects, although this is already not apperceived during the 'visual diagnosis' they make. Therefore, we consider it very important that you insist on using the below detailed interpretation aspects and their sequence during the interpretation process.

1.9.1. Basic rhythm

By basic rhythm, we mean the site of impulse formation that will induce depolarization of the ventricles. This may be supraventricular, ventricular or pacemaker rhythm. We speak about nomotopic impulse formation if the impulse is generated in the SA node. It is referred to as heterotopic impulse formation if the impulse depolarizing the ventricles is originating outside the SA node.

Each cardiac muscle cell and each cell of the cardiac conduction system is able to generate an impulse, it is only the rate of impulse generation that is different. A hierarchical system is built up, in which it is always the fastest impulse that predominates and will be the impulse depolarizing the ventricles. The impulse generation of the subordinate centers undergoes overdrive-suppression by the superior center. The subordinate centers may only become those generating the impulses if they do not receive an impulse from the superior center providing overdrive-suppression or if their own impulse-generating rate rises above

that of the superior center. The intrinsic impulse-generating rate of various areas of the heart is different:

- sinus node: 60-100 bpm;
- AV junction: 40-60 bpm;
- ventricles: 25-40 bpm.

Impulse generation	Intrinsic rate	Acceleration, but < 100 bpm	Acceleration, > 100 bpm
SA node	60-100 bpm	-	Sinus tachycardia
AV-junction	40-60 bpm	Accelerated junctional rhythm	Junctional tachycardia
Ventricles	25-40 bpm	Accelerated idioventricular rhythm	Ventricular tachycardia

Table 1/4. Hierarchical structure of impulse generation in the heart and consequences of an altered impulse-generating rate of the subordinate centers.

Subordinate impulse-generating centers will have an important role when the superior center, for any reason, does not generate an impulse or generates it too slowly. At this time, the subordinate centers ensure the continuous operation of the heart, however, at a lower heart rate. Similar to Uninterruptible Power Supply (UPS) devices, this as a safety net can prevent, for a certain period of time, from a critical decrease in heart rate or a possible cardiac arrest. This hierarchical structure is protecting you from asystolia. During the failure of one of the systems, the subordinate one takes over the control.

Normally, the impulse depolarizing the ventricles originates from the sinus node, the basic rhythm is therefore sinus rhythm. Sinus rhythm can be recognized from the following signs:

- the P wave polarity is normal, i.e. positive in the limb leads and in leads V2-6, negative in lead aVR and biphasic in lead V1 (It frequently occurs that one cannot see P waves in some leads because they are isoelectric there.),
- the shape of P waves does not change significantly within a lead,
- the distance of P waves from one another does not differ significantly (> 0.12 sec) (although it may be greater than this in sinus arrhythmia).

The other types of basic rhythm (supraventricular and ventricular rhythms) will be described later. *The normal findings are sinus rhythm.*

1.9.2. Heart rate

By the heart rate, we generally mean the ventricular rate, which is normally the same as the atrial rate (e.g. in sinus rhythm), however, it should be stressed that the ventricular and atrial rate may also be different from each other. It may occur that atrial impulse generation is so fast that the AV node cannot keep up with it and some impulses are therefore blocked, e.g. in atrial flutter, (atrial rate: 300 bpm, ventricular rate: 150 bpm) or in atrial fibrillation (atrial rate: 500 bpm, ventricular rate: 113 bpm.) In some cases, ventricular impulse generation is faster than the atrial one, so supraventricular impulses cannot prevail in the ventricles in such cases, atrioventricular dissociation develops and electrical operation of the atria and ventricles is separated from each other, e.g. in ventricular tachycardia (atrial rate: 82 bpm, ventricular rate: 140 bpm.) If electrical separation of the atria and ventricles from each other is not due to the fact that the impulse depolarizing the ventricles is faster than the atrial one, the atrial rate may be faster than the ventricular one e.g. in AV block (atrial rate: 82 bpm, ventricular rate: 35 bpm.)

After all these, we wean ventricular rate by the 'heart rate'. The heart rate can be determined in several ways.

1. One way is that 60 is divided by the RR interval measured in seconds, which gives an accurate value (e.g. there are 2 large squares + 2.5 small squares between two consecutive R waves = 2x0.2 sec + 0.1 sec = 0.3 sec → 60/0.3 = 120 bpm).
2. The other option is to count the number of large squares between two R waves and determine the approximate heart rate (in this case, 300 is divided by the number of large squares between two QRS complexes):

- RR= 6 large squares: 50 bpm
  5 large squares: 60 bpm
  4 large squares: 75 bpm
  3 large squares: 100 bpm
  2 large squares: 150 bpm
  1 large square: 300 bpm

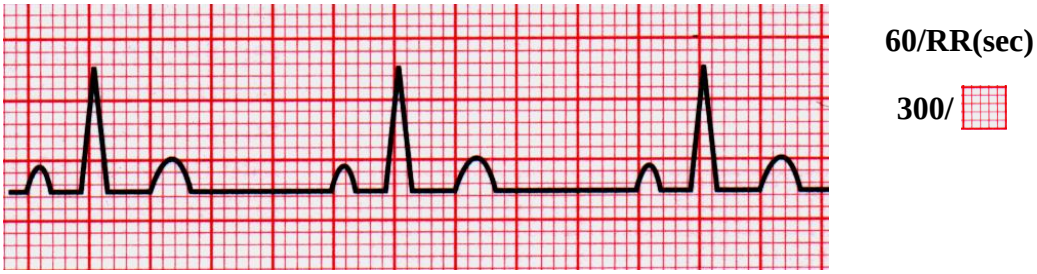


Figure 1/25. Determination of the heart rate (in this case: 60 bpm.)

3. If the heart beating is arrhythmic, the RR intervals vary and the above rules cannot be used. In such a case, it is recommended that the number of R waves is counted in 30 large squares, which is 6 seconds, and by multiplying this number by 10. you obtain the heart rate. For example, in atrial fibrillation, if 8 QRS complexes are observed in 30 large squares (6 sec), the average heart rate is approximately 80 bpm.

At rest, the heart rate of an adult person ranges from 60-100 bpm, which is called normal heart rate. Bradycardia is defined as a heart rate below 60 bpm (clinically significant if it is below 50 bpm), however, during sleep at night, values near 40 bpm may not be regarded as abnormal either. Tachycardia is defined as a heart rate above 100 bpm.

1.9.3. Electrical heart axis

When we refer to the electrical heart axis, we always mean the projection of the axis of the QRS complex on the frontal plane by this. The QRS axis can also be defined both in the horizontal and sagittal planes. The P wave and the T wave also has a spatial vector, i.e. axis, however, we are not dealing with them now. If we are talking about the 'electrical heart axis' hereinafter, we exclusively mean the projection of the QRS complex on the frontal plane by this. Electrical heart axis means the projection onto the frontal plane of the spatial vector of depolarization. This vector is projected onto the frontal plane by the standard limb leads I, II and III as well as leads aVL, aVR and aVF. If these leads are depicted around a circle, with the heart being in its center, each lead views the heart from an accurately defined angle. The positive or negative deflections recorded in each lead are proportional to the deflection of the vector to such direction.

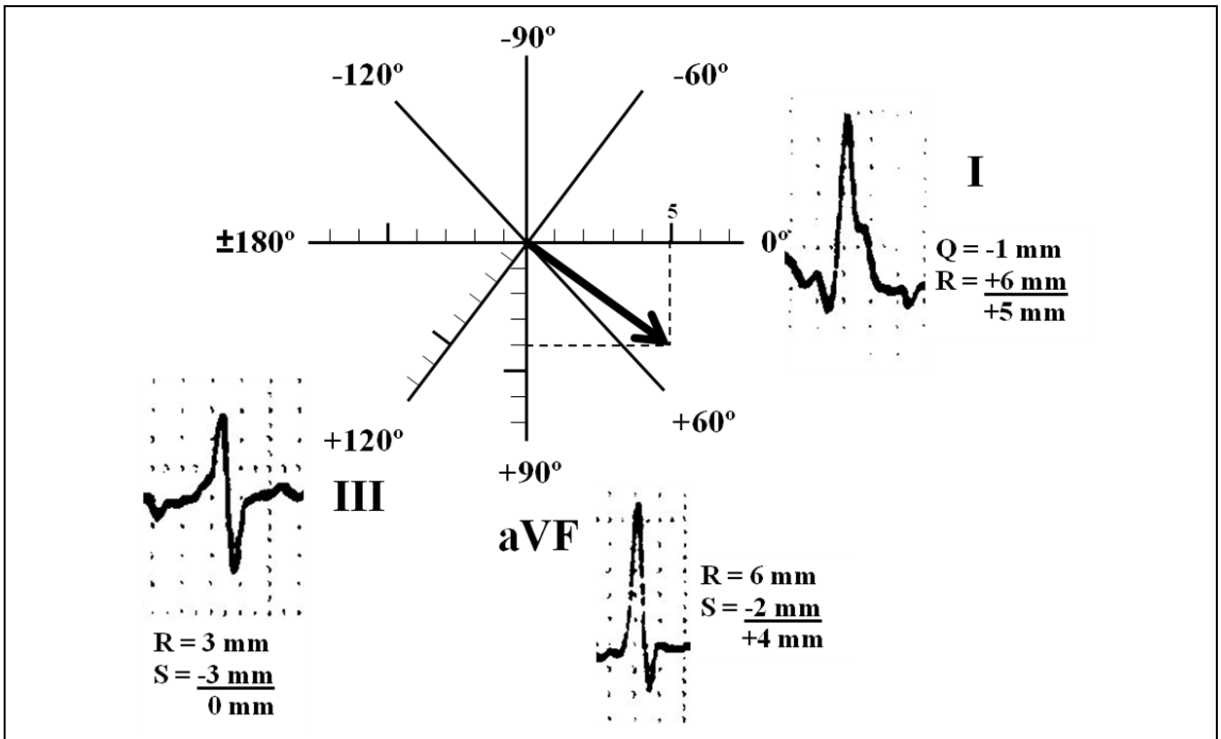


Figure 1/26.

Determination of the electrical heart axis taking the rules of vector drawing into.

Thus, if the depolarization wavefront is just moving toward a certain lead, it is this time that it results in the greatest positive deflection in the given lead. If it moves in the opposite direction, it will create the greatest negative deflection. If its movement is perpendicular to a given lead, the size of the positive and negative deflection will approximately be the same.

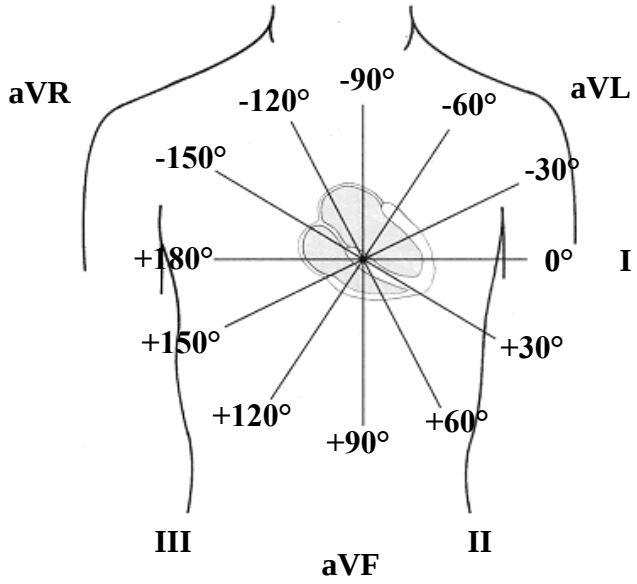


Figure 1/27.

Determination of the QRS axis by the hexaaxial reference system.

In regard to the six limb leads, a hexaaxial reference system is generated, in which the depolarization vector can be calculated accurately based on the direction and size of the deflections in each lead and following the mathematical rules of vector drawing, with the depolarization vector defined as the sum of vectors generated in the individual leads. In the hexaaxial reference system, one can accurately determine that the electrical axis of the heart, i.e. its depolarization vector, is pointing to which direction, that is toward how many degrees, in a full circle divided in 360°. However, in the majority of cases, there is no need to determine the angle of the axis with full accuracy, we are only curious about the fact that to which of the four quadrants of the circle the vector is directed. Based on all these, one can define normal QRS axis (0°- +90°), left axis deviation (0°- -90°), right axis deviation (90°- +180°) or so-called indeterminate or north-west axis or ’no man’s land’ (extreme left or right axis); (-90°- +180°).

The easiest way to determine the QRS axis is that we take a look at on the six above leads and decide on which has the greatest positive or negative deflection. The greatest positive deflection of the QRS complex points to the direction of the R wave axis. The greatest negative deflection of the QRS complex points to the opposite direction of the R wave axis. Given that which lead looks onto the depolarization vector of the heart from which angle, one can define the QRS axis by comparing the amplitude of the deflections. This rule can be simplified even further:

- If the greatest positive deflection of the QRS complex (R wave) is in lead II, it is referred to as normal R wave (QRS) axis. In this case, the dominant deflection in leads I and aVF is positive.

- If the greatest positive deflection of the QRS complex (R wave) is in lead I and aVL and the deflection is negative in lead aVF, it is referred to as leftward deviation of the R wave (QRS) axis. If the deflection is positive in lead aVF, there is still normal QRS axis.
- If the greatest positive deflection of the QRS complex (R wave) is in lead III and the deflection is positive in lead aVF and negative in lead I, it is referred to as rightward deviation of the R wave (QRS) axis.
- If there are significant R waves in lead aVR, it is referred to as indeterminate (extreme left or right) QRS axis.

The normal findings are normal QRS axis.

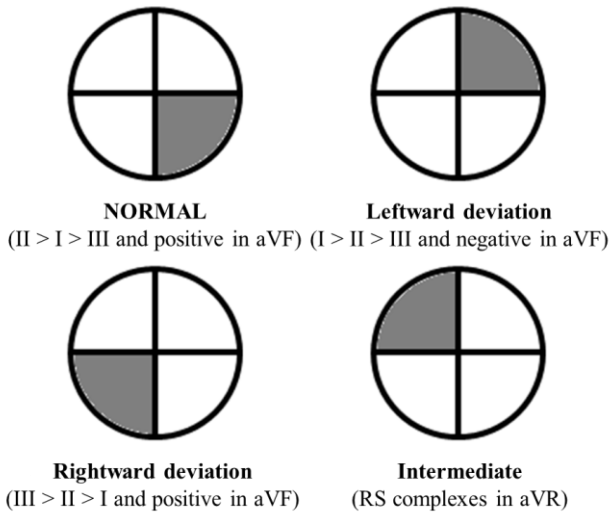


Figure 1/28.

Types of QRS axis.

1.9.4. P wave, PQ interval, QRS, ST, T wave, QT interval

See above for their description.

1.9.5. Wave morphology

P waves may be, notched, bifid, biphasic, broad or negative. QRS complexes may be notched, broad, narrow, negative, with small fragmentations (additional spikes) on them. T waves may be peaked, symmetrical, asymmetrical, tall, negative (T wave inversion).

1.9.6. Opinion (summary)

You are usually able to provide an opinion, however, it may happen that you cannot obtain a fully accurate diagnosis merely from analyzing the 12-lead surface ECG.

Based on all of the above, a normal ECG report sounds as follows: sinus rhythm, 75 bpm, normal QRS axis, normal AV conduction time (PQ:0.16 sec), normal ventricular conduction (QRS:0.08 sec), R wave transition zone in lead V3, normal ventricular repolarization (isoelectric ST segments, concordant T waves). The information in brackets may also be used for reporting, but this is not obligatory. However, being aware of the normal ranges is indispensable.

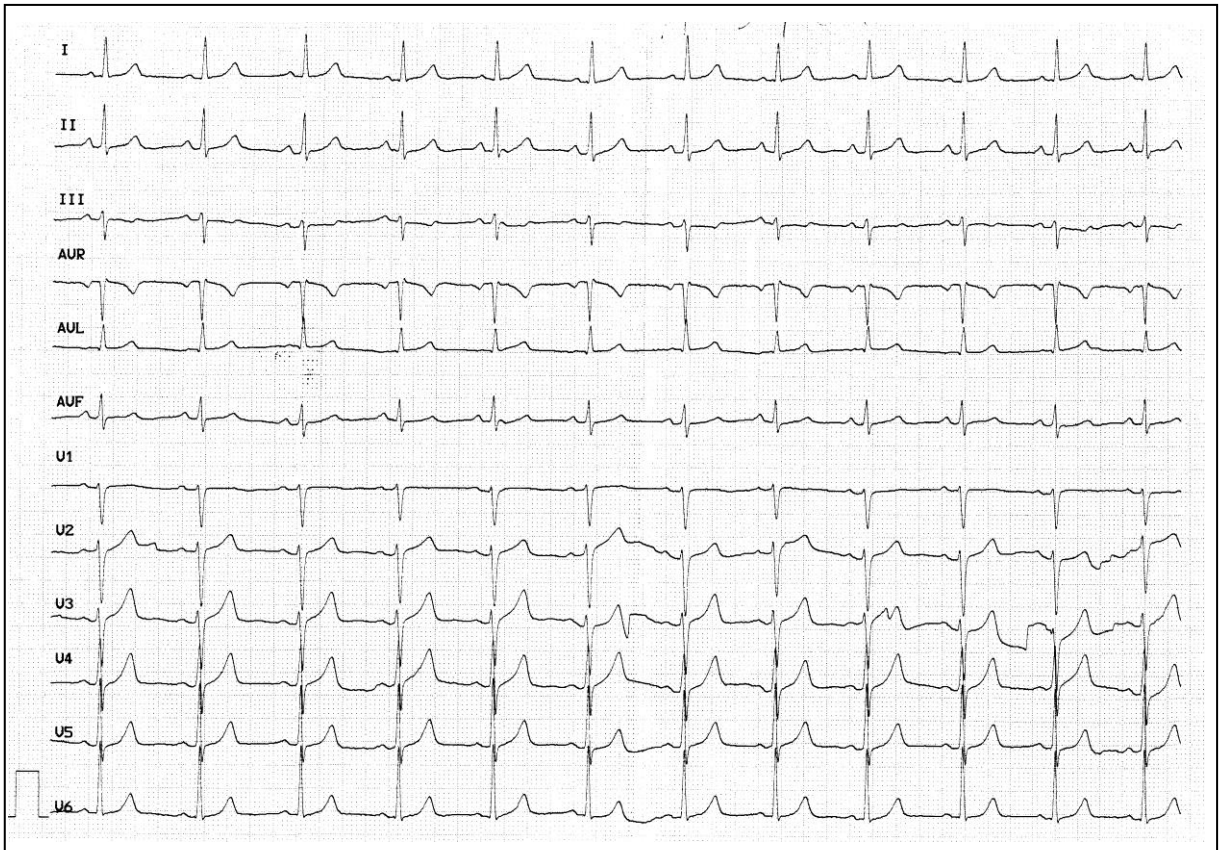


Figure 1/29.

Normal ECG. Sinus rhythm, 80 bpm, normal QRS axis, normal AV conduction time, R wave transition zone in lead V4, narrow QRS complexes, normal ventricular conduction and repolarization.

1.10. Orientation of ECG leads

Until now, it has been described several times that each lead looks onto the heart from a different direction and the activation front is projected by them from this specific direction. Accordingly, there are so-called contiguous leads viewing the heart from similar directions and providing the most information on the heart wall that is closest to them.

V1-4	**- anteroseptal**
I, aVL, V5-6	**- lateral**
II, III, aVF	**- inferior**
VD1-3, V7-9	**- posterior**
V3-6R	**- right ventricular**

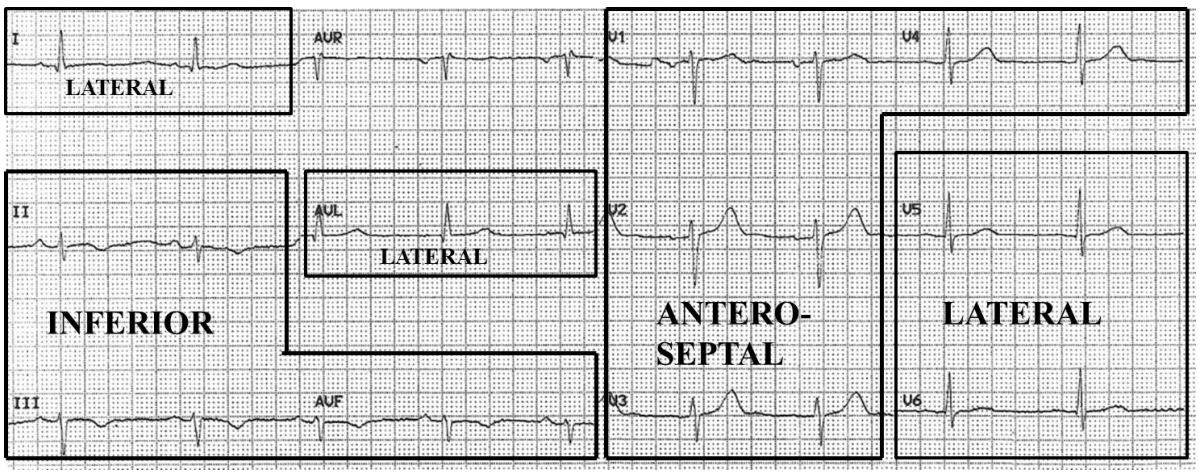


Figure 1/30.

Regions of the left ventricular myocardial wall represented by individual ECG leads.

Naturally, this does not mean that the above leads provide information exclusively on the above regions; it is merely in these leads that the depolarization of these heart walls will obviously result in the greatest deflection, so a pathological abnormality of this region of the heart will be the most conspicuous in these contiguous leads. For example, lead V1 may provide information on the septal region or from the status of the right ventricle and, in addition, it also records so-called reciprocal changes from the posterior region being opposite to this lead. Therefore, one often cannot make conclusions about the basic phenomenon from an abnormality observed in a single lead, only if it appears in several of the contiguous leads. The knowledge of contiguous leads and their orientation is indispensable for ECG interpretation, because it is based on these, for example, that the localization of a myocardial infarction is provided.

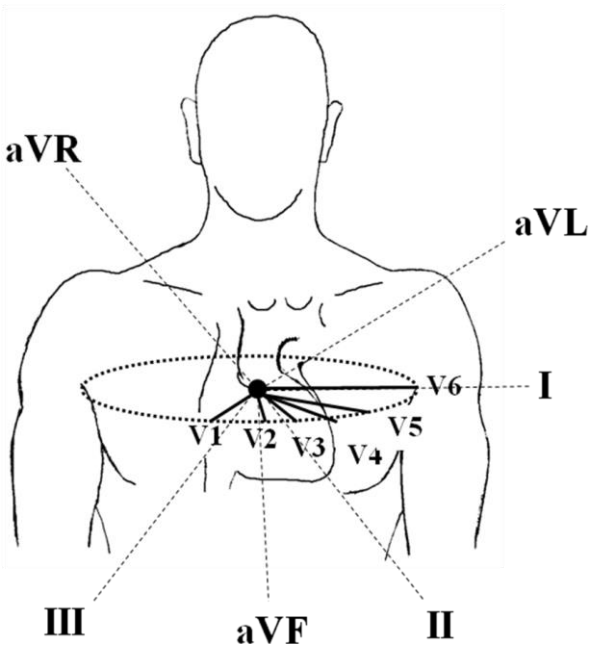


Figure 1/31. Spatial orientation of ECG leads in the frontal and horizontal plane.

FACTS THAT YOU MUST KNOW:

1. During the operation of the ion channels of cells, electric dipole vectors are generated, the sum of which induces a continuously moving electric field with variable direction. An ECG graph is the graphic visualization of the electrical potential differences over time.
2. If the electric vector is directed toward a given lead, it will result in a positive deflection; when it travels away from that lead, this will result in a negative deflection.
3. P waves are formed as a result of atrial depolarization. If the polarity of P waves is positive in all leads, with the exception of lead V1 (biphasic) and aVR (negative), and each P wave is followed by a QRS complex, it is then sinus rhythm.
4. AV conduction time is the time from the onset of the P wave to the onset of the QRS complex (normal range: 0.12-0.2 sec), i.e. the time period during which the impulse originating in the SA node reaches the ventricles (activation of the SA node, atria, AV node, bundle of His, bundle branches and Purkinje fibers are all part of this process).
5. The QRS complex represents ventricular depolarization. The interventricular septum is activated first from left to right, which is followed by the activation of the free walls of the ventricles and, finally, their posterobasal region. The QRS complex is narrow (normal range: < 0.12 sec) since the impulse activates the entire ventricular musculature within a very short time in case the cardiac conduction system is normal.
6. Intramural activation of the ventricles takes place from the endocardium to the epicardium (ventricular activation time), whereas repolarization has the opposite sequence (from the epicardium to the endocardium). This provides an explanation for the fact that the polarity of QRS complexes and T waves is generally the same (concordant).
7. The Q wave is the first negative wave not preceded by a positive deflection. The depth of pathological Q waves reaches or exceeds 25% of the height of the consecutive QRS complex or their width reaches or exceeds 0.03 second, or they occur in leads in which there are no Q waves at all under normal circumstances (V3-4).
8. Each positive wave caused by ventricular depolarization is called an R wave.
9. Any negative wave after an R wave is called an S wave.
10. The R wave transition zone normally occurs in leads V3-4 because it is in these leads that the amplitude of R waves and S waves becomes equal.
11. Structures of the heart with slow conduction include the AV node and ventricular musculature, while the bundle branches and Purkinje fibers have fast conduction properties.
12. Each cell of the myocardium and cardiac conduction system is able to form impulses, however, their rate is different and it is always the impulse with the fastest rate that will prevail and provides overdrive suppression for the rest of the impulses.
13. It is referred to as nomotopic impulse formation if the impulse depolarizing the ventricles originates from the SA node. For heterotopic impulse formation, the impulse depolarizing the ventricles originates out of the SA node.

14. At a chart speed of 25 mm/s, one small square (1 mm) equals 0.04 second (40 msec).
15. Ventricular repolarization is represented on the ECG by the ST segment (slow repolarization or plateau phase) and the T wave (fast repolarization).
16. The position of the ST segment is related to that of the TP segment, and its height is provided at 60-80 msec from the end of the QRS complex (J point). The ST segment may be isoelectric, elevated or depressed.
17. The QT interval (from the beginning of the QRS complex to the end of the T wave) is identical to the action potential duration. Its normal value depends on the heart rate, so it is adjusted for heart rate.
18. In case of rhythmic cardiac activity, the heart rate can be given if 60 is divided by the RR interval measured in seconds. If there is arrhythmic heart beating, one should count the number of QRS complexes in 30 large squares (6 seconds) and multiply it by ten (the latter method can also be used in rhythmic heart beating). The normal heart rate is 50-100 bpm; below this range, it is referred to as bradycardia, and tachycardia for heart rates above this.
19. The QRS axis is provided in the frontal plane. If the greatest positive deflection is visible in lead II, it is nornal QRS axis. If the greatest positive deflection is visible in lead I, one should check the QRS polarity in lead aVF: if the latter one is positive, it is normal QRS axis, if negative, it is left axis deviation. Rarely, it is lead III where the greatest deflection is observable, it is then right axis deviation.
20. Leads II, III, aVF represent the inferior wall of the heart, while leads I, aVL, V5-6 represent the lateral wall and V1-4 the anterior wall.
21. During ECG evaluation, one should always follow the below sequence: basic rhythm, AV conduction time, QRS axis, heart rate, QRS complexes (duration, the presence of pathological Q waves), ST segments, T waves.

CHAPTER 2

DISORDERS OF IMPULSE FORMATION

Normally, impulse formation is nomotopic, that is the impulse is generated in the SA node. While investigating the disorders of impulse formation, one must observe the morphology of P waves, PP intervals, relationship of P waves with the QRS complexes, QRS morphology and axis as well as changes in the QRS axis. For their investigation, a longer ECG recording called a rhythm strip is necessary and one needs to examine leads II, III, aVR and V1-2, because it is in these leads that P waves can most often be analyzed best. Atrial activation normally propagates as a vector directed downwards from the SA node, which is located at the upper pole of the right atrium, and being positive in leads II, III and aVF, while during retrograde atrial activation as a vector propagating from the AV junction toward the upper pole and having just the opposite direction. Retrograde propagation results in narrow, negative P waves in leads II, III and aVF and narrow, positive P waves in lead V1, since the two atria demonstrate simultaneous activation in this case. Retrograde P waves are indicated as P'.

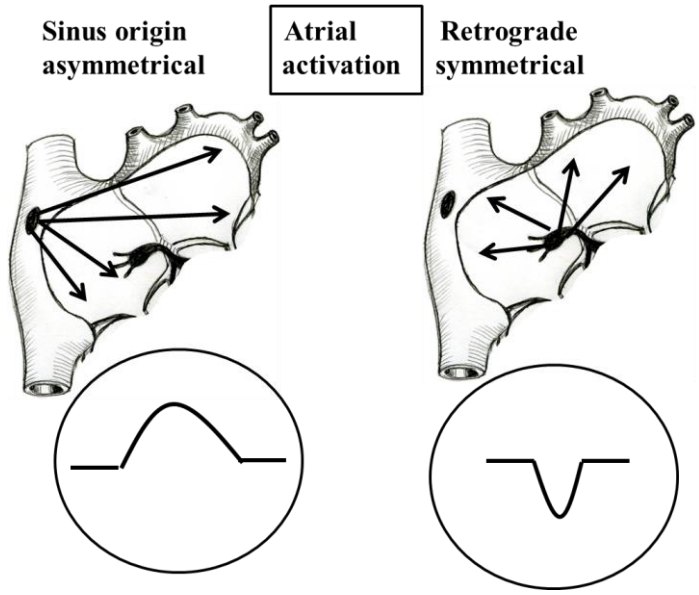


Figure 2/1.

P waves originating from the sinus node are positive in the inferior leads, with a greater amplitude and longer duration than negative P' waves being conducted in a retrograde fashion. This difference can be explained by an asymmetrical and consecutive or, on the other hand, a simultaneous and symmetrical activation of the right and left atrium, respectively.

The disorders of impulse formation can be divided into two groups:

Disorders of nomotopic impulse formation: The site of the disorder of impulse generation is in the sinus node. Disorders of impulse formation may have two forms here. One of them is the disturbance of, or a change in, the automaticity or pacemaker function of the *SA node*. The other one is the disorder of *sinoatrial impulse conduction*, and this latter one may even be classified as a conduction disorder actually.

Disorders of heterotopic impulse formation: The impulse is not generated in the sinus node. It has two forms: for *active heterotopic impulse* formation, the pacemaker rate of a subordinate impulse-generating center exceeds that of the sinus node and it takes over the pacemaker role (analogous to the proverb 'Power in numbers'). For *passive heterotopic impulse formation*, no

impulse is generated in the SA node and a subordinate center therefore takes over the pacemaker role at its own intrinsic rate (analogous to the proverb 'When the cat's away the mice will play').

2.1. Disorders of nomotopic impulse formation

The impulse resulting in contraction of the heart originates from the SA node.

2.1.1. Sinus tachycardia

It does not necessarily represent a disorder of impulse generation because it may even be a normal response to physical exercise, this is why the terms appropriate and inappropriate sinus tachycardia are distinguished. In sinus tachycardia, the rate of the impulse originating from the SA node is 100-180 beats per minute (bpm).

The most common causes include:

- physiological: physical or psychological stress, caffeine, alcohol;
- non-cardiac causes: fever, anemia, pain, hyperthyroidism, pulmonary embolism;
- medications: epinephrine, atropine, β-adrenergic receptor agonists;
- cardiac diseases: myocardial infarction, heart failure, myocarditis, shock.

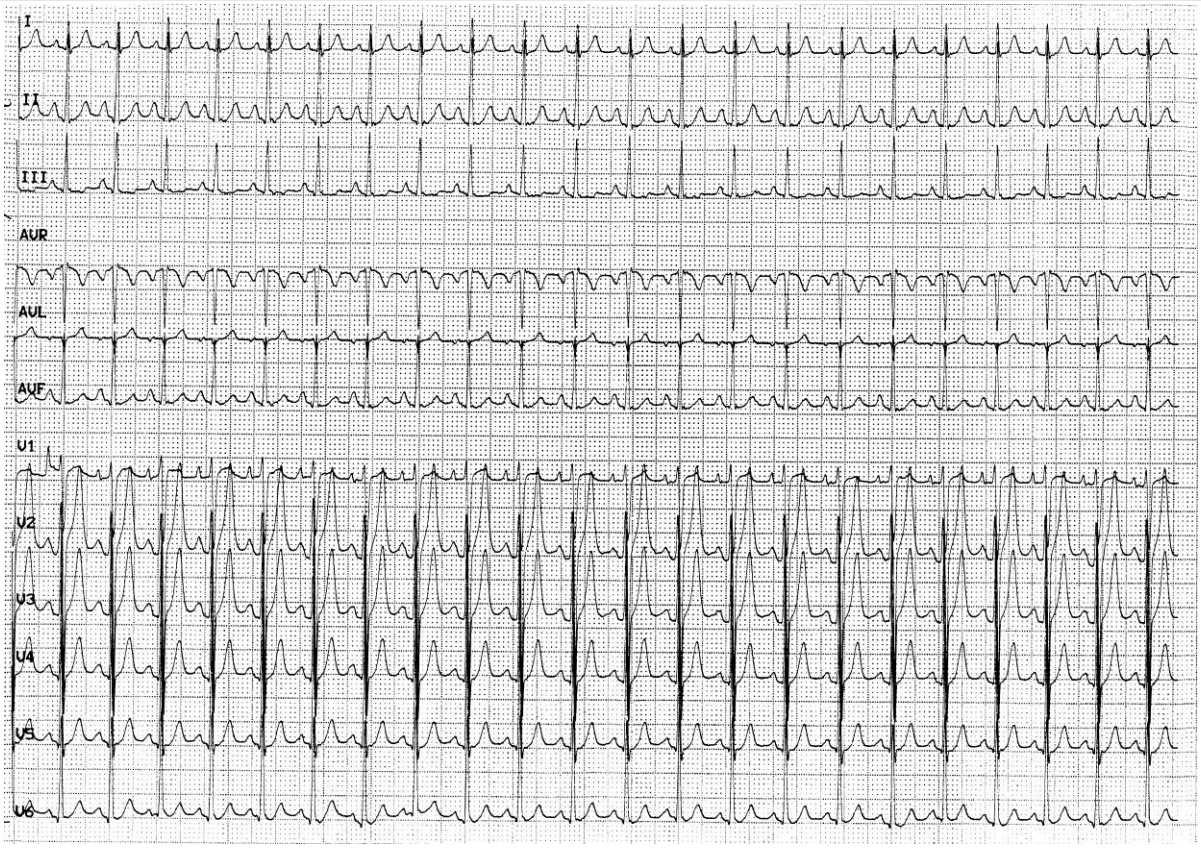


Figure 2/2.

Sinus tachycardia (a young patient with stress). (Sinus tachycardia, 140 bpm, normal QRS axis, normal ventricular conduction, tall, peaked T waves in leads V2-4 (caused by an increased sympathetic tone), otherwise normal ventricular repolarization.)

2.1.2. Sinus bradycardia

For this entity, there is a decreased rate of impulse formation with a regular P wave morphology, which does not necessarily represent a disorder of impulse generation, since it may also occur in healthy subjects, e.g. during sleep. In sinus bradycardia, the rate of the impulse originating from the SA node is below 60 bpm, however, it is regarded as clinically significant at rates below 50 bpm. By 24-hour Holter monitoring, i.e. continuous ECG recording, one may frequently find heart rates around 40 bpm (in fact, around 30 bpm in young athletes) in the night time hours, which is not considered abnormal, however, if this occurs during daytime in the active period, e.g. during physical exercise, it is then indicative of having a disease.

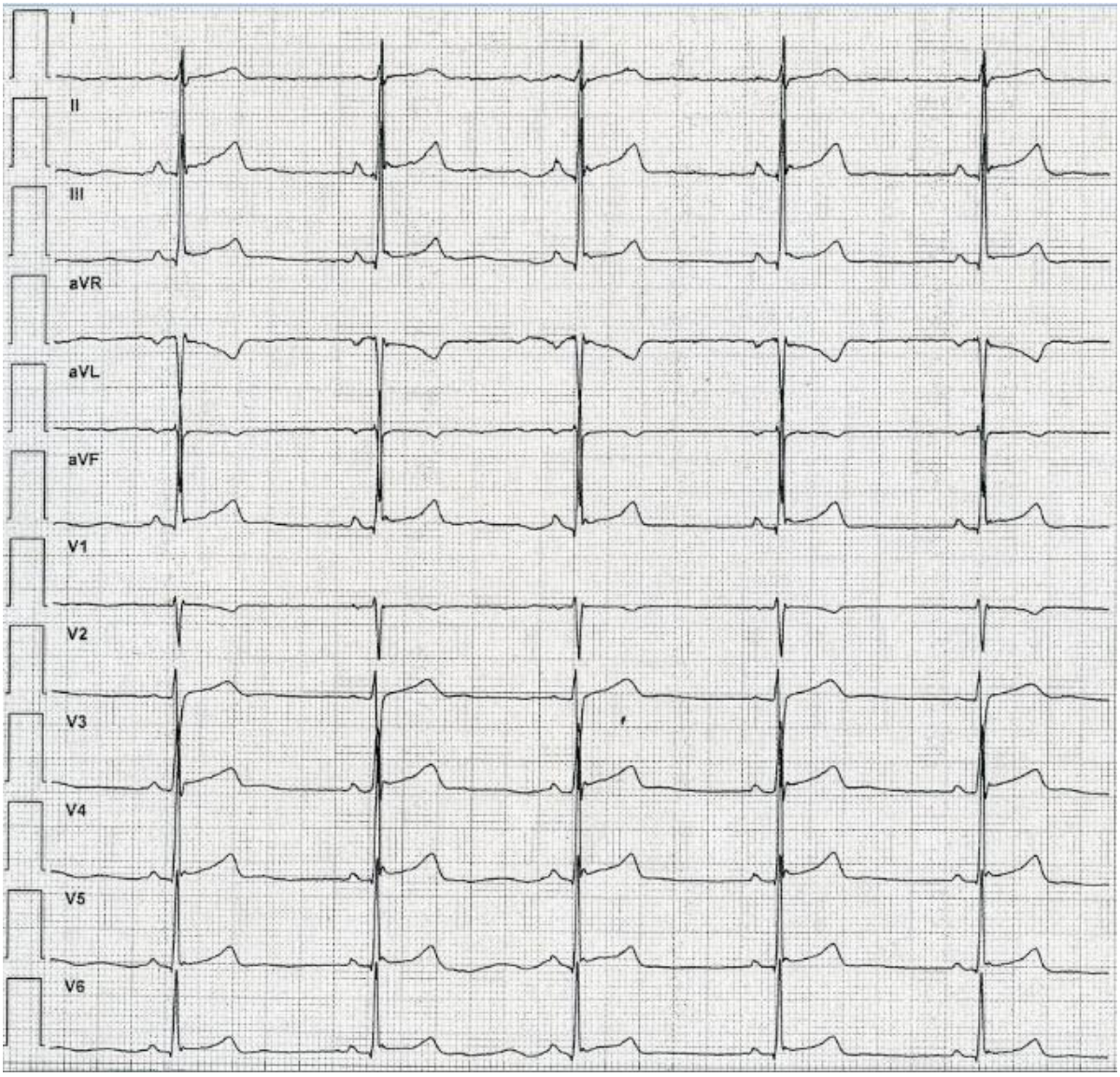


Figure 2/3.

Sinus bradycardia. (Sinus bradycardia, 49 bpm, normal QRS axis, R wave transition zone in lead V3, narrow QRS complexes, early repolarization pattern in leads II, III, aVF and V3-6.)

The most common causes include:

- physiological: physical rest, calmness, regular sports activities, sleep;
- non-cardiac causes: increased vagal tone (e.g. vomiting, abdominal pain), hypothermia, hypothyroidism, peptic ulcer, biliary obstruction, severe increase in intracranial pressure, uremia;
- medications: β-adrenergic receptor blockers, digitalis;
- cardiac diseases: myocardial infarction (especially inferior MI), sinus node disease.

2.1.3. Sinus node reentrant tachycardia

A rare type of arrhythmia with a reentry mechanism and occurring in the sinoatrial region. The only distinguishing feature from sinus tachycardia is that it has a sudden onset and termination, i.e. paroxysmal. During an ongoing arrhythmia, it is nearly indistinguishable from sinus tachycardia merely on the basis of the 12-lead surface ECG as the P wave morphology of this arrhythmia does not differ from that of sinus P waves. The typical heart rate is 120-140 bpm.

2.1.4. Wandering atrial pacemaker

The site of impulse formation is 'wandering' between the SA node and the AV node and, accordingly, there is a continuous change in the shape of P waves and the PQ intervals and at least 3 different types of P wave morphology can be distinguished in a single lead. The underlying cause is fluctuations in vagal tone. The heart rate is nomal.

2.1.5. Sinus arrhythmia

For this type of arrhythmia, impulses originating from the SA node are present typically with normal and constant P wave morphology and PQ intervals, and cyclic changes are only observable in the PP intervals ($\Delta PP > 0.16$ sec), along with a normal heart rate.

It has two forms:

phasic variant: respiratory arrhythmia, the explanation of which should be sought in the Bainbridge reflex. In inspiration, there is an increase in venous filling resulting in an increase in the distension of cardiac walls, which leads to a decrease in vagal tone and, as a consequence, an increase in heart rate. Thus, there is a heart rate increase during inspiration, while a decrease in expiration.

non-phasic variant: it is unrelated to breathing and the causes may include myocardialis infarction, ischemic heart disease, sick sinus syndrome, aortic insufficiency and increased intracranial pressure.

From a differential diagnostic aspect, it should be distinguished from sinus arrest, sinoatrial and AV block and supraventricularis premature beats (SVPBs).

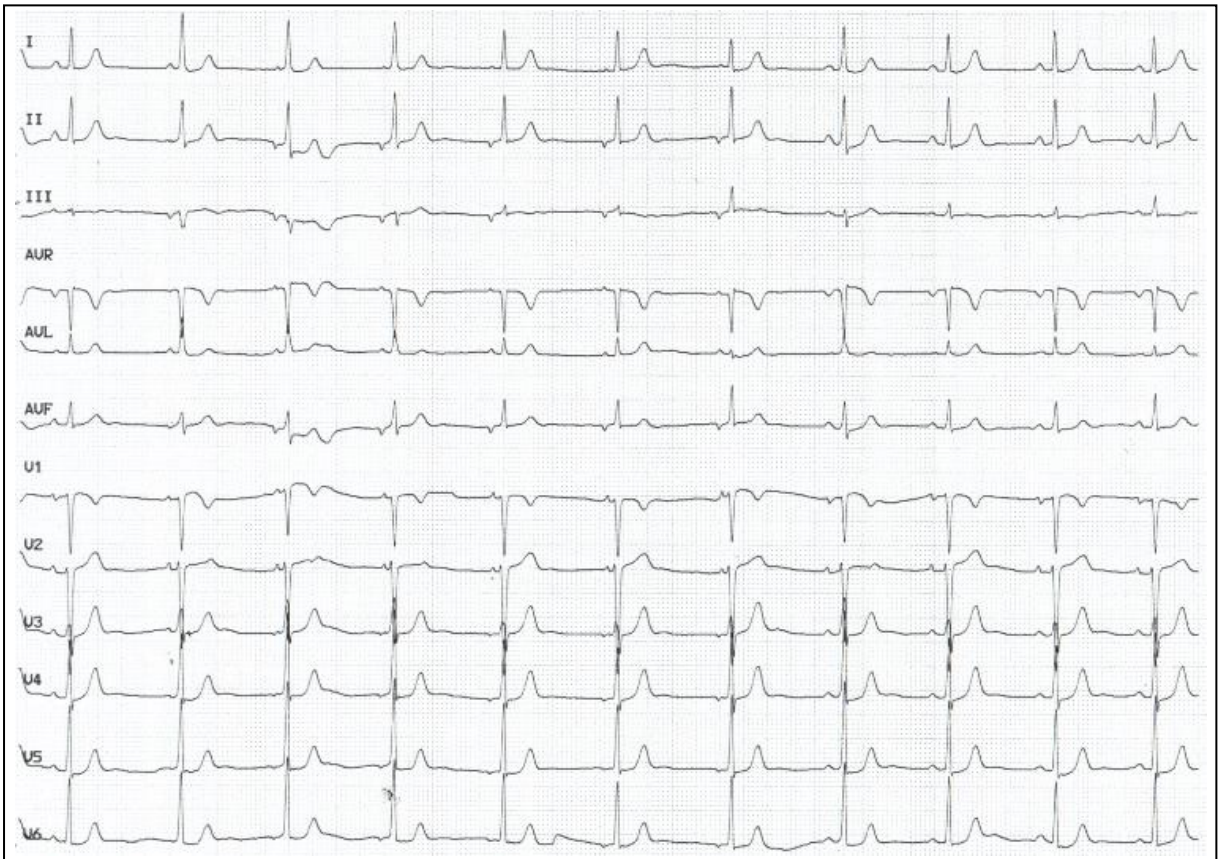


Figure 2/4.

Wandering atrial pacemaker: Sometimes positive P waves (sinus node) and sometimes negative P waves (near the coronary sinus) are visible in leads II, III and aVF. (Wandering atrial rhythm, normal QRS axis, normal atrioventricular conduction time, normal ventricular conduction, trivial horizontal ST segment depression in leads V5-6, otherwise normal ventricular repolarization.)

2.1.6. Sinus node disease

It is also called sinus node dysfunction or sick sinus syndrome (SSS). The disease is characterized by dysfunction of the sinus node accompanied by a disorder of impulse formation in the subordinate centers. The failure in the subordinate center manifests in two ways: either it does not generate impulses in the absence of an impulse from a superordinate center or the cessation of overdrive suppression by the superordinate center may lead to episodes of tachycardia occurring as bursts (i.e. paroxysmally). The significance of this disease lies in the fact that this is the *most common bradyarrhythmia* often leading to asystolia and loss of consciousness, which can be prevented by pacemaker treatment.

Two forms of the sinus node disease can be differentiated:

- intrinsic: anatomical lesion of the sinus node (e.g. ischemia, fibrosis etc.);
- extrinsic: dysfunction caused by an effect of the autonomous nervous system or medications.

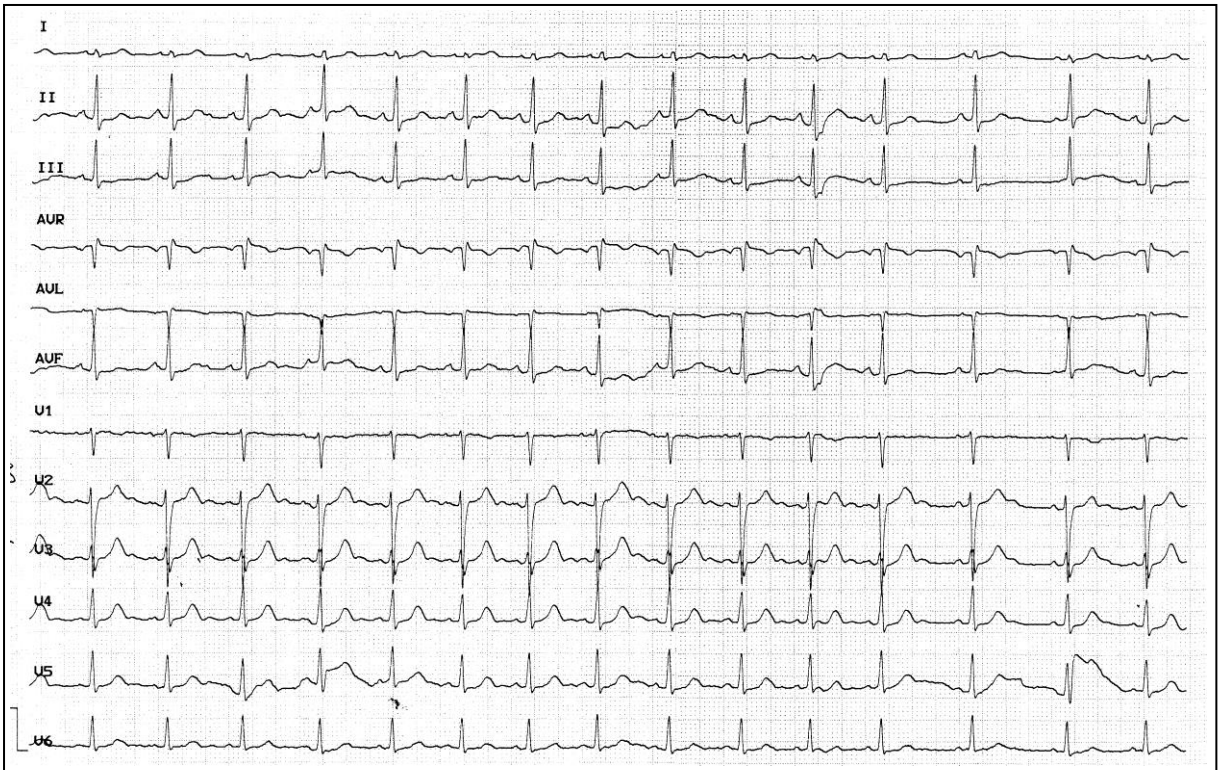


Figure 2/5.

Sinus (respiratory) arrhythmia in a young patient. Please note that it is only the RR intervals, not the P wave morphology, that are altering on the right side of the recording (sinus rhythm at a normal heart rate, sinus arrhythmia, normal QRS axis, normal atrioventricular conduction time, normal ventricular conduction and repolarization.)

The characteristic symptoms of the patient call one's attention to sinus node disease, including (pre)syncope, dizziness, weakness, palpitations, however, there may even be memory loss and personality changes, especially in elderly patients (pseudodementia). Typically, symptoms do not persist constantly, but in an intermittent fashion. It can also be a characteristic sign that if medications suppressing the SA node (e.g. beta-blockers, digitalis) are administered, bradycardia of unusual degree occurs even at low doses of these agents.

The causes of the disease listed in order of frequency are the following: ischemic heart disease (IHD), senile fibrosis, cardiomyopathy, medications (beta-blockers, digitalis, quinidine), myocarditis. It may happen that the disease attacks not only the sinus node, but also the AV node, which is referred to as binodal disease.

ECG manifestations:

Pure bradycardia: Characteristic features are sustained sinus bradycardia (≤ 50 bpm), sinus arrest (sinus pause), which leads to so-called Stokes Adams attacks (syncope) in case of disturbances in the escape activity of the subordinate center (that is in the abscence of an escape rhythm). If missing impulses in the SA node result in a pause longer than 3 seconds, it can be considered significant. In this form, implantation of a permanent pacemaker is often necessitated in symptomatic cases.

Sinoatrial exit block: There are impulses generated in the SA node, but they cannot be conducted to the atria. It is impossible to distinguish complete (third-degree) sinoatrial exit block from sinus arrest on the 12-lead surface ECG. The reason for that is that depolarization of the SA node is not visible on the surface ECG. For details, see disorders of impulse conduction.

Bradycardia-tachycardia syndrome: Episodes of sinus bradycardia are typically interrupted by episodes of atrial fibrillation with a fast ventricular rate, which have a sudden onset and termination. On their cessation (or during cardioversion), a longer pause can frequently be recorded until the sinus node function has been recovered. The atrial tachyarrhythmia is induced by an abnormal excitation of the subordinate centers.

Chronotropic incompetence: It is very typical of sinus node disease and it is mostly based on this that the diagnosis of the disease can be established. It means that the increase in heart rate in response to physical exercise is inappropriate, often not reaching 100 bpm even under significant physical exertion. Exercise stress test is used for its examination.

The atropine test was also used long ago; i.e. after administration of 2 mg of atropine, the heart rate did not increase above 90 bpm or an increase of less than 25% was observed. Moreover, rarely measured parameters also include sinus node recovery time (SNRT) and sinoatrial conduction time (SACT), which can be recorded with intracardiac electrodes during an electrophysiology study.

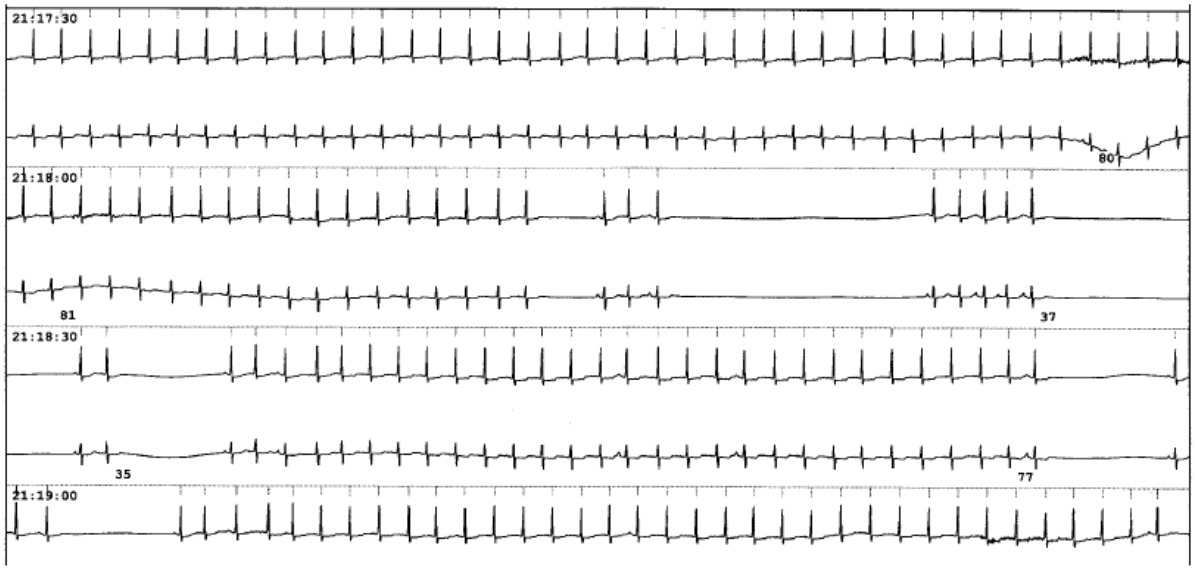


Figure 2/6.

Sinus node disease verified by an ECG recording of the Holter monitor; the long pauses are the result of sinus arrest.

Due to the intermittent presentation of complaints and ECG signs, the most important diagnostic tool to establish sinus node disease is 24-hour Holter monitoring and exercise stress testing.

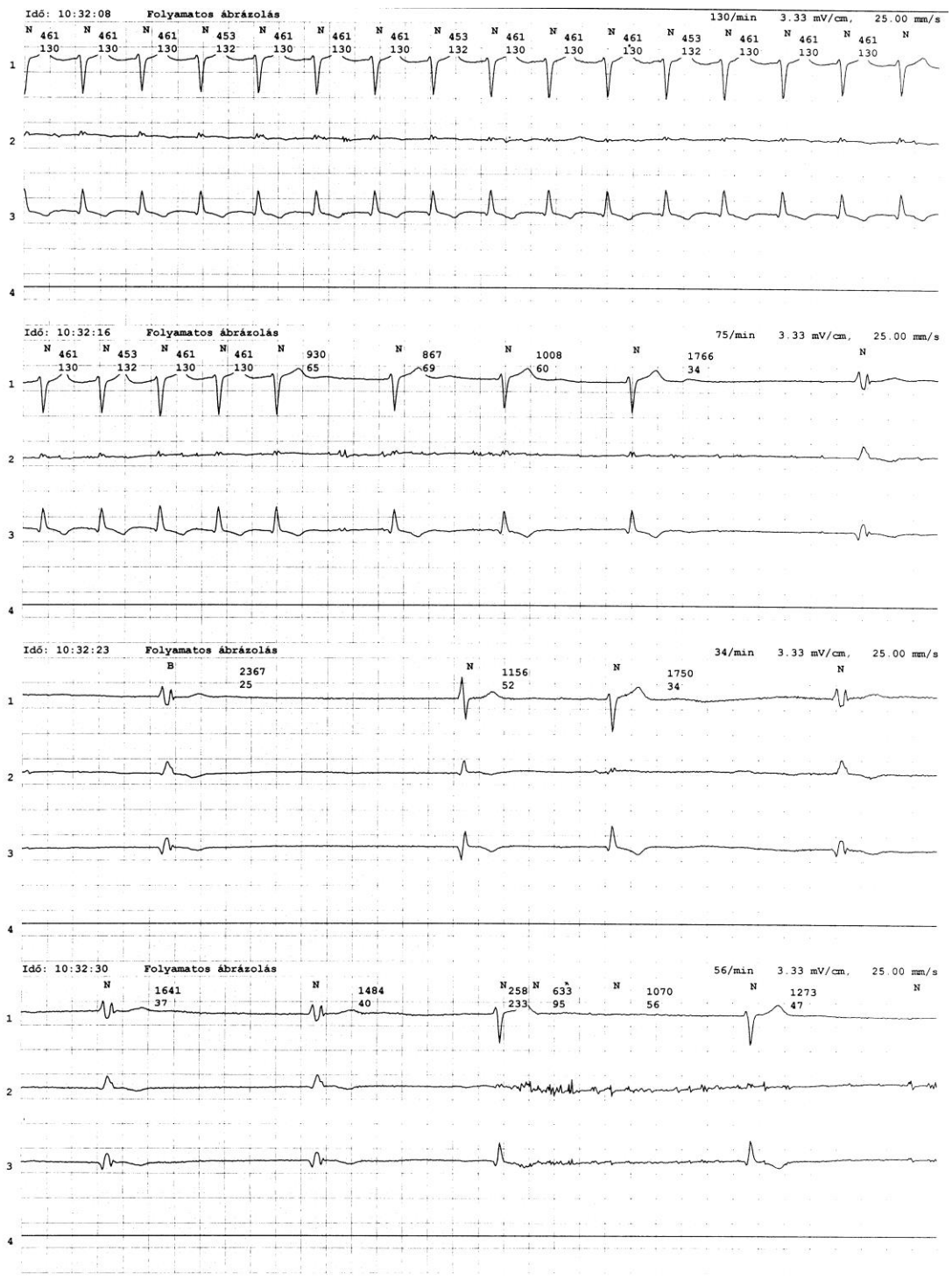


Figure 2/7. Atrial flutter with 2:1 conduction (or maybe atrial tachycardia), which is transformed into atrial fibrillation with a slow ventricular response (i.e. with bradycardia), may also be a characteristic sign of sinus node disease.

2.2. Disorders of heterotopic impulse formation

The impulse inducing contraction of the heart does not originate from the SA node, but outside of it, so it is referred to as *ectopic impulse formation*.

Supraventricular active heterotopic impulse formation: The impulse-generating rate of the subordinate center is greater than that of the SA node.

2.2.1. Premature beats or ectopic beats or extrasystoles

It is only for a *single beat* that the subordinate center is taking over the pacemaker role, i.e. only a single heterotopic beat will prevail. An extrasystole is defined as a beat *occurring* before the expected beat from the sinus node, so *earlier than expected.*

We must review some definitions related to an extrasystole, including:

Coupling interval: the distance between the last regular (sinus) QRS complex and the QRS complex of the extrasystole

Compensatory pause: the time from the QRS complex of the extrasystole to the next regular QRS complex.

Compensated extrasystole: coupling interval + compensatory pause = 2x the time between regular successive QRS complexes (2 RR intervals);

Undercompensated extrasystole: coupling interval + compensatory pause < 2x the time between regular successive QRS complexes (2 RR intervals);

Overcompensated extrasystole: coupling interval + compensatory pause > 2x the time between regular successive QRS complexes (2 RR intervals);

Interpolated extrasystole: coupling interval + compensatory pause = 1x RR interval, the extrasystole does not interfere with sinus activity.

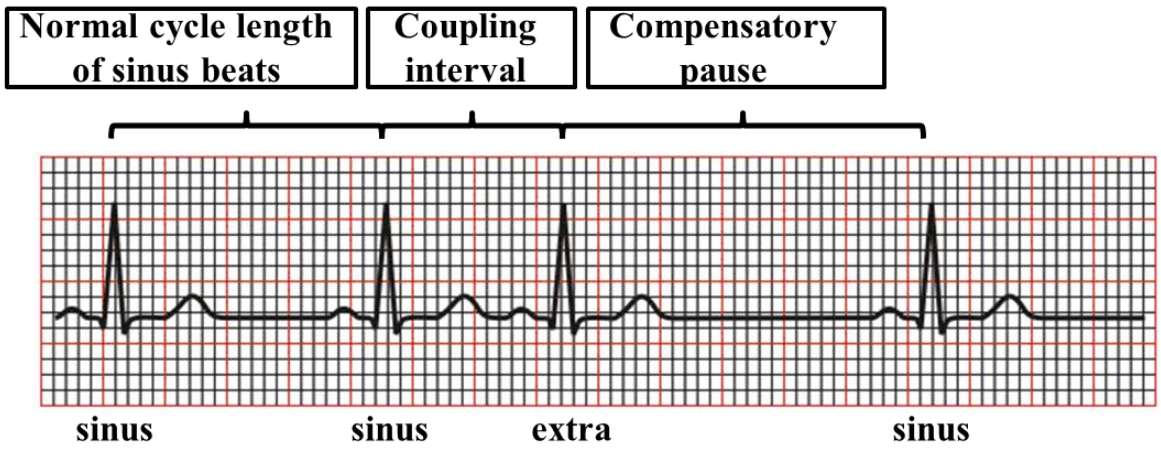


Figure 2/8.

Compensated supraventricular extrasystole. The morphology of the QRS complex of the extrasystole is identical with that of sinus beats and the duration of the coupling interval + compensatory pause = 2x the duration of the normal cycle length of the sinus beat.

Passive heterotopic impulse formation is a phenomenon when the impulse providing overdrive suppression is missing for some reason (e.g. sinus arrest or AV block) and, at this time, the subordinate center starts firing at its own intrinsic pacemaker rate.

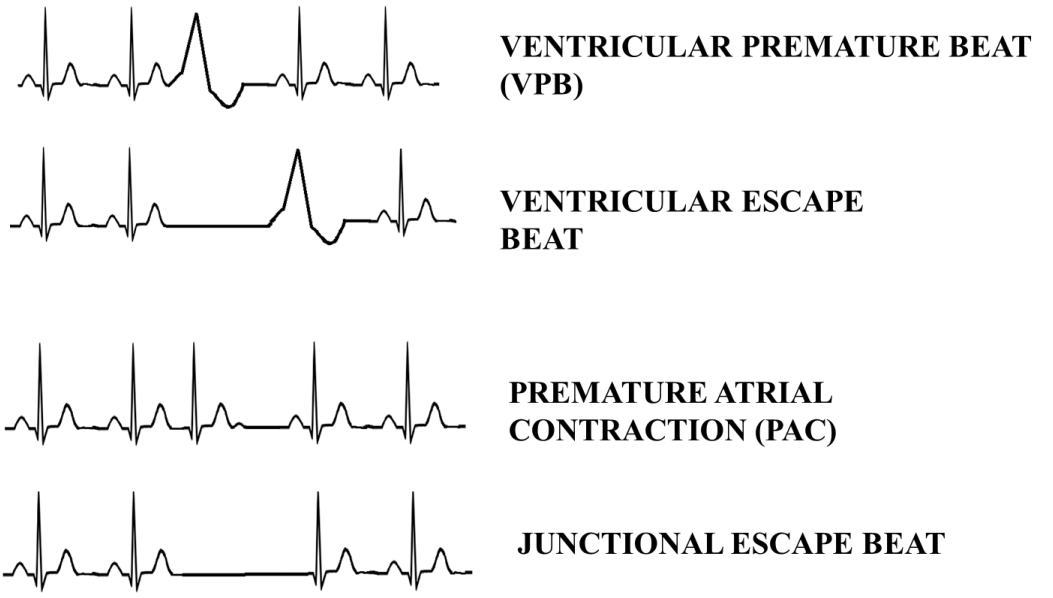


Figure 2/9.

The distinction between active and passive heterotopic impulse formation, i.e. between an ectopic beat and an escape beat, is made based on timing of the specific beat. Moreover, based on the width of this beat, one may make conclusions about its supraventricular or ventricular origin.

Supraventricular premature complexes (SVPCs) or premature atrial contractions (PACs) or atrial premature beats (APBs)

This category includes active heterotopic impulses originating in the bundle of His or regions above that, which may have an atrial and junctional origin. The site of the ectopic focus cannot always be specified accurately on a 12-lead surface ECG recording. The *shape of the P waves is different* from that of sinus P waves; however, the *lack* of presence of P waves is observable perhaps even more frequently since the ectopic P wave is either blending into the concurrently occurring T wave or may even be isoelectric. After arrival of the impulses of PACs to the sinus node, they reset its timing and cause its cycle to appear earlier, so PACs are generally '*undercompensated*' (i.e. they are followed by an incomplete pause). The PQ interval may be normal or prolonged. The *shape of the QRS complexes* of PACs is *identical with the QRS morphology of beats from the sinus node* because ventricular conduction already takes place on the same cardiac structures (bundle of His – bundle branches - Purkinje fibers, etc.).

It is a frequent mistake that, in case of bundle branch blocks, the beat presenting earlier than expected is considered a ventricular premature beat, stating that the QRS complex is wide, but the shape of the QRS complex does not differ from that of the QRS complexes related to sinus beats in this case either, so it can only be a PAC. It may occur that the QRS complexes associated with sinus beats are narrow, then a premature beat occurs, which is wide, but is preceded by a P wave of non-sinus origin unequivocally. In such a case, one should consider the presence of aberrant ventricular conduction, which can be explained by the fact that a supraventricular impulse occurring earlier than expected found one of the bundle branches refractory and was therefore conducted to the ventricles with a bundle branch block pattern. Following this logic, it may also happen

that both bundle branches are in the refractory period at the time of arrival of the impulse of a PAC, thereby resulting in the occurance of a so-called non-conducted (blocked) PAC, which is represented on the surface ECG as a P wave of non-sinus origin presenting earlier than expected and not followed by a QRS complex; however, the rhythm is perturbed by the reset of timing of the SA node.

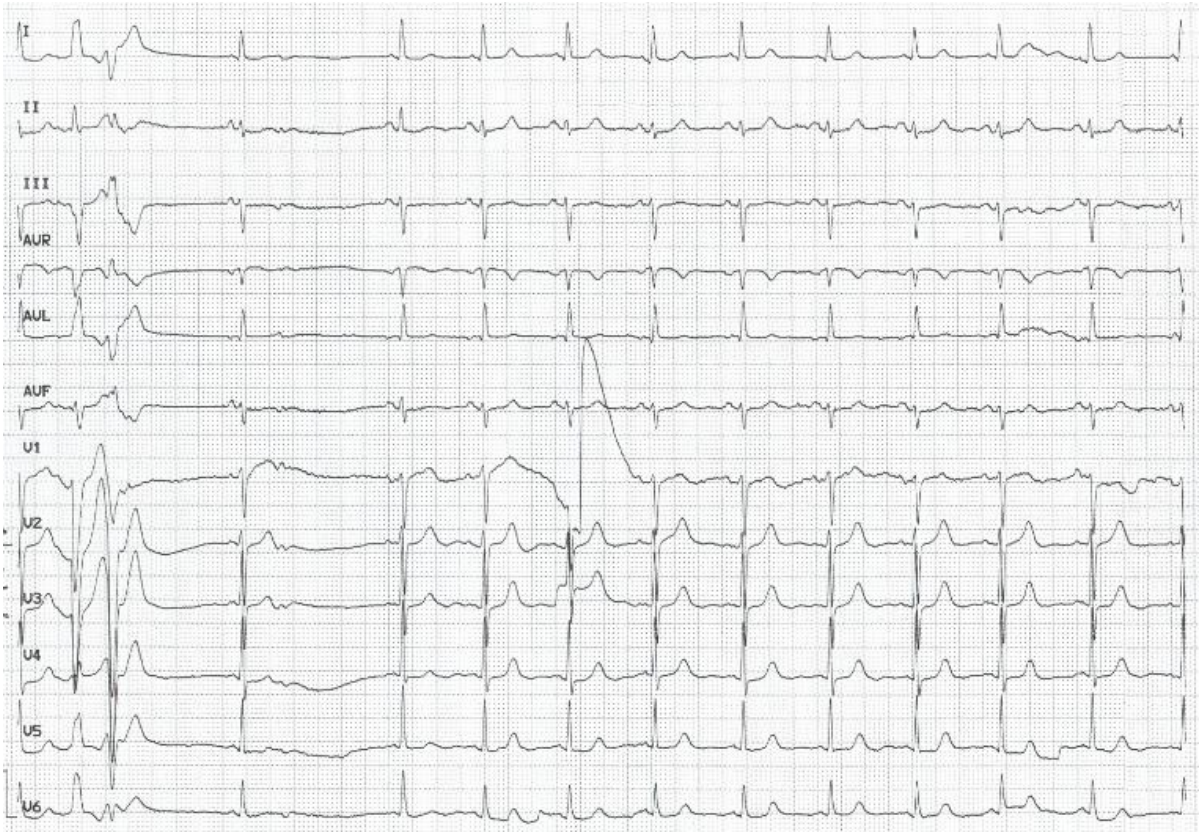


Figure 2/10.

A PAC is visible after the 1st QRS complex, which is conducted with a left bundle branch block morphology (2nd QRS complex); the 3rd beat may be a ventricular premature beat or an 'echo beat' conducted in a retrograde fashion. After the 4th beat, which is a conducted sinus beat, a blocked PAC can be seen, then the subsequent sinus beats are conducted to the ventricles. (Sinus rhythm, left axis deviation, normal AV conduction time, normal ventricular conduction and repolarization, blocked and aberrantly conducted PACs.)

The atrioventricular junction can be divided into three parts from an anatomical aspect: the transitional zone, the compact AV node and the penetrating AV node (bundle of His) with the branching bundles. Atrial cardiomyocytes are connected to the compact AV node through cells of the transitional zone. The anterosuperiorly and posteroinferiorly located fibrous projections of the AV node form the anatomical basis of the AV nodal fast and slow pathway.

The fast pathway is situated outside the Koch' triangle, above the tendon of Todaro, and it is connected to the compact AV node by transitional cells. The course of the slow pathway is situated within the Koch' triangle, between the ostium of the coronary sinus and the tricuspid annulus, and it is connected to the compact AV node by the posteroinferior fibrous projections.

The features of premature junctional complexes (PJCs) include that they are depolarizing the atria in a retrograde fashion and the ventricles in an antegrade fashion nearly simultaneously. Retrograde atrial activation propagates in the caudocranial direction, thus it moves away from the inferior leads and results in negative P waves in leads II, III and aVF (in sinus rhythm, atrial activation occurs inversely, in the craniocaudal direction). These P waves are most often situated before the QRS complexes, but they may also be hidden in, or after, the QRS complexes.

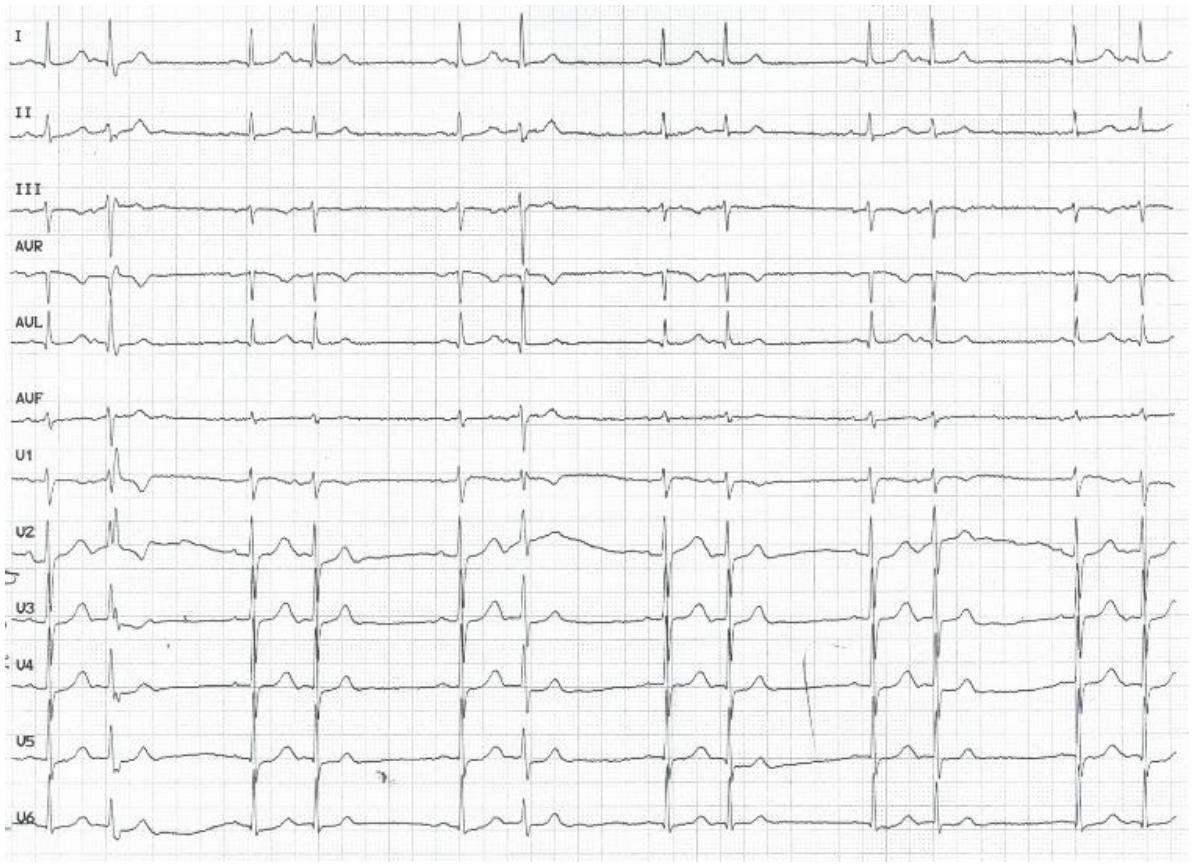


Figure 2/11.

Atrial (supraventricular) bigeminy. The 1st PAC is conducted with a right bundle branch block morphology (rSR' in lead V1), whereas the 3rd PAC with an incomplete right bundle branch block morphology (rSR' in lead V1); the rest of the PACs reach the ventricles through the normal conduction system. (Along with sinus rhythm, PACs in a bigeminal pattern and occasionally with aberrant conduction, normal AV conduction time, normal QRS axis, normal ventricular conduction and repolarization.)

The significance of supraventricular premature complexes lies in the fact that they may cause complaints on one hand and, on the other hand, they may call one's attention to certain medical conditions provoking them; however, they also appear in small numbers in all healthy individuals.

Their causes include atrial (pressure or volume) overload; at this time, they may predict the occurrance of imminent atrial fibrillation (!), ventilation disturbances (lung diseases), myocarditis, hyperthyroidism, digitalis overdose and autonomic instability. PACs alone

should only be treated if they constitute at least 1/6 of all beats and their appearance is associated with complaints, too.

If the abnormal supraventricular center takes over the pacemaker role not only for a single beat, but for several beats or even permanently, it is referred to as **heterotopic** (i.e. of non-sinus origin) **rhythm**. Two consecutive supraventricular beats are referred to as a *supraventricular couplet* or *coupled PACs*, three are called a *supraventricular triplet*, while 3-5 consecutive beats are designated as a *supraventricular run*.

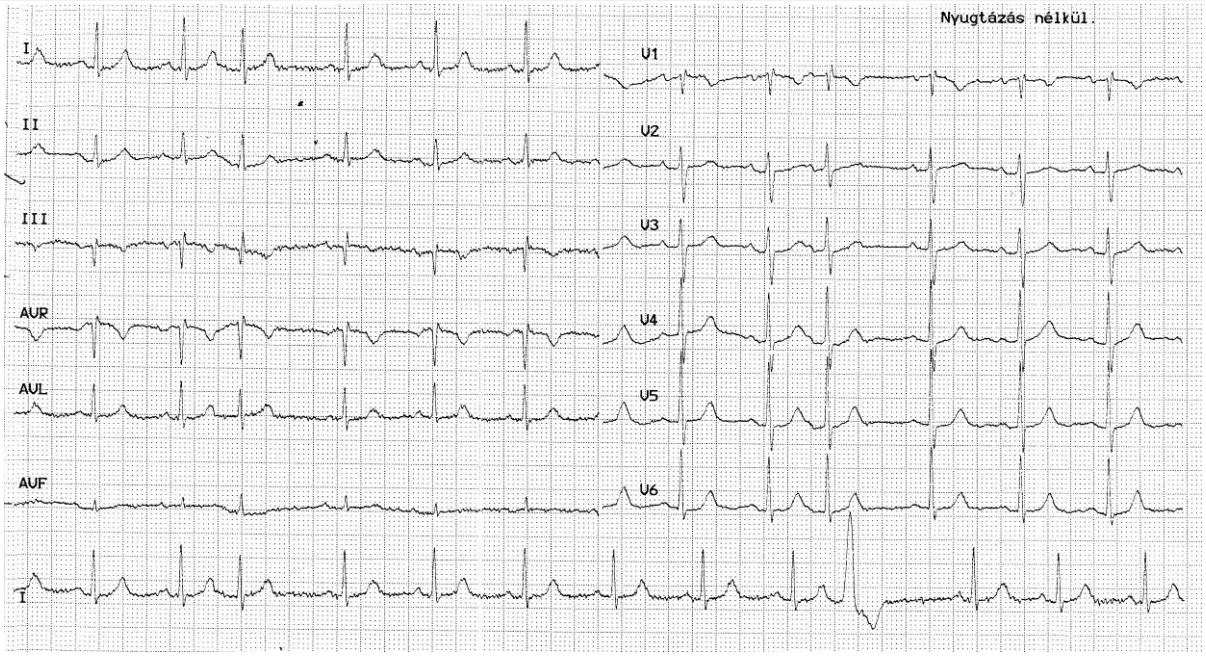


Figure 2/12.

PACs. The 3rd QRS complex appears earlier than expected, but it is narrow and has the same morphology as the QRS complexes related to sinus beats; i.e. it is a premature atrial contraction (3rd beat). In the second part of the rhythm strip (below), a premature beat with a wide QRS is observable, which is a premature ventricular complex.

If 6 or more consecutive ectopic beats are visible, it is referred to as supraventricular tachycardia (SVT), the general characteristics of which are the following:

- 6 or more consecutive supraventricular beats of non-sinus origin;
- The shape of the QRS complexes is identical with that of the QRS associated with sinus beats;
- Atrioventricular conduction is often partially blocked due to decremental conduction properties of the AV node, therefore, the number of P waves exceeds that of QRS complexes (due to a maximal upper heart rate limit of the AV node; e.g. an atrial rate of 240 bpm and a ventricular rate of 120 bpm represents a functional 2:1 AV block). The presence of PACs plus conduction block is frequent in digitalis toxicity, but it may also indicate ischemic heart disease (IHD) or myocarditis.

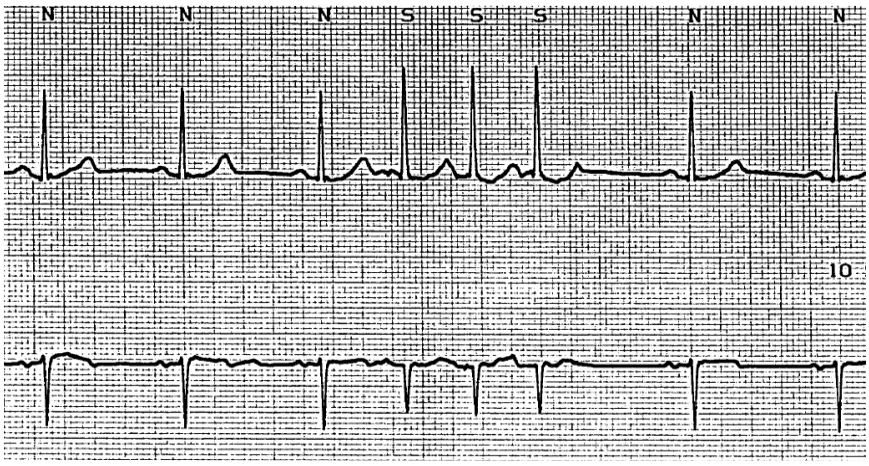


Figure 2/13.

Supraventricular triplet (run).

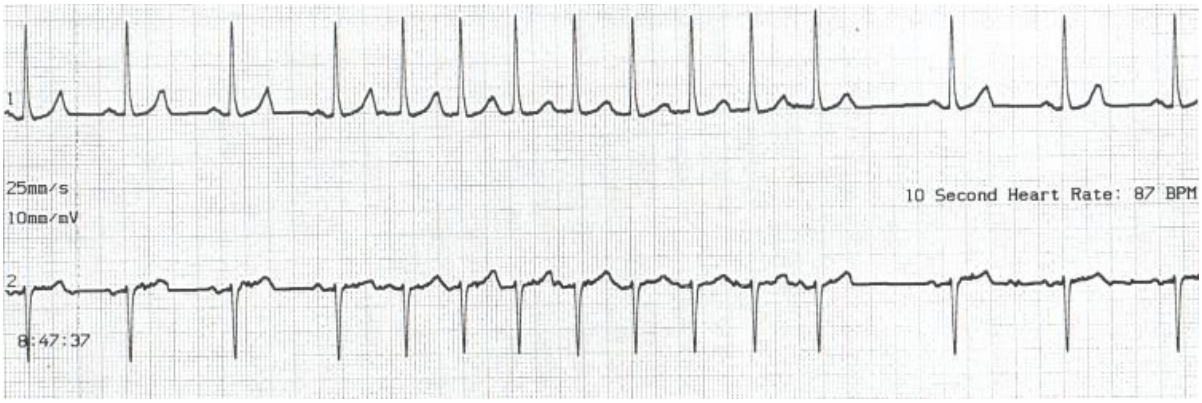


Figure 2/14.

Short supraventricular run.

2.2.2. Real atrial tachycardias

During Holter monitoring, shorter or longer episodes of atrial tachycardia are found in about 1/4 of all recordings. One of the characteristics of atrial tachycardias is that P waves other than sinus P waves occur at a rate of 120-240 bpm, they are therefore frequently accompanied by a functional 2:1 AV block. They arise from the right and left atrium in 75% and 25% of cases, respectively. Basically, two types can be differentiated:

1. paroxysmal:
   - reentrant: this is the most common type, however, it responds to medications only in a limited fashion;
   - based on abnormal automaticity (also frequent);
   - based on triggered activity (DAD): a typical example is digitalis-induced arrhythmia, which is characterized by good responsiveness to medications.
2. incessant:
   - based on abnormal automaticity (automatic atrial tachycardia).

2.2.2.1 Ectopic or automatic atrial tachycardia

The phenomenon of automaticity decreases with age; however, shorter or longer periods of this may be observed even in adults during Holter monitoring. Typically, the incessant form develops in children and its sustained presence results in the development of dilated cardiomyopathy, the so-called tachycardia-induced cardiomyopathy. One characteristic of this arrhythmia is, as it can be anticipated from its name, that *abnormal automaticity* constitutes its pathophysiological basis.

This mechanism is characterized by a gradual 'warm-up' phenomenon at arrhytmia onset (acceleration) and gradual termination (deceleration). Acceleration-deceleration generally occurs over a few beats. This type of tachycardia with the *'warm up'* and *'cool down'* phenomenon is frequently initiated from the right atrium. The atrial rate is 100-180 bpm, conducted beats are visible typically and blocked ventricular conduction is not a characteristic feature. The tachycardia is sensitive to adenosine in 80% of cases.

2.2.2.2. Intraatrial reentrant tachycardia

Its pathophysiological basis is reentry and, accordingly, it is paroxysmal, i.e. occurs as attacks, so it has a sudden onset and termination. The atrial rate may be 120-240 bpm, with a partial AV block in the upper ranges of the rate. When studying the relationship of P waves with the preceding and consecutive QRS complexes, the findings will be PR < RP (long RP tachycardia). The P wave morphology may help decide on the site of initiation. The arrhythmia often originates from the lower regions of the atria (adjacent to the AV junction), which is highlighted by negative P waves observed in the inferior leads.

It can rather be treated with catheter ablation (a search for the atrial focus and its destruction by a radiofrequency current), but class Ic antiarrhythmic agents may also be efficacious in patients with a structurally normal heart.

The causes include myocarditis, chronic obstructive pulmonary disease (COPD) and structural damage of the atrial musculature (e.g. after cardiac surgery).

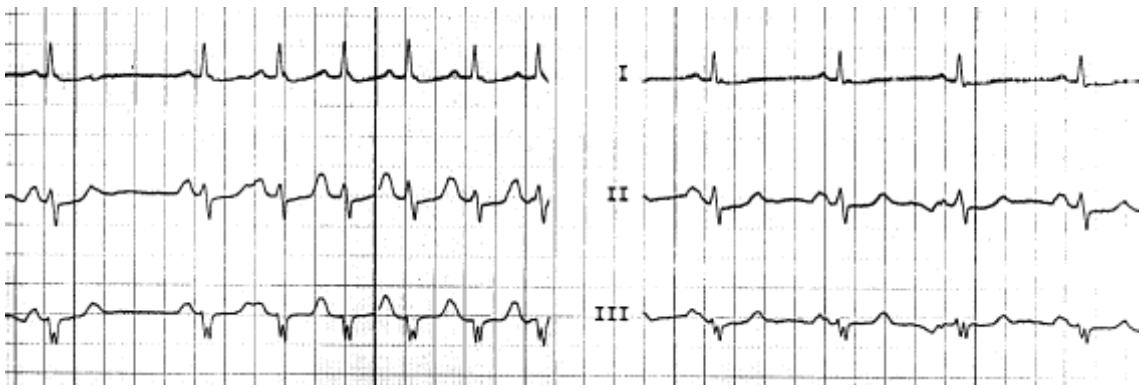


Figure 2/15.

Initiation of a paroxysmal atrial tachycardia with 1:1 conduction, and the ECG tracing after its cessation. In leads II and III, you might see the atrial waves superimposed on the T waves.

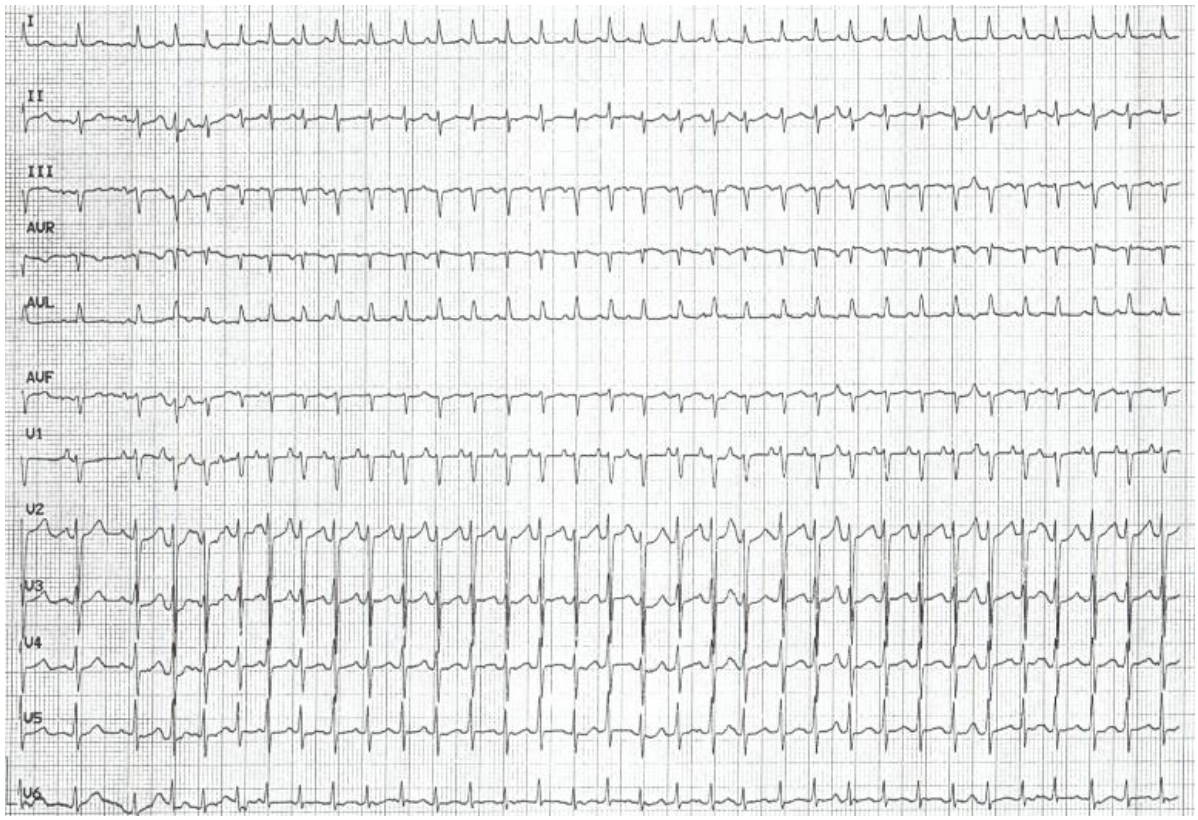


Figure 2/16.

Atrial tachycardia (long RP, 1:1 conduction, right atrial origin). In lead V1, narrow P waves of non-sinus origin are clearly visible. (Atrial tachycardia at a rate of 210 bpm and 1:1 AV conduction, left axis deviation, narrow QRS complexes, normal ventricular repolarization.)

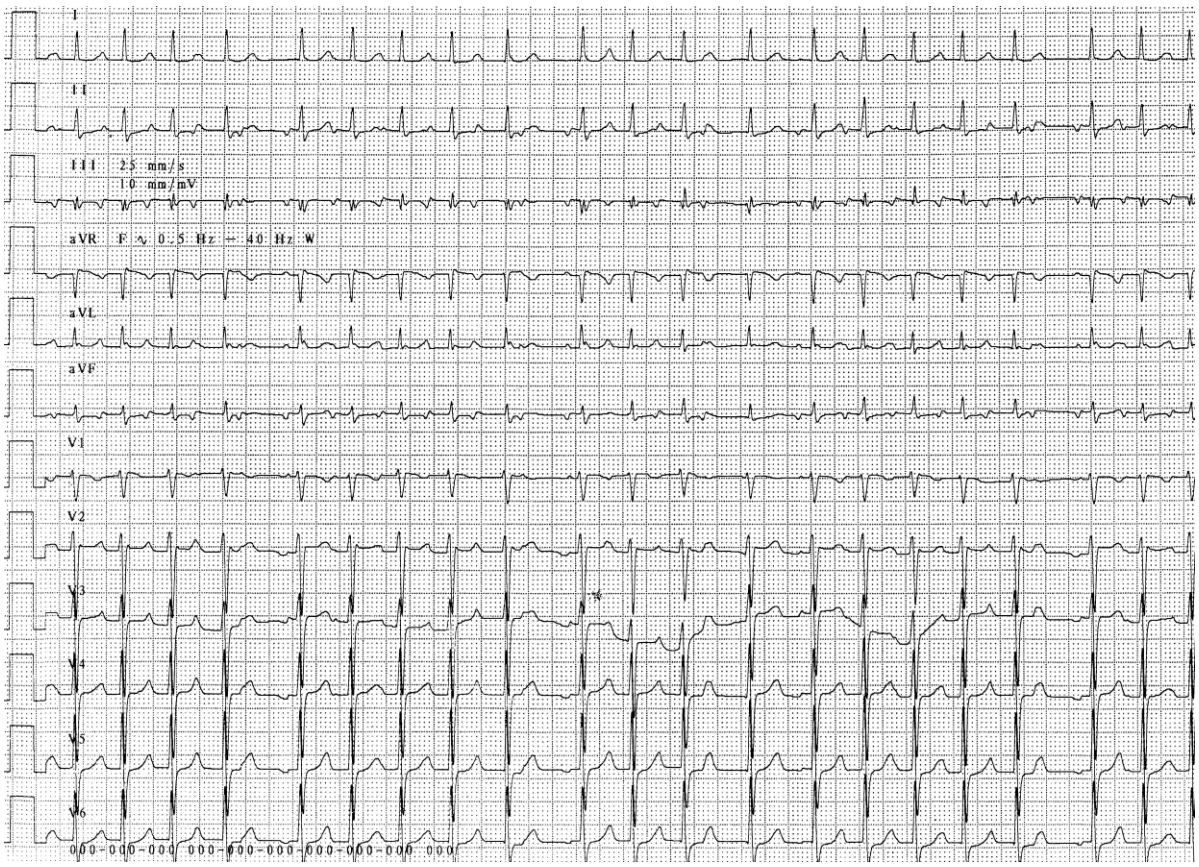


Figure 2/17.

Atrial tachycardia with Mobitz type I (Wenkebach) AV block. Gradually prolonged conduction after ectopic P waves, followed by a blocked P wave, is clearly observable in lead aVF. (Atrial tachycardia at a rate of 150 bpm and with Mobitz type I AV block, normal QRS axis, narrow QRS complexes, normal ventricular repolarization.)

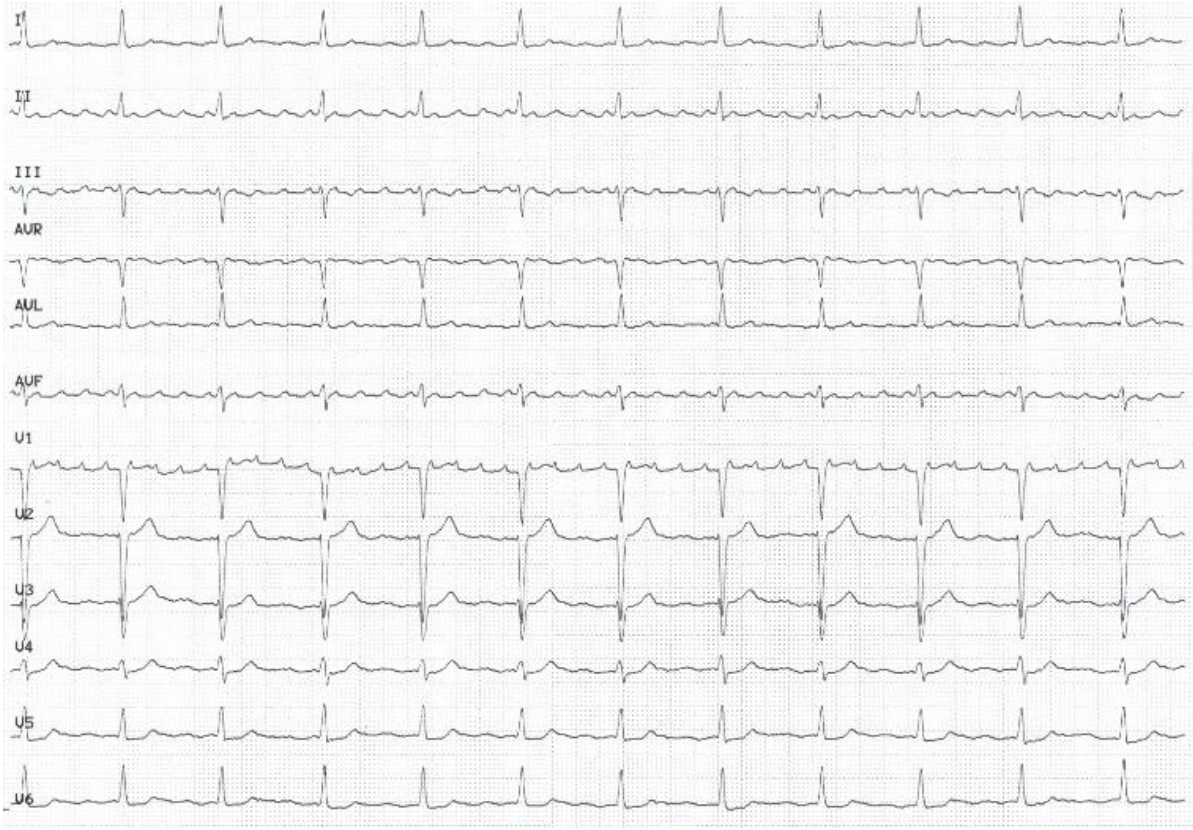


Figure 2/18.

Atrial tachycardia with 4:1 AV conduction block. A P wave morphology other than that of sinus P waves is clearly observable in lead V1. In this case, atrial rate is exceeding 250 bpm, but in spite of that it is a rapidly firing atrial focus, not atrial flutter, we are dealing with. (Atrial tachycardia with 4:1 conduction and with a ventricular rate of 67 bpm, normal QRS axis, R wave reduction in leads V1-3, otherwise normal ventricular conduction and repolarization.)

2.2.2.3. Multifocal atrial tachycardia (MAT)

The P wave morphology is similar to what has been described for wandering atrial pacemakers, that is P waves with at least three different morphology are detectable. The most important point is to differentiate it from atrial fibrillation, where the baseline is never isoelectric, while there is an isoelectric line between the P waves in MAT; however, RR intervals are changing continuously and the rhythm is irregular in both arrhythmias. Atrial rate may be 120-240 bpm, due to which partial AV block may also develop. Its causes include lung diseases, theophyilline overdose and electrolyte disturbances.

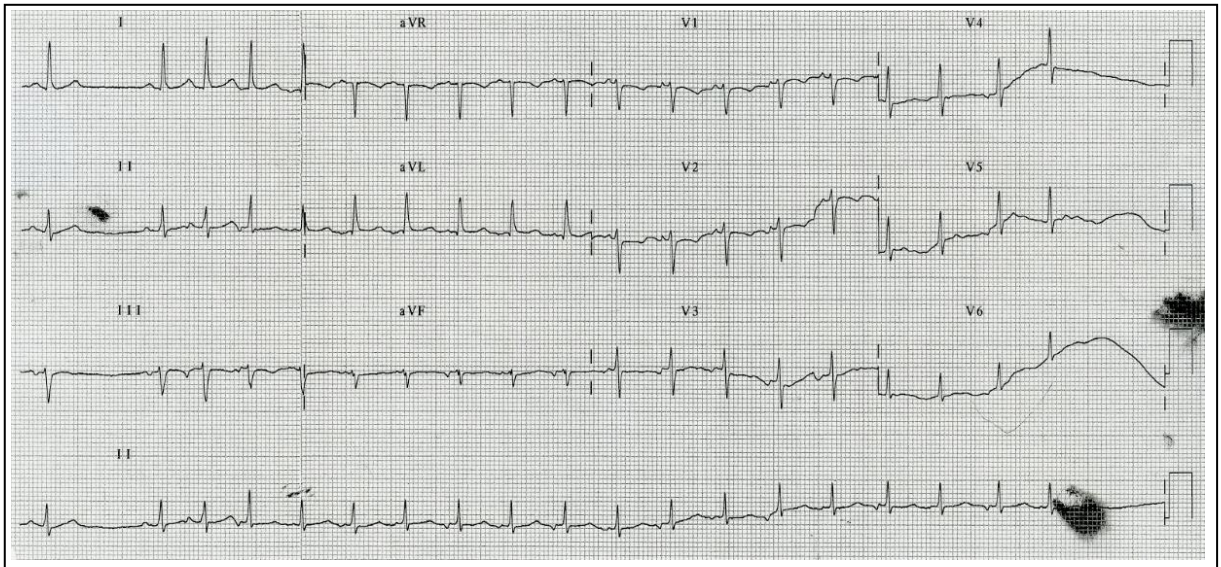


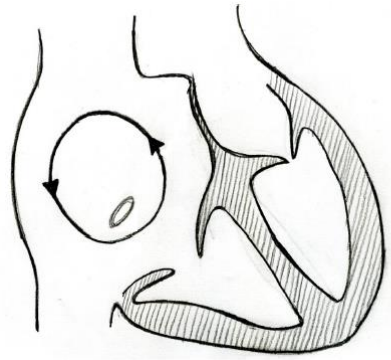
Figure 2/19.

Multifocal atrial tachycardia. You can clearly see a variety of P wave morphology on the rhythm strip (of lead II). (Multifocal atrial tachycardia at a ventricular rate of 130 bpm, left axis deviation, normal ventricular conduction and repolarization.)

2.2.3. Atrial flutter

Most often, it is a *macro-reentry* generated in the *right atrium*. Typically, the reentrant circuit makes a full circle in the right atrium at a speed of 300 bpm.

The border of the reentrant circuit is constituted by the tricuspid valve anteriorly, while posteriorly by a region formed by the superior vena cava, inferior vena cava and crista terminalis as well as demonstrating both anatomical and functional conduction block. The activation wavefront propagates downwards in the anterolateral wall of the right atrium and upwards in the interatrial septum.



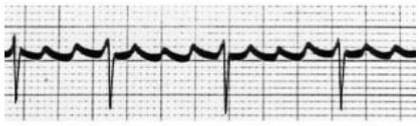


Figure 2/20. Atrial flutter. Macroreentry circuit in the right atrium.

Atrial flutter often appears paroxysmally, that is as attacks; however, a perpetuated form is also prevalent, and transformation into atrial fibrillation may also occur. This type of arrhythmia accounts for 10% of the so-called PSVTs (paroxysmal supraventricular tachycardia,) being more prevalent in males with a male to female ratio of 5:1. It is alternating with atrial fibrillation in 30-50% of cases or may coexist with it (coarse atrial fibrillation or

flutter = atrial fibrillation in the left atrium + atrial flutter in the right atrium). The arrhythmia can easily be recognized based on the 12-lead surface ECG because the *'saw-tooth' appearance* of fluctuations of the baseline – F waves – *in the inferior leads* (II, III and aVF) is striking right away. 2:1 AV block is characteristic of atrial flutter and, if the ratio is 4:1, one should think of an AV conduction abnormality. The ratio of conduction block may even demonstrate a beat-to-beat variation, making the heart beating arrhythmic (similar to what is seen in atrial fibrillation). If, during the physical examination or ECG interpretation, the heart rhythm is found to be regular and *a heart rate of 150 bpm is identified, one should always consider the presence of atrial flutter*.

The causes include atrial injury (e.g. after cardiac surgery), COPD, cardiac valve diseases and hyperthyroidism.

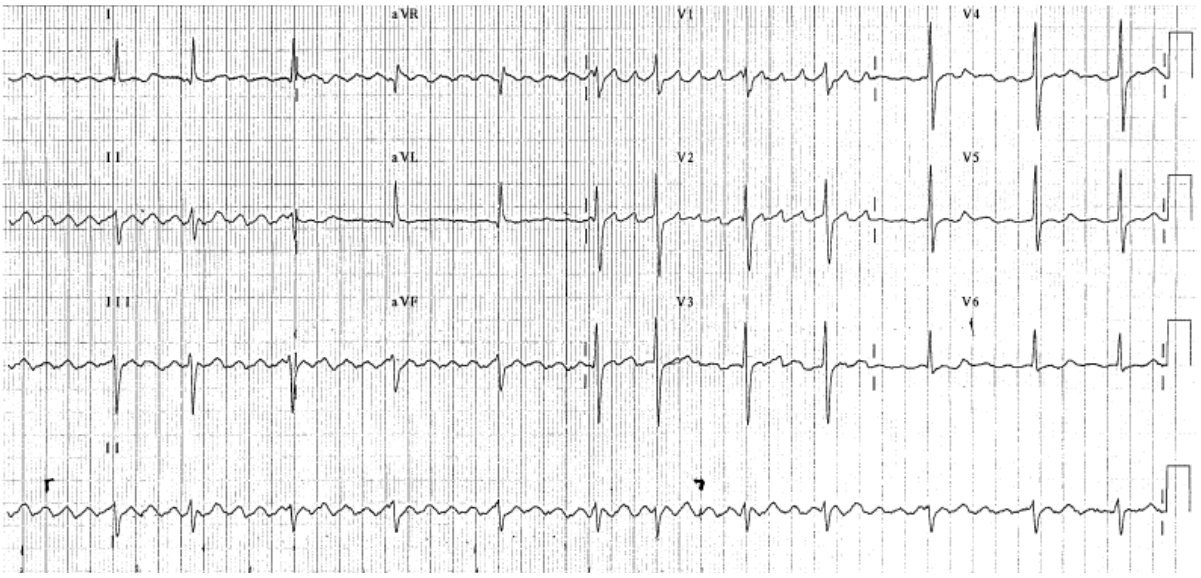


Figure 2/21.

Atrial flutter with variable AV conduction. The 'saw-tooth' pattern of the baseline is clearly observable and one might also see that some flutter waves are conducted, while others are not. (Atrial flutter with variable AV conduction and accompanied by a ventricular response with a normal heart rate, left anterior fascicular block, tall R waves in lead V1, early transition, narrow QRS complexes, signs of trivial left ventricular strain.)

According to the direction of rotation of the right atrial reentrant loop, several types of flutter can be differentiated:

- typical: Type I: The atrial rate is 240-340 bpm, with a counterclockwise rotation of the reentrant loop (negative F waves in leads II, III and aVF, and positive F waves in lead V1),

  Type II or reverse typical form: The atrial rate is 340-440 bpm, with a clockwise rotation pattern (positive F waves in leads II, III and aVF, and negative F waves in lead V1)

- atypical: This includes waveforms other than those described above, which is incisional tachycardia or left atrial macroreentry occurring due to scars developed after cardiac surgery performed for the correction of a congenital heart defect or due to a valve disease.

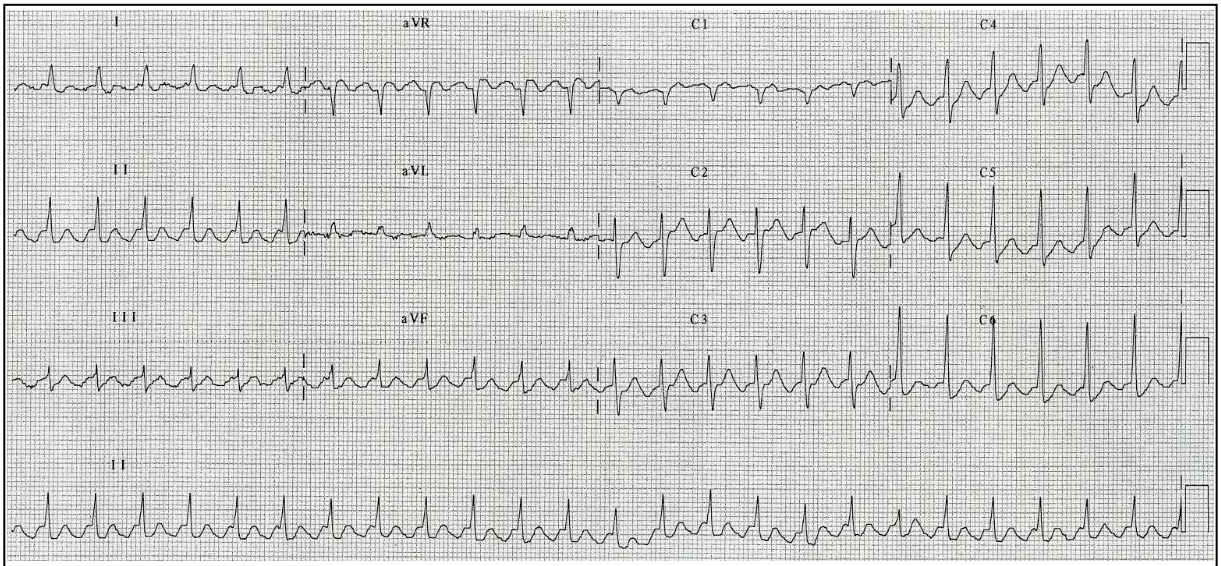


Figure 2/22.

Atrial flutter with 2:1 AV conduction. The ventricular rate of 150 bpm is very characteristic, so in such a case one should first think of atrial flutter. Note the flutter (F) waves between the QRS complexes or blending into the QRS in lead II. (Atrial flutter with 2:1 conduction and a ventricular rate of 150 bpm, normal QRS axis, normal ventricular conduction and repolarization.)

Regarding its treatment, one might speak about acute termination, which can be attained by electrical cardioversion or overdrive pacing; nevertheless, radiofrequency catheter ablation is successful in more than 90% of cases with a chronic recurrence. Ablation is performed at the so-called cavotricuspid isthmus of the right atrium, where disruption of the reentrant circuit is the easiest. In chronic cases, anticoagulation is necessary just as in those with atrial fibrillation. Please remember that for termination of the arrhythmia, class Ic antiarrhythmic agents may only be co-administered with a beta-blocker, otherwise 1:1 atrioventricular conduction may develop, which might result in the occurance of a life-threatening tachycardia.

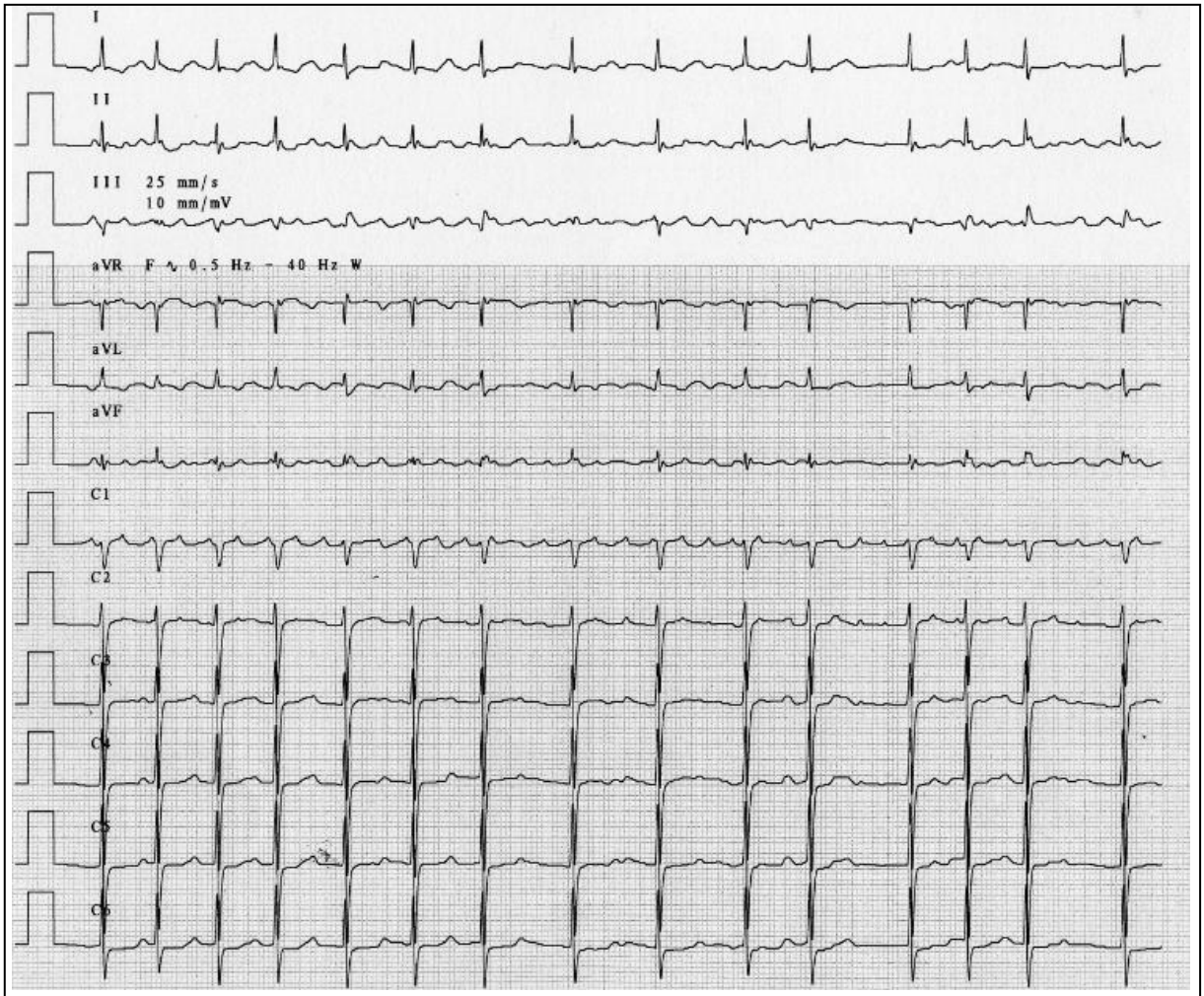


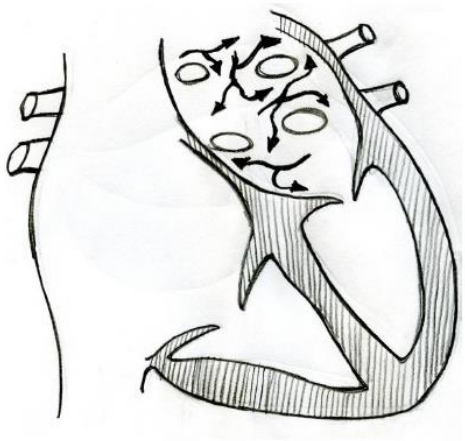
Figure 2/23.

Type II or reverse typical flutter. (Reverse typical flutter with variable AV conduction and an average ventricular rate of 110 bpm, low voltage QRS deflections in the limb leads, otherwise normal ventricular conduction, signs of trivial left ventricular strain)

2.2.4. Atrial fibrillation

Atrial fibrillation is a tachycardia with functional intra-atrial reentry, which can be induced during atrial stimulation. From a pathophysiological aspect, the following 'triad' of factors is necessary for its development and maintenance: ectopic activity (e.g. PACs) as a trigger, macro-reentry circuits and an arrhythmia substrate. The arrhythmia is generated by an inhomogeneity in impulse conduction and repolarization, moreover, slow atrial conduction and short atrial refractoriness are in favor of this inhomogeneity. The arrhythmia is most often initiated by coupled, or shorter or longer series of, premature beats originating from small groups of atrial cardiomyocytes growing into the pulmonary veins. The atrial premature beat may also occur after extreme bradycardia or following a pause with a longer duration. In the maintenance of atrial fibrillation, the development of micro-reentrant circuits plays an important role. They may occur due to an anatomical or functional conduction block, and concurrent presence of a minimum of 5 to 6 activation fronts with variable directions is necessary. One condition of arrhythmia recurrence is that a substrate must be present, that is

an electrophysiological and structural damage of the atrial musculature; the underlying causes of this may include hypertension and consequential left ventricular hypertrophy, coronary artery disease as well as myocardial fibrosis.



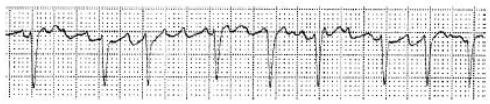


Figure 2/24.

Atrial fibrillation. Atrial micro-reentrant circuits.

The incidence of atrial fibrillation increases with age, with 0.5% in the overall population (over the age of 60: 2-4%; over the age of 65: 5%; over the age of 75: 12%; and 20-30% in acute myocardial infarction (AMI)). To recognize atrial fibrillation based on the 12-lead surface ECG is indispensable; its importance is highlighted by the fact that this is a common arrythmia and the most common underlying cause of cardiogenic embolism of the brain (i.e. cardioembolic stroke).

The ECG characteristics are the following:

- *irregular RR intervals (absolute arrhythmia):* By the term ’irregular’, you should think of the variable RR intervals, not the shape of the QRS complexes.

  (In atrial fibrillation, there is only one case that regular ventricular rhythm occurs; i.e. if complete AV block or ventricular paced rhythm is present concurrently. In any other case, the distance of QRS complexes from one another is irregular as well as regularly repeated group of beats cannot be observed either.)

- *lack of P waves* (A common mistake is that oscillations resembling a P wave are potentially observable on a slightly noisy ECG tracing in atrial fibrillation. In this case, one should always pay attention to the fact whether the deflection considered by one as a P wave is repeated at regular intervals, because if not, it is most likely to be a fibrillatory (f) wave. Another common mistake is that an ’oscillating’ baseline due to myopotentials is falsely interpreted to be atrial fibrillation; nevertheless, there are detectable P waves. For details, see section ’Noises and artifacts’.)

In atrial fibrillation, the atrial rate typically ranges from 450 to 650 bpm, so it is very frequent. Owing to the AV node, a maximum of about 180 impulses can be conducted to the ventricles. If the ventricular rate is above 180 bpm, one should consider the concurrent presence of an accessory pathway (for details, see section on FBI tachycardia). Depending on the ventricular rate, atrial fibrillation may also be designated as bradyfibrillation, tachyfibrillation or atrial fibrillation with a normal ventricular rate.

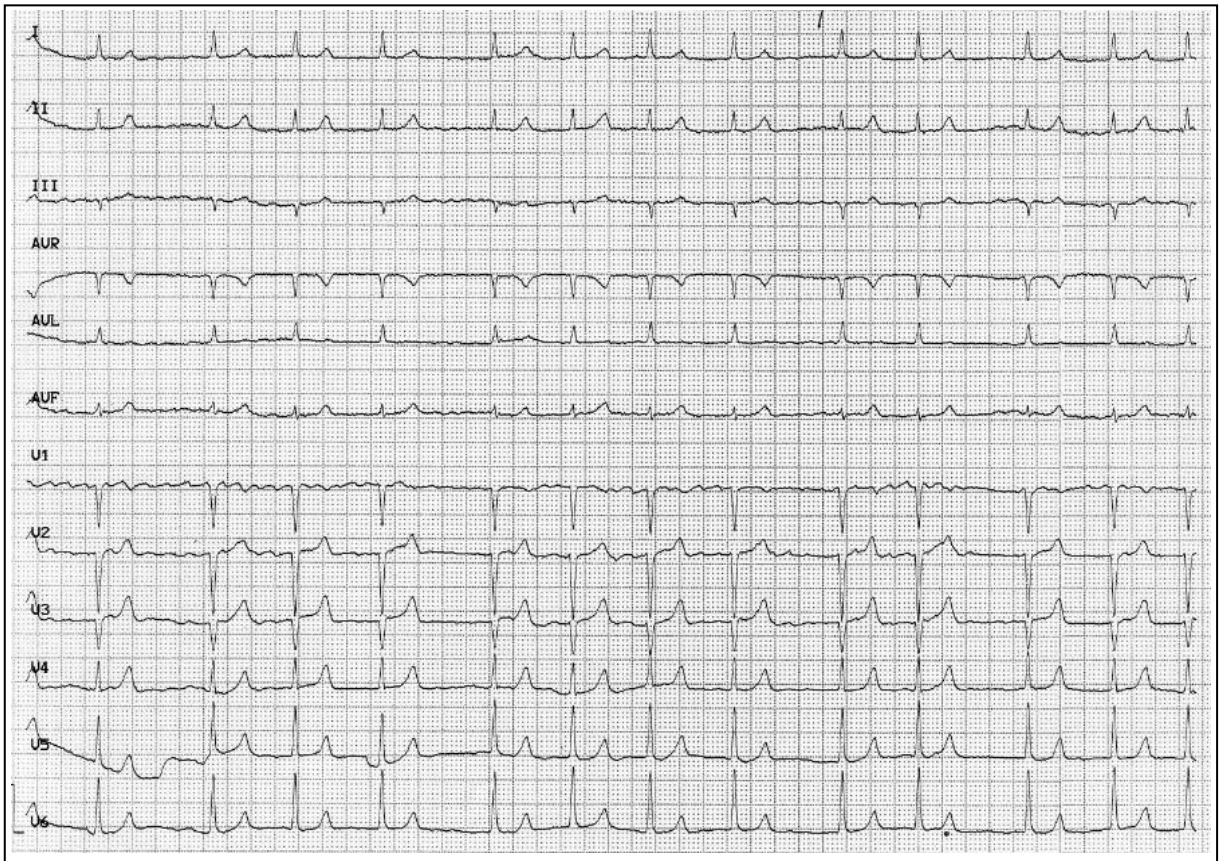


Figure 2/25.

Atrial fibrillation with a normal ventricular rate; QRS complexes appearing at irregular intervals are visible, along with the absence of P waves. (Atrial fibrillation with a normal ventricular rate, normal QRS axis, QS complexes in leads V1-3, normal ventricular repolarization.)

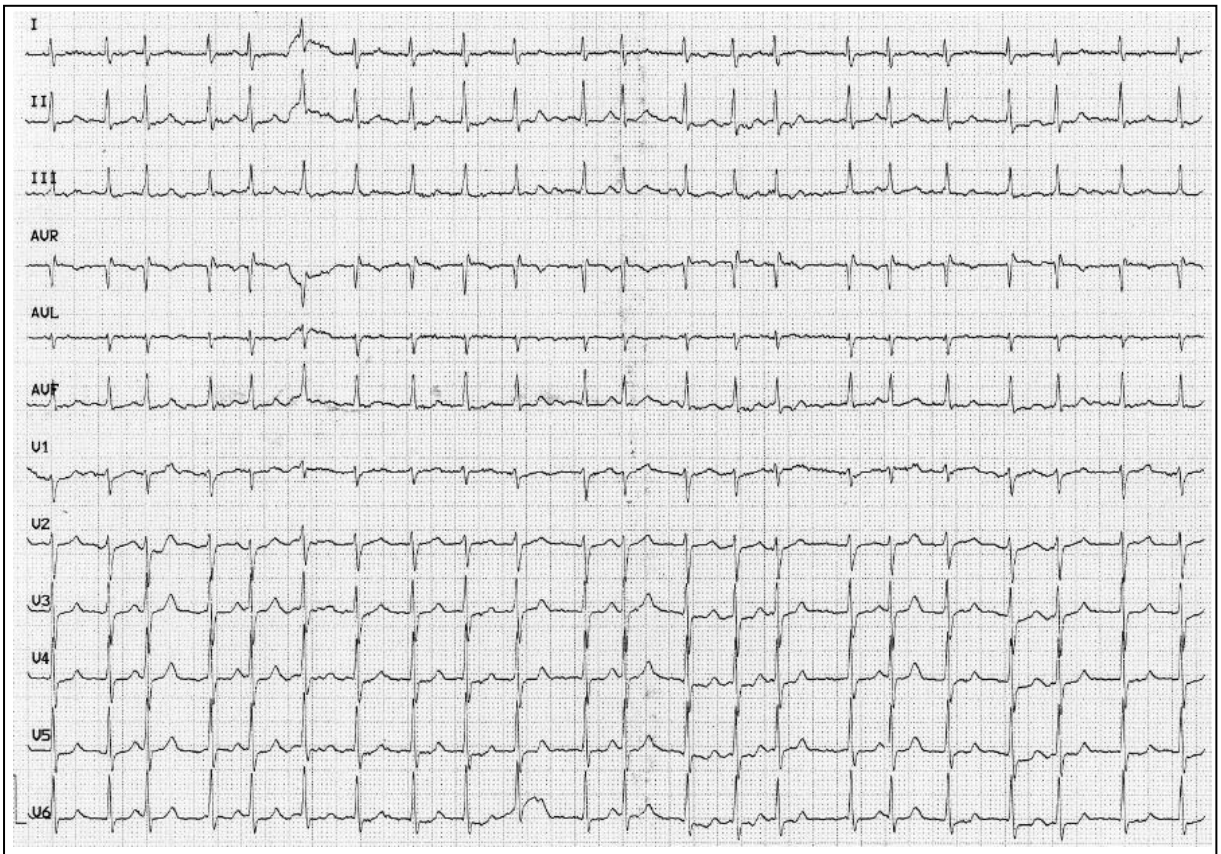


Figure 2/26.

Tachyfibrillation. Absence of the P waves and rapid ventricular response (QRS complexes) occurring at irregular intervals is observable. (Tachyfibrillation, normal QRS axis, normal ventricular conduction and repolarization.)

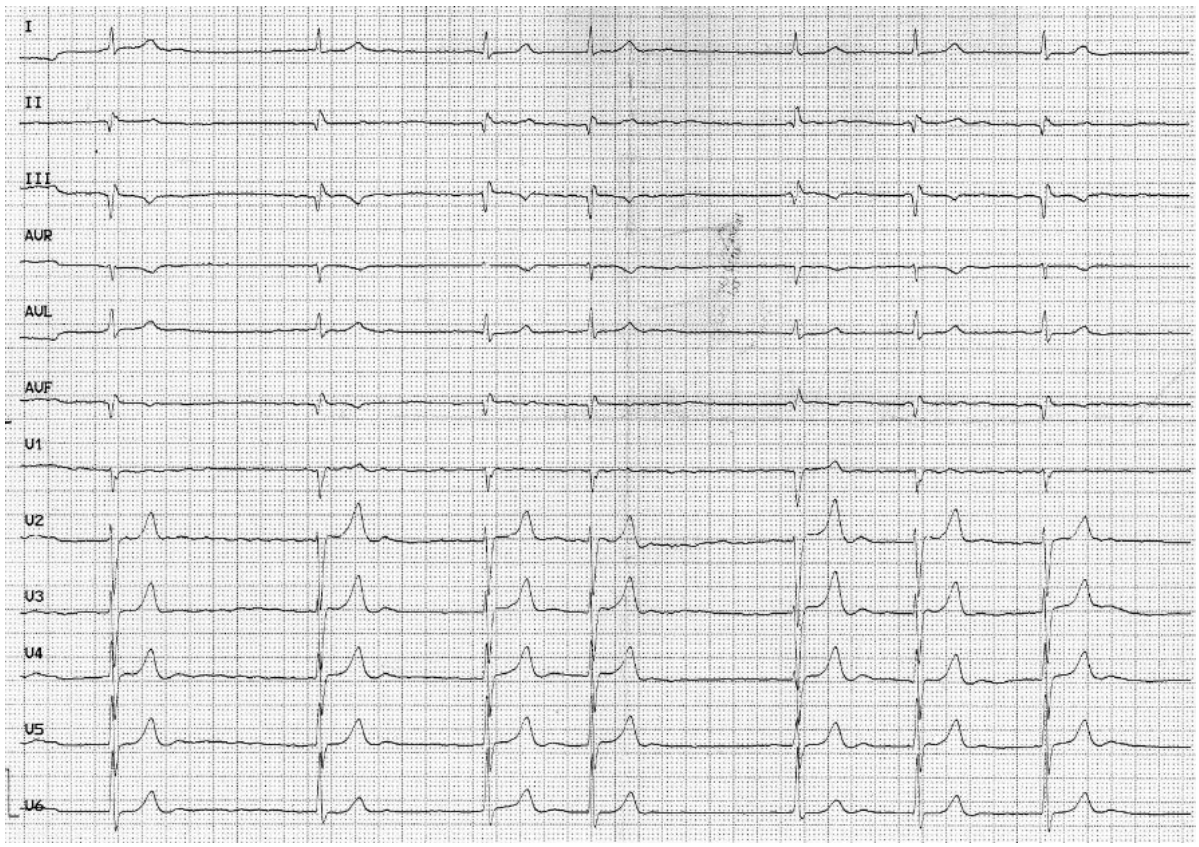


Figure 2/27.

Bradyfibrillation. P waves are missing and an irregular ventricular response (QRS complexes) at a very low rate is detectable. (Bradyfibrillation, left axis deviation, Q waves in leads II, III, aVF – ECG signs of scar after an inferior infarction, narrow QRS complexes, negative T waves in leads III, aVF, otherwise normal ventricular repolarization.)

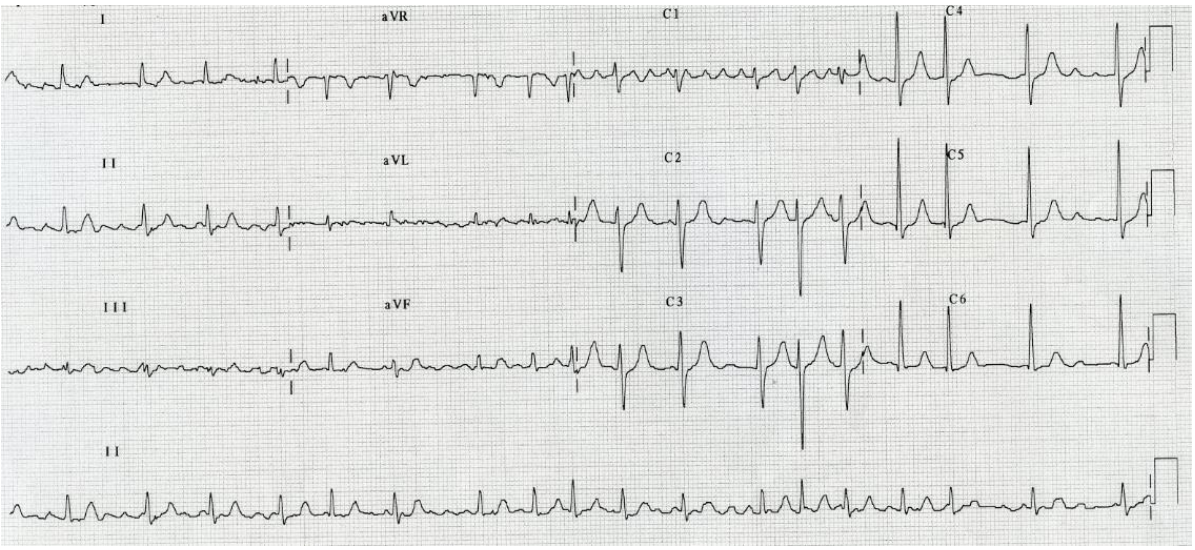


Figure 2/28.

Coarse atrial fibrillation with a fast ventricular rate. It frequently occurs that atrial activity appears to be regular in lead V1, but this is still not atrial flutter or atrial tachycardia. (Atrial fibrillation at a ventricular rate of 120 bpm, normal QRS axis, normal ventricular conduction and repolarization.)

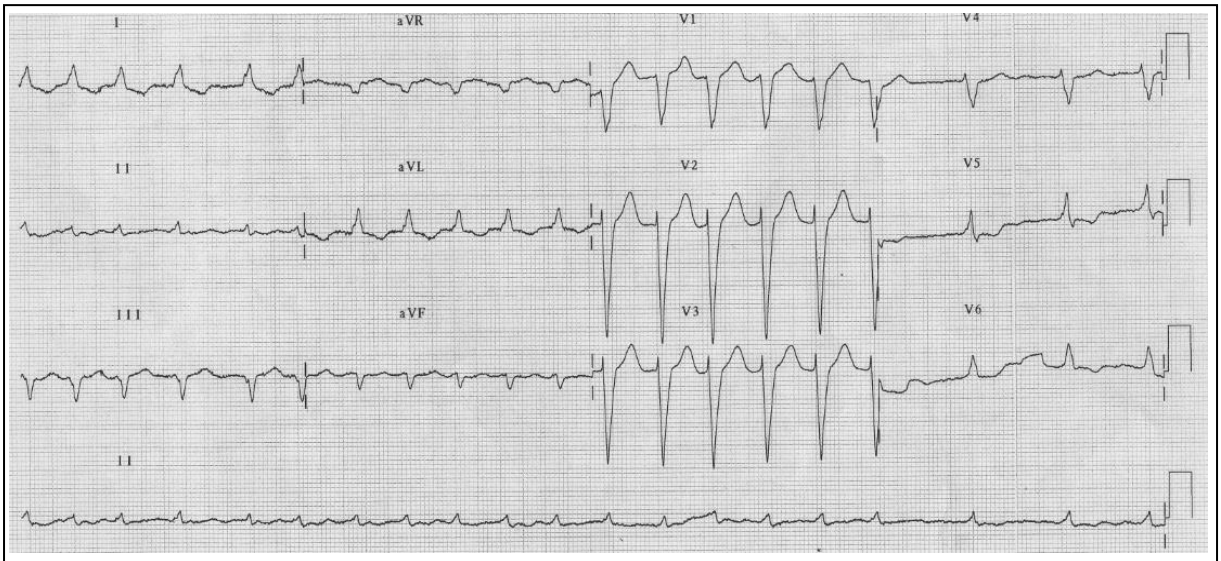


Figure 2/29.

Coexistence of atrial fibrillation and wide QRS complexes (left bundle branch block) can falsely be interpreted as ventricular tachycardia, especially at rapid ventricular rates. Take a look at the rhythm strip and the irregularity of the rhythm becomes evident immediately.

High atrial rates also result in the cessation of effective contraction of the atria, with the atria only performing a characteristic worm-like and writhing movement (atrial

trembling), due to which there is a *decrease in cardiac output by 15-20%* on one hand (which is further impaired by the tachycardia and the reduction in diastolic filling caused by irregular heart beating) and, on the other hand, the *stasis of blood flow* developing in the left atrial appendage in the absence of contractions creates the possibility of *thrombus formation*, which can be a source of *embolism* in the systemic circulation (e.g. cerebral emboli). Nearly one in four cerebral infarctions (a subtype of ischemic stroke) are due to embolism caused by atrial fibrillation. The various forms of atrial fibrillation are differentiated on the basis of its occurrance and duration, including:

- new-onset (acute): developed in < 48 hours, and it is also paroxysmal at the same time. Its distinction is warranted by the fact that usually no left atrial thrombus is forming in this period yet. In such a case, restoration of sinus rhythm may be attempted even without prior anticoagulant treatment.
- paroxysmal: it has a sudden onset and spontaneous cessation (within 7 days).
- persistent: it can only be terminated either by pharmacological or electrical cardioversion, so it does not cease spontaneously.
- long-standing persistent: this type of atrial fibrillation has been existing for >1 year, but pharmacological or electrical cardioversion may be attempted yet to restore sinus rhythm.
- permanent: it cannot be reverted into sinus rhythm, with the arrhythmia persisting for > 6-12 months and being a perpetuated form (sometimes this form is also referred to as 'chronic', but 'permanent' is the preferred term being in use because it better reflects the essence of the disease.)

- vagal AF: it usually occurs at night and at rest in middle-aged males.
- adrenergic AF: it occurs during the day and is provoked by physical exertion (this is referred to as the so-called 'lone' atrial fibrillation).

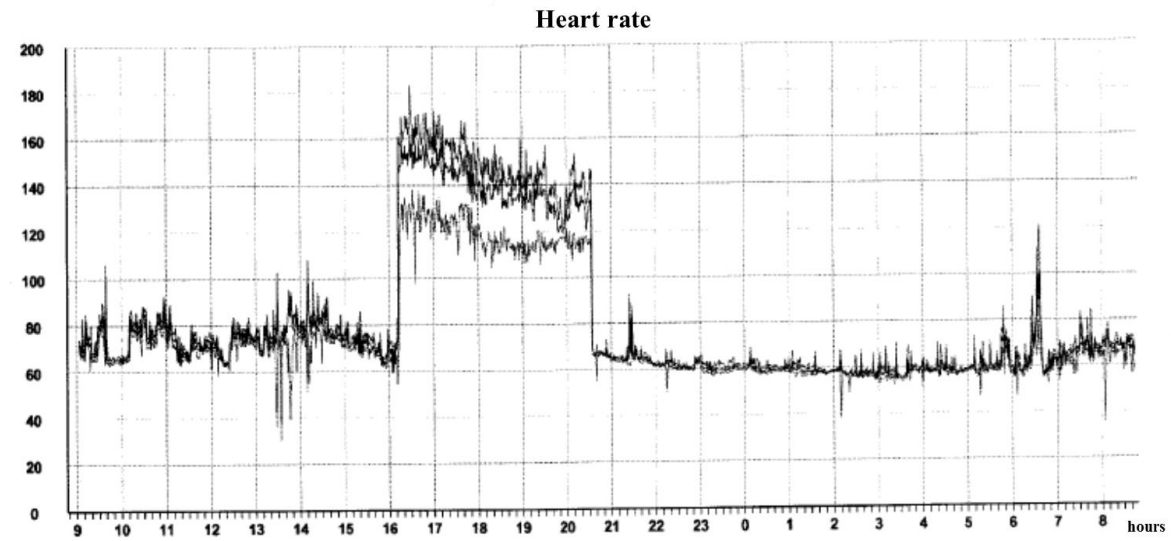


Figure 2/30.

Paroxysmal atrial fibrillation was the underlying cause of this tachycardia occurring during Holter monitoring and ceasing spontaneously.

The causes of AF include:

IHD, valve diseases (mitral stenosis!), hypertension (left ventricular hypertrophy), cardiomyopathy, hyperthyroidism (even subclinical!), COPD, sick sinus syndrome (SSS), alcohol consumption, smoking, ASD, peri-myocarditis, pulmonary embolism (PE), post cardiac or lung surgery, lone (familial?).

An important rule is that the longer an episode of atrial fibrillation persists, the more difficult it is to terminate this because the atrial musculature undergoes structural remodeling, while the cells (ion channel density) go through so-called electrical remodeling (together referred to as atrial remodeling), due to which the arrhythmia becomes self-perpetuating; in other words, *'atrial fibrillation begets atrial fibrillation'.* Pharmacological cardioversion usually proves to be unsuccessful after 1-2 weeks, however, performance of an electrical cardioversion facilitated by antiarrhythmic medications may be attempted.

It is not our objective to describe the treatment of AF in a detailed fashion. Pharmacological (class Ic or III antiarrhythmic agents) or electrical cardioversion (*rhythm-control strategy*) followed by drug treatment maintaining sinus rhythm (class II or III agents), or catheter ablation procedures (e.g. pulmonary vein isolation, which might provide sustained results) may be used for the termination of atrial fibrillation as well. Moreover, in cases of permanent AF, the intention to merely achieve a normal ventricular rate (*rate-control strategy*) provides similarly good life expectancy (with class II agents, digitalis). Good rate control is defined as a ventricular rate below 110 bpm, however, in symptomatic cases or in patients with tachycardia-induced cardiomyopathy, rather below 80 bpm. For both strategies, *anticoagulant* treatment must play an extremely important role!

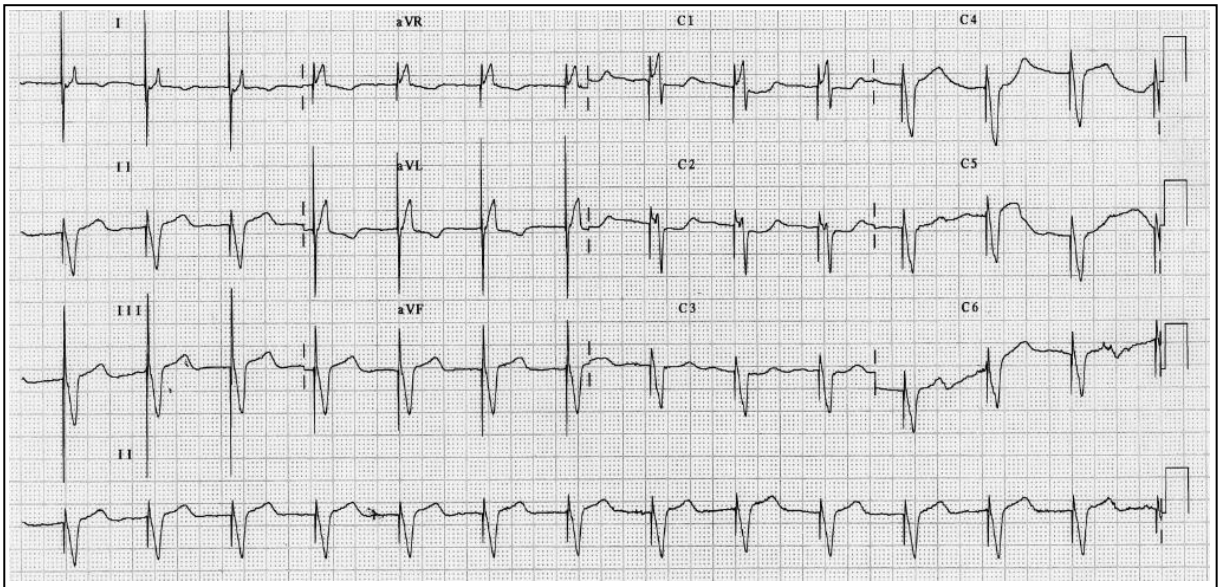


Figure 2/31. Patients may have fibrillation in the atria even along with regular ventricular rates (stimulation by a biventricular pacemaker in this case), which carries the risk of embolism likewise. Anticoagulant treatment is obligatory here as well. (Atrial fibrillation, ventricular paced rhythm at a rate of 78 bpm, secondary repolarization abnormalities.)

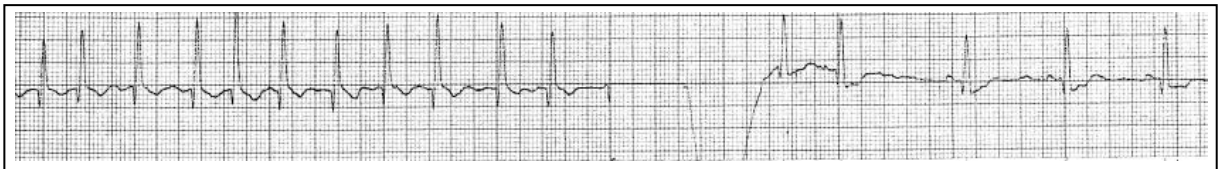


Figure 2/32.

An ECG tracing recorded during electrical cardioversion of atrial fibrillation.

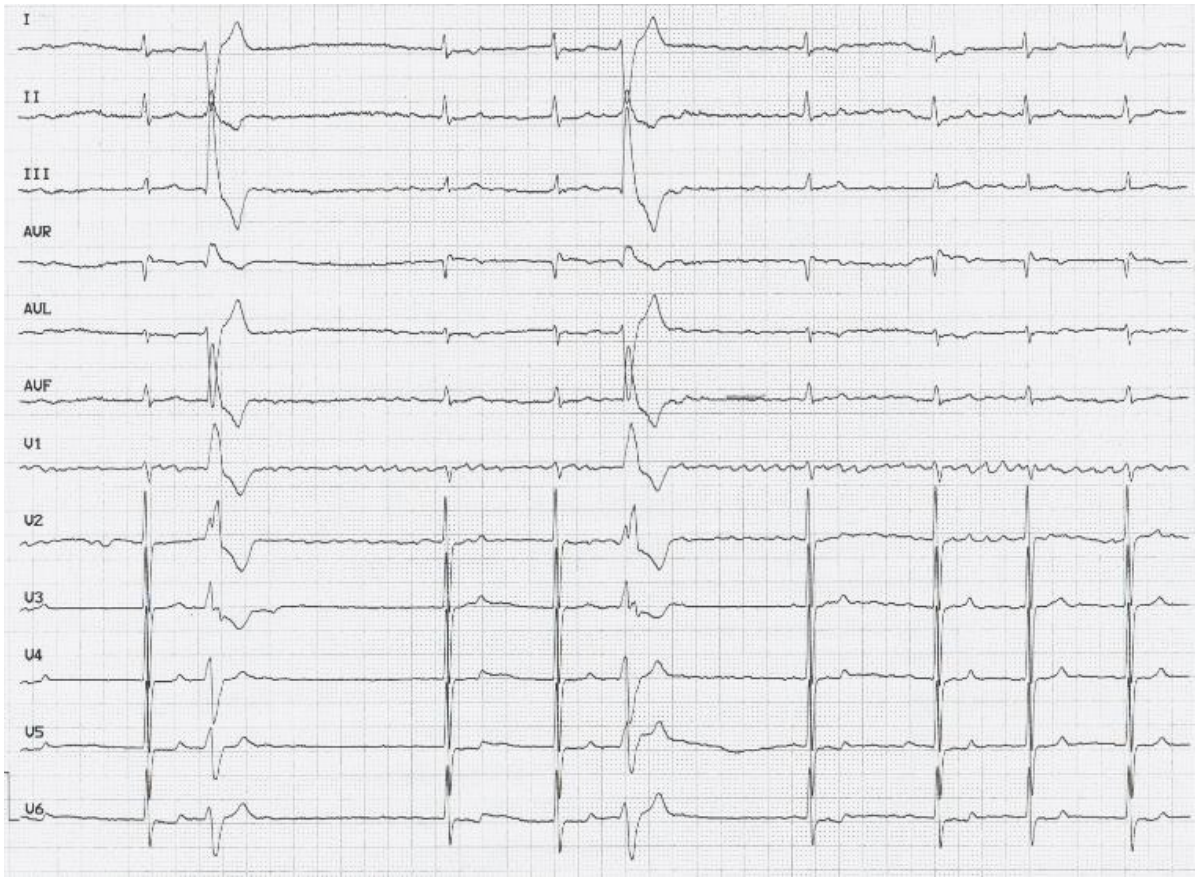


Figure 2/33.

The underlying cause of bradyfibrillation and frequent ventricular premature beats is impaired AV conduction and enhanced ectopic activity caused by digoxin overdose. (Coarse atrial fibrillation with a low ventricular rate, normal QRS axis, early transition, normal ventricular repolarization, frequent VPBs.)

In atrial fibrillation, there is only one case where regular ventricular rhythm is observable; that is in case of 3rd degree AV block.

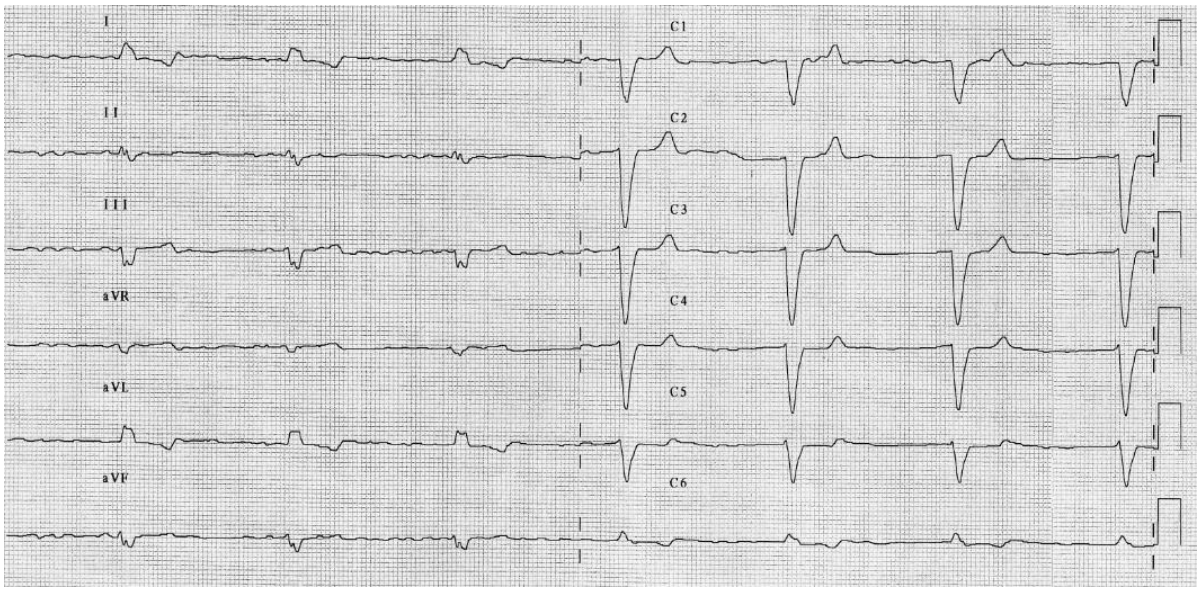


Figure 2/34.

Atrial fibrillation, 3rd degree AV block with a regular ventricular escape rhythm at a rate of 35 bpm

2.2.5. AV nodal reentrant tachycardia (AVNRT)

Knowledge of the pathophysiology of the AV node is indispensable for understanding AVNRT. It is not only the AV node, but also the adjacent structures that take part in AV conduction.

These form the triangle of Koch, which is bounded by the tendon of Todaro, posteriorly; the coronary sinus, anteriorly; and the tricuspid valve, inferiorly. The AV node is located at the tip of the triangle. The infero-posterior, middle and antero-superior portion of the triangle belongs to the posteroseptal, midseptal and anteroseptal region of the right atrium, respectively.

Inputs of the AV node:

To understand the arrhythmia, one has to learn a few characteristics of the AV junction. The AV node has a so-called double input in about 25% of people (*AV nodal dual pathway physiology*), developing during the ontogenesis. One of the inputs is situated anterolaterally and has a long effective refractory period (ERP) – this one is called the *β or fast pathway* and corresponds with the compact AV node anatomically; while the other one reaches the AV node from the posteroseptal direction and has a short ERP - this one is called the α or *slow pathway* and anatomically it is constituted by impulse-conducting structures belonging to the AV junction located posteroseptally from the AV node. Of the two pathways, only the fast pathway has a role normally, because it is through this pathway that the atrial impulse reaches the AV node, with no conduction through the slow pathway in this case.

However, the dual AV nodal pathway creates the possibility for the development of a reentrant tachycardia involving the AV node and the surrounding two pathways, which is referred to as AVNRT.

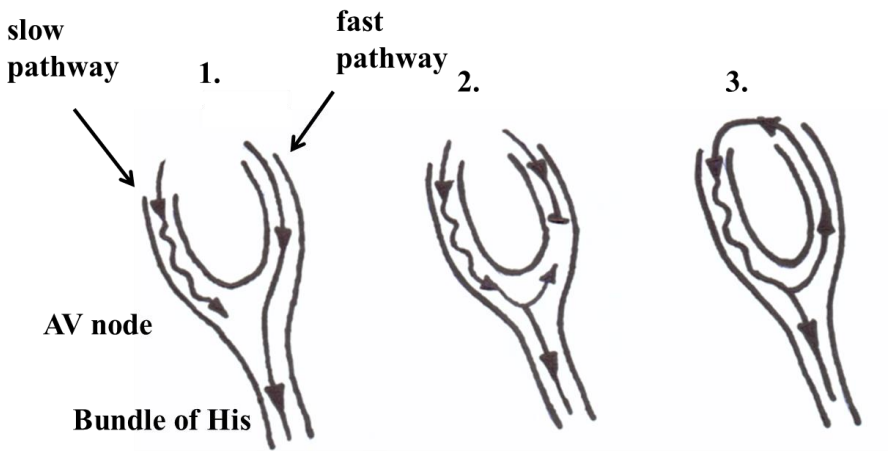


Figure 2/35.

AVNRT (slow-fast). 1. During normal AV conduction, the impulse is conducted into the AV junction through the fast pathway. 2. If the fast pathway is blocked, the impulse is conducted through the slow pathway. 3. The impulse conducted through the slow pathway is then turning back to the fast pathway.

Forms of AVNRT:
- slow-fast or typical (90%): the antegrade limb of the reentrant circuit is the slow pathway, and the retrograde one is the fast pathway
- fast-slow – atypical (10%): the antegrade limb of the reentrant circuit is the fast pathway, and the retrograde one is the slow pathway
- slow-slow – atypical (1%): there are two slow pathways conducting in both directions

Initiation of an AVNRT:

- Typical `slow-fast` AVNRT: a PAC finds the fast pathway, due to its long refractoriness, in an unexcitable stage and the impulse is therefore conducted through the slow pathway. During the period of slow conduction, the fast pathway repolarizes (becomes able to conduct impulses) and the impulse conducted through the slow pathway is jumping onto the fast pathway through the AV node, from where it is conducted further in a retrograde fashion, then the impulse is again jumping onto the slow pathway, thereby initiating the circus movement.
- Atypical `fast-slow` AVNRT: the circus movement is initiated by a VPB, which is conducted in a retrograde fashion through the slow pathway to the atria, then in an antegrade fashion through the fast pathway to the ventricles.

Thus, reentry develops in the perinodal tissues. The significance of AVNRT is that it is the most common type of PSVT, accounting for 55% (!) of cases (prevalence: 0.2%) and mainly occurring in females aged between 30 and 40 years.

ECG signs of AVNRT:

- *Regular, narrow QRS complex tachycardia* at a ventricular rate of 140-220 bpm, with *no P waves visible* before the QRS complexes.
- 'slow-fast' form: Due to simultaneous activation of the atria and ventricles, P waves are hidden in the QRS complexes since the two atria are activated nearly at the same time, and the retrograde P waves (i.e. blending into the end of the QRS complex) will therefore become narrow and peaked ('pseudo S waves' in leads II, III and aVF, r' waves in lead V1).

  Typically, the P wave is situated within the QRS complex in 56% of cases, moreover, at the end of the QRS in 36%, after the QRS in 4% and at the beginning or before the QRS in 2% of cases, respectively.

- 'Fast-slow' form: retrograde (i.e. negative in the inferior leads) P waves are visible well after the QRS complex, RP>PR (long RP tachycardia.)
- 'Slow-slow' from: retrograde P waves are visible in the ST segment (often halfway between the two QRS complexes) or RP<PR (very similar to orthodromic AVRT); it must be distinguished from permanent junctional reciprocating tachycardia (PJRT, Coumel's tachycardia) and atrial tachycardia, which are also long RP tachycardias.
- An AV block observed during the tachycardia makes the diagnosis of AVNRT unlikely, with the exception of the extremely rare infra-Hisian block, which does not terminate the arrhythmia. In such a case, the number of P waves exceeds that of the QRS complexes.

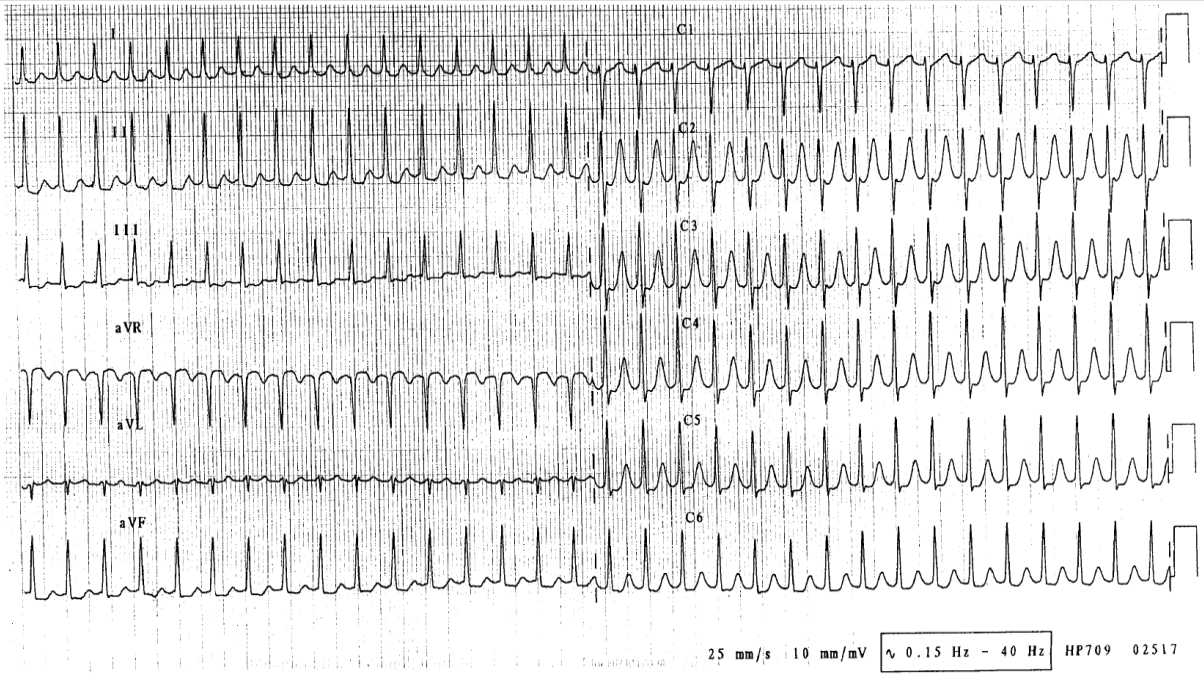


Figure 2/36. Typical slow-fast AVNRT. No unequivocal P waves can be differentiated (the tiny positive deflection at the end of the QRS complexes in lead V2 may be a P' wave conducted to the atria in a retrograde fashion), and the tachycardia is perfectly regular at a ventricular rate of 166 bpm. The ST segment depression observed occasionally is merely the consequence of tachycardia, and not a sign of coronary artery disease. (AVNRT at a ventricular rate of 166 bpm, normal QRS axis, normal ventricular conduction, secondary ST segment depression of 1-1.5 mm can be seen diffusely.)

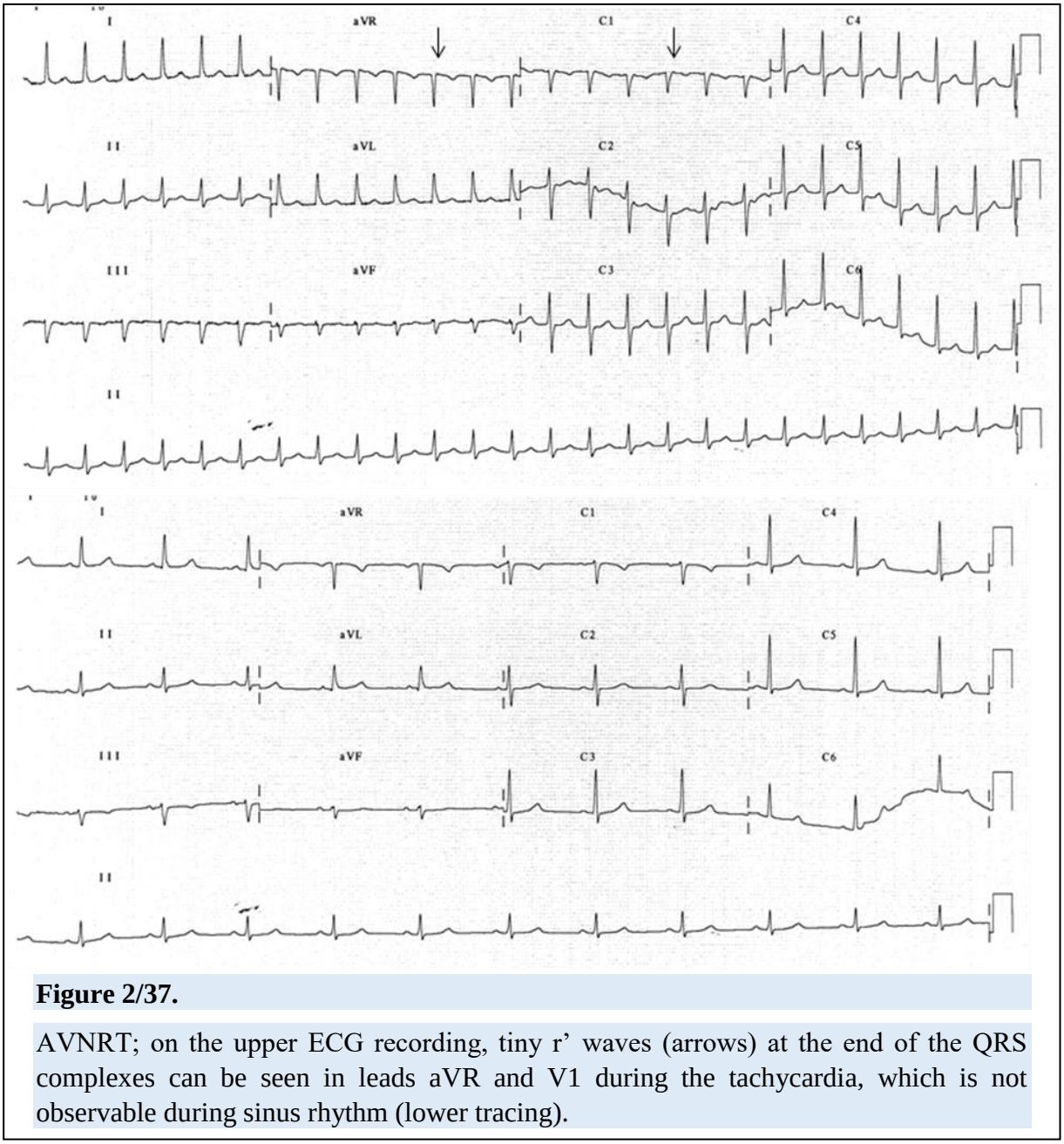


Figure 2/37.

AVNRT; on the upper ECG recording, tiny r' waves (arrows) at the end of the QRS complexes can be seen in leads aVR and V1 during the tachycardia, which is not observable during sinus rhythm (lower tracing).

Regarding its treatment, an acute attack should first be attempted to be terminated by vagal maneuvers, e.g. Valsalva maneuver, carotid sinus massage (CSM), applying cold water on the face, then, with regard to medications, iv. adenosine or verapamil may be given. Vagal maneuvers may be useful for distinguishing AVNRT from other types of PSVT, because atrial fibrillation or atrial flutter is not terminated after the use of vagal maneuvers, only the ratio of the conduction block will increase and there will be slowing of the ventricular rate. The treatment of AVNRT also involves a definitive solution, during which ablation of the slow pathway is performed with the use of radio frequency (RF) energy. The arrhythmia never recurs again in 90-95% of cases afterwards

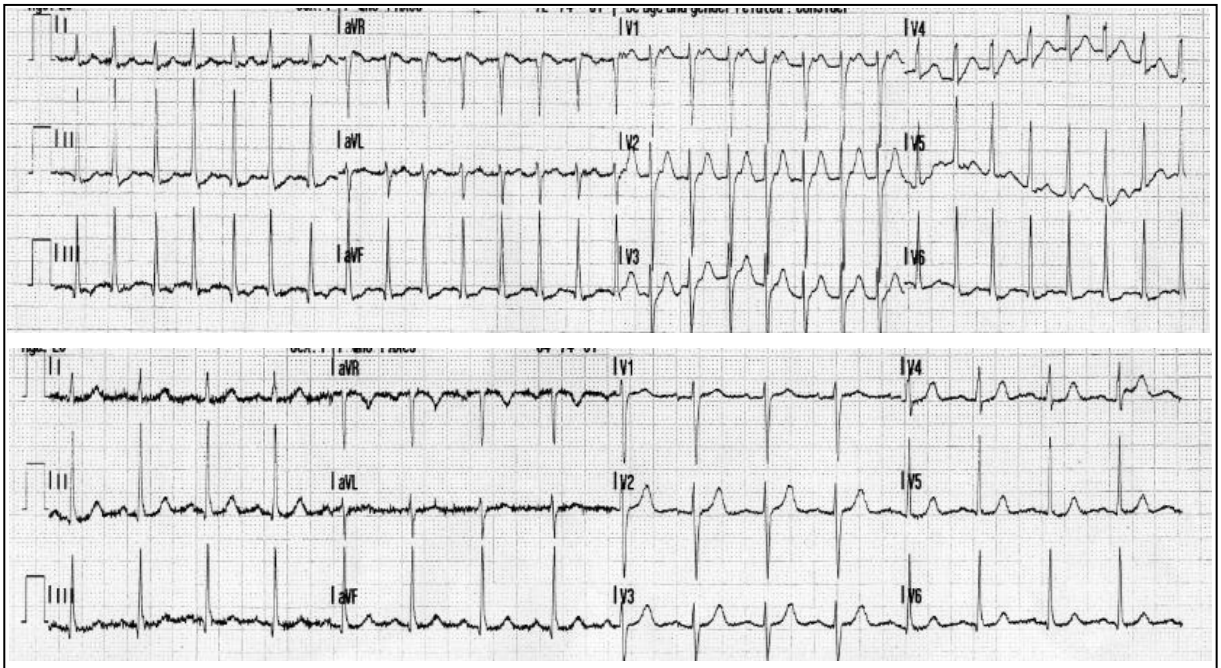


Figure 2/38.

AVNRT; on the upper ECG recording, retrograde P waves occurring after the end of the QRS complexes are visible in lead V1 during the tachycardia and, on the lower tracing, no delta waves are observable during sinus rhythm, so the presence of an AVNRT is more likely.

2.2.6. Junctional tachycardia

As a reminder, we refer back to the intrinsic rate of the AV junction, which is 40-60 bpm. A ventricular rate of 60-100 bpm is referred to as accelerated junctional rhythm, while a rate above this is called junctional tachycardia. Thus, the ventricular rate is 100-200 bpm, with the *arrhythmia being regular normally and retrograde P waves* (i.e. negative in leads II, III, aVF) are detectable before, or more rarely, blending into, the QRS complexes.

Its forms include the following:

- non-paroxysmal junctional ectopic tachycardia: it frequently occurs in children, may induce tachycardiomyopathy (100-250 bpm) and is often intractable;
- non-paroxysmal AVJT: abnormal automaticity, 80-130 bpm, causes: hypokalemia, digitalis, ischemia;
- PJRT (Coumel): RP > PR (a special type of long RP orthodromic AVRT).

Differential diagnosis of narrow QRS complex tachycardias.

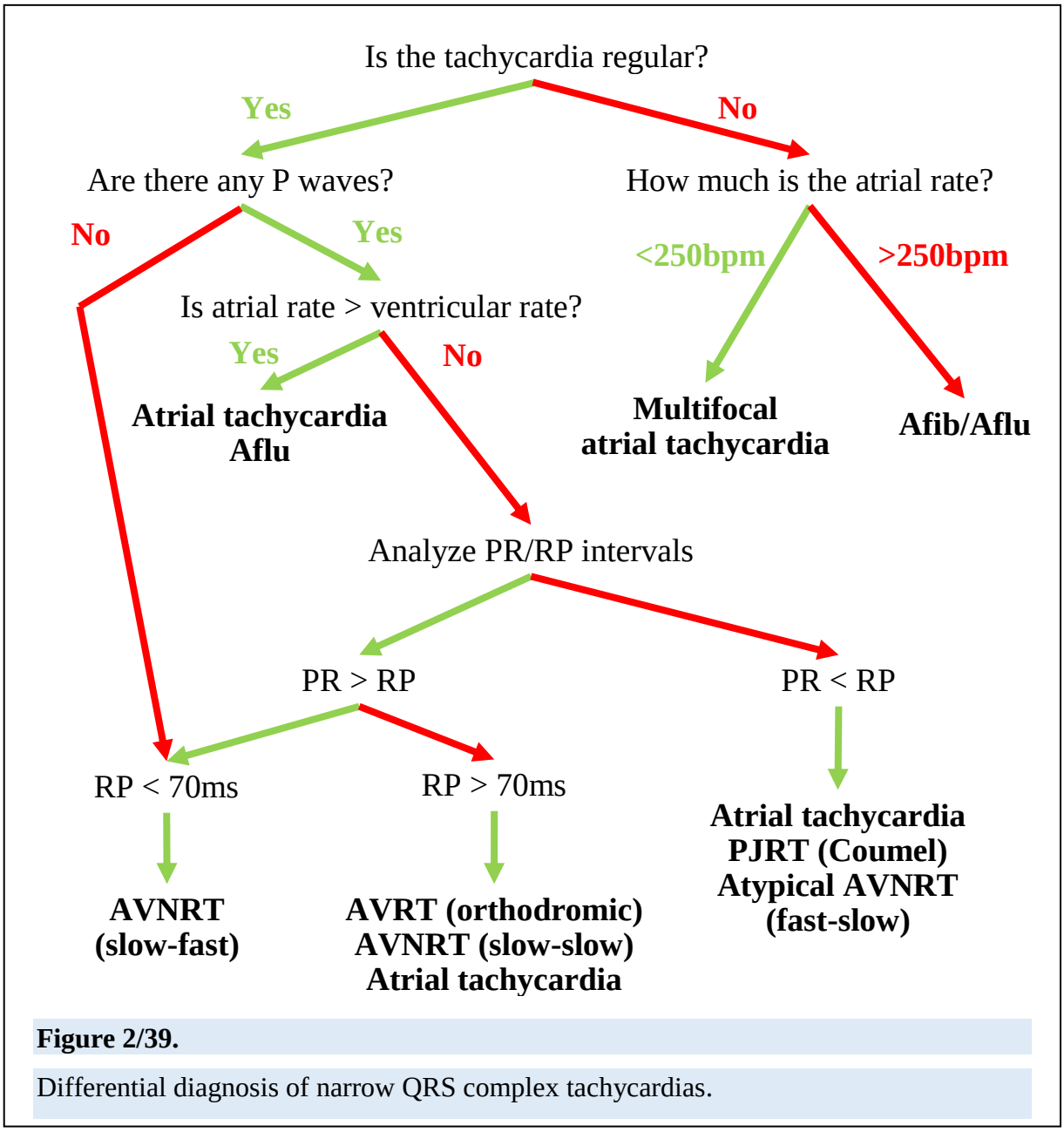


Figure 2/39.

Differential diagnosis of narrow QRS complex tachycardias.

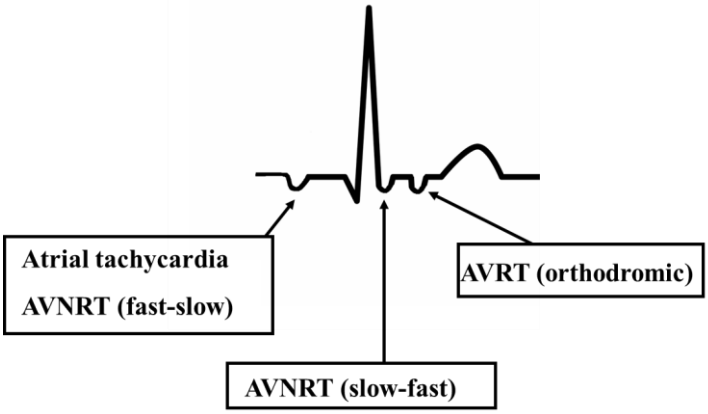


Figure 2/40.

Different regular narrow QRS complex tachycardias can be differentiated based on location of the retrograde P waves.

In SVT, it is important to analyze the presence or absence of P waves; that is if no P waves are visible and the rhythm is irregular, one should primarily think of the presence of atrial fibrillation ('f' waves) or atrial flutter with variable AV conduction ('F' waves). It may happen that waves resembling P waves are visible in certain leads with regular RR intervals, at this time, determination of the atrial rate may be a help: if that is >250 bpm, it is atrial flutter, if <250 bpm, it is atrial tachycardia. If there are visible P waves, but their morphology is variable and the RR intervals are irregular, it is multifocal atrial tachycardia. It may help distinguish orthodromic AVRT (in WPW syndrome) from AVNRT if you determine the relationship of P waves with the preceding and consecutive R waves, that is to compare PR and RP intervals. In orthodromic AVRT with a rapid heart rate, sometimes one might see QRS alternans (a beat-to-beat variation in the amplitude of QRS complexes), which is considered characteristic for AVRT (although it may occur in AVNRT).

There are two important tools available yet for the differential diagnosis of SVTs: carotid sinus massage (CSM) and adenosine test.

In response to CSM, different SVTs may remain unchanged, but the typical response to the procedure is as follows:

Sinus tachycardia shows a gradual slowing.

AVNRT and orthodromic AVRT suddenly converts back to sinus rhythm.

In atrial tachycardia, Afib and Aflu, there is a decrease in ventricular rate, with an increase in the conduction block ratio (less P waves are conducted to the ventricles).

After administration of ADENOSINE (6-12 mg iv. bolus):

If there is no change, it raises suspicion of an inappropriate dose or that the arrhythmia has a ventricular origin after all.

If there is gradual slowing of the arrhythmia, one should think of sinus tachycardia or atrial tachycardia.

If there is sudden termination of the arrhythmia, it may be AVNRT or AVRT, but also atrial tachycardia.

If supraventricular impulse formation remains unchanged and the arrhythmia persists, but there is an increase in the conduction block ratio (i.e. the ventricular rate decreases), it may be both Afib, Aflu and atrial tachycardia.

FACTS THAT YOU MUST KNOW:

1. A QRS complex presenting earlier than the expected sinus beat is the consequence of a premature beat, while that presenting later results from an escape beat.
2. For a regular narrow QRS complex tachycardia, one should search for retrograde P' waves in the limb leads and lead V1. If they cause a deformation in the end of the QRS complexes (and they are not there in sinus rhythm) or one cannot see retrograde P' waves, this is likely due to the fact because they have blended into the QRS complexes. In both cases, we are dealing with AVNRT. If retrograde P' waves are distinct from QRS complexes, we are likely to be dealing with AVRT or atrial tachycardia (AT). If possible, one should have a look at the ECG during sinus rhythm and search for the signs of WPW syndrome.
3. If you find the rhythm to be irregular and there are no consequently occurring P waves, you should always consider the presence of atrial fibrillation even if the QRS complexes are wide, since a conduction abnormality is also present in the latter case.
4. Atrial flutter may have a regular or irregular appearance depending on the conduction block ratio, however, it is clearly distinguishable from atrial tachycardia or atrial fibrillation based on the characteristic 'saw-tooth' pattern of the baseline and the absence of an isoelectric line.
5. The administration of adenosine may help make distinctions in the differential diagnosis of tachycardias either involving (AVNRT, AVRT) or just passively involving (AT, Aflu, Afib) the AV node, because the medication terminates the former arrhythmias, while for the latter arrhythmias, QRS complexes temporarily disappear due to the AV block and the underlying arrhythmia becomes apparent, but it is not terminated.

2.3. Active heterotopic ventricular impulse formation

2.3.1. Ventricular premature beats

The general description of premature beats as well as an overview on the nomenclature related to their appearance has already been made for supraventricular premature beats. Let's now review the characteristics of ventricular premature beats (VPBs). A VPB is an ectopic beat originating from (ventricular) structures below the bundle of His and *presenting earlier than expected*. VPBs are not preceded by P waves or, if they do occur, there is no relationship between the P waves and QRS complexes or the P waves rather present after the VPBs due to the retrograde AV conduction. Ventricular premature beats generally show full compensation with a constant coupling interval. Since the impulse is not conducted through the normal conduction system, therefore the QRS complex becomes wide. The reason for widening of the QRS complex is that conduction velocity in the cardiac conduction system significantly exceeds the conduction velocity between cardiac muscle cells. *The impulses conducted through the myocardium result in wide premature beats with a bizarre morphology, for which repolarization also becomes discordant.* The latter term means that the repolarization abnormality has an opposite direction compared to the dominant deflection of the QRS complex, that is ST segment depression and negative T waves are visible for a dominantly positive deflection of the QRS complex, while ST segment elevation and positive T waves are detectable in case of a negative deflection. *The ST segment and T wave abnormalities are not indicative of ischemia or other abnormalities, they are merely secondary changes.* The explanation for secondary ST segment and T wave abnormalities should be sought in the alteration of the repolarization sequence caused by a change in the depolarization sequence.

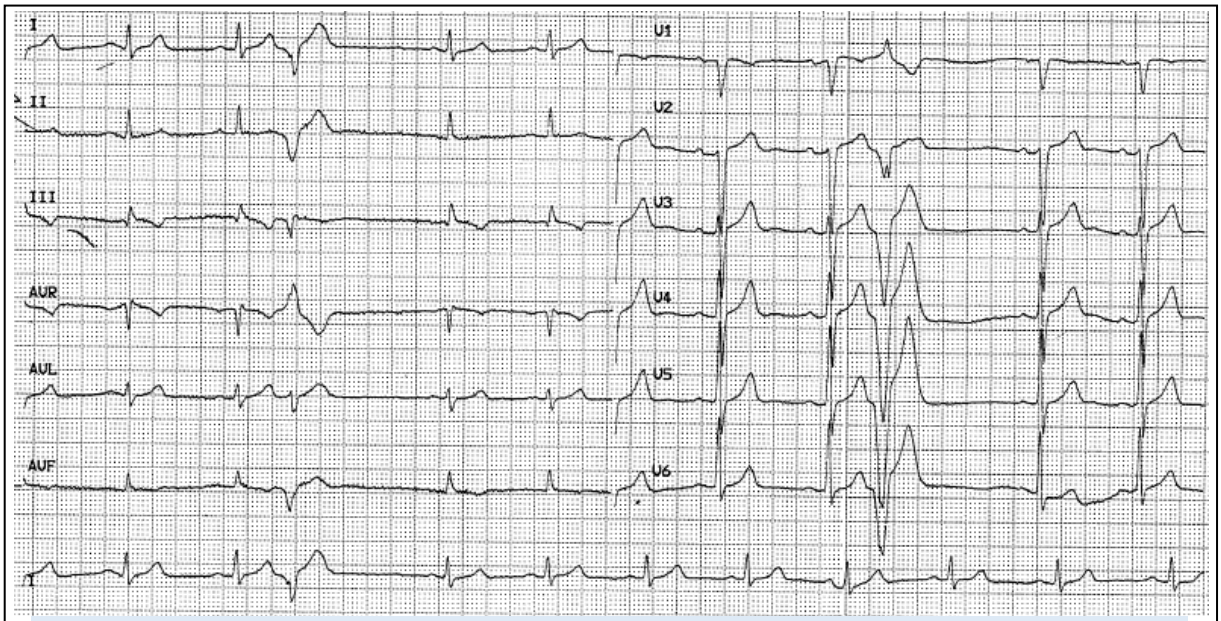


Figure 2/41. VPB. A ventricular premature beat is a beat presenting earlier than expected, which has a wide QRS complex resulting in secondary ST segment abnormalities, which are not the signs of ischemia (e.g. ST segment elevation in leads I, II, V2-6, downsloping ST segment depression and negative T waves in lead aVR – these are non-pathological). (Sinus rhythm at a normal heart rate, normal QRS axis, normal AV conduction time, normal ventricular conduction, trivial ST segment depression and negative T waves in lead III and aVF, otherwise normal ventricular repolarization.)

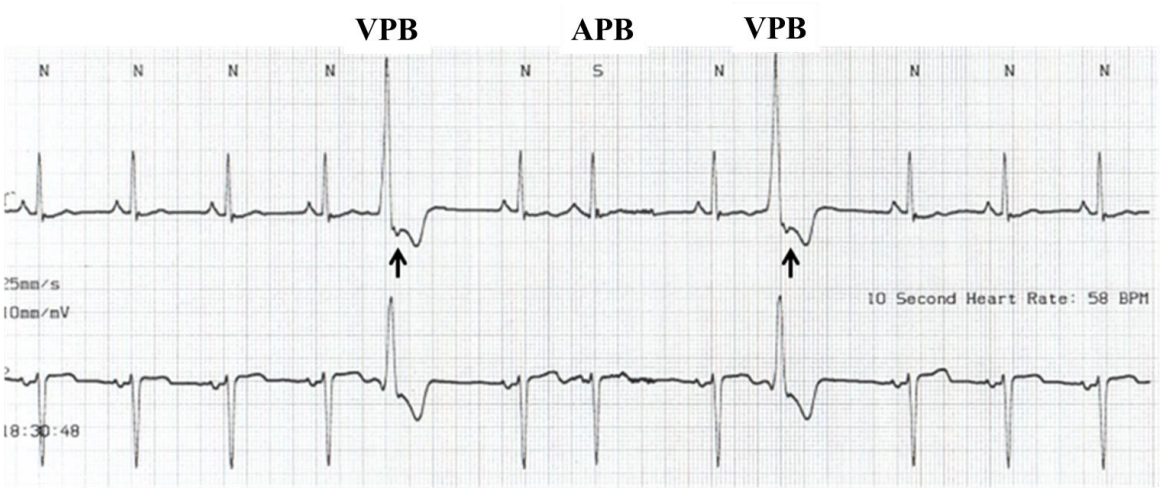


Figure 2/42.

VPB and APB. Please note the retrograde P waves (arrows) occurring on the ST segment after the QRS complex of the ventricular premature beat. Between the sinus beats presenting between the two VPBs, an APB also occurs. Please note the morphological difference between the sinus P waves, retrograde P waves as well as the P wave preceding the APB.

Ventricular premature beats can be classified based on several aspects; e.g. site of origin or characteristics of their appearance. The site of origin may be anywhere in the left and right ventricular myocardium. A VPB arising from the peri-infarct zone may be a sign of residual ischemia and may also have a prognostic value regarding the occurrance of malignant ventricular arrhythmias. If VPBs are classified on the basis of their appearance, they may have a bigeminal, trigeminal or quadrigeminal pattern. The term *bigeminy* is used when every second beat is a ventricular premature beat; in trigeminy, two sinus beats are consecutively followed by a VPB, while in quadrigeminy, it is always after three sinus beats that a premature beat occurs.

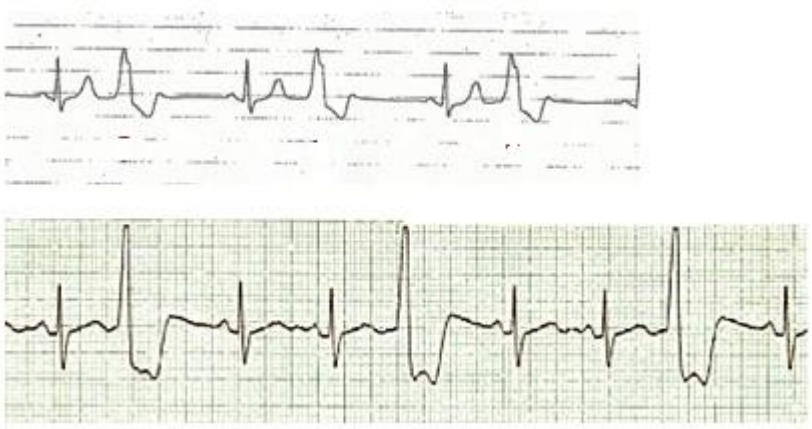


Figure 2/43.

Ventricular premature beats (VPBs) in a bigeminal (above) and trigeminal (below) pattern.

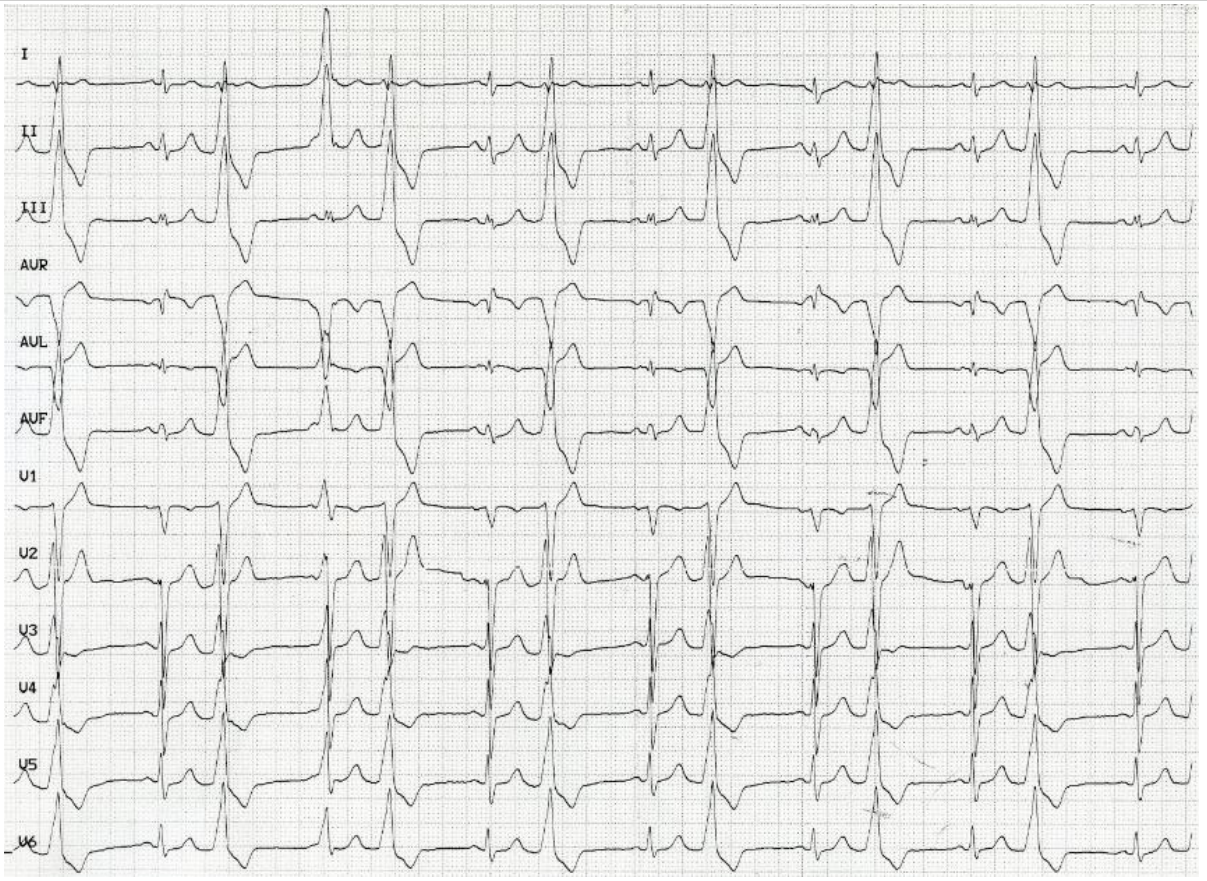


Figure 2/44. Ventricular premature beats with a bigeminal pattern. Every second beat comes early and has a wide QRS complex, i.e. it is a VPB (the 4th beat is the result of fusion of beats). (Sinus rhythm, normal QRS axis, normal AV conduction time, normal ventricular conduction and repolarization, ventricular premature beats in a bigeminal pattern.)

Long RR intervals are in favour of the development of premature beats, so it often occurs that ventricular premature beats in a bigeminal pattern are observable in a sustained fashion, because the compensatory pause of premature beats favours the development of a next premature beat. The phenomenon described above is called the rule of bigeminy.

It is also due to this that it is difficult to influence bigeminy with medications, since the most commonly used beta blocker treatment, due to lowering of the sinus rate, may potentially even favour the development of bigeminy. If the patient perceives the presentation of bigeminy and complaints occur, he/she would rather be encouraged to actively perform physical exertion (squatting, walking at a faster pace) at the time of the arrhythmia, because this may lead to the cessation of bigeminy by an increase in the sinus rate.

Based on their appearance, premature beats can be divided into further morphological groups. If they arise from a single focus, one may refer to them as *monomorphic* (unifocal) VPBs; if they arise from several focuses, they are called *polymorphic* (multifocal) VPBs. If a VPB has a short coupling interval, it may happen that its occurrance coincides with the top or downstroke of the T wave of the sinus beat. This is the so-called *R-on-T phenomenon*, which has a considerable importance in case of an acute myocardial infarction, because an extra impulse occurring at a vulnerable period of repolarization (supernormal phase) may easily result in the development of ventricular fibrillation (malignant VPB).

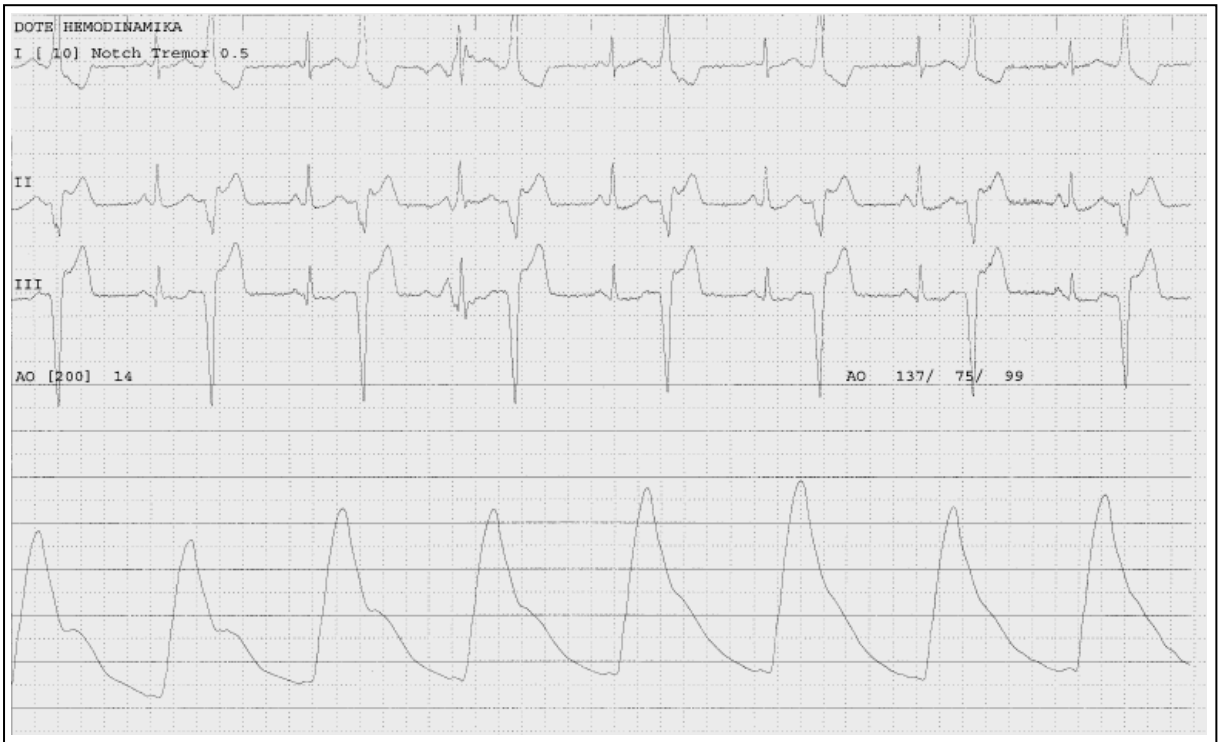


Figure 2/45. Hemodynamic consequences of bigeminy. One might see that, based on the ECG recording, the heart rate is 86 bpm, but that of 43 bpm is obtained from the ventricular pressure tracing and the counted pulse rate, because the ventricular premature beats, resulting in ineffective contractions, hardly cause any changes in the ventricular pressure (they only appear on the downstroke of the LV pressure tracing as a small notching). This is the reason for the fact that in bigeminy the patient or the doctor sometimes observes severe bradycardia on palpation of the pulse, however, this is not accompanied by signs of organ hypoperfusion, e.g. dizziness.

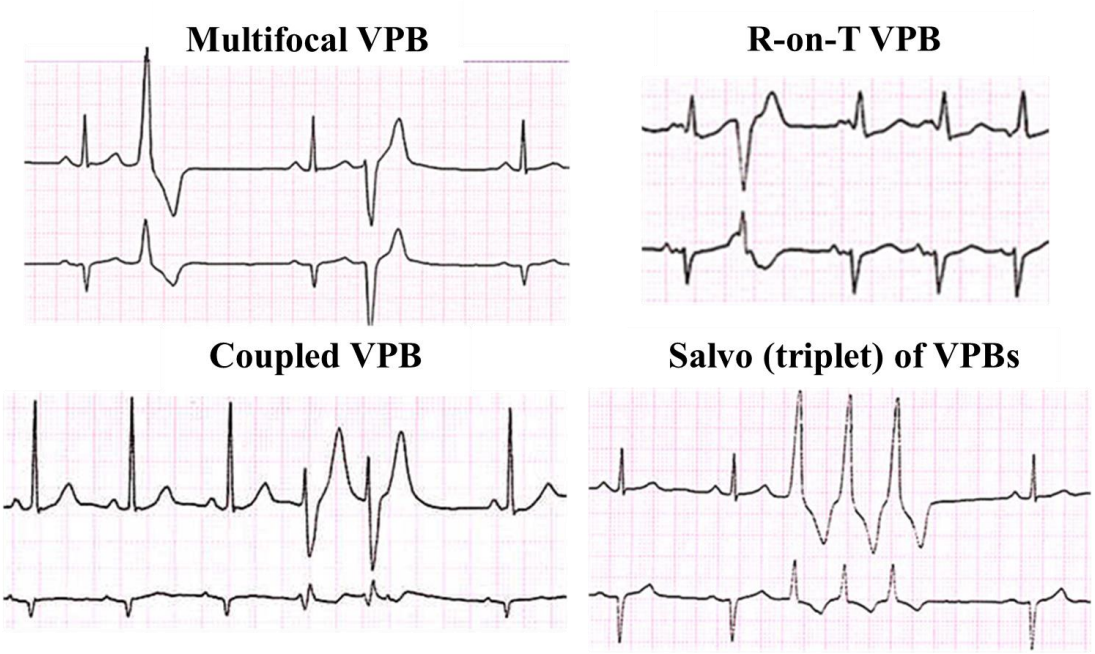


Figure 2/46. Various forms of appearance of ventricular premature beats (VPBs.)

However, the prognostic importance of R-on-T phenomenon without the context of myocardial infarction is doubtful. Complex ventricular premature beats are defined as consecutive ventricular impulses presenting in a repetitive manner. In such a case, they are called coupled beats or ventricular *couplet*s. If 3-5 consecutive premature beats occur, it is referred to as a run or '*salvos*' of VPBs.

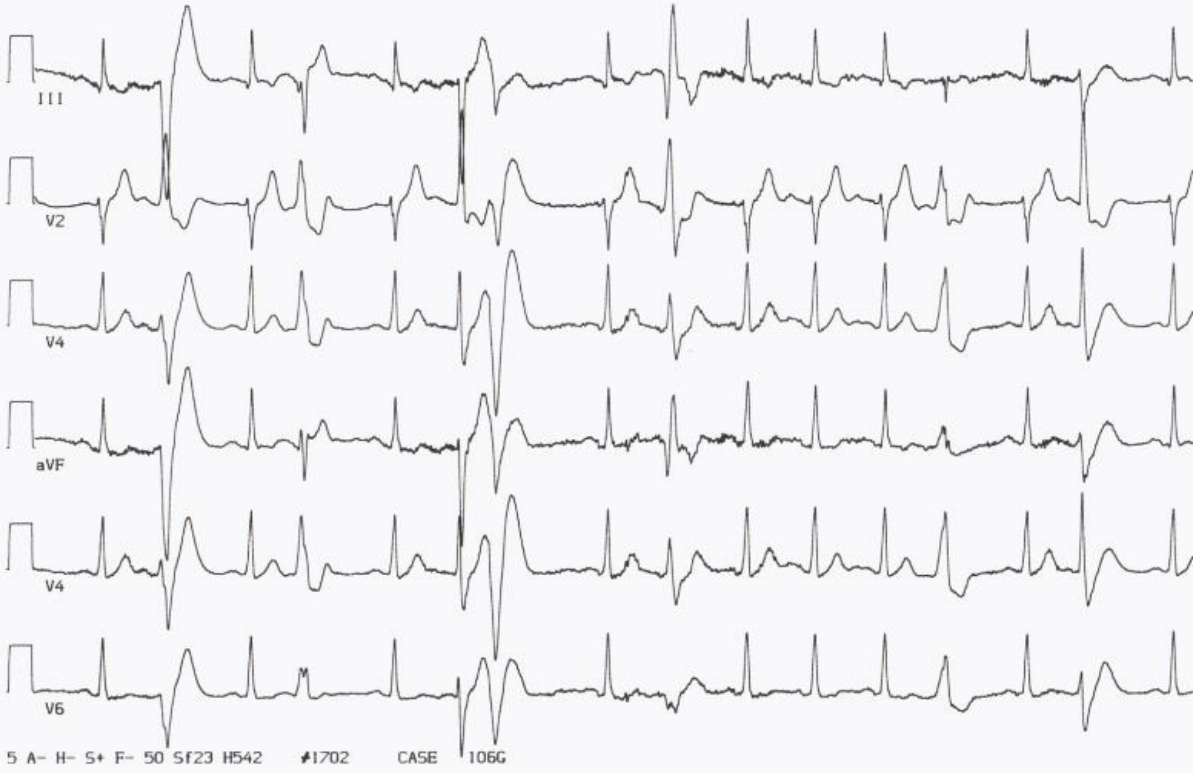


Figure 2/47.

Frequent polymorphic ventricular premature beats and coupled VPBs. The basic rhythm is atrial fibrillation (among the beats with a wide QRS complex, ones with aberrant conduction may also occur, e.g. the 9th and perhaps the 15th beat).

Ventricular premature beats (VPBs) may arise from several regions of the left or right ventricle. Based on the site of origin, premature beats originating from the outflow tract, interventricular septum and free walls of the ventricles can be distinguished. Their distinction may be of importance due to the reason that VPBs arising from the free wall of the left ventricle or the interventricular septum often develop due to ischemia, while those arising from the outflow tract frequently occur even without the presence of ischemia. Ventricular premature beats originating in the outflow tract have a significance only if they account for more than 15-20% of total daily beats (Holter monitoring), because frequently occurring premature beats may cause tachycardia-induced cardiomyopathy. In the distinction, QRS axis of the premature beat as well as the bundle branch block pattern provide help. A positive deflection in leads II, III and aVF is indicative of an origin in the outflow tract (RVOT, LVOT), while a negative or biphasic (i.e. equally positive and negative) deflection implies a septal origin or that from the free wall. An RBBB pattern suggests left ventricular origin, while an LBBB pattern implies right ventricular or septal origin.

In case if 6 or more consecutive and rapid ventricular beats, the phenomenon is called *ventricular tachycardia* (see that section for details). The clinical significance of the

previously used Lown Grading System for VPBs is questionable nowadays, so its knowledge is not absolutely necessary. However, one should be aware of the fact that *ventricular premature beats may occur both in individuals with a healthy heart or in those with a cardiac disease*, therefore, from treatment aspects, it is very important to take the underlying structural cardiac abnormalities into consideration (role of echocardiography!). A few thousands of VPBs should not necessarily be treated in subjects with a healthy heart, however, e.g. in dilated cardiomyopathy or after a myocardial infarction, they may be predictors of malignant ventricular arrhythmias. If the presence of no structural heart disease can be confirmed, it is important to look for the cause of ventricular premature beats, which might be a disturbance of electrolyte balance (potassium or magnesium deficiency), digitalis overdose, excessive alcohol consumption, caffeine intake or smoking. Ventricular premature beats occurring in subjects with a structurally normal heart and being asymptomatic should not be treated. For differential diagnosis, it is important to mention ventricular parasystole and supraventricular premature beats with aberrant ventricular conduction.

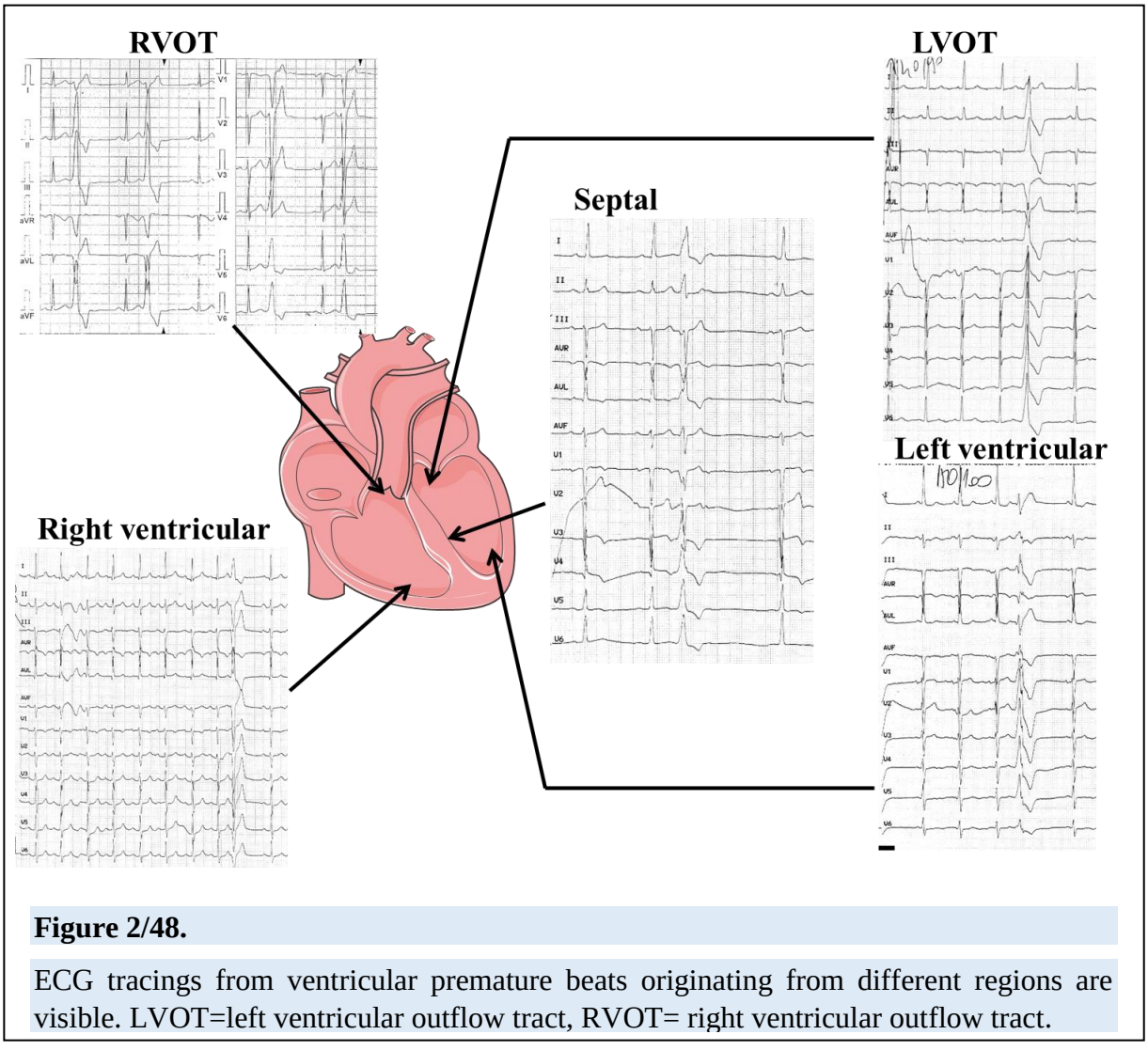


Figure 2/48.

ECG tracings from ventricular premature beats originating from different regions are visible. LVOT=left ventricular outflow tract, RVOT= right ventricular outflow tract.

2.3.2. Ventricular parasystole

This phenomenon is a type of active heterotopic ventricular impulse formation belonging to the group of pararrhythmias, in which two centers with pacemaker activity are working independent of each other; one of them is the sinus node (beats with a narrow QRS complex) and the other one is the ventricular parasystolic focus (beats with a wide QRS complex). The rate of the SA node is higher than that of the parasystolic focus, so the latter one only prevails if the ventricles are not in the refractory period.

ECG signs include:

a. variable coupling interval (which is fixed for VPBs);

b. fixed interectopic intervals (it may also be fixed for VPBs; just think about bi- or trigeminy) and;

c. the presence of frequent fusion beats (e.g. the basal portion of the ventricles is activated by the sinus impulse, while the apical portion by the parasystolic focus).

Parasystole does not require treatment because the parasystolic focus is often refractory to drug treatment and suppression of the impulse formation in the SA node may result in more frequent operation of the parasystolic focus, if, for example, beta adrenergic receptor antagonists are administered.

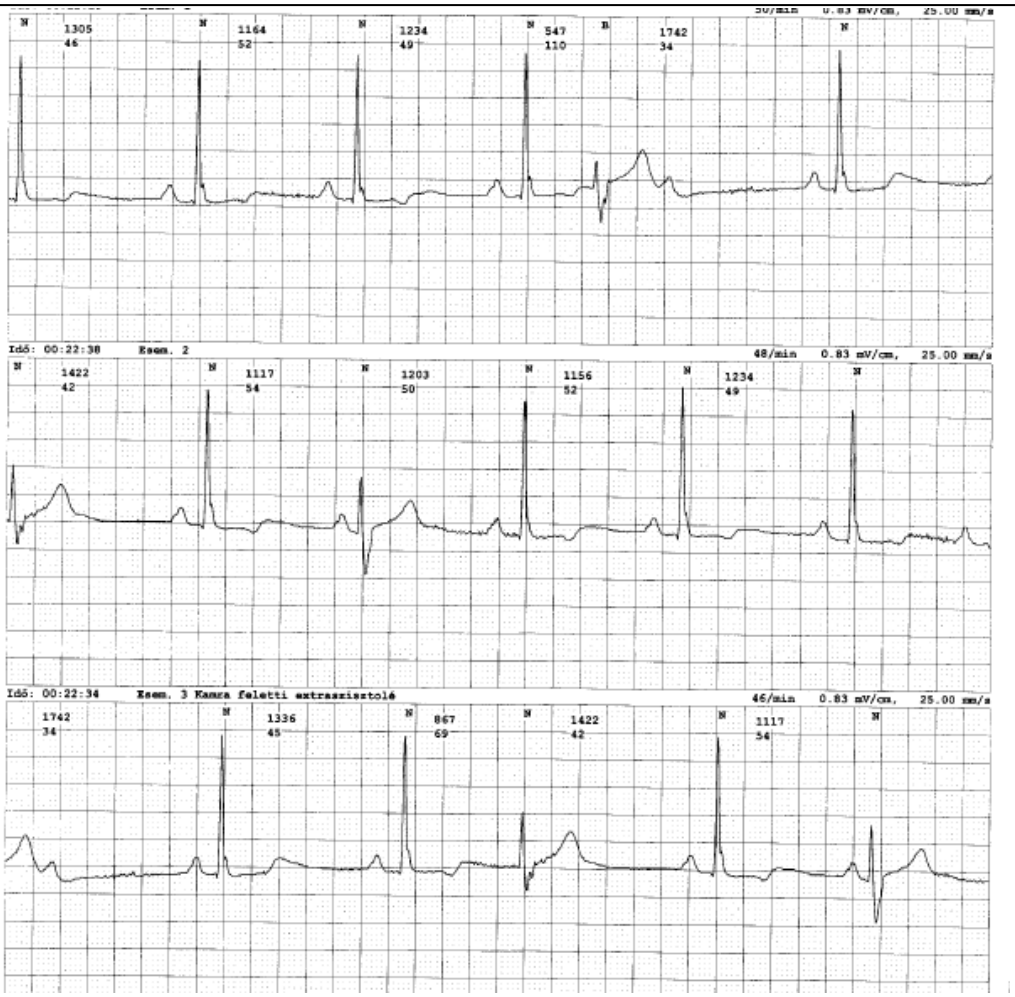


Figure 2/49.

Parasystole. Ventricular ectopic activity with a variable coupling interval. In the first line, a blocked sinus P wave is visible after the 5th parasystolic beat, and several fusion beats can be seen in the second and third line, respectively.

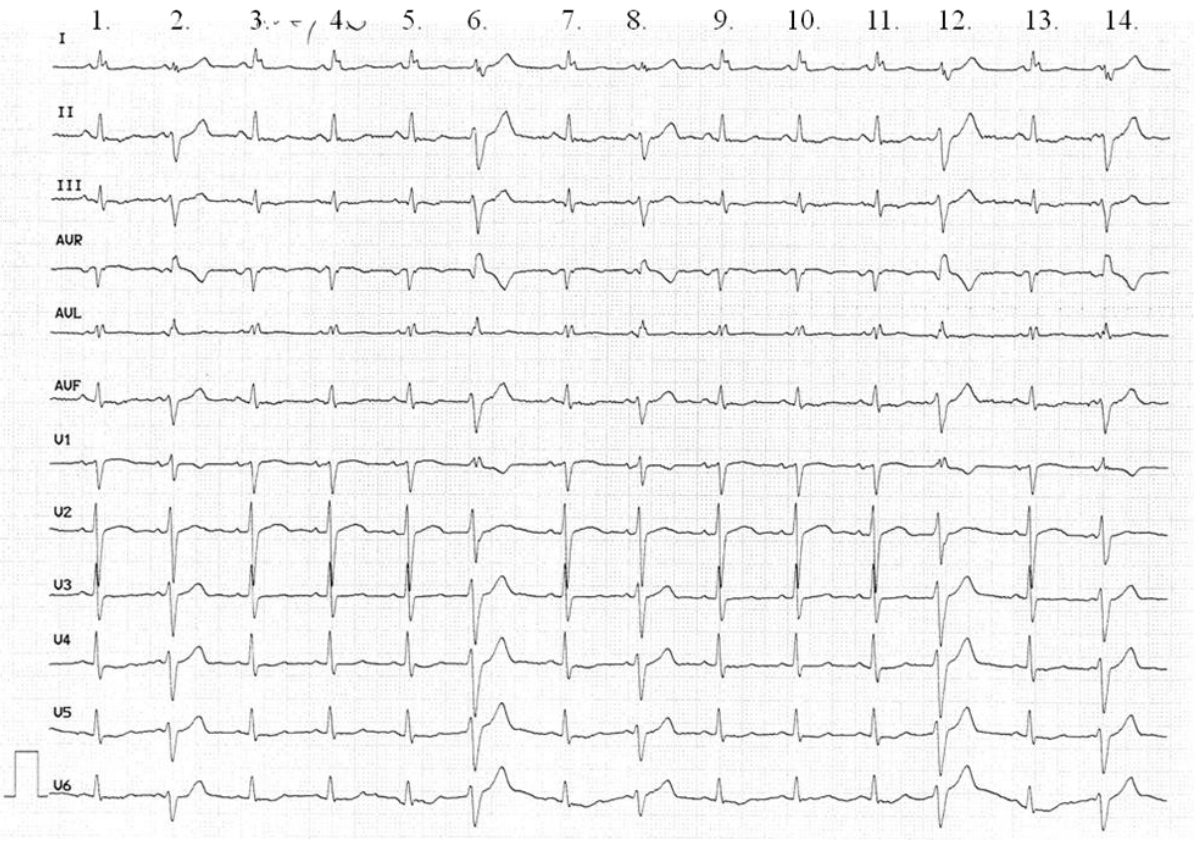


Figure 2/50.

Ventricular parasystole, 2nd, 6th, 8th, 12th and 14th beat: one might see the variable coupling interval and, for the 2nd, 8th and partially for the 14th beat, fusion with the sinus beat is observable. (Sinus rhythm, 85 bpm, normal QRS axis, fragmented QRS complexes in leads I, aVL, otherwise narrow QRS complexes, normal ventricular repolarization, frequent parasystole)

2.3.3. Interference dissociation

Interference dissociation is the phenomenon when, similar to parasystole, two centers capable of impulse formation compete with each other, but the 'firing' rate of the subordinate center exceeds that of the superior one, so it is the subordinate center that results in ventricular contractions more frequently. It is generally associated with structural heart disease, but may also occur in healthy individuals.

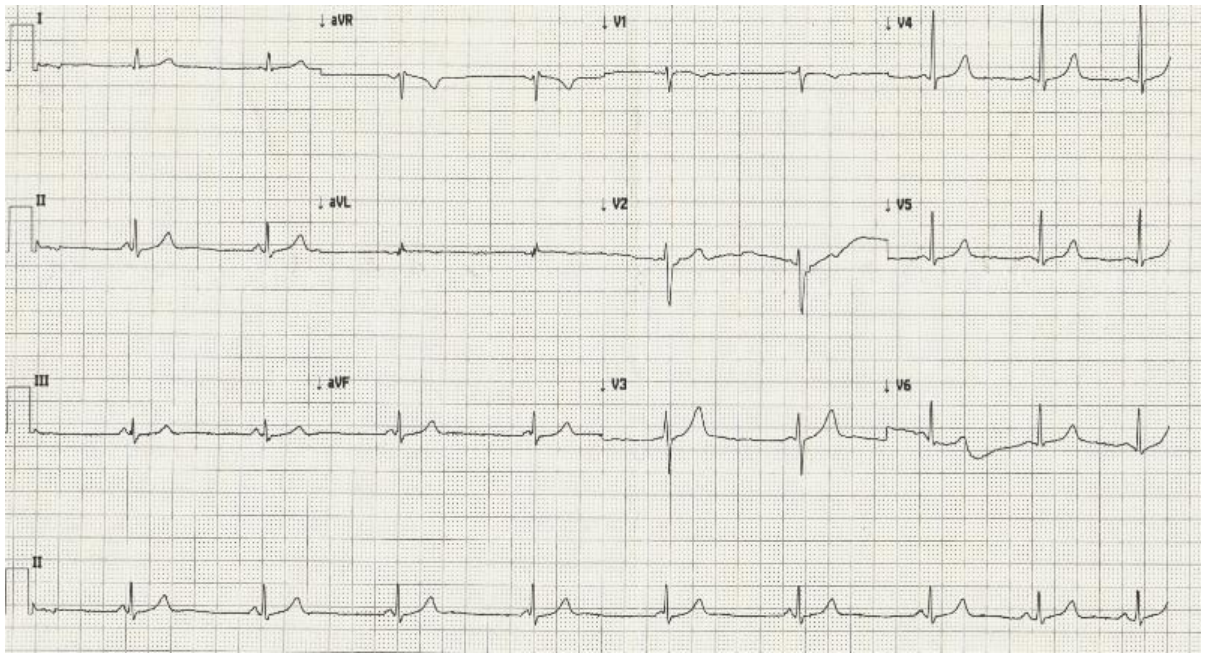


Figure 2/51. Isorhythmic (interference) AV dissociation. One may observe on the rhythm strip that there is a change in the PQ intervals and the P waves in the middle third of the tracing are 'wandering into' the QRS complexes, thus there is no relationship between the P waves and QRS complexes; it is not the conducted P wave that depolarizes the ventricles, but probably a junctional rhythm (Sinus and dissociated junctional rhythm, 50 bpm, normal QRS axis, normal ventricular conduction and repolarization.)

2.3.4. Accelerated idioventricular rhythm

The intrinsic impulse-generating rate of the ventricular musculature is 25-40 bpm, which cannot prevail if there is a sinus impulse providing overdrive suppression. The impulse-generating rate of ventricular muscle cells may increase under pathological circumstances and it transiently suppresses the predominance of sinus impulses. The rate of ventricular 'firing' is between 40-100 bpm in such a case and the QRS complexes are wide. In case of a rate above 100 bpm, one would, by definition, speak about ventricular tachycardia, and not accelerated idioventricular rhythm (see later). Regular and accelerated 'firing' of a ventricular focus may often be observed during *reperfusion* of a *myocardial infarction* or in digitalis toxicity. In the former case, this is a *benign* phenomenon requiring no treatment.

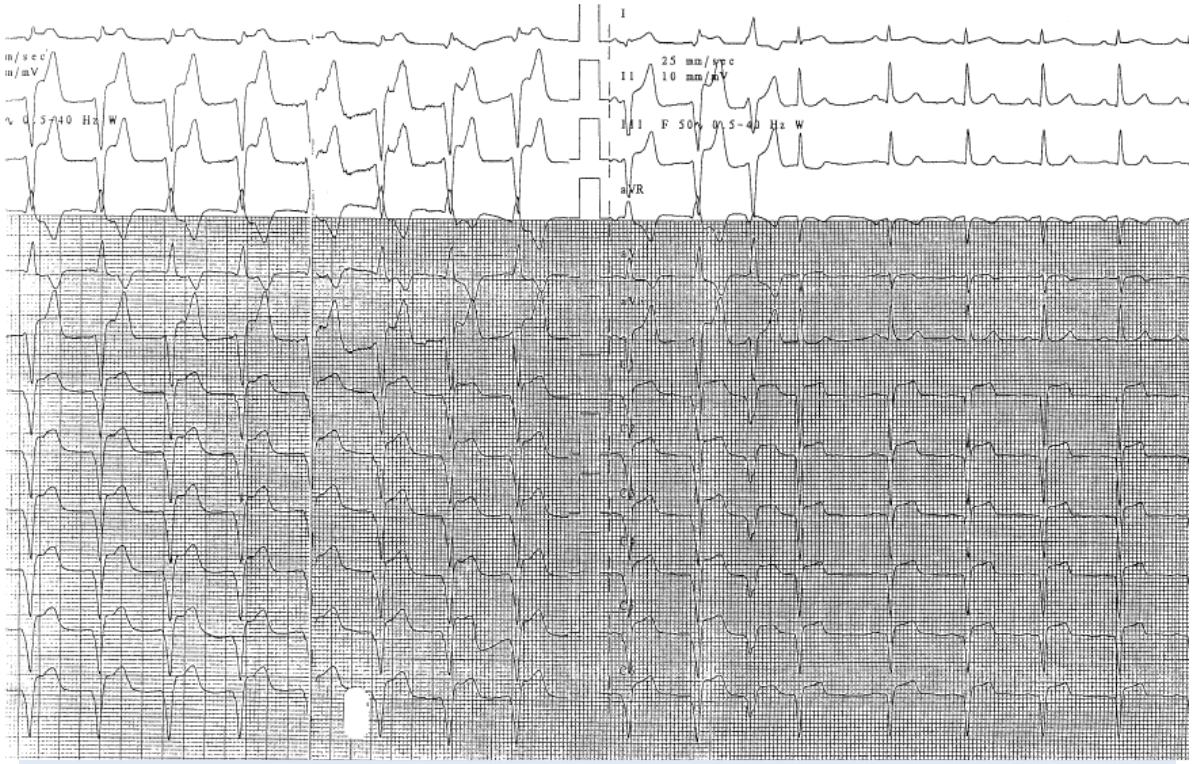


Figure 2/52. Accelerated idioventricular rhythm. The ventricular rhythm can be observed in the first 2/3 of the recording with a rate of 85 bpm, then in the last 1/3 of the tracing, normal sinus beats are visible with ST segment elevation in leads V1-6. The underlying cause was reperfusion of an acute anterior myocardial infarction. (Accelerated idioventricular rhythm with secondary repolarization abnormalities, followed by sinus rhythm, normal QRS axis, normal AV conduction time, QS complexes in leads V1-6, trivial ST segment elevation in leads I, aVL and dome-shaped ST segment elevation of 2-3 mm as well as positive T waves in the precordial leads.)

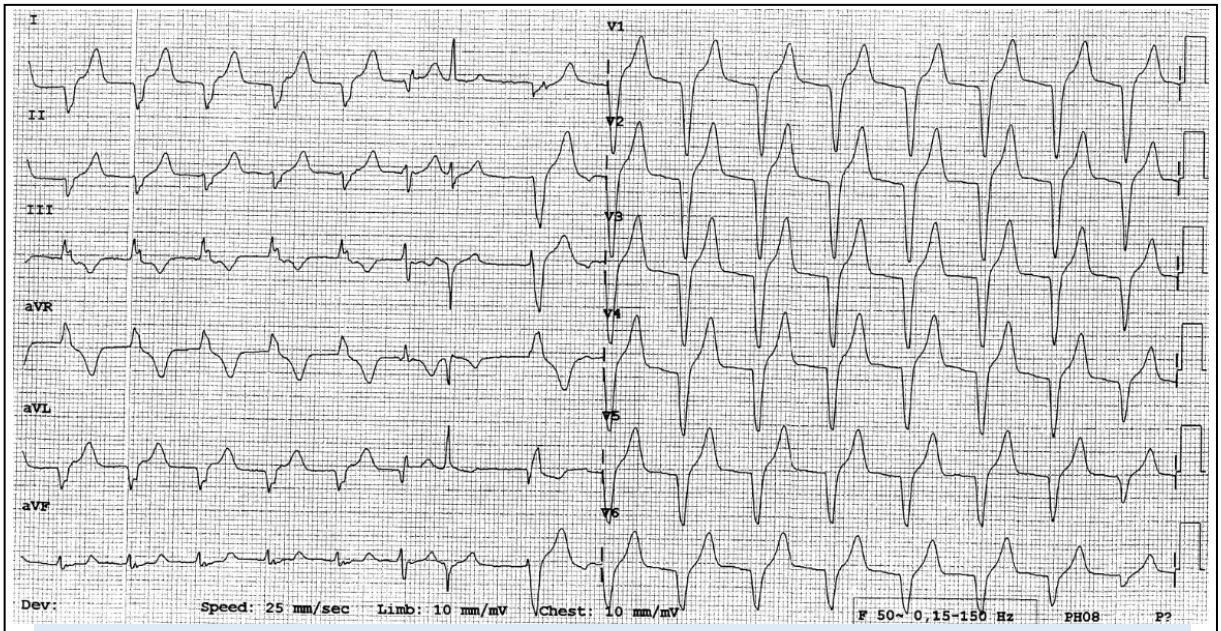


Figure 2/53. Accelerated idioventricular rhythm. The 6th beat in the limb leads is a fusion beat and the 7th beat is a sinus capture beat, after which again accelerated idioventricular rhythm is visible. (Accelerated idioventricular rhythm at a rate of 95 bpm, with extreme right axis deviation and secondary repolarization abnormalities as well as with a fusion beat and a conducted sinus beat.)

2.3.5. Ventricular tachycardia

If 6 or more consecutive ventricular beats are visible at a rate above 100 bpm (100-250 bpm), it is referred to as venticular tachycardia (VT). The most common causes of VT include ischemic heart disease (including AMI), cardiomyopathies (DCM, HCM, ARVD), disturbances of electrolyte balance, carditis. The QRS complexes are typically wide, with a duration usually above 140 ms. The arrhythmia is characterized by discordance of the QRS complexes, ST segments and T waves as well as by secondary repolarization abnormalities, as it has been described for ventricular premature beats. Depending on the origin of the venticular tachycardia (left or right ventricle), the QRS morphology may be similar to that observed during a left or right bundle branch block. VTs with a right bundle branch block morphology are always originating from the left ventricle; however, cases of VT presenting with a left bundle branch block pattern can have either septal or right ventricular origin. Based on their morphology, VTs may be monomorphic (the shape of QRS complexes is identical) or polymorphic (variable QRS morphology). The latter one is often observable in ischemic heart disease.

A special form of polymorphic VTs is torsade de pointes venticular tachycardia associated with a long QT interval, which, beyond congenital prolongation of the QT interval, may be caused by hypokalemia, hypomagnesemia and certain medications (macrolide antibiotics, antihistamines, antipsychotics and antidepressants) and, moreover, bradycardia is in favour of its development (pause-dependent form.)

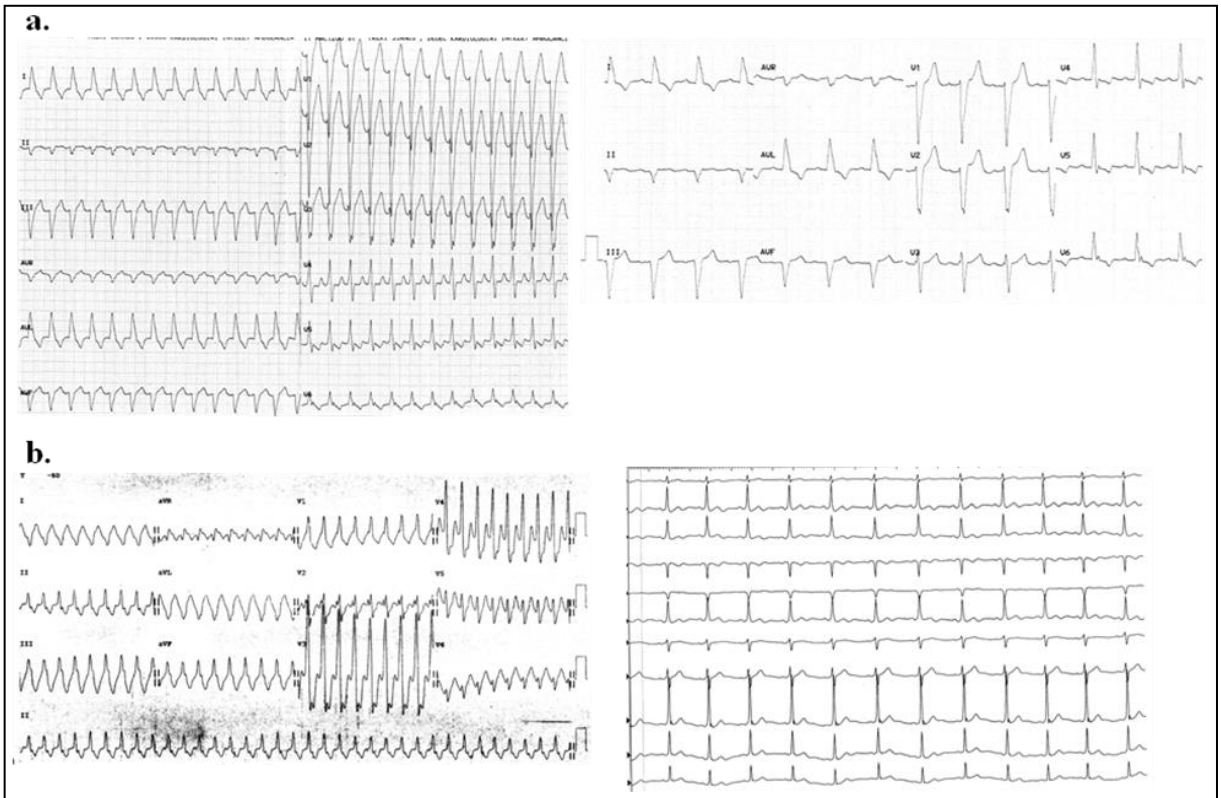


Figure 2/54. Wide QRS complex tachycardia with supraventricular origin. For case **a.)**, atrial flutter with 2:1 conduction is visible, which is superimposed upon the preexisting left bundle branch block conduction pattern (see on the right-sided tracing in sinus rhythm) (i.e. there is no change in the QRS axis and triphasic QRS complexes are detectable in lead V6, which implies a supraventricular origin). For case **b.)**, antidromic AVRT mediated by a left-sided lateral accessory pathway is observable, which is difficult to distinguish based on morphological criteria, but becomes apparent based on the delta waves and short PQ interval detectable in sinus rhythm (right-sided ECG tracing.)

Ventricular tachycardia is often difficult to distinguish from supraventricular arrhythmias associated with a wide QRS complex (aberrant conduction or antidromic AVRT specific for WPW syndrome – see there for details).

Refractoriness may be different in certain areas of the heart, so it may occur that a supraventricular arrhythmia accompanied by a rapid atrial rate is conducted to the ventricles with a bundle branch block morphology, which may falsely be interpreted as a VT. From treatment aspects, their rapid distinction is extremely important, possibly on the basis of signs on the surface ECG. It is important to note *that based on the current blood pressure of the patient as well as on the hemodynamic stability or the ventricular rate of the arrhythmia, one cannot draw conclusions about supraventricular and ventricular origin.* Sometimes clinically stable condition is observable even in patients with a fast VT, whereas a PSVT with a rapid ventricular rate may be associated with syncope and hypotension.

Based on duration of the ventricular tachycardia, sustained (sVT) and non-sustained forms (nsVT) can be differentiated, the limit of which has been established at 30 seconds. Knowledge of this may be of importance regarding treatment, since the therapy of sVT and nsVT may differ from each other (medications, ICD, DC shock, etc.)

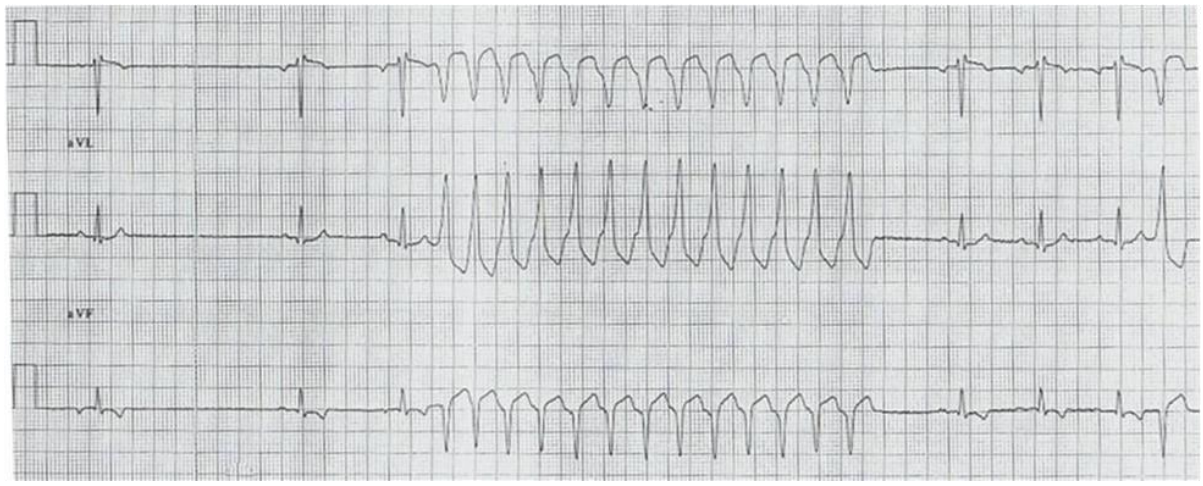


Figure 2/55.

Non-sustained ventricular tachycardia (nsVT.)

In a wide QRS complex tachycardia accompanied by hemodynamic collapse, origin of the arrhythmia is neutral with regard to first medical care, because synchronized electrical cardioversion is necessitated anyway. If the patient has an intact consciousness and there is no impending circulatory collapse, it is in this case that differential diagnosis and, based on this, the choice of adequate therapy becomes necessary. In ¾ of cases, wide QRS complex tachycardias are of ventricular origin. Please remember to take the most common causes into consideration when estabilishing a diagnosis; e.g. for a wide QRS complex tachycardia of a 70-year-old male smoker having had a myocardial infarction previously, it is no use forcing the diagnosis of antidromic AVRT because it is improbable. The obvious diagnosis in this case is ischemic VT.

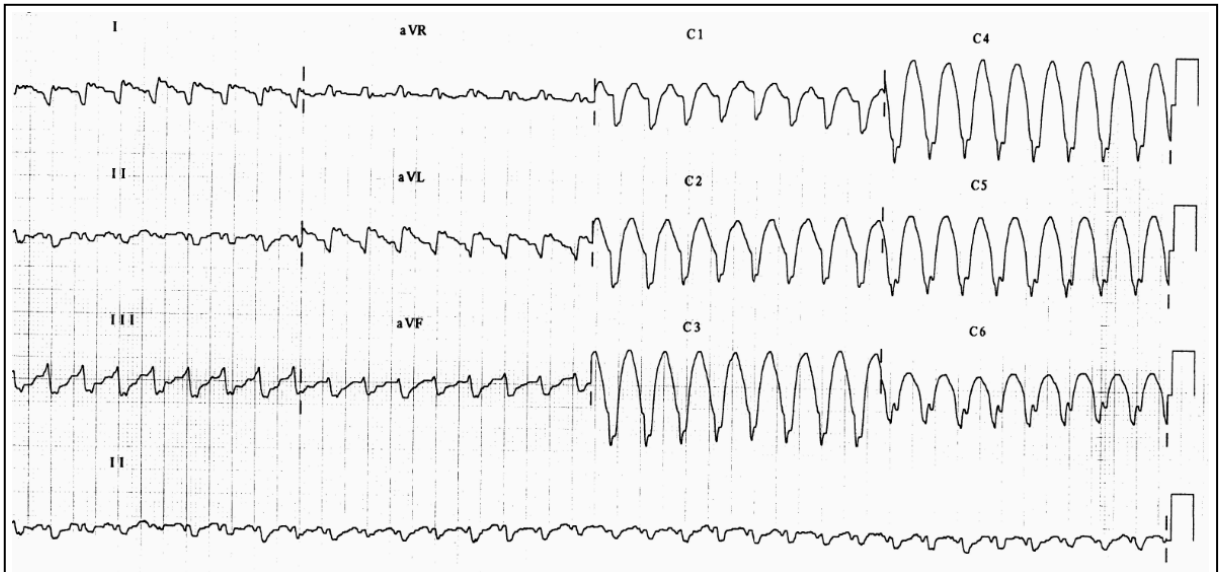


Figure 2/56. Ventricular tachycardia (VT). Please note the regular wide QRS complexes with an LBBB morphology, not preceded by P waves. The dominantly positive deflections in lead aVR ('northwest axis') and the negative deflections throughout the precordial leads (lack of RS complexes) also implies the presence of VT. (Fast ventricular tachycardia with extreme right axis deviation and secondary repolarization abnormalities)

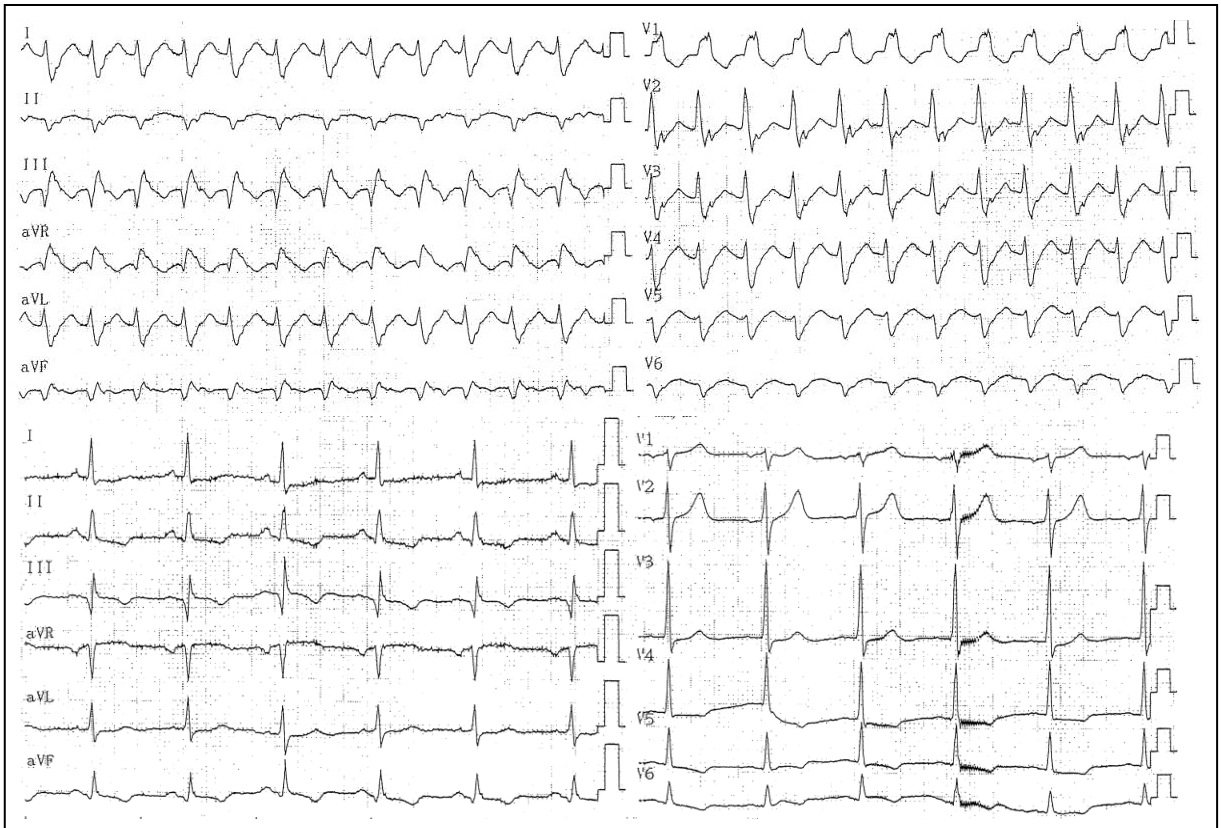


Figure 2/57. Ventricular tachycardia originating from the scar of an inferior myocardial infarction (on the bottom tracing recorded in sinus rhythm, Q waves are visible in leads III and aVF). Inferior Q waves can also be observed during VT. The extreme right (superior) axis deviation (positive deflections in lead aVR) and the AV dissociation in lead aVF (P waves are detectable occasionally) reinforce the diagnosis.

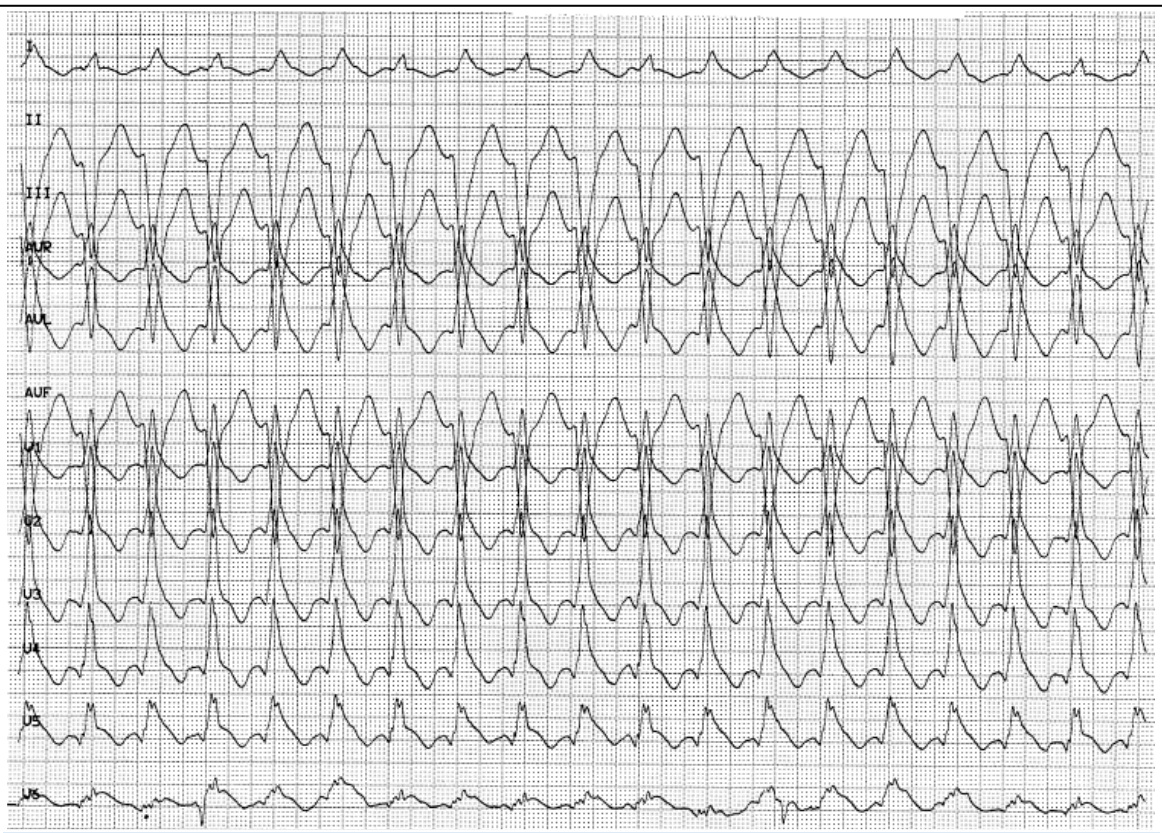


Figure 2/58. Slow ventricular tachycardia. A heart rate of 109 bpm, regular wide QRS complexes with an RBBB+LAFB morphology (fascicular VT?), not preceded by P waves. The positive deflections in lead aVR ('northwest axis') may assist in the differential diagnosis. (Slow ventricular tachycardia, extreme left axis deviation, secondary repolarization abnormalities.)

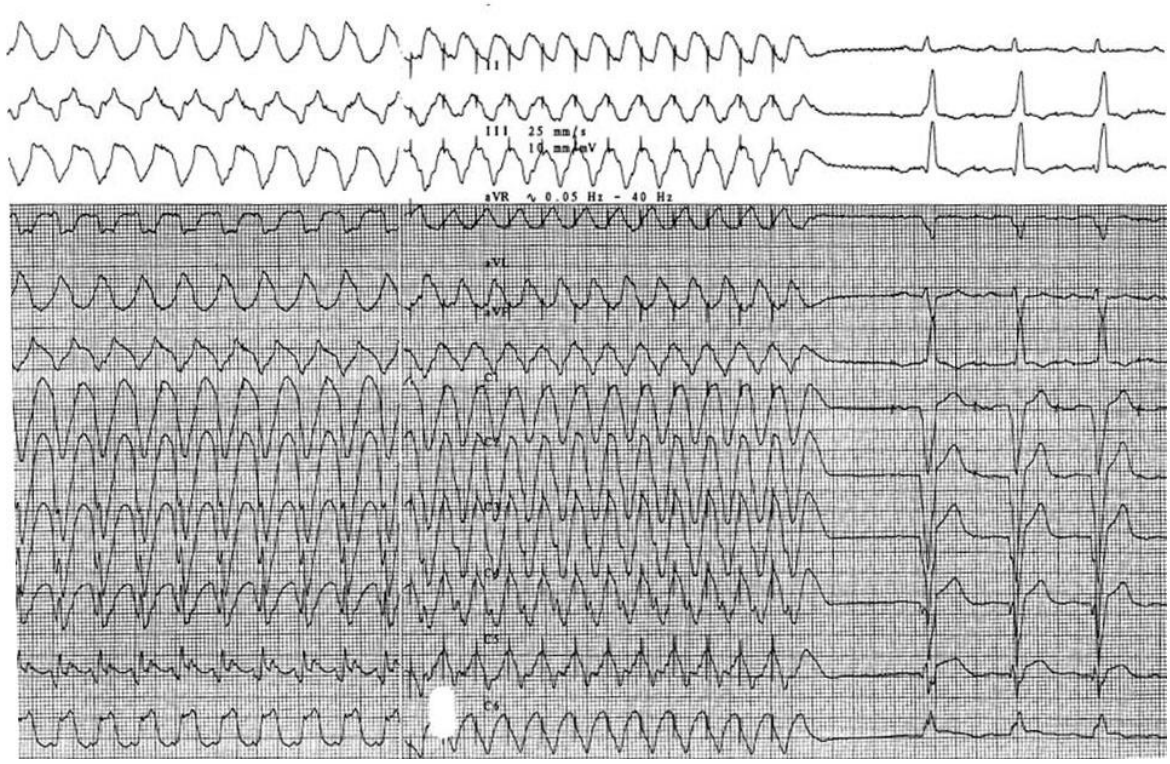


Figure 2/59. Cessation of a fast ventricular tachycardia by antitachycardia pacing (ATP) delivered during ICD therapy.

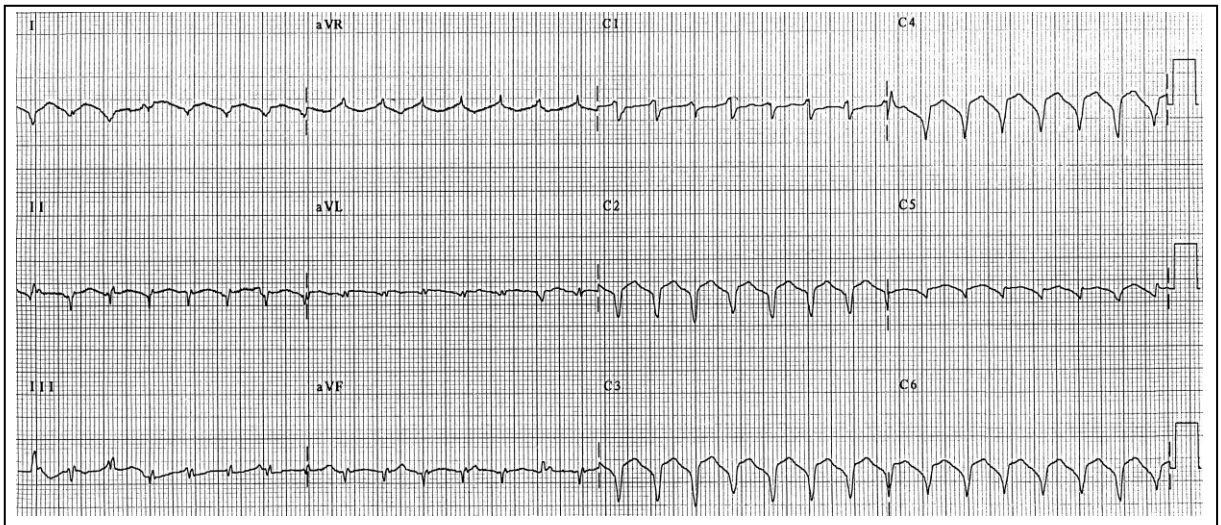


Figure 2/60.

Ventricular tachycardia in severe dilated cardiomyopathy. Please note the signs of AV dissociation in lead aVF: a P wave can be seen after the 2nd and 4th beat, in addition, the 5th beat is a fusion beat (different morphology). (Fast ventricular tachycardia, extreme right axis deviation, low voltage.)

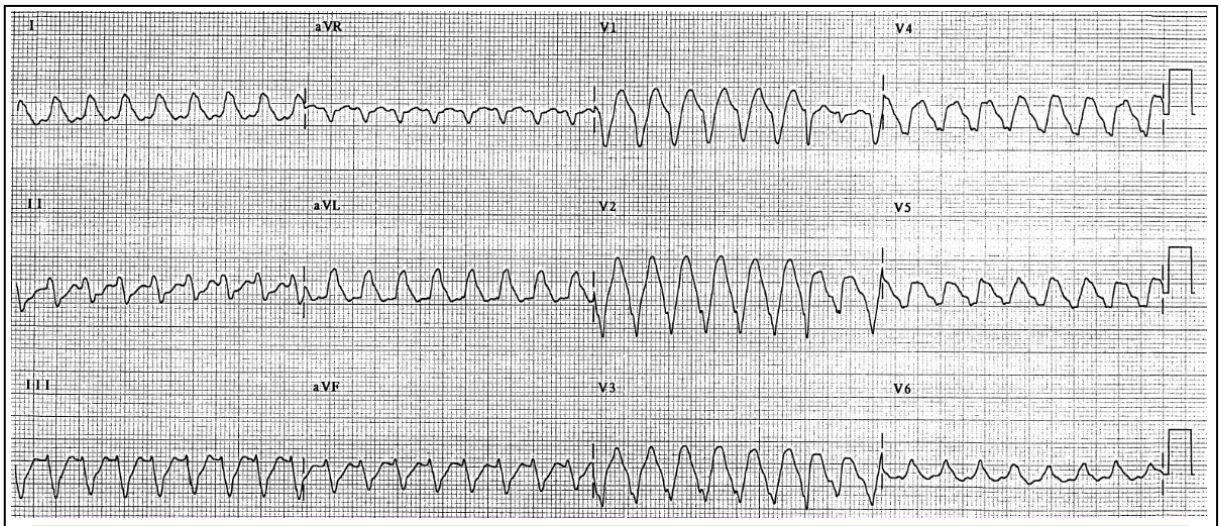


Figure 2/61.

Fast ventricular tachycardia. AV dissociation is visible in leads V1-3, that is the 7th beat is a fusion beat and the 8th beat is a capture beat. Moreover, typical slurring of the downstroke of the QRS complexes is observable in leads V2-3, which is another morphological sign of VTs. (Fast ventricular tachycardia, secondary repolarization abnormalities.)

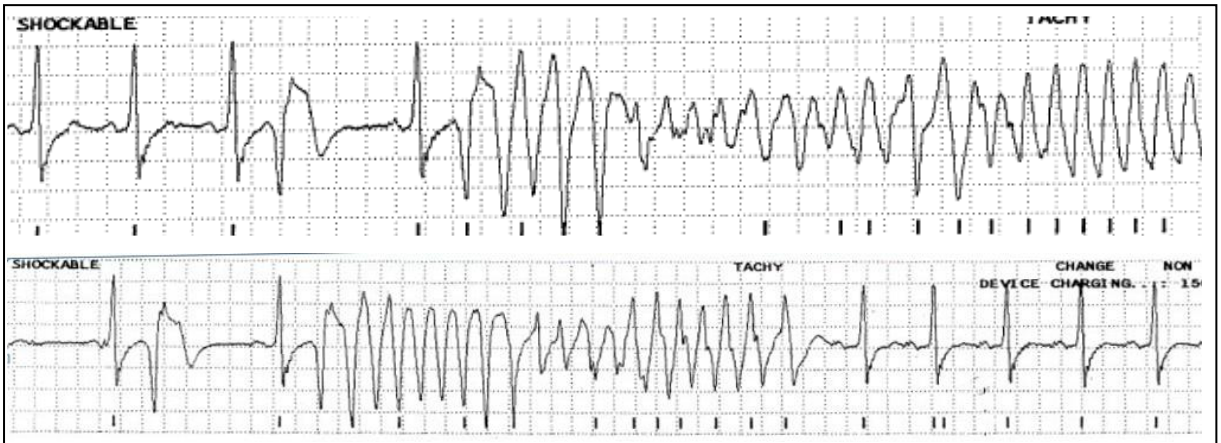


Figure 2/62.

Torsade de pointes (TdP) polymorphic ventricular tachycardia, initiated by an R-on-T phenomenon. A characteristic feature is a swinging baseline on the ECG, induced by a continuous change, i.e. rotation, in the QRS axis. TdP is a typical arrhythmia in QT prolongation (either proarrhythmia or congenital etiology) and it does not even occur without its presence.

Some more rarely occurring, specific forms of VT will be presented in the examples below.

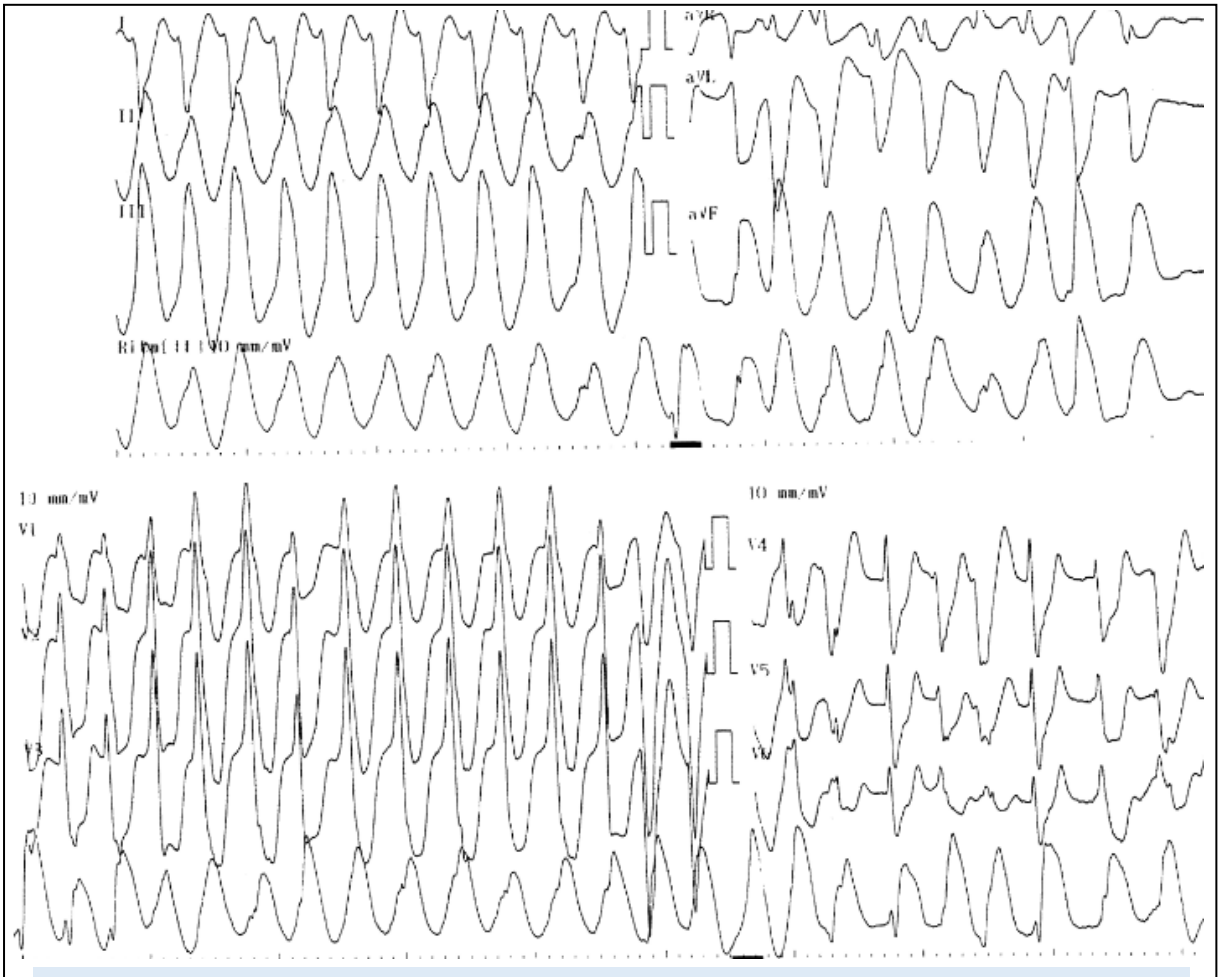


Figure 2/63. Polymorphic VT in acute myocardial infarction.

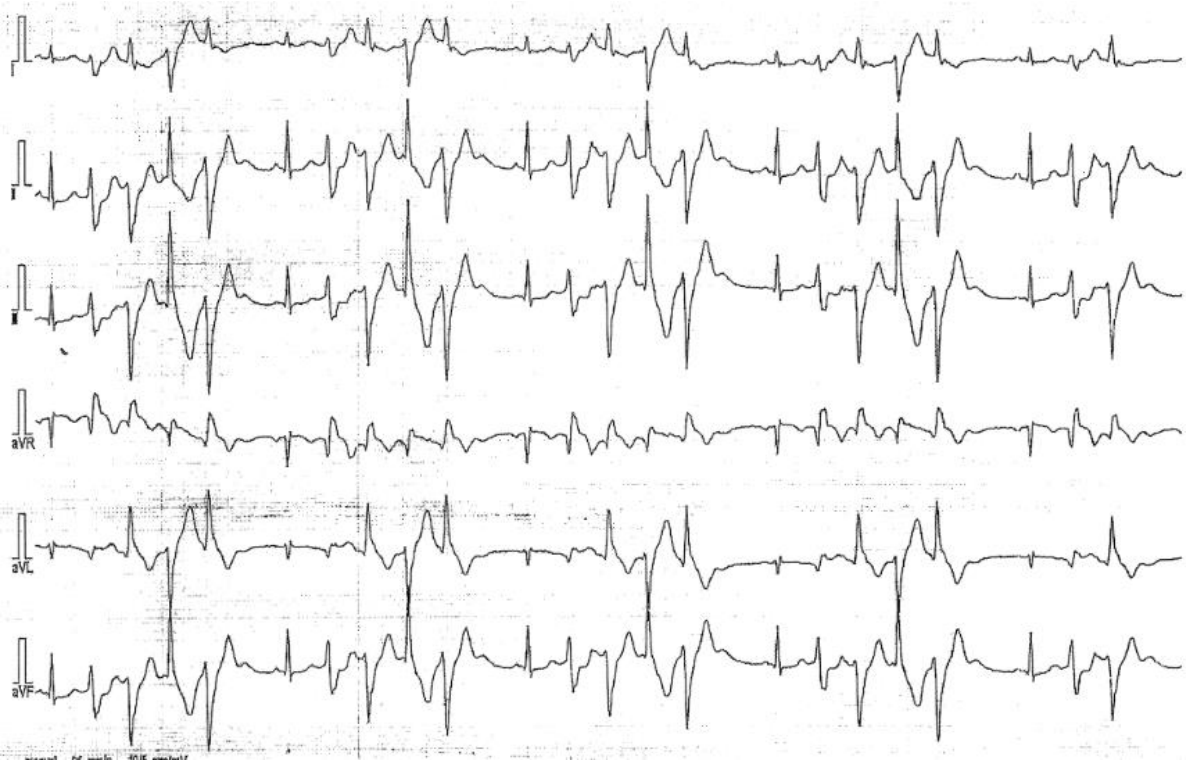


Figure 2/64.

Catecholamine-sensitive (catecholaminergic) polymorphic ventricular tachycardia, interrupted by sinus beats. The arrhythmia is induced by physical exertion and is sensitive to verapamil.

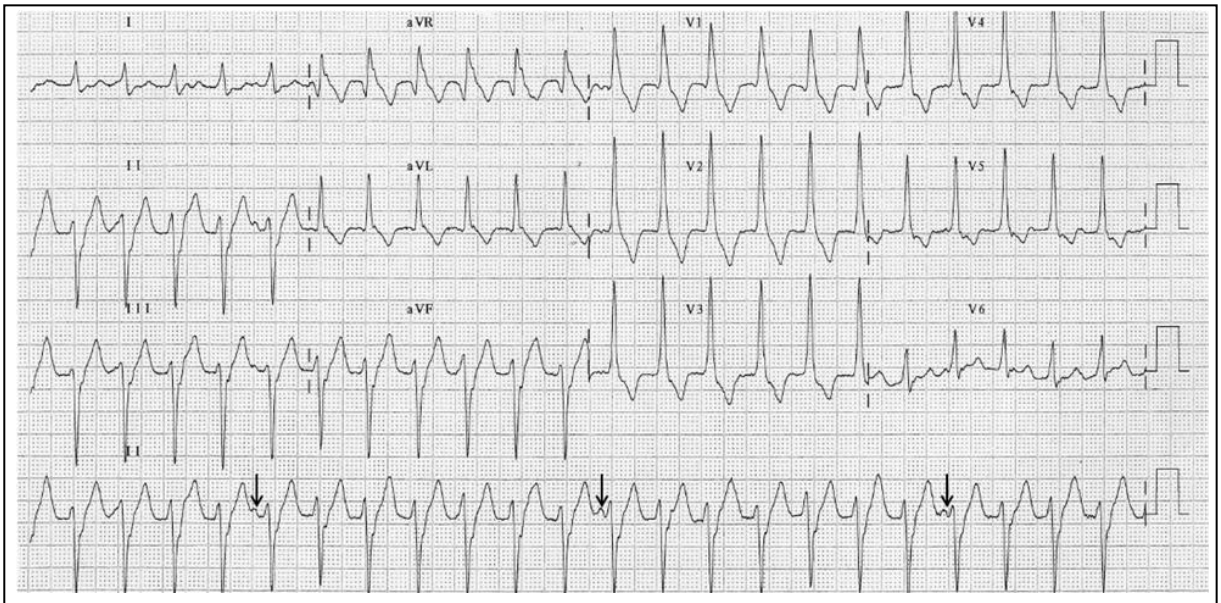


Figure 2/65.

Verapamil-sensitive fascicular VT. The presence of P waves on the rhythm strip (arrows) is a sign of AV dissociation.

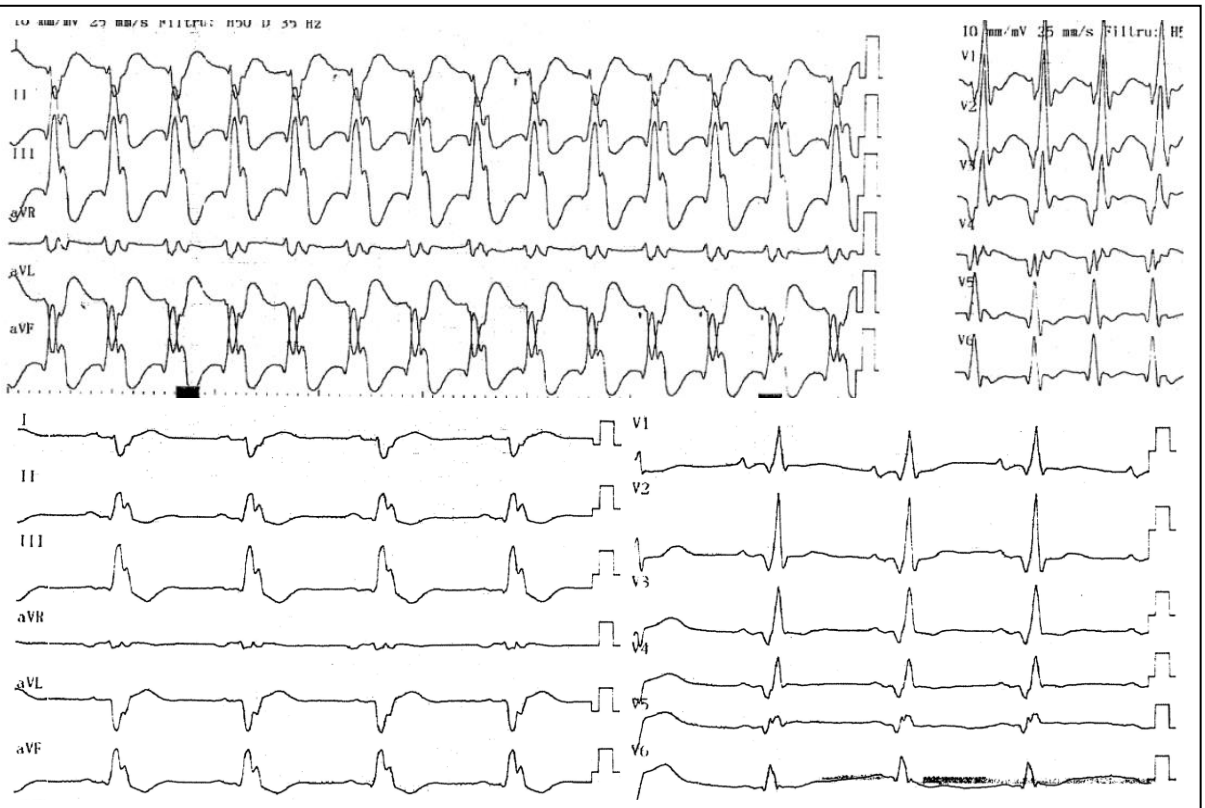


Figure 2/66.

Bundle branch reentrant ventricular tachycardia and the ECG tracing below recorded in sinus rhythm are different from each other only to a very small degree. The preexisting RBBB+LAFB morphology remains unchanged if the tachycardia arises from the left anterior fascicle.

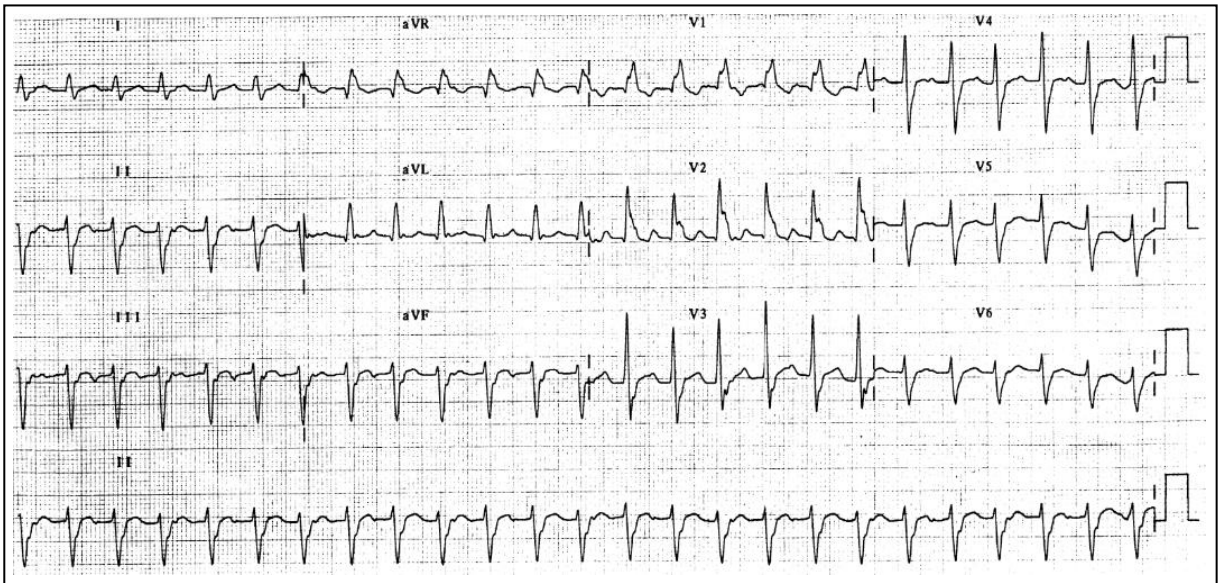


Figure 2/67.

Fascicular VT. It often has an RBBB+LAFB morphology since it is originating from the left posterior fascicle. One might observe in lead III that every third beat is conducted in a retrograde fashion to the atria (negative P wave on the ST segment) – being consistent with 3:1 ventriculoatrial (VA) conduction.

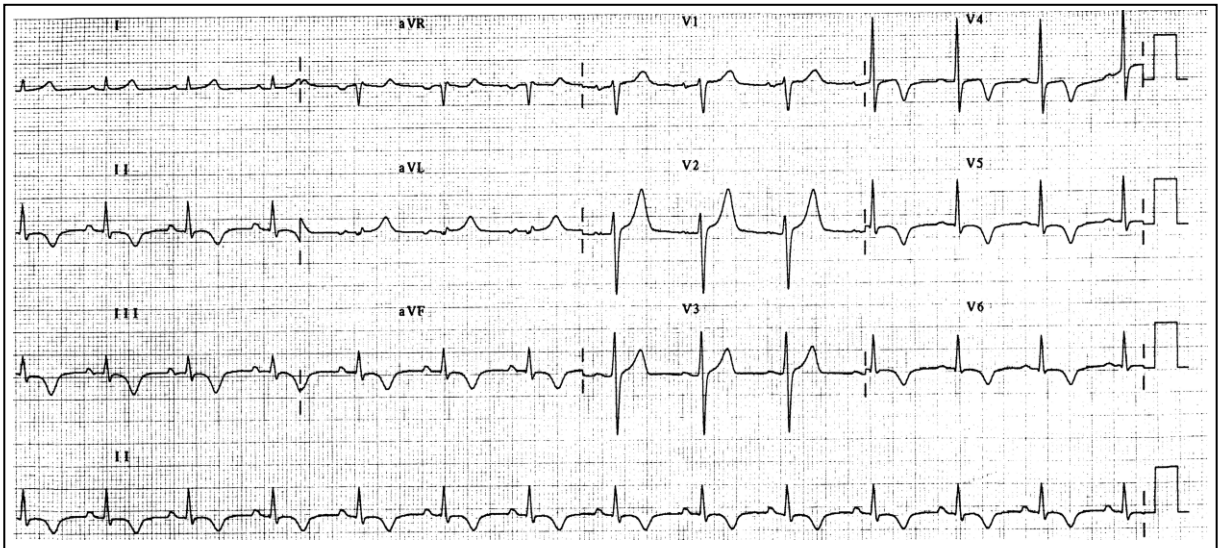


Figure 2/67. continuation Below this, an ECG recorded during sinus rhythm is visible.

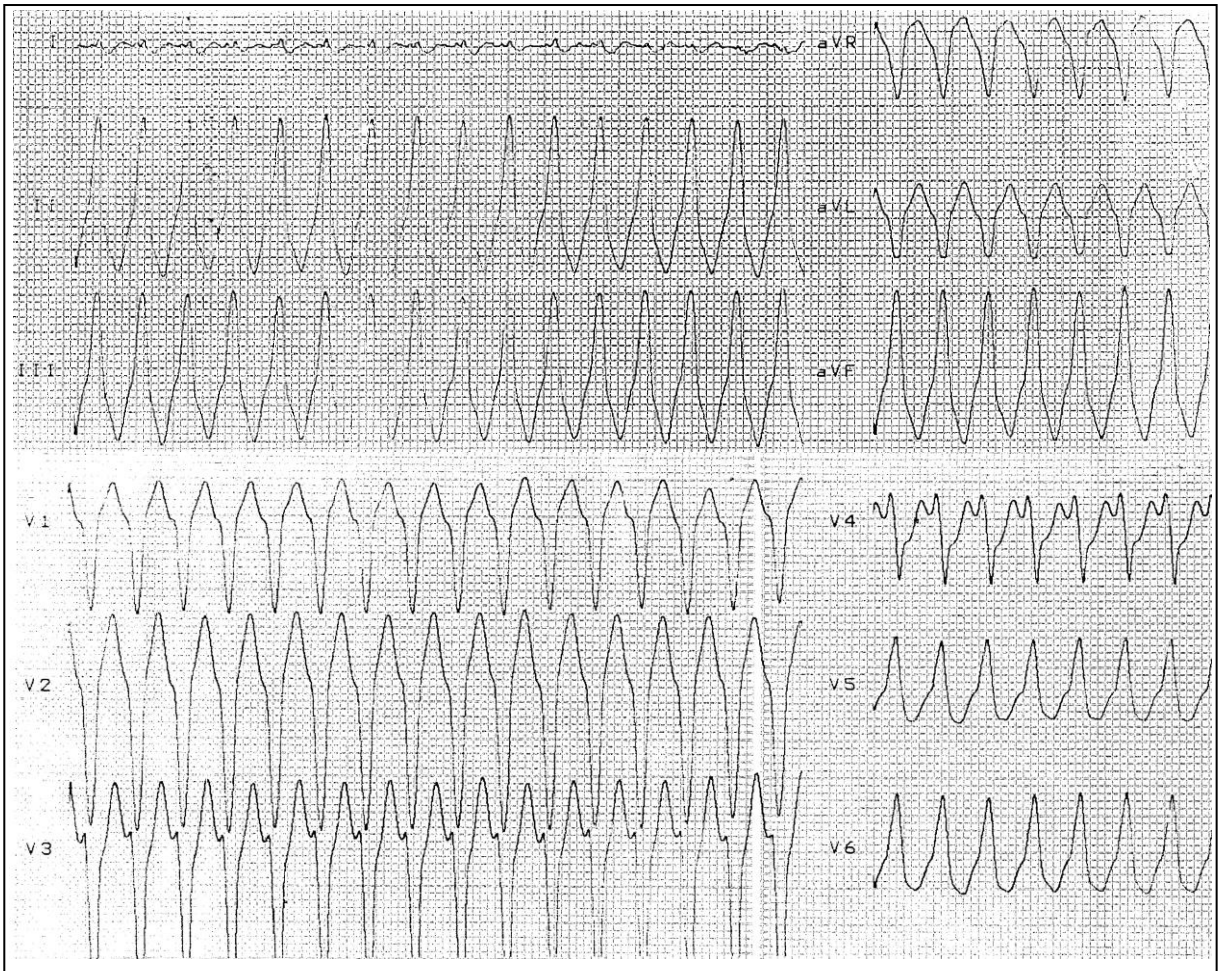


Figure 2/68.

Right ventricular outflow tract tachycardia (RVOT-VT). Positive deflections of the QRS in leads II, III, aVF demonstrate an axis pointing to the inferior direction, while the LBBB morphology shows a right ventricular origin.

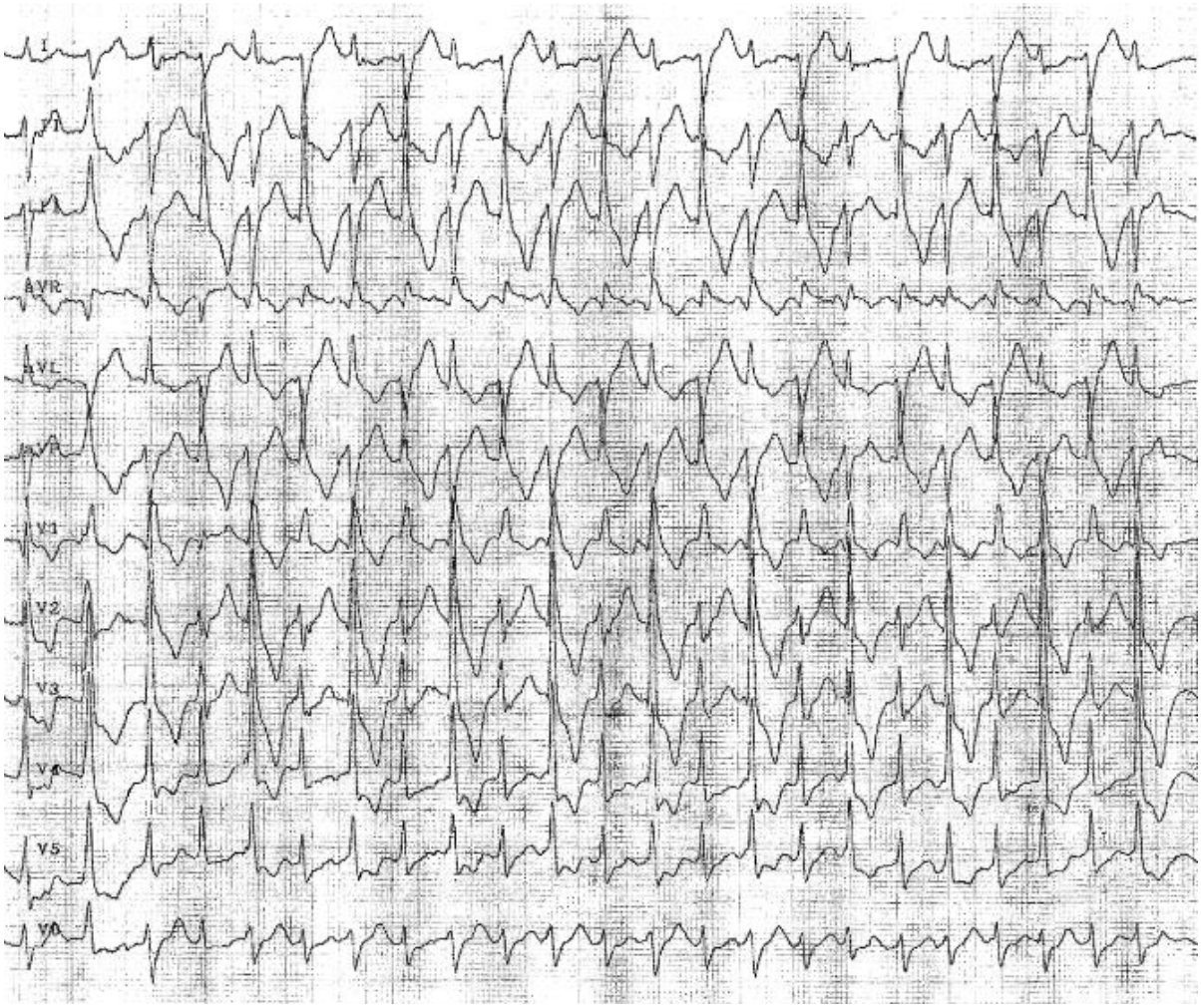


Figure 2/69.

Bidirectional ventricular tachycardia. Along with an RBBB morphology, impulses are conducted to the ventricles once on the left anterior fascicle (LPFB pattern) and once on the left posterior fascicle (LAFB pattern), in an alternating fashion.

It was mentioned previously that the overwhelming majority of wide QRS complex tachycardias is of ventricular origin, therefore, what appears to be a VT at first sight and after taking anamnestic data into account, then it really is a VT. Nevertheless, let's review some criteria which provide guidance on the differentiation between ventricular and supraventricular origin. Apart from ventricular tachycardia, the following conditions may be associated with a wide QRS complex tachycardia:

1. preexisting bundle branch block+SVT;
2. antidromic AVRT;
3. an SVT passively involving the existing accessory pathway;
4. prolongation of impulse conduction: class IA, IC and III antiarrhytmic agents and hyperkalemia(!);
5. pacemaker stimulation.

The differentiation criteria include:

1. A search for the signs of AV-dissociation (a definite sign for VT):

   Less P waves than QRS complexes; the P waves are hidden in, or deform, the QRS complexes.

   Capture beat: primarily for VTs with a less frequent heart rate, a supraventricular impulse with appropriate timing is conducted with a narrow QRS complex, wedged between two beats with a wide QRS complex.

   Fusion beat: a supraventricular impulse with proper timing and a ventricular impulse begin to depolarize the ventricles from two directions at the same time (e.g. the sinus impulse depolarizes from the base of heart, and the ventricular impulse from the cardiac apex), so a mixed pattern is created from the two different beats.

   VA (retrograde ventriculoatrial) conduction can be observed in 1/3 of VTs (it may be blocked by carotid sinus massage, thereby proving ventricular origin).

2. The wider the QRS complex, the more likely it is to have a ventricular origin; for a left bundle branch block pattern, a QRS width above 160 msec (since the left ventricular wall is thicker) renders a ventricular origin probable, while that above 140 msec does so in case of a right bundle branch block pattern. QRS complexes may rarely be narrow or at least having a width of around 120 msec, if the tachycardia arises from the subjunctional or septal region and it immediately penetrates into, or is directly generated in, the cardiac conduction system (e.g. fascicular tachycardia). It is a rare phenomenon, but a definite sign of having a VT, if the QRS complexes recorded during sinus rhythm are wider than those recorded during the tachycardia. The reason for this narrowing is that in case of sinus rhythm and concurrent bundle branch block, the ventricles demonstrate an asymmetrical activation, while during a VT (e.g. think about a septal origin), activation becomes symmetrical and the entire ventricular depolarization is completed earlier.

3. If there is a shift in the QRS axis by more than 40˚ compared to what is observed during normal sinus rhythm, it makes ventricular origin likely. An indeterminate ('northwest') axis, that is tall R waves in lead aVR, also implies ventricular origin. Left axis deviation along with a right bundle branch block pattern suggests ventricular origin, and right axis deviation along with a left bundle branch block pattern definitely ensures the diagnosis of VT.

4. Positive concordance of the QRS complexes (the resultant vector of all deflections is positive) in the precordial leads makes the diagnosis of VT probable, while negative concordance of those (the resultant vector of all deflections is negative) is a definite marker of the presence of a VT.

5. For left and right bundle branch block patterns, there are certain morphological criteria of the QRS complex, which may help in the differentiation process:

	VT	**SVT**
LBBB	V1 r > 40 ms	V1 r < 40 ms
	rS > 60 ms (slurring on the downstroke of the S wave)	rS <60 ms (smooth and rapid downstroke of the S wave)
	V6 qR	V6 no q Wave, trifasic QRS
RBBB	V1 R, Rr', Rs	V1 trifasic rsR' or rR'
	V6 R/S < 1	V6 R/S > 1

Table 2/1. Differentiation between VT and SVT based on QRS morphology.

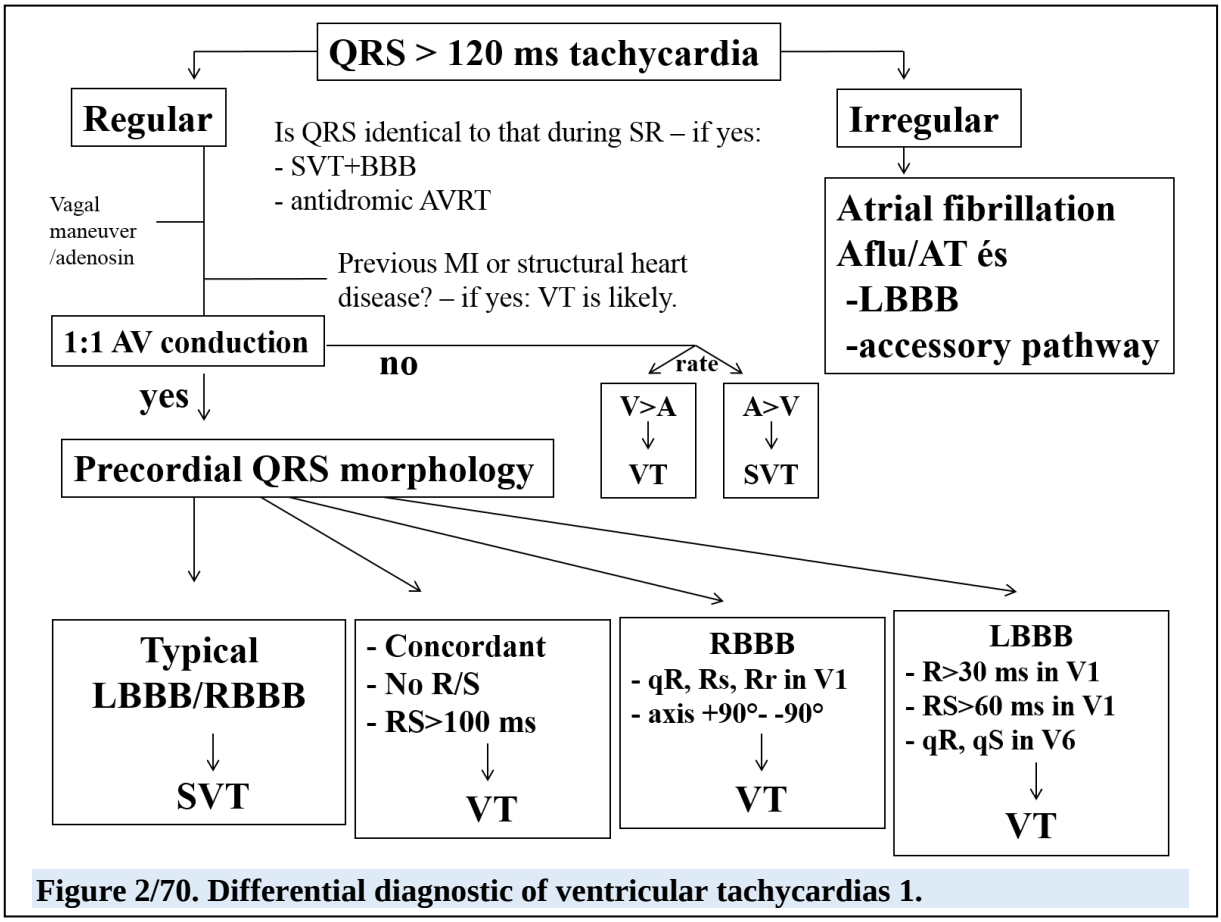


Figure 2/70. Differential diagnostic of ventricular tachycardias 1.

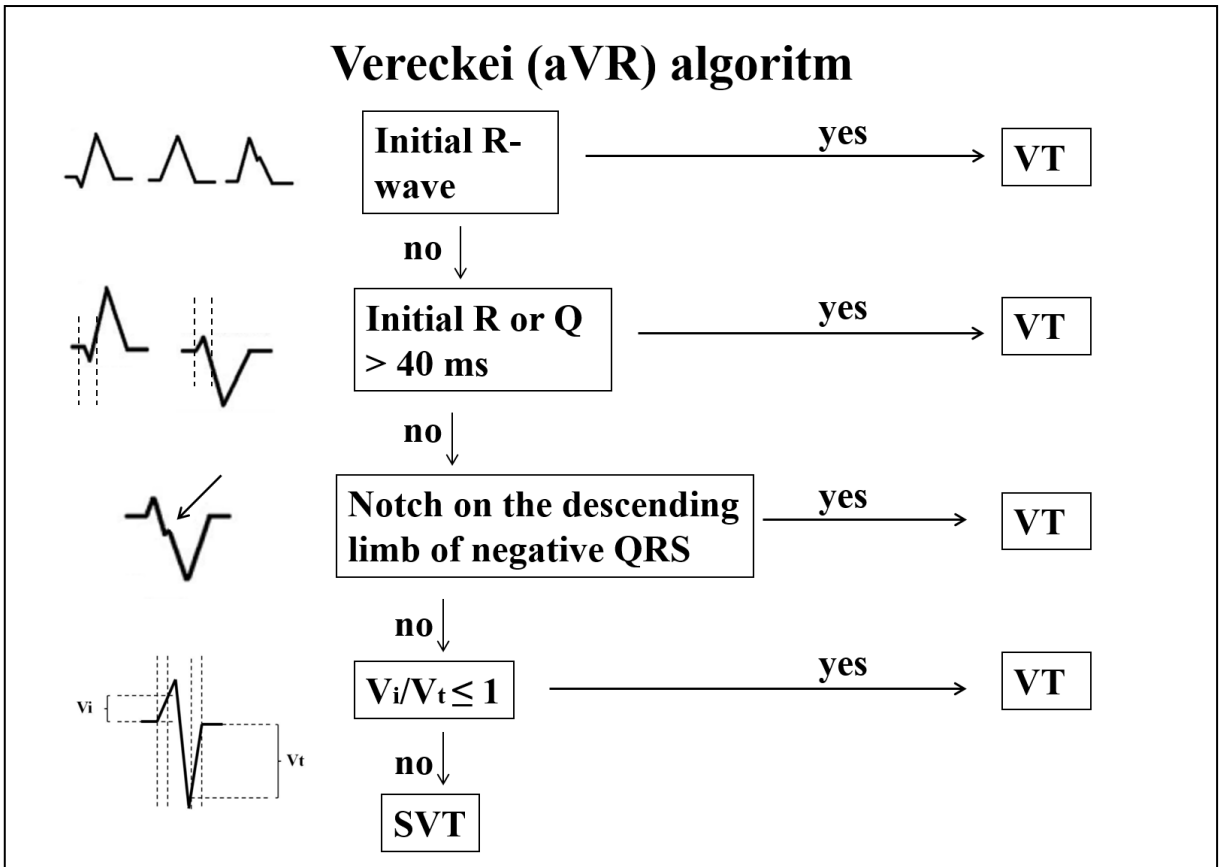


Figure 2/71. Differential diagnostic of ventricular tachycardias 2.

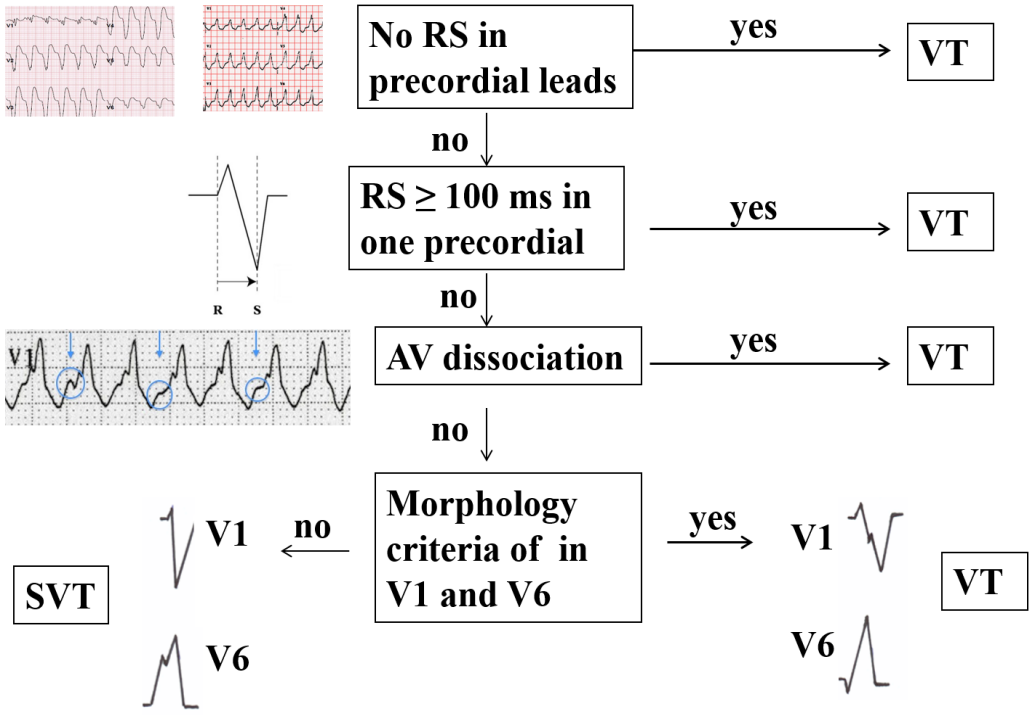


Figure 2/72. Differential diagnostic of ventricular tachycardias 3.

Several algorhythms have been created to differentiate between VT and SVT, of which those from two working groups will be mentioned. The algorhythm by Brugada et al. examines the appearance of RS morphology in the chest leads of a wide QRS complex tachycardia as a first step and, if no such finding is observable, the presence of VT is probable; however, if there is any, then you should find the chest lead with the longest time from the onset of the QRS complex until the deepest point of the S wave. If this RS time is ≥ 100 ms, the presence of VT is probable; if not, examination of the AV dissociation and the above morphological signs of QRS complexes is recommended further. The other algorhythm written by Vereckei and Duray recommends the following: you should look for the signs of AV dissociation first, then examine lead aVR. A tall initial R wave in aVR (’northwest’ or superior axis) indicates the presence of a VT with great accuracy; however, if there are no tall R waves, both the width of the initial r or q wave (> 40 ms) and slurring of the downstroke of the QRS complex is assessed as a sign of VT. Afterwards, the algorhythm recommends an investigation of the above morphological signs of QRS complexes and, if based on all these, the diagnosis is still questionable, velocities of the initial and terminal deflection of QRS complexes (i.e. initial and terminal ventricular activation velocities) are compared to each other. If there is a slow impulse propagation velocity at the initial portion of the QRS complex, while being faster compared to this at the end of the QRS, this renders the diagnosis of VT probable (Vi/Vt ≤ 1).

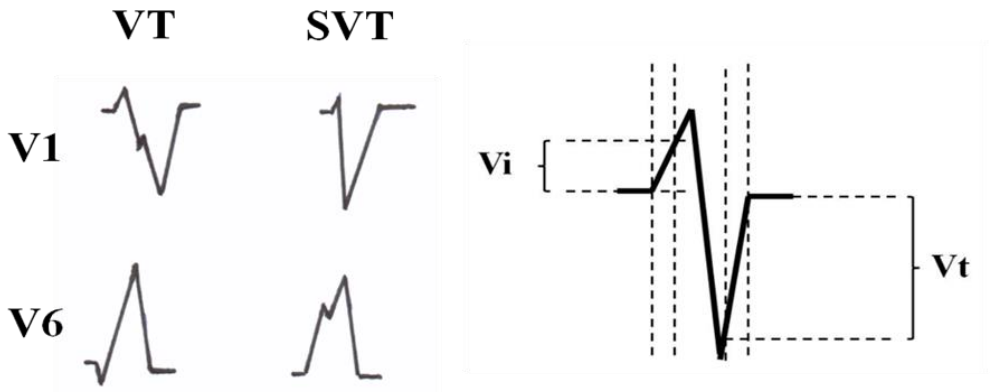


Figure 2/73.

Differentiation between VT and SVT based on QRS morphology.

2.3.6. Ventricular flutter

It is the result of a very rapid (150-300 bpm) ventricular activation with wide, bizarre-shaped and monomorphic beats, resulting in an immediate hemodynamic collapse. QRS complexes are recognizable yet, but ST segments and T waves, as opposed to a VT, can no longer be differentiated. Initiation of immediate cardiopulmonary resuscitation (CPR) and synchronized cardioversion is necessary. Its causes are identical to those of a VT.

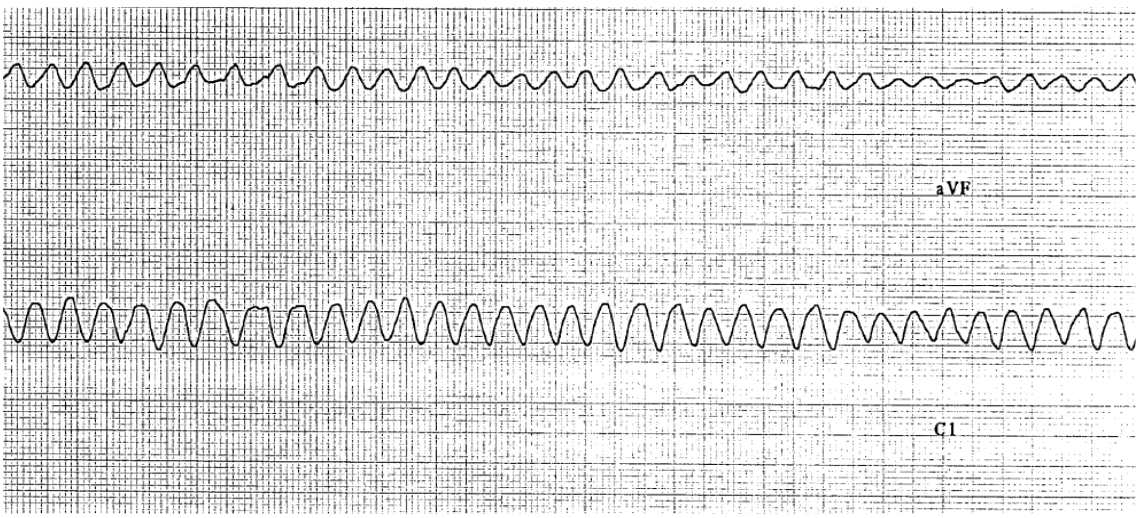


Figure 2/74.

Ventricular flutter. The QRS complexes can be distinguished yet, but other waves cannot.

2.3.7. Ventricular fibrillation

It is a completely disorganized ventricular electrical activity, no longer resulting in effective contractions. Neither atrial nor any regular ventricular activity is observable, because this is an extreme arrhythmia. Immediate cardiopulmonary resuscitation and unsynchronized DC cardioversion (defibrillation) is necessary. Its causes are identical to those of a VT.

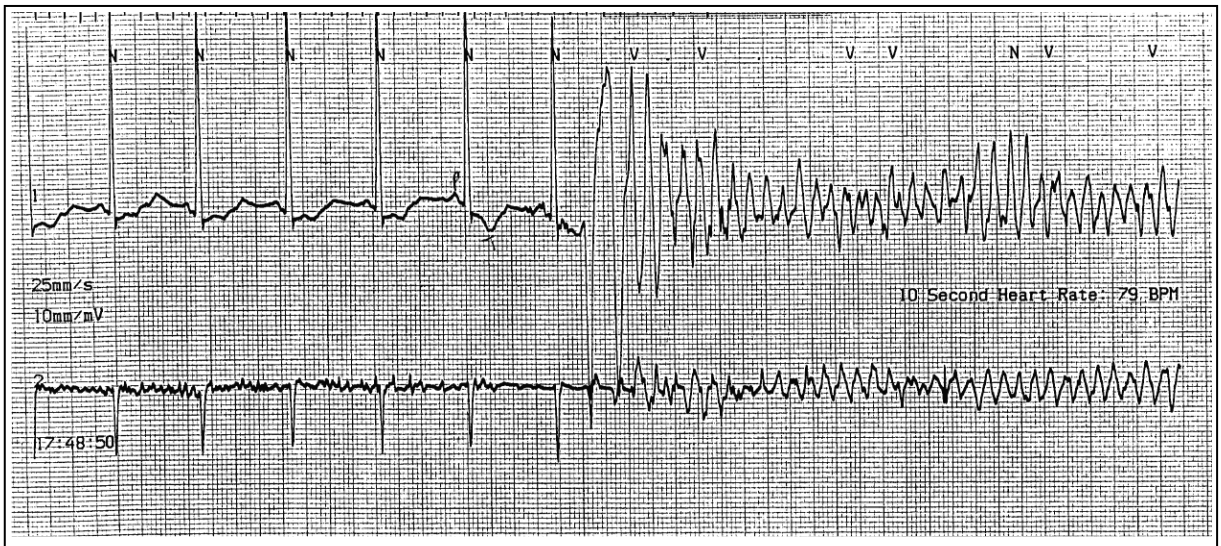


Figure 2/75.

Ventricular fibrillation observed during Holter monitoring, which is initiated by a VPB showing an R-on-T phenomenon.

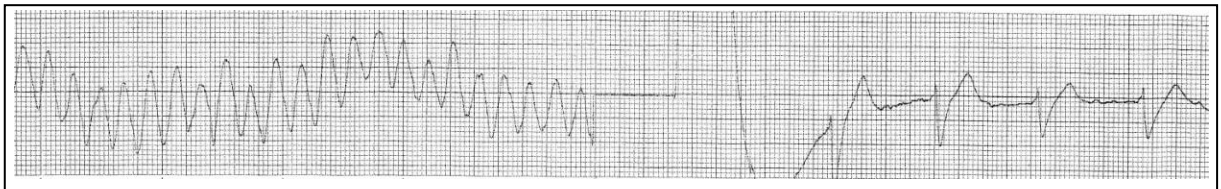


Figure 2/76.

Ventricular fibrillation and defibrillation.

FACTS THAT YOU MUST KNOW:

1. A ventricular premature beat is a beat with a wide QRS complex occurring earlier than expected, often being a harmless phenomenon.
2. Accelerated idioventricular rhythm is not ventricular tachycardia, but a benign phenomenon during reperfusion.
3. In case of a regular wide QRS complex tachycardia, you should think of the presence of ventricular tachycardia first.
4. In an unconscious or hemodynamically unstable patient who has wide QRS complex tachycardia or ventricular fibrillation, you should perform electrical cardioversion or defibrillation without any delay.

CHAPTER 3

DISORDERS OF IMPULSE CONDUCTION

Classification:

1. Sinoatrial conduction abnormalities
2. Atrioventricular conduction disturbances (AV block)
3. Intraventricular conduction disturbances (fascicular block, bundle branch block)

3.1. Sinoatrial exit blocks

3.1.1. First degree sinoatrial block

it has no sign on the 12-lead surface ECG because every sinus impulse reaches the atria, however, only in a delayed fashion, and no direct signs of SA node depolarization are visible; only its consequences, that is the P waves, appear on the ECG.

3.1.2. Second degree sinoatrial block

Second degree type 1 (SA Wenkebach block): Following regular sinus beats, no P wave is visible where its occurrance is expected, and the duration of the pause caused by the missed beat is less than the sum of two normal RR intervals. Differential diagnostics include blocked PACs and sinus arrhythmia.

Second degree type 2 (SA - Mobitz II): Following regular sinus beats, no P wave is visible where its occurrance is anticipated, and the duration of the pause caused by the missed beat is just the same as the sum of two normal RR intervals.

Type 2:1: It cannot be distinguished from sinus bradycadia, nevertheless, its presence may be implied by a sudden doubling in heart rate observed during temporary normalization of the SA conduction.

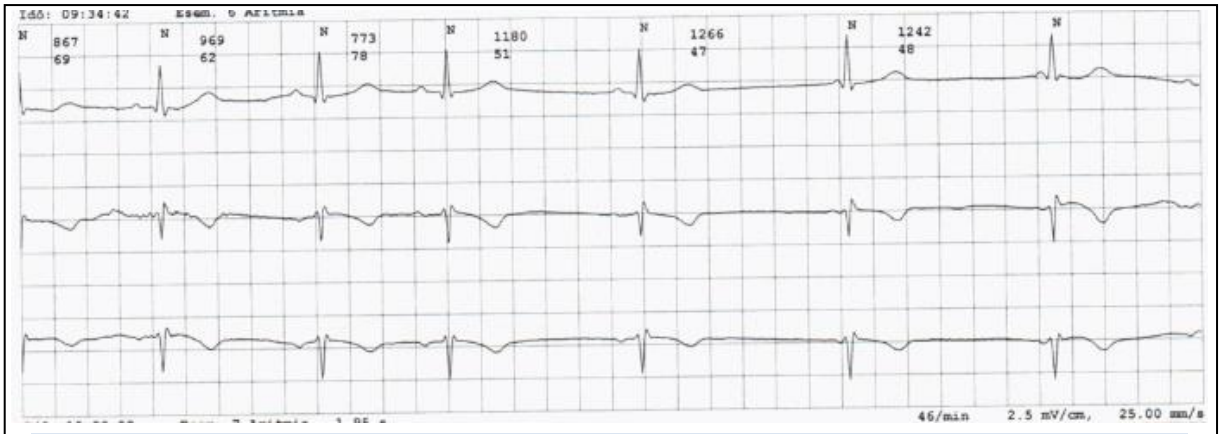


Figure 3/1.

Second degree type 1 (Wenkebach) SA block after the 3rd QRS complex, followed by isorhythmic AV dissociation (block) with junctional escape rhythm.

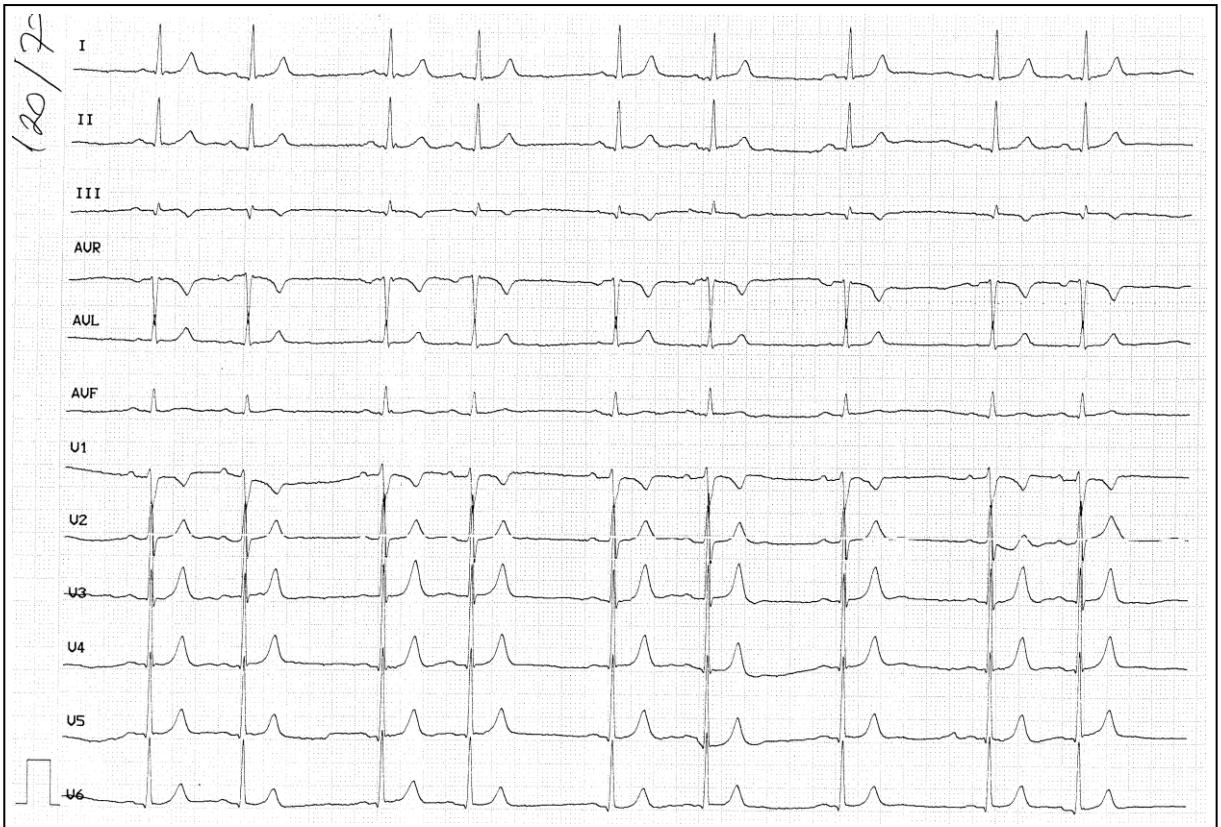


Figure 3/2. Sinoatrial Wenkebach phenomenon. Longer RR intervals represent blocked impulses from the SA node. (Sinus rhythm at a normal heart rate, sinoatrial Wenkebach, normal QRS axis, normal atrioventricular conduction time, normal ventricular conduction and repolarization.)

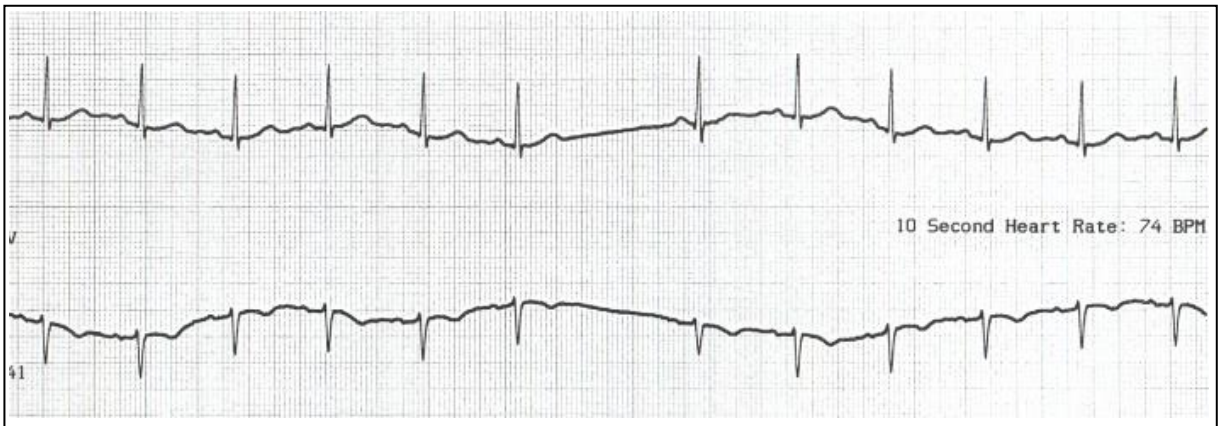


Figure 3/3. Second degree type 2 (Mobitz II) sinoatrial block Duration of the pause is just the same as the sum of two RR intervals.

3.2.3. Third degree sinoatrial block

Following regular sinus beats, no P wave appears where its occurrance is anticipated, with the duration of missed beats being longer than the sum of two normal RR intervals. This form cannot be differentiated from sinus arrest merely on the basis of the surface ECG.

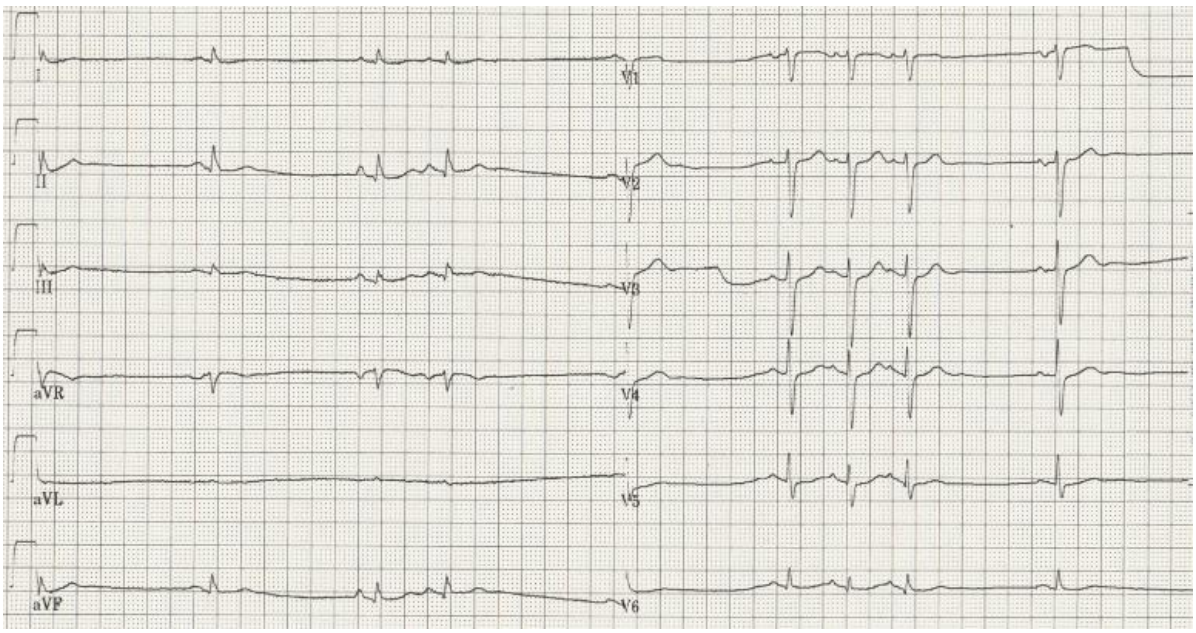


Figure 3/4.

The suspicion of 3rd degree SA block (see above for its definition) is being raised, however, the presence of frequent PACs cannot be excluded either because there is variable P wave morphology in lead V1. (Sinus rhythm, normal QRS axis, normal atrioventricular conduction times along with third degree sinoatrial block, normal ventricular conduction and repolarization.)

3.2. AV blocks

The most common cause of their occurrance is ischemic heart disease, and they particularly appear in association with inferior myocardial infarction.

Blood supply of the cardiac conduction system:

- Sinus node: it is supplied by the right coronary artery in about 50% of cases, whereas by the left circumflex coronary artery (LCx) regarding the other half;
- The AV node and the proximal portion of the bundle of His is supplied by the right coronary artery (more precisely, it is supplied by the branch providing blood supply for the inferior wall (so it may be a dominant LCx as well);
- The distal portion of the bundle of His, the right bundle branch and the left anterior fascicle are perfused by septal branches of the left anterior descending artery (LAD);
- The left posterior fascicle receives blood both from the right coronary artery and the septal branches of the LAD.

The blood supply pattern is the reason for the fact that occlusion of the right coronary artery resulting in an inferior myocardial infarction is frequently accompanied by bradyarrhythmias, while for anterior myocardial infarctions, it is only in case of a very proximal LAD occlusion that impulse conduction abnormalities occur. Beyond ischemia, common causes of AV conduction disturbances include senile myocardial fibrosis, but it may also be caused by myocarditis (e.g. Lyme disease), excessive use of some medications

(digitalis, beta adrenoceptor antagonists) and electrolyte abnormalities (K^+!). AV block presenting in young individuals at nights or in athletes is merely the consequence of increased vagal tone, without the presence of underlying structural heart disease.

With the help of an intracardiac His bundle electrogram, AV blocks, from a functional aspect, can be classified as supra-Hisian (nodal), intra-Hisian and infra-Hisian (bundle branch) blocks. The importance of this lies in the fact that supra-Hisian (AH prolongation) blocks are benign in nature, whereas intra-Hisian and infra-Hisian blocks (notching of the His bundle potential or HV prolongation) often evolve into higher degree blocks, thereby requiring pacemaker therapy more frequently. PQ=A(atrium)-H(His)-V(ventricle) - it can be recorded by intracavitary His bundle electrogram.

Delayed or blocked conduction may occur at the level of the atria - AV node - bundle of His - bundle branches. For 1st degree AV blocks, delayed conduction may appear at all four sites. Wenkebach periodicity, 2:1 AV block and 3rd degree AV block may be associated with a conduction disorder localized in the AV node, bundle of His or bundle branches. However, Mobitz type II AV blocks are always subnodal, i.e. they originate as a result of failure in the His-Purkinje system.

3.2.1. First degree AV block

Though with a prolonged conduction time, yet each sinus impulse reaches the ventricles, it is therefore often not favoured to use the designation ‘block’, and it is rather referred to as prolonged AV conduction. PQ intervals are above 0.2 (0.24) sec.

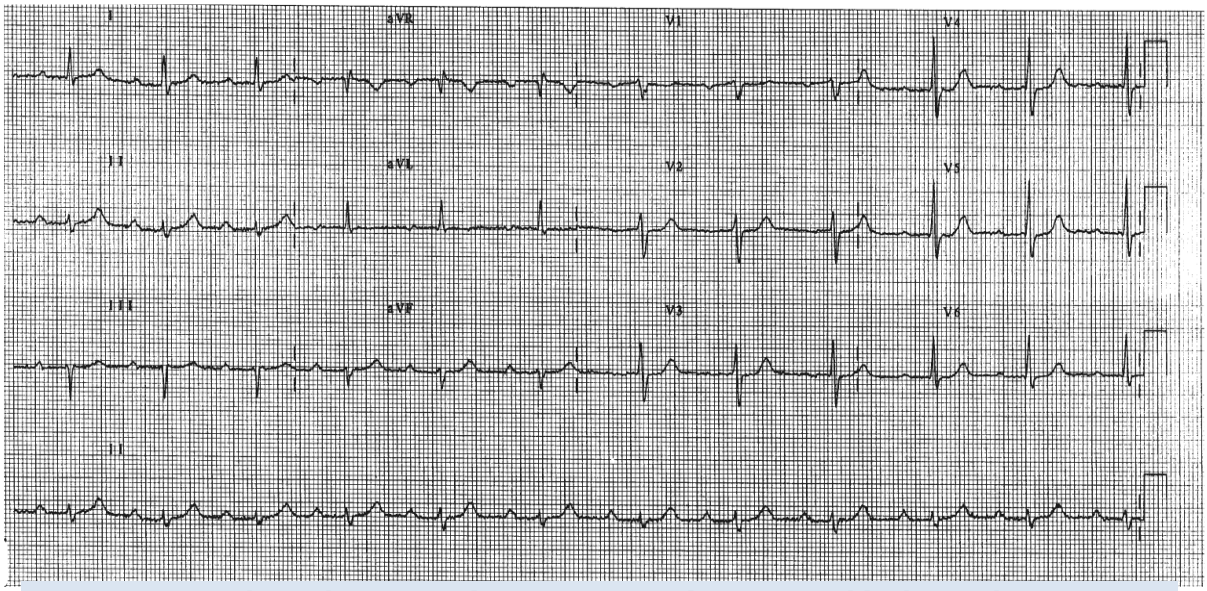


Figure 4/5. Prolonged AV conduction or 1st degree AV block. Each P wave is conducted, but with a prolonged AV conduction time. The underlying cause is old inferior myocardial infarction. Please note the Q waves in leads III and aVF (scar). (Sinus rhythm, prolonged AV conduction, left axis deviation, QS complexes in leads III, aVF(?), narrow QRS complexes, normal ventricular repolarization.)

3.2.2. Second degree AV block

Some sinus impulses are reaching the ventricles, while others are not conducted, but become blocked instead.

Its forms include:

3.2.2.1 *Mobitz type I block or Wenkebach periodicity*

It is caused by supra-Hisian (AV nodal Wenckebach) block in ¾ of cases and is a benign phenomenon that is it will not progress into a conduction abnormality of higher degree. Its ECG feature is a blocked P wave occurring after gradual prolongation of the PQ interval. *The PQ interval preceding the blocked P wave is therefore always longer than the PQ interval after the block.*

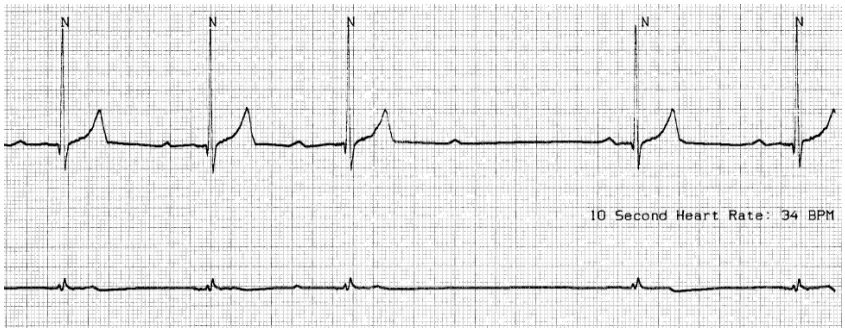


Figure 3/6. Wenkebach periodicity. Please note the progressive prolongation of the PQ interval before the block. Pre-block PQ interval > Post-block PQ interval.

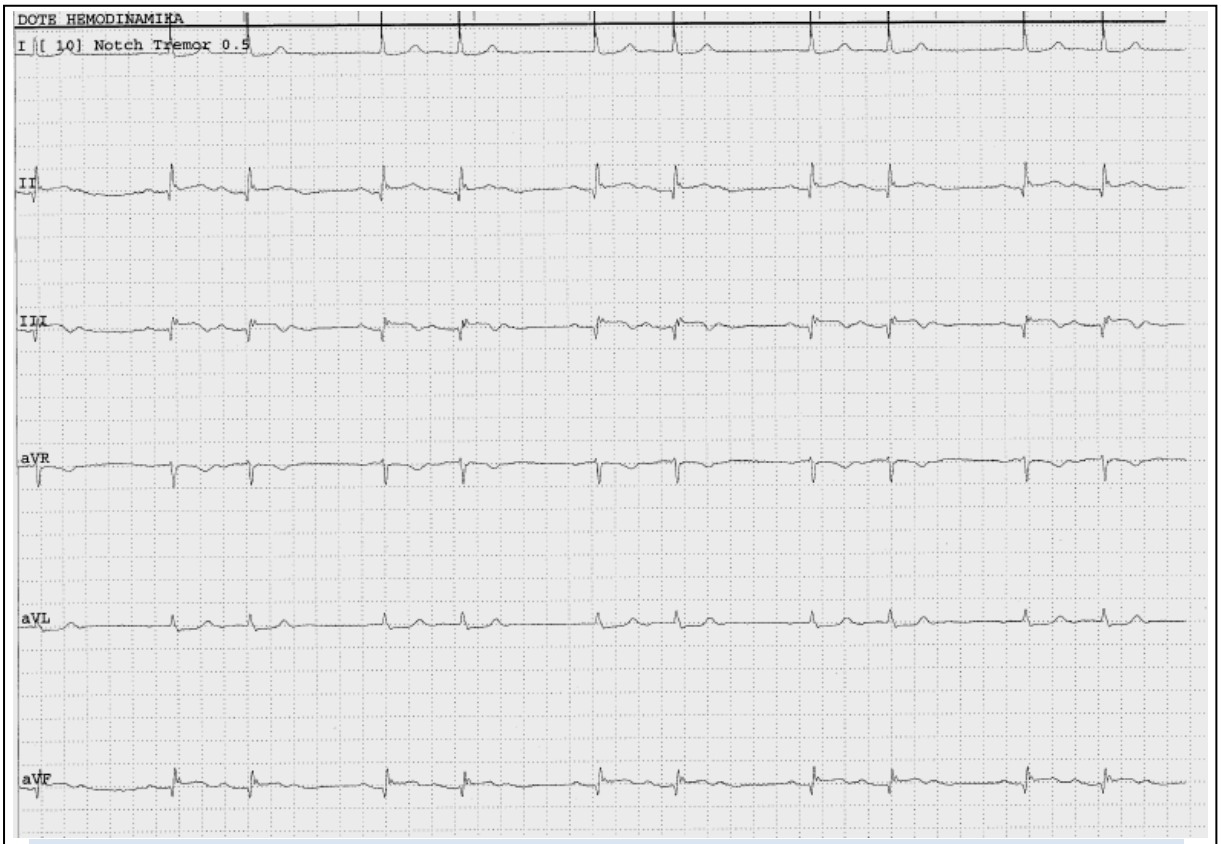


Figure 3/7. Second degree AV block (Wenkebach periodicity). Every 3rd P wave is blocked and the abnormality can mostly be tracked in lead III. (The underlying cause of the abnormality is inferior myocardial infarction caused by occlusion of the right coronary artery providing the AV nodal artery - slight ST segment elevation is still visible in leads II, III and aVF.)

The difference between the shortest (usually ≥ 200 ms) and longest PQ interval is ≥ 100 ms. Characteristics of the more rarely occurring Wenckebach periodicity of the His-Purkinje system include that it is

accompanied by a bundle branch block QRS pattern, with a shortest PQ of ≤0.16 sec and a change in the PQ intervals of ≤50%.

3.2.2.2. *Mobitz type II block*

It is always caused by intra-Hisian or infra-Hisian block, i.e. it is subnodal in location. Its characteristic feature is an 'unexpected' block of P waves without any progressive prolongation of the PQ interval. The PQ interval may be normal or prolonged, but it is typically constant. The PQ interval preceding the blocked P wave is identical with the PQ interval measured after the block.

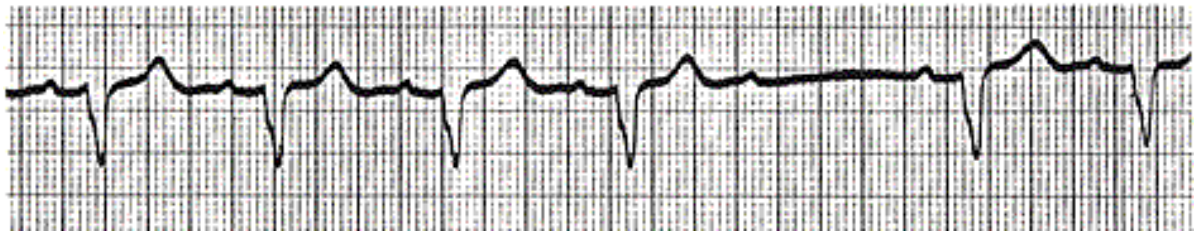


Figure 3/8. Mobitz type II AV block. Please note the absence of PQ prolongation before the blocked P wave, i.e. PQ intervals before and after the blocked P wave are identical in duration.

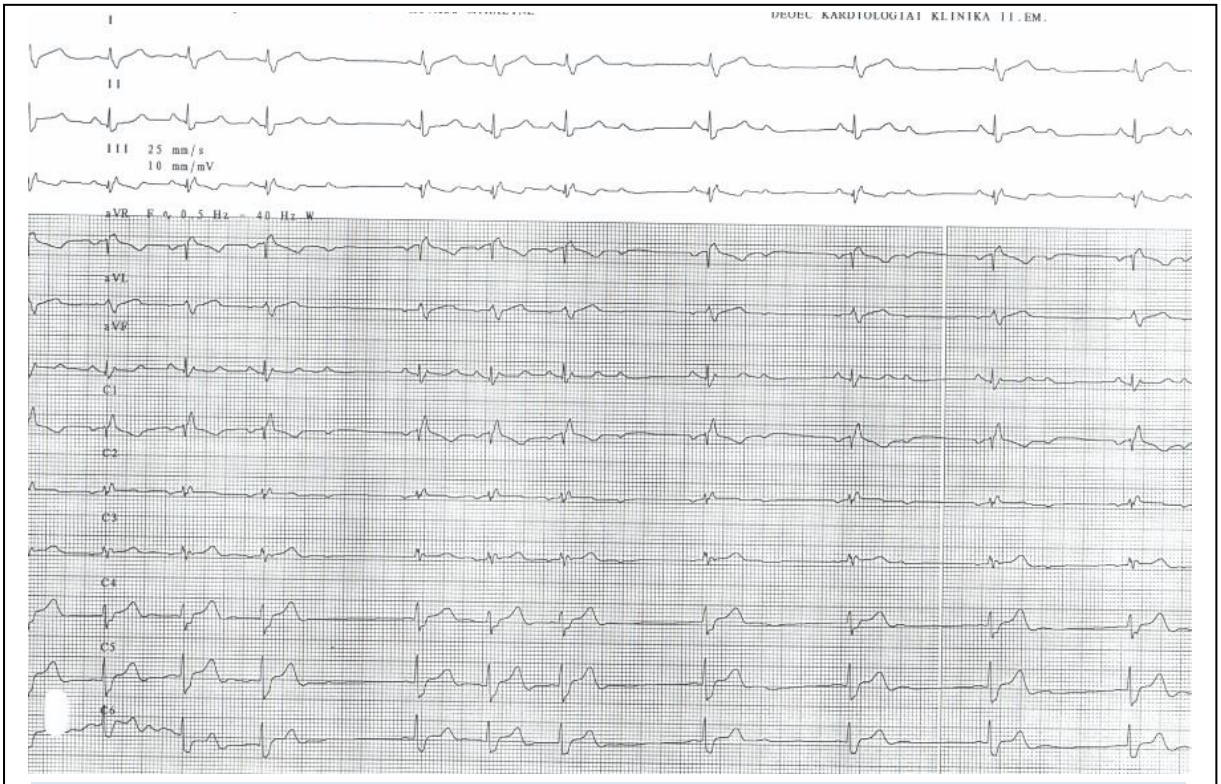


Figure 3/9. Mobitz type II AV block, continuing as 2:1 AV block. The block is most probably at the subnodal level (the PQ interval of the conducted beats is normal, but the QRS complexes show a right bundle branch block pattern), therefore it is an indication for pacemaker implantation. (Sinus rhythm, normal QRS axis, Mobitz type II and 2:1 AV block, right bundle branch block, secondary repolarization abnormalities.)

Narrow QRS complexes render the presence of an intra-Hisian conduction disturbance probable, while wide QRS complexes imply that of an infra-Hisian block. It frequently occurs during Holter monitoring that one considers the presence of several types of 2nd degree AV block probable within a single recording; however, the chances of a patient having concurrent Mobitz type I and II AV block are very low. Therefore, if classical Wenkebach periodicity, or possibly 2:1 AV block, is recorded during the Holter monitoring on several occasions, the slight variations in block morphology in various parts of the recording should not be interpreted as a Mobitz type II block, because it has serious therapeutic consequences.

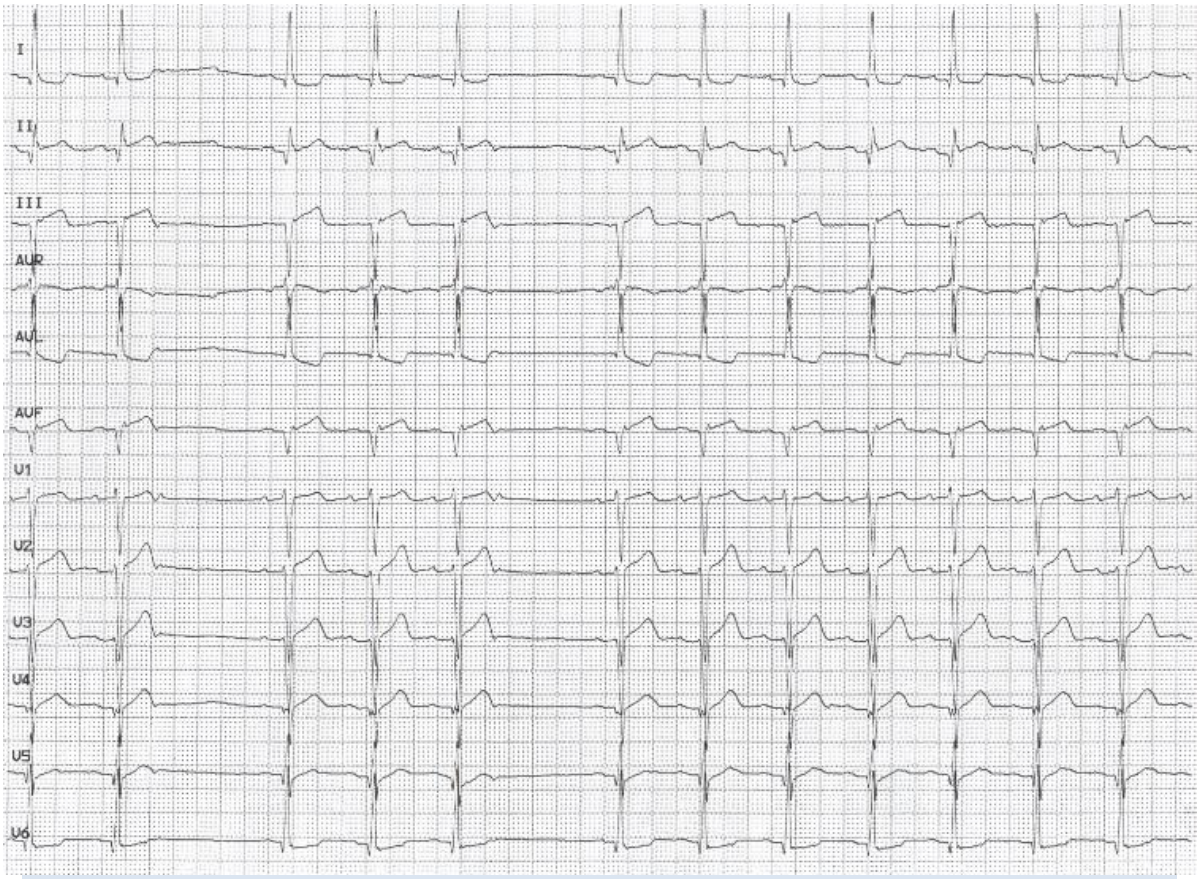


Figure 3/10. Pseudo-Mobitz type II AV block. Blocked P wave is observable (in lead V1) after the 2nd and 5th QRS complexes, but those P waves do not arrive in accordance with the normal sinus cycle length, but earlier, so they are blocked atrial premature beats actually. The underlying cause of the conduction abnormality is subacute inferior myocardial infarction. (Sinus rhythm, left axis deviation, narrow QRS complexes, normal AV conduction time, Q waves, ST segment elevation and biphasic T waves in leads II, III and aVF, reciprocal ST segment depression in leads I, aVL, blocked PACs.)

3.2.2.3. *2:1 AV block*

Every other P wave is blocked, therefore it is impossible to say anything about PQ prolongation or whether or not it is constant. The site of block may be in the AV node or infranodally as well.

Since nodal blocks have a better prognosis than infranodal ones, let's therefore see some characteristics on the surface ECG for their distinction:

- if the PQ≥0.28 ms, it is AV nodal block;
- if the PQ≤0.16 ms, it is a block in the His-Purkinje system;

- if the QRS complexes are narrow, it is a block in the AV node or bundle of His;
- if the QRS complexes are wide, the block may be anywhere, but it is usually a pathology of the bundle branches;
- blocks originating from the AV node are improved by atropine and physical exercise, while impaired by carotid sinus massage and, on the contrary;
- subnodal blocks are impaired by atropine and physical exercise, while improved by carotid sinus massage.

	NODAL	SUBNODAL
Atropine	Improves	Impairs
Physical exercise	Improves	Impairs
Carotid sinus massage	Impairs	Improves
PQ	≥0.28 ms	≤0.16 ms
QRS	Narrow	Wide (narrow in bundle of His blocks)

Table 3/1. Differentiation between nodal and subnodal AV blocks.

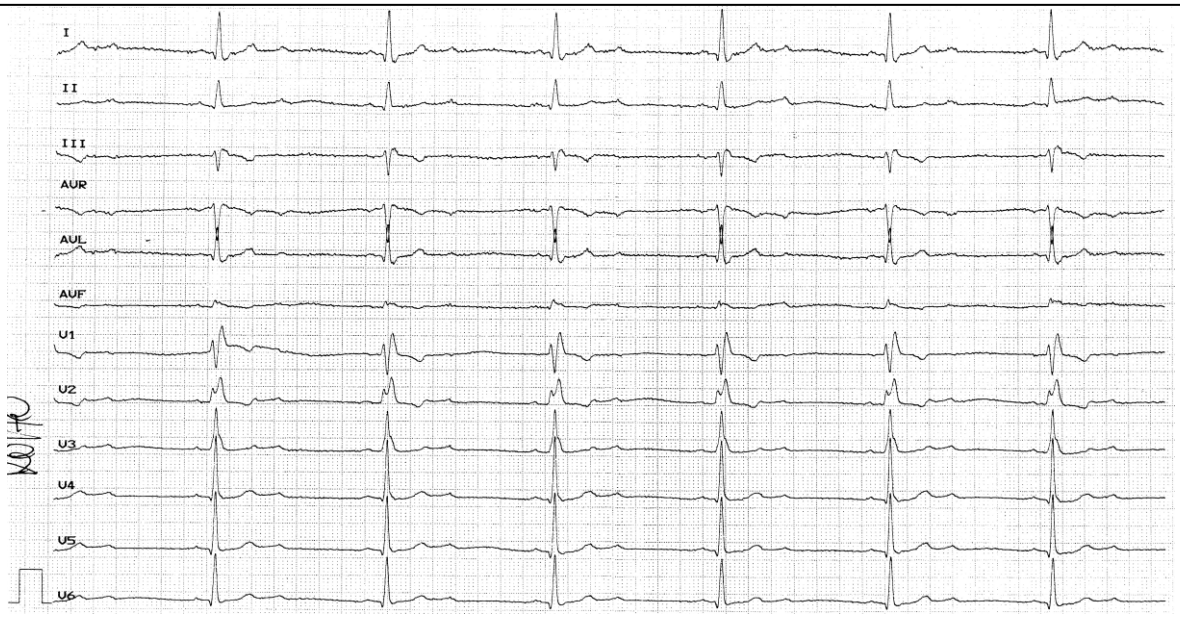


Figure 3/11. 2:1 AV block. The right bundle branch block pattern and the normal AV conduction time support an infra-Hisian origin, so it is an absolute indication for pacemaker implantation. (Sinus rhythm, 38 bpm, normal QRS axis, 2:1 AV block, right bundle branch block, secondary repolarization abnormalities.)

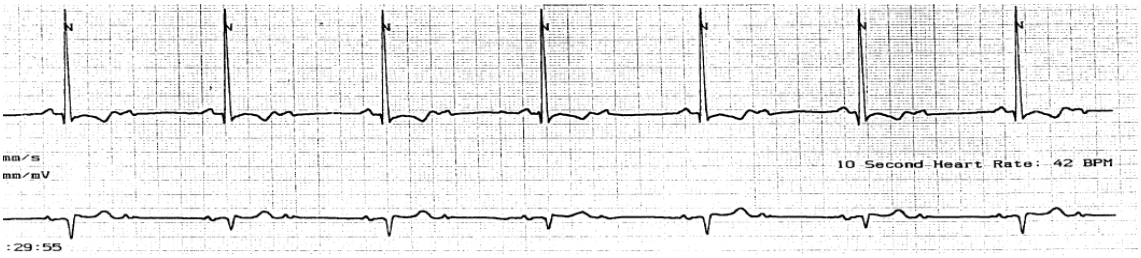


Figure 3/12. 2:1 AV block. Please note the P waves occurring after the T waves, not followed by a QRS complex. (Differential diagnosis (DDx): U waves! However, they have a different morphology and P-to-U distance ≠ U-to-P distance, while P-P intervals are constant.)

3.2.2.4. *High grade AV block*

Several consecutive P waves are blocked, but there is no complete AV block since conducted beats also occur after the block. It is generally a malignant phenomenon and has a predisposition for progression.

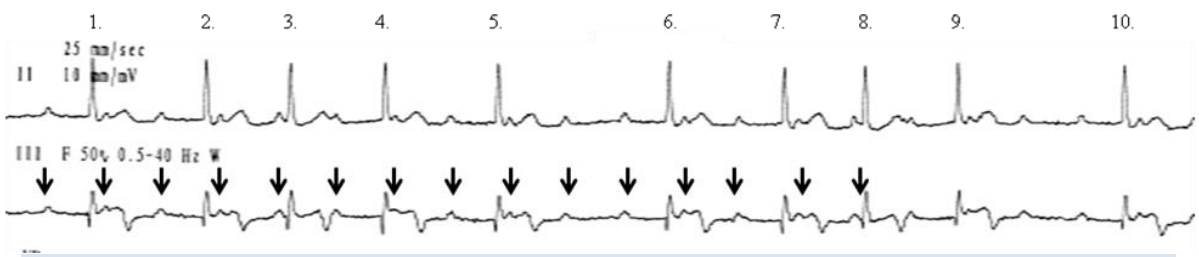


Figure 3/13. High grade AV block associated with inferior myocardial infarction. The 1st, 2nd, 4th, 5th, 6th, 7th, 9th and 10th QRS complexes are conducted beats. The 3rd and 8th QRS complexes are likely to be junctional escape beats and several blocked consecutive P waves are visible between the 5th and 6th as well as 9th and 10th beats. P waves are indicated by arrows.

3.2.3. Third degree AV block

There are no conducted supraventricular impulses. Complete electrical dissociation of the atria and ventricles is observable. In this case, the impulses controlling the atria and ventricles are different from each other, with the former one controlled by sinus impulses and the latter one by a junctional or ventricular escape rhythm.

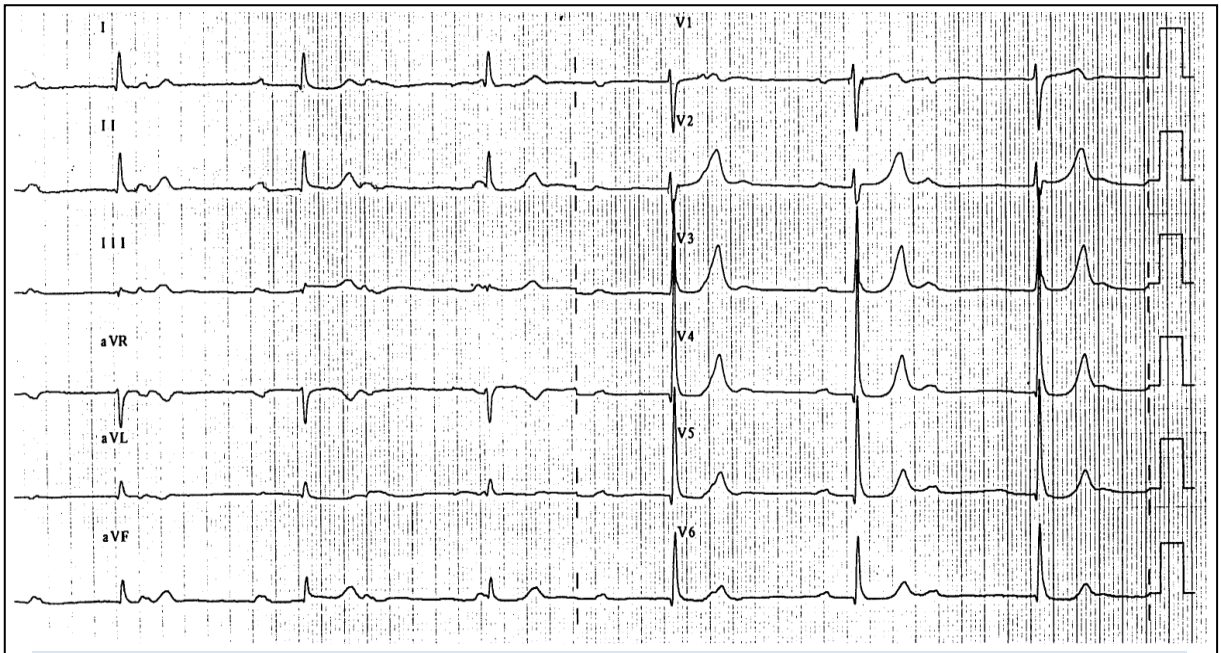


Figure 3/14. Third degree AV block with junctional escape rhythm. Please note the P waves occurring in lead II and that there is no regularity or correlation between the occurrance P waves and QRS complexes (complete AV dissociation). It is the narrow QRS complexes and a heart rate of 40 bpm that indicates junctional origin of the escape rhythm. (Sinus rhythm, 40 bpm, normal QRS axis, 3rd degree AV block, junctional escape rhythm, normal ventricular repolarization.)

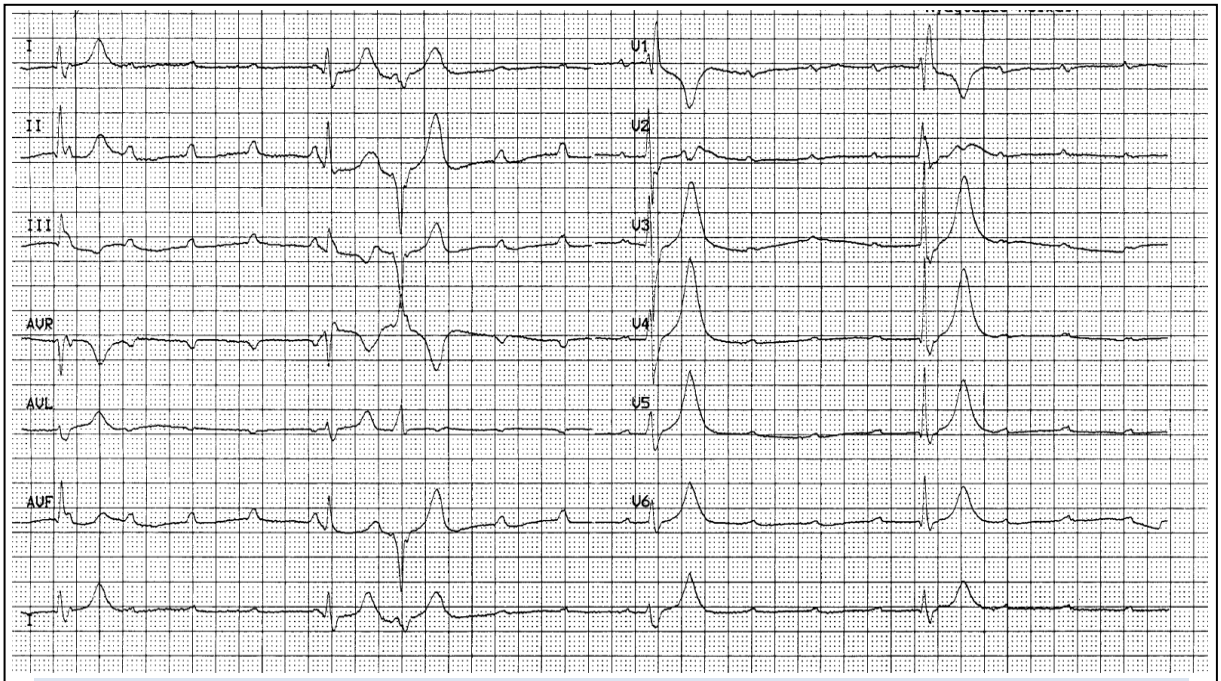


Figure 3/15.

Third degree AV block with ventricular escape rhythm. The 3rd QRS complex is not a ventricular escape beat, but a ventricular premature beat. (Sinus rhythm, 3rd degree AV block, ventricular escape rhythm at a rate of 30 bpm, a VPB.)

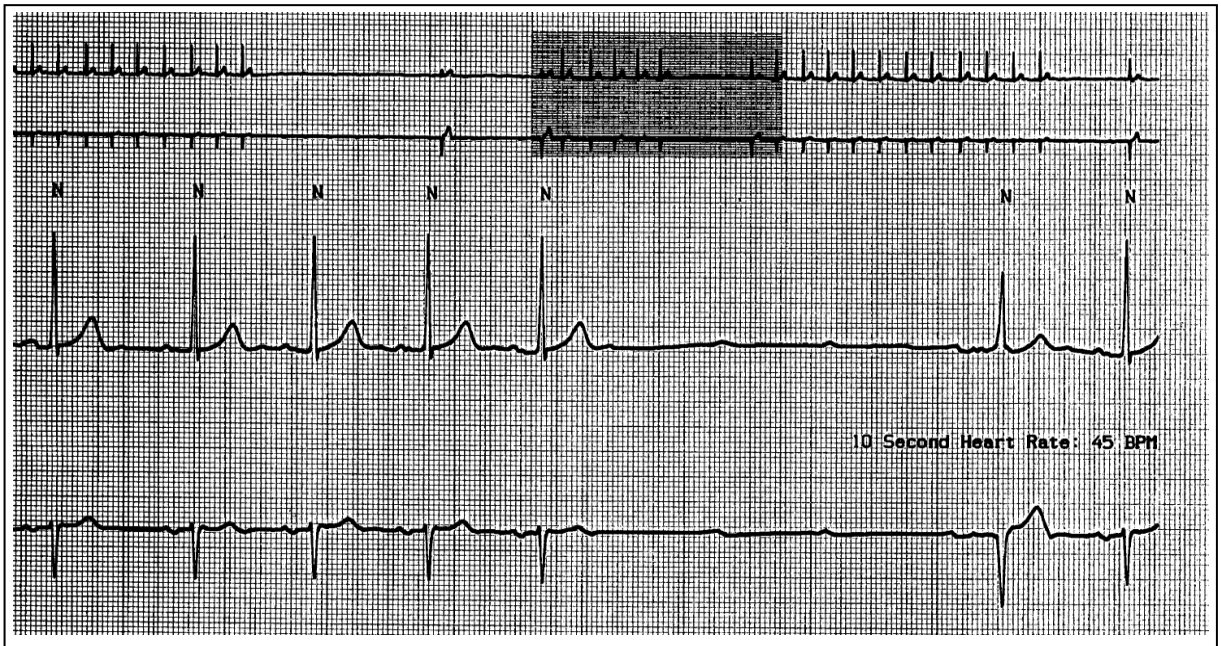


Figure 3/16.

Paroxysmal AV block occurring after a blocked premature atrial complex (it is caused by phase 4 block of the action potential.)

3.3. Intraventricular conduction disturbances

The bundle of His penetrating into the interventricular septum, after a short course, splits into two bundle branches; moreover, the left bundle branch subdivides into an anterior and posterior fascicle. Block or delayed conduction may develop anywhere in this system, thereby leading to intraventricular conduction disturbances. Bundle branch blocks always prolong the QRS duration (≥120 ms), while hemiblocks (fascicular blocks) do not, they rather result in a change in the electrical axis of the heart (QRS<120 ms).

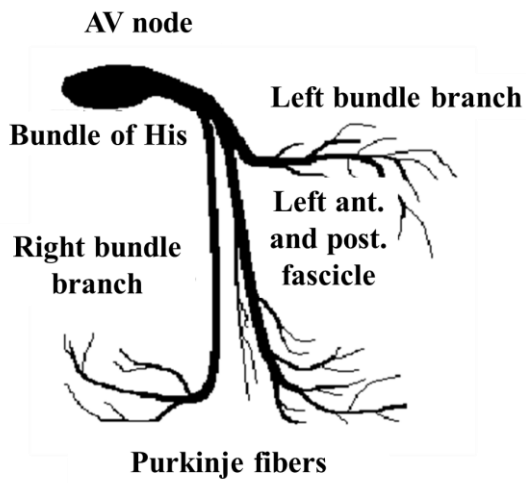


Figure 3/17.

The AV junction and the ventricular conduction system.

3.3.1. Left anterior fascicular block (LAFB)

It is a frequent abnormality, the reason for which is that the anterior fascicle is thinner and more vulnerable. Sometimes it occurs even in completely healthy individuals. Congenital malformations (e.g. ASD), hypertension (left ventricular hypertrophy, damage caused by fibrosis) and left ventricular dilatation may have a role in its development. The left anterior fascicle takes part in carrying the electrical impulse to the anterosuperior region of the left ventricle, so it is this region in LAFB that is activated for the last time. This is why LAFB results in abnormal left axis deviation (QRS between -30° and -90°), the signs of which are rS complexes in leads II, III, aVF and tall R waves in leads I, aVL. The QRS complexes remain narrow. It is important to distinguish LAFB from other causes of left axis deviation; it is mostly the ECG signs of an old inferior myocardial infarction with necrosis that results in a similar appearance. In a lot of cases, their distinction is only possible with the help of concurrent echocardiography.

3.3.2. Left posterior fascicular block (LPFB)

Since the posterior fascicle is thicker and less vulnerable (having a dual blood supply from the LAD and RCA), isolated LPFB is therefore a very rare condition, rather occurring together with RBBB. The left posterior fascicle takes part in carrying the electrical impulse to the posteroinferior region of the left ventricle, so it is this region in LPFB that is activated for the last time. This is why abnormal right axis deviation (QRS axis > 120°) can be observed on the ECG in LPFB, with the amplitude of R waves in lead III exceeding that observed in lead II. The QRS complexes are narrow. qR complexes are observable in leads II, III and aVF, while rS complexes in leads I and aVL (just the opposite of LAFB). Lead misplacement (reversal) of the right and left arm electrodes

results in an ECG appearance similar to that of LPFB, however, in such cases, an examination of the axis (polarity) of P waves may give a clue. The sample ECG also demonstrates such a case, because lead misplacement is more common than real LPFB.

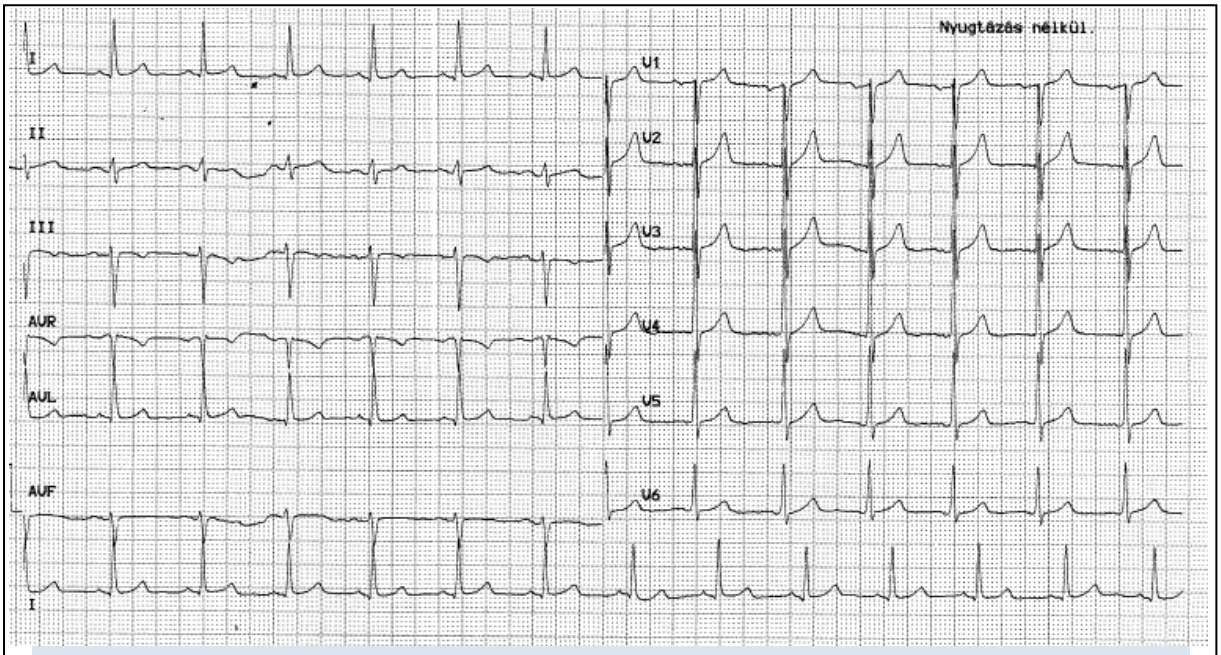


Figure 3/18. Left anterior fascicular block. Left axis deviation - tall R waves in leads I, aVL, while rS complexes in leads II, III, aVF. Note that the QRS complexes are narrow. (Sinus rhythm, 80 bpm, normal AV conduction time, left anterior fascicular block, narrow QRS complexes, normal ventricular repolarization.)

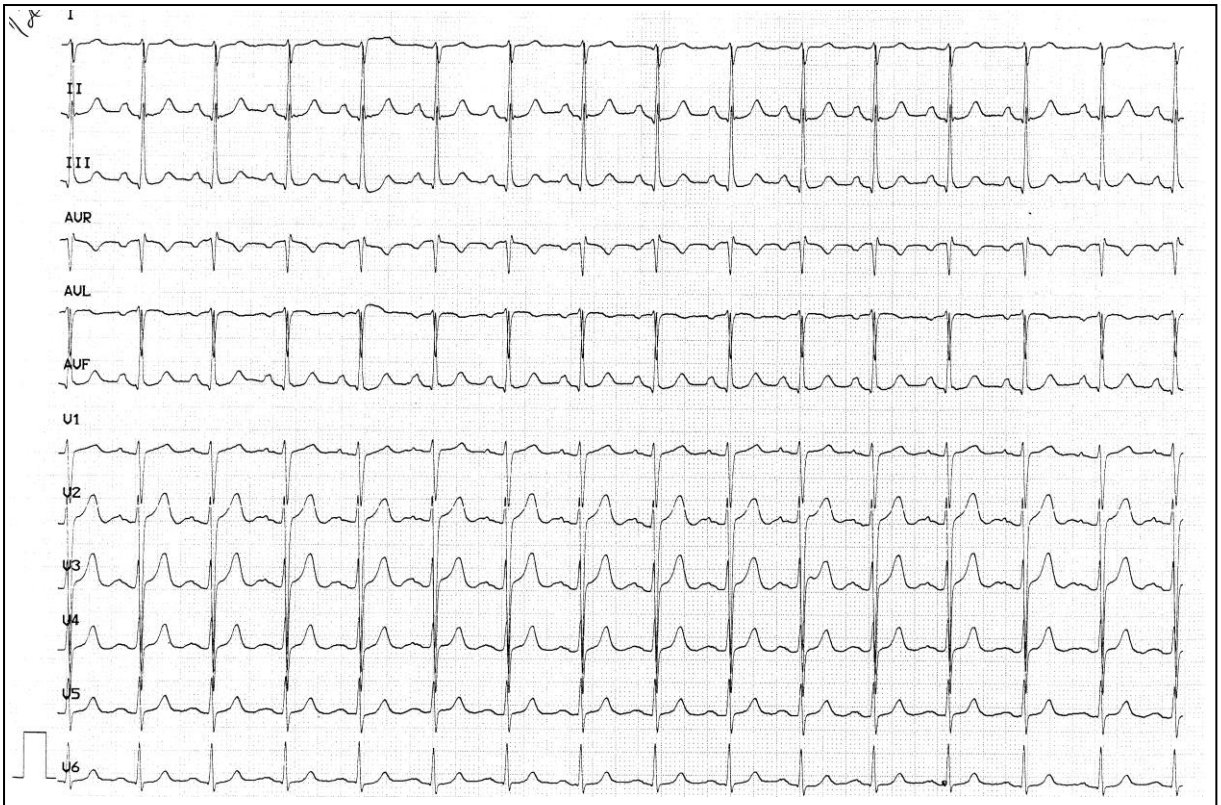


Figure 3/19. 'Pseudo left posterior hemiblock' caused by reversal of the right and left arm electrodes. It is the negative P waves in lead aVL giving one a clue that it is only a technical problem, and not real LPFB.

	II, III, aVF	I, aVL
BAH	rS	Rs
BPH	Rs	rS

Table 3/2. Distinction of LAFB and LPFB by studying the limb leads. Take notice of the fact that the two abnormalities are mirror images of each other, because the difference of the two QRS axes is appr. 180 degrees.

3.3.3. Left bundle branch block (LBBB)

It is a common type of intraventricular conduction disturbances, the underlying cause of which is structural damage of the left ventricle in 90% of cases and the absence of underlying left ventricular dysfunction is rarely seen. Its causes include ischemic heart disease, *cardiomyopathy* (dilated cardiomyopathy), left ventricular hypertrophy and severe cardiac valve disease. Note that the QRS *becomes widened*, i.e. ≥120 ms. The wider the left bundle branch block pattern, the more severe the underlying disease and the worse the prognosis. For a QRS width above 150 ms, it is rarely that a structurally normal heart can be found, with the left ventricular dysfunction generally being severe above a width of 180 ms. All three previously mentioned parts of the QRS complex take part in the widening process; the entire QRS complex is blocked. Wide and notched R waves are visible in leads I, aVL and V5-6, with an absence of small q waves. The explanation for the latter phenomenon should be sought in the activation sequence of the interventricular septum, which is not left-to-right for LBBB, but the left ventricle rather becomes activated from the right ventricle, resulting in an opposite, right-to-left septal activation pattern.

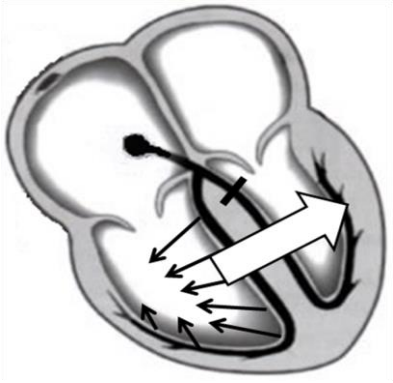


Figure 3/20.

For LBBB, the right ventricle becomes activated from the right bundle branch as normal, while the left ventricle and the interventricular septum are activated from right to left.

.

Note that the dominant deflection of the QRS complex in lead V1-2 is negative. It is easy to remember that if the QRS complexes are wide, you should always look at lead V1 first. If negative deflections (i.e. QS complexes) are seen in lead V1, you can almost be sure that this is LBBB. If this simple rule won't help, measure the ventricular activation time (VAT) in lead V6, which will be prolonged (≥50 ms) here in LBBB. For inexperienced ECG interpreters, it may occur that QRS complexes are falsely interpreted as narrow, because they examine only a few leads and the QRS duration indeed appears to be normal there; it is therefore important to examine the QRS complexes in all of the leads. It is very important to know that in LBBB the abnormal sequence of depolarization alters the sequence of repolarization as well; this is why discordant ST-segment–T-wave configurations develop,

due to which it is extremely difficult, sometimes even impossible, in LBBB to recognize the ECG signs of an acute myocardial infarction or ischemia. It is simply mentioned during the interpretation that *secondary ST segment and T wave abnormalities* have been detected. The secondary ST segment elevation in leads V1-3 is not a consequence of myocardial infarction, and the ST segment depression in leads I, aVL and V5-6 is not indicative of myocardial ischemia either. It is mentioned in several ECG books that acute presentation of an LBBB in the setting of chest complaints should be evaluated as a sign of myocardial infarction; however, blood supply of the left bundle branch, due to the dual blood supply of the left posterior fascicle, may rarely be injured in a way to result in an LBBB, so it has only a theoretical significance.

Cardiac resynchronization therapy (CRT) of heart failure with a pacemaker targets patients with LBBB, because there is a temporal delay in the activation of the septal and lateral wall in these patients, thereby impairing the efficiency of left ventricular ejection. Resynchronization therapy is not efficacious in about 30% of cases (non-responders). It appears that the wider the LBBB, the more effective the CRT. To decrease non-response to CRT, stricter criteria for LBBB (Strauss LBBB criteria) have been introduced recently, which indicate the presence of LBBB with maximum specificity and differentiate it from nonspecific intraventricular conduction delay, which is often caused by LAFB and left ventricular hypertrophy or by myocardial scarring.

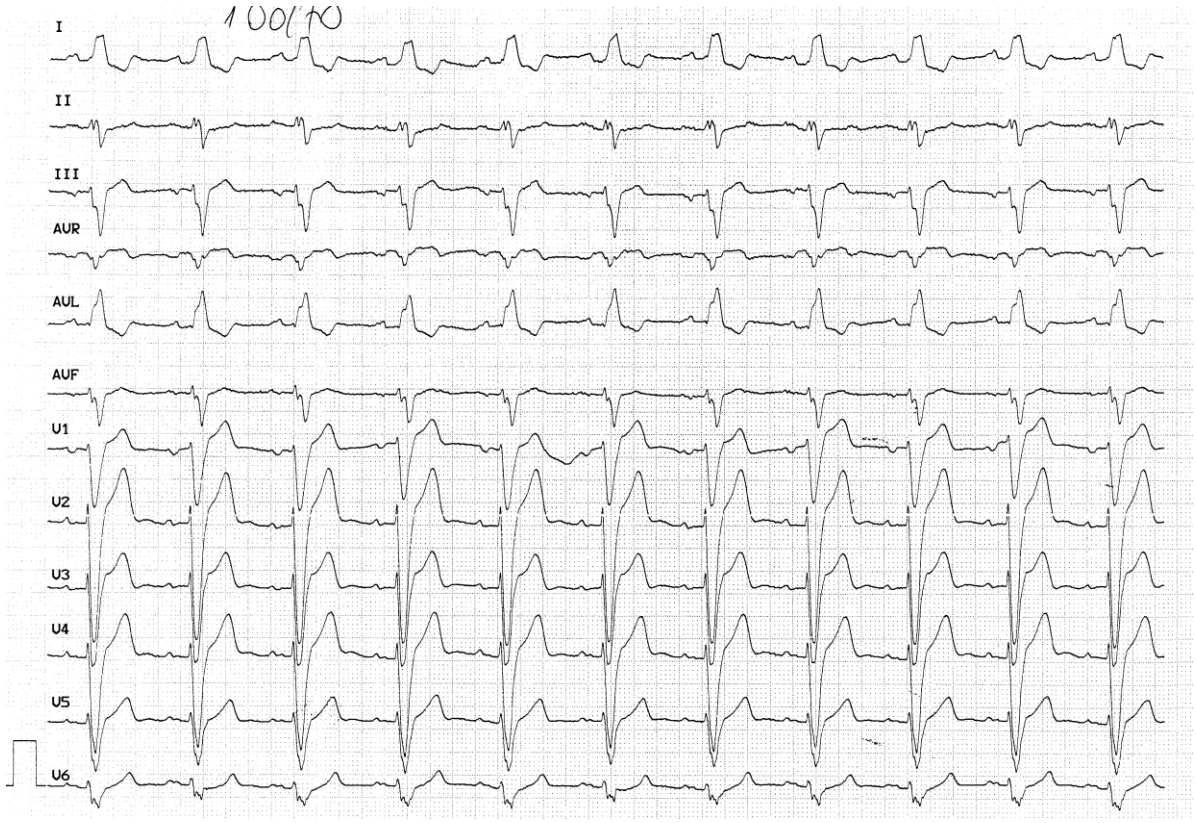


Figure 3/21. Typical left bundle branch block (also based on Strauss LBBB criteria). The QRS complexes are wide and the dominant deflection in V1 is negative. Please note that significant downsloping ST segment depression is visible in leads I, aVL as well as marked ST segment elevation in leads V1-4. These are only caused by the bundle branch block, so they are secondary repolarization abnormalities. (Sinus rhythm, 70 bpm, normal AV conduction time, left bundle branch block, secondary repolarization abnormalities.)

Strauss LBBB criteria are as follows:

1. QRS ≥ 140 ms (males) and QRS ≥ 130 ms (females);

2. mid-QRS slurring at 40 ms from the beginning of the QRS in at least two of the following leads: I, aVL, V1-2, V5-6.

By using these criteria, QRS widening is caused by ‘false LBBB’ in about 1/3 of cases interpreted as an LBBB. Recognizing cases with ‘false LBBB’ does not seem insignificant, since it may have importance in the screening of non-responders to CRT prior to pacemaker implantation. The term ‘incomplete LBBB’ should be avoided because the presence of such an entity is difficult to imagine; and it is rather due to conduction slowing caused by LAFB and left ventricular hypertrophy or by myocardial scarring even in this case.

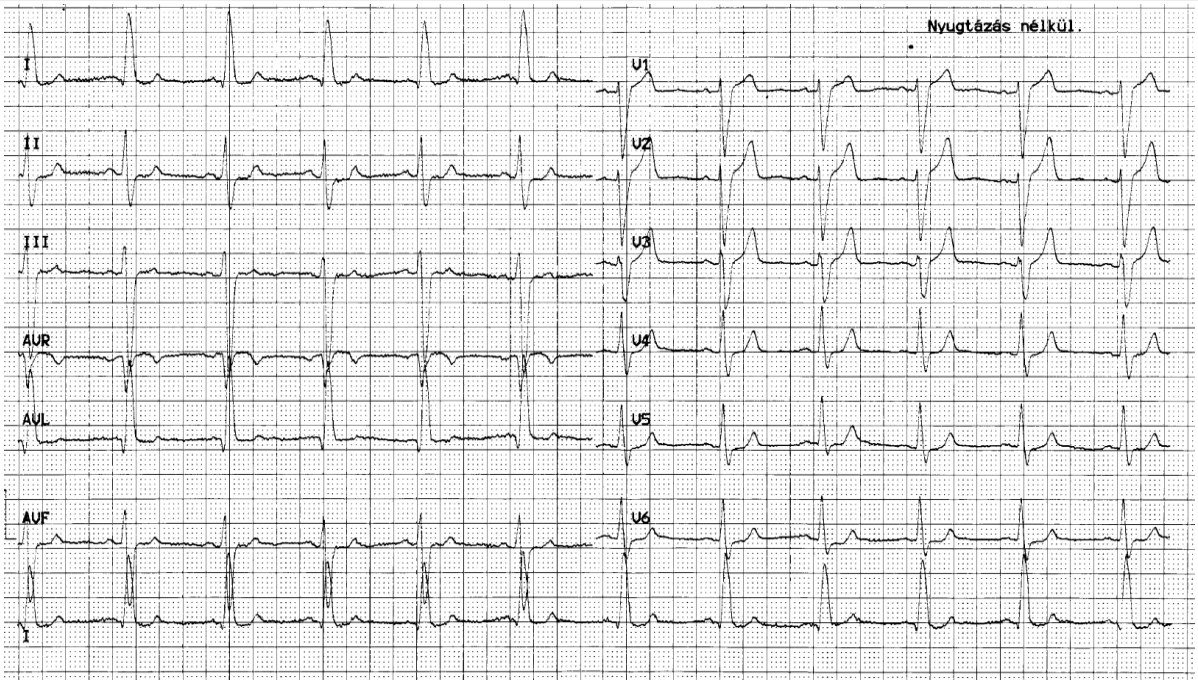


Figure 3/22.

Left bundle branch block (but it isn’t based on Strauss LBBB criteria; there is no mid-QRS slurring). The QRS complexes are wide with a dominantly negative deflection in lead V1, but the repolarization abnormality is not pronounced in this case because the QRS complexes are not very wide either. (Sinus rhythm, 80 bpm, normal AV conduction time, left bundle branch block (or nonspecific intraventricular conduction disturbance), secondary repolarization abnormalities.)

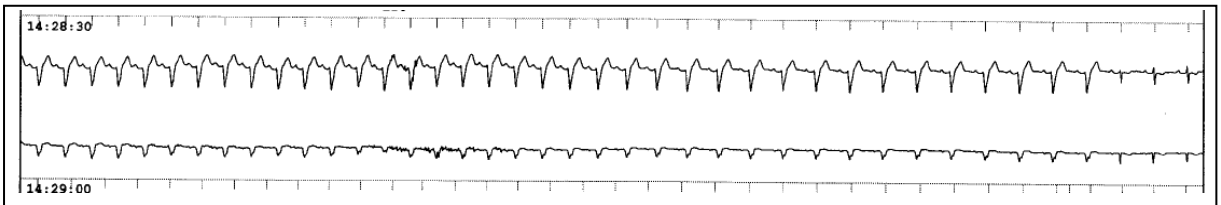


Figure 3/23.

Intermittent (rate-dependent) LBBB. Sinus rhythm, with the last 3 beats having a narrow QRS complex. (Holter recording.)

3.3.4. Right bundle branch block (RBBB)

Its presentation does not always refer to a pathological condition; however, the most common causes include pulmonary embolism (especially the occurrance of acute RBBB in a medical condition associated with chest complaints), chronic lung disease (cor pulmonale), atrial septal defect (ASD), ischemic heart disease and cardiomyopathy.

Note that the QRS becomes widened, i.e. ≥ 120 ms. However, the initial portion of the QRS complex is narrow as normal ('unblocked portion'), because septal and left ventricular activation take place in the usual sequence. There is a delay in right ventricular activation due to the bundle branch block, resulting in the widening of the last third of the QRS complex and thereby widening the entire QRS complex.

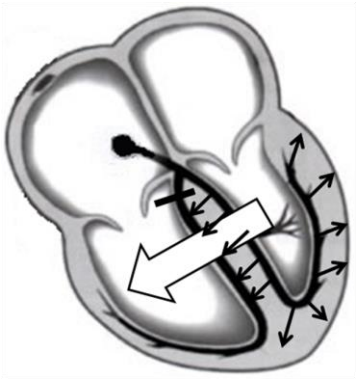


Figure 3/24.

In RBBB, septal and left ventricular activation take place in the usual sequence, due to which the initial third of the QRS complex will be narrow; moreover, right ventricular activation will be delayed, thereby widening the end of the QRS complex.

RBBB also causes the QRS complex to become widened, although this is not unambiguous at first glance and in each lead. On investigation of the QRS width, it is therefore advisable to look at several leads.

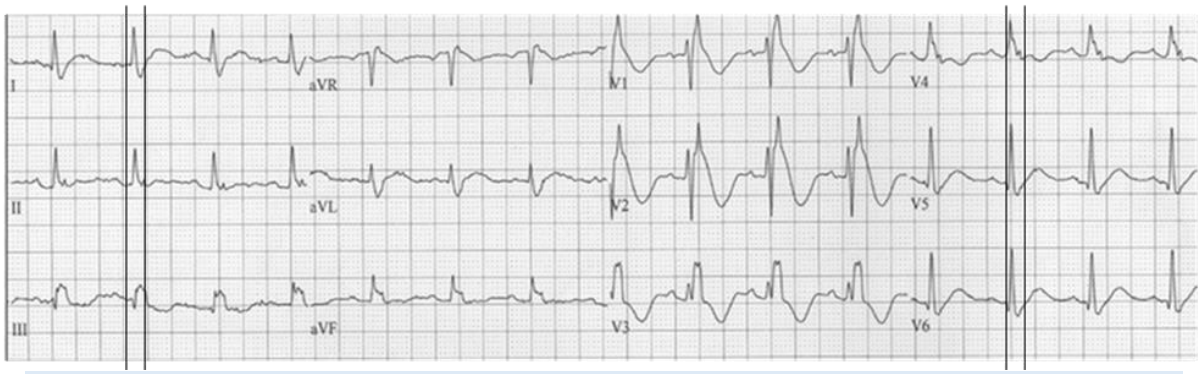


Figure 3/25.

Please note leads II, aVR and, possibly, V5-6, in which QRS complexes are seemingly narrow. Identical points of the unequivocally wide QRS complexes above or below these leads are located on the isoelectric line in these leads, this is why they appear to be narrow.

The above rule can also be applied for RBBB that is if QRS complexes are wide, look at lead V1 first. If *there are positive QRS deflections in lead V1*, it is RBBB. If it does not help either, one must check ventricular activation time in lead V1, which is prolonged in

RBBB (≥30 ms). In lead V1, a QRS configuration resembling the letter ‘M’ (rSR’, formerly called Wilson type RBBB) is often visible, while wide S waves are seen in leads I, aVL, V5-6. In lead V1, the QRS complexes may have several shapes for RBBBs with a different level of the block or developing in various diseases.

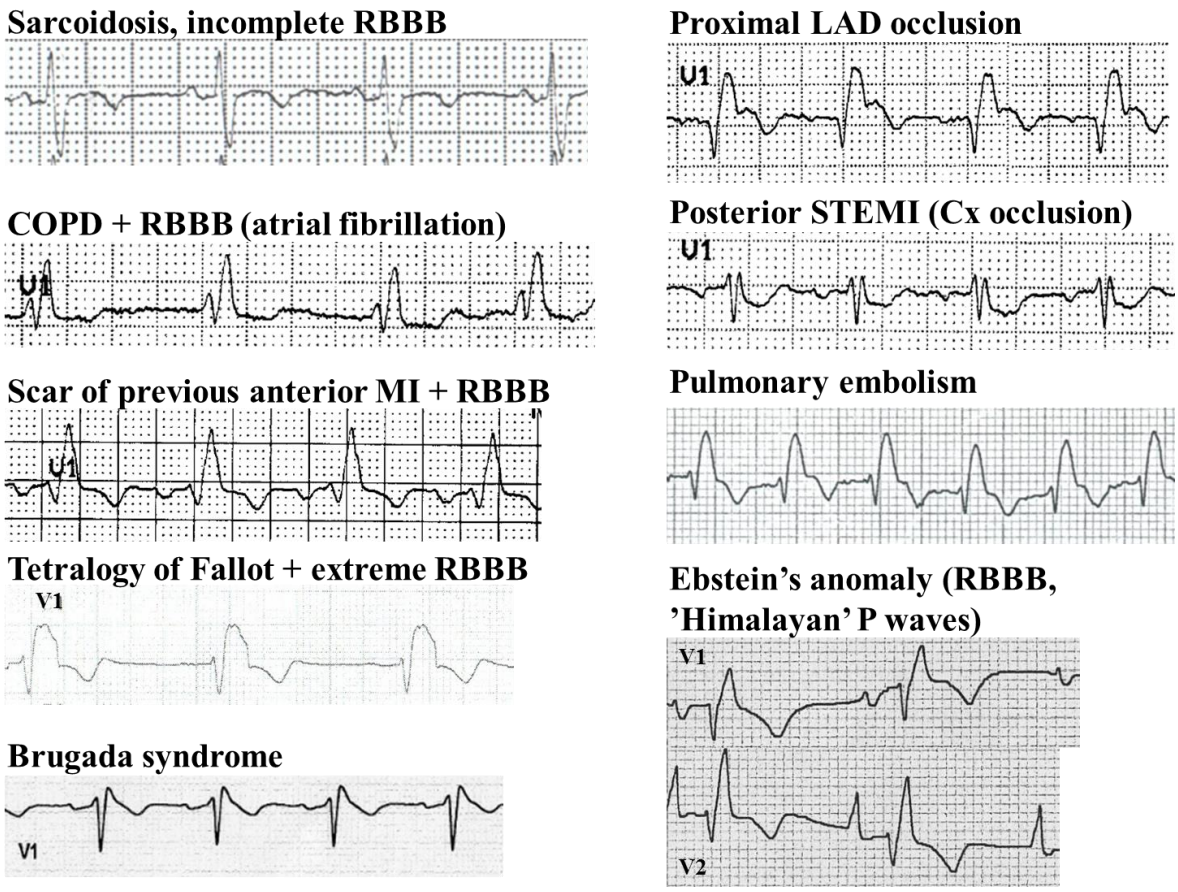


Figure 3/26.

The presence of RBBB may accompany numerous diseases, of which the ECG pattern in lead V1 of the most important diseases are shown on this figure. In most of the cases, blood supply of the bundle branch is deteriorated or right ventricular structural damage or conduction delay for other reasons is the underlying cause of the block.

Secondary ST segment abnormalities in RBBB are far less pronounced than in LBBB, so it is easier to recognize ischemic signs and myocardial infarction (anterior and inferior) for this type of bundle branch block.

RBBB (+LAFB) develops in 6% of anterior myocardial infarctions, which is particularly true for proximal LAD occlusions with an involvement of the first septal branch, because this branch provides blood supply for the right bundle branch. RBBB associated with an anterior myocardial infarction is a poor prognostic sign because it reflects a proximal coronary occlusion.

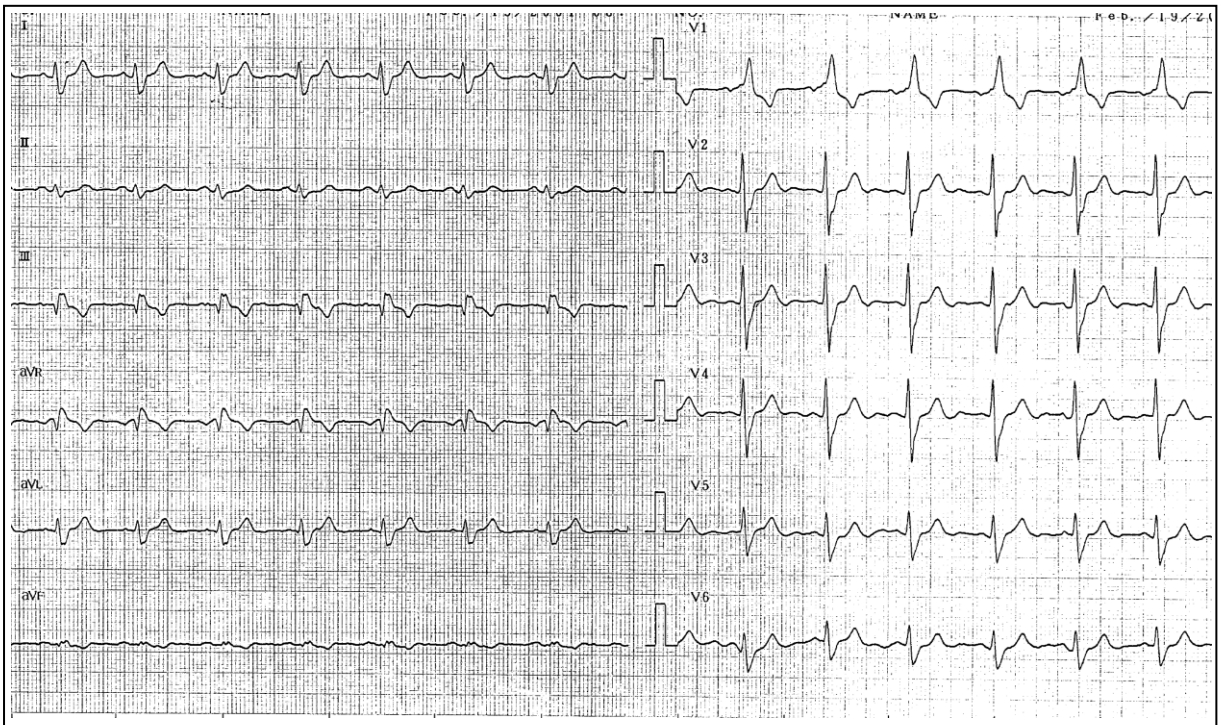


Figure 3/27. Right bundle branch block. The QRS complexes are wide and positive deflections (R waves) are observable in lead V1. (Sinus rhythm, 75 bpm, extreme right axis deviation, normal AV conduction time, right bundle branch block, secondary repolarization abnormalities.)

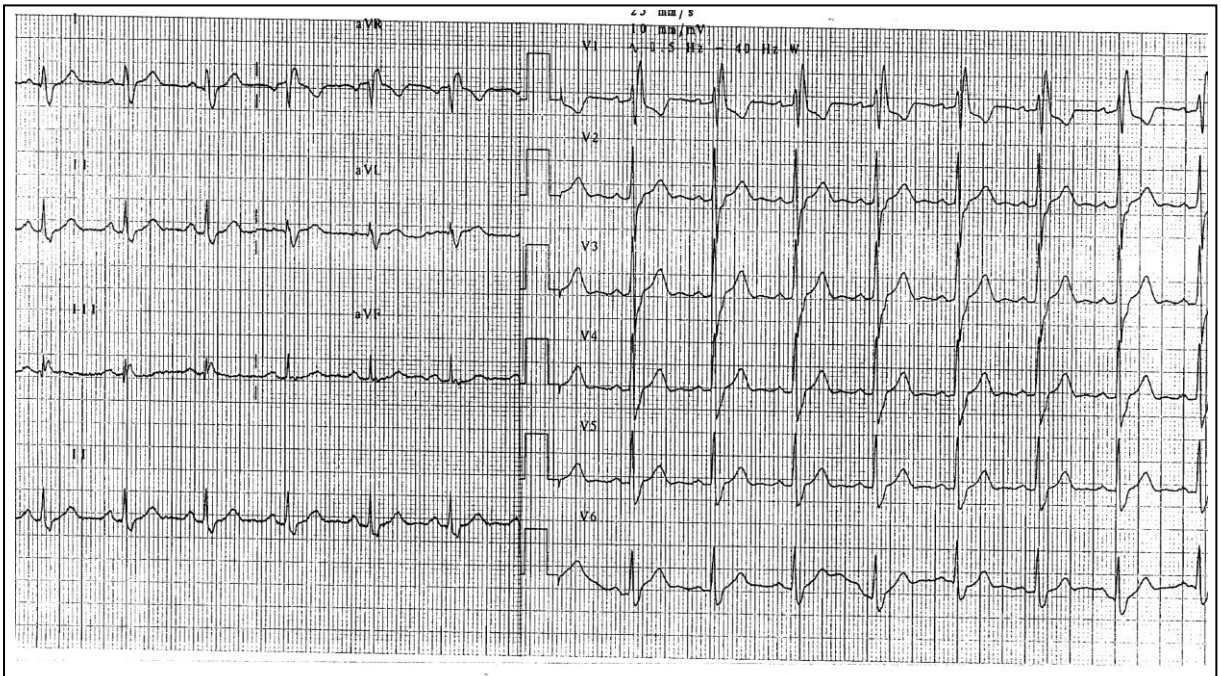


Figure 3/28. Right bundle branch block. The QRS complexes are wide and dominantly positive deflections (rSR') are observable in lead V1. (Sinus rhythm, 80 bpm, normal QRS axis, normal AV conduction time, right bundle branch block, secondary repolarization abnormalities.)

Incomplete RBBB is defined as a QRS morphology (rSr' pattern) reminding of an RBBB, but the QRS width is between 100-120 msec. If the underlying cause of this persists, it predicts the occurrance of complete RBBB.

3.3.5. Combined blocks

3.3.5.1. bifascicular block

- RBBB + LAFB (this is a frequent and usually harmless condition, also called Lenègre syndrome, and it often indicates the presence of ostium primum ASD, especially in young individuals, but can also be seen after an anterior myocardial infarction);
- RBBB + LPFB (this is a rare, but dangerous form, which may indicate the presence of ostium secundum ASD);
- bundle branch block + first degree AV block.

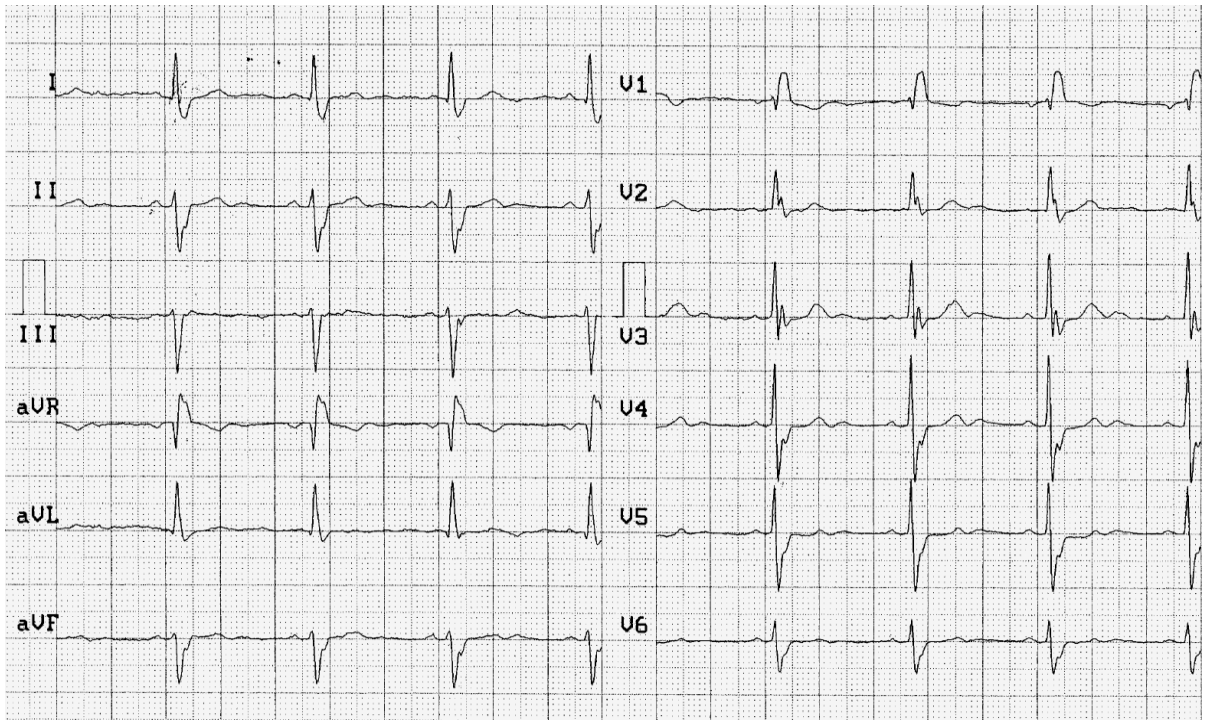


Figure 3/29.

Right bundle branch block (wide QRS, positive deflections in lead V1) and left anterior fascicular block (extreme left axis deviation) - Lenègre syndrome. (Sinus rhythm, 60 bpm, normal AV conduction time, left anterior fascicular block and right bundle branch block, secondary repolarization abnormalities.)

3.3.5.2. trifascicular block (often represent an indication for pacemaker implantation)

- RBBB + LAFB or LPFB + first degree AV block;
- alternating LBBB and RBBB;
- RBBB or LBBB + Mobitz type II AV block;
- RBBB + alternating LAFB and LPFB;
- complete AV block.

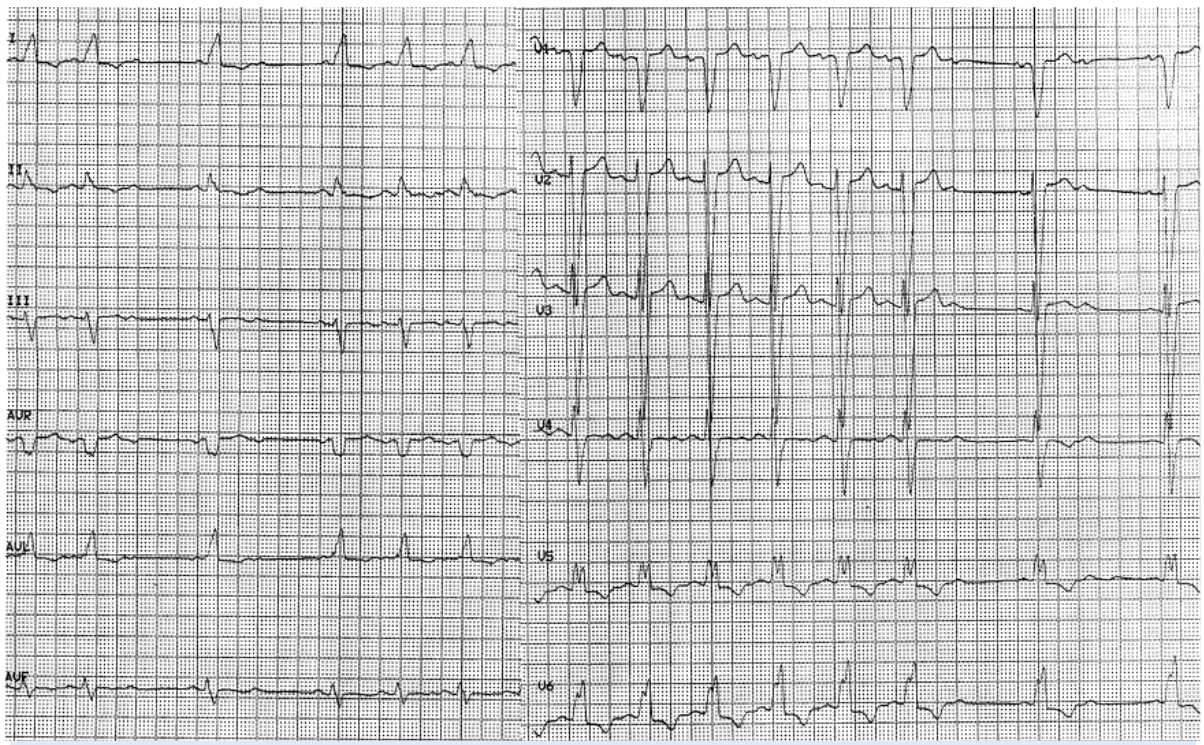


Figure 3/30. Trifascicular block. Intermittent 2:1 AV block (Mobitz type II) and left bundle branch block. Please note the beats conducted with an LBBB pattern in lead V1, followed by a blocked P wave. Mobitz type II block therefore occurs in the right bundle branch. There is a very high risk of complete AV block and asystolia. (Sinus rhythm, intermittent 2:1 AV block, left bundle branch block, secondary repolarization abnormalities.)

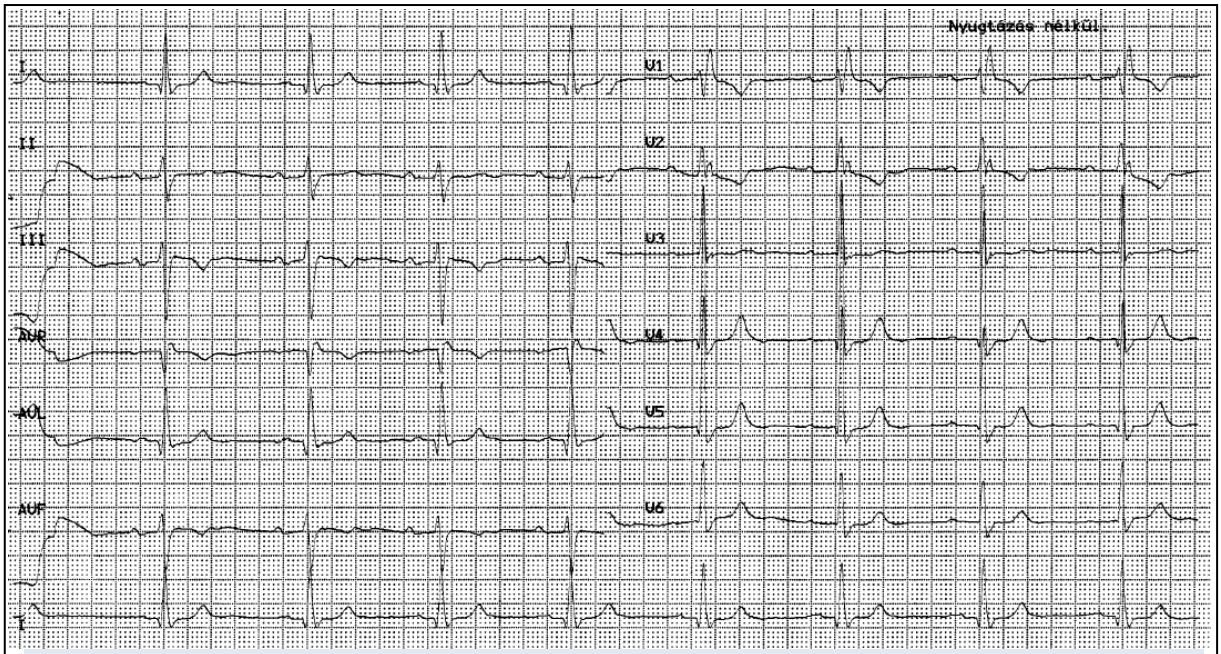


Figure 3/31. Trifascicular block: first degree AV block + left anterior fascicular block + right bundle branch block. This form is benign in nature with a low risk of asystolia, however, it should be explicitly avoided to give medications resulting in additional prolongation of the conduction time. (Sinus rhythm, 52 bpm, minimally prolonged AV conduction, left anterior fascicular block and right bundle branch block, secondary repolarization abnormalities)

FACTS THAT YOU MUST KNOW:

1. First degree AV block is actually prolongation of conduction, however, each P wave is conducted to the ventricles. In second degree AV block, some P waves are conducted, while others are blocked. In third degree AV block, there is complete atrioventricular dissociation, with no P waves being conducted.
2. Mobitz type I (Wenkebach) 2nd degree AV block is generally a benign condition (nodal origin), whereas Mobitz type II block always has an infra-Hisian origin and is a malignant phenomenon. The two of them are differentiated from each other based on the relationship between the PQ intervals before and after the blocked P wave.
3. A beat with a narrow QRS complex occurring later than expected (following a pause) is a junctional escape beat, while that with a wide QRS complex is a ventricular escape beat.
4. In case of abnormal left axis deviation and rS complexes in leads II, III and aVF, one should think of the presence of left anterior fascicular block. The QRS complexes are narrow in this case.
5. In case wide QRS complexes, one should always look at lead V1. If there are positive deflections here, one is dealing with a right bundle branch block, if they are negative, it is left bundle branch block.
6. In case of wide QRS complexes, more specifically, for an LBBB morphology, ST segments will be elevated or depressed secondary to abnormal ventricular activation, and not as a sign of ischemia or infarction. This is also true for widening of the QRS complexes for any reason.
7. In case of acutely developed right bundle branch block and chest pain, one should consider the presence of pulmonary embolism.

CHAPTER 4

ECG SIGNS OF OVERLOAD OF CARDIAC CHAMBERS

4.1. ECG signs of atrial overload

The atrial musculature, due to its structural characteristics, is not capable of thickening, but it becomes dilated instead. It reacts to pressure and volume overload on the same way, so these cannot be distinguished by electrocardiography. Here we would like to refer back to the chapter on the origin of P waves, the understanding of which provides much help for the evaluation of signs of overload. As a reminder, the right atrium is activated first due to the position of the SA node, which is then followed by left atrial depolarization within a short time (this separation is clearly observable in lead V1, since the P waves are biphasic here, that is positive and negative deflections representing the right and left atrium, respectively.)

4.1.1. P mitrale ('broad P waves')

This occurs due to an overload of the left atrium, which was typically observed in mitral stenosis, so this is why it is called P mitrale; however, it may develop in any other case with elevated left ventricular filling pressures (left ventricular hypertrophy, cardiomyopathies, significant valve diseases of the left side of the heart, impaired myocardial relaxation in IHD).

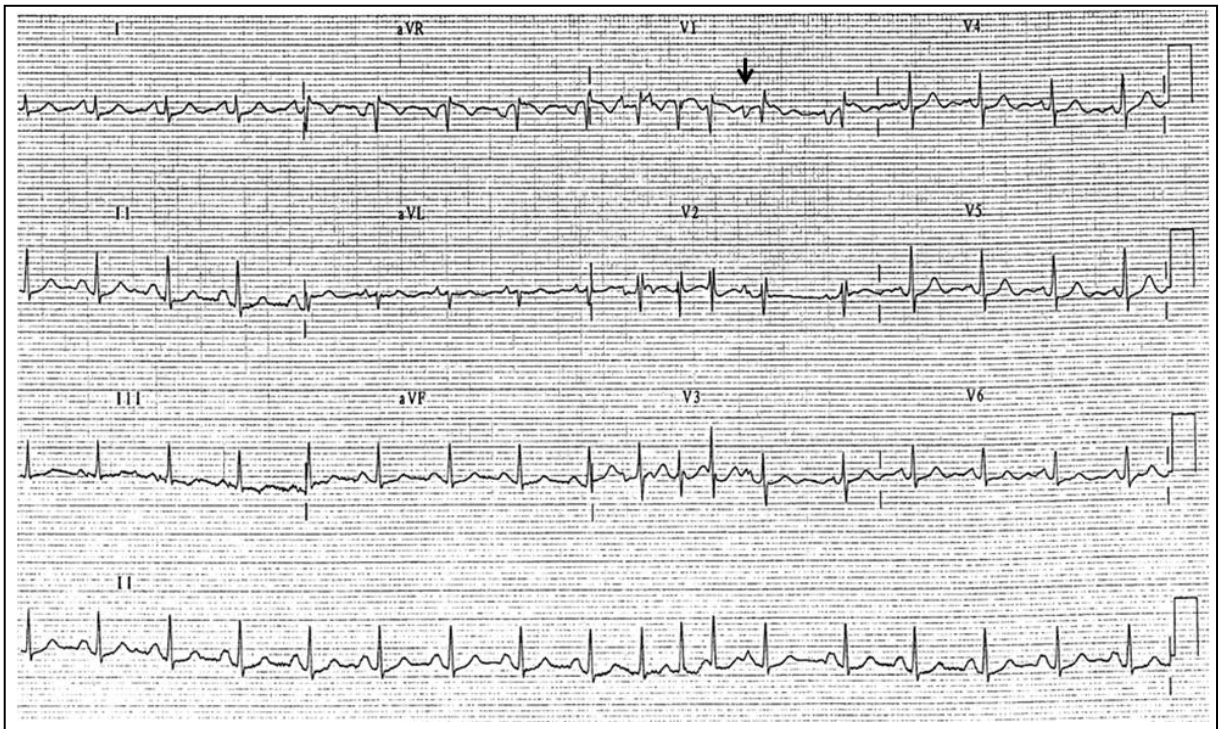


Figure 4/1. P mitrale (being characteristic in leads II and V1), in addition, a supraventricular triplet is also observable, which may indicate imminent atrial fibrillation caused by atrial strain (mitral stenosis). (Sinus rhythm, P mitrale, 105 bpm, normal QRS axis, normal AV conduction time, a QRS pattern consistent with incomplete right bundle branch block in leads V1-2, narrow QRS complexes, trivial ST segment depression in leads II, III, aVF (right ventricular overload), SV triplet.)

- Broad and notched P waves in leads I, II, III, aVF and in V5-6;
- In lead V1, there will be an increase in the amplitude / width of the second, negative phase and it will become dominant in the overall deflection of P waves.

4.1.2. P pulmonale ('tall P waves')

Its name stems from the fact that its underlying cause is often elevated pressure in the right side of the heart, as well as consequential right atrial overload, caused by pulmonary diseases (chronic lung disease, valve diseases resulting in an overload of the right side of the heart.)

Tall, peaked P waves in leads II, III, aVF.

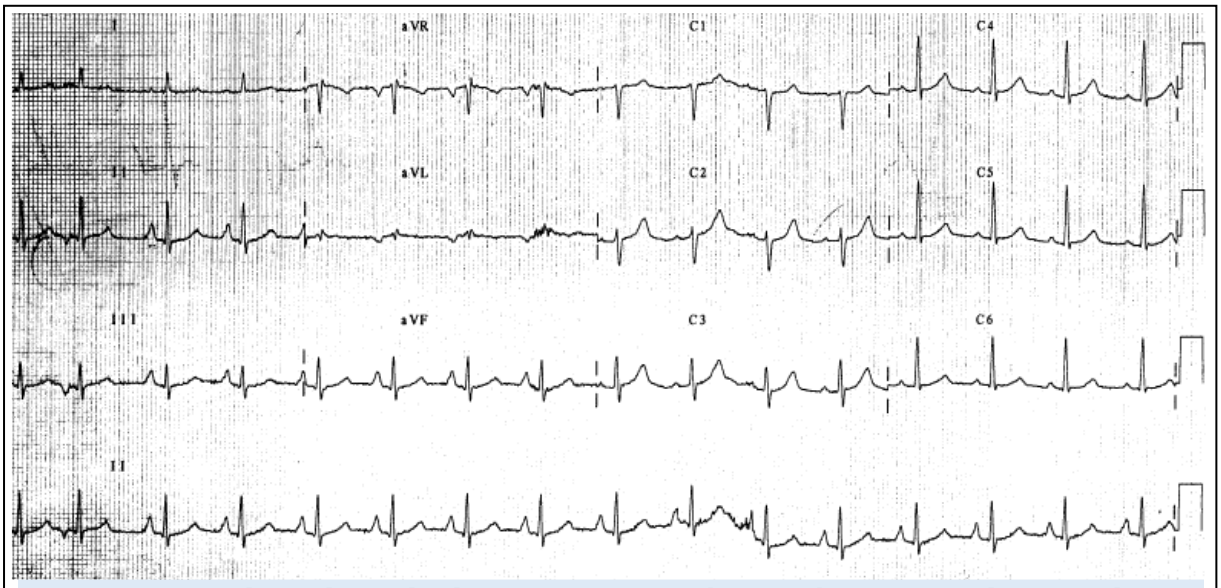


Figure 4/2. P pulmonale (in leads II, III, aVF) in chronic obstructive pulmonary disease (COPD.)

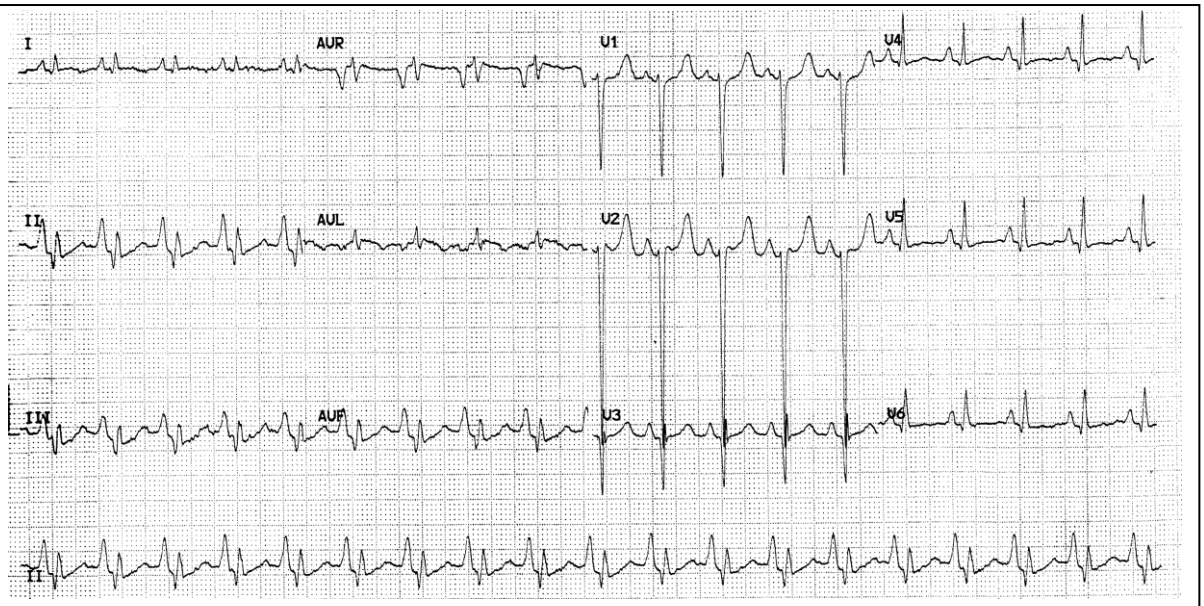


Figure 4/3. Extreme form of P pulmonale in a patient with severe COPD due to an additional elevation in pulmonary artery pressures caused by acute left-sided heart failure. (Sinus tachycardia, P pulmonale, normal QRS axis, normal AV conduction time, Q waves in leads II, III and aVF, deflections with a low amplitude in the limb leads, i.e. low voltage, normal ventricular repolarization.)

4.1.3. P biatriale

In a combination of the above diseases, the ECG abnormalities described above are present in a mixed fashion. Its presence is characteristic in heart failure when there is also chronic elevation of the pulmonary pressures.

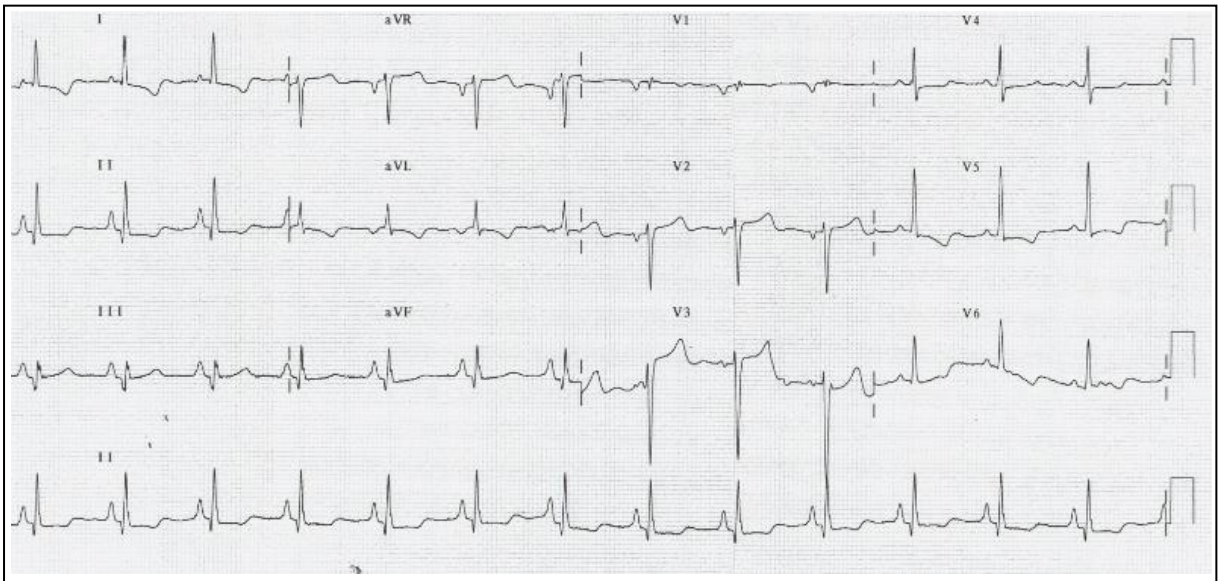


Figure 4/4.

P biatriale; tall, peaked P waves in leads II, III, aVF and deep inverted P waves in lead V1. (Sinus rhythm, P biatriale, normal QRS axis, normal AV conduction time, Q waves in leads II, III, aVF, otherwise normal ventricular conduction, signs of left ventricular strain.)

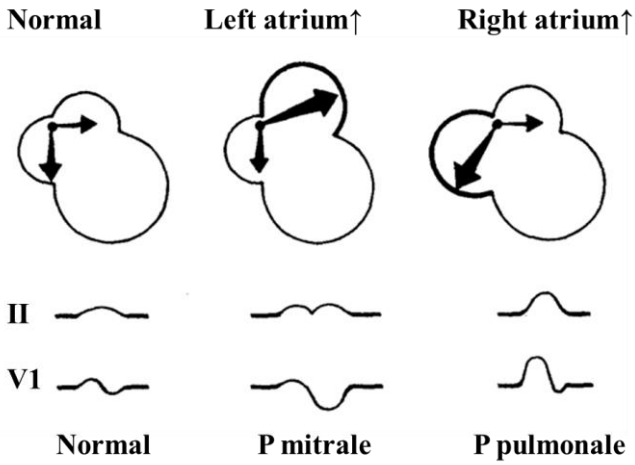


Figure 4/5.

Schematic representation of the ECG signs of atrial enlargement.

4.2. ECG signs of ventricular overload

Overload of the ventricles may induce several reactions (overload concept), that is they are able to compensate for the extra load not only by dilatation, but also by hypertrophy. This is why more specific ECG abnormalities are observable depending on the underlying pathology. A distinction is made between pressure and volume overload. *The overload of the heart during systole („strain")* is typically *pressure overload* (e.g. in aortic stenosis), while

during diastole it is typically referred to as *volume overload*. Systolic or pressure overload may develop during contraction of any of the ventricles against an increased resistance. Diastolic or volume overload occurs when there is an increase in the diastolic inflow of blood in any of the ventricles (e.g. in valvular regurgitation). If the overload persists permanently, compensatory ventricular hypertrophy develops as a result of overstretching of the myocardial fibers, while ventricular dilatation occurs in end-stage disease.

4.2.1. Signs of left ventricular pressure overload or strain

Causes include aortic stenosis and severe, untreated systemic hypertension.

- Downsloping ST segment depression and negative T waves in leads I, aVL and V5-6;
- ST segment elevation of up to 2 mm or slightly even greater than that may frequently be observed in leads V1-3 (this is not myocardial infarction!), especially in case of an overload with a sudden occurrance.

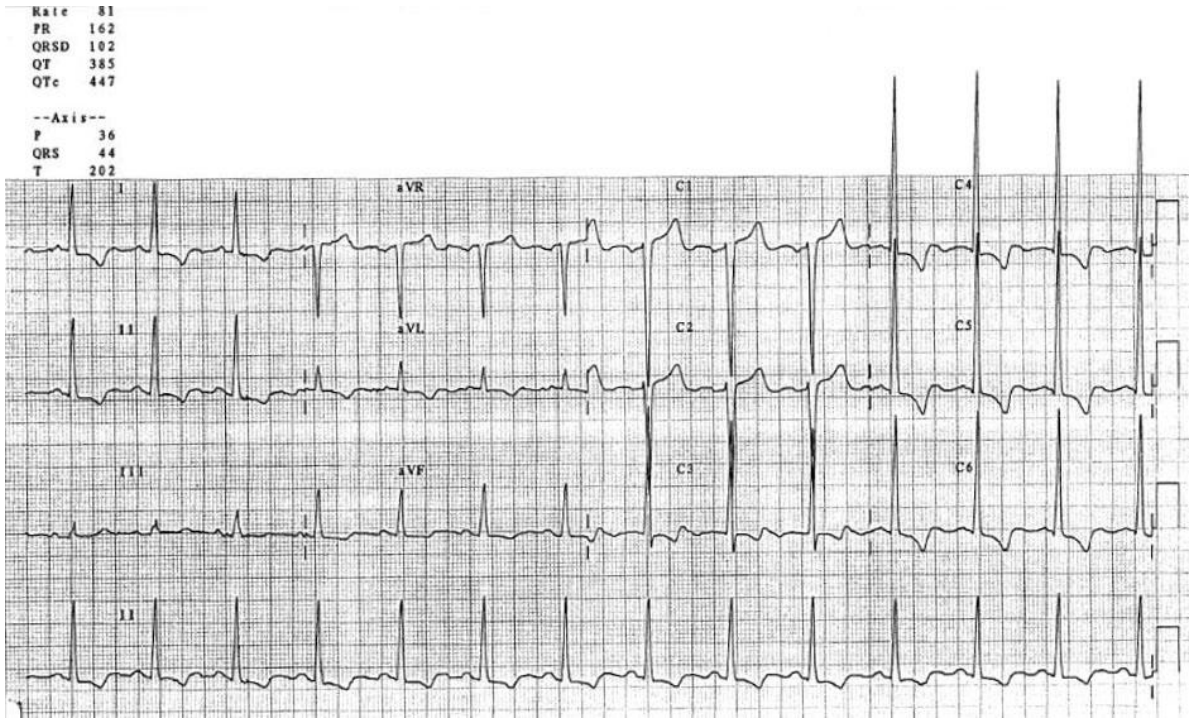


Figure 4/6.

Left ventricular hypertrophy (high voltage) and signs of strain (downsloping ST segment depression and negative T waves in leads I, aVL, V(3-4)5-6) in severe aortic stenosis. (Sinus rhythm, normal QRS axis, 85 bpm, left ventricular hypertrophy and signs of strain)

4.2.2. Signs of left ventricular diastolic overload

Causes include aortic insufficiency, mitral insufficiency and congenital heart defects with a left-to-right shunt (e.g. VSD).

- Concave ST segment elevation (more rare) or horizontal ST segment depression and positive T waves in leads I, aVL, V5-6;
- Tall R waves and deep (>2 mm), but narrow Q waves in leads V5-6 (this is not myocardial infarction!).

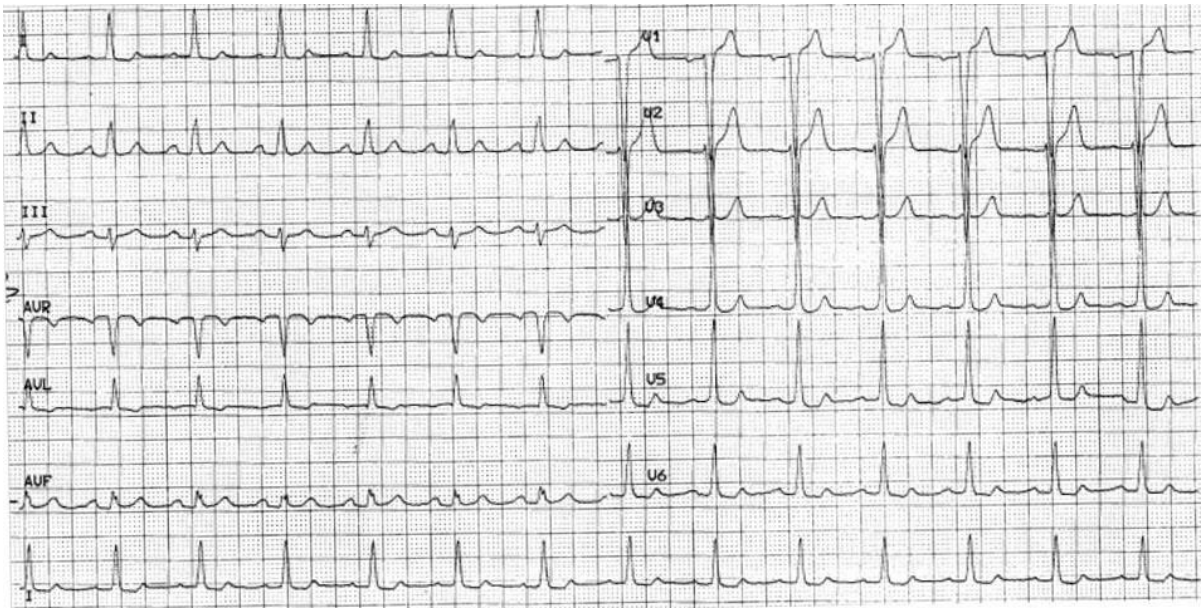


Figure 4/7.

Left ventricular volume overload; horizontal ST segment depression and positive T waves in leads I, aVL, V4-6. The underlying cause is severe aortic insufficiency. (Sinus rhythm, 90 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction, signs of left ventricular diastolic overload.)

4.2.3. Signs of right ventricular systolic overload

Causes include pulmonary hypertension, pulmonary embolism and pulmonary stenosis.

Convex ST segment depression and T wave inversion (’right ventricular strain’) in leads V1-3.

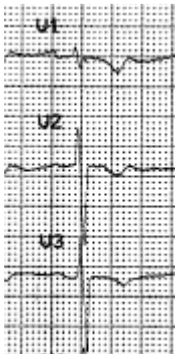


Figure 4/8. Right ventricular strain caused by pulmonary hypertension.

4.2.4. Signs of right ventricular diastolic overload

Causes include congenital heart defects with a left-to-right shunt (e.g. ASD) and tricuspid insufficiency.

- An ECG pattern reminding of right bundle branch block in leads V1-2, accompanied by classical triphasic (RSR) QRS complexes (for details, see RBBB)

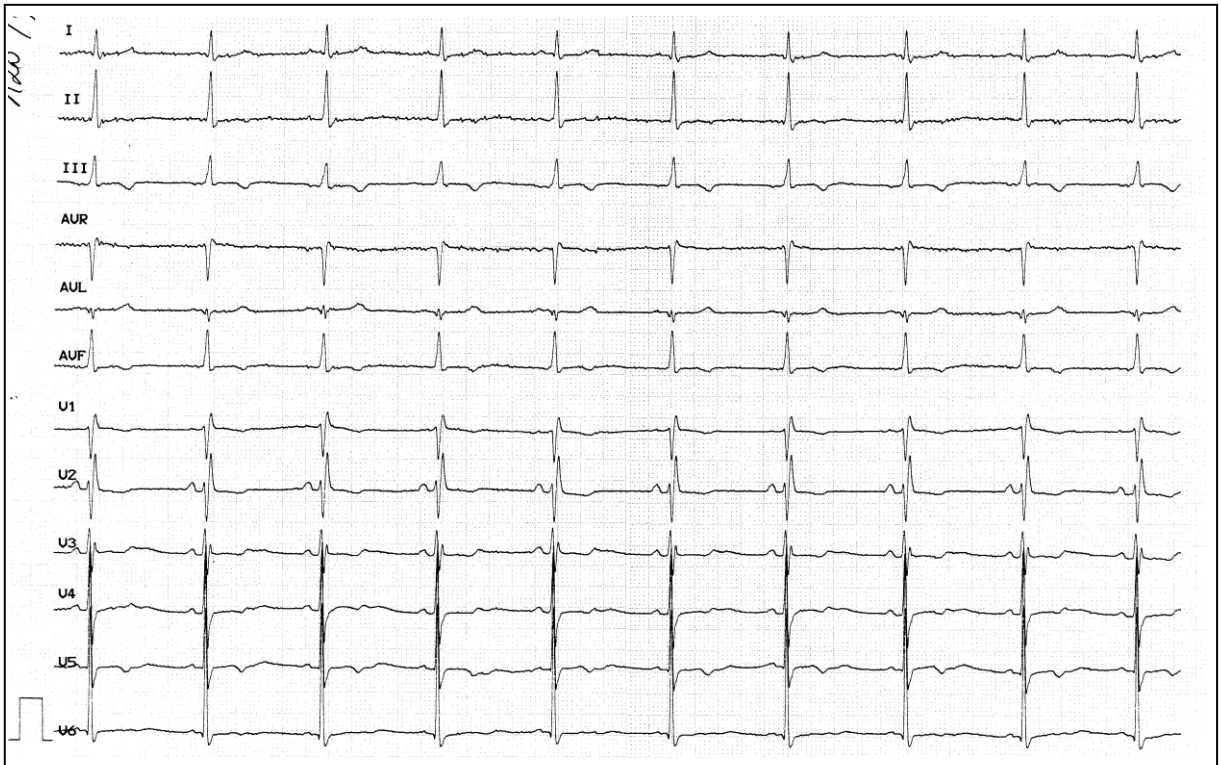


Figure 4/9.

Systolic overload caused by pulmonary hypertension - incomplete right bundle branch block, trivial ST segment depression and negative T waves in leads V1-4 and II, III, aVF. (Sinus rhythm, 70 bpm, normal QRS axis, normal AV conduction time, incomplete right bundle branch block, right ventricular hypertrophy and signs of strain.)

4.2.5. Signs of acute overload of the right side of the heart (pulmonary embolism!)

The right atrium and ventricle suffers a simultaneous and sudden overload, with this resulting in characteristic ECG abnormalities. It is very important to note that in case of sharp chest pain and dyspnea in association with newly developed right bundle branch block, the diagnosis of pulmonary embolism is very likely. In addition, there are several other 'small' signs which might be a clue.

- Sinus tachycardia (!);
- T wave inversion in leads V1-3 (!) - for details, see right ventricular systolic overload;
- 'S1Q3T3' pattern (i.e. deep S waves in lead I, deep Q waves and negative T waves in lead III)(!);
- Downsloping ST segment depression and negative T waves in leads II, III, aVF;
- Right axis deviation;
- Early transition in the precordial leads;
- P pulmonale.

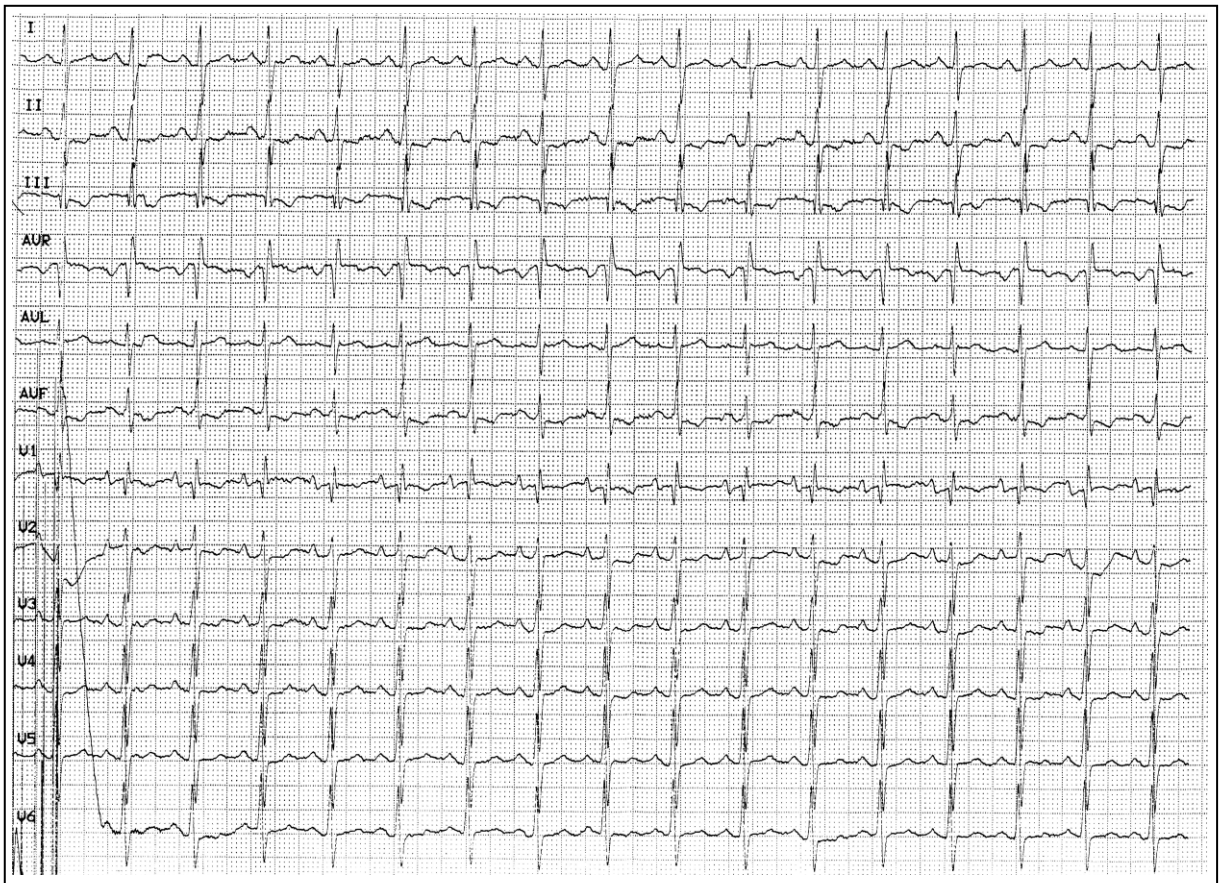


Figure 4/10.

Pulmonary embolism. One's suspicion is reinforced by the SIQIIITIII pattern, sinus tachycardia, early transition, moreover, the ST segment depression and negative T waves in leads II, III, aVF and V1-3. The right ventricular pressure in this patient was 70 mmHg. (Sinus tachycardia, right axis deviation, normal AV conduction time, P pulmonale, tall R waves in leads V1-2, SIQIIITIII pattern, trivial ST segment depression in leads II, III, aVF and V1-3.)

4.3. ECG signs of ventricular hypertrophy

4.3.1. Left ventricular hypertrophy

Due to the greater muscle mass, the heart undergoes counterclockwise rotation (in the horizontal plane), resulting in late (and generally very rapid) transition in the precordial leads as well as left axis deviation occurs.

1. *Sokolow-Lyon index (QRS voltage criteria*), based on which left ventricular hypertrophy can be diagnosed in nearly 1/3 of cases; i.e. one should add the amplitude of S waves in lead V1 to that of R waves measured in lead V6 and, if the sum reaches or exceeds 35 mm, it is referred to as hypertrophy.

   Values of the Sokolow-Lyon index vary with age. They are accepted as normal up to 60 mm for an age between 16-20 years, whereas up to 40 mm for that between 20-30 years.

2. Activation of a greater muscle mass requires more time, as a result of which there will be a prolongation of the ventricular activation time (VAT≥45 ms in leads V5-6).
3. An additional sign may be R waves > 26 mm observed in lead V5 or V6, those > 14 mm in lead I, > 11 mm in lead aVL and > 20 mm in lead aVF, or if RI+SIII > 25 mm. Lewis index may also be mentioned: RI-SI+SIII-RIII > 17 mm. In certain cases, based on widening of the QRS complexes, an ECG pattern reminding of left bundle branch block (small r waves or QS complexes in leads V1-3) may also be observable.

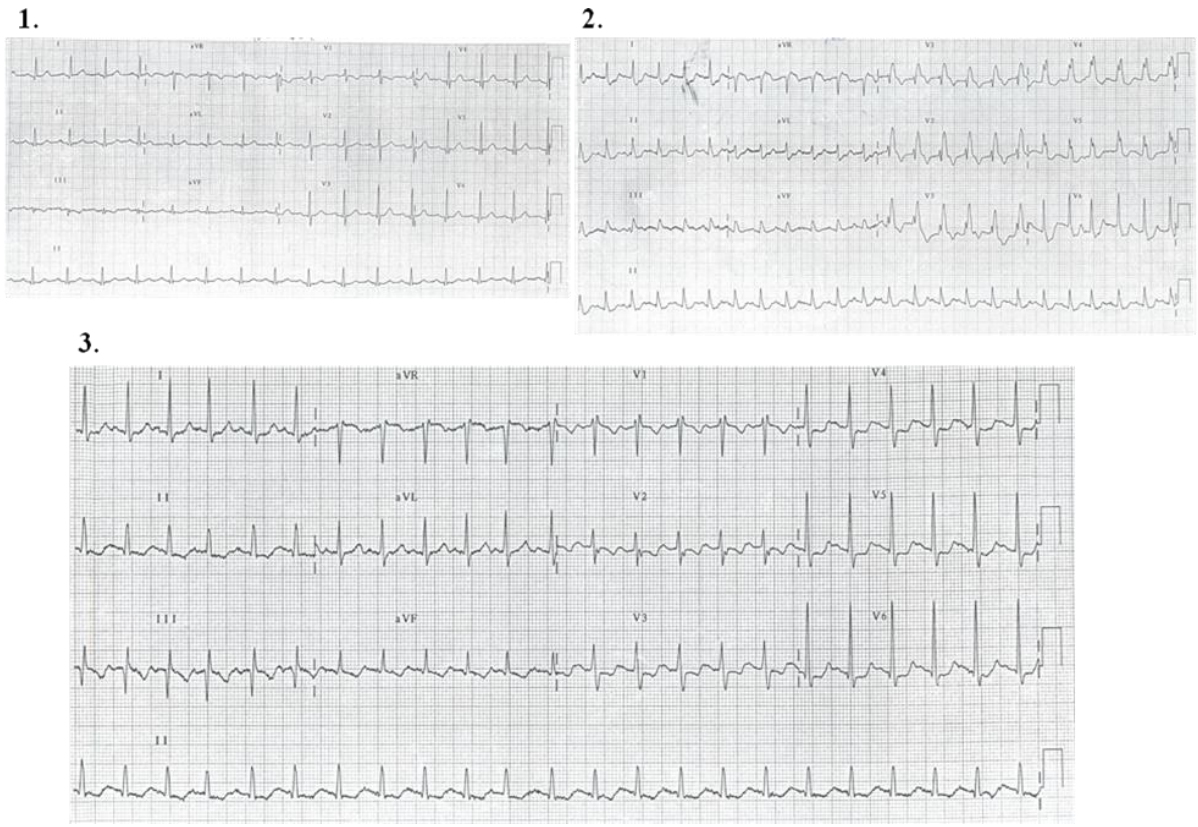


Figure 4/11.

Pulmonary embolism. Changes in the electrocardiographic pattern can be observed in a single patient. **1.** On the admission ECG, signs of an old postero-inferior myocardial infarction scar (Q waves in leads III, aVF, tall R waves in lead V1, the latter one being 'reciprocal' changes corresponding with posterior Q waves) are visible. **2.** Two days later and in association with the development of sharp chest pain and dyspnea, complete right bundle branch block along with sinus tachycardia at a rate of 140 bpm is detectable. **3.** A few hours later, with a relief of pain over time, the degree of tachycardia is reduced, and incomplete right bundle branch block (rSr' in lead V1), right ventricular strain (ST segment depression in leads II, III, aVF and the precordial leads), negative T waves in leads V1-4 as well as S1Q3T3 pattern are visible.

In addition to the above, *signs of left ventricular strain* (c.) can often be found demonstrating that the cause of hypertrophy is most frequently pressure overload. (For details, see the ECG of the patient with aortic stenosis). Left ventricular hypertrophy is indicated with a greater sensitivity (in 50 % of cases) if *the height of R waves measured in lead aVL is added*

to the depth of S waves measured in lead V3 and the calculated sum reaches or exceeds *28 mm and 20 mm in males and females*, respectively.

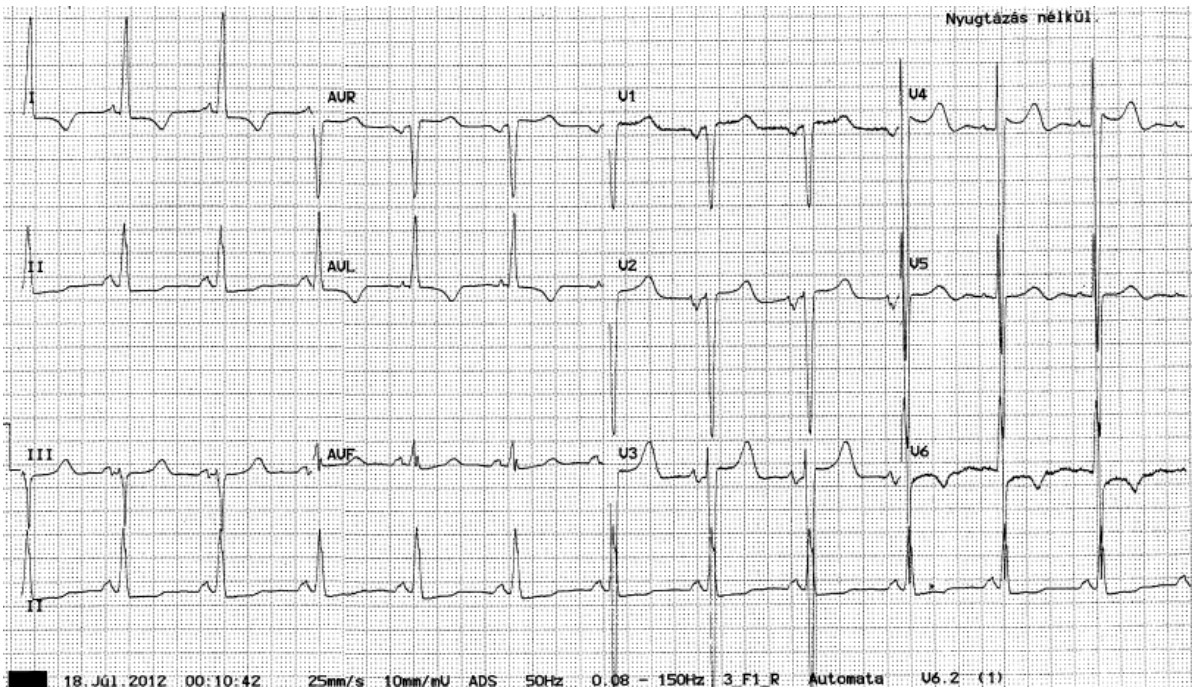


Figure 4/12.

Left ventricular hypertrophy. High voltage, signs of left ventricular strain and ST segment elevation in the precordial leads(!). The latter finding may throw suspicion on the presence of acute coronary syndrome; however, the coronary angiography showed no stenosis in this case, but the left ventricular wall thickness proved to be 19 mm by echocardiography (normally ≤ 12 mm). (Sinus rhythm, normal QRS axis, normal AV conduction time, high voltage seen diffusely in the leads, severe left ventricular hypertrophy, signs of strain.)

Ventricular hypertrophy may exclusively be localized to the interventricular septum (e.g. in hypertrophic cardiomyopathy); in this case, RS complexes are observable in lead V1-3 (or QR complexes in lead V1) as well as deep, but not wide, Q waves (septal Q) in leads V5-6, occasionally in leads II, III and aVF.

4.3.2. Right ventricular hypertrophy

Due to the greater muscle mass, the heart undergoes clockwise rotation (in the horizontal plane) resulting in early transition in the precordial leads as well as right axis deviation occurs. Due to thickening of the right ventricular musculature, the right ventricle may become predominant in the formation of the QRS complex, which was previously determined by the depolarization of the far greater left ventricular muscle mass. At a young age and in children, it is the right ventricle that dominates the deflection of ventricular activation, which cannot be considered as a sign of right ventricular hypertrophy.

- The R/S ratio in leads V1-2 is ≥ 1 (R≥S - qR or R), in addition, early transition and right ventricular strain is observable (downsloping ST segment depression and negative T waves in leads V1-3, which may also appear in leads aVL and aVF);

  According to the Sokolow-Lyon criteria, if the amplitude of R waves in lead V1 exceeds 7 mm or R/S ≥ 1 or there are two R waves, moreover, left ventricular activation time is > 30 ms. Lewis index is < -14 mm.
- Complete or incomplete right bundle branch block;

  (An rSR pattern in lead V1 implies hypertrophy of the right ventricular outflow tract typically occurring in ostium secundum ASD, while if this is associated with left axis deviation, the presence of ostium primum ASD is likely).
- Right axis deviation;
- P pulmonale;
- Low voltage (especially in emphysema).

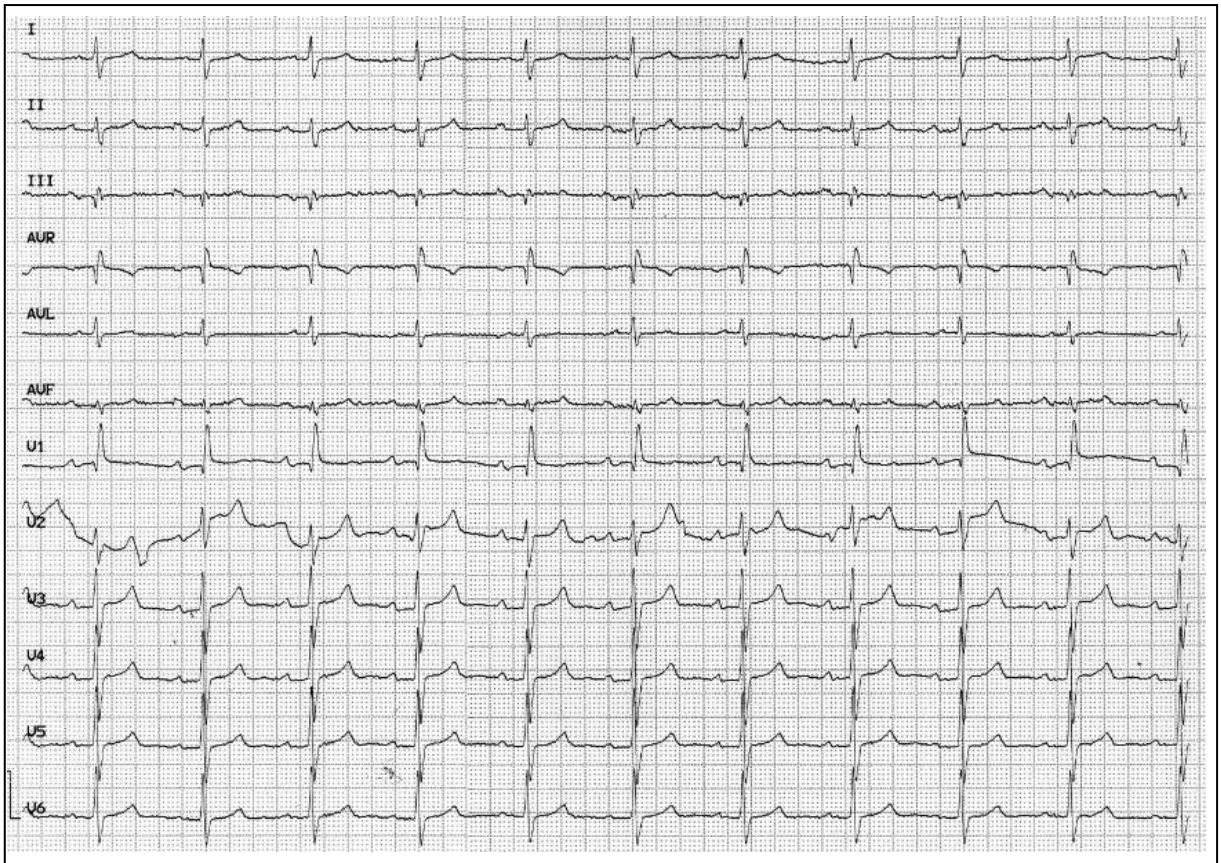


Figure 4/13.

Right ventricular hypertrophy developed due to pulmonary valve stenosis. Extreme right axis deviation or superior axis, tall R waves in lead V1. (Sinus rhythm, 65 bpm, extreme right axis deviation, normal AV conduction time, tall R waves in lead V1, i.e. a sign of right ventricular hypertrophy.)

Normally, R waves are smaller than S waves in lead V1, so R/S ratio is < 1. It occurs in many cases that R/S ratio in lead V1 is > 1 or R wave amplitude exceeds 4 mm, so the R wave is the dominant deflection.

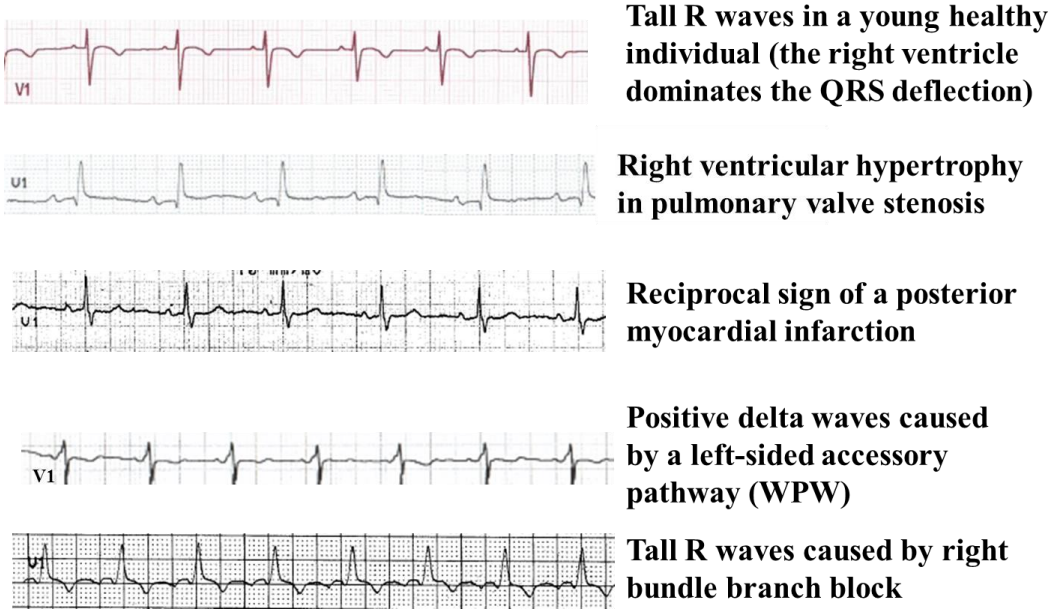


Figure 4/14.

On the left side of the figure, cases with tall R waves in lead V1 are visible, whereas on the right side of the figure, an explanation for the observed ECG pattern can be found.

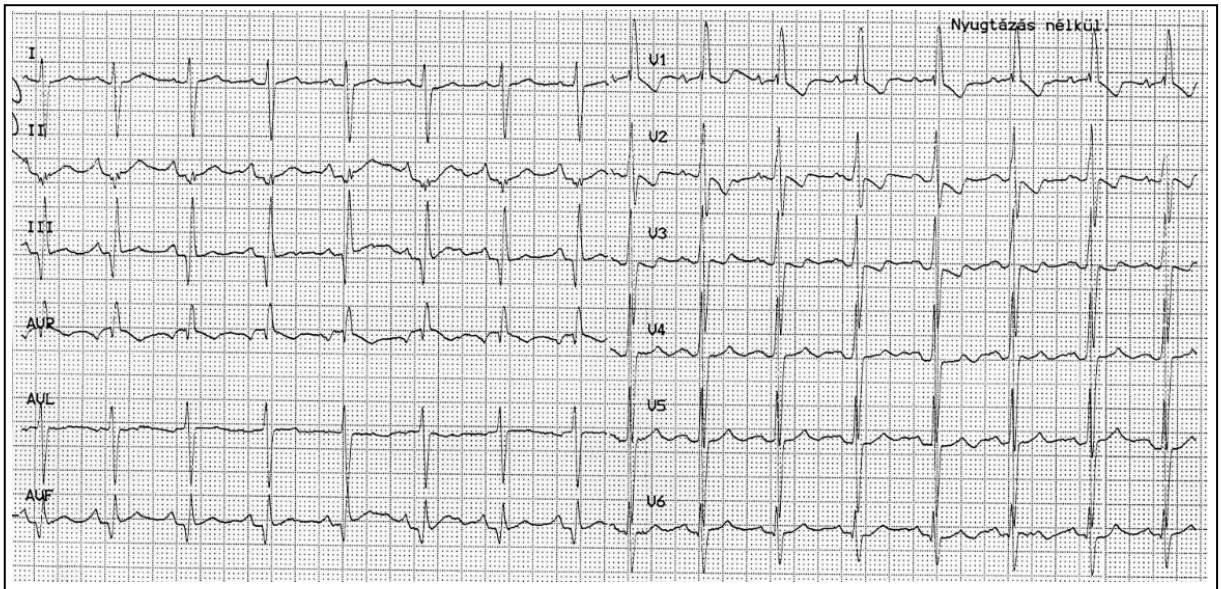


Figure 4/15.

Overload of the right side of the heart in Tetralogy of Fallot. P pulmonale as well as right ventricular hypertrophy and signs of strain. (Sinus rhythm, 100 bpm, right axis deviation, normal AV conduction time, incomplete right bundle branch block, severe right ventricular hypertrophy and signs of strain.)

FACTS THAT YOU MUST KNOW!

1. Tall, peaked P waves (P pulmonale) in leads II, III, aVF indicate the presence of right atrial enlargement (often in COPD) Broad and notched P waves (P mitrale) are the signs of left atrial overload.
2. Systolic overload of the left ventricle (untreated hypertension, aortic stenosis) results in the signs of left ventricular strain; i.e. downsloping ST segment depression and negative T waves in leads I, aVL, V5-6.
3. Left ventricular hypertrophy can be diagnosed by electrocardiography based on the presence of deflections with a high amplitude in nearly less than 50 % of cases, however, high voltage is generally a typical finding.
4. Signs of chronic systolic overload of the right ventricle include tall R waves (right ventricular hypertrophy), ST segment depression and negative T waves (right ventricular strain) in leads V1-3.
5. In case of acutely developed right bundle branch block, one should consider the presence of pulmonary embolism. T wave inversion occurring in leads V1-4 is a more frequent ECG sign of pulmonary embolism than the S1Q3T3 pattern.

CHAPTER 5

SIGNS OF MYOCARDIAL ISCHEMIA, MYOCARDIAL INFARCTION

5.1. Pathophysiology of myocardial ischemia

Ischemia is defined as a condition when the coronary circulation is not able to fulfil current demands and disturbances of blood supply and hypoxia develops. Hypoxia initiates reversible and irreversible changes in myocardial cell function, which have typical clinical symptoms and ECG signs. Disorders of the coronary circulation are caused by atherosclerotic plaques in the majority of cases. As a result of plaque rupture, blood coagulation is activated and the coronary artery will be filled with a thrombus causing total occlusion. This is how myocardial infarction develops most of the time.

5.2. Coronary circulation of the heart and coronary artery dominance

Anatomy of coronary arteries:

The system of the left coronary artery starts with the left main stem (**LM**), which is then divided into two branches: the left anterior descending artery (**LAD**) and the left circumflex artery (**Cx**). **LAD** provides blood supply to the anterior 2/3 of the interventricular septum (**septal branches**) as well as the anterolateral wall (**diagonal branches**) and apical region of the left ventricle. The origin of the first diagonal branch may be before or after the first septal branch. The region of the LAD between the first septal and diagonal branch is called the middle third of LAD. **Cx** provides blood supply to the posterior region of the left ventricle and, with its **marginal branches** (**OM** - obtuse marginal branch), to the adjacent posterolateral region. Trifurcation of the left main stem may also occur and it gives off, beyond the LAD and Cx, the so-called intermediate (**IM**) branch supplying the region consistent with the blood supply of the first diagonal and marginal branch (i.e. lateral wall). The right coronary artery (**RCA**) supplies with its proximal branches the right ventricle and is divided into two branches distally: the posterior descending artery (**PDA**, ramus interventricularis posterior) is responsible for supplying the inferior wall of the left and right ventricle as well as the posterior 1/3 of the interventricular septum, while the posterolateral branch (**PL**), supplementing the role of Cx, takes part in the blood supply of the posterior and posterolateral region. LAD supplies 50-60 % of the left ventricular muscle mass, while Cx and RCA does 25-30 % and 15-20 % of that, respectively.

Coronary artery dominance: The above blood supply pattern of the coronary circulation can be seen most frequently, however, the presence of several individual variations is possible. The above described branching pattern is characteristic of dominance of the right coronary artery, which is observable in about 85-90 % of cases. It is referred to as dominance of the left coronary artery if it is the Cx, not the RCA, that provides the posterior descending artery, which occurs in about 10 to 15 % of cases. In some cases, one may observe that both the RCA and Cx give off branches to the region supplied by the posterior descending artery, which is referred to as co-dominant coronary circulation.

Knowledge of the **blood supply of the impulse formation and cardiac conduction system** provides help in the explanation of conduction disturbances associated with myocardial infarction. The SA node receives its blood supply from the RCA and Cx in 55 % and 45 % of cases, respectively.

The AV node is supplied by the coronary artery coursing toward the inferior wall, being the right coronary artery in the majority of cases, and this explains why AV blocks develop so frequently in inferior myocardial infarctions.

The upper portion of the bundle of His is perfused by the RCA, while the lower part by the LAD. Blood supply of the right bundle branch and the left anterior fascicle is provided by the LAD through the first septal perforator branch. The left posterior fascicle has dual blood supply that is both the LAD and RCA gives off branches to this area; this is why this fascicle is injured so rarely during ischemia.

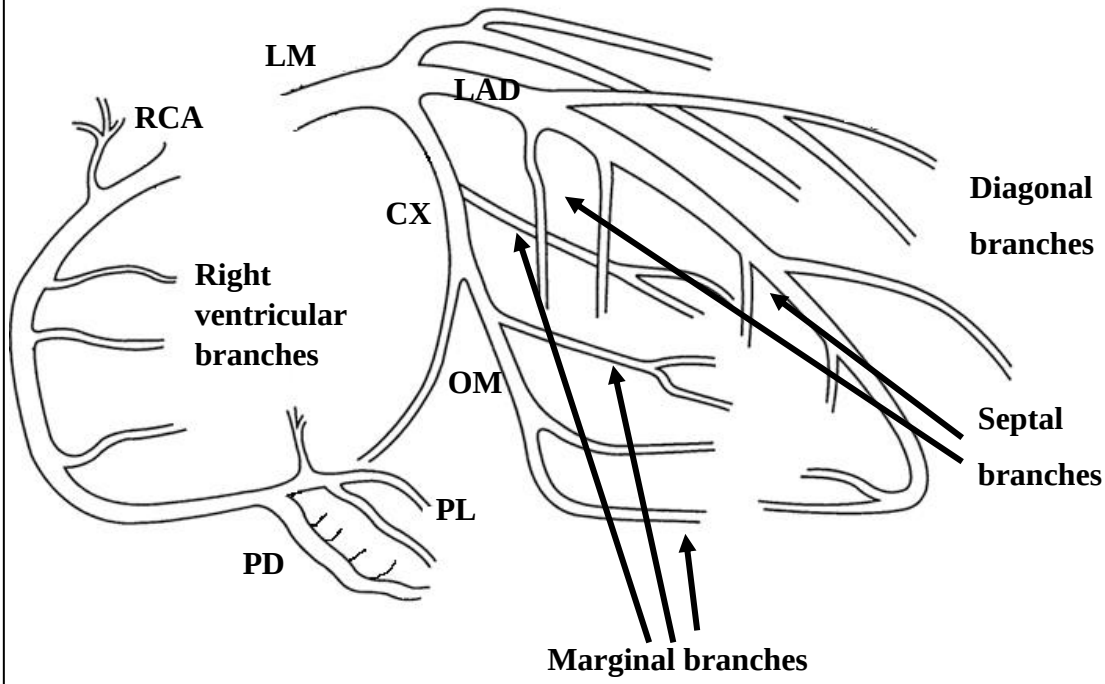


Figure 5/1.

Coronary anatomy. (LM= left main stem, LAD= left anterior descending branch, Cx= left circumflex artery, RCA= right coronary artery, OM= obtuse marginal branch, PD= posterior descending branch, PL= posterolateral branch.)

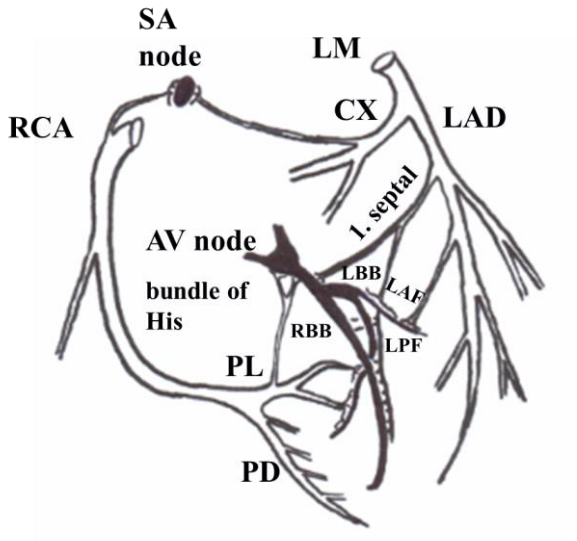


Figure 5/2.

Blood supply of the impulse-generating and cardiac conduction system. See text for an explanation.

5.3. Definition of angina pectoris and myocardial infarction

Angina pectoris is a condition when there is reduced coronary blood flow and myocardial hypoxia develops due to a coronary artery stenosis. This is associated with typical squeezing or pressure-like chest pain and more or less typical ECG abnormalities. No myocardial necrosis and any associated enzyme or biomarker (troponin) release occurs.

In **myocardial infarction**, complete or partial thrombotic occlusion of a blood vessel develops. The chest pain is similar to angina, often even more intense, and is associated with characteristic ECG abnormalities as well as a release of troponin and markers of myocardial necrosis.

5.4. Levels of myocardial damage

Depending on the severity of the coronary artery stenosis, current myocardial oxygen demand and the level of development of collateral branches, several stages of myocardial damage can be differentiated. The coronary artery system penetrates through the myocardium from the epicardial to the endocardial region. Perfusion disturbances therefore present in the subendocardium at first, while in more severe cases, disorders of the transmural coronary blood flow will also develop.

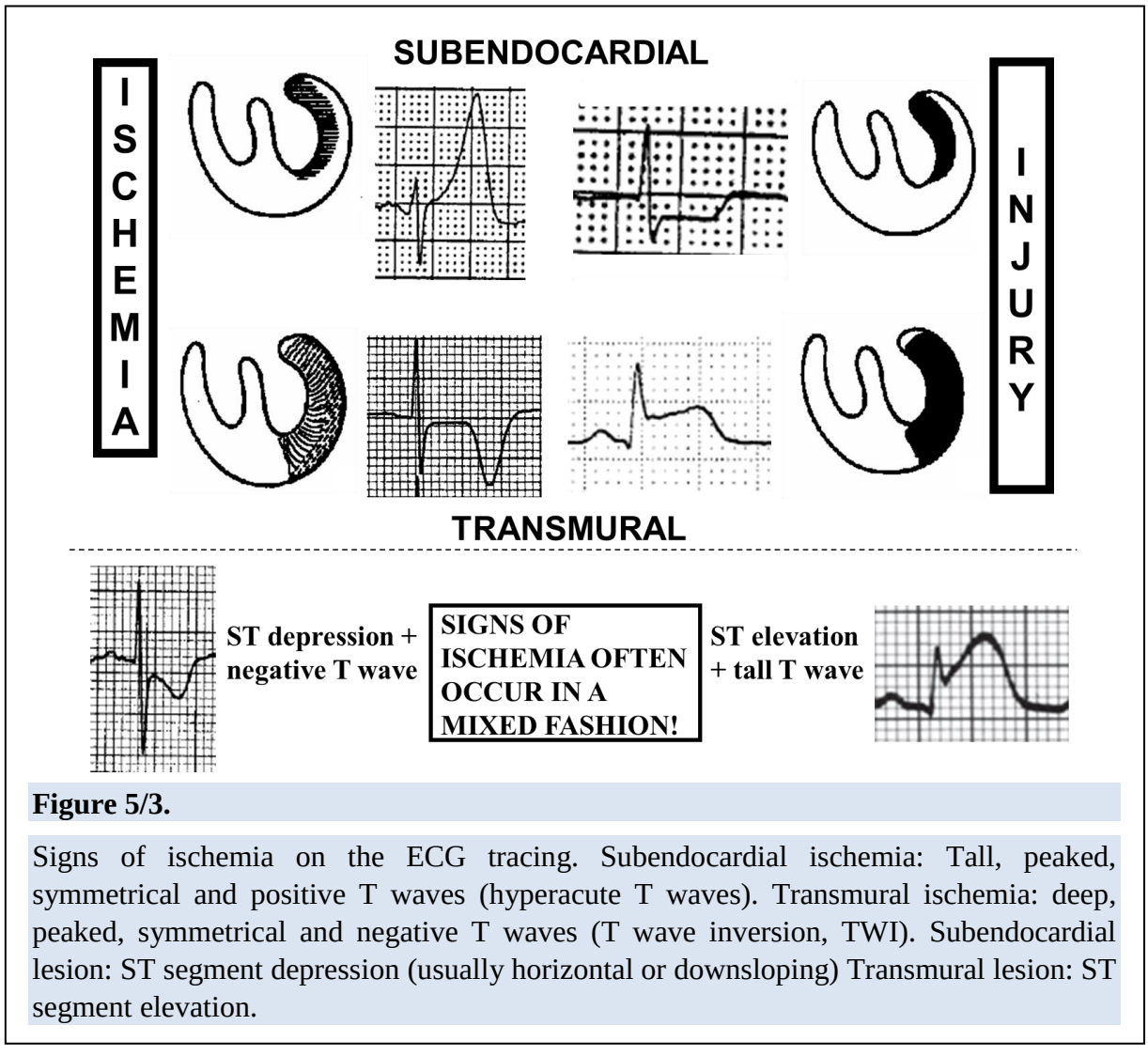


Figure 5/3.

Signs of ischemia on the ECG tracing. Subendocardial ischemia: Tall, peaked, symmetrical and positive T waves (hyperacute T waves). Transmural ischemia: deep, peaked, symmetrical and negative T waves (T wave inversion, TWI). Subendocardial lesion: ST segment depression (usually horizontal or downsloping) Transmural lesion: ST segment elevation.

Reversible hypoxic myocardial injury induces repolarization abnormalities on the ECG resulting in *T wave* abnormalities or, in more severe cases, deviation of the *ST segment*. One might frequently observe in clinical practice that ST segment and T wave abnormalities appear on the ECG recording in association with each other.

In irreversible hypoxic myocardial injury, it is the depolarization that will be altered, e.g. pathological Q waves will occur.

In angina attacks or non ST-segment elevation myocardial infarction (NSTEMI), the most typical finding is a combination of ST segment depression and/or negative T waves. Moreover, in ST-segment elevation myocardial infarction (STEMI), ST segment elevation and tall, peaked T waves (dome-shaped pattern) occur in the acute phase.

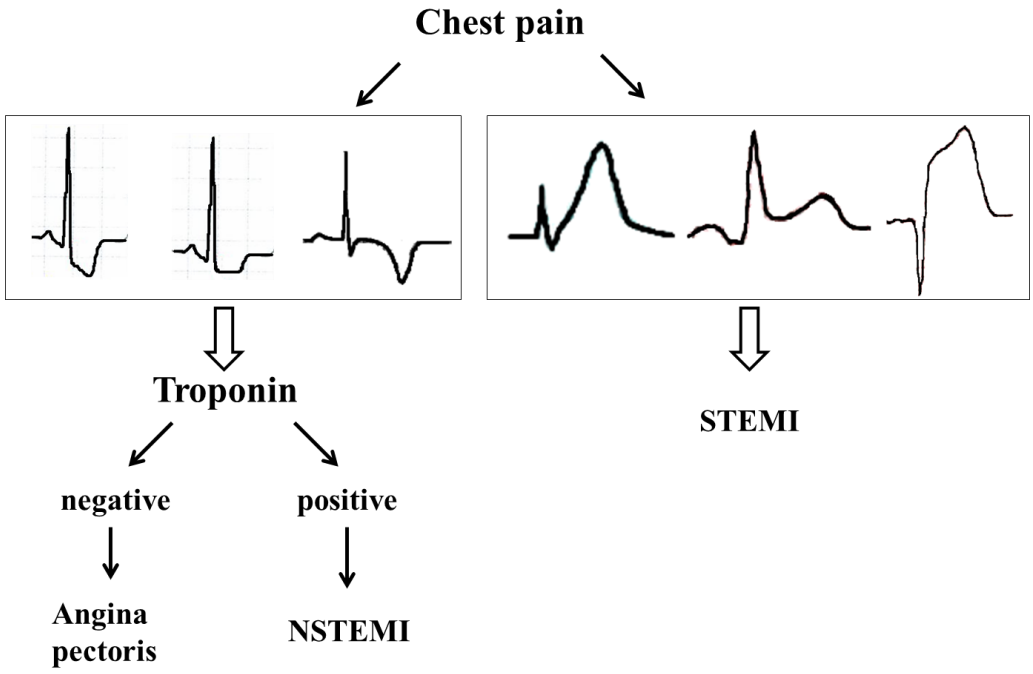


Figure 5/4.

It is mandatory to perform an ECG if chest pain occurs. If you observe the above abnormalities, signs of ischemia can be differentiated based on the following algorithm. The level of emergency will be decided on the basis of your diagnosis.

5.5. Characteristic T wave abnormalities

Normal T waves are concordant with the QRS complexes and not higher than ¾ of the preceding QRS complex. T waves may be flat (isoelectric), biphasic and negative, moreover, there are tall, peaked T waves that is the so-called hyperacute T waves (in hyperacute phase of AMI); however, deep, peaked and symmetrical T wave inversion also exists. These T wave abnormalities may all indicate the presence of ischemia but may also appear in several other normal and pathological cases. For example, in excessive sympathetic and parasympathetic tone, changes in the K^+ level, pericarditis and myocarditis (recent or previous) and secondary changes (in bundle branch blocks!)

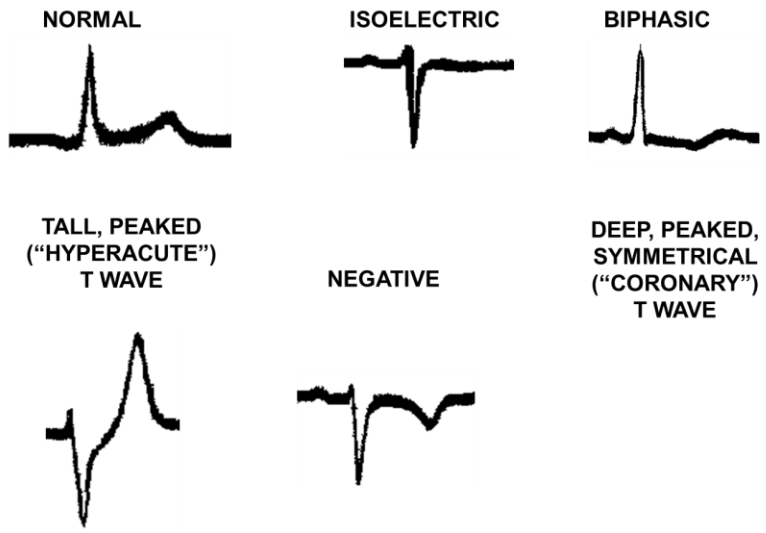


Figure 5/5.

Characteristic T wave abnormalities.

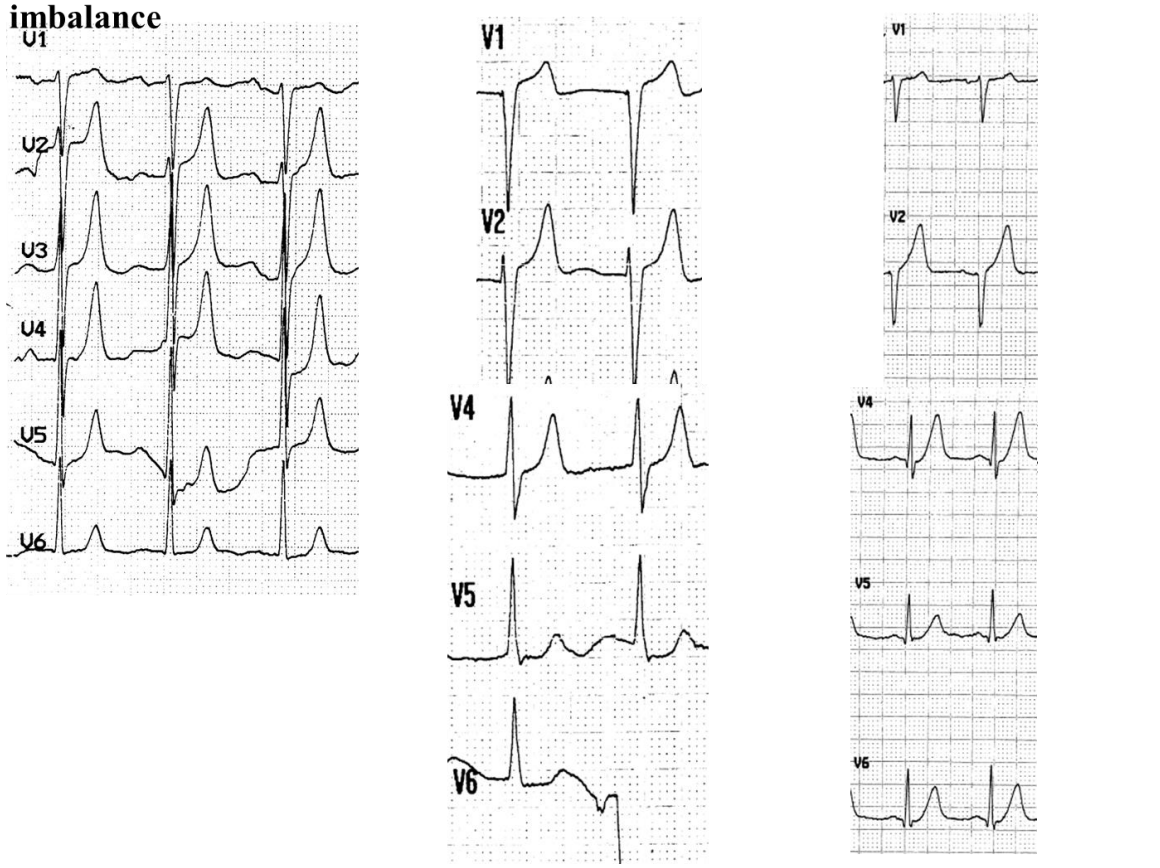


Figure 5/6.

Tall, peaked T waves may appear without the presence of heart disease due to autonomic imbalance or in hyperkalaemia, but it may also be a sign of acute occlusion of the coronary arteries. It is often difficult to distinguish hyperacute T waves caused by myocardial infarction from tall T waves of non-cardiac origin; however, the former one has a somewhat wider base and the complaints as well as dynamic changes on serial ECG recordings may be a clue during the differentiation process.

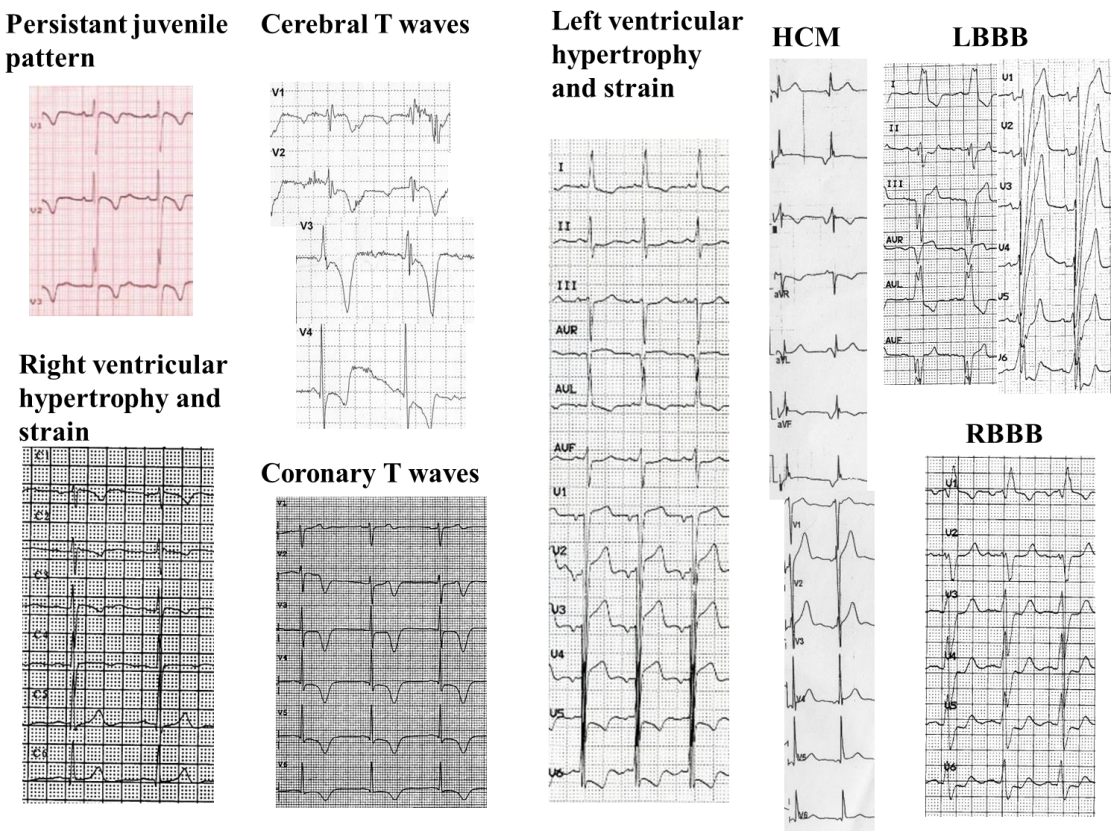


Figure 5/7.

Negative T waves can be seen very often. T wave negativity in leads V1-3 may be persistance of a pattern being normal at a young age also in adulthood, in addition, it may be a sign of right ventricular hypertrophy and overload (pulmonary hypertension or embolism). Diffuse precordial T wave inversion may occur in completely healthy individuals, but it may also be caused by cerebral T waves with a wide base and bizarre shape occurring after traumatic brain injury or intracerebral hemorrhage resulting in parenchymal destruction or, by coronary artery disease. Negative T waves and ST segment depression in leads I, aVL, V5-6 are signs of left ventricular strain in severe aortic stenosis, nevertheless, T wave inversion in a wide variety of locations is also visible in hypertrophic cardiomyopathy (HCM). As a secondary abnormality, negative T waves may also be visible in left and right bundle branch block.

In cases of severe ischemia with primary involvement of the anterior wall, the so-called Wellens' syndrome or sign may be observed. During this, typical changes of the T waves can be characteristically followed on serial ECG tracings rather than deviation of the ST segment. The essence of this condition is that T waves become biphasic in association with an anginal complaint, or shortly thereafter. Initially, it is only the terminal portion of T waves that becomes negative, often followed by the occurrance of deep T wave inversion. Negative (coronary) T waves frequently persist even after revascularisation as an accompanying sign of myocardial stunning.

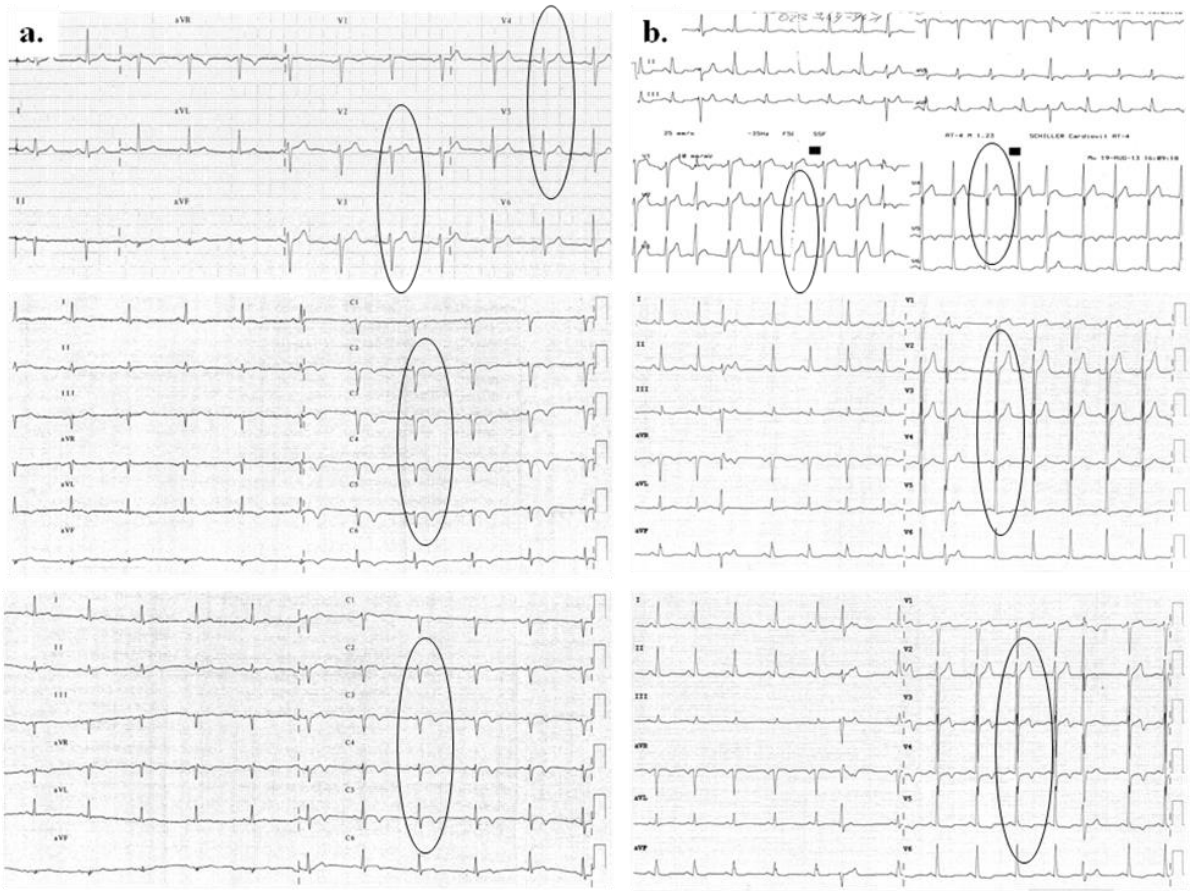


Figure 5/8. Wellens' syndrome. Typical T wave abnormalities in the precordial leads (especially leads V2-4) are observable on serial ECG tracings recorded during chest complaints caused by a subtotal LAD stenosis. In the series of ECG recordings in column a.), normal T waves are observable on the 1st ECG, while initially taller and peaked, hyperacute T waves in column b.), both of which gradually become biphasic, eventually negative, without any marked changes regarding the ST segments. The underlying cause of this finding is almost always a subocclusive stenosis of the LAD.

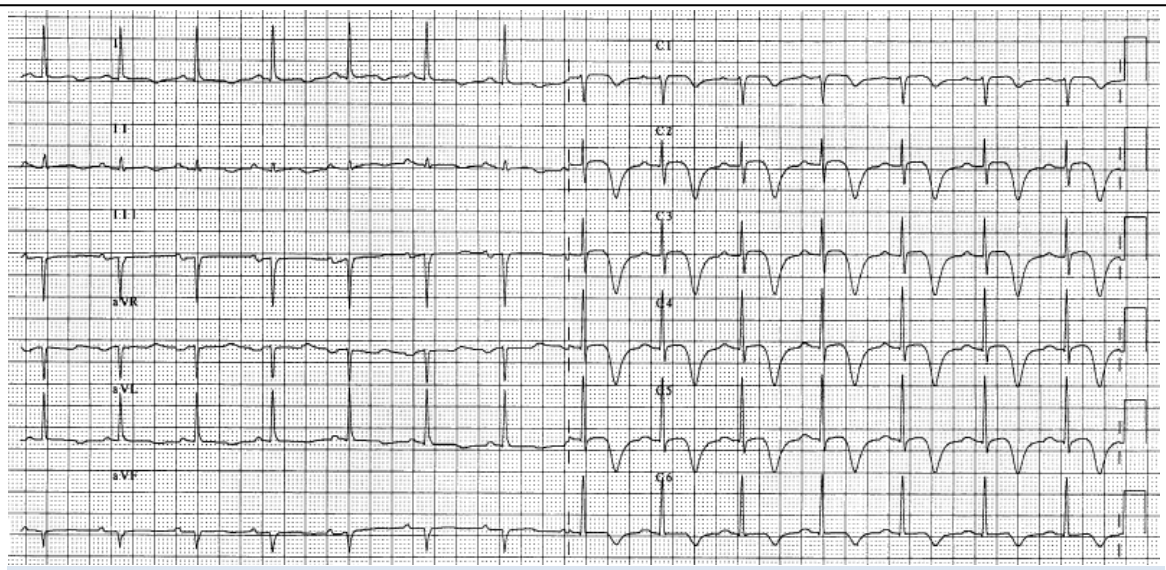


Figure 5/9. Deep T wave inversion in the precordial leads (i.e. negative, symmetrical and peaked T waves), together with ECG signs of a scar from an inferior myocardial infarction. (Sinus rhythm, 80 bpm, left axis deviation, normal AV conduction time, QS complexes in leads III, aVF, narrow QRS complexes, deep T wave inversion in the precordial leads.)

U wave inversion being discordant with the dominant QRS and T wave deflection and occurring in the precordial leads is a highly specific sign of subtotal LAD stenosis.

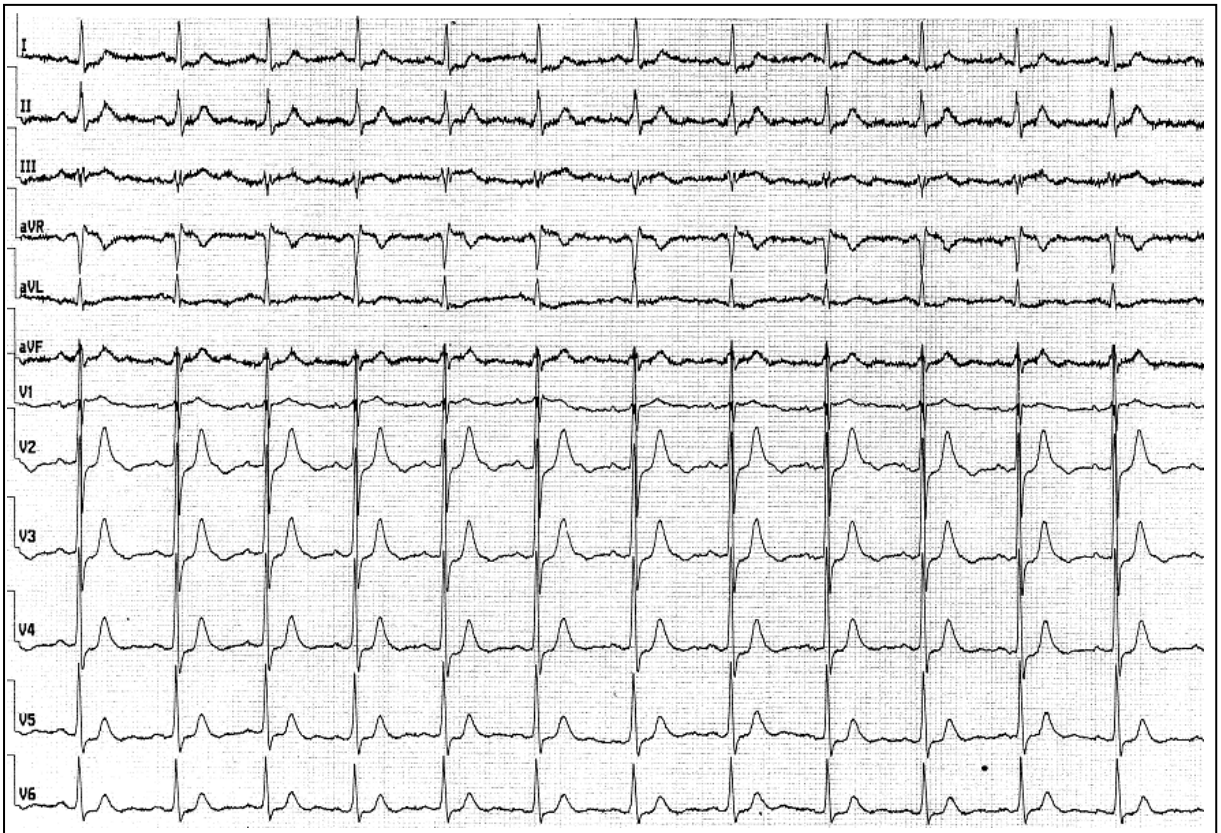


Figure 5/10.

The underlying cause of U wave inversion in the precordial leads (V2-4) was subtotal proximal LAD stenosis. Of course, it is not the only sign of ischemia that is visible on the ECG; this is also indicated by the significant and trivial ST segment depression in leads I, aVL and in the precordial leads, respectively, as well as by the ST elevation in lead aVR. (Sinus rhythm, 81 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction, horizontal ST segment depression of 1 mm in leads I, aVL and trivial ST depression in the precordial leads, U wave inversion in addition to positive T waves in the latter leads.)

5.6. Characteristic changes of the ST segment

Normally, ST segments are isoelectric (relative to the TP segment). Several forms of ST depression and elevation can be differentiated from morphological aspects, with these forms having different clinical significance.

ST segment depression may be upsloping (normal finding during physical exercise and tachycardia), may have a ‘sagging’ or ‘scooped’ appearance (in digitalis toxicity), or it may be horizontal or downsloping. If the ST segment measured at 60-80 ms from the J point is below the TP segment by ≥ 0.5 mm, it is referred to as ST segment depression, which is clinically significant if ≥ 1 mm. The latter two forms, especially the downsloping form, can be regarded as characteristic of myocardial ischaemia, aside from a few exceptions (e.g. left

ventricular strain). ST segment depression in the limb leads does not necessarily localize the site of ischemia, however, that in the precordial leads has a localization value. This is particularly true for ST segment changes presenting during exercise stress testing.

ST segment elevation does not always develop due to myocardial infarction either; for example, it may be a consequence of benign early repolarization (in this case, small r' waves are detectable directly before the beginning of the ST segment, at the end of the QRS complex). Based on its form, ST segment elevation may be concave (it may sometimes occur even normally) or 'saddle-shaped' (this, if present everywhere except leads aVR and V1, is characteristic of pericarditis). Convex and dome-shaped ST elevation can most often be seen in myocardial infarction. Deviations of the ST segment may also be induced by conditions causing T wave abnormalities (see there for details).

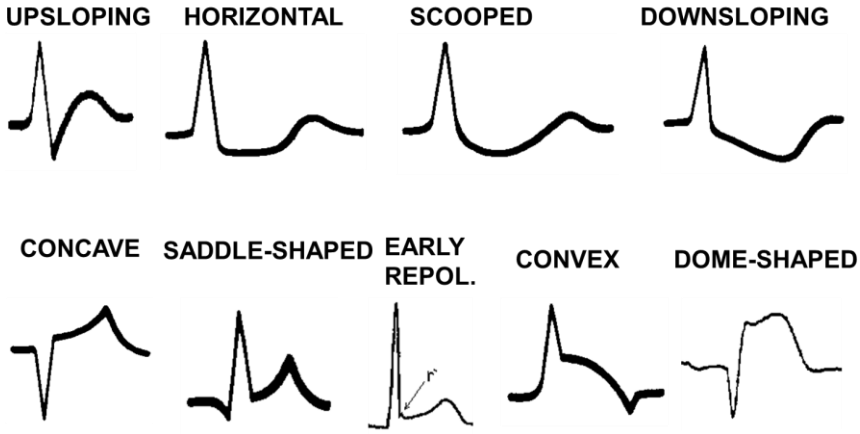


Figure 5/11.

Characteristic changes of the ST segment.

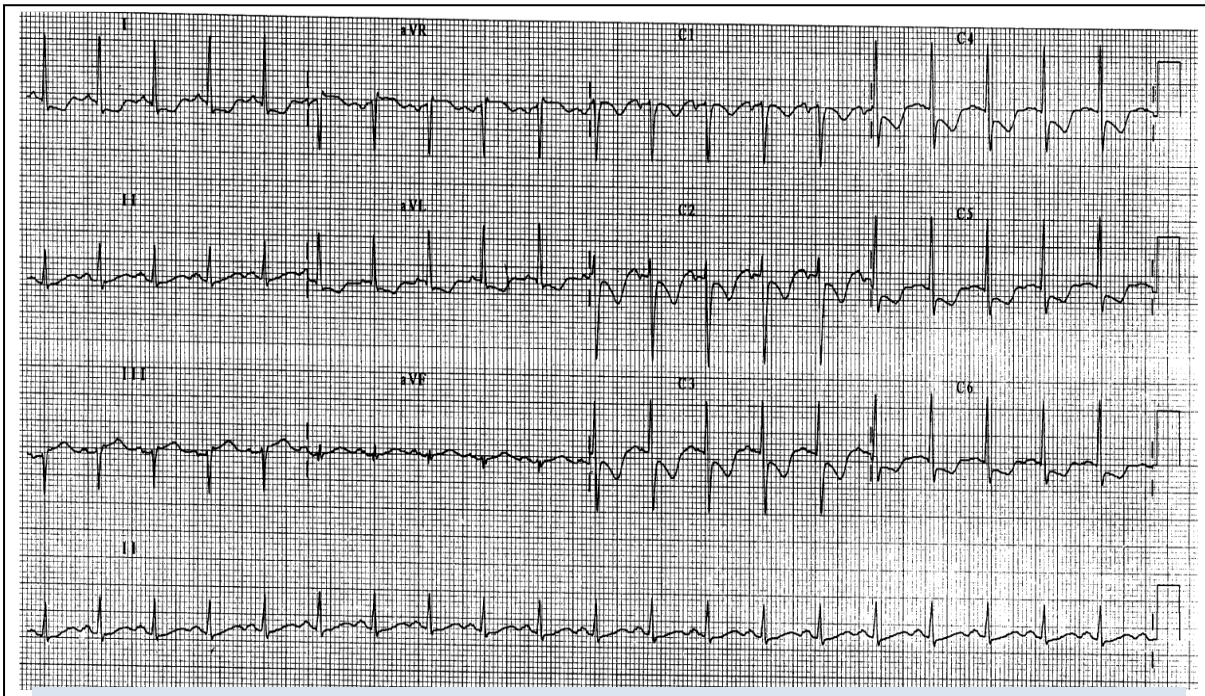


Figure 5/12. ECG signs of ischemia (in severe triple-vessel disease). Downsloping ST segment depression and negative T waves in leads I, aVL, V2-6. The pathological Q waves in leads III and aVF may be signs of a scar from an old inferior myocardial infarction. (Sinus tachycardia, normal QRS axis, normal AV conduction time, Q waves in leads III and aVF, downsloping ST segment depression of 1.5 to 2 mm and negative T waves in leads I, aVL, V2-6.

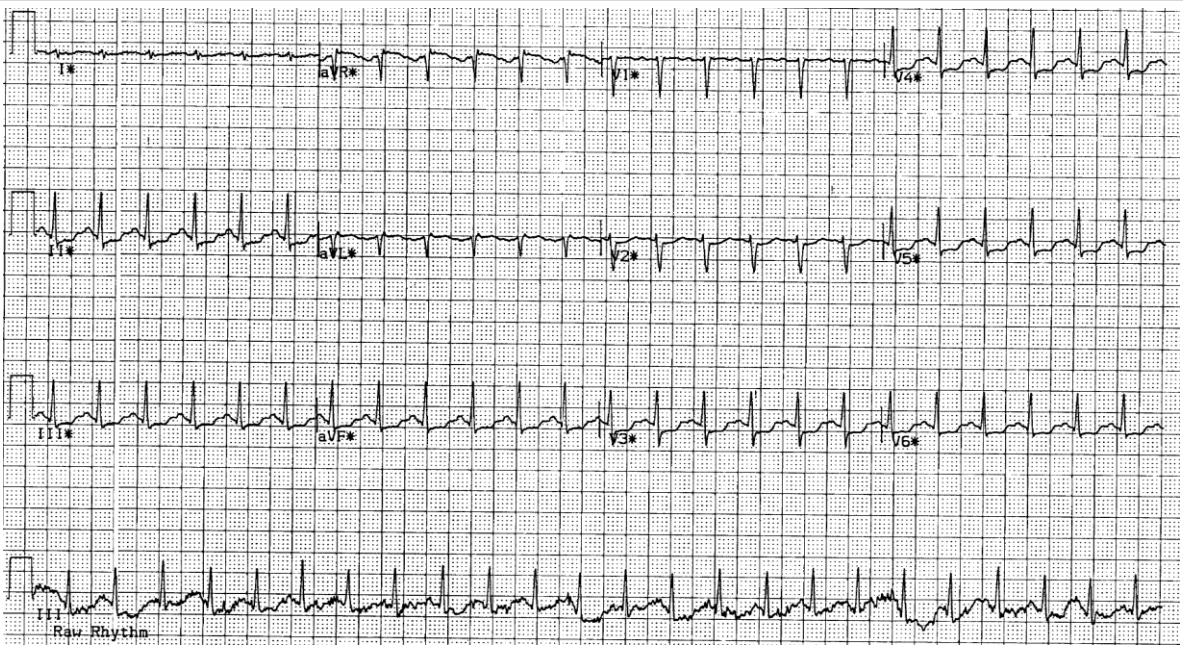


Figure 5/13. ECG signs of ischemia. Horizontal ST segment depression is visible in leads II, III, aVF and V3-6. (Sinus tachycardia, normal QRS axis, normal AV conduction time, normal ventricular conduction, horizontal ST segment depression of 1.5 to 2 mm and biphasic T waves in leads II, III, aVF and V3-6.)

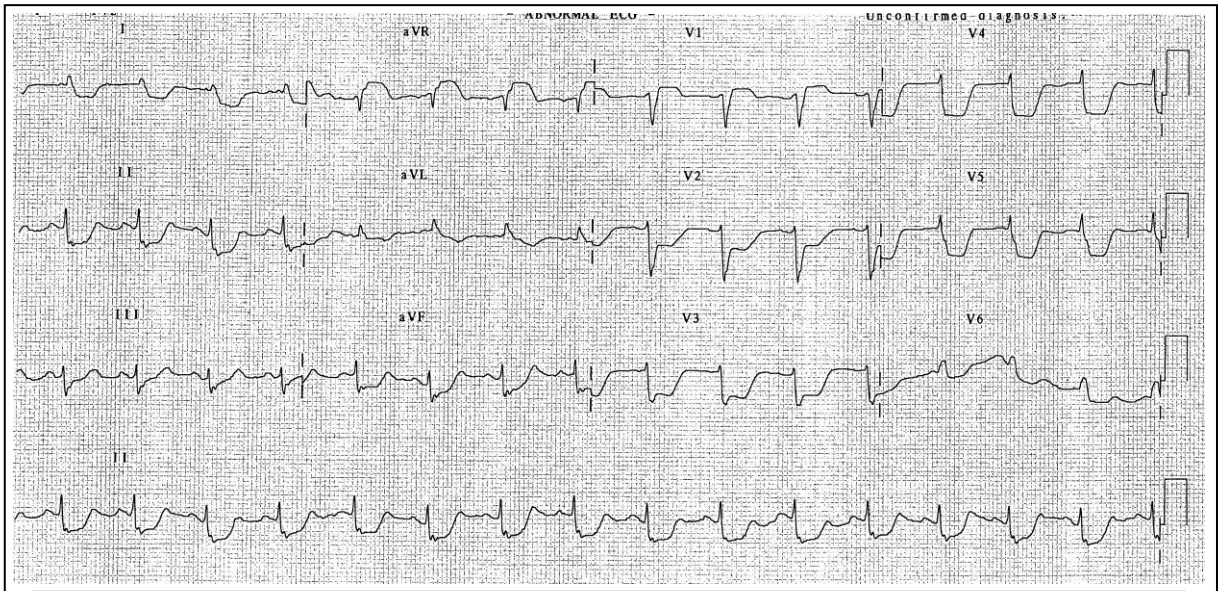


Figure 5/14.

Typical ECG pattern indicative of a critical stenosis (or transient occlusion) of the left main stem. Diffuse severe ST segment depression in all of the leads, except lead aVR and V1 where ST segment elevation is visible. This ECG pattern is consistent with global, severe ischemia of the left ventricle. (Sinus rhythm, 90 bpm, normal QRS axis, normal AV conduction time, R wave reduction and nonspecific intraventricular conduction disturbance in the precordial leads, ST segment depression of 5 to 6 mm all over the leads, ST segment elevation of 2 to 3 mm in leads aVR and V1.)

ST elevation is considered significant if it reaches or exceeds 1 mm, or 0.5 mm in leads V7-9 and VD1-3. However, in regard to V1-3, decision is made based on the table below whether ST segment elevation is significant or insignificant.

Gender / Age	< 40 years	> 40 years
Males	2.5 mm	2 mm
Females	2 mm	1.5 mm

Table 5/1. ST elevation considerations in contrast of the age.

5.7. Presence of Q waves on the ECG

Not too deep and not too wide q waves are detectable even on a normal ECG tracing. The cause of this is septal depolarisation with a direction of propagation from left to right and it may result in smaller q waves due to positional changes of the heart. In left axis deviation, q waves can be seen in leads I, aVL and V5-6, while in leads III and aVF in right axis deviation, but these waves generally do not exceed a height of 2 mm and are narrow (<40 msec). Deep and seemingly pathological q waves often occur in lead III, however, their amplitude decreases significantly or they even disappear in deep inspiration (by a change in the rotation and orientation of the heart). This is referred to as positional q waves. The amplitude of Q waves occurring in leads II, III and aVF after an inferior myocardial infarction may also change significantly, or they may even disappear, in deep inspiration, so it may be unlucky to use the term 'positional q waves'; moreover, q waves presenting only in lead III in an isolated fashion have no importance by themselves anyway. Under normal circumstances, QS complexes can be observed in leads aVR and V1, so this is not an abnormal finding; however, no q waves are normally present in leads V2-4.

Explanation for the development of q waves: By convention, negative deflections are visible on the ECG if the depolarization wavefront is moving away from a specific lead. Since ventricular depolarization is directed from the endocardium toward the epicardium, it is therefore always moving toward a given electrode (positive deflections), except for septal depolarization, which has a left-to-right direction and this is why small q waves can be seen in the left ventricular lateral leads (i.e. depolarization is moving away from these leads). In myocardial infarction, the necrotic regions are markers of electrical silence, so they practically are 'electrical windows' through which one can "look into" the chamber of the left ventricle and can record electric potentials (Q waves, QS complexes) moving away from the given electrode (endocardium → epicardium). (This is one possible explanation, there are other theories as well.)

Characteristics of pathological Q waves are the following:

- they are *wide* (>0.03 sec in the limb leads and >0.04 sec in the chest leads)(!);
- they are *deep* (their depth is greater than *25 %* of the height of consecutive R *wave*s or >4 mm) (!);
- they occur in leads where they are not present normally (leads V2-4);
- they appear *in at least two contiguous leads* concurrently.

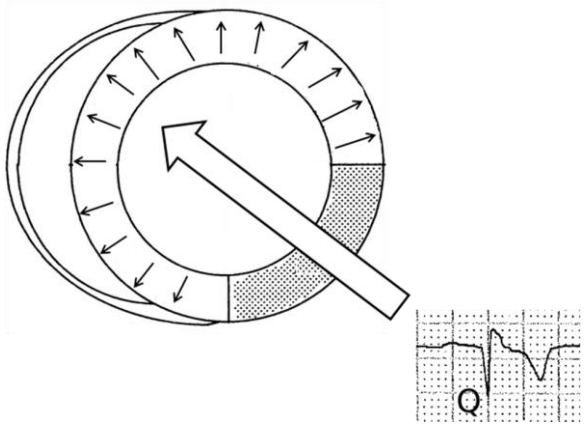


Figure 5/15.

Explanation for the development of q waves. Through the scarred region with electrical silence, the given electrode 'views' the depolarization of the opposite wall directed from the endocardium to the epicardium, which will appear as a wave moving away from that lead, i.e. negative (Q) wave.

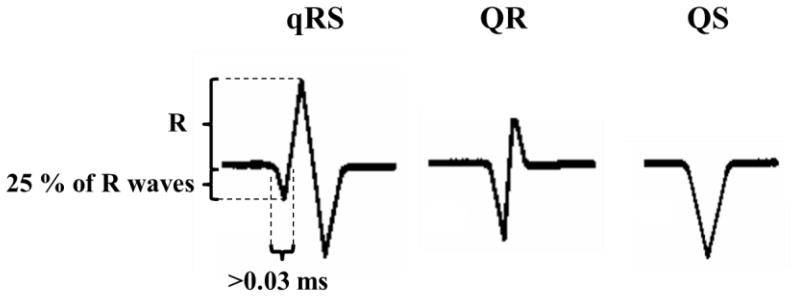


Figure 5/16.

Pathological Q waves.

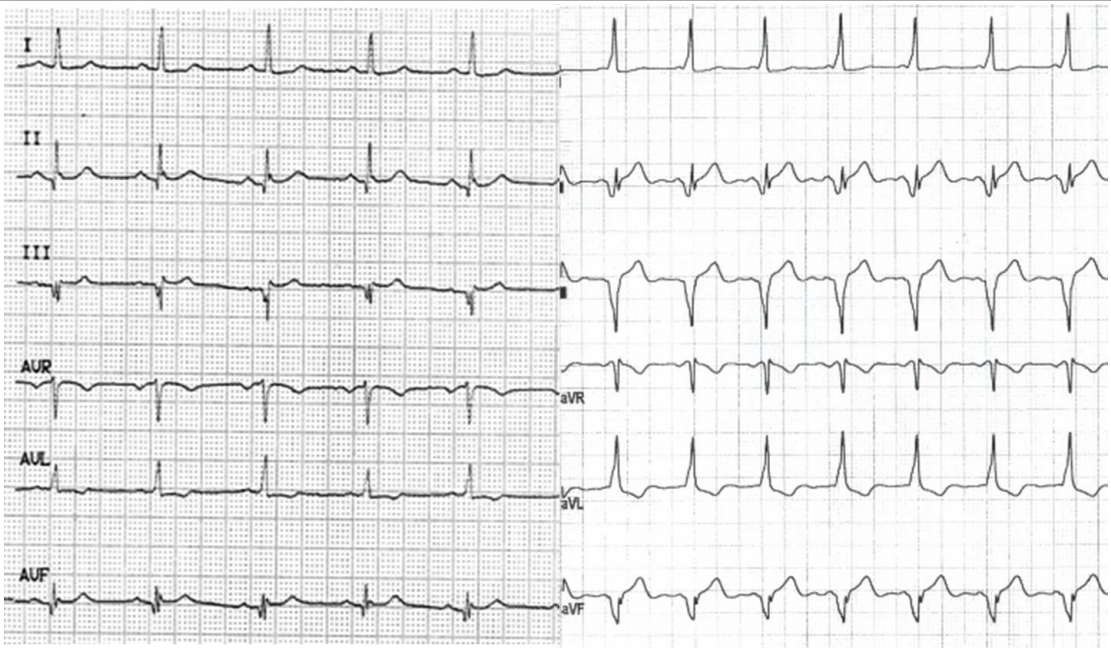


Figure 5/17. Non-infarction Q waves in leads II, III and aVF. On the left side of the figure, septal Q waves are visible in hypertrophic cardiomyopathy (HCM). On the right side, negative delta waves caused by an accessory pathway can be observed in leads II, III and aVF.

Conditions where pathological Q waves occur:

- myocardial infarction (they are missing in certain infarctions; non-Q wave myocardial infarction = subendocardial / nontransmural necrosis);

- left ventricular or septal hypertrophy (septal Q waves: in leads II, III, aVF or V5-6);
- left ventricular diastolic overload (in leads I, aVL, V5-6), acute cor pulmonale (in lead III);
- left bundle branch block (QS complexes in leads V1-2);
- WPW syndrome (negative delta waves in leads II, III, aVF).

5.8. Dynamics of disease course in myocardial infarction

Acut coronary artery occlusion is usually associated with a classic clinical picture and specific ECG abnormalities. Of the factors determining life expectancy of the patient, it is time that is of primary importance. This is why it is important to know the sequence of consecutive ECG changes over time and the explanation of their development. Please remember that an ECG recorded during chest pain provides information only about the few seconds when it has just been performed, so it is important to record a series of ECG tracings (e.g. after 10 minutes or at peak intensity of the pain) in cases of uncertainty.

The first few hours are called the hyperacute phase. The first ECG abnormality observed after a coronary artery occlusion is the occurrance of tall and peaked T waves, also known as hyperacute T waves (transmural ischemia), and dome-shaped (convex) ST segment elevation (transmural injury), which persist for hours, sometimes even for days.

In the acute phase, three stages can be differentiated based on the severity of transmural injury. For the first stage, only trivial ST elevation is associated with the tall T waves; for the second stage, ST elevation starts from the lower half of the downstroke of the QRS complex; while for the third stage, it commences from the upper third (peak) of the QRS and fuses with the tall T wave. In the latter case, the shape of the ECG tracing mostly reminds of that of an action potential recorded from a cardiomyocyte, or a tombstone according to others. The latter metaphor is more appropriate also due to the fact because ST segment elevation myocardial infarctions with such a pattern have the highest mortality rates.

The events of the first day are called the acute phase. In the second half of the acute phase, ST elevation slightly decreases and becomes curved, T waves become inverted and pathological q waves begin to evolve. In the subacute phase of myocardial infarction, changes initiated in the acute phase become even more characteristic. Later stages (weeks or months after the event) are not referred to as a chronic phase, but as a ‘pattern of scarring’ on the ECG as a sign of prior and complete myocardial infarction.

There is no longer ST segment elevation in this phase, however, pathological Q waves have already developed (except non-Q-wave myocardial infarction) and T waves can be both positive and negative.

It is frequently seen especially after an anterior (or rarely, inferior) myocardial infarction that ST elevation persists even months after the event; this was previously attributed to left ventricular aneurysm formation, but nowadays rather to extensive dyskinesia (i.e. pathological systolic motion) of the cardiac walls.

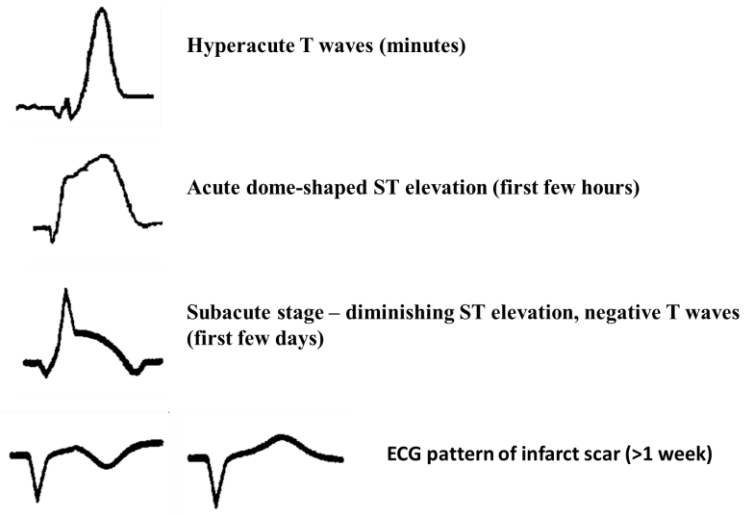


Figure 5/18.

ECG changes in myocardial infarction over time.

The above described typical course of the ECG pattern was substantially altered by the recent medical treatment (thrombolysis, primary percutaneous coronary intervention - PCI or PTCA). Whether revascularization is complete or incomplete, it may make the clinical presentation even more diverse, so it may happen that after mechanical opening (i.e. primary PCI) of the occluded vessel, ST elevation will show regression within minutes. However, if blood circulation is not restored at tissue level, then, in spite of the success in opening the obstructed vessel, necrosis will still progress and ST segment resolution will occur much later. Changes of the ST segment (ST segment resolution) following revascularization are a sensitive marker of restoration of myocardial blood flow at tissue level and a predictor of mortality and long-term consequences of myocardial infarction.

If ST segment resolution one hour after the reperfusion treatment (thrombolyis, PCI) is less than 30 %, the expected reperfusion did not occur at tissue level; however, if exceeding 50 % and 70 % in anterior and inferior myocardial infarction, respectively, and pain has also ceased, complete reperfusion could then be achieved.

Figure 5/19.

Why ST elevation is referred to as ‘dome-shaped’?

5.9. Explanation for the ECG changes in myocardial infarction

In a fully evolved myocardial infarction, all three stages of hypoxic injury of the myocardium can be observed on the ECG recording. The different zones of injury form concentric circles in the myocardium. It is the central zone (supplied exclusively by the occluded coronary artery) that suffers the most injury, hence, (irreversible) **necrosis** develops here, represented on the ECG as **pathological Q waves**. Areas adjacent to the zone of necrosis suffer injury to a lesser degree. Reversible injury is observable (due to collateral branches and other mechanisms) here, so **ST segment elevation** develops in accordance with the zone of **injury** and, by the electrical activity of the outermost zone of **ischemia**, the development of **negative T waves** can be explained. In the absence of reperfusion, there is centrifugal propagation of the necrosis, thereby involving a larger and larger area.

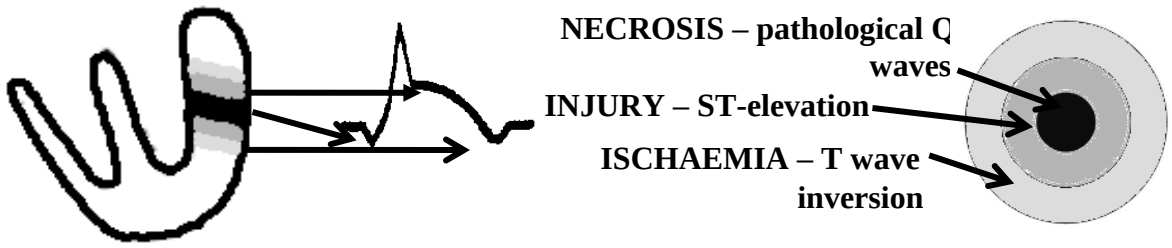


Figure 5/39.

Explanation for the ECG changes in myocardial infarction.

5.10. Interpretation of reciprocal changes

Under normal circumstances, a single lead provides information not only on the nearest ventricular segment, but indirect signs may also be detected from the cardiac wall lying opposite to the respective segment (e.g. for leads V1-3, one can obtain information not only on the left ventricular anterior wall and interventricular septum, but also on the posterior segment opposite to this region). In myocardial infarction, it is the above described ECG abnormalities that provide information on the area with necrosis, moreover, reciprocal changes can be recorded from the cardiac walls opposite to the necrotic region and the deviation of ECG waves will just be the opposite. (For example, in anteroseptal myocardial infarction Q waves, ST elevation and negative T waves are visible in leads V1-4. If the dorsal electrodes, i.e. VD1-3 or V7-9, are placed, tall R waves, ST segment depression and positive T waves will be observed here.) The lack of reciprocal changes distinguishes other medical conditions associated with ST elevation (pericarditis, benign early repolarization, etc.) from myocardial infarction (except for Prinzmetal or vasospastic angina; reciprocal changes are also detectable in this case). Reciprocal changes can only be interpreted in identical planes, thus, an inferior myocardial infarction (limb leads II, III and aVF) cannot have reciprocal changes in the precordial leads because these represent two different planes; in addition, it is about bipolar leads in one case and unipolar leads in the other case. In inferior myocardial infarction, ST segment depression presenting in leads consistent with the anterior wall is a reciprocal change of posterior extension of the infarction rather than that of inferior myocardial infarction. Knowledge of reciprocal changes is helpful not only because they help

distinguish other medical conditions associated with ST elevation from myocardial infarction, but also because it may happen that ST segment depression in the precordial leads (V1-4) on a 12-lead surface ECG is falsely interpreted as a sign of ischemia of the anterior wall, although it is posterior ST segment elevation myocardial infarction (STEMI) actually.

This interpretation issue may delay establishing a correct diagnosis and the start of reperfusion therapy, thereby impairing life expectancy of the patient. It is therefore important to always record an ECG in the dorsal leads (VD or V7-9) in case precordial ST segment depression is present, especially if ST segment depression is associated with positive, rather than negative, T waves.

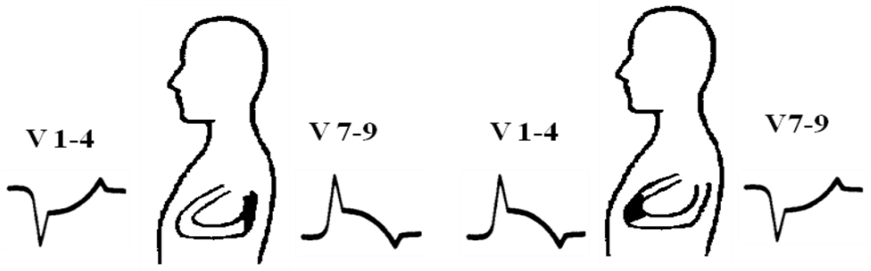


Figure 5/21.

Interpretation of reciprocal changes in myocardial infarction.

5.11. Localization of myocardial infarction

Based on their location, three major groups can be differentiated: anterior (LAD), inferior (RCA) and posterior (CX) infarctions. On the basis of the above described ECG changes (ST segment elevation, pathological Q waves), the following classification by location can be given: - ANTERIOR (anterior-septal-lateral): in V1-6, I, aVL;

- INFERIOR: II, III, aVF;

- POSTERIOR (postero-lateralis): I, aVL, V5-9 (VD1-3).

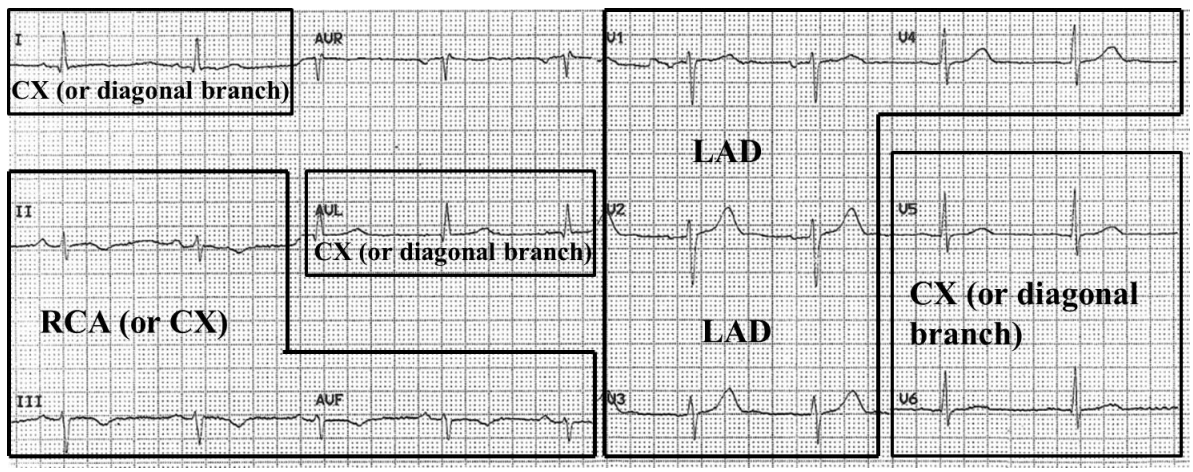


Figure 5/22.

Based on the site of appearance of the ST segment elevation, it can often be determined that the occlusion of which blood vessel resulted in these changes.

Simply put, anterior (territory of the LAD) and non-anterior wall (CX and RCA territory) myocardial infarctions can be differentiated. Localization of the level of blood vessel occlusion assists in the evaluation of the amount of myocardial mass in jeopardy.

Anterior STEMI (LAD occlusion)

- ST segment elevation in the chest leads

  - if ST elevation of ≥2 mm is observable in lead V1, the level of occlusion is proximal to the origin of the first septal branch;

  + in lead aVR as well – very proximal or ostial LAD occlusion;

  + in lead aVL as well – the level of occlusion is before the origin of the first diagonal branch.

Non-anterior wall STEMI

- RCA occlusion:

  - ST elevation in lead III > II; ST depression in lead I;

  - right ventricular involvement: V3-4R;

  - posterior wall involvement: precordial ST segment depression.

- CX occlusion:

  - ST elevation in lead II ≥ III; ST elevation or isoelectric ST segments in lead I;

  - no ST elevation on the 12-lead surface ECG - true posterior STEMI (with reciprocal ST segment depression in the precordial leads);

  - lateral wall involvement: I, aVL, V5-6.

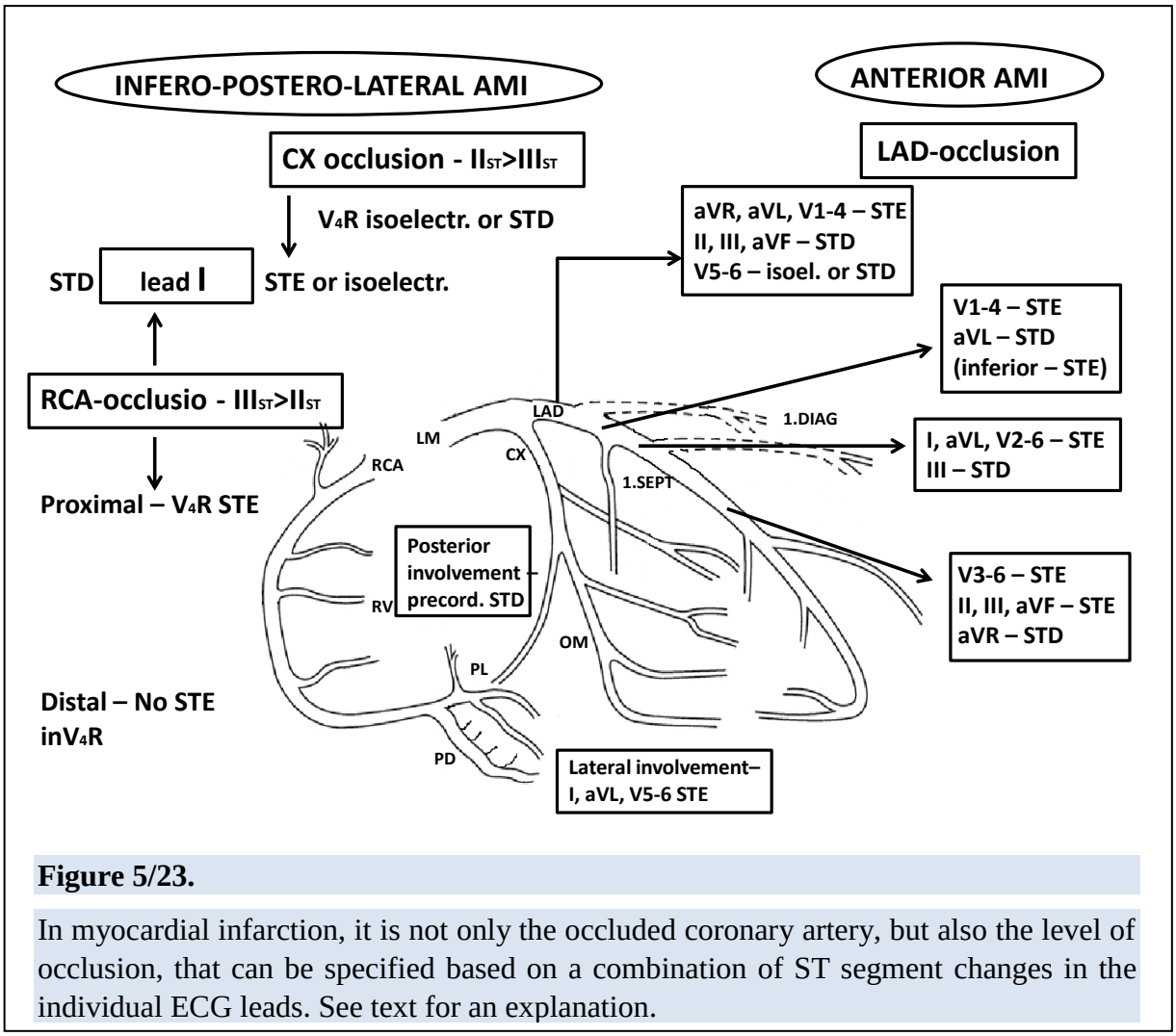


Figure 5/23.

In myocardial infarction, it is not only the occluded coronary artery, but also the level of occlusion, that can be specified based on a combination of ST segment changes in the individual ECG leads. See text for an explanation.

Right ventricular infarction: it generally occurs in association with an inferior myocardial infarction, i.e. in 2/3 of cases. One might suspect a proximal right coronary artery occlusion, if ST elevation is visible in V3-4R out of the right ventricular leads of the ECG recorded in inferior wall ST segment elevation myocardial infarction.

One should also think of the presence of right ventricular infarction if ST elevation occurs in leads V1-3 in association with, or without, ST elevation in the inferior region. This finding is differentiated from anterior wall myocardial infarction based on the fact that while there is a decrease in the degree of ST elevation in leads V1-3 in right ventricular infarction, it increases in case of an anterior wall infarction. Isolated right ventricular infarction is a rare entity, it can develop when there is left dominant coronary circulation and the right coronary artery only gives off right ventricular branches.

It is important to consider right ventricular involvement in inferior wall STEMI since this may result in substantial changes in the therapeutic strategy, e.g. the administration of nitrates is contraindicated in right ventricular involvement.

Importance of the use of additional leads cannot be stressed enough. Placement of right-sided leads is mandatory in inferior myocardial infarction, just as that of leads V7-9 in case of ST segment depression in the precordial leads.

Sample ECGs being typical of myocardial infarctions with different duration are visible on the following pages. Please note that ST segment elevations with a different morphology can be detected in acute occlusion of a coronary artery, however, they are all typical cases of acute ST segment elevation myocardial infarction (STEMI).

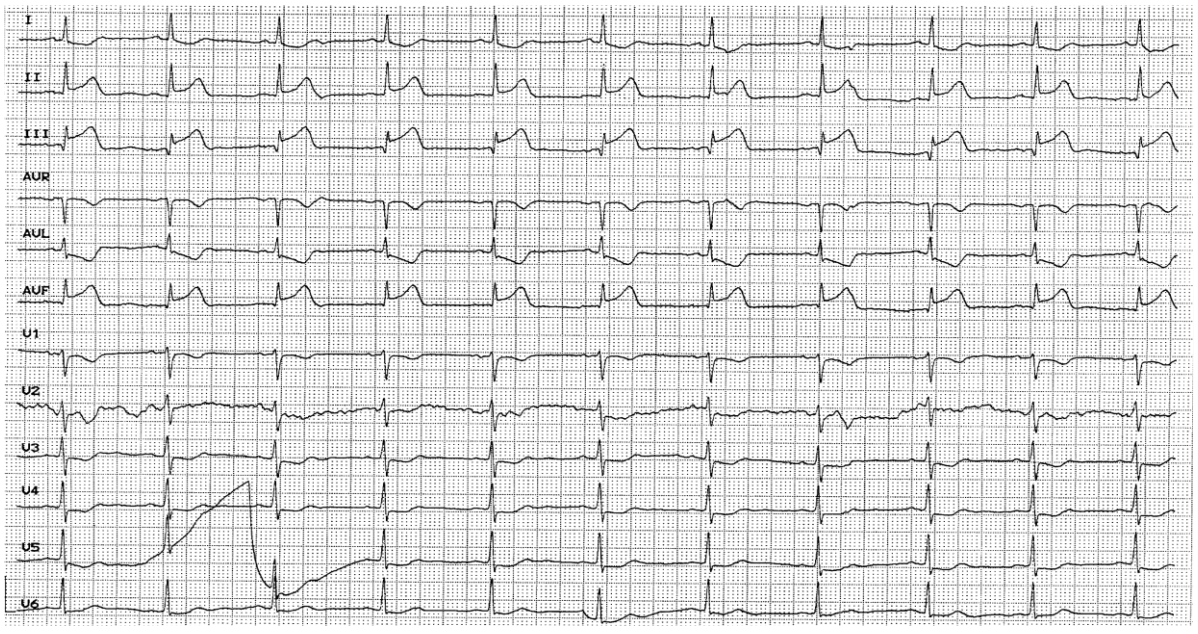


Figure 5/24. Inferior STEMI. ST segment elevation and positive T waves in leads II, III and aVF, reciprocal ST segment depression in leads I and aVL. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 80 bpm, normal AV conduction time, normal QRS axis, normal ventricular conduction, ST segment elevation of 1 to 3 mm and positive T waves in leads II, III and aVF, marked reciprocal changes in leads I and aVL and trivial ones in the precordial leads.)

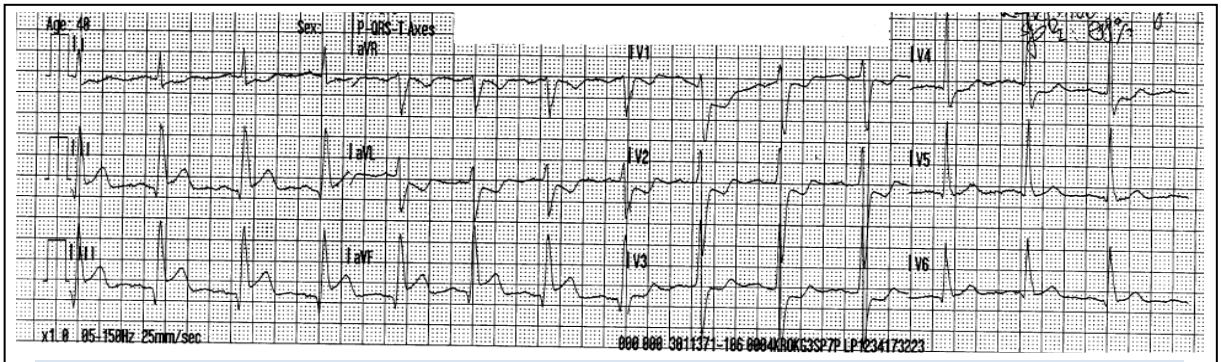


Figure 5/25.

Inferior STEMI. ST segment elevation and positive T waves in leads II, III and aVF, reciprocal ST segment depression in leads I and aVL, the ST segment depression in leads V1-4 indicates posterior extension of the infarction. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 90 bpm, normal AV conduction time, normal QRS axis, q waves, ST segment elevation of 2 mm and positive T waves in leads II, III and aVF, reciprocal ST segment depression in leads I, aVL and V1-4.)

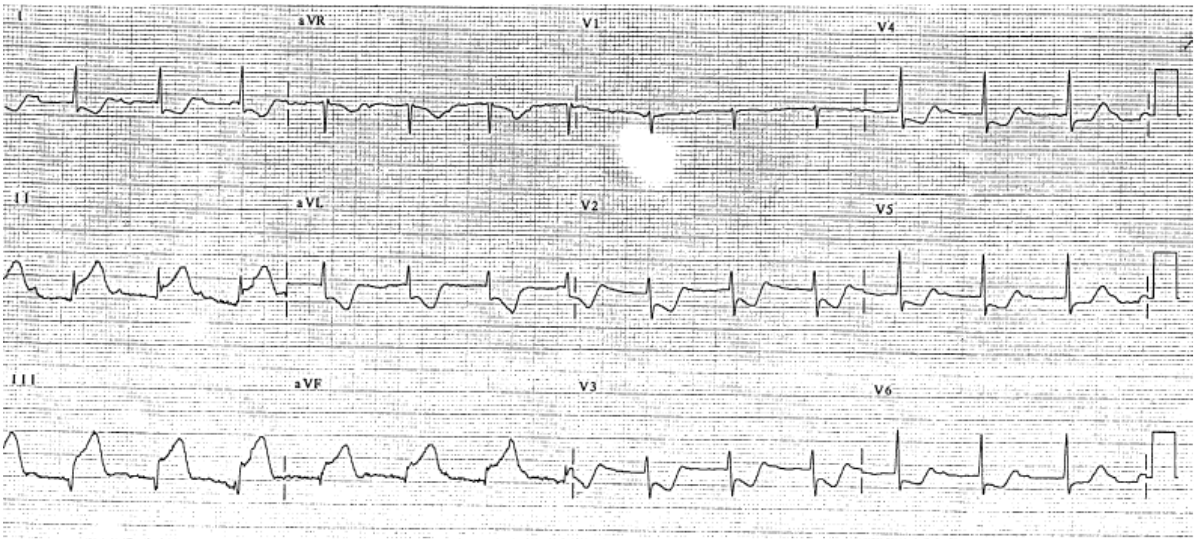


Figure 5/26.

Inferior STEMI. Dome-shaped ST segment elevation in leads II, III and aVF, the reciprocal ST segment depression in leads I and aVL and the significant ST segment depression in the precordial leads are a marker of extensive posterior wall involvement. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 80 bpm, significantly prolonged AV conduction, Q waves in leads III, aVF, dome-shaped ST segment elevation of 3 to 4 mm in leads II, III and aVF, marked reciprocal ST segment depression in leads I, aVL and V2-6.)

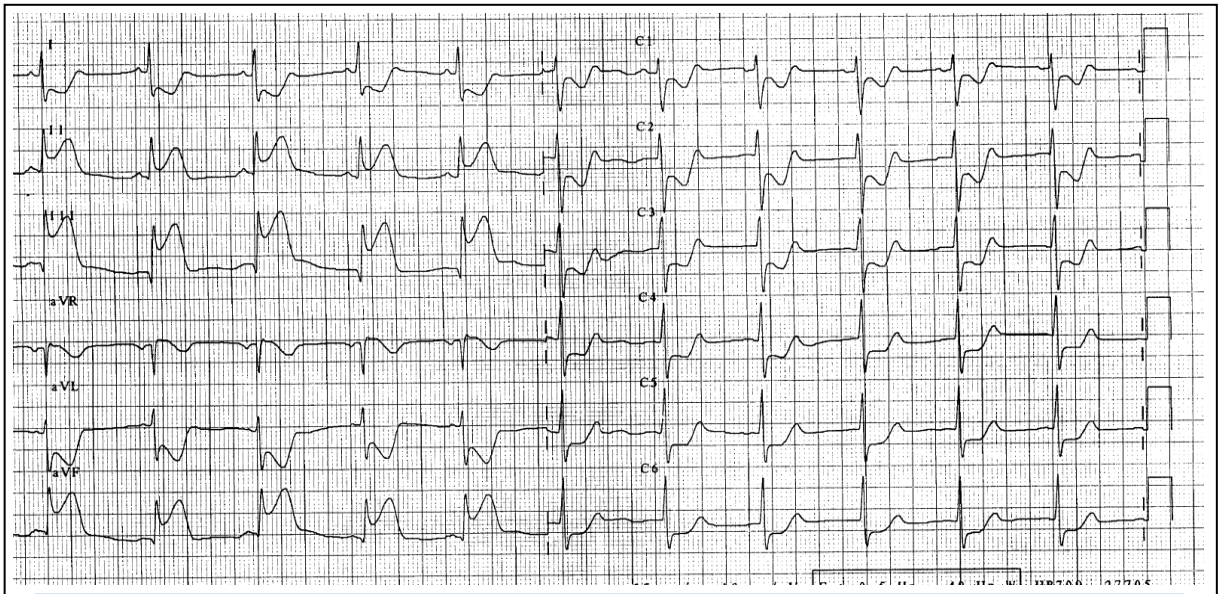


Figure 5/27. Inferior STEMI. Dome-shaped ST segment elevation in leads II, III and aVF, the reciprocal ST segment depression in leads I and aVL and the significant ST segment depression in the precordial leads are a marker of extensive posterior wall involvement. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 65 bpm, normal AV conduction time, q waves in leads III, aVF, dome-shaped ST segment elevation of 3 to 5 mm in leads II, III and aVF, marked reciprocal ST segment depression in leads I, aVL, V1-6.)

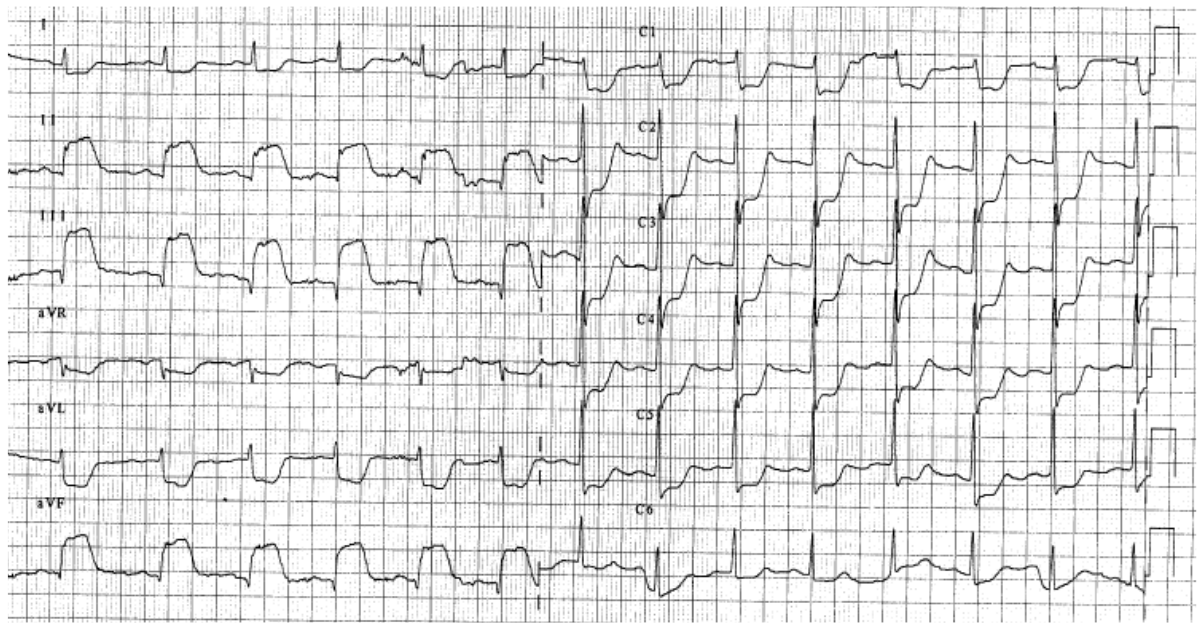


Figure 5/28. Inferior STEMI. Giant, dome-shaped ST segment elevation in leads II, III and aVF. The reciprocal ST segment depression in leads I and aVL and the significant ST segment depression in the precordial leads are a marker of extensive posterior wall involvement. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 80 bpm, normal AV conduction time, Q waves in leads II, III and aVF, dome-shaped ST segment elevation of 6 to 9 mm in leads II, III and aVF, marked reciprocal ST segment depression in leads I, aVL, V1-6.)

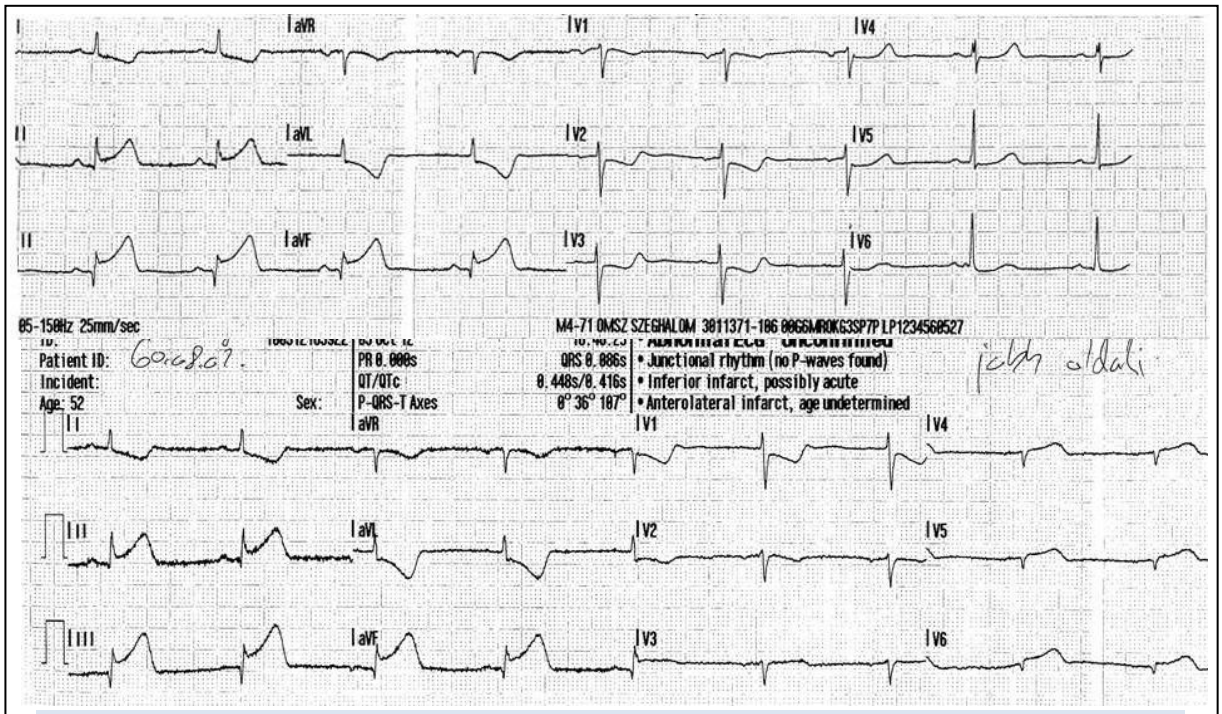


Figure 5/29. Inferior STEMI with right ventricular and posterior wall involvement. On the upper tracing, classic ST segment elevation occurring in leads II, III and aVF is detectable, along with reciprocal ST segment depression in leads I and aVL and ST segment depression in leads V1-3 as a reciprocal change of posterior ST elevation. On the right side of the lower ECG tracing, ST segment elevation occurring in the right ventricular leads (V4-6 = V4-6R) indicates right ventricular involvement (proximal RCA occlusion). (Sinus rhythm, 55 bpm, normal AV conduction time, normal QRS axis, Q waves in leads III and aVF, ST segment elevation of 2 to 3 mm in leads II, III and aVF, reciprocal ST segment depression in leads I, aVL and V1-3, ST segment elevation of 1 mm in the right ventricular leads.)

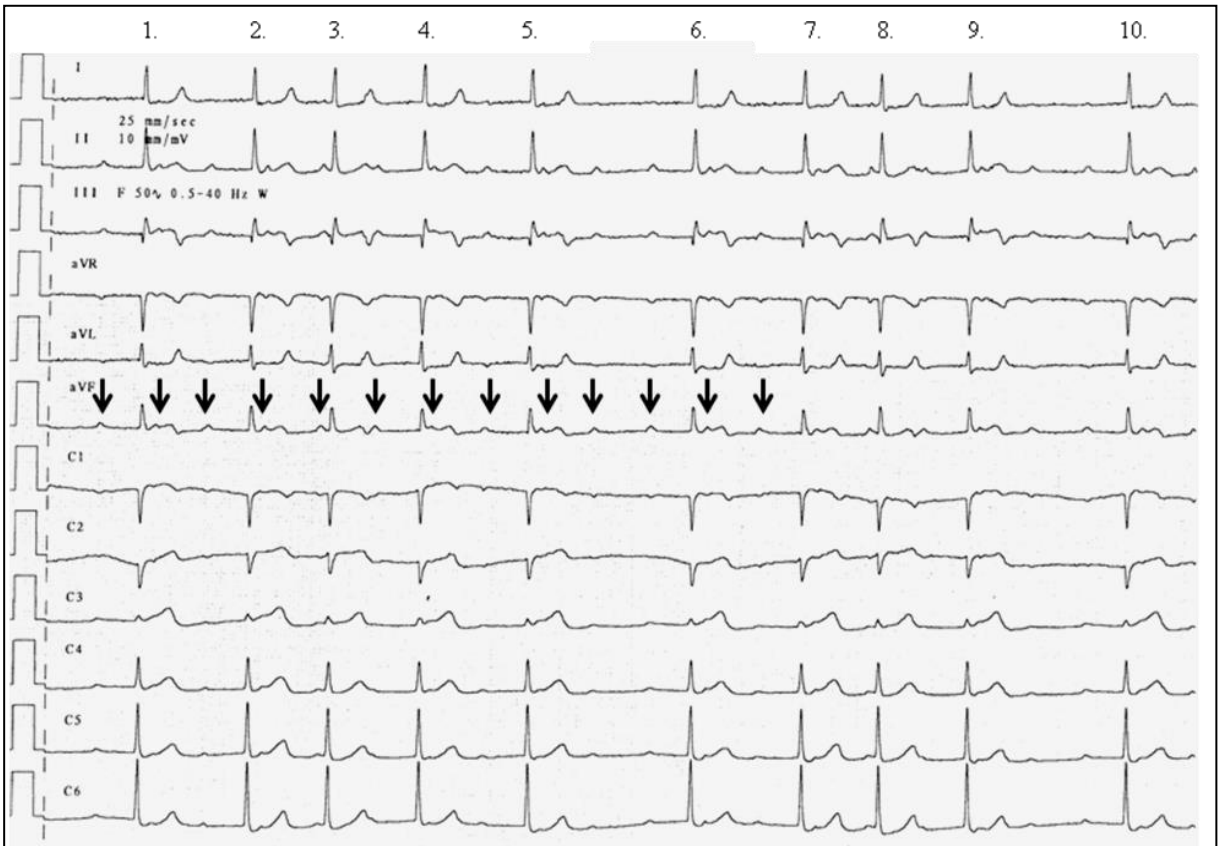


Figure 5/30. Inferior STEMI with high-degree AV block. Trivial ST elevation is visible in leads III and aVF as well as reciprocal ST segment depression in leads I and aVL. Furthermore, several blocked P waves and, in some cases, conducted beats along with prolonged AV conduction are observable (1st, 2nd, 4th, 5th, 6th, 7th, 9th and 10th QRS complex) The underlying cause of these findings was RCA occlusion, which provided the atrioventricular nodal branch and the branch supplying the upper portion of the bundle of His. (Sinus tachycardia, normal QRS axis, high-degree AV block with a normal average ventricular rate, normal ventricular conduction, trivial ST segment elevation in leads III and aVF, reciprocal ST segment depression in leads I and aVL.)

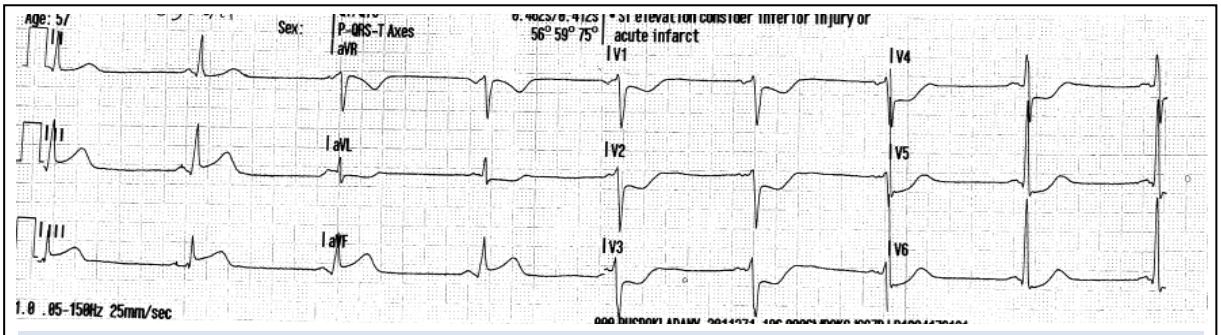


Figure 5/31. Inferior STEMI caused by CX occlusion. The size of ST segment elevation in leads II and III is about the same and the reciprocal ST segment depression in the precordial leads suggesting posterior involvement is more pronounced than the inferior ST elevation. (In the previous cases with RCA occlusion, the degree of ST elevation in lead III exceeded that in lead II in each case, and the degree of ST segment depression in the precordial leads was lower than that of the inferior ST elevation.) (Sinus rhythm, 50 bpm, normal QRS axis, normal AV conduction time, small q waves and ST segment elevation of 2 mm in leads II, III and aVF, reciprocal ST segment depression in leads I, aVL and the precordial leads)

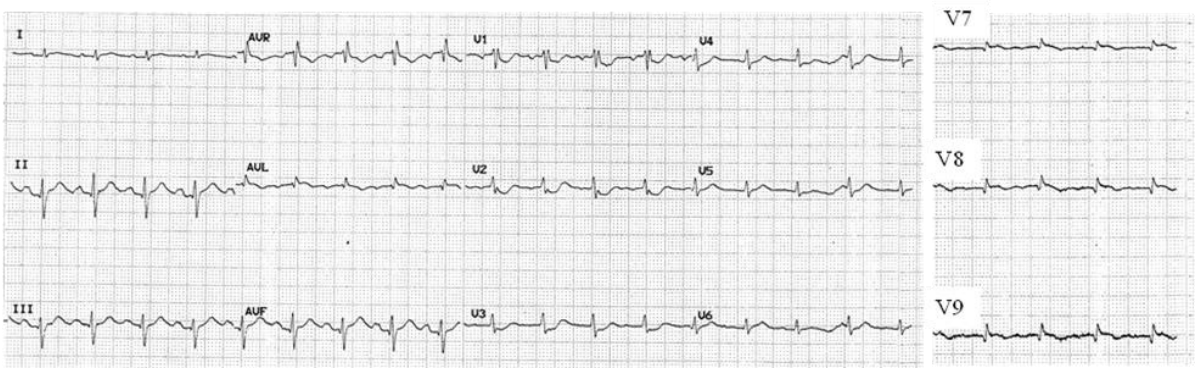


Figure 5/32.

True posterior STEMI. Only ST segment depression in leads V1-3 is visible on the 12-lead surface ECG, however, ST segment elevation in the dorsal leads may help reveal that it is STEMI actually. The underlying cause of these findings was total occlusion of the Cx. (Sinus tachycardia, right axis deviation, normal AV conduction time, incomplete right bundle branch block, trivial and unequivocal ST segment elevation with negative T waves in leads aVL and V7-9, respectively, ST segment depression of 1 to 1.5 mm in leads V1-3.)

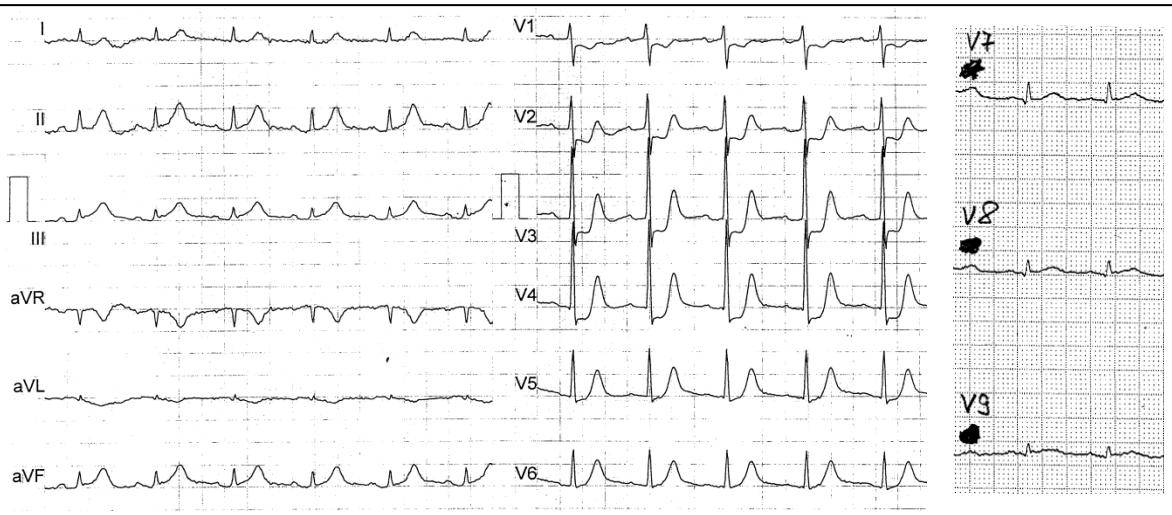


Figure 5/33.

True posterior STEMI. Precordial ST segment depression and trivial elevation of the ST segment in leads II, III and aVF can be seen on the 12-lead surface ECG, however, an evaluation of leads V7-9 led to an unambiguous diagnosis. The underlying cause of these findings was Cx-OM occlusion. (Sinus rhythm, normal QRS axis, 75 bpm, normal AV conduction time, tall R waves in lead V1 (reciprocal change of Q waves), otherwise normal ventricular conduction, ST segment depression of 2 to 3 mm and positive T waves in leads V2-5, trivial ST segment elevation and positive T waves in leads V7-9.)

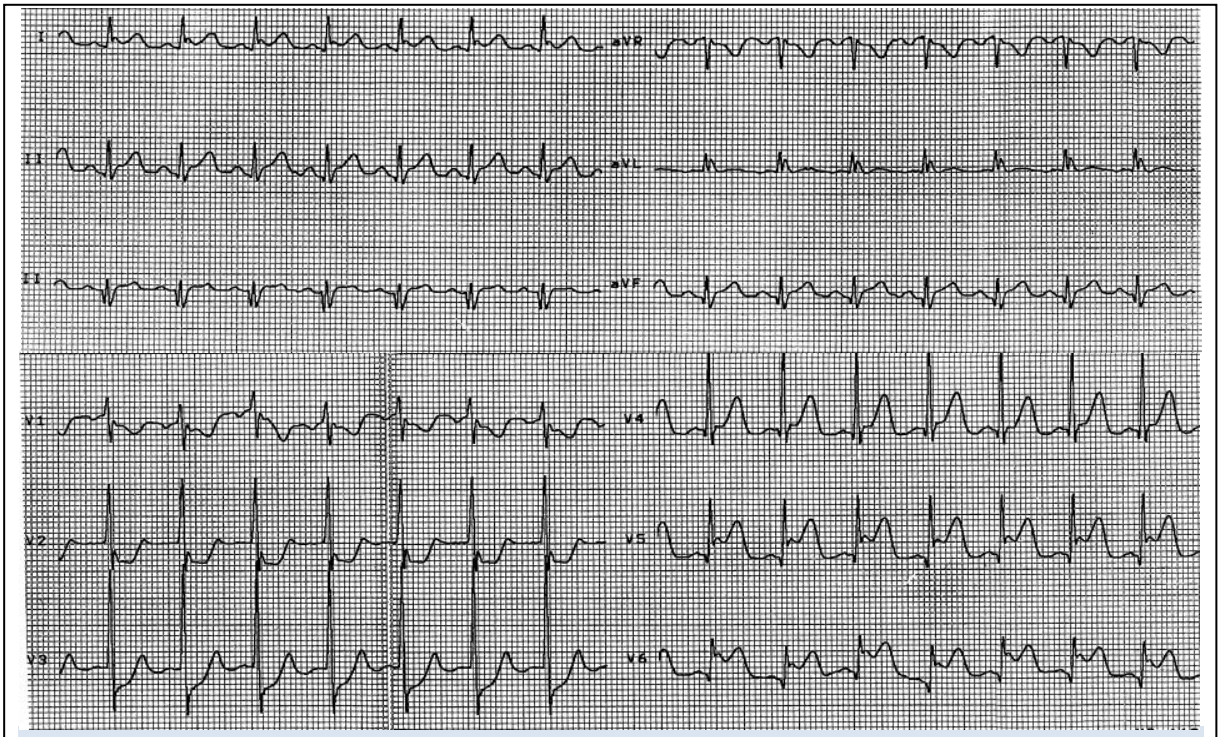


Figure 5/34. Postero-lateral STEMI (Cx occlusion). Only reciprocal changes of the posterior wall involvement are visible in leads V1-3, while there is unequivocal ST elevation in leads representing the lateral wall (V5-6), however, there is trivial ST elevation in leads I, II, III and aVF as well. (Sinus rhythm, 100 bpm, normal QRS axis, normal AV conduction time, q waves in leads III, aVF and V4-6, tall R waves in lead V1 (reciprocal change of Q waves), trivial ST segment elevation in leads I, II and aVF, dome-shaped ST elevation of 3 to 4 mm in leads V5-6, reciprocal ST segment depression and positive T waves in leads V1-3.)

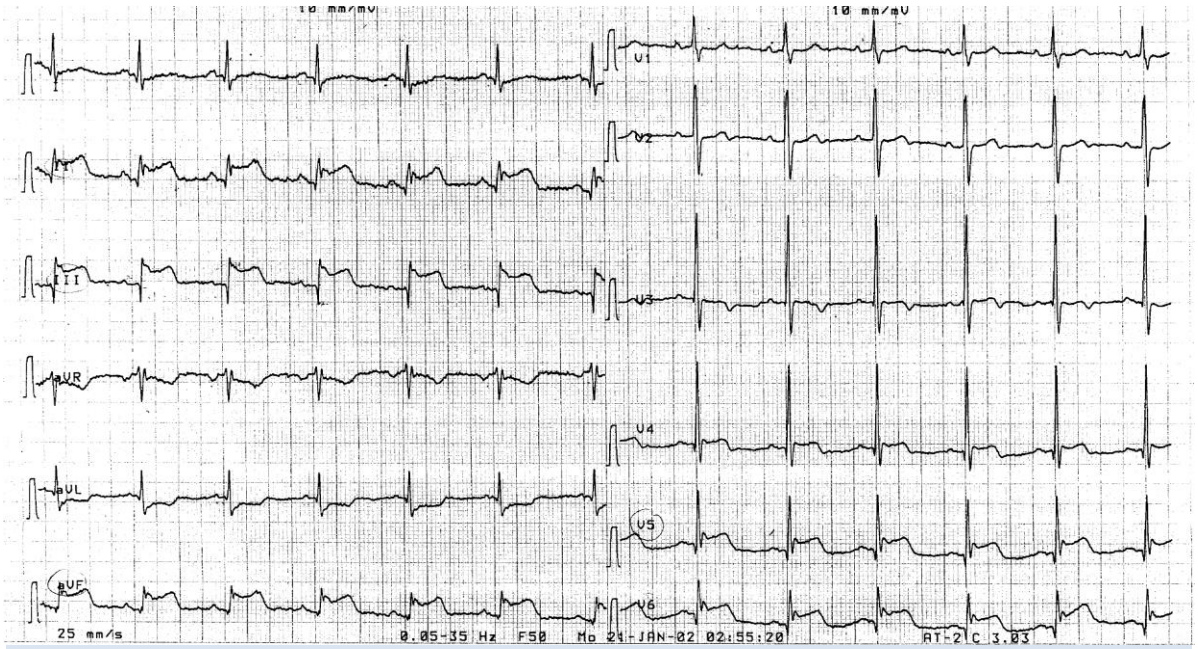


Figure 5/35. Inferolateral STEMI. Dome-shaped ST segment elevation in leads II, III, aVF and V5-6. The underlying cause of these findings was occlusion of a well-developed circumflex artery. (Sinus rhythm, 75 bpm, normal QRS axis, normal AV conduction time, tall R waves in lead V1 (reciprocal change of Q waves), Q waves, ST segment elevation of 3 mm and positive T waves in leads II, III, aVF and V5-6.)

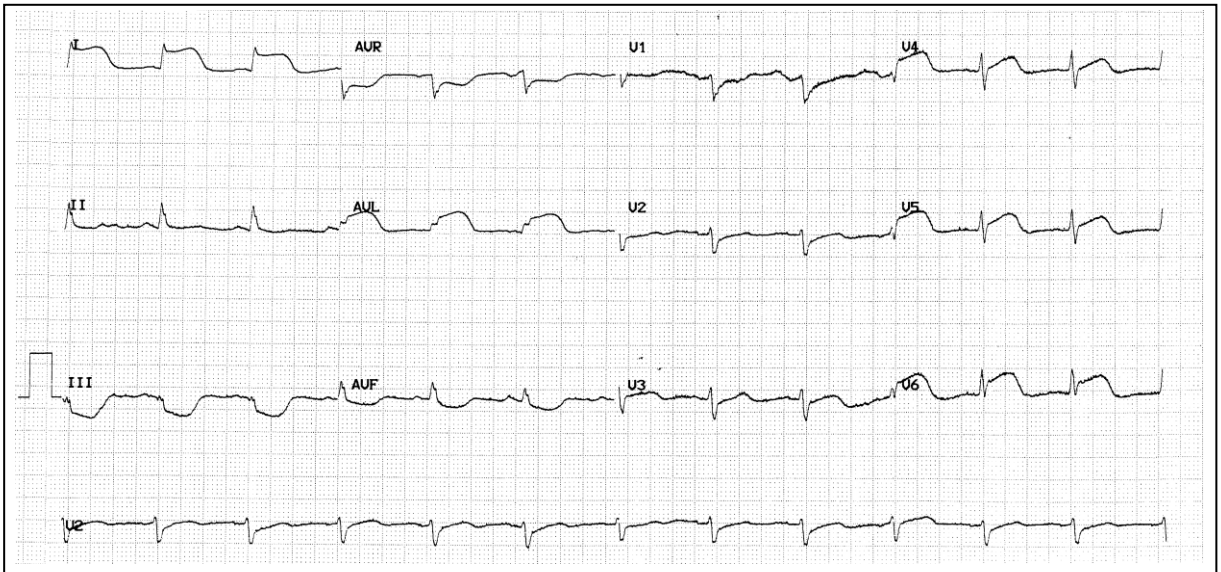


Figure 5/36.

Lateral STEMI (occlusion of the intermediate artery). ST segment elevation in leads I, aVL and V4-6, reciprocal ST segment depression in leads III and aVF. (Sinus rhythm, 75 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction, dome-shaped ST elevation of 4 mm and positive T waves in leads I, aVL and V5-6.)

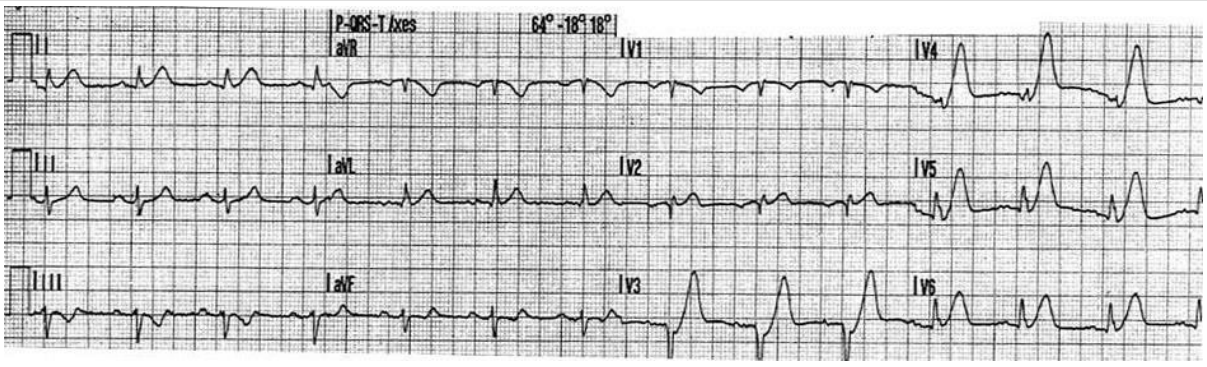


Figure 5/37.

Hyperacute anteroseptal-lateral myocardial infarction. There are hyperacute T waves in leads V2-6, even the ST segment elevation is not visible either. The underlying cause of these findings was occlusion of the middle third of the LAD (between the origin of the first septal and first diagonal branch). (Sinus rhythm, 75 bpm, left axis deviation, normal AV conduction time, QS complexes in leads V1-3, reduced R wave height in the rest of the precordial leads, trivial hyperacute T waves in leads I and aVL, classic hyperacute T waves and trivial J point elevation in leads V3-6.)

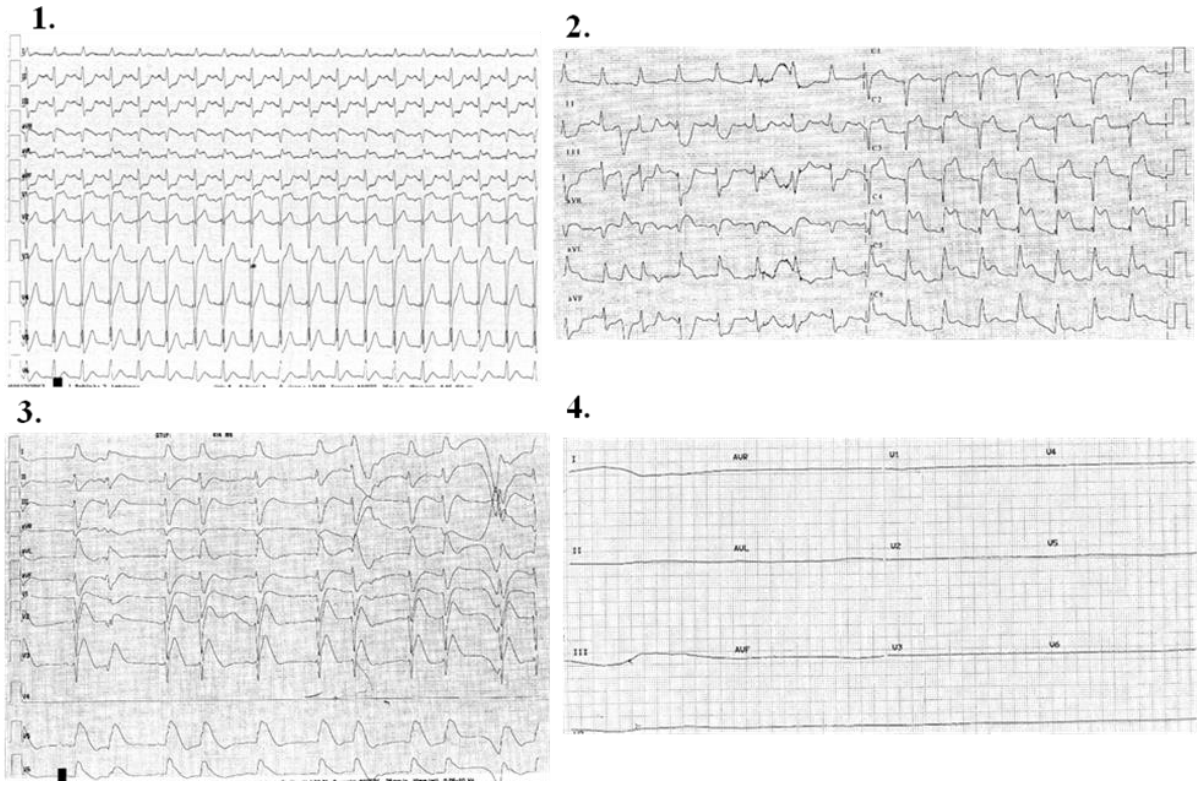


Figure 5/38.

Dynamic ECG changes caused by occlusion of the left main stem. On the **first** ECG of the patient presenting with atypical complaints (without any chest complaints), ST elevation is visible only in lead aVL, however, the presence of typical hyperacute T waves in the precordial leads and reciprocal ST segment depression in the inferior leads should be an alarming sign. The **2nd** ECG was recorded two hours later, already along with chest complaints at this time, where the presence of extensive ST segment elevation in the anterior precordial leads is already obvious. However, the patient had a circulatory collapse minutes after this and the **3rd** ECG was recorded at that time, on which the border of the QRS complex and ST segment cannot be differentiated any longer. Characteristic ECG changes that resemble a tombstone can be recorded in leads V4-6, the designation of which foreshadows the prognosis, too. The patient had already been receiving cardiopulmonary resucitation at that time and was transported for a coronary angiography, where although the occlusion of the left main stem could be opened transiently, yet the patient's circulation could not be stabilized and death occurred (**4th** ECG.)

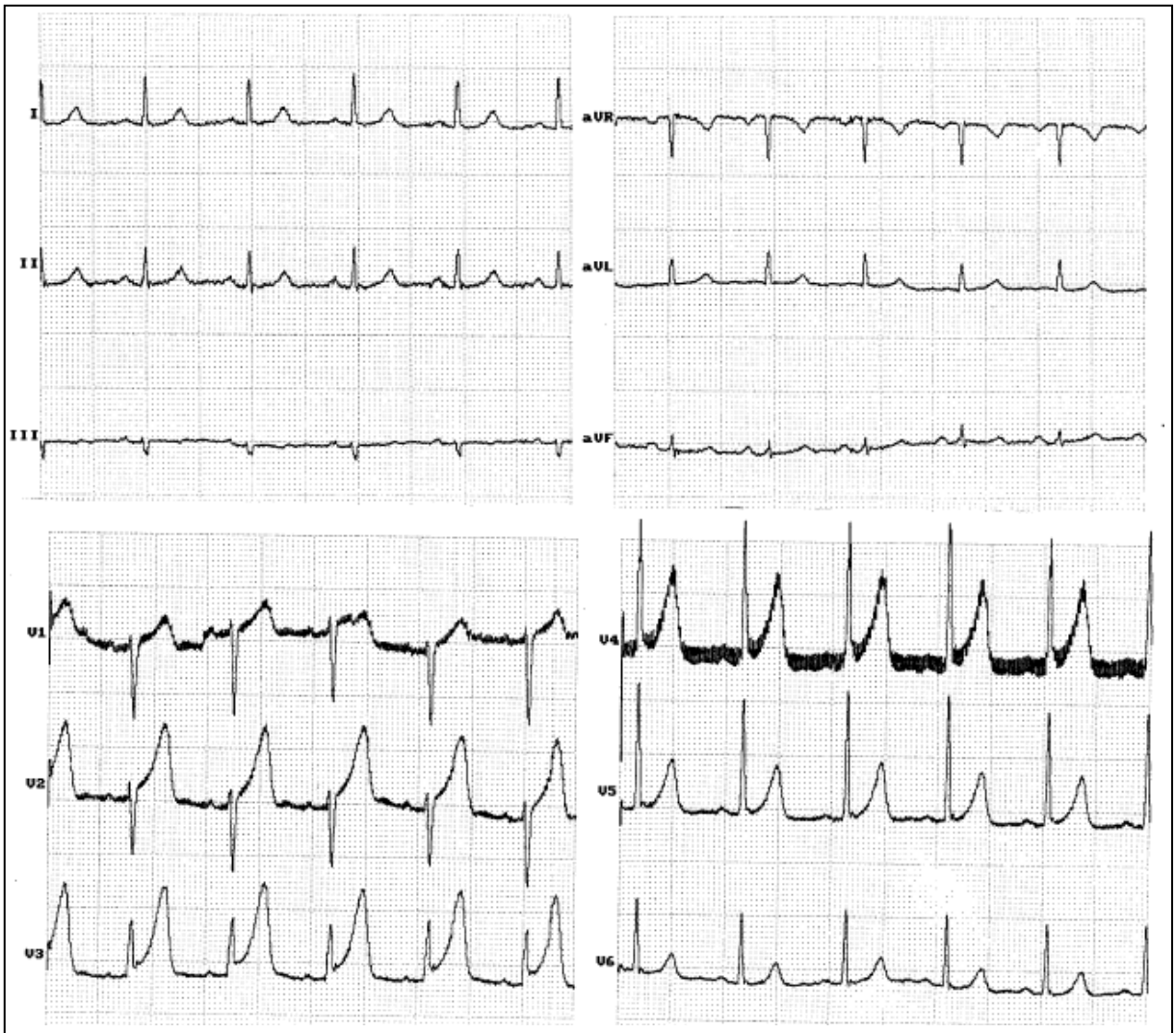


Figure 5/39.

Hyperacute anteroseptal myocardial infarction. Hyperacute T waves and incipient ST segment elevation are visible in leads V2-4. The underlying cause of these findings was occlusion of the middle third of the LAD. (Sinus rhythm, 75 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction, trivial J point elevation and tall, peaked, hyperacute T waves in leads V3-6.)

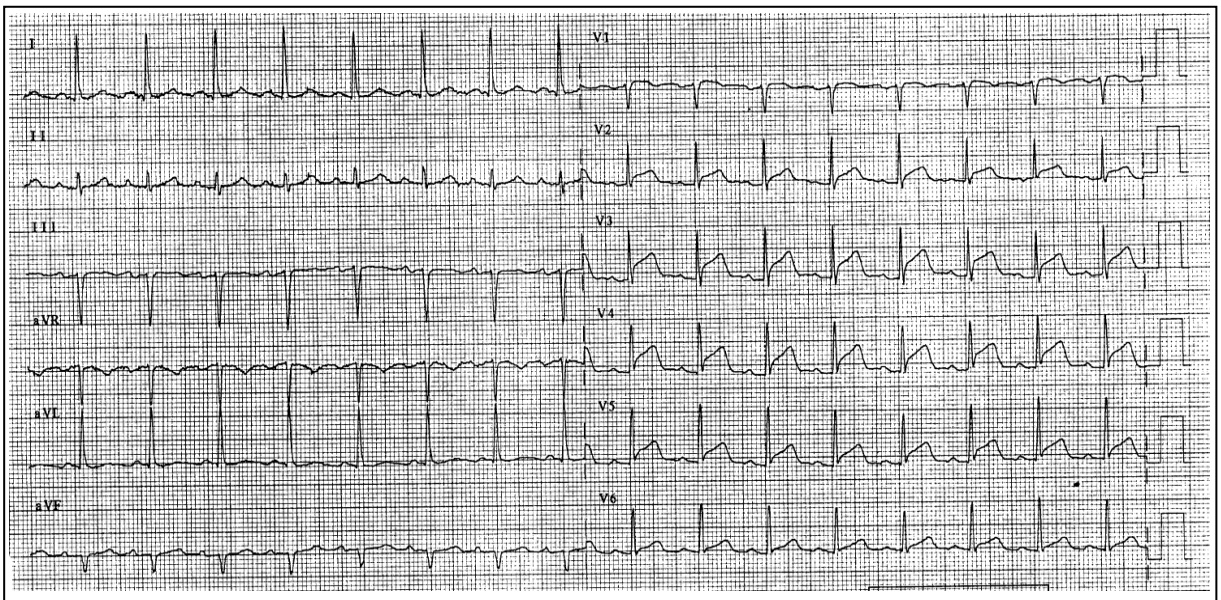


Figure 5/40.

Anteroseptal STEMI. There is dome-shaped ST segment elevation in leads V2-5. The underlying cause of these findings was occlusion of the middle third of the LAD (below the origin of the first septal and first diagonal branch). (Sinus rhythm, 100 bpm, left axis deviation, normal AV conduction time, normal ventricular conduction, dome-shaped ST segment elevation (STE) and positive T waves in leads V2-5.)

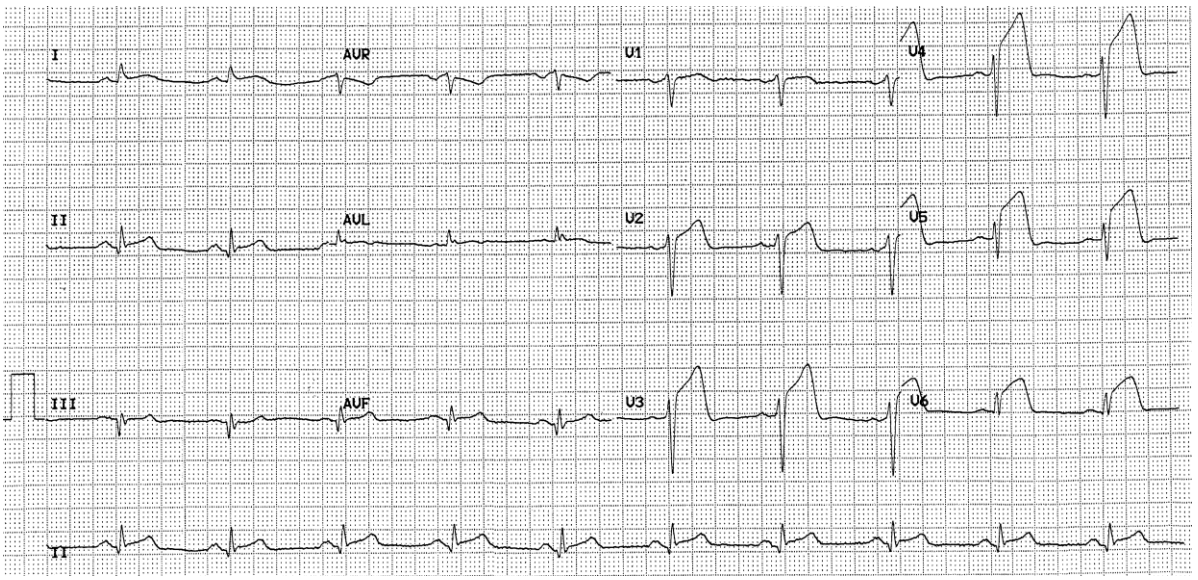


Figure 5/41.

Anteroseptal STEMI. STE is visible in leads I and V2-6, caused by an occlusion of the middle third of the LAD (distal to the origin of the first septal and first diagonal branch). (Sinus rhythm, 65 bpm, left axis deviation, normal AV conduction time, normal ventricular conduction, trivial ST elevation in lead I, dome-shaped ST segment elevation of 4 to 8 mm and positive T waves in leads V2-6.)

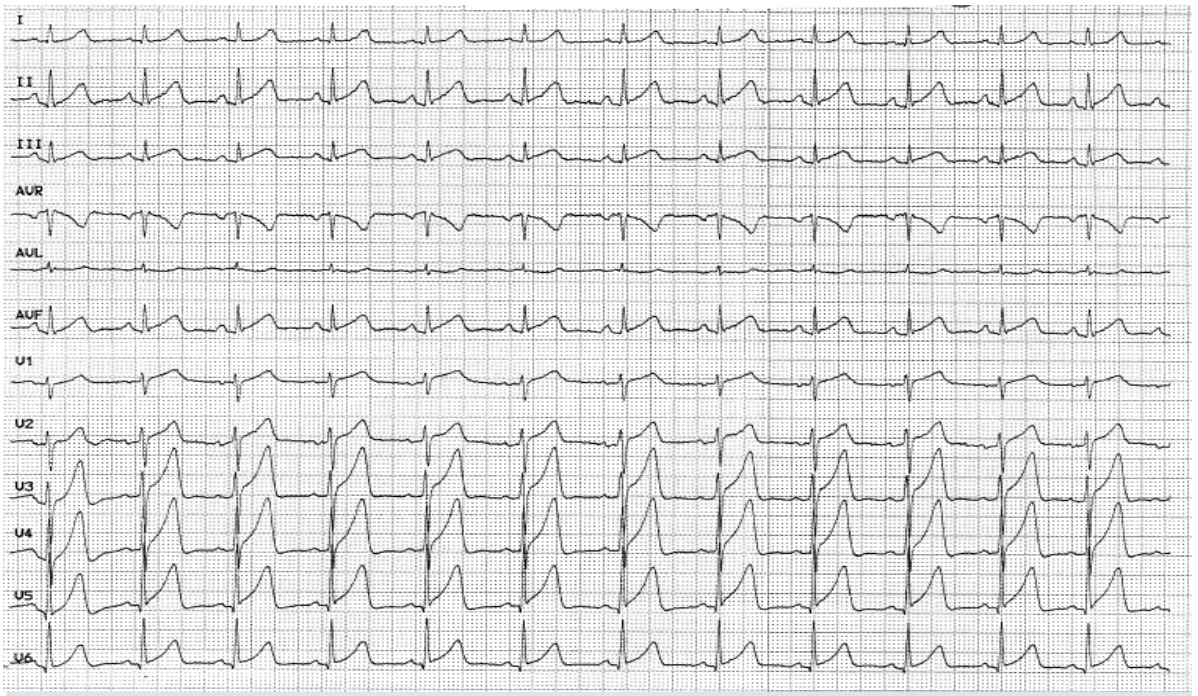


Figure 5/42. Anteroseptal STEMI. There is ST segment elevation in leads V2-5. The underlying cause of these findings was occlusion of the middle third of the LAD (below the origin of the first septal and first diagonal branch). (Sinus rhythm, 70 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction, ST segment elevation of 2 to 3 mm and hyperacute T waves in leads V2-5.)

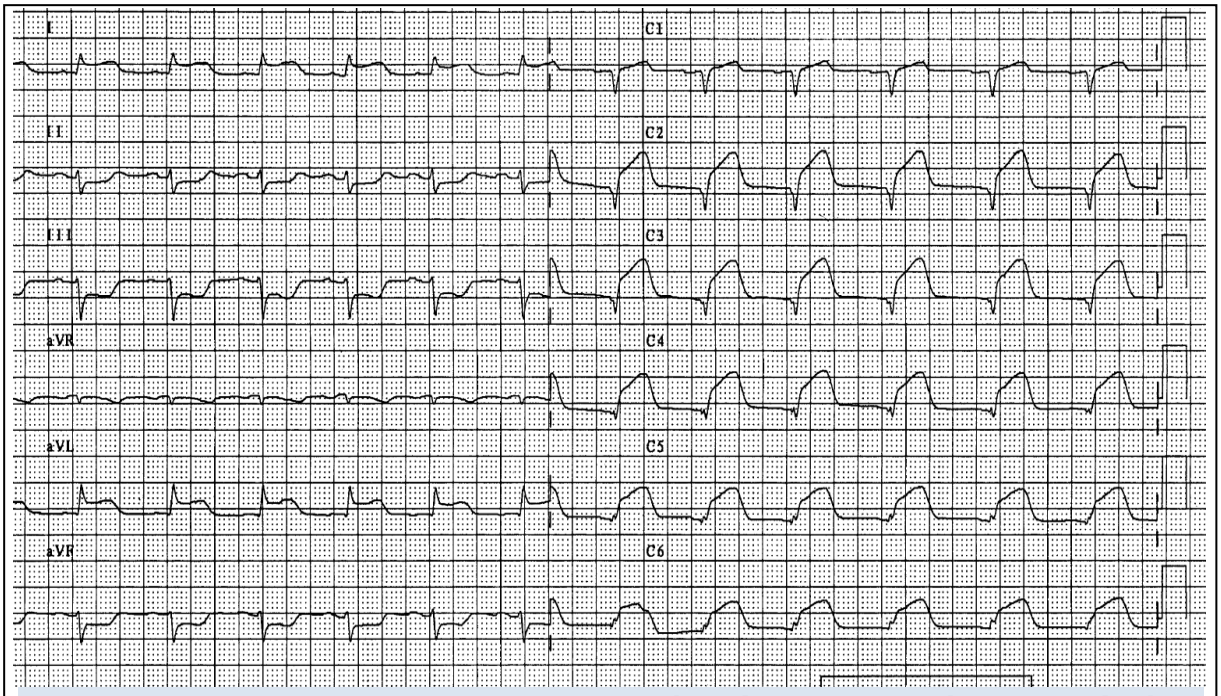


Figure 5/43. Extensive anterior STEMI. Dome-shaped ST segment elevation in leads I, aVL and V1-6. Reciprocal ST segment depression in leads II, III and aVF. The underlying cause of these findings was occlusion of the proximal third of the LAD (distal to the origin of the first septal branch and proximal to that of the first diagonal branch). (Sinus rhythm, 80 bpm, left axis deviation, normal AV conduction time, low QRS voltage and QS complexes in the precordial leads, dome-shaped ST segment elevation of 2 to 5 mm in leads I, aVL and V1-6, reciprocal ST segment depression in the inferior leads.)

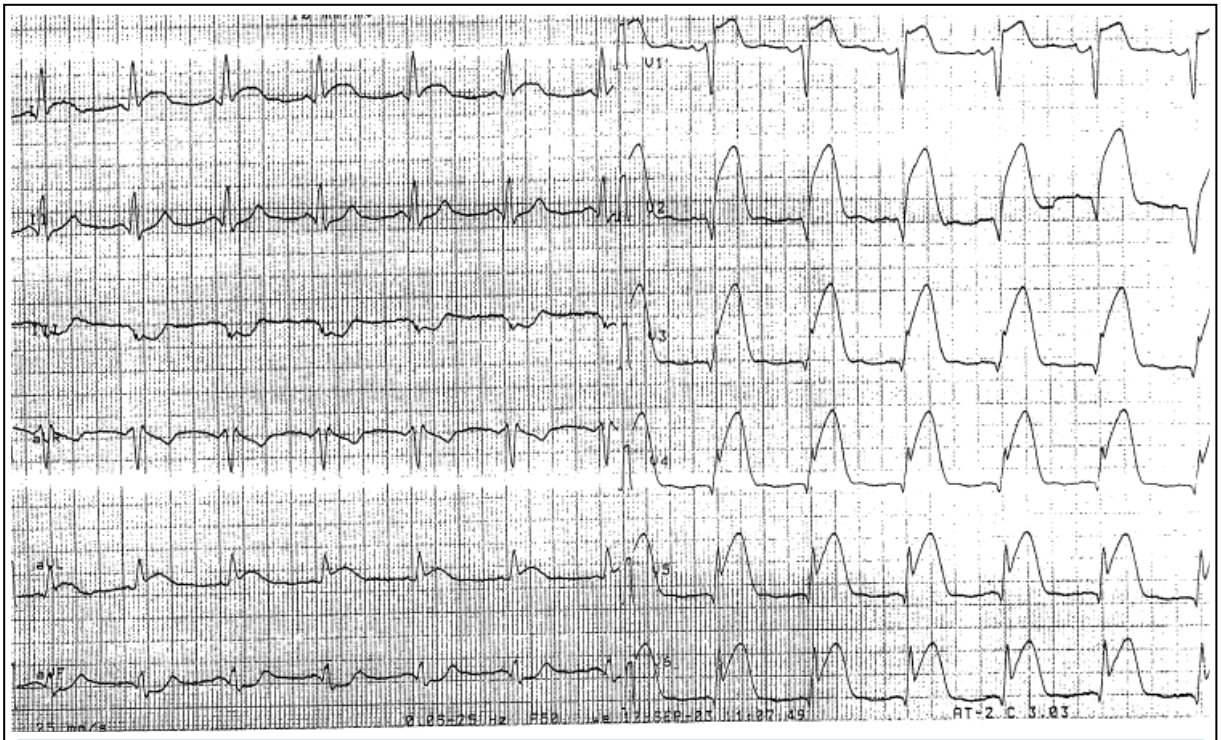


Figure 5/44. Extensive anterior STEMI. Dome-shaped (’tombstoning’) ST segment elevation in leads I, aVL and V1-6. Reciprocal ST segment depression in leads III and aVF. The underlying cause of these findings was occlusion of the proximal third of the LAD. (Sinus rhythm, 75 bpm, normal QRS axis, normal AV conduction time, Q waves in the precordial leads, narrow QRS complexes, ST segment elevation of 1 mm in leads I, aVL, dome-shaped ST segment elevation of 4 to 6 mm in leads V1-6, reciprocal ST segment depression in the inferior leads.)

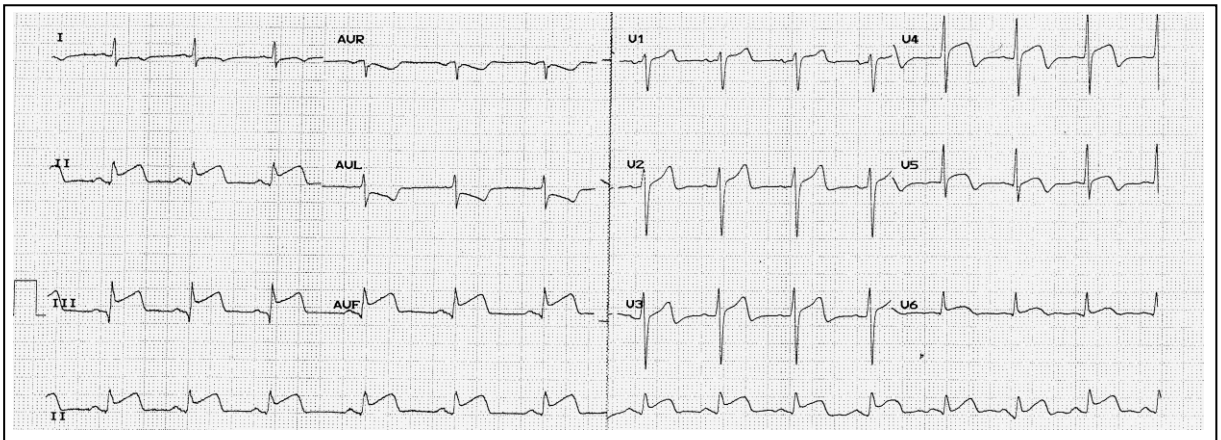


Figure 5/45. Anterior STEMI due to distal LAD occlusion. ST elevation in leads II, III and aVF may be misleading, however, concurrent ST segment elevation in leads V3-6 rather suggests involvement of the anterior wall. This pattern is often falsely interpreted as a double vessel myocardial infarction caused by the LAD coursing beyond the left ventricular apex and also supplying the inferior wall, however, this explanation is correct only in some cases. ST segment elevation presenting in the inferior leads can be explained by the direction of the vector of the ST segment triggered by the distal LAD occlusion, and does not necessarily represent concurrent inferior myocardial infarction. (Sinus rhythm, 78 bpm, normal AV conduction time, normal ventricular conduction, dome-shaped ST segment elevation of 3 to 4 mm in leads II, III, aVF and V3-6.)

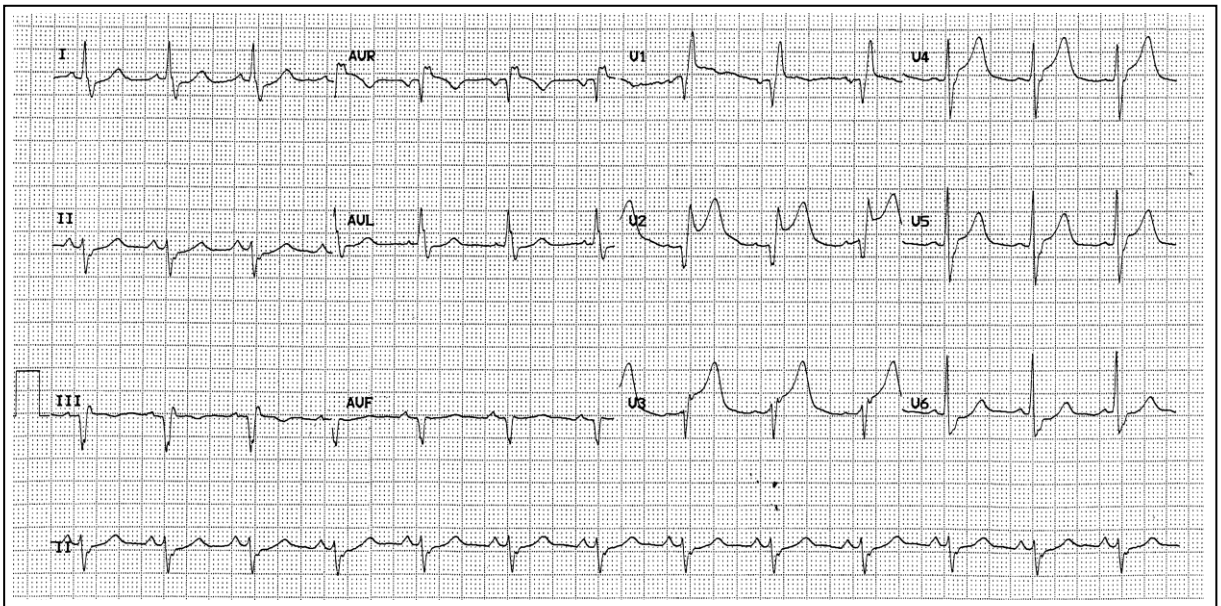


Figure 5/46.

Anterior STEMI with right bundle branch block and ECG pattern of scarring from a previous inferior myocardial infarction. ST segment elevation is observable in leads V1-4. Since the right bundle branch and left anterior fascicle is supplied by the first septal branch of the LAD, and ST elevation is also detectable in lead aVR, the level of occlusion is therefore at the very proximal portion of the LAD. (Sinus rhythm, 80 bpm, left axis deviation, normal AV conduction time, QS complexes in leads III, aVF and V1-3, right bundle branch block, ST segment elevation of 2 to 4 mm and tall positive T waves in leads V1-4.)

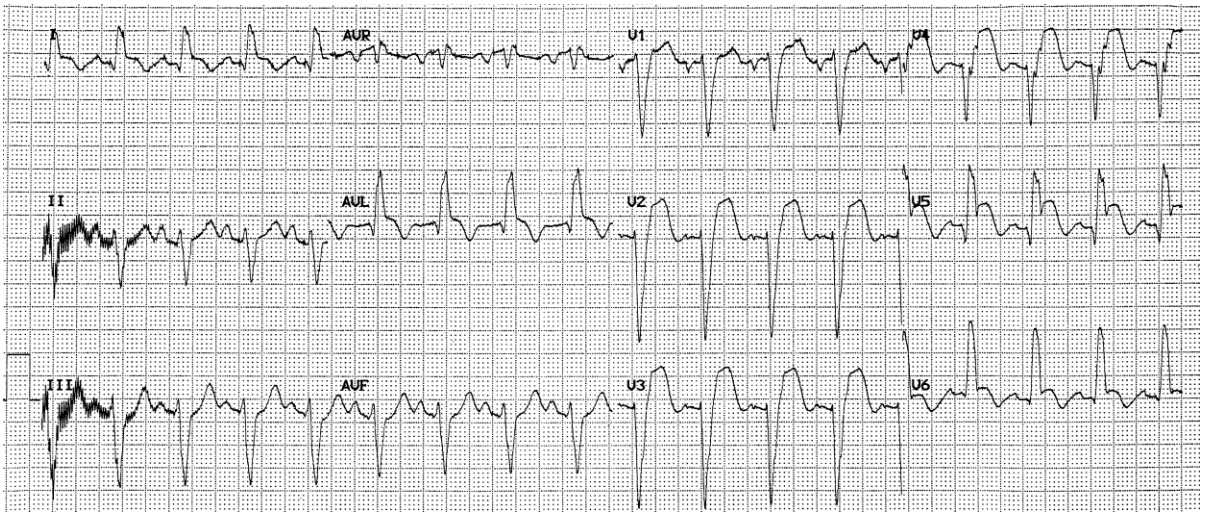


Figure 5/47. Anterior myocardial infarction along with left bundle branch block (LBBB). The ST segment elevation in the precordial leads exceeds 5 mm. Instead of discordant ST segment depression, concordant ST elevation is visible in leads I, aVL and V5-6, moreover, small q waves are also present in the same leads, which can never be seen in isolated LBBB. (Sinus tachycardia, 105 bpm, left axis deviation, normal AV conduction time, left bundle branch block, concordant ST segment elevation of 1 mm in leads I, aVL and V6, ST segment elevation of 4 to 7 mm in leads V1-5.)

Acute myocardial infarction presenting along with LBBB can be recognized based on the *Sgarbossa criteria*:

1. For positive QRS complexes (in leads I, aVL and V5-6, or more rarely, in II, III, aVF), there is ST segment elevation of ≥1 mm;

2. ST segment depression of ≥1 mm in leads V1-3;

3. For negative QRS complexes (in leads V1-3), there is discordant ST segment elevation of ≥5 mm.

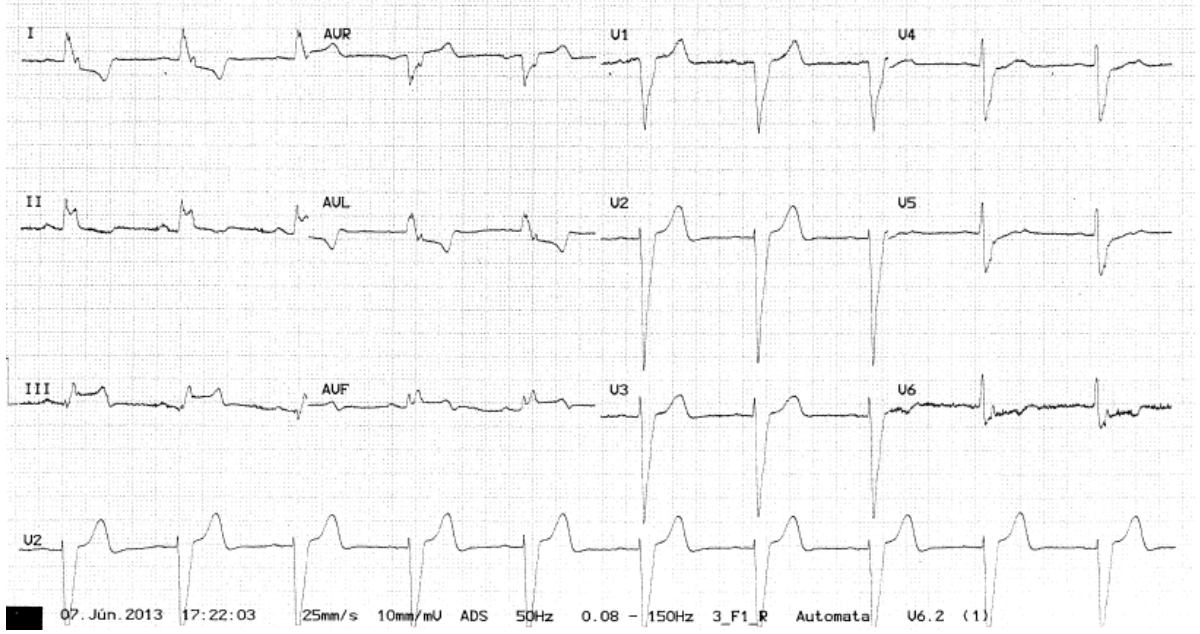


Figure 5/48. Inferior STEMI along with left bundle branch block. ST segment elevation is clearly visible in leads III and aVF, which cannot be the consequence of bundle branch block, since the polarity of QRS complexes is positive and the ST elevation is concordant with them. (Sinus rhythm, normal QRS axis, 60 bpm, normal AV conduction time, left bundle branch block, secondary repolarization abnormalities, concordant ST segment elevation of 1 to 2 mm in leads III and aVF.)

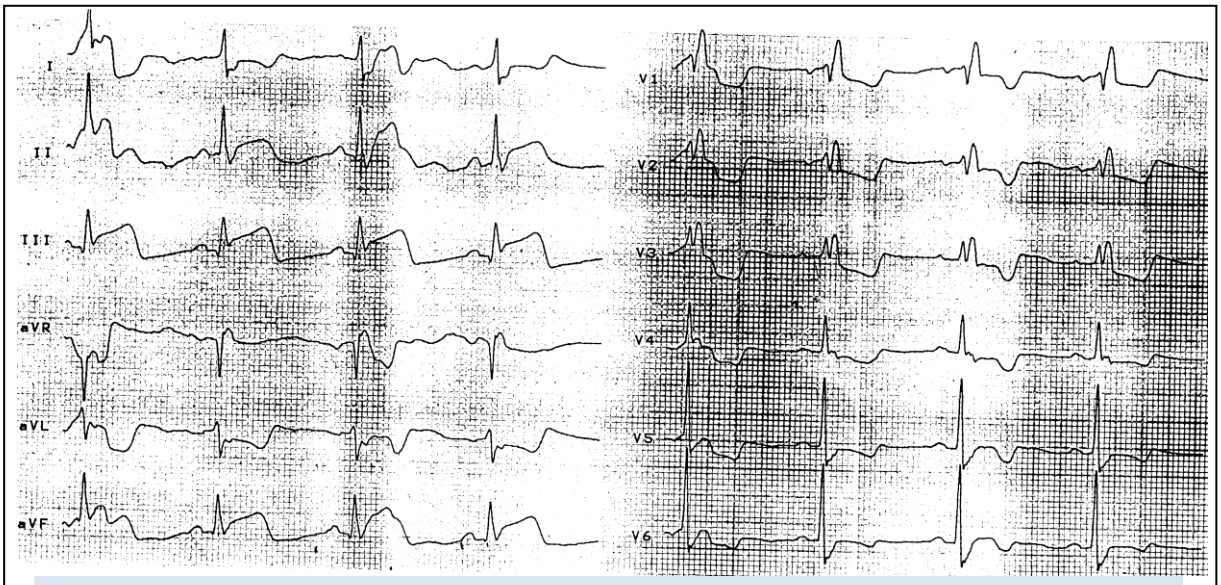


Figure 5/49. Inferior myocardial infarction presenting along with right bundle branch block (RBBB) The ST elevation in leads II, III and aVF is unequivocally recognizable even along with the presence of RBBB. RBBB results in less distortion of the ECG because left ventricular activation occurs in the normal sequence, so it is easier to recognize ST segment elevation along with RBBB than in LBBB. (Sinus rhythm, 55 bpm, normal QRS axis, normal AV conduction time, right bundle branch block, dome-shaped ST segment elevation of 3 to 4 mm in leads II, III and aVF, otherwise secondary repolarization abnormalities.)

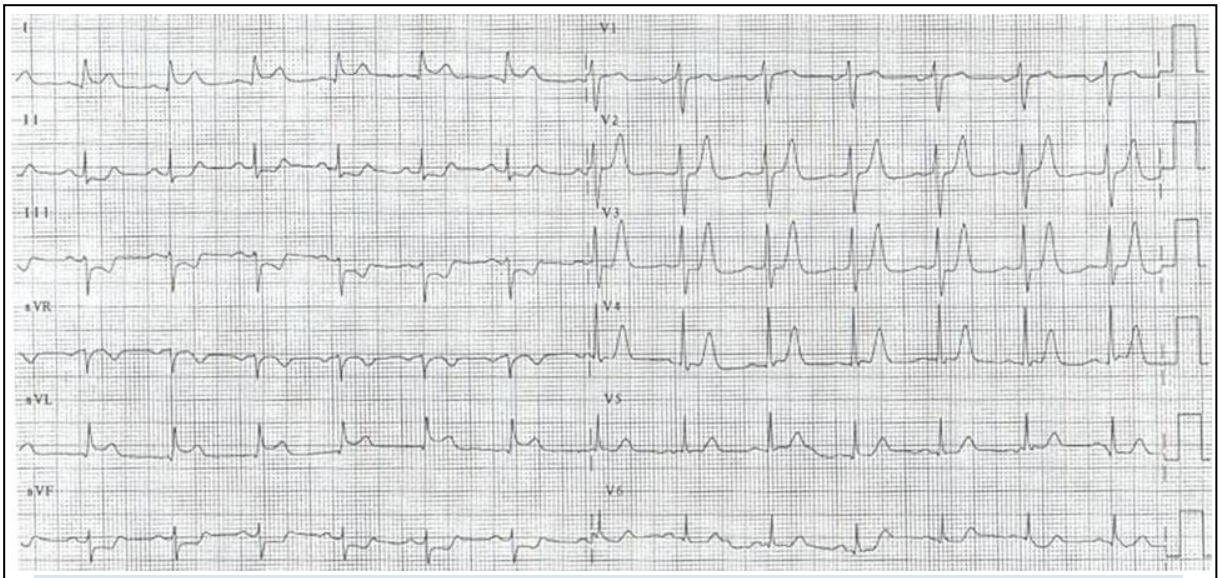


Figure 5/50. High lateral STEMI caused by occlusion of the first diagonal branch. ST elevation in leads I and aVL, reciprocal ST segment depression in leads II, III and aVF. (Sinus rhythm, normal QRS axis, normal AV conduction time, normal ventricular conduction, ST segment elevation of 2 mm in leads I and aVL, reciprocal ST segment depression in leads II, III and aVF.)

In the subacute phase of myocardial infarction, ST elevation decreases, the polarity of T waves may reverse (T wave inversion) and pathological Q waves occur.

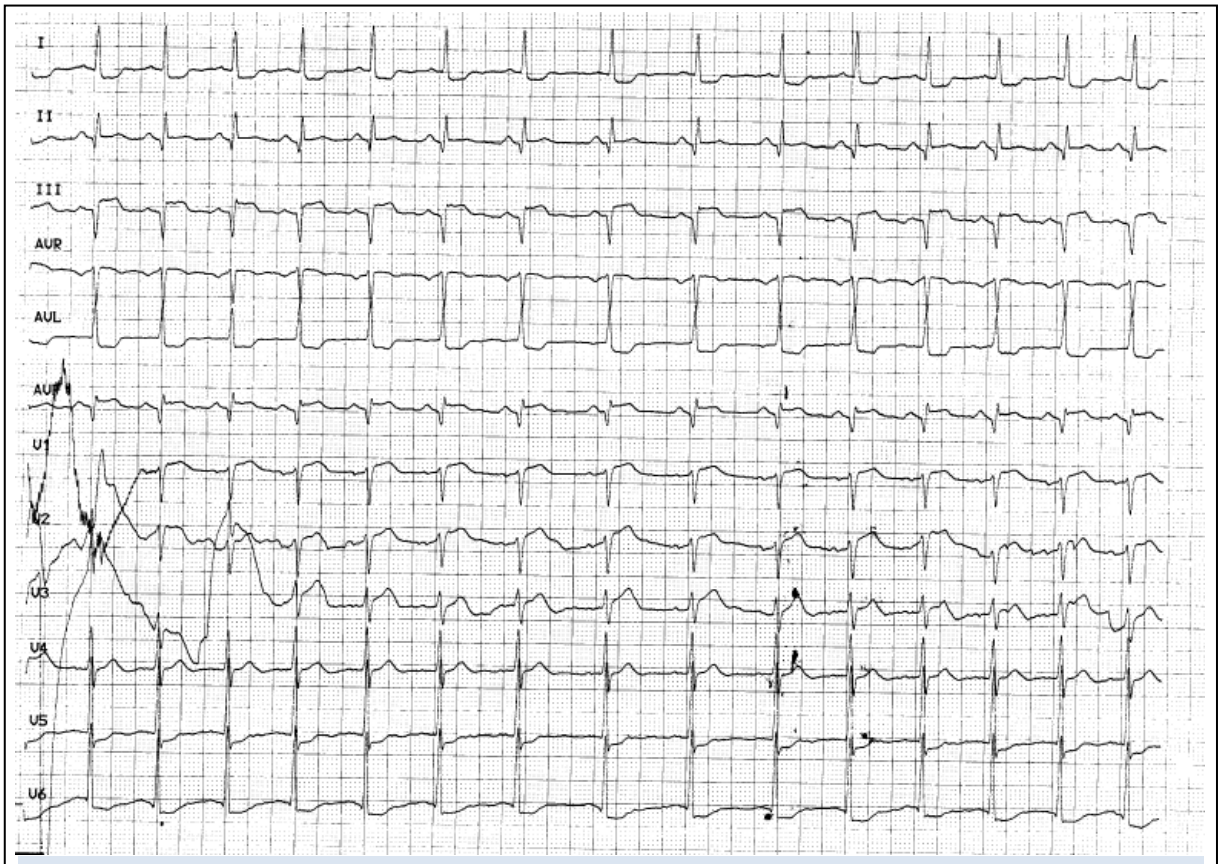


Figure 5/51. Subacute inferior STEMI. There are pathological Q waves in leads II, III and aVF, while ST elevation is still present in leads III and aVF. Reciprocal ST segment depression is visible in leads I, aVL and V5-6. (Sinus rhythm, 100 bpm, normal QRS axis, normal AV conduction time, Q waves in leads II, III and aVF, ST segment elevation of 1 to 2 mm and biphasic T waves in leads III, aVF, reciprocal ST segment depression in leads I, aVL and V5-6.)

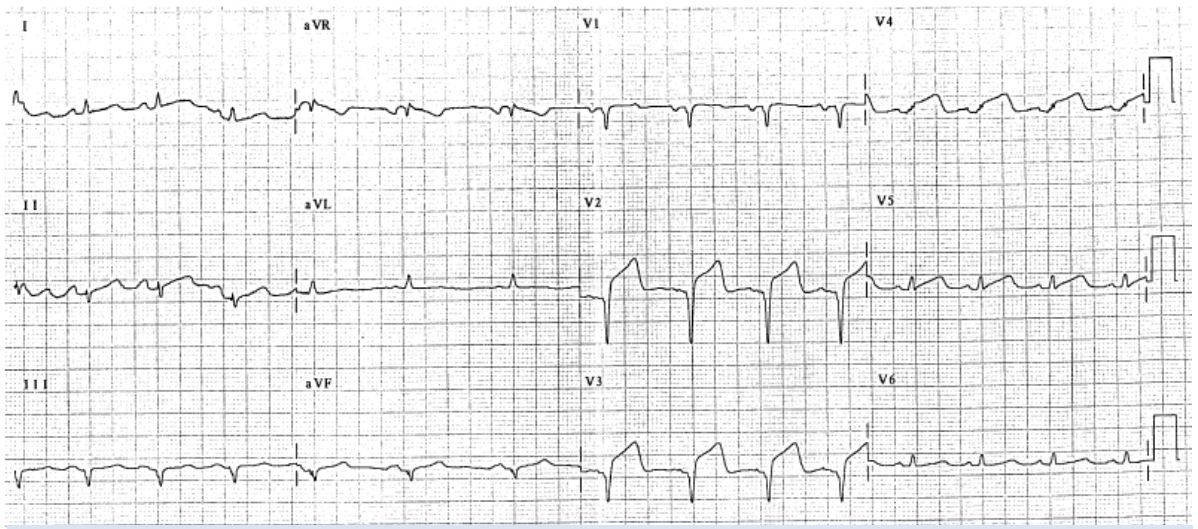


Figure 5/52. Subacute anteroseptal STEMI. QS complexes are already observable in leads V1-4, however, ST segment elevation is also present yet. The QS complexes in leads III and aVF represent an ECG pattern of scarring from an old inferior myocardial infarction. (Sinus rhythm, 100 bpm, left axis deviation, P mitrale, low voltage in the limb leads, QS complexes in leads III, aVF and V1-3, ST segment elevation of 3 to 5 mm and positive T waves in leads V2-4.)

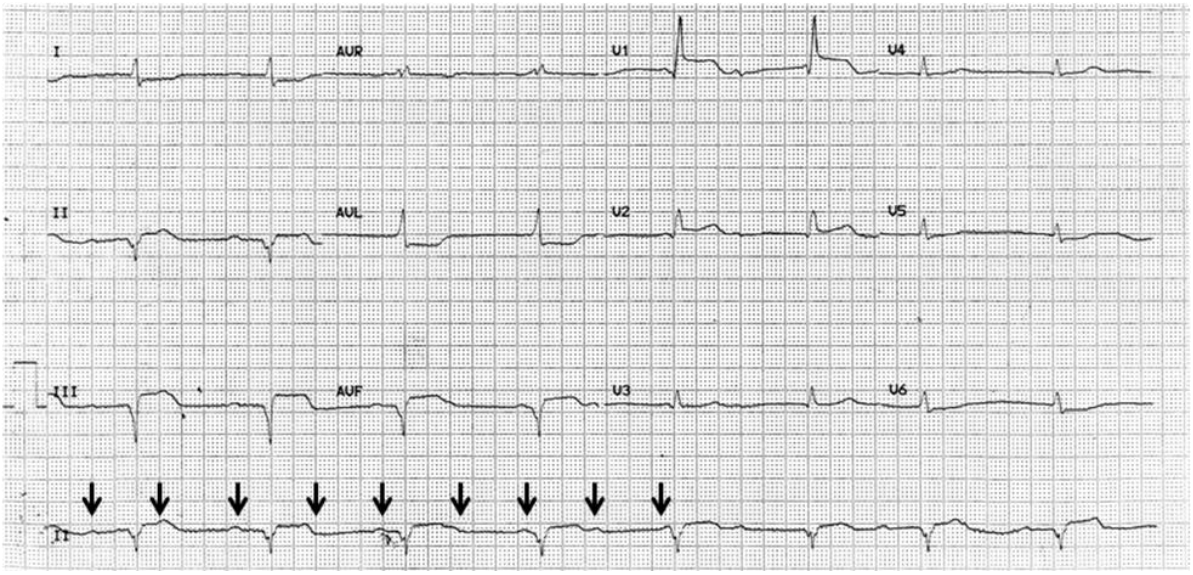


Figure 5/53.

Inferior STEMI with right ventricular involvement, third degree AV block and junctional escape rhythm. Typical ST segment elevation, biphasic T waves and QS complexes (does not imply a very recent occlusion) in leads II, III and aVF. ST elevation in leads V1-2 indicates right ventricular infarction (there is an increase in the degree of ST elevation from V1 to V3 for LAD occlusion, while it decreases in right ventricular infarction). Complete AV dissociation is clearly observable on the lower rhythm strip (third degree AV block and junctional escape rhythm, P waves are indicated by arrows). Moreover, tall R waves in leads V1-2 are a reciprocal change of posterior Q waves. The underlying cause of these findings was proximal RCA occlusion. (Sinus rhythm, third degree AV block, junctional escape rhythm at a ventricular rate of 50 bpm, left axis deviation, QS complexes in leads II, III and aVF, tall R waves in lead V1 (reciprocal change of posterior Q waves), ST segment elevation of 1 to 3 mm and biphasic T waves in leads II, III, aVF and V1-2.)

It is generally characteristic of the **ECG pattern of an infarct scar** that ST segments become isoelectric, T wave inversion may develop and pathological Q waves are present. It may occur that ST elevation persists (especially if it appears in the anteroseptal leads) and, in this case, one should think of the presence of left ventricular dysfunction / aneurysm formation.

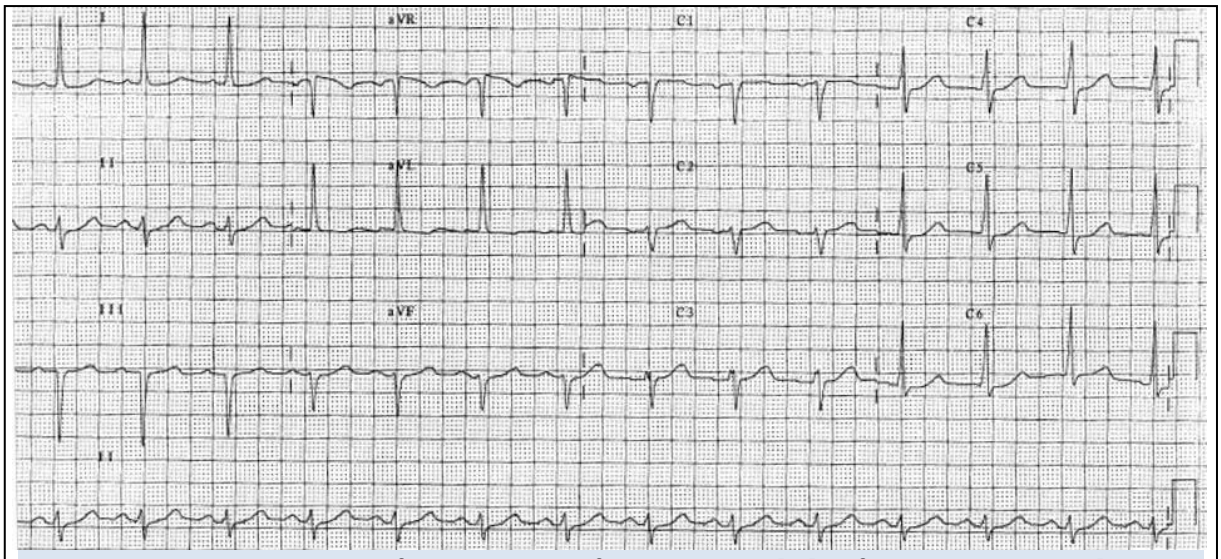


Figure 5/54. ECG pattern of scarring in inferior myocardial infarction. QS complexes in leads III and aVF. Isoelectric ST segments and positive T waves. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, normal AV conduction time, left axis deviation, QS complexes in leads III and aVF, narrow QRS complexes, normal ventricular repolarization.)

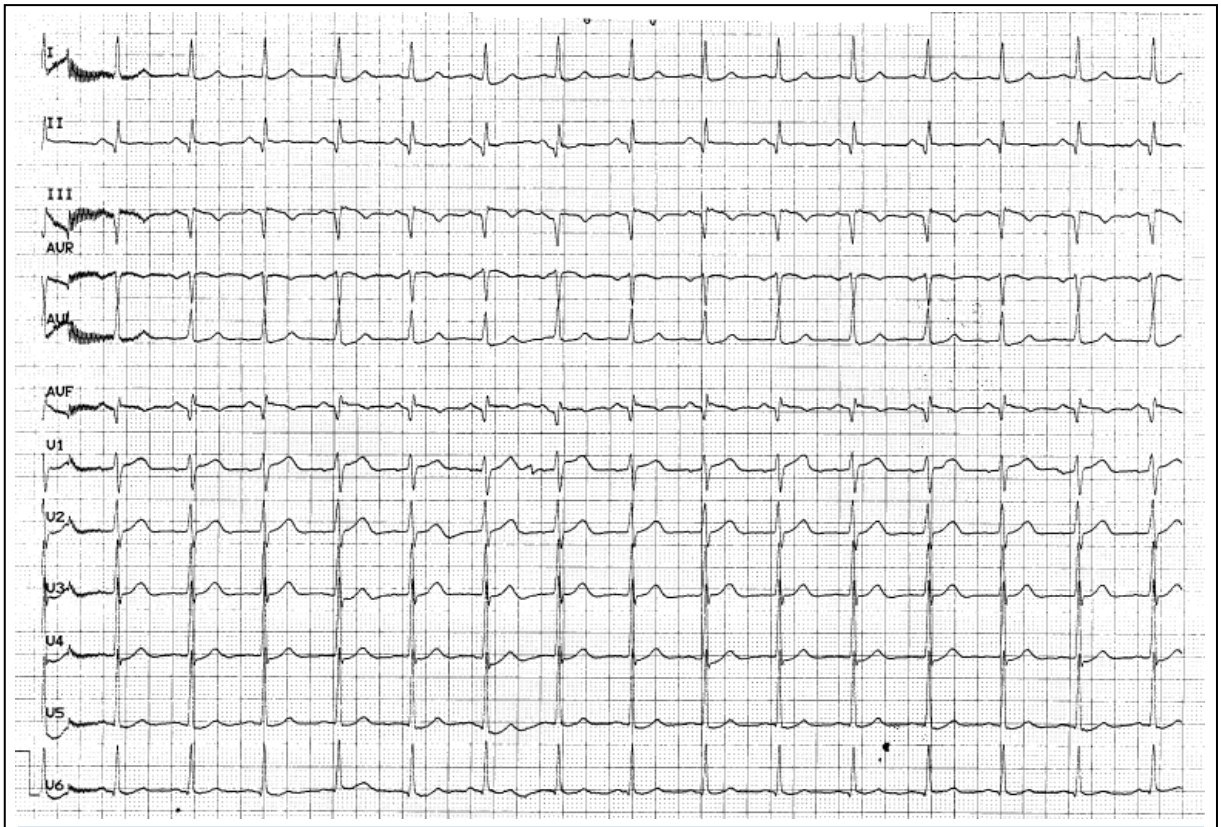


Figure 5/55. ECG pattern of scarring in inferior myocardial infarction. Pathological Q waves in leads II, III and aVF. Negative T waves in leads III and aVF and left axis deviation. Tall R waves in lead V1 are consistent with reciprocal Q waves indicating posterior involvement. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 100 bpm, normal QRS axis, normal AV conduction time, Q waves in leads II, III and aVF, early transition (tall R waves in lead V1 - reciprocal change of Q waves), negative T waves in leads III and aVF, isoelectric ST segments.)

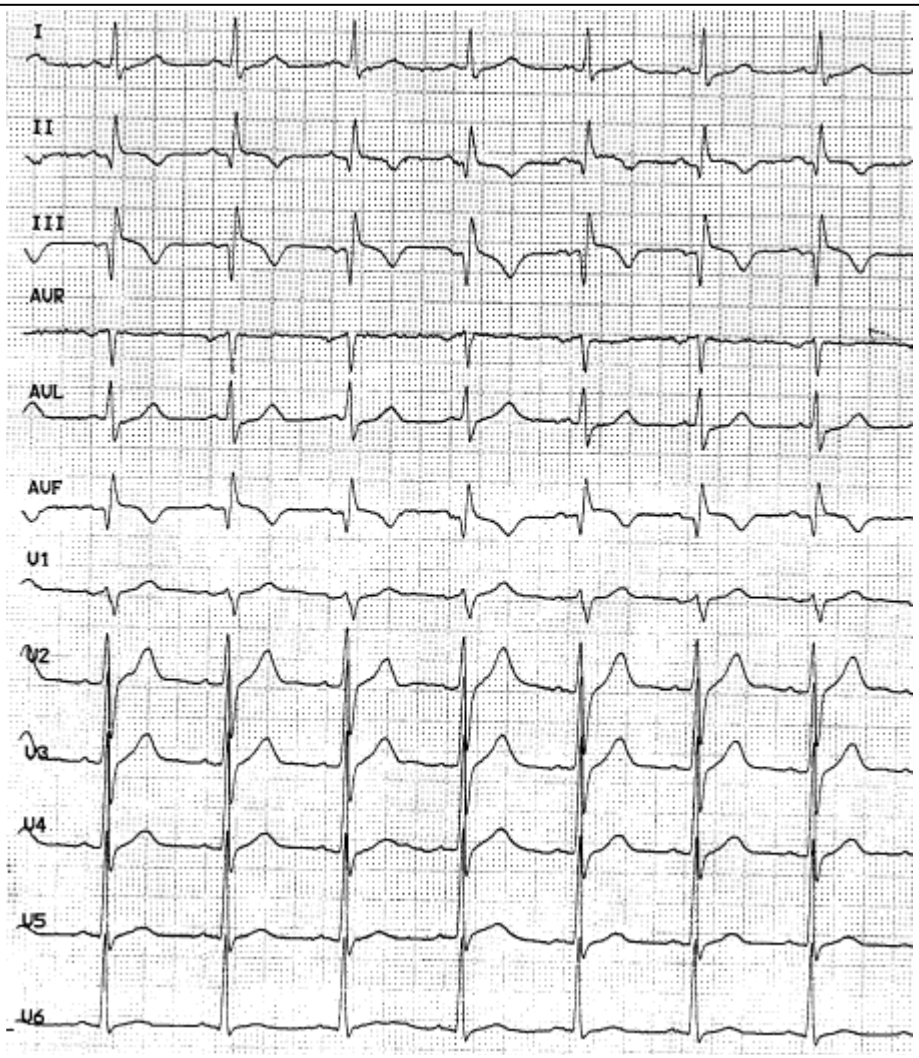


Figure 5/56.

ECG pattern of scarring in inferior myocardial infarction. There are pathological Q waves and negative T waves in leads II, III and aVF, with the ST segments being isoelectric. The underlying cause of these findings was occlusion of the right coronary artery. (Sinus rhythm, 75 bpm, normal QRS axis, normal AV conduction time, Q waves and negative T waves in leads II, III and aVF, isoelectric ST segments.)

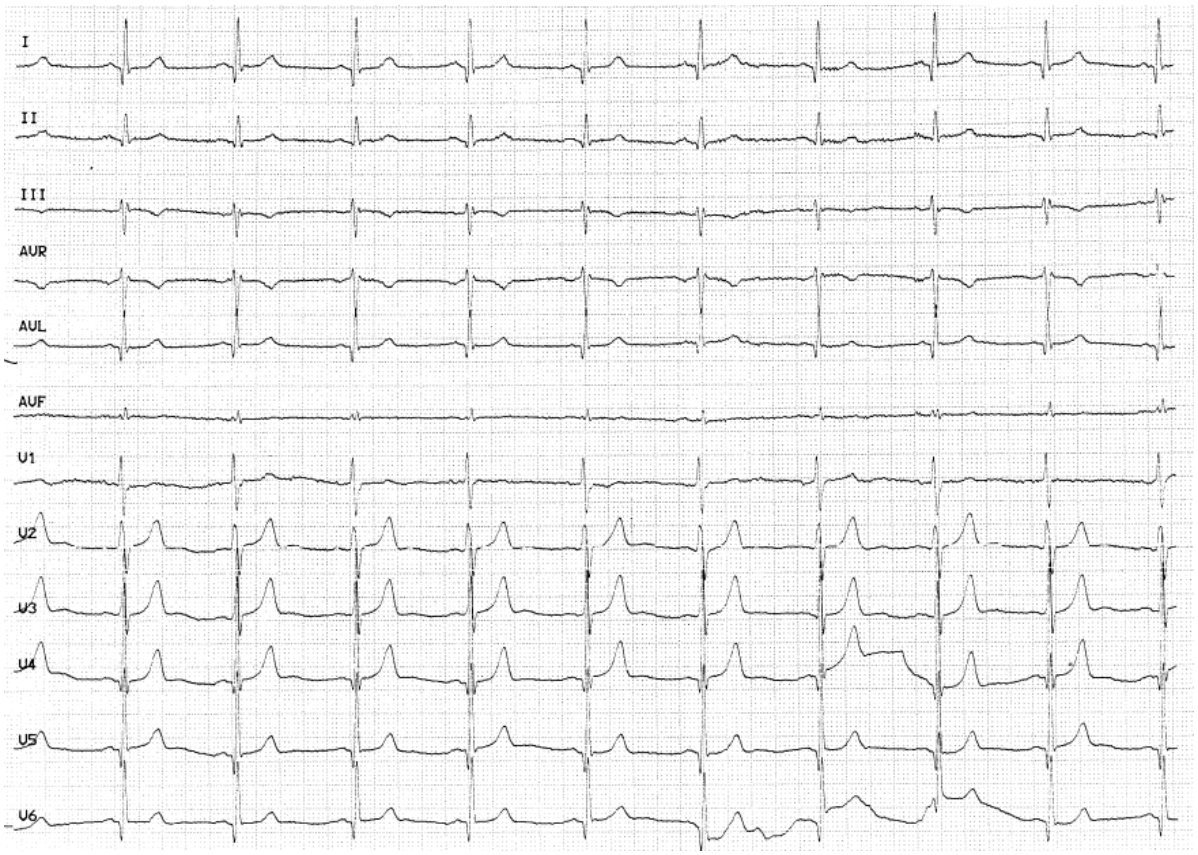


Figure 5/57.

ECG pattern of scarring in postero-lateral myocardial infarction (Cx). Pathological Q waves in leads I, aVL, V5-6, tall R waves in lead V1 (reciprocal change of Q waves). (Sinus rhythm, normal QRS axis, normal AV conduction time, Q waves in leads I, aVL and V5-6, R/S=1 in lead V1 - reciprocal change of Q waves, normal ventricular repolarization.)

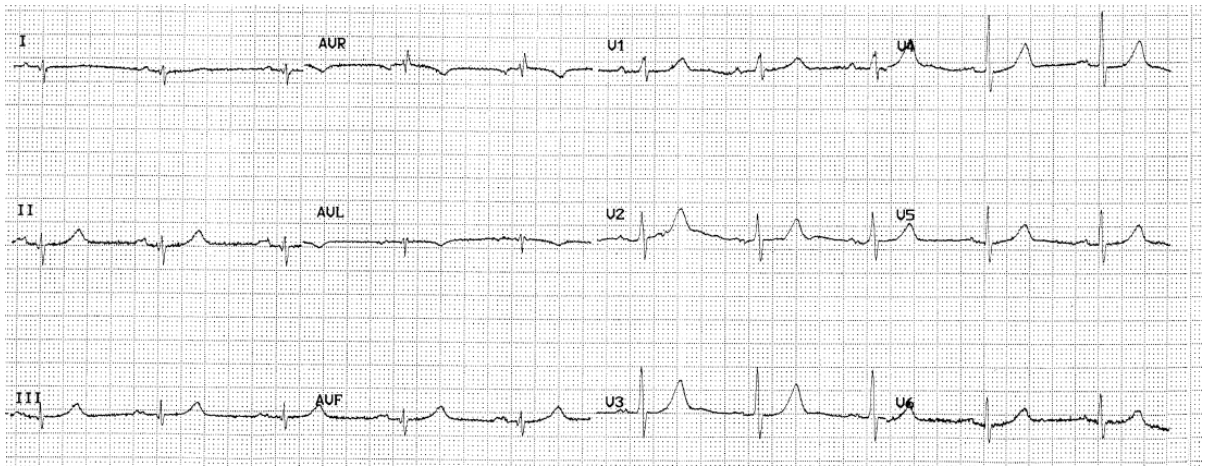


Figure 5/58.

ECG pattern of scarring in posterior myocardial infarction (Cx). Tall R waves in lead V1 are a reciprocal change of posterior Q waves. (Sinus rhythm, 55 bpm, indeterminate (superior) QRS axis, normal AV conduction time, tall R waves in lead V1 (reciprocal change of Q waves), narrow QRS complexes, normal ventricular repolarization.)

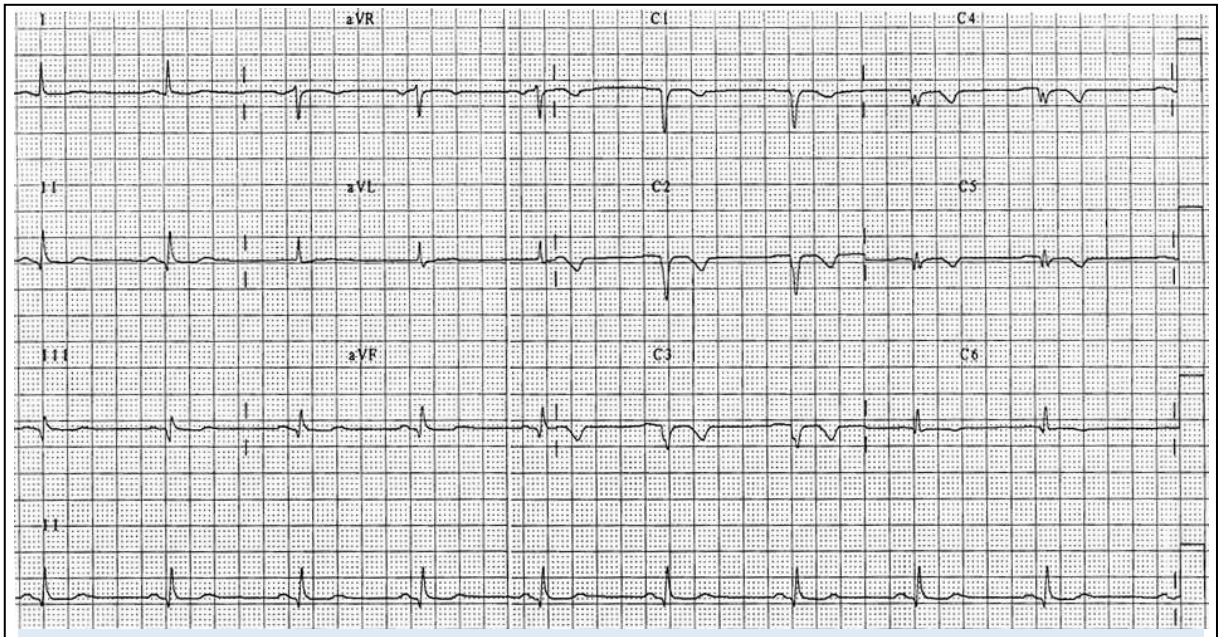


Figure 5/59. ECG pattern of scarring after anteroseptal and inferior myocardial infarction. There are QS complexes in leads V1-4, pathological Q waves both in leads V5 and II, III, aVF, isoelectric ST segments, T wave inversion in the leads representing the anterior wall. The underlying cause of these findings was occlusion of the middle third of the LAD reaching beyond the cardiac apex (and partially supplying the inferior wall as well). (Sinus rhythm, 57 bpm, normal QRS axis, normal AV conduction time, low voltage all over the leads, pathological Q waves in leads II, III, aVF and V4-6, QS complexes in leads V1-3, negative T waves in the precordial leads.)

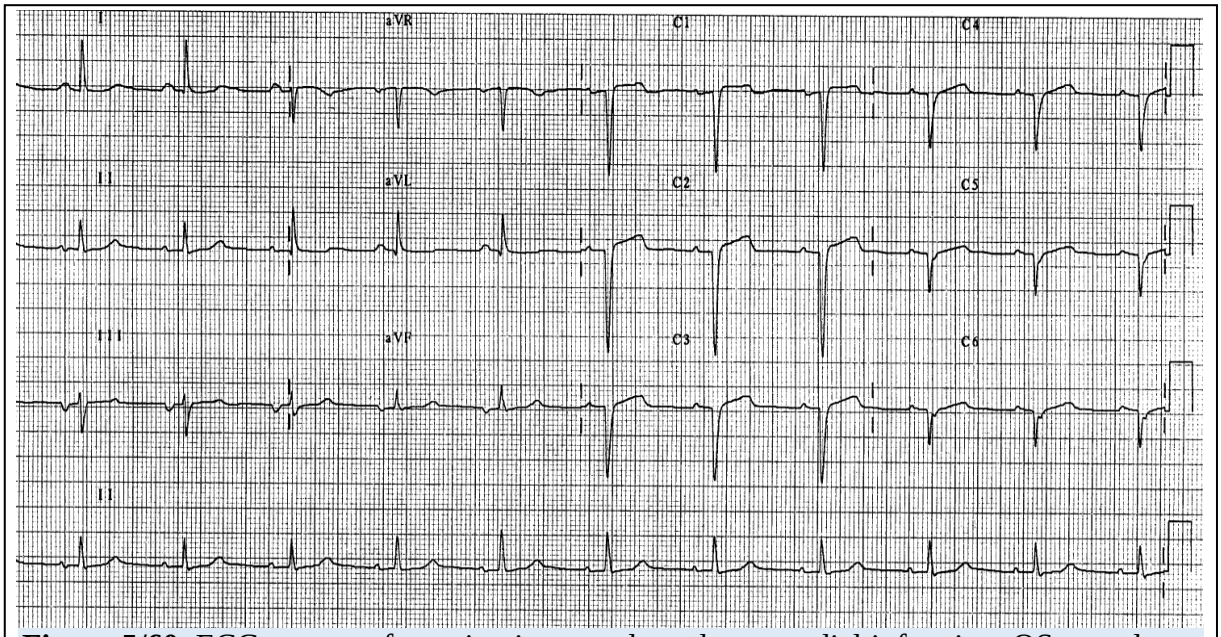


Figure 5/60. ECG pattern of scarring in anterolateral myocardial infarction. QS complexes and trivial, but not significant, ST elevation is present in leads V1-6. The underlying cause of these findings was occlusion at the border of the proximal and middle third of the LAD. It is also observable that P waves are of non-sinus origin. Negative P waves in leads III and aVF are characteristic of impulse formation from the lower portion of the right atrium. (Ectopic atrial rhythm at a ventricular rate of 70 bpm, normal QRS axis, normal AV conduction time, QS complexes and persistent ST elevation of 1 to 2 mm in leads V1-6.)

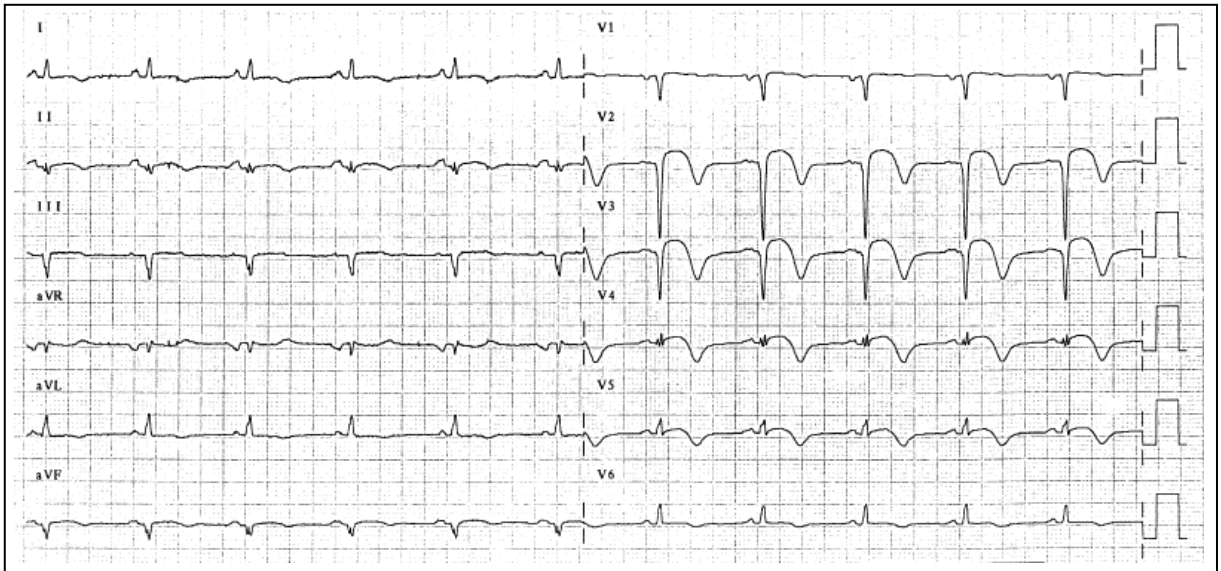


Figure 5/61. ECG pattern of scarring in double vessel (i.e. anteroseptal and inferior) myocardial infarction as well as signs of apical aneurysm formation. There are QS complexes in leads V1-4 and III, aVF, persistent ST elevation and negative T waves in leads V2-4. The underlying cause of these findings was prior occlusion of the right coronary artery and the middle third of the LAD. (Sinus rhythm, P mitrale, 70 bpm, left axis deviation, normal AV conduction time, low voltage in the limb leads, Q waves in leads II, III and aVF, QS complexes in leads V1-3, persistent ST elevation of 2 to 3 mm in leads V2-4, negative T waves in leads I, aVL and the precordial leads.)

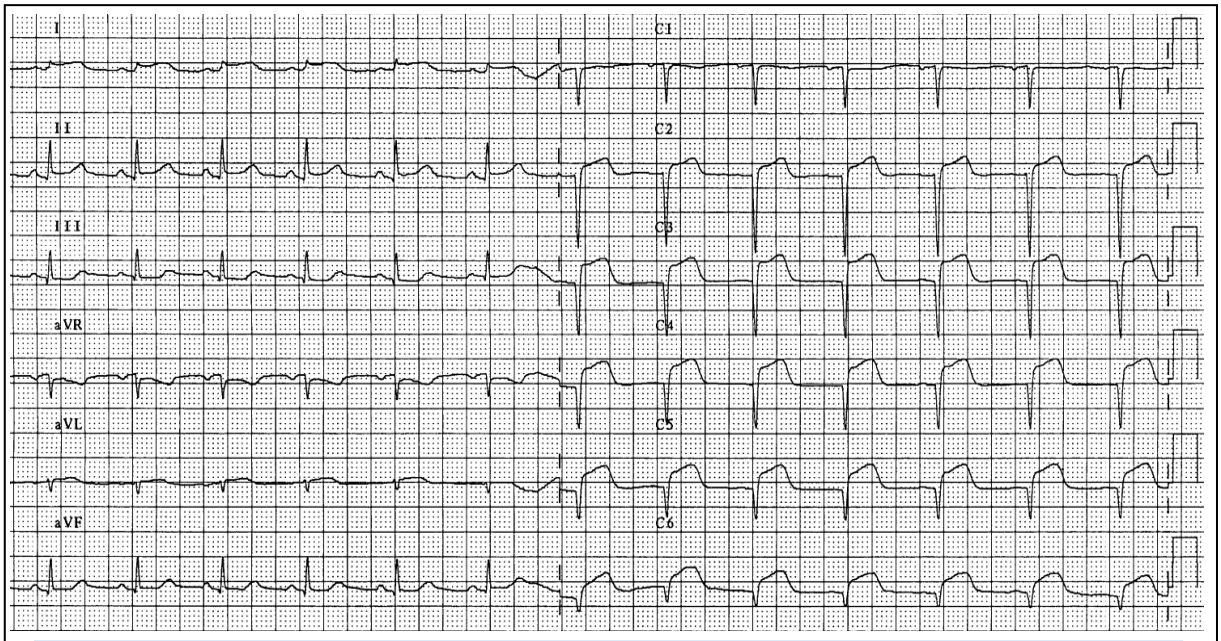


Figure 5/62.

ECG pattern of scarring in extensive anterior myocardial infarction. QS complexes in leads aVL, V1-6, persistent ST elevation in leads I, aVL, V1-6. The underlying cause of these findings was proximal occlusion of the LAD. (Sinus rhythm, normal QRS axis, 80 bpm, normal AV conduction time, QS complexes in leads V1-6, trivial ST elevation in leads I and aVL, persistent ST elevation of 3 to 4 mm in leads V2-6.)

5.12. Non–ST-elevation myocardial infarction (NSTEMI)

About half of myocardial infarction cases are not associated with ST segment elevation, they are rather accompanied by ST segment depression or a nearly normal ECG. In these cases, characteristic complaints and elevation of cardiac troponin levels as well as that of myocardial necrosis markers may help establish the diagnosis. The two characteristic manifestations of NSTEMI are infarction caused by total blood vessel occlusion or distal embolisation from a non-occlusive thrombus.

In the occlusive form, there is complete cessation of antegrade coronary blood flow, but blood getting through the collaterals reduces the degree of necrosis and prevents from the development of ST elevation. Another cause may be that a true posterior myocardial infarction (Cx occlusion) is erroneously described on the 12-lead surface ECG as NSTEMI, however, ST elevation would become apparent if dorsal leads (V7-9) were also placed. If a patient has typical chest pain and ST segment depression reaching or exceeding 2 mm as well as positive T waves are observable in leads V2-4, one should think of the presence of occlusion of the circumflex artery (reciprocal changes of posterior myocardial infarction) and one's activities should be performed in accordance with the protocol for the treatment of ST elevation myocardial infarction.

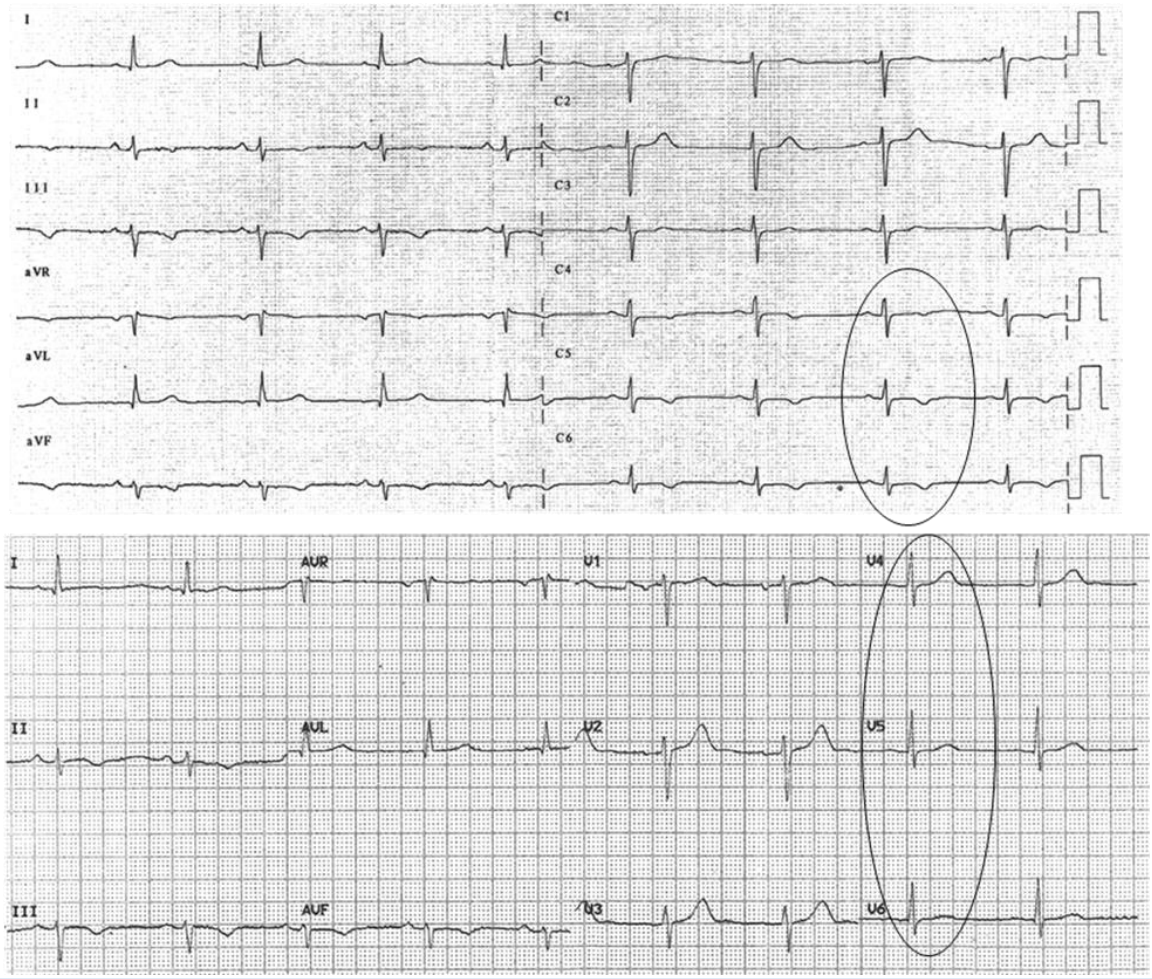


Figure 5/63. Typical NSTEMI caused by total occlusion of the obtuse marginal (OM) branch of the circumflex artery. The ECG pattern at the time of admission is visible on the upper tracing, while that after recanalization of the vessel can be detected on the lower tracing. The only difference is the inversion of negative T waves in leads V4-6.

If the area supplied by the given coronary artery (generally a side branch, e.g. a diagonal or marginal branch) is too small, ST elevation does not necessarily develop even in this case, and this condition is also represented as an NSTEMI.

In the non-occlusive form, platelet activation has a far greater importance, in that white thrombus is formed in these cases and the formation of an appositional red thrombus is only less characteristic, because blood coagulation is activated only to a lesser degree in this form. Fragments breaking off from the platelet plug adhering to the wall of a blood vessel, thereby resulting in distal embolisation, cause elevated troponin and myocardial necrosis marker levels as well as typical complaints, however, no characteristic ST elevation develops due to the relatively small area affected currently.

5.13. Non-Q-wave myocardial infarction

Historically, myocardial infarctions can be divided into two types based on the degree of transmurality. Only the majority of myocardial infarctions (but not all of them) is associated with typical ECG abnormalities. In about 10 % of cases, no Q waves develop even in completed infarctions. These are called non-Q-wave myocardial infarctions. In the initial phase, both ST segment depression and elevation may occur and the infarction may be symptomatic or produce only few symptoms. The essence of this condition is that necrosis does develop in such cases, indicated by elevated levels of myocardial necrosis markers and the presence of regional wall motion abnormalities observed by echocardiography. The pathogenetic basis of this condition lies in the fact that the infarction does not involve full thickness of the myocardium, the necrosis is subendocardial (e.g. only the subendocardium suffers an injury due to rapid recanalization after an occlusion). Non-Q-wave myocardial infarctions are considered an incomplete event, therefore, if such findings are observed (e.g. in the medical history), additional examinations are necessary to assess the level of risk. In a significant number of cases with STEMI, the necrosis is transmural and Q waves also evolve, while in cases with NSTEMI, the extent of necrosis is often subendocardial, or at least non-transmural, so these cases more often result in a non-Q-wave event.

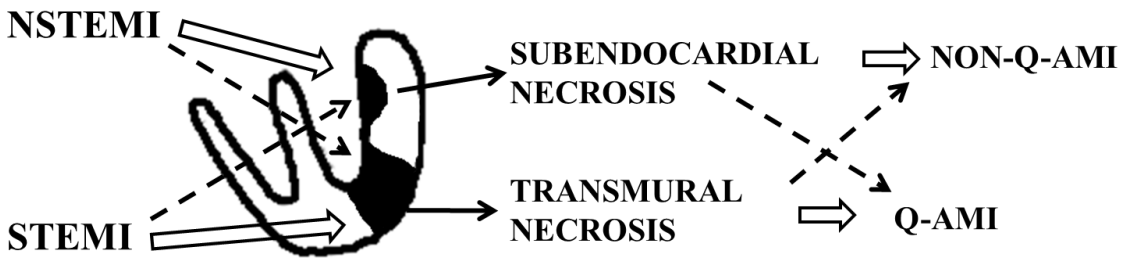


Figure 5/64. Development of Q-wave and non-Q-wave myocardial infarctions.

5.14. Further medical conditions associated with ST segment elevation

Many other heart diseases may cause ST segment elevation. Based on medical history, symptoms, other ECG signs and shape of the ST segment, the underlying pathology can be judged in the majority of cases.

In **Prinzmetal's angina**, the observed complaints and ECG changes (even reciprocal changes) are identical to what is seen in myocardial infarction, however, the abnormalities

completely disappear in 20 to 30 minutes and no necrosis develops. The underlying cause of this condition is vasospasm (dynamic coronary stenosis) or significant proximal stenosis (static stenosis) of a large epicardial coronary artery. Cases associated with transient ST segment elevation and chest pain belong to the same class of emergency where cases with STEMI do, because slowing of the blood flow caused by a subtotal stenosis occurs much more frequently than true coronary vasospasm.

In **pericarditis**, ST segment elevation is generally visible in all of the leads except aVR, in addition, there are no reciprocal changes and ST elevation is concave or 'saddle-shaped' rather than convex or 'dome-shaped'. One does not always encounter this typical ECG pattern in pericarditis; in fact, it may frequently occur that, at the onset of symptoms, ST elevation appears in a localized fashion, e.g. in leads representing the inferior wall.

In **severe left ventricular pressure overload (strain)**, it is not infrequent that convex ST segment elevation of up to 2 to 3 mm presents in leads V1-4.

Persistent ST segment elevation can be observed in the presence of **extensive left ventricular dyskinesia / aneurysm** (this ST elevation may also be dome-shaped and it can be differentiated from myocardial infarction based on the medical history and echocardiographic appearance).

In **bundle branch blocks**, repolarization also becomes abnormal due to an irregular sequence of depolarization, and these changes are called secondary or consequential ST segment abnormalities (discordance of QRS complexes - ST segments).

ST elevation may also be detected as a normal finding, e.g. in **tachycardia** developed during physical exertion, and the so-called **benign early repolarization** is not a pathological condition either. In the latter condition, small r' waves are visible at the initial portion of the ST segment.

The appearance of benign early repolarization in the precordial leads does not indicate any pathology, however, correlation could be detected between r' waves in leads II, III, aVF and sudden cardiac death, therefore, this abnormality must be examined further in such cases.

Elucidating the cause of ST segment elevation may pose a problem in cases where prominent negative P waves appear in several leads due to impulse formation in the lower portion of the atria. This phenomenon can be explained by projection of the repolarization vector of the P wave (which points to the opposite direction compared to that of the P wave) onto the ST segment.

We would like to demonstrate the above described conditions on the following ECG tracings.

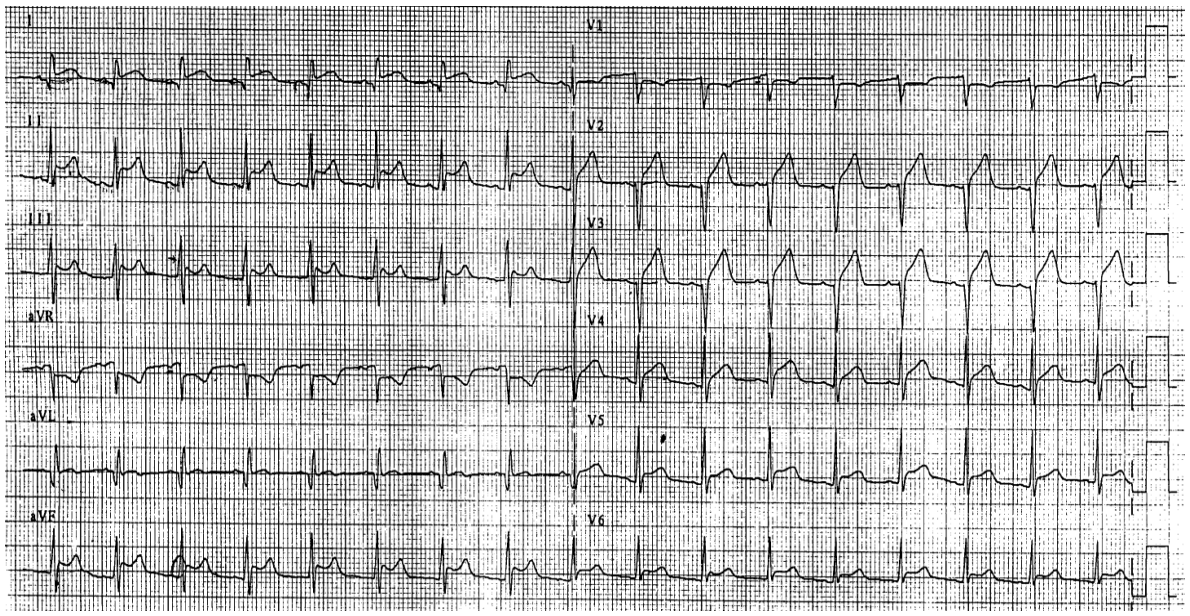


Figure 5/65. Pericarditis. ST segment elevation is visible in all of the leads except lead aVR and V1. Typical ‘saddle-shaped’ ST segment elevation can be observed in leads II, III, aVF, which is considered pathognomonic for pericarditis. (Sinus tachycardia, 110 bpm, normal QRS axis, normal AV conduction time, QS complexes in leads V1-3, ‘saddle-shaped’ ST elevation in leads II, III and aVF, ‘dome-shaped’ ST elevation in leads I, aVL and V2-6, downsloping ST segment depression of 1 mm in leads aVR and V1.)

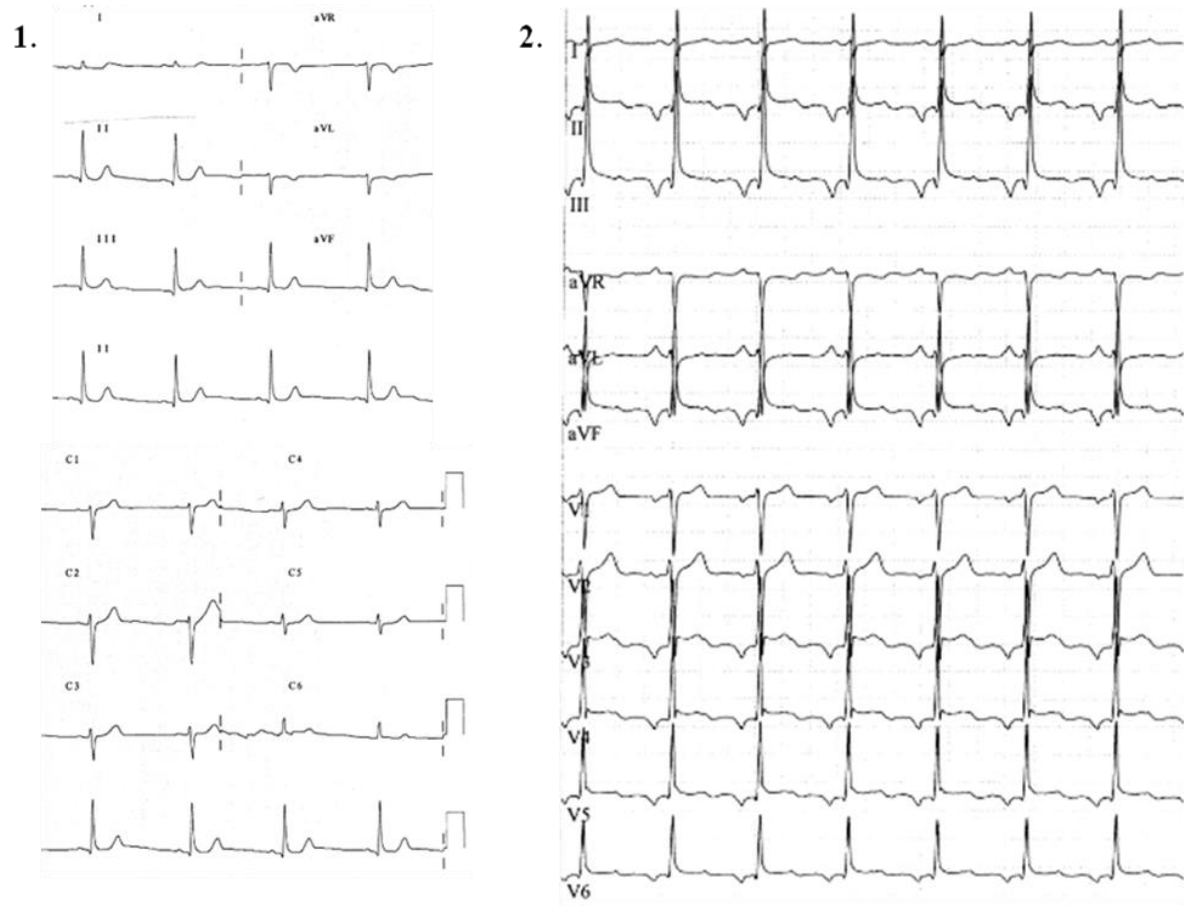


Figure 5/66. ST segment elevation caused by repolarization of P waves (Tp) during impulse formation in the lower portion of the atria. Both ECGs were obtained from the same patient, however, the first one was recorded during sinus rhythm and the second one during ectopic atrial rhythm.

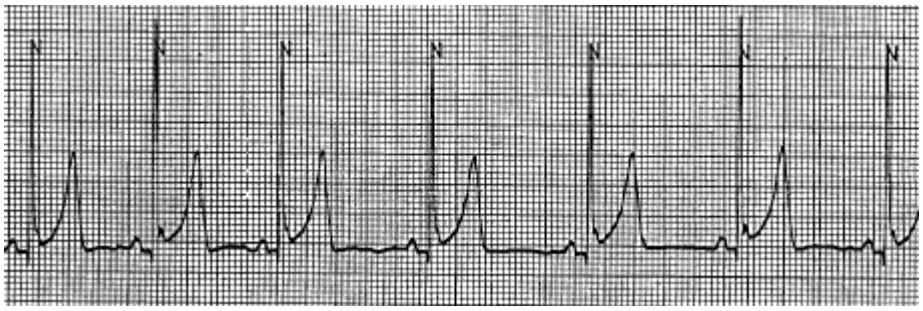


Figure 5/67.

Benign early repolarization. On the ECG tracing, r' waves at the end of QRS complexes can be observed in a magnified form, which are consistent with benign ST elevation.

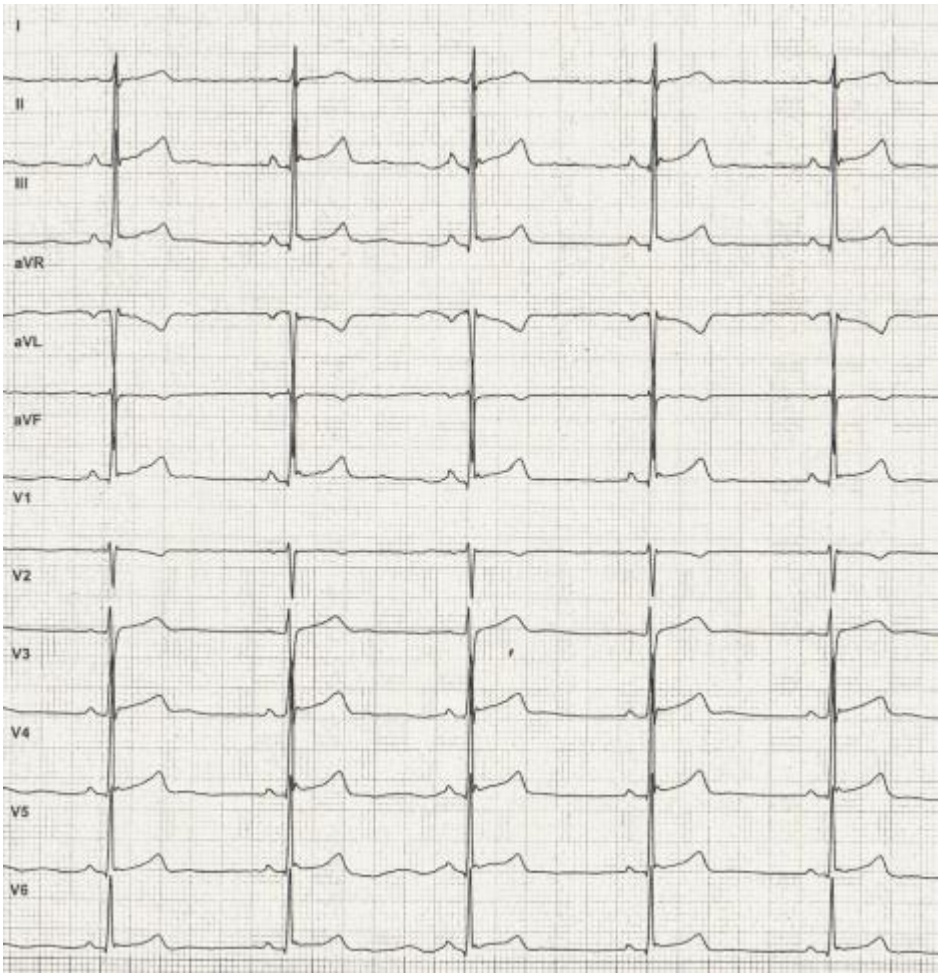


Figure 5/68.

Benign early repolarization. ST segment elevation and small r' waves occurring at the end of QRS complexes are visible in leads II, III, aVF and V3-6. (Sinus bradycardia, 50 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction, ST segment elevation caused by early repolarization in leads II, III, aVF and V3-6, otherwise normal ventricular repolarization.)

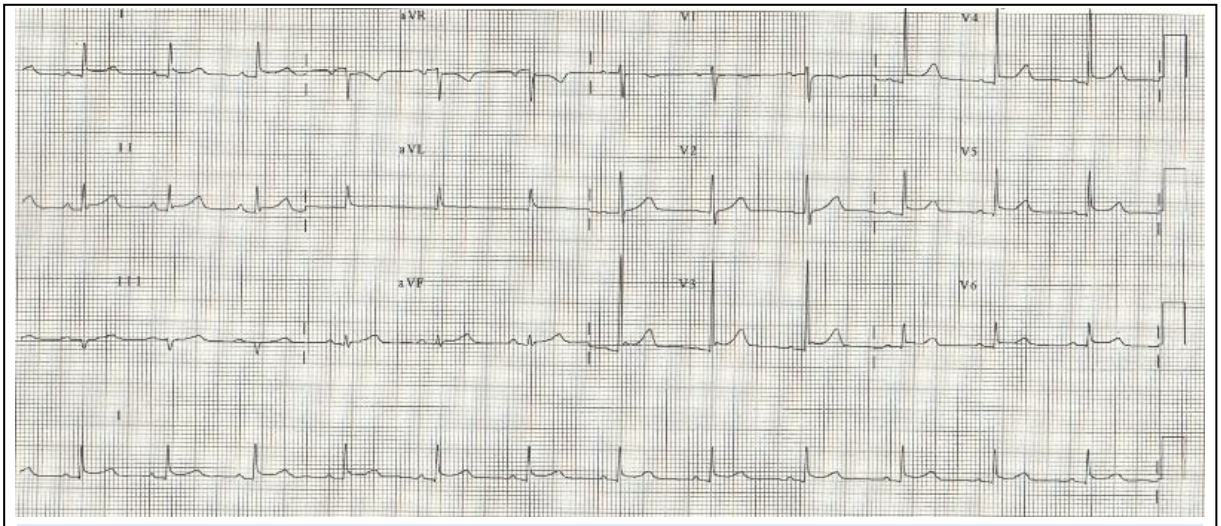


Figure 5/69. Pericarditis. Only trivial concave ST segment elevation appears in some of the leads, however, it is deemed to be larger due to the PR segment depression presenting in several leads (in I, II, aVL and V3-6). PR segment depression is often detectable in pericarditis, while it is associated with an acute coronary syndrome only rarely (e.g. in atrial infarction). (Sinus rhythm, 78 bpm, normal QRS axis, normal AV conduction time, PR depression and trivial concave ST segment elevation in leads I, II and V3-6, PR elevation and trivial ST segment depression in lead aVR)

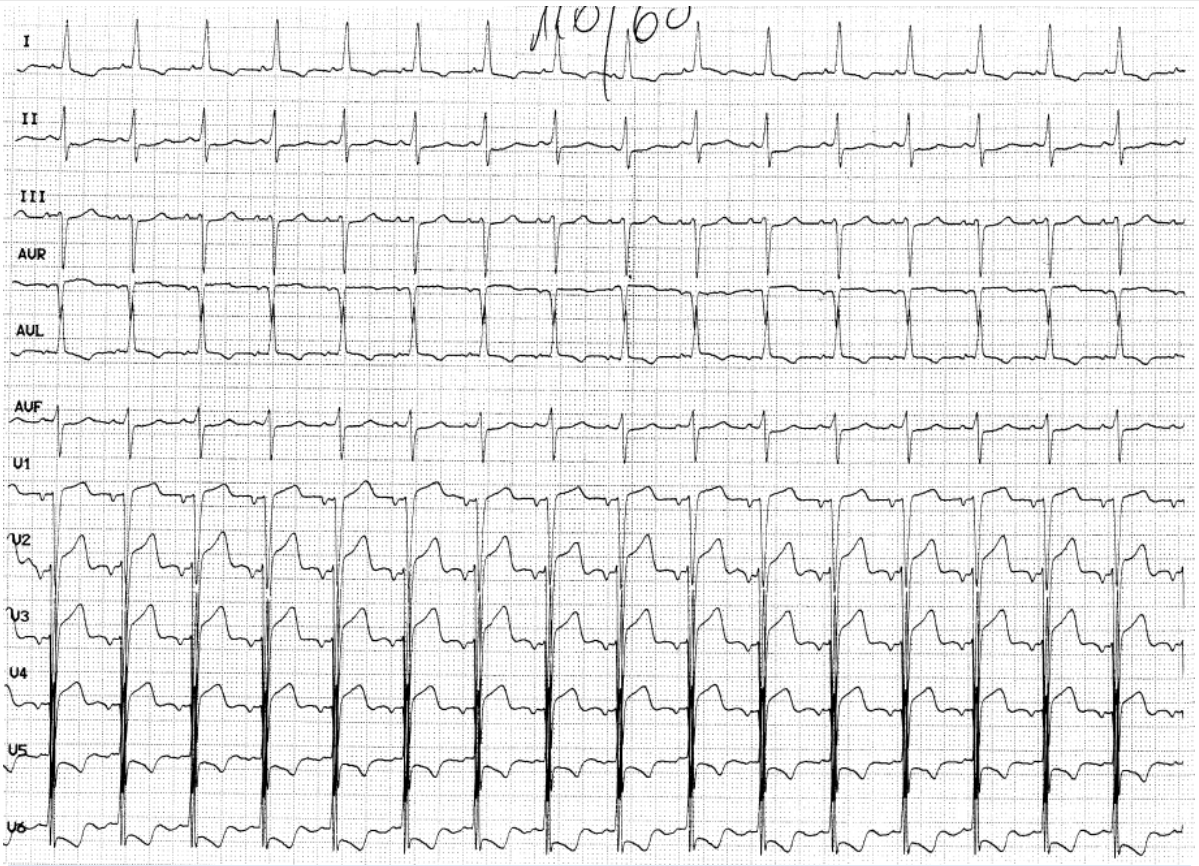


Figure 5/70. ST segment elevation in leads V1-4 caused by left ventricular strain in severe aortic stenosis. If the patient had presented due to chest pain, emergency coronary angiography may not have been avoided since the type of ST elevation corresponds with that observed during an anterior myocardial infarction. (Sinus rhythm, 100 bpm, normal AV conduction time, P mitrale, left axis deviation, downsloping ST segment depression of 1 to 2 mm and negative T waves in leads I, aVL, V2-6, convex ST segment elevation of 2 to 3 mm and positive T waves in leads V1-4.)

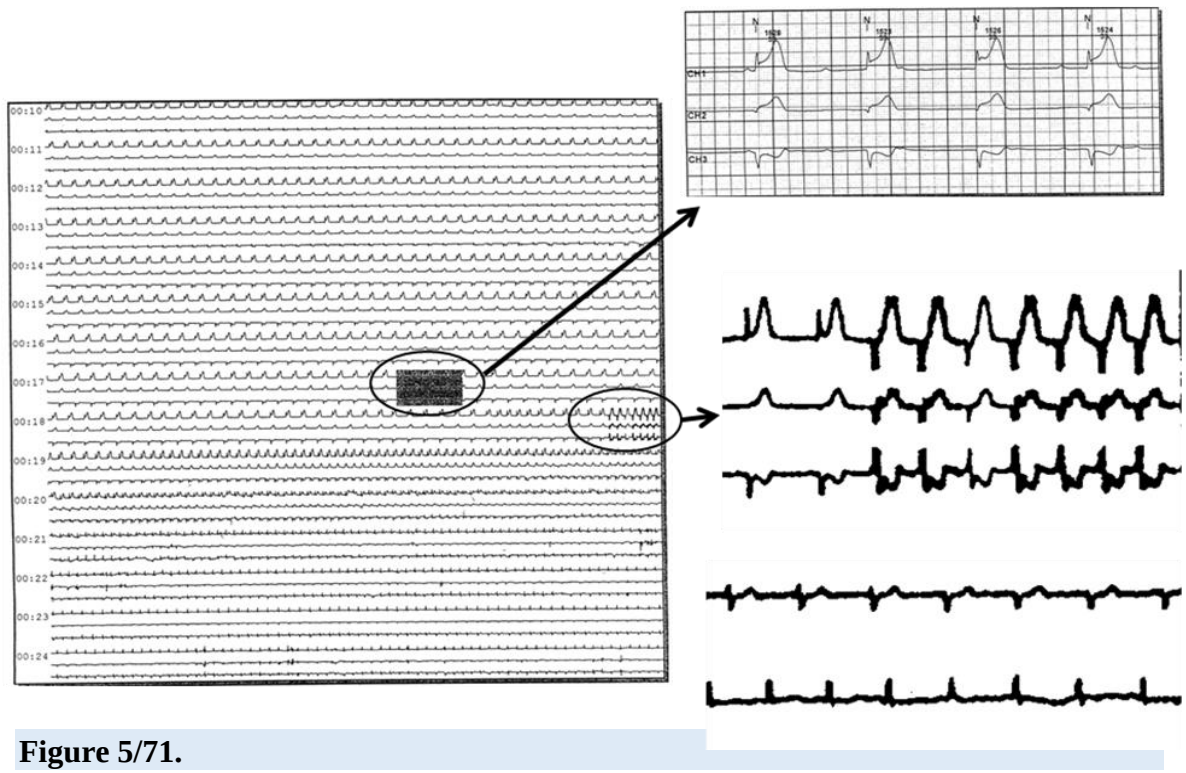


Figure 5/71.

Prinzmetal's angina recorded during Holter monitoring and caused by prolonged vasospasm of the right coronary artery. It is accompanied by ST elevation and, in fact, third degree AV block. During cessation of the ST elevation and resolution of the vasospasm, reperfusion arrhythmia (accelerated idioventricular rhythm) presents and, afterwards, along with cessation of the ST elevation, AV conduction normalizes as well. No organic abnormalities were shown during the coronary angiography.

FACTS THAT YOU MUST KNOW:

1. In case of ST segment depression occurring in several contiguous leads as well as chest pain, one should always think of the presence of myocardial ischaemia. It is mandatory to record an ECG if chest pain presents.
2. Myocardial infarction may be associated both with ST segment depression and elevation; distinction of the cases associated with ST segment depression (NSTEMI) from angina pectoris is possible based on troponin levels.
3. In case of ST segment depression in the precordial leads or inferior myocardial infarction, one should use additional ECG leads. These are leads V7-9 in the former case and right ventricular leads in the latter case.
4. ST segment elevation in leads II, III and aVF suggests inferior myocardial infarction, that in leads V1-4 implies anteroseptal myocardial infarction (if leads I, aVL, V5-6 are also involved beyond V1-4, that is extensive anterior infarction).
5. If ST segment elevation occurs in contiguous leads, one first should think of the presence of myocardial infarction (STEMI).
6. If there is ST segment elevation on the ECG, one should look for the presence of reciprocal changes because they are present only in myocardial infarction.
7. ST segment elevation in leads V1-4 with concurrent LBBB should not be interpreted as myocardial infarction, because it is only a secondary repolarization abnormality. Widening of the QRS complex for other reasons results in significant repolarization abnormalities, which are not signs of ischemia.
8. Persistant ST segment elevation is often present after an anterior myocardial infarction.
9. One should look for the presence of pathological Q waves in the contiguous leads. Please note that Q waves in leads III, aVR and V1 are not pathological findings by themselves.

CHAPTER 6

EXERCISE STRESS TEST

The exercise stress test is the most simple noninvasive testing method carrying indirect information on the coronary circulation, myocardial perfusion as well as functional status of the myocardium. It is often performed if the presence of ischemic heart disease is suspected and the patient has typical complaints, or for the detection of residual ischemia in the postinfarction state. Most commonly, stationary bicycle or treadmill stress testing is performed in clinical practice. ECG and blood pressure of the patient is continuously monitored during the test.

Attainment of a certain heart rate is set as an objective during an exercise stress test. This is the so-called target heart rate, which is dependent upon the age of patients. One can distinguish *maximal heart* rate defined as 220-age, and *submaximal heart rate*, which is 85% of the maximal heart rate. During an exercise stress test, the test is usually continued along with gradually increasing levels of exercise until submaximal heart rate has been attained, unless there are other indications for termination. Since the use of some medications may prevent from reaching the submaximal heart rate, while others may obscure the ECG abnormalities caused by ischemia, their *intake should therefore be withheld transiently* prior to the test. The patient must not receive *beta blockers, digitalis preparations, calcium channel blockers and nitrates* 24 to 48 hours prior to the test, because there may frequently be a false positive or false negative test result with the use of these medications and the test cannot be evaluated appropriately.

Sensitivity and specificity of the test ranges from 70 to 75% even in the presence of typical chest complaints. This means that in case of positive test results, significant coronary artery stenosis is found in 70 to 75% of cases or, alternatively, in case of the presence of a significant coronary artery stenosis, the test will be positive in 70 to 75% of cases. Positivity of the test is often an indication for coronary angiography. It is therefore important to follow the appropriate guidelines when performing an exercise stress test in order to avoid the risk associated with coronary angiography in a patient who surely does not have coronary artery disease. (For example, stabbing chest pain in a 40-year-old, non-smoker female with few risk factors should not necessarily be an indication for exercise stress testing because positivity of such a test would certainly represent false positivity.)

Causes of false positivity during an exercise stress test include valve diseases (aortic stenosis, mitral insufficiency, mitral valve prolapse), digitalis effect, electrolyte disturbances (K^+), hyperventillation, anemia, bundle branch block, WPW syndrome and left ventricular hypertrophy.

It is also observable that the test will be positive far more frequently (95%) in significant left main stem or triple-vessel disease, whereas it is more often negative in single-vessel disease (even in severe cases) or for borderline stenoses.

Indications for exercise stress testing:

- It is advisable to be performed if the suspicion of coronary artery disease is raised and there are no contraindications to the test.
- After a myocardial infarction (>10 days) to detect residual ischemia if no reperfusion treatment was applied.
- Monitoring after a revascularization therapy (PTCA, CABG).
- To assess exercise capacity in heart failure.
- Searching for exercise-induced arrhythmias and chronotropic incompetence (SSS) or to determine refractoriness of the accessory pathway in WPW syndrome.
- To assess exercise tolerance and perioperative risk before high-risk surgery (lungs, great vessels).

Contraindications for exercise stress testing:

- Acute stage of AMI (first few days);
- Unstable angina;
- Severe aortic stenosis and hypertrophic cardiomyopathy;
- Current high blood pressure (RRsyst.>160 mmHg);
- Hypokalaemia;
- Severe or unstable heart failure (NYHA class III-IV);
- Severe disturbances of impulse formation and conduction, ventricular arrhythmias;
- Acute pericarditis, myocarditis, febrile illness, anemia;
- Left ventricular thrombus, acute thromboembolism.

Indications for termination of an exercise stress test:

- Severe angina;
- Severe ST segment depression (>2-3 mm) or angina + ST depression (>1 mm);
- RR > 230/120 mmHg;
- a decrease in RRsyst. (>10 mmHg) or heart rate (raises suspicion of left main stem stenosis or severe left ventricular dysfunction!);
- ST segment elevation;
- Bundle branch block, ventricular tachycardia, AV block, atrial fibrillation, frequent VPBs;
- Sudden paleness, cyanosis, cold sweats;
- Fatigue, dizziness, dyspnea or at the patient's request.

6.1. Basics in exercise physiology

During physical exercise, oxygen consumption of the body increases gradually in parallel with myocardial oxygen consumption. Oxygen supply of an organ can be enhanced in two ways:

a. it increases the extraction of oxygen from a given amount of blood flowing through this organ,
b. it increases the amount of blood flowing through a given area.

Oxygen extraction is already at the maximum level in the myocardium, so the myocardium can address the need of increased oxygen demand only through the latter mechanism. Under normal circumstances, blood flow of the heart can be increased four or five-fold. However, below the level of a stenotic coronary artery, dilatation is observable at the level of arterioles even under resting conditions, which cannot be increased further in parallel with physical exercise, therefore, coronary circulation becomes insufficient. As a result of insufficiency of the coronary circulation, the above detailed signs of ischemia are detectable during the test. The exercise level at which ECG signs occur indicates the severity of ischemia. Not only ST segment and T wave abnormalities may imply the presence of myocardial ischemia, but also progressive decrease of the blood pressure or the occurrance of arrhythmias (atrial fibrillation, ventricular extrasystole or ventricular tachycardia).

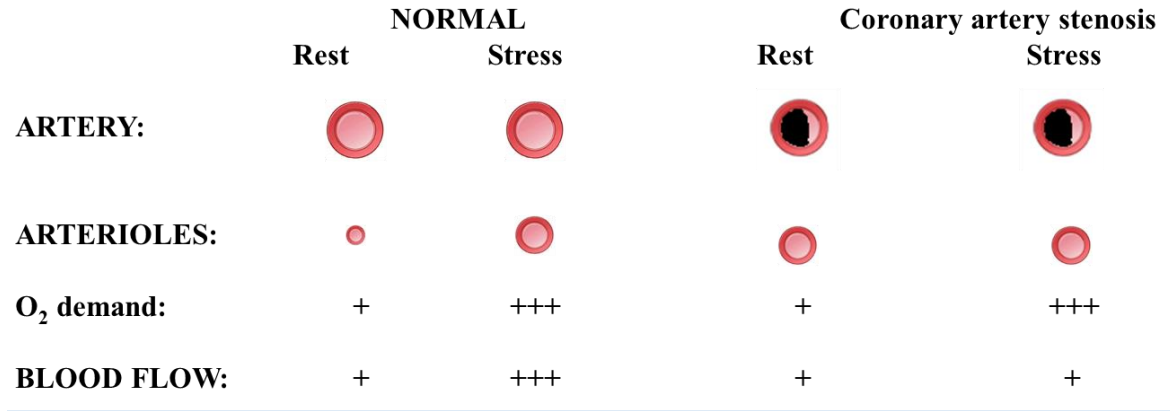


Figure 6/1. Relative coronary insufficiency Blood flow at tissue level is primarily determined by the diameter of arterioles. In case of an epicardial coronary artery stenosis, arterioles are already dilated, so further increase in blood flow at tissue level cannot be achieved any more.

The diameter of blood vessels, beyond circulating humoral factors, is determined by the status of the autonomic nervous system. Since most blood vessels are under sympathetic innervation, current sympathetic tone is therefore the major determinant in the cross section of a given blood vessel segment and the amount of blood flow passing through it. Physical exercise results in activation of the sympathetic nervous system, the effects of which are introduced via adrenergic receptors. It is also due to this that knowledge of adrenergic receptor function is important.

Adrenergic receptors:

(Blood vessels are under sympathetic innervation, except pia mater and erectile tissues. Most blood vessels are innervated by sympathetic adrenergic nerves, while those of the muscles by sympathetic cholinergic nerves.)

α1-receptor: They are situated in blood vessels (small arteries and arterioles)! *Vasoconstriction* occurs in response to their stimulation.

stimulator: norepinephrine (it is used to increase blood pressure),

inhibitor: prazosin (it is used to treat high blood pressures).

α2-receptor: They have a presynaptic position! They inhibit the release of norepinephrine.

stimulator: guanfacine (antihypertensive agent),

inhibitor: yohimbine (it is used in erectile dysfunction).

β1-receptor: They are mainly situated in the heart (SA node, cardiac conduction system, ventricular musculature), but can also be found in fatty tissue and juxtaglomerulal cells of the kidney (renin production). Positive chrono-, ino-, dromo-, bathmo- and lusitropic effects are exerted through these receptors. They enhance lipolysis and their stimulation in the kidneys results in renin production.

stimulator: dobutamine (it is used for the treatment of heart failure),

inhibitor: metoprolol (an antihypertensive and antiarrhythmic agent).

β2-receptor: They are also situated in blood vessels (coronary arteries, skeletal muscles), but can also be found in other smooth muscles (bronchus, uterus, etc.). Binding of a ligand to its receptor results in vasodilatation and smooth muscle relaxation (bronchodilatation, uterine relaxation).

stimulator: epinephrine, salbutamol (used in bronchial asthma),

inhibitor: butoxamine.

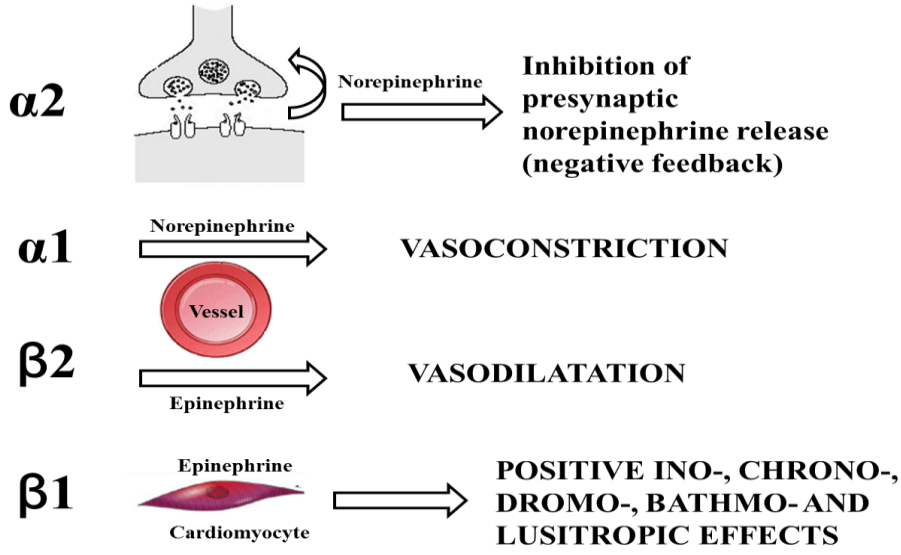


Figure 6/2. The most important processes mediated by catecholamine receptors.

Following that, let's review redistribution of the circulation and cardiac output during physical exercise. Blood pressure is fundamentally determined by two factors: cardiac output and total peripheral resistance. Cardiac output is the product of stroke volume and heart rate, while peripheral resistance is determined by the cross section of blood vessels and blood viscosity. The '25 percent rule' can be used as a rough estimate, which carries information promoting memorization rather than accurate data; that is 25% of the cardiac output is carried to the *heart* (5-6%) and *brain* (15-18%), 25% to the *liver* and *splanchnic* region, 25% to the *kidneys* and 25% to the *muscles* and *skin*. During dynamic work, oxygen demand is increased in working muscles, resulting in vasodilation in the given region as a consequence (β2 effect). On the other hand, vasoconstriction develops in the nonworking muscles, splanchnic region, liver and kidney arterioles (α1 effect). The stimulation of β 1 receptors increases contractility, and thereby also stroke volume, through a positive inotropic effect and heart rate will rise as part of the chronotropic response. Both effects promote an *increase in cardiac output*, which therefore demonstrates a linear increase being *proportional to the degree of rise in oxygen utilization*. By this process, increased cardiac output can direct up to 80-85% of circulating blood to the working muscles, while about 15-20% of blood is getting to the muscles at rest. The rise in partial tissue CO2 concentration, temperature and acidity promotes extraction of oxygen from the blood, which may increase three-fold in muscles (as opposed to the myocardium). Blood vessels of the skin undergo constriction initially, then they become dilated to assist in heat dissipation, thereby decreasing peripheral resistance. As a result of the above processes, systolic blood pressure will always increase, however, vasodilator response in the working muscles and skin may decrease peripheral resistance to such a degree that diastolic pressure will decrease. Diastolic blood pressure response largely depends on the compliance of blood vessels, due to which diastolic blood pressure may stagnate or even rise, depending upon how the vascular system is able to utilize its vasodilator capacity or this capacity is already limited due to the extensive atherosclerosis.

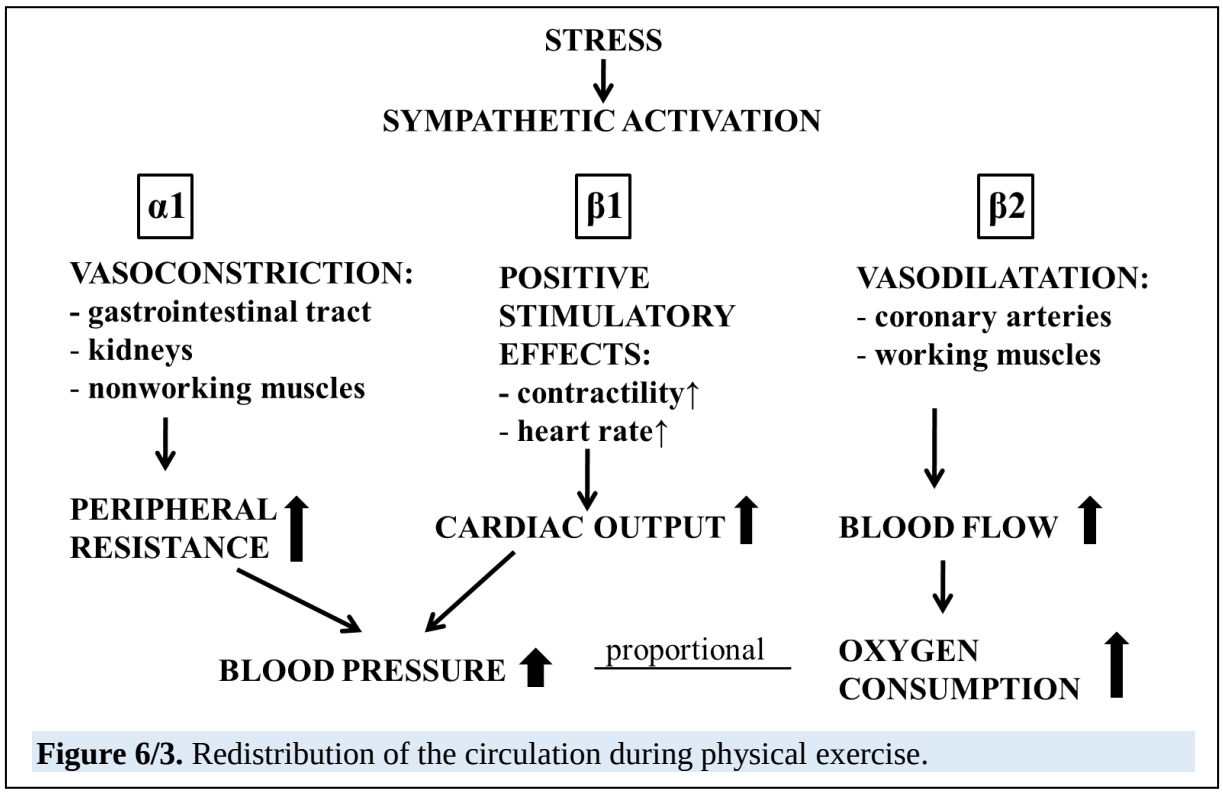


Figure 6/3. Redistribution of the circulation during physical exercise.

The circulation and oxygen uptake of the heart is partly determined by general hemodynamic effects, and partly by specific factors. The specific situation of the heart lies in the fact that while blood flow is determined by the systolic or mean arterial pressure in the majority of organs, it takes places in the coronary arteries during diastole. This is because blood vessels undergo compression in systole, especially subendocardially. Myocardial blood flow is driven by the difference of diastolic pressure in the aorta and left ventricular diastolic pressure. Therefore, *each factor that shortens diastole, decreases diastolic blood pressure or increases left ventricular diastolic pressure will impair coronary perfusion*. In addition, special redistribution processes take place intramyocardially; e.g. in response to tissue metabolites (e.g. adenosine), vasodilatation occurs in the blood vessels belonging to the blood supply territory of an intact coronary artery, while arterioles behind a stenotic coronary artery are not capable of dilating any longer. As a consequence, blood is bypassed through intramural collaterals to the intact region demonstrating no ischemia, and blood supply of the ischemic region relatively decreases. This phenomenon is called the *coronary steal effect* (or Robin Hood effect) and is used to establish a diagnosis during stress myocardial perfusion imaging scans (SPECT). The diagnostic accuracy of these nuclear scans exceeds that of the exercise stress test, ranging from appr. 75 to 90%.

6.2. Basic concepts and activities related to an exercise stress test

Cardiac oxygen consumption increases in parallel with cardiac output, however, these can only be estimated with noninvasive techniques and their accurate measurement is complicated (spiroergometry). At rest, oxygen consumption of the body is *3.5 ml/min/kg of body weight*, which is called 1 MET (metabolic equivalent). MET= 14,7 - (0.13 x age). Up to 9 to 10 METs can be achieved during physical exercise, meaning that the body could increase its oxygen consumption nine- to ten-fold versus the resting state. Below 4 METs, between 4 to 7 METs and above 7 METs, it is referred to as poor, moderate and good exercise tolerance, respectively.

If a patient perceives even dressing up or short walks as stressful, exercise tolerance is around 2-3 METs (NYHA III), if he can climb 2 flights of stairs without stopping, it is above 4 METs (NYHA II), and if he can perform hard gardening activities or jogging, it is above 7 METs (NYHA 0-I).

If exercise stress test results are negative at ≥7 MET, it is very likely that no critical coronary artery stenosis is present.

Changes in systolic blood pressure and pulse rate are considered the two values that are easy to measure and change in parallel with oxygen utilization, so by monitoring these the level of rise of oxygen uptake can be provided. The product of systolic blood pressure and heart rate is referred to as the *double product* or *rate pressure product*, which is also proportional to oxygen consumption. It is typical of stable angina that it always occurs at an identical value of the double product. Oxygen consumption of the body demonstrates a linear increase for some time during physical exercise, then it reaches a certain point after which flattening of the curve is observable and it cannot be increased any longer (*maximal oxygen consumption*). Beyond this point, additional energy can only be gained by anaerobic metabolism. The level of oxygen uptake where lactic acid content of the blood starts to rise exponentially after the previous linear increase is called *anaerobic threshold*. From this point onwards, energy gain can only be increased via anaerobic metabolism.

6.3. Types of exercise stress tests

Mechanical exercise:

1. Static - handgrip strength test (such as standing with heavy shopping bags): This form of exercise primarily results in an increase in diastolic blood pressure, and that of the heart rate only later, so it is not used in clinical practice.
2. Dynamic - stationary bike or treadmill stress testing: This form of exercise primarily results in an increase in heart rate and that of the systolic blood pressure only secondly. The Bruce protocol is used most commonly, for which the exercise level is increased by 25 Watts (W) every three minutes starting at 25 Watts, meanwhile blood pressure measurements are performed every three minutes in addition to continuous multi-channel ECG monitoring and automated ST segment analysis until the patient reaches the target heart rate or any other termination criteria are met.

Pharmacodynamic stress tests:

1. Dipyridamole stress testing: This substance prevents the reuptake and degradation of adenosine produced in tissues. Under normal conditions, the life span of adenosine in the circulation can be measured in seconds due to the rapid reuptake and degradation, however, adenosine levels increase in case dipyridamol is used. Adenosine exerts its major vasodilator effect on the microvasculature (primarily on blood vessels with a diameter below 150 μm). The relative coronary insufficiency of the region behind a stenosis becomes detectable as a result of an increasable perfusion in the intact regions.
2. Dobutamine stress testing: Dobutamine is a sympathomimetic agent with selective β1-adrenergic receptor agonist properties, so it can fully mimic cardiac effects produced by physical exercise.

Combined methods of stress testing:

1. Dipyridamole myocardial stress perfusion imaging (SPECT);
2. Dobutamine or dipyridamole stress echocardiography.

6.4. Physiological ECG changes during exercise:

1. tachycardia (positive chronotropic effect);
2. a decrease in the PQ interval (positive dromotropic effect);
3. an increase in the P wave amplitude;
4. right axis deviation;
5. decreasing R and T wave amplitude;
6. upsloping or junctional ST segment depression (between 1 and 1.5 mm).

6.5. Pathological ECG changes during exercise:

1. upsloping or junctional ST segment depression, if the ST segment is below the isoelectric line by ≥1.5 mm even at 60 msec after the end of the QRS complex (J point);

2. downsloping ST segment depression is the most specific one;
3. ST segment elevation in leads with Q waves indicates deteriorating regional wall motion abnormalities, while in leads with no Q waves it may be a sign of significant proximal epicardial coronary artery spasm or stenosis. ST segment elevation in leads V1 and aVR only is of minor importance in itself.

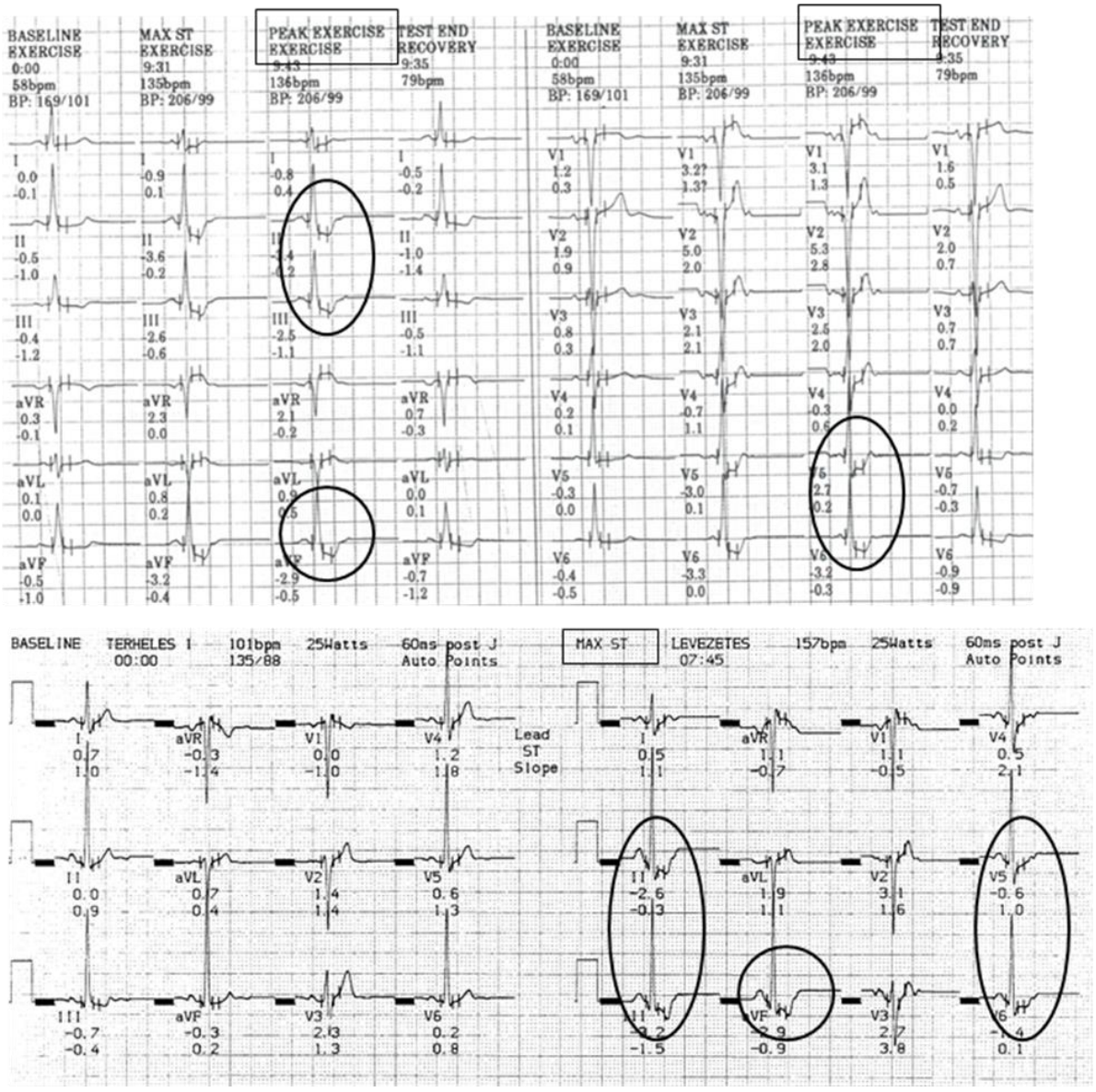


Figure 6/4.

Above: summary sheet of a positive exercise stress test. Please note that significant downsloping ST segment depression developed at peak exercise also in leads V5-6 in addition to leads II, III and aVF, moreover, typical chest pain also presented. **Below**: a false positive exercise stress test. Although significant ST segment depression in leads II, III and aVF is also visible here, however, there are no such findings in leads V5-6 and no chest complaints presented either. The underlying cause of the ST segment depression is likely to be projection onto the ST segment of the P wave repolarization caused by the increased P wave amplitude.

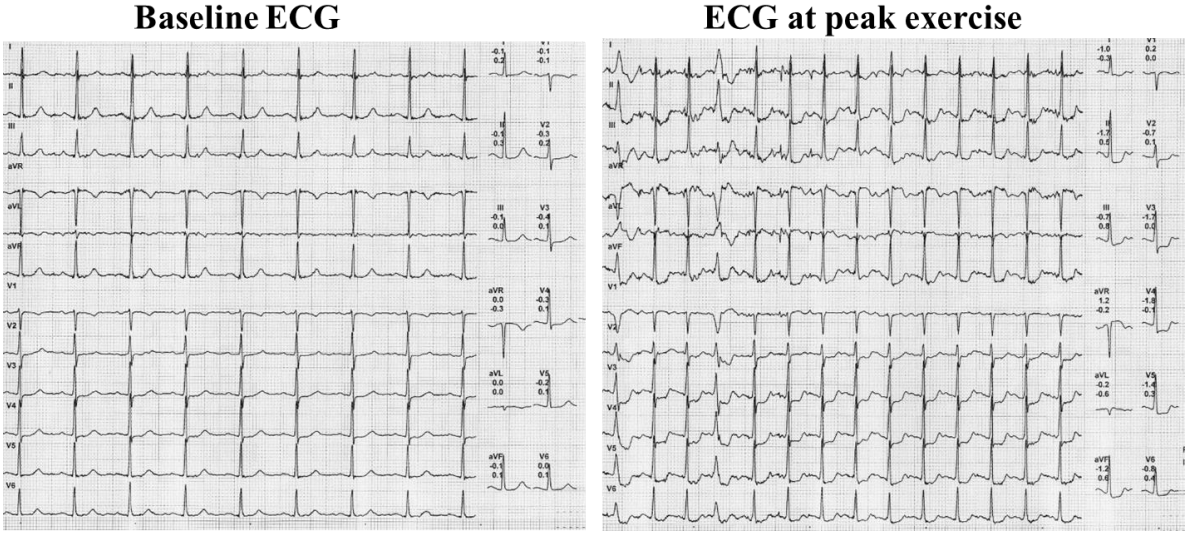


Figure 6/5. In severe stenosis of the origin of LAD, diffuse ST segment depression and angina developed during stress testing as well as there was an increase in ventricular ectopic activity.

It is important to mention that the site where ST segment depression occurs cannot localize ischemia for an exercise stress test. Namely, for an ST segment depression occurring in leads II, III and aVF, one should avoid using the term 'inferior ischemia'. It is always the respective leads, exercise level (in Watts), submaximal heart rate and double product that should be described and one should also take a note on the patient's complaints as well as arrhythmias presenting during exercise. For example: At the submaximal heart rate (146 bpm) attained at a moderate exercise level (100 W, 5 MET), downsloping ST segment depression of 1.5 to 2.1 mm and T wave inversion developed in leads II, III and aVF and it was accompanied by typical angina. No arrhythmias occurred, the maximum RR was 195/108 mmHg. During an exercise stress test, it is almost always in leads II, III, aVF or V3-6 that ST segment depression presents, and almost never in the rest of the leads. Based on observations, it is ST segment depression in lead V5 that reflects ischemia the most, while *ST depression in leads II, III and aVF often proves to be false positive*. Not only the development of ST segment depression, but also the temporal dynamics of its cessation may be informative. True signs of ischemia persist for at least 3 minutes after termination of the exercise. An abnormality persisting for more than 6 minutes may be a sign of severe left main stem stenosis. The longer and longer the ST segment depression persists in the recovery phase, it is the most important distinguishing feature between a true and false positive exercise stress test. It may occur that ST segment abnormalities develop 2-3 minutes after the cessation of exercise, but this is frequently a sign of false positivity. Precordial U wave inversion (LAD) or an increase in amplitude (Cx, RCA) is a rather specific sign of severe coronary artery stenosis. The prognostic value of ventricular premature beats (and short NSVTs) developing during exercise is doubtful.

FACTS THAT YOU MUST KNOW:

1. Beta-blockers and digitalis preparations should be withheld prior to the performance of an exercise stress test.
2. ST segment depression presenting in leads V5-6 (or the rest of the precordial leads) during an exercise stress test is far more pathognomonic of ischemia rather than that in leads II, III and aVF if it is accompanied by typical chest complaints and persist for minutes even in the recovery phase.
3. Patients may have a severe coronary artery stenosis in case of a negative result of the exercise stress test; on the contrary, it frequently occurs with positive test results that the patient has no coronary artery disease.
4. Above 4 and 7 METs, it is referred to as moderate and good exercise capacity, respectively.

CHAPTER 7

PACEMAKER

The first pacemaker implantation was performed in 1957, since when enormous development took place in the pacemaker techniques from both medical and electrotechnical aspects. Beyond the traditional indication (missing ventricular electrical systole), resychronization of asynchronous ventricular contractions accompanying heart failure may be performed nowadays by them in order to optimize ventricular contractility, which may significantly improve the cardiac output and symptoms of patients with heart failure. In addition, 'teachable' devices with a very developed communication system and equipped with antitachycardia pacing function are also available, which are able to terminate life-threatening arrhythmias (e.g. ventricular fibrillation) also by delivering an intracardiac shock (ICD – implantable cardioverter defibrillator). These latter ones will not be described in this chapter, only general information on pacemakers will be provided. Moreover, indications for pacemaker implantation will not be described in a detailed fashion in this chapter either, because they are regulated by the current guidelines in cardiology.

A pacemaker typically consists of two parts: The generator is generally implanted below the skin in the clavicular region or below the pectoral major muscle, from which one or more pacemaker leads are introduced through the venous system into the right side of the heart. The pacemaker leads may be unipolar and bipolar. For unipolar leads, one of the poles is situated at the tip of the electrode in the heart and the other one in the pulse generator, while for bipolar leads, both poles are located within the heart cavity (ring electrode). On the surface ECG, *the signal of unipolar leads (i.e. spike) has a large amplitude, whereas it has a small amplitude for bipolar leads*. The sequence of atrial and ventricular activation, and thereby signs on the surface ECG, change fundamentally following pacemaker stimulation. For example, when a ventricular lead in the right ventricular apex starts to operate, the morphology of QRS complexes most readily reminds of those observed in left bundle branch block (LBBB).

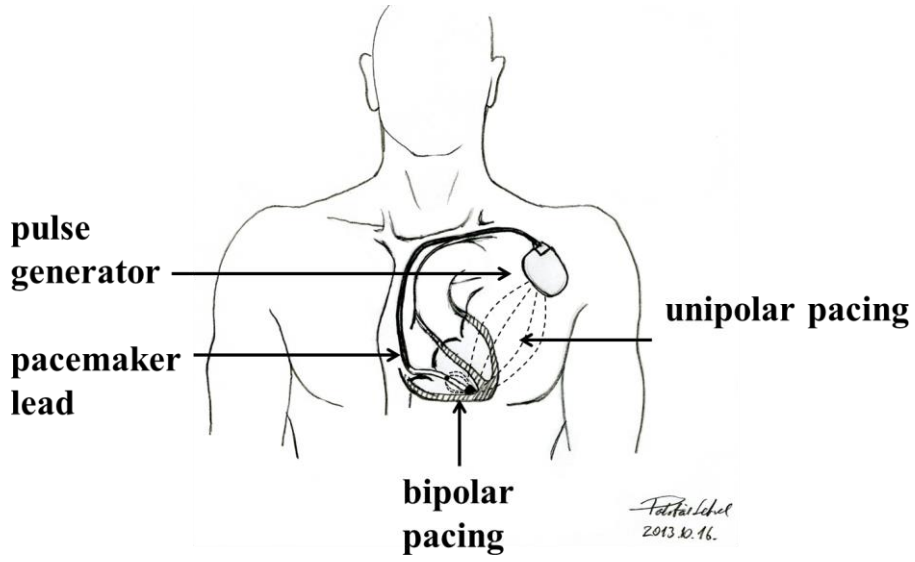


Figure 7/1. Pacemaker - uni- and bipolar pacing.

The reason for that is that the sequence of impulse propagation is very similar to that seen in LBBB, i.e. the left ventricle becomes activated from the right ventricle and there is also right to left activation of the interventricular septum. The change in morphology and widening of the QRS complexes also alters the repolarization sequence, frequently making the diagnosis of the signs of ischemia or acute myocardial events impossible. Nevertheless, it is important to know that in the 'demand' pacing mode (when the pacemaker does not generate impulses currently), the activation sequence may be normal. Therefore, one cannot say that ischemia can never be diagnosed based on the ECG in a patient living with a pacemaker.

7.1. Pacemaker coding system

Pacemakers are divided into groups by a specific code system consisting of 5 letters. Following their order, the letters have the below described meaning:

I. It indicates the site of pacing, which may be the atrium (A), ventricle (V) or both (D = dual).

II. It marks the site of sensing, which also may be the atrium (A), ventricle (V) and both (D) or none (0) of them.

III. This represents the response to pacing, which may be inhibition (I), i.e. intrinsic activity of the heart inhibits the operation of a pacemaker; triggering (T), i.e. activity of the heart triggers the operation of a pacemaker (e.g. sequential ventricular pacing induced by a P wave); and both (D) or none (0) of them.

IV. Programmability: rate responsive (R = rate modulation) that is a sensor is sensing current motion, electrical impedance, etc. of the patient, based on which the device adjusts the heart rate to current needs, e.g. the pacing rate is set to higher levels during jogging.

V. Special functions: antitachycardia pacing (P), shock delivery or cardioversion (S), or both (D).

Based on the above, the major types of permanent pacemakers are the following:

1. AAI: Atrial demand mode inhibited by the P waves. This means that the pacemaker lead is in the right atrium and sensing and pacing is performed here. Normal atrial activity inhibits pacemaker operation, while if it is absent, the pacemaker intervenes and paces the atrium at the preset rate. Such a device may only be implanted if atrioventricular conduction is impeccable, most commonly in isolated sinus node dysfunction.
2. VVI: Ventricular demand pacing inhibited by intrinsic QRS complexes The pacemaker lead is situated in the right ventricle and sensing and pacing is performed here. Ventricular activation interrupts the pacemaker activity, however, a pacing spike will be delivered in the absence of the QRS complex and this pacing ensures ventricular depolarization at the preset rate.

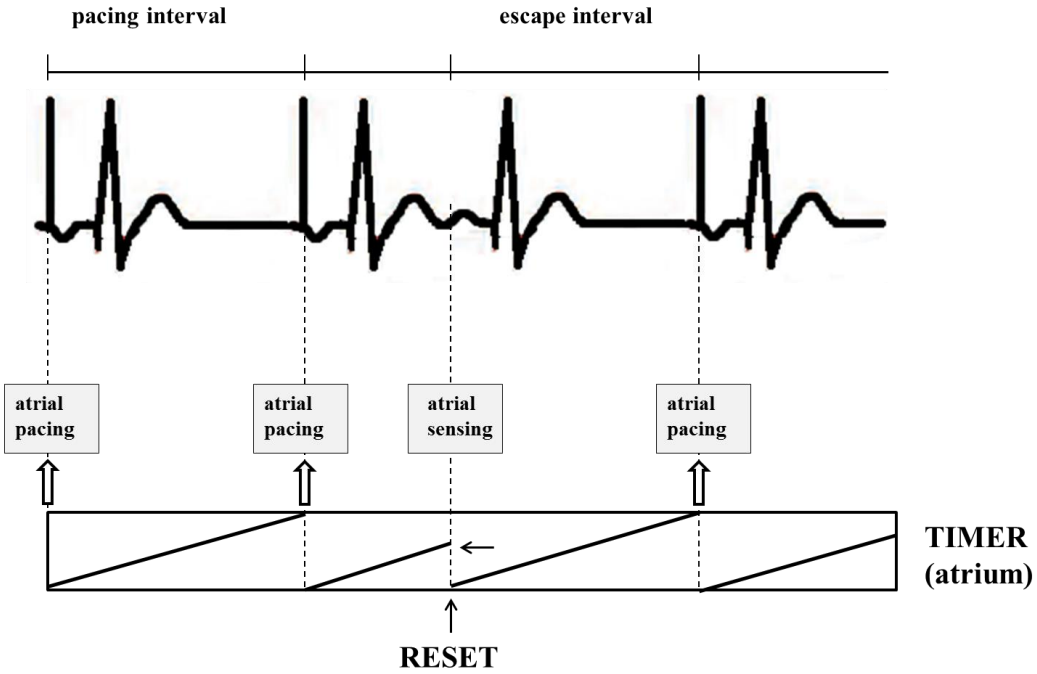


Figure 7/2. Regulation of pacing in the AAI mode The first and second P waves are generated by pacing, but the third beat is generated by the heart itself, which is sensed by the pacemaker and resets its timer. After the sensed beat, no new intrinsic P wave occurs within the escape interval, so the pacemaker will generate a pacing spike. The pacing and escape intervals may be identical, however, the escape interval may be longer in rate hysteresis.

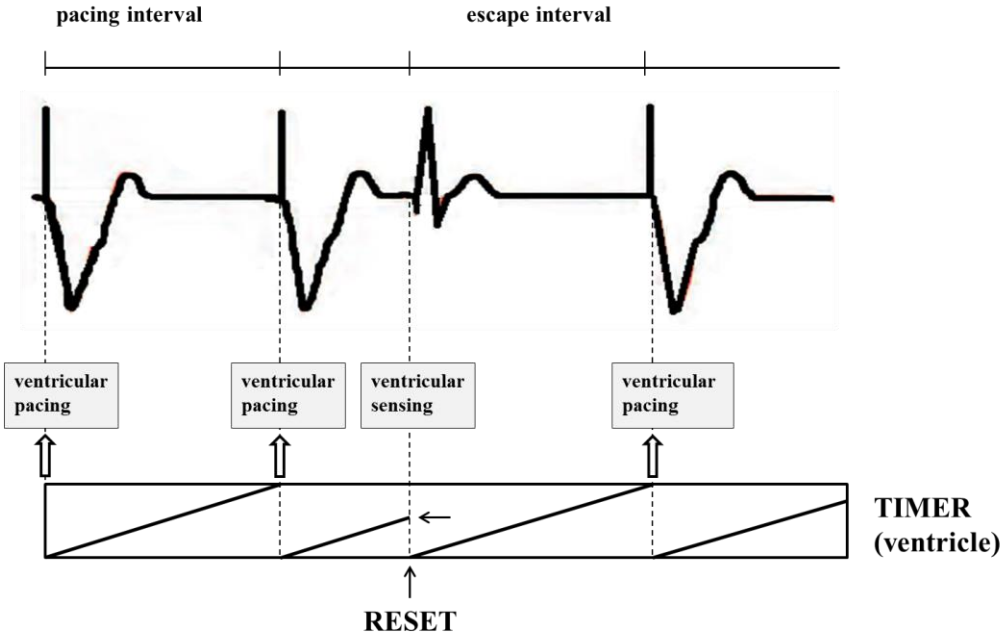


Figure 7/3. Regulation of pacing in the VVI mode The first and second QRS complexes are generated by pacing, but the third beat is generated by the heart itself, which is sensed by the pacemaker and resets its timer. After the sensed beat, no new intrinsic QRS complex occurs within the escape interval, so the pacemaker will generate a spike. The pacing and escape intervals may be identical, however, the escape interval may be longer in rate hysteresis.

3. DDD: combined atrioventricular or AV sequential pacing: It is a device containing two pacemaker leads, one of which is situated in the right atrium and the other one in the right ventricle. Operation of the pacemaker is terminated by the normal activity of the respective heart chamber, while its absence results in the occurrance of pacemaker spikes. Both consecutive contraction of the atria and ventricles as well as AV delay are achieved with the help of the device. This has an important role in ensuring that enough time remains for atrial contraction and emptying and no simultaneous atrial and ventricular activation could occur when atrioventricular valves are closed. Optimal setting of the atrioventricular delay (AV conduction time) creates the possibility of preserving intrinsic ventricular activity that may occur, since our goal is to preserve the intrinsic rhythm and the pacemaker should intervene only if it is absolutely necessary. This, beyond improving the patient's quality of life, increases the battery life of the device.

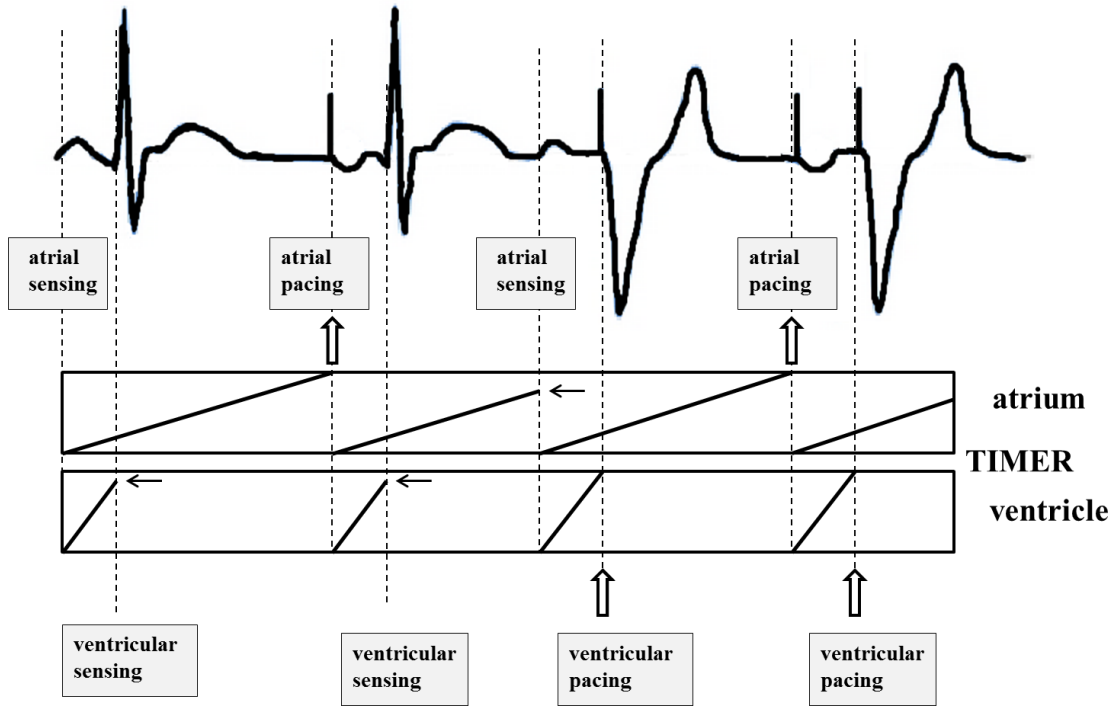


Figure 7/4.

Achievement of AV sequential pacing The first P wave is sensed by the atrial lead of the pacemaker, which will then be conducted with a normal PQ interval and the resulting QRS complex will be sensed by the ventricular lead. During the second beat, the atrial timer reaches the preset lower rate limit and atrial pacing is applied, which is conducted to the ventricles and will be sensed by the ventricular lead. During the third beat, sensing of the intrinsic atrial activity inhibits the delivery of an atrial pacing stimulus, however, reaching the AV delay threshold preset on the ventricular timer, the ventricular lead will deliver a spike due to the delay seen in atrioventricular conduction. For the fourth beat, the atrial lead will generate a spike after reaching the lower rate limit and the ventricular lead will also deliver a stimulus after reaching the AV delay threshold. Horizontal arrows indicate where the timer reset itself before reaching the threshold value (good sensing function), whereas vertical arrows show the site of pacemaker spikes.

Along with DDD pacing, chances of the device to falsely or not deliver spikes are significantly greater, so to avoid this, refractory periods are incorporated into the sensing of atrial and ventricular leads. Ventricular sensing is switched off from the beginning of the QRS complex (ventricular spike) until the end of the T wave (a period of 200-300 ms) in order to avoid sensing of the intrinsic stimulus, QRS complex or the T wave (VRP=ventricular refractory period). Atrial sensing is switched off from the beginning of the QRS complex (ventricular spike) until the segment following the T wave (at about 400 ms) (PVARP=postventricular atrial refractory period) in order to avoid atrial sensing of a ventricular event (spike, QRS complex, T wave) (i.e. pacemaker crosstalk) as well as the sensing of retrograde P waves. There are additional options available to reduce the chance of pacemaker crosstalk. In the short period after the delivery of an atrial spike, ventricular sensing is switched off completely (PAVB = postatrial ventricular blanking); moreover, the so-called ventricular safety period (VSP) begins concurrently with, but lasts longer than, the former period, during which (but beyond the VPB, i.e. ventricular blanking period) events sensed by the ventricular lead result in the delivery of a ventricular spike at the end of the VSP (ventricular safety pacing), through which the delivery of a ventricular stimulus throughout the duration of the T wave becomes avoidable. Intrinsic P waves do not activate the PAVB or VSP.

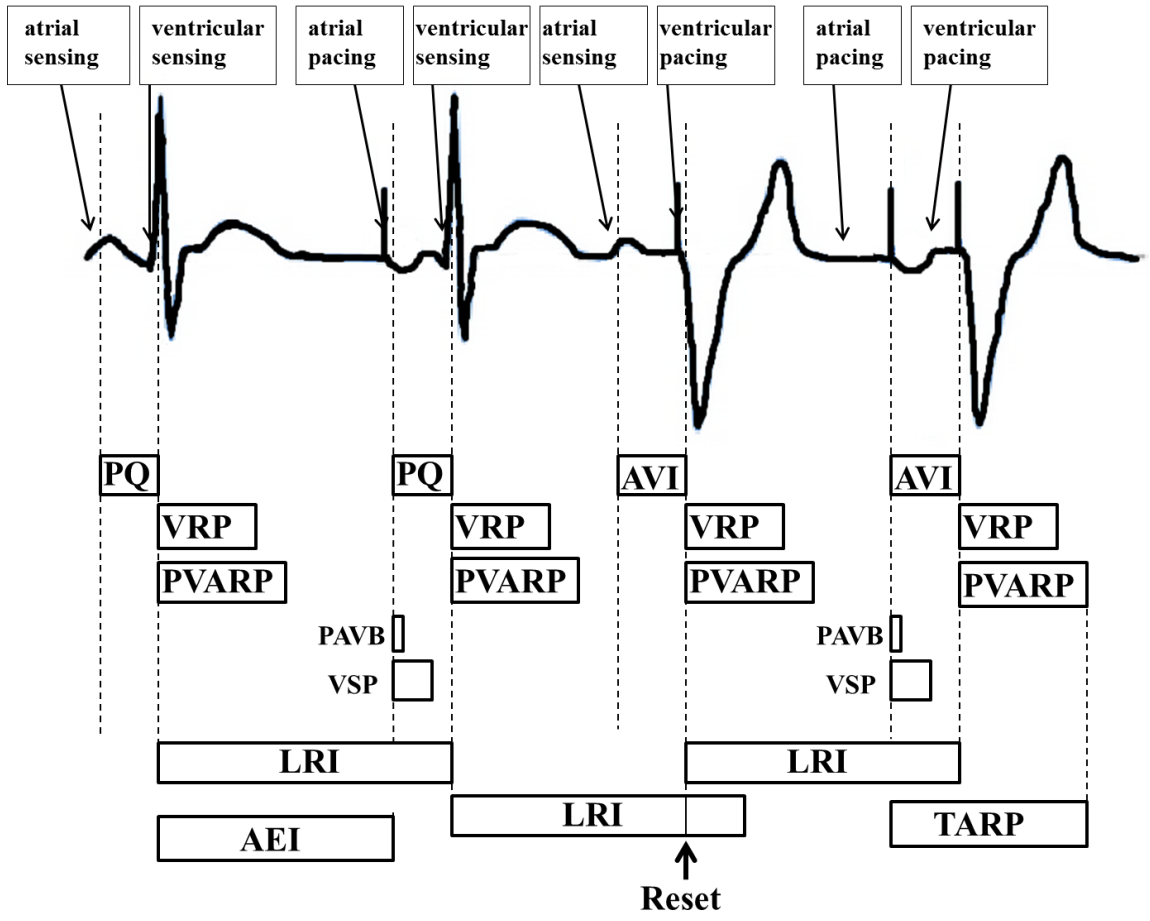


Figure 7/5. Most important time intervals of a DDD pacemaker PQ = normal atrioventricular conduction, which is always shorter than AVI, AVI= atrioventricular interval (20-50 ms longer than PQ), VRP: ventricular refractory period (during this time, no new ventricular stimulus can be delivered by the device), PVARP: postventricular atrial refractory period (during this time, no new atrial stimulus can be delivered by the device), PAVB: postatrial ventricular blanking (10-60 ms), VSP: ventricular safety period, LRI: lower rate interval, AEI: atrial escape interval (atrial pacing rate for consecutive events), TARP: total atrial refractory period.

4. VDD: this is a dual-chamber, single-lead ventricular pacing system controlled by the presence P waves, the most important benefit of which compared to the VVI pacing mode is that ventricular activity is always adjusted to the P waves, thereby maintaining AV sequential contraction of cardiac chambers.

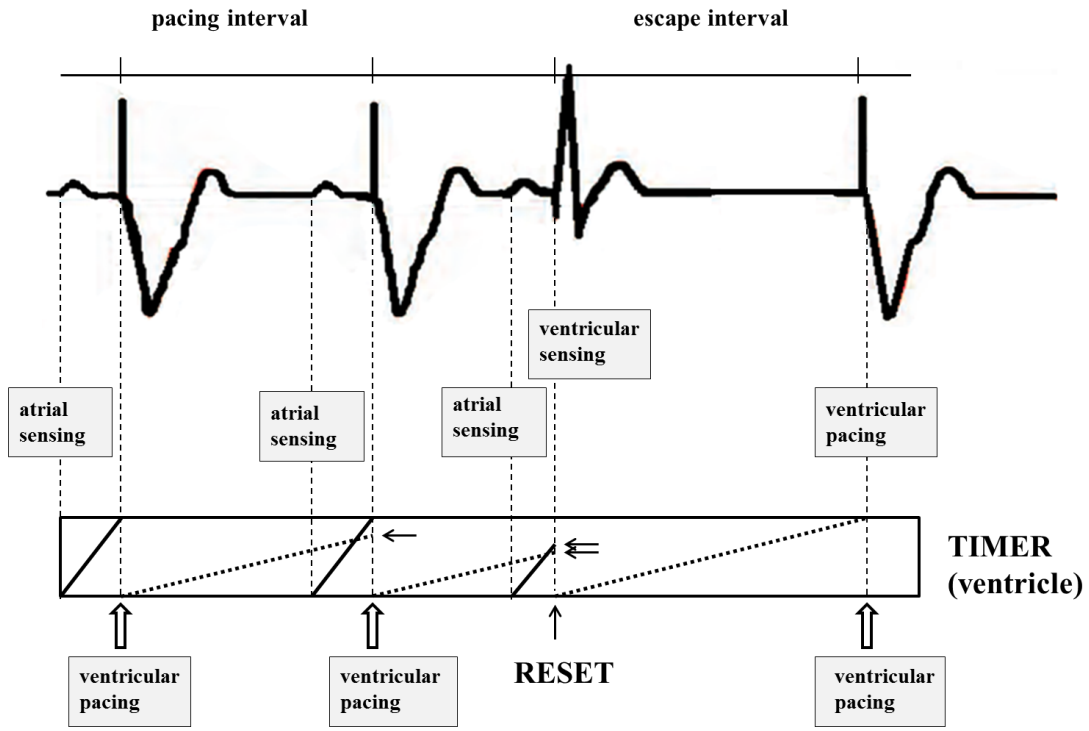


Figure 7/6. VDD pacing mode. The first and second beats are paced ventricular beats synchronized to the P waves, since P waves are not conducted to the ventricles within the preset time. The P wave of the third beat is followed by a conducted QRS complex, which stops ventricular pacing and resets the timer. The fourth beat is the result of ventricular pacing not preceded by a P wave, so the ventricular escape interval (dashed line) reaches the threshold value and a ventricular spike will be delivered even without the presence of a P wave.

5. Rate modulation (R) can be activated for any types of pacemakers, which helps adapt the heart rate to current needs in case of chronotropic incompetence (AAIR, VVIR, DDDR.)

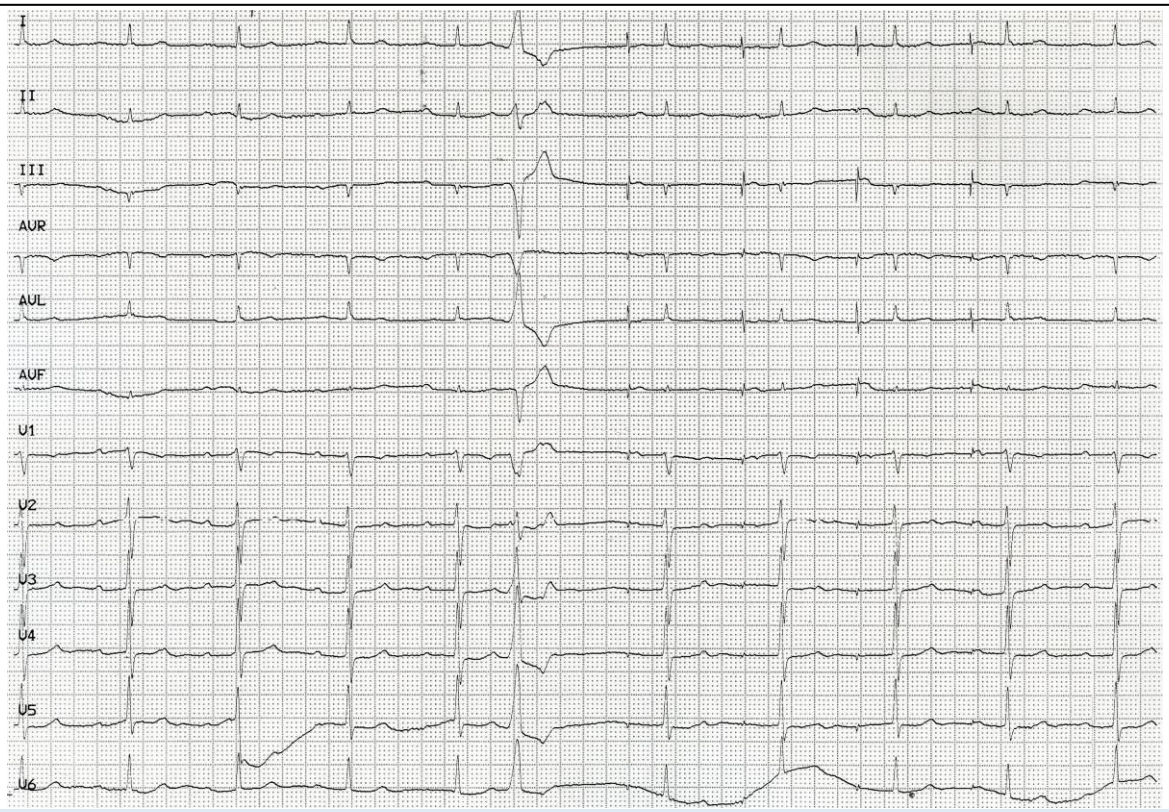


Figure 7/7. AAI atrial paced rhythm. On the first half of the tracing, intrinsic rhythm at a normal heart rate is observable with slightly prolonged conduction, then the compensatory pause of a ventricular premature beat induces atrial pacing and, ultimately, the last beat is again the result of intrinsic atrial activity. (Please note that the QRS complexes are narrow because the ventricles are activated through the normal cardiac conduction system throughout the time.) (Sinus and atrial paced rhythm, 60 bpm, normal QRS axis, prolonged AV conduction time, normal ventricular conduction and repolarization, a VPB.)

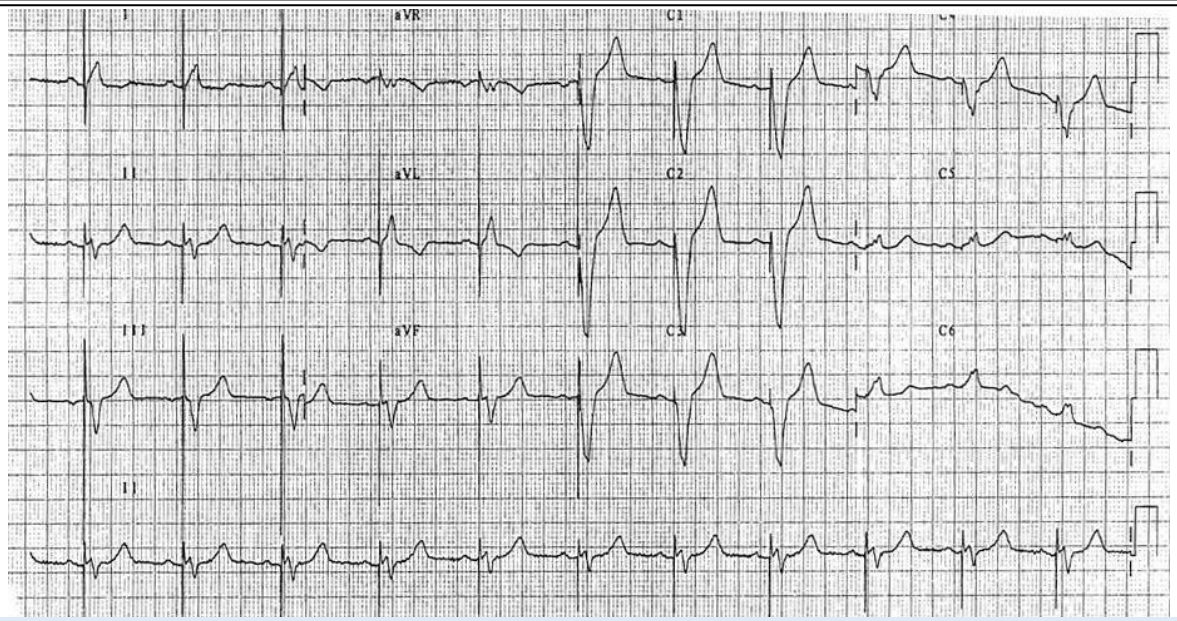


Figure 7/8. VDD ventricular paced rhythm. It is obvious that ventricular pacing is synchronized to the P waves. (The QRS complexes are wide because the ventricles are activated by the pacemaker.) (Sinus rhythm, 70 bpm, good ventricular tracking, secondary repolarization abnormalities.)

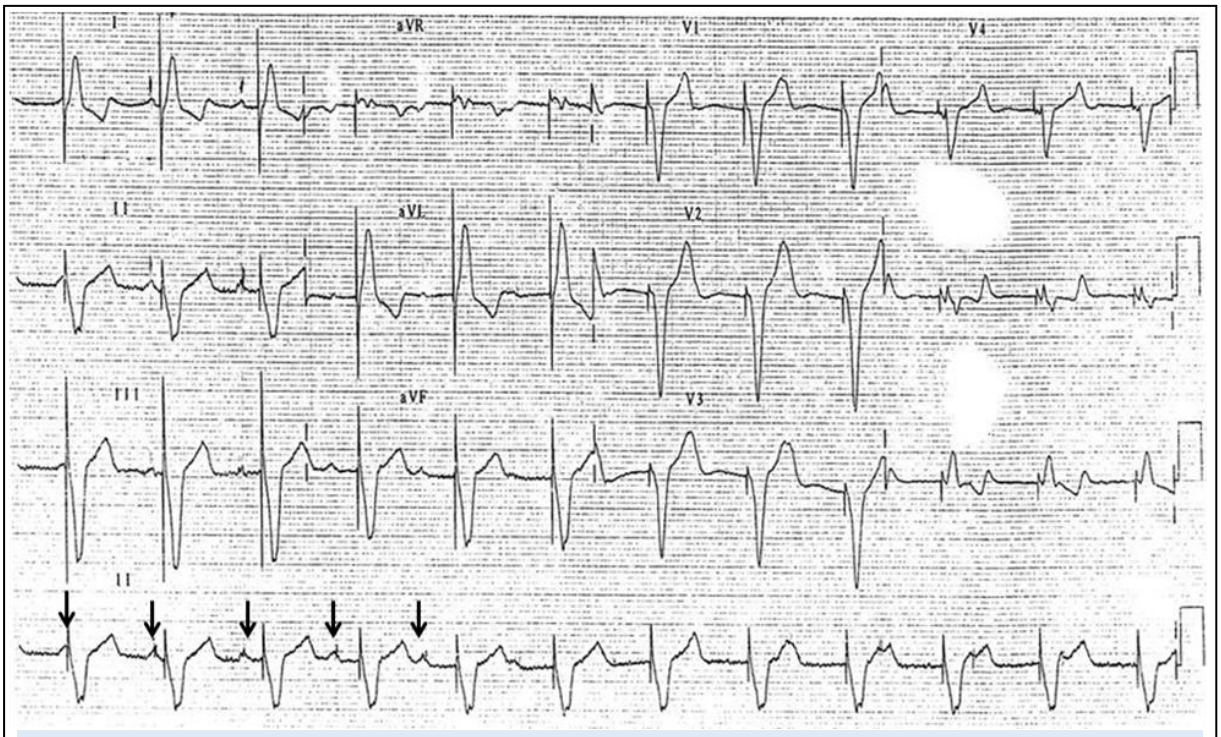


Figure 7/9.

VVI ventricular paced rhythm. (The QRS complexes are wide because the ventricles are activated by the pacemaker.) It can be observed on the lower rhythm strip that the occurrance of P waves (arrows) and pacemaker spikes is not synchronized to each other. (80 bpm, ventricular paced rhythm, left axis deviation, secondary repolarization abnormalities, sinus node activity is the underlying basic rhythm.)

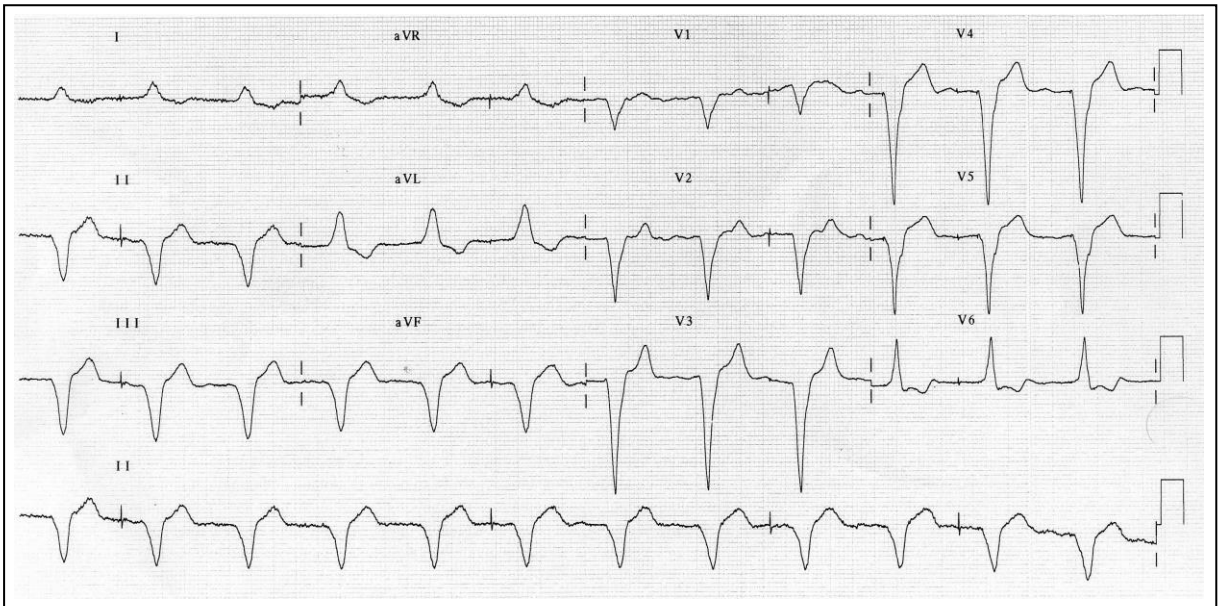


Figure 7/10.

DDD pacemaker. Since the patient has atrial fibrillation, atrial pacing is therefore inefficient (atrial undersensing). Ventricular pacing is realized via a bipolar lead, due to which pacing spikes are very small, however, still visible in leads V4-5. (Atrial fibrillation, atrial undersensing, left axis deviation, 70 bpm, ventricular paced rhythm, secondary repolarization abnormalities.)

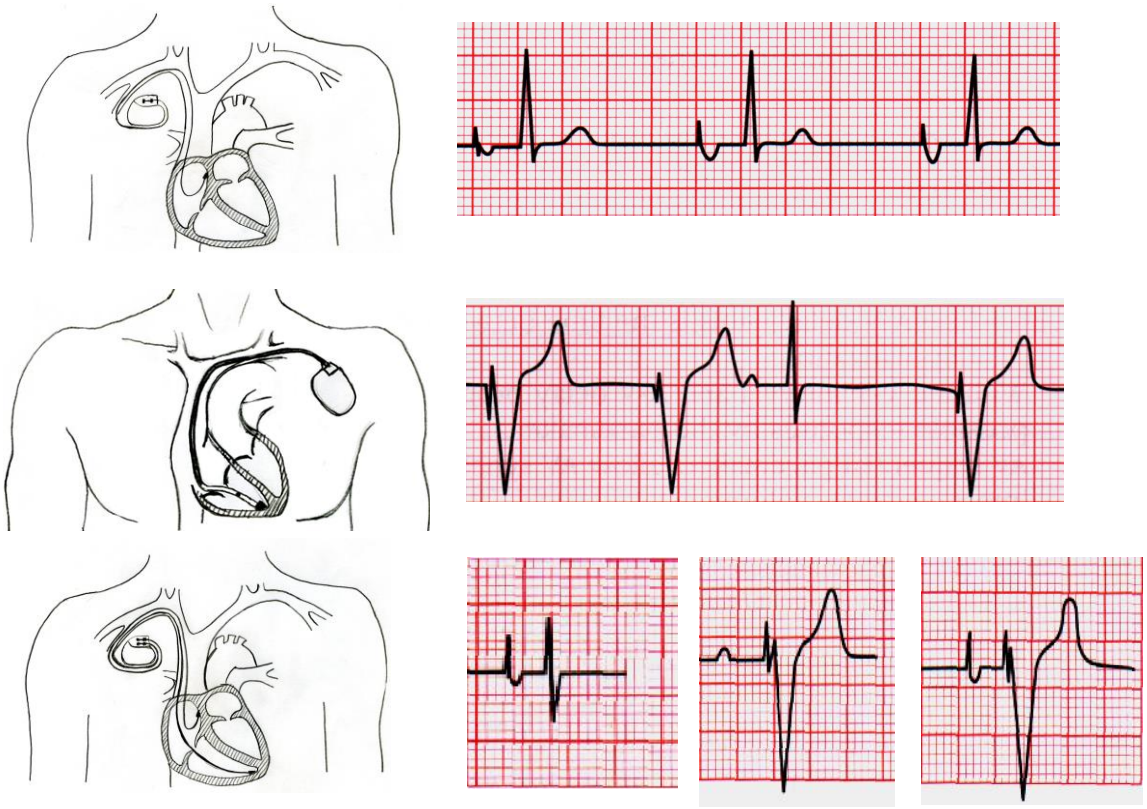


Figure 7/11. Basic types of pacemakers and their principle of operation. For AAI pacemakers, there is a spike before each P wave, so normal atrial (sinus) activity is missing in this case. For VVI pacemakers, the third QRS complex is a conducted beat sensed by the pacemaker and the pacemaker stops its operation temporarily. For DDD pacemakers, it may occur that only atrial or ventricular pacing is observable, however, dual-chamber pacing may also take place depending on which cardiac chamber has a delayed or missing activation. For VDD pacemakers (not shown by this figure), there is no atrial pacing, only ventricular pacing synchronized to P waves (consistent with the P-spike-QRS pattern on the middle ECG image illustrating the operation of a DDD pacemaker).

7.2. Monitoring pacemaker function

The most important tool is 12-lead ECG, which, if recorded during pacing by the pacemaker, carries much information; however, if the pacemaker is only in the demand mode, 12-lead ECG alone does not provide information on what would happen if operation of the pacemaker became necessary. In such cases, placement of a magnet onto the pulse generator may be of help, which suspends the demand mode and starts to pace with a preset fixed rate (V00). Operation of the pacemaker can also be promoted by carotid sinus massage, which activates functioning of the device by lowering the heart rate. If pacemaker malfunction is suspected, it may be recommended to perform 24-hour Holter monitoring, chest radiography or fluoroscopy to check lead position or detect lead fracture. In addition to the above, all settings of the pacemaker can be interrogated with a special device, moreover, battery voltage of the pulse generator can be monitored and programming of the device is also possible.

Normal pacemaker function that is considered abnormal:

1. Pseudofusion beat: This phenomenon develops when intrinsic cycle length of the heart and that of the pacemaker are very close to each other, e.g. the preset minimal heart rate is 60 bpm and the intrinsic heart rate is also around 60 bpm. At this time, it may occur that the impulse depolarizing the ventricles is entirely supraventricular and is originating from the intrinsic structures of the heart; however, sensing of the pacemaker is delayed by a few milliseconds, therefore it delivers a spike but does not take part in the depolarization of the ventricles.
2. Fusion beat: It represents dual activation that is a certain portion of the ventricles is activated from the intrinsic supraventricular structures, while the cardiac apex is activated by the pacemaker lead, later the two depolarization wavefronts meet in the middle and generate a fusion beat. As a consequence, the shape of the beat reminds both to the intrinsic QRS complexes and to those generated by the pacemaker.
3. Rate hysteresis: It is separation of the programmed lower rate and the pacing rate, e.g. the pacemaker is switched on only below a heart rate of 40 bpm, but it paces the heart at a rate of 60 bpm at this time. This may play a role in the consideration that in periods at night with physiological bradycardia, it is not the pacemaker that should stimulate the heart but stimulation should be realized via the normal cardiac conduction system. By this function, not only unnecessary pacing by the pacemaker can be avoided, but battery life will also be increased.

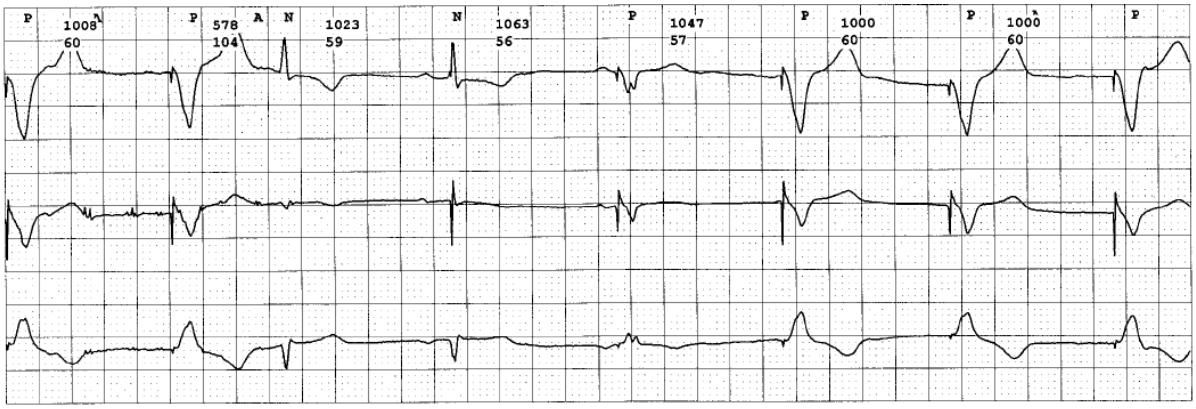


Figure 7/12.

The 1st and 2nd beat results from ventricular pacing, the 3rd beat is an intrinsic beat, the 4th beat is the result of concurrent appearance of an intrinsic beat and a pacing artifact (spike) by the pacemaker (pseudofusion beat), the 5th beat is a fusion beat and the 6th to 8th beats are again ventricular paced beats.

True pacemaker malfunction:

1. Ineffective pacing:
   - exit block (failure to capture): a spike is not followed by activation of cardiac structures, the causes of which may include lead fracture, battery depletion or increased stimulation threshold at the electrode site (e.g. due to scarring, fibrosis, ischemia, hyperkalemia or antiarrhythmic medication use);
   - absence of pacing spikes (output failure);
   - alteration in the programmed pacing rate.

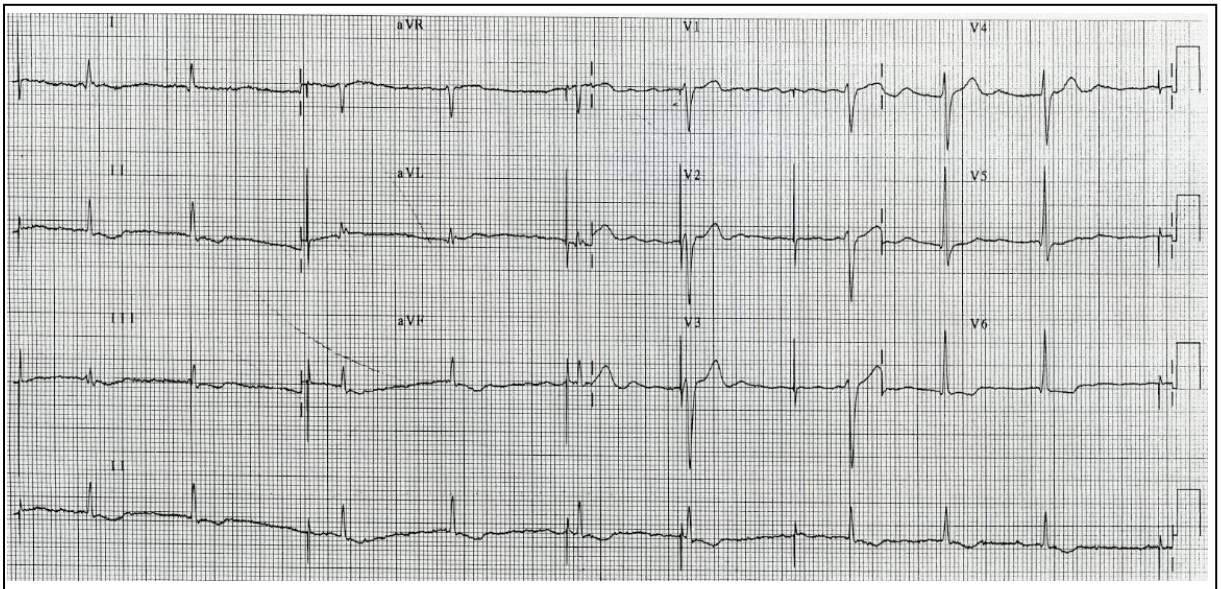


Figure 7/13.

Exit block. Please note the ineffective pacing spikes on the rhythm strip. (Atrial fibrillation at a normal ventricular rate, normal QRS axis, R wave reduction in leads V1-4, downsloping ST segment depression of 1 mm and negative T waves in leads II, III, aVF and V6, ineffective pacing spikes.)

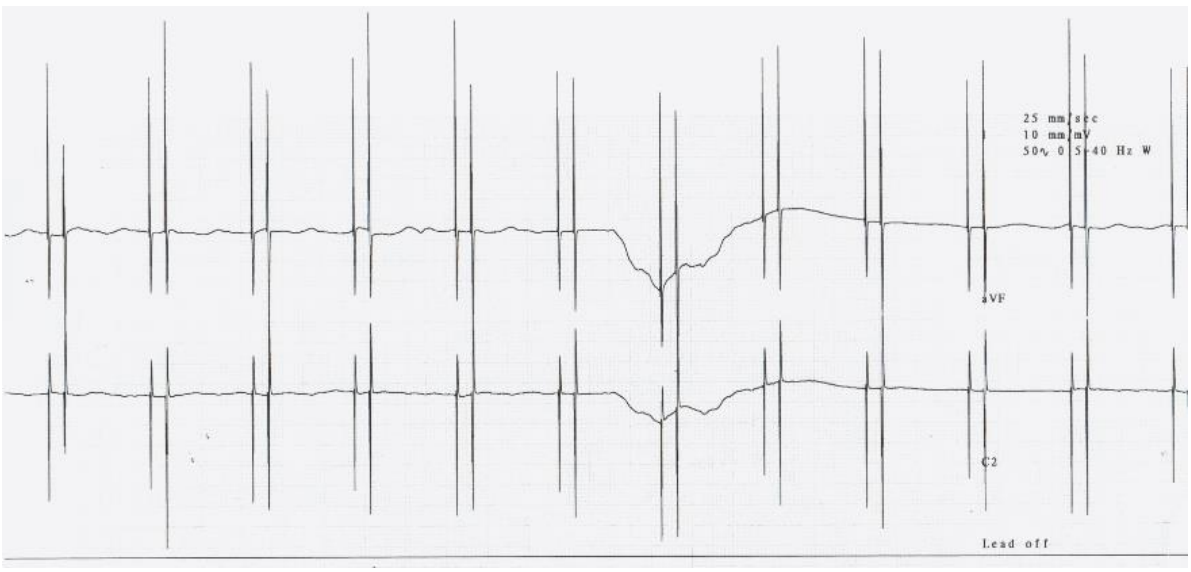


Figure 7/14.

Ineffective pacing in a patient with hyperkalemia and asystolia.

2. Problems with sensing:

   - *Undersensing* (undersensing or lack of sensing): a spike occurring after normal intrinsic electrical activity of the heart. The device fails to sense normal cardiac electrical activity. Causes may include lead dislodgement, lead conductor fracture, pulse generator failure, electrolyte disturbances, problems with settings, etc.

- *Oversensing:* the device senses such findings as normal electrical activity that are electrical signals originating from outside the heart (myopotentials from skeletal muscles), or which are arising from the heart but they are not depolarization waves (e.g. T wave oversensing), and this blocks normal pacemaker function, although that would be just necessary. Causes may include devices generating strong electric fields (high voltage, arc welding, electrocautery, etc.), T wave sensing, etc. Oversensing may generate asystolia for VVI pacemakers and arrhythmias for dual-chamber devices.

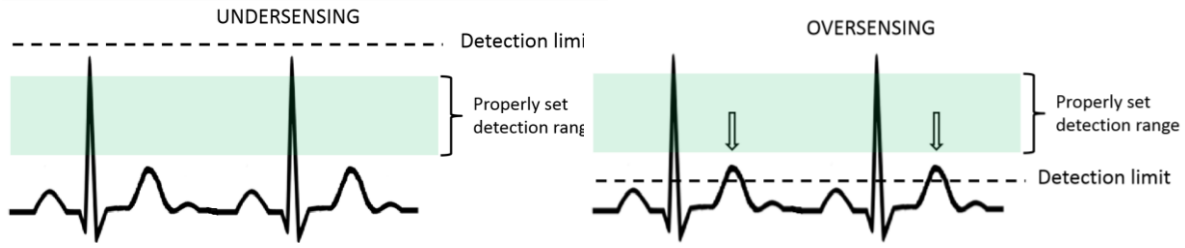


Figure 7/15.

An explanation for undersensing and oversensing. By setting the sensing threshold of the device, false sensing can be terminated.

It is very important to observe some rules in patients living with a pacemaker, in order to avoid erroneous operation or damage of the device. Circumstances which do not interfere with the operation of pacemakers include physical activity, carrying or lifting a load, housework or gardening activities two months after the implantation, transcutaneous electrical nerve stimulation (TENS), cobalt-60 radiation therapy, linear accelerator, ultrasonography, radiography, computed tomography (CT), metal detector gate (the latter one may induce only a very short interference, since the gate is passed within a moment and the gate indicates presence of the device). Circumstances which (may) interfere with the operation of pacemakers include cell phones (if used within 20 cm, this is why it is advisable to use the contralateral ear), high-voltage electric field (arc welding, induction coils, furnaces), MRI is prohibited, electrocautery (the device should be switched off if possible or set to V00; cautherization should be performed at least 15 cm from the pulse generator and the indifferent electrode (grounding) of the electrocauther should be placed in a way that the electric current should not pass through the pacemaker), direct radiation, betatron, short waves and microwaves (a microwave oven does not interfere with them) and, malfunction may occur in close proximity to radars. ESWL may be performed, but at least 15 cm from the device and the pacemaker must be set to VVI (or V00) mode. Defibrillation may be performed, but the paddles should not be placed onto the pulse generator, moreover, electroshock therapy should be avoided.

3. *Pacemaker syndrome:* This condition occurs for VVI pacemakers, during which atrial and ventricular contractions are not synchronized in patients with sinus rhythm and it sometimes results in simultaneous activation of the atria and ventricles and both cardiac chambers contract concurrently. The condition may occur spontaneously when atrial and paced ventricular rate are close to each other, or as a result of activation of the atria due to retrograde atrial conduction of ventricular paced beats. Thus, atrial contraction occurs while atrioventricular valves are closed, resulting in retrograde atrial emptying, which will be perceived by the patient as an unpleasant sensation (palpitations,

distension in the neck region) and cardiac output will also decrease. In order to avoid pacemaker syndrome, VVI pacemakers are now implanted only in patients with atrial fibrillation.

4. *Pacemaker-mediated tachycardia:* It may occur for AV sequential pacemakers that ventricular impulses are conducted in a retrograde fashion after normal atrioventricular activation, which results in atrial and consecutive ventricular depolarization. This type of arrhythmia forms an endless loop (i.e. a reentrant circuit.)

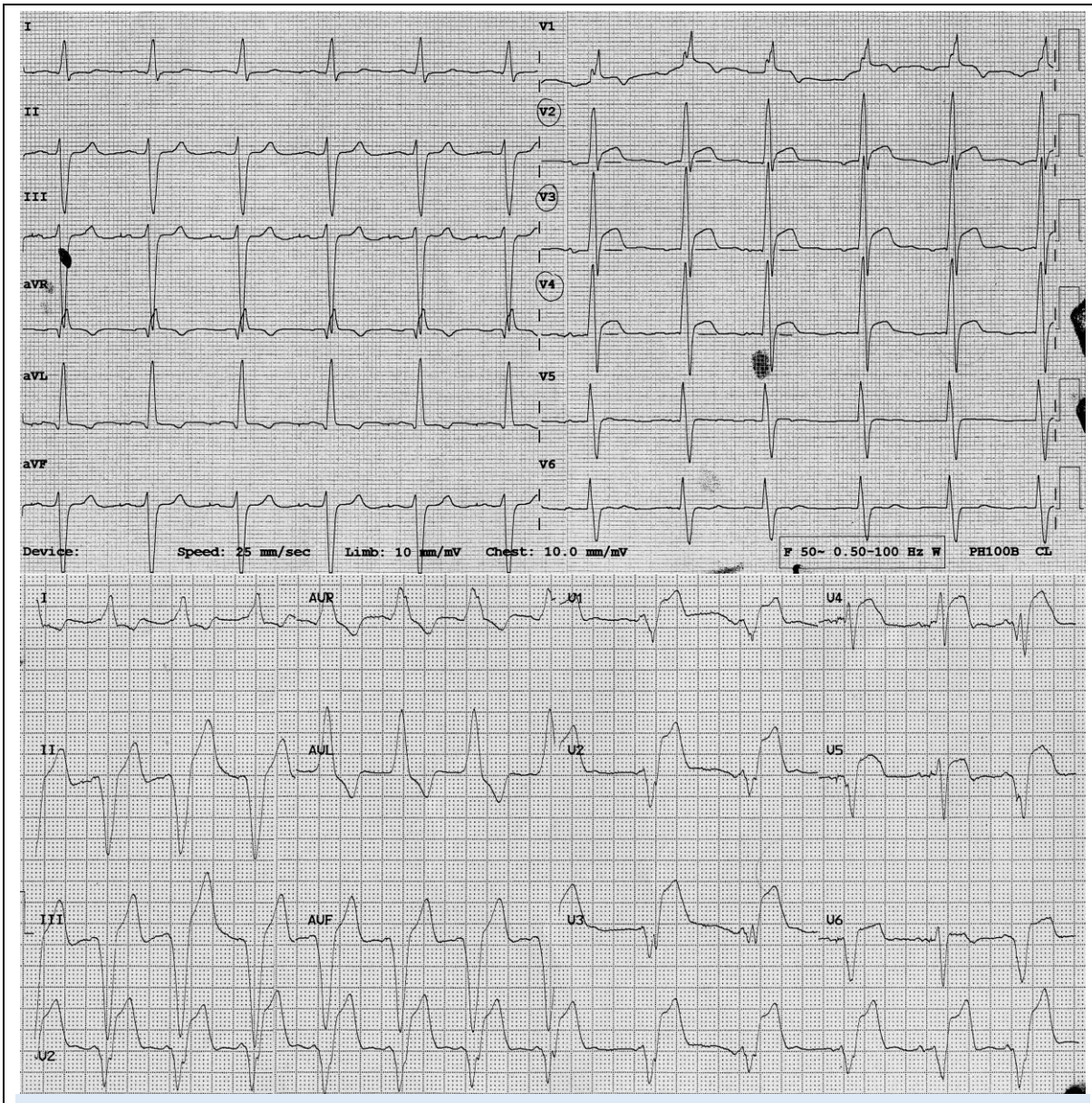


Figure 7/16.

ECG. Only atrial pacing is visible on the upper ECG tracing and ventricular conduction is realized with a left anterior fascicular block and right bundle branch block pattern, so dome-shaped concordant ST segment elevation is clearly visible in leads V1-5. Intrinsic atrial activity can be seen on the first half of the lower ECG tracing, while atrial pacing followed by ventricular paced rhythm is observable on its second half, which has a left bundle branch block pattern due to right ventricular pacing; however, the presence of giant precordial ST segment elevation is obvious even along with this ECG pattern and the Cabrera's sign (slowing of impulse conduction caused by necrosis) is already detectable on the upslope of the QRS complexes in leads V2-3.

How to recognize myocardial infarction in patients with a pacemaker:

1. Acute myocardial infarction: Since right ventricular pacing is associated with a left bundle branch block pattern, therefore we refer to the Sgarbossa criteria if one wishes to recognize myocardial infarction; these criteria were described in detail in the chapter on ischemia.
2. Searching for the signs of an old myocardial infarction is often a more difficult task, and during combined assessment of several signs, one can manage to explore in only 3/4 of all cases that the patient had a myocardial infarction. The majority of these signs is notching of the QRS complex due to the slowing of impulse conduction caused by the infarct scar, while the rest of them are based on a search for Q waves occurring in the lateral leads. QS complexes presenting in leads II, III and aVF during pacing is a normal finding, the occurrance of which is less common in LBBB. For biventricular pacing, searching for the signs of an old myocardial infarction is an even more difficult task, since slowing of the impulse conduction caused by the infarct scar is nearly impossible to be detected on the surface ECG in the presence of biventricular pacing.

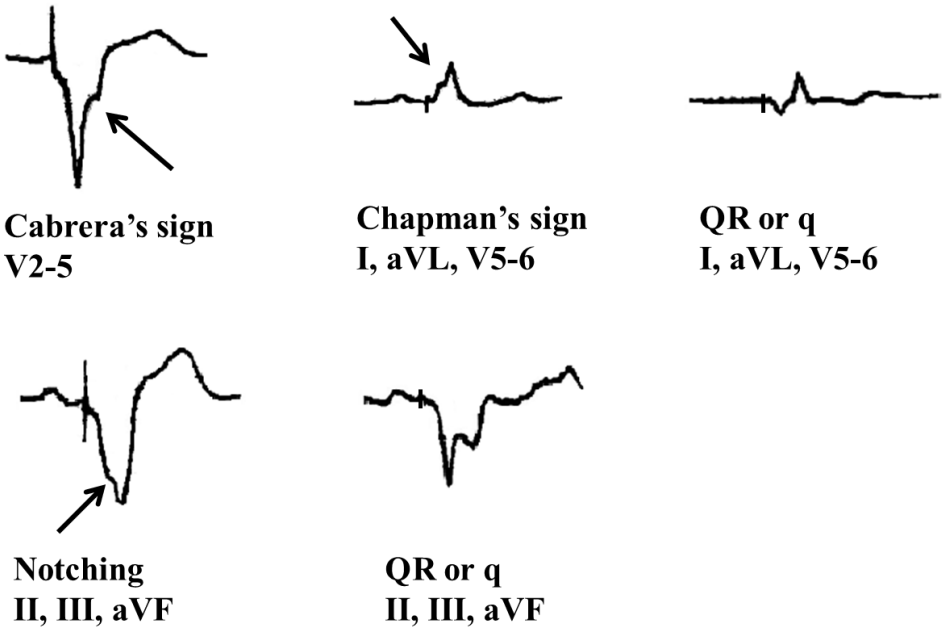


Figure 7/17.

Cabrera's sign and Chapman's sign are notching of the upslope of the QRS complex in leads V2-5 and in the lateral leads, respectively. Q waves in the lateral and inferior leads and notching of the downslope of the QRS complexes in the inferior leads may also be consistent with infarct scar.

A special form of pacemakers is a biventricular pacemaker, which is one of the most important tools in the treatment of patients with heart failure accompanied by left bundle branch block. QRS complexes are also wide during biventricular pacing, however, fusion of the depolarization wavefronts and cessation of the delay of contraction of the lateral wall significantly improves synchronous contraction and mechanical function of the ventricular walls in patients with heart failure and concomitant LBBB. Left ventricular mechanical delay and dyssynchrony caused by LBBB leads to a significant deficit in cardiac output, the synchronization of which (CRT= cardiac resynchronisation therapy) sometimes results in dramatic improvement and often initiates reverse remodelling. In the setting of biventricular

pacing, one of the pacemaker leads is situated in its usual position in the right ventricle, while the other lead is introduced through the coronary sinus into one of the posterolateral branches, which is pacing the left ventricle from the epicardial surface. Accordingly, concurrent left ventricular pacing can be recognized based on signs on the surface ECG, because the dominant deflection of the QRS complex will be negative in leads I and aVL as well as significant positive deflection of the QRS can be observed for this type of pacing. In case of decreased left ventricular function, there is a high risk of malignant arrhythmias (ventricular tachycardia, ventricular fibrillation), so biventricular pacemakers (CRT-P) are frequently combined with a defibrillator (ICD = implantable cardioverter defibrillator), abbreviated as CRT-D.

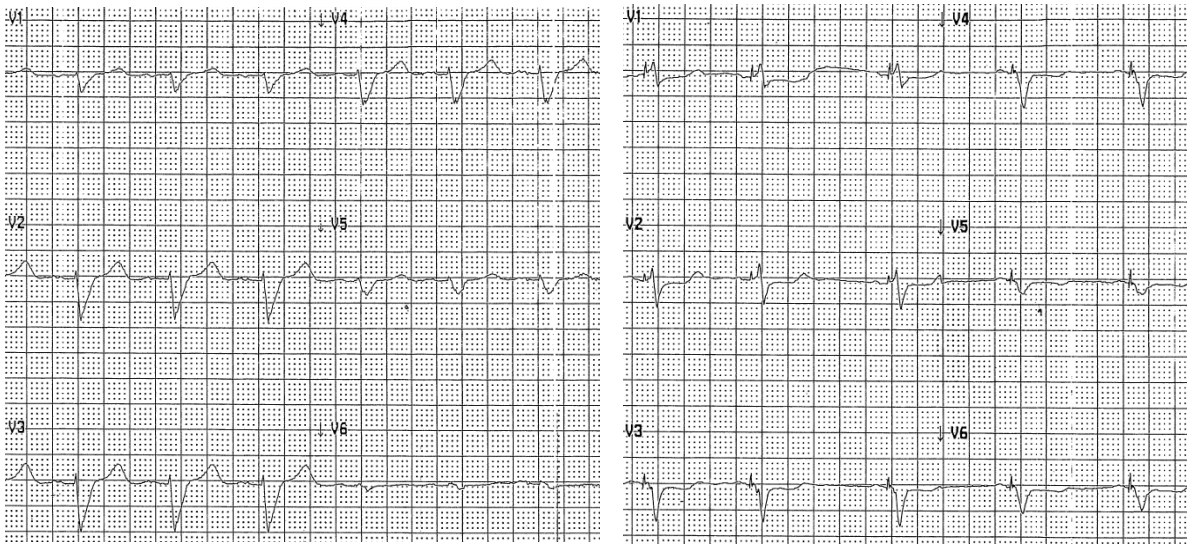


Figure 7/18.

In the above case, intraventricular conduction disturbance with a left bundle branch block pattern occurring in postinfarction ischemic cardiomyopathy can be seen on the left-sided tracing. In response to biventricular pacing (right-sided tracing), not only the QRS complexes became apparently narrower, but the patient's complaints also showed marked regression.

FACTS THAT YOU MUST KNOW!

1. Merely based on the fact that the patient has a pacemaker, it cannot always be identified from the ECG if the pacemaker is only in the demand mode and intrinsic activity of the heart is normal currently.
2. Pacing of the ventricles results in widening of the QRS complexes, which is accompanied by secondary ST segment and T wave abnormalities and these are not signs of ischemia or infarction. In the setting of atrial pacing, if atrioventricular and ventricular conduction is normal, QRS complexes remain narrow.
3. For bipolar pacing, pacemaker spikes can hardly be seen sometimes and one should search them in leads V3-4 in such cases.
4. If a ventricular pacing spike is not followed by a QRS complex or long (2 sec) pauses are visible, one should think of the presence of pacemaker malfunction.
5. To avoid the development of pacemaker syndrome and preserve atrioventricular synchrony, an AV sequential pacemaker (VDD or DDD) is implanted in patients with sinus rhythm.

CHAPTER 8

MECHANISM OF CARDIAC ARRHYTHMIAS AND DISORDERS OF IMPULSE CONDUCTION AS WELL AS REFRACTORINESS

We are going to deal with the development of cardiac arrhythmias and their electrophysiological explanation only briefly and in passing, in a non-exhaustive manner. Basically, three mechanisms inducing arrhythmias can be differentiated:

8.1. Abnormal automaticity

The mechanism when a subordinate center takes over the pacemaker role is called automaticity. It is referred to as passive heterotopic rhythm if it occurs because of a failure in the operation of the superior center and the latter one is not able to generate action potentials. At this time, the subordinate center takes over control in a passive manner, the result of which is referred to as escape beats and escape rhythm. For details, see the later section on passive heterotopic rhythms, under AV blocks. If the impulse generation of the subordinate center is faster than that of the superior one (Ca^{2+} overload), it is then referred to as active heterotopic rhythm. (For example, there is a decrease in the amount of ATP due to ischemia, due to which K^{ATP} channels are opening and reduced function of the $3Na^{+}/2K^{+}$ pump is observable, so depolarization occurs as a result.)

Clinical features of this type of arrhythmia:

- it is not initiated and terminated by a premature beat;
- it occurs, and is terminated, gradually;
- the occurance of the QRS complexes may be somewhat irregular;
- it it not terminated by vagal maneuvers or overdrive pacing;
- it is frequently associated with (functional) AV block;
- Examples: focal atrial tachycardias, accelerated idioventricular rhythm, particular types of ventricular tachycardia (VT) (e.g. reperfusion-induced VT.)

8.2. Reentry

It is the most frequent type of the arrhythmia mechanisms. For this type, circus movement of the electrical impulse is generated in the myocardium. The route of the circus movement may either be an anatomically defined pathway (e.g. an atrioventricular accessory pathway in WPW syndrome) or a functional pathway determined by the electrical inhomogeneity of cardiomyocytes (e.g. in case of ischemia or in an infarction scar or around that scar). A prerequisite of its development is the presence of a dual-pathway electrophysiology, through which the impulse is normally passing through simultaneously. However, if one of the pathways is blocked, it becomes possible for the impulse to turn back towards the blocked pathway and, by the time it gets to the region having been blocked previously, the repolarization of this region will have already taken place and this area is no longer in the refractory period, so it is excitable again and the impulse may be conducted

through it in a retrograde fashion, which then turns back onto the other pathway. This is how a reentry circuit is formed (its illustration is similar to that of 'a snake biting its tail').

Prerequisites:

- unidirectional block;
- functional (inhomogeneity of the repolarization or refractoriness) or anatomical dual pathway;
- different wavelength of the electrical impulse (conduction velocity, refractoriness.)

Clinical features of this type of arrhythmia:

- it is initiated and terminated by a premature beat (the coupling interval and cycle length of the tachycardia are inversely related to each other);
- paroxysmal (it presents as sudden attacks);
- regular;
- it can be terminated by increasing the refractoriness;
- Examples: atrial fibrillation, atrial flutter, AVRT, AVNRT, bundle branch reentrant ventricular tachycardia (BBRVT), ventricular tachycardia (90% of VTs), ventricular fibrillation.

8.3. Triggered activity

The impulses arriving at the end of the plateau phase of the action potential or during the fast repolarization may induce arrhythmias with a special mechanism. The two forms are early afterdepolarization (EAD) and delayed afterdepolarization (DAD).

EAD:
- It may be caused long QT, bradycardia, hypokalemia, ischemia or drug effect (it is caused by membrane potential oscillations of phase 2 and 3 of the action potential.
- Extremely long repolarization of the Purkinje and midmyocardial cells as well as prolonged action potential duration creates the possibility of its development (pause-dependent.)
- The underlying cause is reactivation of I_{CaL} (phase 2) → Ca^{2+} overload → $3Na^{+}/1Ca^{2+}$ exchanger activation → depolarization; or it may also be generated by decreased function of I_{Kr} (phase 3.)
- Examples: torsade de pointes ventricular tachycardia, rare types of atrial tachycardia.

DAD:
- It is caused by membrane potential oscillations presenting in phase 4 of the action potential.
- The underlying cause is Ca^{2+} overload (ischemia, catecholamines, digoxin, reperfusion.)
- Clinical features:
  - It is rather tachycardia that facilitates its development.
  - It may be initiated and terminated by a premature beat.
  - Examples: mainly digitalis-induced atrial tachycardias and accelerated junctional rhythm and ventricular tachycardias.

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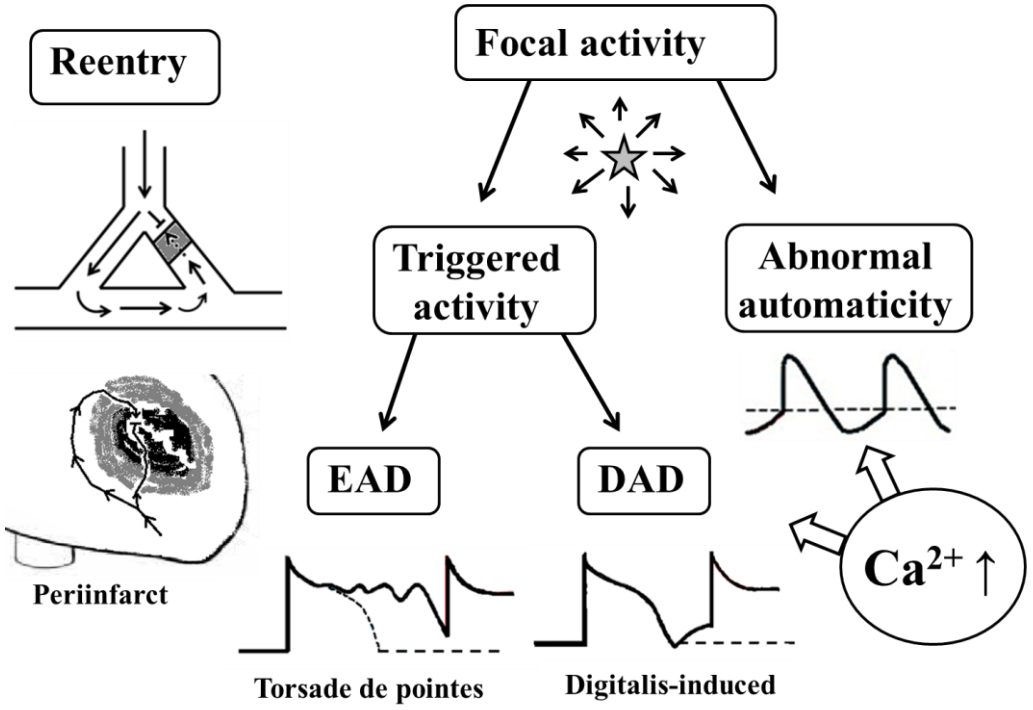


Figure 8/1.

Mechanism of cardiac arrhythmias. During reentry, there is blocked conduction on one of the pathways of a dual pathway separated either anatomically (e.g. around a scar, accessory pathway, etc.) or functionally (variable refractoriness), then the impulse conducted through the other pathway, after turning around, can already be conducted in a retrograde fashion through the previously blocked region. The arrhythmias based on focal activity are often related to Ca^{2+} overload of the cells. In abnormal automaticity, the action potential of cells is therefore becomes similar to that of nodal tissues, but it generates an impulse faster. Ca^{2+} overload in triggered activity results in an extra impulse occurring in phase 3 (EAD: early afterdepolarization) or phase 4 (DAD: delayed afterdepolarization) of the action potential.

Disorders of impulse conduction can be permanent (e.g. in case of a bundle branch block being present constantly) or transient. The mechanisms generating transient conduction disorders can be observed in phase 3 and 4 of the action potential. Accordingly, one may refer to them as phase 3 and phase 4 blocks.

Phase 3 block is a phenomenon occuring in normal hearts, the essence of which is that an impulse presenting earlier than expected cannot be conducted through a given cardiac structure (e.g. the bundle branches) since that is still in the refractory period (i.e. have not repolarized yet). Thus, the impulse will be blocked and the given beat will be conducted with a bundle branch block morphology. The impulse presenting in phase 3 of the action potential is therefore not conducted. This type can even be observed under physiological circumstances, e.g. in tachycardia-dependent bundle branch blocks, but it may also be present in a series of so-called fast-slow cycles (e.g. bigeminy or Ashman's phenomenon). The explanation for this latter phenomenon is that in case of a longer cardiac cycle, the refractory period of the action potential, due to its prolongation, will also increase and then, suddenly in case of a short cycle, the refractoriness cannot adapt to this and the impulse becomes blocked. This phenomenon is frequently seen during initiation of a supraventricular premature beat or paroxysmal supraventricular tachycardia (PSVT) (i.e. functional bundle branch block), or in tachyfibrillation (Ashman's

phenomenon). The bundle branch being blocked is usually the right bundle branch because its action potential / refractoriness is longer even normally than that of the left bundle branch.

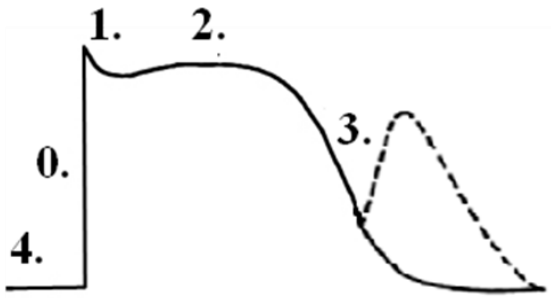


Figure 8/2.

Occurrance of a phase 3 block. The impulse (e.g. an atrial premature beat) occurring in this phase of the action potential (i.e. early) will be conducted with a functional block of the bundle branches due to refractoriness of the bundle branch not having been repolarized yet.

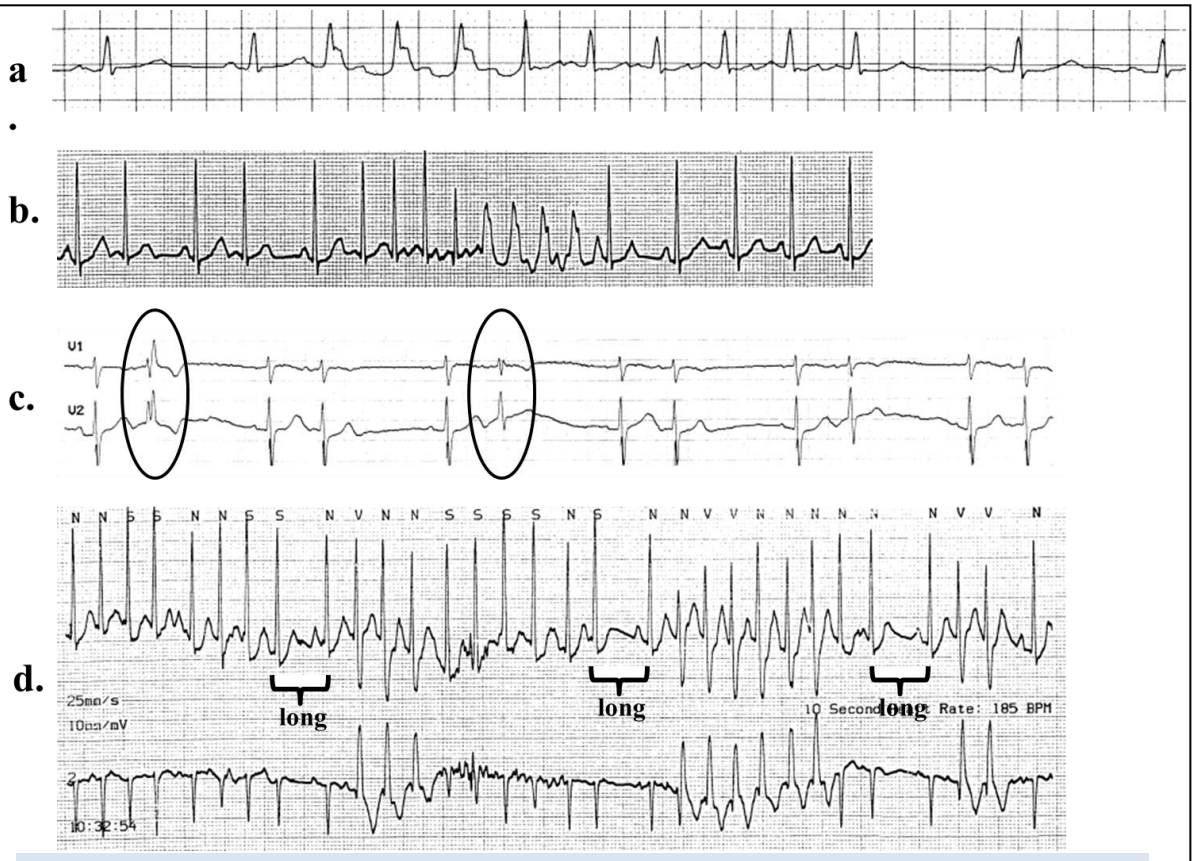


Figure 8/3. Examples for a phase 3 block. **a.)** Atrial tachycardia, the first three beats of which are conducted with a bundle branch block morphology because shortening of the coupling interval occurs too fast, and shortening of the action potential as well as accomodation of the refractoriness takes some time and the conduction normalizes afterwards. **b.)** Following frequent premature atrial complexes (PAC), a short run of atrial fibrillation occurs, the first four beats of which are conducted normally and the second four beats are conducted with a bundle branch block morphology due to the shorter coupling interval (rate-dependent bundle branch block). **c.)** Atrial bigeminy, the first PAC is conducted with a complete bundle branch block morphology, while the third PAC with an incomplete bundle branch block morphology (see circles). **d.)** Ashman's phenomenon, i.e. in tachyfibrillation, conduction will be transformed into one with a bundle branch block morphology following the presence of cardiac cycles of slightly longer duration (long-short sequence).

Phase 4 block is a more rare phenomenon and, contrary to the phase 3 block, it does not occur in normal hearts. The essence of this phenomenon is that an impulse presenting after a long pause paradoxically presents with a functional block (e.g. AV block, bundle branch block). The explanation is to be sought in the slow diastolic depolarization and the threshold potential moving toward zero (0), due to which the depolarizing impulse cannot reach the threshold potential. During the long cycle, there will be an increase in the resting potential of cells, whereas the threshold potential is moving toward zero, so the cells become unexcitable. Due to its appearance after a long cycle, this form is also called 'bradycardia-dependent block'. It is represented on the ECG as a long pause, at the beginning of which a P wave is visible and at the end of which there is a conduction pattern characteristic of a bundle branch block. A typical example is paroxysmal AV block.

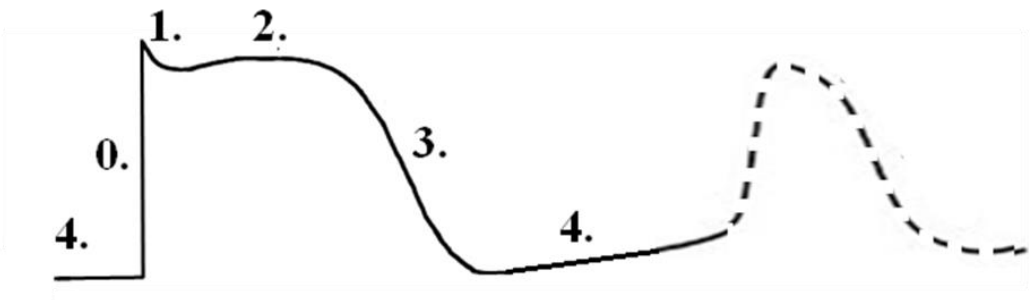


Figure 8/4.

During a phase 4 block, phase 4 of the action potential is prolonged significantly and the membrane potential of cells demonstrates a gradual decrease. This decrease may reach a level where the tissue is incapable of conducting impulses any longer and the threshold potential is also moving toward zero, due to which the tissue becomes unexcitable.

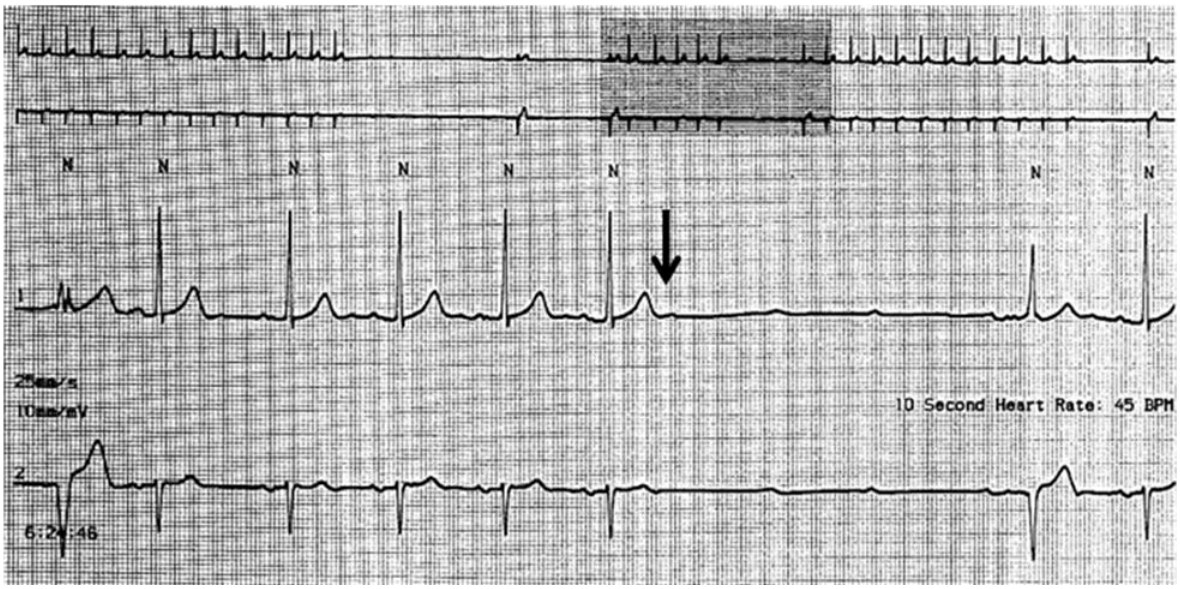


Figure 8/5. See under.

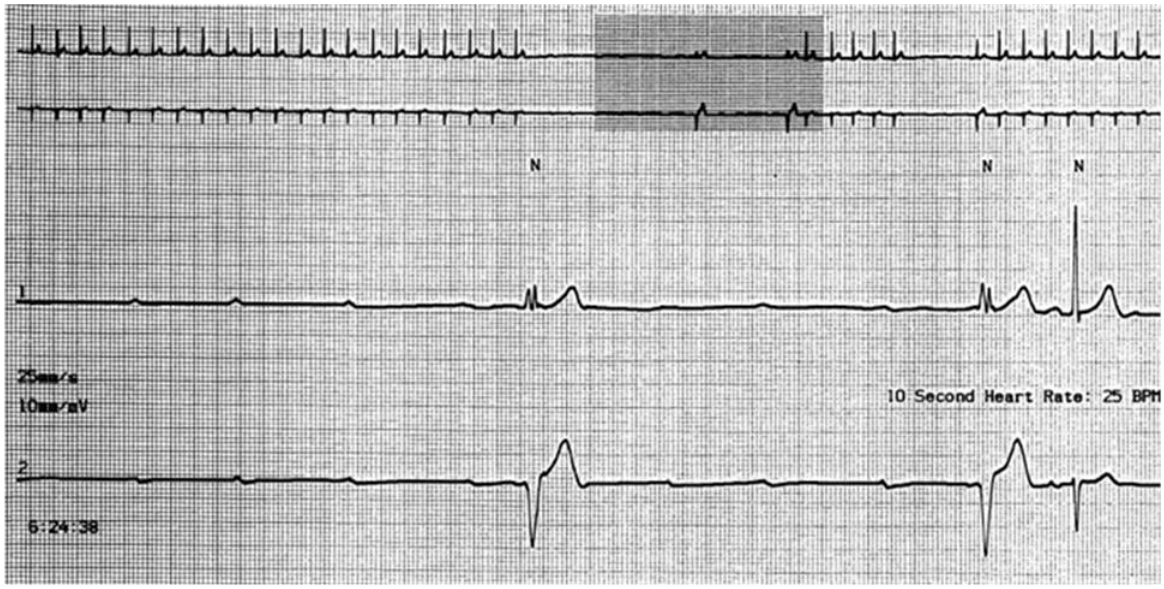
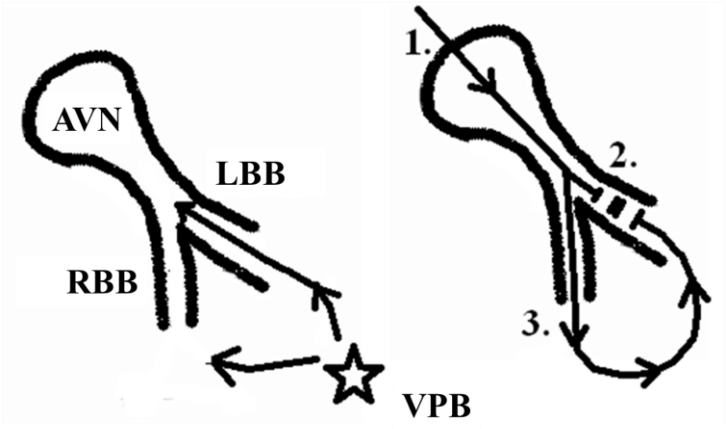


Figure 8/5. - continuation

Paroxysmal AV block is a classic example of phase 4 block. Please note that it is initiated by an atrial premature beat (arrow), then several P waves are blocked and, finally, junctional and ventricular escape beats are visible.

The functional block of bundle branches may also be induced by, beyond the above two mechanisms, the so-called concealed retrograde conduction or retrograde invasion of the individual bundle branches. You can think of it as a ventricular premature beat penetrating in a retrograde fashion into one of the bundle branches, while the supraventricular impulse is conducted down through the other bundle branch (with a bundle branch block morphology), then, after turning round, it again penetrates into the other bundle branch, thereby creating a circus movement of the impulse, which can only be interrupted by a new ventricular premature beat.

Figure 8/6.



Ventricular conduction disorder caused by retrograde invasion. On the first half of the image, you can see a ventricular premature beat (VPB) penetrating in a retrograde fashion into the left bundle branch (LBB).

On the right side of the image, the supraventricular impulse (1.) is passing through the AV node (AVN) and finds the left bundle branch in the refractory period (2.), so it can only be conducted down through the right bundle branch (RBB) (3.) and, afterwards, the conducted impulse is again penetrating into the left bundle branch, which will therefore be in the refractory period on arrival of the next beat. The circus movement of the impulse can only be terminated by another VPB.

Excitability of the heart:

It is a response of the ventricular cardiac muscle cells to a stimulus that exceeds a particular threshold value. This response means the development of a new action potential. Throughout the action potential, the responsiveness of cardiomyocytes changes. In view of this, there can be different phases of the excitability, including:

1. Absolute refractory period: It is the time interval from the onset of the action potential, during which the ventricular cardiac muscle cells are unexcitable. It is characterized by different activation phases of the Na^+ channel being responsible for the depolarization of cardiomyocytes with a fast conduction. The Na^+ channel is inactivated during phase 3 of the action potential.

2. Effective refractory period: It is a period longer than the previous one, including the absolute refractory period and the short time interval during which strong stimuli may induce depolarization locally. This may result in prolongation of the duration of the previous action potential or in the development of a new action potential, which may contribute to the development of arrhythmias.

3. Relative refractory period: An impulse stronger than the threshold potential may induce an action potential. This is different from the normal action potential and it may also have a role in the development of arrhythmias.

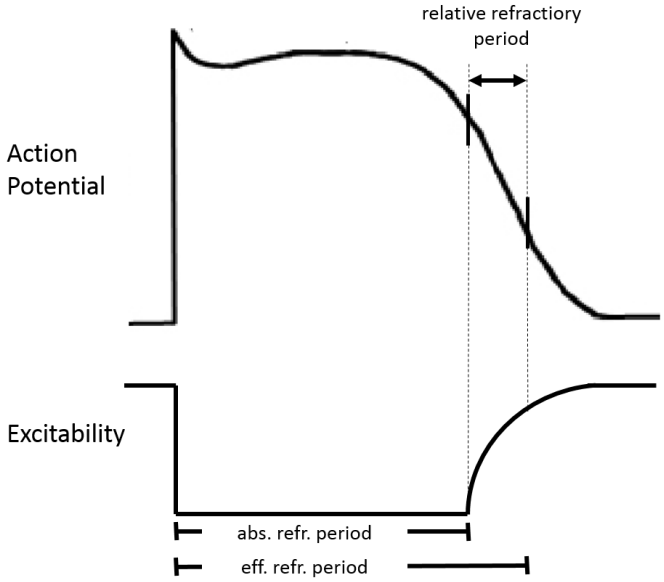


Figure 8/7.

Phases of excitability of ventricular cardiomyocytes.

Chapter 9

EFFECTS OF ELECTROLYTE DISTURBANCES AND DIGITALIS ON THE ECG TRACING

In general, the diagnosis of electrolyte disturbances is primarily a task of the laboratory. ECG abnormalities are usually observable only in marked electrolyte disturbances. These ECG abnormalities are nonspecific, but may arise as a differential diagnostic problem during the analysis of an ECG tracing. However, it is important to know that certain ECG abnormalities can only be evaluated as a sign of heart disease if ion levels have been normalized and they still persist (electrolyte disturbances - ECG signs of ischemia - ST segment depression). Individual abnormalities are listed as follows:

9.1. Hypokalemia

(causes: diuretic use, vomiting, diarrhoea, insulin, Conn's and Cushing's syndrome)

- prominent U waves;
- flat, wide and inverted T waves;
- QT prolongation;
- ST segment depression (!);
- conduction disturbances, PQ prolongation;
- ventricular and supraventricular arrhythmias (!) – torsade de pointes VT.

9.2. Hyperkalemia

(causes: kidney disease, diuretic use, ACE inhibitor use, cell lysis, potassium (K) intake)

- tall, peaked T waves with a narrow base and almost originating from the end of the wide QRS complexes, with the ST segments being short (!);
- a reduction in the R wave amplitude;
- QRS widening;
- prolongation of the PQ interval;
- ST segment depression (!);
- asystolia.

9.3. Hypocalcemia

(causes: chronic renal failure, secondary hyperparathyroidism, hypoparathyroidism, diuretic use)

- QT prolongation (!) - due to prolongation of the ST segment;
- flat or inverted T waves;
- ST segment depression (!).

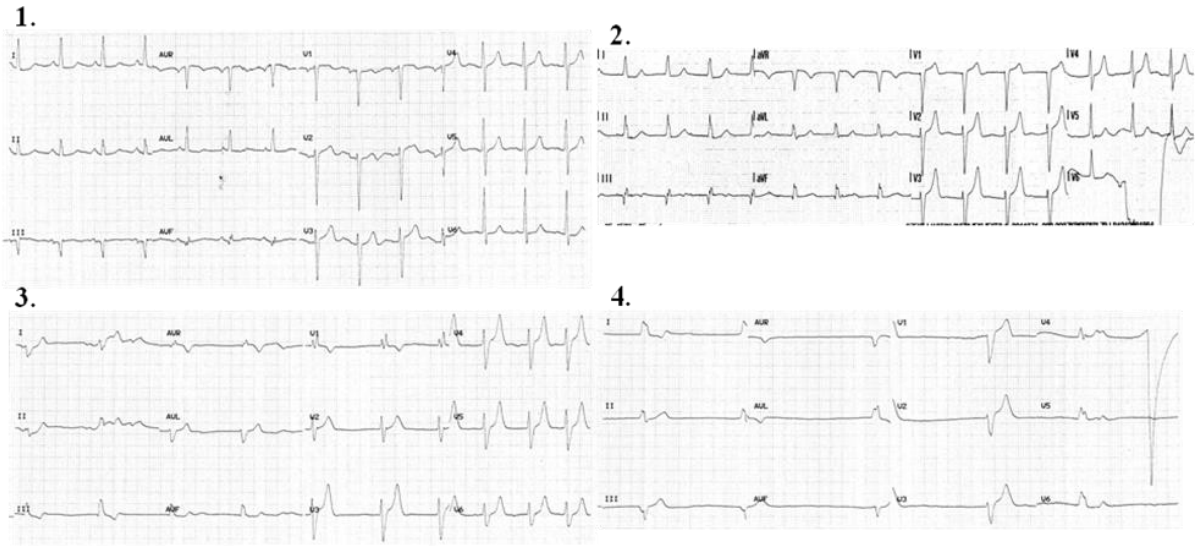


Figure 9/1.

Hyperkalemia. One can observe the ECG abnormalities from normokalemia to severe hyperkalemia in a single patient. 1. Normokalemia. 2. Moderate hyperkalemia (6.2 mmol/L) - tall, peaked T waves. 3. Moderate hyperkalemia (6.8 mmol/L) - tall, peaked T waves, disappearing P waves, bradycardia, widening of the QRS complexes. 4. Severe hyperkalemia (7.5 mmol/L) - severe bradycardia, wide QRS complexes.

9.4. Hypercalcemia

(causes: primary hyperparathyroidism, bone metastases, myeloma multiplex, thiazide diuretics)

- shortening of the QT interval(!) and, rarely, ST segment elevation;
- abnormalities similar to those seen in digitalis effect.

9.5. Digitalis effect

- scooped ST segment depression;
- T wave inversion;
- shortening of the QT interval;
- PQ prolongation, bradycardia;
- increased ectopic activity (VPBs, PACs, atrial tachycardia with high degree AV block.)

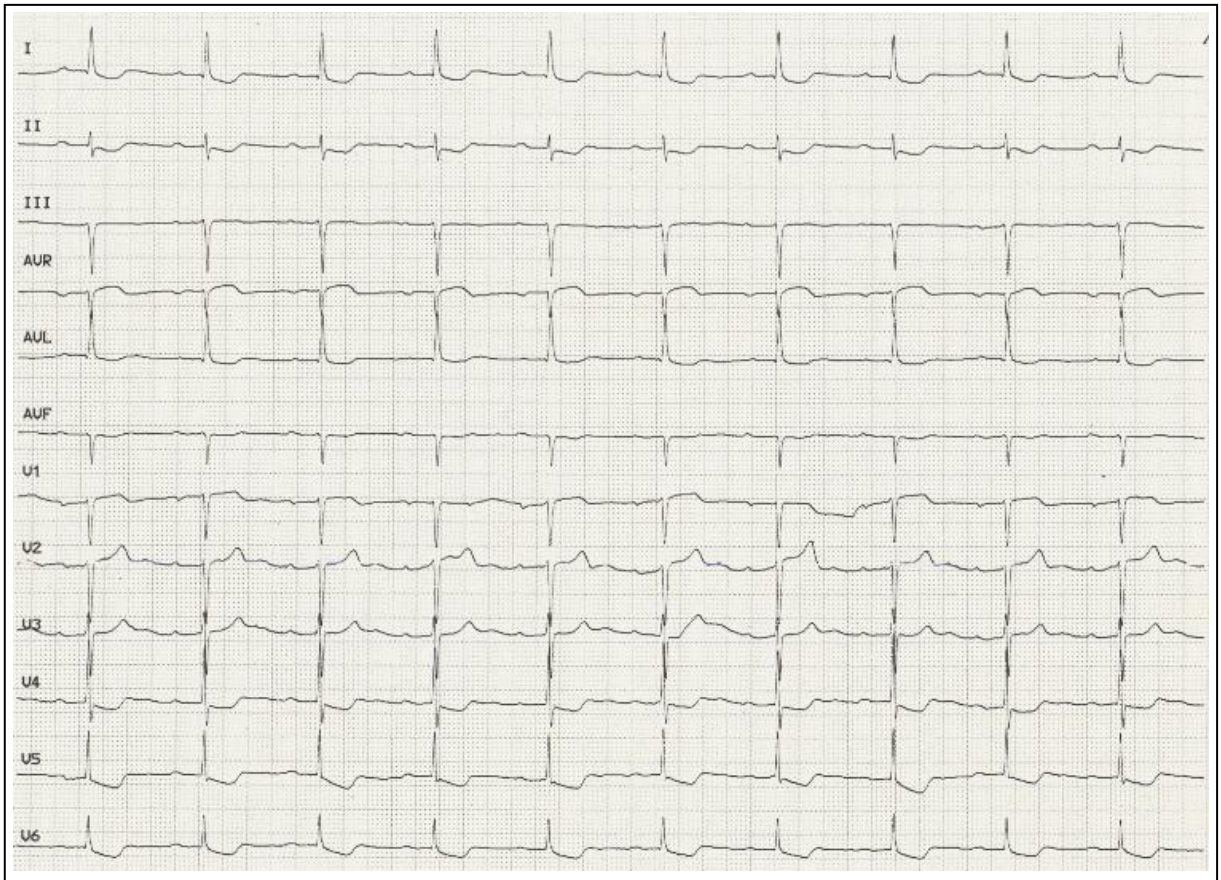


Figure 9/2.

Digitalis toxicity. In addition to ECG signs of a scar from an old inferior myocardial infarction, prolonged AV conduction, bradycardia and ST segment depression with a typical scooped pattern is visible. The patient took 8 tablets (?!) of digitoxin daily. (Sinus rhythm, 60 bpm, left axis deviation, prolonged AV conduction (260 msec), narrow QRS complexes, ST segment depression with scooping as well as biphasic T waves in leads I, II, aVL and V4-6.)

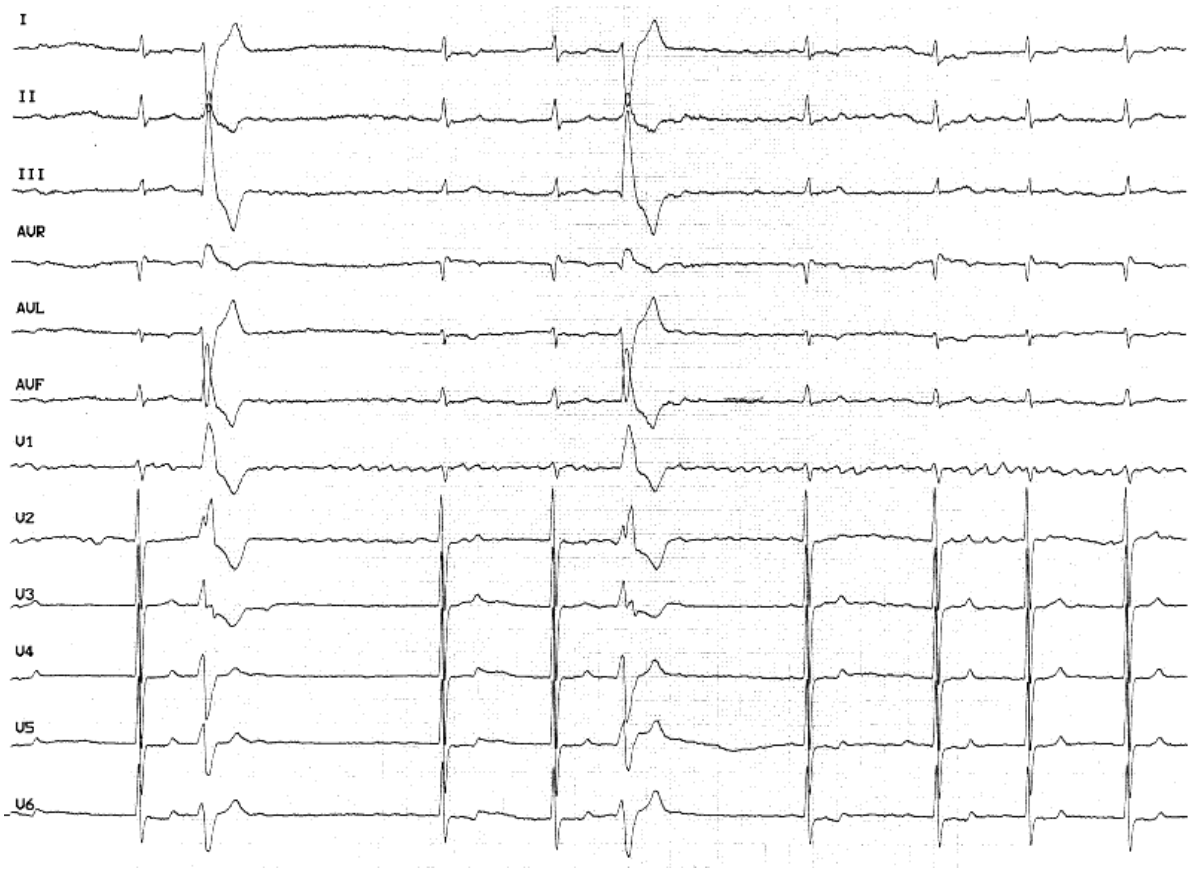


Figure 9/3.

Digitalis toxicity. Bradyfibrillation and frequent ventricular ectopic activity. (Bradyfibrillation, normal QRS axis, early transition, narrow QRS complexes, signs of trivial left ventricular strain, frequent VPBs.)

Chapter 10

Wolff-Parkinson-White (WPW)-syndrome

WPW syndrome is a congenital impulse conduction disturbance. Normally, the atria and ventricles are totally isolated electrically from each other by a fibrous ring called anulus fibrosus (cardiac skeleton). There is only one place in this isolation through which impulses can be conducted, i.e where the bundle of His passes through this ring. If isolation of the anulus fibrosus is not perfect in some areas, electrical communication between the atria and ventricles can be realized not only through the AV junction, but also via this *accessory pathway* (bundle of Kent). Thus, for an accessory pathway, supraventricular impulses can reach the ventricles from two directions. Since decremental conduction properties described for the AV junction do not, or only rarely, prevail for the bundle of Kent, an impulse can therefore be conducted here without any delay. This is the reason for the *shortening of the PQ* interval in WPW syndrome. A certain portion of the ventricles becomes depolarized through the accessory pathway, while the other portion by the normal cardiac conduction system. The portion of the ventricles depolarized by the accessory pathway is represented on the ECG as a *delta wave*, resulting in widening of the initial part of the QRS complex. This phenomenon is called *ventricular preexcitation*. No short PQ intervals and delta waves can be seen in about 30% of patients with an accessory pathway, since there is no antegrade conduction through the pathway in such cases.

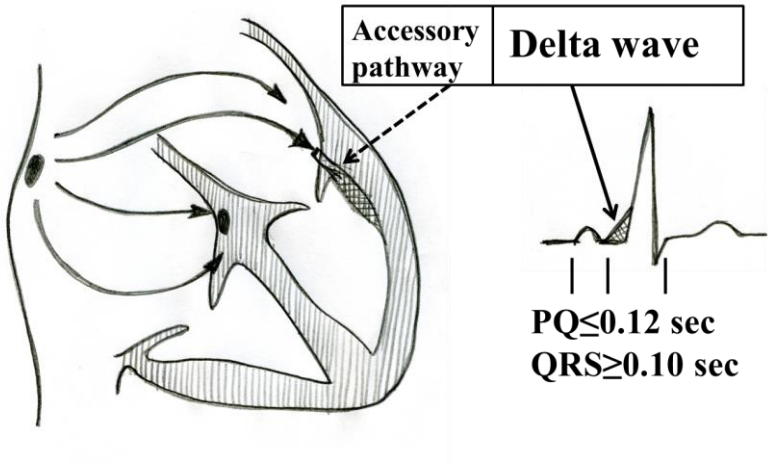


Figure 10/1.

WPW syndrome. An explanation for preexcitation, short PQ intervals and delta waves.

Accessory pathways may be present nearly all around the entire atrioventricular ring, maybe except for the left-sided anteroseptal region. Accessory pathways more often have a left-sided location and are most commonly, in more than half of the cases, situated at the ventricular free wall; in addition, they are posteroseptal in 25% of cases and less than 20% of them are situated in the right-sided fibrous ring. The latter two types are present in a combined fashion in about 10% of cases that is there are several accessory pathways at the same time. Right-sided and multiple pathways more often occur in congenital anomalies involving the right side of the heart (e.g. Ebstein's anomaly). Based on the surface ECG, the site of accessory pathways can be identified with an accuracy of about 85-90% based on the appearance of delta waves; however, one third of the pathways demonstrates no conduction in sinus rhythm, i.e. these are called concealed accessory pathways, so their existence cannot be diagnosed based on the surface ECG until characteristic tachycardia develops. An accessory

pathway localized based on the analysis of the surface ECG may assist the electrophysiologist in planning the catheter ablation procedure. Broadly speaking, it is true that if QRS complexes (delta waves) are negative in leads V1-2 and/or there are positive delta waves in leads I and aVL, the accessory pathway is right-sided. Deformation of the ECG tracing caused by a right-sided accessory pathway is similar to that resulting from left bundle branch block. For a left-sided lateral accessory pathway, QRS complexes (delta waves) are positive in leads V1-2 (with the QRS pattern reminding of incomplete right bundle branch block with monophasic R waves) and/or there are negative delta waves in leads I and aVL. Negative delta waves (Q waves) in leads III and aVF imply the presence of a posteroseptal accessory pathway, while positive delta waves in lead aVF are indicative of an anterior pathway.

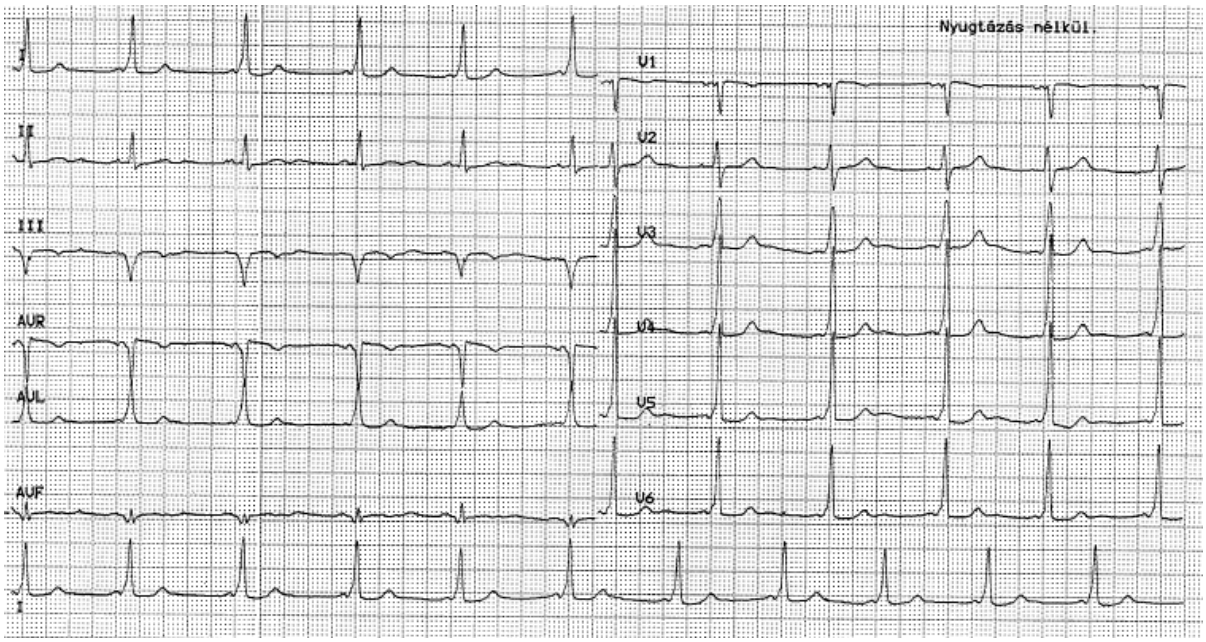


Figure 10/2.

WPW syndrome (left-sided posteroseptal accessory pathway). There is short PQ interval, moreover, positive and negative delta waves at the initial portion of the QRS complex are clearly observable in leads I, aVL, V3-6 as well as in III, aVF and V1, respectively. (Sinus rhythm, 70 bpm, normal QRS axis, short PQ interval, delta waves, pre-excited QRS complexes, secondary repolarization abnormalities.)

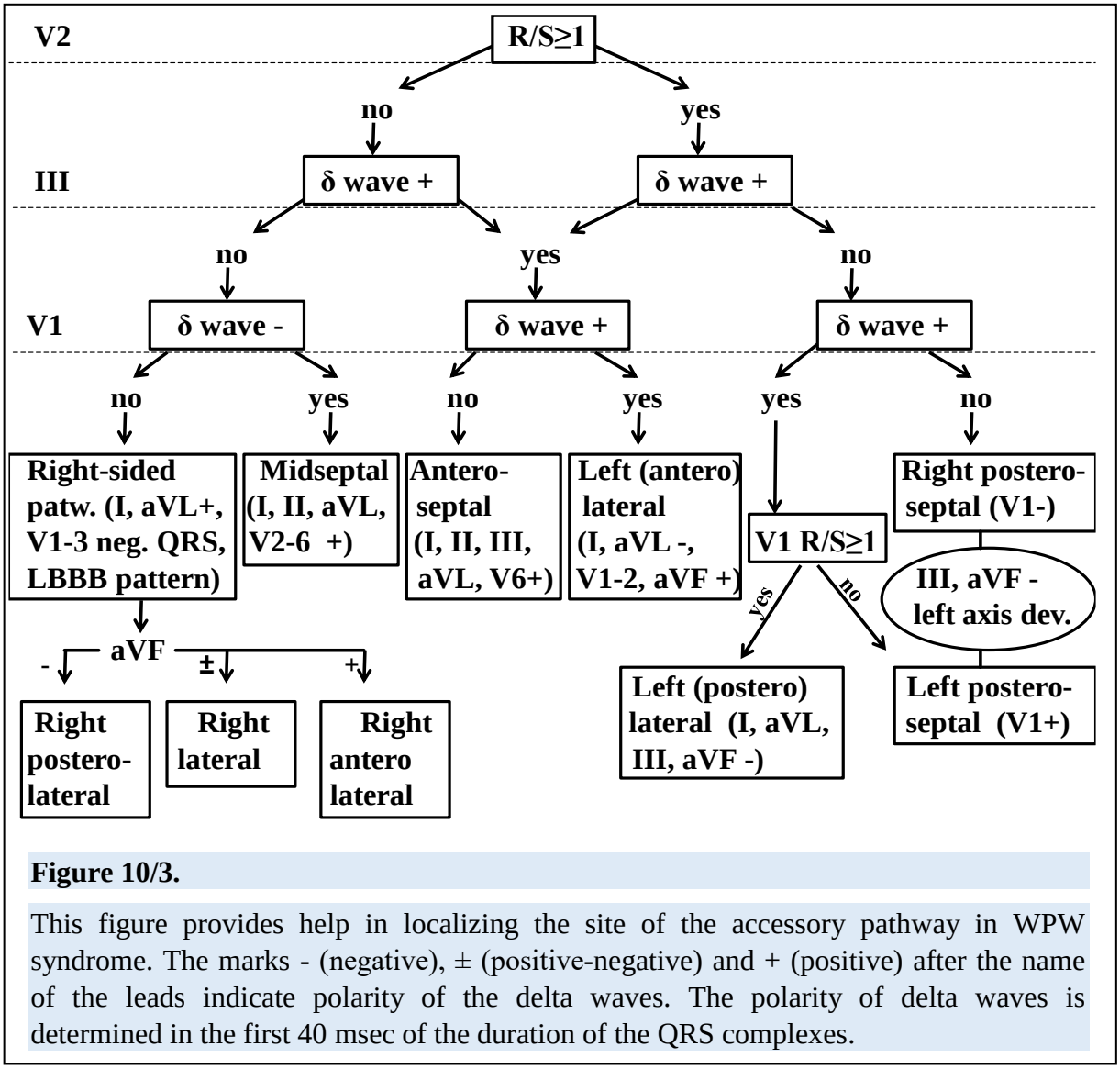


Figure 10/3.

This figure provides help in localizing the site of the accessory pathway in WPW syndrome. The marks - (negative), ± (positive-negative) and + (positive) after the name of the leads indicate polarity of the delta waves. The polarity of delta waves is determined in the first 40 msec of the duration of the QRS complexes.

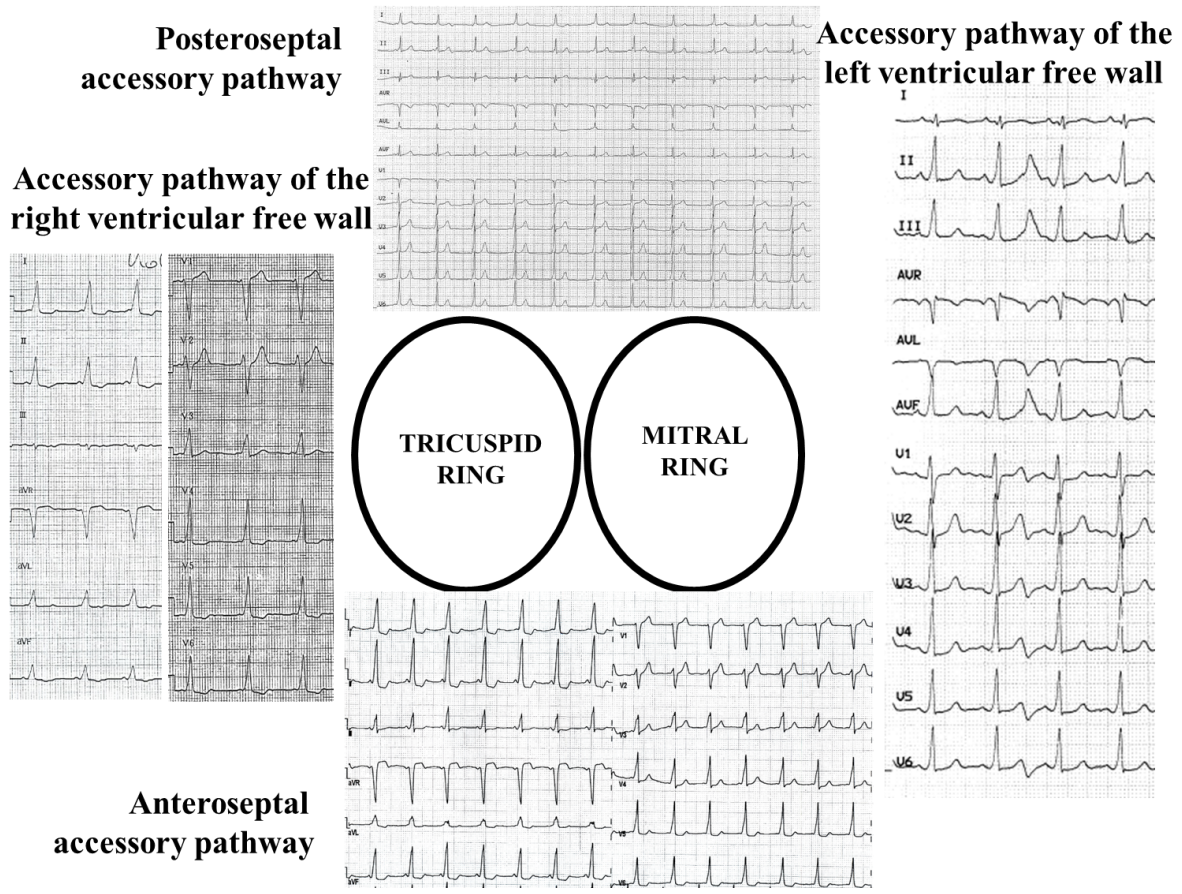


Figure 10/4.

The risk of WPW syndrome lies in the fact that an anatomically defined dual pathway (an accessory pathway plus the AV junction) favours the development of reentrant arrhythmias. Such specific forms of tachycardias typical of WPW syndrome are orthodromic and antidromic atrioventricular reentrant tachycardia (AVRT). In orthodromic AVRT, supraventricular impulses reach the ventricles through the AV junction and are then conducted back to the atria via the accessory pathway and again to the ventricles through the AV junction thereafter. This results in a narrow QRS complex tachycardia.

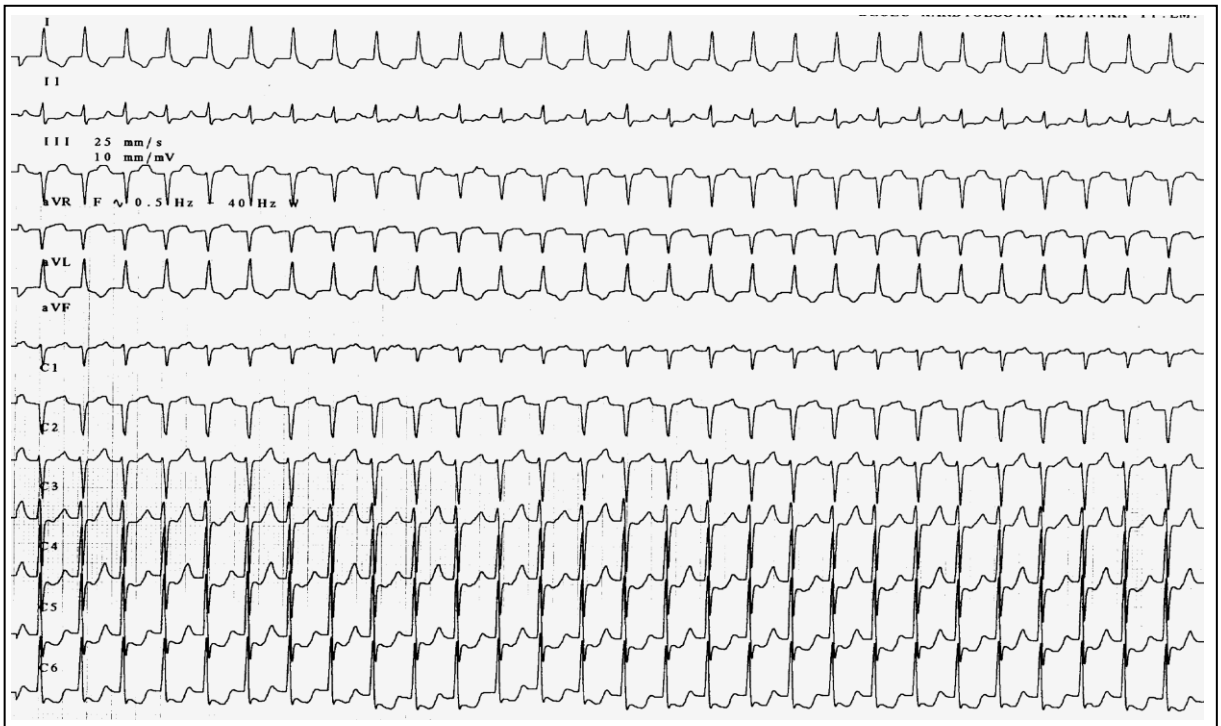


Figure 10/5.

Orthodromic AVRT. Regular narrow QRS complex tachycardia. Retrograde P waves on the ST segment are detectable in lead aVF. (Orthodromic AVRT, 160 pbm, left axis deviation, secondary repolarization abnormalities.)

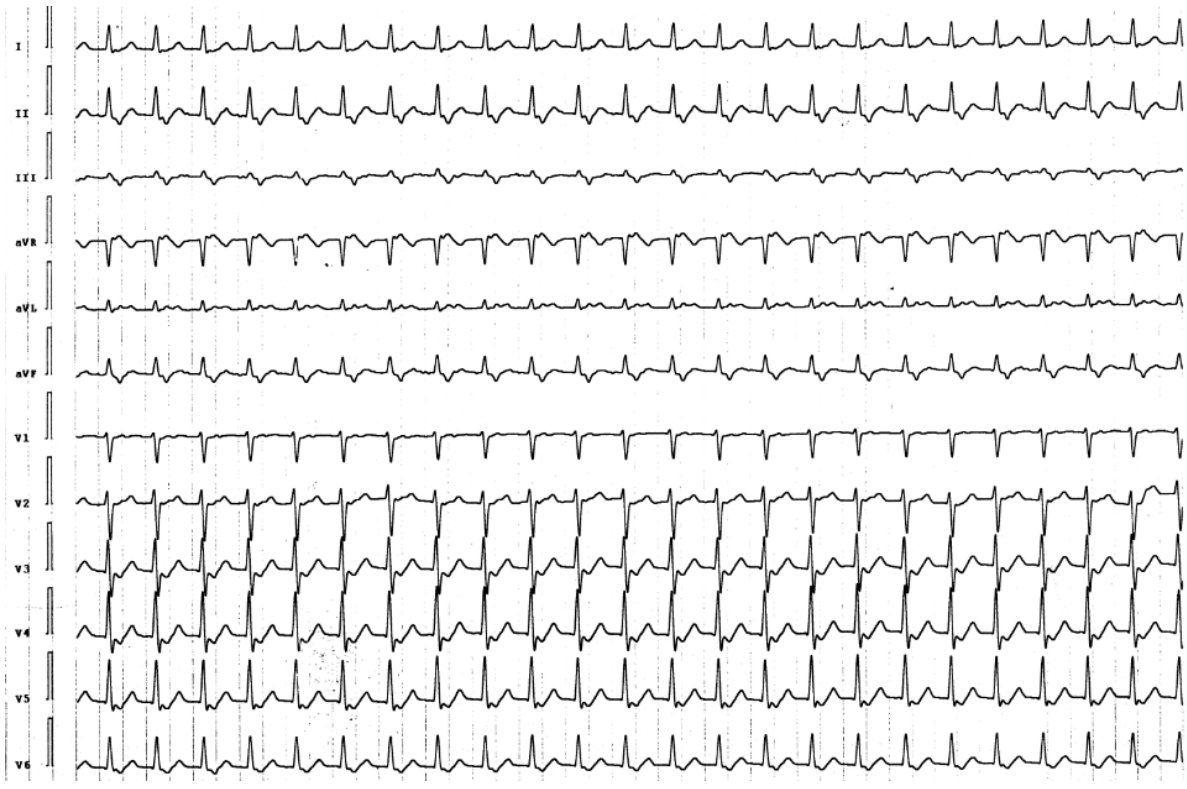


Figure 10/6. Orthodromic AVRT. Retrograde P waves are clearly visible on the ST segment (most readily in leads II, III and aVF). (Orthodromic AVRT, 155 pbm, normal QRS axis, secondary repolarization abnormalities.)

The circus movement of antidromic AVRT has just the opposite direction that is supraventricular impulses reach the ventricles through the accessory pathway and are then conducted back to the atria via the AV junction. Since the ventricles are depolarized through the accessory pathway, therefore the entire QRS complex will be pre-excited, so a wide QRS complex tachycardia will be seen. Antidromic AVRT cannot often be differentiated from ventricular tachycardia merely based on morphological signs, however, analysis of the ECG recorded in sinus rhythm is always a help (short PQ interval, delta waves.)

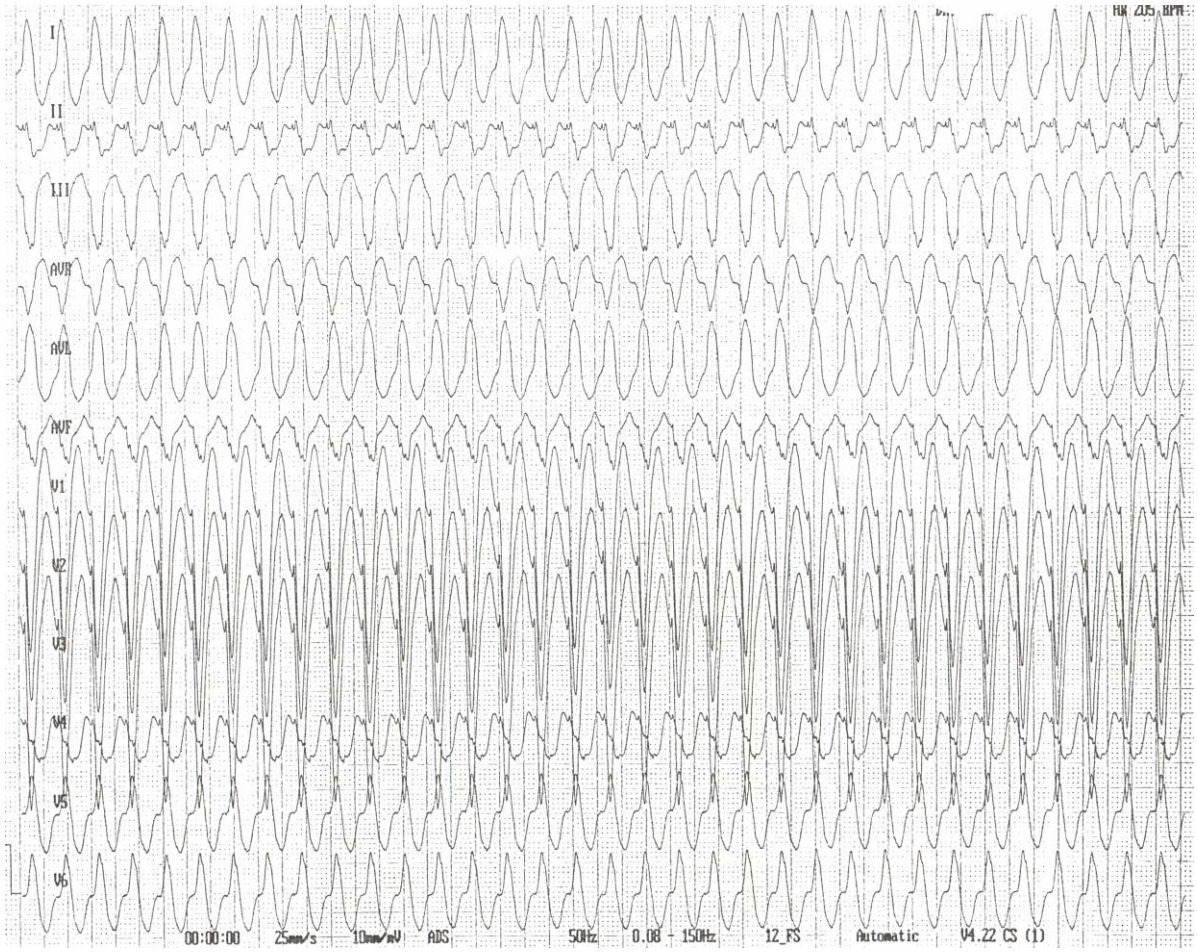


Figure 10/7.

Antidromic AVRT caused by an accessory pathway cannot often be differentiated from ventricular tachycardia based on the surface ECG. (Wide QRS complex tachycardia, 190 bpm, antidromic AVRT, secondary repolarization abnormalities.)

Another risk of WPW syndome is that certain supraventricular arrhythmias reach the ventricles not in a delayed fashion with a conduction block, but they can be conducted with a 1:1 ratio. The very rapid ventricular rate occurring in the latter case (e.g. in atrial fibrillation) can easily lead to ventricular fibrillation (FBI tachycardia.)

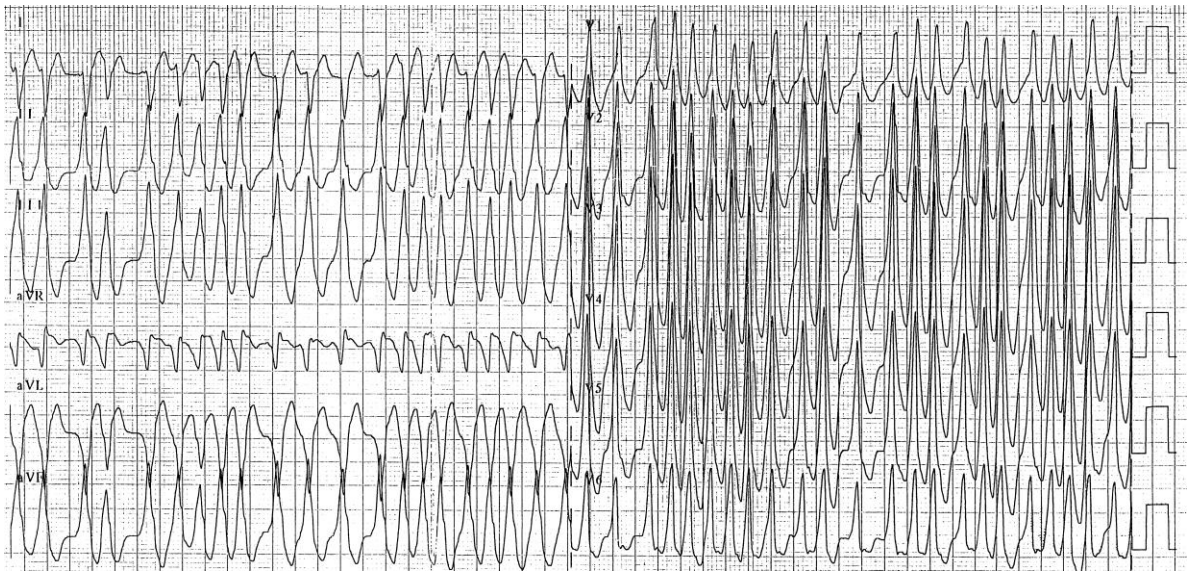


Figure 10/8. FBI tachycardia (fast, broad, irregular). In atrial fibrillation, supraventricular impulses are not filtered by the AV junction, so they are immediately conducted to the ventricles through the accessory pathway, thereby generating the above wide QRS complex tachycardia as a result of preexcitation. (Atrial fibrillation, pre-excited wide QRS complex tachycardia at a mean ventricular rate of 300 bpm, secondary repolarization abnormalities.)

A short refractory period of the accessory pathway (≤ 250 msec) is in favour of the development of the above life-threatening arrhythmia, because 4 or even more supraventricular impulses can reach the ventricles each second in such a case. The refractory period of the accessory pathway can most easily be studied by means of a noninvasive test by exercise stress testing. The disappearance of delta waves in relation to the increase in heart rate during physical exercise indicates the refractory period of the accessory pathway.

FACTS THAT YOU MUST KNOW!

1. The ECG of 2/3 of the patients with an accessory pathway shows a short PQ interval and delta waves, which may carry the risk of the development of reciprocal tachycardia or, in case of atrial fibrillation, even that of life-threatening FBI tachycardia.
2. Repolarization abnormalities and pathological Q waves are frequently observable in preexcitation, which are not signs of ischemia or infarct scar.
3. The refractory period of the accessory pathway can also be examined in a non-invasive manner by exercise stress testing, thereby accessory pathways with a short refractory period can be screened out, for which there is persistant conduction even at high heart rates and delta waves do not disappear either.
4. In case of a regular narrow (wide) QRS complex tachycardia, one should think of the presence of orthodromic (antidromic) AVRT, if signs of preexcitation are visible in sinus rhythm.
5. An irregular wide QRS complex tachycardia with a rapid heart rate may be FBI tachycardia caused by atrial fibrillation as well.

CHAPTER 11

WIDENING OF THE QRS COMPLEX

Widening of the QRS complex stems from the fact that the impulse depolarizing the ventricles does not use the cardiac conduction system for propagation or uses it only partially. When beats or a rhythm with wide QRS complexes is observed, it is a frequent mistake that they are automatically considered to have been originating from the ventricles. Supraventricular impulses may be conducted to the ventricles with a bundle branch block pattern due to a preexisting bundle branch block or a functional bundle branch block developed as a result of a short coupling interval. The essence of functional bundle branch block is that the given bundle branch could not completely be repolarized yet, so the next impulse finds it in the refractory period. Since the refractory period of the right bundle branch is longer than that of the left-sided one, functional bundle branch blocks therefore more often have a right bundle branch block pattern. If an impulse with a wide QRS complex is of ventricular origin, the widening is caused by slow conduction of the impulse through the myocardium (instead of via the cardiac conduction system). As it was mentioned previously, the impulse conduction velocity shows marked differences in various areas of the heart. The conduction velocity of bundle branches and Purkinje fibers is nearly tenfold of that of ventricular cardiomyocytes. An impulse originating in the ventricles therefore reaches each point of the heart more slowly, even when it penetrates into the cardiac conduction system somewhere.

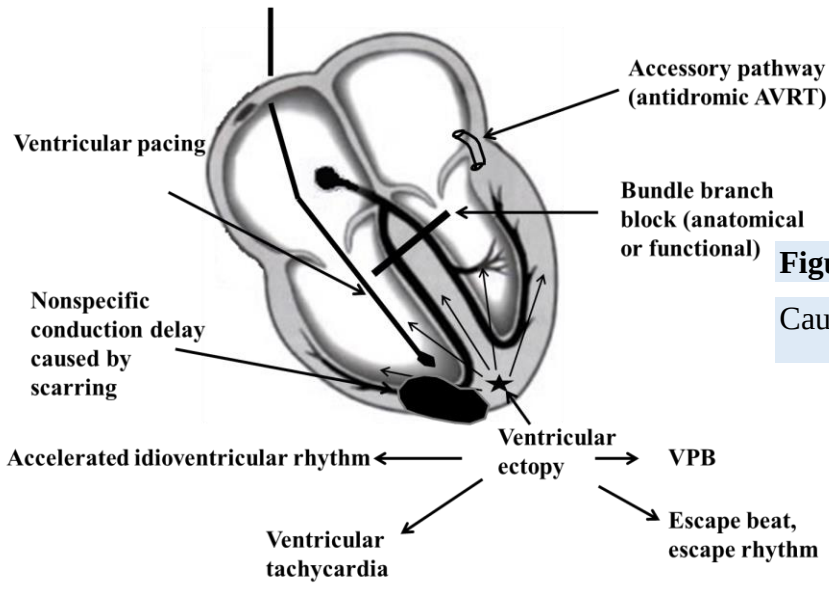


Figure 11/1.

Causes of QRS widening.

Taking all of the above into consideration, let's review the cases associated with widening of the QRS complex.

1. Fixed bundle branch block: Please remember that patients might be in sinus rhythm, but they may have atrial fibrillation as well. In the latter case, no P waves can be seen and it is therefore a frequent mistake that QRS complexes are interpreted as having a ventricular

origin. Inexperienced interpreters may really get confused by the observed ECG picture when there is high ventricular rate. However, one should keep in mind that cardiac rhythms of ventricular origin are regular in the vast majority of cases, while there is variable distance between the consecutive QRS complexes in atrial fibrillation that is the rhythm is irregular.

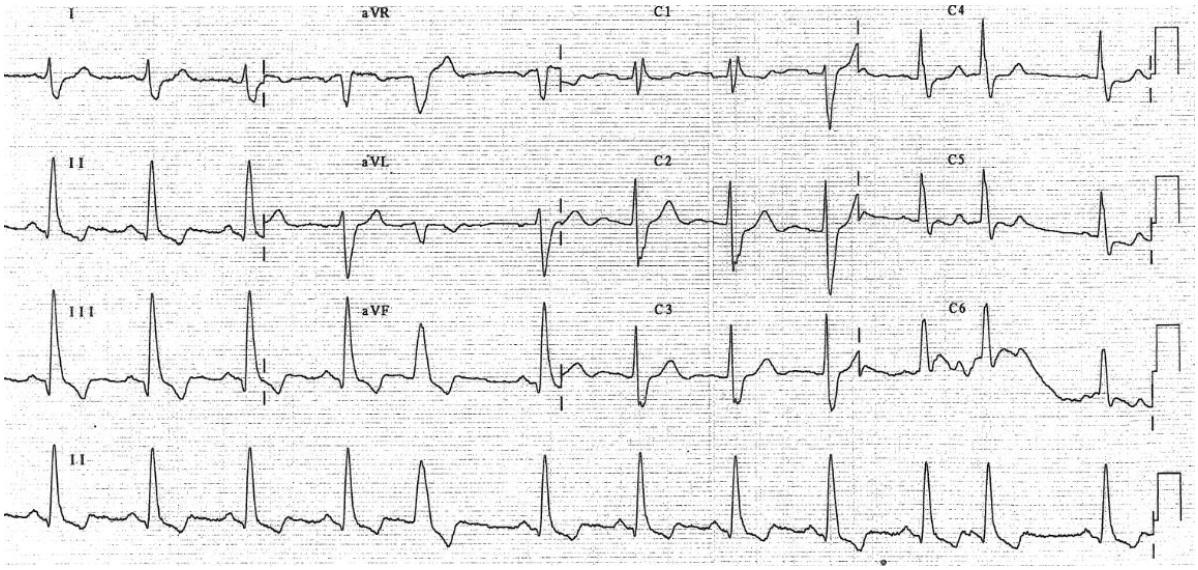


Figure 11/2.

RBBB and LPFB results in a wide QRS complex pattern along with sinus rhythm. The 5th beat also has a wide QRS complex, but the QRS morphology and axis is significantly different from that of the QRS complexes of sinus beats, so it is a VPB, while the 11th beat has an identical morphology with those of sinus beats, it is therefore a PAC.

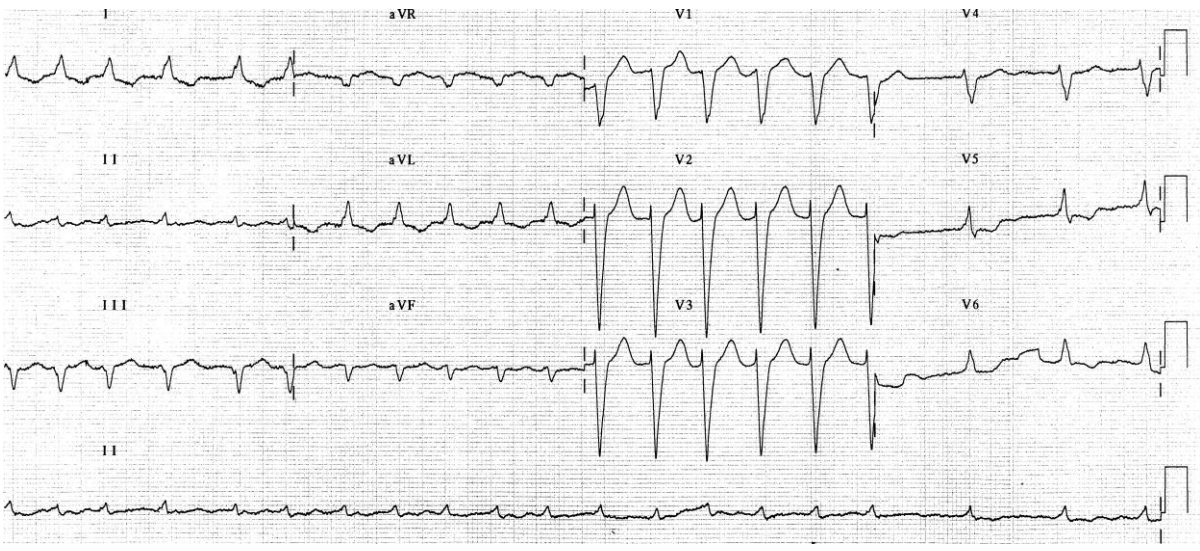


Figure 11/3.

Atrial fibrillation and LBBB. If there are no P waves and the QRS complexes are wide, it is not a cardiac rhythm of ventricular origin just because of these. Regularity of the rhythm in leads V1-3 may be misleading, however, the rhythm strip and leads V4-6 give one a clue.

It may occur in bundle branch blocks and prolonged AV conduction (especially in sinus tachycardia) that the P wave is blending into the T wave of the preceding complex and may be difficult to notice.

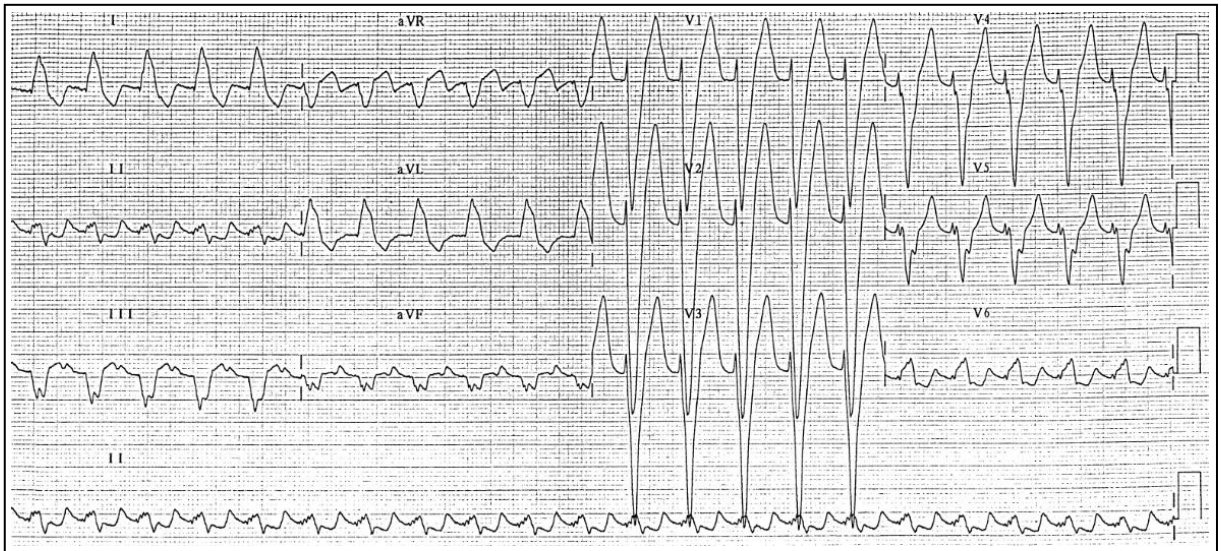


Figure 11/4.

The patient with known ischemic cardiomyopathy and left bundle branch block has sinus tachycardia with prolonged AV conduction, which can easily be interpreted as ventricular tachycardia if one fails to examine leads III and aVF where P waves are clearly detectable (sinus tachycardia, prolonged AV conduction (240 ms), left axis deviation, left bundle branch block, secondary repolarization abnormalities.)

2. Functional bundle branch block: Refractoriness cannot immediately adapt to sudden shortening of the RR interval, therefore it frequently occurs that an impulse with a short coupling interval, e.g. a supraventricular premature beat, will become completely or partially blocked. In the latter case, the beat will be conducted with a bundle branch block pattern. For an atrial fibrillation with marked variability of the coupling interval, wide QRS complexes with aberrant conduction (Ashman phenomenon, see its description for details) frequently occur, which sometimes cannot easily be differentiated from ventricular premature beats. This also accounts for the fact that during initiation of a paroxysmal supraventricular tachycardia (PSVT), the first few QRS complexes might as well be wide (for details, see section on phase 3 blocks.)

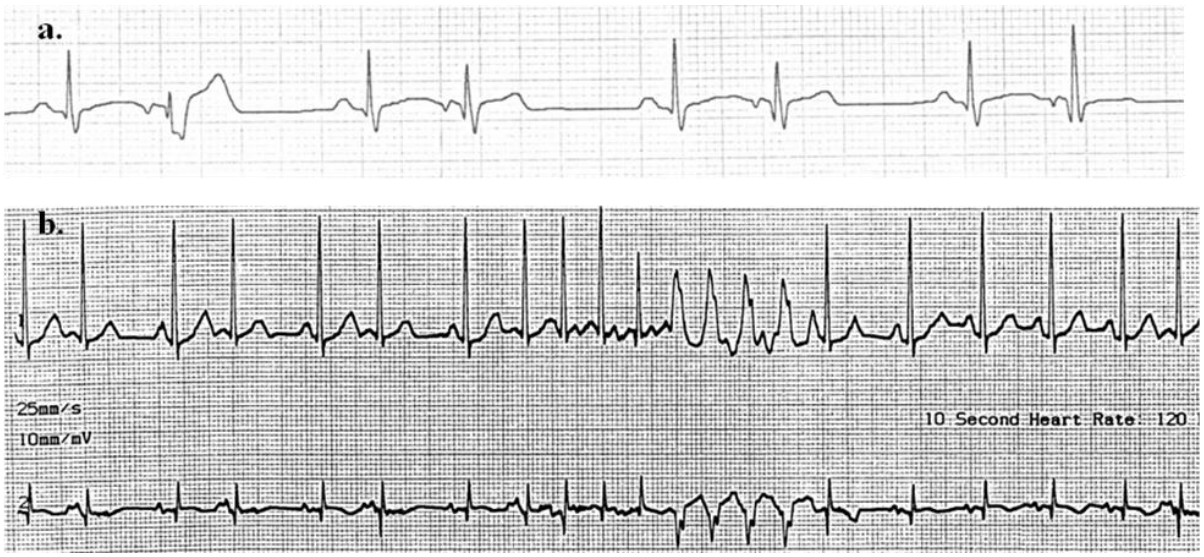


Figure 11/5.

a. Supraventricular premature complexes are conducted with an RBBB pattern with a variable QRS width. **b.)** Tachyfibrillation following the frequent occurrance of PACs is observable on the Holter recording, the 5th to 8th beat of which is aberrantly conducted to the ventricles due to the functional bundle branch block.

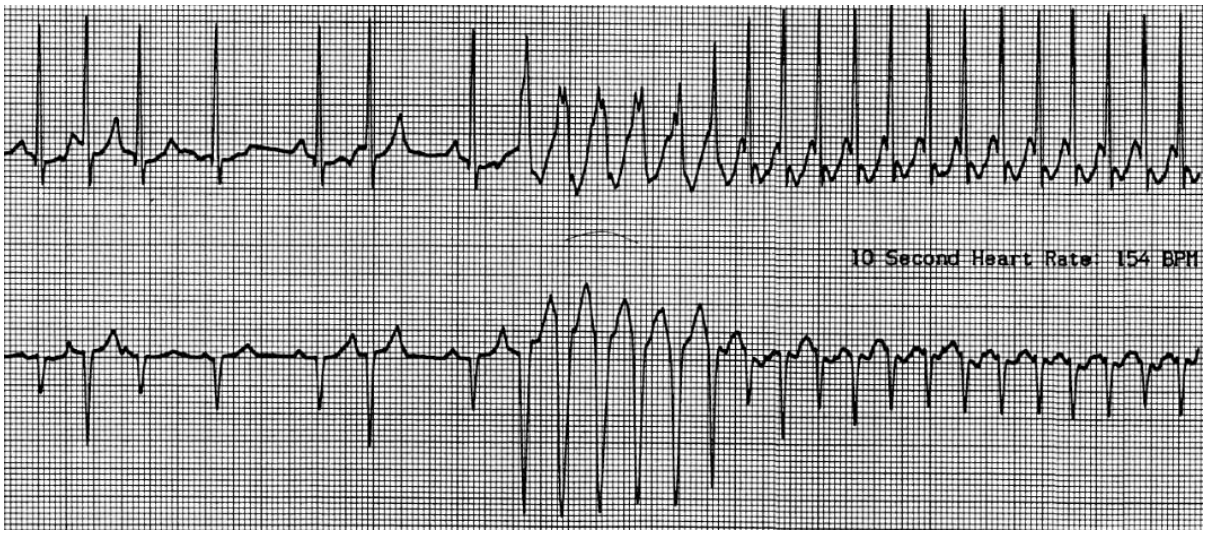


Figure 11/6. Wide QRS complexes are visible at the onset of an orthodromic AVRT mediated by a concealed accessory pathway, which is the consequence of functional bundle branch block.

3. Nonspecific intraventricular conduction disturbance (IVCD): Especially after a myocardial infarction with large extent of the necrosis or developed in multiple locations or, alternatively, in severe cardiomyopathy, wider QRS complexes can frequently be observed, which, however, do not meet the morphological criteria of left and right bundle branch block either. Widening of the QRS complexes in this case is caused by slowed conduction in the peri-infarct zone or extensive myocardial fibrosis, so it is not in the bundle branches where impulse conduction is blocked.

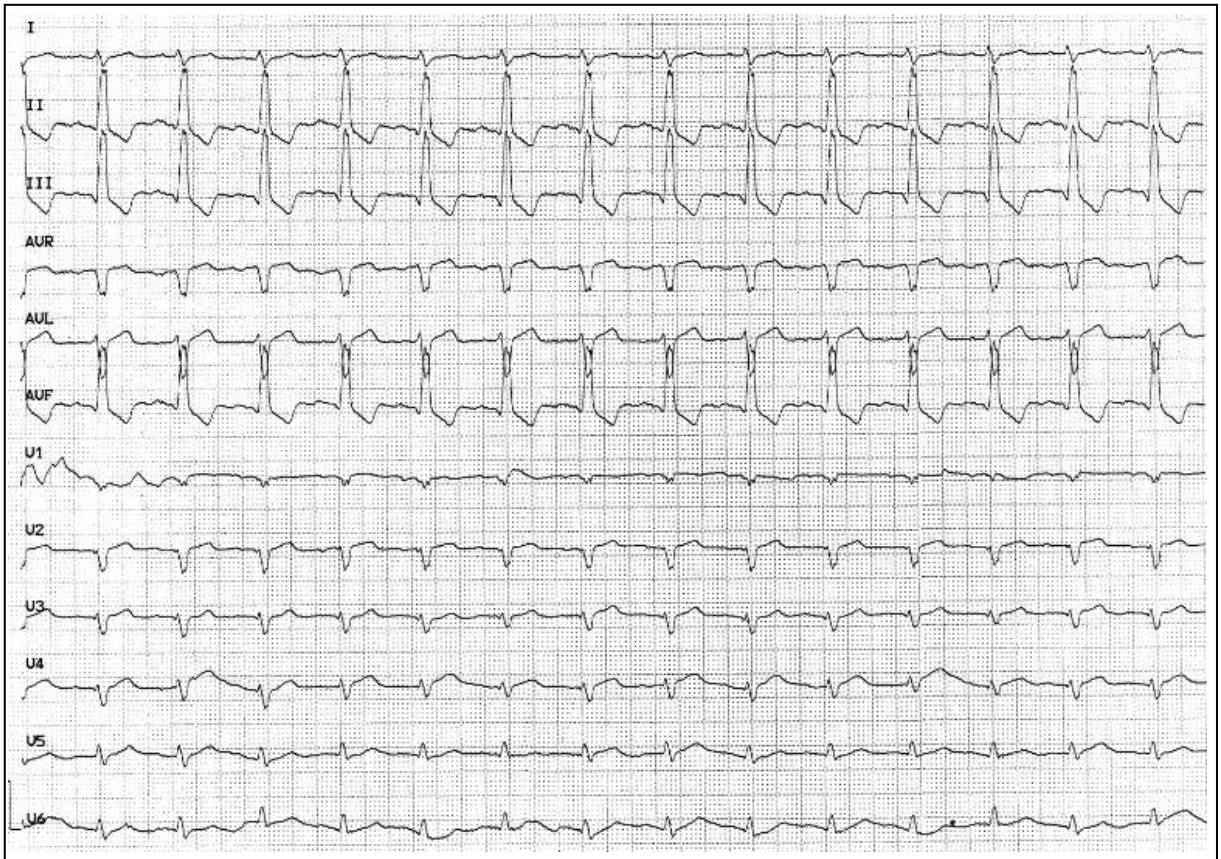


Figure 11/7.

Nonspecific intraventricular conduction disturbance. The patient has left posterior fascicular block, but this does not provide an explanation for widening of the QRS complex. During the coronary angiography, chronic proximal occlusion of the LAD and the right coronary artery was observed. (Sinus rhythm, normal AV conduction time, right axis deviation, left posterior fascicular block, low voltage in the precordial leads, nonspecific intraventricular conduction disturbance, secondary repolarization abnormalities.)

4. An impulse originating in the ventricles may be an escape beat, premature beat or a series of any previous type of beats. The impulses arising in the ventricles are slowly propagating to each region of the myocardium, thereby resulting in wide QRS complexes.

5. QRS complexes are also wide during ventricular pacing because the condition, from the aspect of impulse propagation, is similar to ventricular premature beats, which are also generated focally. Due to the ventricular lead positioned in the right ventricle, the left ventricle is activated from the right ventricle. The same is detectable in left bundle branch block (LBBB) that is the left ventricle is depolarized from the right ventricle. This is why the ECG pattern of right ventricular pacing is very similar to that seen in left bundle branch block.

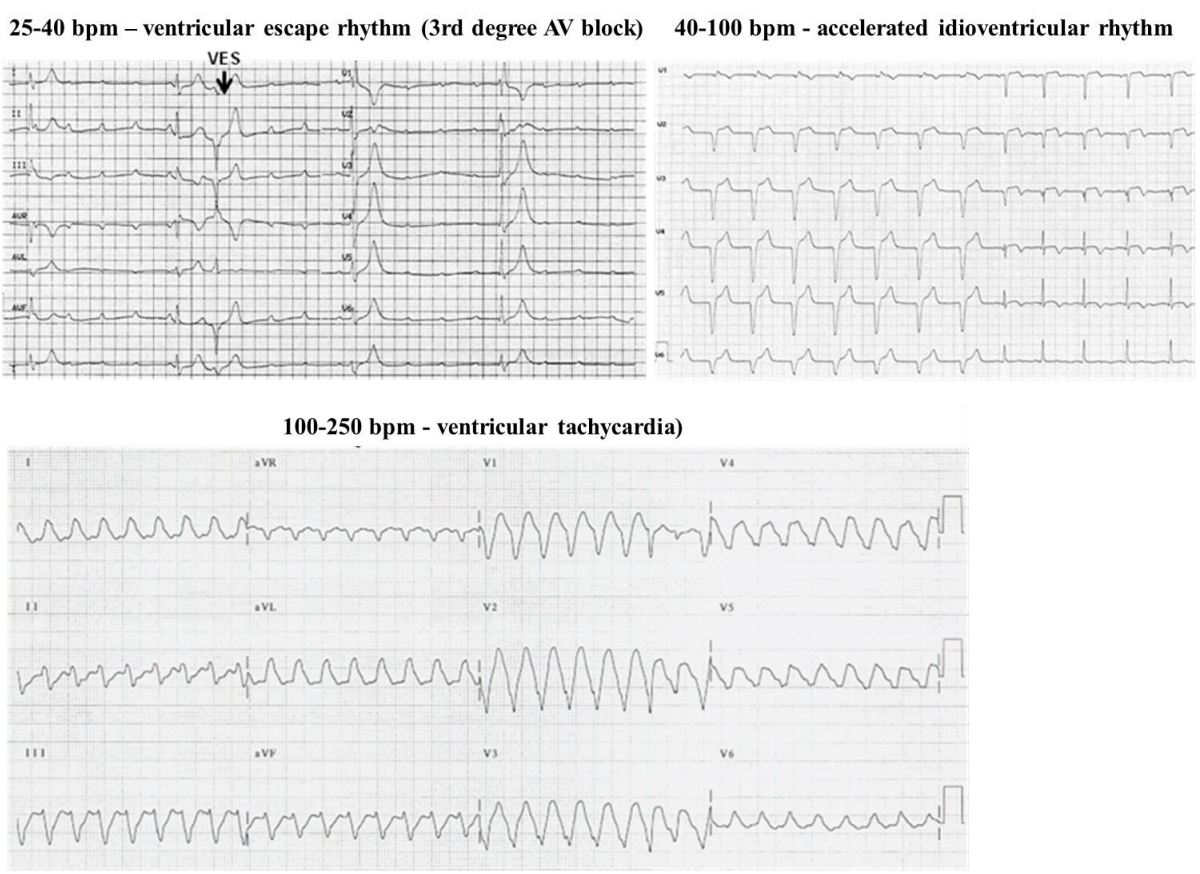


Figure 11/8. Impulse formation takes place in the ventricles in all three cases, but the causes and the heart rate are different. In the first case, ventricular escape rhythm caused by 3rd degree AV block is visible. Moreover, accelerated idioventricular rhythm developed during reperfusion of an anterior myocardial infarction can be seen on the second ECG tracing, while fast ventricular tachycardia on the third ECG (the 7th and 8th beat in lead V1-3 are fusion beats occurring as a result of fusion of ventricular and sinus beats.)

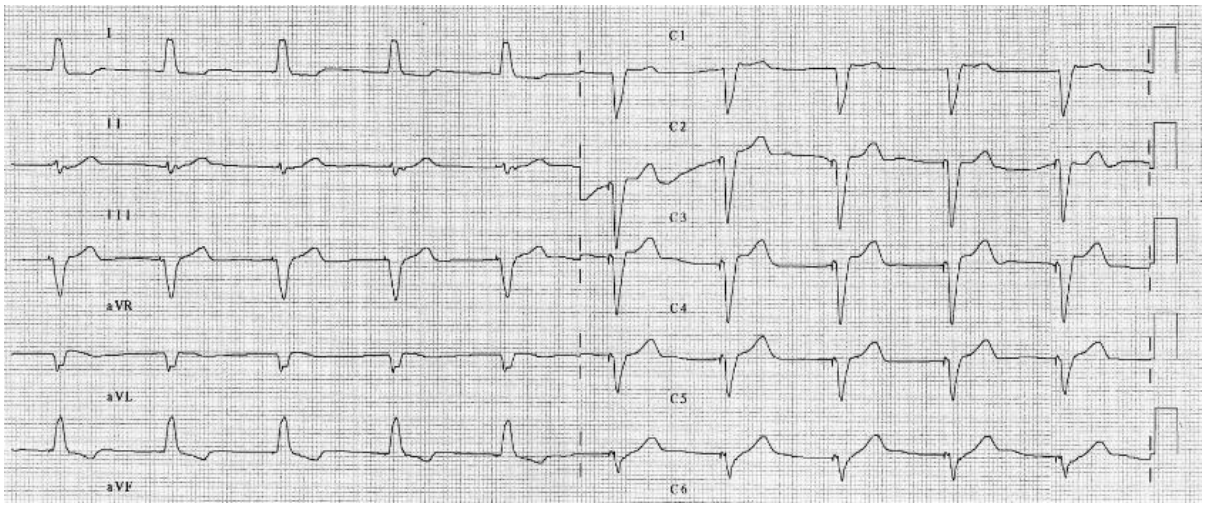


Figure 11/9. ECG for VVI pacemakers, the pacemaker lead is situated in the right ventricle and if continuous pacing (bipolar pacing with a small amplitude in this case) is applied, this results in widening of the QRS complexes demonstrating an LBBB pattern. (Ventricular paced rhythm, 60 pbm, left axis deviation, secondary repolarization abnormalities.)

CHAPTER 12

NOISES AND ARTIFACTS ON THE ECG

In clinical practice, only less than half of the ECG recordings have an impeccable quality from technical aspects. A significant number of ECG recordings may be interspersed with minor or major artifacts, which generally are potentials resulting from electrical interference or skeletal muscle activity. The most common types are motion artifacts (movement of the limbs, breathing, tremor, shivering), therefore some examples of these will be shown. Artifacts resulting from motion often make it difficult to determine the basic rhythm. Fine oscillations of the baseline may create the idea in one to think of the presence of atrial fibrillation, although P waves can be discovered with a vigorous examination and the rhythm does not seem irregular either. Consider the fact that noise (oscillations and fluctuations of the baseline) always presents randomly, while signals (e.g. P waves) consequently appear on the same respective point of the tracing. One should carefully examine the time interval of 200-240 ms before the QRS complexes. It may occur that the amplitude of P waves is low in many of the leads, therefore, one should have a look at each lead carefully in case of uncertainty. If consequently occurring P waves are visible in one of the leads, the rhythm is sinus rhythm. In addition, it should be added that even noise can produce a wave being similar to P waves (or sometimes f waves show a pattern being similar to P waves); however, when examining the segments before the rest of the QRS complexes in the given lead, these waves cannot be found any longer because the basic rhythm is atrial fibrillation.

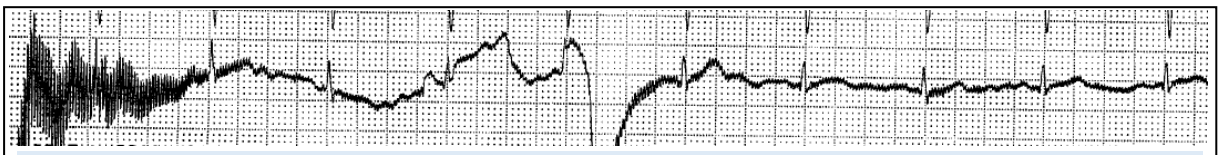


Figure 12/1. Electrical interference.

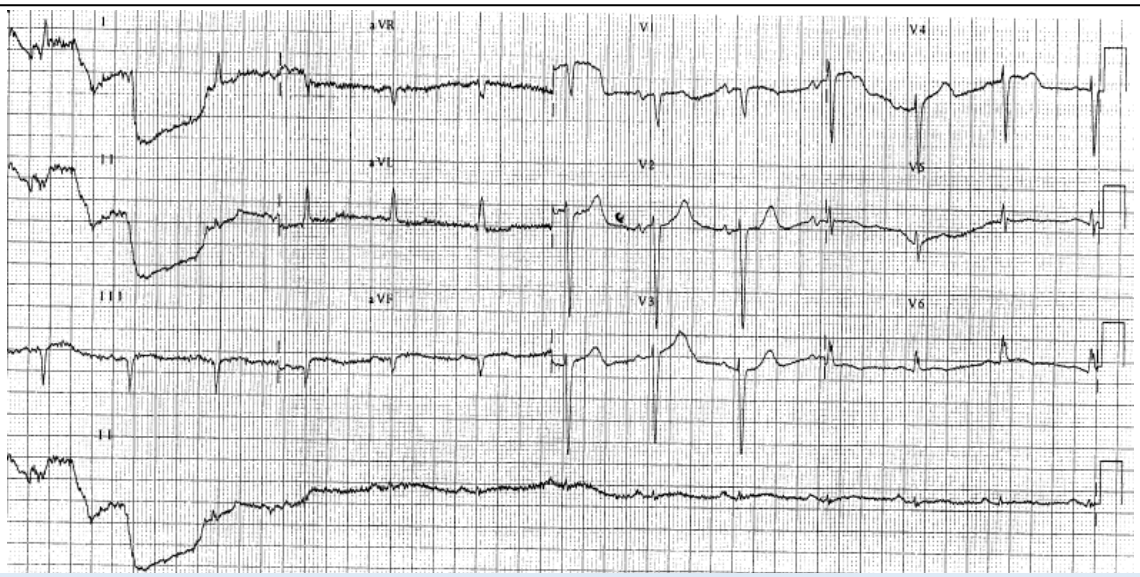


Figure 12/2. Fluctuations (wandering) and oscillations of the baseline may cause some difficulties in determining the basic rhythm in the limb leads, however, lead aVF and the right-sided chest leads unequivocally indicate that this is sinus rhythm.

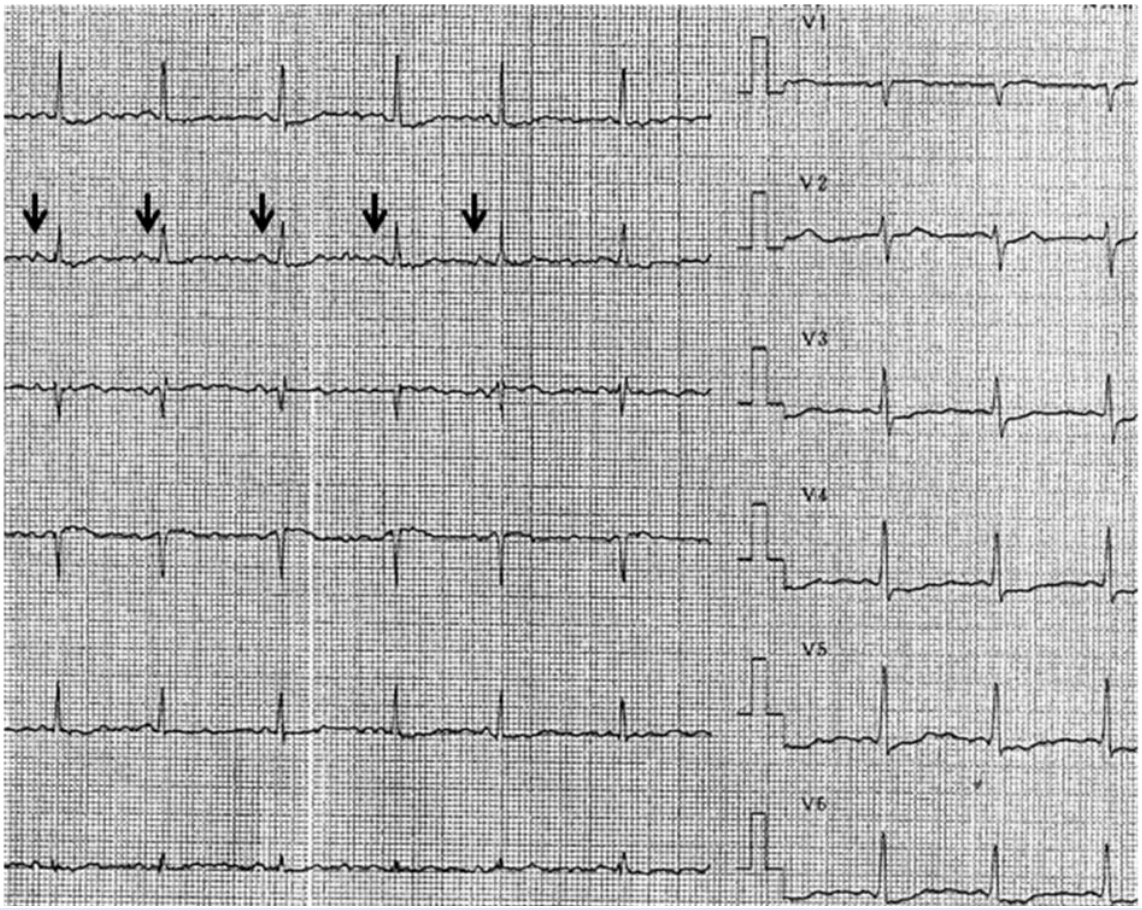


Figure 12/3. A baseline with a lot of noise may make it difficult to identify the basic rhythm, however, since noise occurs at random locations, while P waves at their usual place (arrows), it is no doubt that this is sinus rhythm.

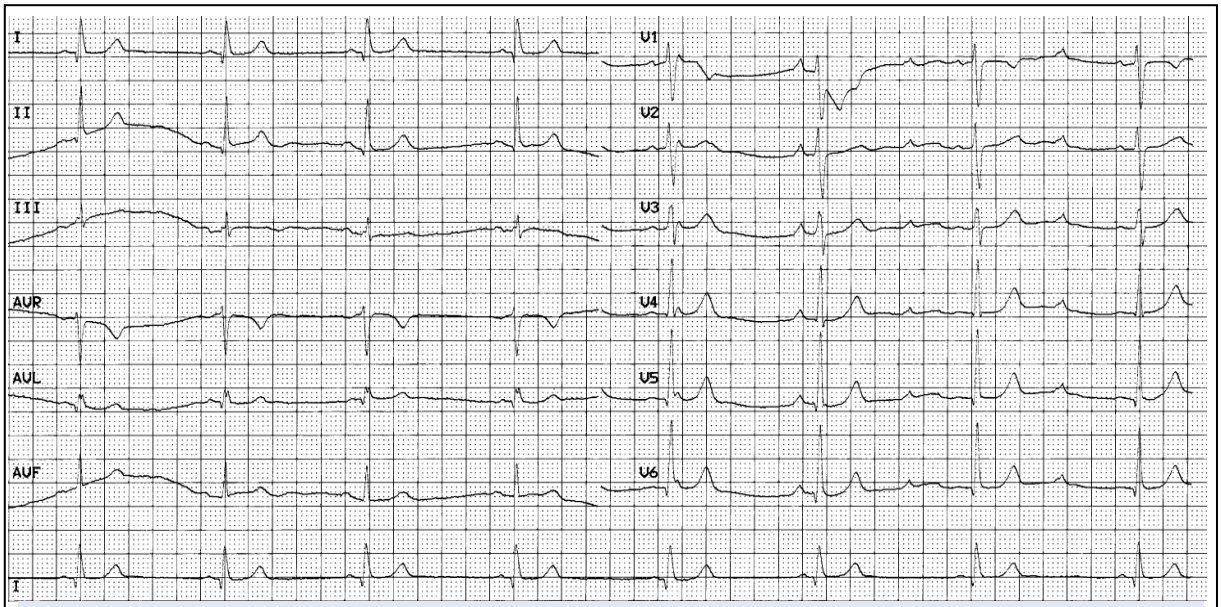


Figure 12/4. Please note the deflections similar to P waves between the 2nd and 3rd as well as 3rd and 4th QRS complexes in the chest leads, which may raise the possibility of 2:1 AV block. However, this is not the case here and the patient has normal sinus rhythm without any abnormalities regarding AV conduction. The odd wave being similar to the P wave does not appear between the 1st and 2nd QRS complexes in the precordial leads and the rhythm is completely regular as well.

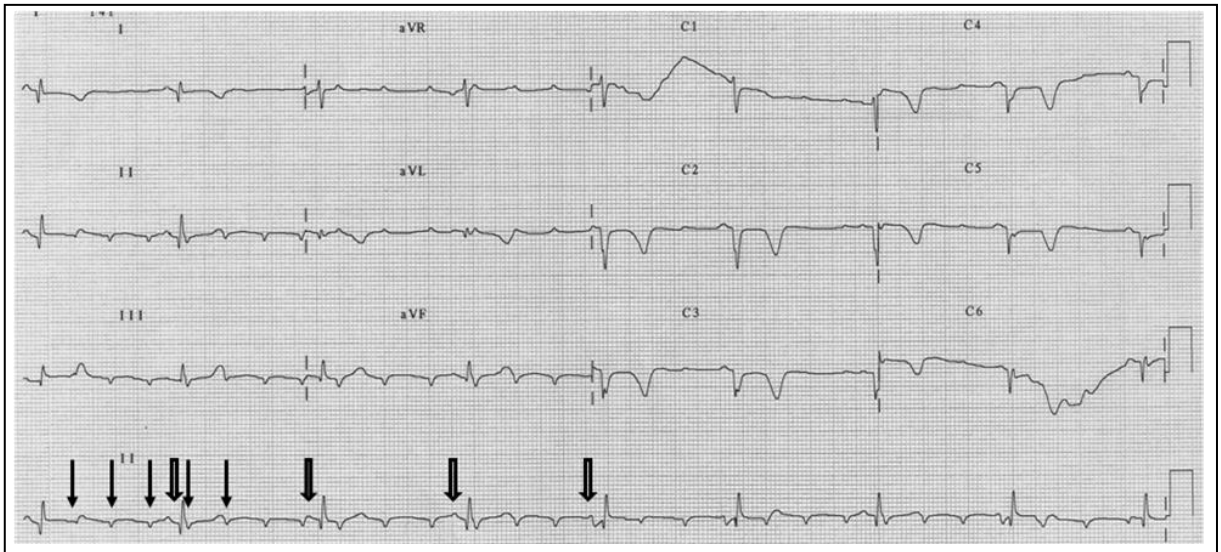


Figure 12/5.

ECG. The negative „P waves" (thin arrows) occurring in leads II, III and aVF raise the possibility of atrial tachycardia, but based on the concurrent presence of the real P waves (empty arrows), the former abnormality can only be a noise.

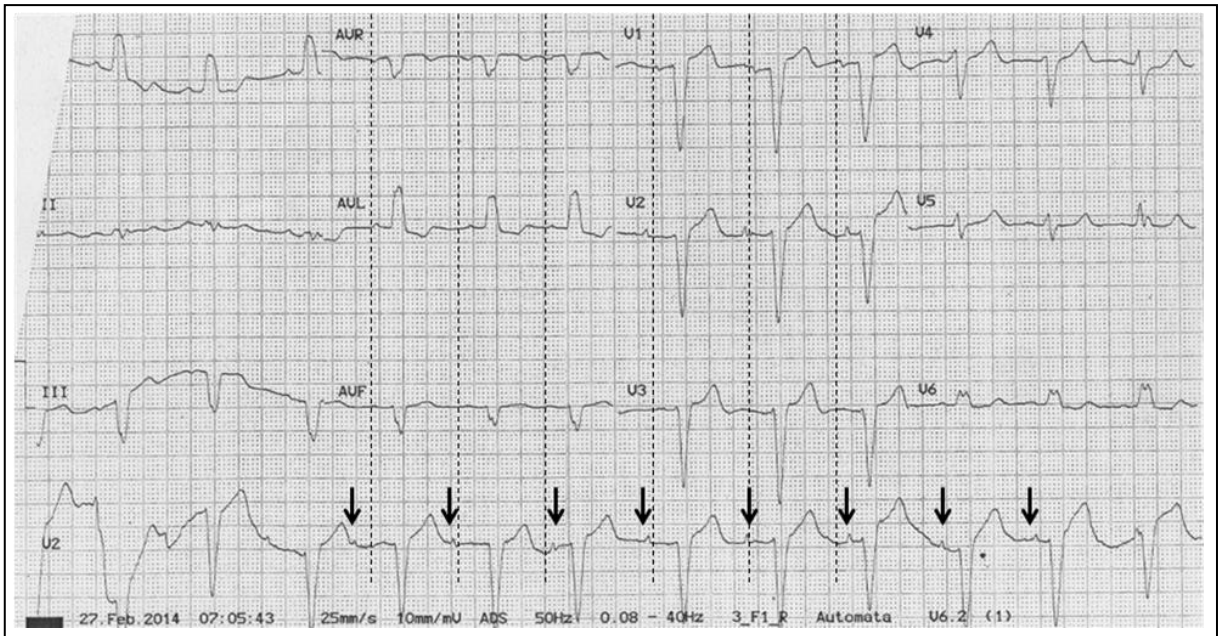


Figure 12/6.

Artifacts (arrows) mimicking P waves on the rhythm strip. Dashed lines indicate the initial portion of the real P waves, which does not coincide with the artifacts.

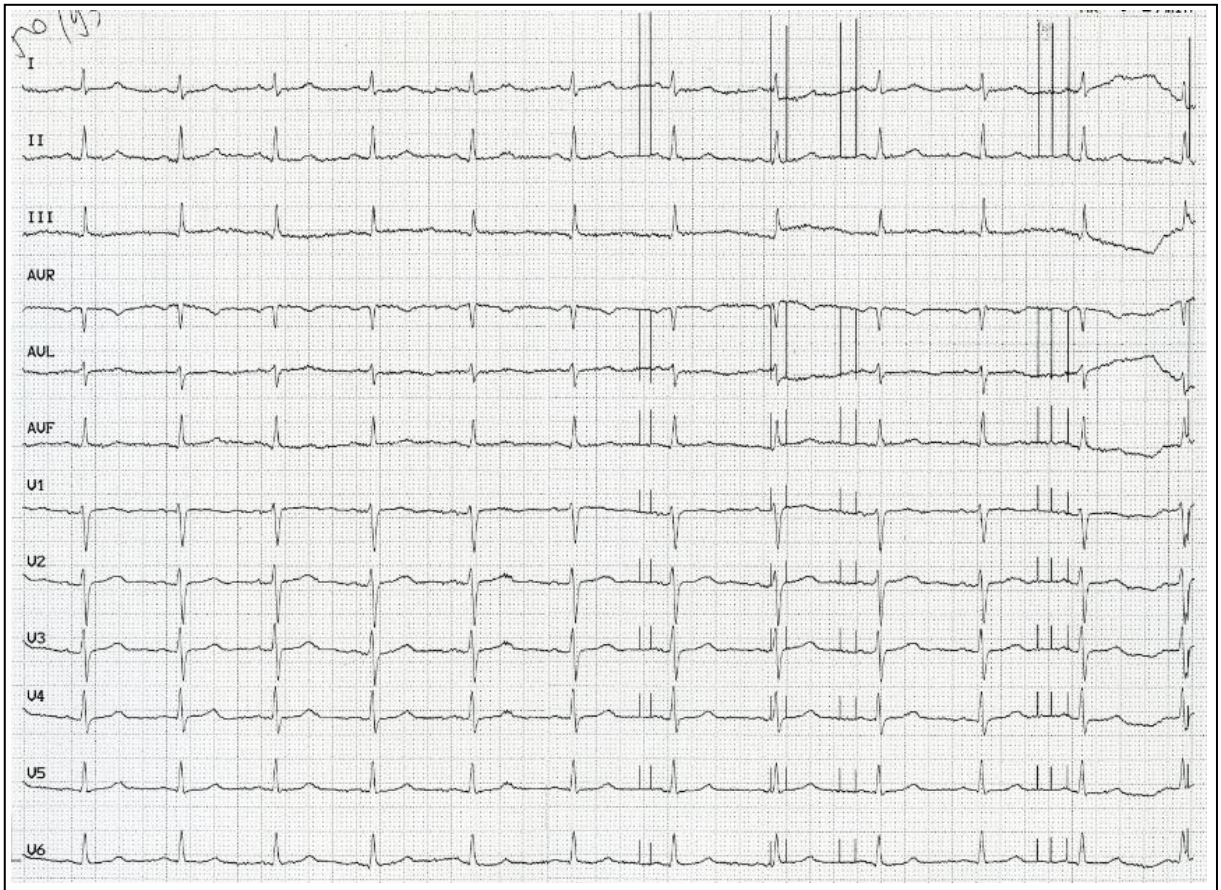


Figure 12/7.

Noise mimicking pacing spikes. No pacing spikes with such a short cycle length can occur on the ECG and the patient has no pacemaker anyway.

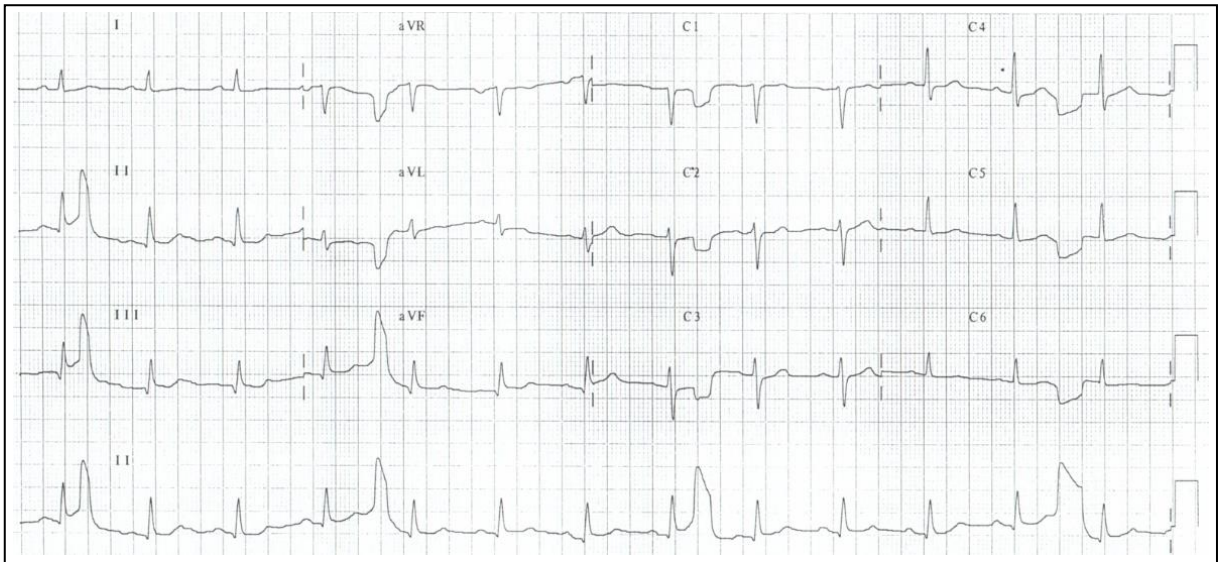


Figure 12/8.

Noise mimicking ventricular premature beats. It is interpolated between the QRS complexes completely at a random manner, moreover, VPBs also have repolarization which cannot be observed here.

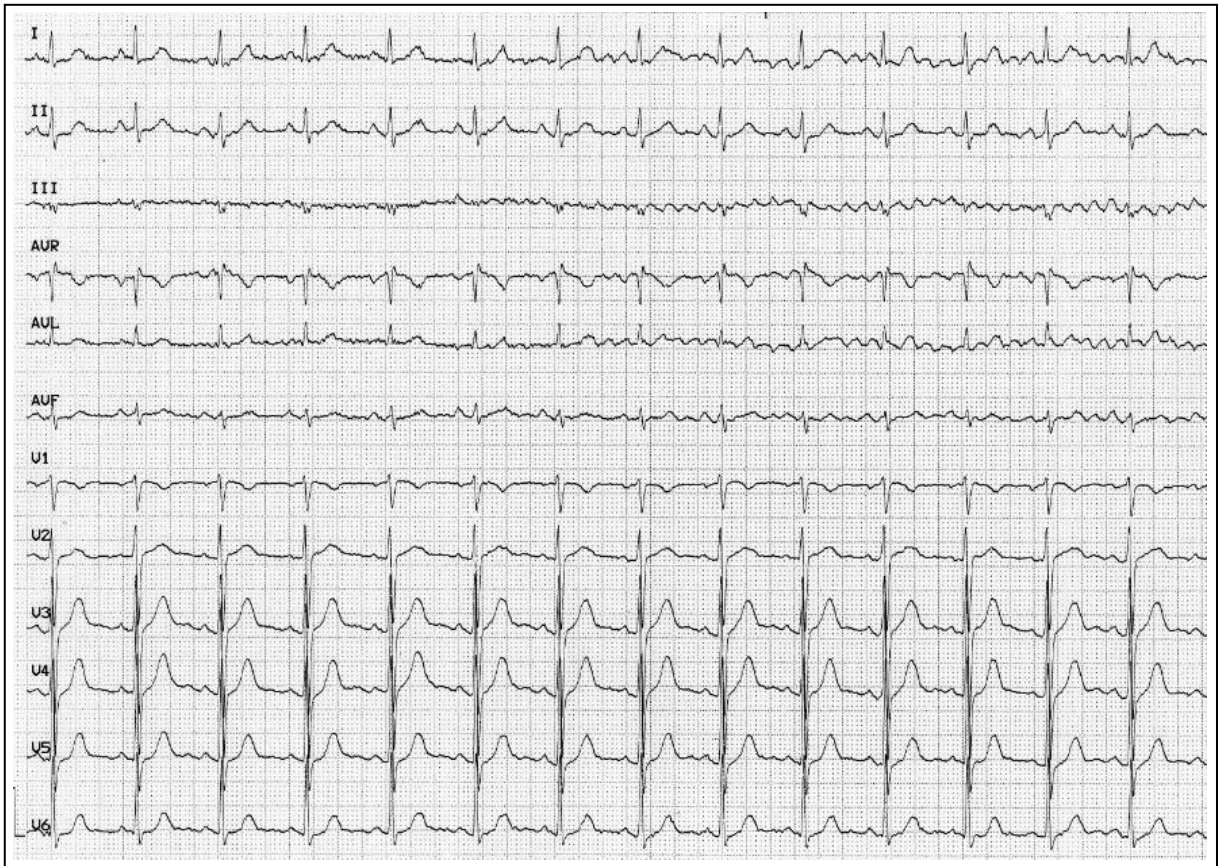


Figure 12/9.

Noise mimicking atrial fibrillation in the chest leads. However, unequivocal P waves can be seen throughout the tracing in lead II.

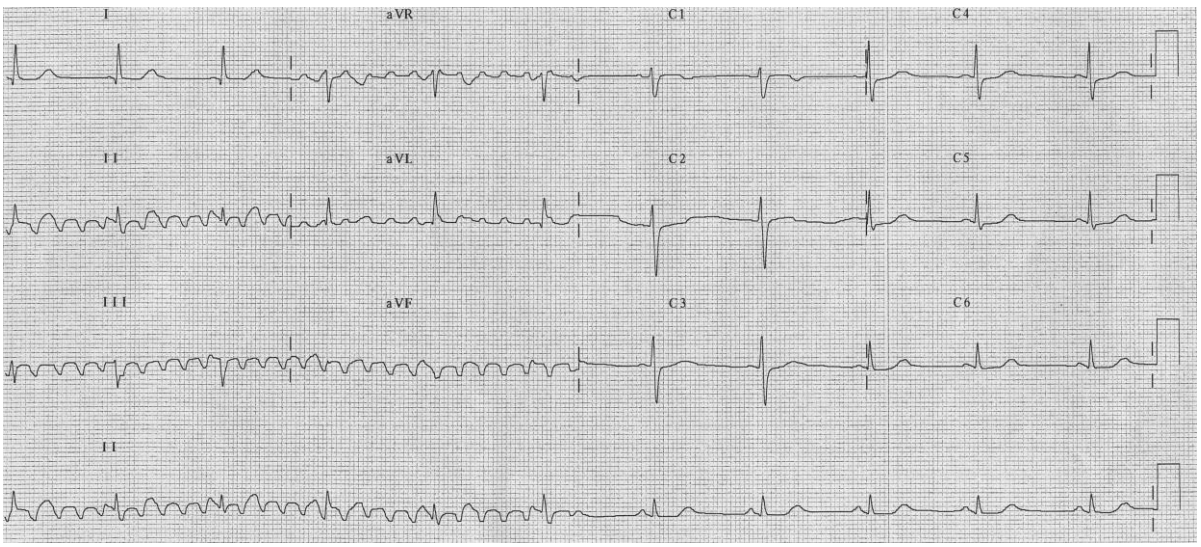


Figure 12/10.

Noise reminding of atrial flutter in the limb leads. Please note on the rhythm strip that regular P waves occur along with constant RR intervals on the right side of the tracing, which exclude the possibility of atrial flutter.

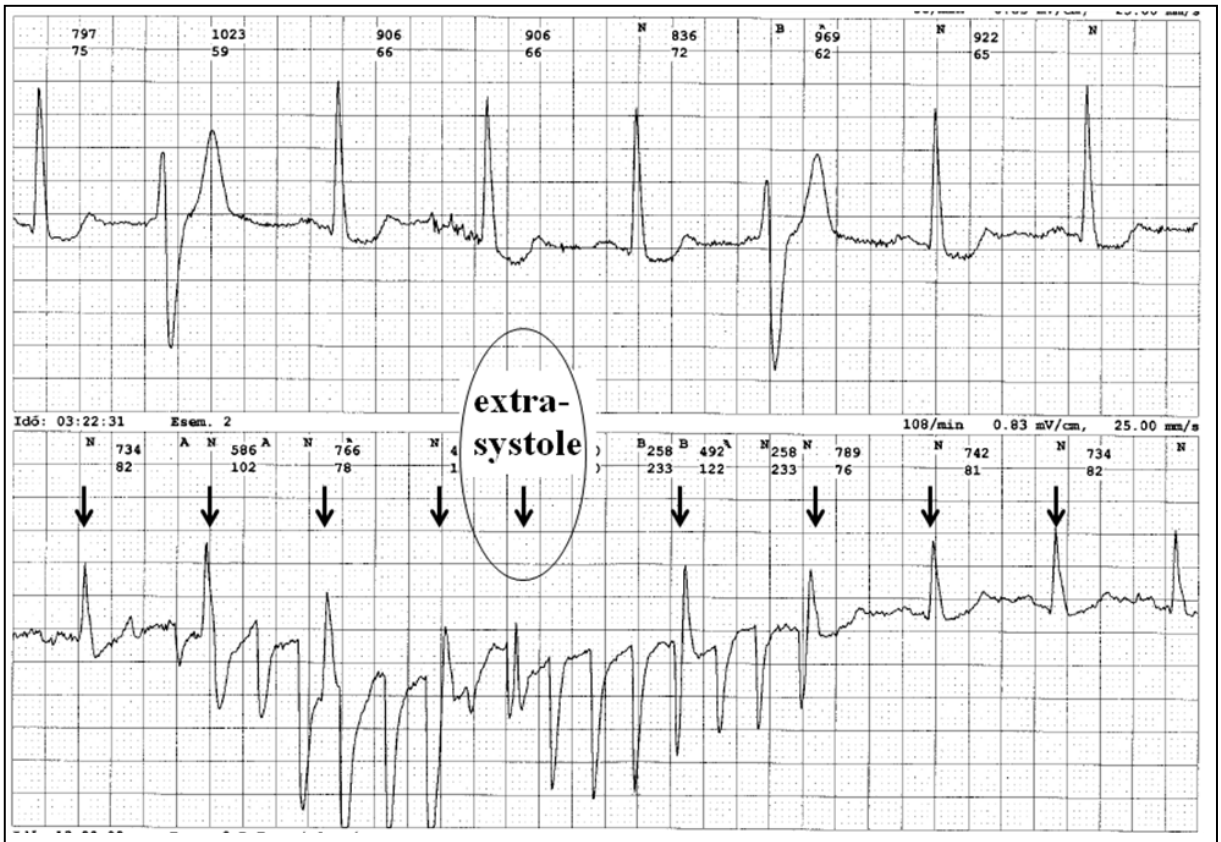


Figure 12/11.

Ventricular premature beats are observable on the upper part of the ECG recording. In addition, a waveform pattern similar to ventricular tachycardia is visible in the middle part of the tracing; the arrows indicate QRS complexes originating from the SA node, so the presence of VT can be excluded. (The Holter electrodes caused irritation of the skin and resultant scratching, which was the underlying cause of this finding.)

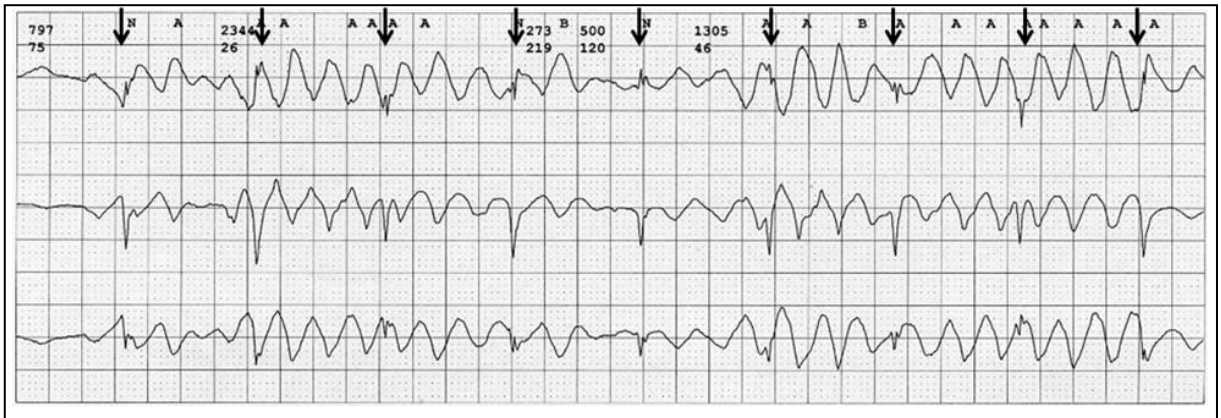


Figure 12/12.

Wandering of the baseline mimicking ventricular arrhythmia detected on the Holter recording. The arrows indicate normal QRS complexes.

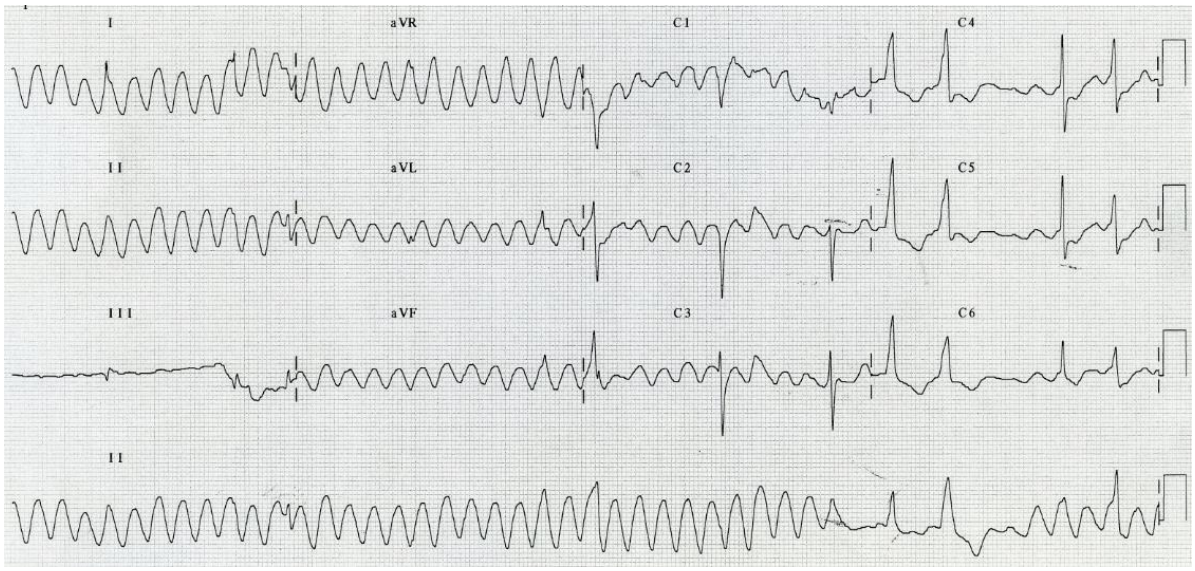


Figure 12/13.

Noise reminding of fast ventricular tachycardia. The right-sided precordial leads and lead III help establish the correct diagnosis, however, beats with a wide QRS complex in the left-sided precordial leads may represent a complete wrong turn for the diagnosis.

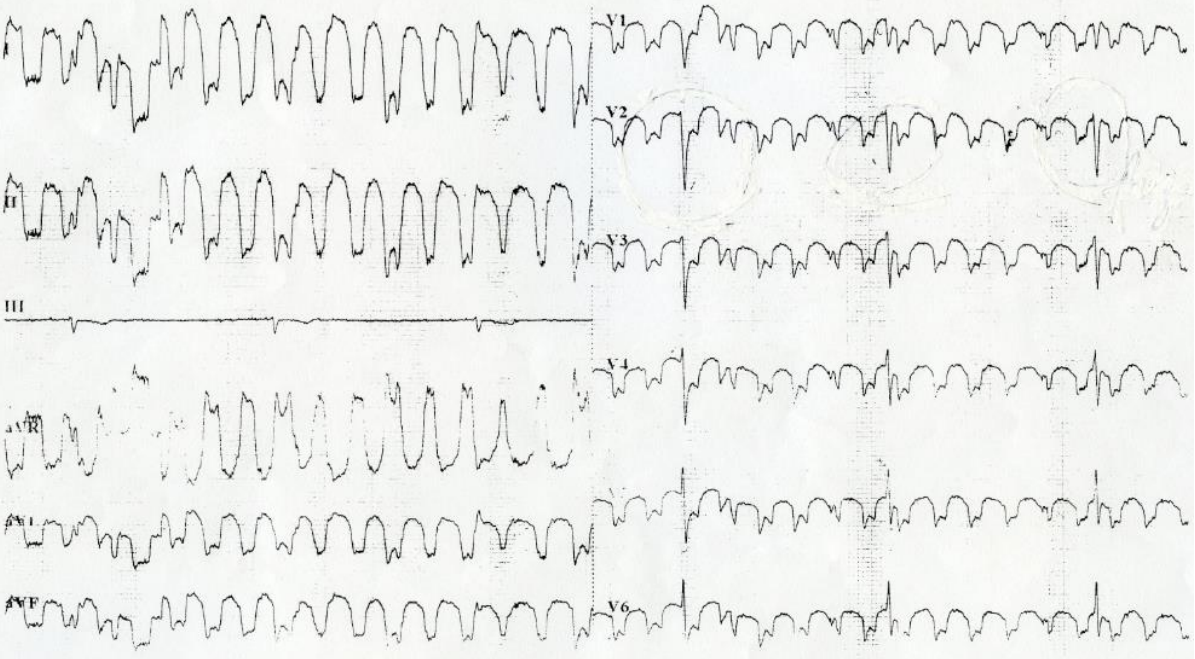


Figure 12/14.

Noise mimicking polymorphic ventricular tachycardia, which finding is unambiguously unveiled by lead III. Instead of tachycardia, it is just the opposite that is bradycardia caused by digitalis toxicity. Oscillations of the baseline can be explained by Parkinson's disease of the patient.

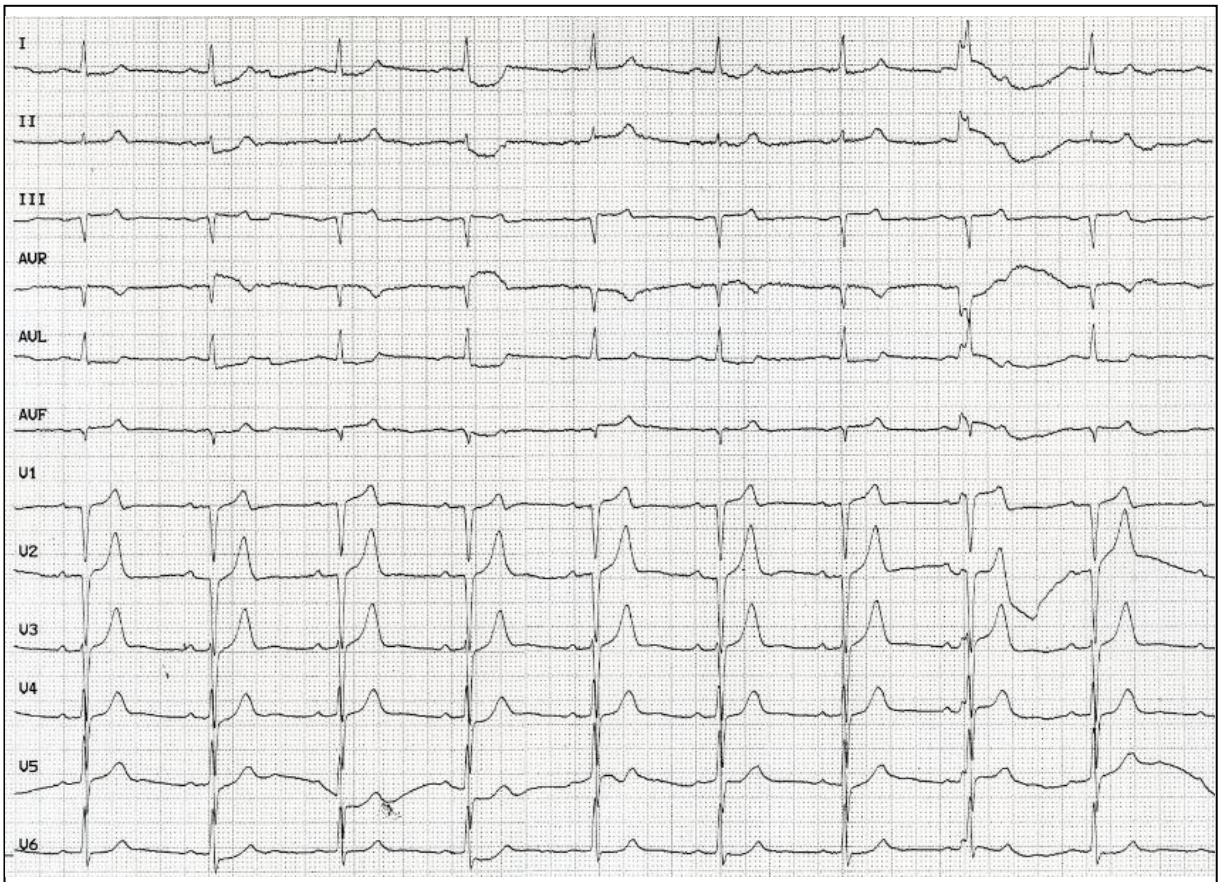


Figure 12/15.

After the 2nd and 4th QRS complexes, ST segment elevation can be observed in lead aVR as well as ST segment depression in leads I, II and aVL, but these are not real abnormalities. Along with a constant morphology of the QRS complexes (narrow QRS), no beat-to-beat variation of the ST segment is possible.

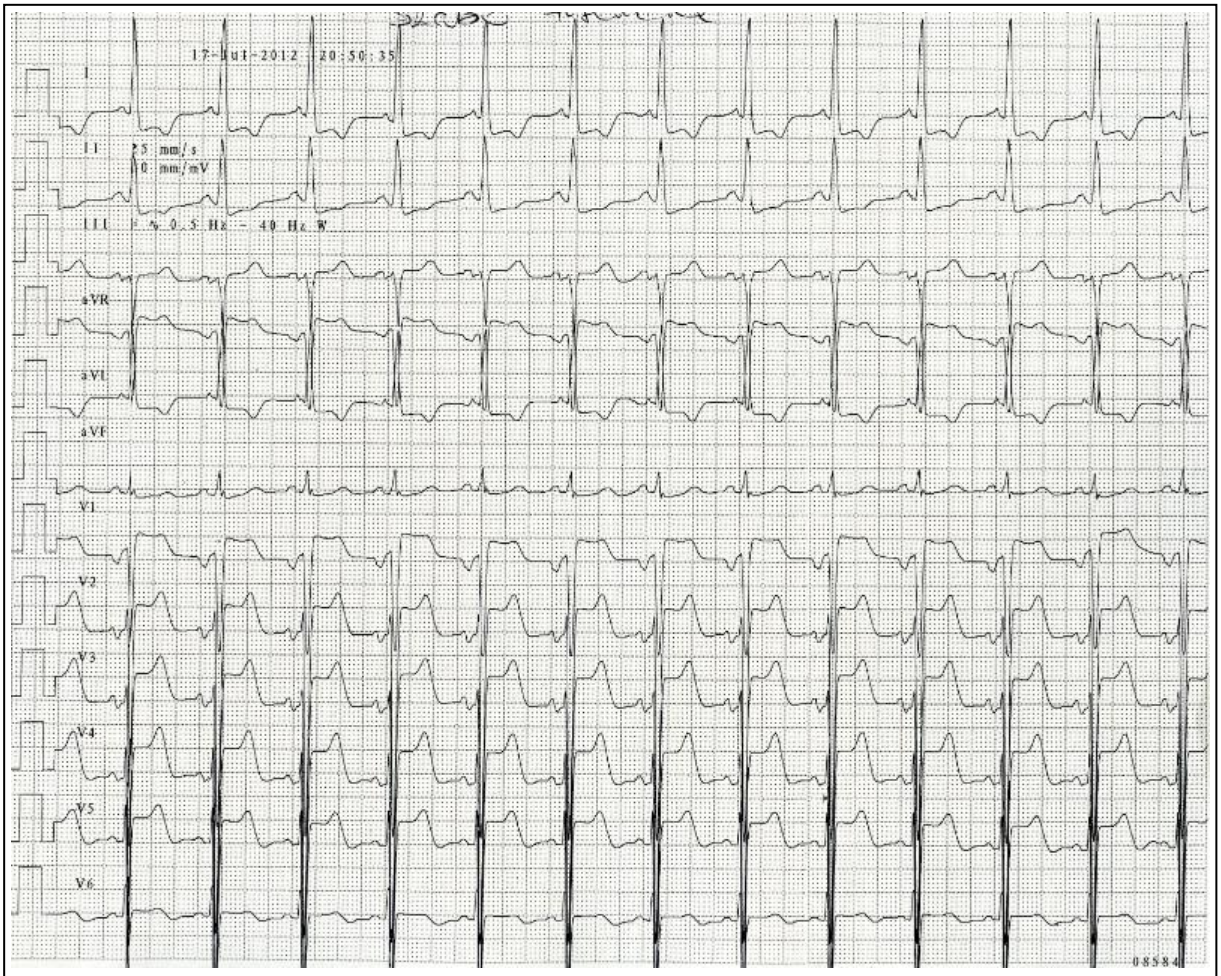


Figure 12/16.

False ST segment elevation in the precordial leads occurring with the use of certain ECG instruments. Such an ECG appearance may occur if the manual recording mode is used instead of the automatic mode while recording an ECG. In this case, signs of left ventricular strain occurring due to left ventricular hypertrophy of the patient are presented in the precordial leads in an exaggerated fashion, rather mimicking acute anterior myocardial infarction, however, the patient had no recent chest pain and the calibration signal is also normal-sized.

FACTS THAT YOU MUST KNOW!

1. Noise always appears on the ECG at random, while signals occur consequently.
2. If something unusual is observed on the ECG that has never been detected before (and provided that one has widespread knowledge on the ECG), it might be noise.
3. Fine oscillations and fluctuation of the baseline is not atrial fibrillation.

CHAPTER 13

REVIEW OF THE ECG ANALYSIS AND PRACTICE SESSION

Let's now go through the aspects of ECG analysis and review how we get to an ECG diagnosis.

1. **If there are P waves** and they come from the sinus node based on their morphological signs, the rhythm is sinus rhythm; if their morphology is different from that seen in sinus rhythm, see above in section on the differential diagnosis of narrow QRS complex tachycardias for further details. The next step is the PQ interval; if it is short and delta waves are visible at the initial portion of the QRS complexes, it is WPW syndrome; if it is long, one has to search for an AV block. At heart rates below 50 bpm, it is referred to as bradycardia, while at those above 100 bpm, it is called tachycardia. For the QRS complexes, the following features are examined: a.) are they narrow or wide b.) are there any pathological Q waves? If QRS complexes are *narrow*, it always represents supraventricular impulse formation, while in the opposite case, i.e. if QRS complexes are wide, the impulses may have both supraventricular and ventricular origin. Let's now review what might result in wide QRS complexes

- Conduction with a *bundle branch block pattern*: if QRS complexes are wide, one should always check lead V1. If the dominant deflection of the QRS complex is negative here, it is left bundle branch block (LBBB), while if that is rather positive, it is right bundle branch block (LBBB). The impulses depolarizing the ventricles are supraventricular in both cases, it is only the sequence of ventricular conduction that changes and its duration becomes prolonged, thereby widening the QRS complex.
- A special case is *WPW syndrome*, during which slow conduction of the region of the ventricles having been depolarized by the abnormal atrioventricular pathway is responsible for the development of delta waves and occasional widening of the QRS complexes. Impulse formation is also supraventricular in such cases.
- Impulses of *ventricular origin*: each beat and rhythm arising from the ventricles is accompanied by wide QRS complexes. The reason for that is that these impulses get to each cardiac cell not through the normal cardiac conduction system, but via the myocardium, which has relatively slow conduction properties. Based on the above, there is widening of the QRS complexes both for ventricular premature beats, ventricular escape rhythm, accelerated idioventricular rhythm and ventricular tachycardia.
- A special situation is pacing by a *pacemaker*, during which the ventricular lead is situated in the right ventricular apex. In such a case, the left ventricle is activated from the right ventricle just as in LBBB, and this is why the QRS morphology in right ventricular pacing reminds of that seen in LBBB.

As a next step, let's examine if there are any <u>pathological Q waves</u>:

- The most important features of these include that they appear in at least two contiguous leads, they are deep (≥ 25% of the amplitude of QRS complexes) and/or wide (≥ 0.04 sec, i.e. one 'small square'); these may indicate the presence of a previous myocardial infarction (infarct scar).

One should dispense with the analysis of the ST segments in the vast majority of cases if there is shortening of the PQ interval (WPW syndrome) or the *QRS complexes are wide*, because ST segment deviation occurring in such cases is only a *secondary abnormality*, i.e the sequence and velocity of repolarization will also change secondary to the abnormal depolarization sequence and slowed conduction. Otherwise, analysis of the ST segment should be performed as described in chapter called 'ECG signs of ischemia'. For ST segment depression, a deviation of 0.5 mm should already be indicated, just as that of 1 mm (~2 mm in leads V1-3) for ST segment elevation because they may imply the presence of ischemia.

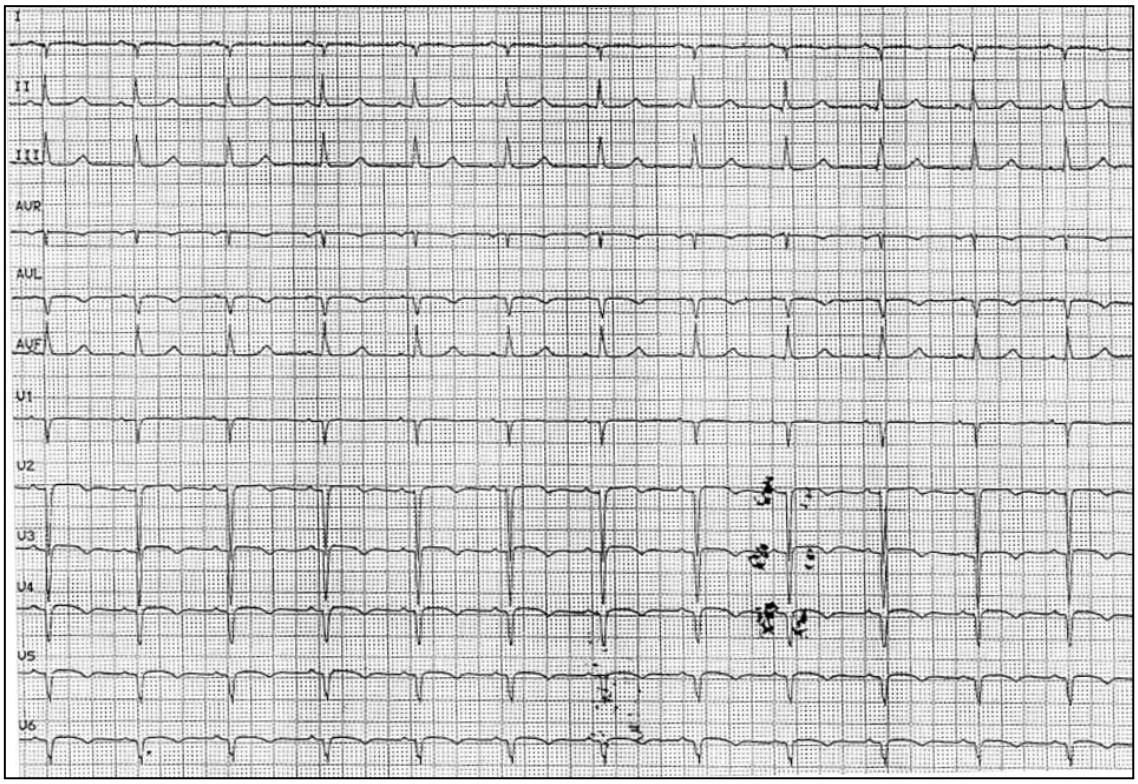
T wave abnormalities are often nonspecific and should be evaluated together with the patient's complaints.

For example, there is different importance of negative T waves in a 30-year-old female with stabbing chest pain and deep T wave inversion in a 70-year-old male smoker with typical angina.

2. If there are no P waves and the occurrance of QRS complexes is irregular, one should think of the presence of atrial fibrillation (atrial flutter) as one of the most common arrhythmias that should obligatorily be recognized by everyone. If QRS complexes appear in a regular pattern, one should go through the algorhythm used in the differential diagnosis of supraventricular arrhythmias. For a regular supraventricular tachycardia with a heart rate around 150 bpm, the presence of atrial flutter with a 2:1 conduction block is most likely. For a regular narrow QRS complex tachycardia in a young patient, one should think of the presence of AVNRT in females as the most likely underlying cause, while of AVRT in patients with known WPW syndrome (and in males).

Practice session for differential diagnosis

1.



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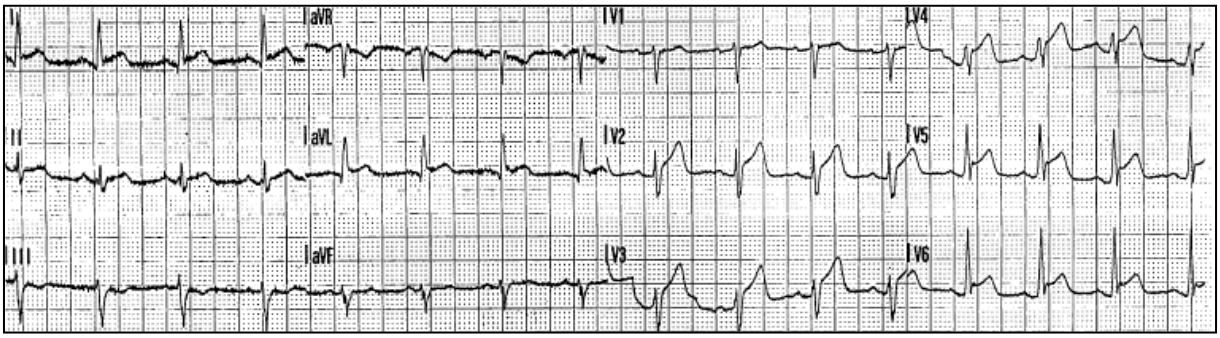
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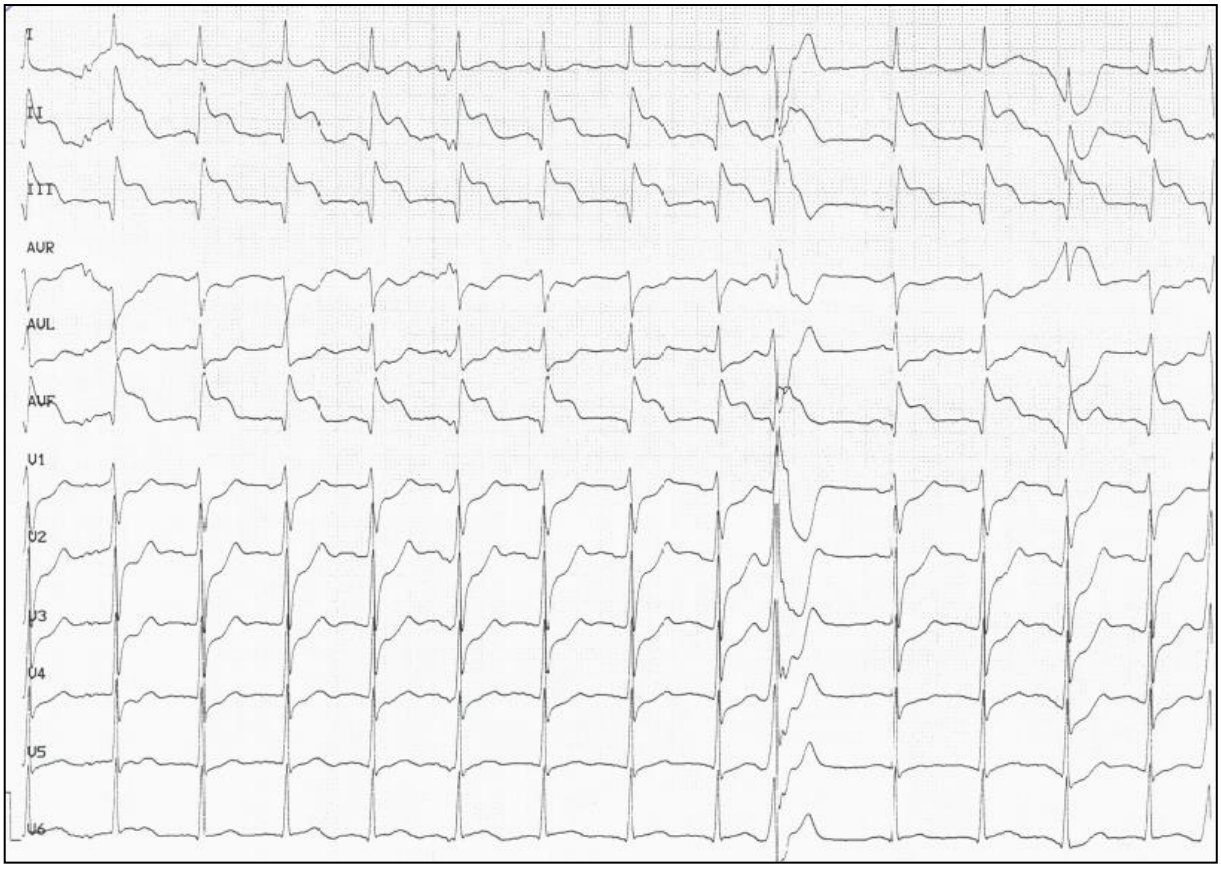
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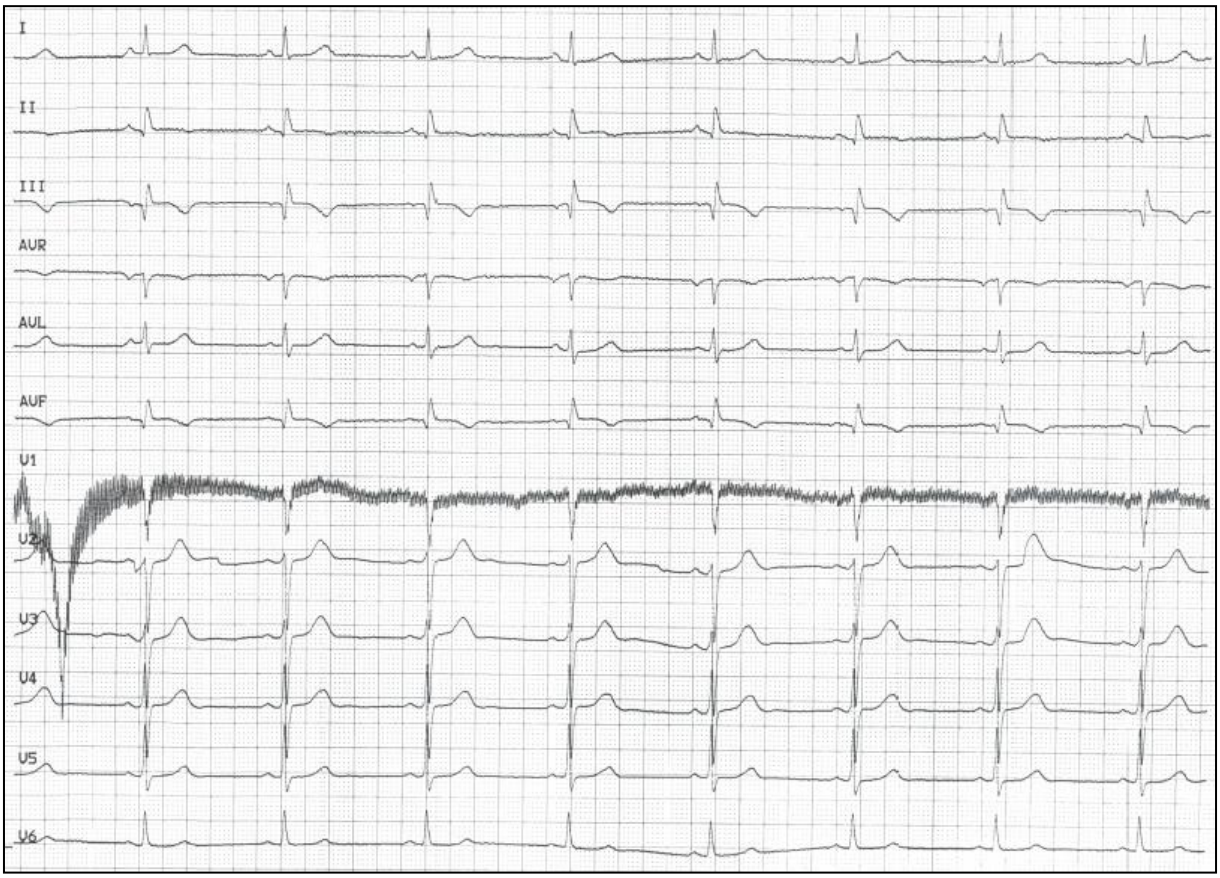
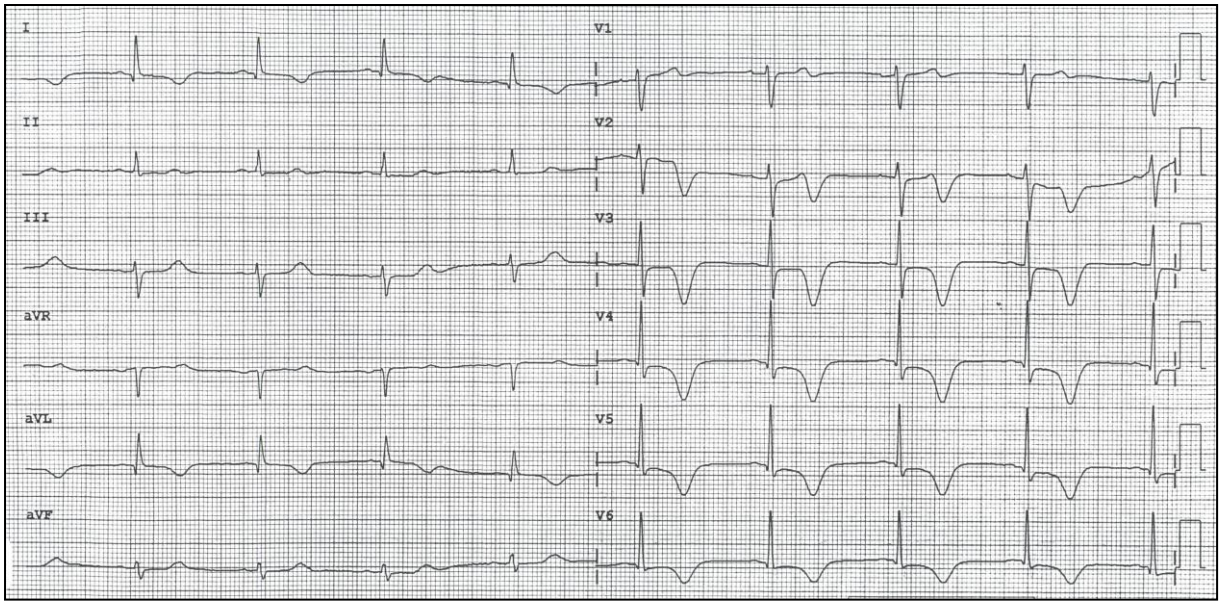
4.


I
II
III
AUR
AUL
AUF
U1
U2
U3
U4
U5
U6

5.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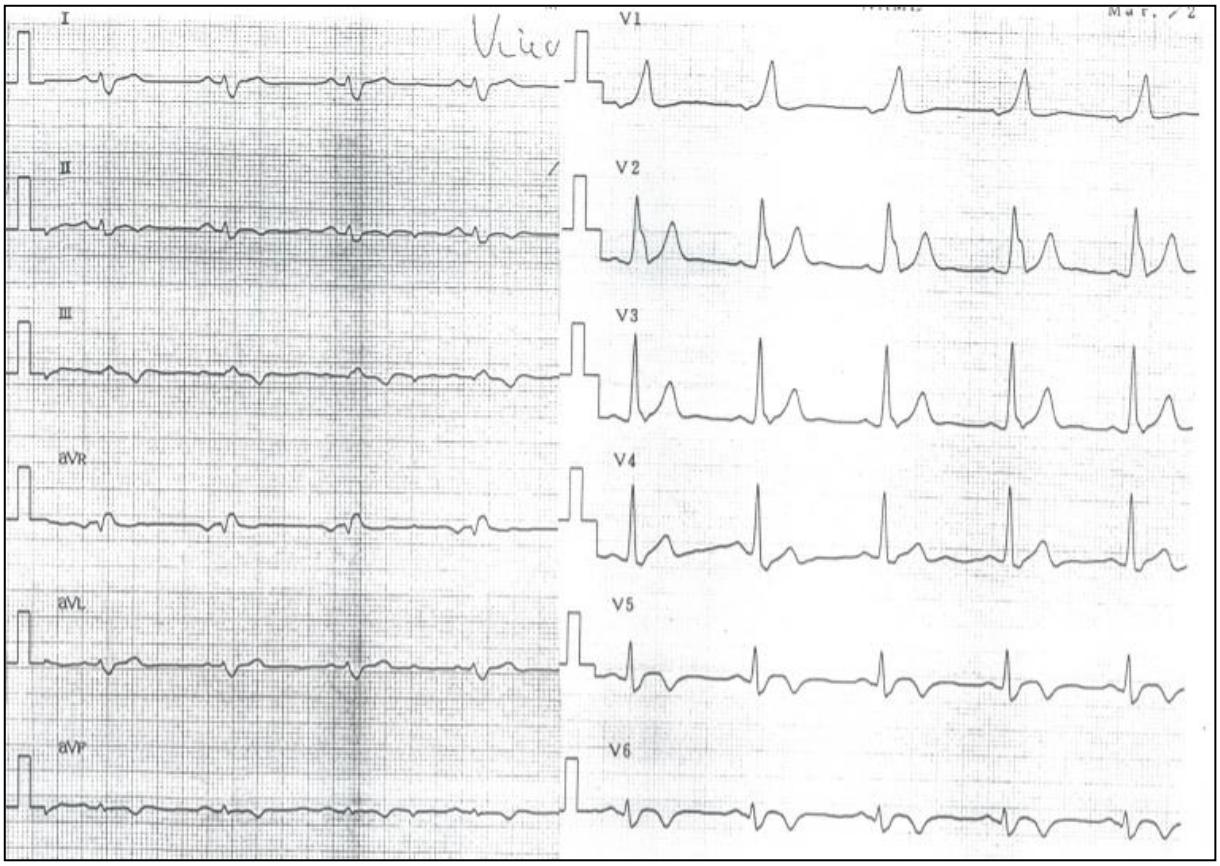
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

6.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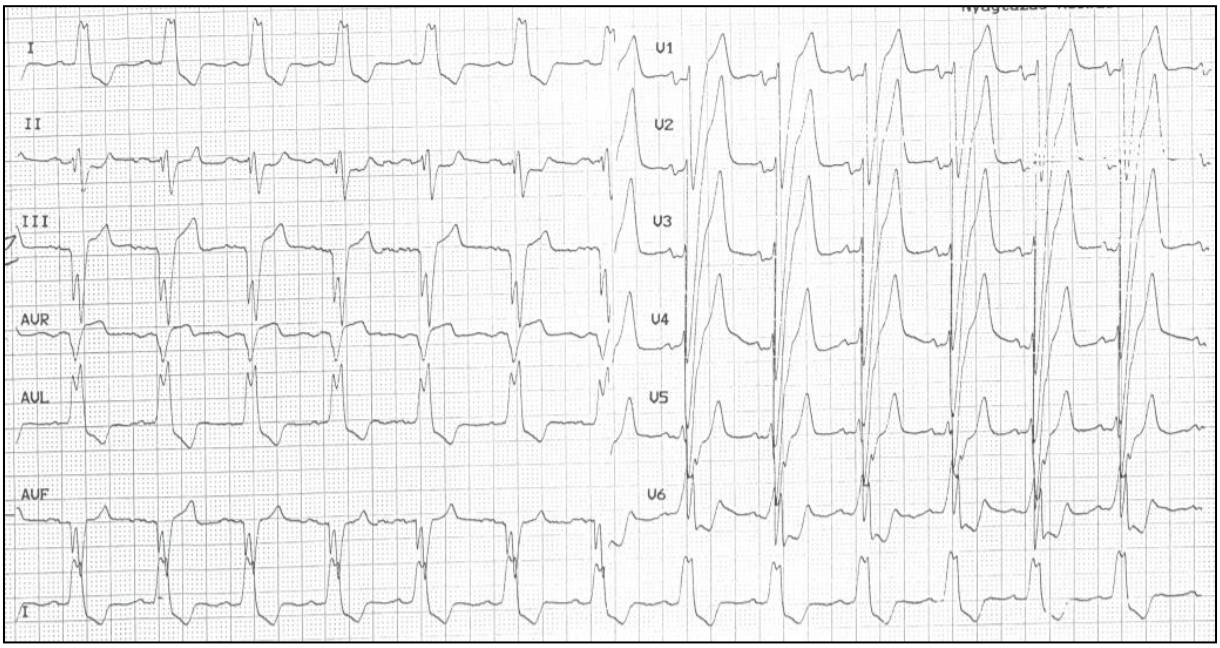
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

7.


I
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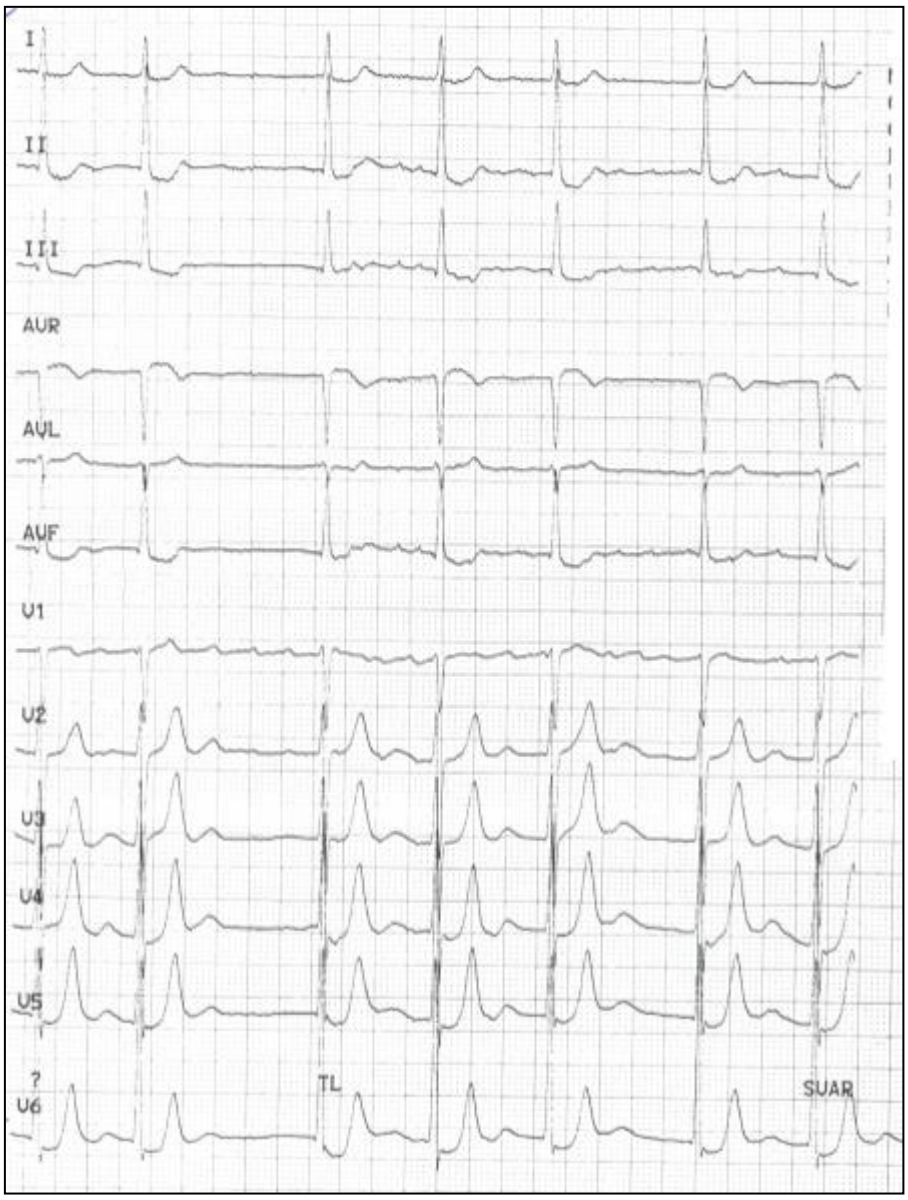
AUL
AUF
I
U1
U2
U3
U4
U5
U6

8.


I
II
III
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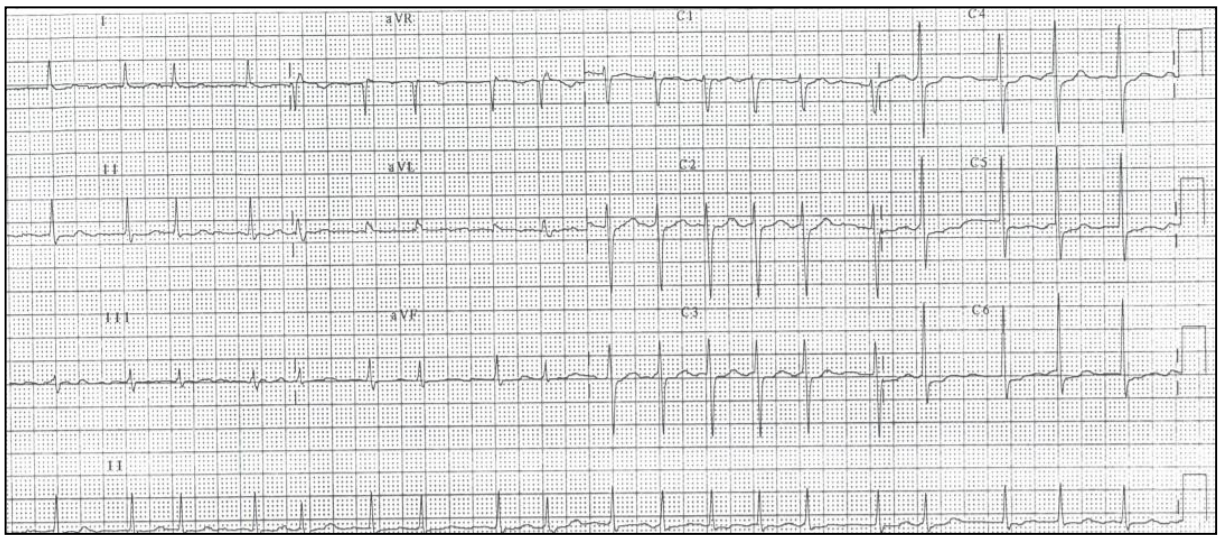
AUL
AUF
U1
U2
U3
U4
U5
?
U6
TL
SUAR

9.


I
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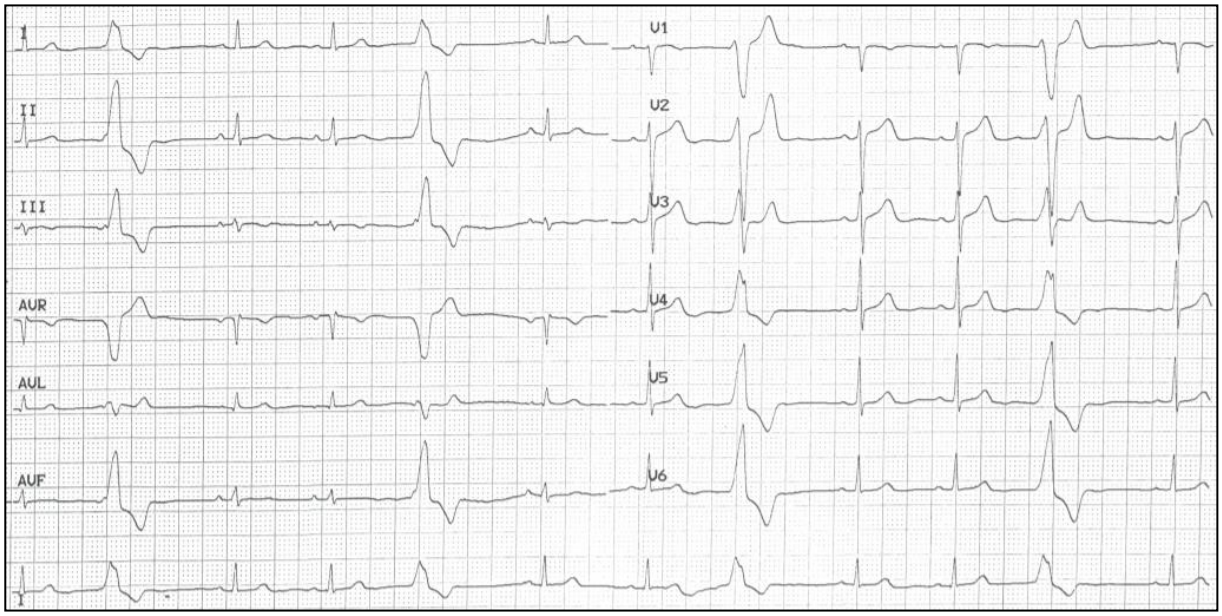
C1
C4
II
aVL
C2
C5
III
aVF
C3
C6
II

10.


I
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AUF
U1
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U3
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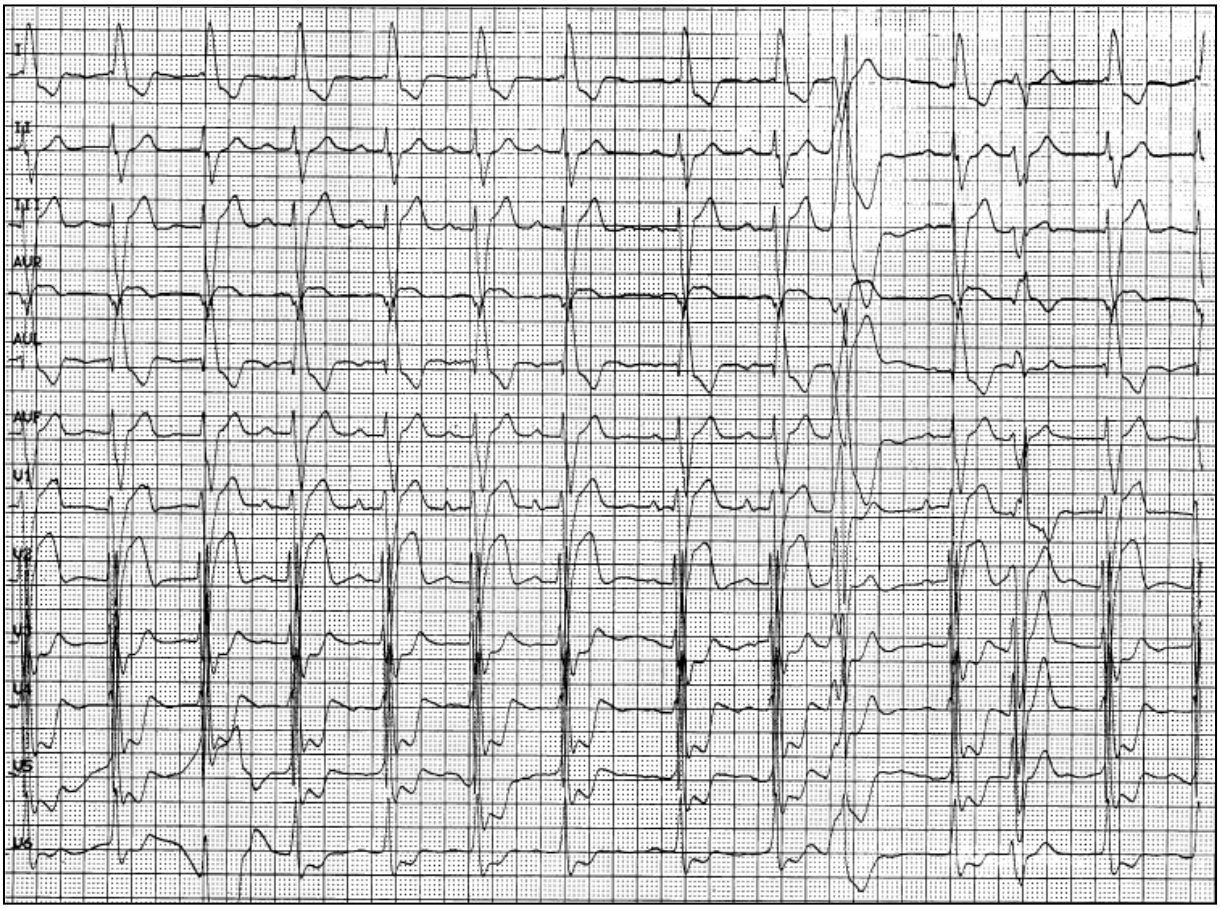
U6

11.


I
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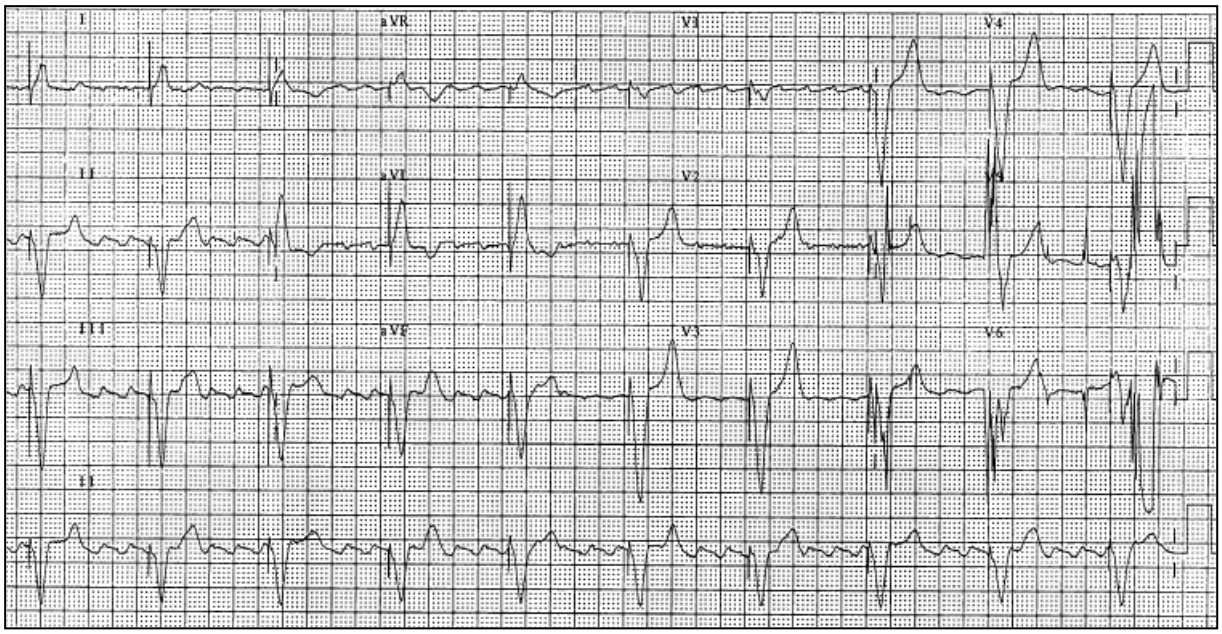
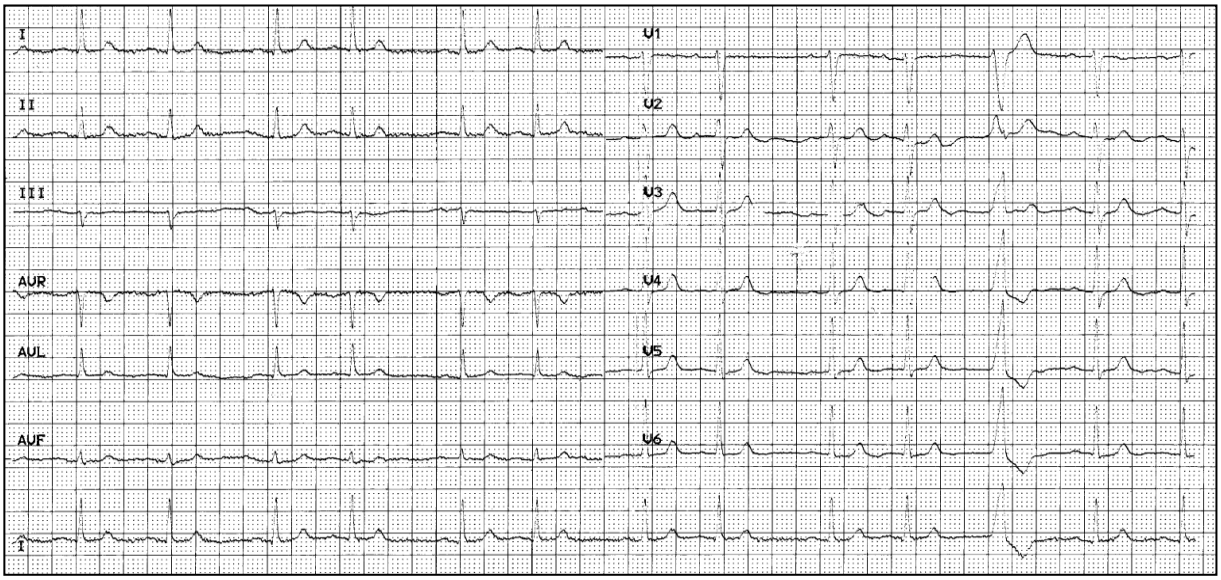
12.


I
aVR
V1
V4
II
aVL
V2
III
aVF
V3
V6
II

13.


I
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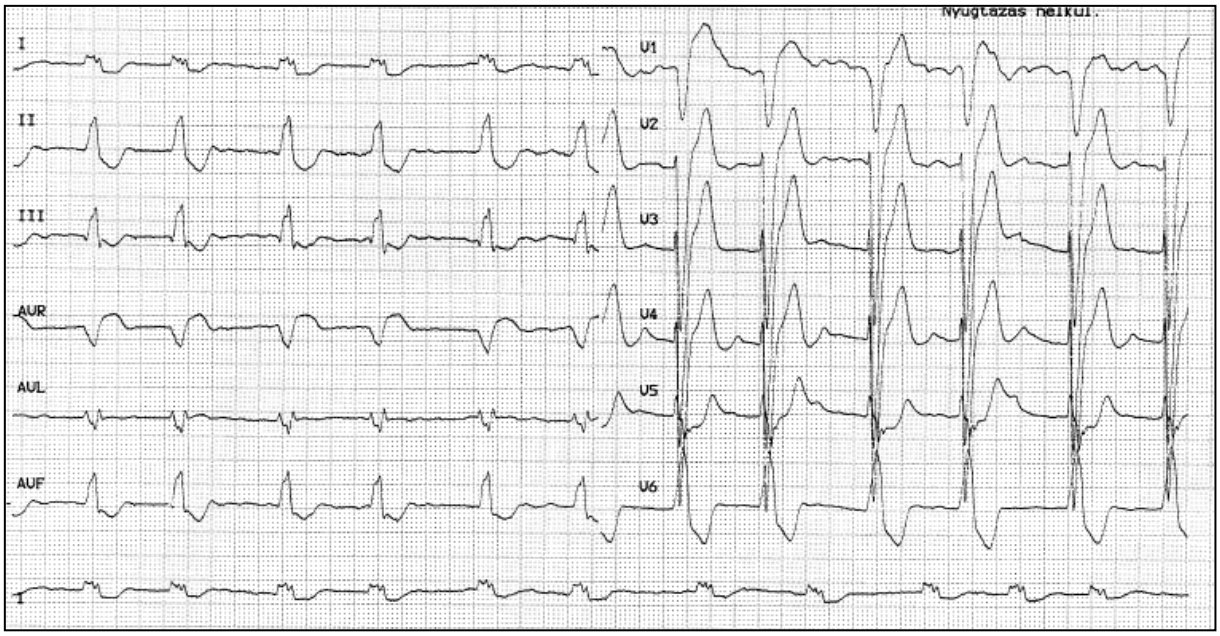
U4
U5
U6

14.


Nyugtázás nélkül.
I
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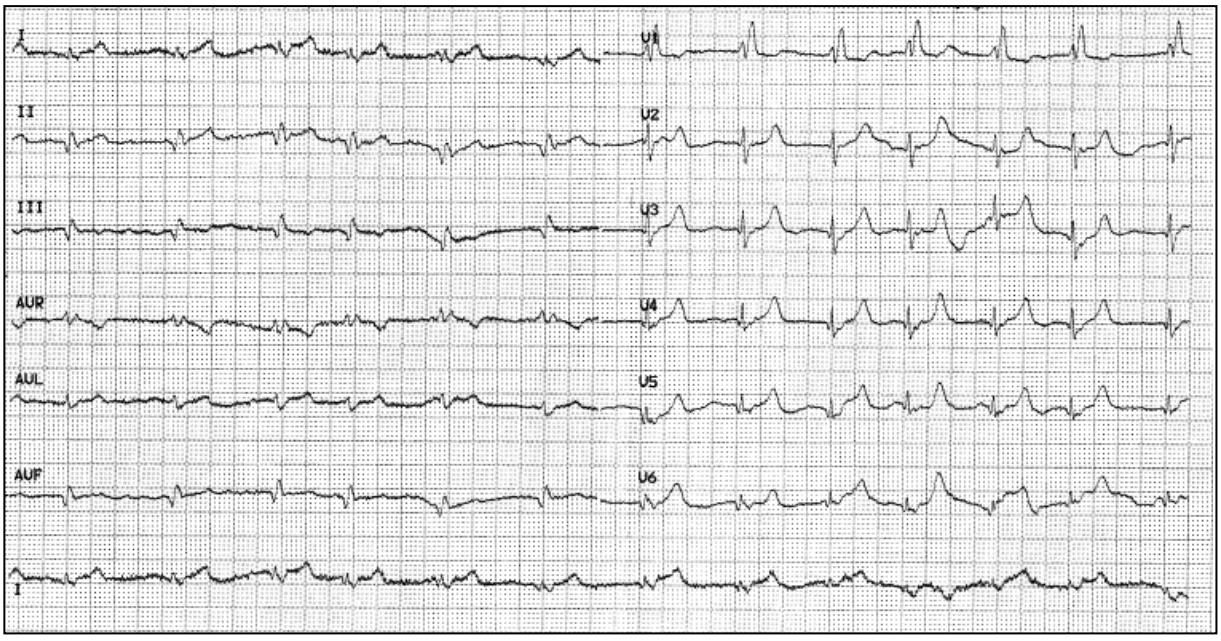
V1
V2
V3
V4
V5
V6

15.


I
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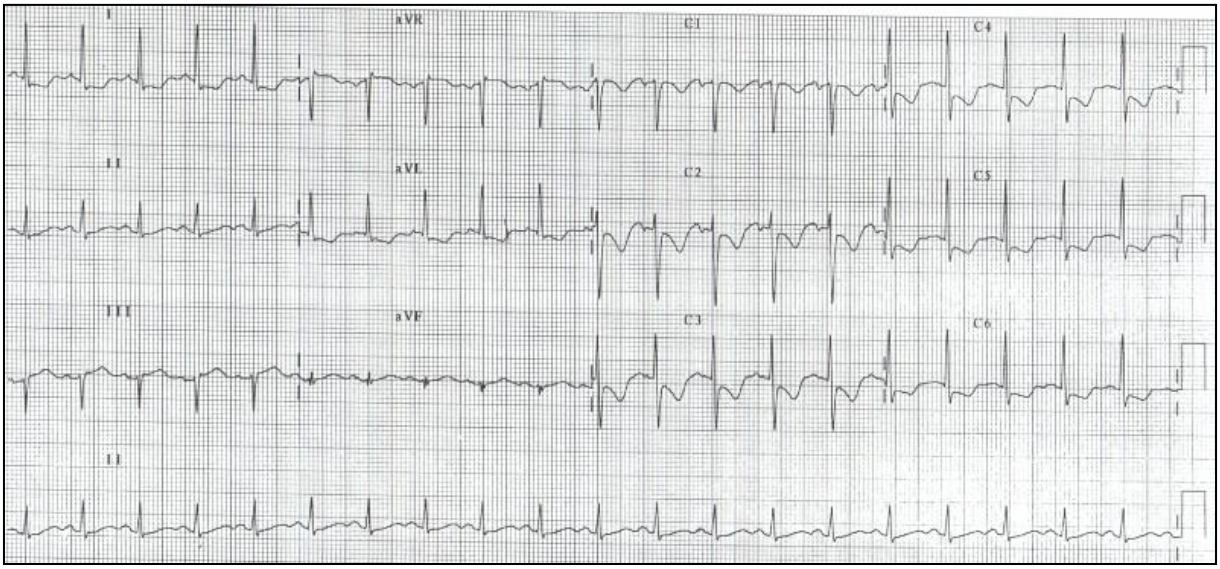
V4
V5
V6

16.



ECG reports

1. Sinus rhythm, 72 bpm, right axis deviation, normal AV conduction time, narrow QRS complexes, QS complexes in leads I, aVL and V1-6, negative T waves in leads V1-5, isoelectric ST segments - Summary: ECG pattern of infarct scar of an extensive anterior myocardial infarction, right axis deviation.
2. Sinus rhythm, 95 bpm, left axis deviation (left anterior fascicular block), narrow QRS complexes, small (physiological) q waves in leads I and aVL, dome-shaped ST segment elevation of 1 to 4 mm and positive T waves in leads I, aVL and V2-6 - Summary: acute extensive anterior myocardial infarction, left anterior fascicular block.
3. Sinus rhythm, 82 bpm, normal QRS axis, normal AV conduction time, Q waves, significant dome-shaped ST segment elevation and positive T waves in leads II, III and aVF, reciprocal ST segment depression in leads I, aVL and V1-4, a ventricular premature beat - Summary: acute inferior myocardial infarction, ventricular premature beat. (Mild fluctuation of the baseline is detectable in some parts of the tracing - motion artifact.)
4. Sinus rhythm, 50 bpm, normal QRS axis, normal AV conduction time, pathological Q waves and negative T waves in leads II, III and aVF, isoelectric ST segments, otherwise normal ventricular repolarization - Summary: ECG pattern of scar from an old inferior myocardial infarction. (Oscillations of the baseline in lead V1 is only noise and not produced by the myocardium.)
5. Sinus rhythm, 50 bpm, left axis deviation, normal AV conduction time, trivial (of 0.5 to 1 mm) ST segment depression and classic deep T wave inversion (i.e. negative, symmetrical and peaked T waves) in leads I, aVL and V3-6 - Summary: ECG signs of ischemia, probably due to severe stenosis of a large epicardial coronary artery.

6. Sinus rhythm, 60 bpm, normal QRS axis, normal AV conduction time, right bundle branch block, secondary repolarization abnormalities. - Summary: right bundle branch block.
7. Sinus rhythm, 75 bpm, left axis deviation, normal AV conduction time, left bundle branch block, secondary repolarization abnormalities. - Summary: left bundle branch block.
8. Atrial fibrillation, 72 bpm, normal QRS axis, normal ventricular conduction (narrow QRS complexes, no pathological Q waves), horizontal ST segment depression of 1 to 1.5 mm and positive T waves in leads I, II, III, aVF and V3-6 - Summary: atrial fibrillation, ECG signs of ischemia or left ventricular volume overload.
9. Atrial fibrillation, 120 bpm, normal QRS axis, normal ventricular conduction and repolarization - Summary: tachyfibrillation.
10. Sinus rhythm, 67 bpm, normal QRS axis, normal AV conduction time, normal ventricular conduction and repolarization, ventricular premature beats - Summary: ventricular premature beats in a trigeminal pattern.
11. Sinus rhythm, 78 bpm, AV conduction time: 0.22 sec, left axis deviation, left bundle branch block, secondary repolarization abnormalities, a ventricular premature beat - Summary: bifascicular block (1st degree AV block, left bundle branch block), a ventricular premature beat.
12. Ventricular paced rhythm (wide QRS complexes with an LBBB morphology), 60 bpm, normal QRS axis, wide QRS complexes, secondary repolarization abnormalities Please note that the underlying rhythm depolarizing the atria is atrial flutter. - Summary: VVI pacemaker rhythm, atrial flutter.
13. Sinus rhythm, 70 bpm, left axis deviation, normal AV conduction time, normal ventricular conduction, R wave transition zone in lead V3, normal ventricular repolarization, an atrial and a ventricular premature beat - Summary: a PAC and a VPB.
14. Atrial fibrillation, 70 bpm, normal QRS axis, left bundle branch block, secondary repolarization abnormalities - Summary: atrial fibrillation, LBBB.
15. Atrial fibrillation, 80 bpm, normal QRS axis, pathological Q waves in leads II, III and aVF, right bundle branch block, secondary repolarization abnormalities - Summary: atrial fibrillation, ECG pattern of scar from an inferior myocardial infarction, right bundle branch block.
16. Sinus tachycardia, left axis deviation, normal AV conduction time, normal ventricular conduction, descending ST depression of 2 to 3 mm and negative T in leads I, II, aVL, V2-V6 (ECG recorded during angina pectoris.)

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