Management of peri-arrest arrhythmia

Sunita Sanghavi FRCA
Juergen Rayner-Klein FRCA FIMC RCSEd

Arrhythmias, potentially serious electrical abnormalities of the heart, cause most sudden cardiac death (SCD). Cardiac arrhythmias are a well-recognised complication of myocardial infarction but can also be precipitated by metabolic or endocrine abnormalities or drugs (Table 1). They may precede ventricular fibrillation (VF) or follow successful defibrillation. Emergency cardiovascular care no longer focuses just on the patient in established cardiac arrest. Advanced life support (ALS) providers must be able to recognise promptly and effectively treat patients ‘on their way to a cardiac arrest’ and those recovering in the immediate post-resuscitation period.

There are peri-arrest arrhythmia algorithms for: (i) broad complex tachycardia; (ii) narrow complex tachycardia; (iii) atrial fibrillation; and (iv) bradycardia. These algorithms, provided by the European Resuscitation Council (ERC) and adopted by the Resuscitation Council (UK), aim to facilitate the safe, effective and timely initial treatment of arrhythmias by the non-specialist ALS provider. They universally recommend seeking expert help expeditiously at various stages whilst assessment and life-saving first-line emergency treatment is undertaken. They also clearly re-assure the non-specialist and outline recommendations in case of non-life-threatening arrhythmias.

General approach to arrhythmias

Both tachyarrhythmias and bradyarrhythmias may present clinically with haemodynamic compromise. The approach to a patient with an arrhythmia depends on the: (i) adverse clinical effects of the presenting heart rhythm; (ii) diagnosis of the rhythm abnormality from the electrocardiogram; and (iii) recognition of any identifiable precipitating factors or underlying cardiac abnormalities.

Adverse clinical effects

Shock

Clinical evidence of low cardiac output is seen as pallor, generalised sweating, cold clammy extremities due to increased sympathetic activity, depressed conscious level, oliguria and hypotension.

Heart failure

Tachyarrhythmias reduce the coronary blood flow and thus reduce the efficiency of the heart as a pump. Acute cardiac pump failure may manifest itself as pulmonary oedema if the left ventricle is predominantly affected or may present with raised jugular venous pressure, hepatic congestion and pitting peripheral oedema if the right ventricle is affected. There may be a combination of all these physical signs if both sides of the heart are affected.

Table 1 Precipitating factors of arrhythmias

<table>
<thead>
<tr>
<th>Underlying cardiac disease</th>
<th>Ischaemic heart disease (acute or recent myocardial infarction, angina)</th>
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<td>Mitral valve disease</td>
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<td>Left ventricular aneurysm</td>
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<td>Congenital heart disease</td>
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<td>Pre-excitation syndromes (short PR interval)</td>
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<td>Long QT syndrome (congenital or acquired)</td>
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<td>Metabolic abnormalities</td>
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<td>↑ Ca²⁺ or ↓ Ca²⁺</td>
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<td>↑ PaCO₂</td>
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<td>Acidosis</td>
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<td>Endocrine abnormalities</td>
<td>Thyrotoxicosis</td>
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<td>Phaeochromocytoma</td>
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<td>Drugs</td>
<td>Anti-arrhythmics</td>
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<td>Sympathomimetries (β₂-agonists, cocaine)</td>
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<td>Antidepressants</td>
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<td>Adenylate cyclase inhibitors (aminophylline, caffeine)</td>
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<td>Alcohol</td>
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**Angina**
Coronary blood flow may be critically reduced during arrhythmias resulting in myocardial ischaemia.

**Excessive tachycardia**
This arrhythmia reduces the time available for coronary blood flow during diastole and occurs at heart rates > 200 beats min⁻¹ in narrow complex tachycardias and at heart rates > 150 beats min⁻¹ in broad complex tachycardias.

**Excessive bradycardia**
This is defined as a heart rate < 40 beats min⁻¹ and leads to inadequate perfusion in patients with a low stroke volume. Even a heart rate below 60 beats min⁻¹ may be poorly tolerated in patients with reduced cardiac reserve.

**Diagnosis**
The initial treatment of peri-arrest arrhythmias depends on the correct diagnosis of the arrhythmia from the electrocardiogram and then the use of the appropriate algorithm. This process is simplified by applying the following systematic approach to diagnosis:
1. Is the heart rate > 100 beats min⁻¹ or < 60 beats min⁻¹?
2. Are the QRS complexes broad (= 120 msec or 3 small squares on the ECG) or narrow (< 120 msec or 3 small squares on the ECG).
3. Is the rhythm regular or irregular?

**General management**
In any situation that may result in, or follows, cardiac arrest, it is mandatory to administer supplementary inspired oxygen and establish intravenous access. Electrolyte abnormalities should be corrected urgently.

There are four options available for the immediate management of arrhythmias: (i) electrical DC cardioversion; (ii) anti-arrhythmic drugs; (iii) cardiac pacing; and (iv) vagal manoeuvres. Any anti-arrhythmic intervention may potentially be pro-arrhythmic and lead to clinical deterioration as a direct consequence of the drug, rather than as a result of lack of effect. Therefore, caution is advised in refractory cases.

**Electrical cardioversion**
Electrical cardioversion is the application of a synchronised DC current across the patient’s chest along the main vector of the cardiac axis. It is a relatively reliable way of converting a tachyarrhythmia back to sinus rhythm. Cardioversion is carried out in the same way as defibrillation and requires gel pads to be applied to the chest. The recommended energy format is an ascending sequence of 100 J, 200 J and 360 J or the equivalent biphasic energy. Electrical cardioversion is painful and requires adequate sedation or anaesthesia in conscious patients. The timing of the electrical discharge must be synchronised to occur with the R wave of the ECG rather than with the T wave, as delivery during ventricular repolarisation could lead to ventricular fibrillation (the R on T phenomenon). A ‘synchronisation mode’ switch in manual defibrillators enables the R wave to be sensed and to trigger the delivery of electrical energy. There may be a slight lag period between pressing the discharge button and the energy delivery until the next R wave is detected. However, the paddles of the defibrillator must not move during this period in order to ensure that the QRS complexes are detected.

**Anti-arrhythmic drugs**
Anti-arrhythmic drugs are capable of inducing or sustaining an arrhythmia, particularly if the myocardium is impaired or damaged. When a second arrhythmic drug is added, these anti-arrhythmic effects escalate exponentially. Anti-arrhythmic drugs are slower in onset of effect and less reliable than electrical cardioversion in converting a tachycardia to sinus rhythm. They are, therefore, usually reserved for patients without adverse clinical signs.

If an appropriate dose of a single anti-arrhythmic drug fails to terminate an arrhythmia, electrical cardioversion should be considered as the second line of treatment if symptoms persist. Patients in congestive cardiac failure or with impaired left ventricular function should be treated very cautiously with anti-arrhythmic drugs to avoid precipitation of congestive cardiac failure or further impairment of pump function.

Amiodarone has become a first-line drug in the management of tachycardias due to its broad range of action and less negative inotropic effects when compared to lidocaine (Fig. 1).

**Cardiac pacing**
Cardiac pacing is indicated in:
1. Bradycardias with adverse clinical signs that are refractory to atropine (external transcutaneous pacing).
2. Profound bradycardias that cause loss of cardiac output and ventricular standstill with P waves (external percussion pacing).
3. Complete heart block with broad QRS complexes (external transcutaneous pacing).

4. Paroxysms of torsades de pointes (external, transcutaneous overdrive pacing).

Systems for non-invasive external transcutaneous pacing are integrated into some modern defibrillators. Multifunction, adhesive electrodes capable of ECG monitoring, pacing, cardioversion and defibrillation enable the operator to pace the heart quickly, without the need for central venous access and with minimal specialised training.

Percussion pacing is the delivery of a series of chest thumps with a closed fist over the precordium at a point, found usually by trial and error, which will result in consistent ventricular stimulation.

All these described pacing methods are only temporary measures. If they are successful and result in an adequate cardiac output being established, then expert help must be sought and more definitive treatment established (transvenous pacing, permanently implanted pacemaker).

**Vagal manoeuvres**

Vagal manoeuvres, which stimulate the vagal nerve, will slow the heart rate and terminate supraventricular tachycardias in up to 25% of cases. A Valsalva manoeuvre may be simulated by asking the patient to blow into a 20 ml syringe with enough force to push the plunger back. Carotid sinus massage is a firm rotatory pressure over the carotid sinus against the transverse process of the third cervical vertebra. Carotid sinus massage may cause an atheromatous plaque to embolise into the cerebral circulation and cause a stroke.

**Specific management of arrhythmias**

**Broad complex tachycardias**

Broad complex tachycardias (Fig. 2) are, in the peri-arrest context, assumed to be ventricular in origin and are treated as sustained ventricular tachycardias. The key decisions are:

1. Is there a pulse?
2. Are there adverse clinical signs?
   - Systolic blood pressure < 90 mmHg
   - Chest pain
   - Heart failure
   - Heart rate > 150 beats min⁻¹
3. Is the plasma potassium concentration normal?
4. Is the broad complex tachycardia refractory to first line treatment?

If there is no pulse present, treatment follows the cardiac arrest protocol for VF/pulseless VT.
Management of peri-arrest arrhythmia

Broad Complex Tachycardia
(Treat as sustained ventricular tachycardia) *

If not already done, give oxygen and establish i.v. access

Pulse? No

Use VF protocol

Yes

Adverse signs?
• Systolic BP < 90 mmHg
• Chest pain
• Heart failure
• Rate > 150 beats min⁻¹

If potassium known to be low,
see panel

• Amiodarone 150 mg i.v.
  over 10 min, or
• Lidocaine i.v. 50 mg over 2
  min repeated every 5 min
to a maximum dose of 200 mg

Seek expert help

Synchronised DC shock †
100 J : 200 J : 360 J
or equivalent biphasic energy

If necessary, further amiodarone 150 mg
i.v. over 10 min, then 300 mg over 1 h
and repeat shock

Further cardioversion
as necessary

For refractory cases consider
additional pharmacological agents:
amiodarone, lidocaine,
procainamide or sotalol;
or overdrive pacing
Caution: drug induced
myocardial depression

Doses throughout are based on an adult of average body weight

* Note 1 For paroxysms of torsades de pointes, use magnesium as above
  or overdrive pacing (expert help strongly recommended).
† Note 2 DC shock is always given under sedation/general anaesthesia.

Fig. 2 Broad complex tachycardia algorithm.
If there is a pulse but adverse clinical signs are present, then synchronised DC cardioversion is indicated. This always requires careful sedation or general anaesthesia. It is important to seek expert help early. If the potassium concentration is low, then potassium 60 mmol (maximum rate 30 mmol h⁻¹) and magnesium (5 ml 50% in 30 min) are given intravenously. The administration of amiodarone (150 mg i.v. over 10 min) may stabilise the cardiac rhythm. However, further cardioversion attempts, overdrive pacing or additional pharmacological treatment (lidocaine, procainamide, sotalol) may be necessary.

If there are no adverse signs, then administer either amiodarone (150 mg i.v. over 10 min) or lidocaine (50 mg i.v. over 2 min, repeated every 5 min to a maximum of 200 mg). Again, expert help must be sought and synchronised cardioversion attempted if the pharmacological interventions fail. Plasma potassium concentrations should be corrected as described above.

Narrow complex tachycardias

Narrow complex tachycardias (Fig. 3) are presumed to be supraventricular as they originate within the atrium or above the bundle of HIS. The key decisions are:

1. Is there a pulse?
2. Does the ECG look like atrial fibrillation?
3. Could this be Wolff-Parkinson-White syndrome?
4. Are there adverse clinical signs?

If the narrow complex tachycardia is believed to be atrial fibrillation, the specific algorithm for atrial fibrillation (Fig. 4) should be followed. Vagal stimulating manoeuvres (Valsalva or carotid sinus massage) can be tried to terminate the tachycardia. These vagal manoeuvres are to be used with caution as they might cause a sudden bradycardia, which may trigger ventricular fibrillation particularly in the context of myocardial ischaemia and digitalis toxicity.

The next step is the administration of a rapid bolus of adenosine 6 mg intravenously. Adenosine is a naturally occurring purine nucleotide which slows transmission across the AV node. Adenosine has an extremely short half-life of 10−15 sec and, therefore, must be given into a fast-running intravenous infusion. It is most effective for terminating paroxysmal supraventricular tachycardias with re-entrant circuits that include the AV node. Adenosine can be repeated up to three times (12 mg, every 1−2 min) if the starting dose of 6 mg proves unsuccessful. This dose is currently outside the UK licence. Methylxanthines (theophylline) block the effect of adenosine. Patients on dipyridamole or carbamazepine or those with denervated hearts experience a markedly exaggerated effect that may be hazardous.

Blockage of conduction across the AV node by adenosine may promote conduction across an accessory pathway. In patients with Wolff-Parkinson-White syndrome (WPW), administration of adenosine for a supraventricular tachycardia may result in a dangerously rapid ventricular response. Adenosine may rarely precipitate atrial fibrillation in WPW syndrome with a dangerously rapid ventricular response.

The presence of adverse clinical signs demands synchronised DC cardioversion under sedation or anaesthesia. This is followed by intravenous administration of amiodarone 150 mg over 10 min and then 300 mg over 1 h before attempting DC cardioversion again. In the absence of adverse clinical signs, esmolol (40 mg in 1 min with an infusion of 4 mg min⁻¹), verapamil (5−10 mg), amiodarone (300 mg over 1 h) or digoxin (maximum dose 500 mg i.v. over 30 min, twice) can be used. Verapamil should not be used in WPW syndrome and has potentially dangerous therapeutic interactions in patients taking β-blocking drugs.

Atrial fibrillation

Atrial fibrillation is the result of continuous rapid (in excess of 400 beats min⁻¹) activation of the atria by multiple meandering wavelets. The atria respond electrically at this fast rate but there is practically no atrial mechanical pump action. Only a proportion of electrical impulses are conducted to the ventricles. The appropriate treatment for atrial fibrillation (Fig. 4) is determined by the patient’s relative clinical risk from the arrhythmia. The key decisions are:

1. Is the patient at high, intermediate or low clinical risk?
2. Are there clinical signs of poor perfusion?
3. Is there known structural heart disease?
4. Is the onset of the arrhythmia known to be within 24 h?

High risk

Patients at high risk from atrial fibrillation have a heart rate > 150 beats min⁻¹ and display adverse signs of on-going chest pain or critical perfusion. Immediate expert help should be sought before heparinisation and treatment with synchronised DC cardioversion under sedation or anaesthesia. If cardioversion fails or atrial fibrillation recurs, amiodarone 300 mg is administered intravenously over 1 h before a further cardioversion attempt or a second 300 mg dose of amiodarone.
Narrow Complex Tachycardia
(Presumed supraventricular tachycardia)

If not already done, give oxygen and establish i.v. access

Vagal manoeuvres
(caution if possible digitalis toxicity, acute ischaemia, or presence of carotid bruit for carotid sinus massage)

Adenosine 6 mg by rapid bolus injection; if unsuccessful, follow, if necessary, with up to 3 doses each of 12 mg every 1–2 min
*(Caution with adenosine in known Wolff-Parkinson-White syndrome)

Choose from:
- Esmolol: 40 mg over 1 min + infusion 4 mg min⁻¹
  (i.v. injection can be repeated and infusion increased incrementally to 12 mg min⁻¹)
  OR
- Verapamil 5-10 mg i.v. **
  OR
- Amiodarone: 300 mg i.v. over 1 h, may be repeated once if necessary
  OR
- Digoxin: maximum dose 500 µg i.v. over 30 min x 2

If necessary, amiodarone 150 mg i.v. over 10 min, then 300 mg over 1 h and repeat shock

Doses throughout are based on an adult of average body weight
A starting dose of 6 mg adenosine is currently outside the UK licence for this agent.

* Note 1 Theophylline and related compounds block the effect of adenosine. Patients on dipyridamole, carbamazepine, or with denervated hearts have a markedly exaggerated effect which may be hazardous.
† Note 2 DC shock is always given under sedation/general anaesthesia.
** Note 3 Not to be used in patients receiving β-blockers.

Fig. 3 Narrow complex tachycardia algorithm.
Atrial fibrillation

If appropriate, give oxygen and establish i.v. access

**High risk?**
- Heart rate > 150 beats min⁻¹
- Ongoing chest pain
- Critical perfusion

Yes
Seek expert help

Immediate heparin and synchronised DC shock†
100 J : 200 J : 360 J or equivalent biphasic energy

Amiodarone 300 mg i.v. over 1 h.
If necessary, may be repeated once

**Intermediate risk?**
- Rate 100-150 beats min⁻¹
- Breathlessness

Yes
Seek expert help

Consider anticoagulation:
- Heparin
- Warfarin for later synchronised DC shock†, if indicated

Yes

**Low risk?**
- Heart rate < 100 beats min⁻¹
- Mild or no symptoms
- Good perfusion

Yes

Onset known to be within 24 hours?

Yes

Heparin
Amiodarone: 300 mg i.v. over 1 h, may be repeated once if necessary or
Flecainide 100–150 mg i.v. over 30 min and/or synchronised DC shock†, if indicated

Poor perfusion and/or known structural heart disease?

Yes

Initial rate control
- β-Blockers, oral or i.v. OR
- Verapamil i.v. (oral) ** OR
- Diltiazem, oral (or i.v. if available) ** OR
- Digoxin, i.v. or oral OR
Consider anticoagulation:
- Heparin
- Warfarin for later synchronised DC shock†, if indicated

No

Onset known to be within 24 hours?

Yes

Initial rate control
- Amiodarone: 300 mg over 1 h, may be repeated once if necessary AND
Anticoagulation:
- Heparin
- Warfarin
Synchronised DC shock†, if indicated

No

Attempt cardioversion:
- Amiodarone: 300 mg over 1 h, may be repeated once if necessary OR
- Synchronised DC shock†, 100 J : 200 J : 360 J or equivalent biphasic energy

Yes

Attempt cardioversion:
- Heparin
- Synchronised DC shock†, 100 J : 200 J : 360 J or equivalent biphasic energy

Amiodarone 300 mg i.v. over 1 h.
If necessary, may be repeated once

Doses throughout are based on an adult of average body weight

† Note 1 DC shock is always given under sedation/general anaesthesia.
** Note 2 NOT TO BE USED IN PATIENTS RECEIVING β-BLOCKERS

Fig. 4 Atrial fibrillation algorithm.
Bradycardia
(Includes rates inappropriately slow for haemodynamic state)

If appropriate, give oxygen and establish i.v. access

Adverse signs?
- Systolic BP < 90 mmHg
- Heart rate < 40 beats min⁻¹
- Ventricular arrhythmias requiring suppression
- Heart failure

Interim measures
- Atropine 500 µg i.v.
- Repeat to maximum 3 mg
- Transcutaneous (external) pacing
  or
- Epinephrine (adrenaline) i.v.
  2–10 µg min⁻¹

Risk of asystole?
- Recent asystole
- Möbitz II AV block
- Complete heart block with broad QRS
- Ventricular pause > 3 s

Observe

Seek expert help
Arrange transvenous pacing

Fig. 5 Bradycardia algorithm.
Intermediate risk

Patients at intermediate risk from atrial fibrillation present with a heart rate of 100–150 beats min⁻¹ and are breathless. Expert help should be sought. If there is evidence of poor perfusion and/or known structural heart disease, then these patients are treated as high risk provided the onset of atrial fibrillation is known to have been within the preceding 24 h. If the onset of atrial fibrillation is unknown, these patients undergo initial rate control with an infusion of amiodarone 300 mg over 1 h, which may be repeated once with immediate anticoagulation. Synchronised electrical cardioversion to sinus rhythm may be attempted 3–4 weeks later.

Patients at intermediate risk from atrial fibrillation who do not show evidence of poor perfusion or have structural heart disease are treated according to the onset time of the arrhythmia. If the onset is known to have been within the preceding 24 h, synchronised cardioversion is attempted after immediate treatment with an intravenous infusion of amiodarone 300 mg in over 1 h, repeated once if necessary, or by an intravenous infusion of flecainide 100–150 mg over 30 min. If the onset of atrial fibrillation is unknown, then the presenting heart rate can be controlled using intravenous or oral β-blockers, verapamil, diltiazem or digoxin. The patient can be anticoagulated and undergo synchronised electrical cardioversion 3–4 weeks later.

Low risk

Patients are at low risk from atrial fibrillation if their heart rate is < 100 beats min⁻¹, have good perfusion and minimal clinical symptoms. If the onset is known to have been within the preceding 24 h, these patients should be anticoagulated and receive an intravenous infusion of amiodarone 300 mg over 1 h (repeated once if necessary) or an intravenous infusion of flecainide 100–150 mg over 30 min. In addition, they can undergo synchronised DC electrical cardioversion under sedation or anaesthesia if required. If the time of onset of atrial fibrillation is unknown, these patients should be anticoagulated and undergo synchronised DC electrical cardioversion 3–4 weeks later.

Bradycardia

Bradycardia is defined in the algorithm (Fig. 5) as a heart rate of < 60 beats min⁻¹. A relative bradycardia exists when the heart rate is inappropriately slow for the haemodynamic state of the patient. The key decisions are:

1. Are there clinical adverse signs?
   - Systolic blood pressure < 90 mmHg
   - Heart rate < 40 beats min⁻¹ (absolute bradycardia)
   - Ventricular arrhythmias requiring treatment
   - Heart failure

2. Is there a satisfactory response to intravenous atropine 500 µg?

3. Is there a risk of asystole?

4. Is there a need to consider repeat doses of intravenous atropine, transcutaneous (external) pacing or intravenous epinephrine?

Atropine 500 µg i.v. is administered if adverse signs are present and can be repeated up to a total dose of 3 mg as an interim measure. A satisfactory response to atropine then requires an assessment of the risk of asystole. The risk of asystole is suggested by a recent episode of asystole, a Möbitz type II AV block, 3rd degree heart block with broad QRS complexes or a ventricular pause > 3 sec. If there is a risk of asystole, then immediate expert help must be summoned whilst interim measures, such as transcutaneous external pacing or an intravenous infusion of epinephrine 2–10 µg min⁻¹, titrated to response, are tried. If there is no risk of asystole, the patient can be simply observed.

Key references


Web sites

European Resuscitation Council www.erc.edu
Resuscitation Council (UK) www.resus.org

See multiple choice questions 70–77.