

In congestive cardiomyopathy it is possible to notice an abnormal conduction in the ventricles which leads to some abnormalities in the QRS complex. We can notice the presence of some nonspecific ST segment and T wave changes as seen in other cardiomyopathies. In restrictive cardiomyopathies we can notice a low voltage QRS complex and disappearance of progression in the precordium.

3.25.6.8 Electrocardiographic changes in metabolic and electrolyte disorders

The cardiac tissue is electrically active, and that is why the electrolyte changes can affect its electrical activity.

In hyperkalaemia the appearance of tall T waves and a prolonged ventricular complex which becomes mated with the T wave to form one peak. P wave is small and PR interval is prolonged.

In hypokalaemia T waves become flattened or inverted. Whereas the U wave becomes very clear and the QT interval is markedly prolonged. Its prolongation is partly due to the U wave which is mated into the T wave. These signs signalize the possible occurrence of arrhythmias especially when digitalis or antiarrhythmic drugs (group 1) are applied. The ECG changes in hypokalaemia are similar to those of hypocalcaemia. **Hypocalcaemia** prolongs the ST interval as well as the T wave, this will lead to QT interval prolongation. Hypocalcaemia on the other side is not as dangerous as hypokalaemia in generating cardiac arrhythmias. Non specific ECG changes occur in hyperthyroidism, hypothyroidism, diabetic ketoacidosis, and of course after the use of antiarrhythmic drugs. The prolongation of PR and QT interval as well as some non specific changes in ST segment and T wave are the most commonly seen.

3.26 The electrophysiological basis in the generation of cardiac arrhythmias

The term arrhythmia constitutes all changes of the cardiac rhythm that render the rhythm different from the normal sinus rhythm. The sinus rhythm is initiated in the SA node. The activation is progressing then from the atria to the ventricles having a normal PR interval that doesn't exceed 0,20s and the heart rate in adults ranges between 60–100/min.

The development of electrocardiology and especially the intracardial electrophysiological study and the Holter monitoring could influence many opinions about cardiac arrhythmias. Many findings were used to explain changes in the conventional electrocardiography.

From the histological and functional point of view we can differentiate two types of cardiac cells: contractile cells, which duty is to provide the pumping function of the heart and specialized cells for the formation and conduction of activation. The contractile cells have the ability to perform mechanical works and conduct electrical impulses from cell to cell.

The myocardial cells are connected to one another by intercalated disks by which they form interconnected muscle fibers. The intercalated disks serve the conduction of mechanical energy, yet they have electrical characteristics which render the activation conduction from cell to cell much easier.

The intercalated disks are formed by union of plasma membranes of the neighboring cells. It is actually a doubled plasma membrane. The junction is step, shaped with the alternation of areas oriented vertically and horizontally to the disk plane. This leads to an increment of the total surface area of the membranous contact. There are special junction structures in the intercalated disks being: The fascia adherens, macula adherens (desmosom), and a nexus (gap). Fascia adherens is the most common structure which form both plasmatic membranes which are wavy and inserted to each other. They run in a parallel manner and are separated by a 20–30 nm wide slot. Fascia adherens on the other side is the place where myofillaments are inserted. The

desmosom or macula adherens is a structure having 0,2–0,5 micrometer diameter in which the neighboring plasma membranes are situated parallel to each other. Nexus or the gap is a special structure which is found in the longitudinal segments of the intercalated discs. The electrochemical impulse spreads through the gap from cell to cell. It is composed of tight junctions of the neighboring plasma membranes. The membrane is structurally different in these areas. The gaps contain small channels 2–2,5 nm in diameter through which small ions can diffuse from cell to cell. Gaps and desmosomes do present even in the lateral surfaces of the neighboring cells.

According to other electrophysiological characteristics we can differentiate what is known as **fast and slow cells**. The fast cells are characterized by a large diameter and high speed of conduction. Their resting potential is -90 mV. The ascending edge of the action potential is very steep. On the action potential curve we can see the overshoot to positive values and the plateau is very clear as well. Purkinje cells and functioning cells of the myocardium have the characteristics of the fast cells. The resting potential of the slow cells is between -50 and -70 mV. Even the threshold potential is low (-30 till -40 mV). The stimulation of slow cells starts endogenously by increasing the membrane potential (from -70 to -40 mV) to reach the threshold level. The ascending part of the action potential curve is not steep and there is no overshooting. There is no plateau on the action potential curve. These are the characteristics of both SA node cells and AV node cells.

From the functional point of view we can find two types of cells, the **myocardial functioning cells** and **cells of the cardiac conductive system**. The main function of the conductive system of the heart is the generation of impulses and their conduction from the SA node where they are usually generated to the atrial and ventricular myocardium. According to the ultrastructure we can differentiate three types of cells: the P cells, the transitional cells, and Purkinje cells.

P cells or the pacemaker cells present in large number in the AV junctional area. These cells generate impulses and hence are marked as the automatic cells. Comparing those cells with the contractile cells they contain small number of mitochondria, and less developed sarcoplasmic reticulum. The slow conduc-

tion in the AV node is partially caused by the absence of the intercalated disks.

The transitional cells form a heterogeneous group of cells which are present among the P cells, Purkinje cell, and myocardial fibers. Purkinje cells are mainly present in the branches of His bundle and in the Purkinje network. If we compare them with the contractile cells we will find that they have less mitochondria and linearly situated myofibrils. These cells are also composed of many intercalated disks with a simple T system which might be absent. This system might be the basis of fast conduction of activation. From the conductive point of view the contractile elements are less effective. The Purkinje cells and similar contractile cells are on the other hand considered to be electrophysiological.

The basic electrophysiological characteristics of the heart cells are automaticity, excitability, and conductivity. **Automaticity** is the ability of some cardiac cells to generate impulses which can spread to the surrounding area. **Excitability** is the ability of all the cells in the heart to respond to an effective impulse. In case of repeated impulses these cells need a certain time for recovery (refractory period). Conductivity is the ability of the cell to conduct impulses. The impulse conduction is related to the electrical phenomena of the transmembranous transmission but it is as well related to the heart as whole. The mentioned electrophysiological characteristic could be disturbed and might lead into a disturbance of cardiac rhythm. This is the reason why we explain them in relation with the generation of arrhythmia.

3.26.1 Automaticity

Automatic cells (slow cells) are characterized by diastolic transmembranous potential which is modified by **spontaneous diastolic depolarization** (gradual change in membrane potential in phase 4). The diastolic transmembrane potential is not horizontal as in the contractile cells but is slow ascending, till it reaches the threshold potential. In the contractile cells (fast cells) the formation of transmembrane action potential is the result of impulse affect, which was formed in the pacemaker cells.

The impulse leads to an electrical flow which progresses across the cellular membrane. This electrical flow will reduce the diastolic transmembrane potential of the regional contractile cells (in phase 4) to the threshold potential. The change of the mem-

brane potential to the threshold potential and the beginning of phase 0 will stimulate an electrical flow which spreads across the membranes and its origin is in the automatic cells.

The automatic cells have even with no external interference the **ascending phenomena in phase 4**. This event proves that a slow diastolic depolarization takes place on the membranes. During diastole the continuous Na^+ or Ca^{2+} flow is evident. It is marked as the I_{Bi} which is equalized by the K^+ efflux $I_{\text{K}2}$ or IP flow (see fig. 3.34 page 230).

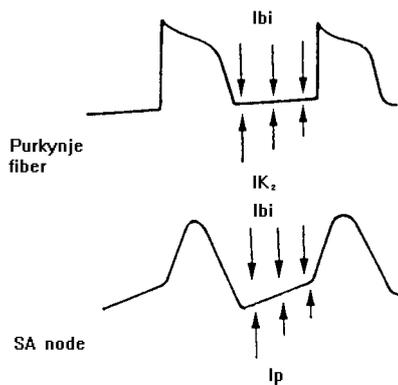


Figure 3.34: Ascend phase 4th of transmembrane action potential of an automatic cells

Inactivation of the potassium efflux explains the formation of transmembranous action potential, when sodium and calcium influx overwhelms or exceeds the potassium efflux. I_{P} inactivation is on the other hand responsible for the diastolic depolarization of the SA node cells, and the $I_{\text{K}2}$ inactivation is responsible for diastolic depolarization of the Purkinje cells. In normal conditions phase 4 is faster in the SA node than in the Purkinje cells. The reason is that the I_{P} is inactivated faster than the $I_{\text{K}2}$. From the mentioned facts we can learn that automaticity has its origin in the changes of ionic flow. Automaticity is produced when there is an excess in permeability and conductivity of K^+ , Na^+ , and Ca^{2+} . Transmembranous action potential (phase 0) in the automatic cells grows more slowly and reaches less values due to its generation from lower values of the diastolic transmembrane potential.

The structures of the conducting system have the duty of impulse conduction to other structures. Apart from this they have the ability to generate impulses which in certain conditions can be propagated to the surrounding regions. Automaticity can generate in the cells of A-V junctional area, but even in the bundle of His and Purkinje system where fast cells are found. But this automaticity is smaller (slower) and normally the sinus automaticity is the dominant one.

The automatic cells reach the threshold potential fast. That is why cells which need the shortest time for reaching the threshold potential dominate over others and they become the center of impulses. The time taken by the diastolic depolarization to reach the threshold potential is basically determined by three factors.

1. The ascending speed of diastolic transmembrane potential (phase 4). The fast and soon reached threshold potential determines high automaticity
2. Level of the threshold potential. When more negative level of the threshold potential with same ascending speed of diastolic transmembrane potential the threshold potential is reached sooner. The final result is a faster automaticity meaning that a more negative threshold potential will increase the heart rate and vice versa.
3. Level of the diastolic transmembrane action potential (DTP). As this level is more negative on the beginning of phase 4 more time is needed for the spontaneous diastolic depolarization to reach the level of the threshold potential. The speed of impulse formation is hence decreased. Contraversly when the starting DTP is not very negative the threshold potential is reached very soon and the speed of impulse generation is faster.

3.26.2 Automaticity disturbances

Decrement of the SA node automaticity can happen due to the following causes:

1. Lowering of the DTP speed
2. Increment of the maximal diastolic potential (e.g.: due to hyperpolarisation)

3. Decrement of the threshold potential (reaching the 0 level)

These causes can occur in combination (see fig. 3.35 page 232).

Increment of the SA node automaticity can happen due to opposite causes of the mentioned above.

When the SA node automaticity is decreased it could be compensated for by the automaticity of the AV junctional area and its frequency is only between 20–40/min. If the automaticity in the AV area was disturbed either, a pacemaker will generate in the subjunctional area and its frequency is only between 20–40/min. During certain structural conditions with slower activity the SA node automaticity can not be interfered. These structures are depolarized with every excitation and they start to repeat the cycle of renewing the membrane potential, which starts to get slower during any given disturbance. Impulses which start to generate as a result of the reduced automaticity of the SA node, can be single or repeated. These impulses from AV junctional area or from the ventricles are referred to as escape impulses when they are repeated they may generate an independent rhythm (escape rhythm).

In some case the **ectopic, atrial, the AV junctional, or the ventricular automaticity can increase** so much that it exceeds the sinus rhythm. In these cases we are not dealing with a defensive but with a hyperactive rhythm. Impulses generating from an increment of automaticity can be isolated or can be repeated. Isolated impulses, which activate the heart do not depend on the original rhythm, they stimulate what is known as **parasytostole**. Isolated impulses which occur in relation with the original rhythm generate what is known as **extraxystole**. Impulses can originate from in one or more centers.

It was proved experimentally that depolarization is sometimes incomplete or delayed. After reaching the membrane potential -50 till -70 mV an early post potentials can occur and depolarization may start again. Delayed post potentials (see fig. 3.36 page 232) are the result of oscillating diastolic depolarization. Arrhythmias caused by post potentials are marked as trigger arrhythmias. They differ from arrhythmias caused by a disturbance in the automaticity. Trigger activity is noticed with arrhythmias that occur post acute coronary occlusion in its late stages (see fig. 3.36 page 232).

3.26.3 Excitability disturbances

Excitability is the ability of all cardiac cells to response on stimulus.

Cardiac cells need a certain time to renew their own excitability. The period needed for their excitability renewal corresponds with the final part of the TAP (voltage dependent excitability renewal). Slow cells need a longer period to renew their excitability (time dependent excitability renewal). This means that those cells don't have their excitability renewed, when in phase 3 they reach a certain level of potential. Their excitability will be renewed after some time of the TAP ends. Potential renewal of excitability corresponds with the transmembrane action potential. The relation of excitability with TAP can be divided into 5 periods (phases) (see fig. 3.37 page 231):

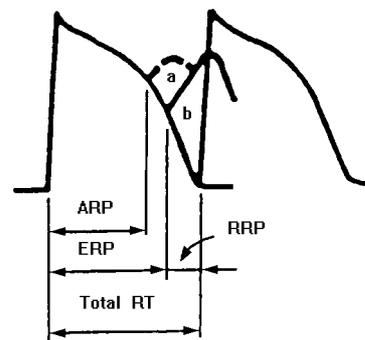


Figure 3.37: Relationship of excitability to TAP

1. Complete refractory period. In this period the cellular membrane is absolute refractory towards any impulse. Absolute Refractory period ARP (see fig. 3.37 page 231)
2. Period of local response. It is a very short period in which some local changes of potential can occur, yet the potential can not be propagated. The effective refractory period ERP consist of the ARP and a period of local response
3. Period of partial excitability. Consist of the last part of phase 3. The response is obtained only

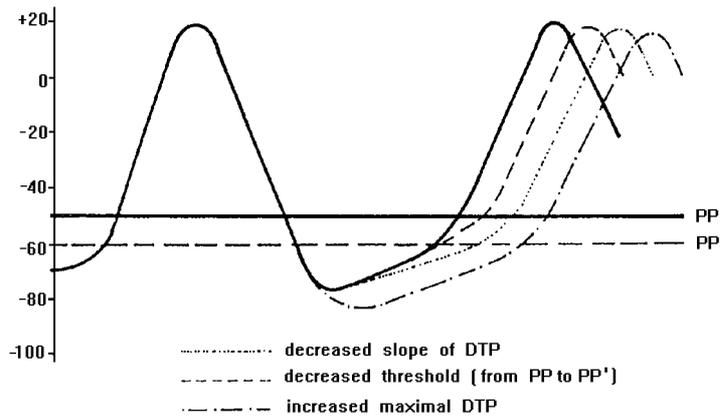


Figure 3.35: Automacity decrease in SA node

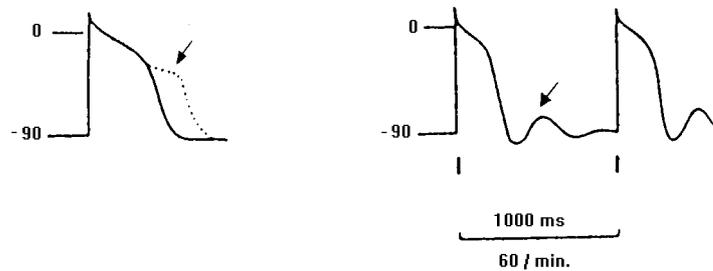


Figure 3.36: Early and late postpotentials

by an above threshold stimulus and it can be propagated. This phase is marked as the relative refractory period (RRP)

4. Period of normal excitability. The cell responds to any stimulus which has the threshold intensity.
5. Period of supernormal excitability. In this short section at the end of phase 3. The cell responds to a sub threshold stimulus.

3.26.4 Conduction disorders

The impulse which generates in the SA node is propagated through a special conductive system to the atria and to the ventricles. The progress of activation is basically an electrochemical change, which takes place on the cellular membranes. This is realized without chemical mediators, but the speed of the activation progress can be changed by material which usually affect the TAP (those are catecholamins, acetylcholin and anti arrhythmic substances).

The cytoplasm of muscle fibers and interstitial fluid have a low resistance and are good electric

transmitters. Cellular membranes are structures with high resistance and they separate these 2 media. The conduction is different in the fast and slow cells. For fast cells the regenerative conduction is typical. For slow cells in the junctional area, mainly the AN and H ones the decremental conduction is typical. The speed of conduction in different cardiac structures is:

- atria 1–2 m/s
- AV node 0,05 m/s
- His–Purkinje system 1,5–4 m/s
- Ventricles 0,3 m/s

The speed of conduction is determined by 2 basic factors:

1. How fast the TAP level is reached (dV/dt phase 0)
2. Ultrastructural characteristics

Thin fibers (contractile and transitional cells) and those not having intercalated disks (P cells) conduct activation slowly. Thick fibers with intercalated disks (Purkinje cells) conduct activation fast.

In the sinoatrial junction and AV junction area the activation is progressing slowly. This is caused by the presence of large number of P cells and transitional cells. In these structures conduction disturbances can be in form of supernormal conduction, gap phenomena, slow conduction, and a hidden conduction and reentry mechanism.

Conduction faster than normal. An early impulse can be blocked during propagation. Sometimes an ectopic impulse is created even earlier and this impulse could be propagated. On one side we are dealing with a supernormal excitability of the surrounding cells but on the other hand there is a supernormal conduction. This could be the result of transitional hyperpolarisation of cellular membranes. It was shown experimentally that a supernormal conduction can be time or tension dependent. During pre-mature stimuli the supernormal conduction may lead to gap phenomena. Premature impulse slows the progression of activation. The earlier impulse comes the slower. The activation reaches the distal zones outside of refractory period.

The result is that there is shortening of the complete activation progressing time. Another gap phenomenon is noticed with antigrade and retrograde conduction. Differentiation could only be obtained by intracardial electrocardiologic monitoring.

Conduction via abnormal pathways. These conductions are mainly shown in WPW (Wolf Parkinson White) syndrome and in short PR interval syndrome. It could occur even with other disorders. In these cases the activation is progressing via unusual pathways which might or might not be histologically evident.

Slowing of conduction. There are many types of slow conduction. It could only concern a certain part of conductive system of the heart. In the electrocardiologic terminology we use the term block. We can distinguish many types of blocks. First degree heart block. This term refers to a condition when impulses are constantly created but their propagation is somehow a bit slower than normal.

Second degree heart block It is marked as Mobitz I. A classic picture are Wenckebach periods during which the P-Q interval is gradually prolonging till they reach a state where no impulse is propagated anymore. When the relation is not strong the block is marked as Mobitz II.

Third degree heart block This type occurs when no impulse is conducted to the ventricle. Atria and ventricles have an independent activity on each other. The AV dissociation can be caused by an extremely long refractory period of the AV junction. It can be a picture of complete block. In this case no impulse is transmitted from atria to ventricles. In case of incomplete block and AV dissociation some impulses can be conducted from atria to ventricles. In other cases it is possible that a fast activity in the junctional area could be transmitted in a retrograde direction back to the atria. There is formation of AV dissociation with interference. Sinoatrial block is a condition during which no impulse is conducted from the SA node to the atria. Usually in these cases there is the formation of a substitute impulse in the AV-junctional area.

Aberrant Ventricular Conduction. Is formed as a result of differences in the refractory period between some parts of the transitional abnormal distribution of supra ventricular impulses. We can see ventricular complexes which have abnormal shapes. The condition can occur as a result of prolongation

of the refractory period in one of the branches or when the impulse reaches the ventricle through an accessory pathway. In physiological conditions the refractory period is longer in the right arm. The classical aberrant conduction in ventricles depends upon the length of diastole. It could be seen during extrasystolia or with tachycardia. Electrocardiographic picture of wide QRS with a supra-ventricular tachycardia may disappear with a certain drop in the heart rate. During fast heart rate the impulses in the right branch of bundle of His are in the refractory phase. The picture of aberrant conduction can occur even when the cycle is prolonged. It is caused by an increased diastolic depolarization, or when the threshold potential reaches near 0 levels, or by some hidden conductive pathway, or by a combination of many factors. Aberrant conduction occurs even with normal cycle of an intermittent ventricular block. It can occur with incomplete depolarization in a certain area of a conductive system of the heart or with incomplete production of TAP.

Concealed Conduction Could be a partial depolarization which can not be differentiated using the electrocardiogram. Concealed conduction is the cause of a large number of phenomena in arrhythmology. Usually it is concerned with AV junction or branch of bundle of His.

Reentry disorders of conduction are sometimes dealing with a certain area. We can talk about an extra bundle branch or local block. In the surrounding tissue the activation can be conducted normally. A block in such a place could be unidirectional. This means that activation can reach this area from the opposite side. In this case activation can spread repeatedly via same pathways. The progress of activation is slow.

We are talking about classical reentry phenomena. The obstacle of the spread of activation doesn't have to be an anatomical structure. When a circular activation is formed the conditions will be in favor of slow progress of activation and of shortening the refractory period.

A. reentry with normal conductivity and unidirectional block

This type occurs in slow cells during some abnormal conditions. In this case extrasystolies are formed. These extrasystolies have constant interval after the previous systole. Basically depending on

these facts three types of reentry can occur

1. enclosed conductive circle
2. unidirectional block
3. a proper speed for conducting activation.

An enclosed reentry circle can have many types. It can occur in the atria with or without participation of the junctional area. In His-Purkinje system a vicious circle could occur either in the peripheral Purkinje fibers or at the level of junction of Purkinje fibers with the contractile myocardium. In this case the vicious circle is very small. It is a typical example of microreentry. It is usually the basic reason for ventricular extrasystolies and hyperactive ventricular rhythm. (see fig. 3.38 on page 235). An enclosed circle could be formed in the branches and the fascicles which have parallelly situated fibers. The proper conditions for the circle closure occur when two independent waves of activation are progressing simultaneously. This condition is known as reentry with summation of activation.

A closed circle can happen in AV junctional area (see fig. 3.39 on page 235). Reentry phenomenon then leads to the appearance of junctional reciprocal tachycardia. It is a regular supraventricular tachycardia. Reentry cycle can exist with the participation of intranodal structures, or with the participation of juxtanodal structures.

Intranodal circle can have a pathway with a short refractory period and a prolonged conduction, or it can take the b pathway with a long refractory period and a shortened conduction. Sometimes there might be formation of a pathway in the extranodal structures which have the character of specialized abnormal pathways. Reciprocal tachycardia with AV junctional area participation can have:

1. slow antigrade and fast retrograde conduction
2. fast antigrade and slow retrograde conduction.

Unidirectional block. In this case the determinant is that a part of tissue can be activated from the tissue which is in the area of block. Overmore if the myocardium is not in the refractory period it could be reexcited. A small circle could be formed, (a microreentry) or a large circle could be formed, a macroreentry, which has the character of intra and extra nodal circle. When it is intranodal it has the

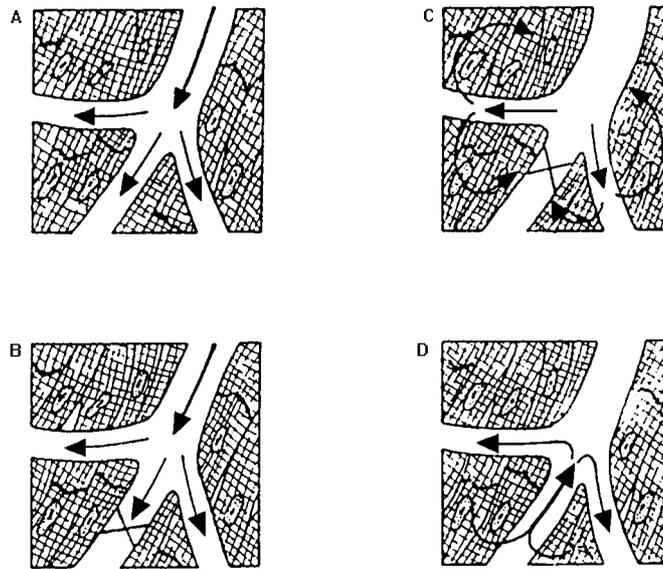


Figure 3.38: Unidirectional block of one Purkinje fiber

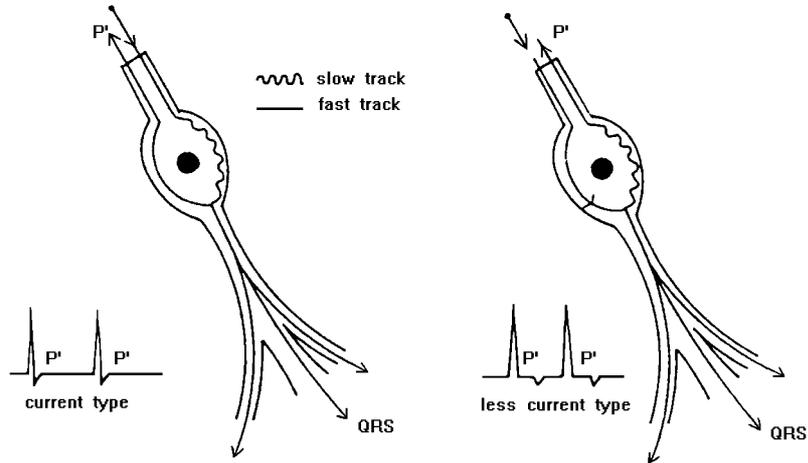


Figure 3.39: Closed circuit in AV junctional region

characteristic of B pathways. The impulse can be transmitted from the atria via the A pathway towards the ventricles and can be transmitted retrogradely back to the atria. The atria may be re-

activated. As a result of this the reentry continues with the participation of both pathways.

When the cycle consists of atria, AV-junctional area, and ventricular myocardium it is very difficult

or even impossible to determine the exact place of the unidirectional block.

A proper speed of conduction. When a reentry of this type is formed the progress of activation can not be very fast, otherwise it hits a zone which is in the refractory period. It also can not be very slow. Because of the mentioned the atria would be then activated first by the SA node.

B. Reentry resulting from disturbance of refractory period

In this case some zones with shortened refractory period may occur. In neighboring zones the refractory period is prolonged or different (non homogeneous). This situation usually occurs in areas where slow and fast cells exist. During fibrillation there may be a microreentry in different areas of the myocardium.

3.26.5 Sinoatrial node dysfunction

SA node is the dominant pacemaker of the heart because it is the fastest one to reach the threshold potential level. The speed of diastolic depolarization is under the control of vegetative nervous system i.e. (autonomic nervous system). It is getting fast during activation of the organism and slows down during sleep and rest. The increment in impulse formation at rest is usually obtained by sympathetic activation via the B adrenergic receptors. Slowing on the other hand is realized through the activation of parasympathetic fibers via the muscarinic receptors. Sinus rhythm in the adults reaches a frequency between 60–100 beat/min. In children the frequency is higher, in newborn it is about 120–140/min. Trained individuals (athletes) have a frequency which is below 50/min, most probably as a result of an increment in parasympathetic tonus.

The normal SA node function can be disturbed due to decrement in the direct vascular supply of the SA node. This usually happens in coronary sclerosis, mainly the right coronary artery. Another type of dysfunction is senile amyloidosis of atrial myocardium. Sinus bradycardia occurs in hypothyroidism, hypothermia, liver diseases, brucellosis, vasovagal syncope, hypoxia, hypercapnia, acidosis, acute hypertension, and in other conditions. If bradycardia occurs quickly, it can evoke haemodynamic changes, which are manifested as an acute

drop in the cardiac output. There might be no impulse formation in the SA node (sinus arrest) or there might be a block of the SA node. Sometimes the SA node dysfunction occurs together with AV dysfunctions. Elsewhere the SA node dysfunction and arrest is manifested by syncope and a possible junctional rhythm. Yet more often the SA dysfunction is shown as an inability to increase cardiac frequency during situations which are usually accompanied by tachycardia. The SA node dysfunction can appear after the use of pharmaceuticals which usually don't affect the heart rate in healthy individuals.

Sick sinus syndrom has a very wide range of symptoms. It is manifested by bradycardia, sinoatrial block, or sinus arrest. Atrial tachyarrhythmia as well as fibrillation and flutter are usually joined with tachyarrhythmia and bradyarrhythmia. Sinoatrial block of first degree presents a prolonged progression of activation from the SA node to the surrounding tissue. When the atrial activation (P wave) is the first to be shown in the ECG, the first degree block can not be diagnosed from the electrocardiogram. Only by a special examination being an intracardiac registration if possible.

Second degree sinoatrial block is a disorder, during which some impulses from the SA bundle are not transmitted to the surrounding tissue. It is expressed as the absence of one P wave and the related QRS complex.

Third degree sinoatrial block is actually a complete sinoatrial block. It cannot be differentiated from sinus arrest on the ECG. This disorder could be registered by the use of intracardiac registration bradycardia.

Tachycardia syndrom is shown as tachyarrhythmia on the ECG. In this case the retrograde progression of activation from the atria can lead to SA node suppression. The most important thing is to recognize the relation between the SA node dysfunction and other symptoms from which the patient suffers.

Very important is the 24 hour monitoring of the electrical function of the heart. By pharmacological tests it is possible to influence the autonomic nervous system, to find out if there is a primary error in the SA node. By using a complete chemical (pharmacological) block of the autonomic nervous system it is possible to prove the SA node dysfunctions caused by the autonomic nervous system and to exclude primary SA node dysfunction. An effective method is

the intracardiac registration with complete pharmacological block, so that it is possible to measure the refractory time for the SA node. First we provide the atrial pacing and after that we notice how much time is needed for the activity of the node to occur. The SA node recovery (refractory time) is less than 550 ms, its prolongation is a sign of SA node dysfunction. The carotid sinus syndrome can be verified by short massaging of this area and monitoring the ECG. Patients with SA node dysfunction may or may not have various symptoms in relation with this dysfunction. In the asymptomatic SA dysfunction it is not necessary to do the electrocardiological monitoring or cause any trauma to the patients by other examinations. SA node dysfunction often occurs together with AV conduction defects. Here the haemodynamic symptoms are mostly caused by the AV conduction disorder and not by the SA node dysfunction alone. In these cases it is necessary to provide a complete electrocardiographical examination together with the electrocardiographic registration of the His bundle and a programmed atria R and ventricular stimulation. In patients with SA node dysfunction, which is connected with haemodynamic outcomes a permanent pacemaker is very likely the best solution. In a disorder which is manifested only during a paroxysm or during an attack it is better to use ventricular or biventricular or even atrial on demand pacemaker (this pacemaker paces only when needed).

3.26.6 Disturbances of AV conduction

There is a morphologically defined structure between the atria and ventricles. This structure provides the transmission of activation from atria to ventricles.

Apart from activation conduction the atrioventricular junction shares in the cardiac action coordination by slowing down the conduction of activation, so that to enable the atria and ventricles to work in a coordinate manner from the haemodynamical point of view.

The AV junction is composed of structures together known as the AV junctional area. It is composed of the AV compact node, which is supplied by sympathetic and parasympathetic fibers. The progress of activation is influenced by the autonomic nervous system. Slow transmission of activation is

known to occur in trained individuals with a marked vagotonia. The AV junctional area as well as the AV node changes during many diseases. AV blocks can occur even as congenital defects. The progress of activation across the AV area can be changed in myocardial infarction of the posterior wall or in right coronary artery spasm. The AV junction is affected by many drugs, mainly digoxin, beta blockers, calcium blockers, and some toxic substances. The AV conduction disorders are seen in some viral infections like viral myocarditis, in rheumatic fever, infectious mononucleosis, in amyloidosis, and in neoplastic diseases of the heart. AV block can occur simultaneously with the bundle branch block. This usually occurs in sclerotic fibrosis of the cardiac skeleton, aortic and mitral valves. Sclerodegenerative changes of the AV junctional area can occur in an isolated manner. Hypertension and aortic or mitral stenosis are diseases, which usually accelerate the degeneration of the conductive system, or may cause its calcification and fibrosis.

First degree AV block. Often marked as the prolongation of AV conduction. There is prolongation of the PR interval for more than 0,20s on ECG. This interval represents the progress of activation through the atria, AV bundle and His-Purkinje system. The prolongation can occur in any of these parts. The QRS complex is normal. When the PR interval is changing the error usually lies in the AV bundle. In case the error is more distal in the His-Purkinje system, it is always accompanied by QRS prolongation. Only by using the intracardiac electrography it is possible to locate the exact place of the error.

Second degree AV block. Also known as the intermittent AV block. In this case some impulses are not conducted to the ventricles. On the ECG we can notice a gradual prolongation of the PR interval, until one ventricular systole doesn't occur. This is also known as Mobitz 1 second degree AV block. Before this the disorder was known as Wenckebach periods or Wenckebach block. The error is usually localized in the AV node. The QRS complex is usually unchanged. This block may occur in myocardial infarction, in digitalis intoxication, post beta blockers and calcium antagonists. The seriousness of this lies in the haemodynamic changes. When there are no haemodynamic changes, the prognosis is excellent (see fig. 3.40 page 238).

Sometimes the impulse is not conducted to the

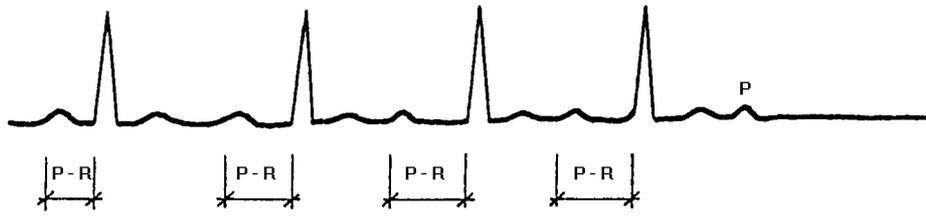


Figure 3.40: AV block of 2nd type, Mobitz type I.

ventricles without prolongation of the PR interval. This is known as Mobitz type 2 second degree heart block. The primary error is usually localized in the His-Purkinje system. Often there is prolongation of the QRS complex. The most important thing is to know whether the error progresses to a complete block or not. It is usually found in the anteroseptal infarct, or in sclerodegenerative or calcificating disorders of the fibrous skeleton of the heart. In what is called a higher degree block, two or more successive impulses are not expressed in the ventricles. If the resulting haemodynamics is affected a pacemaker is necessary (see fig. 3.41 on page 238).



Figure 3.41: AV block of 2nd type, Mobitz type II.

Third degree heart block, also known as complete AV block. No impulse is transmitted from atria to ventricles. If there is a ventricular substitutive rhythm, the frequency reaches 40–55/min. Complete congenital AV block originates in the AV bundle. The main error is usually localized in the His bundle (see fig. 3.42 on page 238). This complete AV block causes syncope and ventricular arrest. But usually this block is preceded by some mild dysfunction. That is why it is important to define them, know their progression, and if possible determine the probability of a substitutive rhythm occurrence dur-

ing the complete AV block. The last mentioned demand is the most important one. The resulting picture of the complete AV block depends if there is or if there is not a substitutive rhythm, and the type of this rhythm. In good conditions the substitutive rhythm can generate in His bundle. Here the frequency reaches 40–60/min. Because the progress of activation is usually normal, the ventricular complex is not deformed. Yet when the activation is generated in distal parts of the conductive system e.g. in Purkinje fibers, the frequency is about 25–45/min. The progress of activation in the ventricles is abnormal, and shows itself as a wide QRS complex.

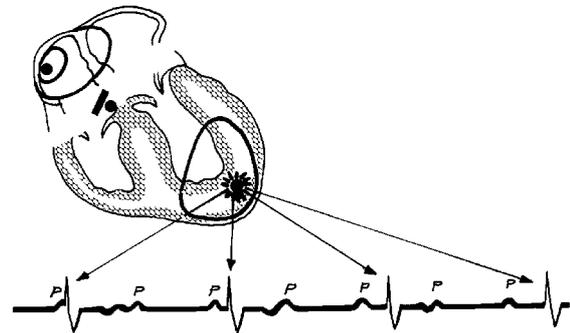


Figure 3.42: AV block of 3rd type

AV dissociation represents a disorder in which the control of the ventricles is separated from the control of the atria. This error doesn't have to be combined with the complete AV block. AV dissociation can occur in extreme sinus bradycardia. It can also occur when there is an ectopic junctional or ventricu-

lar pacemaker that has a higher frequency of impulse formation than the SA node. Here an interference in the rhythms can occur. This condition occurs in case of accelerated junctional or ventricular rhythm, in ischemic disease of the heart, in myocardial infarction, or after cardiac surgery.

AV blocks that have haemodynamic symptomatology can be solved by the use of pacemaker. Prior to this it is necessary to perform an intracardiac electrocardiographic examination. The obtained results enable us to understand the pathophysiological mechanism of the disorder. Valuable information are obtained from the electrocardiographic registration of the bundle of His, and from programmed stimulation. Electrocardiography is necessary in patients with syncope and a bundle branch or a bifascicular block the His bundle electrocardiography is necessary. If the HV interval is longer than 100 ms, this means that there is an infra-His disorder, which should be solved by pacemaker. In AV block we should differentiate between the intra or infra-His block, or finally if there is block in the His-Purkinje System.

It is possible to use a trans venous insertion of catheter electrodes to the apex of the right ventricle with an external monitoring to handle a complicated case of atrioventricular conduction disturbance. Electrodes for permanent pacing is usually placed in the right atrium or right ventricle via the subclavian vein. The source of power is fixed subcutaneously in the sub clavicular area. Epicardial electrodes are used when, due to other reasons thoracotomy is performed. The **pacemaker** can have a fixed frequency or it can work *on demand*, it may also be *rate adaptive*. The electrodes can be connected to atrium or ventricle or it can be connected to both arteries or ventricles. From the pathological point of view it is not easy to simulate the optimal function of the conductive system of the heart. Pacing can cause haemodynamic changes in the atria and the atrioventricular valves. Such change can manifest itself as marked difficulties for the patient. The combination of symptoms is known as the pacemaker Syndrom.

3.27 Tachyarrhythmia

All kinds of tachyarrhythmias can be divided into two types depending on their origin. A group of tachyarrhythmias which cause are changes in the spread, and progression of activation. Another group is caused by a defect in the formation of stimuli. The electrophysiological bases of conduction disturbances were considered in the previous chapter. The reentry mechanism is the most common of cardiac dysarrhythmias. With no detailed electrophysiological differentiation it is shown that the following four conditions are needed for the reentry to occur:

1. An electrophysiological nonhomogeneity of two or more areas in the heart, which are activated, or can be activated following each other, and hence they can form a potentially morphological base for a close conductive circle. This nonhomogeneity results from the difference in the conductivity of these structures or the difference in their refractory period.
2. A unidirectional block means, that there is part of the conductive loop that can be activated from the neighboring area, which is part of the conductive loop and part of the block.
3. The refractory period should be shortened to a level that permits reexcitability on repeated activation.
4. Re excitation leads to the formation of loop activation.

The repeated circulation of an impulse can form the basis for the formation of arrhythmia's. These arrhythmia's can be initiated repeatedly. Yet they can end as an extra systole or as a fast stimulation. This characteristic renders them different from the trigger arrhythmias.

Arrhythmias that are generated from disturbance in the impulse formation, can be further divided into, those caused by automaticity change, and those resulting from triggered activation. Normally the myocardial cells lack the pacemaker action of the SA node, in other words this activity is very low in them. The pacemaker activity is obtained due to the