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АРИТМИИ У ДЕТЕЙ

CARDIAC ARRITHMIAS IN CHILDREN

Учебно-методическое пособие

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В пособии представлены современные данные о причинах и механизмах развития нарушений сердечного ритма у детей, а также клинические и электрокардиографические проявления аритмий. Отражены принципы оказания неотложной помощи, включая фармакологическую и электроимпульсную терапию в зависимости от варианта аритмии.

Предназначено для студентов 6-го курса факультета иностранных учащихся, изучающих педиатрию на английском языке

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Anatomy and physiology of the heart

The cardiovascular system exists to provide tissue perfusion, to ensure the body's cells are provided with oxygen and nutrients at the same time as removing metabolic wastes. The heart can be described as a hollow organ located centrally in the chest directly behind the sternum, between the lungs, and it is a component of the mediastinum. It is supported at its base (which is at the top) by the great vessels and it rests on the diaphragm with its apex (which is at the bottom) directed anteriorly and to the left. Two-thirds of the mass of the heart lies to the left of the body's midline (See Fig. 1).

The heart provides the impetus to drive blood flow throughout the body. The function of the heart is to circulate blood and therefore oxygen and nutrients to the tissues; the blood then removes metabolic wastes from the tissues. It is a four-chambered double-pump. The atria receive returning blood and direct it to the ventricles. The ventricles provide the impetus to circulate the blood through the systemic and pulmonary circulations. The systemic and pulmonary circulations are illustrated in Fig. 2.

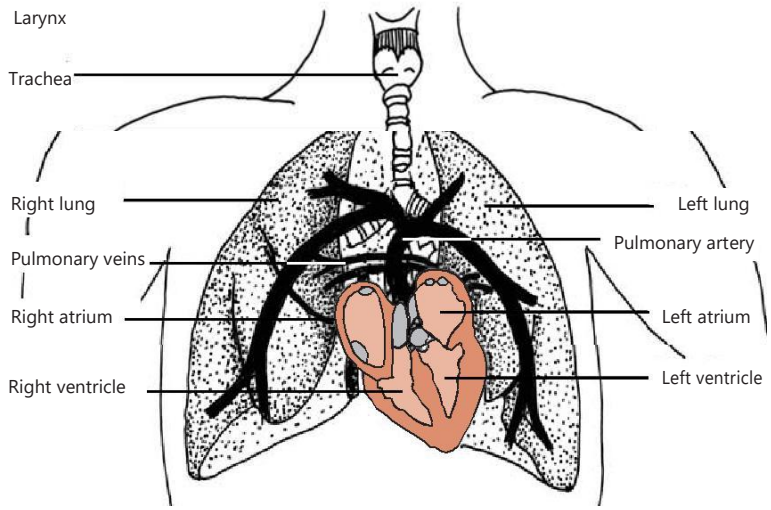

Fig. 1. *The heart in situ*

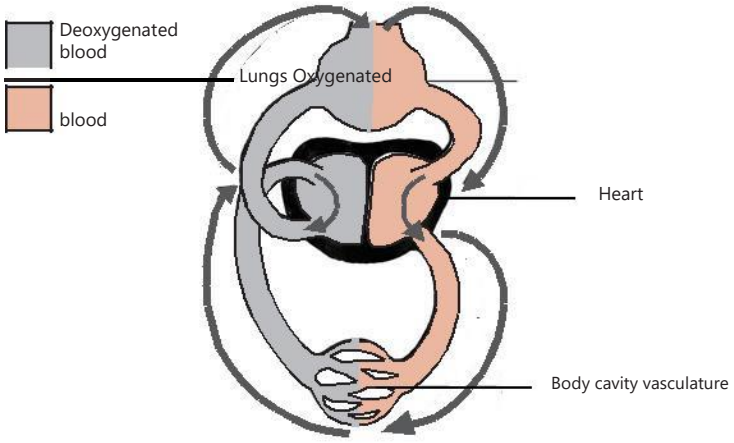

Fig. 2 *The systemic and pulmonary circulations*

The two sides of the heart are divided by the atrial and ventricular septa. The atrial septum is largely muscular tissue, and the ventricular septum is divided into two distinct parts – the membranous septum (the small, superior portion that borders the right atrium) and the larger muscular portion (which forms the true division between the left and right ventricles) as well as forming the wall of the left ventricle and functioning as part of the left ventricle.

The heart consists of three distinct layers: the epicardium, myocardium and endocardium. Epicardium -is the thin, transparent layer of the heart wall. It also forms the visceral layer of the pericardium, which is made up of two sacks, an outer one consisting of fibrous tissue, and an inner one consisting of mucous membrane.

Myocardium - is the middle layer and it is made of specialized cardiac muscle cells called myocytes. Cardiac muscle fibres (Fig. 3) are involuntary, striated and branched, arranged as interlacing bundles of fibres. They are responsible for cardiac contraction. Each cell has branches that lie in close relation to the next cell, forming junctions known as intercalated discs. These fibres facilitate the passage of nervous impulses from one cell to the next, therefore each individual cell does not need its own nerve supply.

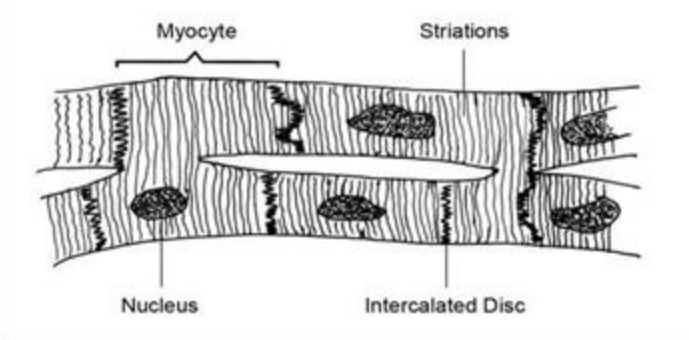

Fig. 3 *Cardiac muscle fibres*

Endocardium - is the inner layer. It is a membrane consisting of flattened squamous epithelial cells lining the inside of the myocardium and covering the heart valves and tendons.

The blood flow through the heart is described below.

1. Superior and inferior venae caves return deoxygenated blood from the systemic circulation to the right atrium from all areas of the body.

2. Blood passes from the right atrium to the right ventricle via the tricuspid valve.

3. Blood passes into the pulmonary artery via the pulmonary valve to the lungs where it picks up oxygen and releases carbon dioxide.

4. Oxygenated blood returns to left side of the heart via the four pulmonary veins.

5. Blood from the left atrium passes into the left ventricle via the mitral valve.

6. Blood passes from the left ventricle into the aorta via the aortic valve to be distributed around the systemic circulation.

Conduction system of the heart

The heart's conduction system relies on the sinoatrial node (the pacemaker of the heart) and the atrioventricular node, which are small groups of specialized neuromuscular cells in the myocardium that initiate and then conduct electrical impulses over the heart muscle, causing it to contract. These specialized cells have the ability to discharge electrical impulses automatically that is without the influence of a nerve supply. This is known as automaticity. In other words, even if there is no stimulation from the central nervous system, the heart will continue to beat automatically. However, the system can be stimulated or depressed by nerve impulses initiated from the brain. The lower down the conducting system the impulse is triggered, the slower the rate will be.

There are two nodes that are central to the conduction process:

First is the sinoatrial (SA) node. This small mass of specialized neuromuscular cells is located in the wall of the right atrium near the opening of the superior vena cava. The SA node is often described as the pacemaker of the heart because each heart beat is normally triggered by the impulses initiated by the SA node. The SA node normally discharges at 60–100 beats per minute (b.p.m.).

The second is the atrioventricular (AV) node. This mass of specialized neuromuscular cells is situated in the wall of the atrial septum near the AV valves. Normally the AV node is stimulated by the wave of electrical impulses initiated by the SA node, sweeping over the atrial myocardium via internodal tracts or pathways. The AV node normally discharges at 40–60 b.p.m.

However, it is also capable of initiating electrical impulses if there is no stimulation from the SA node or the central nervous system. The bundle of His (also called the AV bundle) consists of a mass of specialized neuromuscular fibers originating from the AV node and passing downwards in the septum that separates the left and right ventricles. This bundle of fibres then divides into 2 branches, one feeding each ventricle – these are the left and right bundle branches. Within the myocardium of the ventricles, the branches further divide into a network of fine filaments called the Purkinje fibres (or fibres of Purkinje). The bundle of His and the Purkinje fibres (together referred to as the His–Purkinje fibres) convey electrical impulses from the AV node to the myocardium of the ventricles. The His–Purkinje fibres normally discharge at 20–40 b.p.m.

The electrical impulses initiated by the SA node stimulate the atrial myocardium to contract. This first wave of impulses and contraction stimulate the AV node to continue the wave of contraction to the apex of the heart via the His–Purkinje fibres and then upward over the myocardium. In this way, the ventricular wave of contraction begins at the apex of the heart (at the bottom) and blood is forced upward into the pulmonary artery and aorta to leave the heart at its base (at the top). The function of the bundle branches is to conduct unified electrical impulses

throughout the ventricles, thus causing brief, powerful and unified contraction of the ventricles (See fig.4).

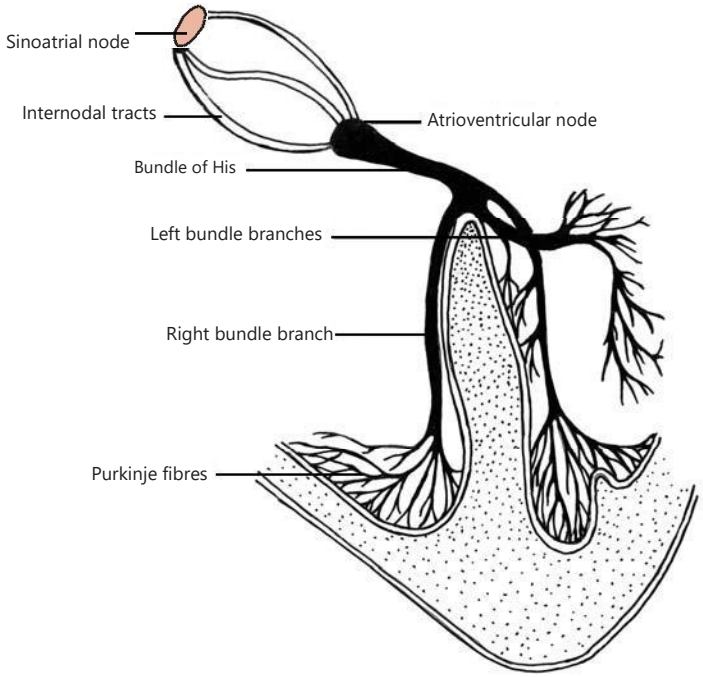

Fig. 4. *The conduction system of the heart*

Electrophysiology of depolarisation and repolarisation

Without an electrical stimulus, the heart would not beat. The heart has specialized cells with 4 characteristics that ensure the heart continues to pump with or without stimulation from the central nervous system, these are:

1. Automaticity (spontaneous initiation of an electrical impulse).
2. Excitability (the cells respond to an electrical stimulus as a result of electrolyte shifts).
3. Conductivity (the electrical impulse is transmitted from one cell to the next).
4. Contractility (the cell contracts as a result of an electrical stimulus).

There are two types of cardiac muscle cells - contractile and non-contractile. The contractile cells make up 99% of all cardiac muscle cells and provide the powerful contraction that propels blood around the body. The conducting system, which initiates and controls those contractions, is made up of non-contractile cells. The human body contains various electrolytes in solution through which electrical currents will flow. In the heart, each cardiac cell contains such electrolyte fluids and is also surrounded by them. The main electrolytes responsible for electrical activity within the heart are sodium (Na+), potassium (K+) and calcium (Ca++). In resting (or polarised) cardiac cells, the inside of the cell is relatively negatively charged in comparison to

the outside of the cell, which is positively charged; this creates what is known as the resting potential.

When myocardial cells are stimulated by an electrical impulse, a change takes place in the cell membrane's permeability and various electrolytes move across the cell membrane by diffusion or active transport so that the inside of the cell becomes positively charged.

The process by which the inside of a cell becomes more positive in relation to the outside is called depolarisation and it is this movement of electrolytes that generates the electrical flow, which is picked up by the ECG. This is known as the action potential (Fig. 5).

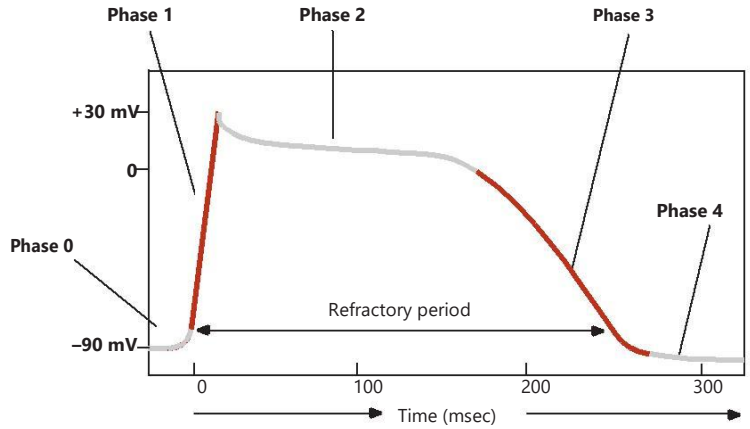

Fig. 5 *The action potential*

There are five phases of the action potential.

Phase 0—Rapid depolarization. This happens because positively charged Na+ and Ca++ move into the cell. Na+ moves in rapidly and Ca++ more slowly, through slow calcium channels. The inside of the cell therefore becomes more positively charged.

Phase 1—Early repolarization. The sodium channels close so no more Na+ can enter the cell. Therefore the inside of the cell cannot become any more positively charged.

Phase 2—Plateau phase. Ca++ continues to flow in slowly, whilst positively charged K+ starts to flow out of the cell, so the overall charge starts to become more negative.

Phase 3—Rapid repolarization. The calcium channels close, so no more Ca++ enters the cell and K+ flows out rapidly, so the inside of the cell becomes more negative more quickly.

Phase 4—Resting phase. The Na+, K+ and Ca++ return to their original state.

A depolarised cell is electrically negative on the outside compared with any of its neighbouring non-stimulated cells. A potential difference therefore exists between the cells and current flows between them until they have all been depolarised. When the cell is depolarised it becomes excited and ready for action. It also stimulates the cell or cells next to it so there is a

smooth wave of depolarisation all the way down the conducting tissue of the heart. The refractory period - is how long it takes for an excitable membrane that has returned to its resting state following excitation to be ready for a second stimulus. During the so-called relative refractory period, it is possible for a very strong electrical impulse to depolarise the cell early. During the absolute refractory period, the cells cannot be stimulated at all.

A single heart beat.

Each beat of the heart is initiated by the SA node. On the ECG trace it is made up of a normal P wave, a QRS complex and a T wave (Fig. 6).

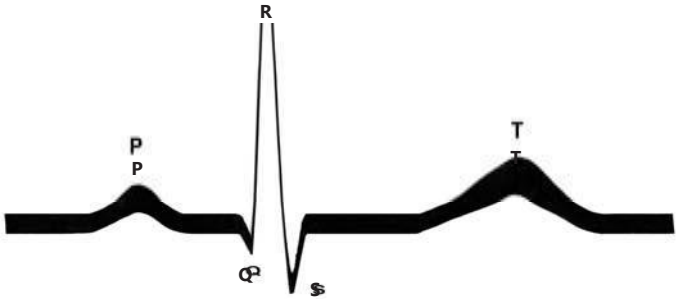

Fig. 6 *One heart beat*

The rhythm should be regular with a rate of approximately 72 beats (in adult) per minute (b.p.m.). When this is the case, the heart is said to be in sinus rhythm, which is the normal rhythm of the heart (fig. 7). If there is any deviation from this sinus rhythm, an arrhythmia is present. You will not be able to recognise an arrhythmia if you cannot first recognise normal sinus rhythm, as this is the rhythm against which all other rhythms are compared.

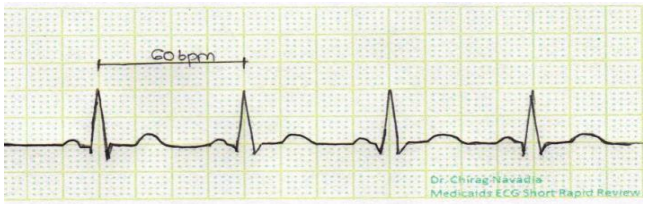

Fig. 7 *Sinus rhytm*

Waveforms.

There are 4 stages within each heart beat and each is represented in terms of waves, namely the P, Q, R, S and T waves.

Stage 1 – Sinoatrial node impulse. The SA node fires an electrical impulse, which spreads over the atria via the internodal tracts, resulting in atrial depolarisation. This is denoted by the P wave. Depolarisation causes the atria to contract. The P wave is 0.06–0.12 seconds in duration.

Stage 2 – Atrial repolarization. There is atrial repolarisation (i.e. the atria relax). However, the wave of repolarisation is usually not seen as it is hidden by the more powerful QRS complex.

Stage 3 – Ventricular depolarization. Ventricular depolarisation occurs next as the wave of depolarization passes down the bundle of His, the right and left bundle branches and the Purkinje fibres. It is represented by the QRS complex. Ventricular depolarisation causes the ventricles to contract. The QRS complex should last for less than 0.12 seconds and is measured from the beginning of the Q wave to the end of the S wave. Remember, all three waves (Q, R and S) may not be present because of the speed of contraction, but the heart's activity can still be normal.

Stage 4 – Ventricular repolarization. Ventricular repolarisation is represented by the T wave as the myocardial cells return to their resting charge.

Intervals and segments of ECG

Intervals contain waves. Segments are the lines between the waves where there is no electrical activity and the trace does not deflect either above or below the baseline (i.e. they are isoelectric). The intervals and segments in one heart beat are shown in Fig. 8.

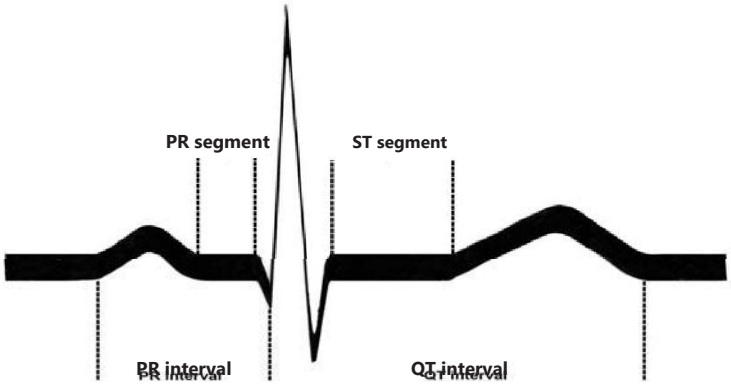

Fig. 8. *Intervals and segments in one heart beat*

PR interval

The PR interval is the time from the beginning of the P wave to the beginning of the QRS complex. It indicates the time taken for the impulse to pass from the SA node to the ventricle. It therefore reflects atrial depolarisation and is between 0.12 and 0.2 seconds in duration. A long PR interval may indicate that there is a conduction delay through the atria or AV node. A short PR interval suggests the impulse originated away from the SA node.

PR segment

The PR segment indicates the time between the end of the P wave and the beginning of the QRS complex.

ST segment

The ST segment indicates the time between the end of the S wave and the beginning of the T wave and represents the beginning of ventricular repolarisation. The beginning of the ST

segment is known as the J point. The ST segment should be isoelectric and if it is raised or depressed it may indicate the presence of myocardial ischaemia.

QT interval

The QT interval represents the time from the beginning of the Q wave to the end of the T wave and therefore reflects the time taken for the ventricles to depolarise and repolarise. It usually lasts for 0.36–0.44 seconds.

Cardiac arrhythmias are very common and produce symptoms such as dizziness, palpitations and syncope. They are generally benign although, in a critically ill patient they can create further complications for the patient with an already compromised cardiovascular system. In addition to these relatively benign arrhythmias, there are arrhythmias that are dangerous for the patient if they are left untreated and there are arrhythmias that bring about sudden death! The cardiac monitor can give you prior warning of problems that may lie ahead, many of which can be resolved or abated to some degree.

Classification of arrhythmias

Cardiac arrhythmias are generally classified by their site of origin, for example SA node (sinus rhythms), atrial (supraventricular), junctional or ventricular arrhythmias. These arrhythmias result from a disturbance in impulse formation. However, heart blocks result from a disturbance in impulse conduction

Cardiac arrhythmias may arise for several reasons:

1. From abnormal electrical conduction within the heart.
2. Through re-entry circuits whereby an impulse travels where it has already been.
3. From enhanced automaticity (an irritable focus within the conducting system firing an impulse when it shouldn’t).

Sinus node arrhythmias.

In sinus arrhythmia the heart rate stays within normal limits but it is irregular. The rhythm generally corresponds to the respiratory cycle, increasing during inspiration and decreasing during expiration, because of the effect breathing has on the vagus nerve (see Fig. 9).

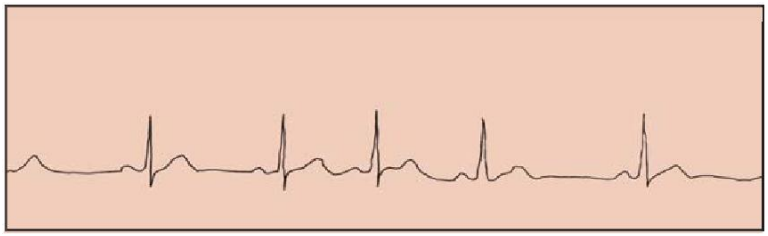

Fig. 9. *Sinus arrhythmia*

The P wave, QRS complex and T wave are normal but the RR intervals are irregular. It is usually insignificant and requires no treatment but it may be indicative of a more serious problem. ***Causes of sinus arrhythmia:*** Sick sinus syndrome, Digoxin toxicity, Myocardial infarction, Raised intracranial pressure

Management. Sinus arrhythmia often occurs naturally in children and athletes. Unless the patient is symptomatic, it is no cause for concern and requires no treatment. If sinus arrhythmia has an underlying cause (such as those listed above) it should be closely monitored and the cause dealt with swiftly.

Sinus bradycardia

Sinus bradycardia (Fig. 10) has a regular rhythm and a rate of less than 60 beats per minute. The P wave, QRS complex and T wave are normal.

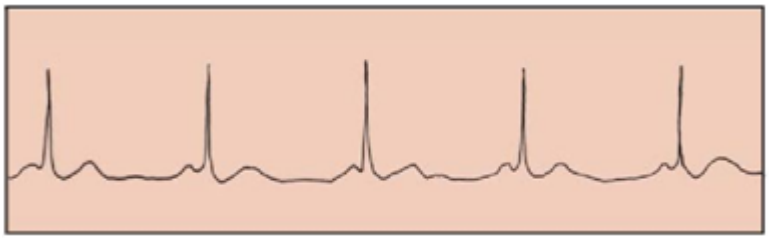

Fig. 10. *Sinus bradycardia.*

It often occurs in athletes and during relaxation and sleep when the body's metabolic demands are reduced. There is no cause for concern. However, if the patient has an underlying condition, a decrease in cardiac output may occur – resulting in hypotension – and the patient may be predisposed to further serious arrhythmias. A bradycardia of less than 45 b.p.m. is not well tolerated and will produce symptoms of reduced cardiac output.

Causes: Increased vagal stimulation caused e.g. by sleep, vomiting, carotid sinus massage, inferior, myocardial infarction, pneumothorax, Raised intracranial pressure, Hypothermia, hypovolaemia, Hypoxia, Acidosis, Electrolyte disturbances, Some drugs (e.g. digoxin, calcium-channel blockers, beta-blockers).

Management.

For as long as the patient is asymptomatic, no treatment is necessary. However, if the patient is compromised by the bradycardia, swift treatment is necessary. A heart rate of less than 60 b.p.m. may cause the patient to collapse or suffer symptoms of inadequate perfusion, particularly if there is a sudden bradycardia – in which case the adult bradycardia Resuscitation algorithm should be used. Assess airway, breathing and circulation (ABC), ensure the patient's airway is clear, and give them oxygen and respiratory assistance if required. Transcutaneous or transvenous pacing may be required with drug intervention to support the patient's circulation prior to pacing (e.g. using atropine and/or inotropic agents).

Sinus tachycardia

Sinus tachycardia has a regular rhythm. The P waves, QRS complexes and T waves are normal and regular with a rate of 100–160 b.p.m.(fig. 11)

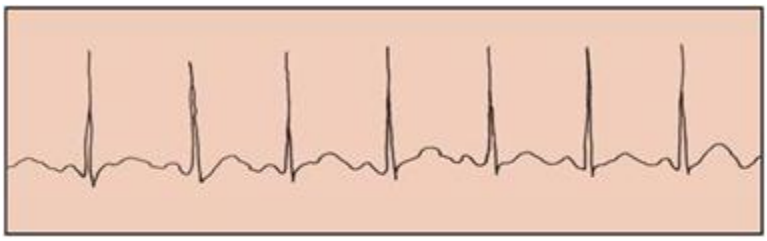

Fig. 11. *Sinus tachycardia.*

In sustained sinus tachycardia, the P waves may increase in amplitude or be superimposed on the preceding T wave, sometimes making identification difficult.

Causes: Strenuous exercise, Anxiety, Haemorrhage, Hypovolaemia, Pain, Acute myocardial infarction, Initial stage of cardiogenic shock.

Sinus tachycardia is of little or no significance when there is no underlying cause or it occurs as a response to strenuous exercise or a high-anxiety state. In the critically ill patient, it may be caused by pain and/or hypovolaemia for which treatment should be given swiftly. Sinus tachycardia can have serious consequences as it increases cardiac work and therefore oxygen consumption and can therefore lead to heart failure. In patients with underlying heart disease, it can be an ominous prognostic sign.

Management.

If there is no underlying cause, the tachycardia is transient, and the patient is asymptomatic, no treatment is necessary – but the patient should be observed. With a sustained sinus tachycardia, however, even if the patient is asymptomatic, the cause should be determined and treated. Sinus tachycardia can severely reduce cardiac output because of the reduction in ventricular filling time, so there is less blood in the ventricles to pump into the circulation. In addition to this, the heart has to work harder as it tries to maintain the circulation and therefore it needs more oxygen (which it cannot get because of the reduction in cardiac output) which is why this chain of events can lead to heart failure. Symptoms of reduced cardiac output are produced and treatment should be instigated to slow the heart rate and increase the power of ventricular contraction. Here are the most common treatments:

1. For tachycardia caused by haemorrhage or hypovolaemia – stop the bleeding and replace fluid.
2. For tachycardia with another cause, give beta-adrenergic blockers and/or calcium-channel blockers.

In the critically ill patient it may also be necessary to support the circulation with inotropic agents to increase cardiac contractility, which will increase blood pressure and improve oxygen delivery to the tissues.

Atrial (supraventricular) arrhythmias.

Premature atrial contractions (PACs)

Premature atrial contractions are also known as atrial premature beats (APBs) or atrial ectopic beats. They are simply beats that occur sooner than the expected beat. They occur because an irritable focus outside of the SA node fires, causing the heart to contract. It takes a little time for the SA node to reset itself so there is a slight pause before the next beat, although this is not long enough to be a full compensatory pause. PACs occur in patients with underlying heart disease but they also occur quite normally and cause no problems in people who are disease free (fig. 12).

Causes: Smoking, Alcohol consumption, Exhaustion, Caffeine consumption, Pyrexia, Infection, Coronary heart disease, Valves disease, Lung disease, Hypoxia , Respiratory failure

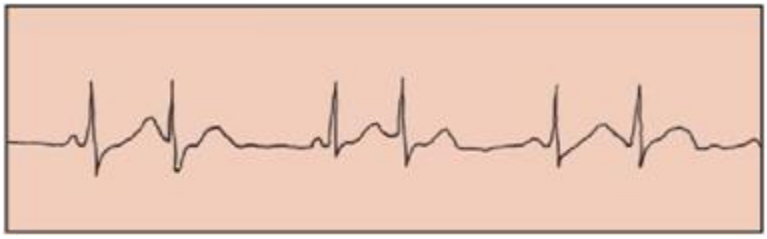

Fig. 12. *Premature atrial contractions.*

In patients with an underlying cardiac problem they can lead to further arrhythmias or heart failure or to symptoms of a reduced cardiac output (if the PACs are frequent). They therefore need to be closely monitored and treatment should be instigated where necessary.

Management.

If the patient has no cardiac disease and is asymptomatic, treatment is rarely necessary. If the PACs are caused by a removable cause such as caffeine, alcohol or other such irritants, then the patient should be advised to reduce intake of such substances. In patients who have an underlying disease with symptoms resuling from the PACs, drug treatment may be commenced to increase the atrial refractory period (e.g. digoxin).

Atrial flutter.

Atrial flutter is a form of supraventricular tachycardia (fig. 13). It is present when there is an irritable focus within the atria firing at a rate of approximately 300 b.p.m. Not all of these impulses pass through the AV node but when one does, the ventricles contract. So the atrial rate is much higher than the ventricular rate. P waves are not distinguishable and it is recognised by a typical so-called saw-toothed appearance, caused by the flutter waves. The flutter originates from a single focus within the atria. If it takes two impulses (flutter waves) to stimulate ventricular contraction; the conduction ratio is 2:1 three flutter waves; 3:1 four flutter waves; 4:1

and so on. A 2:1 block is most common and you will see an atrial rate of approximately 300 b.p.m. with a ventricular rate of approximately 150 b.p.m.

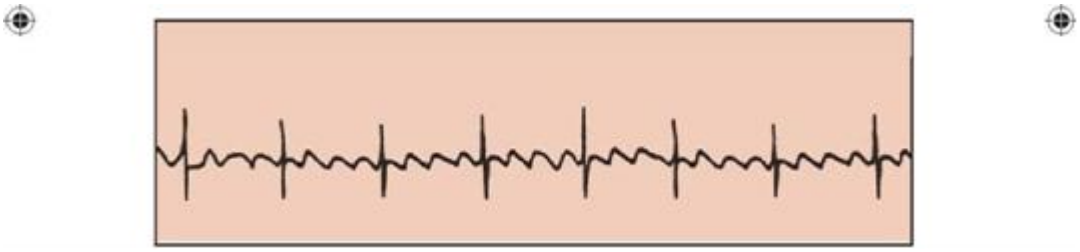

Fig. 13. *Atrial flutter.*

Causes: Hypoxia, Acute myocardial infarction, Cardiac surgery, Valvular disease, Chronic obstructive pulmonary disease, Infection, Hypovolaemia

Management.

If the ventricular rate is normal, the patient may be asymptomatic, but if the ventricular rate is badly affected and is too slow or too high, symptoms of reduced cardiac output and perfusion disturbances will be seen. The goals of treatment are:

1. To control the ventricular rate.
2. To restore sinus rhythm.
3. To prevent recurrences of atrial flutter.

If the patient is unstable, synchronous direct-current (DC) cardioversion is commonly the initial treatment of choice. An antiarrhythmic agent, beta-blocker or calcium-channel blocker may be used if the symptoms are less severe. If the heart rate is high, it may be necessary to terminate the arrhythmia by the use of a temporary pacing wire.

Atrial fibrillation

Atrial fibrillation is a very common arrhythmia. It occurs when there are many foci in the atria all firing through re-entry circuits, in a chaotic fashion. As a result, the atria lose their kick and cardiac output can be reduced by up to 25%. There is no electrical synchronisation within the atria and therefore no P waves are present. The foci in the atria may fire at up to 600 times each minute and thus they quiver instead of contracting. Instead of P waves, there are f waves, which show as an erratic baseline waveform. The rate is very irregular and may be very rapid.

Causes: Smoking, Caffeine, Alcohol, Anxiety and stress, Hypoxia, Hypotension, Electrolyte disturbances, Acute myocardial infarction, Pulmonary embolism, Chronic obstructive pulmonary disease.

Atrial fibrillation can be a sustained arrhythmia or can occur paroxismaly. The ventricular rate can vary. It depends how many of the impulses pass through the AV node to stimulate

ventricular contraction. The QRS complexes are described as irregularly irregular and T waves are unidentifiable (fig. 14).

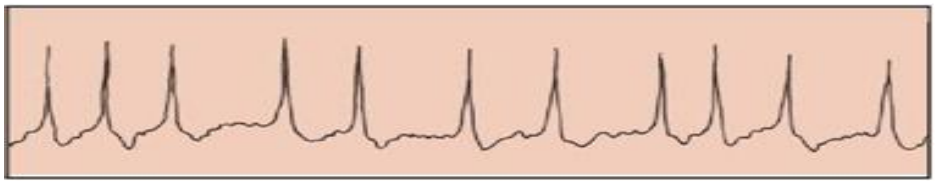

Fig. 14. *Atrial fibrillation.*

Management.

If the patient is unstable and the arrhythmia has been present for less than 48 hours, the treatment of choice is electrical cardioversion. If the ventricular rate is very rapid, carotid sinus massage may be useful for slowing the heart rate. If cardioversion does not convert the heart back to sinus rhythm, or if AF is persistent, the decision to perform pharmacologic conversion may be made. In this case, an intravenous antiarrhythmic agent will be used. In the absence of structural heart disease (coronary artery disease or left ventricular dysfunction), a class Ic drug such as flecainide or propafenone is used. In the presence of structural heart disease, amiodarone should be the drug of choice. Anticoagulation is also necessary because when the atria are fibrillating they do not empty correctly so there is an increased chance of clot formation.

Atrial tachycardia

Atrial tachycardia is often referred to as supraventricular tachycardia (SVT). It is indeed a type of supraventricular tachycardia but so are all types of tachycardia that originate above the ventricles (i.e. they are supraventricular). Atrial tachycardia is caused by an abnormal focus in the atria which fires at a rate in excess of 150 b.p.m. (and can reach 250 b.p.m.). It is often associated with stress and/or stimulants but usually manifests in digoxin toxicity and primary or secondary heart conditions and it can lead to more serious arrhythmias. P waves are often superimposed on the T waves of the preceding beats and thus the T wave may appear distorted. The P waves are usually followed by a normal QRS complex (fig. 15).

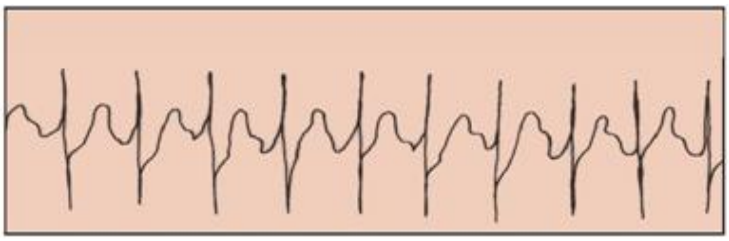

Fig. 15. *Atrial tachycardia.*

The rhythm is often regular but it may be irregular if there is an AV block present and it can occur as paroxysmal atrial tachycardia. Multifocal atrial tachycardia (MAT) occurs when there are several foci that intermittently fire.

Management.

If there is no underlying cause, the tachycardia is transient and the patient is asymptomatic, no treatment is necessary – but the patient should be observed. However, with a sustained atrial tachycardia, even if the patient is asymptomatic, the cause should be determined and treated.

Causes: Smoking, Caffeine, Stress, Primary or secondary cardiac disease, Digoxin toxicity (most common cause).

Sustained atrial tachycardia, as with sinus tachycardia, can severely reduce cardiac output because of the reduction in ventricular filling time (so there is less blood in the ventricles to pump into the circulation). In addition to this, the heart is working harder in an attempt to maintain the circulation and therefore needs more oxygen, which it cannot get because of the reduction in cardiac output – this chain of events can lead to heart failure. Symptoms of reduced cardiac output are produced and treatment should be instigated to slow the heart rate and increase the power of ventricular contraction. Depending on how unstable the patient is as a result of the arrhythmia, the condition is treated with adenosine or amiodarone or electrical cardioversion. Carotid sinus massage can sometimes be successful in slowing the heart rate in the first instance. Carotid sinus massage can be used either to diagnose or to treat atrial tachycardia but it is to be avoided in older patients. When the carotid sinus is massaged, the vagus nerve is stimulated and therefore the firing of the SA node is inhibited, slowing the heart rate down. However, a number of complications are associated with carotid sinus massage – bradycardia, asystole, reduced blood pressure as a result of vasodilation, ventricular arrhythmias and cerebral vascular accident. Therefore, it should only be carried out by experienced healthcare professionals when resuscitation equipment is to hand.

ECG of patient with Wolff–Parkinson–White (WPW) with the accessory pathway located in the inferoseptal heart wall and atrial fibrillation episode and supraventricular paroxysmal tachycardia in the same patient are presented on Fig.16 and 17. Patient with WPW syndrome that presents a very fast AF triggering a ventricular fibrillation (arrow).

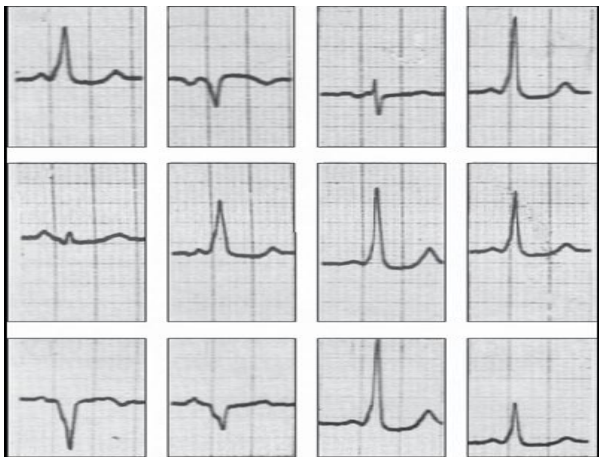

Fig. 16. *Wolff–Parkinson–White (WPW) patient with the accessory pathway located in the inferoseptal heart wall (Type III WPW).*

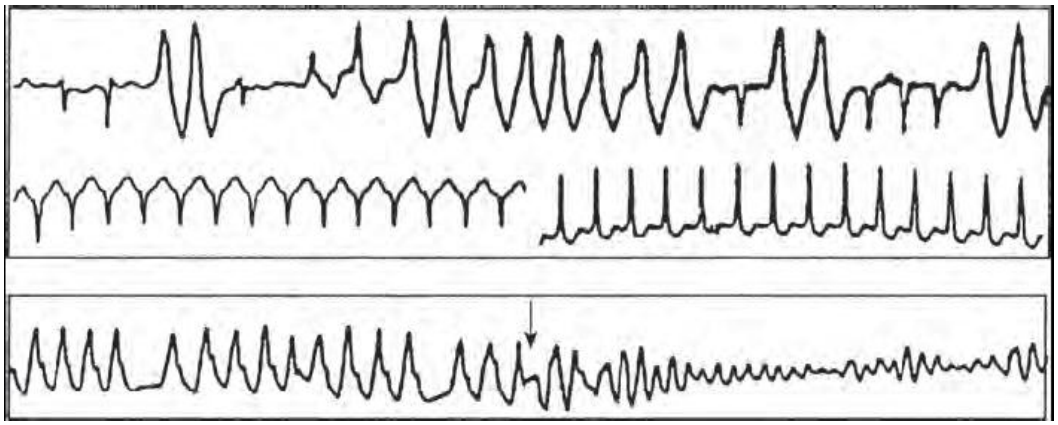

Fig. 17. *Atrial fibrillation episode and supraventricular paroxysmal tachycardia in patient with WPW syndrome.*

Arrhythmias arising at the AV junction.

In critical and acute care areas, the most common arrhythmias seen (that arise at the AV junction, i.e. around the area of the AV node and bundle of His) are junctional escape rhythm and AV block.

Junctional escape rhythms.

If the SA node fails to initiate impulses and another focus within the atria does not take over as pacemaker, the AV node may take over. This causes abnormal conduction as the impulse spreads upwards over the atria (instead of downwards). The rate is slower than normal because, as mentioned previously, the lower down the conducting system the impulses are generated, the slower the rate will be. When monitoring in lead II, a junctional escape rhythm can be determined by a rate of 60 b.p.m. or less, with inverted P waves. The P waves may be inverted because the positive deflection denotes the downward wave of depolarisation; as the impulse originates in the AV node the wave of depolarisation passes in an upward direction over the atria and will therefore be seen as a negative deflection on the ECG (fig. 18).

However, P waves may be absent if atrial and ventricular depolarisation occurs simultaneously because the P wave will be hidden within the more powerful QRS complex. It is possible to see inverted P waves when the rhythm arises low in the atria; inverted P waves do not automatically demonstrate nodal rhythm.

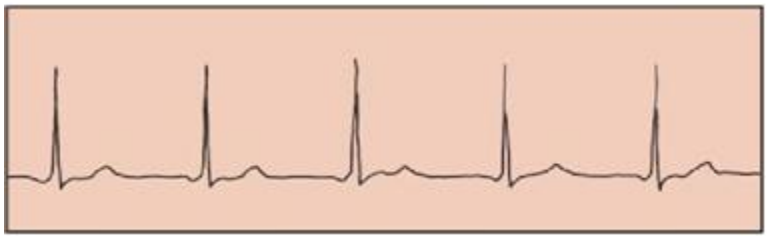

Fig. 18. *Junctional escape rhythms.*

You can determine this by considering the PR interval. If the PR interval is less than 0.12 seconds long, it is nodal rhythm, but if there is a normal PR interval, the rhythm originated in the atria and you are looking at something other than junctional escape rhythm.

Causes: Hypoxia, Digoxin toxicity, Cardiac disease, Sick sinus syndrome, Myocardial infarction, Cardiac surgery, Drugs that may cause bradycardia, Complete heart block.

Management.

If the patient is asymptomatic, generally the arrhythmia itself is not treated but, if necessary, the underlying cause is. Junctional escape rhythm, as the name suggests, serves as an 'escape' mechanism to maintain the heart rate during periods of bradycardia or asystole and it should not be suppressed. In patients with complete heart block, or symptomatic sick sinus syndrome, a permanent pacemaker may be needed. An anticholinergic agent such as atropine may be required if symptomatic bradycardia is present.

N.B. An accelerated junctional escape rhythm will result in tachycardia.

First-degree AV block

First-degree AV (or heart) block is generally not dangerous in itself and the arrhythmia is not treated as it does not usually affect cardiac output. At first glance it will appear to be normal sinus rhythm, but on closer examination, it is recognised by an extended PR interval of greater than 0.2 seconds. This means there is a delay at the AV junction before the impulse passes into the bundle of His. The P wave will be followed by a normal QRS complex and T wave and the rate will not be affected. First-degree AV block may occur normally in a healthy patient (fig.19).

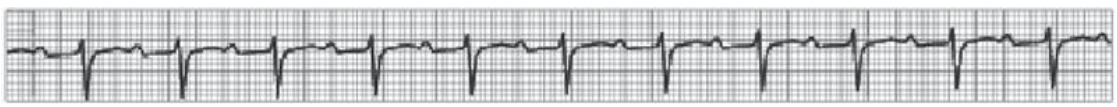

Fig. 19. *First-degree AV block*

Management.

If the patient is asymptomatic, the arrhythmia is not treated. However, a delay in conduction means there is a problem in the conducting system and therefore should be monitored. The underlying cause should be treated, if necessary, because first-degree block can progress to more serious forms of heart block.

Causes: Myocardial infarction, Degenerative cardiac disease, Most commonly caused by drugs that depress AV conduction: (e.g. beta-blockers, digoxin, calcium-channel blockers)

Second-degree AV block

There are two types of second-degree heart block known as Mobitz type I and Mobitz type II. Mobitz type I is also commonly known as Wenckebach phenomenon (because it was discovered by a man called Wenckebach).

Mobitz type I (Wenckebach phenomenon).

This is a cyclical rhythm recognised by a progressively increasing PR interval. It continues to increase until eventually the impulse is blocked and cannot stimulate ventricular depolarisation so the next QRS complex is dropped. The sequence then begins again (fig. 20).

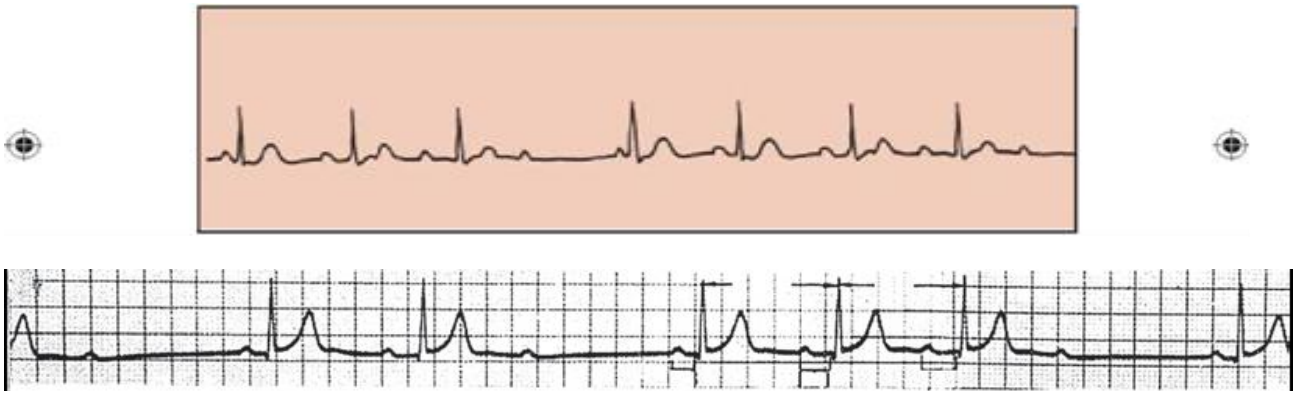

Fig. 20. *Second-degree AV block – Mobitz type I*

Management.

If the patient is asymptomatic, the arrhythmia is not treated as it is usually transient. However, as with first-degree AV block, a delay in conduction means there is a problem in the conducting system and this should therefore be monitored.

Causes: Myocardial infarction, Degenerative cardiac disease, Drugs that depress AV conduction (most commonly) such as beta-blockers, digoxin and calcium-channel blockers.

The underlying cause should be treated, if necessary, because Mobitz type I block can progress to more serious forms of heart block. If the rhythm is prolonged and causes a reduction

in cardiac output, it may be necessary to give atropine. A temporary transvenous pacing wire may be required to support the patient until the arrhythmia has resolved.

Mobitz type II.

A Mobitz type II second-degree AV block is more serious than a type I block but is less common. This arrhythmia usually requires intervention because it frequently progresses to high-grade block or complete heart block. It is easily recognised as frequent impulses fail to pass through the AV node and are seen as missed beats (no QRS complex) or it takes two P waves to stimulate a QRS complex. This is known as a 2:1 block, meaning every other QRS complex is dropped (fig. 21).

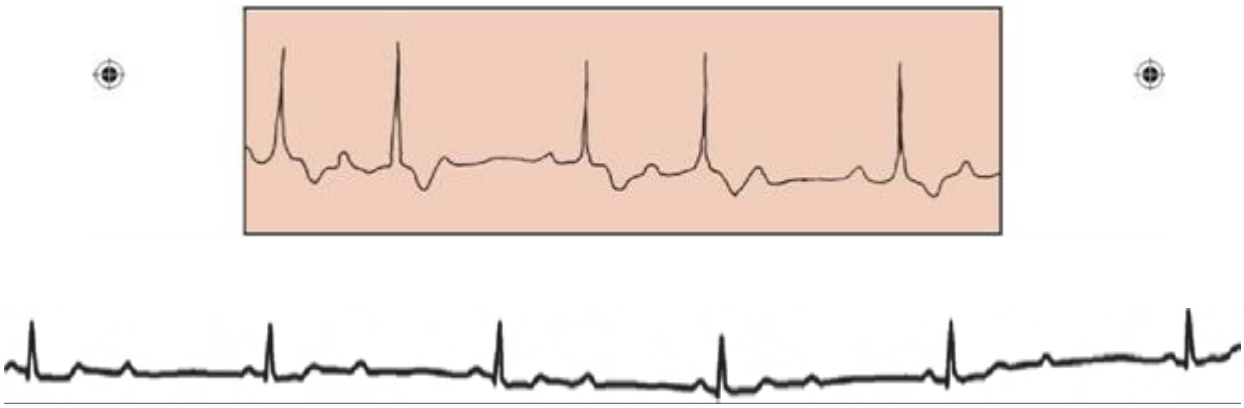

Fig. 21. *Second-degree AV block – Mobitz type II*

A high-grade AV block exists if there is a 3:1 or higher ratio of P waves to QRS complexes. This will result in a drastic reduction in cardiac output and rapidly progresses to complete heart block.

Causes: Myocardial infarction, Degenerative heart disease, Digoxin toxicity

Management.

Transcutaneous pacing or a temporary transvenous pacing wire is indicated for all patients with Mobitz type II block, including asymptomatic patients, as patients with Mobitz II second-degree AV block usually progress to complete heart block. An anticholinergic drug such as atropine may also be required.

Third-degree AV block.

Third-degree heart block, also known as complete heart block, is potentially life-threatening and requires immediate intervention. The atria depolarise normally but the impulse cannot pass through the AV node, so no impulses are passed from the atria to the ventricles. A focus in the ventricles may take over but the ventricles beat at a slower rate than the atria and the atria and ventricles depolarise and contract independently of one another. A focus in the His–

Purkinje system may take over as the pacemaker, causing ventricular depolarisation. The ventricular rate will be slow and the patient will immediately suffer symptoms of a reduced cardiac output (fig. 22).

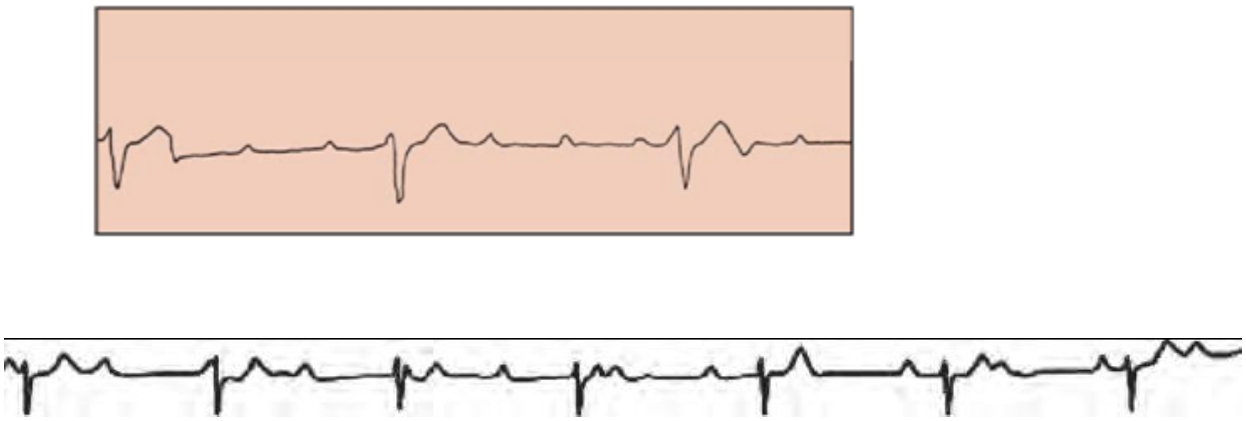

Fig. 22. *Third-degree AV block. A complete AV dissociation is observed.*

Therefore a normal and regular atrial rate will be seen, usually with normal P waves which do not relate to the QRS complexes. P waves can sometimes be disguised by a QRS complex or T wave. The ventricular rate is also regular but much slower than the atrial rate and the QRS complexes are usually widened because they are initiated lower down the conducting system than normal. If a ventricular focus does not take over as pacemaker, ventricular 'standstill' will follow which is seen as a row of P waves with no QRS complexes.

Causes: Myocardial infarction, Degenerative cardiac disease, Digoxin toxicity, Drugs that depress AV conduction (beta-blockers and calcium-channel blockers).

Management.

In a critically ill patient, this arrhythmia will usually drastically reduce cardiac output. It is a medical emergency and it requires immediate drug therapy in order to improve the ventricular rate. Atropine is usually given and inotropic agents may be required to support the patient's circulation. The insertion of a temporary transvenous pacing wire is often necessary until the cause of the block has been resolved. If the block is permanent, a permanent pacemaker will subsequently be required.

VENTRICULAR ARRHYTHMIAS.

Ventricular extrasystoles.

Ventricular extrasystoles, also known as ventricular ectopic beats or premature ventricular contractions (PVCs), arise from an irritable focus within the ventricles, firing randomly. They are very common and can occur quite naturally in healthy patients. They do not necessarily cause any symptoms or problems. They are easily recognised as they have wide and bizarre QRS complexes which are therefore easy to diagnose. During a ventricular extrasystole, there is a reduced (or no) cardiac output and if they don't occur frequently, they are usually of no

importance and are left untreated. However, if they are very frequent or occur in salvos (i.e. two or more together), or occur in a repeating pattern (such as in bigeminy or trigeminy), or occur in a patient with cardiac disease or problems, then they can cause a reduction in cardiac output or lead to more serious ventricular arrhythmias, in which case treatment will be necessary. There are various types of ventricular extrasystoles that result in various arrhythmias.

Causes: Use of substances such as alcohol, caffeine, tobacco and cocaine, Electrolyte imbalance, Hypoxia, Hypothermia, Myocardial infarction, Metabolic acidosis, Digoxin toxicity.

Unifocal ventricular extrasystoles

Unifocal ventricular extrasystoles are all the same shape and size and are caused by an irritable ectopic focus in the ventricles firing before it should. Conduction through the myocardium is abnormal and ventricular depolarisation is delayed, resulting in a wide QRS complex. Occurring infrequently, these arrhythmias are of little or no consequence but if there is a salvo of three or more then this is – by definition – ventricular tachycardia (Hand, 2002) and this indicates an irritable or unstable myocardium (fig. 23).

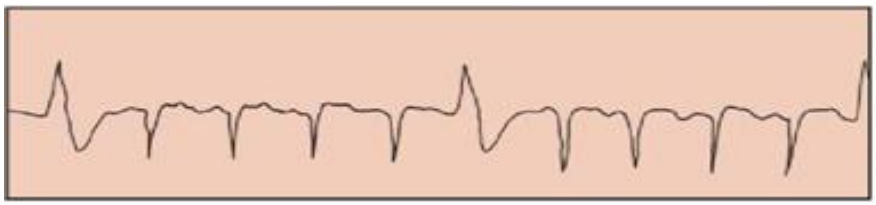

Fig. 23. *Uniifocal ventricular extrasystoles.*

Multifocal ventricular extrasystoles are more dangerous than unifocal ventricular extrasystoles because there is more than one irritable focus in the ventricular muscle. They can be easily recognised by their differing shapes and sizes. These arrhythmias can lead to more serious arrhythmias, or even lethal arrhythmias such as ventricular fibrillation (fig. 24).

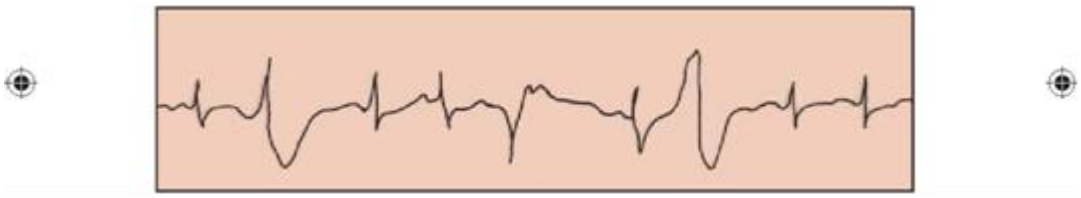

Fig. 24. *Multifocal ventricular extrasystoles*

Bigeminy is caused by an abnormal focus in the ventricular muscle and is recognised by a unifocal ventricular ectopic beat after each normal sinus beat. If this is prolonged, it causes reduced cardiac output and hypotension and will require intervention (fig.25).

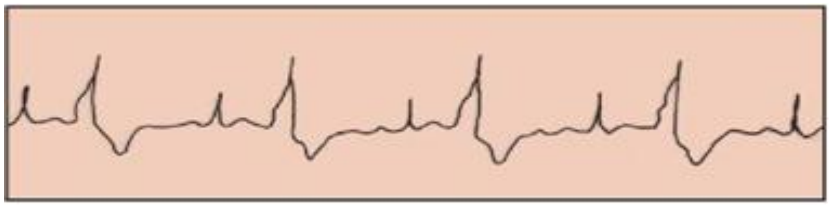

Fig. 25. *Bigeminy*

R-on-T phenomenon

R-on-T phenomenon occurs when a ventricular extrasystole occurs at the peak of the T wave of the preceding beat (i.e. during ventricular repolarisation, when the heart is resting). This is very dangerous and can lead to ventricular tachycardia or ventricular fibrillation (Fig. 26).

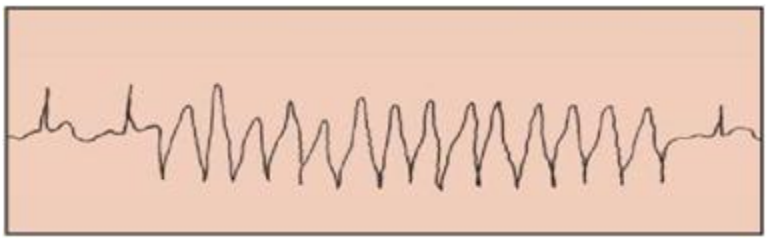

Fig. 26. *R- on T phenomen, ventricular tachycardia*

Management of ventricular extrasystoles and related rhythms.

If ventricular extrasystoles are infrequent and the patient is asymptomatic, no treatment is necessary. However, if they are frequent and/or produce symptoms of reduced cardiac output, treatment will need to be instigated. The treatment depends upon the cause of the problem. For example:

- If caused by hypoxia, oxygen administration is required or the oxygen concentration should be increased.
- If caused by acidosis, correct the acidosis.
- If caused by hypokalaemia (low potassium), hypocalcaemia (low calcium) or hypomagnesaemia (low magnesium), give intravenous supplementation.

N.B! If they are frequent and/or dangerous, give antiarrhythmic drugs. However, remember that antiarrhythmic agents may worsen existing arrhythmias or cause new rhythm disturbances; this is known as the proarrhythmic effect. Intravenous amiodarone is used for the acute treatment of ventricular extrasystoles, and beta-blockers and calcium-channel blockers may also be used.

Ventricular tachycardia

Ventricular tachycardia (VT), also known as broad complex tachycardia, is present if a focus in the ventricular muscle fires at high frequency and more than three ventricular ectopic beats occur in a row at a rate of 100–250 b.p.m. In effect it causes rapidly repeated ventricular extrasystoles. There are no P waves present, the QRS complexes are wide and slightly irregular

and may vary slightly in shape. It may occur paroxysmally and cause no symptoms, but sustained VT is very dangerous and is usually a life-threatening arrhythmia that often precedes ventricular fibrillation. Therefore it requires immediate intervention. VT can be monomorphic or polymorphic and either can be pulseless.

Monomorphic ventricular tachycardia

Monomorphic VT is caused by a single ectopic focus firing rapidly and all the complexes look the same (Fig. 27).

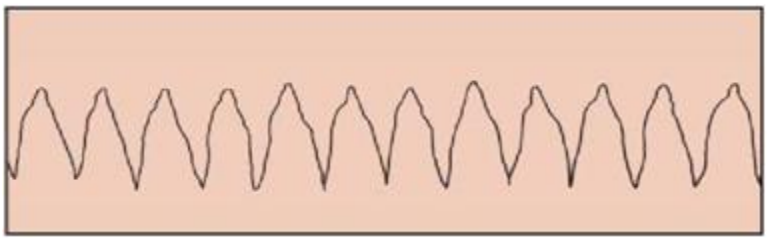

Fig. 27. *Monomorphic ventricular tachycardia*

Polymorphic ventricular tachycardia

Polymorphic VT occurs when there are more than one ectopic foci firing and the complexes therefore change in shape and size (Fig. 28). An example of this is **torsades de pointes**, which may be paroxysmal and is often caused by drugs.

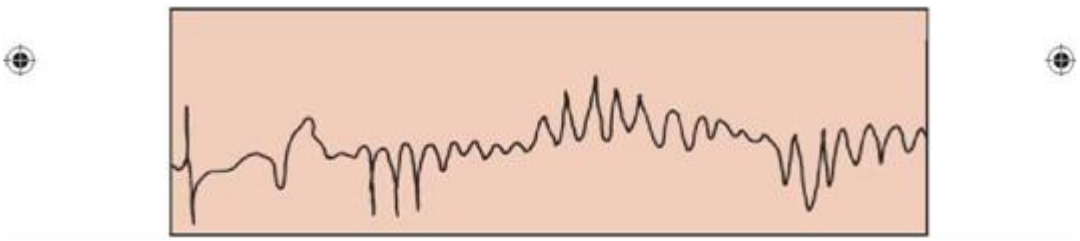

Fig. 28. *Monomorphic ventricular tachycardia «***torsades de pointes»**

When VT is sustained, the patient often becomes pulseless and will collapse as a result because ventricular filling time is severely reduced, as is the force of contraction. Therefore, cardiac output is reduced and, consequently, so is the blood pressure. The condition may degenerate into ventricular fibrillation, causing immediate cardiac arrest. Immediate action is therefore necessary.

Management. The management of patients with VT depends on the symptoms produced.

- If the patient is pulseless, it is treated in the same way as ventricular fibrillation, namely by defibrillation and cardiopulmonary resuscitation (following the Resuscitation algorithm).

- If the pulse is present but the patient is unstable, DC synchronised cardioversion is needed immediately.
- A stable patient will be treated with amiodarone and the cause corrected. If amiodarone is ineffective, DC synchronised cardioversion will be needed.

Causes: Myocardial infarction, Proarrhythmic effects of some antiarrhythmic agents, Electrolyte imbalance, Heart failure, Valvular disease.

Ventricular fibrillation

Ventricular fibrillation (VF) is caused by numerous irritable foci within the myocardium firing rapidly and chaotically (fig.29). The effect of this is that the ventricles quiver rather than contract and thus there is no cardiac output; the physiological effects are the same as asystole. However, there is a greater chance of survival as there is still some electrical activity. The finer the trace, the less chance there is of survival. Untreated VF will usually cause sudden death.

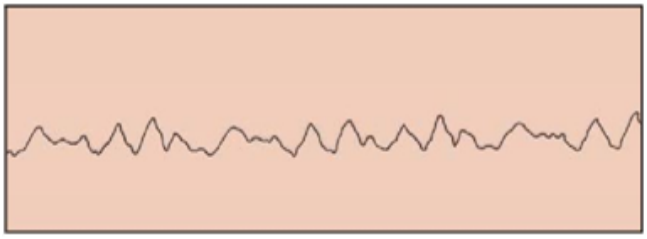

Fig. 29. *Ventricular fibrillation*

Management

If VF is suspected, immediately check the patient (VF can be mimicked by external electrical interference). If the patient is unresponsive, treatment in the form of defibrillation must be instigated immediately following the Resuscitation algorithm, as the patient is in cardiac arrest. If there is any doubt about whether the rhythm is asystole or fine VF do not attempt defibrillation. Instead, continue chest compression and ventilation.

Consider giving a single precordial thump (a sharp blow to the lower third of the sternum) when cardiac arrest is confirmed rapidly after a witnessed and monitored sudden collapse and there is no defibrillator immediately to hand. A precordial thump should be undertaken immediately after confirmation of cardiac arrest but only by healthcare professionals trained in the technique. Cardiopulmonary resuscitation must be initiated if defibrillation is delayed for any reason to ensure oxygen delivery. Intravenous epinephrine is administered if defibrillation does not immediately revert the rhythm to sinus rhythm.

Causes: Hypoxia, Electrolyte imbalances, Acid–base disturbances, ventricular tachycardia and other arrhythmias, Digoxin toxicity, Hypothermia, Heart disease.

Asystole.

Asystole literally means ‘no systole’ and therefore there is no electrical activity and no mechanical contraction of the heart (this is ventricular ‘standstill’). Without immediate treatment, asystole is rapidly irreversible and death ensues. The ECG trace is seen as a flat line (fig. 30).

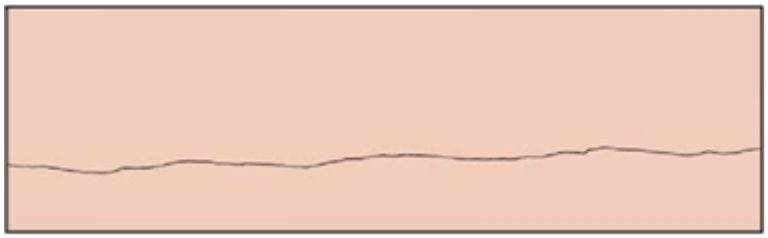

Fig. 30. *Asystole.*

Management

If asystole is suspected, check the patient immediately. Asystole can be mimicked by something as simple as electrode detachment. Ensure that fine VF is excluded by turning up the gain on the monitor. If the patient is pulseless and unresponsive, cardiopulmonary resuscitation must be instigated immediately, together with a single dose of intravenous atropine and repeated doses of epinephrine, following the Resuscitation algorithm. It should be noted that treatment for a patient in asystole (particularly sustained asystole) is unlikely to be successful.

Causes: Untreated ventricular fibrillation, Severe hypoxia, Severe electrolyte imbalances, Severe acid–base disturbances, Myocardial infarction, Hypothermia.

Many of the arrhythmias discussed within this workbook require no treatment, because they will not affect cardiac output (e.g. occasional unifocal ventricular extrasystoles). However, many arrhythmias may compromise a patient’s cardiovascular system and some of them are even life-threatening. It is important to remember that what may appear to be a benign arrhythmia may lead to a more serious arrhythmia – particularly in a patient who is already critically ill – and this is why the importance of accurate monitoring cannot be stressed enough. Appropriate management requires precise diagnosis and appropriate treatment of the underlying cause.

WHEN AN ARRHYTHMIA IS DETECTED

- Check ABCs (airway, breathing, circulation).
- Commence cardiopulmonary resuscitation without delay (if necessary).
- If the patient is asymptomatic then often no treatment is required.
- If cardiac output is affected then treatment is necessary.

Treatment is aimed symptomatically and/or causally. Table 1 summarizes appropriate interventions for the differrant arrhythmias.

Drug and non-drug interventions

The interventions listed in Table 1 are instigated if the patient is demonstrating symptoms of reduced cardiac output. Where no symptoms are apparent, or are unlikely, treatment is usually not necessary. Arrhythmias marked with an asterisk (*) require the treatment stated, regardless of the symptoms.

Table 1 Specific interventions for arrhythmias.

Arrhythmia	Non-drug intervention	Drug intervention
Sinus arrhythmia	Treat cause if symptomatic	Treat cause if symptomatic
Sinus bradycardia	Transcutaneous or transvenous pacing	Atropine
Sinus tachycardia	If caused by haemorrhage, stop bleeding and replace fluid	Beta-blockers Calcium-channel blockers
Premature atrial contractions	Treat cause if symptomatic	Digoxin (if problematic)
Atrial flutter	Direct-current synchronised cardioversion (if tachycardia, use transcutaneous or transvenous pacing)	Beta-blockers Verapamil
Atrial fibrillation	Direct-current synchronised cardioversion (if less than 48 hours from onset of arrhythmia) Carotid sinus massage	Flecainide or propafenone (if no structural heart disease) Amiodarone (if structural heart disease) Anticoagulant drugs
Atrial tachycardia	Carotid sinus massage Direct-current synchronised cardioversion	Adenosine Amiodarone
Junctional escape rhythm	Treat cause if symptomatic	Atropine (if arrhythmia causes symptomatic bradycardia)
First-degree AV block	Treat underlying cause	Treat underlying cause
Mobitz type I second-degree heart block	Transcutaneous pacing Transvenous pacing	Atropine
Mobitz type II second-degree heart block*	Transcutaneous pacing Transvenous pacing	Atropine
Third-degree AV block*	Transcutaneous pacing Transvenous pacing	Atropine
Unifocal ventricular extrasystoles	Treat underlying cause	Amiodarone Beta-blockers Calcium-channel blockers Lidocaine
Multifocal ventricular extrasystoles	Treat underlying cause	Amiodarone Beta-blockers Calcium-channel blockers Lidocaine
Bigiminy	Treat underlying cause	Amiodarone Beta-blockers Calcium-channel blockers Lidocaine
R-on T phenomenon*	Treat underlying cause	Amiodarone Beta-blockers Calcium-channel blockers Lidocaine

Monomorphic ventricular tachycardia*	If pulseless: defibrillation and/or cardiopulmonary resuscitation If pulse present: (patient unstable) DC synchronised cardioversion; (patient stable, amiodarone ineffective) DC synchronised cardioversion	Epinephrine Amiodarone
Polymorphic ventricular tachycardia*	If pulseless: defibrillation and/or cardiopulmonary resuscitation If pulse present: (patient unstable) DC synchronised cardioversion; (patient stable, amiodarone ineffective) DC synchronised cardioversion	Epinephrine Amiodarone
Ventricular fibrillation*	Defibrillation and/or cardiopulmonary resuscitation	Epinephrine
Acystole*	Cardiopulmonary resuscitation	Epinephrine Atropine

*Require treatment regardless of symptoms

Drugs used for arrhythmia treatment

Table 2 lists the drugs used for arrhythmia treatment, according to the Vaughan-Williams classification of antiarrhythmic drugs (Anaesthesia UK, 2008; British Medical Association and Royal Pharmaceutical Society of Great Britain, 2009).

Table 2. Drugs used for arrhythmia treatment

Class	Mechanism	Drug(s)	Uses
I	Sodium-channel blockers that prolong the action potential	Quinidine Procainamide Disopyramide	Supraventricular arrhythmias
Ib	Sodium-channel blockers that shorten the action potential	Lidocaine	Ventricular extrasystoles
Ic	Sodium-channel blockers that don't affect action potential	Flecainide Propafenone	Ventricular extrasystoles
II	Beta-blockers	Atenolol Esmolol Propanalol	Sinus tachycardia Atrial fibrillation Atrial tachycardia Ventricular extrasystoles
III	Potassium-channel blockers	Amiodarone	Atrial fibrillation Ventricular extrasystoles Stable ventricular tachycardia
IV	Calcium-channel blockers	Verapamil Diltiazam	Sinus tachycardia Atrial flutter Atrial fibrillation Atrial tachycardia Ventricular extrasystoles
V	Other mechanisms	Digoxin Atropine	Premature atrial contractions Atrial fibrillation Atrial tachycardia Sinus bradycardia Junctional escape rhythm

		Epinephrine	Second-degree heart block (Mobitz I and II) Third-degree heart block Asystole Ventricular fibrillation Asystole

Defibrillation

The delivery of a direct electrical current through the heart depolarises the myocardium allowing the sinoatrial node to resume its normal pacemaker function.

Automatic external defibrillators ***(AED)*** should be used wherever possible. All healthcare professionals should consider the use of an AED to be an integral component of basic life support. They are described as sophisticated, reliable, safe, computerised devices that deliver defibrillatory shocks to victims of cardiac arrest by giving visual and voice prompts to rescuers.

Manual defibrillator - self-adhesive defibrillator pads, attached to the defibrillator through an interface cable, rather than paddles because this will reduce the risk of sparks and the operator will not have to lean over the patient.

When using defibrillator paddles, water-based gel pads should be placed between the paddles and the patient's skin. The paddles should be placed in the appropriate position:

•The right (sternal) electrode is placed below the clavicle to the right of the sternum.

•The apical paddle is placed vertically in the mid-axillary line, level with the V6 ECG electrode's position

The paddles should be pressed firmly to the chest wall with an optimal force of 8 kg A shock of between 150– 360 Joules is delivered when the patient is in ventricular fibrillation.

Safety considerations.

Turn off oxygen supplementation or remove the oxygen mask or nasal cannulae and move them at least 1 meter away before the shock is delivered. Sparks from poorly applied paddles may cause a fire in an oxygen-rich environment. Remove any transdermal drug patches before a shock is delivered. This is particularly important for GTN (glyceryl trinitrate) patches because they may explode. Defibrillation should only be carried out by trained healthcare professionals.

Cardioversion.

Cardioversion is carried out in the same manner as defibrillation but the direct-current (DC) shock is delivered in synchrony with the peak of the R wave. The defibrillator has a 'synchronise' switch which is utilised for cardioversion. The other major differences are that the conscious patient will usually require sedation, and the intensity of the shock is usually less. Current evidence suggests that the majority of patients will be successfully cardioverted by

shocks of 200 Joules, and it appears that only a very small percentage of patients benefit from stronger shocks. Note that antero-posterior electrode placement may be more effective than the traditional antero-apical position in cardioversion of atrial fibrillation, although either position is acceptable.

Pacing.

Temporary pacing. A pacemaker is a device that generates an electrical impulse from a pulse generator, which is then transmitted through the heart causing depolarisation. This in turn brings about contraction of the myocardium. Temporary pacemakers are used in emergency and acute situations until the arrhythmia has resolved or until a permanent pacemaker can be fitted if necessary.

Transcutaneous pacing - is used in an emergency situation and is a simple, non-invasive process of applying external electrodes to the anterior chest wall, or on the patient's anterior chest wall and one on the patient's back. Electrical impulses are generated and these pass through the skin to the myocardium and thus pace the heart.

Putting it all together.

When considering a rhythm strip, it is helpful to approach diagnosis with a systematic evaluation that will help you to determine the rhythm. The following six steps can be applied systematically to each arrhythmia you deal with.

Step 1-Consider whether the rhythm is life-threatening. If it is life-threatening, summon help and instigate the appropriate Resuscitation algorithm.

Step 2-Consider the heart rate (Bradycardia - Less than 60 b.p.m., Normal rate Between 60 and 100 b.p.m., Tachycardia More than 100 b.p.m.)

Step 3-Consider the P waves. If P waves are present:

- Are they normal in appearance?
- Does one P wave occur before each QRS complex?

Step 4-Consider the PR interval

- Is the PR interval of normal duration (0.12–0.2 seconds)?
- Is the interval prolonged?
- Is the interval shortened?
- Is it consistent?

Step 5—Consider the QRS complex

- Is the QRS complex normal in duration (0.06–0.12 seconds)?
- Is the QRS consistent in duration?
- Are the complexes of normal shape and configuration?
- Is the QRS consistent in form?

Step 6-Consider the RR interval

- Is the RR interval consistent?
- If it is inconsistent, is there some pattern in the variation?

Now, bearing these six steps in mind, we can work out with arrhythmias.

LITERATURE

Main literature

1. *Pediatrics*: Texbook for students of the Faculty of Foreign Students / N.S. Paramonova, V.A. Zhemoytiak, P.R.Gorbachevsky,- Grodno: GrSMU,2010. – 264p.
2. *Nelson* Textbook of Pediatrics, 19th ed. /edited by Robert M. Kliegman et al. –USA,-2011, P. 5320-5338.
3. Cardiac Arrhythmia RecognitionAn easy learning guide. Fiona Foxall 2010.P.1-152.

Additional literature:

4. *Lissauer, T* Illustrated textbook of paediatrics / T. Lissauer, G. Clayden ed.4. Elsevier, 2012. P. 277-285.
5. *Bayes de Luna, Antoni.* Clinical arrhythmology / Antoni Bayes de Luna ; with the collaboration of Diego Goldwasser .[et al.].2011, p.95-290.

CONTENTS

Учебное издание

Козыро Инна Александровна

Чичко Алексей Михайлович

АРИТМИИ У ДЕТЕЙ

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