Peri-Arrest Arrhythmias

Introduction

Cardiac arrhythmias are well-recognised complications of myocardial infarction. They may precede ventricular fibrillation (VF) or follow successful defibrillation. The treatment algorithms described in this section 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

The principles of treating peri-arrest arrhythmias remain unchanged from Guidelines 2000. The bradycardia algorithm is virtually unchanged. Previous Resuscitation Council (UK) guidelines have included three separate tachycardia algorithms: broad-complex tachycardia, narrow-complex tachycardia, and atrial fibrillation. In the peri-arrest setting many treatment principles are common to all the tachycardias. For this reason, they have been combined into a single tachycardia algorithm.

Sequence of actions

In all cases, give oxygen and insert an intravenous cannula while the arrhythmia is assessed. 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^{++}).

Assessment and treatment of all arrhythmias address two factors: the condition of the patient (stable versus unstable) and the nature of the arrhythmia.

Adverse signs

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

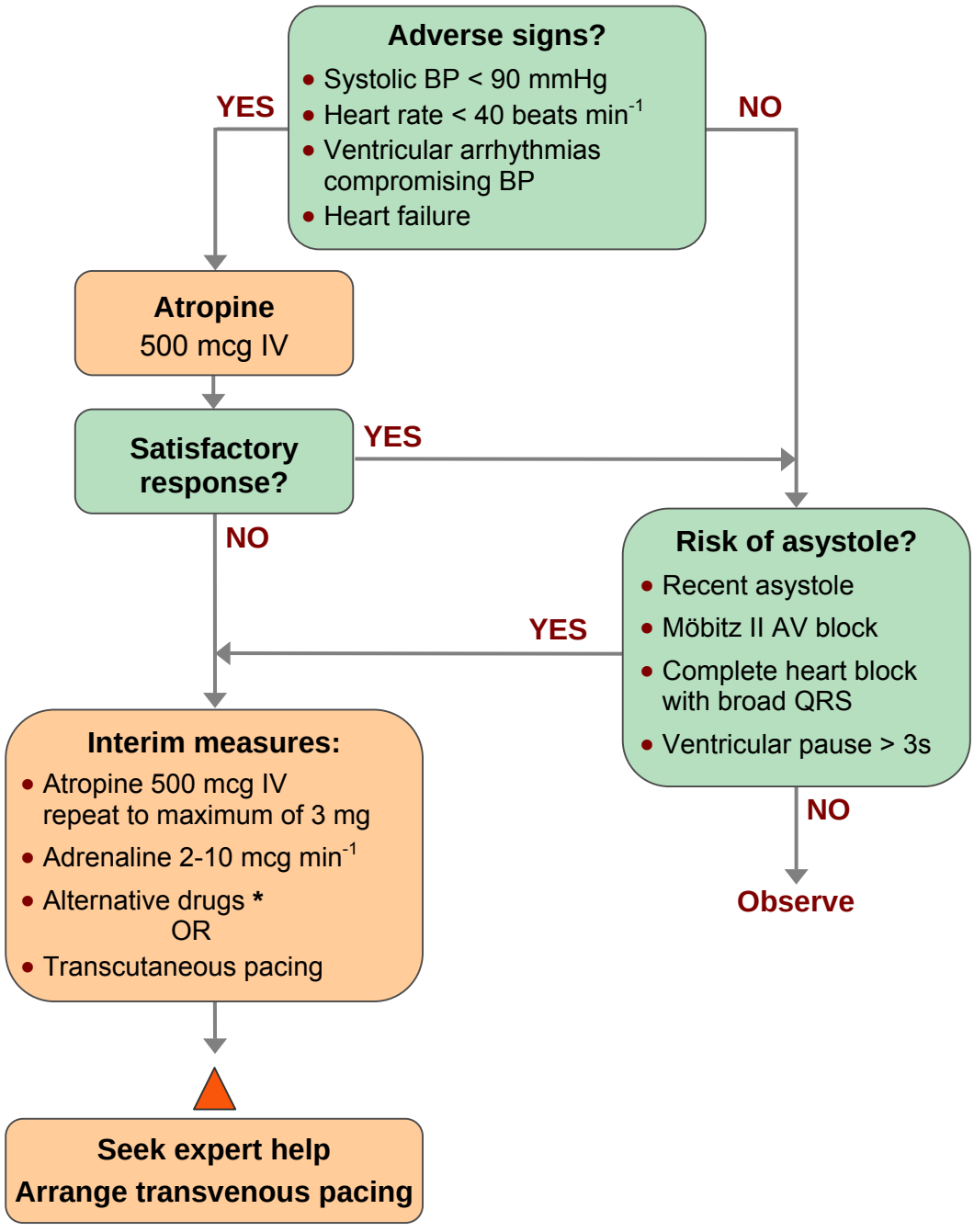
Clinical evidence of low cardiac output

Pallor, sweating, cold, clammy extremities (increased sympathetic activity), impaired consciousness (reduced cerebral blood flow), and hypotension (e.g. systolic blood pressure < 90 mmHg).

Bradycardia Algorithm

(includes rates inappropriately slow for haemodynamic state)

If appropriate, give oxygen, cannulate a vein, and record a 12-lead ECG



*** Alternatives include:**

Aminophylline
Isoprenaline
Dopamine
Glucagon (if beta-blocker or calcium-channel blocker overdose)
Glycopyrrolate can be used instead of atropine

Excessive tachycardia

Very high heart rates (e.g. > 150 min^{-1}) reduce coronary blood flow and can cause myocardial ischaemia. Broad-complex tachycardias are tolerated by the heart less well than narrow-complex tachycardias.

Excessive bradycardia

This is defined as a heart rate of < 40 beats min^{-1}, but rates of < 60 beats min^{-1} may not be tolerated by patients with poor cardiac reserve.

Heart failure

Pulmonary oedema indicates failure of the left ventricle, and raised jugular venous pressure and hepatic engorgement indicate failure of the right ventricle.

Chest pain

The presence of chest pain implies that the arrhythmia, particularly a tachyarrhythmia, is causing myocardial ischaemia.

Treatment options

Having determined the rhythm and presence or absence of adverse signs, there are broadly three options for immediate treatment:

- anti-arrhythmic (and other) drugs;
- attempted electrical cardioversion;
- cardiac pacing.

Anti-arrhythmic drugs act more slowly and less reliably than electrical cardioversion in converting a tachycardia to sinus rhythm. Thus, drugs tend to be reserved for stable patients without adverse signs, and electrical cardioversion is usually the preferred treatment for the unstable patient displaying adverse signs.

Once an arrhythmia has been treated successfully, repeat the 12-lead ECG to enable detection of any underlying abnormalities that may require long-term therapy.

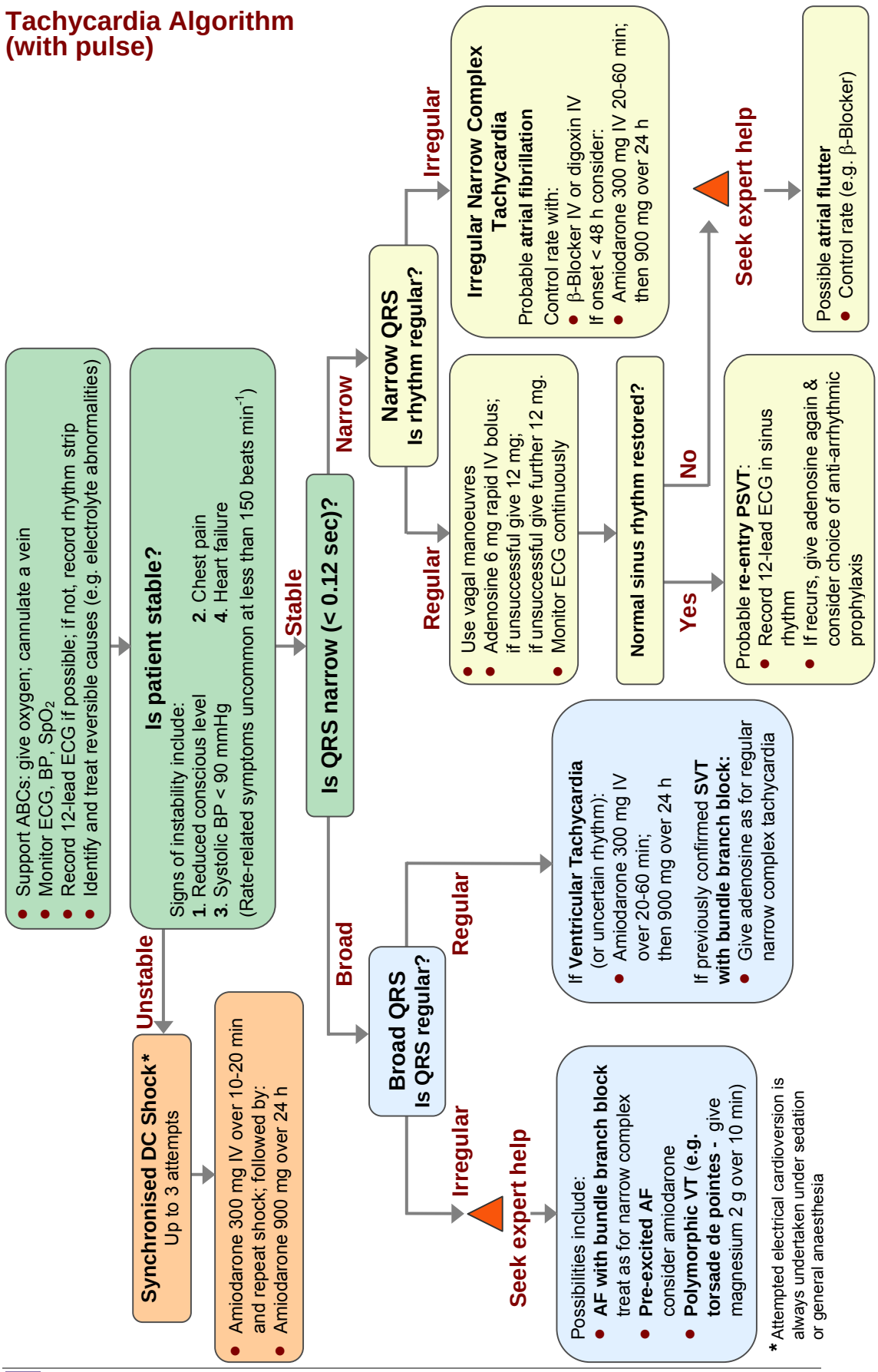
Bradycardia

Bradycardia is defined as a heart rate of < 60 beats min^{-1}. However, it is more helpful to classify a bradycardia as absolute (< 40 beats min^{-1}), or relative when the heart rate is inappropriately slow for the haemodynamic state of the patient.

The first step in the assessment of bradycardia is to determine if the patient is unstable. The following adverse signs may indicate instability:

- systolic blood pressure < 90 mm Hg;
- heart rate < 40 beats min^{-1};
- ventricular arrhythmias requiring suppression;
- heart failure.

Tachycardia Algorithm (with pulse)



If adverse signs are present, give atropine 500 microgram intravenously and, if necessary, repeat every 3 to 5 min, to a total of 3 mg. If a satisfactory response is achieved with atropine, or if the patient is stable, next determine if there is a risk of asystole, as indicated by:

- recent asystole;
- Möbitz type II AV block;
- complete (3rd degree) heart block (especially with broad QRS or initial heart rate less than 40 min^{-1});
- ventricular standstill of more than 3 sec.

Cardiac pacing is likely to be required if there is a risk of asystole or if the patient is unstable and has failed to respond satisfactorily to atropine. Under these circumstances the definitive treatment is transvenous pacing. One or more of the following interventions can be used to improve the patient's condition while waiting for the appropriate personnel and facilities:

- transcutaneous pacing;
- adrenaline infusion in the range of 2-10 microgram min^{-1} titrated against the response.

Other drugs that can be given for symptomatic bradycardia include dopamine, isoprenaline, and theophylline. Consider giving intravenous glucagon if beta-blockers or calcium-channel blockers are a potential cause of the bradycardia.

Complete heart block with a narrow QRS is not an absolute indication for pacing because atrioventricular junctional ectopic pacemakers (producing a narrow QRS) may provide a reasonable and stable heart rate.

Pacing

Transcutaneous pacing

Initiate transcutaneous pacing immediately if there is no response to atropine, if atropine is unlikely to be effective, or if the patient is severely symptomatic, particularly if there is high-degree AV block (Möbitz Type II 2^{o} block or 3^{o} block).

Fist pacing

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

Tachycardias

If the patient is unstable and deteriorating, with signs and symptoms caused by the tachycardia (e.g. impaired conscious level, chest pain, heart failure, hypotension, or other signs of shock), attempt synchronised cardioversion immediately. 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 or significant co-morbidity may be symptomatic and unstable at lower heart rates. 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.

Synchronised electrical cardioversion

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 convert with lower energies: start with 70-120 J biphasic (100 J monophasic).

If a patient with tachycardia is stable (no serious signs or symptoms caused by the tachycardia), and is not deteriorating, there is time to evaluate the rhythm using the 12-lead ECG. Then determine treatment options and, if required, consult an expert. If the patient becomes unstable proceed immediately to synchronised electrical cardioversion. If a patient develops a tachyarrhythmia during, or as a complication of some other illness (e.g. infection, heart failure), treatment of the other medical problem is required also.

Broad-complex tachycardia

In broad-complex tachycardias the QRS complexes are ≥ 0.12 sec in duration, and are usually ventricular in origin. Broad-complex tachycardias may be caused also by supraventricular rhythms with aberrant conduction.

In the unstable periarrest patient assume that the rhythm is ventricular in origin and attempt electrical cardioversion. 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. In a stable patient, VT can be treated 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 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. 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 (cardiac arrest algorithm).

Narrow-complex tachycardia

Regular narrow-complex tachycardias include:

- 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 atrial fibrillation (AF), or sometimes atrial flutter with variable AV conduction ('variable block').

Regular narrow-complex tachycardia

Sinus tachycardia

This is a common physiological response to a stimulus such as exercise or anxiety. In a sick patient it may occur in response to many stimuli such as pain, fever, anaemia, blood loss, and heart failure. Treatment is almost always 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.

AVNRT and AVRT (paroxysmal supraventricular tachycardia)

AV nodal re-entry tachycardia is the commonest type of paroxysmal supraventricular tachycardia (PSVT), 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 block produces a tachycardia of about 150 min^{-1}. Much faster rates (170 min^{-1} or more) are unlikely to be caused by atrial flutter with 2:1 block.

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 the 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. Record an ECG (preferably multi-lead) during the injection. If the ventricular rate slows transiently, but the arrhythmia then returns, 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.
- 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.
- If adenosine is contra-indicated, or fails to terminate a regular narrow complex tachycardia without demonstrating that it is atrial flutter, give 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 electrical cardioversion.

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 aim is to control heart rate, options include a beta blocker, digoxin, magnesium, or combinations of these.

If the duration of AF is less than 48 h, and rhythm control is considered appropriate, this may be attempted using amiodarone (300 mg IV over 20-60 min followed by 900 mg over 24 h). 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.

Further reading

Blomstrom-Lundqvist C, Scheinman MM, Aliot EM et al. ACC/AHA/ESC Guidelines for the Management of Patients with Supraventricular Arrhythmias. European Heart Journal 2003; 24: 1857-1897.

Fuster V, Ryden LE, Asinger RW et al. ACC/AHA/ESC Guidelines for the Management of Patients with Atrial Fibrillation. European Heart Journal 2001; 22: 1852-1923.