Guidelines: Peri-arrest arrhythmias

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1. The guideline process

The process used to produce the Resuscitation Council UK Guidelines 2015 has been accredited by the National Institute for Health and Care Excellence. The guidelines process includes:

• Systematic reviews with grading of the quality of evidence and strength of recommendations. This led to the 2015 International Liaison Committee on Resuscitation (ILCOR) Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. 1,2

• The involvement of stakeholders from around the world including members of the public and cardiac arrest survivors.

• Details of the guidelines development process can be found in the Resuscitation Council UK Guidelines Development Process Manual.

• These Resuscitation Council UK Guidelines have been peer reviewed by the Executive Committee of Resuscitation Council UK, which comprises 25 individuals and includes lay representation and representation of the key stakeholder groups.

2. Summary of changes since 2010 Guidelines

There are relatively few changes from Guidelines 2010. The basic principles of assessment and treatment of a suspected cardiac arrhythmia are unchanged. Use of oxygen therapy is not recommended unless the patient is hypoxaemic, in which situation the concentration of oxygen delivered should be guided by monitoring arterial oxygen saturation whenever possible. There is stronger
emphasis on the use of antithrombotic therapy in atrial fibrillation (AF) and the importance of assessing thromboembolic risk in people with AF.

3. Introduction

Cardiac arrhythmias are relatively common in the ‘peri-arrest’ period. An arrhythmia may precede the development of ventricular fibrillation (VF) or asystole or may develop after successful defibrillation. Although arrhythmias are common in the setting of acute myocardial infarction, there are many other causes. Some rhythm abnormalities are usually benign and others usually dangerous; each rhythm encountered requires assessment and treatment in the context of the individual clinical circumstances at the time.

If a patient with an arrhythmia is not acutely ill there may be other treatment options, including the use of drugs (oral or parenteral), that are less familiar to the non-expert. In this situation advice should be sought from the most appropriate experts (e.g. cardiologists).

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. They are based on current national and international guidelines for management of arrhythmia.3-9

4. Sequence of actions

- Assess a patient with a suspected arrhythmia using the ABCDE approach
- In particular, note the presence or absence of ‘adverse features’
- Give oxygen immediately to hypoxaemic patients and adjust delivery according to observed arterial oxygen saturations
- Insert an intravenous (IV) cannula
- Whenever possible, record a 12-lead ECG; this will help identify the precise rhythm, which may guide immediate treatment and/or be crucial to planning later treatment
- Correct any electrolyte abnormalities (e.g. K⁺, Mg²⁺, Ca²⁺).

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

**Adverse features**

The presence or absence of adverse symptoms or signs will dictate the appropriate immediate treatment for most arrhythmias. The following adverse features indicate that a patient is at high risk of early deterioration and death (‘unstable’), either because of the arrhythmia itself or because of underlying heart disease with the arrhythmia superimposed:

- **Shock** – hypotension (systolic blood pressure <90 mm Hg), 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).

[H3] 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 treatment options can be categorised under four headings:

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

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, continue to assess the patient
(ABCDE) and re-peat a 12-lead ECG to detect any other abnormalities that may require treatment, either immediately or in the longer term.

5. Tachycardia

The approach to an adult with tachycardia and a palpable pulse is shown in the Adult Tachycardia (with pulse) algorithm (Figure 1).

![Figure 1. Adult tachycardia (with pulse) algorithm](image)

**If a patient is unstable**

If a patient with a tachyarrhythmia is unstable (i.e. has adverse features likely to be caused or made worse by the tachycardia) synchronised cardioversion is the treatment of choice. In patients with otherwise normal hearts adverse symptoms and signs are uncommon from arrhythmia with ventricular rate <150 \text{ min}^{-1}.

Patients with impaired cardiac function, structural heart disease or other serious medical conditions (e.g. severe lung disease) are more likely to be symptomatic and unstable during arrhythmias with heart rates between 100 and 150 \text{ 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 may be followed by an infusion of
Synchronised cardioversion

If the patient is conscious, carry out cardioversion under sedation or general anaesthesia, 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 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.

If the patient is stable

If a patient with a tachyarrhythmia has no adverse features consider whether any treatment is required. If so, consider using drug treatment in the first instance. Assess the ECG and determine the QRS duration. If the QRS duration is 0.12 s or greater (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

Many broad-complex tachycardias (QRS ≥0.12 s) are ventricular in origin. In other cases broad-complex tachycardia may be a supraventricular rhythm with aberrant conduction (bundle branch block). In an unstable 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 from the ECG if the rhythm is regular or irregular.

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

In a stable patient, if the broad-complex tachycardia is thought to be VT, treat with amiodarone 300 mg IV 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 (usually after expert assessment of previous episodes of identical rhythm) and the patient is stable use the strategy indicated for regular, narrow-complex tachycardia (below). Where there is uncertainty, seek urgent expert help whenever possible.

**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 sustained 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. Do not give amiodarone for definite torsade de pointes. Correct electrolyte abnormalities, especially hypokalaemia. Give magnesium sulfate 2 g IV over 10 min (= 8 mmol, 4 mL of 50% magnesium sulfate). 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 are present, 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
- AV nodal re-entry tachycardia (AVNRT) – the commonest type of regular narrow-complex tachyarrhythmia
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 usually 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 rare 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)

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\(^{-1}\)) are unlikely to be caused by atrial flutter with 2:1 conduction. Regular tachycardia with slower rates (e.g. 125–150 min\(^{-1}\)) 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 apply vagal manoeuvres and/or give adenosine to an unstable patient with a regular narrow-complex tachycardia while preparations are being made urgently for synchronised cardioversion. 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 IV 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 (i.e. no transient slowing or termination of the tachyarrhythmia) to adenosine 6 mg IV, give a 12 mg IV bolus. If there is no response give one further 12 mg IV 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. Termination of a regular narrow-complex tachycardia in these ways identifies it as being AVNRT or AVRT. 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 verapamil 2.5–5 mg IV 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 or made worse by the arrhythmia, start antithrombotic therapy (see below) and attempt synchronised cardioversion.

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

• no treatment
• 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 low-molecular-weight heparin in weight-adjusted 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 attempted cardioversion, whether or not it is successful. Seek expert advice on the duration of anticoagulation, which should be a minimum of four weeks, often substantially longer, unless the risk of bleeding is prohibitive. Use of scoring systems to assess thromboembolic risk (e.g. the CHA$_2$DS$_2$-VASc score) and the risk of bleeding (e.g. the HAS-BLED score) are recommended when considering the balance of risk and benefit from anticoagulant therapy in people with AF (and atrial flutter).

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 IV preparation of diltiazem is available in some countries but not in the UK. Digoxin or amiodarone may be used in patients with heart failure. Amiodarone may be used to assist with rate control but is more useful in maintaining rhythm control. Whenever 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 or propafenone. Do not use flecainide or propafenone in the presence of heart failure, known left ventricular impairment or ischaemic heart disease, or a prolonged QT interval. Amiodarone (300 mg IV over 20–60 min followed by 900 mg over 24 h) may also be used (unless the QT interval is prolonged) 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 to 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.

6. Bradycardia

The approach to an adult with bradycardia and a palpable pulse is shown in the Adult Bradycardia algorithm (Figure 2).
Bradycardia is defined as a heart rate in an adult of <60 min⁻¹. Causes include:

- physiological (e.g. during sleep, in athletes)
- cardiac causes (e.g. atrioventricular block or sinus node disease)
- non-cardiac causes (e.g. vasovagal, hypothermia, hypothyroidism,
Assess a patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for adverse features. If no adverse features are present, continue to monitor and reassess the patient (ABCDE). Seek expert help to plan any necessary further assessment and treatment.

Consider treating any reversible causes of bradycardia identified in the initial assessment. If adverse features signs are present start to treat the bradycardia. Initial treatment for most patients is pharmacological; pacing is indicated for patients unresponsive to pharmacological treatment or with risks factors for asystole.

**Treatment using drugs**

If adverse features are present, give atropine 500 mcg IV and, if necessary, repeat every 3-5 min to a total of 3 mg. Doses of atropine of less than 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 infarction; the resulting increase in 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.

If bradycardia with adverse signs persists 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. Serious cases of digoxin or other drug toxicity should be discussed with the National Poisons Information Service. Consider using theophylline (100–200 mg by slow IV injection) for bradycardia complicating acute inferior wall myocardial infarction, spinal cord injury or cardiac transplantation.

**Transcutaneous pacing**
Initiate transcutaneous pacing immediately if there is no response to atropine, or if atropine is contraindicated. Transcutaneous pacing can be painful and may fail to achieve effective electrical capture (i.e. a QRS complex after each pacing stimulus) or fail to achieve a mechanical response (i.e. palpable pulse). Check for 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 for life-threatening, extreme bradycardia, while waiting for pacing equipment or personnel. Give repeated rhythmic thumps with the side of a 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 initiate 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 or complete (third-degree) AV block (especially with broad QRS or initial heart rate <40 beats min\(^{-1}\)).

**7. Accreditation of the 2015 Guidelines**

NICE has accredited the process used by Resuscitation Council UK to produce its Guidelines development Process Manual. Accreditation is valid for 5 years from March 2015. More information on accreditation can be viewed at [https://www.nice.org.uk/about/what-we-do/accreditation](https://www.nice.org.uk/about/what-we-do/accreditation).

**8. References**


Related content
- The ABCDE Approach

Downloads
- Adult Tachycardia Algorithm (PDF) 68.16 KB
- Adult Bradycardia Algorithm (PDF) 65.47 KB