



COMENIUS UNIVERSITY IN BRATISLAVA
JESSENIUS FACULTY OF MEDICINE IN
MARTIN



ELECTROCARDIOGRAPHY

**Dysfunction of the electrophysiological processes in the heart –
mechanisms and manifestation**

Textbook for students of the 3rd year – background for the seminars related to the electrocardiography

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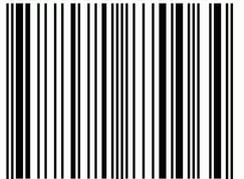
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1.0 PREFACE

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It is generally known that heart works very effectively during rest condition; however it can increase its output according to tissue oxygen and substrate demand in couple of seconds. This quick and effective process is an evidence of perfect nervous and humoral regulation. Regulation of the heart activity is fascinating. Detailed study of the heart physiology clearly points out the perfection of this phenomenon at one side, but also a complexity of it at the other side. Complexity of this regulation may be also the background for the delicacy of these processes. One of the manifestations of “imperfections” in this complex process could be quite common presence of disturbances of electrical and/or mechanical work of the heart.

During our entire practice as a teacher of pathophysiology, we frequently faced the lack of knowledge in students on the heart function during pathological and even during physiological conditions.

The most problematic part seems to be interpretation of ECG curve caused by disturbances of the impulse production or conduction and many more processes. Many of our students managed to understand the ECG thanks to their endurance and the help from the teachers during the pre-gradual study. Some of them, however, never broke the barrier of understanding of ECG and they only relied on their ability to memorize and remember the most important changes of the electrocardiogram. Memory used to be bad servant sometimes, and it may happen that the remembered figures vanish with the time. Memorizing instead of understanding ECG is not the best way how to manage one of the most important examination methods in cardiology, and it was frustrating for both students and teachers. The lack of knowledge at the student side was frequently explained as a consequence of not well written literature (textbooks) that would be student-friendly and that would explain electrophysiological processes in the heart with their manifestation on the ECG curve. The retention of knowledge can only be achieved by understanding of the task rather than the memorizing textbooks. This was main motivation for preparing better teaching textbook on ECG.

The main objective of this textbook is to explain the mechanisms leading to the typical electrophysiological changes in the heart in physiological and pathological conditions and also to show the manifestation of these processes on the ECG curve.

The authors tried to avoid using mathematical and electrophysiological terms in the text. This will lead to some degree of simplification of all tasks; however, the authors do believe that simple explanations can be more effective for understanding than sophisticatedly written text. The authors do believe that this small textbook can be useful and can help the students to “read” electrocardiogram properly. We all know the situations in cardiology when quick decision is necessary to save a life.

2.0 STRUCTURE AND ELECTROPHYSIOLOGY OF THE HEART

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Since the students in the third year of medical study successfully passed the anatomy, histology and physiology exams, and they know where to find relevant information about this task, we will only point out some important features, which are necessary for understanding of the electrical processes in the heart.

The main tissue in the heart is **striated musculature** (myocardium), which has some of the features of the **smooth muscle** (for example, it cannot be controlled voluntarily). Myocardium does belong to the **excitable tissues** of the body. The cells of the myocardium are polarized, which means that there is a difference among positively and negatively charged ions between internal and external side of their membrane. The basic structural component of the myocardium is a **myocardial cell**. These cells have an ellipse shape and they are connected into a kind of network by the end to end pattern. These connected cells form a **myocardial fibre**. Myocardial cells are connected into a unique entity which is called **functional syncytium** (cellular unit). This specific connection of the cells is not only morphological, but functional and it enables rapid activation (electrical and later mechanical as well) of the myocardium of the atria (left and right atrium are synchronized when it comes to the electrical activation) and ventricles (with a little delay necessary for ventricular filling). Activation of both ventricles is synchronized too. This synchronized activation is the basal condition for the optimal work of the heart as a pump. Existence of such synchronicity is caused by

a) specialized conductive system in the heart

b) cell-to-cell connections with the low resistance located in the intercalar discs, they are called “nexus” or gap junctions. Electrical conductivity between the neighbouring cells is very good. Thanks to these structures and connections, the heart reacts to the electrical impulse as a unit in very short time (atria are depolarized within 0.1 second from the SA node, so are ventricles).

When it comes to the electrophysiology, we can clearly distinguish two types of the cells in the heart. **Working myocardial cells** (the musculature itself) and the cells of the **specific excitation and conduction system**. The working myocardial cells normally do not have the ability to produce impulses spontaneously (automatically) without external trigger. They cannot depolarize spontaneously under normal conditions, but they are depolarized by the electrical impulse generated in the specific excitable cells – pacemaker cells. Why is that? These cells are different not only morphologically. The cells in the excitable and conductive system have less contractile elements and mitochondria, they have the chain of enzymes enabling them to produce energy by glycolysis, but most importantly they have different membrane. The membrane of the cells in the excitable and conductive system of the heart contains “slow sodium channels” which are responsible for ability of spontaneous depolarization. Sodium flows to the cells thus decreasing their membrane potential (towards zero) until it reaches the value which can open quick sodium channels, which in turn produces action potential (or impulse). These cells have special enzymatic equipment, which make them less sensitive to the lack of energy, and their performance in case of reduced oxygen delivery is much better than cardiomyocytes.

Specialized excitable and conductive system of the heart consist of sinus node (SN), preferential conduction pathways in the atria, atrio-ventricular node (AVN), bundle of His, bundle branches (left and right) and Purkinje fibres (Fig. 1).

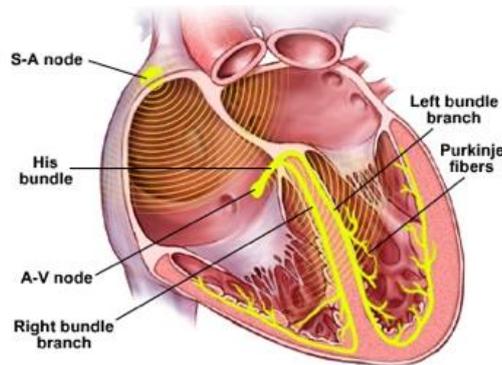


Fig. 1. Excitable and conductive system of the heart

The cells in this system have several differences when compared to the working cardiomyocytes. The most important differences necessary for understanding ECG are:

- **Ability of spontaneous depolarization (SD)** – it is a depolarization during the heart diastole which increases the membrane potential to the value of the threshold potential that will open the rapid sodium channels and allows the quick Na^+ influx, leading to the action potential onset. This impulse spreads through the myocardium of the atria causing its depolarization (which is followed quickly by a contraction too). The cells of the conductive system do not contract much, as they lack the contractile proteins. The speed of spontaneous depolarization in different parts of the conductive system is different. The fastest depolarization is present in the cells of the sinus node, bit slower in the cells of the AV node and the slowest spontaneous depolarization have cells of the Purkinje fibres in the ventricles.

Therefore, we can see the hierarchy in the ability and speed of spontaneous depolarization from the sinus node, down through the AV node, finally to the Purkinje fibres. This orchestration must be preserved, because if not, it may lead to dysrhythmias. The speed of the spontaneous depolarization is related to the **value of the resting membrane potential (RMP)**. Cells with the low RMP have higher ability to depolarize spontaneously than the cells with the higher RMP. This is caused by the features of the cell membrane which contain voltage gated ion channels. These channels open after the repolarization of the cells reaches certain value and positively charged ions start “flowing” through the membrane and it starts depolarization process. This type of depolarization is the fastest in the cells of the sinus node, that’s why the sinus node is the structure that drives and controls the rate of the depolarization/contraction cycles in the entire heart.

Although the cells of the working myocardium cannot produce impulses (cannot depolarize spontaneously) under normal conditions, they can get these abilities during pathological conditions. What happens to these cells? One of the reasons for this abnormal behaviour of working myocardial cells is the reduction of their membrane potential below -50 mV. This happens when the cells are damaged for example by

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hypoxia, ischemia, inflammation, hyperkalaemia or some other pathological processes. These mechanisms will be analysed in greater details in the particular sections of this textbook.

Note: When we say that RMP is decreased, we mean that it has less negative value, it is closer to zero. And vice versa, shift of the membrane potential to more negative value is considered as increase. Its absolute value increases actually. From the mathematical point, these statements are confusing, but they are generally accepted in electrophysiology, and we must get familiar with them to understand ECG. So, movement of the potential towards zero is the decrease of it.

- **Heart tissues do not have good conduction abilities.** This disadvantage is minimized by some other cells properties, for example in the cells with the high RMP conduction from cell to cell leads to the production of the “new” action potential (new impulse) in every single cell, therefore, the **value of the depolarizing potential does not decrease with the number of depolarized cells. This is called depolarization without decrement.** This phenomenon does not apply to the cells with the low membrane potential. In this case the impulse will spread with decrement, what means that it can vanish along the way. This could be one of the mechanisms leading to heart blocks. Cells in different parts of the conductive system have different values of resting membrane potential and this is the reason why they have also different conduction speeds when carrying the impulse to the next parts of the conductive system or to the myocardium itself. This is attributed again to the value of their membrane potential. Why is that? Let’s take a look at this simple explanation.

Parts of the conductive system containing cells with the high RMP can conduct impulses quickly, parts containing cells with the lower RMP conduct impulses more slowly. If the RMP of the cells is very low, they cannot conduct the impulse at all (the conduction will be blocked). One of the main drives for the depolarization speed is the difference of potentials between already depolarized and not yet depolarized part of the cell (or the muscle fibre). Huge difference of potentials leads to the quick conduction of the impulse from cell to cell (except from the conduction through the nexus) in a way of local electrical currents.

The background for the ECG curve are the currents present in the myocardium during depolarization and then repolarization of the cardiomyocytes spreading through the extracellular space in the direction of depolarization. These local currents reduce resting membrane potentials of the cells located in front of the depolarization wave, thus preparing them for upcoming depolarization. Therefore, the difference between the depolarized and not yet depolarized parts of the heart leads to formation of local electrical currents which increase the speed of conduction. Local currents can be effective to a distance of couple of millimetres what means that the impulse can “skip” couple of cells with decreased conductivity e.g. damaged cells (the length of the cell is 50 -100µm). This mechanism can be useful in the spreading of the impulse to the parts of the heart located behind the damaged field.

- **Preferential conduction pathways in the atria** – the cells forming these pathways differ from the working cardiomyocytes and also from the cells of conductive system, and it is possible to identify three fascicles –

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frontal (Bachman's), middle (Wenckebach's) and dorsal fascicle (Thorle's). These fascicles cannot be considered as internodal, because they do not begin straight in the sinus node, and they do not terminate straight in the AV node. Their main role is to provide fast and synchronized depolarization of the atrial musculature. Even though, they cannot be excluded as a potential source of escape depolarizations in the case of the sinus block, or sinus arrest (secondary pacemakers).

- **Delay of the impulse in the AV node** – it is physiological delay of the conduction from the atria to the ventricles in the AV nodal conductive tissue. **The main role of this delay is to provide an optimal time for the ventricles to be filled with the blood from the atria**, that's why the depolarization of the ventricles starts later after the previous depolarization of the atria. Therefore, the contraction of the ventricles will start after the ventricles achieve optimal end-diastolic volume. Functional background for the nodal delay is the existence of the cells with very low resting membrane potential (in the central part of the AV node the cells have potential only -50 mV). Activation of these cells by the depolarization wave coming from the atria down to the ventricles opens only the slow sodium channels; therefore, they only generate an action potential with the low amplitude. This will reduce the speed of conduction (as we explained that the amplitude of the membrane potential and action potential determine the conduction speed). Conduction speed in this part of the conduction system is approximately 0.02 m/sec. This is the place in the heart with the slowest conduction.

- **Other specific functions of the AV nodal part**

The AV nodal junction is the only electrical connection between the atria and ventricles in physiological conditions. This is the only place where depolarization can spread to the ventricles, otherwise the atria and ventricles are electrically separated (isolated). Damage to this zone leads to disconnection and the impulse would not reach the ventricles. This is the reason why the AV zone has a specific architecture that guarantees a higher degree of safety of conduction to the ventricles. Simplified description would say that the number of the conducting fibres decreases in the direction from the atria to the His bundle. This principle of the organization of the conductive fibres is called **convergence**. The architecture provides conditions for **one-directional** (forward) spreading of the impulse from the atria to the ventricle without decrement, and also it **worsens or disables the retrograde spreading of the depolarization from the ventricles back to the atria**. In case of the retrograde spreading, the impulse should depolarize higher number of fibres and this would lead to the decrease of its magnitude to the value when it would not depolarize the other cells any longer – therefore the conduction will stop.

- **Conductive system in the ventricles**

This system consists of **right and left bundle branches and the Purkinje fibres**. It is important to note that this system has very high conduction speed (**4 m.s⁻¹**) **and the action potential in these cells lasts longer than in the neighbouring cardiomyocytes. These cells also have ability of spontaneous diastolic depolarization, but the rate of it is slow** (20-40 min⁻¹). To compare the rate of SDD in sinus node (>60 SDD per

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minute) **and the structures of the AV node** ($40-60 \text{ min}^{-1}$). Conduction from the Purkinje fibres to the cardiomyocytes has typical spatiotemporal pattern and it is the underlying condition for spreading of the contraction wave across the ventricular musculature. **Depolarization starts at the left side of the interventricular septum, approximately in its middle part and the depolarization spreads from the left to the right side of the septum. Later the depolarization spreads quickly across the ventricular musculature from the subendocardium to subepicardium.** Both ventricles are depolarized synchronously; however, the depolarization of the left ventricle takes a bit longer time. Depolarization starts at the apex of the heart and it is directed towards the basis of the heart (also the contraction of the ventricles starts at the apex and continues to the basis). **The last part of the left ventricle which is depolarized is the basal part of the left ventricle and the upper part of the septum.** The reason why depolarization of the left ventricle takes longer is that left ventricle contains bigger mass of the musculature when compared to the right side.

3.0 ELECTROPHYSIOLOGY - BACKGROUND FOR THE ECG CURVE

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ECG curve recorded from the body surface represents the summation of all action potentials of depolarized and subsequently repolarized cells of the myocardium. Depolarization of the cells in the conductive system is not displayed on the curve, because even after summation of all depolarizations it is below the detection threshold of the ECG recorded by body surface electrodes. Direct recording of electrical activity from the conductive system can be performed by the intracardial electrodes and is performed by specialized clinics.

The background for the ECG curve is the electrical current caused by the depolarization and repolarization of the myocardial cell. These currents are present inside of the cell, around it and they also “run” across the cell membrane (Fig 2A-E).

Fig. 2A. If the cells of the myocardial fibre are at rest (**diastole**) and if we measure the electrical potential by electrodes placed on the membrane surface we would get no changes of the potential, because the surface of the membrane has the same potential everywhere. When this device is attached to a recorder, we would get the flat line (isoelectric line – zero potential). As one electrode dives deeper into the cell and the other remain on the membrane surface, the voltmeter will immediately detect **negative potential**, because there is a dominance of the negative ions inside of the cell. Difference of the potential between internal and external sides of the membrane is 85-90 mV. It is a clear evidence that the membrane is polarized.

Fig. 2B. When the depolarization of the myocardial fibre begins and if both electrodes are outside of the membrane they will detect electrical activity which has two main phases. First is **depolarization** which means that the polarity of the membrane will reverse – the negative charge will be outside of the membrane and vice versa. The process of depolarization is quick, that’s why the recorded changes are short and the amplitude of the deflection is quite high (depends on the value of RMP and the number of depolarized cells). **The magnitude and the slope of the deflection depends on the RMP** – high membrane potential leads to quick and strong depolarization. The result of this process is also an onset of the potential change between the part of the fibre which has been depolarized already and the rest of the fibre which is not yet depolarized. **The potential change leads to the flow of the electrical current directed from the depolarized part towards not depolarized part.** Since this current is oriented towards the recording electrode, it will record positive deflection. This principle is important!

If the depolarization wave spreads towards the recording electrode, it will record positive deflection. If it spreads away from the electrode, then it will record negative deflection.

This is important for understanding of mechanisms leading to the positive and negative deflections on the ECG and also movements of the ST segment in a manner of elevation or depression.

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Fig. 2C. When depolarization wave “activates” all fibres, its polarity will be reversed along the entire length of the membrane. Inside of the cells, as well as outside of the cells, there would not be any difference of the potential between two electrodes, therefore we would not have any electrical currents. The electrode will therefore record zero potential (isoelectric) line. Immediately after the depolarization the myocytes contract so the electrical activity is immediately followed by mechanical force.

Fig. 2D. Repolarization starts at the same point where the depolarization has started. In our case it is the left side of the fibre. In the initial phase, the left side of the fibre is repolarized already while the right side of it is still depolarized. This difference will again lead to the onset of electrical current between these two places with different potentials. There is one important information – polarity at the both sides of the fibre is reversed, therefore the direction of the current in this case will be oriented toward the electrode at the left side. Current flows from the depolarized part to the part which is still repolarized. On the recorded trace it will manifest as a negative deflection. From the figure it is clear, that the **repolarization wave lasts longer and its amplitude is lower than the wave of depolarization**. The reason is that in fact repolarization is slower process – it consumes much more energy and the entire biochemical processes providing energy in this case are simply slower.

Fig. 2E. Repolarization process restores the physiological polarity of the fibre, potential differences vanish, therefore the recorded trace will return to zero. **It is important to note that repolarization currents do not cause the contraction of the cardiomyocytes because the cells are in the refractory phase and they cannot respond by neither electrical nor mechanical action.** These processes described above repeat every time the normally polarized cells are activated by the optimal impulse. Response to an activation is one of the typical features of excitable tissues.

The trace recorded from the single fibre is very similar to the ECG curve recorded during the ventricular electrical activity. It is not surprising, because the ECG curve (Fig. 3) is the sum of all action potentials present in all cells. Logically, the recording of the trace of whole heart (ECG) is much more complicated, because the heart itself is not only a fibre, but a complex organ. Anyway, the principles described in these figures are valid for whole heart and we will come back to them in many of our explanations.

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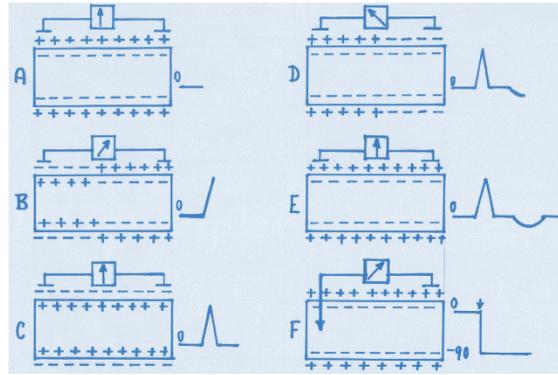


Fig. 2. Scheme of the depolarization and repolarization in the fibre of the cardiomyocytes and the recording of this activity by two electrodes placed on the “external” part of the cell membrane. F – resting membrane potential

Dominant pacemaker of the heart is the sinus node. Impulses produced by the cells in the sinus node spread across the atria in a radial direction (similarly to the waves on lake surface after thrown stone). Depolarization spreads through the atrial musculature and also through the preferential conducting pathways in the atria. **Final vector representing atrial depolarization is directed to the left, downwards and to the front.** It is recorded on the ECG trace as the P wave. It has typical duration (max 0.1 s) and amplitude (max 2.5 mV) and polarity (P wave can be positive or negative) in particular leads.

Depolarization spreads from atria to ventricles through the AV nodal zone (AV node, bundle of His, bundle branches and Purkinje fibres). Potential produced by the depolarization of the conductive system is too small to be recorded by ECG device. During activation of conductive system the trace shows PQ (PR) segment – depending on the first deflection of the ventricular depolarization (Q or R). PQ segment is measured from the end of the P wave to the beginning of the ventricular activity, while PQ interval involves also the P wave.

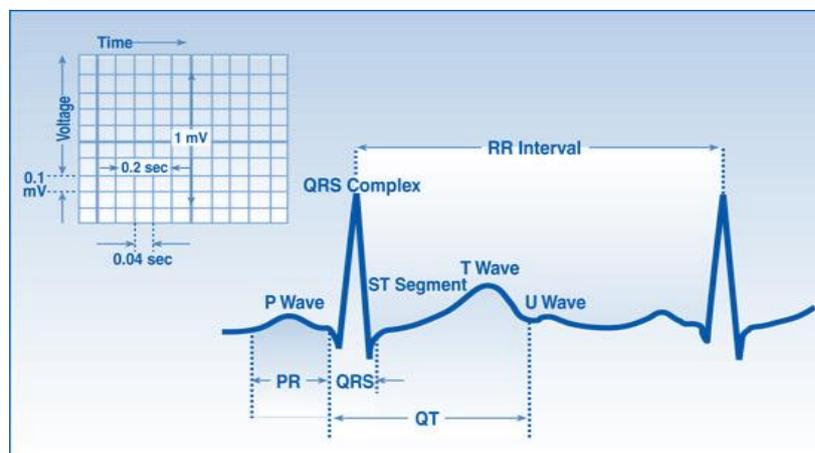


Fig. 3. Physiologic ECG trace - waves, intervals, deflections (Yanowitz FG, 2007)

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PQ segment lasts 0.1 s, while PQ interval lasts 0.12-0.20 s. It is important to measure whether it is shortened or prolonged, and it can also decline from the isoelectric line in case that it interferes with the repolarization potential of the atria. This is however very unusual. Normally, the atrial repolarization is hidden in the QRS complex. There are some conditions when atrial repolarization can be recorded on the trace – mainly in the case of atrial infarction or complete AV dissociation.

Depolarization of the ventricles manifest as a complex of fast deflections named QRS complex. It represents spatiotemporal pattern of the ventricular electrical activity. Both of these (direction and duration of this activation) are important sources of the information about the heart pathology. The pattern of the ventricular activation was described above, but it is important to note that amplitudes of the Q, R and S deflections are influenced by many factors – e.g. position of the heart in the chest, mass of the ventricular musculature, position of the recording electrode, conductivity (resistance) of the structures between the heart and the electrode, body temperature etc. Ventricular depolarizations ends with the S deflection in some leads.

Ventricular repolarization starts with the ST segment. This part of the trace normally copies the isoelectric line and it has horizontal position. It does not mean that there are no electrical currents in the heart during the ST. They are here, they run across the membrane and inward and outward currents are equal (Na^+ and Ca^{2+} inward, K^+ outward). In fact, ST segment is the representation of the “finalization” of the depolarization of the ventricular musculature. ST segment does not necessarily lie in the isoelectric line even in physiological conditions. Determination of the ST position is very important in recognition of many heart pathologies – change of the ST position (up or downwards) named elevations and depressions are typically found in ischemia (these changes will be described in details later). Sometimes it is difficult to determine whether the ST segment is or is not in the normal position. For that purpose it is very useful to determine the J point (junction point). The J point is a transition of the trace from the S or R to the ST segment. This point is situated at the same position as the onset of Q from the isoelectric line. Even on the ECG curve of healthy individuals this point can be elevated in the leads V_1 – V_4 up to 3 mm (0.3 mV if calibration matches 1 mV = 10 mm). In pathological circumstances, this J point is usually situated deep below this level (more info in the section myocardial ischemia).

Repolarization of the ventricles is represented by the T wave. Its amplitude, direction and duration are influenced by same factors as the QRS complex. Higher speed and higher magnitude of the repolarization current will lead to the peaked T wave with higher amplitude. This is the process in which the polarity of the cardiomyocytes achieves the resting values, but it does not mean that the ion composition of the intra and extra cellular space will become the same as well. It is important to consider that during depolarization considerable of Na^+ entered the cells, and considerable amount of K^+ left cells during the repolarization. Complete restoration of the ion balance is achieved during the heart diastole.

Quite common question of the students is **why the T wave has the same polarity as the main amplitude of the QRS complex** (concordant position of the T and QRS) in case that repolarization current has the opposite direction than depolarization current (explained on the stripe of the heart musculature in the previous section (Fig. 2). From what we already know this can sound confusing, because this would mean that

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repolarization starts at the spots where the depolarization had finished – and it is in the epicardial layers of the musculature. How is it then? We need to consider several facts. First, repolarization depends a lot on the availability of the energy and we know that for “energy resources” a cell need the oxygen and the substrates (blood flow). If we consider the availability of the oxygen and the nutrients we will understand that the lowest blood supply is in the subendocardial layer of the musculature, because this part is exposed to extremely severe intraventricular pressure during the repolarization (this phase of electrical activity fits to the mechanical systole of the ventricle), and this reduces blood flow via the intramyocardial branches of the coronary circulation. Contrary, subepicardial layer has much better blood supply and it is not exposed to strong mechanical force; also the intramyocardial arteries are not compressed as much as the vessels bringing the oxygen to the subepicardial layer. These are the reasons leading to the delay of repolarization in the subendocardial layers, so it starts in the epicardial layers and goes in reversed direction – back to the subendocardial layer. If this current is recorded on the trace, it will show concordant position of the T and QRS. If we speak about the one fibre or separated muscle string – then yes, repolarization “goes” in the same direction as depolarization, but we have to consider complexity of the heart architecture, and the mechanical forces influencing coronary circulation during the heart systole that lead to real delay of repolarization in the deep layers of the myocardial musculature.

3.1 QRS WAVES – TERMINOLOGY

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Deflections of the QRS complex represent depolarization of the ventricular musculature. In some leads they all can be detected, in some leads we may miss Q or S deflections, they also have different amplitudes in different leads, Why is that? Why the QRS looks different from lead to lead?

The reason is that the system of 12 leads is positioned on the body surface, and their recording of electrical activity of the heart from different spots, it means, that they “observe” depolarization and repolarization current, but always from different positions, and we discussed already what happens if the current spreads toward the recording electrode, and what happens if it spreads in reversed direction. **We can conclude that every single lead “records” the depolarization and repolarization waves from its own angle or aspect.** For example, lead aVR positioned on the right arm looks mainly at the atria and it records the currents produced by the atrial depolarization (P wave) - it is always negative in aVR; aVF located in the “inferior position” or the left leg looks to the heart from below and it will record positive P wave, because the currents goes towards it.

Different position of the electrodes (leads) and their ability to record the depolarization and repolarization waves is responsible for different directions and amplitude of the waves and deflection on the ECG trace in particular leads.

The principles applied to the terminology of the QRS complex are generally accepted, but they still seem to be a problem for medical students. This is the reason we decided to look at this task a little closer.

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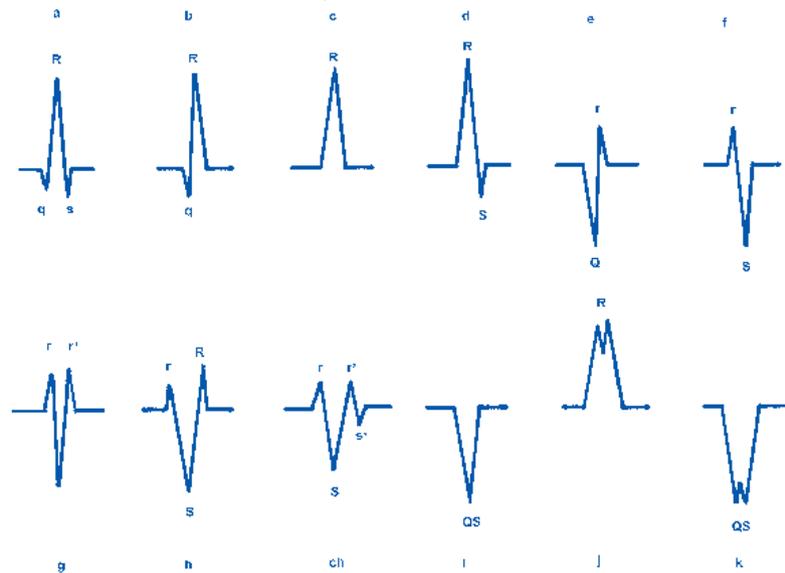


Fig. 4: Most common types of the QRS complexes and their terminology

Q is defined as the first negative deflection of the QRS complex. R is defined as the first positive deflection of the QRS complex. A negative deflection following the R is the S deflection. If there are two positive spikes with similar or equal amplitude, they would be R and R'. If there are two positive spikes, one higher, the other smaller they would be R and r (according to the amplitude – capital letters for the high amplitude deflections). Problematic are QRS complexes on the fig. 4 (g, ch) and k. In case of the 4i – students cannot decide, whether it is Q or S, since we have only one negative deflection. How to name the deflections then? This shape is recognized as QS. If a split of the wave lead to the onset of the new spike (negative or positive), but the line of the split does not cross the zero line, it cannot be recognized as real individual deflection. When the line of the split crossed the zero line, we then have a new deflection, and we will give it a name (RqrS). If the amplitude of the deflections is low, the names are small letters (q, r, s), if they have high amplitude, the names are capital letters (Q,R,S).

3.2 RECORDING OF THE ECG CURVE – SYSTEM OF LEADS

Standardized ECG record consists of 12 leads. Electrodes recording potential in those leads are positioned on the body surface. **They are 3 standard Einthoven bipolar leads (electrodes positioned on the upper and lower extremities), 3 augmented Goldberg unipolar leads and chest unipolar leads.** They are indirect leads, because the electrical activity is not recorded directly from the surface of the heart, but from the body surface. Except from these, there are more sophisticated methods, which can be used in case that special tests are needed e.g. recording directly from the heart surface, from heart chambers (direct recording), oesophageal electrodes (semi direct recording) and also from the less standard spots of the chest or high resolution ECG recording and many more. **All ECG leads record the same electrical activity of the heart, but**

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the traces are different, because every single lead has different position towards the direction of the depolarization and repolarization wave.

It is important to note polarity of the electrodes of bipolar leads whose electrodes are located on the extremities. In a very simple way it is possible to say that the electrode on the right hand is during the depolarization negative as first, because it is the first electrode which detects the electrical activity of the depolarized parts of the heart (depolarized parts of the right atrium – because here is the sinus node and it is main pacemaker in physiological conditions). Meanwhile, the electrode on the left hand is still “positive” – in lead I. The depolarization current runs from the right to left hand electrode. The last electrode that detects electrical activity is the electrode on the left leg, it means, it is “positive” towards both electrodes located on the hands – electrical current goes from the electrode on the left hand down to the electrode on the leg, and same way for the right hand side. The current goes from the electrode on the right hand down to the left leg (Fig. 5). Standard bipolar leads therefore records differences of potentials between the spots of the electrodes position, which are caused by the electrical activity of the heart. The Einthoven law declares that the amplitude of the potentials recorded in the lead II is a sum of potential recorded in the lead I and III. Standard bipolar leads do not “see” all parts of the heart perfectly, and even some part of the heart are „hidden” to those leads. They only record potentials in the frontal plane, that’s why the postero-anterior potentials are not recorded. Therefore, they are not able to record necessarily all pathologies in the electrical activity of the heart. These leads together form a triangle (Einthoven’s triangle) with the heart in the middle of it.

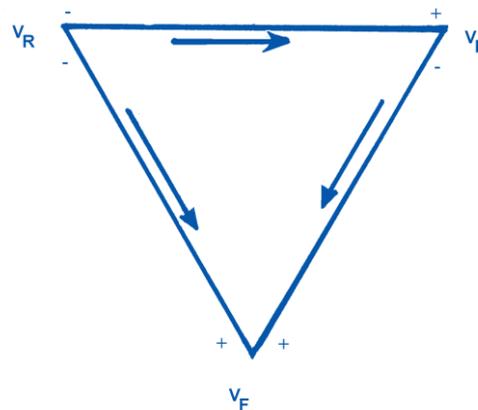


Fig. 5: Bipolar Einthoven leads and polarity of used electrodes

Unipolar Goldberg leads (augmented leads) Fig 6. record real value of the electrical potential in the place of their position – **aVR is located at the right arm, aVL on the left arm and aVF on the lower left extremity.** These leads were introduced by Goldberg and the names come from the abbreviations (augmented Voltage Right, Left, and Foot). The recording from the lead aVF is a sum of the activity recorded by leads II and III, similarly in case of aVL and aVR. All three electrodes are positive during the electrical activity of the heart and their polarity changes during the depolarization and repolarization. These leads also record electrical activity only in frontal plane. A combination of the Einthoven’s triangle (leads I, II and III) with Goldberg

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augmented leads form a hexagonal system which lets us to detect more information about the electrical processes in the heart.

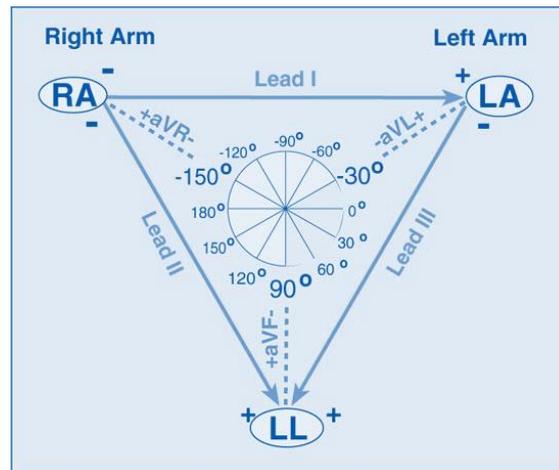


Fig. 6: Standard bipolar leads and unipolar leads (RA – right arm, LA – left arm, LL left leg (Yanowitz FG, 2007)

Unipolar chest (precordial) leads record electrical activity of the heart from the surface of the chest wall (Fig. 7). Therefore they record electrical activity of the heart from different points and angles and every single lead “sees” a bit different picture of it. These leads V1-V6 record electrical currents which spread through the myocardium in the horizontal plane in the antero-posterior and right to left direction.

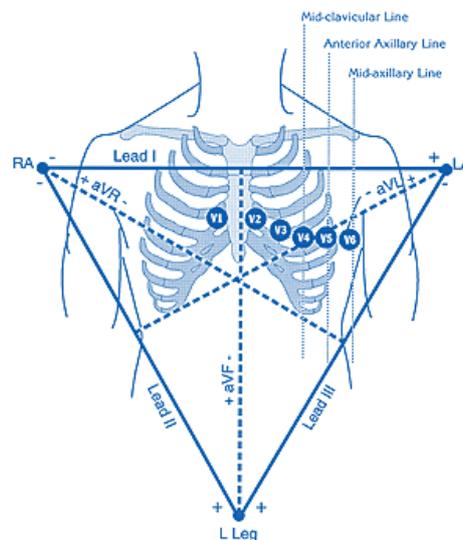


Fig 7: Precordial leads and their relationship to limb leads (Yanowitz FG, 2007)

They are positive, therefore, if there will be a depolarization wave, they will record positive deflection and when the depolarization goes away from them, they will record negative deflection. Leads **V₁₋₂** are

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positioned mainly above the right part of the heart, therefore they can “see” the changes of electrical activity in this part of the heart. Leads **V₅₋₆** are **positioned above the left ventricle** that’s why they can detect activity from the left ventricle. Electrodes **V₃₋₄** are **positioned above the anterior part and the interventricular septum** and they usually record equal amplitudes of the R and S deflections. Typical shape of the QRS complex in the leads V₁₋₂ is small r and deep S and the amplitude of the R increases while the amplitude of the S decreases towards V₅₋₆. These are typical physiological shapes of the QRS complexes in the precordial leads. It is determined by following factors:

- a) **position of the recording electrode** towards the depolarization/repolarization wave
- b) **mass of the musculature** in the septum, left and right ventricle – bigger musculature will produce higher potentials therefore higher amplitudes on the trace
- c) **spatiotemporal pattern of the depolarization and repolarization in the ventricles** (first septum, then ventricles and finally the basis of the ventricle from subendocardium to subepicardium).
- d) **position of the heart in the chest**

Figure 5D shows that potential produced by depolarization of the septum is directed towards V₁₋₂, that’s why the first deflection in these leads will be positive (small r and no Q or q in V₁₋₂. Leads V₅₋₆ record first deflection and it is negative q, because depolarization wave is heading to the V₁₋₂ and away from V₅₋₆.

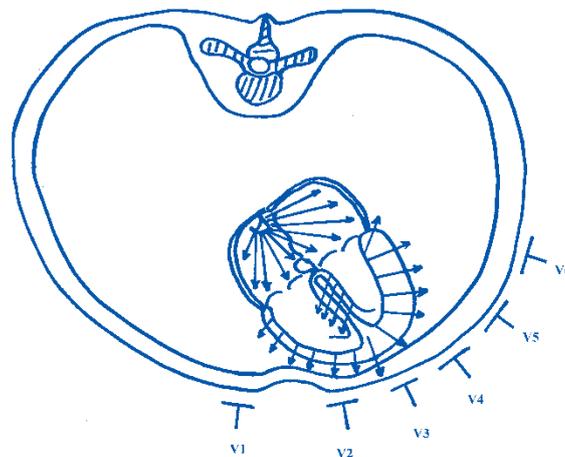


Fig. 8: Diagram of the directions of the depolarization waves in the heart and position of the chest leads

To understand the morphology of the QRS complexes it is important also to note that

- the magnitude of the final depolarization potential in the right ventricle is much smaller in comparison to the left ventricle and
- the direction of the vectors in the left and right ventricles is just opposite and simultaneous, and the magnitude of the final vector will have the direction of the bigger one (Fig 9).

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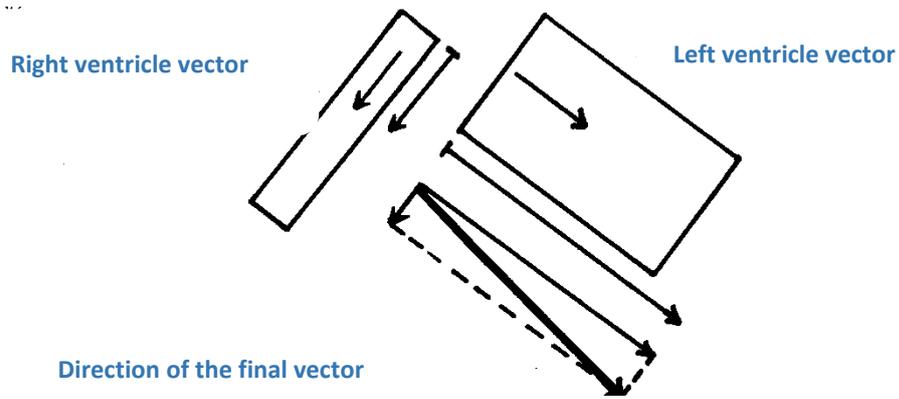


Fig. 9: Factors influencing the electrical axis of the heart

Construction of the final vector of ventricular depolarization with a proper direction and magnitude is not that easy, because the heart is three-dimensional and the potentials have, in fact, very variable directions. In general, it is important to accept the fact that in case of normal position of the heart in the chest, the axis of ventricular depolarization is oriented from the AV node **downwards and to the left side**. Based on this information we can explain the configuration of the QRS in the chest leads as follows:

V₁-V₂ record deep S, because the final vector is heading away from them

V₅-V₆ record high R, because the final vector is heading towards them

Now we can understand, that relatively small change of the heart position in the chest (obese people have horizontal position of the heart while skinny people usually have vertical position of the heart) may lead to the change of the S and R deflection in the precordial leads, because what has changed is the position of the electrodes towards the currents spreading across the heart.

4.0 GUIDELINE FOR THE ECG EVALUATION

J. Hanacek

ECG curve is a source of important information about the electrophysiological processes in the heart. To identify pathological changes, one must be familiar first with the physiological ECG curve and then, he/she must follow some guidelines to “read” the ECG curve properly. Students must learn and practice the systematic approach to the ECG reading and interpretation. Here we offer one guide how to do it. This “reading” process can be divided into a general aspect in which we evaluate the heart action, rhythm, rate, electrical axis and transitional zone, and the individual part, in which we evaluate individual part of the ECG starting from the P wave, ending with the T, and during this process we evaluate duration, polarity and amplitudes of the waves and deflections, duration of segments and intervals. How the outline for the reading should look like?

1. Make sure you have the proper recording, **control the ID of your patient, age, gender and time of recording**. Some processes may evolve with time. **Consider the patients’ complaints and history**.
2. **Check the technical quality of the recording**, whether it is “readable” at all, **check the paper speed and calibration**, also try to see if there are some interferences with the alternating current or bad contact of the electrodes which may cause **artefacts**.
3. **Check the heart action** – whether it is regular, irregular or regularly irregular, try to see if every single P wave is followed by QRS complex, see also the mismatching between P waves and ORS complexes rates
4. **Check the heart rate** – the rate of ORS complexes, P waves if not matched
5. **Check the heart rhythm – physiological heart rhythm is the sinus rhythm**

It is important to see whether the heart action is orchestrated by the sinus node (nomotopic production of the impulses). If the impulses are produced from some ectopic focus (heterotopy), then it is important to recognize where is the focus (atria, AV zone, ventricles), and finally whether it is active or passive heterotopy.

6. Construct the electrical axis of the heart

7. Check the transitional zone in the precordial leads

8. Evaluate carefully all waves, segments, intervals and deflections of the ECG, measure the amplitudes, durations and polarities starting with the P wave and ending with the T wave.

Every single abnormality should be mentioned in summary of ECG evaluation and in the final interpretation.

4.1 HEART RATE (RATE OF ELECTRICAL ACTIVITY)

Why it is important to add “electrical” activity, why not only the heart rate? The reason is that sometimes there can be a pathological process which will be characterized by quite high rate of the electrical activity, which will be, however, not followed by mechanical force. Remember, electrical and mechanical

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activities of the heart are not the same processes even they are connected and both are necessary for the optimal heart performance.

There are many ways how we can calculate the heart rate, and we offer an easy way which is also recommended in many literature sources.

Look at the original ECG record (Fig. 11). You can see there horizontal and vertical lines dividing it into a small and large squares. The distance between every vertical lines is 1 mm = 0.04 sec (1 sec divided into 25 = 0.04 sec) in case of the standard paper speed, which is 25 mm/s the distance between bold vertical lines is 5 mm (1sec:25 = 0.2 sec). These information are very useful for the calculations of the heart rate.

Let's find the R deflection which lies on (or very closely) to the bold vertical line. Imagine another one R on the next bold vertical line, and again, and again – first stripe on the figure. The heart rate would be 300 beats per minute, because we have one R every 0.2 sec (which is 5 in one sec). The minute has 60 sec, therefore it will be $5 \times 60 = 300$ beats per minute.

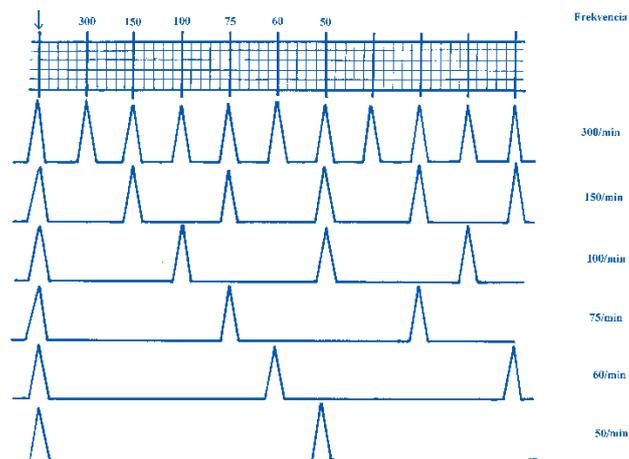


Fig. 10: Method of the quick calculation of the heart rate (Dubbin D, 1989)

Similar mathematical formula can be used to calculate the heart rate from the record where R deflections matches with every second bold line (second stripe on the figure). In this the heart rate will be 150 bpm. If the R matches with every third (100 bpm), fourth (75 bpm), fifth (60 bpm) bold vertical line, you can always use this formula where heart rate = 300: number of large squares (separated by the bold vertical lines). To use this method it is necessary to remember this simple formula, however, this method has a limitation and cannot be used when heart action is irregular. In these cases you need to use alternative methods which take into account a longer part of the trace, because it contain more “irregularities” and eventually parts with the higher and lower rates. Valid method which should be used to calculate the heart rate in case of irregular heart action is measurement of 3, 6 or 10 sec segments from the trace (Fig. 11). We count the number of QRS complexes present in these segments, and then multiply by either 20, 10 or 6.

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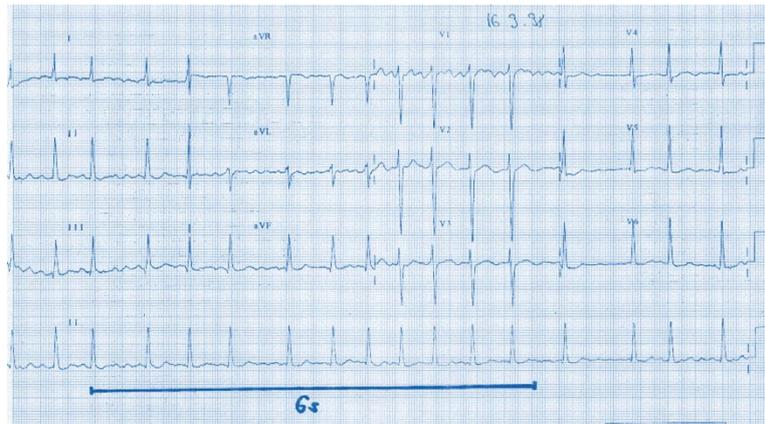


Fig. 11: ECG trace with the irregular heart action, we marked the 6 seconds segment and counted the QRS complexes, then multiplied by 10. (11 QRS x 10 = 110 bpm)

4.2 HEART RHYTHM

Heart rhythm is not an equivalent to heart rate, however these two parameters are frequently mixed. While heart rate determines how many times in the minute the heart depolarizes, the rhythm determines where is the place which rhythmically produces the impulses causing depolarization. Therefore rhythm cannot be a number, but it defines the part of the heart producing impulses.

In the heart of the healthy individual, the impulses are produced in the sinus node, that's why we call it the sinus rhythm. In pathological conditions any part of the heart can become a source of the impulses that will orchestrate the work of the heart. Based on the location of such a focus we can distinguish:

- ◆ **atrial rhythm** – impulses are produced in the atria outside of the sinus node
- ◆ **atrio-ventricular rhythm** – impulses are produced in the AV junctional zone
- ◆ **ventricular rhythm** – impulses are produced in the ventricles by either cardiomyocytes or ventricular conductive system

It is important to point out that ECG is the most appropriate method to identify the heart dysrhythmias – presence of the abnormal heart rhythms. That's why it is critically important to identify the rhythm of the heart action, whether it is sinus rhythm or some other rhythms. Presence of the sinus rhythm on the record indicates that the heart damage is not that serious even though its function is abnormal at the moment. The factors leading to such dysrhythmias come very likely outside of the heart, and it is necessary to identify them.

4.2.1 SINUS RHYTHM

Students frequently fail at the question how to identify the sinus rhythm. Their answer is that the hallmark of the sinus rhythm is the presence of the P wave, which is followed by the QRS complex. His

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answer is however not exact and is incomplete. The P wave will be present on the trace and followed by the QRS also if the impulses are produced by the ectopic focus somewhere in the atria or in the AV zone with the retrograde conduction to the atria. Therefore, it is important to set exact criteria how to recognize the sinus rhythm.

In case of sinus rhythm, we need to confirm that the impulse is produced in the “upper” part of the atria and it spreads down to the AV junction, and this can be confirmed by the evaluation of the polarity of the P wave in the leads aVR and aVF (aVR on the right arm and aVF on the left leg) which can nicely detect the direction of the current depolarizing the atria. P wave must also have the proper duration and shape, what confirms that the depolarization of the atria is not chaotic and it runs physiologically. The list of the criteria for the sinus rhythm are:

- **each P wave is followed by the QRS complex**
- **each P wave has normal shape**
- **P wave is negative in the aVR and positive in the aVF**
- **PQ interval is 0.12 - 0.2 s**
- **heart rate is between 60-90 bpm at the rest condition**

If criteria for sinus rhythm are fulfilled, but it seems to be too fast (more than 90 bpm – it is also sinus rhythm, but with increased heart rate, which is called sinus tachycardia, a vice versa if the heart rate is less than 60 bpm. These criteria are completed by the finding of normal QRS complexes unless there is no pathology in the ventricles that would eventually disturb the spreading of the depolarization through the ventricles.

4.2.2 ATRIAL RHYTHM

This type of the heart rhythm is not commonly explained in the textbooks, but logically, the existence of the preferential conductive pathways in the atria, and perhaps some pathological conditions may lead to the onset of the ectopic foci in the atria, that would become a pacemaker, leading to presence of the atrial rhythm. The atria are frequently the source of many dysrhythmias, for example flutter, premature atrial beats, fibrillation etc. The atrial rhythms can be recognized by the changed shape, duration and eventually also polarity of the P wave, depending on the location of this ectopic focus in the atrial musculature.

P wave is present in front of each QRS complex, but it has abnormal shape, and the duration may be prolonged as the spreading of the impulse from the ectopic focus may not actually follow the preferential pathways, and may spread via some alternative pathways to finalize depolarization of the atria. Also, the shorter PQ interval may indicate that the impulses are not produced in the sinus node but perhaps somewhere closer to the ventricles so the impulse can reach the AV zone in a shorter time. QRS complex is not changed, because the conduction in the ventricular system is normal.

4.2.3 AV – JUNCTIONAL RHYTHM

AV zone consist of three parts. First localized in the lower right atrial wall is called atrio-nodal zone (AN zone). Second - middle part is called nodal zone (N zone) and it is still localized in the lowest part of the right atrial wall and third zone which is called nodal-His zone (NH zone). It which creates transition from the nodal part of the His bundle and it penetrates the annulus fibrosus and reaches ventricles. The source of the impulses (source of the AV rhythm) can be any part of this zone. Based on the terminology, we can talk about the AV rhythm in the case that these impulses produced in the AV zone drives the heart action longer than 2-3 normal depolarizations, although it may last minutes, days or months. The reasons why the AV zone became pacemaker are:

- **sinus node does not produce impulses, or the impulses form the sinus node are blocked**, this must last at least for couple of seconds, and afterwards the AV zone will start pacing itself

- **cells in the AV zone became more automatic than the cells of the sinus node, so they can produce impulses with the higher rate than the sinus node** – this disturbance appears on the record as soon as it happens in the AV zone

In the first case, the AV zone became a pacemaker because it was necessary (sinus node was not producing impulses, therefore the heart would stop working). So, there is enough time to create impulses by AV system. This is called passive AV junctional rhythm and the rate of the impulses produced by the AV zone is lower than physiological rate of the sinus node cells, and it is approximately 40-50 bpm, while in the sinus rhythm it is 60-90 bpm in the rest condition.

In the second case the AV zone got the role of the pacemaker actively – that's why we call this active AV junctional rhythm. The cells of the AV zone can now produce impulses with the rate considerably higher than the typical rate for the sinus node cells.

What hallmarks are typical for the AV junctional rhythm? They depend on:

- the site in the AV zone which is producing the impulses
- **possibility of the retrograde spreading of impulses back to the atria**
- **speed of the conduction forward (to the ventricles) and backward (back to atria)**, because in the case we may have three alternatives – the impulse will reach the atria first (then the ventricles), the impulse will reach the ventricles first and the retrograde wave will be slower, or the impulse will hit the atria and the ventricles in the same time (Fig. 12).

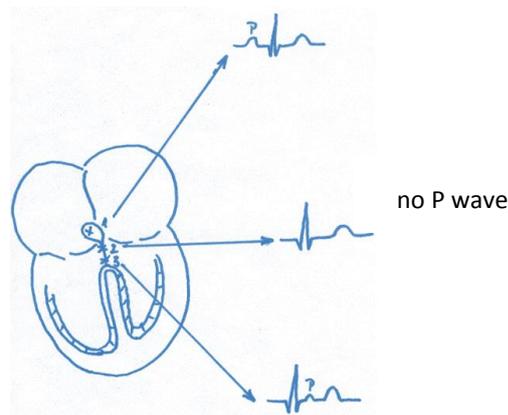


Fig. 12 shows position of the P wave in the ECG trace determined by the order in which the impulse from AV zone will reach the atrial and ventricles.

If the impulses are produced in the upper parts of the AV node (at the side where it connects to the conductive fibres from the atria), then it is very likely that it will depolarize the atria earlier than the ventricles (because to the ventricles, it must go through the AV zone, which normally cause the delay of the impulse transition). On the trace, there will be P wave in front of the QRS, but the P wave will be “retrograde”, it will have opposite polarity, it will be positive in aVR and negative in aVF (because the depolarization wave goes away from it). PQ will be very short. If the impulses are produced in the “middle” part of the AV zone, the conduction time forward and backward are very similar, and the atria and ventricles will be depolarized in the same time. P wave would not be seen, because it will be hidden in the QRS complex. The reason is that the potentials from the ventricles are much stronger and they cover the electrical activity of the atria. Sometimes, if the synchronization of the atria and ventricles is not perfect, the P wave can be visible in a form of small deformation of the beginning or terminal parts of the QRS complex.

If the impulses are produced in the “lower” parts of the AV zone, and if here is still a possibility for the retrograde conduction to the atria (from the functional point), this will take much longer than conduction to the ventricles. On the trace, we will see the QRS complex, and just after it finishes, the P wave will appear, which is abnormal, of course. In this case, mechanical systole of the atria cannot contribute to the optimal ventricular filling. This type of the AV junctional rhythm manifest as a QRS complex, and the “retrograde” P wave behind it. In all three cases, the shape of the QRS will be normal, because the ventricular depolarization is normal.

4.2.4 VENTRICULAR RHYTHM

Conductive system of the ventricles and sometimes also the working cardiomyocytes can be a source of ectopic impulses, therefore they can become a pacemaker making the hart work according to the ventricular rhythm. Similarly to the AV junctional rhythm, also ventricular rhythm can be either passive or active (Fig. 13a).

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The cause of the **passive ventricular rhythm** is AV block of third degree, what means that impulses from the SA node, through the atria cannot reach to the ventricles due to the pathological process affecting the AV zone (Fig. 13b).

In this condition, atria are depolarized by the impulse from the sinus node in the regular pacing pattern, but the impulse cannot get through the AV zone. This leads to the need of the extra pacing for the ventricles which is possible thanks to the ability of cells in the ventricular conductive system to depolarize spontaneously, just like the cells of the sinus node, or AV node but the rate of the spontaneous depolarizations is lower. The source of the ventricular rhythm in the case of AV block is the part of the conductive system located behind the block (a place somewhere in the AV junction).

Active ventricular rhythm is a consequence of a pathological process affecting ventricles, leading to the offset of the one or more ectopic foci with abnormal high automaticity (Fig 13.B). This is not the rhythm which is activated as a consequence of the lack of pacing from atria, but the foci (focus) in the ventricles have higher automaticity – higher rate of spontaneous diastolic depolarizations. In this case, sinus node is pacing normally and depolarizing atria, AV connection is normal and can conduct the impulses to the ventricles, however, this is not possible, because the ventricles are activated from the “hyperreactive” focus of extra beats. And the ventricular conductive system of the musculature is either depolarized or in refractory phase which is caused by the high rate of pacing of the hyperreactive focus in the ventricles.

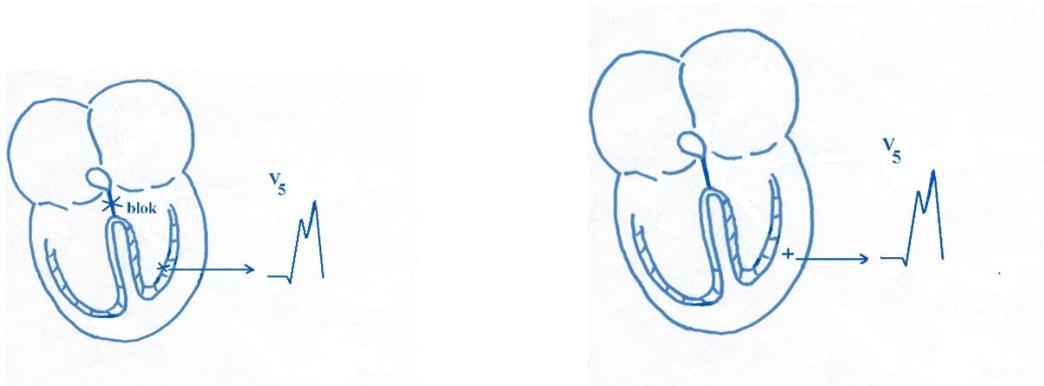


Fig 13a, b: Passive ventricular rhythm which offsets after the AV block versus active ventricular rhythm, which is a consequence of “hyperactive” ectopic ventricular focus producing impulses with high rate

In both types of these rhythms we can see **atrio-ventricular dissociation**. This term describes independent activation of the atria and the independent activation of ventricles with no temporal relationship between them. In the first case, however, the ventricles are depolarized with the slower rate, in the second case (hyperactive focus) the ventricular pacing is faster than the pacing of the sinus node.

In case of the ventricular rhythms the trace can record bradycardia or tachycardia (depending on the type of the ventricular rhythm), but the main hallmarks are changes of the configuration, shape and total duration of the QRS complex. The pacemaker is located in the ventricles (right or left) and the depolarization spreads from it through the musculature to the other ventricle – the direction and the pattern of depolarization is abnormal, and this abnormal depolarization wave influences the shape and duration of the QRS.

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Abnormalities of the QRS complex are consequences of the abnormal direction of the depolarization wave spreading toward and away from particular electrodes, and wide QRS is a consequence of the prolonged and delayed depolarization of the musculature of the right ventricle (remember that conduction speed of the bundle branches and Purkinje fibres is 4m/s, while in the muscle it is only 1m/s. Abnormal QRS complex is followed by the abnormalities in the ST segment and T wave, because also the repolarization of the ventricles is opposite to normal. QRS complexes are typically in discordant position (there is however one exception – ventricular rhythm produced from the bundle of His before it splits to the bundle branches (suprabifurcational rhythm). This place is morphologically located in the ventricles, but from the functional aspect, the impulse will reach the ventricles by the physiological way and it also spreads in a normal way across the ventricles – therefore in this case the QRS complexes are not wide and even the rate is not necessarily changed from the normal values. To identify this abnormality, we need to focus on the presence of the AV dissociation, which can be recognized by these findings:

- **distances between P waves are the same and stable from beat to beat**
- **distances between QRS complexes are also stable and same from beat to beat** if there is just one ectopic focus
- **there is no temporal relationship between the P waves and QRS complexes !!!**

There is no real PQ segment in the AV dissociation, there can be something that mimics PQ segment between the P waves and QRS complex, but in reality this part of the trace cannot be even called PQ segment, because the conduction of the impulse from the atria to the ventricles does not even exist. (PQ segment is defined as time of atrioventricular conduction which does not exist here).

4.3 DETERMINATION OF THE ELECTRICAL AXIS OF THE HEART

Electrical axis of heart ventricles refers to the direction of depolarization which spreads throughout of the ventricles. It is manifested by amplitude and shape of QRS complex. Because this axis is characterized by magnitude and direction, it is called a vector (white arrow in Fig. 14, left side). Electrical axis of ventricles is sum of electrical depolarization each of single cardiomyocyte, so, it is mean vector. We can use small vectors to demonstrate ventricular depolarization which begins in the endocardium and proceeds through ventricular wall to epicardium (Fig. 14 – right side).

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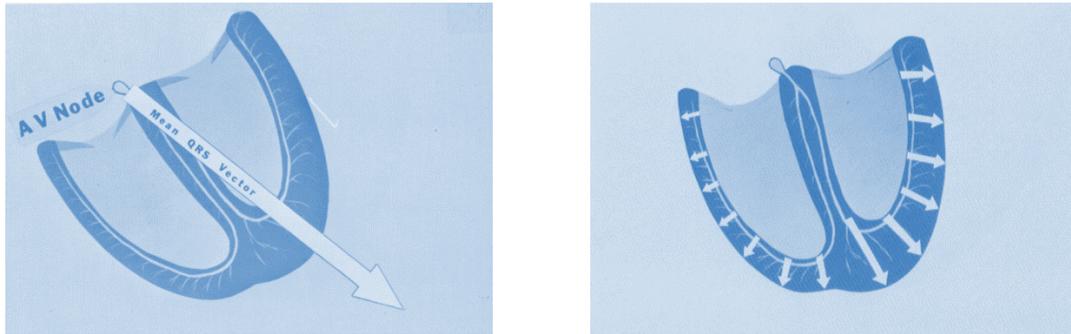


Fig. 14. Graphics representing normal direction of the electrical axis of the heart 14a, and second phase of the ventricular depolarization from subendocardium to subepicardium (Dubbin D, 1989)

The ventricular conductive system transmits electrical impulse with high speed, so depolarization of endocardial part of ventricular muscle in all areas begins at the same time. You can see at the scheme that left ventricular wall has larger vectors than right ventricle. If we add up all the small vectors developing during depolarization of each muscle cell, we obtain one large mean ventricular vector – it is called mean QRS vector. Because the vectors representing left ventricle are larger, the mean QRS vector points slightly to the left. The origin of the QRS vector is always the AV node and actual position of the top of vector is moving during ventricular depolarisation creating virtual circle/ellipse - when recorded the vectocardiogram is drawn.

Position of mean QRS vector is noted in degrees within the circle drawn over the patient's chest (in the frontal plain, only – Fig. 15). Zero (0°) is on the patient left, horizontally, and the lower part of this circle is positive (+) degrees, while the top half is negative degrees. The normal mean QRS vector points downward and to the left, or between 0° and $+90^{\circ}$ degrees. If the heart is displaced or size of ventricles are profoundly changed the mean QRS vector is also displaced – when heart is in vertical position (e.g. in young tall and slim boys) QRS vector is displaced to the right, too, and when there is rather horizontal position of the heart (e.g. in obese people diaphragm is pushed up and heart ventricles, too) the QRS axis will move to the left. Similarly, with hypertrophy (enlargement) of one ventricle, the greater electrical potential in hypertrophied ventricle will displace the QRS vector toward the hypertrophied ventricle (more will be presented in chapter devoted to ventricular hypertrophy and dilatation).

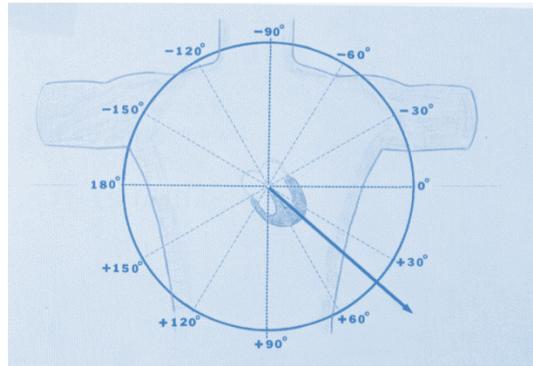


Fig. 15 represents the electrical field around the heart in 2D projection (Dubin D, 1989)

The mean QRS vector gives us useful information about the position of the heart and its movement in the chest cavity. It also gives us information on hypertrophy of one heart ventricle or, on acute myocardial infarction (there is sudden loss of some percentage of myocardial muscle – usually in left ventricle, which can be accompanied by sudden shifting QRS vector to the right). Changes of the heart axis are also present in hemiblocks. As you can see these principles of axis position are logical and understandable. This is one important reason why it is necessary to use this diagnostic tool whenever the 12 lead ECG is recorded.

How to determine QRS vector direction? There is more than one method to do this. One of them seems to us quite simple and useful for medical students and non-cardiologist. The method was offered by Dubin (1989), and it is based on amplitude of QRS vector in two leads, only - in I. standard limb lead and aVF (unipolar limb lead).

Firstly, visualize a circle surrounding the heart with the centre created by AV node (Fig. 16 – left side). The QRS vector has the starting point at AV node, and the tip of the vector will be directed somewhere on the surface of this virtual circle. So, introduce the lead I (horizontal line) into the circle (left arm with positive electrode and right side with negative) at the level of AV node. When we now create instead circle around the AV node the sphere (Fig. 16 – right side) we can now consider the sphere in two halves – right side is negative, and left side is positive. In this situation depolarization in the lead I is spreading from right side to left, and it is accompanied by positive deflection of QRS complex. If we find in the lead I positive deflection of QRS complex (we should count amplitude of all positive and negative deflections in the lead I) then mean QRS vector points to the positive half of sphere, it means to the left – left axis deviation (Fig. 16 – bottom). This information is insufficient for precise determination of ventricular electrical axis. We only know that it is directed to the left side (from $+90^\circ$ to -90°) or to right side (again from $+90^\circ$ to -90° in opposite side). If the QRS complex is in the same lead negative the mean QRS vector points to the right (to negative half of circle) – right axis deviation. Dubin (1989) thinks that lead I is the best lead for detecting right axis deviation.

Electrocardiography

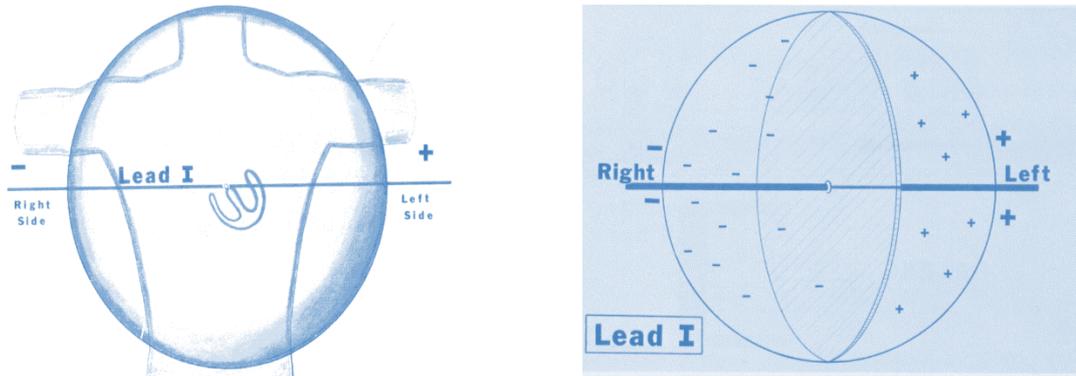


Fig 16 a, b: determination of the electrical axis from the lead I – whether it is located in the left or right hemisphere (Dubbin D, 1989).

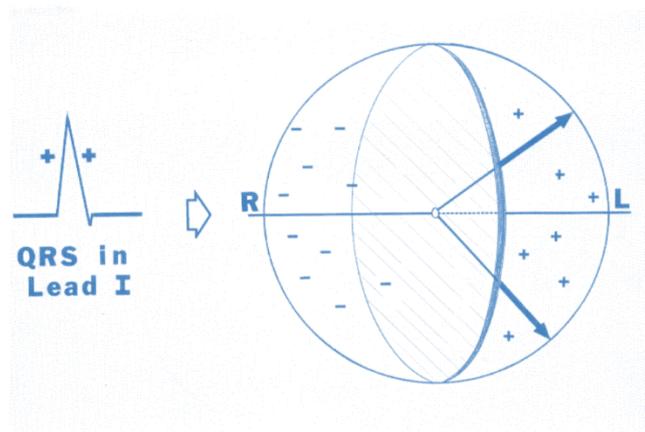


Fig. 16c Determination of the electrical axis based on the polarity of the deflection in the QRS in the first lead. (Dubbin D, 1989)

For determination of the quadrant of the sphere to which the QRS vector is directed it is necessary to use another lead – lead aVF. In this lead positive electrode is localized at left leg (it is unipolar electrode). Create now the virtual sphere around the aVF lead (with centre in the AV node – Fig. 17- left side). We can neglect lead I with the sphere around it for a time being. Concentrate to polarity of the sphere around the lead aVF. It can be also divided on to two halves – upper half is negative and lower half is positive (possible explanation – depolarization of the heart starts in its upper part, so it is very early depolarized - surface is negatively charged. ECG machine makes the sensor on left leg positive). Because QRS vector is normally directed downwards to the left, it is directed to positive aVF electrode, so, QRS deflection should be positive (Fig. 17 – right side). Opposite, if we find negative deflection of QRS complex in aVF lead it is clear that QRS vector will be directed from bottom up, so in opposite direction as under physiological condition. By this method we can determine only whether the QRS vector is directed to upper (negative) or lower (positive) part of sphere around the aVF lead.

Electrocardiography

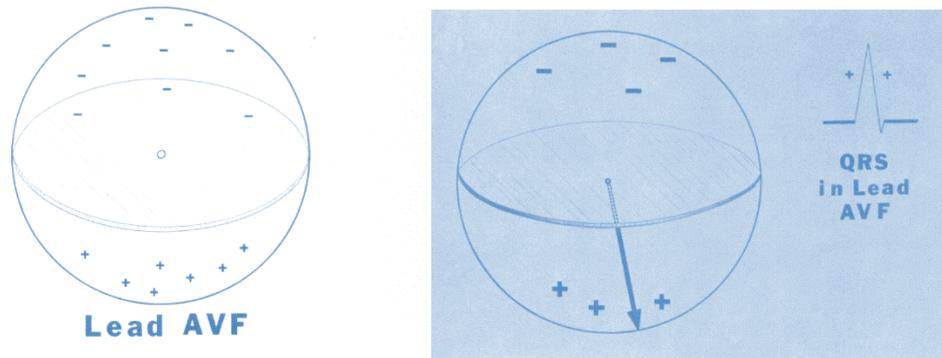


Fig. 17, 17a: Determination of the axis using the polarity of QRS in the lead aVF (Dubbin D, 1989)

When we use information from both these leads we can construct direction of QRS vector in frontal plane (Fig. 18). If the QRS complex is positive in lead I and also in lead aVF (double thumbs up sign), the vector should point downward and to the patient's left. It is clear that should be quadrant between 0° - $+90^\circ$, so it is the normal range of ventricular axis.

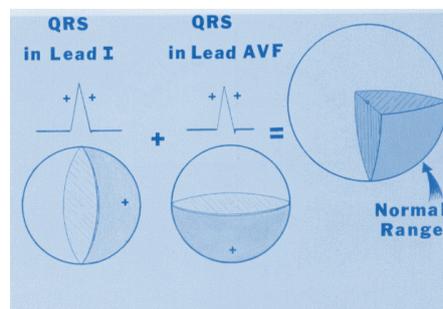


Fig. 18 Electrical axis of the heart according to the polarity of the QRS complex in lead I and lead aVF (Dubbin D, 1989).

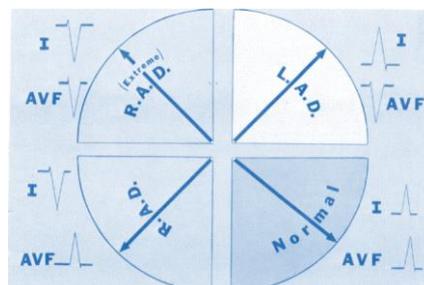


Fig. 19 possible combinations of the polarities of QRs on lead I and aVF as the determinant of the electrical axis (Dubbin D, 1989)

Electrocardiography

In figure 19 are depicted for possible axis quadrants where mean QRS vector may point to. If the vector points upward (from the AV node) and to the patient's left, this is left axis deviation (L.A.D.) – QRS complex is positive in lead I and negative in lead aVF. If QRS complex is negative in lead I and positive in lead aVF – this is right axis deviation (R.A.D.).

There are three dimensions to the heart and also to the sphere drawn around it in the chest. Position of the mean QRS vector also move in three dimensions, so in sagittal, frontal and horizontal planes. The horizontal plane (virtually present at the level of AV node) divides the chest into upper and lower parts. Chest leads, V_1-V_6 , form the horizontal plane (Fig. 20 – left side). So, if you like to determine changes (rotation) of mean QRS vector in horizontal plane, you should examine the chest leads. For better understanding the movement/rotation of QRS vector in horizontal plane we can use lead V_2 as example (at the 4th intercostal space to the left of sternum), and we can create sphere around it with the centre again in AV node. All the chest leads are positive (done by ECG machine), so patient's back is negative (Fig. 20 – right side). The front of the chest is than positive and back of the chest is negative. When there is physiological position of the heart (ventricles) in the chest the QRS in lead V_2 is negative, so mean QRS vector points backward because generally posteriorly positioned thick left ventricle (depolarization of left ventricle is directed away from positive V_2 electrode).

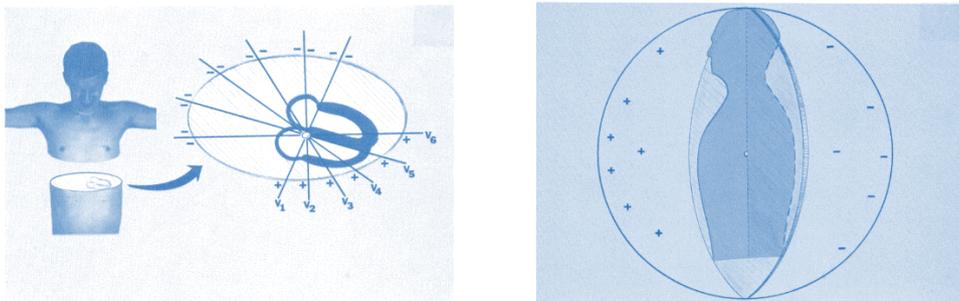


Fig.20, a, b (Dubbin D, 1989)

The orientation of V_2 electrode makes it very important lead for the determination of pathological processes (namely ischemia) present at anterior and posterior wall of the left ventricle. As you will see later (in chapter devoted to myocardial infarction) ventricular electrical processes should be taken into account in the right chest leads, because anterior and posterior myocardial infarctions will be reflected in these leads.

4.4 OUTLINE FOR THE ECG EVALUATION AND INTERPRETATION

There is a lot of information hidden in the ECG curve, but they need to be found, recognized and interpreted properly to give us a message about the electrical activity of the heart. To “read” the curve properly, one needs the knowledge about the electrophysiology of the heart and careful guided reading of the trace.

Students can get knowledge by studying, however, the reading and interpretation of the curve also needs practice. Frustration caused by inability to do it correctly as soon as possible may lead to discouragement of students, but in this case, you only need to keep study and practice. Beginners can and may do mistakes. The problem could be if even a trained person does not recognize important pathology by very quick, superficial and irresponsible evaluation of the trace.

In the ECG “reading”, we strongly recommend to follow a guideline how to do it, what to look at first, then how to “read” the other parts of the trace and after all to get familiar with these guidelines. Here is one of the outlines which can be used to read and interpret the ECG curve properly. Every single abnormality should be mentioned in the final interpretation of the ECG curve.

1. Make sure you have the proper record, **control the ID of your patient, age, gender and time of recording**. Some processes may evolve with time. **Consider the patients complaint and the history**.
2. **Check the technical quality of the record carefully**, whether it is “readable” at all, **check the paper speed and calibration** of potential also try to see if there are some interferences with the alternating current or bad contact of the electrodes which may cause **artefacts**.
3. **Check the heart action** – whether it is regular, irregular or regularly irregular, try to see if every single P wave is followed by QRS complex, see also the mismatching between P waves and ORS complexes rates, polarity of P waves
4. **Check the heart rate** – the rate of ORS complexes, P waves if not matched
5. **Check the heart rhythm – physiological heart rhythm is the sinus rhythm**
6. It is important to see whether the heart action is orchestrated by the sinus node (nomotopic production of the impulses). If the impulses are produced from some ectopic focus (heterotopy), then it is important to recognize where is the focus (atria, AV zone, ventricles), and finally whether it is active or passive heterotopy.
7. **Construct the electrical axis of the heart**
8. **Check the transitional zone in the precordial leads**
9. **Evaluate carefully all waves, segments, intervals and deflections of the ECG, measure the amplitudes, durations and polarities starting with the P wave and ending with the T wave.**

5.0. MOST COMMON DYSRHYTHMIAS - MECHANISMS AND MANIFESTATION ON ECG

J Hanacek

There are many interindividual variations of the physiological ECG and even more variations are known when it comes to the disturbances of the electrical function of the heart. First of all, student must understand the simple disturbances, causes leading to them and also their manifestation on ECG. The authors prepared a system of disturbances of the electrical function of the heart and divided them into the disturbances of the impulse production and disturbances of the impulse conduction. Even though these two may combine in real pathological process, this classification is useful for educational purposes.

5.1 DISTURBANCE OF IMPULSE PRODUCTION

Disturbances of the impulse production are classified according to the site, which is affected by the pathological process. They are **nomotopic** and **heterotopic** disturbances of the impulse production.

- **Nomotopic disturbances of the impulse production** are all changes of the electrical activity of the heart caused by some physiological and pathological processes affecting directly the sinus node. This type of disturbance has only an active form (active nomotopy), because there is no “higher” pacemaker than SA node for the heart, therefore “passive nomotopy” that would appear after the higher pacemaker fails to produce impulses does not exist.

- **Heterotopic disturbances of the impulse production** are all changes of the electrical activity of the heart with a source (an arrhythmogenic substrate) located outside the SA node. It can be located anywhere in the atria, AV junction or ventricles and it may come in two forms – passive and active heterotopy. They manifest in passive and active form.

5.1.1 NOMOTOPIC DISTURBANCES OF THE IMPULSE PRODUCTION

a) Sinus tachycardia

It is characterized by increased rate of pacing in the SA node above 90 (100) bpm. Production of impulses is faster, but regular. Regularity can be influenced by the effect of the respiration (inspiration increases the heart rate, while expiration reduces it). This disturbance is easily recognized on the ECG trace, because we can recognize the “sinus rhythm”, however the heart rate is increased above the predicted values. Causes of the sinus tachycardia are many:

- **increased body temperature** (also temperature of SA node) increases the metabolic speed and the oxygen consumption and it also increases the electrical process in the cells of the SA node (spontaneous diastolic depolarization)

- **physical exercise or emotional stress** increases the concentration of catecholamine in blood and they reduce the impact of the vagus nerve on SA node

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- **hyperthyreosis** – T3 & T4 increase the metabolic speed and potentiate the effect of catecholamines
- **hypokalaemia, anaemia, pregnancy...**



Fig. 21: Sinus tachycardia – sometimes it looks like the P wave of the upcoming depolarization shows up very closely after the T wave, sometimes there is a fusion of both waves into one waveform (Yanowitz FG, 2007)

- SA node has intrinsic (inherited) ability to produce impulses spontaneously independent on the nerves, and this “inherited” pacing rate would be 100 – 110 bpm. This activity is however influenced by the vagus nerve. This innervation lead to the inhibitory effect on the SA node cells, therefore they pace with the rate 60 – 80 bpm. Cutting the right vagus nerve leads to tachycardia, so does the medication with atropine, which blocks the parasympathetic muscarinic receptors (responding normally to acetylcholine) on the SA node cells.

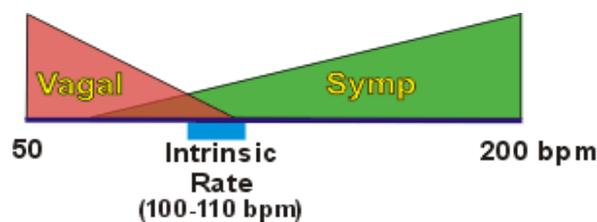


Fig. 22. The effect of the sympathetic and the vagus nerves on the intrinsic SA node activity

- Changes of SA node activity are achieved by the efferent cardiac vagal and sympathetic nerves. The pacing rate increases by the activation of the sympathetic system in physiological and pathological conditions (physical exercise, stress, fever) but only if the vagus activity is inhibited at the same time. Sympathetic efferents release norepinephrine which activates adenylyl-cyclase and it increases production of cAMP. This product influences function of the membrane in channels in a way that increases the rate of spontaneous diastolic depolarization. Vagus nerve has opposite effect – acetylcholine reduces production of c AMP which in turn reduces the speed of spontaneous depolarizations of the pacemaker cells. Acetylcholine also hyperpolarizes the membranes of the pacemaker cells – and this slows the pacing as well.

Pacemaker activity is influenced dramatically by the age. The maximal heart rate that can be achieved in an individual is estimated by an equation

$$\text{Maximal heart rate} = 220 \text{ bpm} - \text{age in years}$$

Therefore a 20-year-old person will have maximal heart rate of about 200 bpm and this will decrease to about 170 bpm when the person is 50 years old. This maximal heart rate is genetically determined and cannot be modified by exercise training or external factors (Klabunde, 2013).

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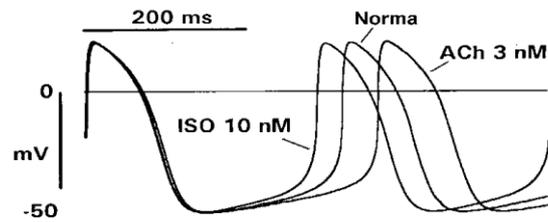


Fig. 23: Effect of sympathetic system (ISO) and parasympathetic system (ACh) on the speed of SDD in the SA node cells. Mind the delays of the action potential offset (Francesco, 1993).

b) Sinus bradycardia

It is characterized by slow production of the impulses by the SA node cells, and the final heart rate is below 60 bpm. This bradycardia is physiological finding in the well-trained athletes and during sleep in healthy individuals as a manifestation of the “economy” of the heart work in condition characterized by reduced cardiac output as a response to the lower oxygen demands of the tissues during night rest. Presence of sinus bradycardia in other conditions than these two can indicate a pathology of heart or a pathology of the system that controls its rate

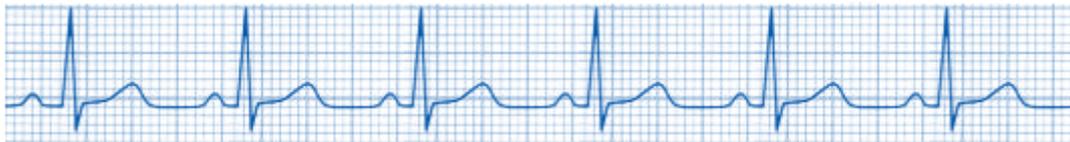


Fig. 24: Sinus bradycardia – distance between T and P is prolonged (Yanowitz FG, 2007)

Causes of bradycardia:

- **bradycardia in old people** – it can be a consequence of degenerative changes in the SA node or vagotony
- **bradycardia caused by ischemia of SA node tissue** – in subjects with ischemic heart disease with the ischemic damage of the right atrium
- **hypothermia** – as a consequence of reduced speed of metabolic processes in the body including SA node
- **hyperkalaemia** – reduces the speed of SDD and also cell to cell conduction – this may lead to the delay in the spreading of the depolarization to the atria
- **hypofunction of the thyroid gland** – lack of the T3 & T4 decreases metabolic speed and the heart is less sensitive to action of catecholamines
- **increased intracranial pressure, jaundice, typhoid fever** – reflex influence on the heart via vagus nerve
- **medication** – digitalis (stimulates parasympathetic system) beta-blockers, Ca channel blockers

Why it is important to evaluate the activity of SA node? Because the hormonal and neural influences on the SA node can effectively adjust the heart rate (also cardiac output) to the actual oxygen demand, so the oxygen delivery by the cardiovascular system matches to the oxygen consumption.

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Heart rate and stroke volume are two important parameters determining the cardiac output. Increase of heart rate leads also to the increase of the cardiac output until the heart reaches the rate 160-180 bpm. Faster rates do not contribute to the increase of the cardiac output, because the diastole becomes too short with increased heart rate. It reduces end-diastolic volume therefore the stroke volume of following systole is reduced – this cannot increase cardiac output any longer.

Extreme heart rates in tachycardia can reduce cardiac output, so does bradycardia. Extremely low heart rates may result into the hypoperfusion of body tissues and their damage caused by the lack of oxygen.

c) Respiratory sinus arrhythmia (RSA)

It is the change of the rate of SDD in the sinus node (and also the heart rate) related to respiration. It is best observable in children and adolescents. The rate of impulse production increases in inspiration and decreases in expiration (Fig. 16). There is no exact match between the stages of the respiratory cycle and the heart rates accelerations/decelerations, because the heart rate changes follow the respiratory changes with little delays. Magnitude of the RSA decreases with the age and also in serious heart diseases. Clinical experience shows that presence of RSA indicates that the cardiovascular system is not that damaged because the heart and the regulatory systems influencing its functions are still working. Serious diseases of the heart or regulatory mechanisms lead to the reduced magnitude of RSA, or it may eventually vanish. Every rule has an exception – patients with heart failure and Cheyne-Stokes breathing (abnormal periodic breathing pattern) can have RSA. RSA can be recognized on the ECG trace by RR intervals which fluctuates (shorter and longer RR intervals) related to the breathing cycle. Since the respiratory phases are not usually recorded with the ECG, we can only assume that what we see on the trace could be RSA.



Fig. 25: Respiratory sinus arrhythmia (Yanowitz FG, 2007)

There are more than just one mechanisms leading to the RSA:

- **Central influences** - activation from the inspiratory centre in the medulla (brainstem) irradiates to the cardio-motor centre where it inhibits vagal motoneurons and this reduces their inhibitory effects on the SA node – it means that the effect of the sympathetic system becomes dominant during inspiration
- **Reflex influences from slowly adapting stretch receptors in the lungs** – during inspiration they inhibit activity of the cardioinhibitory centre and it leads to the dominance of cardioexcitatory centre
- **Bainbridge's reflex** – distension of the right atrium by increased venous return in inspiration (negative pressure in the chest during inspiration leads to the augmentation of the venous return from lower parts of the body) leads to the acceleration of the heart rate by local and reflex mechanisms

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- Reflexes from arterial baroreceptors and local direct mechanical influences on the SA node
- Oscillation of the pH, PaCO₂, PaO₂ – hypercapnia increases magnitude of RSA, hypoxia reduces it

d) Sinus arrest (stop of the activity in the SA node)

Cause of this problem can be strong activation of the vagus nerve (its branches innervating the area of the SA node and/or increased sensitivity of the SA node to acetylcholine). Another cause can be the damage of SA node by pathological processes – ischemia, inflammation, degenerative changes and toxic influences) - Activity of the SA node may interrupt for couple of seconds or minutes, however, it may also be permanent. The manifestation of sinus arrest on the ECG is lack of P wave, QRS, ST and T in the expected time. If this disturbance is only temporary, it will manifest by the pause between two normal activities of the heart (doubled, triple distances of “normal” heart actions). If the sinus arrest lasts longer, this may lead to the activation of secondary pacemaker activity from the centres with lower automacy. This secondary pacemaker may be located in the atria or in the AV zone. The heart will therefore be activated from this focus, which was activated as a must, otherwise the heart would stop. This is called passive depolarization or passive heterotopy. If the focus paces just once, it is called escaped beat, if it remains pacing, it will be the escape rhythm (atrial or AV junctional rhythm).

Sinus arrest and sinus block are two different disturbances, however, it is not possible to differentiate them on the ECG trace. There is the absence of the P, QRS, ST and T in both circumstances. The difference is that in sinus block the impulses are produced by the cells in the SA node, but they cannot depolarize the atrial musculature (they cannot get through) because of the conduction disturbance located around the SA node. The identification of sinus arrest or sinus block would be possible by using a special electrode inserted straight to the SA node (and this is not conventional ECG, but specialized electrocardiographic method).

Clinical significance of these disturbances (presence of signs and symptoms) depends on the time between the SA node stops and the activation of secondary pacemakers. If it takes longer; the patient may experience cerebral ischemia as a consequence of asystole. Clinical presentation of **Adams – Stokes’s syndrome** includes **asystole** (the lack of ventricular contraction), **syncope** (short lasting unconsciousness from hypoperfusion of CNS) and **cramps of striated muscles** (because of increased cortical activity provoked by acute cortical ischemia).

e) Sick sinus syndrome (SSS)

It is a term describing the dysfunction of the SA node caused by its damage which manifests by episodes of bradycardia alternating with episode of tachycardia from the supraventricular area (paroxysmal supraventricular tachycardia, atrial flutter or atrial fibrillation). The other name of this disorder is the syndrome of brady-tachyarrhythmia. Adams – Stokes’s syndrome may be present as well.

5.1.2 HETEROTOPIC DISTURBANCES OF THE IMPULSE PRODUCTION

The definition of this group of arrhythmias clearly states that the impulses activating the heart are produced ectopically – outside of the SA node. This may be caused by several mechanisms:

- **SA node stopped pacing** (strong vagus activation, damage of the node by pathological process). Secondary or tertiary pacemaker cells in the heart then become the source of the extra pacing. They can be supraventricular (atrial, or AV junctional) or ventricular pacemakers and they are usually cells of the physiological conductive system of the heart. Since they are activated in case of sinus node problems, they substitute its function – this is called passive heterotopy and the “new” pacemakers have typically lower rate of SDD than the SA node.
- **some cells located outside of the sinus node assume the ability of SDD and they started pacing with the higher rate than is normal rate of the SA node** – this is called active heterotopy, because the ectopic pacemaker is more active than SA node.

Pathological process causing this arrhythmia can last couple of seconds – then the duration of this arrhythmia will be also short. This process will manifest by one or several abnormal activations of the heart on the ECG trace. Passive heterotopy will manifest as isolated escaped depolarizations (escape beats) and active heterotopy will manifest as a premature depolarization (premature beats of the atria or ventricles). Escaped depolarization appear on the trace later than expected depolarization from SA node, active heterotopy offsets earlier than expected.

Longer duration of the pathological process (hours, days, even years) leads to the situation, when the ectopic focus itself becomes dominant pacemaker over the long period of the time and it orchestrates the activation of the heart. It will manifest as a presence of heterotopic rhythms either passive (escape rhythms) or active heterotopic rhythms.

Pathological processes leading to the offset of either active or passive heterotopy can also have different intensity in time, so sometimes the intensity may reduce leading to a remission; eventually get worse again (exacerbation, recidivism). This is one of the reasons why it is important to mark a date/hour on every single ECG record taken in a patient. Comparison of older records and fresh records can predict the course of the disease (pathological process). Depending on how long these changes persist on the ECG recording we can classify:

- **temporary disturbances** – disturbances which vanish with time, and never show up again
- **intermittent** – disturbances which appear and go repeatedly and there are periods of remission in between
- **permanent** – disturbances which appear with the onset of the pathological process and never disappear.

5.1.2.1. PASIVE HETEROTOPY

A) ESCAPE DEPOLARIZATIONS

If SA node stops pacing (for any reason) or if there is a SA node block, then the lower order pacemaker is usually located in the AV zone. Impulses in this area are however produced with lower speed, that's why the ECG will show passive heterotopic activity from the AV zone with a delay (it will show up later than the expected activation from the SA node). This is the first and important hallmark of the escaped depolarizations (beats), because this is also the one and only difference between them and premature beats produced in the same area. P waves, eventually QRS complexes will have the same shape (the same position of the ectopic focus towards the recording electrodes), but the premature beats will appear earlier than expected, while the escaped beats will appear later than expected.



Fig. 26. Junctional escape beat

Escaped AV junctional depolarization activates the ventricles by the same way as the normal impulse from the SA node. Depolarization of the atria depends on many factors (described in details in the section about AV rhythm). If atrial depolarization appears, it will be typically retrograde, which will manifest by the change of the polarity and eventually also the shape of the P wave. P wave will be positive in aVR and negative in aVF.

If both SA node and AV node fails in pacing or the impulses are produced, but cannot be conducted to the ventricles (complete AV block) – in this case the ventricles attain pacing activity. The tertiary pacemakers may be located in the conductive system of the ventricles (bundle of His, bundle branches or Purkinje fibres) however these cells are able to depolarize spontaneously very slowly. If it happens - the ECG will record escaped ventricular depolarization. The shape of the QRS complex will be modified, it yields a typical giant ventricular complex when ectopic focus is localized in one of the ventricles, but QRS complex can be also slim, not prolonged, if the ectopic focus is localized in the His bundle. The delay between the ventricular escaped beat and the previous normal sinus driven ventricular activation is longer than the interval which was recorded in case of the activation of secondary pacemaker in the AV junctional zone. **Again, morphology of QRS complexes of ventricular escaped beat and ventricular premature beat are the same if the focus that has produced them is at the same place of the ventricle, the difference in the time of their onset - premature beat appears earlier than expected and escaped beat appears later than expected.**

B) HETEROTOPIC ESCAPED RHYTHMS

These are the heart rhythms which appear and remain present in case of long lasting inactivity of the SA node. AV junctional rhythm appears in case of inactivity of SA node, and/or ventricular rhythms in case of inactivity of both SA node and AV node or in case of complete AV block. AV junctional rhythms are characterized by the rate of pacing lower than in SA node (40-60 bpm). More details about the AV junctional rhythm are in the section 4.2.3.

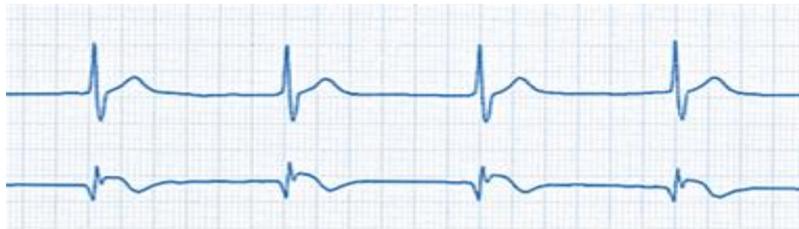


Fig. 27. AV junctional rhythm – no P wave, slow heart rate, but still normal duration and configuration of the QRS complexes, www.frca/ECG

C) VENTRICULAR RHYTHM

The source of this rhythm is usually conductive system of the ventricle. The rate of SDD is approximately 20-40 bpm. Typical is change of the QRS complex duration (more than 0.12 sec) and abnormal configuration. The cause of this finding is abnormal spreading of the depolarization wave through the ventricular musculature from the one point in the ventricles instead of spreading via physiological conduction system which provides quick depolarization with narrow QRS complexes. The impulses are produced in one ventricle and then from here they spread to the other ventricle. Depolarization of the ventricles is not synchronized and it also does not follow described physiological pattern (septum first, then both ventricles, and finally the basis of the left ventricle). This will lead to the changes in the R and S deflection in the chest leads. This is caused by changed direction of the mean QRS vector of ventricular depolarization. Since the conduction form cell to cell in the working myocardium of the ventricles is slower than the conduction via conductive system, this will lead to delay and the QRS complex will be wide. Since the repolarization is abnormal too, the ST – T segment will be in discordant position towards QRS complex. If the cause leading to the activation of the ventricular rhythm was complete AV block, the atria will still be activated by continuing normal pacing from the SA node. P wave may be present on the recording. Some of them may be also hidden in the QRS complexes, but the P and QRS complexes have no temporal relationship. In this case the hart works according to the two rhythms – atria are activated by the SA node (sinus rhythm) and the ventricles are activated by the ventricular ectopic focus. This process of independent action of the atria and ventricles is called AV dissociation. AV dissociation with extreme bradycardia (ventricular rate is 20-40 bpm) may lead to the heart failure.

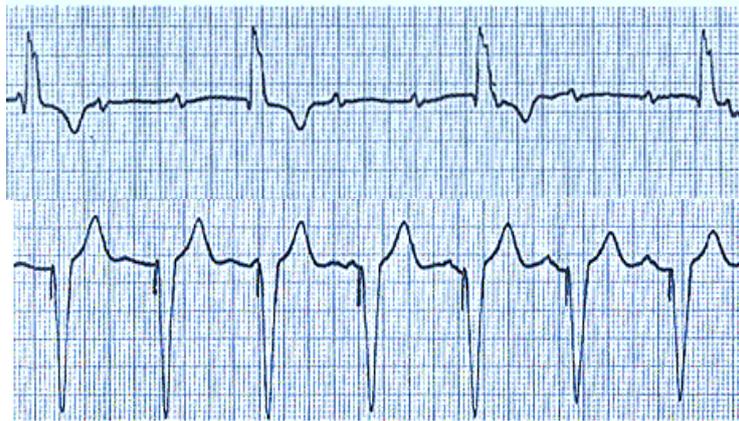


Fig. 28: The upper panel shows ventricular rhythm from the left ventricle with the low heart rate, wide QRS and discordant T. There are P waves in between the QRS complexes; they represent the activation of atria from SA node. Hemodynamic consequences (low cardiac output) were treated by pacemaker implanted to the ventricular system. Small spikes visible in front of every QRS are the spikes of pacemaker activity. www.anaesthesiauk.com/.../ECG/ECGresource14.jpg

5.1.2.2. ACTIVE HETEROTOPY

Remember! The term “active heterotopy” means that impulses depolarizing heart muscle are produced outside of SA node and they are produced faster than is the normal rate of the SA node.

• Causes and mechanisms leading to the active heterotopy

The underlying condition leading to the onset of the active heterotopy is **reduction of resting membrane potential** of the cells in the conduction system outside of the SA node and/or of the cells of the heart muscles (working myocardium).

What is happening to these cells with the change of the RMP?

- **change of conduction speed from cell to cell** (mild reduction of RMP leads to the faster conduction, severe reduction of the RMP lead to the slower conduction, eventually heart blocks)
- **change of the refractory phase** (shorter or longer than normally)
- **change of the automaticity** (increased ability to produce impulses spontaneously)

There are also other conditions which may lead to the onset of active heterotopy. For example, prolonged **QT interval** or the change of the activity of autonomic nervous system innervating the heart. They will be discussed below.

• Main types of pathological processes leading to an active heterotopy

In general, any pathological process affecting the heart tissue can damage the cells to the extent that their metabolism will fail in production of ATP or the membrane of cells is damaged, therefore it cannot

Electrocardiography

maintain the semipermeable state and optimal operation of the ion channels. Consequences of lack of ATP and membrane dysfunction lead to the reduction of RMP.

Pathological processes leading to the lack of ATP and membrane dysfunction are: hypoxia, ischemia, inflammation, mechanical forces (stretch of the muscle in case of high preload or afterload), changes of the ion concentration in extracellular fluid, organic/inorganic toxins, medication, degenerative changes.

• Types of arrhythmias caused by active heterotopy depending on the site of origin

- 1) **Supraventricular** • atrial & • junctional
- 2) **Ventricular**

SUPRAVENTRICULAR ARRHYTHMIAS

a) atrial - premature atrial depolarizations (PAD) = extrasystole

- supraventricular tachycardia
- atrial flutter
- atrial fibrillation

All of these arrhythmias have the same background – it is a production of extra beats in the atria or AV junctional zone (supraventricular parts of the heart), they differ only by the number of produced impulses (isolated impulses lead to the PAD, while permanent pacing will lead to tachycardia). It also depends whether they are produced by one ectopic focus of extra beats (monotopic) or more ectopic foci (polytopic). More than one ectopic focus may indicate that the damage of the atria and AV zone is more serious when comparing to the damage leading to monotopic arrhythmia.

PREMATURE ATRIAL DEPOLARIZATION (PAD) EXTRASYSTOLE

This term means that the atria are suddenly depolarized earlier than they would be depolarized by the activity from the SA node and the source of this depolarization is an ectopic focus producing extra beats in the atria. They are present in case of increased tonus of the vagus nerve, after ischemic damage to the atrial cells, dilatation or hypertrophy of one or both atria and also other pathological processes. Premature atrial depolarization is recognized on the ECG according these hallmarks:

- **depolarization begins earlier than normally** – distance between the last normal depolarization and PAD is shorter than distance between two normal depolarizations
- **P wave is different – in its shape and polarity** - influenced by the position of the ectopic focus in the atria and by direction of the depolarization across the atria
- **polarity of the P way may be reversed** – if the ectopic focus is located close to the AV zone in the lower parts of the atria – depolarization in atria spreads in this case in a retrograde direction
- **PQ (PR) segment can (but not necessarily) be shorter** – the reason is that the impulse will reach the AV zone much faster if the focus is e.g. in the lower part of the atria

Electrocardiography

- **QRS complex would not be changed** – because the ectopic impulse is picked up from the atria similarly to the normal impulse - only in case that the ventricular conductive system is functioning properly

If the premature depolarization from atria reaches the AV zone in its refractory phase, it cannot get through the AV zone to the ventricles – blocked atrial premature beat. There will be only premature P wave which is not followed by the QRS complex.



Fig. 29: Premature atrial depolarization and its main hallmarks – it appears earlier than expected, it has modified P wave, QRS complex is normal, T is concordant and there is no compensatory pause.

SUPRAVENTRICULAR TACHYCARDIA (SVT)

SVT is every single tachycardia which is characterized by quick onset and quick termination and the focus – arrhythmogenic substrate is located in the atria or AV zone. This type of tachycardia was previously called paroxysmal atrial tachycardia; however, it has been identified that the mechanisms leading to it are based on re-entry mechanisms. Therefore, supraventricular re-entry tachycardia is much better name. 70% of the patients affected by SVT are females in their 40 – 50ties. Some of the SVTs are dangerous by extremely high rates and its duration.

SVT is triggered by either increased automaticity of the primary pacemaker (SA node) or secondary (in atria) and tertiary foci in the AV zone which are normally latent. SVT can be triggered also by a pathological process leading to the conditions that “allow” existence of the re-entry mechanism. The most common type of SVT is **AV nodal re-entry tachycardia**. The underlying condition for this type of arrhythmia is existence of at least two functionally distinct conductive pathways in the AV zone. One of them is characterized by fast conduction of impulses with relatively long refractory phase (fast pathway). The other one has a slow conduction speed and quite short refractory phase.

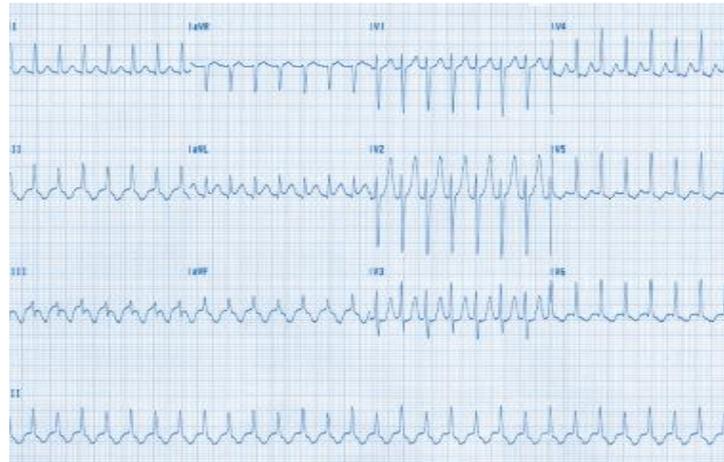


Fig. 30 shows supraventricular tachycardia.

Another example of the re-entry tachycardia in the AV nodal zone is that one affecting individuals with the WPW syndrome (Fig. 31).

During sinus rhythm (on the left side of the panel) the slurred initial portion of the QRS complex (called delta wave) is due to early depolarization of the part of the ventricular muscle through rapid anterograde conduction over the accessory pathway (AP). During orthodromic atrioventricular re-entrant tachycardia (middle section of the panel) no delta wave is seen because all anterograde conduction is over AV zone and through normal His-Purkinje system. Retrograde P waves are visible shortly after each QRS complex. During antidromic atrioventricular re-entrant tachycardia there is maximal preexcitation with wide, bizarre QRS complexes, because ventricular depolarization results entirely from anterograde conduction over the accessory pathway.

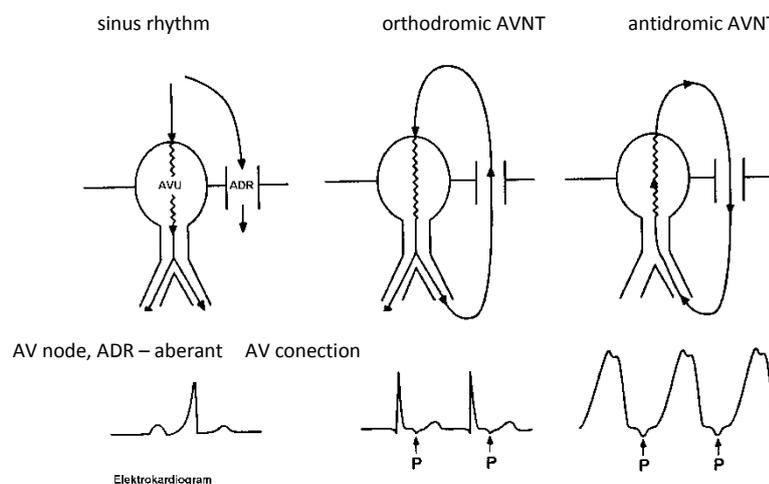


Fig 31. Different forms of the re-entry tachycardia in WPW syndrome (Ganz LI, Friedman PL, 1995)

Electrocardiography

In general, SVT is caused by the presence of the ectopic focus (re-entry mechanisms) with the high activity leading to the activation of the heart (either from atria or AV zone) leading to the fast heart rate. This process can be interrupted by the manoeuvres which increase the activity of the parasympathetic part of the nervous system – Valsalva manoeuvre, Mueller manoeuvre, oculo-cardial or sino-carotic reflex.

This electrical disturbance is identified on the ECG based on the fast heart rate, changes of the P wave shape and polarity (sometimes P and T get together into one waveform – this happens due to fast activity), QRS complexes are usually narrow, because the ventricular part of the depolarization is normal. On the Holter monitoring it is possible to identify the onset of the tachycardia (usually with couple of PAD) extrasystole beginning, and also the termination of it (extrasystolic termination).

WANDERING PACEMAKER

It is an arrhythmia that occurs when the natural cardiac pacemaker site shifts between the SA node, the atria and/or AV node. This shifting of the pacemaker from the SA node to adjacent tissue is identifiable on ECG lead II by morphological changes of the P wave. Sinus beats have smooth upright P waves, while atrial beats have flattened, notched or biphasic P waves. It is often seen in very young, very old individuals, athletes and rarely causes symptoms or requires treatment. Wandering pacemaker is caused by varying vagal tone with increased vagal tone SA node slows, allowing a pacemaker in the atria or AV node, which may become slightly faster. After vagal tone decreases, the SA node assumes its natural pace.

ATRIAL FLUTTER

It is the disturbance of the electrical activity of the heart caused by an ectopic focus with even higher activity than is typical for common tachycardia - one focus of extra depolarizations produces impulses with the rate 250-350 bpm. Although this rate of produced impulses is too high, it still depolarizes whole atria at once, depolarization however spreads across the muscle, which is not optimally repolarized after previous depolarization, that's why the record of this activity shows **specific type of the P wave with the shape of saw teeth** Fig. 30. They are “flutter

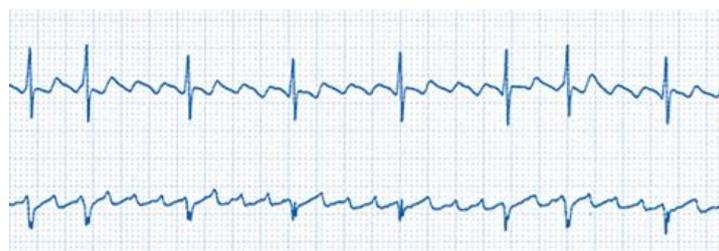


Fig. 32. Atrial flutter with typical flutter waves, and AV block – not all F waves are followed by the QRS (Yanovicz, 2007).

Electrocardiography

waves" (sharp, all same shape and quite high amplitude). All of these depolarizations are approaching the AV zone but not all of them will be also conducted to the ventricles. Some of the conduction fibres in the AV zone have longer AP and refractory phase. When the impulse hits the AV zone in the time of its refractory phase, it is not being conducted through – the QRS complexes will be missing. When the impulse hits the AV zone outside its refractory phase, the impulse will reach ventricles and the QRS will be present on the recording. This phenomenon is known as a rate filter, which allows only every second, third or fourth depolarization to be conducted to the ventricles. The final ratio between atrial versus ventricular depolarization will be 2:1, 3:1, 4:1. 3:1, for example, means that from 300 impulses produced in the atria only 100 will depolarize the ventricles. Ventricular rate 100 bpm is quite mild form of tachycardia and the haemodynamical consequences are not usually very serious. If the AV zone lost its filtering ability, all impulses will go to the ventricles, which will lead to the ventricular rate of 300 bpm – that would be the ventricular flutter already with serious reduction of cardiac output.

Filtering ability of AV zone does not depend only on the rate of the impulses produced in the atria, but also on the quality of this part of the conduction system – pathological processes may easily lead to reduced conduction speed (blocks of 2:1, 3:1), but also to the gain of the conduction ability. This will lead to "full rhythm" when all impulses produced in the atria will depolarize also the ventricles.

Flutter is one of disturbances with typical ECG trace. The main hallmarks of it are typical saw teeth shaped F waves and only every second, third or fourth F wave is followed by QRS complex. QRS complexes are narrow (in case the block in AV is still functioning). The degree of the block is not determined by simple counting the F waves and QRS complexes, but rather by calculating the rate of F waves and rate of QRS complexes and calculating their final ratio, because some of the F waves can be hidden in the QRS complex. QRS complex have higher rate than normally, their shape is normal because the depolarization of the ventricles runs in physiological way. In case of the full flutter rhythm the QRS complexes may be also abnormal, because the spreading of the impulse in the ventricles with such a high rate becomes abnormal too and there is no sufficient repolarization. In case of high rate of the ventricular depolarization, the contraction itself would not be completely synchronized and this together with the extremely short diastolic phase may lead to the heart failure, even to the cardiogenic shock.

Atrial flutter is quite common disturbance of heart electrical activity, because the pathological processes causing it are quite common – e.g. ischemic heart disease, mitral stenosis, hyperthyreosis, hypokalaemia, hypercalcaemia and high alcohol intake (more than 250g of pure alcohol per week).

ATRIAL FIBRILATION AND PAROXYSMAL ATRIAL FIBRILATION

These are the most common arrhythmias in clinical practice. Paroxysmal atrial fibrillation takes approx. 40% of all atrial fibrillations. Incidence of both types increases with the age and with the presence of the degenerative processes in the heart, therefore there is a high chance that not only cardiologists with deal with it, but also GPs and non-cardiologists can (and will) eventually have a patient with atrial fibrillation. The

Electrocardiography

risk of this arrhythmia is that the atria do not have proper contraction; therefore there is not effective emptying of the atria during diastole leading to the risk of thrombosis. Embolus created from atrial thrombus may travel along the blood stream to the brain or peripheral arteries, leading to the life threatening complications. From this point of view, atrial fibrillation could be dangerous type of electrical disturbance.

Fibrillation is an arrhythmia caused by irregular production of impulses in multiple foci in the atria independently and these foci are producing impulses during the systole and diastole. The rate of the impulses is 400-600 bpm and every single focus can produce impulses most likely based on the re-entry mechanism. Impulses spread around the foci in the radial direction until they “meet” the depolarization front from the other foci or they hit the area which was depolarized just a tiny moment before and it is in the refractory phase (therefore it cannot be depolarized). The diameter of the circular area depolarized from each of the foci is different, smaller or bigger, therefore the mass of muscles depolarized by these impulses are logically smaller or bigger. None of these foci can activate whole atria (difference from the flutter). The ECG trace will show small depolarization waves with irregular pattern and different amplitudes (f – fibrillation waves) and the recording lacks P wave – because there is no synchronized depolarization of whole atria. The action of the ventricles is also irregular.



Fig. 33 Atrial fibrillation (Yanowitz FG, 2007)

Imagine the lake surface which is calm – this is the atrial musculature without any impulse. When you throw a stone to the middle of the lake (but only one stone – one ectopic focus) it will ripple the water by the circles of waves spreading from the place of immersed stone to the periphery of the lake. This could be an analogy for the flutter – one focus depolarizes all atria as one stone ripple the surface of entire lake. Imagine now the raindrops (many small foci all over the lake – multiple foci producing impulses in atrial fibrillation). They will ripple the water in completely different way – the circles of waves will be small and will be centred on the rain drops that caused them. This is how we can understand the partial; neither synchronized nor organized activation of the atria in subjects with atrial fibrillation.

Atrial fibrillation on the ECG manifest by the presence of fibrillation waves (f) instead of P wave, which are sometimes identified as the “artefacts” caused by technical difficulties while taking the recording. To be sure that the recording lacks the P, wave one must take a look at the V₁-V₂ – these electrodes are placed on the chest wall practically closest to the atria, and if there is any coordinated atrial activation, they will record nice P

Electrocardiography

waves. If the record lacks P wave in the V_1 - V_2 , very likely there is no P wave and we can move forward to identify further hallmarks of atrial fibrillation. In atrial fibrillation, the ventricular action is always irregular identified by on the unequal distances between RR deflections. This finding correlates also with the physical examination of a patient in whom we find *pulsus frequens, irregullaris et inaequalis* – which mean that the ventricular contractions are irregular, fast and every single stroke volume is different, because every single diastole is different in duration as well. Some of the stroke volumes are too small to cause elastic deformation of the vessels wall (a pulse), that's why very frequent finding could be a pulse deficit - more heartbeats detected over the heart than by a pulse measurement.

Not synchronized atrial contraction will limit the ventricular filling during the diastole, because of lack of the third part of the diastolic filling. Failure of the systolic function of the atria leads to the reduction of end – diastolic ventricular volume and this may lead to the reduction of the cardiac output.

The most common causes leading to the atrial fibrillation are mitral stenosis and mitral regurgitation (with atrial hypertrophy and/or dilatation), hyperthyroidism, pericardial diseases and atrial septal defects.

JUNCTIONAL SUPRAVENTRICULAR ARRHYTHMIAS

Junctional arrhythmias are caused by increased ability of the cells located in the AV zone to produce impulses with the higher rate than being produced by SA node – active heterotopy. The main types of this disorder are junctional premature depolarizations (extrasystole) and junctional tachycardia. AV-nodal tachycardia was described already, so we will focus on the junctional premature depolarizations now. This premature depolarization begins somewhere in the AV junctional zone. The source of the extrasystole is one focus in the AV zone with higher automacy producing these extra impulses. The shape of the ECG curve is similar to that seen in junctional escaped beat, but the difference is that premature depolarization appears on the ECG earlier than expected depolarization from SA node (Fig. 34).

Junctional premature depolarization may appear occasionally, in bursts, or may eventually trigger junctional tachycardia (idiojunctional tachycardia).

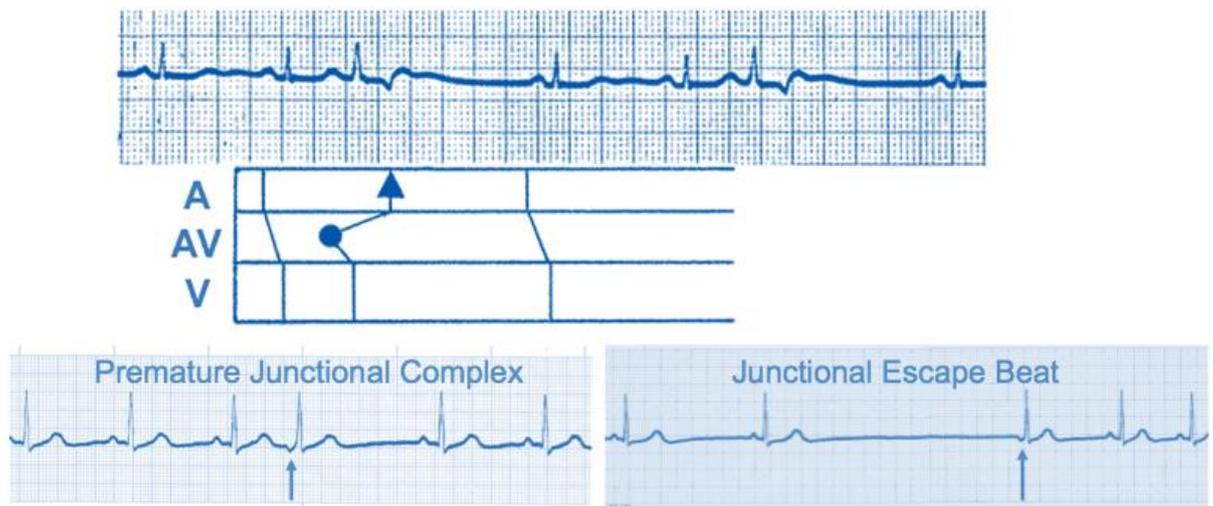


Fig 34. Junctional premature depolarization and difference between premature depolarization and escape beat Yanowitz FG, 2007

VENTRICULAR ACTIVE HETEROTOPY

These are arrhythmias caused by the existence of one focus producing extra depolarizations (unifocal) or more active foci of extra activity (multifocal) disturbances. The ectopic focus may be located in the ventricular conductive system or eventually in the musculature of the ventricles. Depending on the severity of pathological process causing these disturbances they may appear on the ECG recording occasionally in a form of unifocal premature ventricular depolarizations (PVD) or their activity may be prolonged leading to the couplets, triplets or bursts of the PVD. They may eventually lead to a short run of ventricular tachycardia (more than 8 PVC in a series form one focus). If the activity of this ectopic focus remains persistent, it may be a source of ventricular tachycardia, ventricular flutter or ventricular fibrillation – the last one, however, is caused by multiple foci of extra electrical activity). Logically, prolonged (persistent) electrical dysfunction of the ventricles is influencing mechanical performance of the ventricle that may lead to the reduction of cardiac output.

Multifocal PVDs are a product of more than one ectopic focus located in the ventricles. Multiple places with electrical dysfunction always indicate a serious damage of the heart electrical system and also mechanical function. Multifocal PVDs are characterized by different shapes of the QRS (they all begin at different places – therefore they must have different shapes of the QRS complexes) and also the distance between the previous normal systole and PVDs are different. Except from multifocal PVDs there is one type – multiform PVC but it comes from one focus. They are typically seen in patients with digoxin intoxication.

Main mechanisms leading to the ventricular heterotopy are:

- a) onset of abnormal pacemaker mechanisms in the damaged cells caused by the lack of ATP and dysfunction of the membrane ion channels. For example, damage of the cells leads to the reduction of RMP and it

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inactivates the outward K^+ current during the diastole and activates inward Na^+ and Ca^{2+} currents to the cell what leads to increased probability to depolarization of cells.

b) mechanisms of delayed depolarization (afterdepolarization)

Afterdepolarization is a depolarization that begins before the complete repolarization (early afterdepolarization) or immediately after ventricular repolarization (delayed afterdepolarization). It is a pathological phenomenon caused by damage of cardiomyocytes and ventricular conduction system. The source of delayed depolarizations are clusters of surviving cardiomyocytes which are surrounded by the ischemic myocardium, very likely, these surviving cells are not damaged so seriously as the neighbouring cells. These cells are not depolarized simultaneously with the rest of the “healthy” (unaffected) musculature, but they are depolarized later by an impulse brought to them by some alternative ways. The result is the onset of the action potential which can be a trigger for the neighbouring cells – this mechanism may lead to the onset of PVD and other types of ventricular arrhythmias.

VENTRICULAR PREMATURE BEATS (CONTRACTIONS) PVC (EXTRASYSTOLE)

The principle of the PVD is the presence of **one ectopic focus in the ventricles with high automaticity** – this focus is able to produce impulses with the rate higher than are the impulses from the SA node. This focus produces impulses earlier than the normal impulse incoming through the AV zone can reach and depolarize the ventricles. Ventricles cannot be depolarized by impulse from SA node because they are already depolarized by the PVD or they are in their refractory phase. Therefore, this impulse will be blocked in the AV zone but another impulse from the SA node will be conducted to the ventricles, and this gap will lead to the presence of the **complete compensatory pause typical for PVD.**

The shape of the PVD QRS depends on the position of the ectopic focus in the ventricles (because the direction of the depolarization currents influences the final shape of the QRS on the recording. Here the spreading of the depolarization is abnormal, that’s why the **QRS complex will be abnormal as well.** Every student should be able to recognize PVC on the ECG, because this can be negative prognostic factor related to the onset of more serious ventricular arrhythmias.



Fig. 35. Polytopic PVC in a bigeminy with complete compensatory pause (Yanowitz FG, 2007)

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Hallmarks typical for PVD are:

PVC appears earlier than expected depolarization from the SA node. Therefore, there will be QRS complex more close to the previous cycle. Sometimes, the PVD appears immediately after the termination of the previous T wave (phenomenon R on T), which is really bad prognostic sign and it indicates a high risk of ventricular flutter or ventricular fibrillation, mainly in subjects with myocardial ischemia.

Duration of PVC QRS takes longer than 0.12 sec – because the impulse does not depolarize the ventricles via conductive system with the speed (2-4m/s) but via working muscle (cell to cell conduction- 1m/s).

Configuration and amplitudes of QRS complex – during normal ventricular conduction the left and the right ventricles depolarize simultaneously. As a result, depolarization going toward the left (in the left ventricle) is somewhat opposed by simultaneous depolarization going toward the right (in right ventricle), and relatively small (normal) QRS complexes results. But PVD originates in one ventricle which is depolarized the first, before the other ventricle. So there is no simultaneous opposed direction of depolarization QRS vectors. The result is that deflections of PVD are very tall on the ECG record. PVD have greater deflections than normal QRS complexes.

Absence of the P wave – because the depolarization starts from the ventricle and it is happening simultaneously as the atrial depolarization driven by the SA node. This physiological P wave is hidden in the QRS complex produced by the ventricular depolarization driven from the ectopic focus.

T wave is discordant - because the repolarization of the ventricle is also abnormal.

There is full compensatory pause – which means that the next normal depolarization from the SA node cannot depolarize the ventricles as they are in their refractory phase.

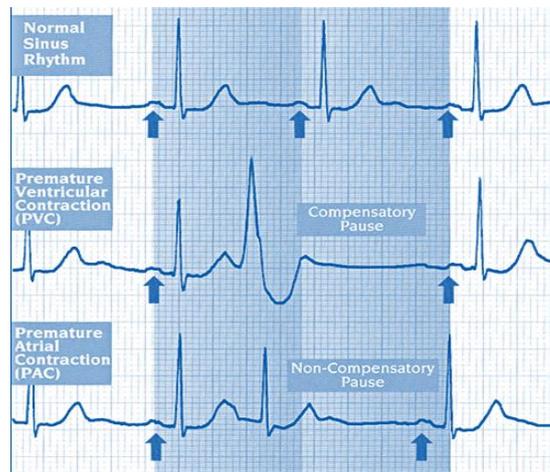


Fig. 36: Compensated and non-compensated premature beat (Yanowitz FG, 2007)

OTHER FORMS OF THE ACTIVE VENTRICULAR HETEROTOPY

Doublets (2 PVD), **triplets** (3 PVD), **salvos** (4-7 PVD) and **episodes of PVD** (more than 8 PVC) are **clustered premature ventricular depolarizations**. They indicate the current status of the ectopic focus producing these extra impulses which is, of course, influenced by intensity of the pathological process affecting the ventricle. The number of PVD produced by the ectopic focus correlates with the severity of pathological process – so if the count of the PVD increases, it may indicate that the process is also getting worse.



Fig. 37: Triplet of PVC (Yanowitz FG, 2007)

This could be dangerous for the haemodynamics, because this focus may get active to the extent that it will result into the **ventricular tachycardia, ventricular flutter or even ventricular fibrillation**. Every of these may lead to death.

VENTRICULAR TACHYCARDIA

It can have a paroxysmal form - **it comes and goes very quickly in an attack** – in this case it is usually temporary with the spontaneous termination and it can have also **persistent nonparoxysmal form**. The attack usually starts with the PVD and it is followed by the paroxysm of the ventricular tachycardia. There are two phenomena which can be observed – **extrasystolic beginning** and **extrasystolic termination** as this paroxysm may starts and may also terminate with the bursts of PVD.

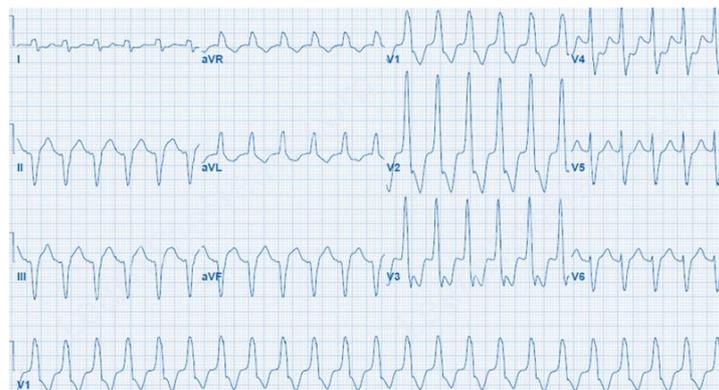


Fig 38: Ventricular tachycardia (Yanowitz FG, 2007)

Ventricular tachycardia is a result of **one super active ectopic focus which produces impulses with the rate 150-250 bpm**. Since we have only one ectopic focus, all ventricular complexes have the same morphology; however, with the fast speed we are unable to distinguish clearly individual parts of the QRS complexes or we are not able to identify whether there is any compensatory pause or not. Sometimes, the recording can show one “quite normal” QRS complex, the reason is that sometimes one of the normal SA node impulses can get through the AV zone and hit the ventricles outside of their depolarization or refractory phase. The result of this is called **fusion beat** (first part of the QRS belongs to the “normal” depolarization wave and the second part of the QRS belongs to the PVD). Presence of either fusion beat confirms the diagnosis of ventricular tachycardia.

Of course, such a high ventricular rate cannot produce optimal cardiac output, especially in the heart that was previously affected by chronic ischemic heart disease or valvular disorders. Reduction of the cardiac output may vary from mild forms to the life threatening intensity.

Ventricular tachycardia **Torsade de Pointes - TdP** (Fig. 39) is characterized by a gradual change in the amplitude and twisting of the QRS complexes around the isoelectric line (see the image below). Torsade de pointes, often referred as a torsade, is associated with prolonged QT interval, which may be congenital or acquired. Torsade usually terminates spontaneously but frequently recurs and may degenerate into ventricular fibrillation. There are many factors predisposing for the onset of TdP – prolonged QT interval, hypokalaemia, hypomagnesaemia, hypocalcaemia, myocardial ischemia and paradoxically some antiarrhythmic drugs which prolong duration of the action potential (amiodarone).

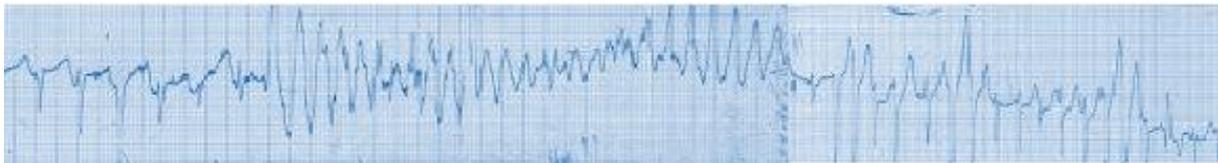


Fig. 39. Arrhythmia Torsade de Pointes with spontaneous conversion to the sinus with some PVC (Vincent GM, 2002)

Prolonged QT interval (Fig. 41, 42) is a phenomenon which is not difficult to recognize on the ECG recording. The background for this prolonged QT is either congenital or acquired abnormality characterized by prolonged repolarization of the ventricles. In individuals with this abnormality, the QT_c interval is longer than 0.47 s in males and 0.48 s in females. This finding on the ECG may not have clinical manifestation, but it is always a risk for the onset of arrhythmias and eventually sudden cardiac death in the future. The risk of arrhythmias or sudden cardiac death in this case has nothing to do with the damage of the coronary arteries. Syncope or sudden cardiac death is frequently related to the physical strain or emotional stress. Additional findings on the ECG record in individuals with prolonged QT are changes of the T wave (biphasic T wave, T wave with a notch) and prominent U wave. Some patients may have sinus bradycardia and paradoxically, physical exercise does not lead to the tachycardia, but the bradycardia persists even during exercise. Most common

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cause of death in these patients is ventricular tachycardia **Torsade de Pointes**, frequently resulting to ventricular fibrillation. This may lead to the sudden cardiac death (the individual will die) or so called abortive sudden cardiac death, when the individual has had early intervention.

The main mechanisms responsible for this disturbance are dysfunctional K^+ channels (participating in the repolarization). That's why this process belongs to the "channelopathies". Dysfunction of the K^+ channels is frequently provoked by sympathetic activity and they respond quite nicely to a medication with beta-blockers.

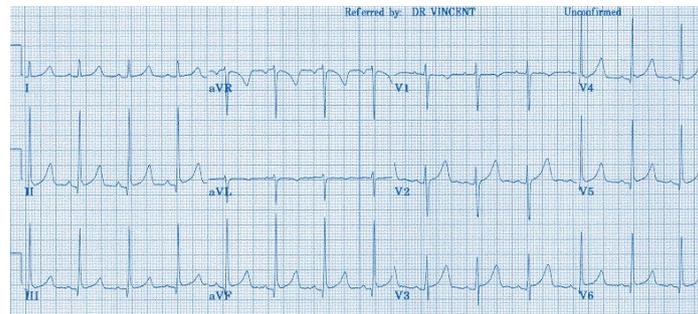


Fig. 40. Syndrome of long QT interval in 11-year-old boy with normal T waves. Mean duration of the QT is 0.48 sec (Vincent GM, 2002)

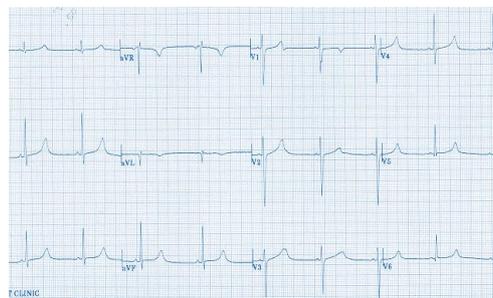


Fig. 41: Syndrome of prolonged QT in 15 years old boy, basis of the T wave is wide (Vincent GM, 2002)

One of the most common reasons leading to the ventricular arrhythmias (active ventricular heterotopy) is a **presence of afterdepolarization**. The sources of afterdepolarization are small islets of the surviving cardiomyocytes surrounded by the ischemic myocardium which is not depolarized simultaneously with the non-ischemic cells, but **their depolarization is delayed**. The consequence of this can be an onset of late potential during repolarization which is a trigger of PVD and other types of ventricular arrhythmias. Delayed depolarization (afterdepolarization) of the islets of surviving cardiomyocytes in the field otherwise damaged cells by ischemia can be caused also by delayed conduction through the ischemic focus (there is low RMP and higher resistance of the gap junctions).

PVC are frequently firmly connected to the previous normal depolarization. We can recognize connection of one normal depolarization with one PVD (**bigeminy**), two normal depolarizations and one PVD (**trigeminy**) or eventually between three normal depolarizations and one PVD (**quadrigeminy**).

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The cause of this phenomenon is a mechanism of early afterdepolarization of the ventricular muscle in the repolarization phase. This term “**early afterdepolarization**” describes sort of deformations or oscillations that appear in the phase three or four of the action potential (fig. 42).

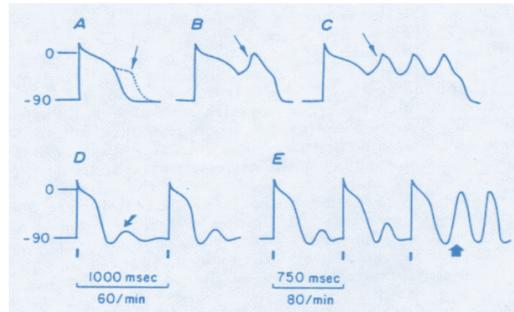


Fig. 42: Mechanisms leading to early and delayed afterdepolarizations in the Purkinje fibres. A – normal depolarization, interrupted line represents prolonged repolarization; B, C – onset of one or more early post potentials, which can trigger an arrhythmia, D – late post potential of subthreshold intensity which does not cause myocardial depolarization, E – suprathreshold late postpotential leading to the myocardial depolarization (arrow).

Described oscillations and deformities have amplitudes of microvolts, and they are very likely caused by the factors limiting the intensity of the repolarization current (outward leak of the K^+ out of the cells) and/or prolong the duration of the depolarization current (inward Na^+ and Ca^{2+} current). It is important to note that such oscillations of the membrane potential in a group of cells may be a trigger leading to the depolarization of the neighbouring cells and this may lead to the PVD.

Specific type of the PVD is the ventricular **parasystole** – **which is the result of dual rhythm**. QRS complex of the parasystole is the same as it would be for PVD, but parasystole is a consequence of dual pacing. One caused by the ectopic focus in the ventricles and one caused normally by SA node. Ventricular ectopic focus produces impulses with the slow rate and this focus is characterized by the entrance block – it means that ongoing depolarization of the ventricles cannot depolarize it and it prevents it from deactivation. Impulse produced by this focus can spread to the ventricle (there is not exit block) if the muscle is not depolarized or in the refractory phase. So not every impulse produced by this focus can depolarize the ventricles. Thanks to this protective mechanism only few of the produced impulses from ectopic focus can depolarize the ventricular muscle. Therefore, the evidence of parasystole require longer stripe of the ECG and search for the shortest interval between two ectopic depolarizations. If the inter-ectopic interval - i.e. timing between the ectopic depolarizations are some multiples (1x, 2x, 3x etc.) of the basic rate of the parasystolic focus, just after that observation we can conclude that what we see is a parasystole or parasystolia.

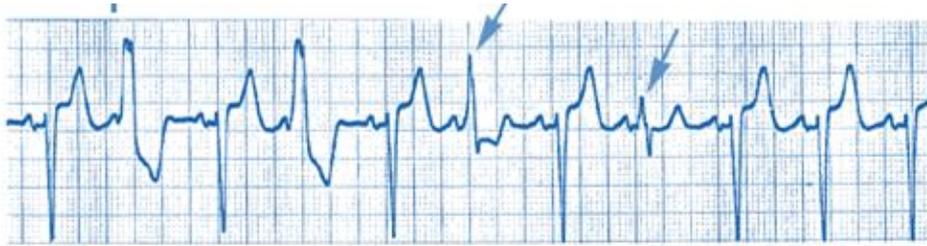


Fig. 44: Ventricular parasystole (Yanowitz FG, 2007)

VENTRICULAR FLUTTER

This is arrhythmia which is directly endangering the human's life. It is caused by the high rate activity (250-350 bpm) of one ectopic focus in the ventricles. Each of these impulses depolarizes both ventricles, but the recording does not have the characteristics of normally shaped QRS complexes but rather plane waveforms, because the action is very fast. The ECG trace records smooth sinusoidal waves instead of normal QRS complexes Fig. 45. www.anaesthetist.com/.../ecg/images



Fig. 45: Ventricular flutter

Activation of the ventricles with such a high rate does allow neither effective contraction nor relaxation of the muscle and it also limits the diastolic filling of the ventricle. Cardiac output is reduced, nearly to zero and there is considerable hypoperfusion in the systemic circulation. Another risk is that this arrhythmia may transit to the ventricular fibrillation.

VENTRICULA FIBRILLATION

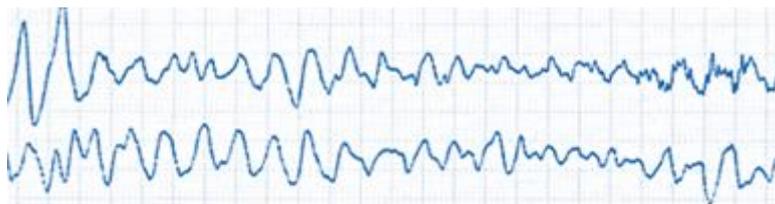


Fig. 46: Ventricular fibrillation

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This is a fatal arrhythmia caused by the uncoordinated activity of the multiple foci of “extra” activity in the heart ventricles. The result of this activity is chaotic activation of small fields of musculature around the foci, and also chaotic contraction of this small area. Ventricle as a complex structure fails to produce any effective contraction and pressure (in fact it does not contract in a pattern of systole of whole ventricles). There is zero cardiac output, therefore the circulation stops. The ECG curve records chaotic irregular electrical activity with irregular amplitudes. It can be effectively treated by defibrillation.

5.1.3 CONDUCTION DISTURBANCES

J. Plevková

5.1.3.1. IMPORTANT NOTES ABOUT THE ANATOMY AND PHYSIOLOGY OF CONDUCTIVE SYSTEM

Cells of the conduction system are characterized by the ability of self-excitation and this ability is called autorhythmicity. Their membrane can spontaneously depolarize during diastole without any external impulse and this process is responsible for rhythmogenesis. Promptness of spontaneous diastolic depolarization is gradually decreasing from the SA node, through AV node down to the ventricles. Cells of the SA node can spontaneously depolarize 70-80 times per min, AV nodal cells 40-60 times per minute and the cells of the ventricular conduction system can depolarize spontaneously 30-40 times per minute.

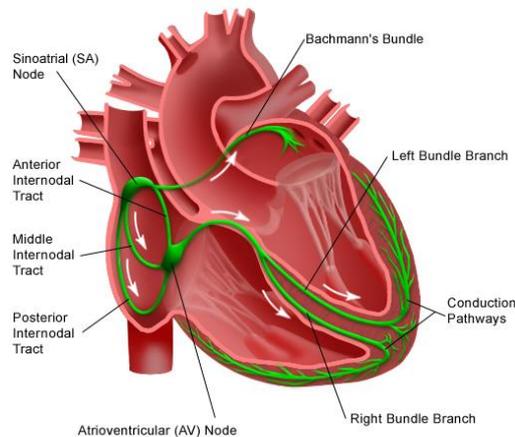


Fig. 47 Conductive system of the heart.

Cells participating in normal physiological rhythmogenesis have specific electrophysiological features that differ from the features of the “working” myocardial cells. The differences are morphological and of course functional. While the main function of the “working cells” is to provide a mechanical force, the main role of the cells in the conductive system is to produce and conduct impulses through the heart. The cells in the SA node, AV node and the ventricular system have specific types of ion channels in cell membrane which are responsible for slow depolarizing inward Na^+ current leading to discharge of action potentials. These mechanisms were described in details in the first section of this textbook. But what happens with the impulse after its production in the SA node?

Cells of the SA node are surrounded by the atrial musculature, which is characterized by quite low conduction speed 0.3 m/s, but the SA node and the AV junctional zone are further connected by preferential conductive pathways. These fascicles cannot be considered as internodal, because they do not start straight in the sinus node and they do not terminate straight in the AV node. Their main role is to provide fast and

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synchronized depolarization of the atrial musculature. Their conduction speed is about 1 m/s. These fibres are similar to the Purkinje fibres and they form three bundles named after Bachman, Thorell and Wenckebach.

- **Heart tissues do not have good conduction abilities.** This disadvantage is minimized by some other changes, for example in the cells with the high RMP conduction from cell to cell lead to the production of the “new” action potential (new impulse) in every single cell, therefore, the value of the depolarizing potential does not decrease with the number of depolarized cells. This is called **depolarization without decrement**. This phenomenon does not apply to the cells with the low membrane potential. In this case the impulse will spread with the decrement, therefore it can vanish along the way from the cell to cell. This is one of the mechanisms leading to heart blocks. Cells in the different parts of the conductive system have different values of the resting membrane potential and this is the reason why they have also different conduction speed when carrying the impulse to the next parts of the conductive system or to the myocardium itself. This is attributed again to the value of their membrane potential.

Parts of the conductive system containing cells with high RMP can conduct depolarization quickly, parts containing cells with lower RMP conduct depolarization slowly. If the RMP of the cells is very low, they cannot conduct depolarization at all (**the conduction will be blocked**). One of the main drives for the depolarization speed is the difference of potentials between already depolarized and not yet depolarized part of the cell (or the muscle fibre). Huge difference of potentials leads to quick conduction of the impulse from cell to cell (except from the conduction through the nexus) in a way of local electrical currents.

The background for the ECG curve are the currents present in the myocardium during depolarization and then repolarization of the cardiomyocytes spreading through the extracellular space in the direction of depolarization. These local currents reduce resting membrane potentials of the cells located in front of the depolarization wave, thus preparing them for upcoming depolarization. Therefore, the difference between the depolarized and not yet depolarized parts of the heart leads to formation of **local electrical currents which increase the speed of conduction**. Local currents can be effective to a distance of couple of millimetres what means that the impulse can “skip” couple of cells with decreased conductivity e.g. damaged cells (the length of the cell is 50 -100µm). This mechanism can be useful in the spreading of the impulse to the parts of the heart located behind the damaged field.

- **Delay of the impulse in the AV node** – it is physiological delay of the conduction from atria to ventricles in AV nodal conductive tissue. The main role of this delay is to provide an optimal time for the ventricles to be filled with blood from the atria, that’s why depolarization of the ventricles starts later after the previous depolarization of the atria. Therefore, the contraction of the ventricles will start after the ventricles achieve optimal end diastolic volume. Functional background for nodal delay is the existence of the cells with very low resting membrane potential (in the central part of the AV node the cells have potential only -50 mV). These are called “slow cells” or “transitional cells”, their conduction speed is 0.02 – 0.05 m/s. Activation of these cells by depolarization wave coming from atria down to ventricles opens only the slow sodium channels, therefore, they only generate an action potential with the low amplitude. This will reduce the speed of conduction (as we

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explained that the amplitude of the membrane potential and action potential determine the conduction speed). This is the place in the heart with the slowest conduction. Slow conduction is typical also for the node itself 0.05 m/s. and total delay of the impulse in the AV zone is 0.13 s.

• other specific functions of the AV nodal part

The AV nodal junction is the only electrical connection between the atria and ventricles in the physiological conditions. This is the only place where depolarization can spread to the ventricles, otherwise atria and ventricles are electrically separated (isolated). Damage to this zone leads to disconnection and the impulse will not reach the ventricles. This is the reason why the AV zone has a specific architecture that guarantees higher degree of safe conduction to ventricles. Simplified description would say that the number of the conducting fibres decreases in the direction from the atria to the bundle of His. This principle of the organization of the conductive fibres is called convergence. This architecture provides conditions for one. It worsens or enables the retrograde spreading of the depolarization from the ventricles back to atria. In case of the retrograde spreading, the impulse should depolarize higher number of fibres and this would lead to the decrease of its magnitude to the value when it would not depolarize other cells any longer – therefore the conduction will stop. It is also important to note that the cells in AV zone have less of gap junction connections which increases the resistance against the electrical current.

Lower resting membrane potential, less gap junctions and convergence of the conductive fibres - all of them contribute to the delay of the impulse in the AV junctional zone.

Very important feature of the conduction in the heart is a **unidirectional character**. Depolarization normally spreads only in the anterograde fashion (means forward from SA node, to atria, to AV zone and to ventricles). Some pathological circumstances may lead to the possibility of retrograde spreading of the depolarization. This is possible in the AV zone, where we normally have many conductive pathways with different resting membrane potentials and different refractory phases. To set some clear examples of retrograde spreading – junctional rhythm with the retrograde atrial activation or initiation of the re-entry mechanism in AV junctional zone.

In case of physiological concentrations of ions in extracellular fluids, normal body temperature and availability of ATP conduction in the heart is always:

- onedirectional
- spreads without decrement
- keeps the particular speed in atria, AV zone and ventricles

5.1.3.2 CAUSES AND MECHANISMS LEADING TO THE CONDUCTION DISTURBANCES

Causes and mechanisms leading to the impaired conduction either via conductive pathways or from cell to cell are the same or very similar for every part of the heart. In general – the conduction will be slower (leading to “heat blocks”) in these cases:

- **decrease of the resting membrane potential (movement towards zero)** – the higher the membrane potential the higher speed of conduction. The main mechanisms leading to the reduced resting membrane potential are – lack of ATP (hypoxia, ischemia, inflammation, hypertrophy (leading to the relative lack of the energy), tension in the wall (increases consumption of ATP, ion changes in extracellular fluid) – all processes leading to the lack of ATP may influence the operation of the ion transporters and permeability of the membrane which determines the electrical processes in the heart
- **presence of the inflammatory infiltrate** (influences the resistance of the extracellular compartment in the heart and may decrease the spread of depolarizing currents in the tissue)
- **accumulation of amyloid**
- **excessive fibrosis in the heart (mainly as a consequence of degenerative or reparative processes)**
- **hypertrophy and dilatation** – may lead to the lack of the ATP, but they also prolong the time necessary for depolarization of entire muscle
- **change of the ion channel function** - by parasympathetic dominance (acetylcholine reduces conductivity), beta-blocker, Ca channel blockers, digoxin, antiarrhythmic drugs – lidocaine, phenytoin)

5.1.3.3 SINUS BLOCK

The term sinus block characterizes a disturbance in which the impulses are generated in sinus node, but for some reasons they cannot be conducted to the atrial muscle.

There will be a flat line - no P wave, no QRS, no ST, T because the heart is not depolarized. This disturbance can be only occasional (then the flat line will be seen only occasionally), but it can be also longer lasting or even permanent sinus block. In these cases, some of the secondary pacemakers in the AV junctional zone become the pacemaker and the heart will work according to the AV junctional rhythm. Therefore, escaped beats or escaped rhythms are also frequent findings on the ECG trace.

Sinus block has the same ECG manifestation on the standard 12 lead record as sinus arrest. In sinus arrest, impulses are not generated but in sinus block impulses are generated but they cannot get through. This can only be recognized by a special electrode placed directly to the SA node during an invasive procedure.

The electrophysiological classifications describe three degrees of SA block:

- **1st degree SA block** – this disorder is characterized by delayed conduction from the SA node to the atrial muscle. It cannot be recognized on standard ECG and it takes special electrocardiology approach to identify this

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disorder. Clinical significance of this disturbance is debatable since there is no haemodynamic effect because all impulses generated in the SA node will depolarize the heart in a physiological way.

- **2nd degree SA block** – this disorder is characterized by inability to conduct some of the SA node generated impulses to the atria – therefore it is occasional lack of conduction. This will manifest by the absence of the P wave, QRS, ST-T (Fig. 38). Haemodynamic impact depends on how many impulses are not conducted. If it is every second or third impulse, the cardiac output may be considerably reduced.

- **3rd degree SA block** means that no impulses are conducted to the atria with the necessary activation of the secondary pacemaker in AV zone. Therefore, the most common manifestation of complete SA block is the presence of junctional rhythm (unless the patient is on Holter monitor and we have the chance to observe the period of the SA block till the restoration of the AV junctional rhythm) as a passive heterotopy.

Clinical manifestation of SA block can be “cardiogenic syncope”, a transient loss of consciousness caused by reduced cerebral blood flow with a tendency to spontaneous recovery – mainly after the secondary rhythm is restored. Most common causes of SA block are vagotony, digitalis toxicity or hyperkalaemia.



Fig 48. SA block (occasional) lack of P wave, QRS, ST-T on the trace. The arrows are focused on the points of “expected” but not shown activities. This is second degree SA block, where only some impulses are not conducted.

5.1.3.4 DISTURBANCES OF CONDUCTION IN THE AV ZONE

These disturbances can not only manifest by slowing or complete block of conduction, but also by accelerated conduction via alternative pathways which will excite ventricles much earlier that it would happen normally. So now we will talk about the **AV blocks and syndromes of preexcitation**.

5.1.3.4.1 AV BLOCKS

1st degree AV block

1st degree AV block is characterized by the delay of the atrio-ventricular conduction to more than 0.2 sec. Although the AV zone has the lowest conduction speed and it is desirable for the heart to work properly, in this case the delay is longer than that seen in physiological circumstances – the PQ is longer than 0.21 sec (normal is 0.12 – 0.20s). Some extreme delays may lead to the PQ prolongation to 0.4 s.

All impulses that reach the AV zone are conducted, but with a delay. There is no haemodynamic impact on the cardiac output, because all impulses will reach and also depolarize the ventricles number of SA generated impulses is equal to the number of ventricular depolarizations/contractions, therefore every single P wave is followed by the QRS complex. This disturbance does not lead to the bradycardia.

The causes of the 1st degree AV block are dominance of the vagus nerve, digitalis medication, β -blockers, Ca channels blockers, rheumatic fever, diphtheria or streptococcus toxins. It can be also caused by diaphragmatic myocardial infarction, myocarditis or degenerative heart diseases.

In case of really long diastole, the first heart sound is bit silent, because the leaflets of the mitral and tricuspid valve are too close to each other by this increased end diastolic volume, so their closure would not be that noisy as normally.



Fig. 49 AV block first degree with prolonged PQ, but every single P wave is followed by QRS complex. All impulses are conducted through the AV zone, but with a delay.

2nd degree AV blocks

Typical feature of this disturbance is that some of the impulses that reach the AV zone are not conducted to the ventricles. On the ECG trace some of the P waves are not followed by the QRS complexes. Based on the pathomechanisms leading to the particular blocks, we distinguish two main types of this block - Wenckebach periods (= Mobitz type I) and Mobitz type II.

Wenckebach's periods (Mobitz I)

Mobitz I is characterized by progressive slowing of the conduction in the AV zone from beat to beat, until the conduction of one impulse fails. This repeats periodically in cycles.

On ECG, this block manifests by progressive prolongation of the PQ interval above physiological values, the intervals are longer from beat to beat because of progressive worsening of conduction via AV zone. When

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the conduction is at its worst, then the impulse does not reach the ventricles and there is not QRS complex following the P wave. What is the cause of these periodic changes in the AV zone?

The cause of the progressive worsening of the conduction in AV zone and final block of the “last impulse” in a sequence are changes of electrophysiological features of the cells in the AV zone. As it was mentioned already, the conduction speed depends on the amplitude of RMP, which is of course influenced by availability of ATP. Ischemia, hypoxia or increased workload may lead to the depletion of ATP and also changes of the RMP. In case of the ATP lack, mainly the repolarization to the optimal RMP is difficult. Therefore, the next conduction in a sequence is depolarizing the cells with reduced RMP (logically, this will be slower). After this conduction, the repolarization is again not optimal, what decrease the RMP even further. Next impulse is depolarizing cells with more reduced RMP (this will be slower than the previous one). This will repeat several times until the impulse approaching the AV zone finds it “not repolarized” yet) – means the AV zone will be still depolarized or in its refractory phase – so the impulse will not be conducted (this will manifest by missing QRS complex on the recording). During this pause, the AV zone repolarizes properly and the upcoming depolarization will be conducted to the ventricles with normal conduction time and the sequence of progressive prolongation of PQ will repeat.

Severity of this disorder depends on the count of impulses which were conducted to the ventricle out of impulses generated in the sinus node. This can be expressed in ratio atria to ventricles = $n:n-1$, where n is the count of impulses generated in the SA node. The highest possible ratio of Wenckebach periods is 3:2 (ratio between atrial and ventricular rate), because there are 3 PQ intervals where the progressive prolongation can be observed. If there are only 2 PQ, we can see that one is normal, one is bit longer or shorter, but we would not be able to see this progressive prolongation any longer.

The pathological process leading to the Mobitz I AV block is localized in the AV node itself (in 75% of cases) and if it is not caused by myocardial infarction, the mortality is not high. There is however a risk of progression to the complete AV block, but it is very low in contrary to the Mobitz II where the risk of complete AV block in very high in fact.

This disturbance - Wenckebach period (Mobitz I) can appear in the vagus nerve dominance, in athletes, adolescents, after the heart septum surgery, valvular surgery, digitalis medication, β -blockers, Ca channels blockers or medication with antiarrhythmic drugs, e.g. amiodarone. Clinical manifestation is rare; it can be a sort of accidental finding on ECG recording taken for other reasons. If it manifests, then the patients are reporting palpitations, syncope or they feel that the heart skipping a beat.



Fig. 50. Wenckebach's period on the ECG in lead V1. There is progressive prolongation of the PQ, and finally one missing QRS complex after a P wave appearing in normal time.

Mobitz II

This block is characterized by the presence of normal and constant PQ intervals, and some of the P waves are not followed by QRS complex. Pathological processes leading to this type of disorder typically affect the section of the AV zone below the compact node, therefore distal parts of the AV zone are affected (very likely bundle of His). There is very high risk of this block to become complete AV block (3rd degree AV block) and this is also the reason why Mobitz II has much higher mortality than Mobitz I. The main causes of Mobitz II are myocardial infarction and myocarditis. Severity of this disturbance and its impact on the ventricular rate (and cardiac output) is expressed by a ratio n:1, where n is a number of impulses produced in the SA node (70-80 bpm) which are followed by a blocked conduction. Therefore, for example in Mobitz II 3:1 – every third impulse is conducted, in Mobitz II 2:1 – every second impulse is conducted. Now it is clear that if only one third or one half of total SA node depolarization is conducted to the ventricles, it may lead to the 1/2 or 1/3 reduction of the cardiac output. Patients cannot live with such a ventricular rate, they can however survive, but not live - that's why they are treated by the implantation of the permanent external wire pacemaker.



Fig 51: AV block 2n degree, Mobitz II 2:1. Missing QRS complexes are marked by arrows

3rd degree AV block

Also is called complete AV block. This disturbance is characterized by complete inability of the AV zone to conduct impulses. Impulses are normally produced in the SA node, but they are not conducted to the ventricles. What that means actually for the heart? Atria are depolarized by the impulse from the SA node in a normal pacing process, so the P waves are present, they are regular, with a normal shape and polarity. But the ventricles are depolarized from the ventricular conductive system. Therefore, the QRS complexes will appear on the record in a random position in relation to P waves, there is no relationship between atrial and ventricular depolarization. This is called AV dissociation. QRS complexes may be narrow (if the tertiary pacemaker is located above the bifurcation of the His) or they can be wide (> 0.12 s) if the pacemaker is located below the bifurcation of the bundle of His. The ventricular rhythm leads to bradycardia with the rate 20-40 bpm.

Acute complete AV block is not followed by the activation of alternative pacemaker immediately and this will lead to the asystole. Clinical manifestation of this process is Adams-Stokes syndrome characterized by asystole, unconsciousness and cramps of striated muscles. The ventricular or distal junctional rhythm begins after several seconds, and this interval is called pre-automatic phase. Even though the substituting pacemaker

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generates impulses, their rate is frequently not sufficient to provide optimal cardiac output; therefore, patients usually end up with implanted wire pacemaker.

Causes of the 3rd degree AV block are mainly myocardial ischemia – MI located at diaphragmatic wall, congenital heart diseases, and surgical interventions on heart septum.



Fig. 52: The upper panel shows complete AV block with ventricular rhythm (wide, slow (30 bpm) QRS complexes with abnormal configuration). The patient received an artificial pacemaker to treat severe bradycardia. Spikes of the pacemaker are nicely seen in front of every single QRS complex.

5.1.3.4.2 ACCELERATED AV CONDUCTION – PREEXCITATION

In previous section we discussed mainly disturbances leading to the heart blocks caused by slowing of the conducting or by cell to cell uncoupling. These disturbances may have negative impact on the cardiac output, because for some of them a lack of ventricular depolarizations is typical. Now we are going describe a disorder in which ventricles are depolarized by abnormal pathways which bring the impulse to ventricles much earlier than it would be brought there by AV junctional system. These disorders are called syndromes of short PQ or preexcitation syndromes and most known of them is WPW syndrome.

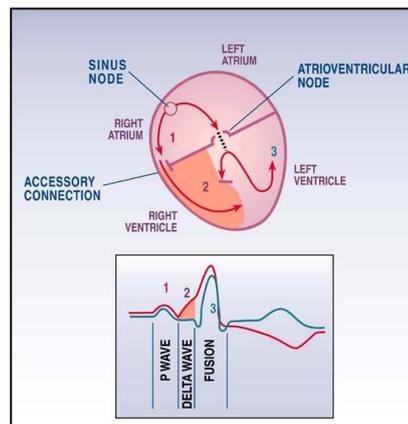


Fig. 53: Diagram showing abnormal conductive connection between the atria and ventricles outside the AV zone and the impact of this abnormal pathway on the ECG curve (Yanowitz FG, 2007)

Cause leading to preexcitation is a presence of abnormal conductive pathway (a sort of a shortcut) between atria and ventricles, which are normally electrically separated. The only connection where the depolarization may pass from the atria to the ventricle is the AV junctional zone. This part of the conductive system is characterized by ability to slow down the conduction, because a little delay is necessary to provide an optimal duration of the diastolic ventricular filling.

Accessory abnormal connections are muscle connections between the right atrium and right ventricle, or between the left atrium and left ventricle and they are frequently consequences of imperfect electrical separation of atria from ventricles. This process normally finalizes during the embryologic development of the heart and may be connected with the congenital malformation of annulus fibrosus, mitral or a tricuspid valve. Approximately 0.2% of population may have these connections, however they are clinically silent, they could be identified on the ECG record taken for some other reason (preventive check, sport medicine check etc.). What makes them different from the AV zone? Their conduction speed is higher than in AV zone and they can conduct impulses in both directions – anterograde and retrograde spreading of the depolarization.

Accessory pathways can be located inside the AV zone and outside of it. If they run along the tissue forming the AV junctional zone, they make the connections between the atria and the bundle of His directly, by-passing the AV zone. This leads to manifestation of syndrome of short PQ without delta wave also called LGL syndrome (named after Lown-Ganong-Lewine).

If the accessory pathway exists outside of the AV node this leads to the WPW – Wolf-Parkinson-White syndrome of preexcitation with presence of delta wave. Fast anterograde conduction of the depolarization from the atria to the ventricle lead to the depolarization of the small area in the ventricle even before the impulse passing through the AV zone can hit it. This leads to the extremely short PQ interval and delta wave as a marker of “premature” activation of ventricular musculature surrounding the accessory pathway. The delta wave makes the QRS complex to look wider than it normally is, because there is a fusion of the delta wave and

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QRS – as a demonstration of the fusion of two depolarization lines – one normal and one abnormal arriving to the ventricles via abnormal conductive pathway.

The significance of these connections is negative, because they can conduct with quite fast speed and can conduct also both ways – frequently they may become a part of the AV nodal re-entry circuits giving a rise to SVT with considerable haemodynamic consequences.

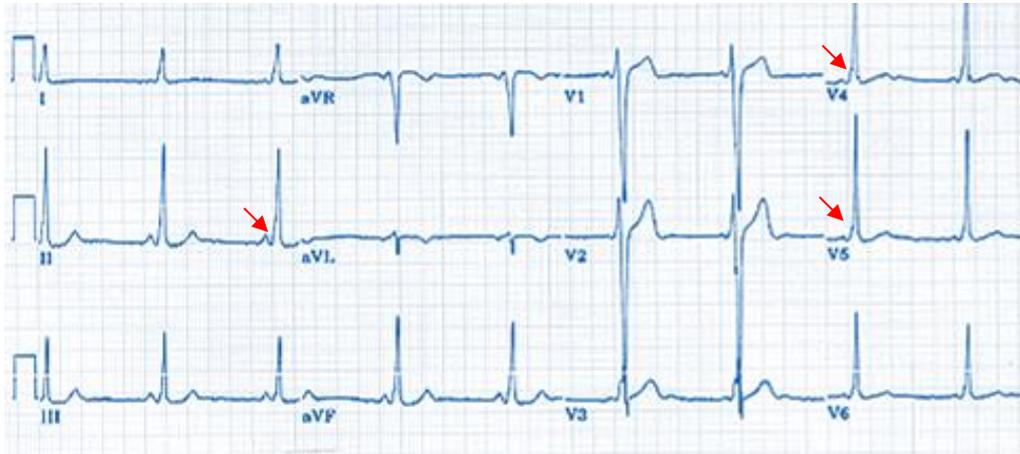


Fig. 54. WPW syndrome with extremely short PQ and delta waves (arrows)

Condition of if the impulse in the ventricles

This system consists of **right and left bundle branches and the Purkinje fibres**. It is important to note that this system has very high conduction speed (4 m.s^{-1}) and the action potential in these cells lasts longer than in the neighbouring cardiomyocytes. These cells also have ability of spontaneous diastolic depolarization, but the rate of it is slow ($20\text{-}40 \text{ min}^{-1}$). To compare the rate of SDD in sinus node ($>60 \text{ SDD per minute}$) and the structures of the AV node ($40\text{-}60 \text{ min}^{-1}$). Conduction from the Purkinje fibres to the cardiomyocytes has typical spatiotemporal pattern and it is the underlying condition for the spreading of the contraction wave across ventricular musculature. **Depolarization starts at the left side of the interventricular septum, approximately in its middle part and the depolarization spreads from the left to the right side of the septum. Later the depolarization spreads quickly across the ventricular musculature from the subendocardium to subepicardium.** Both ventricles are depolarized at the same time, however, the depolarization of the left ventricle takes a bit longer. Depolarization starts at the apex of the heart and it is directed towards the basis of the heart (also the contraction of the ventricles starts at the apex and continues to the basis). The last part of the left ventricle which is depolarized is the basal part of the left ventricle and the upper part of the septum (Fig. 45). The reason why depolarization of the left ventricle takes longer is that left ventricle contains stronger mass of the musculature when compared to the right side.

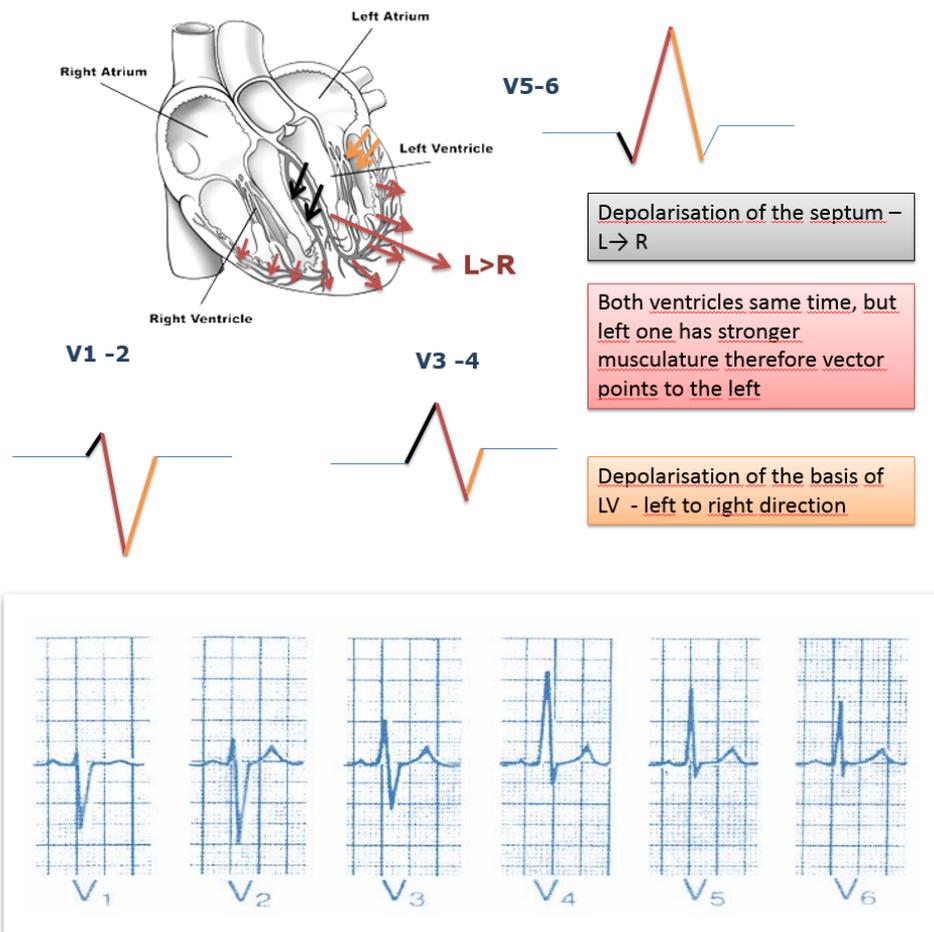


Fig. 55: Direction of the main depolarization vectors (sum all depolarization currents and their direction) towards employed electrodes (V₁₋₂), (V₃₋₄) and (V₅₋₆), and “normal” shapes of the QRS complexes in the precordial leads – rS in right precordial, RS in “septal” and qRs in left precordial leads.

Understanding of the ventricular conductive problem requires the knowledge about basic principles of the ECG recording, position of the leads in the precordium and the principles why the ECG electrodes record positive, and why they record negative deflections. Just to repeat – if the depolarization goes “towards” the electrode, the device will record positive deflection, if the depolarization goes “away” from the electrode, it will record negative deflection. The final direction of the vector describing depolarization of the certain parts of the heart muscle is a sum of all vectors of all depolarizing currents present in that area.

The sum of all depolarization in the certain area also determines the amplitude of the vector, therefore the magnitude of the deflections on the record (Q, R, S amplitude). For example, hypertrophy of the

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heart chambers leads to production of stronger depolarization currents – therefore the S and R deflections in the particular leads exceed the normal voltage criteria.

Duration of the depolarization and repolarization is influenced also by the mass of the muscle, but also by the availability of ATP in the cells and ion distribution. If depolarization and repolarization take longer time, the duration of particular deflection of the ventricular complex will be longer than predicted duration.

Physiological depolarization of the ventricles is very fast (QRS complexes <0.11s), follows physiological spatiotemporal pattern (normal morphology of QRS in precordial leads) and it is followed by physiological repolarization (ST – T), where T is normally concordant with the main amplitude of the QRS complex.

Some important notes that help us understand disturbances of the impulse conduction in the ventricles

- Left and right bundle branches are located in the septum, underneath the endocardium
- Right bundle branch depolarizes distal third of septum and the right ventricle
- Left bundle branch depolarizes upper two thirds of septum and the left ventricle
- Conduction in the Purkinje fibres is very fast, directed from the subendocardium to subepicardium
- Conduction speed in the ventricular musculature is 0,03-0,5 m/s

Ventricular blocks – bundle branches blocks

Right or left bundle branch blocks are caused by the slowing or complete block of the impulse conduction after the impulse normally produced in the SA node depolarized atria and was successfully conducted through the AV zone. Typical feature of the bundle branch blocks is desynchronized depolarization and also repolarization of ventricles, overall electrical activity in the ventricles is prolonged and the higher amplitudes of ventricular deflections caused by desynchronized movement of the depolarization wave through ventricles. There are several aspects of these blocks that should be discussed.

1. Time aspect

Interestingly, ventricular conduction problems (bundle branches blocks – BBB) may not influence haemodynamic parameters in a way leading to a clinical presentation. Some of them can be accidental findings on the ECG record taken for other reasons. 24-hour ECG recording – Holter monitoring reveals that in some subjects, normal physiological record is suddenly interrupted by several QRS complexes showing the bundle branches blocks morphologies. This type of bundle branch blocks is called **occasional BBB**. This is in the contrast with the **persistent BBB**, which is present on every single QRS complex. Persistent BBB are very likely caused by the pathological process which is not dynamic, there was a pathological process, but it's already gone, terminated (e.g. scar tissue after the MI, or disseminated intramyocardial fibrosis). Occasional BBB are very likely caused by dynamic processes – for example hypertrophic ventricle may have enough energy in the form of ATP in the rest condition, but during the tachycardia the consumption of energy is higher, and the lack

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of ATP may lead to the slowing or complete block of conduction. During this period the block will be present on the ECG and it will be gone after the circumstances provoking it will terminate.

If the block was caused by a pathological process which was successfully treated (myocarditis for example), it was present on the ECG and now disappeared, it was so-called **temporary BBB**.

2. Complete vs incomplete blocks

According to the hallmarks present on the ECG record it is possible to recognize **complete bundle branch block** (total block) and **incomplete bundle branch block** (partial block). The typical findings in BBB are **deformations - abnormal configurations of the QRS complexes** which are caused by the abnormal pattern of ventricular depolarization, because in case of the block of one of the bundle branches, the ventricle is not depolarized by “conductive system” but the ventricle with blocked bundle branch will depolarize with a delay and the source of the depolarization are the electrical currents from the other ventricle. The delays and the time necessary for depolarization of the ventricle with the blocked conductive system will cause changes in the duration of QRS complexes. They will be prolonged to 0.12 s in precordial leads. Abnormal QRS complex with duration 0.12 s and less is considered to be a partial BBB. The term partial bundle branch blocks describes the conduction through the bundle branch which is only slower than normally. Abnormal QRS complex with the duration more than 0.12 s is considered to be complete BBB – the conduction via this pathway is blocked (not possible).

Causes leading to disturbances of ventricular conduction are many, some of them are pathological processes, while some of them are considered physiological.

a/ **myocardial hypertrophy in athletes** – it is compensatory mechanism to increased haemodynamic strain in athletes (only this one is physiological process), **myocardial hypertrophy caused by pathological processes** leading to either increased preload or afterload (aortic stenosis, regurgitation, hypertension for left ventricle and pulmonary hypertension, pulmonary valve stenosis, COPD for right ventricle) – increased mass of muscle requires longer time to get depolarized. There is no electrophysiological problem with the conduction, it only takes longer. However, tachycardia in hypertrophic heart may lead to the ischemia affecting mainly subendocardial parts, therefore the block may be “occasional” related to tachycardia or the periods of increased oxygen consumption.

b/ **congenital heart diseases** affecting the septum or valves, leading to malformations of AV junction or septum itself.

c/ **ischemic heart disease** – the tissue scar after the MI located in the anteroseptal and also diaphragmatic location may lead to the ventricular conduction problems. They will be persistent types of blocks because the scar tissue represents the end point of the process and the recovery is not possible. Acute MI can also lead to the BBB. Subjects with angina pectoris usually have disseminated intramyocardial fibrosis and this process may lead to the partial blocks or occasional blocks which will appear in periods characterized by increased consumption of oxygen, otherwise they would not be present on the ECG recording.

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d/ **inflammation, amyloidosis** – inflammatory infiltration of the myocardial interstitial and deposition of amyloid also influence from cell to cell conduction in the heart.

e/ **dilatation and the extreme haemodynamic strain of the heart** – these processes do not influence ventricular conductive system, but both circumstance increase ATP consumption in the myocardium, therefore the lack of ATP may lead to the slower cell-to-cell conduction in the ventricles. This may mimic the BBB figures – mainly prolonged QRS complexes and abnormal configurations of QRS in the chest leads. Finally, BBB may appear on the ECG of otherwise healthy individuals and this finding has no reasonable explanation.

Right bundle branch block (RBBB)

Most common causes of RBBB are hypertrophy of the right ventricle (cor pulmonale), ischemic heart disease and degenerative changes of the myocardium. In right bundle branch block, the depolarization of the ventricular system starts normally, because it is normally initiated by the left bundle branch. The first phase of the ventricular depolarization – depolarization of the septum leads to presence of vector oriented from the left to the right side, therefore V_{1-2} record positive **r** deflection and V_{5-6} record **small negative q**. The depolarization of the right ventricle is not happening, because the RBBB is present, however the left ventricle depolarizes normally. The vector of this depolarization is oriented to the left side. V_{1-2} will record **S** (negative deflection), while the V_{5-6} will record positive deflection with high amplitude caused by left ventricle depolarization – **R**.

The right ventricle must be depolarized as well, and it will happen by the local electrical currents spreading from the left ventricle – since these currents will be directed to the right side, the V_{1-2} will record positive deflection **R'** or **r'** summing up to the shape **RSR'** or **RSR'** (known as bunny ears) in the right precordial leads. The leads in the left precordium V_{5-6} will record a current heading from the left ventricle to the right side, therefore they will record **negative and wide S deflection**. Overall activation of the ventricular system lasts more than 0.12 sec in complete block and 0.12 sec or less in incomplete RBBB. **Typical hallmarks of RBBB on ECG are:**

- **prolonged duration of QRS complex > 0.12 sec**
- **V_{1-2} rSR' or RSR' (the bunny ears)**
- **V_{5-6} abnormal deep and wide S**

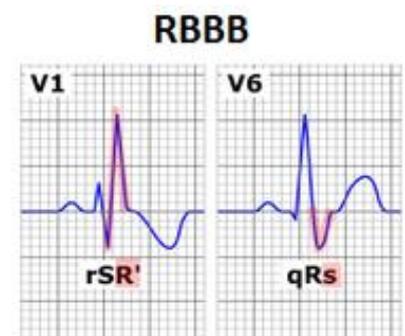


Fig. 56: Right bundle branch block (Yanowitz FG, 2007)

Left bundle branch block (LBBB)

In case of total LBBB the impulse produced in the SA node depolarizes the atria, AV zone, but it cannot continue along the left bundle branch to the septum and left ventricle. Conduction in the right bundle branch is intact. Depolarization continues from the AV zone along the right bundle branch to the right ventricle, and septum does not depolarize as a first – this is a reason why the leads V₁₋₂ will miss small r and leads V₅₋₆ will miss small q (septal depolarization). Impulse continues in the right bundle branch towards the heart apex, and it also depolarizes the right ventricle via the Purkinje fibres. It also slowly moves to the left ventricle but not via normal conductive system, but the left ventricle is depolarized by the local electrical currents produced in the right ventricle. The impulse is conducted slowly from cell to cell. Obviously the depolarization of the both ventricles is not synchronized. Depolarization of the apex and the right ventricle could be partially synchronized, but the depolarization vectors of the septum and right ventricle are inverse (septum is now depolarized **from the right side to the left!** from the right bundle branch and the right ventricular vector is oriented to the right) and equivalent when it comes to the amplitude. The final vector of this stage of depolarization can be zero (not causing a deflection) as result of these temporal and spatial relationships between the septal depolarization and right ventricle depolarization.

Depolarization of the left ventricle continues producing strong vector oriented to the left side, upwards anticlockwise and this vector is not interrupted by the vectors of the right ventricle or the septum, because as we said before, these two (septum and RV gives together a zero potential). Depolarization of the left ventricle is slow, delayed and it lead to the recording of wide abnormal QRS complex. In leads V₁₋₂ it looks like a W (broad deep S, deformed), but it represents a vector of the left ventricle going away from V₁₋₂. On the contrary, V₅₋₆ are recording deformed wide R (M-like) because they are recording the vector directed towards them. Standard lead I records R with split peak. Duration of ventricular complexes is longer than 0.12 sec.

Causes of the LBBB are ischemic heart disease, inflammation and congenital heart diseases. Hypertrophy or dilatation can also manifest as a LBBB.

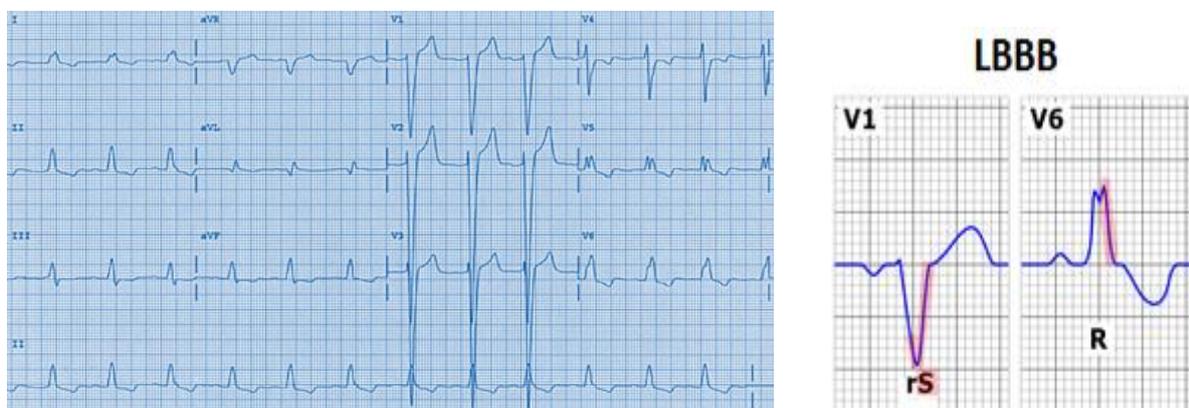


Fig 57. Left bundle branch block (Yanowitz FG, 2007)

6.3. Fascicular blocks

Anatomical, histopathological and finally electrophysiological studies proved that the left bundle branch splits into anterior and posterior fasciculus. These tiny parts can be affected by pathological processes separately leading to so-called hemiblocks – left anterior and left posterior hemiblock. They are commonly associated with the RBBB.

6.3.1. Left anterior hemiblock

Isolated damage of the left anterior fascicle manifests on the ECG recording as “dominance” of the left ventricle. There is considerable rotation of electrical axis to the left side to more than -45° and mild prolongation of the QRS. Causes of this hemiblock are anterior MI, aortic stenosis and cardiomyopathies.

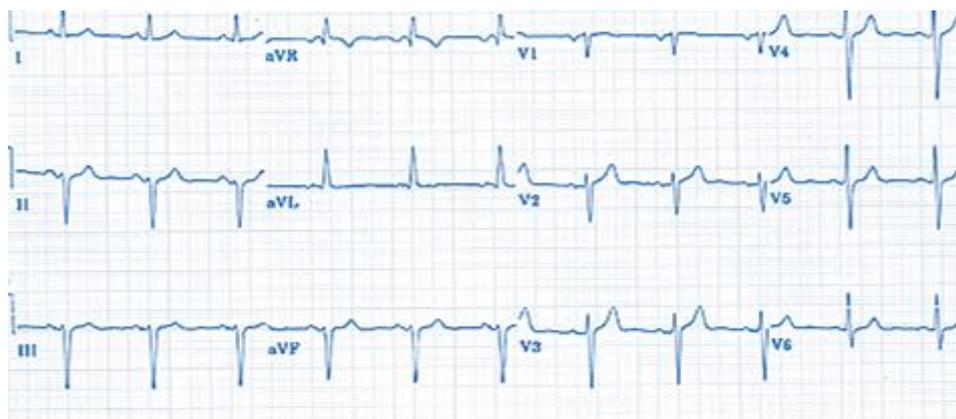


Fig. 58. Left anterior hemiblock with severe left axis deviation (Yanowitz FG, 2007)

6.3.2. Left posterior hemiblock

This disturbance manifest as a “dominance” of the right ventricle with the right or extreme right deviation of electrical axis which of course can be found in patients with the right ventricle hypertrophy or in the subjects with extreme vertical position of the heart. Posterior hemiblock is however less common than anterior hemiblock and the causes of different sensitivity to the pathological processes are:

- left posterior fasciculus has blood supply from two coronary arteries, therefore it is more resistant to ischemia
- left posterior fascicle is bold what makes is also more resistant to damage
- it is located in haemodynamically less strained part of the ventricle
- it is the first tiny branch of the bundle of His

While the anterior fasciculus is supplied only from one coronary artery and it is located in the part of ventricle characterized by high haemodynamic strain.

Electrocardiography

ECG manifestation of hemiblocks frequently combines with the RBBB. Combination of RBBB with anterior hemi block is one of the most common types of the disturbances of ventricular conduction. In this case, the right precordial leads record typical features of RBBB with the deviation of the heart electrical axis to left side to -60° , even more.



Fig. 59: RBBB in combination with the left anterior hemiblock (Yanowitz FG, 2007)

Combination of the RBBB and left posterior hemiblock leads to the deviation of electrical axis to extreme right position, while the precordial leads are recording typical BRRR pattern (bunny ears). Clinical significance of the bifascicular blocks is high, because they indicate serious and quite extensive damage of the ventricular conductive system and importantly, they may eventually become trifascicular blocks (total blocks of the ventricular conductive system).

6.0 MYOCARDIAL ISCHEMIA AND ITS ECG MANIFESTATION

J Hanacek

This section does not explain the causes and mechanisms leading to the myocardial ischemia, but rather consequences of ischemia on electrophysiological features of the cells, electrical processes in the heart and their manifestation on the ECG recording. It is also important to note that ECG is a key examination method used to establish a diagnosis of myocardial ischemia and since these situations are life-threatening (myocardial infarction) it is of high importance for every medical student to be able to identify the main changes of the ECG induced by ischemic damage of the myocardial cells. We all know that in these circumstances, the time and quick decisions matter.

Myocardial ischemia causes changes of the two main functions of the heart – electrical and mechanical. These changes are further characterized by considerable dynamics when it comes to time, place and intensity. The main pathological process leading to the changes of electrical and mechanical functions is reduced oxygen delivery to myocardium and the lack of oxygen leads to the complex metabolic & adaptation processes resulting into:

- **changes of intra and extra cellular homeostasis of ions (K^+ , Ca^{2+} , Na^+ , Mg^{2+})**
- **accumulation of the anaerobic metabolic end products, mainly lactic acid and adenosine**
- **reduced production of energy (ATP and creatinphosphate)**
- **increased production of reactive oxygen species**
- **activation of local autonomic reflexes (stimulation or inhibition of vagal and sympathetic nerve fibres in the myocardium)**
- **increased release of neurotransmitters (adenosine, catecholamine, acetylcholine)**

All of the aforementioned changes have negative effect on the membranes of heart cells, both the muscle and conduction system. The main changes are

- reduction of the resting membrane potential
- onset of foci with abnormal automacy
- changes of the refractory phase
- slow conduction speed, cell to cell uncoupling, conduction of the impulse with the decrement
- onset of abnormal electrical currents between the ischemic zone and the healthy myocardium surrounding the affected area

These electrophysiological changes culminate into the changes of excitability and conductivity of the cells are represent so called arrhythmogenic substrate and a trigger leading to arrhythmias based on the re-entry and abnormal automacy mechanisms.

Complex of the changes we described above can have **different intensity and spatiotemporal dynamics** because the **ischemic focus is heterogenic – there are more areas affected with different severity of**

Electrocardiography

ischemia, therefore the cells may have distinct electrophysiological properties. These factors lead to quite typical time sequence of the changes detectable on the ECG, their character and intensity. Intensity and time course of the changes in the focus with complete ischemia – anoxia (no flow) will be different than the changes in the focus with some perfusion preserved– hypoxia (low flow). In the first case, there is a complete lack of oxygen and quite fast accumulation of anaerobic end products. The zone with some perfusion also accumulates anaerobic products, but their gain is slower and may exceed the concentration anaerobic products in the no flow zone. The reason is that fast gain of lactate and other molecules inhibits the enzymes participating in the anaerobic glycolysis process. In case of slow accumulation of these products, the enzymes are not inhibited and this is the reason why concentration of anaerobic metabolites in the low flow ischemia zone is even higher. This explains more severe electrophysiological abnormalities in the low flow ischemia than in the no flow ischemia. These changes are present mainly in the border zone between low flow and no flow zones.

Mechanisms and consequences of the main electrophysiological changes caused by myocardial ischemia

a) decrease RMP of ischemic cells – the potential decreases towards zero from -90mV to -70mV, -50mV...) the causes are accumulation of K^+ in extracellular compartment, anoxia, decrease of extra and intracellular pH and accumulation of lysophosphoglycerides. Decreased RMP in these cells and normal value of RMP in intact cells lead to the onset of so-called **wound potential and wound current** (explanations will follow soon).

b) decreased amplitude of the AP of ischemic cells – the main cause of this change is attenuated difference of electrical potential outside vs inside of the cell and this will lead to the slow Na^+ inward current during the cell depolarization (slower than normally). There is also a problem with repolarization (ATP dependent) and the cells have difficulties to achieve the potential -90mV. This worsens with increasing rates of depolarization/repolarizations cycles. With the increased heart rate the RMP will be progressively slower and also the amplitude of the AP will be smaller – these processes are rate dependent.

c) changes in the duration of AP – when the ischemia begins, **the AP gets shorter** (the reason is reduced potassium outward current via ATP dependent K^+ channel – ATP regulates its permeability). Later the gain of extracellular K^+ itself limits the outward K^+ current and the P begins to prolong. Change of the extracellular pH has an inverse effect and this limits progressive prolongation of AP (this would happen otherwise but the pH does not let it).

d) changes of the refractory period, excitability of ischemic cells and the onset of abnormal automacy – **refractory period** gets shorter at the beginning of the ischemia that correlates with the shortening of AP in this period (the cause is hypoxia and progressive gain of extracellular K^+). Later the refractory period prolongs - likewise the AP, the cause is more severe hypoxia and progressive gain of extracellular K^+ . Prolongation of the refractory period is, however, longer than the prolongation of AP. It is known that the cells of the myocardium outside the conductive system are not able to generate impulses, but the ischemic damage can make them to become pacemakers. This pathological automacy in the ischemic focus is initially inhibited by the gain of

Electrocardiography

extracellular K^+ concentration, but in the cells of the borderline zone we have limited gain of the K^+ ex and at the same time enhanced inward Ca^{2+} current. This may promote automacy. These cells typically have afterdepolarizations that may trigger ventricular dysrhythmias.

e) cell to cell uncoupling – is caused by the damage of the gap junctions due to reduced extracellular pH and by the increase of the Ca^{2+} in ischemic cells. This electrical uncoupling **seems to be a protective mechanism related to the protection of intact cells and their disconnection from the cells damaged by ischemia, anyway, it leads to the onset of ventricular arrhythmias.**

f) changes of the conductivity – ability of ischemic cells to conduct impulses decreases due to several factors. The first is reduced RMP (because of gain of extracellular K^+), and it also reduces the amplitude of the AP. It was already explained in the section about heart blocks that cells with reduced membrane potential are not able to produce strong enough local currents to boost the depolarization spreading. Slowing of the conduction is promoted by other mechanisms as extracellular pH drops and damage to the gap junctions. The result is either slow conduction or conduction with the decrement leading to the hart blocks.

Slowing of the conduction can be rate dependent which means that it gets worse with the increased heart rate. In the normal heart rate, eventually mild bradycardia there are no blocks on the recording, but tachycardia leads to impaired conduction, therefore partial and even complete blocks may occur on the ECG recording.

Complex metabolic processes in ischemic myocardium lead to the activation of local autonomic reflexes (activation of the nerve endings of the sympathetic or parasympathetic origin in the heart). This may also contribute to the electrical instability of the myocardium. It is important to mention some information about the distribution of the sympathetic and parasympathetic nerve endings in the heart. Vagus nerve endings are denser in the postero-inferior parts of the ventricles and in the subendocardial layer of the heart wall. Sympathetic nerve fibres are denser in the anterior wall of the heart ventricles and in the subepicardial layer of the wall. This unequal distribution has electrophysiological and also some other types of consequences. Ischemia localized mainly in the anterior wall of the heart activates mainly sympathetic nervous system at the local level (in the heart) but also at the systemic level leading to the sympathoadrenal response (acceleration of the heart rate and strength of the heart work plus peripheral vasoconstriction. Ischemia localized in the diaphragmatic wall (infero-posterior) leads to the inhibition of the sympathetic effects on the heart – this will manifest by bradycardia and vasodepressor effects. Ischemia of subendocardial layer will also manifest by bradycardia and hypotension and it is usually not accompanied by typical stenocardia (silent ischemia). Typical stenocardia is rather provoked by the ischemic lesion located in the anterior wall and in subepicardial layer.

6.1 ONSET AND PROGRESSION OF ISCHEMIA - MANIFESTATION ON THE ECG

Life threatening types of the ischemic heart disease (MI) have quite uniform presentation on the ECG recording, since they are particularly dangerous, students should be able to recognize them as soon as possible.

Electrocardiography

Ischemic focus – its size, location, intensity and the progress of changes have some typical features which are detectable on the ECG in case you know what to search for.

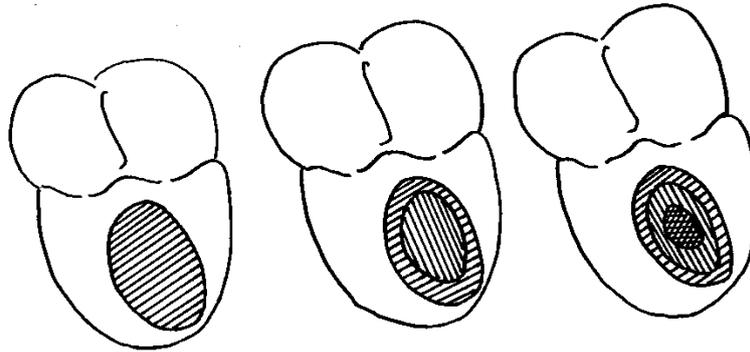


Fig 60. Ischemic focus is not homogenous mass of the muscle with the same severity of the damage, but it rather progresses with the time from one zone to fully stratified ischemic focus (explanation in the text).

After the blood flow in the coronary artery stops, all area supplied by this artery becomes ischemic – **“zone of ischemia”**. The periphery of this primary ischemic focus can be supplied by the collateral circulation or from the neighbourhood that may improve the oxygen supply in the periphery, so the ischemic zone gets smaller. In the very central parts of the zone, however, ischemia gets severe and causes metabolic and biochemical changes that were mentioned already – this zone is called **“zone of damage”**. If the blood flow is not restored either spontaneously or by treatment, the central part of the focus become necrotic – **“zone of necrosis”**. **The primary ischemic focus stratifies with time into zone of necrosis (centre), zone of damage around it, and finally zone of ischemia in the periphery of the focus. This sequence describes usual development of myocardial ischemia after the occlusion of the coronary artery which is a main cause of myocardial infarction.**

Described processes lead to the characteristic changes of the electrophysiological features of the cells, which can be detected with an ECG device. These changes are present in the depolarization and also repolarization parts of the curve. Based on the position of recording electrodes and the changes in those leads it is possible to identify the location of the ischemia. Some of the leads are placed directly “above” the ischemia (of course there is a chest wall in between) but those leads can “see “ and record the most typical changes. Other leads “look” at the ischemic focus from different positions and perspectives – therefore they will record electrical activity in the ischemic focus from their own angle and perspective. Some of the leads are not able to even detect those changes. Logically, ECG changes will be different from lead to lead and they will have different intensity. It is not random, there are some rules and principles which can help to identify the exact location of the ischemic focus.

Manifestation of ischemia on the ECG is variable, strongly influenced by time. The ischemic focus will influence the ECG in a different way at its very beginning, after several minutes, hours and weeks (fig. 56). **Ischemic changes start in the repolarization phase – particularly by change of the shape and amplitude of the**

Electrocardiography

T wave, later by the changes of the position of ST segment and finally ischemia influences also the depolarization part – QRS complex.

- **change of the T wave** - is caused by the activation of the potassium channels governing the flow of the K^+ out of the cells. This will lead to the acceleration of the repolarization and the manifestation of it will be **peaked T wave with the high amplitude**.
- **change of ST segment** – in the 12 lead ECG, some of the leads record elevation, while some of the lead record depression of ST segment. These changes are induced by the presence of the “wound current”, which runs between the intact cells and ischemic cells (the current flows from negative to positive electrodes in the real life, so does in the myocardium). The intracellular space of the non-ischemic cells is more positive (higher positive potential during transpolarization phase of AP) than in ischemic cells. They have much lower RMP. Wound current has the highest amplitude during considerably delayed depolarization of the ischemic myocardium. The final position of ST segment in particular leads depends on the direction of the current and position of the electrode. If the current “goes” to the electrode, it will record elevation (ST segment will move upwards), if the current goes away from the electrode, the electrode will record depression of the ST segment.
- **Pathological Q** – is the manifestation of necrosis. It is usually recorded in case of transmural necrosis. In this case – necrotic wall is not producing any electrical activity, therefore the electrodes above the focus “looks” into the ventricle and record so called “chamber” potential. We know that the depolarization spreads from subendocardium to subepicardium – therefore the chamber potential is negative (all impulses going away from the chamber electrode). Some books explain that the necrosis serves as a window and the electrode looks through the window to the chamber and records the potential inside of the chamber.

Progression of the ischemic focus does not stop here, if the patient survives the acute MI, the zone of the damage disappears (it turns either to the necrosis or it will be recovered by the treatment or spontaneous recanalization) – this will lead to the return of the ST segment back to its normal zero position. If circulation restores, even the ischemic zone disappears (normalisation of the T wave) and the final witness of the MI is the scar (persistent Q deflection) after the necrosis. Fibrotic tissue forming the scar does not generate nor conduct impulses.

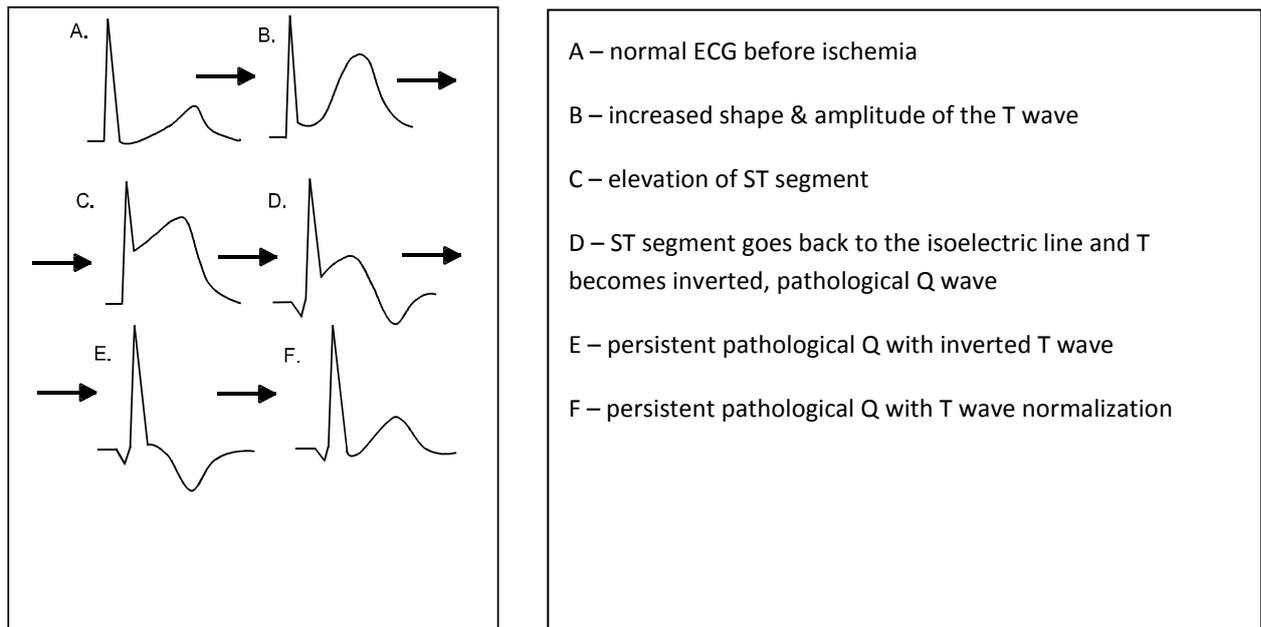


Fig 61. Diagram of the progression of the changes caused by acute myocardial ischemia in time

Typical changes caused by myocardial infarction does not appear on the ECG immediately after the onset of the clinical symptoms (stenocardia), but it takes some time until they are detectable. It is not possible to say exactly when do they appear on the trace – for example changes assigned as C –E – it is not possible to say when do they appear and for how long they will be present on the record, because their presence is influenced by many factors – intensity of ischemia, existence and function of collateral circulation, haemodynamic strain, oxygenation of the blood and many more. It is only possible to say that stages assigned as A and B appear immediately after the onset of ischemia (minutes, hours) and F – which appears after the formation of the scar or after complete reperfusion of previously ischemic myocardium.

ECG record helps also to localize the ischemic lesion and also the extension of it. It is important to identify whether ischemia is located on the anteroseptal part of the heart or diaphragmatic position. Anteroseptal infarction will manifest mainly in precordial leads V_{1-4} because they are close to it, and the diaphragmatic infarction will manifest in the leads II, III and aVF, because their position allows to see and record potentials from the infero-posterior (diaphragmatic wall) of the heart.

6.2 ANTERIOR WALL STEMI

Occurs in proximal occlusion of ramus interventricularis anterior (RIA), or trunk of left descending coronary artery (LAD). The different **infarct patterns** are named according to the leads with maximal ST elevation:

- Septal = V_{1-2}
- Anterior = V_{2-5}
- Anteroseptal = V_{1-4}
- Anterolateral = V_{3-6} , I, aVL
- Extensive anterior / anterolateral = V_{1-6} , I, aVL

Electrocardiography

In early anteroseptal STEMI (B-C), main changes are seen in the lead V_{1-3} - elevations of ST segments, the recording demonstrates the presence of the zones of ischemia and injury, zone of necrosis is not present. R in the leads V_{4-6} is not affected, does not have reduced amplitude and it still has normal shape - it indicates that the lateral part of the ventricle is not affected (Fig. 55a).

Fully developed anteroseptal STEMI will involve also a pathological Q deflection (QS) in the leads V_{1-2} - electrodes are located above the necrotic myocardium, elevation is still present (wound current still present) in V_{1-2} and also coronary T which is typical for chronic ischemia. (The Alan E. Lindsay ECG tutorial, 2007)



Fig 62 a and 62 b

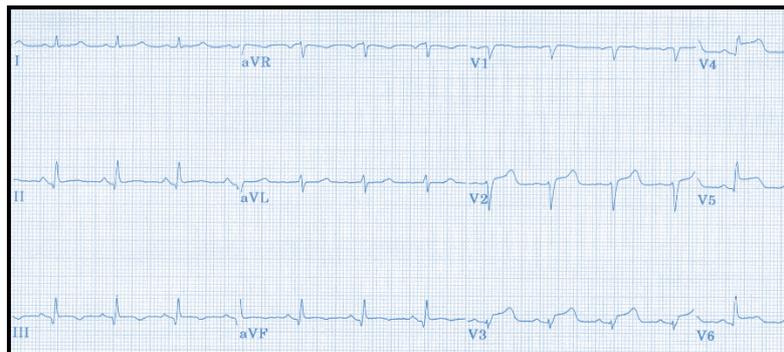


Fig. 62c Anterolateral STEMI (Yanowitz FG, 2007)

6.3 DIAPHRAGMATIC STEMI

Myocardial infarction caused by the occlusion of RAD affecting the postero-inferior, also called diaphragmatic wall does not lead to the typical changes in the chest leads (V_{1-6}). This is caused mainly by the fact that the chest wall is bit apart from the focus located on the diaphragmatic wall, that's also why the main changes are present in leads II, II and aVF – the trace here will be modified by the presence of elevations of ST segments and changed T waves in early acute MI, and later, if the necrosis will develop, these electrodes will also record pathological Q. Sometimes “opposite leads” aVL and V_{1-2} may record depressions of the ST

Electrocardiography

segments and the “mirror” changes caused by the wound current, which is directed towards lower leads (II, III, aVF – they “see” elevations and the mirror leads see depressions of ST segments).



Fig. 63: Acute DIA STEMI – peaked T wave and ST elevations in II, III and aVF, reciprocal depressions in I and aVL, also in V₁₋₃. Leads V₄ and V₅ are switched (Yanowitz FG, 2007).

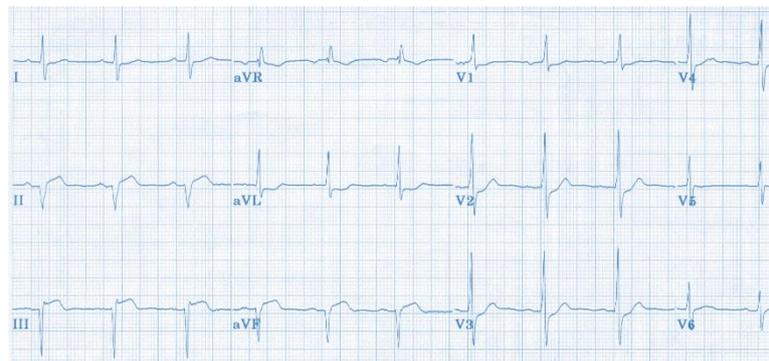


Fig. 64 b: Fully developed DIA STEMI – pathological Q in II, III, aVF, ST segment still bit elevated, high amplitude R in precordial leads as an inverse picture of deep Q in DIA leads (Yanowitz FG, 2007).

Leads detecting the major changes of the ECG curve may indicate exact location of the ischemia

1	MI of anterior wall	V ₁₋₆ , also I, aVL
2	MI anteroseptal	V ₁₋₄
3	MI anterolateral	V ₄₋₆
4	MI high lateral	I, aVL
5	MI diaphragmatic	II, III, aVF
6	MI diaphragmatic–lateral	I, III, aVF, V ₅₋₆
7	MI circular	V ₁₋₆ , II, III, aVF
8	MI posterior wall	V ₇₋₉
9	MI right ventricle	V ₁ , V _{3R} –V _{6R}

6.4 NON-STEMI ACUTE CORONARY SYNDROME

Acute coronary syndromes are caused by variety of processes affecting coronary circulation, while the STEMI is typically caused by the occlusive thrombosis of the coronary artery, unstable angina and non ST segment elevation MI are rather caused by dynamic type of the process inside the coronary circulation – non-occlusive thrombosis. Occlusive thrombosis leads to the transmural lesion of the myocardial wall, while in the NONSTEMI the ischemia affects only the part of it. (Fig. 65)

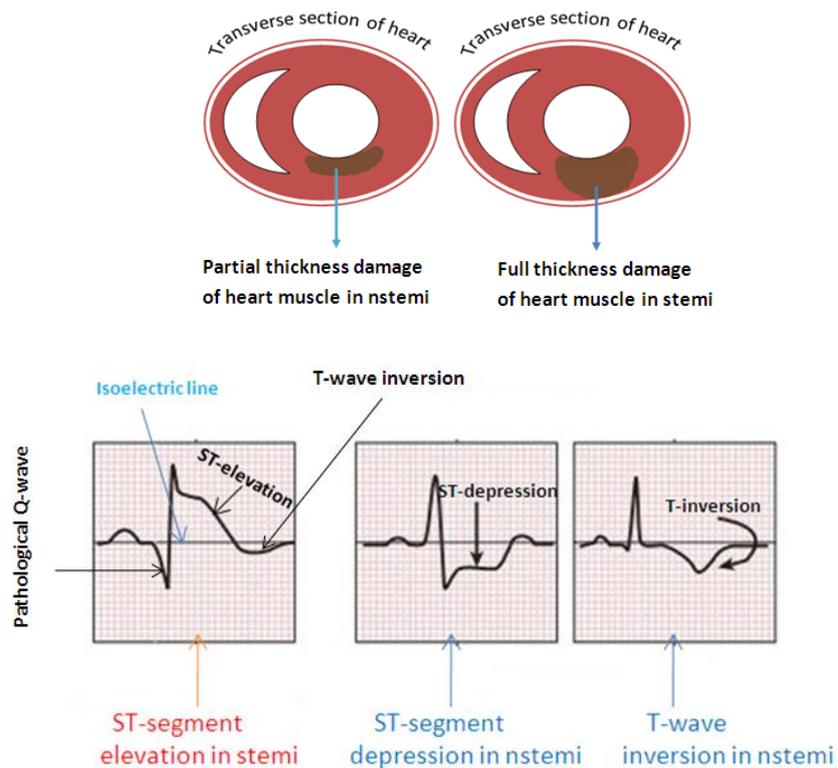


Fig. 65. Non-STEMI coronary syndrome

In this case, the clinical presentation is completely the same – stenocardia, restlessness, breathlessness etc. but the ECG shows no ST elevations. There is depression of ST segment or a transient elevation of ST segments with deep inverted T waves – Fig 65.

6.5 ANGINA PECTORIS

Angina pectoris represents the chronic form of ischemic heart disease which is characterized by the presence of ischemic pain (stenocardia) during the physical exercise or emotional stress – typically when the myocardial oxygen demands increase. It lasts less than 15 minutes and responds to either rest or

Electrocardiography

nitroglycerine. The ECG during the rest has no marks of ischemia, because there is no ischemia in fact. The ECG changes can be provoked by exercise ECG. The changes on the ECG correlate to the presence of the pain, and they are depressions of ST segments in leads V₄₋₆ – usually leads recording electrical activity over the left ventricle. Left ventricle is normally the biggest consumer of the oxygen and the lack of it appears there. Physical exercise is not only connected with the increased oxygen consumption, but also with tachycardia, which limits the diastole duration and as we know already, coronary arteries are considerably compressed by the contracting muscle.



Fig. 66. ECG recorded during the rest versus ECG taken during the exercise, the heart rate increases and the trace shows depressions of ST segments. <http://medvin2u.net/angina-pectoris-in-detail/>

6.6 PRINZMETAL ANGINA PECTORIS

Symptoms typically occur at rest, rather than on exertion (thus attacks usually occur at night). Two-thirds of patients have concurrent atherosclerosis of a major coronary artery, but this is often mild or not in proportion to the degree of symptoms. Prinzmetal's should be suspected by a cardiologist when the pain occurs at rest and/or in clusters, and in the absence of a positive treadmill stress test, as Prinzmetal's is exercise tolerant and can generally only be diagnosed after other forms of cardiac disease have been ruled out. It is associated with specific ECG changes (elevation rather than depression of the ST segments). The mechanism that causes such intense vasospasm, as to cause a clinically significant narrowing of the coronary arteries is so far unknown.

Electrocardiography

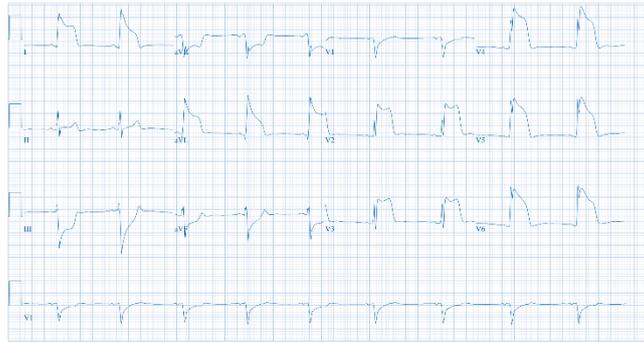


Fig. 67. ST segments elevations in subjects with Prinzmetal angina pectoris
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2755812/>

7.0 ECG CHANGES CAUSED BY THE ELECTROLYTE DISTURBANCES

J. Plevková

Potassium is the main intracellular cation and the ratio between intracellular and extracellular potassium concentrations (K^+_{ICT}/K^+_{ECT}) determines the resting membrane potential in excitable tissues. The movement of potassium to the cells and maintenance of high gradient of concentrations depend of the activity of Na^+/K^+ ATP-ase. Disturbances of potassium homeostasis and its distribution can be caused by many pathological processes and from the quantitative point, there are two main situations - hypo or hyperkalaemia in the extracellular compartment.

Hyperkalaemia is defined as a concentration of K^+ in ECF above 5 mmol/l, clinically significant hyperkalaemia is present above 6 mmol/l and directly life-threatening concentration is above 7 mmol/l. These changes manifest on ECG trace. Causes of hyperkalaemia are renal failure, acidosis, crush syndrome or adrenal insufficiency, of course it may appear as iatrogenic hyperkalaemia in case of inappropriate substitution of K^+ (by parenteral treatment). In hyperkalaemia, all chest leads record high amplitude and peaked T waves, negative T wave present in children in chest leads becomes positive in hyperkalaemia. High concentrations of potassium reduce the RMP, limit the amplitude of AP, its duration and impair conduction of depolarization in the heart. P wave becomes flat, PQ interval becomes extremely prolonged and QRS complex is deformed. Sometimes the fusion of the QRS and T wave is visible (Fig. 58). Very high concentration of potassium causes cardiac arrest in diastole. In distribution hyperkalaemia (acidosis, catabolism or hyperglycaemia), the effect of potassium on the heart may not be that severe.

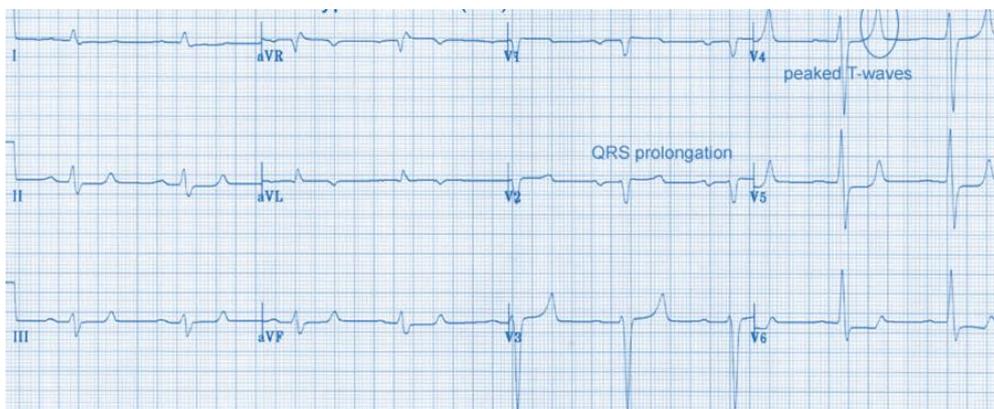
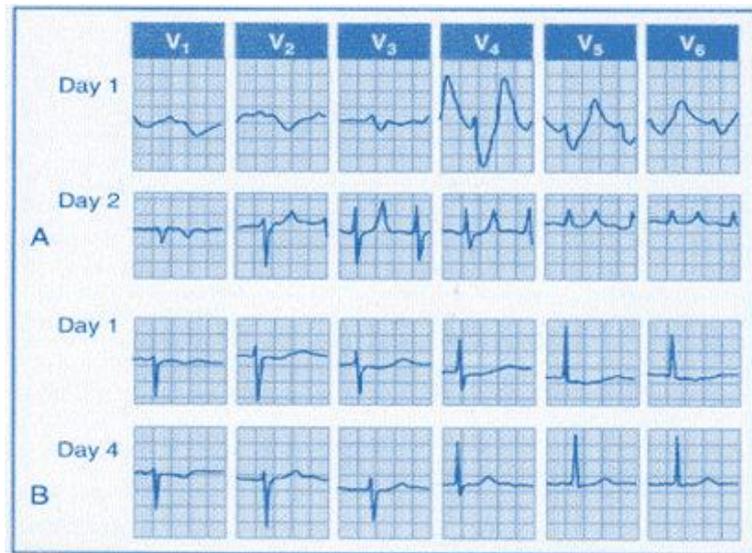


Fig. 68 Hyperkalaemia on the ECG trace

Hypokalaemia (inappropriate doses of some diuretics, hyperaldosteronism, hepatic failure, quick correction of acidosis) does not influence the ECG that much. Changes occur when the potassium concentration in ECF falls below 3.0 mmol/l. The changes in repolarization phase occur, the ST segment

Electrocardiography

decreases below the isoelectric line and the amplitude of the T wave decreases as well and sometimes it may fall into the negative amplitudes. RT interval (overall electrical activity of ventricles) is prolonged and the U wave shows up. The fusion of T and U waves may cause the difficulties in the measurement of the QT interval, which is as you know already, a risk factor for onset of abnormal ventricular activity – Fig. 69.



Calcium is mainly distributed in the extracellular fluid, the cells have quite low concentration of it. Calcium is present in the ECF in two main forms – free calcium and calcium in bond to plasmatic proteins. The free calcium occurs in a form of complexes with citrate, phosphate or bicarbonate, however clinically very significant is the ionized form of free calcium. Disturbances of the calcium homeostasis can influence the electrophysiological processes in the myocardium, what of course can be recorded on the electrocardiogram.

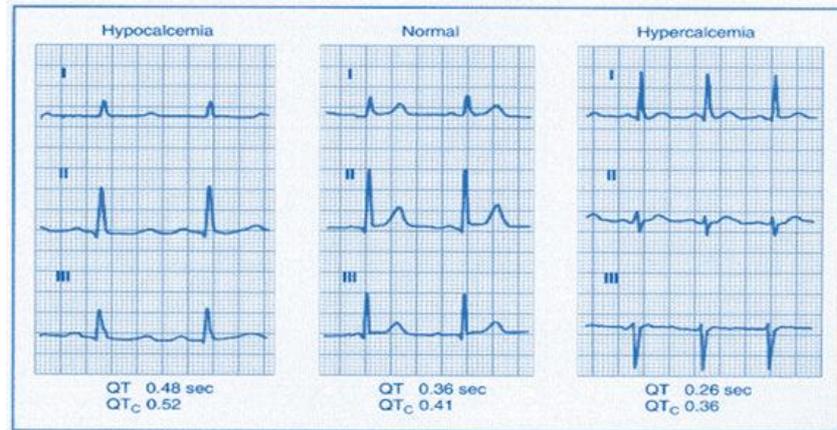
Calcium ions play a key role in the chain of excitation-contraction processes in the heart. Depolarization of the cell membrane irradiates to the cell via the T tubuli towards sarcoplasmic reticulum. During the depolarization, calcium ions are immediately released from the reticulum, diffuse to the cytoplasm where they interact with the regulatory proteins troponin and tropomyosin. Another source of Ca^{2+} is their transport across the membrane of the T tubuli. The heart contraction would be considerable weaker without the extracellular Ca resources. Concentration of Ca^{2+} influences duration of the plateau phase of the action potential and they contribute to the optimal conduction of the impulse.

Hypercalcaemia > 2.5 mmol/l and severe hypercalcaemia > 3.5 mmol/l are caused by hyperparathyroidism, paraneoplastic processes, bone tumours and metastases. Increased concentration of ionized calcium increases muscle contractility but decreases neuro-muscular irritability. Increased inotropic state of the myocardium is frequently associated with PVC, short QT interval, short ST segment and T wave sometimes begins just after the R terminates.

Decreased concentration of ionized calcium in extracellular fluid below 2.25 mmol/l, eventually severe hypocalcaemia < 1.6 mmol/l leads to the prolonged ST and QT interval. T wave is usually peaked and has high

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amplitude, contractility of the heart decreases. Hypocalcaemia occurs in disturbances of Ca/phosphate balance (parathormone), malabsorption, renal failure, pancreatitis, disturbances of the bone mass regulation and some other pathological processes – Fig. 70.



8.0 HYPERTROPHY OF THE HEART CHAMBERS ON ECG

The response of the heart to increased haemodynamic load is a hypertrophy. This process represents a type of adaptation of the heart to increased work load and it also represent a compensatory mechanism. Its main function is to maintain optimal cardiac output that matches with tissue oxygen demands in case of increased preload or afterload.

It is believed that the direct stimulus which initiates hypertrophy is increased tension in the myocardial wall which leads to extremely increased consumption of ATP in the myocardial wall. Hypertrophy changes the ratio between the radius of the heart chambers and the thickness of the wall and it returns the tension in the wall to the previous values and optimizes consumption of ATP. The heart reacts by **eccentric type of hypertrophy to the volume overload** (processes with increased preload e.g. valve regurgitations) and by **concentric type of hypertrophy to the pressure work** (processes with increased afterload – e.g. aortic stenosis, systemic hypertension). Some students mix hypertrophy with dilatation - these terms describe different processes.

Hypertrophy (myocardial wall thickening) – is defines as long lasting structural remodelling based adaptation of the heart to the extra volume or pressure load. The number of cardiomyocytes does not increase; it is only an enlargement of already existing cardiomyocytes (they do not multiply).

Dilatation of the heart represent enlargement of the heart during which the wall of the heart is absolutely or relatively thinner than the enlargement of the radius of the heart chambers. There can be a primary dilation – when normal not hypertrophic heart suddenly dilates as a response to the acute haemodynamic strain or it can be secondary, when formerly hypertrophic heart dilates as an end stage of chronic cardiovascular diseases.

Hypertrophy of the atria and ventricles have an impact on the ECG curve:

- Enlargement of the muscle in the haemodynamically overloaded parts of the heart leads to the change of anatomical position of the heart – there are real anatomical changes
- Hypertrophic mass of the atrial or ventricular musculature represents much stronger source of the electrical potential which are influencing position of “electrical axis of the heart”
- Overall electrical activity of the hypertrophic walls takes longer time

All of these facts modulate the final ECG.

8.1. HYPERTROPHY AND OVERLOAD OF ATRIA

Depolarization of the atria is represented by the P wave. **Hypertrophy and overload of atria therefore modify the shape, duration and amplitude of P wave.**

Hypertrophy of the right atrium causes shift of the atrial depolarization vector to the right side and more to the vertical position, while normal position of atrial vector follows a virtual connection between the SA node and the AV node. ECG curve typically records high amplitude of the P wave and the P wave is also peaked

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and symmetric in the leads II, III, aVF and in precordial leads V_{1-2} , which are the closest recording electrodes. Sometimes the chest leads V_{1-2} record biphasic P wave. Since the most common causes leading to hypertrophy of the right atrium are pathological processes in the lungs and lung circulation (COPD, pulmonary hypertension) the P was given a name **P pulmonale**.

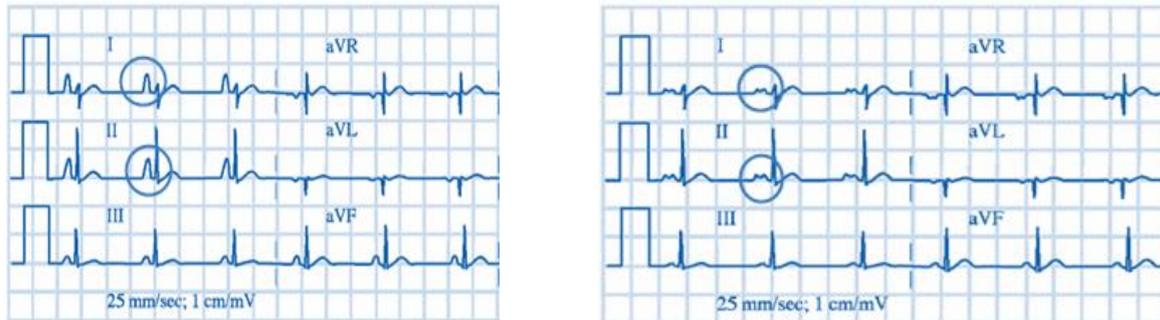
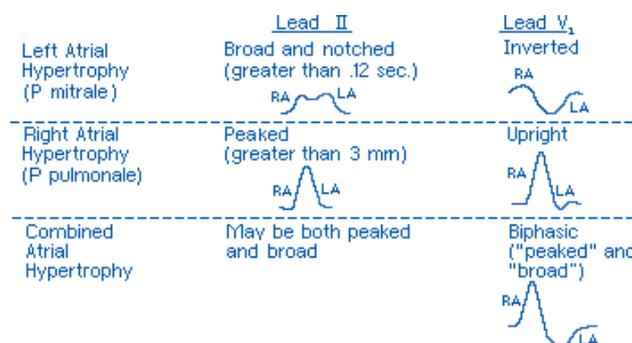


Fig. 71: Hypertrophy of the right atrium “P pulmonale “ and hypertrophy of the left atrium P mitrale (Yanowitz FG, 2007)

P wave recorded in case of the left atrium hypertrophy is called **P mitrale**, because the most common causes leading to the left atrium hypertrophy are diseases of the mitral valve (mitral stenosis, mitral regurgitation). Vector of the atrial depolarization rotates to the left side, to the horizontal position and the duration of the P wave is typically prolonged to more than 0.11 sec. **P mitrale usually have two peaks** – the second one with higher amplitude represents depolarization of the thicker wall in the left atrium. Chest leads V_{1-2} may eventually record biphasic P wave. Hypertrophy of both atria may lead to the presence of so-called P cardiale – peaked, broad P wave, eventually and biphasic in the precordial leads V_{1-2} . – Fig. 72



8.2. HYPERTROPHY AND OVERLOAD OF VENTRICLES

Hypertrophy of the right ventricle

Hypertrophy of the right ventricle changes position of the electrical axis in the frontal plane in the standard leads I, II, III - the axis rotates to the right side (right lower quadrant) and T waves can be discordant. More typical features of the hypertrophy are seen in the chest leads – mainly in the leads from the right precordium V_{1-2} (V_3). While the QRS complexes are typically negative in the right precordium (rS shape),

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hypertrophy of right ventricle produces positive R deflections with high amplitude. They represent a potential of hypertrophic right ventricle which produces much stronger electrical potentials than the left one. QRS complex is typically shaped into qR and T wave is negative. Presence of such QRS complexes (positive in the right precordial leads move transitional zone to the left side. Electrodes recoding in the left precordium “see” deep and broad S deflection which confirms the dominance of the right ventricle (presence of the vectors oriented “away” from V_{5-6}). ECG criteria of right ventricular hypertrophy:

1. V_1 and aVR – **high R wave**, or $R > S$, or in V_1 $R > S$ than 2 mm
2. $R(V_1) + S(V_5) = 10,5$ mm
3. Axis – **right deviation**
4. In leads II, III, aVF **depression of ST segment** and **negative T waves**
5. **Transitory zone: left shift**
6. In leads II, III, aVF the P wave $> 2,5$ mm
7. ECG manifestation of **incomplete RBBB**



Fig. 73: Right ventricle hypertrophy – the right precordial leads V_{1-2} record qR, and the left precordial leads V_{5-6} record deep and broad S, hallmarks of incomplete RBBB with rotation of electrical axis of the heart to the right.

Hypertrophy of left ventricle

Standard leads do not show typical changes that would support the diagnosis of LV hypertrophy, however, rotation of the axis to the left side (horizontal and even more to the left) and broad QRS complexes with the borderline duration 0.11 sec. Precordial leads record the changes of the QRS shapes, as well as changes of the amplitudes of the particular deflections caused by the electrical potential produced by the huge musculature.

Right precordium leads V_{1-2} record small r with very deep and broad S (rS), borderline duration of QRS, slight elevations of ST segments. Leads in the left precordium record very high R deflection and commonly depressions of ST segment. In case of the overload, the repolarization changes become more visible, mainly the depression of ST and T inversion in the left precordium. Remember, the left ventricle is the strongest part of the heart and it has the highest oxygen consumption. The leads V_{5-6} are witnessing the relative lack of oxygen provoked e.g. by tachycardia or increased preload or afterload – this relative lack of oxygen will lead to the

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relative lack of ATP and slight hypoxia in the deep layers of left ventricle – that's why those leads detect depression of ST with inverted T wave. Relative depletion of the ATP causing changes of the ST segment in the hypertrophic myocardium are:

- increased tension in the hypertrophic ventricular wall leads to increased consumption of ATP
- huge musculature and its increased tension compress coronary arteries not only during systole, but also during diastole (this is normally not happening in nonhypertrophic myocardium) and it limits coronary blood supply
- myocardial cells are hypertrophic while the capillary network is not – this leads to the mismatch between the oxygen supply and oxygen demand by huge musculature

Evaluation of left ventricle enlargement needs analysis of more criteria. e.g. rotation of electrical axis to the left side is not absolute indicator of LV hypertrophy, but in combination with hallmarks of LV overload (depression of ST, T inversion) makes the diagnosis of LV enlargement more probable.

Although we defined some ECG criteria for LV enlargement, finally clinical diagnosis is made after the heart ultrasound which makes it possible to exactly assure the thickness of the walls, septum, proper work of valves and pressure gradients in the heart. Anyway, it is useful to know how to apply e.g. Sokolow index to detect possible LV enlargement.

SOKOLOW INDEX (ECG)

1. $S(V_1) + R(V_5/V_6) \geq 35$ mm or $S(V_1/V_2) + R(V_6) \geq 40$ mm
2. Sum of highest R a S waves in precordial leads ≥ 45 mm
3. Axis in frontal plane – left deviation (0° or less)
4. Depression of ST segment and negative T wave in V_5 , V_6 and I
5. $T(V_1) > T(V_6)$

Some pathological processes may lead to false positive diagnosis of LV enlargement – obesity, emphysema, fever, because they lead to the change of the anatomical position of the heart (obesity) or eventually to increased workload for LV (fever).

Just for demonstration, we wanted to show some other indexes and criteria which can be used to prove ECG findings typical for LV hypertrophy.

- Sokolow + Lyon (*Am Heart J*, 1949;37:161)
 - $S V_1 + R V_5$ or $V_6 > 35$ mm
- Cornell criteria (*Circulation*, 1987;3: 565-72)
 - $SV_3 + R aVL > 28$ mm in males
 - $SV_3 + R aVL > 20$ mm in females
- Framingham criteria (*Circulation*, 1990; 81:815-820)
 - $R aVL > 11$ mm, $R V_{4-6} > 25$ mm
 - $R V_5$ or $V_6 > 35$ mm, $R I + S III > 25$ mm

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Fig. 74. Hypertrophy of LV, high amplitudes of the S in V_{1-3} and high R in V_{5-6} with the T wave inversions in the left precordial leads, LAD