Peri-arrest Arrhythmias

CHAPTER 11

Learning outcomes

To understand:

- **The importance of arrhythmias that may precede or follow a cardiac arrest**
- **How to assess peri-arrest arrhythmias**
- **The principles of treatment of peri-arrest arrhythmias**

Introduction

Rhythm abnormalities that occur in the peri-arrest period may be considered in two main categories:

- **Arrhythmias that may lead to cardiac arrest** - many rhythm abnormalities occur without causing cardiac arrest: they are a relatively common complication of acute myocardial infarction (AMI) but are also common in patients with other cardiac abnormalities and in people who do not have coronary disease or structural heart disease. Untreated, some of these arrhythmias may lead to cardiac arrest or to avoidable deterioration in the patient's condition. Others may require no immediate treatment.

- **Arrhythmias that occur after initial resuscitation from cardiac arrest** - these often indicate that the patient's condition is still unstable and that there is a risk of deterioration or further cardiac arrest.

You should be able to recognise common arrhythmias and to know how to assess whether or not they require immediate treatment. The treatment algorithms described in this section have been designed to enable the non-specialist advanced life support (ALS) provider to treat a 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 treatment options, including the use of drugs (oral or parenteral) that will be less familiar to the non-expert. In this situation you should, whenever possible, seek advice from cardiologists or other senior doctors with the appropriate expertise.

Sequence of actions

When an arrhythmia is present or suspected, start by assessing the patient using the ABCDE approach, including early establishment of cardiac monitoring (see Chapter 8). Assess the patient specifically for adverse features (see below). Insert an intravenous cannula and, if appropriate, give oxygen. Whenever possible, record a 12-lead ECG at the earliest opportunity. This will help to identify the precise rhythm, either before treatment or retrospectively, if necessary with the help of an expert. Clinical assessment is of limited value in identifying the precise rhythm abnormality.

When you assess any patient with an arrhythmia address two factors:

1. the condition of the patient (presence or absence of adverse features)

2. the nature of the arrhythmia.

Adverse features

The presence or absence of adverse signs or symptoms will dictate the urgency and choice of treatment for most arrhythmias. The following adverse features indicate that a patient is unstable and at risk of deterioration, wholly or partly because of the arrhythmia:

- Shock - hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold extremities, confusion or impaired consciousness.

- Syncope - transient loss of consciousness because of global reduction in blood flow to the brain.

- Heart failure - pulmonary oedema and/or raised jugular venous pressure (with or without peripheral oedema and liver enlargement).

- Myocardial ischaemia - typical ischaemic chest pain and/or evidence of myocardial ischaemia on a 12-lead ECG.

- Extremes of heart rate - in addition to the above adverse features it may be appropriate to consider extremes of heart rate as adverse signs in themselves, requiring more urgent assessment and treatment than less extreme tachycardia or bradycardia with no adverse signs.

  (a) Extreme tachycardia: when heart rate increases, diastole is shortened to a greater degree than systole. Rhythm abnormalities that cause very fast heart rates (e.g. > 150 min^{-1}) reduce cardiac output dramatically (because diastole is very short and the heart does not have time to fill properly) and reduce coronary blood flow (because this mostly occurs during diastole),

potentially causing myocardial ischaemia. The faster the heart rate, the less well it will be tolerated.

(b) Extreme bradycardia: in general, the slower the bradycardia the less well it will be tolerated and heart rates below 40 min^{-1} are often tolerated poorly. This is especially so when people have severe heart disease and cannot compensate for the bradycardia by increasing stroke volume. Some people with very severe heart disease require faster than normal heart rates to maintain cardiac output, and even a 'normal' heart rate may be inappropriately slow for them.

Treatment options

Depending on the clinical status of the patient (i.e. the presence or absence of adverse features) and the nature of the arrhythmia, 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, percussion 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 as a complication of some other condition (e.g. infection, AMI, heart failure), make sure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary.

Subsequent monitoring and treatment

After successful treatment of an arrhythmia continue to monitor the patient until you are confident that the risk of further arrhythmia is low. Remember always to record a 12-lead ECG **after** successful treatment of an arrhythmia because this may show abnormalities (or absence of abnormalities) that will be important in planning future management. Correct all reversible factors that may predispose to further arrhythmia. Ensure that appropriate further expert help and advice is obtained at the most appropriate time for the patient.

Tachyarrhythmia

If the patient has adverse features

These imply that the patient's condition is unstable and at risk of deterioration; if this appears to be because of the presence of tachyarrhythmia, attempt to correct this using synchronised cardioversion (Figure 11.1). In people with otherwise normal hearts, adverse signs and symptoms are uncommon if the heart 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 terminate the arrhythmia, and adverse features persist, give amiodarone 300 mg IV over 10 - 20 min and attempt further synchronised cardioversion. The loading dose of amiodarone can be followed by an infusion of 900 mg over 24 h, given into a large vein (preferably via central venous cannula).

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 deliver a synchronised shock. This delivers the shock to coincide with the R wave. An unsynchronised shock could coincide with a T wave and cause ventricular fibrillation (VF).

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-energy shocks: start with 70 - 120 J biphasic (100 J monophasic). For atrial fibrillation and flutter use anteroposterior defibrillator pad positions when it is practicable to do so.

When delivering the shock, press the shock button and keep it pressed until after the shock has occurred - there may be a slight delay before the shock is delivered.

If a second shock is needed, reactivate the synchronisation switch if necessary.

If the patient has no adverse features

If there are no adverse features consider using drug treatment in the first instance. Assess the ECG and measure the QRS duration. If the QRS duration is 0.12 s (3 small squares at ECG paper speed 25 mm s^{-1}) or more this is a broad complex tachycardia. If the QRS duration is < 0.12 s it is a narrow complex tachycardia. Following any drug therapy, continue to reassess the patient (ABCDE)

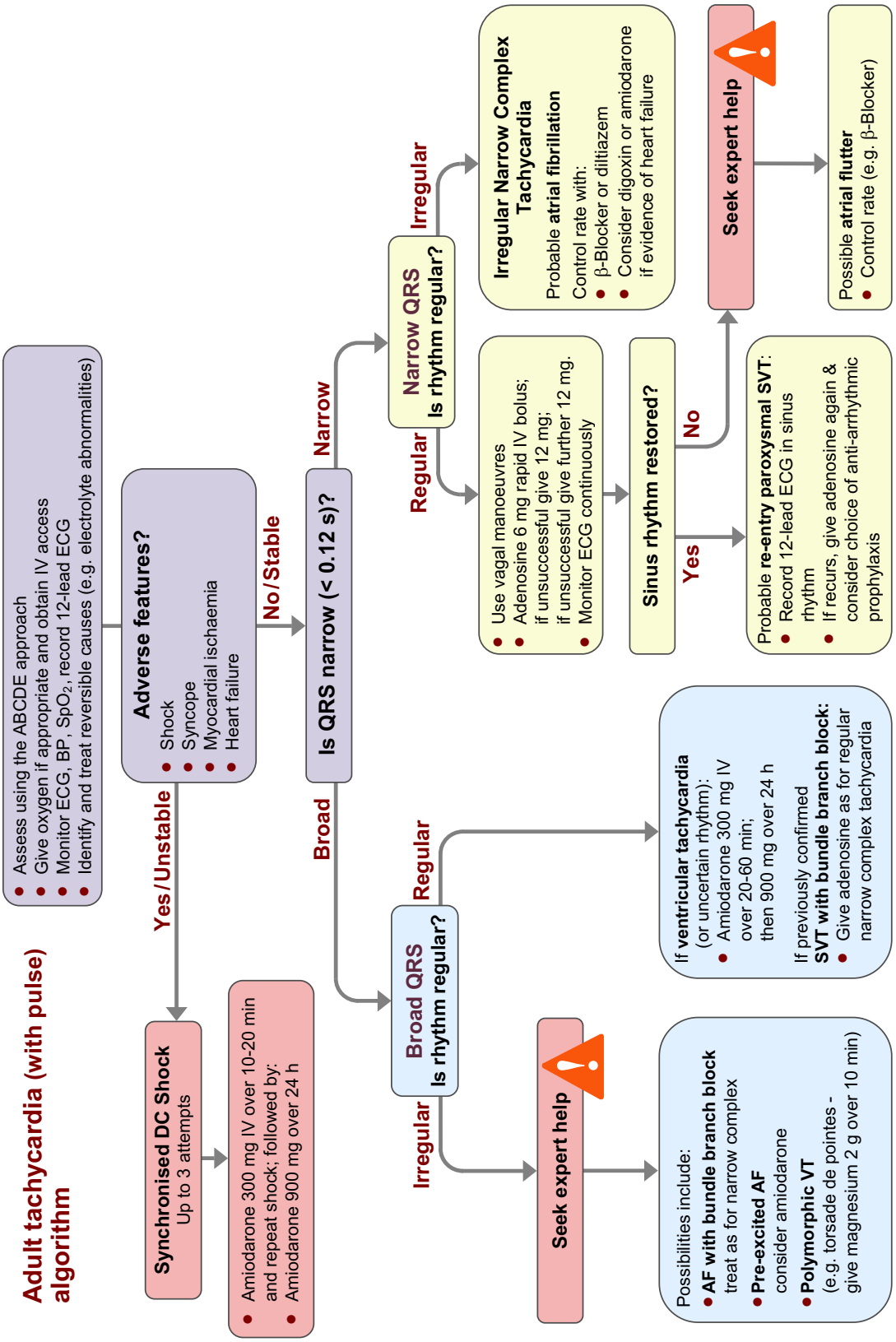

Figure 11.1 Tachycardia algorithm

and monitor heart rate and rhythm to assess the response to treatment. Some anti-arrhythmic drugs cause myocardial depression, which may cause or worsen heart failure or hypotension, and in some cases an anti-arrhythmic drug may cause other tachyarrhythmia or provoke severe bradycardia.

Broad-complex tachycardia

Broad-complex tachycardia (QRS ≥ 0.12 s) may be ventricular in origin or may be a supraventricular rhythm with aberrant conduction (i.e. bundle branch block). In the patient with adverse features the distinction is irrelevant. Attempt synchronised cardioversion as described above. If a patient has a broad-complex tachycardia but no adverse features, next determine whether the rhythm is regular or irregular.

Regular broad-complex tachycardia

A regular broad-complex tachycardia may be ventricular tachycardia (VT) or a supraventricular rhythm with bundle branch block.

If the broad complex tachycardia is considered to be VT, treat with amiodarone 300 mg intravenously over 20 - 60 minutes, 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 treatment 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 provide 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 by stopping immediately all drugs known to prolong the QT interval. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulphate 2 g IV over 10 min. 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 sinus tachycardia, atrioventricular nodal re-entry tachycardia (AVNRT) - the commonest type of regular narrow-complex tachyarrhythmia, atrioventricular re-entry tachycardia (AVRT) - due to WPW syndrome, and 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 situations will make the situation worse. Do not attempt to treat sinus tachycardia with cardioversion or anti-arrhythmic drugs.

AVNRT and AVRT (paroxysmal supraventricular tachycardia)

Atrioventricular 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 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 usually well above the upper limit of sinus rate at rest (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.

Atrioventricular 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 with no visible atrial activity on the ECG. Like AVNRT, it may cause frightening symptoms.

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 on 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 (125 - 150 min^{-1}) may be caused by 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 tachyarrhythmia

If the patient has adverse features and is at risk of deterioration because of the tachyarrhythmia, perform synchronised cardioversion. In this situation it is reasonable to attempt vagal manoeuvres (see below) or to give intravenous adenosine (see below) to a patient with a regular narrow-complex tachyarrhythmia while preparations are being made for synchronised cardioversion. However, do not delay electrical cardioversion if these treatments fail to terminate the arrhythmia.

In the absence of adverse features:

1. 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 12-lead) during each manoeuvre. If the rhythm is atrial flutter with 2:1 conduction, slowing of the ventricular response will often occur and reveal flutter waves.

2. If the arrhythmia persists and is not atrial flutter, give adenosine 6 mg as a very 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 12-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.

3. 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).

4. 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.

Rapid narrow-complex tachycardia with no pulse

Rarely, a very rapid (usually > 250 min^{-1}) narrow-complex tachycardia can impair cardiac output to such an extent that the pulse may be impalpable and consciousness impaired. If the patient is pulseless and unconscious this situation is pulseless electrical activity (PEA) and you should start CPR. As the arrhythmia is potentially treatable by DC shock the most appropriate treatment then is immediate synchronised cardioversion, so this is an exception to the non-shockable limb of the ALS algorithm (Chapter 6).

Irregular narrow-complex tachycardia

An irregular narrow-complex tachycardia is most likely to be AF with a rapid ventricular response or, less commonly, atrial flutter with variable AV conduction. Record a 12-lead ECG to identify the rhythm.

If the patient has adverse features and is at risk of deterioration because of the tachyarrhythmia, perform synchronised cardioversion. In the absence of contraindications, start anticoagulation, initially with low-molecular-weight heparin or unfractionated heparin (see below), at the earliest opportunity. Do not allow this treatment to delay cardioversion.

If there are no adverse features, immediate treatment options include:

- rate control by drug therapy;
- rhythm control using drugs to achieve chemical cardioversion;
- rhythm control by synchronised 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 > 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least 3 weeks, or unless trans-oesophageal echocardiography has detected no evidence 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 - 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 may be used in patients in whom beta blockade is contraindicated or not tolerated. 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 < 48 h and rhythm control is considered the appropriate strategy, chemical cardioversion may be appropriate. 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 be used to attempt chemical cardioversion but is less often effective and takes longer. Electrical cardioversion remains an option in this setting and will restore sinus rhythm in more patients than chemical cardioversion.

Seek expert help if a 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 may cause a relative increase in pre-excitation.

Bradyarrhythmia

Bradycardia is defined as a resting 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 signs (Figure 11.2). Treat any reversible causes of bradycardia identified in the initial assessment.

If the patient has adverse features

If adverse features are present start to treat the bradycardia. Initial treatment is usually pharmacological; pacing is used for patients in whom initial pharmacological treatment is ineffective or inadequate and those with risk factors for asystole.

Pharmacological treatment for bradycardia

If adverse features 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 < 500 mcg can cause paradoxical slowing of the heart rate. In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate. Use atropine cautiously in the presence of acute myocardial ischaemia or myocardial infarction; the resulting increase in heart rate may worsen ischaemia or increase the size of the infarct.
If bradycardia with adverse signs persists despite atropine, cardiac pacing should be considered. 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 caused by 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. 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. Other options for second-line drug therapy include infusion of isoprenaline (5 mcg min^{-1} starting dose), adrenaline (2 - 10 mcg min^{-1}), or dopamine (2.5 - 10 mcg kg^{-1} min^{-1}).

Cardiac pacing for bradycardia

In a patient with bradycardia and adverse features, if there is no response to atropine or if atropine is unlikely to be effective, initiate transcutaneous pacing immediately (see chapter 10). In the presence of severe bradycardia, use percussion pacing as an interim measure until transcutaneous pacing is achieved. 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 beats min^{-1}.

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; remember that sedation may compromise respiratory effort so continue to reassess the patient at frequent intervals. Attempt to identify the cause of the bradyarrhythmia.

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

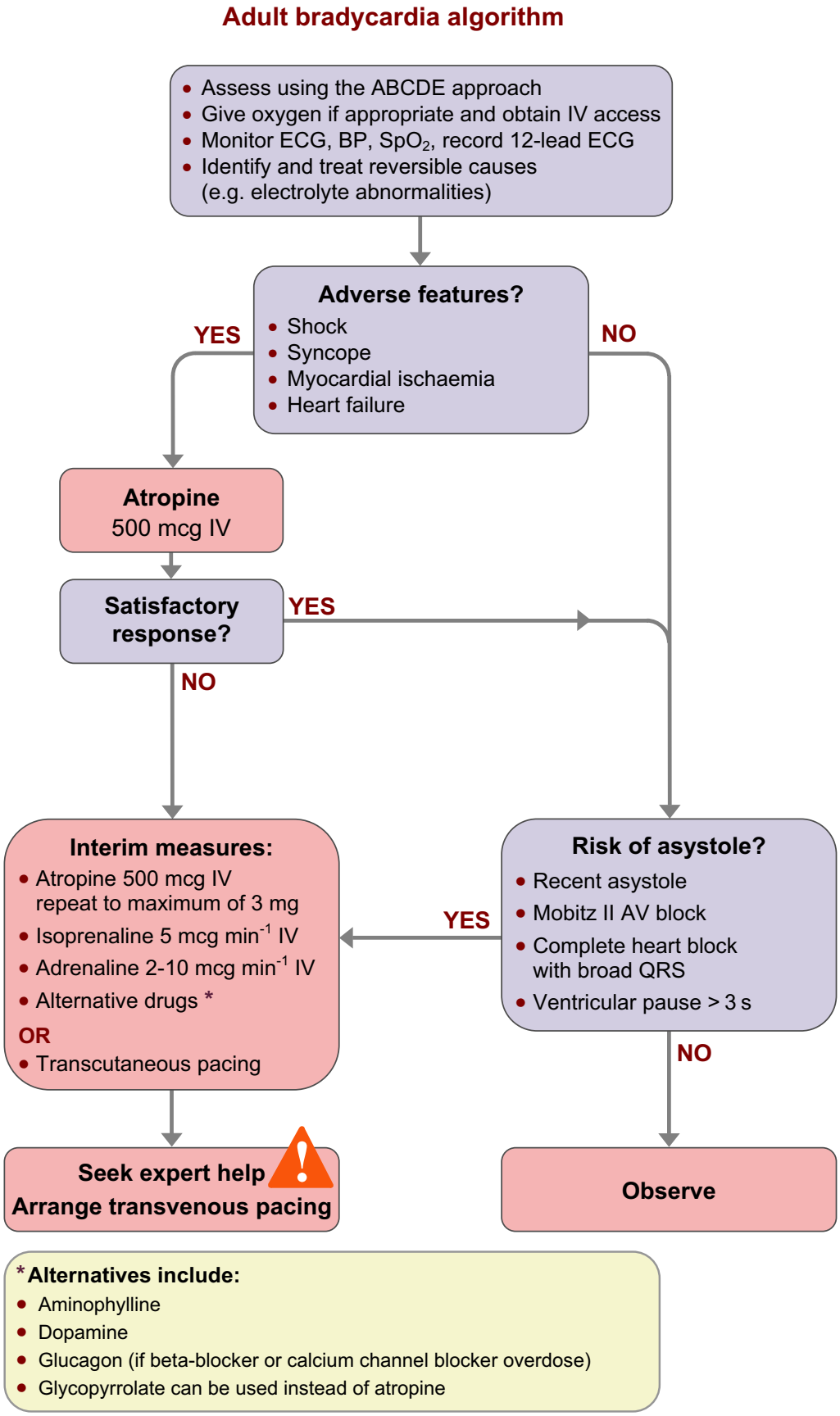

Figure 11.2 Bradycardia algorithm

If the patient has no adverse features

In a patient with bradycardia and no adverse features or high risk of progression to asystole, do not initiate immediate treatment. Continue to monitor the patient. Assess the patient to identify the cause of the bradycardia. If the cause is physiological or reversible (e.g. by stopping suppressant drug therapy) no further treatment may be needed. Seek expert help to arrange appropriate further assessment and treatment for those with other causes of bradycardia.

Key learning points

- Arrhythmias occurring after resuscitation from cardiac arrest and ROSC may need treatment to stabilise the patient and prevent recurrence of cardiac arrest.
- In other settings some arrhythmias may require prompt treatment to prevent deterioration, including progression to cardiac arrest, and others do not require immediate treatment.
- The urgency for treatment and the best choice of treatment is determined by the condition of the patient (presence or absence of adverse features) and by the nature and cause of the arrhythmia.
- Assessment of a patient with an arrhythmia should follow the ABCDE approach.
- Whenever possible the arrhythmia should be documented on a 12-lead ECG.

Further reading

Blomstrom-Lundqvist C, Scheinmann M M (Co-chairs). American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines. ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias. www.escardio.org

Moya A, Sutton R (Co-chairs). The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC). Guidelines for the diagnosis and management of syncope (version 2009). www.escardio.org

Ryden L, Fuster F (Co-chairs). American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation. www.escardio.org

Vardas P E (Chairperson). The Task Force for Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology. Guidelines for cardiac pacing and cardiac resynchronization therapy. www.escardio.org

Zipes D P, Camm J A (Co-chairs). A report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden death. www.escardio.org