8 Peri-arrest arrhythmias

Introduction

Cardiac arrhythmias are relatively common in the peri-arrest period. They are common in the setting of acute myocardial infarction and may precipitate ventricular fibrillation (VF) or follow successful defibrillation. The treatment algorithms described in this chapter have been designed to enable the non-specialist advanced life support (ALS) provider to treat the patient effectively and safely in an emergency; for this reason they have been kept as simple as possible. If patients are not acutely ill there may be several other treatment options, including the use of drugs (oral or parenteral) that will be less familiar to the non-expert. In this situation there will be time to seek advice from cardiologists or other senior doctors with the appropriate expertise.

Guideline changes

There are relatively few changes from Guidelines 2005. Initial assessment of patients with suspected peri-arrest arrhythmias now uses the ABCDE approach (see the preventing cardiac arrest chapter). A single set of adverse features for tachy- and brady-arrhythmias has been introduced for consistency.

Sequence of actions

Assess the patient using the ABCDE approach. In all cases, give oxygen and insert an intravenous cannula and assess the patient for adverse features. Whenever possible, record a 12-lead ECG; this will help determine the precise rhythm, either before treatment or retrospectively, if necessary with the help of an expert. Correct any electrolyte abnormalities (e.g. K^+, Mg^{++}, Ca^{++}).

When you assess and treat any arrhythmia address two factors: the condition of the patient (stable versus unstable – determined by the absence or presence respectively of adverse features) and the nature of the arrhythmia.

Adverse features

The presence or absence of adverse symptoms or signs will dictate the appropriate treatment for most arrhythmias. The following adverse features indicate that a patient is potentially unstable because of the arrhythmia:

- Shock – hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness.
- Syncope – transient loss of consciousness due to global reduction in blood flow to the brain.
- Myocardial ischaemia – typical ischaemic chest pain and/or evidence of myocardial ischaemia on 12-lead ECG.
- Heart failure – pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).

Treatment options

Depending on the nature of the underlying arrhythmia and clinical status of the patient (in particular the presence or absence of adverse features) immediate treatments can be categorised under four headings:

1. Electrical (cardioversion for tachyarrhythmia or pacing for bradyarrhythmia)
2. Simple clinical intervention (e.g., vagal manoeuvres, fist pacing)
3. Pharmacological (drug treatment)
4. No treatment needed

Most drugs act more slowly and less reliably than electrical treatments, so electrical treatment is usually the preferred treatment for an unstable patient with adverse features.

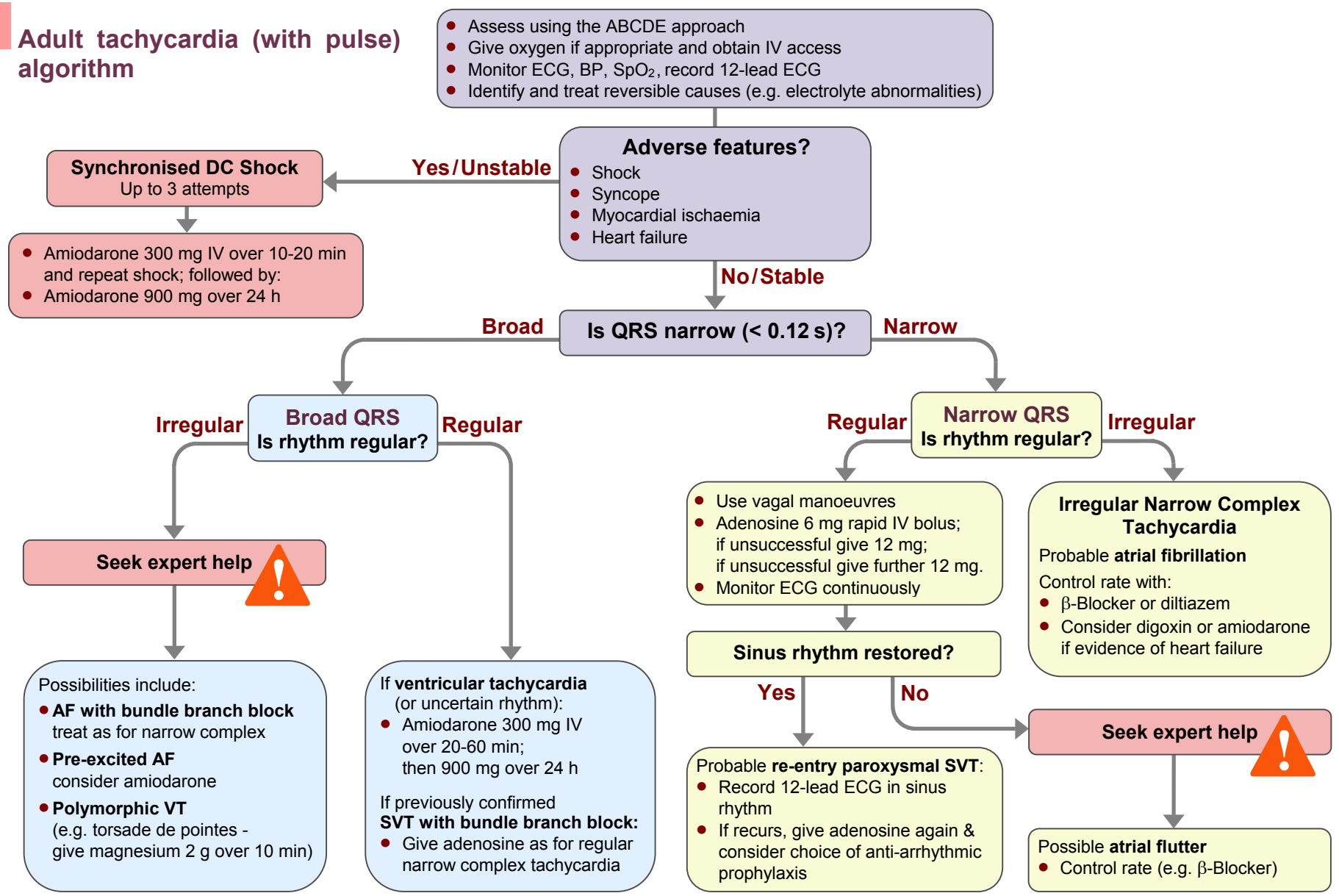
If a patient develops an arrhythmia during, or as a complication of some other condition (e.g. infection, acute myocardial infarction, heart failure), make sure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary. Once an arrhythmia has been treated successfully, repeat a 12-lead ECG to detect any abnormalities that may require treatment in the longer term.

Tachycardias

If the patient is unstable

If the patient is unstable and deteriorating (i.e., has adverse features caused by the tachycardia) synchronised cardioversion is the treatment of choice. In patients with otherwise normal hearts, serious signs and symptoms are uncommon if the ventricular rate is < 150 min^{-1}. Patients with impaired cardiac function, structural heart disease or other serious medical conditions (e.g. severe lung disease) may be symptomatic and unstable during arrhythmias with heart rates between 100 and 150 min^{-1}. If cardioversion fails to restore sinus rhythm, and the patient remains unstable, give amiodarone 300 mg IV over 10 - 20 min and re-attempt electrical cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h.

Adult tachycardia (with pulse) algorithm



Synchronised cardioversion

Carry out cardioversion under general anaesthesia or conscious sedation, administered by a healthcare professional competent in the technique being used. Ensure that the defibrillator is set to synchronised mode. For a broad-complex tachycardia or atrial fibrillation, start with 120-150 J biphasic shock (200 J monophasic) and increase in increments if this fails. Atrial flutter and regular narrow-complex tachycardia will often be terminated by lower energies: start with 70-120 J biphasic (100 J monophasic).

If the patient is stable

If there are no adverse features consider using drug treatment in the first instance (if any treatment is required). Assess the ECG and determine the QRS duration. If the QRS duration is greater than 0.12 s (3 small squares on standard ECG paper speed of 25 mm s^{-1}), this is a broad-complex tachycardia. If the QRS duration is less than 0.12 s, it is a narrow-complex tachycardia.

Broad-complex tachycardia

Broad-complex tachycardias (QRS ≥ 0.12 s) are usually ventricular in origin. Broad-complex tachycardias may be also caused by supraventricular rhythms with aberrant conduction (bundle branch block). In the unstable, peri-arrest patient assume that the rhythm is ventricular in origin and attempt synchronised cardioversion as described above. Conversely, if a patient with broad-complex tachycardia is stable, the next step is to determine if the rhythm is regular or irregular.

Regular broad-complex tachycardia

A regular broad-complex tachycardia is likely to be VT or a supraventricular rhythm with bundle branch block.

If the broad complex tachycardia is thought to be VT, treat with amiodarone 300 mg intravenously over 20-60 min, followed by an infusion of 900 mg over 24 h. If a regular broad-complex tachycardia is known to be a supraventricular arrhythmia with bundle branch block, and the patient is stable, use the strategy indicated for narrow-complex tachycardia (below).

Irregular broad-complex tachycardia

This is most likely to be atrial fibrillation (AF) with bundle branch block, but careful examination of a 12-lead ECG (if necessary by an expert) may enable confident identification of the rhythm. Other possible causes are AF with ventricular pre-excitation (in patients with Wolff-Parkinson-White (WPW) syndrome), or polymorphic VT (e.g. torsade de pointes), but polymorphic VT is unlikely to be present without adverse features. Seek expert help with the assessment and treatment of irregular broad-complex tachyarrhythmia.

Treat torsade de pointes VT immediately by stopping all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium

sulphate 2 g IV over 10 min (= 8 mmol, 4 ml of 50% magnesium sulphate). Obtain expert help, as other treatment (e.g. overdrive pacing) may be indicated to prevent relapse once the arrhythmia has been corrected. If adverse features develop, which is common, arrange immediate synchronised cardioversion. If the patient becomes pulseless, attempt defibrillation immediately (ALS algorithm).

Narrow-complex tachycardia

Examine the ECG to determine if the rhythm is regular or irregular.

Regular narrow-complex tachycardias include:[262]

- sinus tachycardia;
- AV nodal re-entry tachycardia (AVNRT) – the commonest type of regular narrow-complex tachyarrhythmia;
- AV re-entry tachycardia (AVRT) – due to WPW syndrome;
- atrial flutter with regular AV conduction (usually 2:1).

An irregular narrow-complex tachycardia is most likely to be AF or sometimes atrial flutter with variable AV conduction ('variable block').

Regular narrow-complex tachycardia

Sinus tachycardia

Sinus tachycardia is not an arrhythmia. This is a common physiological response to stimuli such as exercise or anxiety. In a sick patient it may occur in response to many conditions including pain, infection, anaemia, blood loss, and heart failure. Treatment is directed at the underlying cause; trying to slow sinus tachycardia that has occurred in response to most of these conditions will make the situation worse. Do not attempt to treat sinus tachycardia with cardioversion or anti-arrhythmic drugs.

AVNRT and AVRT (paroxysmal supraventricular tachycardia)

AV nodal re-entry tachycardia is the commonest type of paroxysmal supraventricular tachycardia (SVT), often seen in people without any other form of heart disease. It is relatively uncommon in the peri-arrest setting. It causes a regular, narrow-complex tachycardia, often with no clearly visible atrial activity on the ECG. The heart rate is commonly well above the typical range of sinus rhythm at rest (60-100 min^{-1}). It is usually benign, unless there is additional, co-incidental, structural heart disease or coronary disease, but it may cause symptoms that the patient finds frightening.

AV re-entry tachycardia occurs in patients with the WPW syndrome, and is also usually benign, unless there is additional structural heart disease. The common type of AVRT is a regular narrow-complex tachycardia, usually having no visible atrial activity on the ECG.

Atrial flutter with regular AV conduction (often 2:1 block)

This produces a regular narrow-complex tachycardia. It may be difficult to see atrial activity and identify flutter waves in the ECG with confidence, so the rhythm may be indistinguishable, at least initially, from AVNRT or AVRT.

Typical atrial flutter has an atrial rate of about 300 min^{-1}, so atrial flutter with 2:1 conduction produces a tachycardia of about 150 min^{-1}. Much faster rates (160 min^{-1} or more) are unlikely to be caused by atrial flutter with 2:1 conduction. Regular tachycardia with slower rates (e.g. 125-150) may be due to atrial flutter with 2:1 conduction, usually when the rate of the atrial flutter has been slowed by drug therapy.

Treatment of regular narrow-complex tachycardia

If the patient is unstable, with adverse features caused by the arrhythmia, attempt synchronised electrical cardioversion. It is reasonable to give adenosine to an unstable patient with a regular narrow-complex tachycardia while preparations are being made for synchronised cardioversion. However, do not delay electrical cardioversion if adenosine fails to restore sinus rhythm.

In the absence of adverse features:

- Start with vagal manoeuvres. Carotid sinus massage or the Valsalva manoeuvre will terminate up to a quarter of episodes of paroxysmal SVT. Record an ECG (preferably multi-lead) during each manoeuvre. If the rhythm is atrial flutter, slowing of the ventricular response will often occur and reveal flutter waves.
- If the arrhythmia persists and is not atrial flutter, give adenosine 6 mg as a rapid intravenous bolus. Use a relatively large cannula and large (e.g., antecubital) vein. Warn the patient that they will feel unwell and probably experience chest discomfort for a few seconds after the injection. Record an ECG (preferably multi-lead) during the injection. If the ventricular rate slows transiently, but then speeds up again, look for atrial activity, such as atrial flutter or other atrial tachycardia, and treat accordingly. If there is no response to adenosine 6 mg, give a 12 mg bolus. If there is no response give one further 12 mg bolus. Apparent lack of response to adenosine will occur if the bolus is given too slowly or into a peripheral vein.
- Vagal manoeuvres or adenosine will terminate almost all AVNRT or AVRT within seconds. Failure to terminate a regular narrow-complex tachycardia with adenosine suggests an atrial tachycardia such as atrial flutter (unless the adenosine has been injected too slowly or into a small peripheral vein).
- If adenosine is contra-indicated, or fails to terminate a regular narrow complex tachycardia without demonstrating that it is atrial flutter, consider giving a calcium-channel blocker, for example verapamil 2.5 - 5 mg intravenously over 2 min.

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV block. Record a 12-lead ECG to identify the rhythm. If the patient is unstable, with adverse features caused by the arrhythmia, attempt synchronised cardioversion.[263]

If there are no adverse features, treatment options include:

- rate control by drug therapy;
- rhythm control using drugs to encourage chemical cardioversion;
- rhythm control by electrical cardioversion;
- treatment to prevent complications (e.g. anticoagulation).

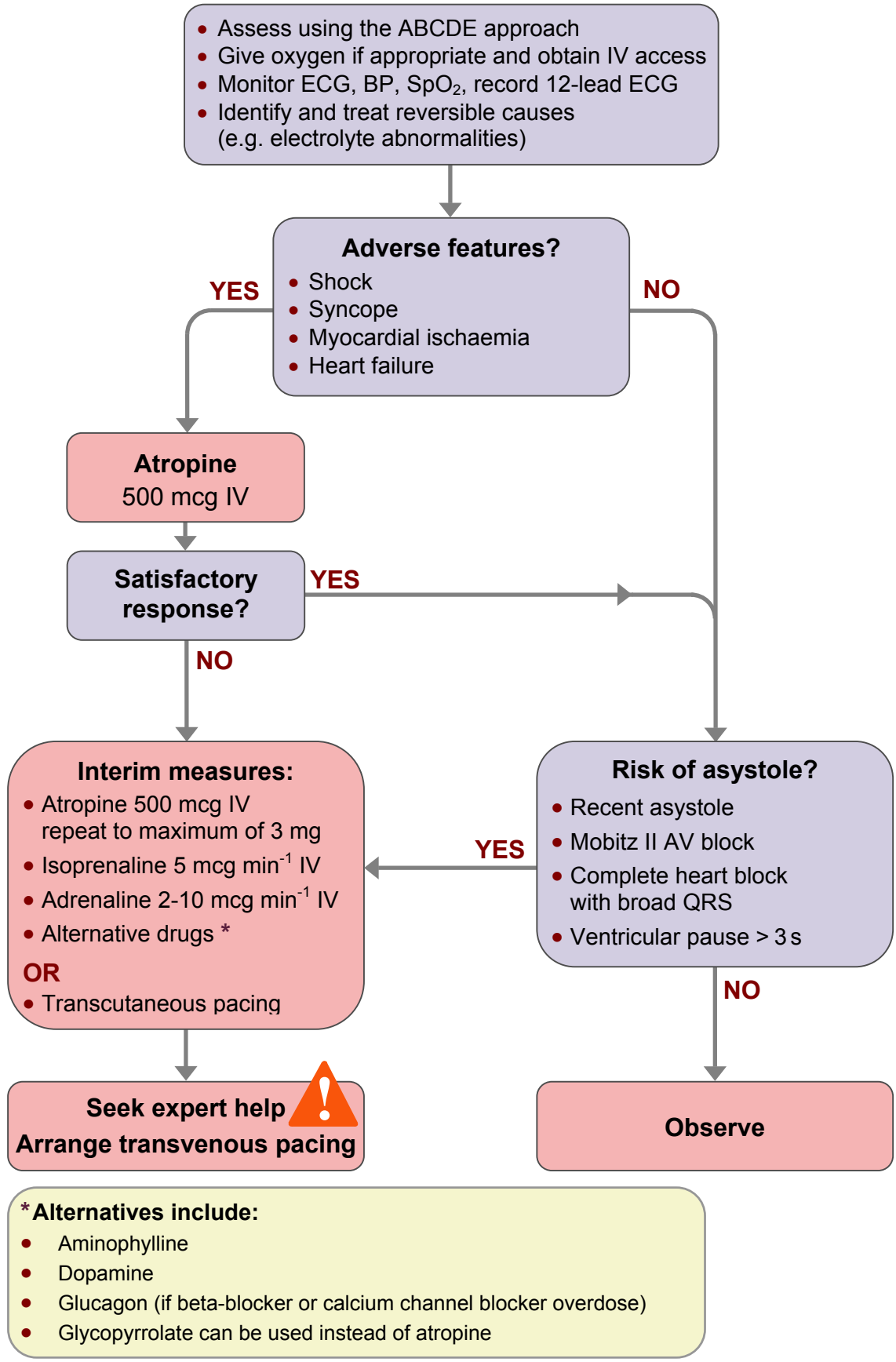
Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF the greater is the likelihood of atrial thrombus developing. In general, patients who have been in AF for more than 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least three weeks, or unless trans-oesophageal echocardiography has shown the absence of atrial thrombus. If the clinical situation dictates that cardioversion is needed more urgently, give either regular low-molecular-weight heparin in therapeutic dose or an intravenous bolus injection of unfractionated heparin followed by a continuous infusion to maintain the activated partial thromboplastin time (APTT) at 1.5 to 2 times the reference control value. Continue heparin therapy and commence oral anticoagulation after successful cardioversion. Seek expert advice on the duration of anticoagulation, which should be a minimum of 4 weeks, often substantially longer.

If the aim is to control heart rate, the usual drug of choice is a beta-blocker. Diltiazem or verapamil may be used in patients in whom beta blockade is contraindicated or not tolerated. An intravenous preparation of diltiazem is available in some countries but not in the UK. Digoxin may be used in patients with heart failure. Amiodarone may be used to assist with rate control but is most useful in maintaining rhythm control. Magnesium is also used but the data supporting this are limited. When possible seek expert help in selecting the best choice of treatment for rate control in each individual patient.

If the duration of AF is less than 48 h and rhythm control is considered appropriate, chemical cardioversion may be attempted. Seek expert help with the use of drugs such as flecainide. Do not use flecainide in the presence of heart failure, known left ventricular impairment or ischaemic heart disease, or a prolonged QT interval. Amiodarone (300 mg intravenously over 20-60 min followed by 900 mg over 24 h) may also be used but is less likely to achieve prompt cardioversion. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

Seek expert help if any patient with AF is known or found to have ventricular pre-excitation (WPW syndrome). Avoid using adenosine, diltiazem, verapamil, or digoxin in patients with pre-excited AF or atrial flutter as these drugs block the AV node and cause a relative increase in pre-excitation.

Adult bradycardia algorithm



Bradycardia

Bradycardia is defined as a heart rate of < 60 min^{-1}. It may be:

- physiological (e.g., in athletes);
- cardiac in origin (e.g., atrioventricular block or sinus node disease);
- non-cardiac in origin (e.g., vasovagal, hypothermia, hypothyroidism, hyperkalaemia);
- drug-induced (e.g., beta blockade, diltiazem, digoxin, amiodarone).

Assess the patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for adverse features. Treat any reversible causes of bradycardia identified in the initial assessment. If adverse signs are present start to treat the bradycardia. Initial treatment is pharmacological, with pacing being reserved for patients unresponsive to pharmacological treatment or with risks factors for asystole.

Pharmcological treatment

If adverse signs are present, give atropine, 500 mcg, intravenously and, if necessary, repeat every 3-5 min to a total of 3 mg. Doses of atropine of less than 500 mcg have been reported to cause paradoxical slowing of the heart rate.[264] In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate.[265] Use atropine cautiously in the presence of acute coronary ischaemia or myocardial infarction; increased heart rate may worsen ischaemia or increase the zone of infarction. Do not give atropine to patients with cardiac transplants. Their hearts are denervated and will not respond to vagal blockade by atropine, which may cause paradoxical sinus arrest or high-grade AV block in these patients.[266]

If bradycardia with adverse signs persist despite atropine, consider cardiac pacing. If pacing cannot be achieved promptly consider the use of second-line drugs. Seek expert help to select the most appropriate choice. In some clinical settings second-line drugs may be appropriate before the use of cardiac pacing. For example consider giving intravenous glucagon if a beta-blocker or calcium channel blocker is a likely cause of the bradycardia. Consider using digoxin-specific antibody fragments for bradycardia due to digoxin toxicity. Consider using theophylline (100-200 mg by slow intravenous injection) for bradycardia complicating acute inferior wall myocardial infarction, spinal cord injury or cardiac transplantation.

Pacing

Transcutaneous pacing

Initiate transcutaneous pacing immediately if there is no response to atropine, or if atropine is unlikely to be effective. Transcutaneous pacing can be painful and may fail to achieve effective electrical capture (i.e. a QRS complex after the pacing stimulus) or fail to achieve a mechanical response (i.e. palpable pulse). Verify electrical capture on the monitor or ECG and check that it is producing a pulse. Reassess the patient's condition (ABCDE). Use analgesia and sedation as necessary to control pain; sedation may compromise respiratory effort so continue to reassess the patient at frequent intervals.

Fist pacing

If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment.[267-269] Give serial rhythmic blows with the closed fist over the left lower edge of the sternum to stimulate the heart at a rate of 50-70 min^{-1}.

Transvenous pacing

Seek expert help to assess the need for temporary transvenous pacing and to intitiate this when appropriate. Temporary transvenous pacing should be considered if there is documented recent asystole (ventricular standstill of more than 3 s), Mobitz type II AV block; complete (third-degree) AV block (especially with broad QRS or initial heart rate <40 beats min^{-1}).