Clinical practice review

Cardiac Arrhythmias: Diagnosis and Management. The Tachycardias

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ABSTRACT

Objective: To review the diagnosis and management of cardiac arrhythmias in a two-part presentation.

Data sources: Articles and published peer-review abstracts on tachycardias and bradycardias.

Summary of review: Normal cardiac rhythm originates from impulses generated within the sinus node. These impulses are conducted to the atrioventricular node where they are delayed before they are distributed to the ventricular myocardium via the His-Purkinje system. Abnormalities in cardiac rhythm are caused by disorders of impulse generation, conduction or a combination of the two and may be life-threatening due to a reduction in cardiac output or myocardial oxygenation.

Cardiac arrhythmias are commonly classified as tachycardias (supraventricular or ventricular) or bradycardias. The differentiation between supraventricular and ventricular tachycardias usually requires an assessment of atrial and ventricular rhythms and their relationship to each other.

In the critically ill patient the commonest tachycardia is sinus tachycardia and treatment generally consists of management of the underlying disorder. Other supraventricular tachycardias (SVTs) include, atrial flutter, atrial fibrillation and paroxysmal supraventricular tachycardia (PSVT) all of which may require cardioversion, although to maintain sinus rhythm, antiarrhythmic therapy is often needed. Adenosine is useful in the management and treatment of many SVTs although its use in PSVT with Wolff-Parkinson-White syndrome is hazardous. Multifocal atrial tachycardia is a characteristic supraventricular tachycardia found in the critically ill patient. While it usually responds to intravenous magnesium sulphate, its management also requires removal of various precipitating factors.

Ventricular tachycardia (VT) and ventricular fibrillation (VF) require urgent cardioversion and defibrillation respectively. Torsade de pointes should be differentiated from these ventricular arrhythmias as antiarrhythmic therapy may be contraindicated.

Conclusions: Supraventricular and ventricular tachycardias in the critically ill patient often have underlying disorders that precipitate their development (e.g. hypokalaemia, hypomagnesaemia, anti-arrhythmic proarrhythmia, myocardial ischaemia, etc). While antiarrhythmic therapy and cardioversion or defibrillation may be required to achieve sinus rhythm, correction of the associated abnormalities is also required. (Critical Care and Resuscitation 2002; 4: 35-53)

Key words: Critical illness, atrial flutter, atrial fibrillation, paroxysmal supraventricular tachycardia, Wolff-Parkinson White syndrome, torsade de pointes, multifocal atrial tachycardia, ventricular tachycardia, ventricular fibrillation

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NORMAL CARDIAC RHYTHM

The rhythm of a normal resting adult heart is initiated from impulses generated from the sinoatrial (SA) node with a rate varying between 60 - 100 beats per minute (bpm). During sleep the rate may decrease to 30 - 50 bpm, with episodes of sinus pauses up to 3 seconds, sinoatrial block, junctional rhythms, first degree and second degree atrioventricular nodal block occurring often enough (particularly in trained athletes) to be considered normal variants.

The impulses generated from the SA node spread via specialised internodal atrial conducting pathways to the atrioventricular (AV) node, where they are delayed before they are finally distributed to the ventricular myocardium via the His-Purkinje system. Normally, with exercise the heart rate increases to at least 85% of the age predicted maximum of 220 - age in years, with failure to do so being termed ‘chronotropic incompetence’. Sinus arrhythmia is defined as sinus rhythm with P-P variations of more than 10%. It is due to cyclical variations in vagal tone commonly related to respiration (the rate is faster with inspiration and slower with expiration), and is often seen in individuals with sinus bradycardia. It disappears with exercise, breath holding and atropine and is more likely to be seen in individuals who do not have cardiac disease.

Frequent multifocal ventricular ectopic beats have been noted in up to 12% of normal adults during a 24 hr period, indicating that, in the absence of underlying cardiovascular disease, ventricular ectopic beats are not a prelude to something more sinister.

CARDIAC ARRHYTHMIAS

An arrhythmia is defined as any cardiac rhythm other than regular sinus rhythm. It is caused by a disorder of impulse generation, impulse conduction or a combination of the two, and may be life-threatening due to a reduction in cardiac output, reduction in myocardial blood flow or precipitation of a more serious arrhythmia. While the term ‘dysrhythmia’ would appear to be better suited as a label for an abnormal cardiac rhythm (as the term arrhythmia suggests an absence of rhythm), the term ‘arrhythmia’ will be used in this review as it has now become the accepted medical term.

Diagnosis of an arrhythmia

The assessment of an arrhythmia requires the determination of the site of the conduction disturbance, the atrial and ventricular rhythms present and the relationship between the atrial and ventricular impulses. When using the standard ECG leads, the cardiac rhythm is often best considered from leads II or V1 as they provide the maximum P and QRS wave amplitudes to allow the supraventricular and ventricular impulse relationship to be determined. In unusual circumstances, a trace of up to 60 seconds may be required.

However, leads II and V1 are not ideal for the recognition of myocardial ischaemia, although V5, which shows approximately 90% of all ST segment changes due to anterior, inferior or posterior ischaemia, is. A modification of both leads II and V5, where the right arm lead is mounted on the manubrium sterni, the left arm lead is mounted on the xiphisternum and left leg lead is placed in V5 position, facilitates the diagnosis of both rhythm and ischaemic changes. Using this lead, the setting of lead I results in maximal P wave amplitude and the setting of lead II offers optimal ischaemic detection, with the added advantages of reducing interference and electrical artifact.

To perform the cardiac ECG ‘rhythm trace’ the patient should be in a warm environment to reduce ‘shivering’ artifact, and the recording should not be performed during body movement, as muscle or limb movement artifact can simulate ventricular tachycardia.

Description of an arrhythmia

Arrhythmias may be described from their following characteristics:

1. Rate (e.g. tachycardia or bradycardia)
   a. tachycardia is defined as three or more consecutive impulses from the same pacemaker at a rate exceeding 100 bpm in adults (i.e. > 8 years of age).
   b. bradycardia is defined as three or more consecutive impulses from the same pacemaker at a rate less than 60 bpm.
2. Rhythm (e.g. regular or irregular)
3. Origin of impulse (i.e. supraventricular, ventricular, or artificial pacemaker)
4. Impulse conduction (i.e. atrioventricular, ventriculo-atrial or block)
5. Ventricular rate
6. Special phenomena (e.g. pre-excitation)

Management of an arrhythmia

In general, the management of an arrhythmia focuses on:

- correcting precipitating causes (e.g. hypokalaemia, hypomagnesaemia, hypercapnia, hypoxia, metabolic alkalosis, drug poisoning or toxicity),
- restoring sinus rhythm or providing a supraventricular rhythm with an acceptable ventricular rate, and
- preventing a relapse.

The specific management of an arrhythmia depends upon whether it is an ectopic impulse, a sustained arrhy-
thmia (supraventricular or ventricular tachycardia, or bradycardia) or a special phenomenon.

ECTOPIC IMPULSES

An ectopic impulse is one that arises from any site other than the SA node. An escape impulse is one which arises from a different pacemaker from the underlying pacemaker due to a delay in the arrival of the expected impulse of the prevailing rhythm. An extrasystole is a premature impulse (i.e. arises earlier than one would anticipate by observing the prevailing rhythm) and usually shows a fixed, and probably causal, relationship to the preceding activation of the same cardiac chamber.\(^{13}\)

An increase in both atrial and ventricular ectopics may be associated with:

- physiological conditions (e.g. cold, emotion, fatigue, pregnancy)
- drugs (e.g. alcohol, tea, coffee, tobacco, aminophylline, tricyclics, digoxin, quinidine, sympathomimetics)
- disease (e.g. ischaemic heart disease, cardiomyopathies, pulmonary embolism, COPD, systemic disease, fever, renal colic, hypokalaemia and biliary tract disease).

Atrial ectopics

Atrial ectopics commonly occur in early cardiac failure and often herald the onset of atrial fibrillation or atrial flutter, particularly when they are associated with acute myocardial infarction, post operative thoracotomy, rheumatic fever or thyrotoxicosis. They can also initiate paroxysmal supraventricular tachycardias in susceptible patients. The premature atrial complex may arise from any location in the atria and are recognised as an early P wave with a morphology that differs from the sinus P wave. There are three different effects that the atrial ectopic may produce on the rhythm of the heart:

1. It may discharge the SA node, so that the pause following it is the same as normal,
2. It may not discharge the SA node, so that there is a compensatory pause before the next sinus beat, or
3. It may be partially or completely blocked in the AV node, to prolong the PR interval or exhibit a P wave with no ventricular response.

Junctional ectopics

As the AV node in vitro does not have the property of automaticity, it is believed that junctional ectopics arise from the bundle of His. The impulses are conducted retrogradely to the atria and antegrade to the ventricles with the P wave being hidden by the QRS or appearing just before, or after, the QRS wave and inverted in leads II, III and aVF.

Ventricular ectopics

Ventricular ectopics are of little clinical significance in normal individuals.\(^{15,16}\) They assume significance when they are associated with clinical evidence of heart disease, as an indicator of cardiac disease rather than a disorder that needs to be treated, as treatment to suppress them may be associated with an increase rather than a decrease in mortality.\(^{15,16}\)

Ventricular ectopic beats are characterised by a wide QRS complex that is not preceded by a P wave. It often bears a relatively fixed relationship to the preceding sinus complex. If fixed coupling does not exist but the intervals between the ventricular ectopic beats are regular, then the ectopic beat has an entry block and ventricular parasystole is said to be present.

If every sinus beat is followed by a ventricular ectopic, then ventricular bigeminy is said to be present. If every second sinus beat is followed by a ventricular ectopic, then ventricular trigeminy is said to be present, etc. If every sinus beat is followed by two ventricular ectopic beats then this is described as ventricular couplets (if a sinus beat is followed by three ectopics then ventricular tachycardia is present if the ventricular rate is greater than 100 beats per minute).

The ventricular ectopic impulse is often not conducted retrogradely and therefore, while it usually blocks the sinus beat, it usually does not alter the sinus rate. Post-extrasystolic T wave changes (i.e. alteration in the T wave vector, amplitude or contour compared with the preceding sinus beat T wave) occur in 68% of normal individuals and 81% of patients with coronary heart disease, and are not indicative of heart disease.\(^{17}\)

Warning ventricular ectopic beats preceding ventricular tachycardia (VT) or ventricular fibrillation (VF) have been described as, greater than 1 in 10 beats, two or more beats in succession, multifocal (i.e. have differing contours in any one lead and differing coupling times to the preceding impulse of the prevailing rhythm), R on T phenomenon (i.e. refers to an ectopic impulse which is superimposed on the T wave of a preceding impulse), and arising from the left ventricle (i.e. show a RBBB pattern). However, these ‘warning arrhythmias’ have been detected in up to 60% of patients who do not develop VF,\(^{18,19}\) and episodes of VF not preceded by these ‘warning arrhythmias’, have been observed in up to 40% of patients with acute myocardial infarction.\(^{20-24}\)

The R on T ventricular ectopic as a predictor of VF in patients with acute myocardial infarction is neither a specific nor sensitive phenomenon,\(^{25,26}\) and it may have been confused with the vulnerable phase for fibrillating an animal ventricle by stimulation techniques not analogous to ectopic beats.\(^{27}\)
SUSTAINED ARRHYTHMIAS

The sustained arrhythmias are classified as either tachycardias or tachyarrhythmias (if there are three or more complexes with a rate of greater than 100 beats per minute) or bradycardias or bradyarrhythmias (if the rate of the complexes are less than 60 beats per minute). The tachycardias may be caused by disorders of impulse conduction (i.e. re-entry) or impulse generation (i.e. enhanced automaticity or triggered activity). The bradycardias may be caused by a decrease in impulse formation or conduction.

Tachycardias

Uncontrolled tachycardias can induce cardiac failure (even a reversible cardiomyopathy), cardiac ischaemia and may degenerate into ventricular fibrillation. In the management of a patient with tachycardia, the clinician is required to assess whether the source of the rhythm is supraventricular (e.g. atrial) or ventricular. However, the relationship between an atrial and ventricular rhythm may be extremely difficult to define and may require methods to reduce the ventricular rate (e.g. increase the AV block by vagal stimulation; Figure 1), enhance the atrial ECG complexes (e.g. intra-atrial or oesophageal leads) or observe aortic and mitral valve movement during echocardiography to clarify the relationship.

Vagal stimulation

This is often used to diagnose and treat supraventricular tachycardias by slowing the atrial rate and increasing the AV block. The standard responses to vagal stimulation are reduction of sinus rate (even transient second- or third-degree block), slowing of the ventricular response in atrial fibrillation, transient increase in the AV block in atrial flutter (Figure 1), conversion of 50 - 60% of junctional tachycardias (i.e. paroxysmal supraventricular, idio-nodal, and multifocal atrial tachycardia) to sinus rhythm and no effect in VT. In latent digoxin toxicity, bigeminal or multifocal ventricular ectopics may occur.

Vagal stimulation may be performed by physical methods (e.g. carotid sinus massage, Valsalva manoeuvre, facial immersion, etc) or drugs.

Physical methods

Carotid sinus massage (Czermak’s manoeuvre). Carotid sinus massage is performed with ECG monitoring, the patient supine and the head turned to the opposite side. The carotid bifurcation is localised by palpating the carotid impulse at the angle of the jaw. While observing the ECG trace, pressure or massage is applied for no longer than 3 s at the bifurcation of the carotid artery. To increase the vagal effect, the manoeuvre should be performed at the end of inspiration or during expiration. The carotid sinus on one side is stimulated first. If there is no effect, after a delay of 1 minute, the opposite carotid sinus is stimulated. A pause of greater than 3 s or a decrease in systolic blood pressure of more than 50 mmHg is abnormal, and indicative of carotid sinus hypersensitivity. This manoeuvre is contraindicated in patients who have AV block, or known cerebrovascular disease.

Valsalva manoeuvre. This is performed by asking the patient not to take a deep breath before blowing into an aneroid manometer up to 40 mmHg for 10 - 15 s. The vagal response occurs during the period of termination of the manoeuvre.

Facial immersion. This is performed by asking the patient to hold his/her breath on inspiration while a towel with cold water is placed over the jaw. This produces a profound vagal response with peripheral vasodeconstriction, unlike a vasovagal attack which is associated with vasodilation.

Other physical methods. These include, ocular pressure (Aschner-Dagnini manoeuvre), vomiting and squatting. In one study of 35 patients with induced and sustained junctional tachycardia, the Valsalva manoeuvre in the supine position was successful in terminating the arrhythmia in 54%, right carotid sinus massage in 17%, left carotid sinus massage in 5% and facial immersion in 17% of cases.

Drugs

Ephedrine, phenylephrine, edrophonium and neostigmine have all been used to either directly or indirectly stimulate the vagus. These agents are now rarely used.

Atrial tachycardias

Sinus tachycardia

In sinus tachycardia the rate is usually between 100 - 160 bpm but may increase to 220 bpm particularly with severe sympathetic stimulation or drug effect. The causes include anxiety, tetanus, delirium, phaeochromocytoma, thyrotoxicosis, fever (with an increase of 8 bpm for every 1°C increase in temperature, if the fever is due to an infection), pain, shock, drug withdrawal and sympathomimetic agents. Treatment should be directed at the underlying cause (e.g. pain, hypoxia, etc.). Beta-blockers may be required if the tachycardia has been generated by an inappropriate sympathetic hyperactivity, for example in patients with tetanus, drug withdrawal, delirium tremens, thyrotoxic crisis or phaeochromocytoma.

Atrial flutter

Atrial flutter in its common form has an atrial rate that varies between 250 - 350 bpm (usually it is almost
exactly 300 bpm), and is often caused by a single re-entrant circuit in the right atrium. In type 1 atrial flutter, the circuit runs anticlockwise around the right atrium, in type 2 atrial flutter it runs clockwise. Unlike atrial fibrillation, there is a discrete atrial mechanical systole after each electrical flutter wave, explaining why arterial embolism is rare with atrial flutter.

Atrioventricular regurgitation does not occur in atrial flutter, whereas it always occurs with atrial fibrillation. Classically, the ECG trace shows a sawtooth pattern in leads II and III, characterised by a regular atrial rhythm and, in the untreated patient, a 2:1 AV block with a ventricular rate around 150 bpm. There is no isoelectric line between the P waves, which appear inverted in II, III and aVF in 70% (typical pattern or type 1) and upright in 30% (atypical pattern or type 2). A variant is flutter/fibrillation, where the atrial activity alternates between both rhythms, and in this condition arterial embolism can occur. Atrial flutter is commonly caused by those disorders which also cause atrial fibrillation.

The treatment of choice is cardioversion with low-voltage direct current shock (e.g. 50 J). While prior anticoagulation was not initially recommended unless the patient varied between atrial flutter and fibrillation, recent reports of embolic events following cardioversion for atrial flutter without anticoagulation have prompted many to recommend otherwise. Higher energies (100 J) may be required if the flutter has been prolonged. However, in one study of 330 patients with atrial flutter a higher conversion rate was reported using 100 J when compared with 50 J (85% c.f. 70%), irrespective of the duration of the arrhythmia. If a right atrial pacing wire is present then 15 s of rapid atrial pacing (with a pacing cycle length 10 ms less than the atrial rate and progressively decreased to 150 ms or 400 per minute for 15 - 30 seconds) will also convert the arrhythmia.

To maintain sinus rhythm, class Ia, III or IV anti-arrhythmic drugs (Table 1) may be required following the cardioversion (e.g. procainamide, amiodarone or verapamil). Verapamil, digoxin, or amiodarone may also be used instead of cardioversion to slow the ventricular rate or convert the arrhythmia to sinus rhythm. Verapamil will convert the rhythm to sinus in 10 - 30% of cases. If class Ia or III drugs are used to convert the arrhythmia (which have conversion rates varying between 30% - 50%), these drugs may, in rare instances, increase the AV conduction before the rhythm reverts, causing a 1:1 ventricular response and dangerous tachycardia that may proceed to VF. To reduce this hazard, digoxin is often used first to increase the AV block and control the ventricular rate before class Ia or type III agents are used.

Atrial fibrillation

This may be caused by mitral valve disease (the incidence is 41% in mitral stenosis and 75% in mitral regurgitation), cardiomyopathy, ischaemic heart disease, thyrotoxicosis, myxoedema, pneumonia, systemic infection, alcohol intoxication and withdrawal, hypothermia, thoracotomy, post cardiac surgery, lung and mediastinal malignancy, rheumatic fever, pre-excitation syndromes, pulmonary embolism, constrictive pericarditis, COPD, ‘sick’ sinus syndrome, hypovolaemia and idiopathic (i.e. lone atrial fibrillation). The arrhythm-
Table 1  Classification of antiarrhythmic drugs

<table>
<thead>
<tr>
<th>Class</th>
<th>Action</th>
<th>Antiarrhythmic drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>Block inward sodium current</td>
<td>Quinidine, procainamide, disopyramide</td>
</tr>
<tr>
<td>Ib</td>
<td></td>
<td>Lignocaine, tocainide, mexiletine, phenytoin</td>
</tr>
<tr>
<td>Ic</td>
<td></td>
<td>Flecainide, encainide, lorcaaine</td>
</tr>
<tr>
<td>II</td>
<td>Beta adrenergic receptor blockers</td>
<td>Propranolol, metoprolol, atenolol, sotalol</td>
</tr>
<tr>
<td>III</td>
<td>Prolong the action potential</td>
<td>Amiodarone, bretylium, sotalol, ibutilide, dofetilide</td>
</tr>
<tr>
<td>IV</td>
<td>Calcium channel blockers</td>
<td>Verapamil, diltiazem, tiapamil</td>
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Agents not classified (Digoxin, Adenosine)

Figure 2. Atrial fibrillation with a ventricular rate varying between 190 - 200 bpm

genic atrial premature beats that appear to trigger atrial fibrillation arise predominantly from the pulmonary veins.47

The clinical features include palpitations, irregular pulse, variable intensity of the second heart sound, fatigue, cardiac failure and embolic phenomena (there is a six-fold increase in strokes in patients with chronic atrial fibrillation).48 The ECG reveals a variable R-R interval and fibrillatory (f) waves particularly in lead II and V1 (Figure 2). The f waves may be coarse (i.e. waves greater than 0.5 mm from trough to peak) particularly if the atrial fibrillation is of recent onset), or fine, particularly if the atrial fibrillation is chronic, or caused by ischaemic heart disease or congestive cardiomyopathy. All patients with atrial fibrillation should have an ECG, chest X-ray, serum electrolytes, echocardiogram and thyroid function studies.35

Treatment is aimed at correction of any underlying causes or precipitating factors and control of rapid ventricular response (to a resting heart rate of < 110, to increase coronary perfusion, reduce cardiac work and reduce left atrial pressure). This will be achieved by either restoration of sinus rhythm and prevention of recurrence of atrial fibrillation or slowing ventricular rate with AV blocking drugs and anticoagulation to prevent thromboembolic complications. 49,50

Symptomatic improvement in patients with atrial fibrillation will be similar in those who have rhythm control (e.g. reversion to sinus rhythm with antiarrhythmic agents to maintain sinus rhythm) compared with those who have rate control (e.g. negative chronotropic agents and anticoagulation), although exercise tolerance is better (yet hospital admission is more frequent) when rhythm control is compared with rate control.51

The agent most often used to slow the ventricular response is digoxin (unless atrial fibrillation is present in a patient who has Wolff-Parkinson-White syndrome where intravenous procainamide is recommended),52 particularly in patients who have left ventricular systolic dysfunction. Digoxin does not alter the incidence of spontaneous reversion of acute atrial fibrillation to sinus rhythm, which occurs within 24 hr in 60 - 80% of acute cases.53 Amiodarone has also been used which reduces the ventricular rate (and does not increase the risk of death in patients with chronic heart failure)54 increases the incidence of spontaneous reversion to sinus rhythm55 (although this was not found in another study56) and maintains more than 50% of patients in sinus rhythm for one year following cardioversion.57

Calcium channel blockers (e.g. verapamil, diltiazem) and beta-adrenergic receptor blockers (e.g. sotalol), have also been used to reduce ventricular rate, although their negative inotropic effects make them not as useful as digoxin in patients with left ventricular systolic dysfunction.59 Recently, pure class III agents have been used to pharmacologically convert atrial fibrillation (e.g.
either intravenous dofetilide 8 µg/kg or ibutilide 0.025 mg/kg, converting approximately 30% to sinus rhythm), although polymorphic ventricular tachycardia may develop in 4 - 8% of patients. Oral dofetilide in patients monitored in hospital for the first three days (250 µg daily or twice daily, depending on the creatinine clearance - patients with prolongation of QTc, bradycardia, hypokalaemia or severe renal failure were excluded) was associated with an increase in the spontaneous conversion rate to sinus rhythm (12% cf 1% after 1 month) in patients with atrial fibrillation and chronic heart failure with no effect on mortality (although 3% patients developed torsade de pointes, usually within the first three days).58

Class I agents (e.g. quinidine, procainamide, disopyramide, flecainide) tend not to be used particularly when left ventricular dysfunction coexists as they are often associated with an adverse prognosis.59

In a prospective, multicentre and randomised trial of patients with atrial fibrillation of less than 6 months duration, amiodarone (200 mg daily) was more effective compared with sotalol (80 - 160 mg 12-hourly) and propafenone (150 mg 8 to 12-hourly) in maintaining sinus rhythm following cardioversion.60

In the presence of pyrexia or shock due to pulmonary embolism or hypovolaemia, the ventricular rate is often rapid and controllable only by correcting the underlying condition (e.g. fluid administration for hypovolaemia).61

Anticoagulation should be considered for all patients who have chronic or intermittent atrial fibrillation (with the exception of ‘lone’ atrial fibrillation before the age of 60 years, in the absence of cardiopulmonary disease and hypertension) to reduce the incidence of stroke.62-64

In five independent randomised trials, embolic complications associated with chronic nonrheumatic atrial fibrillation occurred in approximately 6% of patients, and was reduced by two-thirds or more (e.g. to 1.5%) by warfarin (to keep the INR between 2.0 and 3.0; although the optimal value may be closer to 3.0).65 The incidence of intracranial bleeding was less than 0.5% and about 100 patients needed to be treated to prevent 2 - 3 serious strokes.66 While aspirin did not consistently reduce the embolic complications in patients with atrial fibrillation,66 it is recommended in those patients with atrial fibrillation in whom warfarin is contraindicated.67

Minidose warfarin (i.e. 1.25 mg daily) does not reduce the incidence of embolic stroke.58

Cardioversion may be considered if atrial fibrillation is acute (i.e. less than 7 days) or associated with shock, hypertrophic cardiomyopathy or Wolff-Parkinson-White (WPW) syndrome. It is usually not considered if atrial fibrillation has been present for longer than 6 months or the left atrium is dilated (greater than 4.5 cm), because recurrence commonly occurs unless the underlying disorder has been corrected.

If atrial fibrillation has been present for more than 48 hr then warfarin is given for 3 weeks prior to the cardioversion and continued for 4 weeks after the cardioversion to reduce the incidence of post-cardioversion stroke, which occurs from 6 hr to 6 days after the cardioversion as atrial mechanical function may not be normal for up to several weeks after the return of sinus rhythm.70 One large multicentre, rand-omised, controlled trial in patients with atrial fibrillation demonstrated that patients who had no transoesophageal echocardiographic evidence of left atrial thrombus did not require a three week period of anticoagulation before cardioversion but could be anticoagulated with warfarin then cardioverted (with anticoagulation continuing for a further 4 weeks) without increasing the incidence of an embolic stroke. This study prompted the suggestion that this approach be recommended in patients who have an increased risk of haemorrhage with warfarin or in those who have atrial fibrillation of less than 3 weeks duration.72

However, while transoesophageal echocardiography may detect patients with atrial fibrillation who have atrial thrombosis (and therefore select those who require anticoagulation prior to cardioversion),73 it may still miss small atrial thrombi and thereby incorrectly simulate safe conditions for cardioversion,70 (e.g. cerebral embolism after cardioversion without anticoagulation in a patient with negative findings on transoesophageal echocardiography, has been reported).74

If the f waves in V1 are greater than 2 mm, cardioversion using 100 J may be chosen first; if the f waves in V1 are less than 2 mm then 200 J is commonly used.75

Quinidine has been considered the mainstay of therapeutic agents to prevent recurrence of atrial fibrillation following cardioversion. However, in most trials 20 - 50% of patients treated with quinidine, compared with 10 - 25% of patients given placebo, have remained in sinus rhythm for 1 year after electrical conversion of atrial fibrillation.76 Quinidine therapy may also be at a cost of an increase in long term mortality.77,78 Other class Ia agents, (e.g. procainamide, disopyramide) are no more successful or any better tolerated.76 While the class III agents of amiodarone and sotalol provide alternatives, the side-effects of the former and the reduction in cardiac function with the latter make them less than ideal, although currently they are the agents of choice. Low dose amiodarone (e.g. < 400 mg/day) appears to have a lower 5 year mortality when compared to quinidine when used to maintain sinus rhythm after cardioversion.79 Flecainide has also been used to maintain sinus rhythm.80
Recently, surgical procedures to provide an electrically isolated corridor of tissue from the sinus node to the atrioventricular node (e.g. ‘atrial corridor procedure’, although atrial transport function is usually not restored and thus atrial thrombus remains a risk; furthermore sinus node dysfunction is common and permanent pacing may be required), or multiple atrial incisions to disrupt the atrial re-entrant pathways (e.g. ‘maze procedure’, which usually maintains atrial transport function, although return of atrial fibrillation, atrial atrial flutter or complete heart block may occur) have been used to maintain sinus rhythm in patients who have had disabling atrial fibrillation uncontrolled by pharmacological therapy. Radiofrequency ablation has also been used successfully in patients in whom atrial fibrillation originated from ectopic beats in the pulmonary veins.

The heart performs more efficiently with the return of sinus rhythm by responding appropriately to stress and restoring the AV valve competence, and an acute decrease in heart rate and increase in stroke volume are usually observed. However, an acute change in cardiac output following conversion is not consistently observed and may be due to a delay in the return of the atrial contraction.

Supraventricular tachycardias

Paroxysmal supraventricular tachycardia (PSVT) or paroxysmal atrial tachycardia (PAT)

This is caused by re-entry in 96% of cases (Figure 3). The re-entry path is at the AV junction in 70%, sinus node in 1 - 2%, atria in 1 - 2%, and is an AV nodal bypass tract in 15% of cases. The remaining 4% or so of cases are due to an ectopic focus.

Reentry involving an AV bypass tract usually travels antegrade through the AV node and retrogradely through the bypass tract. If the bypass tract also conducts antegrade then pre-excitation exists (i.e. WPW syndrome); if the bypass tract only manifests retrograde conduction it is termed a concealed bypass tract. In the latter case the QRS complex during sinus rhythm is normal.

1. Atrioventricular nodal reentrant tachycardia (AVNRT)

AV nodal re-entrant tachycardia has a rate that varies between 160 and 220 bpm, a regular R-R interval and a retrograde P wave (which is often difficult to distinguish in the standard ECG trace) that is buried, precedes or proceeds the narrow QRS complex. While patients who have a left bundle branch block (LBBB) or right bundle branch block (RBBB) may also have PSVT (LBBB or RBBB patterns are not uncommonly associated with PSVT), if the PSVT produces aberrant conduction and a widened QRS, the pattern is RBBB in 85%, RBBB with LAD in 10% and LBBB in 5%, of cases.

The re-entrant pathway is permitted by a fast and slow pathway that exists within the AV node. The fast (anterior) pathway exhibits rapid conduction and a long refractory period while the slow (posterior) pathway exhibits slow conduction and a short refractory period. As only the fast pathway conducts during sinus rhythm, the PR interval during sinus rhythm is normal. However, an appropriately timed atrial ectopic dissociates conduction between two pathways and permits the establishment of circulating electrical activity that spreads to both the atrial and ventricular myocardium causing the tachyarrhythmia. In up to 90% of cases the antegrade conduction proceeds via the slow pathway and the retrograde conduction via the fast pathway.

2. Atrioventricular re-entrant tachycardia (AVRT)

Atrioventricular re-entrant tachycardia incorporates a concealed AV bypass tract as part of the re-entrant circuit. The AV bypass tract allows retrograde conduction only.

3. Sinus node and intraatrial re-entrant tachycardias

The sinus node and intraatrial re-entry tachycardias
incorporate a re-entrant pathway within the sinus node or within the atrium, respectively. These arrhythmias are less common than AVNRT and AVRT.

Clinical features
While the rapid ventricular rate may be tolerated for 1 - 3 days or longer, cardiac failure, hypotension, shock and pulmonary oedema may develop if the rate is allowed to continue unabated. PSVT may occur in the absence of heart disease or may be associated with hypokalaemia, WPW syndrome, ischaemic heart disease, rheumatic heart disease, thyrotoxicosis, pheochromocytoma, cardiomyopathy, mitral valve prolapse or tetanus. In 50% of patients without underlying heart disease, the attack is associated with polyuria caused by an inhibition of vasopressin and release of a natriuretic peptide. This occurs 20 - 30 minutes after the onset of the attack, with micturition occurring every 30 - 90 min up to 8 hr later.

Treatment
The treatments for AVNRT, AVRT and sinus node and intra-atrial re-entrant tachycardias are similar and includes a chronological sequence of:

a. Vagal stimulation
b. Drugs
   i. Short acting intravenous drugs (i.e. an action which is terminated within 1 - 2 min):
      - Adenosine (3 - 15 mg/70 kg, which has now taken the place of verapamil as the treatment of choice, except in asthmatics).
      - Esmolol (35 mg/70 kg)
      - Edrophonium (5 - 20 mg/70 kg)
   ii. Intermediate acting drugs
      - Verapamil 5 - 10 mg intravenously (0.075 - 0.15 mg/kg) infused over 1 - 3 min (terminates 80% of cases). This is the treatment of choice in asthmatic patients with PSVT, or if the PSVT recurs after the effect of adenosine has worn off. However, verapamil, should not be used as a second line drug if adenosine has failed, as verapamil is unlikely to succeed and it will only compound the negative inotropic effect of both agents, causing severe hypotension.
      - a delta interval which does not exceed 0.12 s
      - a delta wave (a slurred thickened proximal portion of the QRS complex). A negative delta wave in V1 is diagnostic of a right-sided bypass tract (Figure 4).
      - a QRS which equals or exceeds 0.12 s. A Q wave in III and aVF often mimics an inferior myocardial infarct.
   c. Cardioversion 50 - 100 J (or rapid atrial pacing)
   d. Surgical ablation or cryo or radiofrequency catheter ablation to interrupt the re-entrant pathway.

4. Atrioventricular pre-excitation (Wolff-Parkinson-White or WPW syndrome)
Pre-excitation exists if the whole or some part of the ventricular muscle is activated earlier than normally by the impulse that originates from the atrium passing through accessory AV conducting fibres rather than through the normal AV conduction system. The connections may occur anywhere around the cardioskeletal ring. The atrial vector is normal (i.e. the P wave is upright in II), differentiating pre-excitation from nodal impulses or ventricular impulses with retrograde atrial activation.

The characteristics of WPW syndrome are episodic supraventricular tachycardia in patients who have specific ECG abnormalities that consist of:

- a PR interval which does not exceed 0.12 s
- a delta wave (a slurred thickened proximal portion of the QRS complex). A negative delta wave in V1 is diagnostic of a right-sided bypass tract (Figure 4).
- a QRS which equals or exceeds 0.12 s. A Q wave in III and aVF often mimics an inferior myocardial infarct.

The classical ECG changes of WPW syndrome occur in 1 - 2 per 1000 of the general population and tachycardias occur in 40 - 80% of these patients, 75% of which are re-entrant supraventricular tachycardias (95% due to retrograde conduction through the bypass tract causing an orthodromic tachycardia with a normal QRS complex and 5% due to antegrade conduction through the bypass tract causing an antidromic tachycardia with a wide QRS complex) and 20% atrial fibrillation (usually wide complex and rapid ventricular rate); atrial flutter is rare.

The WPW syndrome may be associated with mitral
valve prolapse, ischaemic heart disease, hypertrophic subaortic stenosis, cardiomyopathy, ‘sick’ sinus syndrome, rheumatic fever, Ebstein’s anomaly, bicuspid aortic or pulmonary valves, coarctation of the aorta, ventricular septal defect and atrial septal defect. False-positive exercise tests may also occur with WPW syndrome.

The treatment of PSVT associated with WPW includes increasing vagal tone, adenosine, lignocaine, procainamide, amiodarone, propranolol and cardioversion. Sotalol has also been recommended as the drug of first choice in patients with acute PSVT and for long-term management.

The treatment of atrial fibrillation with WPW includes cardioversion, or drugs which act on both the atrial and accessory pathways (i.e. class Ia, Ic or III). Beta-blockers or lignocaine are ineffective, and digoxin, verapamil and adenosine are contraindicated as they may increase the ventricular rate leading to VT and VF by blocking antegrade AV conduction, shortening atrial refractoriness (facilitating the induction of AF) and enhancing the conduction through the anomalous pathway. The treatment of atrial fibrillation with WPW syndrome may be reversed by using 8 mmol of magnesium sulphate, intravenously. By prolonging the refractory period of the AV and His-Purkinje system as well as the accessory pathway, one study found that ibutilide terminated AF in 95% of patients with pre-excitation.

Ablation of the anomalous pathway. In patients in whom the episodes of supraventricular tachycardia are numerous, debilitating and not controlled by long-term antiarrhythmic treatment, epicardial mapping, His bundle ECG and atrial pacing to facilitate operative identification and surgical ablation of the anomalous pathway, have been used. Currently, however, radio-frequency intracardiac ablation is being offered in specialised centres as a safe alternative to medical treatment in patients who have only minor symptoms. Patients with pre-excitation who are asymptomatic have a benign clinical course and only warrant careful follow-up.

5. Nonparoxysmal junctional tachycardia

This rhythm usually results from conditions that enhance automaticity (e.g. theophylline, catecholamine toxicity) or triggered activity (e.g. digoxin toxicity) in the AV junction. The rate usually varies between 70 and 130 bpm, the QRS complex is identical to that observed with sinus rhythm and the rate may be influenced by vagotonic and vagolytic agents. Treatment is directed towards eliminating the underlying factors.

6. Multifocal atrial tachycardia (MAT)

This is a non-reentrant atrial tachycardia that causes supraventricular tachycardia in 1 - 2% of cases. The rate varies from 100 to 220 bpm and is characterised by an absence of one predominant atrial focus with three or more P waves of different morphologies in a single ECG lead, an isoelectric baseline between the P waves, and a variation in PR, PP and RR intervals (Figure 5). It differs from the condition of ‘wandering pacemaker’, which usually refers to multifocal supra-ventricular escape complexes in the presence of sinus bradycardia.
Multifocal atrial tachycardia is required to be differentiated from atrial fibrillation because digoxin is often used in the latter but is not effective for the former. Multifocal atrial tachycardia usually occurs in the critically ill elderly (i.e. 70 years or more) male patient, is often associated with COPD, cardiac failure or ischaemic heart disease, and is usually precipitated by hypoxia, hypercapnia, sepsis, electrolyte disorders, sympathomimetics, digoxin or methylxanthines. While the mortality due to the arrhythmia may be low, patients who develop this arrhythmia have an in-hospital mortality of about 45%. Treatment consists of efforts to correct drug toxicity (e.g. sympathomimetic, digoxin or theophylline toxicity) and correct electrolyte abnormalities (i.e. hypokalaemia, hypomagnesaemia or alkalosis). If the ventricular rate is deemed too rapid then therapy of choice is intravenous magnesium sulphate, 10 mmol in 2 - 3 min (which may be followed by an infusion of 20 - 40 mmol of magnesium sulphate in 4 - 6 hr) or intravenous verapamil 5 - 10 mg.

If verapamil is used then pretreatment with 1 g of intravenous calcium gluconate before administering verapamil has been used to prevent any hypotensive effect, without preventing its anti-arrhythmic effect. Metoprolol (10 mg i.v. over 5 minutes) has also been used, and in one study was more effective than verapamil in reducing ventricular rate and converting MAT to sinus rhythm. However, as many of these patients are elderly with COPD, metoprolol may be associated with adverse side effects (e.g. severe bronchospasm, hypotension). Treatment with digoxin, quinidine, procainamide, lignocaine, phenytoin or cardioversion is usually ineffective. Metoprolol (10 mg i.v. over 5 minutes) has also been used, and in one study was more effective than verapamil in reducing ventricular rate and converting MAT to sinus rhythm. However, as many of these patients are elderly with COPD, metoprolol may be associated with adverse side effects (e.g. severe bronchospasm, hypotension). Treatment with digoxin, quinidine, procainamide, lignocaine, phenytoin or cardioversion is usually ineffective.

**Wide complex tachycardias**

The main question that arises in the management of a patient with a tachycardia is ‘is it supraventricular or is it ventricular?’ If the QRS complex is wide (Figure 6), the bedside diagnosis of the tachycardia is difficult. The features that help distinguish supraventricular from ventricular tachycardias are given in Table 2. If there is doubt about the origin of the impulse then the patient should be cardioverted, particularly if there is cardiac failure or hypotension. If the patient is not haemodynamically compromised, intravenous lignocaine (1.5 mg/kg as an intravenous bolus which may be repeated after 10 min) followed, if required, by adenosine (6 mg as an intravenous bolus, which is followed 2 min later, if required, by 12 mg as an intravenous bolus) is currently recommended. Adenosine is used.
Table 2. Differentiation between supraventricular (SVT) and ventricular tachycardia (VT)

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>Rare</td>
<td>Not uncommon</td>
</tr>
<tr>
<td>Pulse rate (beats/min)</td>
<td>160-220</td>
<td>130-180</td>
</tr>
<tr>
<td>JVP cannon A waves</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Beat-to-beat variation in blood pressure</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Varying intensity of S1</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Reversion with vagal stimulation</td>
<td>Common</td>
<td>Rare</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pressure trace recordings</th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure: cannon A waves</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Blood pressure: beat-by-beat variation</td>
<td>Rare</td>
<td>Common</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Echocardiograph</th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>atrioventricular dissociation</td>
<td>Rare</td>
<td>Common</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECG features</th>
<th>SVT</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR irregularity</td>
<td>Rare</td>
<td>Not uncommon</td>
</tr>
<tr>
<td>Atrioventricular relationship</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. Relationship between the QRS and the preceding P wave</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>b. Fusion beats</td>
<td>Never</td>
<td>Not uncommon</td>
</tr>
<tr>
<td>c. Capture</td>
<td>Never</td>
<td>Not uncommon</td>
</tr>
<tr>
<td>d. Retrograde atrial activation</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>(inverted P in II upright P in aVR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>e. A 2:1 or 3:1 ventriculoatrial conduction with vagal stimulation</td>
<td>Never</td>
<td>Common</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>QRS duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. A narrow QRS complex</td>
</tr>
<tr>
<td>b. If broad complex (&gt; 0.14 s)</td>
</tr>
<tr>
<td>LBBB pattern</td>
</tr>
<tr>
<td>RBBB pattern</td>
</tr>
<tr>
<td>Precordial QRS mainly:</td>
</tr>
<tr>
<td>Positive</td>
</tr>
<tr>
<td>Negative</td>
</tr>
<tr>
<td>Biphasic</td>
</tr>
<tr>
<td>QRS axis</td>
</tr>
</tbody>
</table>

as it has a rapid offset of action, will terminate any arrhythmia that has an obligatory participation of the AV node, and will not cause haemodynamic compromise or change the tachycardia to a life threatening arrhythmia (although it may do so in patients who have WPW with atrial fibrillation).\(^{102,103}\)

In patients with wide complex tachycardias there is little difference between bolus doses of adenosine and adenosine triphosphate in relation to the minimal effective dose and incidence of side effects.\(^{37,127}\) Amiodarone or procainamide may also be tried as these agents may terminate both ventricular or supraventricular arrhythmias. While verapamil may on rare occasions terminate ventricular tachycardia,\(^{128,129}\) it is contraindicated in patients with wide complex tachycardia of unknown origin as the majority are of ventricular origin and verapamil is ineffective and potentially hazardous in most of these patients.\(^{119,130,131}\)

Ventricular tachycardias

**Ventricular tachycardia.** This is defined as three or more consecutive ectopic ventricular impulses having approximately the same contour and separated by a fixed interval. It is sustained if either its duration is greater than 30 s or if it has to be terminated by
cardioversion or pacing in less than 30 s because of severe hypotension.

The rate usually ranges from 130 - 180 bpm (if it is greater than 180 bpm it is often considered as a form of ventricular flutter), the R-R interval may be slightly irregular (i.e. 0.01 - 0.02 s variations) or regular (Figure 6). Characteristically there is AV dissociation, although rarely retrograde conduction may occur and give rise to a P wave following, or less than 0.12 s before, the QRS complex. Fusion beats or ‘capture’ are pathognomonic of ventricular rhythm (Figure 7). The QRS complex during VT is usually uniform (mono-morphic); if it varies from beat to beat it is said to be polymorphic. Bidirectional VT is due to either two ventricular foci or one focus with aberration of the conducting impulses (e.g. alternating bundle-branch block).

The causes of VT include ischaemic heart disease, ventricular aneurysm, prolonged QTc syndrome, WPW syndrome, rheumatic fever, cardiomyopathy, drug toxicity (e.g. tricyclic antidepressants, digoxin, quinidine), hypokalaemia and hypomagnesaemia.

The clinical features of hypotension, shock, angina or cardiac failure are not pathognomonic of VT and may be associated with the disorder due to the underlying myocardial disease.

Treatment of VT involves cardioversion, if the patient is haemodynamically compromised, followed by class I or III agents, (e.g. amiodarone, sotalol, lignocaine, procainamide, bretylium). If the patient is not in cardiac failure or is not hypotensive, class I or III agents without cardioversion are often used.

In a multicentred study of patients with ventricular tachycardia, sotalol was found to be more effective than imipramine, mexiletine, procainamide or quinidine in preventing death and recurrences of ventricular tachyarrhythmias.132 In a double blind study of patients with acute sustained VT, sotalol (100 mg i.v. over 5 minutes converted 69% to sinus rhythm) was more effective than lignocaine (100 mg i.v. over 5 minutes converted 20% to sinus rhythm) in terminating VT, and the incidence of adverse effects was similar for both drugs.133

**Accelerated idioventricular rhythm.** This is also known as slow ventricular tachycardia and has a rate which ranges from 60 - 120 bpm (Figure 7). It usually occurs in patients with acute myocardial infarction, cardiomyopathy, or digoxin intoxication and is often transient, resistant to antiarrhythmic therapy, but rarely causes significant haemodynamic disturbance. Apart from correction of drug toxicities and electrolyte abnormalities, specific treatment for accelerated idioventricular rhythm is rarely necessary.

**Torsade de pointes**

This is a ventricular arrhythmia which has characteristics of both VT and VF. Some believe that it is an atypical VT,134 whereas others consider it an early phase of VF.135 However it is commonly considered to be a polymorphic VT which is preceded by QT prolongation (Figure 8) and is characterised by:

- a ventricular rate exceeding 200 bpm
- a widened QRS deflection which shows a continual change in amplitude giving the appearance of a modulated sine wave, or spindle
- a complete reversal of the QRS and T wave deflections as they appear to twist around the isoelectric line (hence the name)
- atrioventricular dissociation

The arrhythmia is associated with disorders listed in Table 3.136

The attacks are often short lived and self-limiting, and the patient often does not lose consciousness as a mean arterial pressure ranging from 20 - 40 mmHg is
Figure 8. Sinus rhythm at 88 bpm with a prolonged QTc interval, two ventricular ectopic beats, another sinus beat then torsade de pointes.

Figure 9. Ventricular fibrillation

usually present. However, the episodes may recur and become prolonged, particularly when hypokalaemia, hypomagnesaemia and an underlying bradycardia coexist, and may deteriorate into VF with clinical signs of cardiac arrest.

Table 3 Disorders associated with torsade de pointes

<table>
<thead>
<tr>
<th>Disorder</th>
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<tbody>
<tr>
<td>Complete atrioventricular or sinoatrial block</td>
</tr>
<tr>
<td>Prolonged QT syndromes</td>
</tr>
<tr>
<td>Hypokalaemia, hypomagnesaemia</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td>Myocarditis</td>
</tr>
<tr>
<td>Central nervous system disorders</td>
</tr>
<tr>
<td>Liquid-protein diets</td>
</tr>
<tr>
<td>Drugs</td>
</tr>
<tr>
<td>quinidine, procainamide, disopyramide, cisapride</td>
</tr>
<tr>
<td>phenothiazines (particularly thioridazine),</td>
</tr>
<tr>
<td>antihistamines, tricyclic antidepressants,</td>
</tr>
<tr>
<td>chloral hydrate,</td>
</tr>
<tr>
<td>erythromycin, azithromycin, fluconazole</td>
</tr>
</tbody>
</table>

Treatment of the arrhythmia includes resuscitation and defibrillation if it is prolonged or degenerates to VT or VF, and correction of the underlying disorder (i.e. hypokalaemia, hypomagnesaemia and drug toxicity). Otherwise, intravenous magnesium sulphate (bolus 5 - 10 mmol) is the agent of choice. Other treatments include potassium supplements to keep the plasma potassium at 4 mmol/L or greater, or (if the patient has an underlying bradycardia), producing a ventricular rate of 100 beats/min or greater using intravenous atropine (0.6 - 2.4 mg), an infusion of isoprenaline (2 to 6 µg/ min) or a pacemaker. Class Ia drugs are to be avoided as they tend to sustain the arrhythmia. Phenytin and lignocaine are generally ineffective, although bretylium (which now is no longer available) has been useful.

Ventricular fibrillation

This is characterised by a loss of an orderly sequence of ventricular myocardial contraction caused by a random and chaotic spread of electrical activity. The ECG reveals completely irregular, chaotic and deformed deflections of varying height, width and shape. (Figure 9). Death results from loss of cardiac output and absence of cerebral perfusion, unless direct current defibrillation successfully restores sinus rhythm. Diseases associated with ventricular fibrillation include, ischaemic heart disease, cardiomyopathies, electrocution, disorders associated with prolonged QTc syndrome.
and drugs (i.e. phenothiazines, antihistamines and pro-arrhythmic effect of quinidine, disopyramide, etc.).13,14

The treatment involves external cardiac massage and defibrillation at the earliest opportunity and correction of any underlying disorder.

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REFERENCES


75. Wagner GS, McIntosh HD. The use of drugs in achieving successful DC cardioversion. Prog Cardiovasc Dis 1969;11:431-442.


D. DURHAM, ET AL.


126. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 1992;268:2172-2288.


135. Standard and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 1992;268:2172-2288.

136. Stratmann HG, Kennedy H L. Torsades de pointes: de finition a nd m anagement. Mod C onc


