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 Tachyarhythmia

All kinds of tachyarhythmias can be devided into two types depending on their origin. A group of tachyarhythmias 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 dysarhythmias. With no detailed electrophysiological differentiation it is shown that the following four conditions are needed for the reentry to occur:

1. An electrophysiological nonhomogenesity 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 nonhomogenesity 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 devided into, those caused by automacity 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

speed of the spontaneous diastolic depolarization. In the originally rhythmogenic cells its duration is very long and it is always disturbed by the coming activity from the neighboring areas. Increment in the automacity of the potential pacemaker cells can affect the following conditions in the heart:

1. An increment in the endogenous and exogenous catecholamins.
2. Electrolyte changes.
3. Hypoxia or ischaemia.
4. An increment in the muscle fiber tension (dilatation).
5. The effects of some substances.

The high automacity may lead to tachycardia, which is initiated from an ectopic excitatory locus. Tachycardia resulting from an ectopic automacity can not be initiated neither stoppe

Trigger activity phenomena ca
isolated cells, when there is an inc
cal concentration of catecholamins,
hypercalcemia, and in toxic effect
these cases there will be an intrac
tion of calcium, which is caused b
This is marked as early depolarizati
ization). If the amplitude is high e
activation will occur. For the init
ger activity it is necessary that a
occurs first. That is why trigger
caused by pacing. To give an exac
mechanism of defect formation we have to perform an intracardial registration of the activation progression, and a programmed stimulation. The result of this examination is useful for proper treatment.

3.27.1 Extrasystole

Extrasystole is known as that cardiac systole which occurs early and disturbs the original heart rhythm. We can divide the extrasystole depending on many principles. One of these is according to the location of the impulse formation which is the main cause of the extrasystole formation.

Atrial extrasystole can occur in totally healthy people. Those individuals usually don't know about this condition. The 24 hr monitoring could prove the existence of extrasystole in nearly 60 % healthy individuals. More sensitive individuals could notice palpitation. The occurrence of atrial and supra ventricular tachycardia can be evoked by alcohol, smoking and adrenergic stimulation.

Atrial extrasystoles are based on extra stimulus which can form at any time in the atria. On the ECG these extrasystoles are shown as a morphologically changed P wave during sinus rhythm. The more the ectopic center is away from the SA node the more distinguished is the P wave. The activation starting in the ectopic locus reaches the AV node and spreads to the ventricles via the usual pathway. That is why the QRS complex is unchanged. Sometimes the activation may come from the atria to the AV node very quickly. AV node may still be in the refractory period. In such case the PR interval is prolonged and the QRS complex has a different morphology compared with the normal non extra systolic complexes (see fig. 3.43 page 240).

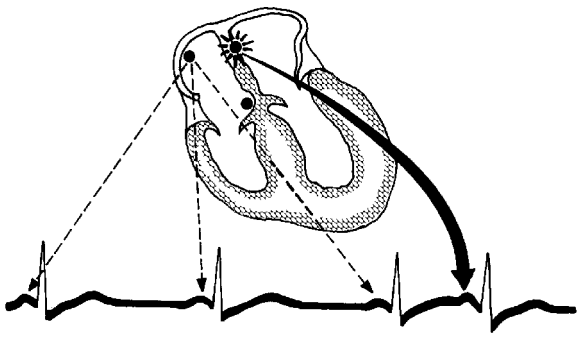

Figure 3.43: Supraventricular extrasystole

AV junctional extrasystole. AV junction includes many morphologically defined structures. The compact AV node is composed of cells that have no pacemaker activity. That is why the extrasystoles can have its origin in all other structures of the AV junctional area apart from the compact AV node. The most common place for extra stimulus generation is in the bundle of His.

The AV junctional extrasystole is less common. They occur in heart diseases. The activation spreads in an anti grade manner to the ventricles, but also in a retrograde manner from the ventricles to atria. On the ECG the QRS complexes have normal configuration, yet the interval between the beginning of the

P wave and the QRS complex is shorter. During the retrograde activation the P wave could be inverted in lead II, III and AVF. The P wave can either occur after the QRS complex or it can disappear in the QRS complex (see fig. 3.44 page 241).

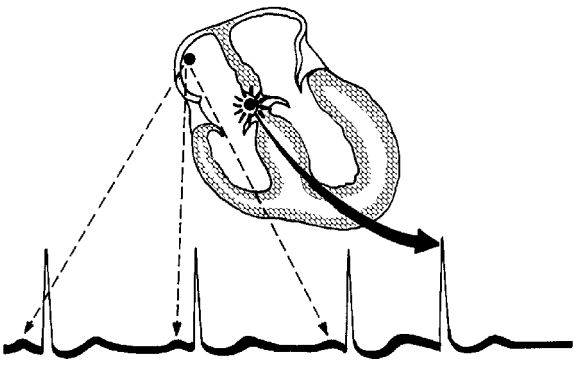

Figure 3.44: AV junctional extrasystole

Ventricular extrasystole. Occurs relatively often. It was found that in nearly 60 % of adults was revealed individual ventricular extrasystole during the 24 hour monitoring. What is important to know is whether these extrasystoles occur in a healthy or in a diseased heart. In a healthy heart the individual extrasystoles are not considered a risk factor, which means an increased morbidity or mortality. The extrasystoles occur in 80 % of patients who suffered with myocardial infarction. It is important to know whether we are dealing with individual extrasystoles or not and how often they occur. If their number is more than 10 per hour and if they occur in clusters, they are usually connected with an increased mortality in these patients. The mortality though is related to the present, and markedly noticed ventricular dysfunction. Ventricular dysfunction might lead to heart failure as a pump. Ventricular tachycardia or fibrillation is once again very dangerous because they may lead to sudden death.

Ventricular extrasystole are easy to recognize on the ECG due to their wide and morphologically changed shape of the QRS complex. Apart from this there is no P wave preceding the QRS complex (see fig. 3.45 on page 241).

Ventricular extrasystoles are usually strongly related to the preceding normal complex. If there is

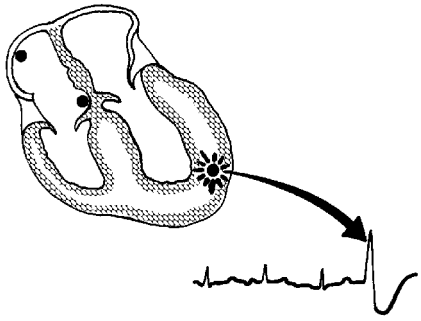

Figure 3.45: Ventricular extrasystole

no strong relation (i.e. the distance between the normal systole and extrasystole have different duration) then we have to count the distance between the individual extrasystoles, and if this distance is one of the multiples of the same distance here we are dealing with what is known as parasystole (see fig. 3.46 on page 242).

In the ventricles there is a locus with an abnormal automacity and this locus is protected against the normal coming activation. The function of this locus is regular, and that is why the distances between the extrasystoles has the same relation.

Ventricular extrasystoles are either isolated or repeated (see fig. 3.47 on page 242).

Bigeminy is a condition in which each normal systole is followed by an extrasystole, whereas **trigeminy** is a case where each two normal systoles are followed by an extrasystole.

If one systole is followed by another one here we are talking about a coupled extrasystoles. When three or more extrasystoles occur together we are dealing with a cluster of extrasystoles or ventricular tachycardia. If the extrasystoles originate from one place they are known as monomorphic, because they have similar shapes. On the other hand extrasystoles which have different shapes originate from different areas, and so are called polymorphic or multimorphic. Ectopic activation of the ventricles doesn't spread in a retrograde manner to the atria. This is why the SA node is not exhausted by this activity. The activation from the SA node reaches the ventricle in the refractory period. So the next impulse coming from the SA node is effective. The inter-

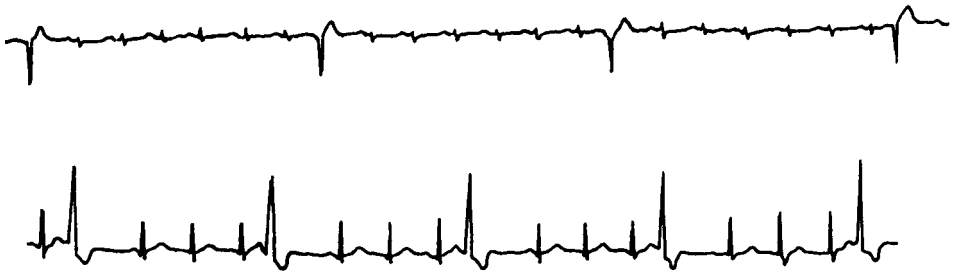

Figure 3.46: Parasystolia

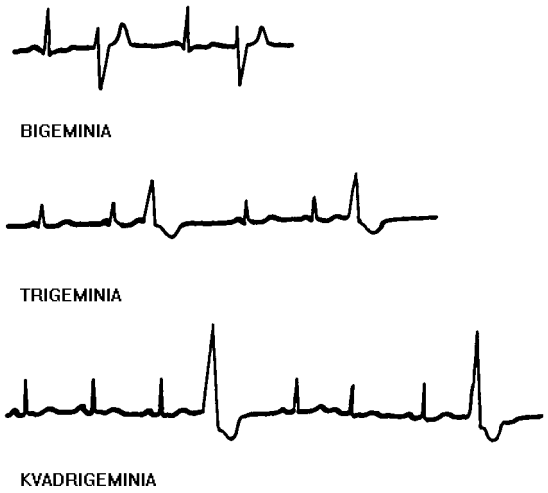

Figure 3.47: Recurred extrasystoles

val following the extrasystole is longer than normal and it is called **the compensatory pause**. The interval preceding the extrasystole together with the compensatory pause equals two RR intervals. Occasionally the activation may spread to the atria. Here a negative P wave can appear in lead II, III and AVF. The compensatory pause will be shorter because the sinoatrial node is now exhausted. Sometimes the activation reaches only the AV node, so in the following normal systole there will be prolongation of the PR interval which proves the hidden retrograde spread of activation. Ventricular extrasystole can occur as a result of a marked sinus bradycardia but here there will be no retrograde spread or compensatory pause. This is a case of interpolated extrasystole. If the extrasystole occurs very shortly after the last complex it is known as the R on T phenomena, it might be a risk factor in the ventricular fibrillation occurrence.

Individual ventricular extrasystoles do not require any treatment. In otherwise healthy heart treatment is questionable. Treating ventricular arrhythmia's don't seem to affect the risk of the occurrence of sudden death. Moreover the antiarhytmic drugs may cause lethal arrhythmias. The situation is of course different in patients with MI where the ventricular tachycardia may proceed into ventricular fibrillation.

3.27.2 Tachycardia

Under the term tachycardia we mean tachycardia which occur suddenly and reaches over 100/min. Paroxysmal tachycardia starts by atrial or ventricular extrasystole. They occur on the base of reentry mechanism. Only in case of intoxication they occur as a result of trigger activity.

Paroxysmal (attack) tachycardia can be an acute and dramatic situation which starts suddenly and ends suddenly. The important factor here is the haemodynamic state. If the haemodynamic state is stable we can provide a longer ECG monitoring. On the ECG we consider the shape of P wave and its presence, the morphology of the QRS complexes, the relation between atrial and ventricular activation and possible differences between the sinus rhythm

and tachycardia. In case of good haemodynamic state we may try to perform carotid sinus massaging, or any other vagal reflex. *Affecting* the automatic nervous system can sometimes end the tachycardia. Yet, massaging the carotid sinus can in this case evoke asystolia or ventricular fibrillation. The electrocardiologic monitoring during the presence of a flexible stimulating electrode situated in the oesophagus can give us some valuable information and we can also try a vagomimetic maneuver.

3.27.2.1 Sinus tachycardia

Sinus tachycardia is not *classical* or *primary arrhythmia*. It is basically an extremely high cardiac frequency. It occurs in stress situation, fever, blood loss, anxiety, during work, during thyrotoxicosis, hypoxemia, hypoxia, and in heart failure. It usually starts insidiously and ends gradually. We can notice signs of sinus rhythm on the ECG even if they are sometimes slightly changed. Massaging the carotid sinus terminates or at least decreases the tachycardia.

3.27.2.2 Atrial fibrillation

The primary cause of atrial fibrillation is a chaotic activation of atrial myocardium in very small sections. Atria are dilated and it is possible to see non coordinate contraction of small sections if we look to the atrial surface. It is not possible to see P waves in any of the 12 leads of ECG. We can see many deviations between the QRS complexes. These deviations are of different shapes and sizes. Their frequency reaches about 300–600/min. The atrial activation reaches the AV node in large numbers but it dies out at the AV node because of the long refractory period of the AV node, or sometimes due to a hidden conduction. The activation reaches the ventricles irregularly, but in large numbers. **The ventricular irregularity is totally irregular**. On auscultation with simultaneous pulse examination we can notice a pulse deficit. The cause of which is what is known as a non systole outcasting ie. due to the short diastole there is not enough time for the ventricular filling and when the ventricle contracts there will not be opening of the semiluminar aortic valves and hence the pulse wave doesn't occur. Atrial fibrillation can come in paroxysms or can be a continuous process. It can occur following cardiosurgery, or after intoxication by alcohol or during life threatening situations. It more often occurs in patients with cardiopulmonary diseases, in acute hypoxia, hypercapnia, or in metabolic disorders. It occurs in rheumatic fever, mitral valve diseases, hypertension, atrial septal defect and in thyrotoxicosis.

Atrial fibrillation with **a high ventricular frequency** can lead to hypotension or syncope. Atrial fibrillation may lead to thrombus formation in the atria and these thrombi may reach the lungs or the greater circulation If this condition is long lasting it might lead to heart failure. Together with mitral stenosis it may lead to lung oedema (see fig. 3.48 page 243).

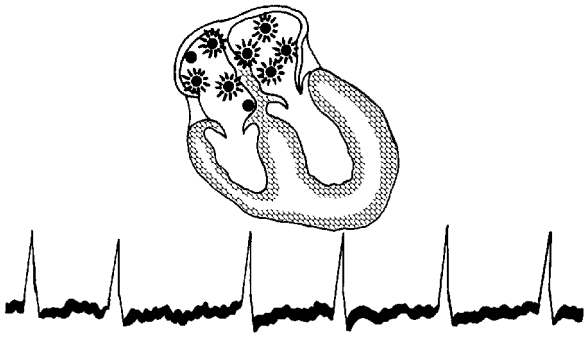

Figure 3.48: Atrial fibrillation

3.27.2.3 Atrial flutter

Ectopic activation is the usual cause of atrial flutter and this activation leads to 250–350 contraction/min in the atria. This condition usually occurs in patients with cardiac diseases, pericarditis, acute respiratory insufficiency, and after cardiosurgery. The thrombi are formed less frequently. The ECG shows a typical picture of saw-toothed waves between the ventricular complexes, the ventricular complexes on the other hand have normal configuration. Only part of the impulses are transmitted form atria to the ventricles. This is the reason, why the ventricular contraction is regular, and usually without tachycardia.

Detailed electrocardiological studies revealed that atrial flutter is maintained depending on the principles of the reentry mechanism (see fig. 3.49 on page 244).

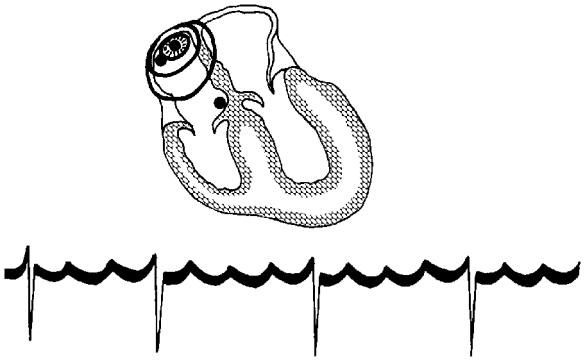

Figure 3.49: Atrial flutter

3.27.3 Paroxysmal supraventricular tachycardia

Nearly in all cases we are dealing with the mechanism of reentry. The close cycle of activation is running via the SA node to the atria, the AV node and then back to the SA node. This circle can sometimes be formed of the AV area with an anti grade progression of activation and a retrograde pathway for activation progression via a by-pass tract. The anti grade by-pass tract is present in the WPW syndrome. Yet we more often deal with a hidden retrograde by-pass tract.

3.27.3.1 AV nodal reentry tachycardia

is the most common form of the paroxysmal supraventricular tachycardia. It is characterized by narrow ventricular complexes. Tachycardia is initiated by atrial extrasystoles, which have a prolonged PR interval. During the tachycardia P waves may differ in their relation to the QRS complex. Yet their position during the tachycardia is stable. Prolongation of the PR interval is caused by the prolongation of AH interval which is shown on the electrogram of bundle of His. This is most probably caused by a doubled conduction through the AV node. β pathway is characterized by a fast conduction with a long refractory period. α pathway on the other hand is characterized by slow conduction and a short refractory period. During the sinus rhythm only the β (fast) pathway is used, and this is why the PR interval is normal (not prolonged). When atrial extrasystoles occur, they spread towards the AV junction. They cannot continue spreading via the β pathway because it is still in the refractory period of the previous activation, and this refractory period is quite long. So this is why the activation continues its progression via α pathway. The progression of activation via this pathway is so slow, that there is quite enough time for the β pathway to recover from its refractory period. The activation reaches the ventricles via the β pathway and via the same pathway there will be atrial echo and so tachycardia continues. For the tachycardia to continue an optimal coordination between the refractory period and the speed of activation in the AV bundle is necessary. Ventricular and atrial activation occur simultaneously. That is why P waves are imbedded in the QRS complex (see fig. 3.50 page 244).

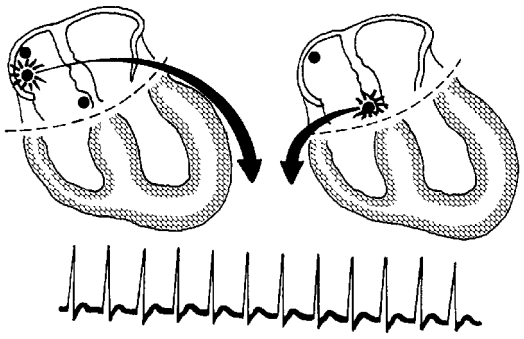

Figure 3.50: Supraventricular tachycardia

3.27.3.2 AV reentry tachycardia

A determinant factor for this supraventricular tachycardia is a by- pass tract that is incorporated in the loop activation and this AV-by-pass tract can conduct activation strictly in a retrograde direction. The activation progresses from the atria via the AV node and His-Purkinje system to the ventricles and from there by a retrograde direction via *a hidden* by-pass tract back to the atria. In this paroxysmal tachycardia unlike in the WPW syndrom the hidden (concealed) by-pass tract cannot transmit or conduct activation an anti grade direction when there is sinus rhythm. Tachycardia starts by atrial or ventricular extrasystole. P waves occur after the QRS com-

plexes. The electrocardiographic mapping of atrial activation have a great value in determining the origin of this arrhythmia. The main proof of presence of a *hidden* by-pass tract is the fact that we can activate the atria by activating the ventricles by a stimulus in time when the His bundle is in the refractory period. This defect can be treated by surgical ablation.

3.27.3.3 Sinoatrial reentry tachycardia

This paroxysmal supraventricular tachycardia starts by atrial extrasystoles. P waves are similar as in the sinus rhythm, but there is prolongation of the PR interval. By this it differs from the sinus tachycardia, in which the PR interval is shorter. If the reentry is intraatrial P waves have different configuration and the PR interval is prolonged.

3.27.3.4 Atrial tachycardia without reentry

They occurs in cardiac and pulmonary diseases, hypokalaemia and after the application of some substances. We are commonly dealing with that is known as multifocal tachycardias. They are characterized by different P wave morphology. This commonly occurs after overdosing with digitalis.

3.27.3.5 Preexcitation syndrom

Impulse propagation from atria to the ventricles occurs over the exactly anatomically determined structures. Sometimes, some accessory pathways may exist in the heart by which the impulse can pass much faster than through AV junctional area and AV node. The impulse, which is conducted via the accesory pathways by-passes AV node to activate the ventricle. Therefore, there is no physiological slowing of conduction in the AV node. The activation, which travels via the accessory pathway, reaches the ventricles earlier that the activation that travels via the normal pathway. Thierefore, this activation is called as premature (preexcitation syndrom) see fig. 3.51 page 246).

The existence of aberrant pathways was proved histologically. The terms, such as Kent, Mahaim, James bundles are not currently used. The accessory pathways are anatomically devided into:

1. Accesory atrioventricular junctions, which are situated away from the AV junction
2. Nodal – ventricular junctions, that join the atrioventricular myocardium
3. Fasciculo–ventricular junctions, that join the penetrating bundle with the ventricular myocardium
4. Atrio–fascicular junctions, that join the atria with the pearcing bundle.
5. Intranodal by-pass tract that is located in the AV node.

Its existence was not proven histologically, but electrophysiologically. Ventricular activation occurs via the normal pathway, only sometimes via the accessory pathways. When the activation reaches the ventricles via the accessory pathway there will be shortening of the PR interval and delta wave at the beginning of the QRS complex. The QRS duration is prolonged. The resulting activation is fusion of the normal activation and activation via the accessory pathway.

The term WPW (Wolf-Parkinson-White) syndrom is used in cases where there is preexcitation on the ECG with the presence of paroxysmal tachycardia. In atrial paroxysmal tachycardia the activation travels anterogradely via the accessory pathway (the by-pass tract). In WPW syndrome it is very important to reach the exact diagnosis and location of the accessory pathway. It is also important to know its role in the occurrence of arrhythmia and the probability of life-threatening situation during the fatal disturbances of the rhythm. Pharmacological treatment, pacing, or surgical treatment is usually required.

3.27.3.6 Junctional non–paroxysmal tachycardia

Junctional non-paroxysmal tachycardia can result from increased automacity or trigger activity in the AV-junctional area. Its occurrence is very common in setting of intoxication with digitalis, in association with posterior myocardial infarction, myocarditis, after catecholamines, in rheumatic fever, and in valvular cardiosurgery. The highest frequency is 150/min. The QRS complexes are similar as during sinus rhythm.

Figure 3.51: W–P–W syndrome

3.27.3.7 Sustained ventricular tachycardia

Sustained ventricular tachycardia is defined by lasting more than 30 seconds or causing haemodynamic collapse. It may occur in setting of structural heart diseases with altered myocardial architecture. **It occurs commonly in patients with chronic ischaemic heart disease in the setting of healed myocardial infarction**. It can be present in patient with cardiomyopathies without ischemic heart disease, metabolic disorders, toxic cardiac disorder, in prolonged QT interval. Sustained ventricular tachycardia is sometimes not secundary to any cardiovascular disorders. The sustained ventricular tachycardia is almost always associated with haemodynamic deterioration or with the development of acute myocardial ischaemia. The acute myocardial ischaemia can facilitate the development of ventricular fibrillation. Many cases of ventricular fibrillation are initiated as ventricular tachycardia. The typical findings in patients with ventricular tachycardia are shown as wide QRS complexes. Heart rate is over 100/min. The rhythm is usually regular. The atria have own rhythm or they can be activated in a retrograde direction. The onset of tachycardia is usually sudden and sometimes it might be insiduous. The configuration of QRS complexes can be monomorphic if the ectopic impulses origin from the same locus (see fig. 3.52 page 246).

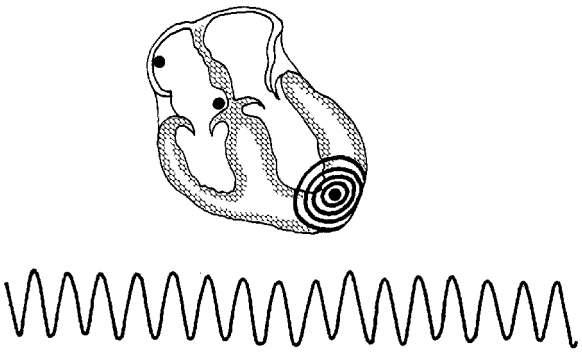

Figure 3.52: Ventricular tachycardia

The QRS morphology of ventricular tachycardia can be also polymorhic, with changing loci of the ectopic impulses. In case of a bidirectional tachycardia the QRS complexes have two configurations and they origin from two different loci. Paroxysmal ventricular tachycardia usually starts with ventricular premature complexes. It is very important to differentiate between the supraventricular tachycardia and the aberrant intraventricular conduction from ventricular tachycardia. Comparing between a rest ECG recording and ECG during the attack of ventricular tachycardia may be helpful. Meantime much attention is focused on detection of markers for life - threatening ventricular tachycardia. These markers are: wide QRS complexes that occur during the sinus rhythm, uniform QRS complexes from the precordium, and unexplainable electric axis deviations of ventricular axis. Using programmed stimulation, premature ventricular stimuli can initiate sustained ventricular tachycardia in 95 % patients, who present with this clinical arrhythmia. This type of stimulated tachycardia is identical with the spontaneous tachycardia. Occasionally, polymorphic ventricular tachycardia or ventricular fibrillation is provoked using programmed stimulation. It is possible that using of programmed stimulation or fast pacing we can also terminate the uniform ventricular tachycardia.

If the tachycardia persists after this, cardioversion is required. The ability to terminate tachycardia by the programmed stimulation provides us with the possibility of using antitachycardia pacemaker for long treatment of the ventricular tachycardia. The use of such pacemaker is not without risk.

Haemodynamic problems associated with ventric-

ular tachycardia depends on the heart rate and on the cardiovascular state. Patients with high frequent ventricular arrhytmias and myocardial dysfunction are at risk of syncope. The most serious problem represents the sustained ventricular tachycardia in post- myocardial infarction patients.

Patients with ventricular tachycardia occuring within the first six weeks after the acute myocardial infarction have a very poor prognosis. It this group of patients is about 85 % mortality within one year. Patients with sustained ventricular tachycardia have **a three fold higher risk of sudden death** than patients with myocardial infarction without ventricular tachycardia. The problem of ventricular tachycardia is very difficult. It is important to consider risk / benefit ratio in every treating procedure, because of some antiarrhythmic drugs instead of arrhytmia prevention can paradoxically provoked arrhythmia. Patients with ventricular tachycardia without underlying heart diseases or a haemodynamic symptoms should not be treated. Whereas patients with sustained ventricular tachycardia, even if they are without clinical symptoms, should be treated because this tachycardia is not without haemodynamic outcomes. Ventricular tachycardia have to be immediatedlly terminated in patients with haemodynamic deterioration, underlying organic heart disease, myocardial ischaemia, heart failure or CNS hypoperfusion. Progammed stimulation may be helpful for the best choice of antiarhytmic drug. Antitachycardial pacing should be employed, if the tachycardia is resistant to pharmacotherapy. Recently there have been used **automatic implantable cardioverter defibrillators (AICD)** in patients with non-stable ventricular tachycardia. Endocardial and peroperative mapping lead to development of new surgical techniques, which allow treatment of the ventricular arrhythmias. Mapping of activation spread allows to localise side of origin of tachycardia. It is common recently to provide ablation of the morphological arrhythmogenic substrate based on accurate mapping by specialized experts.

3.27.3.8 Torsades de pointes

Torsades de pointes is ventricular tachycardia that is characterized by polymorphic QRS complexes with different amplitude and duration. Tachycardia is not regular and the QRS duration of cardiac cycles is variable. QT interval is prolonged. This tachycardia occurs in association with hypokalaemia, hypomagnesaemia, therapz with chinidin, phenothiazides, and tricyclic antidepressant agents, during intracranial processes, and in complete AV block. This polymorphic tachycardia can be accompanied with syncope, ventricular fibrillation and sudden death.

After the termination of tachycardia, it is important to remove factors causing QT prolongation. The application of magnesium is also effective in suppressing the tachycardia. Polymorphic tachycardia with normal QT interval in patients with ischemic heart disease occur after very early ventricular premature complexes (R on T phenomenon). This form of tachycardia has completely. So here we are dealing with tachycardia of completely different substrate.

3.27.3.9 Accelerated ventricular rhythm

This tachycardia is marked as slow ventricular tachycardia. It is seen in acute myocardial infarction and mainly with thrombolitic procedures and treatment. It can also occur post cardiosurgery, rheumatic fever and cardiomyopathies it doesn't cause any marked haemodynamics problems, because its frequency ranges between 60 and 120/min.

3.27.4 Ventricular fibrillation and ventricular flutter

Ventricular fibrillation and ventricular flutter are cardiac rhythm disturbances, which lead to an immediate drop of blood pressure and a reduction of the cardiac output "to zero".

They both occur suddenly. There will be loss of consciousness, and if there is not immediate resuscitation, the patient dies. These arrhythmias usually occur in patients with ischaemic heart disease. They can also occur in WPW syndrome, after the use of antiarrhythmics for QT interval prolongation, or after an electric shock. Studies showed that 75 % of deaths occuring during Holter monitoring were caused by either ventricular fibrillation or flutter. Ventricular fibrillation or flutter are usually initiated with ventricular extrasystoles. Couplets of ventricular extrasystoles evoke ventricular tachycardia which may degenerate to ventricular fibrillation or flutter. In patients with acute myocardial infarction it is usually the acute ischaemia that leads to the ventricular extrasystole, which falls into a vulnerable period (R

on T extrasystole). See fig. 3.53 on page 248 and fig. 3.54 on page 248.

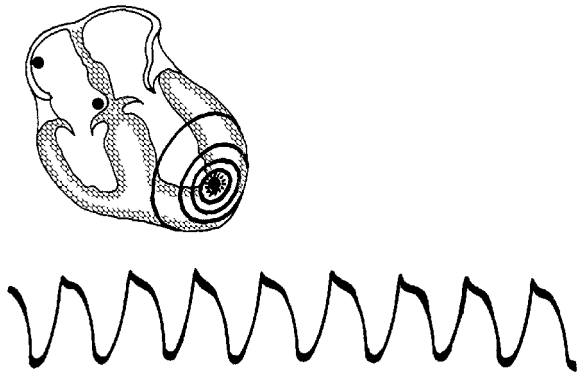

Figure 3.53: Ventricular flutter

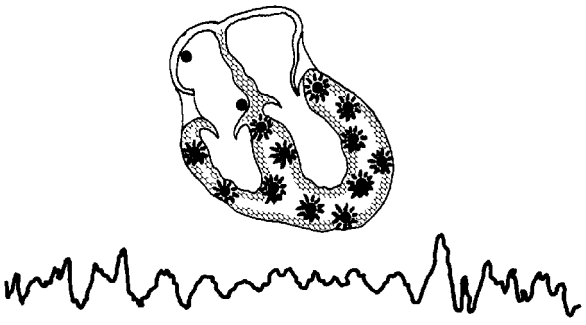

Figure 3.54: Ventricular fibrillation

3.27.5 Pathophysiological outcomes of antiarrhythmic treatment

In antiarrhythmic treatment it is necessary to consider all the pathological changes, which can cause arrhythmia, maintain it, or initiate it. The removal of these nonsuitable conditions is first aim. Then we have to consider the fact that antiarrhythmic drugs have proarrhytmogenic effect. The aim of treatment can be to terminate arrhythmia or to prevent its occurrence.

The choice of the proper antiarhythmics is quite difficult. Antiarrhythmics have some exactly defined characteristics, considering their effect on the electric process on the cellular membranes. Yet the doctor does not know how these processes take place on the patients cellular membranes. He only knows the result which is arrhythmia. That is why we recently appreciate testing the effects of antiarhytmics in programmed stimulation. For example: in induced ventricular tachycardia we found out that antiarrhythmics really lead to the prevention of ventricular tachycardia after stimulation. Yet programmed stimulation and intravascular catheterization are procedures which have some limits due to many risks.

Antiarrhythmics are agents that affect the depolarizing Na^{2+} and Ca^{2+} ion currents, the process of action potential, and cellular automacity. For example, if we depress the depolarizing currents, slowing in the impulse conduction will be resulted and so we can terminate arrhythmia by blocking the impulse conduction in these areas where they are already slow. Another type of antiarrhytmics lead to the prolongation of action potential and hence the prolongation of the refractory period. These effects are defined precisely on isolated myocytes. The effect of antiarrhytmic drug in vivo may differ. Besides the fact that the heart architecture is very complicated and we can not calculate with it in using of antiarrhythmics. Arrhythmia usually have a morphological substrate for a reentry mechanism, in which some cells or cardiac areas are incorporated. But antiarrhythmic drugs effect all cardiac cells equally. Therefore, it is necessary to reevaluate the proarrhythmogenic effects of antiarrhythmics in each individual patient.

3.28 Diseases of the venous system

Veins are capacity vessels which secure the venous return of blood to the heart. Changes in the venous return affect the minute heart volume. The entire