Resuscitation guidelines

The guidelines contain detailed information about basic and advanced life support for adults, paediatrics and the newborn. Also included are guidelines for the use of Automated External Defibrillators and other related topics.

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Contributors and conflict of interest

ILCOR Science worksheets

In March 2015 the Resuscitation Council (UK) received NICE Accreditation for the process used to assemble and produce all its guideline
In July 2012 the process used by the Resuscitation Council (UK) to produce the 2010 Resuscitation Guidelines was accredited by the National Institute for Health and Clinical Excellence (NICE). The NICE Accreditation Scheme recognises organisations that demonstrate high standards in producing health or social care guidance. Users of NICE accredited guidance can therefore have high confidence in the quality of the information provided. The NICE Accreditation was based on the procedures and methodology used in the development of the 2010 Resuscitation Guidelines, as documented in the Resuscitation Council (UK) Guidelines Development Process Manual (2012).

NICE manages the NHS Evidence service, which provides access to authoritative clinical and non-clinical evidence and best practice through a web-based portal.
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.
- These Resuscitation Council (UK) Guidelines have been peer reviewed by the Executive Committee of the Resuscitation Council (UK), which comprises 25 individuals and includes lay representation and representation of the key stakeholder groups.

2. Summary of changes in basic life support and automated external defibrillation since the 2010 Guidelines

- Guidelines 2015 highlights the critical importance of the interactions between the emergency medical dispatcher, the bystander who provides cardiopulmonary resuscitation (CPR) and the timely deployment of an automated external defibrillator (AED). An effective, co-ordinated community response that draws these elements together is key to improving survival from out-of-hospital cardiac arrest.
The emergency medical dispatcher plays an important role in the early diagnosis of cardiac arrest, the provision of dispatcher-assisted CPR (also known as telephone CPR), and the location and dispatch of an AED. The sooner the emergency services are called, the earlier appropriate treatment can be initiated and supported.

The knowledge, skills and confidence of bystanders will vary according to the circumstances, of the arrest, level of training and prior experience. The bystander who is trained and able should assess the collapsed victim rapidly to determine if the victim is unresponsive and not breathing normally and then immediately alert the emergency services. Whenever possible, alert the emergency services without leaving the victim.

The victim who is unresponsive and not breathing normally is in cardiac arrest and requires CPR. Immediately following cardiac arrest blood flow to the brain is reduced to virtually zero, which may cause seizure-like episodes that may be confused with epilepsy. Bystanders and emergency medical dispatchers should be suspicious of cardiac arrest in any patient presenting with seizures and carefully assess whether the victim is breathing normally.

3. Introduction

The community response to cardiac arrest is critical to saving lives. Each year, UK ambulance services respond to approximately 60,000 cases of suspected cardiac arrest. Resuscitation is attempted by ambulance services in less than half of these cases (approximately 28,000). The main reasons are that either the victim has been dead for several hours or has not received bystander CPR so by the time the emergency services arrive the person has died. Even when resuscitation is attempted, less than one in ten victims survive to go home from hospital. Strengthening the community response to cardiac arrest by training and empowering more bystanders to perform CPR and by increasing the use of automated external defibrillators (AEDs) at least doubles the chances of survival and could save thousands of lives each year.

This guideline is based on the International Liaison Committee on Resuscitation (ILCOR) 2015 Consensus on Science and Treatment Recommendations (CoSTR) for Basic Life Support and Automated External Defibrillation and the European Resuscitation Council Guidelines for Resuscitation 2015 Section 2 Adult basic life support and automated external defibrillation. These contain all the reference material for this section.

4. Chain of Survival

The Chain of Survival (Figure 1) describes four key, inter-related steps, which if delivered effectively and in sequence, optimise survival from out-of-hospital cardiac arrest.

1: Early recognition and call for help

If untreated, cardiac arrest occurs in a quarter to a third of patients with myocardial ischaemia within the first hour after onset of chest pain.

Once cardiac arrest has occurred, early recognition is critical to enable rapid activation of the ambulance service and prompt initiation of bystander CPR.

2: Early bystander CPR

The immediate initiation of bystander CPR can double or quadruple survival from out-of-hospital cardiac arrest. Despite this compelling evidence, only 40% of victims receive bystander CPR in the UK.

3: Early defibrillation

Defibrillation within 3–5 min of collapse can produce survival rates as high as 50–70%. This can be achieved through public access defibrillation, when a bystander uses a nearby AED to deliver the first shock. Each minute of delay to defibrillation reduces the probability of survival to hospital discharge by 10%. In the UK, fewer than 2% of victims have an AED deployed before the ambulance arrives.

4: Early advanced life support and standardised post-resuscitation care

Advanced life support with airway management, drugs and the correction of causal factors may be needed if initial

Figure 1. The Chain of Survival

5. Improving survival from out-of-hospital cardiac arrest

The Resuscitation Council (UK) recommends that to improve survival from cardiac arrest:

1. All school children are taught CPR and how to use an AED.
2. Everyone who is able to should learn CPR.
3. Defibrillators are available in places where there are large numbers of people (e.g. airports, railway stations, shopping centres, sports stadiums), increased risk of cardiac arrest (e.g. gyms, sports facilities) or where access to emergency services can be delayed (e.g. aircraft and other remote locations).
4. Owners of defibrillators should register the location and availability of devices with their local ambulance services.
5. Systems are implemented to enable ambulance services to identify and deploy the nearest available defibrillator to the scene of a suspected cardiac arrest.
6. All out-of-hospital cardiac arrest resuscitation attempts are reported to the National Out-of-Hospital Cardiac Arrest Audit. www.warwick.ac.uk/ohcao.

6. The Resuscitation Council (UK) BLS/AED guidelines

The remainder of this section contains guidance on the initial resuscitation of an adult cardiac arrest victim where the cardiac arrest occurs outside a hospital. This includes basic life support (BLS: airway, breathing and circulation support without the use of equipment other than a protective barrier device) and the use of an automated external defibrillator (AED). Simple techniques used in the management of choking (i.e. foreign body airway obstruction) are also included. Guidelines for the use of manual defibrillators and starting in-hospital resuscitation are found in Advanced life support guidelines section.

www.resus.org.uk/resuscitation-guidelines/adult-advanced-life-support/

The guidelines are based on the ILCOR 2015 Consensus on Science and Treatment Recommendations (CoSTR) for BLS/AED and European Resuscitation Council Guidelines for BLS/AED.2,8
7. Key messages from Guidelines 2015

- Ensure it is safe to approach the victim.
- Promptly assess the unresponsive victim to determine if they are breathing normally.
- Be suspicious of cardiac arrest in any patient presenting with seizures and carefully assess whether the victim is breathing normally.
- For the victim who is unresponsive and not breathing normally:
  - Dial 999 and ask for an ambulance. If possible stay with the victim and get someone else to make the emergency call.
  - Start CPR and send for an AED as soon as possible.
  - If trained and able, combine chest compressions and rescue breaths, otherwise provide compression-only CPR.
  - If an AED arrives, switch it on and follow the instructions.
  - Minimise interruptions to CPR when attaching the AED pads to the victim.
- Do not stop CPR unless you are certain the victim has recovered and is breathing normally or a health professional tells you to stop.
- Treat the victim who is choking by encouraging them to cough. If the victim deteriorates give up to 5 back slaps followed by up to 5 abdominal thrusts. If the victim becomes unconscious – start CPR.
- The same steps can be followed for resuscitation of children by those who are not specifically trained in resuscitation for children – it is far better to use the adult BLS sequence for resuscitation of a child than to do nothing.

8. Adult BLS sequence

The sequence of steps for the initial assessment and treatment of the unresponsive victim are summarised in Figure 2. Further technical information on each of the steps is presented in Table 1 and below.

The sequence of steps takes the reader through recognition of cardiac arrest, calling an ambulance, starting CPR and using an AED. The number of steps has been reduced to focus on the key actions. The intent of the revised algorithm is to present the steps in a logical and concise manner that is easy for all types of rescuers to learn, remember and perform CPR and use an AED.
Figure 2. Adult basic life support algorithm

A4-size algorithm: [http://resus.org.uk/_resources/assets/attachment/full/0/6444.pdf](http://resus.org.uk/_resources/assets/attachment/full/0/6444.pdf)

Table 1: BLS/AED detailed sequence of steps

<table>
<thead>
<tr>
<th>SEQUENCE</th>
<th>Technical description</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAFETY</td>
<td>Make sure you, the victim and any bystanders are safe</td>
</tr>
</tbody>
</table>
| RESPONSE | Check the victim for a response  
• Gently shake his shoulders and ask loudly: “Are you all right?”  
If he responds leave him in the position in which you find him, provided there is no further danger; try to find out what is wrong with him and get help if needed; reassess him regularly |
| AIRWAY   | Open the airway  
• Turn the victim onto his back  
• Place your hand on his forehead and gently tilt his head back; with your fingertips under the point of the victim’s chin, lift the chin to open the airway |
| BREATHING| Look, listen and feel for normal breathing for no more than 10 seconds  
In the first few minutes after cardiac arrest, a victim may be barely breathing, or taking infrequent, slow and noisy gasps. Do not confuse this with normal breathing. If you have any doubt whether breathing is normal, act as if it is they are not breathing normally and prepare to start CPR |
# Table 1: BLS/AED detailed sequence of steps

<table>
<thead>
<tr>
<th>SEQUENCE</th>
<th>Technical description</th>
</tr>
</thead>
<tbody>
<tr>
<td>DIAL 999</td>
<td>Call an ambulance (999)</td>
</tr>
<tr>
<td></td>
<td>• Ask a helper to call if possible otherwise call them yourself</td>
</tr>
<tr>
<td></td>
<td>• Stay with the victim when making the call if possible</td>
</tr>
<tr>
<td></td>
<td>• Activate the speaker function on the phone to aid communication with the ambulance service</td>
</tr>
<tr>
<td>SEND FOR AED</td>
<td>Send someone to get an AED if available</td>
</tr>
<tr>
<td></td>
<td>If you are on your own, do not leave the victim, start CPR</td>
</tr>
<tr>
<td>CIRCULATION</td>
<td>Start chest compressions</td>
</tr>
<tr>
<td></td>
<td>• Kneel by the side of the victim</td>
</tr>
<tr>
<td></td>
<td>• Place the heel of one hand in the centre of the victim’s chest; (which is the lower half of the victim’s breastbone (sternum))</td>
</tr>
<tr>
<td></td>
<td>• Place the heel of your other hand on top of the first hand</td>
</tr>
<tr>
<td></td>
<td>• Interlock the fingers of your hands and ensure that pressure is not applied over the victim’s ribs</td>
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<tr>
<td></td>
<td>• Keep your arms straight</td>
</tr>
<tr>
<td></td>
<td>• Do not apply any pressure over the upper abdomen or the bottom end of the bony sternum (breastbone)</td>
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<tr>
<td></td>
<td>• Position your shoulders vertically above the victim’s chest and press down on the sternum to a depth of 5–6 cm</td>
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<tr>
<td></td>
<td>• After each compression, release all the pressure on the chest without losing contact between your hands and the sternum;</td>
</tr>
<tr>
<td></td>
<td>• Repeat at a rate of 100–120 min⁻¹</td>
</tr>
<tr>
<td>GIVE RESCUE BREATHS</td>
<td>After 30 compressions open the airway again using head tilt and chin lift and give 2 rescue breaths</td>
</tr>
<tr>
<td></td>
<td>• Pinch the soft part of the nose closed, using the index finger and thumb of your hand on the forehead</td>
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<tr>
<td></td>
<td>• Allow the mouth to open, but maintain chin lift</td>
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<tr>
<td></td>
<td>• Take a normal breath and place your lips around his mouth, making sure that you have a good seal</td>
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<tr>
<td></td>
<td>• Blow steadily into the mouth while watching for the chest to rise, taking about 1 second as in normal breathing; this is an effective rescue breath</td>
</tr>
<tr>
<td></td>
<td>• Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out</td>
</tr>
<tr>
<td></td>
<td>• Take another normal breath and blow into the victim’s mouth once more to achieve a total of two effective rescue breaths. Do not interrupt compressions by more than 10 seconds to deliver two breaths. Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions</td>
</tr>
<tr>
<td></td>
<td>Continue with chest compressions and rescue breaths in a ratio of 30:2</td>
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<tr>
<td></td>
<td>If you are untrained or unable to do rescue breaths, give chest compression only CPR (i.e. continuous compressions at a rate of at least 100–120 min⁻¹)</td>
</tr>
<tr>
<td>IF AN AED ARRIVES</td>
<td>Switch on the AED</td>
</tr>
<tr>
<td></td>
<td>• Attach the electrode pads on the victim’s bare chest</td>
</tr>
</tbody>
</table>
### Table 1: BLS/AED detailed sequence of steps

<table>
<thead>
<tr>
<th>SEQUENCE</th>
<th>Technical description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>If more than one rescuer is present, CPR should be continued while electrode pads are being attached to the chest</strong></td>
<td>Follow the spoken/visual directions</td>
</tr>
<tr>
<td></td>
<td>Ensure that nobody is touching the victim while the AED is analysing the rhythm</td>
</tr>
<tr>
<td><strong>If a shock is indicated, deliver shock</strong></td>
<td>Ensure that nobody is touching the victim</td>
</tr>
<tr>
<td></td>
<td>Push shock button as directed (fully automatic AEDs will deliver the shock automatically)</td>
</tr>
<tr>
<td></td>
<td>Immediately restart CPR at a ratio of 30:2</td>
</tr>
<tr>
<td></td>
<td>Continue as directed by the voice/visual prompts</td>
</tr>
<tr>
<td><strong>If no shock is indicated, continue CPR</strong></td>
<td>Immediately resume CPR</td>
</tr>
<tr>
<td></td>
<td>Continue as directed by the voice/visual prompts</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CONTINUE CPR</th>
<th><strong>Do not interrupt resuscitation until:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A health professional tells you to stop</td>
</tr>
<tr>
<td></td>
<td>You become exhausted</td>
</tr>
<tr>
<td></td>
<td>The victim is definitely waking up, moving, opening eyes and breathing normally</td>
</tr>
</tbody>
</table>

It is rare for CPR alone to restart the heart. Unless you are certain the person has recovered continue CPR

<table>
<thead>
<tr>
<th>RECOVERY POSITION</th>
<th><strong>If you are certain the victim is breathing normally but is still unresponsive, place in the recovery position</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Remove the victim’s glasses, if worn</td>
</tr>
<tr>
<td></td>
<td>Kneel beside the victim and make sure that both his legs are straight</td>
</tr>
<tr>
<td></td>
<td>Place the arm nearest to you out at right angles to his body, elbow bent with the hand palm-up</td>
</tr>
<tr>
<td></td>
<td>Bring the far arm across the chest, and hold the back of the hand against the victim’s cheek nearest to you</td>
</tr>
<tr>
<td></td>
<td>With your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground</td>
</tr>
<tr>
<td></td>
<td>Keeping his hand pressed against his cheek, pull on the far leg to roll the victim towards you on to his side</td>
</tr>
<tr>
<td></td>
<td>Adjust the upper leg so that both the hip and knee are bent at right angles</td>
</tr>
<tr>
<td></td>
<td>Tilt the head back to make sure that the airway remains open</td>
</tr>
<tr>
<td></td>
<td>If necessary, adjust the hand under the cheek to keep the head tilted and facing downwards to allow liquid material to drain from the mouth</td>
</tr>
<tr>
<td></td>
<td>Check breathing regularly</td>
</tr>
</tbody>
</table>

Be prepared to restart CPR immediately if the victim deteriorates or stops breathing normally
Initial assessment

For clarity, the algorithm is presented as a linear sequence of steps. It is recognised that the early steps of ensuring the scene is safe, checking for a response, opening the airway, checking for breathing and calling the ambulance may be accomplished simultaneously or in rapid succession.

Airway

Open the airway using the head tilt and chin lift technique whilst assessing whether the person is breathing normally. Do not delay assessment by checking for obstructions in the airway. The jaw thrust and finger sweep are not recommended for the lay provider.

Breathing

Agonal breaths are irregular, slow and deep breaths, frequently accompanied by a characteristic snoring sound. They originate from the brain stem, which remains functioning for some minutes even when deprived of oxygen. The presence of agonal breathing can be interpreted incorrectly as evidence of a circulation and that CPR is not needed. Agonal breathing may be present in up to 40% of victims in the first minutes after cardiac arrest and, if correctly identified as a sign of cardiac arrest, is associated with higher survival rates. The significance of agonal breathing should be emphasised during basic life support training. Bystanders should suspect cardiac arrest and start CPR if the victim is unresponsive and not breathing normally.

Immediately following cardiac arrest, blood flow to the brain is reduced to virtually zero. This may cause a seizure-like episode that can be confused with epilepsy. Bystanders should be suspicious of cardiac arrest in any patient presenting with seizures. Although bystanders who have witnessed cardiac arrest events report changes in the victims’ skin colour, notably pallor and bluish changes associated with cyanosis, these changes are not diagnostic of cardiac arrest.

Checking the carotid pulse (or any other pulse) is an inaccurate method for confirming the presence or absence of circulation.

Dial 999

Early contact with the ambulance service will facilitate dispatcher assistance in the recognition of cardiac arrest, telephone instruction on how to perform CPR and locating and dispatching the nearest AED.

If possible, stay with the victim while calling the ambulance. If the phone has a speaker facility, switch it to speaker mode as this will facilitate continuous dialogue with the dispatcher including (if required) CPR instructions. It seems reasonable that CPR training should include how to activate the speaker phone. Additional bystanders may be used to call the ambulance service.

Circulation

In adults needing CPR, there is a high probability of a primary cardiac cause for their cardiac arrest. When blood flow stops after cardiac arrest, the blood in the lungs and arterial system remains oxygenated for some minutes. To emphasise the priority of chest compressions, start CPR with chest compressions rather than initial ventilations.

Deliver compressions ‘in the centre of the chest’

Experimental studies show better haemodynamic responses when chest compressions are performed on the lower half of the sternum. Teach this location simply, such as, “place the heel of your hand in the centre of the chest with the other hand on top”. Accompany this instruction by a demonstration of placing the hands on the lower half of the sternum.

Chest compressions are most easily delivered by a single CPR provider kneeling by the side of the victim, as this facilitates movement between compressions and ventilations with minimal interruptions. Over-the-head CPR for single CPR providers and straddle-CPR for two CPR providers may be considered when it is not possible to perform compressions from the side, for example when the victim is in a confined space.

Compress the chest to a depth of 5–6 cm

Fear of doing harm, fatigue and limited muscle strength frequently result in CPR providers compressing the chest less deeply than recommended. Four observational studies, published after the 2010 Guidelines, suggest that a
Compression depth range of 4.5–5.5 cm in adults leads to better outcomes than all other compression depths during manual CPR. The Resuscitation Council (UK) endorses the ILCOR recommendation that it is reasonable to aim for a chest compression depth of approximately 5 cm but not more than 6 cm in the average sized adult. In making this recommendation, the Resuscitation Council (UK) recognises that it can be difficult to estimate chest compression depth and that compressions that are too shallow are more harmful than compressions that are too deep. Training should continue to prioritise achieving adequate compression depth.

**Compress the chest at a rate of 100–120 per minute (min⁻¹)**

Two studies, with a total of 13,469 patients, found higher survival among patients who received chest compressions at a rate of 100–120 min⁻¹. Very high chest compression rates were associated with declining chest compression depths. The Resuscitation Council (UK) therefore recommends that chest compressions are performed at a rate of 100–120 min⁻¹.

**Minimise pauses in chest compressions**

Delivery of rescue breaths, defibrillation shocks, ventilations and rhythm analysis lead to pauses in chest compressions. Pre- and post-shock pauses of less than 10 seconds, and minimising interruptions in chest compressions (proportion of resuscitation attempt delivering chest compression >60% (chest compression fraction) are associated with improved outcomes. Pauses in chest compressions should be minimised and training should emphasise the importance of close co-operation between CPR providers to achieve this.

**Chest recoil**

Leaning on the chest preventing full chest wall recoil is common during CPR. Allowing complete recoil of the chest after each compression results in better venous return to the chest and may improve the effectiveness of CPR. CPR providers should, therefore, take care to avoid leaning forward after each chest compression.

**Duty cycle**

The proportion of a chest compression spent in compression compared to relaxation is referred to as the duty cycle. There is very little evidence to recommend any specific duty cycle and, therefore, insufficient new evidence to prompt a change from the currently recommended ratio of 50%.

**Feedback on compression technique**

CPR feedback and prompt devices (e.g. voice prompts, metronomes, visual dials, numerical displays, waveforms, verbal prompts, and visual alarms) should be used when possible during CPR training. Their use during clinical practice should be integrated with comprehensive CPR quality improvement initiatives rather than as an isolated intervention.

**CPR provider fatigue**

Chest compression depth can decrease as soon as two minutes after starting chest compressions. If there are sufficient trained CPR providers, they should change over approximately every two minutes to prevent a decrease in compression quality. Changing CPR providers should not interrupt chest compressions.

**Rescue breaths**

CPR providers should give rescue breaths with an inflation duration of 1 second and provide sufficient volume to make the victim’s chest rise. Avoid rapid or forceful breaths. The maximum interruption in chest compression to give two breaths should not exceed 10 seconds.

**Mouth-to-nose ventilation**

Mouth-to-nose ventilation is an acceptable alternative to mouth-to-mouth ventilation. It may be considered if the victim’s mouth is seriously injured or cannot be opened, the CPR provider is assisting a victim in the water, or a mouth-to-mouth seal is difficult to achieve.

**Mouth-to-tracheostomy ventilation**

Mouth-to-tracheostomy ventilation may be used for a victim with a tracheostomy tube or tracheal stoma who requires rescue breathing.
Barrier devices for use with rescue breaths

Barrier devices decrease transmission of bacteria during rescue breathing in controlled laboratory settings. Their effectiveness in clinical practice is unknown.

If a barrier device is used, care should be taken to avoid unnecessary interruptions in CPR. Manikin studies indicate that the quality of CPR is improved when a pocket mask is used, compared to a bag-mask or simple face shield during basic life support.

Compression-only CPR

CPR providers trained and able to perform rescue breaths should perform chest compressions and rescue breaths as this may provide additional benefit for children and those who sustain an asphyxial cardiac arrest or where the EMS response interval is prolonged. Only if rescuers are unable to give rescue breaths should they do compression-only CPR.

The Resuscitation Council (UK) has carefully considered the balance between potential benefit and harm from compression-only CPR compared to standard CPR that includes ventilation. Our confidence in the equivalence between chest-compression-only and standard CPR is not sufficient to change current practice. The Resuscitation Council (UK), therefore, endorses the ILCOR and ERC recommendations that CPR providers should perform chest compressions for all patients in cardiac arrest. CPR providers trained and able to perform rescue breaths should perform chest compressions and rescue breaths as this may provide additional benefit for children and those who sustain an asphyxial cardiac arrest or where the EMS response interval is prolonged.

When an untrained bystander dials 999, the ambulance dispatcher should instruct him to give chest-compression-only CPR while awaiting the arrival of trained help. Further guidance on dispatcher-assisted CPR is given in the Prehospital resuscitation guidelines. www.resus.org.uk/resuscitation-guidelines/prehospital-resuscitation/

9. Use of an automated external defibrillator

AEDs are safe and effective when used by laypeople, including if they have had minimal or no training. AEDs may make it possible to defibrillate many minutes before professional help arrives. CPR providers should continue CPR with minimal interruption to chest compressions both while attaching an AED and during its use. CPR providers should concentrate on following the voice prompts, particularly when instructed to resume CPR, and minimising interruptions in chest compression.

Public access defibrillation (PAD) programmes

Public access AED programmes should be actively implemented in public places with a high density and movement of people such as airports, railway stations, bus terminals, sport facilities, shopping malls, stadiums, centres, offices, and casinos – where cardiac arrests are frequently witnessed and trained CPR providers can quickly be on scene. AEDs should also be provided in remote locations where an emergency ambulance response would be likely to be delayed (e.g. aircraft, ferries and off-shore locations). The potential benefits of AEDs being placed in schools as a method to raise awareness and familiarity with this lifesaving equipment is highlighted in the Education and implementation of resuscitation section. www.resus.org.uk/resuscitation-guidelines/education-and-implementation-of-resuscitation/

Registration of defibrillators with the local ambulance services is highly desirable so that dispatchers can direct CPR providers to the nearest AED.

When implementing an AED programme, community and programme leaders should consider factors such as the development of a team with responsibility for monitoring and maintaining the devices, training and retraining individuals who are likely to use the AED, and identification of a group of volunteer individuals who are committed to using the AED in victims of cardiac arrest. Funds must be allocated on a permanent basis to maintain the programme.

The Resuscitation Council (UK) and British Heart Foundation have produced information endorsed by the National Ambulance Service Medical Directors Group about AEDs and how they can be deployed in the community – A guide to Automated External Defibrillators. www.resus.org.uk/publications/a-guide-to-aeds/
Risks to recipients of CPR

It is extremely rare for bystander CPR to cause serious harm in victims who are eventually found not to be in cardiac arrest. Those who are in cardiac arrest and exposed to longer durations of CPR are likely to sustain rib and sternal fractures. Damage to internal organs can occur but is rare.\textsuperscript{55} The balance of benefits from bystander CPR far outweighs the risks. CPR providers should not, therefore, be reluctant to start CPR because of the concern of causing harm.

Risks to the CPR provider

CPR training and actual performance is safe in most circumstances. Although rare occurrences of muscle strain, back symptoms, shortness of breath, hyperventilation, pneumothorax, chest pain, myocardial infarction and nerve injury have been described in rescuers, the incidence of these events is extremely low. Individuals undertaking CPR training should be advised of the nature and extent of the physical activity required during the training programme. Learners and CPR providers who develop significant symptoms (e.g. chest pain or severe shortness of breath) during CPR training should be advised to stop and seek medical attention.

Although injury to the CPR provider from a defibrillator shock is extremely rare, standard surgical or clinical gloves do not provide adequate electrical protection. CPR providers, therefore, should not continue manual chest compressions during shock delivery. Avoid direct contact between the CPR provider and the victim when defibrillation is performed. Implantable cardioverter defibrillators (ICDs) can discharge without warning during CPR and rescuers may therefore be in contact with the patient when this occurs. However the current reaching the rescuer from the ICD is minimal and harm to the rescuer is unlikely.

Adverse psychological effects after performing CPR are relatively rare. If symptoms do occur the CPR provider should consult their general practitioner.

10. Choking

Choking is an uncommon but potentially treatable cause of accidental death. As most choking events are associated with eating, they are commonly witnessed. As victims are initially conscious and responsive, early interventions can be life-saving.

Recognition

Recognition of airway obstruction is the key to successful outcome, so do not confuse this emergency with fainting, myocardial infarction, seizure or other conditions that may cause sudden respiratory distress, cyanosis or loss of consciousness. Choking usually occurs while the victim is eating or drinking. People at increased risk of choking include those with reduced consciousnes, drug and/or alcohol intoxication, neurological impairment with reduced swallowing and cough reflexes (e.g. stroke, Parkinson’s disease), respiratory disease, mental impairment, dementia, poor dentition and older age.\textsuperscript{56}

Table 2 and Figure 3 present the treatment for the adult with choking. Foreign bodies may cause either mild or severe airway obstruction. It is important to ask the conscious victim “Are you choking?” The victim that is able to speak, cough and breathe has mild obstruction. The victim that is unable to speak, has a weakening cough, is struggling or unable to breathe, has severe airway obstruction.

<table>
<thead>
<tr>
<th>SEQUENCE</th>
<th>Technical description</th>
</tr>
</thead>
<tbody>
<tr>
<td>SUSPECT</td>
<td>Be alert to choking particularly if victim is eating</td>
</tr>
<tr>
<td>CHOKING</td>
<td></td>
</tr>
<tr>
<td>ENCOURAGE TO</td>
<td>Instruct victim to cough</td>
</tr>
<tr>
<td>COUGH</td>
<td></td>
</tr>
<tr>
<td>GIVE BACK</td>
<td>If cough becomes ineffective give up to 5 back blows</td>
</tr>
</tbody>
</table>

Table 2: Sequence of steps for managing the adult victim who is choking
Table 2: Sequence of steps for managing the adult victim who is choking

<table>
<thead>
<tr>
<th>SEQUENCE</th>
<th>Technical description</th>
</tr>
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</table>
| **BLOWS** | • Stand to the side and slightly behind the victim  
  • Support the chest with one hand and lean the victim well forwards so that when the obstructing object is dislodged it comes out of the mouth rather than goes further down the airway  
  • Give five sharp blows between the shoulder blades with the heel of your other hand |
| **GIVE ABDOMINAL THRUSTS** | If back blows are ineffective give up to 5 abdominal thrusts  
  • Stand behind the victim and put both arms round the upper part of the abdomen  
  • Lean the victim forwards  
  • Clench your fist and place it between the umbilicus (navel) and the ribcage  
  • Grasp this hand with your other hand and pull sharply inwards and upwards  
  • Repeat up to five times  
  • If the obstruction is still not relieved, continue alternating five back blows with five abdominal thrusts |
| **START CPR** | Start CPR if the victim becomes unresponsive  
  • Support the victim carefully to the ground  
  • Immediately activate the ambulance service  
  • Begin CPR with chest compressions |

Figure 3. Adult choking algorithm

A4-size algorithm: [http://resus.org.uk/_resources/assets/attachment/full/0/6446.pdf](http://resus.org.uk/_resources/assets/attachment/full/0/6446.pdf)
Treatment for mild airway obstruction

Coughing generates high and sustained airway pressures and may expel the foreign body. Aggressive treatment with back blows, abdominal thrusts and chest compressions at this stage may cause harm and can worsen the airway obstruction. These treatments are reserved for victims who have signs of severe airway obstruction. Victims with mild airway obstruction should remain under continuous observation until they improve, as severe airway obstruction may subsequently develop.

Treatment for severe airway obstruction

The clinical data on choking are largely retrospective and anecdotal. For conscious adults and children over one year of age with complete airway obstruction, case reports show the effectiveness of back blows or ‘slaps’, abdominal thrusts and chest thrusts. Approximately half of cases of airway obstruction are not relieved by a single technique. The likelihood of success is increased when combinations of back blows or slaps, and abdominal and chest thrusts are used.

Treatment of choking in an unresponsive victim

Higher airway pressures can be generated using chest thrusts compared with abdominal thrusts. Bystander initiation of chest compressions for unresponsive or unconscious victims of choking is associated with improved outcomes. Therefore, start chest compressions promptly if the victim becomes unresponsive or unconscious. After 30 compressions, attempt 2 rescue breaths, and continue CPR until the victim recovers and starts to breathe normally.

Aftercare and referral for medical review

Following successful treatment of choking, foreign material may nevertheless remain in the upper or lower airways and cause complications later. Victims with a persistent cough, difficulty swallowing or the sensation of an object being still stuck in the throat should, therefore, seek medical advice. Abdominal thrusts and chest compressions can potentially cause serious internal injuries and all victims successfully treated with these measures should be examined afterwards for injury.

11. Resuscitation of children and victims of drowning

Many children do not receive resuscitation because potential CPR providers fear causing harm if they are not specifically trained in resuscitation for children. This fear is unfounded: it is far better to use the adult BLS sequence for resuscitation of a child than to do nothing. For ease of teaching and retention, laypeople are taught that the adult sequence may also be used for children who are not responsive and not breathing normally. The following minor modifications to the adult sequence will make it even more suitable for use in children:

- Give 5 initial rescue breaths before starting chest compressions.
- If you are on your own, perform CPR for 1 minute before going for help.
- Compress the chest by at least one third of its depth, approximately 4 cm for the infant and approximately 5 cm for an older child. Use two fingers for an infant under 1 year; use one or two hands as needed for a child over 1 year to achieve an adequate depth of compression.

The same modifications of 5 initial breaths and 1 minute of CPR by the lone CPR provider before getting help may improve outcome for victims of drowning. This modification should be taught only to those who have a specific duty of care to potential drowning victims (e.g. lifeguards).

12. Acknowledgements

These guidelines have been adapted from the European Resuscitation Council 2015 Guidelines. We acknowledge and thank the authors of the ERC Guidelines for Adult basic life support and automated external defibrillation: Gavin D Perkins, Anthony J Handley, Rudolph W. Koster, Maaret Castrén, Michael A Smyth, Theresa Olasveengen, Koenraad G. Monsieurs, Violetta Raffay, Jan-Thorsten Gräsner, Volker Wenzel, Giuseppe Ristagno, Jasmeet Soar.
13. References


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

2. Summary of changes in advanced life support since 2010 Guidelines

The 2015 Advanced life support (ALS) guidelines have a change in emphasis aimed at improved care and implementation of these guidelines in order to improve patient outcomes. 3 The key changes since 2010 are:

- Increased emphasis on minimally interrupted high quality chest compressions throughout any ALS intervention.
- Chest compressions must only be paused briefly to enable specific interventions. This includes minimising interruptions in chest compressions to less than 5 seconds when attempting defibrillation or tracheal intubation.
- There is a new section on monitoring during ALS.
- Waveform capnography must be used to confirm and continually monitor tracheal tube placement, and may be used to monitor the quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).
- There are a variety of approaches to airway management during CPR and a stepwise approach based on
patient factors and the skills of the rescuer is recommended.

- The recommendations for drug therapy during CPR have not changed, but there is equipoise for the role of drugs in improving outcomes from cardiac arrest.
- The routine use of mechanical chest compression devices is not recommended, but they may be useful in situations where sustained high quality manual chest compressions are impractical or compromise provider safety.
- Peri-arrest ultrasound may be used to identify reversible causes of cardiac arrest.
- Extracorporeal life support techniques may be used as a rescue therapy in selected patients where standard ALS measures are not successful.
- The ALS algorithm (Figure 1) has been modified slightly to show these changes.

![Adult advanced life support algorithm](http://resus.org.uk/_resources/assets/attachment/full/0/6442.pdf)
3. Introduction

This section on adult advanced life support (ALS) adheres to the same general principles as Guidelines 2010, but incorporates some important changes. The guidelines in this section apply to healthcare professionals trained in ALS techniques. Laypeople, first responders, and automated external defibrillator (AED) users are referred to the Adult basic life support and automated external defibrillation section. [www.resus.org.uk/resuscitation-guidelines/adult-basic-life-support-and-automated-external-defibrillation/](http://www.resus.org.uk/resuscitation-guidelines/adult-basic-life-support-and-automated-external-defibrillation/)

Adult ALS includes advanced interventions after basic life support has started and when appropriate an AED has been used. The transition between basic and advanced life support should be seamless as BLS will continue during and overlap with ALS interventions. Post-resuscitation care guidelines are presented in a new section that recognises the importance of the final link in the Chain of Survival. [www.resus.org.uk/resuscitation-guidelines/post-resuscitation-care/](http://www.resus.org.uk/resuscitation-guidelines/post-resuscitation-care/)

These guidelines are based on the International Liaison Committee on Resuscitation (ILCOR) 2015 Consensus on Science and Treatment Recommendations (CoSTR) for ALS and the European Resuscitation Council 2015 Advanced Life Support Guidelines. These contain all the reference material for this section.

4. ALS treatment algorithm

Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The main difference in the treatment of these two groups is the need for attempted defibrillation in patients with VF/pVT.

Other actions, including chest compression, airway management and ventilation, vascular access, administration of adrenaline, and the identification and correction of reversible factors, are common to both groups. The ALS algorithm provides a standardised approach to the management of adult patients in cardiac arrest.

Drugs and advanced airways are still included among ALS interventions, but are of secondary importance to early defibrillation and high quality, uninterrupted chest compressions. At the time of writing these guidelines, three large randomised controlled trials (RCTs) (adrenaline versus placebo [ISRCTN73485024], amiodarone versus lidocaine versus placebo [NCT01401647] and supraglottic airway (i-gel) versus tracheal intubation [ISRCTN No: 08256118]) are currently ongoing.

**Shockable rhythms (VF/pVT)**

The first monitored rhythm is VF/pVT in approximately 20% of both in-hospital and out-of-hospital cardiac arrests (OHCAs). Ventricular fibrillation/pulseless ventricular tachycardia will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.

**Treatment of shockable rhythms (VF/VT)**

1. Confirm cardiac arrest – check for signs of life and normal breathing, and if trained to do so check for breathing and a pulse simultaneously.

2. Call resuscitation team.

3. Perform uninterrupted chest compressions while applying self-adhesive defibrillation/monitoring pads – one below the right clavicle and the other in the V6 position in the midaxillary line.

4. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.

5. Stop chest compressions; confirm VF/pVT from the ECG. This pause in chest compressions should be brief and no longer than 5 seconds.

6. Resume chest compressions immediately; warn all rescuers other than the individual performing the chest compressions to "stand clear" and remove any oxygen delivery device as appropriate.

7. The designated person selects the appropriate energy on the defibrillator and presses the charge button. Choose an energy setting of at least 150 J for the first shock, the same or a higher energy for subsequent shocks, or follow the manufacturer’s guidance for the particular defibrillator. If unsure of the correct energy level
for a defibrillator choose the highest available energy.

8. Ensure that the rescuer giving the compressions is the only person touching the patient.

9. Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to “stand clear”; when clear, give the shock.

10. After shock delivery immediately restart CPR using a ratio of 30:2, starting with chest compressions. Do not pause to reassess the rhythm or feel for a pulse. The total pause in chest compressions should be brief and no longer than 5 seconds.

11. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.

12. Pause briefly to check the monitor.

13. If VF/pVT, repeat steps 6–12 above and deliver a second shock.

14. If VF/pVT persists, repeat steps 6–8 above and deliver a third shock. Resume chest compressions immediately. Give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR. Withhold adrenaline if there are signs of return of spontaneous circulation (ROSC) during CPR.

15. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/pVT persists.

16. Give further adrenaline 1 mg IV after alternate shocks (i.e. approximately every 3–5 min).

17. If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC (check for signs of life, a central pulse and end-tidal CO₂ if available).
   
   a. If there is ROSC, start post-resuscitation care.
   
   b. If there are no signs of ROSC, continue CPR and switch to the non-shockable algorithm.

18. If asystole is seen, continue CPR and switch to the nonshockable algorithm.

The interval between stopping compressions and delivering a shock must be minimised. Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation. Chest compressions are resumed immediately after delivering a shock (without checking the rhythm or a pulse) because even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation. The duration of asystole before ROSC can be longer than 2 min in as many as 25% of successful shocks. If a shock has been successful immediate resumption of chest compressions does not increase the risk of VF recurrence. Furthermore, the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.

The use of waveform capnography can enable ROSC to be detected without pausing chest compressions and may be used as a way of avoiding a bolus injection of adrenaline after ROSC has been achieved. Several human studies have shown that there is a significant increase in end-tidal CO₂ when ROSC occurs. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Regardless of the arrest rhythm, after the initial adrenaline dose has been given, give further doses of adrenaline 1 mg every 3–5 min until ROSC is achieved; in practice, this will be about once every two cycles of the algorithm. If signs of life return during CPR (e.g. purposeful movement, normal breathing or coughing), or there is an increase in end-tidal CO₂, check the monitor; if an organised rhythm is present, check for a pulse. If a pulse is palpable, start post-resuscitation care. If no pulse is present, continue CPR.

Give amiodarone 300 mg IV after three defibrillation attempts irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest. Consider a further dose of amiodarone 150 mg IV after a total of five defibrillation attempts. Lidocaine 1 mg kg⁻¹ may be used as an alternative if amiodarone is not available but do not give lidocaine if amiodarone has been given already.

**Witnessed, monitored VF/pVT**

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.
- Rapidly check for a rhythm change and, if appropriate, ROSC after each defibrillation attempt.
- Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful.

This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is
already connected to a manual defibrillator – these circumstances are rare. Although there are no data supporting a three-shock strategy in any of these circumstances, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF/pVT.

If this initial three-shock strategy is unsuccessful for a monitored VF/pVT cardiac arrest, the ALS algorithm should be followed and these three-shocks treated as if only the first single shock has been given.

**Precordial thump**

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm. Its routine use is therefore not recommended. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus.

**Non-shockable rhythms (PEA and asystole)**

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse. These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure – this is sometimes described as ‘pseudo-PEA’ (see below). PEA can be caused by reversible conditions that can be treated if they are identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

**Treatment of PEA and asystole**

1. Start CPR 30:2
2. Give adrenaline 1 mg IV as soon as intravascular access is achieved
3. Continue CPR 30:2 until the airway is secured – then continue chest compressions without pausing during ventilation
4. Recheck the rhythm after 2 min:
   a. If electrical activity compatible with a pulse is seen, check for a pulse and/or signs of life
      i. If a pulse and/or signs of life are present, start post resuscitation care
      ii. If no pulse and/or no signs of life are present (PEA OR asystole):
         1. Continue CPR
         2. Recheck the rhythm after 2 min and proceed accordingly
         3. Give further adrenaline 1 mg IV every 3–5 min (during alternate 2-min loops of CPR)
   b. If VF/pVT at rhythm check, change to shockable side of algorithm.

Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because the patient may respond to cardiac pacing when there is ventricular standstill with continuing P waves. There is no value in attempting to pace true asystole.

**5. Treat reversible causes**

Potential causes or aggravating factors for which specific treatment exists must be considered during all cardiac arrests. For ease of memory, these are divided into two groups of four, based upon their initial letter: either H or T:

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- Hypothermia
- Thrombosis (coronary or pulmonary)
• Tension pneumothorax
• Tamponade – cardiac
• Toxins

**The four ‘Hs’**
Minimise the risk of hypoxia by ensuring that the patient’s lungs are ventilated adequately with the maximal possible inspired oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described below, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by hypovolaemia is due usually to severe haemorrhage. This may be precipitated by trauma, gastrointestinal bleeding or rupture of an aortic aneurysm. Stop the haemorrhage and restore intravascular volume with fluid and blood products.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient’s medical history (e.g. renal failure). Give IV calcium chloride in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.

Hypothermia should be suspected based on the history such as cardiac arrest associated with drowning.

**The four ‘Ts’**
Coronary thrombosis associated with an acute coronary syndrome or ischaemic heart disease is the most common cause of sudden cardiac arrest. An acute coronary syndrome is usually diagnosed and treated after ROSC is achieved. If an acute coronary syndrome is suspected, and ROSC has not been achieved, consider urgent coronary angiography when feasible and, if required, percutaneous coronary intervention. Mechanical chest compression devices and extracorporeal CPR can help facilitate this (see below).

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause of cardiac arrest consider giving a fibrinolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts. In some settings extracorporeal CPR, and/or surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

A tension pneumothorax can be the primary cause of PEA and may be associated with trauma. The diagnosis is made clinically or by ultrasound. Decompress rapidly by thoracostomy or needle thoracocentesis, and then insert a chest drain.

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy. The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable.

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or toxic substances may be revealed only by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment is supportive and standard ALS protocols should be followed.

**Use of ultrasound imaging during advanced life support**
When available for use by trained clinicians, focused echocardiography/ultrasound may be of use in assisting with diagnosis and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position has been recommended. Placement of the probe just before chest compressions are paused for a planned rhythm assessment enables a well-trained operator to obtain views within 10 seconds.

Several studies have examined the use of ultrasound during cardiac arrest to detect potentially reversible causes. Although no studies have shown that use of this imaging modality improves outcome, there is no doubt that echocardiography has the potential to detect reversible causes of cardiac arrest. Specific protocols for ultrasound evaluation during CPR may help to identify potentially reversible causes (e.g. cardiac tamponade, pulmonary embolism, hypovolaemia, pneumothorax). Absence of cardiac motion on sonography during resuscitation of patients in cardiac arrest is highly predictive of death although sensitivity and specificity has not been reported.
6. During CPR

High quality chest compressions with minimal interruption

During the treatment of persistent VF/pVT or PEA/asystole, there should be an emphasis on giving high quality chest compression between defibrillation attempts or rhythm checks, whilst recognising and treating reversible causes (4 Hs and 4 Ts), and whilst obtaining a secure airway and intravascular access. Aim for a chest compression pause of less than 5 seconds for rhythm checks, defibrillation attempts, and tracheal intubation. To achieve this rescuers must plan their actions before pausing compressions.

Monitoring during advanced life support

The following methods can be used to monitor the patient during CPR and help guide ALS interventions:

- Clinical signs such as breathing efforts, movements and eye opening can occur during CPR. These can indicate ROSC and require verification by a rhythm and pulse check, but can also occur because CPR can generate a sufficient circulation to restore signs of life including consciousness.32

- Pulse checks when there is an ECG rhythm compatible with an output can be used to identify ROSC, but may not detect pulses in those with low cardiac output states and a low blood pressure.33 The value of attempting to feel arterial pulses during chest compressions to assess the effectiveness of chest compressions is unclear. A pulse that is felt in the femoral triangle may indicate venous rather than arterial blood flow. There are no valves in the inferior vena cava and retrograde blood flow into the venous system can produce femoral vein pulsations.34 Carotid pulsation during CPR does not necessarily indicate adequate myocardial or cerebral perfusion.

- Monitoring heart rhythm through pads, paddles or ECG electrodes is a standard part of ALS. Motion artefacts prevent reliable heart rhythm assessment during chest compressions forcing rescuers to stop chest compressions to assess the rhythm, and preventing early recognition of recurrent VF/pVT. We suggest that artefact-filtering algorithms are not used for analysis of ECG rhythm during CPR unless as part of a research programme.35

- End-tidal CO₂ with waveform capnography. The use of waveform capnography during CPR has a greater emphasis in Guidelines 2015 and is addressed in more detail below.

- The use of CPR feedback or prompt devices during CPR should be considered only as part of a broader system of care that should include comprehensive CPR quality improvement initiatives 36-38 rather than an isolated intervention.

- Blood sampling and analysis during CPR can be used to identify potentially reversible causes of cardiac arrest. Avoid finger prick samples in critical illness because they may not be reliable; instead, use samples from veins or arteries.

- Blood gas values are difficult to interpret during CPR. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state.39 Analysis of central venous blood may provide a better estimation of tissue pH.

- Invasive cardiovascular monitoring in critical care settings (e.g. continuous arterial blood pressure and central venous pressure monitoring). Invasive arterial pressure monitoring will enable the detection of low blood pressure values when ROSC is achieved.

- Ultrasound assessment is addressed above to identify and treat reversible causes of cardiac arrest, and identify low cardiac output states (‘pseudo-PEA’).

Waveform capnography during advanced life support

Use waveform capnography whenever tracheal intubation is undertaken. Although the prevention of unrecognised oesophageal intubation is clearly beneficial, there is currently no evidence that use of waveform capnography during CPR results in improved patient outcomes. The role of waveform capnography during CPR includes:

- Ensuring tracheal tube placement in the trachea (although it will not distinguish between bronchial and tracheal placement).

- Monitoring ventilation rate during CPR and avoiding hyperventilation.
• Monitoring the quality of chest compressions during CPR. End-tidal CO₂ values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.⁴⁰ Whether this can be used to guide care and improve outcome requires further study.⁴¹

• Identifying ROSC during CPR. An increase in end-tidal CO₂ during CPR can indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC.¹⁴,⁴¹-⁴³ If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

• Prognostication during CPR. Precise values of end-tidal CO₂ depend on several factors including the cause of cardiac arrest, bystander CPR, chest compression quality, ventilation rate and volume, time from cardiac arrest and the use of adrenaline. Values are higher after an initial asphyxial arrest, with bystander CPR, and decline over time after cardiac arrest.⁴¹,⁴⁴,⁴⁵ Low end-tidal CO₂ values during CPR have been associated with lower ROSC rates and increased mortality, and high values with better ROSC and survival.⁴¹,⁴⁶,⁴⁷ The inter-individual differences and influence of cause of cardiac arrest, the problem with self-fulfilling prophecy in studies, our lack of confidence in the accuracy of measurement during CPR, and the need for an advanced airway to measure end-tidal CO₂ reliably limits our confidence in its use for prognostication. The Resuscitation Council (UK) recommends that a specific end-tidal CO₂ value at any time during CPR should not be used alone to stop CPR efforts. End-tidal CO₂ values should be considered only as part of a multi-modal approach to decision-making for prognostication during CPR.

Defibrillation

This section predominantly addresses the use of manual defibrillators. Guidelines concerning the use of an automated external defibrillator (AED) are addressed in the Adult basic life support and automated external defibrillation section. www.resus.org.uk/resuscitation-guidelines/adult-basic-life-support-and-automated-external-defibrillation The defibrillation strategy for the 2015 Resuscitation Guidelines has changed little from the former guidelines:

• The importance of early, uninterrupted chest compressions remains emphasised throughout these guidelines, together with minimising the duration of pre-shock and post-shock pauses – even 5–10 seconds delay will reduce the chances of the shock being successful.⁵⁸-⁵³

• Continue chest compressions during defibrillator charging, deliver defibrillation with an interruption in chest compressions of no more than 5 seconds and immediately resume chest compressions following defibrillation.

• Place the right (sternal) electrode to the right of the sternum, below the clavicle. Place the apical paddle in the mid-axillary line, approximately over the V6 ECG electrode position. This electrode should be clear of any breast tissue. It is important that this electrode is placed sufficiently laterally.

• Defibrillation shock energy levels are unchanged from the 2010 Guidelines.

  ◦ Deliver the first shock with an energy of at least 150 J.
  ◦ The shock energy for a particular defibrillator should be based on the manufacturer’s guidance.
  ◦ Those using manual defibrillators should be aware of the appropriate energy settings for the type of device used, but in the absence of this and if appropriate energy levels are unknown, for adults use the highest available shock energy for all shocks.
  ◦ If an initial shock has been unsuccessful it is worth attempting the second and subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy but, based on current evidence, both fixed and escalating strategies are acceptable.
  ◦ If VF/pVT recurs during a cardiac arrest (refibrillation) give subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy.

• There are no high quality clinical studies to indicate the optimal strategies within any given waveform and between different waveforms.² Knowledge gaps include the minimal acceptable first-shock energy level; the characteristics of the optimal biphasic waveform; the optimal energy levels for specific waveforms; and the best shock strategy (fixed versus escalating). It is becoming increasingly clear that selected energy is a poor comparator with which to assess different waveforms as impedance-compensation and subtleties in waveform shape result in significantly different transmyocardial current between devices for any given selected energy. The optimal energy levels may ultimately vary between different manufacturers and associated waveforms. Manufacturers are encouraged to undertake high quality clinical trials to support their defibrillation strategy recommendations.

• No one must touch the patient during shock delivery. Standard clinical examination gloves (or bare hands) do not provide a safe level of electrical insulation.⁵⁴

• Use oxygen safely during defibrillation by:
Removing any oxygen mask or nasal cannulae and place them at least 1 m away from the patient’s chest during defibrillation.

Leaving the ventilation bag connected to the tracheal tube or other airway adjunct. Alternatively, disconnect the ventilation bag from the tracheal tube and move it at least 1 m from the patient’s chest during defibrillation.

Airway management and ventilation

The options for airway management and ventilation during CPR vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers. They include: no airway and no ventilation (compression-only CPR), compression-only CPR with the airway held open (with or without supplementary oxygen), mouth-to-mouth breaths, mouth-to-mask, bag-mask ventilation with simple airway adjuncts, supraglottic airways (SGAs), and tracheal intubation (inserted with the aid of direct laryngoscopy or videolaryngoscopy, or via a SGA).

In comparison with bag-mask ventilation and use of a SGA, tracheal intubation requires considerably more training and practice and can result in unrecognised oesophageal intubation and increased hands-off time. A bag-mask, a SGA and a tracheal tube are frequently used in the same patient as part of a stepwise approach to airway management but this has not been formally assessed. Patients who remain comatose after initial resuscitation from cardiac arrest will ultimately require tracheal intubation regardless of the airway technique used during cardiac arrest. Anyone attempting tracheal intubation must be well trained and equipped with waveform capnography. Personnel skilled in advanced airway management should attempt laryngoscopy and intubation without stopping chest compressions; a brief pause in chest compressions may be required as the tube is passed through the vocal cords, but this pause should be less than 5 seconds. In the absence of these, use bag-mask ventilation and/or an SGA until appropriately experience and equipped personnel are present.

There is no high quality evidence supporting one particular intervention over another. Depending on the circumstances and the skills of the rescuers, use either an advanced airway (tracheal intubation or supraglottic airway (SGA)) or a bag-mask for airway management during CPR.

Basic airway manoeuvres and airway adjuncts

Assess the airway. Use head tilt and chin lift, or jaw thrust to open the airway. Simple airway adjuncts (oropharyngeal or nasopharyngeal airways) are often helpful, and sometimes essential, to maintain an open airway. When there is a risk of cervical spine injury, establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation of the head and neck by an assistant. If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt in small increments until the airway is open; establishing a patent airway takes priority over concerns about a potential cervical spine injury.

Oxygen during CPR

During CPR, give the maximal feasible inspired oxygen concentration. There are no data to indicate the optimal arterial blood oxygen saturation (SaO₂) during CPR, and no trials comparing different inspired oxygen concentrations. In one observational study of patients receiving 100% inspired oxygen via a tracheal tube during CPR, a higher measured partial pressure of arterial oxygen (PaO₂) value during CPR was associated with ROSC and hospital admission. The worse outcomes associated with a low PaO₂ during CPR could, however, be an indication of illness severity.

After ROSC, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration. This is addressed in the Post-resuscitation care section.

Ventilation

Provide artificial ventilation as soon as possible in any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective but the rescuer’s expired oxygen concentration is only 16–17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. A pocket resuscitation mask enables mouth-to-mask ventilation and some enable supplemental oxygen to be given. Use a two-hand technique to maximise the seal with the patient’s face. A self-inflating bag can be connected to a face mask, tracheal tube, or SGA. The two-person technique for bag-mask ventilation is preferable. Deliver each breath over approximately 1 second and give a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time
for chest compression. During CPR with an unprotected airway, give two ventilations after each sequence of 30 chest compressions. Once a tracheal tube or SGA has been inserted, ventilate the lungs at a rate of about 10 breaths min$^{-1}$ and continue chest compression without pausing during ventilation.$^{2,5}$

**Alternative airway devices**

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest.$^{62}$ There is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (2.4–17% in several studies involving paramedics)$^{63-67}$ and dislodgement, is unacceptably high.$^{68}$ Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral perfusion. Several alternative airway devices have been used for airway management during CPR.

There are published studies on the use during CPR of the Combitube, the classic laryngeal mask airway (cLMA), the Laryngeal Tube (LT) and the i-gel, and the LMA Supreme (LMAS) but none of these studies has been powered adequately to enable survival to be studied as a primary endpoint. Instead, most researchers have studied insertion and ventilation success rates. The SGAs are easier to insert than a tracheal tube and,$^{69}$ unlike tracheal intubation, can generally be inserted without interrupting chest compressions.$^{70}$

**Laryngeal mask airway (LMA)**

An LMA is relatively easy to insert, and ventilation using an LMA is more efficient and easier than with a bag-mask. If gas leakage is excessive, chest compression will have to be interrupted to enable ventilation. Although an LMA does not protect the airway as reliably as a tracheal tube, pulmonary aspiration is uncommon when using an LMA during cardiac arrest. The original LMA (classic LMA [cLMA]) has been superseded by several second generation SGAs that have more favourable characteristics, particularly when used for emergency airway management.$^{71}$

**I-gel**

The cuff of the i-gel does not require inflation; the stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube. It is very easy to insert, requiring only minimal training and a laryngeal seal pressure of 20–24 cmH$_2$O can be achieved.$^{72,73}$ The ease of insertion of the i-gel and its favourable leak pressure make it theoretically very attractive as a resuscitation airway device for those inexperienced in tracheal intubation. In observational studies insertion success rates for the i-gel were 93% (n = 98) when used by paramedics for out-of-hospital cardiac arrest (OHCA)$^{74}$ and 99% (n=100) when used by doctors and nurses for in-hospital cardiac arrest (IHCA).$^{75}$ The i-gel is in widespread use in the UK for both IHCA and OHCA.

**LMA Supreme (LMAS)**

The LMAS is a disposable version of the Proseal LMA, which is used in anaesthetic practice. In an observational study, paramedics inserted the LMAS successfully and were able to ventilate the lungs of 33 (100%) cases of OHCA.$^{76}$

**Tracheal intubation**

Tracheal intubation should be attempted only by trained personnel able to carry out the procedure with a high level of skill and confidence. No intubation attempt should interrupt chest compressions for more than 5 seconds. Use an alternative airway technique if tracheal intubation is not possible.

Healthcare personnel who undertake prehospital intubation should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills. Rescuers must weigh the risks and benefits of intubation against the need to provide effective chest compressions. The intubation attempt may require some interruption of chest compressions but, once an advanced airway is in place, ventilation will not require interruption of chest compressions. Personnel skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compressions; a brief pause in chest compressions will be required only as the tube is passed through the vocal cords. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until ROSC;$^{77,78}$ this strategy is being studied in a large prehospital randomised trial.$^{79}$ The intubation attempt should interrupt chest compressions for less than 5 seconds; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, tube placement must be confirmed and the tube secured adequately.

**Videolaryngoscopy**

Videolaryngoscopes are being used increasingly in anaesthetic and critical care practice.$^{80,81}$ In comparison with
direct laryngoscopy, they enable a better view of the larynx and improve the success rate of intubation. Preliminary studies indicate that use of videolaryngoscopes improve laryngeal view and intubation success rates during CPR but further data are required before recommendations can be made for wider use during CPR.

Confirmation of correct placement of the tracheal tube
The Resuscitation Council (UK) recommends using waveform capnography to confirm and continuously monitor the position of a tracheal tube during CPR in addition to clinical assessment. End-tidal CO₂ detectors that include a waveform graphical display (capnographs) are the most reliable for verification of tracheal tube position during cardiac arrest.

Clinical assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and adequate) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement alone (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not reliable. The reported sensitivity (proportion of tracheal intubations correctly identified) and specificity (proportion of oesophageal intubations correctly identified) of clinical assessment varies: sensitivity 7–100%; specificity 66–100%.

Based on the available data, the accuracy of colormetric CO₂ detectors, oesophageal detector devices and non-waveform capnometers does not exceed the accuracy of auscultation and direct visualisation for confirming the tracheal position of a tube in victims of cardiac arrest. Waveform capnography is the most sensitive and specific way to confirm and continuously monitor the position of a tracheal tube in victims of cardiac arrest and must supplement clinical assessment (auscultation and visualisation of tube through cords). Waveform capnography will not discriminate between tracheal and bronchial placement of the tube – careful auscultation is essential. Existing portable monitors make capnographic initial confirmation and continuous monitoring of tracheal tube position feasible in almost all settings, including out-of-hospital, emergency department and in-hospital locations where intubation is performed.

Cricothyroidotomy
If it is impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or alternative airway device, delivery of oxygen through a cannula or surgical cricothyroidotomy may be life saving. A tracheostomy is contraindicated in an emergency, as it is time consuming, hazardous and requires considerable surgical skill and equipment.

Surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient’s lungs until semi-elective intubation or tracheostomy is performed. Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source, runs the risk of barotrauma and can be particularly ineffective in patients with chest trauma. It is also prone to failure because of kinking of the cannula, and is unsuitable for patient transfer. In the 4th National Audit Project of the UK Royal College of Anaesthetists and the Difficult Airway Society (NAP4), 60% of needle cricothyroidotomies attempted failed. In contrast, all surgical cricothyroidotomies achieved access to the trachea. While there may be several underlying causes, these results indicate a need for more training in surgical cricothyroidotomy and this should include regular manikin-based training using locally available equipment.

Drugs for cardiac arrest
The ILCOR systematic reviews found insufficient evidence to comment on critical outcomes such as survival to discharge and survival to discharge with good neurological outcome with any drug during CPR. There was also insufficient evidence to comment on the best time to give drugs to optimise outcome.

Thus, although drugs are still included among ALS interventions, they are of secondary importance to high quality uninterrupted chest compressions and early defibrillation.

Adrenaline
Despite the continued widespread use of adrenaline during resuscitation, there is no placebo-controlled study that shows that the routine use of adrenaline during human cardiac arrest increases survival to hospital discharge, although improved short-term survival has been documented.

The current recommendation is to continue the use of adrenaline during CPR as for Guidelines 2010. We have considered the benefit in short-term outcomes (ROSC and admission to hospital) and our uncertainty about the benefit or harm on survival to discharge and neurological outcome given the limitations of the observational studies.
The Resuscitation Council (UK) has decided not to recommend a change to current practice until there are high quality data on long-term outcomes. Dose response and placebo-controlled efficacy trials are needed to evaluate the use of adrenaline in cardiac arrest. There is an ongoing randomised study of adrenaline vs. placebo for OHCA in the UK (PARAMEDIC 2: The Adrenaline Trial, ISRCTN73485024).

Amiodarone

No anti-arrhythmic drug given during human cardiac arrest has been shown to increase survival to hospital discharge, although amiodarone has been shown to increase survival to hospital admission.6,97-99 Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest. There is an ongoing trial comparing amiodarone to lidocaine and to placebo designed and powered to evaluate for functional survival.6

Vascular access during CPR

The role of drugs during cardiac arrest is uncertain. Some patients will already have intravenous access before they have a cardiac arrest. If this is not the case ensure CPR had started and defibrillation, if appropriate, attempted before considering vascular access.

Peripheral versus central venous drug delivery

Although peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central venous catheter compared with a peripheral cannula,99 insertion of a central venous catheter requires interruption of CPR and can be technically challenging and associated with complications. Peripheral venous cannulation is quicker, easier to perform and safer. Drugs injected peripherally must be followed by a flush of at least 20 mL of fluid and elevation of the extremity for 10–20 seconds to facilitate drug delivery to the central circulation.

Intraosseous route

If intravenous access is difficult or impossible, consider the intraosseous (IO) route. This is now established as an effective route in adults.100-108 Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a vein.109-110 Animal studies suggest that adrenaline reaches a higher concentration and more quickly when it is given intravenously as compared with the intraosseous route, and that the sternal intraosseous route more closely approaches the pharmacokinetics of IV adrenaline.111 The recent availability of mechanical IO devices has increased the ease of performing this technique.112 There are several intraosseous devices available as well as a choice of insertion sites including the humerus, proximal or distal tibia, and sternum. The decision concerning choice of device and insertion site should be made locally and staff adequately trained in its use.

7. CPR techniques and devices

Mechanical chest compression devices

We recommend that automated mechanical chest compression devices are not used routinely to replace manual chest compressions.

Automated mechanical chest compression devices are a reasonable alternative to high quality manual chest compressions in situations where sustained high quality manual chest compressions are impractical or compromise provider safety.2

Interruptions to CPR during device deployment should be avoided. Healthcare personnel who use mechanical CPR should do so only within a structured, monitored programme, which should include comprehensive competency-based training and regular opportunities to refresh skills.

Since Guidelines 2010 there have been three large RCTs enrolling 7582 patients that have shown no clear advantage for the routine use of automated mechanical chest compression for OHCA using the Lund University Cardiac Arrest System (LUCAS)113,114 and AutoPulse devices.115 Ensuring high quality chest compressions with adequate depth, rate and minimal interruptions, regardless of whether they are delivered by machine or human is important.116,117 Mechanical compressions usually follow a period of manual compressions;118 the transition from manual compressions to mechanical compressions whilst minimising interruptions to chest compression and avoiding delays in defibrillation is therefore an important aspect of using these devices. The use of training drills and ‘pit-crew’
techniques for device deployment are suggested to help minimise interruptions in chest compression.\textsuperscript{119-121}

**Extracorporeal cardiopulmonary resuscitation (ECPR)**

Extracorporeal CPR (ECPR) should be considered as a rescue therapy for those patients in whom initial ALS measures are unsuccessful and, or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI) or pulmonary thrombectomy for massive pulmonary embolism).\textsuperscript{122,123} There is an urgent need for randomised studies of ECPR and large ECPR registries to identify the circumstances in which it works best, establish guidelines for its use and identify the benefits, costs and risks of ECPR.\textsuperscript{124,125}

Extracorporeal techniques require vascular access and a circuit with a pump and oxygenator and can provide a circulation of oxygenated blood to restore tissue perfusion. This has the potential to buy time for restoration of an adequate spontaneous circulation, and treatment of reversible underlying conditions. This is commonly called extracorporeal life support (ECLS), and more specifically extracorporeal CPR (ECPR) when used during cardiac arrest. These techniques are becoming more commonplace and have been used for both in-hospital and out-of-hospital cardiac arrest despite limited observational data in select patient groups. Observational studies suggest ECPR for cardiac arrest is associated with improved survival when there is a reversible cause for cardiac arrest (e.g. myocardial infarction, pulmonary embolism, severe hypothermia, poisoning), there is little comorbidity, the cardiac arrest is witnessed, the individual receives immediate high quality CPR, and ECPR is implemented early (e.g. within 1 hour of collapse) including when instituted by emergency physicians and intensivists.\textsuperscript{126-132}

The implementation of ECPR requires considerable resource and training. When compared with manual or mechanical CPR, ECPR has been associated with improved survival after IHCA in selected patients.\textsuperscript{126,128} After OHCA outcomes with both standard and ECPR are less favourable.\textsuperscript{133} The duration of standard CPR before ECPR is established and patient selection are important factors for success.\textsuperscript{122,126,130,132,134-136}

### 8. Duration of resuscitation attempt

If attempts at obtaining ROSC are unsuccessful the resuscitation team leader should discuss stopping CPR with the team. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing, as long as the patient remains in VF/pVT, or there is a potentially reversible cause than can be treated. The use of mechanical compression devices and extracorporeal CPR techniques make prolonged attempts at resuscitation feasible in selected patients. It is generally accepted that asystole for more than 20 minutes in the absence of a reversible cause and with ongoing ALS constitutes a reasonable ground for stopping further resuscitation attempts.\textsuperscript{137}

### 9. Acknowledgements

These guidelines have been adapted from the European Resuscitation Council 2015 Guidelines. We acknowledge and thank the authors of the ERC Guidelines for Adult advanced life support:

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Paediatric basic life support

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

2. Summary of changes in paediatric basic life support since the 2010 Guidelines

- The duration of delivering a breath is about 1 second, to coincide with adult practice.
- For chest compressions, the lower sternum should be depressed by at least one third the anterior-posterior diameter of the chest, or by 4 cm for the infant and 5 cm for the child.

3. Introduction

These guidelines aim to provide clear advice to healthcare professionals and members of the general public about the delivery of basic life support (BLS) to children.

Cardiorespiratory arrest occurs less frequently in children than in adults; thus, both healthcare professionals and lay people are less likely to be involved in paediatric resuscitation. It is therefore important to be familiar with the
knowledge and skills required for paediatric BLS so that the best care possible can be delivered in what is often a stressful situation.

The review of these guidelines has incorporated new information from the ILCOR 2015 review of resuscitation science. They also include practical changes intended to aid training and retention of knowledge and skills required to deliver high quality BLS in children.

There is limited evidence on paediatric resuscitation as there are relatively few studies, particularly randomised controlled trials, in this area. Existing evidence derived from observational studies is of variable quality but new collaborations involving national and international registries should yield valuable data on the resuscitation of children.

What is known is that cardiopulmonary resuscitation (CPR) should start as soon as possible for optimum outcome. This should start with the first person on scene, who is often a bystander (i.e. a lay rescuer).

The majority of paediatric cardiorespiratory arrests are not caused by primary cardiac problems but are secondary to other causes, mostly respiratory insufficiency, hence the order of delivering the resuscitation sequence: Airway (A), Breathing (B) and Circulation (C).

- To promote the delivery of BLS by the general public, this section confirms that using the adult BLS sequence for a child is far better than not performing any CPR, and describes a modification to the adult BLS sequence for use by non-specialists. This modified sequence of BLS will make it more likely that potential rescuers will commence CPR and so improve the outcomes for critically ill children.

- For healthcare professionals with a duty to respond to paediatric emergencies, a more specific form of paediatric BLS is presented as they have an obligation to deliver more targeted care.

4. Guideline notes

Recognition of cardiorespiratory arrest – healthcare provider and lay person

If a layperson or healthcare provider considers that there are no ‘signs of life’, CPR should be started immediately.

Feeling for a pulse is not a reliable way to determine if there is an effective or inadequate circulation, and palpation of the pulse is not the sole determinant of the need for chest compressions. The presence or absence of ‘signs of life’, such as response to stimuli, normal breathing (rather than abnormal gasps) or spontaneous movement must be looked for as part of the child’s circulatory assessment. If a healthcare provider does feel for a pulse in an unresponsive child, they must be certain that one is present for them NOT to start CPR. In this situation, there are often other signs of life present. Lay rescuers should not be taught to feel for a pulse as part of the assessment of need for CPR.

The decision to start CPR should take less than 10 seconds from starting the initial assessment of the child’s circulatory status and if there is still doubt after that time, start CPR.

Compression: Ventilation ratios – healthcare provider and layperson

Although ventilation remains a very important component of CPR in children, rescuers who are unable or unwilling to provide breaths should be encouraged to perform at least compression-only CPR. A child is far more likely to be harmed if the bystander does nothing.

All providers should be encouraged to initiate CPR in children even if they haven’t been taught specific paediatric techniques. CPR should be started with the C:V ratio that is familiar and for most, this will be 30:2. The paediatric modifications to adult CPR should be taught to those who care for children but are unlikely to have to resuscitate them. The specific paediatric sequence incorporating the 15:2 ratio is primarily intended for those who have the potential to resuscitate children as part of their role.

Chest compression quality

Uninterrupted, high quality chest compression is vital, with attention being paid to all components of each chest compression including the rate, depth and allowing adequate time for chest recoil to occur (approximately 50% of the whole cycle should be the relaxation phase).
Training and feedback devices are being developed for adults and children but require absolute, rather than relative, dimensions for depth. In order to facilitate this for children, the measurement data indicate that the approximate dimensions of one-third compression depths in infants and children are about 4 cm and 5 cm respectively.

To maintain consistency with adult BLS guidelines, the compression rate remains at 100–120 min\(^{-1}\). Ideally chest compressions should be delivered on a firm surface otherwise the depth of compression may be difficult to achieve.

Figure 1. Paediatric basic life support algorithm
(Healthcare professionals with a duty to respond)

A4-size algorithm: http://resus.org.uk/_resources/assets/attachment/full/0/6456.pdf
5. Infant and child BLS sequence

Rescuers who have been taught adult BLS, and have no specific knowledge of paediatric resuscitation, should use the adult sequence. The following modifications to the adult sequence will make it more suitable for use in children:

- Give 5 initial rescue breaths before starting chest compression.
- If you are on your own, perform CPR for 1 min before going for help.
- Compress the chest by at least one-third of its depth, approximately 4 cm for an infant and approximately 5 cm for an older child. Use two fingers for an infant under 1 year; use one or two hands for a child over 1 year to achieve an adequate depth of compression.

The compression rate should be 100–120 min\(^{-1}\).


Those with a duty to respond to paediatric emergencies (usually healthcare professional teams) should use the following sequence:

1. Ensure the safety of rescuer and child.

2. Check the child’s responsiveness:
   - Gently stimulate the child and ask loudly, ‘Are you all right?’

3A. If the child responds by answering or moving:
   - Leave the child in the position in which you find him (provided he is not in further danger).
   - Check his condition and get help if needed.
   - Reassess him regularly.

3B. If the child does not respond:
   - Shout for help.
   - Turn the child onto his back and open the airway using head tilt and chin lift:
     - Place your hand on his forehead and gently tilt his head back.
     - With your fingertip(s) under the point of the child’s chin, lift the chin.
     - Do not push on the soft tissues under the chin as this may block the airway.
     - If you still have difficulty in opening the airway, try the jaw thrust method: place the first two fingers of each hand behind each side of the child’s mandible (jaw bone) and push the jaw forward.

Have a low threshold for suspecting injury to the neck. If you suspect this, try to open the airway using jaw thrust alone. If this is unsuccessful, add head tilt gradually until the airway is open. Establishing an open airway takes priority over concerns about the cervical spine.

4. Keeping the airway open, look, listen, and feel for normal breathing by putting your face close to the child’s face and looking along the chest:
   - Look for chest movements.
   - Listen at the child’s nose and mouth for breath sounds.
   - Feel for air movement on your cheek.

In the first few minutes after cardiac arrest a child may be taking infrequent, noisy gasps. Do not confuse this with normal breathing. Look, listen, and feel for no more than 10 seconds before deciding – if you have any doubts whether breathing is normal, act as if it is not normal.
5A. If the child IS breathing normally:

- Turn the child onto his side into the recovery position (see below).
- Send or go for help – call the relevant emergency number. Only leave the child if no other way of obtaining help is possible.
- Check for continued normal breathing.

5B. If the breathing is NOT normal or absent:

- Carefully remove any obvious airway obstruction.
- Give 5 initial rescue breaths.
- Although rescue breaths are described here, it is common in healthcare environments to have access to bag-mask devices. Providers trained in their use should use them as soon as they are available.
- While performing the rescue breaths note any gag or cough response to your action. These responses, or their absence, will form part of your assessment of ‘signs of life’, described below.

Rescue breaths for an infant:

- Ensure a neutral position of the head (as an infant’s head is usually flexed when supine, this may require some extension) and apply chin lift.
- Take a breath and cover the mouth and nasal apertures of the infant with your mouth, making sure you have a good seal. If the nose and mouth cannot both be covered in the older infant, the rescuer may attempt to seal only the infant’s nose or mouth with his mouth (if the nose is used, close the lips to prevent air escape).
- Blow steadily into the infant’s mouth and nose over 1 second sufficient to make the chest rise visibly. This is the same time period as in adult practice.
- Maintain head position and chin lift, take your mouth away, and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence four more times.

Rescue breaths for a child over 1 year:

- Ensure head tilt and chin lift.
- Pinch the soft part of his nose closed with the index finger and thumb of your hand on his forehead.
- Open his mouth a little, but maintain the chin lift.
- Take a breath and place your lips around his mouth, making sure that you have a good seal.
- Blow steadily into his mouth over 1 second sufficient to make the chest rise visibly.
- Maintaining head tilt and chin lift, take your mouth away and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence four more times. Identify effectiveness by seeing that the child’s chest has risen and fallen in a similar fashion to the movement produced by a normal breath.

For both infants and children, if you have difficulty achieving an effective breath, the airway may be obstructed:

- Open the child’s mouth and remove any visible obstruction. Do not perform a blind finger sweep.
- Ensure that there is adequate head tilt and chin lift but also that the neck is not over extended.
- If head tilt and chin lift has not opened the airway, try the jaw thrust method.
- Make up to 5 attempts to achieve effective breaths. If still unsuccessful, move on to chest compression.

6. Assess the circulation (signs of life):

Take no more than 10 seconds to:

- Look for signs of life. These include any movement, coughing, or normal breathing (not abnormal gasps or infrequent, irregular breaths).
- If you check the pulse take no more than 10 seconds:
  - In a child aged over 1 year – feel for the carotid pulse in the neck.
  - In an infant – feel for the brachial pulse on the inner aspect of the upper arm.
  - For both infants and children the femoral pulse in the groin (mid-way between the anterior superior iliac spine and the symphysis pubis) can also be used.
7A. If confident that you can detect signs of a circulation within 10 seconds:

- Continue rescue breathing, if necessary, until the child starts breathing effectively on his own.
- Turn the child onto his side (into the recovery position) if he starts breathing effectively but remains unconscious.
- Re-assess the child frequently.

7B. If there are no signs of life, unless you are CERTAIN that you can feel a definite pulse of greater than 60 min\(^{-1}\) within 10 seconds:

- Start chest compressions.
- Combine rescue breathing and chest compressions.

For all children, compress the lower half of the sternum:

- To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Compress the sternum one finger’s breadth above this.
- Compression should be sufficient to depress the sternum by at least one-third of the depth of the chest, which is approximately 4 cm for an infant and 5 cm for a child.
- Release the pressure completely, then repeat at a rate of 100–120 min\(^{-1}\).
- Allow the chest to return to its resting position before starting the next compression.
- After 15 compressions, tilt the head, lift the chin, and give two effective breaths.
- Continue compressions and breaths in a ratio of 15:2.

The best method for compression varies slightly between infants and children.

Chest compression in infants:

- The lone rescuer should compress the sternum with the tips of two fingers.
- If there are two or more rescuers, use the encircling technique:
  - Place both thumbs flat, side-by-side, on the lower half of the sternum (as above), with the tips pointing towards the infant’s head.
  - Spread the rest of both hands, with the fingers together, to encircle the lower part of the infant’s rib cage with the tips of the fingers supporting the infant’s back.
  - Press down on the lower sternum with your two thumbs to depress it at least one-third of the depth of the infant’s chest, approximately 4 cm.

Chest compression in children aged over 1 year:

- Place the heel of one hand over the lower half of the sternum (as above).
- Lift the fingers to ensure that pressure is not applied over the child’s ribs.
- Position yourself vertically above the victim’s chest and, with your arm straight, compress the sternum to depress it by at least one-third of the depth of the chest, approximately 5 cm.
- In larger children, or for small rescuers, this may be achieved most easily by using both hands with the fingers interlocked.

8. Continue resuscitation until:

- The child shows signs of life (normal breathing, cough, movement or definite pulse of greater than 60 min\(^{-1}\)).
- Further qualified help arrives.
- You become exhausted.

**When to call for assistance**

It is vital for rescuers to get help as quickly as possible when a child collapses:

- When more than one rescuer is available, one (or more) starts resuscitation while another goes for assistance.
• If only one rescuer is present, undertake resuscitation for about 1 min before going for assistance. To minimise interruptions in CPR, it may be possible to carry an infant or small child whilst summoning help.

• The only exception to performing 1 min of CPR before going for help is in the unlikely event of a child with a witnessed, sudden collapse when the rescuer is alone and primary cardiac arrest is suspected. In this situation, a shockable rhythm is likely and the child may need defibrillation. Seek help immediately if there is no one to go for you.

**Recovery position**

An unconscious child whose airway is clear and who is breathing normally should be turned onto his side into the recovery position. There are several recovery positions; each has its advocates. The important principles to be followed are:

• Place the child in as near a true lateral position as possible to enable the drainage of fluid from the mouth.

• Ensure the position is stable. In an infant, this may require the support of a small pillow or a rolled-up blanket placed behind his back to maintain the position.

• There should be no pressure on the chest that impairs breathing.

• It should be possible to turn the child onto his side and to return him back easily and safely, taking into consideration the possibility of cervical spine injury.

• Ensure the airway is accessible and easily observed.

• The adult recovery position is suitable for use in children.

**6. Explanatory notes**

**Definitions**

• A newborn is a child just after birth.

• A neonate is a child in the first 28 days of life.

• An infant is a child under 1 year.

• A child is between 1 year and puberty.

The differences between adult and paediatric resuscitation are largely based on differing aetiology, with primary cardiac arrest being more common in adults whereas children usually suffer from secondary cardiac arrest. The onset of puberty, which is the physiological end of childhood, is the most logical landmark for the upper age limit for use of paediatric guidelines. This has the advantage of being simple to determine in contrast to an age limit, as age may be unknown at the start of resuscitation. Clearly, it is inappropriate and unnecessary to establish the onset of puberty formally; if the rescuer believes the victim to be a child then he should use the paediatric guidelines. If a misjudgement is made, and the victim turns out to be a young adult, little harm will accrue as studies of aetiology have shown that the paediatric pattern of arrest continues into early adulthood.

It is necessary to differentiate between infants and older children, as there are some important differences between these two groups.

**Automated external defibrillators (AEDs)**

Since the publication of Guidelines 2010 there have been continuing reports of safe and successful use of AEDs in children less than 8 years demonstrating that AEDs are capable of identifying arrhythmias accurately in children and are extremely unlikely to advise a shock inappropriately. Nevertheless, if there is any possibility that an AED may need to be used in children, the purchaser should check that the performance of the particular model has been tested in paediatric arrhythmias.

Many manufacturers now supply purpose-made paediatric pads or programmes, which typically attenuate the output of the machine to 50–75 J. These devices are recommended for children between 1 and 8 years. If no such system or manually adjustable machine is available, an unmodified adult AED may be used.

Although shockable rhythms are extremely unusual in infants, and the focus of infant resuscitation should be on high quality CPR, there are rare case reports of the successful use of AEDs in this age group. For an infant in a shockable
rhythm, the risk: benefit ratio favours the use of an AED (preferably with an attenuator) if a manually adjustable model is not available.

7. Choking

Recognition of choking

The management of the choking child remains unaltered from 2010 and the sequence of reversing partial or complete obstruction of the airways is the same. Back blows, chest thrusts and abdominal thrusts all increase intra-thoracic pressure and can expel foreign bodies from the airway. In half of the episodes documented with airway obstruction, more than one technique was needed to relieve the obstruction. There are no data to indicate which technique should be used first or in which order they should be applied. If one is unsuccessful, try the others in rotation until the object is cleared.

When a foreign body enters the airway the child reacts immediately by coughing in an attempt to expel it. A spontaneous cough is likely to be more effective and safer than any manoeuvre a rescuer might perform. However, if coughing is absent or ineffective, and the object completely obstructs the airway, the child will become asphyxiated rapidly. Active interventions to relieve choking are therefore required only when coughing becomes ineffective, but they then must be commenced rapidly and confidently.

The majority of choking events in children occur during play or whilst eating, when a carer is usually present. Events are therefore frequently witnessed, and interventions are usually initiated when the child is conscious.

Choking is characterised by the sudden onset of respiratory distress associated with coughing, gagging, or stridor. Similar signs and symptoms may also be associated with other causes of airway obstruction, such as laryngitis or epiglottitis, which require different management. Suspect choking caused by a foreign body if:

- the onset was very sudden
- there are no other signs of illness
- there are clues to alert the rescuer (e.g. a history of eating or playing with small items immediately prior to the onset of symptoms).

<table>
<thead>
<tr>
<th>General signs of choking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Witnessed episode</td>
</tr>
<tr>
<td>Coughing or choking</td>
</tr>
<tr>
<td>Sudden onset</td>
</tr>
<tr>
<td>Recent history of playing with or eating small objects</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Ineffective coughing</th>
<th>Effective cough</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unable to vocalise</td>
<td>Crying or verbal response to questions</td>
</tr>
<tr>
<td>Quiet or silent cough</td>
<td>Loud cough</td>
</tr>
<tr>
<td>Unable to breathe</td>
<td>Able to take a breath before coughing</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Fully responsive</td>
</tr>
<tr>
<td>Decreasing level of consciousness</td>
<td></td>
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</tbody>
</table>

When a foreign body enters the airway the child reacts immediately by coughing in an attempt to expel it. A spontaneous cough is likely to be more effective and safer than any manoeuvre a rescuer might perform. However, if coughing is absent or ineffective, and the object completely obstructs the airway, the child will become asphyxiated rapidly. Active interventions to relieve choking are therefore required only when coughing becomes ineffective, but they then must be commenced rapidly and confidently.
Relief of choking

Safety and summoning assistance

Consider the safest action to manage the choking child:

- If the child is coughing effectively, then no external manoeuvre is necessary. Encourage the child to cough, and monitor continuously.
- If the child’s coughing is, or is becoming, ineffective, shout for help immediately and determine the child’s conscious level.

Conscious child with choking

- If the child is still conscious but has absent or ineffective coughing, give back blows.
- If back blows do not relieve choking, give chest thrusts to infants or abdominal thrusts to children. These manoeuvres create an ‘artificial cough’ to increase intrathoracic pressure and dislodge the foreign body.

Back blows

- In an infant:
  - Support the infant in a head-downwards, prone position, to enable gravity to assist removal of the foreign body.
  - A seated or kneeling rescuer should be able to support the infant safely across his lap.
  - Support the infant’s head by placing the thumb of one hand at the angle of the lower jaw, and one or two fingers from the same hand at the same point on the other side of the jaw.
  - Do not compress the soft tissues under the infant’s jaw, as this will exacerbate the airway obstruction.
  - Deliver up to 5 sharp back blows with the heel of one hand in the middle of the back between the shoulder
The aim is to relieve the obstruction with each blow rather than to give all 5.

- **In a child over 1 year:**
  - Back blows are more effective if the child is positioned head down.
  - A small child may be placed across the rescuer’s lap as with an infant.
  - If this is not possible, support the child in a forward-leaning position and deliver the back blows from behind.

If back blows fail to dislodge the object, and the child is still conscious, use chest thrusts for infants or abdominal thrusts for children. Do not use abdominal thrusts (Heimlich manoeuvre) for infants.

### Chest thrusts for infants:
- Turn the infant into a head-downwards supine position. This is achieved safely by placing your free arm along the infant’s back and encircling the occiput with your hand.
- Support the infant down your arm, which is placed down (or across) your thigh.
- Identify the landmark for chest compression (lower sternum approximately a finger’s breadth above the xiphisternum).
- Deliver up to 5 chest thrusts. These are similar to chest compressions, but sharper in nature and delivered at a slower rate.
- The aim is to relieve the obstruction with each thrust rather than to give all 5.

### Abdominal thrusts for children over 1 year:
- Stand or kneel behind the child. Place your arms under the child’s arms and encircle his torso.
- Clench your fist and place it between the umbilicus and xiphisternum.
- Grasp this hand with your other hand and pull sharply inwards and upwards.
- Repeat up to 4 more times.
- Ensure that pressure is not applied to the xiphoid process or the lower rib cage as this may cause abdominal trauma.
- The aim is to relieve the obstruction with each thrust rather than to give all 5.

### Following chest or abdominal thrusts, reassess the child:
- If the object has not been expelled and the victim is still conscious, continue the sequence of back blows and chest (for infant) or abdominal (for children) thrusts.
- Call out, or send, for help if it is still not available.
- Do not leave the child at this stage.

If the object is expelled successfully, assess the child’s clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance.

### Unconscious child with choking
- If the choking child is, or becomes, unconscious place him on a firm, flat surface.
- Call out, or send, for help if it is still not available.
- Do not leave the child at this stage.

### Airway opening:
- Open the mouth and look for any obvious object.
- If one is seen, make an attempt to remove it with a single finger sweep.

**Do not attempt blind or repeated finger sweeps** – these can push the object more deeply into the pharynx and cause injury.
Rescue breaths:
• Open the airway and attempt 5 rescue breaths.
• Assess the effectiveness of each breath: if a breath does not make the chest rise, reposition the head before making the next attempt.

Chest compression and CPR:
• Attempt 5 rescue breaths and if there is no response, proceed immediately to chest compression regardless of whether the breaths are successful.
• Follow the sequence for single rescuer CPR (step 7B above) for approximately 1 min before summoning an ambulance (if this has not already been done by someone else).
• When the airway is opened for attempted delivery of rescue breaths, look to see if the foreign body can be seen in the mouth.
• If an object is seen, attempt to remove it with a single finger sweep.
• If it appears that the obstruction has been relieved, open and check the airway as above. Deliver rescue breaths if the child is not breathing and then assess for signs of life. If there are none, commence chest compressions and perform CPR (step 7B above).
• If the child regains consciousness and is breathing effectively, place him in a safe side-lying (recovery) position and monitor breathing and conscious level whilst awaiting the arrival of the ambulance.

8. Acknowledgements
These guidelines have been adapted from the European Resuscitation Council 2015 Guidelines. We acknowledge and thank the authors of the ERC Guidelines for Paediatric life support: Ian K. Maconochie, Robert Bingham, Christoph Eich, Jesús López-Herce, Antonio Rodríguez-Núñez, Thomas Rajka, Patrick Van de Voorde, David A. Zideman, Dominique Biarent.

9. References
Paediatric advanced life support

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

2. Summary of changes in paediatric advanced life support since the 2010 Guidelines

In managing the seriously ill child:

- If there are no signs of septic shock, children with a febrile illness should receive fluid with caution followed by reassessment. In some forms of septic shock, restricted fluid therapy with isotonic crystalloid may be more beneficial than the liberal use of fluids.

In the paediatric cardiac arrest algorithm:

- Many of the features are common with adult practice.
In post-resuscitation care:

- Prevent fever in children who have return of spontaneous circulation (ROSC) from an out-of-hospital cardiac arrest.
- Targeted temperature management of children post-ROSC should comprise treatment with either normothermia or mild hypothermia.

3. Introduction

The causes of cardiorespiratory arrest in children differ from those in adults in that most paediatric arrests arise from decompensated respiratory or circulatory failure (i.e. they are predominantly secondary cardiorespiratory arrests). Although in adulthood, primary arrests resulting from arrhythmias are more common, many young adults have similar causes to children (e.g. trauma, drowning and poisoning), meaning that respiratory failure is also common in this population.

Cardiorespiratory arrest generally has a poor outcome in children hence the identification of the seriously ill or injured child is an absolute priority. Directed interventions at the compensated or decompensated stages of illness/injury can be life-saving and prevent progression to cardiorespiratory arrest. Any unwell child or infant should be assessed in a systematic manner to identify the extent of any physiological disruption and interventions started to correct the situation.

The order of assessment and intervention for any seriously ill or injured child follows the ABCDE principles:

- **Airway** (Ac for airway and cervical spine stabilisation for the injured child).
- **Breathing**.
- **Circulation** (with haemorrhage control in the injured child).
- **Disability** (level of consciousness and neurological status).
- **Exposure** to ensure full examination (whilst respecting the child’s dignity and ensuring body temperature conservation).

Interventions are made at each step in the assessment as abnormalities are identified. The next step of the assessment is not started until the preceding abnormality has been treated and corrected if possible (the exception to this is the child presenting with life-threatening haemorrhage after serious injury when circulatory interventions will be made simultaneously with assessment and management of airway and breathing).

4. Summoning expert advice

Summoning a paediatric rapid response team (RRT) or medical emergency team (MET) may reduce the risk of respiratory and/or cardiac arrest in hospitalised children. This team should include at least one clinician with paediatric expertise and one specialist nurse. They should be called to evaluate a potentially critically ill child who is not already in a paediatric intensive care unit (PICU) or paediatric emergency department (ED).

5. The sequence of actions in cardiopulmonary resuscitation (CPR)

1. **Establish basic life support** (see paediatric BLS section)

2. **Oxygenate, ventilate, and start chest compression**:

   - Ensure a patent airway by using an airway manoeuvre described in the Paediatric basic life support section. [www.resus.org.uk/resuscitation-guidelines/paediatric-basic-life-support/](www.resus.org.uk/resuscitation-guidelines/paediatric-basic-life-support/)
   - Provide ventilation initially by bag-mask, using high-concentration inspired oxygen as soon as this is available.
   - Intubate the trachea only if this can be performed by an experienced operator with minimal interruption to chest compressions. Tracheal intubation will both control the airway and enable chest compression to be given.
continuously, thus improving coronary perfusion pressure.

- Use a compression rate of 100–120 min⁻¹.
- If the child has been intubated and compressions are uninterrupted, ensure that ventilation is adequate and use a slow ventilation rate of approximately 10–12 min⁻¹.

3. Attach a defibrillator or monitor:
   - Assess and monitor the cardiac rhythm.
   - If using a defibrillator, place one defibrillator pad or paddle on the chest wall just below the right clavicle, and one in the mid-axillary line. Pads for children should be 8–12 cm in size, and 4.5 cm for infants. In infants and small children it may be best to apply the pads to the front and back of the chest if they cannot be adequately separated in the standard positions.
   - The defibrillator pads may be used to assess the rhythm, when in monitoring mode.

4. Assess rhythm and check for signs of life:
   - Look for signs of life, which include responsiveness, coughing, spontaneous movements and normal breathing.
   - Assess the rhythm on the monitor:
     - Non-shockable (asystole or pulseless electrical activity (PEA)) OR
     - Shockable – ventricular fibrillation (VT) or pulseless ventricular tachycardia (pVT).

5A. Non-shockable (asystole or PEA):
This is the more common finding in children.

- Perform continuous CPR:
  - Continue to ventilate with high-concentration oxygen.
  - If ventilating with bag-mask give 15 chest compressions to 2 ventilations.
  - Use a compression rate of 100–120 min⁻¹.
  - If the patient is intubated, chest compressions can be continuous as long as this does not interfere with satisfactory ventilation.
  - Once the child's trachea has been intubated and compressions are uninterrupted use a ventilation rate of approximately 10–12 min⁻¹. Note: Once there is return of spontaneous circulation (ROSC), the ventilation rate should be 12–20 min⁻¹. Measure end-tidal carbon dioxide (CO₂) to monitor ventilation and ensure correct tracheal tube placement.
- Give adrenaline:
  - If vascular access has been established, give adrenaline 10 mcg kg⁻¹ (0.1 mL kg⁻¹ of 1 in 10,000 solution).
  - If there is no circulatory access, obtain intraosseous (IO) access.
- Continue CPR, only pausing briefly every 2 min to check for rhythm change.
  - Give adrenaline 10 mcg kg⁻¹ every 3–5 min (i.e. every other loop), while continuing to maintain effective chest compression and ventilation without interruption.
- Consider and correct reversible causes (4Hs and 4Ts):
  - Hypoxia
  - Hypovolaemia
  - Hyper/hypokalaemia, metabolic
  - Hypothermia
  - Thromboembolism (coronary or pulmonary)
  - Tension pneumothorax
  - Tamponade (cardiac)
  - Toxic/therapeutic disturbance
5B. Shockable (VF/pVT)

This is less common in children but may occur as a secondary event and is likely when there has been a witnessed and sudden collapse. It is seen more often in the intensive care unit and cardiac ward.

- **Continue CPR until a defibrillator is available – as 5A above**
  - **Defibrillate the heart:**
    - Charge the defibrillator while another rescuer continues chest compressions.
    - Once the defibrillator is charged, pause the chest compressions, quickly ensure that all rescuers are clear of the patient and then deliver the shock. This should be planned before stopping compressions.
    - Give 1 shock of 4 J kg\(^{-1}\) if using a manual defibrillator.
    - If using an AED for a child of less than 8 years, deliver a paediatric-attenuated adult shock energy.
    - If using an AED for a child over 8 years, use the adult shock energy.
  - **Resume CPR:**
    - Without reassessing the rhythm or feeling for a pulse, resume CPR immediately, starting with chest compression.
    - Consider and correct reversible causes (4Hs and 4Ts).

- **Continue CPR for 2 min, then pause briefly to check the monitor:**
  - If still VF/pVT, give a second shock (with same energy level and strategy for delivery as the first shock).

- **Resume CPR:**
  - Without reassessing the rhythm or feeling for a pulse, resume CPR immediately, starting with chest compression.

- **Continue CPR for 2 min, then pause briefly to check the monitor:**
  - If still VF/pVT, give a third shock (with same energy level and strategy for delivery as the previous shock).

- **Resume CPR:**
  - Without reassessing the rhythm or feeling for a pulse, resume CPR immediately, starting with chest compression.
  - Give adrenaline 10 mcg kg\(^{-1}\) and amiodarone 5 mg kg\(^{-1}\) after the third shock, once chest compressions have resumed.
  - Repeat adrenaline every alternate cycle (i.e. every 3–5 min) until ROSC.
  - Repeat amiodarone 5 mg kg\(^{-1}\) one further time, after the fifth shock if still in a shockable rhythm.

- **Continue giving shocks every 2 min, continuing compressions during charging of the defibrillator and minimising the breaks in chest compression as much as possible.**
  - **After each 2 min of uninterrupted CPR, pause briefly to assess the rhythm:** If still VF/pVT:
    - Continue CPR with the shockable (VF/pVT) sequence.
  - If asystole:
    - Continue CPR and switch to the non-shockable (asystole or PEA) sequence as above.
  - If organised electrical activity is seen, check for signs of life and a pulse:
    - If there is ROSC, continue post-resuscitation care.
    - If there is no pulse (or a pulse rate of <60 min\(^{-1}\)), and there are no other signs of life, continue CPR and continue as for the non-shockable sequence above.

If defibrillation was successful but VF/pVT recurs, resume the CPR sequence and defibrillate. Give an amiodarone bolus (unless two doses have already been given) and start a continuous infusion of the drug.

**Important note**

Uninterrupted, high quality CPR is vital. Chest compression and ventilation should be interrupted only for defibrillation. Chest compression is tiring for providers and the team leader should repeatedly assess and feedback on the quality of the compressions. To prevent fatigue, change providers should every two minutes. This will mean that the team can deliver effective high quality CPR so improving the chances of survival.\(^2,14\)
6. Explanatory notes

Tracheal tubes
Recent studies continue to show no greater risk of complications for children younger than 8 years when cuffed, rather than uncuffed, tracheal tubes are used in the operating room and intensive care unit. Cuffed tracheal tubes are as safe as uncuffed tubes for infants (except neonates) and children if rescuers use the correct tube size, cuff inflation pressure, and verify tube position. The use of cuffed tubes increases the chance of selecting the correct size at the first attempt. Under certain circumstances (e.g. poor lung compliance, high airway resistance, and facial burns) cuffed tracheal tubes may be preferable.¹⁵

Alternative airways
Although bag-mask ventilation remains the recommended first line method for achieving airway control and ventilation in children, the laryngeal mask airway (LMA) is an acceptable airway device for providers trained in its use. It is particularly helpful in airway obstruction caused by supraglottic airway abnormalities or if bag-mask ventilation is not
possible. Other supraglottic airways (SGA) (e.g. i-gel) which have been successful in children's anaesthesia may also be useful, but there are few data on the use of these devices in paediatric emergencies. Supraglottic airways do not totally protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required as their use is associated with a higher incidence of complications in small children compared with older children or adults.

**Capnography**

Monitoring end-tidal CO$_2$ with waveform capnography reliably confirms tracheal tube placement in a child weighing more than 2 kg with a perfusing rhythm and must be used after intubation and during transport of an intubated child. The presence of a capnographic waveform for more than four ventilated breaths indicates that the tube is in the tracheobronchial tree, both in the presence of a perfusing rhythm and during CPR. Capnography does not rule out intubation of a bronchus. The absence of exhaled CO$_2$ during CPR does not guarantee tube misplacement because a low or absent end-tidal CO$_2$ may reflect low or absent pulmonary blood flow.

Capnography may also provide information on the efficiency of chest compressions and a sudden rise in the end-tidal CO$_2$ can be an early indication of ROSC. Try to improve chest compression quality if the end-tidal CO$_2$ remains below 2 kPa as this may indicate low cardiac output and low pulmonary blood flow. Be careful when interpreting end-tidal CO$_2$ values after giving adrenaline or other vasoconstrictor drugs when there may be a transient decrease in end-tidal CO$_2$, or after the use of sodium bicarbonate when there may be a transient increase in the end-tidal values. Current evidence does not support the use of a threshold end-tidal CO$_2$ value as an indicator for stopping the resuscitation attempt.

**7. Drugs used in CPR**

**Adrenaline**

This is an endogenous catecholamine with potent alpha, beta$_1$, and beta$_2$ adrenergic actions. Adrenaline induces vasoconstriction, increases coronary and cerebral perfusion pressure and enhances myocardial contractility. Although firm evidence for its effectiveness is lacking, it is thought to stimulate spontaneous contractions, and increases the intensity of VF so increasing the likelihood of successful defibrillation.

The recommended IV/IO dose of adrenaline in children is 10 micrograms kg$^{-1}$. Subsequent doses of adrenaline are given every 3–5 min. Do not use higher doses of intravascular adrenaline in children because this may worsen outcome.$^{16}$

**Amiodarone**

Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is also slowed and a similar effect occurs in accessory pathways. Amiodarone has a mild negative inotropic action. The hypotension that occurs with IV amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80 and benzyl alcohol), which causes histamine release, than the drug itself.

In the treatment of shockable rhythms, give an initial IV bolus dose of amiodarone 5 mg kg$^{-1}$ after the third defibrillation. Repeat the dose after the fifth shock if still in VF/pVT. If defibrillation was successful but VF/pVT recurs, amiodarone can be repeated (unless two doses have already been given) and a continuous infusion started.

Amiodarone can cause thrombophlebitis when injected into a peripheral vein and, ideally, should be delivered via a central vein. If central venous access is unavailable (likely at the time of cardiac arrest) and so it has to be given peripherally, flush it liberally with 0.9% sodium chloride or 5% glucose.

One recent observational study in children showed that ECG resolution and survival to discharge was similar in a group treated with lidocaine instead of amiodarone but the evidence was not sufficiently robust to recommend a change in practice.$^{17}$

**Atropine**

Atropine is effective in increasing heart rate when bradycardia is caused by excessive vagal tone (e.g. after insertion of nasogastric tube). The dose is 20 mcg kg$^{-1}$. There is no evidence that atropine has any benefit in asphyxial bradycardia or asystole and its routine use has been removed from the ALS algorithms.
Magnesium
This is a major intracellular cation and serves as a cofactor in many enzymatic reactions. Magnesium treatment is indicated in children with documented hypomagnesaemia or with polymorphic VT (torsade de pointes), regardless of cause.

Calcium
Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. However, high plasma concentrations achieved after intravenous injection may be harmful to the ischaemic myocardium and may also impair cerebral recovery. The routine administration of calcium during cardiac arrest has been associated with increased mortality and it should be given only when specifically indicated (e.g. in hyperkalaemia, hypocalcaemia and in overdose of calcium-channel-blocking drugs).

Sodium bicarbonate
Cardiorespiratory arrest results in combined respiratory and metabolic acidosis, caused by cessation of pulmonary gas exchange and the development of anaerobic cellular metabolism respectively. The best treatment for acidaemia in cardiac arrest is a combination of effective chest compression and ventilation (high quality CPR). Administration of sodium bicarbonate generates carbon dioxide, which diffuses rapidly into the cells, exacerbating intracellular acidosis if it is not rapidly cleared via the lungs. It also has the following detrimental effects:

- It produces a negative inotropic effect on an ischaemic myocardium.
- It presents a large, osmotically active, sodium load to an already compromised circulation and brain.
- It produces a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues.

The routine use of sodium bicarbonate in CPR is not recommended. It may be considered in prolonged arrests, and it has a specific role in hyperkalaemia and the arrhythmias associated with tricyclic antidepressant overdose.

Fluids in CPR
Hypovolaemia is a potentially reversible cause of cardiac arrest. If hypovolaemia is suspected, give IV or IO fluids rapidly (20 mL kg\textsuperscript{-1} boluses). In the initial stages of resuscitation there are no clear advantages in using colloid solutions, whatever the aetiology, so use isotonic saline solutions for initial volume resuscitation. Do not use dextrose-based solutions for volume replacement – these will be redistributed rapidly away from the intravascular space and will cause hyponatraemia and hyperglycaemia, which may worsen neurological outcome.

8. Post-resuscitation care

Oxygen
In neonates there is evidence that hyperoxaemia can be detrimental and room air is recommended for use during initial resuscitation of the newborn (see Resuscitation and support of transition of babies at birth www.resus.org.uk/resuscitation-guidelines/resuscitation-and-support-of-transition-of-babies-at-birth). In the older child there is no evidence for any such advantages, so 100% oxygen should be used for initial resuscitation. After ROSC, titrate the inspired oxygen, using pulse oximetry, to achieve an oxygen saturation of 94–98%. In situations where dissolved oxygen plays an important role in oxygen transport such as smoke inhalation (carbon monoxide poisoning) and severe anaemia, maintain a high inspired oxygen (FiO\textsubscript{2}).

Carbon dioxide
In children who do not recover consciousness immediately following ROSC, controlled ventilation may help prevent further secondary brain injury (from cerebral oedema or ischaemia) as blood pH and PaCO\textsubscript{2} levels influence cerebral blood flow. The usual target range for PaCO\textsubscript{2} in this setting is 4.5–5.0 kPa, however, the target value for the PaCO\textsubscript{2} should be towards the anticipated value for the individual patient as the 'normal value' for a child with a chronic respiratory disorder may exceed the quoted ranges based on children without adaptive physiology.

Intravascular fluids and inotropes
Following ROSC, the use of fluids and/or inotropes to avoid hypotension is recommended in context of trying to
Rescue and post-ROSC use of extracorporeal membrane oxygenation (ECMO)

Extracorporeal circulatory techniques such as ECMO may be of benefit to patients with cardiac origins to their cardiorespiratory arrest in a setting where it can be instituted rapidly. The benefit to patients who have other causes for their arrest is unclear.2

Prognostic factors for outcomes of resuscitation

No single prognostic factor is sufficiently reliable to inform decisions about the termination of a resuscitation attempt or the likely outcome. Factors that should influence any decisions include the circumstances of the arrest, initial rhythm, duration of resuscitation and other features such as presence of hypothermia and severe metabolic derangement. Comatose children with ROSC receiving mechanical ventilation who fulfill neurological criteria for death, or in whom withdrawal of life-sustaining treatments is planned should be considered as potential organ donors.

Therapeutic hypothermia

Initial trials of therapeutic hypothermia (TH) in adults following cardiac arrest showed improvement in survival and neurological outcome.23,24 However, subsequent studies failed to replicate these findings. A recent study comparing a target temperature of 33°C with 36°C in comatose adults resuscitated from out-of-hospital cardiac arrest (OHCA) showed no difference in neurological outcome.25

The Therapeutic Hypothermia After Pediatric Cardiac Arrest (THAPCA) study was a large randomised controlled trial that compared mild TH (32–34°C) with therapeutic normothermia (36.8°C) (both groups received active temperature control) for comatose children who survived OHCA.26 The trial did not show any significant difference in survival or 1-year functional outcome between the two groups (it was powered to show an absolute risk difference of 15-20%). There was a tendency toward better outcomes at the lower temperature ranges. There was no difference in the incidence of infection, bleeding, or serious arrhythmias between the two groups hence TH appears to be safe.

A child who has ROSC, but remains comatose after cardiorespiratory arrest, may benefit either from being cooled to a core temperature of 32–34°C (TH) or having their core temperature actively maintained at 36.8°C for at least 24 h post arrest. Do not actively rewarm a successfully resuscitated child with hypothermia unless the core temperature is below 32°C. Following a period of mild hypothermia, re warm the child slowly at 0.25–0.5°C h⁻¹.

Blood glucose control

Neonatal, child and adult data show that both hyperglycaemia and hypoglycaemia are associated with poor outcome after cardiorespiratory arrest but it is uncertain if this is causative or merely an association. Closely monitor plasma glucose concentrations in any ill or injured child including after cardiorespiratory arrest. Do not give glucose-containing fluids during CPR except for treatment of hypoglycaemia.

Hyperglycaemia and hypoglycaemia should be avoided following ROSC but tight glucose control has not shown survival benefits when compared with moderate glucose control in adults and increased the risk of hypoglycaemia in neonates, children and adults.

9. Parental presence

Many parents would want to be present during a resuscitation attempt; they can see that everything possible is being done for their child. Reports show that being at the side of the child is comforting to the parents or carers and helps them to gain a realistic view of attempted resuscitation and death. Bereaved families who have been present in the resuscitation room show less anxiety and depression several months after the death.

Parental presence in the resuscitation room may also encourage healthcare providers' professional behaviour and facilitate their understanding of the child in the context of his/her family.

A dedicated staff member should be present with the parents at all times to explain the process in an empathetic and sympathetic manner. They can also ensure that the parents do not interfere with the resuscitation process or distract the resuscitation team. If the presence of the parents is impeding the progress of the resuscitation, they should be gently asked to leave. When appropriate, physical contact with the child should be allowed.
The resuscitation team leader should decide when to stop the resuscitation; this should be expressed with sensitivity and understanding. After the event, debriefing of the team should be conducted, to express any concerns and to allow the team to reflect on their clinical practice in a supportive environment.

10. Acknowledgements

These guidelines have been adapted from the European Resuscitation Council 2015 Guidelines. We acknowledge and thank the authors of the ERC Guidelines for Paediatric life support: Ian K. Maconochie, Robert Bingham, Christoph Eich, Jesús López-Herce, Antonio Rodríguez-Núñez, Thomas Rajka, Patrick Van de Voorde, David A. Zideman, Dominique Biarent.

11. References


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.
- The involvement of stakeholders from around the world including members of the public and cardiac arrest survivors.
- These Resuscitation Council (UK) Guidelines have been peer reviewed by the Executive Committee of the Resuscitation Council (UK), which comprises 25 individuals and includes lay representation and representation of the key stakeholder groups.

2. Introduction

Until such time that the newly born infant has successfully made the transition to air breathing it is dependent on the placenta and umbilical cord for respiration. The normal function of both of these may be interrupted by a number of pathological events before or during the birth process (e.g. by placental abruption, cord entanglement, etc.) giving rise to a hypoxic insult that may be acute, chronic or both (acute on chronic).

Passage through the birth canal itself is a relatively hypoxic experience for the fetus, since significant respiratory exchange at the placenta is interrupted for the 50–75 s duration of the average contraction. Although most infants tolerate this well, the few that do not may require help to establish normal breathing at delivery. The majority of these infants merely require supported perinatal transition rather than 'resuscitation'.

Resuscitation or support of transition is more likely to be needed by babies with intrapartum evidence of significant fetal compromise, babies delivering before 35 weeks gestation, babies delivering vaginally by the breech, maternal infection and multiple pregnancies. Furthermore, caesarean delivery is associated with an increased risk of problems with respiratory transition at birth requiring medical interventions especially for deliveries before 39
weeks gestation. However, elective caesarean delivery at term does not increase the risk of needing newborn resuscitation in the absence of other risk factors.\textsuperscript{7-10}

Some infants need support at birth for reasons other than hypoxia (e.g. infection), however the initial approach to these infants follows the same algorithm.

Newborn life support (NLS) is intended to provide this help and comprises the following elements:

- Enabling placental transfusion (when able to do so) by delaying the clamping of the umbilical cord.
- Drying and covering the newborn infant, and where necessary taking additional steps, to maintain a normal body temperature (i.e. between 36.5°C and 37.5°C).
- Assessing the infant’s condition and the need for any intervention.
- Maintaining an open airway.
- If the infant is not breathing, aerating the lungs with inflation breaths.
- Continue ventilating apnoeic infants until respiration is established.
- If the heart remains less than 60 min\textsuperscript{-1} after 5 effective inflation breaths and 30 seconds of effective ventilation, start chest compressions.
- Administration of drugs (rarely).

3. Physiology of acute perinatal hypoxia

If subjected to sufficient hypoxia in utero, or during passage through the birth canal, the fetus will attempt to breathe. If the hypoxic insult is continued the fetus will eventually lose consciousness. Shortly after this the neural centres in the brainstem which control these breathing efforts will cease to function because of lack of oxygen. The fetus then enters a period of absent respiratory effort known as primary apnoea.

Up to this point, the heart rate remains unchanged, but soon decreases to about half the normal rate as the myocardium reverts to anaerobic metabolism – a less fuel-efficient process. The circulation to non-vital organs is reduced in an attempt to preserve perfusion of vital organs. The release of lactic acid, a by-product of anaerobic metabolism, causes deterioration of the biochemical milieu, adding to the respiratory acidosis from the accumulation of carbon dioxide.

If the hypoxic insult continues, shuddering agonal gasps (whole-body respiratory gasps at a rate of about 12 min\textsuperscript{-1}) are initiated by primitive spinal reflexes. If the fetus remains in utero, or if, for some other reason, these gasps fail to aerate the lungs, they fade away and the fetus enters a period known as secondary, or terminal, apnoea with no further spontaneous breathing. Until now, the circulation has been maintained but, as terminal apnoea progresses, the rapidly worsening acidosis, dwindling substrate for anaerobic metabolism and on-going anoxia begins to impair cardiac function. The heart eventually fails and, without effective intervention, the fetus dies. The whole process probably takes up to 20 min in the human fetus at term.

Thus, in the face of anoxia, the infant can maintain an effective circulation throughout the period of primary apnoea, through the gasping phase, and even for a while after the onset of terminal apnoea. Therefore the most urgent requirement for any severely hypoxic infant at birth is that the lungs be aerated effectively. Provided the infant’s circulation is functioning sufficiently well, oxygenated blood will then be conveyed from the aerated lungs to the heart. The heart rate will increase and the brain will be perfused with oxygenated blood. Following this, the neural centres responsible for normal breathing will usually function once again and the infant will recover.

Merely aerating the lungs is sufficient in the vast majority of cases.\textsuperscript{11} Lung aeration and subsequent ventilation remains central to all efforts to resuscitate a newborn infant, but in a few cases cardiac function will have deteriorated to such an extent that the circulation is inadequate and cannot convey oxygenated blood from the aerated lungs to the heart. In this case, a brief period of chest compression may be needed as well as aeration of the lungs. In an even smaller number of cases, lung aeration and chest compression will not be sufficient, and drugs may be required to restore the circulation. The outlook in this group of infants remains poor.
4. Important guideline changes

The following are the changes that have been made to the NLS guidelines in 2015, some build on or expand the changes that were introduced in 2010.

- For uncompromised term and preterm infants, a delay in cord clamping of at least one minute from the complete delivery of the infant, is now recommended. As yet there is insufficient evidence to recommend an appropriate time for clamping the cord in infants who are severely compromised at birth. For infants requiring resuscitation, resuscitative intervention remains the immediate priority. Stripping (or ‘milking’) of the cord is not recommended as a routine measure except in the context of further randomised trials.

- The temperature of newly born infants is actively maintained between 36.5°C and 37.5°C after birth unless a decision has been taken to start therapeutic hypothermia. The importance of achieving this has been highlighted and reinforced because of the strong association with mortality and morbidity. Even the mild hypothermia that was once felt to be inevitable and therefore clinically acceptable carries a risk. The admission temperature should be recorded as a predictor of outcomes as well as a quality indicator.

- Preterm infants of less than 32 weeks gestation may benefit from a combination of interventions to maintain their body temperature between 36.5°C and 37.5°C after delivery through stabilisation and neonatal unit admission. These may include:
  - Warmed humidified respiratory gases
  - Thermal mattress alone
  - A combination of increased room temperature with plastic wrapping of head and body with thermal mattress

All of these combinations have been effective in reducing hypothermia. In addition, the delivery room temperature should be at least 26°C for the most immature infants.

- An ECG, if available, can give a rapid accurate and continuous heart rate reading during newborn resuscitation. It does not, however, indicate the presence of a cardiac output and should not be the sole means of monitoring the infant.

- Resuscitation of term infants should commence in air. For preterm infants, a low concentration of oxygen (21–30%) should be used initially for resuscitation at birth. If, despite effective ventilation, oxygenation (ideally guided by oximetry) remains unacceptable, use of a higher concentration of oxygen should be considered. Blended oxygen and air should be given judiciously and its use guided by pulse oximetry. If a blend of oxygen and air is not available use what is available. If chest compressions are administered, supplemental oxygen should be increased.

- Attempts to aspirate meconium from the nose and mouth of the unborn infant, while the head is still on the perineum, are not recommended. The emphasis should be on initiating lung inflation within the first minute of life in non-breathing or ineffectively breathing infants and this should not be delayed. If presented with a floppy, apnoeic infant born through thick particulate meconium it is reasonable to inspect the oropharynx rapidly to remove potential obstructions. Tracheal intubation should not be routine in the presence of meconium and should only be performed for suspected tracheal obstruction.

- Nasal continuous positive airways pressure (CPAP) rather than routine intubation may be used to provide initial respiratory support of all spontaneously breathing preterm infants with respiratory distress. Early use of nasal CPAP should also be considered in those spontaneously breathing preterm infants who are at risk of developing respiratory distress syndrome (RDS).

- The recommended compression: ventilation ratio for CPR remains at 3:1 for newborn resuscitation. Asynchronous compressions are not recommended.

5. Suggested sequence of actions

Keep the infant warm and assess

Infants are born small and wet. They get cold very easily, especially if they remain wet and in a draught. For uncompromised infants, a delay in cord clamping of at least one minute from the complete delivery of the infant, is recommended. Allowing placental transfusion ensures a more gradual transition to extra-uterine life preventing
sudden changes in venous return to the heart and the potential impact of these on blood pressure.

Whatever the situation it is important that the infant does not get cold. In all cases whether intervention is required or not, dry the term or near-term infant, remove the wet towels, and cover the infant with dry towels. Significantly preterm infants are best placed, without drying, into polyethylene wrapping under a radiant heater. In infants of all gestations, the head should be covered with an appropriately sized hat. The temperature must be actively maintained between 36.5°C and 37.5°C after birth unless a decision has been taken to start therapeutic hypothermia. The admission temperature should always be recorded as a predictor of outcomes as well as a quality indicator.

This process will provide significant stimulation and will allow time to assess the infant's breathing, and heart rate. A note should be made of the colour and tone, although these are of lesser importance in determining the immediate approach to be taken they can point towards the severely acidaemic baby (potentially requiring substantial resuscitation) or anaemic baby (potentially requiring urgent transfusion).

Reassess breathing and heart rate regularly every 30 s or so throughout the resuscitation process but it is the heart rate, which is the key observation. The first sign of any improvement in the infant will be an increase in heart rate. Consider the need for help; if needed, ask for help immediately.

A healthy infant will be born blue but will have good tone, will cry within a few seconds of delivery and will have a good heart rate within a few minutes of birth (the heart rate of a healthy newborn infant is about 120–150 min⁻¹). A less healthy infant will be blue at birth, will have less good tone, may have a slow heart rate (less than 100 min⁻¹), and may not establish adequate breathing by 90–120 s. An unwell infant will be born pale and floppy, not breathing and with a slow, very slow or undetectable heart rate.

In the first few minutes, the heart rate of an infant is usually judged best by listening with a stethoscope. It may also be felt by gently palpating the umbilical cord but a slow or absent rate at the base of the umbilical cord is not always indicative of a truly slow heart rate. Feeling for any of the peripheral pulses is not helpful.

An ECG is the most accurate way to obtain a rapid and continuous heart rate reading but may not be immediately available, nor will it give an indication of a cardiac output. A pulse oximeter can give a continuous heart rate and oximetry reading in the delivery room. With practice it is possible to attach a pulse oximeter probe and to obtain a useful reading of heart rate and oxygen saturation about 90 s after delivery.
Airway

Before the infant can breathe effectively the airway must be open. The best way to achieve this is to place the infant on his back with the head in the neutral position (i.e. with the neck neither flexed nor extended). Most newborn infants will have a relatively prominent occiput, which will tend to flex the neck if the infant is placed on his back on a flat surface. This can be avoided by placing some support under the shoulders of the infant, but take care not to overextend the neck. If the infant is very floppy (i.e. has no or very little tone) it is usually necessary to support the jaw with a jaw thrust. These manoeuvres will be effective for the majority of infants requiring airway stabilisation at birth.

Airway suction immediately following birth should be reserved for infants who have obvious airway obstruction that cannot be rectified by appropriate positioning and in whom material is seen in the airway. Rarely, material (e.g. mucus, blood, meconium, vernix) may be blocking the oropharynx or trachea. In these situations, direct visualisation and suction of the oropharynx should be performed. For tracheal obstruction, intubation and suction during withdrawal of the endotracheal tube may be effective. This latter manoeuvre should only be performed by appropriately trained staff and, if performed, should not unduly delay the onset of inflation breaths and subsequent ventilation.
Breathing

Most infants have a good heart rate after birth and establish breathing by about 90 s. If the infant is not breathing adequately aerate the lungs by giving 5 inflation breaths, preferably using air. Until now the infant's lungs will have been filled with fluid. Aeration of the lungs in these circumstances is likely to require sustained application of pressures of about 30 cm H₂O for 2–3 s; these are 'inflation breaths'. Begin with lower pressures (20–25 cm H₂O) in preterm infants.

Use positive end-expiratory pressure (PEEP) of 4–5 cm H₂O if possible. It is of demonstrable benefit in preterm infants but should also be used in term infants, although evidence for its benefit in this group of infants is lacking from human studies. If the heart rate was below 100 min⁻¹ initially then it should rapidly increase as oxygenated blood reaches the heart.

- If the heart rate does increase then you can assume that you have successfully aerated the lungs.
- If the heart rate increases but the infant does not start breathing for himself, then continue ventilations at a rate of about 30–40 min⁻¹ until the infant starts to breathe on his own.
- If the heart rate does not increase following inflation breaths, then either you have not aerated the lungs or the infant needs more than lung aeration alone. By far the most likely is that you have failed to aerate the lungs effectively. If the heart rate does not increase, and the chest does not passively move with each inflation breath, then you have not aerated the lungs.

If the lungs have not been aerated then consider:

- Checking again that the infant's head is in the neutral position?
- Is there a problem with face mask leak?
- Do you need jaw thrust or a two-person approach to mask inflation?
- Do you need a longer inflation time? – were the inspiratory phases of your inflation breaths really of 2–3 s duration?
- Is there an obstruction in the oropharynx (laryngoscope and suction)?
- Will an oropharyngeal (Guedel) airway assist?
- Is there a tracheal obstruction?

Check that the infant’s head and neck are in the neutral position; that your inflation breaths are at the correct pressure and applied for sufficient time (2–3 s inspiration); and that the chest moves with each breath. If the chest still does not move, ask for help in maintaining the airway and consider an obstruction in the oropharynx or trachea, which may be removable by suction under direct vision. An oropharyngeal airway may be helpful.

If the heart remains slow (less than 60 min⁻¹) or absent after 5 effective inflation breaths and 30 seconds of effective ventilation, start chest compressions.

If you are dealing with a preterm infant then initial CPAP of approximately 5 cm H₂O, either via a face mask or via a CPAP machine, is an acceptable form of support in infants who are breathing but who show signs of, or are at risk of developing, respiratory distress. In preterm infants who do not breathe or breathe inadequately, you should use PEEP with your inflation breaths and ventilations as the lungs in these infants are more likely to collapse again at the end of a breath; using PEEP prevents this.

Chest compression

Almost all infants needing help at birth will respond to successful lung inflation with an increase in heart rate within 30 seconds followed quickly by normal breathing. However, in some cases chest compression is necessary. Chest compression should be started only when you are sure that the lungs have been aerated successfully.

In infants, the most efficient method of delivering chest compression is to grip the chest in both hands in such a way that the two thumbs can press on the lower third of the sternum, just below an imaginary line joining the nipples, with the fingers over the spine at the back. Compress the chest quickly and firmly, reducing the antero-posterior diameter of the chest by about one third.

The ratio of compressions to inflations in newborn resuscitation is 3:1.
Chest compressions move oxygenated blood from the lungs back to the heart. Allow enough time during the relaxation phase of each compression cycle for the heart to refill with blood. Ensure that the chest is inflating with each breath. You should increase the oxygen concentration if you have reached this stage of resuscitation. You should also have called for help and a pulse oximeter, if not already in use, will be helpful in monitoring how you are doing.

Do not use asynchronous compressions, even if the infant has a tracheal tube placed, as maintaining air entry into the lung remains as important now as it was during the initial aeration. Compressing the chest during a ventilation breath may reduce air entry, which may be harmful.

In a very few infants (less than one in every thousand births) inflation of the lungs and effective chest compression will not be sufficient to produce an effective circulation. In these circumstances drugs may be helpful.

**Drugs**

Drugs are needed rarely and only if there is no significant cardiac output despite effective lung inflation and chest compression. The outlook for most infants at this stage is poor although a small number have had good outcomes after a return of spontaneous circulation followed by therapeutic hypothermia.

The drugs used include adrenaline (1:10,000), occasionally sodium bicarbonate (ideally 4.2%), and glucose (10%). All resuscitation drugs are best delivered via an umbilical venous catheter or if this is not possible through an intraosseous needle.

The recommended intravenous dose for adrenaline is 10 microgram kg$^{-1}$ (0.1 mL kg$^{-1}$ of 1:10,000 solution). If this is not effective, a dose of up to 30 microgram kg$^{-1}$ (0.3 mL kg$^{-1}$ of 1:10,000 solution) may be tried.

Adrenaline is the only drug that may be given by the tracheal route, although of unknown efficacy at birth. If this is used, it must not interfere with ventilation or delay acquisition of intravenous access. The tracheal dose is thought to be between 50–100 microgram kg$^{-1}$.

Use of sodium bicarbonate is not recommended during brief resuscitation. If it is used during prolonged arrests unresponsive to other therapy, it should be given only after adequate ventilation and circulation (with chest compressions) is established. The dose for sodium bicarbonate is between 1 and 2 mmol of bicarbonate kg$^{-1}$ (2–4 mL of 4.2% bicarbonate solution).

The dose for glucose (10%) is 2.5 mL kg$^{-1}$ (250 mg kg$^{-1}$) and should be considered if there has been no response to other drugs delivered through a central venous catheter.

Very rarely, the heart rate cannot increase because the infant has lost significant blood volume. If this is the case, there is often a clear history of blood loss from the infant, but not always. Use of isotonic crystalloid rather than albumin is preferred for emergency volume replacement. In the presence of hypovolaemia, a bolus of 10 mL kg$^{-1}$ of 0.9% sodium chloride or similar given over 10–20 s will often produce a rapid response and can be repeated safely if needed.

**When to stop resuscitation**

In a newly-born infant with no detectable cardiac activity, and with cardiac activity that remains undetectable for 10 min, it is appropriate to consider stopping resuscitation. The decision to continue resuscitation efforts beyond 10 min with no cardiac activity is often complex and may be influenced by issues such as the availability of therapeutic hypothermia and intensive care facilities, the presumed aetiology of the arrest, the gestation of the infant, the presence or absence of complications, and the parents’ previous expressed feelings about acceptable risk of morbidity. The difficulty of this decision-making emphasises the need for senior help to be sought as soon as possible.

Where a heart rate has persisted at less than 60 min$^{-1}$ without improvement, during 10–15 min of continuous resuscitation, the decision to stop is much less clear. No evidence is available to recommend a universal approach beyond evaluation of the situation on a case-by-case basis by the resuscitating team and senior clinicians.

**Communication with the parents**

It is important that the team caring for the newborn baby informs the parents of the baby’s progress. At delivery, adhere to the routine local plan and, if possible, hand the baby to the mother at the earliest opportunity. If resuscitation is required inform the parents of the procedures undertaken and why they were required. Record all discussions and decisions in the baby’s records as soon as possible after birth.
6. Post-resuscitation care

Therapeutic hypothermia

Term or near-term infants, with evolving moderate to severe hypoxic-ischaemic encephalopathy, should be treated with therapeutic hypothermia. Whole body cooling and selective head cooling are both appropriate strategies. Cooling should be initiated and conducted under clearly-defined protocols with treatment in neonatal intensive care facilities and the capabilities for multidisciplinary care. Treatment should be consistent with the protocols used in the randomised clinical trials (i.e. commence cooling within 6 h of birth, continue the cooling for 72 h before re-warming the infant over a period of at least 4 h). All treated infants should be followed longitudinally. Passive or active therapeutic hypothermia should only instituted following a senior clinical decision.

Glucose

Infants who are preterm or require significant resuscitation should be monitored and treated to maintain blood glucose in the normal range. Hypoglycaemia should be avoided in babies demonstrating signs of evolving hypoxic ischaemic encephalopathy or who are undergoing therapeutic hypothermia. An infusion of 10% glucose rather than repeated boluses is usually best at treating low blood glucose values and maintaining glucose in the normal range.

7. Explanatory Notes

Resuscitation or stabilisation

Most infants born at term need no resuscitation and they can usually stabilise themselves during the transition from placental to pulmonary respiration very effectively. Provided attention is paid to preventing heat loss (and avoiding over-warming) and a little patience is exhibited before cutting the umbilical cord, intervention is rarely necessary. However, some infants will have suffered stresses or insults during labour. Help may then be required which is characterised by interventions designed to rescue a sick or very sick infant and this process can then reasonably be called resuscitation.

Significantly preterm infants, particularly those born below 30 weeks gestation, are treated differently. Most infants in this group are healthy at the time of delivery and yet all can be expected to benefit from help in making the transition. Maintaining the temperature between 36.5°C and 37.5°C is even more important than for term babies. Intervention in this situation is usually limited to keeping an infant healthy during this transition and is more appropriately called stabilisation. Gentle airway support using CPAP rather than ventilation may be adequate for many of these infants. In the past both situations have been referred to as resuscitation and this seems inappropriate and likely to cause confusion.

Umbilical cord clamping

For healthy term infants delaying cord clamping for at least one minute or until the cord stops pulsating following delivery improves iron status through early infancy. For preterm infants in good condition at delivery, delaying cord clamping for up to 3 min results in increased blood pressure during stabilisation, a lower incidence of intraventricular haemorrhage and fewer blood transfusions. However, infants were more likely to receive phototherapy. There are limited data on the hazards or benefits of delayed cord clamping in the non-vigorous infant.

Delaying cord clamping for at least one minute is recommended for all newborn infants not requiring resuscitation. At present there is insufficient evidence to define an appropriate time to clamp the cord in infants who apparently need resuscitation. However, this may be because time is the wrong defining parameter and perhaps the cord should not be clamped until the infant has started breathing (or the lungs are aerated). Stripping (or ‘milking’) of the umbilical cord has been suggested as an alternative to delayed cord clamping when the infant is in need of resuscitation; however there is insufficient evidence to recommend this as a routine measure. Umbilical cord milking did, however, produce improved short term haematological outcomes, admission temperature and urine output when compared to delayed cord clamping in one recent study.
Maintaining normal temperature (between 36.5°C and 37.5°C)

Naked, wet, newborn infants cannot maintain their body temperature in a room that feels comfortably warm for adults. Infants who are compromised are particularly vulnerable to the effects of cold stress, which may will lower arterial oxygen tension and increase metabolic acidosis. Active measures will need to be taken to avoid hypothermia, especially in the preterm infant where a team approach and a combination of strategies may be required. The neonatal unit admission temperature of newborn infants is a strong predictor of mortality at all gestations and in all settings. For each 1°C decrease in admission temperature below this range there is an associated increase in mortality by 28%.

Babies born outside the normal delivery environment may benefit from placement in a food grade polyethylene bag or wrap after drying and then swaddling. Alternatively, well newborns >30 weeks gestation who are breathing may be dried and nursed with skin to skin contact or kangaroo mother care to maintain their temperature whilst they are transferred. They should be protected from draughts.

**Recommendation**

Unless you have decided to implement therapeutic hypothermia, take active steps to maintain the temperature of the newly born infant between 36.5°C and 37.5°C from birth to admission and throughout stabilisation. If the resuscitation is prolonged, consider measuring temperature during the resuscitation.

**Oximetry and the use of supplemental oxygen**

If resources are available, use pulse oximetry for all deliveries where it is anticipated that the infant may have problems with transition or need resuscitation. Oxygen saturation and heart rate can be measured reliably during the first minutes of life with a modern pulse oximeter. Data from healthy spontaneously breathing infants has been used to inform when oxygen should be given (see algorithm).

The sensor must be placed on the right hand or wrist to obtain an accurate reading of the preductal saturation. Placement of the sensor on the infant before connecting to the instrument may result in faster acquisition of signal. In most cases a reliable reading can be obtained within 90 s of birth. Pulse oximetry can also provide an accurate display of heart rate during periods of good perfusion.

In healthy term infants, oxygen saturation increases gradually from approximately 60% soon after birth to over 90% at 10 min. In preterm infants hyperoxaemia is particularly damaging and if oxygen is being used and the saturation is above 95% the risk of hyperoxaemia is high. Therefore the rate of rise in oxygen saturation after birth in preterm infants should not exceed that seen in term infants, although some supplemental oxygen may be required to achieve this.

**Colour**

Using colour as a proxy for oxygen saturation is usually inaccurate. However, noting whether an infant is initially very pale and, therefore, either acidic or anaemic at delivery may be useful as an indicator for later therapeutic intervention.

**ECG monitoring of heart rate**

Clinical assessment of heart rate, whether by palpation of the cord or apex of the heart or by listening with a stethoscope tends to be inaccurate. There is increasing evidence supporting the use of ECG monitoring as a means of rapidly determining the heart rate during resuscitation; it is quicker to provide an accurate reading than pulse oximetry but does require that the ECG leads make good contact with the skin.

**Airway suctioning with or without meconium**

Routine elective intubation and suctioning of vigorous infants at birth, does not reduce meconium aspiration syndrome (MAS). Nor does suctioning the nose and mouth of such infants on the perineum and before delivery of the shoulders (intrapartum suctioning). Even in non-vigorous infants born through meconium-stained amniotic fluid who are at increased risk of MAS, intubation and tracheal suctioning has not been shown to improve the outcome. There is no evidence to support suctioning of the mouth and nose of infants born through clear amniotic fluid.

**Recommendation**

Routine *intrapartum* oropharyngeal and nasopharyngeal suctioning for infants born with clear and/or meconium-stained amniotic fluid is not recommended.
The practice of routinely performing direct oropharyngeal and tracheal suctioning of non-vigorous infants after birth with meconium-stained amniotic fluid was based upon poor evidence. The presence of thick, viscous meconium in a non-vigorous infant is the only indication for initially considering visualising the oropharynx and suctioning material, which might obstruct the airway. If an infant born through meconium-stained amniotic fluid is also floppy and makes no immediate respiratory effort, then it is reasonable to rapidly inspect the oropharynx with a view to removing any particulate matter that might obstruct the airway. Tracheal intubation should not be routine in the presence of meconium and is performed only for suspected tracheal obstruction. The emphasis is on initiating ventilation within the first minute of life in non-breathing or ineffectively breathing infants and this should not be delayed, especially in the bradycardiac infant.

Laryngeal mask

Several studies have shown that laryngeal mask airways (LMAs) can be used effectively at birth to ventilate the lungs of infants weighing over 2000 g, greater than 33 weeks gestation and apparently needing resuscitation. Case reports suggest that LMAs have been used successfully when intubation has been tried and failed – and occasionally vice-versa. One small randomised study has suggested that LMAs may reduce the need for intubation compared to facemask ventilation, however LMAs cost about three times as much and it is not clear how many of the infants resuscitated using an LMA would have responded to good quality facemask ventilation. Data on LMA use in smaller or less mature infants are scarce.

Recommendation

Consider using an LMA during resuscitation of the newborn infant if face mask ventilation is unsuccessful and tracheal intubation is unsuccessful or not feasible. The LMA may be considered as an alternative to a face mask for positive pressure ventilation among newborn infants weighing more than 2000 g or delivered ≥34 weeks gestation. The LMA may be considered as an alternative to tracheal intubation as a secondary airway for resuscitation among newborn infants weighing more than 2000 g or delivered ≥34 weeks gestation. There is limited evidence evaluating its use for newborn infants weighing <2000 g or delivered <34 weeks gestation and none for those infants receiving compressions.

Use of the LMA, nonetheless, should be limited to those individuals who have been trained to use it. Its use has not been evaluated in the setting of meconium stained fluid, during chest compressions, or for the administration of emergency intra-tracheal medications.

Exhaled carbon dioxide

Detection of exhaled carbon dioxide confirms tracheal intubation in neonates with a cardiac output more rapidly and more accurately than clinical assessment alone. It will not, however, distinguish between correct placement with the tip of the tracheal tube in the trachea and incorrect insertion with the tip in the right main bronchus (i.e. too long). False negative readings may occur in very low birth weight neonates and in infants during cardiac arrest (in these cases a brief period of chest compressions may bring about a colour change as more carbon dioxide is delivered to the lungs). False positives may occur with colorimetric devices contaminated with adrenaline, surfactant and atropine.

Drugs in resuscitation at birth

Ventilation and chest compression may fail to resuscitate fewer than 1 in 1000 infants. In this group, resuscitation drugs may be justified. Whilst there is evidence from animal studies for both adrenaline and sodium bicarbonate in bringing about return of spontaneous circulation, there is no placebo-controlled evidence in human infants for the effectiveness of any drug intervention in this situation. Even for adults and children in cardiac arrest, there is insufficient evidence to suggest that vasopressors improve long-term survival.

For this reason use of drugs before achieving lung aeration followed by chest compressions (known to be effective resuscitative interventions) can never be justified.

Glucose

Hypoglycaemia is associated with adverse neurological outcome in a neonatal animal model of hypoxia and resuscitation. Newborn animals that were hypoglycaemic at the time of a hypoxic-ischemic insult had larger areas of cerebral infarction and/or decreased survival compared to controls. However, only a single clinical study has shown an association between hypoglycaemia and poor neurological outcome following perinatal hypoxia. In adults, children and extremely low-birth-weight infants receiving intensive care, hyperglycaemia is associated with a worse outcome. However, in children, hyperglycaemia after hypoxia-ischaemia does not appear to be harmful.
which confirms data from animal studies\textsuperscript{86} some of which suggest it may even be protective.\textsuperscript{87} The situation remains unclear and unfortunately, the range of blood glucose concentration that is associated with the least brain injury following asphyxia and resuscitation cannot be defined based on available evidence.

8. Acknowledgements

These guidelines have been adapted from the European Resuscitation Council 2015 Guidelines. We acknowledge and thank the authors of the ERC Guidelines for Resuscitation and support of transition of babies at birth: Jonathan Wyllie, Jos Bruinenberg, Charles Christoph Roehr, Mario Rüdiger, Daniele Trevisanuto, Berndt Urlesberger.

9. References


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.
- These Resuscitation Council (UK) Guidelines have been peer reviewed by the Executive Committee of the 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

The 2015 Guideline does not include any major changes in core in-hospital resuscitation interventions since the previous guidelines published in 2010. The key changes since 2010 are:

- Continuing emphasis on the use of rapid response systems for care of the deteriorating patient and prevention of in-hospital cardiac arrest.
- Continued emphasis on providing minimally interrupted high quality chest compressions throughout CPR: chest compressions are paused briefly only to enable specific interventions. This includes minimising interruptions in chest compressions to attempt defibrillation.

3. Introduction

These guidelines are aimed primarily at healthcare professionals who are first to respond to an in-hospital cardiac arrest and may also be applicable to healthcare professionals working in other clinical settings. The Resuscitation Council (UK) has produced Quality standards for cardiopulmonary resuscitation and training to support the implementation of this guideline. [www.resus.org.uk/quality-standards/](http://www.resus.org.uk/quality-standards/)

between basic life support (BLS) and advanced life support (ALS) is arbitrary; in practice, the resuscitation process is a continuum. For all in-hospital cardiac arrests, ensure that:

- cardiorespiratory arrest is recognised immediately
- help is summoned using a standard telephone number (e.g. 2222)<sup>3</sup>
- CPR is started immediately and, if indicated, defibrillation is attempted as soon as possible (within 3 min).

All in-hospital cardiac arrests should be reviewed as part of an audit and quality improvement process. Details should be recorded after each event. The National Cardiac Arrest Audit (NCAA) enables hospitals to collect standardised data, and monitor changes in cardiac arrest activity.

4. Sequence for a collapsed patient in hospital

An algorithm for the initial management of in-hospital cardiac arrest is shown in Figure 1.

![In-hospital cardiac arrest algorithm](http://resus.org.uk/_resources/assets/attachment/full/0/6469.pdf)
1. Ensure personal safety

- There are very few reports of harm to rescuers during resuscitation.\(^4\)
- Your personal safety and that of resuscitation team members is the first priority during any resuscitation attempt.
- Check that the patient's surroundings are safe.
- Put on gloves as soon as possible. Other personal protective equipment (PPE) (eye protection, face masks, aprons, gowns) may be necessary especially when the patient has a serious infection such as tuberculosis. Follow local infection control measures to minimise risks.

2. Check the patient for a response

- If you see a patient collapse or find a patient apparently unconscious assess if he is responsive (shake and shout). Gently shake his shoulders and ask loudly: “Are you all right?”
- If other members of staff are nearby it will be possible to undertake several actions simultaneously.

3A. If the patient responds

- Urgent medical assessment is required. Call for help according to local protocols. This may include calling a resuscitation team (e.g. medical emergency team (MET)).
- While waiting for the team, assess the patient using the ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach.
- Give the patient oxygen. Use a pulse oximeter to guide oxygen therapy.\(^5\)
- Attach monitoring: a minimum of pulse oximetry, ECG and blood pressure.
- Record vital signs observations and calculate the early warning score.
- Obtain venous access, and take blood samples for investigation.
- Prepare for handover using SBAR (Situation, Background, Assessment, Recommendation) or RSVP (Reason, Story, Vital signs, Plan).\(^6,7\)

3B. If the patient does not respond

- Shout for help (if not done already).
- Turn the patient on to his back.
- Open the airway using head tilt and chin lift.
- If there is a risk of cervical spine injury, establish a clear upper airway by using jaw thrust or chin lift in combination with manual in-line stabilisation (MILS) of the head and neck by an assistant (if enough people are available). If life-threatening airway obstruction persists despite effective application of jaw thrust or chin lift, add head tilt a small amount at a time until the airway is open; establishing a patent airway, oxygenation and ventilation takes priority over concerns about a potential cervical spine injury.
- Keeping the airway open, look, listen, and feel to determine if the victim is breathing normally. This is a rapid check and should take less than 10 seconds:
  - Look for chest movement (breathing or coughing)
  - Look for any other movement or signs of life
  - Listen at the victim’s mouth for breath sounds
  - Feel for air on your cheek
- If trained and experienced in the assessment of sick patients, check for breathing and assess the carotid pulse at the same time. The assessment should take less than 10 seconds whether you do a pulse check or not.
- Agonal breathing (occasional, irregular gasps) is common in the early stages of cardiac arrest and is a sign of cardiac arrest and should not be mistaken for a sign of life. Agonal breathing and limb movement can also occur during chest compressions as cerebral perfusion improves, but is not indicative of a return of spontaneous
circulation (ROSC). 

- Changes in skin colour (e.g. pallor, cyanosis) in isolation are not diagnostic of cardiac arrest. 
- If the patient is already attached to monitoring in a critical care area this will add to rather than replace the assessment for signs of life.

**4A. If there are signs of life or a pulse**

- Urgent medical assessment is required. Depending on the local protocols, this may take the form of a resuscitation team.
- While awaiting the team, assess and treat the patient using the ABCDE approach.
- Follow the steps in 3A above whilst waiting for the team.
- The patient is at high risk of further deterioration and cardiac arrest and needs continued observation until help arrives.

**4B. If there are no signs of life and no pulse**

- Start CPR and get a colleague to call the resuscitation team and collect the resuscitation equipment and a defibrillator.
- If alone, leave the patient to get help and equipment.
- Chest compressions in a patient whose heart is still beating are unlikely to cause harm. However, delays in diagnosing cardiac arrest and starting CPR will adversely affect chances of survival and must be avoided, so if there is any doubt proceed as if there are no signs of life and no pulse.
- Give 30 chest compressions followed by 2 ventilations.
- The correct hand position for chest compression is the middle of the lower half of the sternum.
- This hand position can be found quickly if you have been taught to ‘place the heel of one hand in the centre of the chest with the other hand on top’ and your teaching included a demonstration of placing hands in the middle of the lower half of the sternum.
- Ensure high quality chest compressions:
  - Depth of 5–6 cm
  - Rate of 100–120 compressions min\(^{-1}\)
  - Allow the chest to recoil completely after each compression
  - Take approximately the same amount of time for compression and relaxation
  - Minimise any interruptions to chest compression (hands-off time)
- If available, use a prompt and/or feedback device to help ensure high quality chest compressions. The use of these devices should be part of a hospital-wide quality improvement program that includes formal debriefing.
- Do not rely on palpating carotid or femoral pulses to assess the effectiveness of chest compressions.
- Resume compressions without any delay; place your hands back on the centre of the patient’s chest.
- If there are enough team members, the person doing chest compressions should change about every 2 min or sooner if they are unable to maintain high quality chest compressions. This change should be done with minimal interruption to compressions. This should be done during planned pauses in chest compression such as during rhythm assessment.
- Use whatever equipment is available immediately for airway and ventilation (e.g. a self-inflating bag-mask, or a supraglottic airway device and bag according to local policy).
- Use an inspiratory time of about 1 second and give enough volume to produce a visible rise of the chest wall. Avoid rapid or forceful breaths.
- Add supplemental oxygen as soon as possible.
- There are usually good clinical reasons to avoid mouth-to-mouth ventilation in clinical settings, and it is therefore not commonly used, but there will be situations where giving mouth-to-mouth breaths could be life-saving (e.g. in non-clinical settings). If there are clinical reasons to avoid mouth-to-mouth contact, or you are unable to do this, do chest compressions until help or airway equipment arrives. A pocket mask or bag-mask should be immediately available in all clinical areas.
- Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill,
and can insert the tracheal tube with minimal interruption (less than 5 seconds) to chest compressions. Waveform capnography must be used routinely for confirming that a tracheal tube is in the patient’s airway and subsequent monitoring during CPR. Waveform capnography can also be used to monitor the quality of CPR, as an indicator of a ROSC and to help with determining prognosis during CPR.

- Once the patient’s trachea has been intubated, continue chest compressions uninterrupted (except for defibrillation or rhythm checks when indicated), at a rate of 100–120 min⁻¹, and ventilate the lungs at approximately 10 breaths min⁻¹ (i.e. do not stop chest compressions for ventilation). If a supraglottic airway (e.g. LMA) device has been inserted it may also be possible to ventilate the patient without stopping chest compressions.

- As soon as a defibrillator arrives, apply the self-adhesive pads to the patient’s chest whilst chest compressions are ongoing. The use of adhesive electrode pads will enable rapid assessment of heart rhythm compared with the use of ECG electrodes.⁴

- Once the pads are applied, pause briefly for a rapid rhythm check – aim for a pause in chest compressions of less than 5 seconds.

- If the rhythm is ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT), restart chest compressions. All other team members must now be informed to stand clear of the patient whilst the defibrillator is charged and a safety check performed. Once the defibrillator is charged and the safety check completed, stop chest compressions, deliver the shock and restart chest compressions immediately.

- Do not delay restarting chest compressions to check the cardiac rhythm.

- Using a manual defibrillator it is possible to reduce the pause between stopping and restarting of chest compressions to less than 5 seconds.

- If staff cannot use a manual defibrillator, use an automated external defibrillator (AED). Switch on the AED and follow the audio-visual prompts.

- Rescuers must not compromise on safety. All actions should be planned by the team before pausing chest compressions. If there are delays caused by difficulties in rhythm analysis or if individuals are still in contact with the patient, chest compressions are restarted whilst a decision is made what to do when compressions are next paused.

- Continue resuscitation until the resuscitation team arrives or the patient shows signs of life. Follow the Advanced life support algorithm.

- Once resuscitation is underway, and if there are sufficient staff present, prepare intravenous cannulae and drugs likely to be used by the resuscitation team (e.g. adrenaline).

- Use a watch or clock for timing between rhythm checks. Any interruption to CPR should be planned. Assess the cardiac rhythm about every 2 minutes.

- Identify one person to be responsible for handover to the resuscitation team leader. Use a structured communication tool for handover (e.g. SBAR, RSVP).

- Locate the patient’s records and ensure that they are available immediately the resuscitation team arrives.

4C. If the patient is not breathing and has a pulse (respiratory arrest)

- Ventilate the patient’s lungs (as described above) and check for a pulse every 10 breaths (about every minute).

- This diagnosis can be made only if you are confident in assessing breathing and pulse or the patient has other signs of life (e.g. warm and well-perfused, normal capillary refill).

- If there are any doubts about the presence of a pulse, start chest compressions and continue ventilations until more experienced help arrives. All patients in respiratory arrest will develop cardiac arrest if the respiratory arrest is not treated rapidly and effectively.

5. If the patient has a monitored and witnessed cardiac arrest

If a patient has a monitored and witnessed cardiac arrest in the catheter laboratory, coronary care unit, a critical care area, or whilst monitored after cardiac surgery, and a manual defibrillator is rapidly available:

- Confirm cardiac arrest and shout for help.

- If the initial rhythm is VF/pVT, give up to three quick successive (stacked) shocks.

- Rapidly check for a rhythm change and, if appropriate check for a pulse and other signs of ROSC after each defibrillation attempt.
• Start chest compressions and continue CPR for 2 min if the third shock is unsuccessful. These initial three stacked shocks are considered as giving the first shock in the ALS algorithm.

• This three-shock strategy may also be considered for an initial, witnessed VF/pVT cardiac arrest if the patient is already connected to a manual defibrillator – these circumstances are rare.

• A precordial thump works only rarely. Delivery of a precordial thump must not delay calling for help or accessing a defibrillator. Consider a precordial thump only when it can be used without delay whilst awaiting the arrival of a defibrillator in a monitored VF/pVT arrest. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of about 20 cm, then retract the fist immediately to create an impulse-like stimulus.

5. Background notes

Hospital and staff factors

The exact sequence of actions after in-hospital cardiac arrest depends on several factors including:

• location (clinical or non-clinical area; monitored or unmonitored patients)
• skills of staff who respond
• number of responders
• equipment available
• hospital system for response to cardiac arrest and medical emergencies (e.g. MET, cardiac arrest team).

Location

Monitored cardiac arrests are usually diagnosed rapidly. Ward patients may have had a period of deterioration and an unwitnessed arrest. Ideally, all patients who are at high risk of cardiac arrest should be cared for in a monitored area where facilities for immediate resuscitation are available. Patients, visitors, or staff may also have a cardiac arrest in non-clinical areas (e.g. car parks, corridors).

Delay in attempting defibrillation can occur when patients sustain cardiac arrest in unmonitored hospital beds and in outpatient departments. In these areas several minutes may elapse before a resuscitation team arrives with a defibrillator and delivers a shock. Automated external defibrillation should be considered to facilitate early defibrillation (aiming for shock delivery within 3 min of collapse) in areas where staff have no rhythm recognition skills or use defibrillators infrequently. However, an automated external defibrillator (AED) should not be used in preference to a manual defibrillator when staff are present who have rhythm recognition and manual defibrillation skills.

Skills of staff who respond

All healthcare professionals should be able to recognise cardiac arrest, call for help, and start resuscitation. Staff should do what they have been trained to do. For example, staff in critical care and emergency medicine may have more advanced resuscitation skills than staff who are not involved regularly in resuscitation in their normal clinical role. Hospital staff who attend a cardiac arrest may have different competencies in managing the airway, breathing, and circulation. Rescuers should use those resuscitation skills in which they have been trained.

The Resuscitation Council (UK) Immediate Life Support (ILS) course is aimed at the majority of healthcare professionals who attend cardiac arrests rarely but have the potential to be first responders or resuscitation team members. The Resuscitation Council (UK) Advanced Life Support (ALS) course is aimed at doctors and senior nurses working in acute areas of the hospital and those who may be resuscitation team leaders and members. The course is also suitable for senior paramedics and some hospital technicians. During training and clinical practice there should be a greater emphasis on non-technical skills (NTS). These consist of situational awareness, decision making, team working, team leadership, task management and communication. Tools such as SBAR or RSVP should be used to ensure rapid effective communication and handovers.

Number of responders

The single responder must ensure that help is on its way. If other staff are nearby, several actions can be undertaken simultaneously. Numbers of hospital staff tend to be fewer during the night and at weekends. This may influence
patient monitoring, recognition, treatment and outcomes. Data from the US shows that survival rates from in-hospital cardiac arrest are lower during nights and weekends. Several studies show that increased nurse staffing is associated with lower rates of failure-to-rescue, and reductions in incidence of cardiac arrest, pneumonia, shock and death.

**Equipment available**

The equipment used for CPR (including defibrillators) and the layout of equipment and drugs should be standardised throughout the hospital. A review by the Resuscitation Council (UK) of serious patient safety incidents associated with CPR and patient deterioration reported to the National Patient Safety Agency showed that equipment problems are a common contributing cause. All resuscitation equipment must be checked regularly to ensure it is ready for use. Specially designed trolleys or sealed tray systems improve speed of access to equipment and reduce adverse incidents.

Hospitals and teams must have monitoring and equipment for transferring patients after they have been resuscitated. This includes portable monitors with a minimum capability of pulse oximetry, ECG, and non-invasive blood pressure measurement. In addition waveform capnography must be used for all patients after tracheal intubation. For further information, refer to the Intensive Care Society’s Guidelines for the Transport of the Critically Ill Adult.

www.ics.ac.uk/ics-homepage/guidelines-and-standards/

**Resuscitation team**

The resuscitation team may take the form of a traditional cardiac arrest team, which is called only when cardiac arrest is recognised. Alternatively, hospitals may have strategies to recognise patients at risk of cardiac arrest and summon a team (e.g. MET) before cardiac arrest occurs. The term ‘resuscitation team’ reflects the range of response teams. In-hospital cardiac arrests are rarely sudden or unexpected. A strategy of recognising patients at risk of cardiac arrest may enable some of these arrests to be prevented, or may prevent futile resuscitation attempts in those patients who are unlikely to benefit from CPR (See Prevention of in-hospital cardiac arrest and decisions about CPR www.resus.org.uk/resuscitation-guidelines/prevention-of-cardiac-arrest-and-decisions-about-cpr).

Surveys show that resuscitation teams rarely have formal briefings and/or debriefings. Resuscitation team members should meet for introductions and plan roles and responsibilities before they attend actual events. Team members should also debrief after each event based on events during the resuscitation. The aim should be to discuss problems and concerns openly and allow learning and improvement in a constructive manner. Ideally this should be based on data collected during the event.

**Diagnosis of cardiac arrest**

Trained healthcare staff cannot assess the breathing and pulse sufficiently reliably to confirm cardiac arrest. Agonal breathing (infrequent and irregular gasps) is a pre-terminal event and common in the early stages of cardiac arrest. It should not be confused as a sign of life/circulation. Agonal breathing can also occur during chest compressions as cerebral perfusion improves, but is not indicative of a ROSC. In addition, immediately after cardiac arrest the sudden cessation of cerebral blood flow can cause an initial short seizure-like episode that can be confused with epilepsy.

Delivering chest compressions to a patient with a beating heart is unlikely to cause harm. However, delays in diagnosis of cardiac arrest and starting CPR will adversely effect survival and must be avoided.

**High quality CPR**

The quality of chest compressions during in-hospital CPR is frequently sub-optimal and interruptions are often prolonged. Even short interruptions to chest compressions can adversely impact on outcome and every effort must be made to ensure that continuous, effective chest compression is maintained throughout the resuscitation attempt. Chest compressions should commence at the beginning of a resuscitation attempt and continue uninterrupted apart from a brief pause for a specific intervention (e.g. rhythm check). Most interventions can be performed without interruptions to chest compressions. The team leader should monitor the quality of CPR, change the person providing chest compressions every 2 minutes (during rhythm assessment) or sooner if the quality of CPR is poor.

**Defibrillation strategy**

The length of the pre-shock pause (i.e. the interval between stopping chest compressions and delivering a shock) is inversely related to the chance of successful defibrillation. Every five-second increase in the duration of the pre-shock pause almost halves the chance of successful defibrillation, therefore it is critical to minimise the pause.

The traditional lengthy ‘top-to-toe’ safety check (e.g. “head, middle, bottom, self, oxygen away”) performed after the
defibrillator has charged and before shock delivery, will therefore diminish significantly the chances of successful defibrillation. The pre-shock pause can be substantially reduced by continuing compressions during charging of the defibrillator and by having an efficient team coordinated by a leader who communicates effectively. The safety check to avoid rescuer contact with the patient at the moment of defibrillation should be undertaken rapidly but efficiently. The post shock pause is minimised by resuming chest compressions immediately after shock delivery. The process of shock delivery should be achievable with less than a 5 second interruption to chest compressions.

Rescuers must not compromise on safety. Roles should be agreed by the team members before attending a cardiac arrest. Always plan actions before stopping chest compressions. If there are delays caused by difficulties in rhythm analysis or if individuals are still in contact with the patient as the shock is about to be delivered, restart chest compressions whilst plans are made to decide what to do when compressions are next stopped. Rescuers should wear gloves during CPR attempts but do not delay starting CPR if gloves are not immediately available.

Although there are no data supporting a three-shock strategy, it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs immediately after onset of VF/pVT. In circumstances where rapid early defibrillation is feasible (e.g. cardiac catheter laboratory, in monitored cardiac surgery patients, patients who have a witnessed and monitored VF/VT and are already connected to a defibrillator) three rapid defibrillation attempts in quick succession, may achieve ROSC without the need for chest compressions.

**National Cardiac Arrest Audit**

All in-hospital cardiac arrests should be reviewed and audited. The National Cardiac Arrest Audit (NCAA) [https://ncaa.icnarc.org/](https://ncaa.icnarc.org/) is a UK-wide database of in-hospital cardiac arrests and is supported by the Resuscitation Council (UK) and the Intensive Care National Audit & Research Centre (ICNARC). NCAA monitors and reports on the incidence of and outcome from, in-hospital cardiac arrests in order to inform practice and policy. It aims to identify and foster improvements in the prevention, care delivery and outcomes from cardiac arrest. Participating in NCAA enables hospitals to collect and contribute to national, standardised data on cardiac arrest, enabling local and national improvements in patient care.49-53

6. References

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Post-resuscitation care

1. The guideline process
2. Summary of changes in post-resuscitation care since the 2010 Guidelines
3. Introduction
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10. Organ donation
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1. The guideline process

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- 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.
- Collaboration with the European Resuscitation Council and European Society of Intensive Care Medicine, and adapting their post-resuscitation care guidelines for use in the UK.\(^3\)
- These Resuscitation Council (UK) Guidelines have been peer reviewed by the Executive Committee of the Resuscitation Council (UK), which comprises 25 individuals and includes lay representation and representation of the key stakeholder groups.
2. Summary of changes in post-resuscitation care since the 2010 Guidelines

This section is new to the Resuscitation Council (UK) Guidelines; in 2010 the topic was incorporated into the section on Advanced life support.

The most important changes in post-resuscitation care since 2010 include:

• There is a greater emphasis on the need for urgent coronary catheterisation and percutaneous coronary intervention (PCI) following out-of-hospital cardiac arrest of likely cardiac cause.

• Targeted temperature management remains important but the target temperature can be in the range of 32°C to 36°C according to local policy. There was a preference for 36°C among the guidelines group because it is easier to implement and there is no evidence that it is inferior to 33°C.

• Prognostication is now undertaken using a multimodal strategy and there is emphasis on allowing sufficient time for neurological recovery and to enable sedatives to be cleared.

3. Introduction

Successful return of spontaneous circulation (ROSC) is the first step towards the goal of complete recovery from cardiac arrest. The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response during CPR and following successful resuscitation have been termed the post-cardiac arrest syndrome. Depending on the cause of the arrest, and the severity of the post-cardiac arrest syndrome, many patients will require multiple organ support and the treatment they receive during this post-resuscitation period influences significantly the overall outcome and particularly the quality of neurological recovery. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient is transferred to the most appropriate high-care area (e.g. emergency room, cardiac catheterisation laboratory or intensive care unit (ICU)) for continued diagnosis, monitoring and treatment. The post-resuscitation care algorithm (Figure 1) outlines some of the key interventions required to optimise outcome for these patients.

Of those comatose patients admitted to ICUs after cardiac arrest, as many as 40–50% survive to be discharged from hospital depending on the cause of arrest, system and quality of care. Of the patients who survive to hospital discharge, the vast majority have a good neurological outcome although many have subtle cognitive impairment.
4. The post-cardiac arrest syndrome

The post-cardiac arrest syndrome comprises:  

- post-cardiac arrest brain injury  
- post-cardiac arrest myocardial dysfunction  
- systemic ischaemia/reperfusion response  
- persistent precipitating pathology.

The severity of this syndrome will vary with the duration and cause of cardiac arrest. It may not occur at all if the cardiac arrest is brief. Post-cardiac arrest brain injury manifests as coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction and brain death. Among patients surviving to ICU admission but subsequently dying in-hospital, brain injury is the cause of death in approximately two thirds after out-of-hospital cardiac arrest and approximately 25% after in-hospital cardiac arrest.  

Cardiovascular failure accounts for most deaths in the first three days, while brain injury accounts for most of the later deaths.  

Withdrawal of life-sustaining therapy (WLST) is the most frequent cause of death (approximately 50%) in patients with a prognosticated bad outcome, emphasising...
the importance of the prognostication plan (see below). Post-cardiac arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypotension, hypercarbia, hypoxaemia, hyperoxaemia, pyrexia, hypoglycaemia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically starts to recover by 2–3 days, although full recovery may take significantly longer.\textsuperscript{12} The whole body ischaemia/reperfusion of cardiac arrest activates immune and coagulation pathways contributing to multiple organ failure and increasing the risk of infection. Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion, vasodilation, endothelial injury and abnormalities of the microcirculation.\textsuperscript{13–15}

5. Airway and breathing

Control of oxygenation

Patients who have had a brief period of cardiac arrest responding immediately to appropriate treatment may achieve an immediate return of normal cerebral function. These patients do not require tracheal intubation and ventilation but should be given with oxygen via a facemask if their arterial blood oxygen saturation is less than 94%. Hypoxaemia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Several animal studies indicate that hyperoxaemia early after ROSC causes oxidative stress and harms post-ischaemic neurons.\textsuperscript{16} A meta-analysis of 14 observational studies showed significant heterogeneity across studies, with some studies showing that hyperoxaemia is associated with a worse neurological outcome and other failing to show this association.\textsuperscript{17}

The animal studies showing a relationship between hyperoxia and worse neurological outcome after cardiac arrest have generally evaluated the effect of hyperoxia in the first hour after ROSC. There are significant practical challenges with the titration of inspired oxygen concentration immediately after ROSC, particularly in the out-of-hospital setting. It may be difficult to obtain reliable arterial blood oxygen saturation values using pulse oximetry in this setting.\textsuperscript{18} A recent study of air versus supplemental oxygen in ST-elevation myocardial infarction (STEMI) showed that supplemental oxygen therapy increased myocardial injury, recurrent myocardial infarction and major cardiac arrhythmia and was associated with larger infarct size at six months.\textsuperscript{19}

Given the evidence of harm after myocardial infarction and the possibility of increased neurological injury after cardiac arrest, as soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%. Avoid hypoxaemia, which is also harmful – ensure reliable measurement of arterial oxygen saturation before reducing the inspired oxygen concentration.

Control of ventilation

Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. Ensure the tracheal tube is positioned correctly, well above the carina. Hypocarbia causes cerebral vasoconstriction and a decreased cerebral blood flow.\textsuperscript{20} After cardiac arrest, hypocapnia induced by hyperventilation causes cerebral ischaemia.\textsuperscript{21} Observational studies using cardiac arrest registries document an association between hypocapnia and poor neurological outcome.\textsuperscript{22,23} Two observational studies have documented an association with mild hypercapnia and better neurological outcome among post-cardiac arrest patients in the ICU.\textsuperscript{23,24} Until prospective data are available, it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal CO\textsubscript{2} and arterial blood gas values. Although protective lung ventilation strategies have not been studied specifically in post-cardiac arrest patients, given that these patients develop a marked inflammatory response, it seems rational to apply protective lung ventilation: tidal volume 6–8 mL kg\textsuperscript{-1} ideal body weight and positive end expiratory pressure 4–8 cm H\textsubscript{2}O.\textsuperscript{15,25}

Insert a gastric tube to decompress the stomach; gastric distension caused by mouth-to-mouth or bag-mask ventilation will splint the diaphragm and impair ventilation. Give adequate doses of sedative, which will reduce oxygen consumption. A sedation protocol is highly recommended. Bolus doses of a neuromuscular blocking drug may be required, particularly if using targeted temperature management (TTM) (see below). Limited evidence shows that short-term infusion (≤48 h) of short-acting neuromuscular blocking drugs given to reduce patient-ventilator dysynchrony and risk of barotrauma in patients with acute respiratory distress syndrome (ARDS) is not associated with an increased risk of ICU-acquired weakness and may improve outcome in these patients.\textsuperscript{26} There are some data suggesting that continuous neuromuscular blockade is associated with decreased mortality in post-cardiac arrest patients;\textsuperscript{27} however, infusions of neuromuscular blocking drugs interfere with clinical examination and may mask seizures. Continuous electroencephalography (EEG) is recommended to detect seizures in these patients, especially
when neuromuscular blockade is used. Obtain a chest radiograph to check the position of the tracheal tube, gastric tube and central venous lines, assess for pulmonary oedema, and detect complications from CPR such as a pneumothorax associated with rib fractures.

6. Circulation

Coronary reperfusion

Acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA): in a recent meta-analysis, the prevalence of an acute coronary artery lesion ranged from 59%–71% in OHCA patients without an obvious non-cardiac aetiology. Many observational studies have shown that emergent cardiac catheterisation laboratory evaluation, including early percutaneous coronary intervention (PCI), is feasible in patients with ROSC after cardiac arrest. The invasive management (i.e. early coronary angiography followed by immediate PCI if deemed necessary) of these patients, particularly those having prolonged resuscitation and nonspecific ECG changes, has been controversial because of the lack of high-quality evidence and significant implications on use of resources (including transfer of patients to PCI centres).

Percutaneous coronary intervention following ROSC with ST-elevation

In patients with ST segment elevation (STE) or left bundle branch block (LBBB) on the post-ROSC electrocardiogram (ECG) more than 80% will have an acute coronary lesion. There are no randomised studies but given that many observational studies reported increased survival and neurologically favourable outcome, it is highly probable that early invasive management is beneficial in STE patients. Immediate angiography and PCI when indicated should be performed in resuscitated OHCA patients whose initial ECG shows ST-elevation, even if they remain comatose and ventilated. The National Institute for Health and Care Excellence (NICE) Clinical Guideline 167 for the acute management of STEMI recommends: ‘Do not use level of consciousness after cardiac arrest caused by suspected acute STEMI to determine whether a person is eligible for coronary angiography (with follow-on primary PCI if indicated)’. This recommendation is based on low quality evidence from selected populations. Observational studies also indicate that optimal outcomes after OHCA are achieved with a combination of TTM and PCI, which can be included in a standardised post-cardiac arrest protocol as part of an overall strategy to improve neurologically intact survival.

Percutaneous coronary intervention following ROSC without ST-elevation

In contrast to the usual presentation of ACS in non-cardiac arrest patients, the standard tools to assess coronary ischaemia in cardiac arrest patients are less accurate. The sensitivity and specificity of the usual clinical data, ECG and biomarkers to predict an acute coronary artery occlusion as the cause of OHCA are unclear. Several large observational series showed that absence of STE may also be associated with ACS in patients with ROSC following OHCA. In these non-STE patients, there are conflicting data from observational studies on the potential benefit of emergent cardiac catheterisation laboratory evaluation. It is reasonable to discuss and consider emergent cardiac catheterisation laboratory evaluation after ROSC in patients with the highest risk of a coronary cause for their cardiac arrest. Factors such as patient age, duration of CPR, haemodynamic instability, presenting cardiac rhythm, neurological status upon hospital arrival, and perceived likelihood of cardiac aetiology can influence the decision to undertake the intervention in the acute phase or to delay it until later in the hospital stay.

Indications and timing of computed tomography (CT) scanning

Cardiac causes of OHCA have been extensively studied in the last few decades; conversely, little is known about non-cardiac causes. Early identification of a respiratory or neurological cause can be achieved by performing a brain and chest CT scan at hospital admission, before or after coronary angiography. In the absence of signs or symptoms suggesting a neurological or respiratory cause (e.g. headache, seizures or neurological deficits for neurological causes, shortness of breath or documented hypoxaemia in patients suffering from a known and worsening respiratory disease) or if there is clinical or ECG evidence of myocardial ischaemia, undertake coronary angiography first, followed by CT scan in the absence of causative lesions.

Haemodynamic management

Post-resuscitation myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias. Perform early echocardiography in all patients in order to detect and quantify the degree of myocardial dysfunction. Post-resuscitation myocardial dysfunction often requires inotropic support, at least transiently. The systematic inflammatory response that occurs frequently in post-cardiac arrest patients may cause
vasoplegia and severe vasodilation. Thus, noradrenaline, with or without dobutamine, and fluid is usually the most effective treatment. The controlled infusion of relatively large volumes of fluid is tolerated remarkably well by patients with post-cardiac arrest syndrome.

Treatment may be guided by blood pressure, heart rate, urine output, rate of plasma lactate clearance, and central venous oxygen saturation. Serial echocardiography may also be used, especially in haemodynamically unstable patients. In the ICU an arterial line for continuous blood pressure monitoring is essential. Cardiac output monitoring may help to guide treatment in haemodynamically unstable patients but there is no evidence that its use affects outcome. Some centres still advocate use of an intra aortic balloon pump (IABP) in patients with cardiogenic shock, although the IABP-SHOCK II Trial failed to show that use of the IABP improved 30-day mortality in patients with myocardial infarction and cardiogenic shock.54,55

A bundle of therapies, including a specific blood pressure target, has been proposed as a treatment strategy after cardiac arrest. However its influence on clinical outcome is not firmly established and optimal targets for mean arterial pressure and/or systolic arterial pressure remain unknown. In the absence of definitive data, target the mean arterial blood pressure to achieve an adequate urine output (1 mL kg$^{-1}$ h$^{-1}$) and normal or decreasing plasma lactate values, taking into consideration the patient’s normal blood pressure, the cause of the arrest and the severity of any myocardial dysfunction.4

During mild induced hypothermia the normal physiological response is bradycardia. Recent retrospective studies have shown that bradycardia is associated with a good outcome. As long as blood pressure, lactate and urine output are sufficient, a bradycardia of $\leq$40 min$^{-1}$ may be left untreated. Importantly, oxygen requirements during mild induced hypothermia are reduced.

Immediately after a cardiac arrest there is typically a period of hyperkalaemia. Subsequent endogenous catecholamine release and correction of metabolic and respiratory acidosis promotes intracellular transportation of potassium, causing hypokalaemia. Hypokalaemia may predispose to ventricular arrhythmias. Give potassium to maintain the serum potassium concentration between 4.0 and 4.5 mmol L$^{-1}$.

Implantable cardioverter defibrillators

Consider insertion of an implantable cardioverter defibrillator (ICD) in ischaemic patients with significant left ventricular dysfunction, who have been resuscitated from a ventricular arrhythmia that occurred later than 24–48 h after a primary coronary event. ICDs may also reduce mortality in cardiac arrest survivors at risk of sudden death from structural heart diseases or inherited cardiomyopathies.2

7. Disability (optimising neurological recovery)

Cerebral perfusion

Animal studies show that immediately after ROSC there is a short period of multifocal cerebral no-reflow followed by transient global cerebral hyperaemia lasting 15–30 min. This is followed by up to 24 h of cerebral hypoperfusion while the cerebral metabolic rate of oxygen gradually recovers. After asphyxial cardiac arrest, brain oedema may occur transiently after ROSC but it is rarely associated with clinically relevant increases in intracranial pressure. In many patients, autoregulation of cerebral blood flow is impaired (absent or right-shifted) for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity. In one study autoregulation was disturbed in 35% of post-cardiac arrest patients and the majority of these had been hypertensive before their cardiac arrest; this tends to support the recommendation made in the 2010 ERC Guidelines: after ROSC, maintain mean arterial pressure near the patient’s normal level.

Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 h after ROSC, there are no high-level data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. Patients need to be sedated adequately during treatment with TTM, and the duration of sedation and ventilation is therefore influenced by this treatment. A combination of opioids and hypnotics is usually used. Short-acting drugs (e.g. propofol, alfentanil, remifentanil) will enable more reliable and earlier neurological assessment and prognostication (see prognostication below). Adequate sedation will reduce oxygen consumption. Use of published sedation scales for monitoring these patients (e.g. the Richmond or Ramsay Scales) may be helpful.
Control of seizures

Seizures are common after cardiac arrest and occur in approximately one-third of patients who remain comatose after ROSC. Myoclonus is most common and occurs in 18–25%, the remainder having focal or generalised tonic-clonic seizures or a combination of seizure types. Clinical seizures, including myoclonus may or may not be of epileptic origin. Other motor manifestations could be mistaken for seizures and there are several types of myoclonus, the majority being non-epileptic. Use intermittent electroencephalography (EEG) to detect epileptic activity in patients with clinical seizure manifestations. Consider continuous EEG to monitor patients with a diagnosed status epilepticus and effects of treatment.

In comatose cardiac arrest patients, EEG commonly detects epileptiform activity: post-anoxic status epilepticus was detected in 23–31% of patients using continuous EEG-monitoring. Patients with electrographic status epilepticus may or may not have clinically detectable seizure manifestations that may be masked by sedation. Whether systematic detection and treatment of electrographic epileptic activity improves patient outcome is not known.

Seizures may increase the cerebral metabolic rate and have the potential to exacerbate brain injury caused by cardiac arrest: treat with sodium valproate, levetiracetam, phenytoin, benzodiazepines, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Propofol is effective to suppress post-anoxic myoclonus. Clonazepam, sodium valproate and levetiracetam are antimyoclonic drugs that may be effective in post-anoxic myoclonus. Routine seizure prophylaxis in post-cardiac arrest patients is not recommended because of the risk of adverse effects and the poor response to anti-epileptic drugs among patients with clinical and electrographic seizures.

Myoclonus and electrographic seizure activity, including status epilepticus, are related to a poor prognosis but individual patients may survive with good outcome (see prognostication). Prolonged observation may be necessary after treatment of seizures with sedatives, which will decrease the reliability of a clinical examination.

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome. A large randomised trial of intensive glucose control (4.5–6.0 mmol L⁻¹) versus conventional glucose control (10 mmol L⁻¹ or less) in general ICU patients reported increased 90-day mortality in patients treated with intensive glucose control. Severe hypoglycaemia is associated with increased mortality in critically ill patients, and comatose patients are at particular risk from unrecognised hypoglycaemia. Irrespective of the target range, variability in glucose values is associated with mortality. Compared with normothermia, mild induced hypothermia is associated with higher blood glucose values, increased glucose variability and greater insulin requirements. Increased blood glucose variability is associated with increased mortality and unfavourable neurological outcome after cardiac arrest. Based on the available data, following ROSC maintain the blood glucose at ≤ 10 mmol L⁻¹ and avoid hypoglycaemia. Do not implement strict glucose control in adult patients with ROSC after cardiac arrest because it increases the risk of hypoglycaemia.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest. Several studies document an association between post-cardiac arrest pyrexia and poor outcomes. The development of hyperthermia after a period of mild induced hyperthermia (rebound hyperthermia) is associated with increased mortality and worse neurological outcome. There are no randomised controlled trials evaluating the effect of treatment of pyrexia (defined as ≥37.6 °C) compared to no temperature control in patients after cardiac arrest and the elevated temperature may only be an effect of a more severely injured brain. Although the effect of elevated temperature on outcome is not proven, it seems reasonable to treat hyperthermia occurring after cardiac arrest with antipyretics and to consider active cooling in unconscious patients.

Targeted temperature management

Animal and human data indicate that mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia. Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen (CMRO₂) by about 6% for each 1°C reduction in core temperature and this may reduce the release of excitatory amino acids and free radicals. Hypothermia blocks the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response associated with the post-cardiac
All studies of post-cardiac arrest mild induced hypothermia have included only patients in coma. One randomised trial and a pseudo-randomised trial demonstrated improved neurological outcome at hospital discharge or at six months in comatose patients after out-of-hospital VF cardiac arrest. Cooling was initiated within minutes to hours after ROSC and a temperature range of 32–34°C was maintained for 12–24 h. In the Targeted Temperature Management (TTM) trial, 950 all-rhythm OHCA patients were randomised to 36 h of temperature control (comprising 28 h at the target temperature followed by slow rewarmin) at either 33°C or 36°C. There was no difference in mortality and detailed neurological outcome at 6 months was also similar. Importantly, patients in both arms of this trial had their temperature well controlled so that fever was prevented in both groups.

The optimal duration for mild induced hypothermia and TTM is unknown although it is currently most commonly used for 24 h. Previous trials treated patients with 12–28 h of targeted temperature management. Two observational trials found no difference in mortality or poor neurological outcome with 24 h compared with 72 h of hypothermia. The TTM trial provided strict normothermia (<37.5°C) after hypothermia until 72 h after ROSC.

The term targeted temperature management or temperature control is now preferred over the previous term therapeutic hypothermia. The Advanced Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) made several treatment recommendations on targeted temperature management:

- Maintain a constant, target temperature between 32°C and 36°C for those patients in whom temperature control is used.
- TTM is recommended for adults after OHCA with an initial shockable rhythm who remain unresponsive after ROSC.
- TTM is suggested for adults after OHCA with an initial nonshockable rhythm who remain unresponsive after ROSC.
- TTM is suggested for adults after IHCA with any initial rhythm who remain unresponsive after ROSC.
- If targeted temperature management is used, it is suggested that the duration is at least 24 h.

Following the TTM trial, many intensive care clinicians in the UK have elected to use 36°C as the target temperature for post cardiac arrest temperature control. This has several advantages compared with a target temperature of 33°C:

- There is a reduced need for vasopressor support.
- Lactate values are lower (the clinical significance of this is unclear).
- The rewarmin phase is shorter.
- There is reduced risk or rebound hyperthermia after rewarmin.

**How to control temperature**

The practical application of TTM is divided into three phases: induction, maintenance and rewarmin. External and/or internal cooling techniques can be used to initiate and maintain TTM.

Animal data indicate that earlier cooling after ROSC produces better outcome but this has yet to be demonstrated in humans. External and/or internal cooling techniques can be used to initiate cooling. If a lower target temperature (e.g. 33°C) is chosen, an infusion of 30 mL kg⁻¹ of 4°C saline or Hartmann’s solution will decrease core temperature by approximately 1.0–1.5°C and is probably safe in a well-monitored environment. Prehospital cooling using this technique is not recommended because there is some evidence of increased risk of pulmonary oedema and re-arrest during transport to hospital.

Methods of inducing and/or maintaining TTM include:

- Simple ice packs and/or wet towels are inexpensive; however, these methods may be more time consuming for nursing staff, may result in greater temperature fluctuations, and do not enable controlled rewarmin. Ice cold fluids alone cannot be used to maintain hypothermia, but even the addition of simple ice packs may control the temperature adequately.
- Cooling blankets or pads.
- Water or air circulating blankets.
- Water circulating gel-coated pads.
- Transnasal evaporative cooling – this technique enables cooling before ROSC and is undergoing further
Intravascular heat exchanger, placed usually in the femoral or subclavian veins.

Extracorporeal circulation (e.g. cardiopulmonary bypass, ECMO).

In most cases, it is easy to cool patients initially after ROSC because the temperature normally decreases within this first hour. Admission temperature after OHCA is usually between 35°C–36°C and in a recent large trial the median temperature was 35.3°C. If a target temperature of 36°C is chosen allow a slow passive rewarm to 36°C. If a target temperature of 33°C is chosen, initial cooling is facilitated by neuromuscular blockade and sedation, which will prevent shivering. Magnesium sulfate, a naturally occurring N-methyl-D-aspartate (NMDA) receptor antagonist, that reduces the shivering threshold slightly, can also be given to reduce the shivering threshold.

In the maintenance phase, a cooling method with effective temperature monitoring that avoids temperature fluctuations is preferred. This is best achieved with external or internal cooling devices that include continuous temperature feedback to achieve a set target temperature. The temperature is typically monitored from a thermistor placed in the bladder and/or oesophagus. As yet, there are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique; however, internal devices enable more precise temperature control compared with external techniques.

Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling. Rebound hyperthermia is associated with worse neurological outcome. Thus, rewarming should be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25–0.5°C of rewarming per hour. Choosing a strategy of 36°C will reduce this risk.

Physiological effects and complications of hypothermia

The well-recognised physiological effects of hypothermia need to be managed carefully:

- Shivering will increase metabolic and heat production, thus reducing cooling rates – strategies to reduce shivering are discussed above. The occurrence of shivering in cardiac arrest survivors who undergo mild induced hypothermia is associated with a good neurological outcome; it is a sign of a normal physiological response. Occurrence of shivering was similar at a target temperature of 33°C and 36°C. A sedation protocol is required.

- Mild induced hypothermia increases systemic vascular resistance and causes arrhythmias (usually bradycardia). Importantly, the bradycardia caused by mild induced hypothermia may be beneficial (similar to the effect achieved by beta-blockers); it reduces diastolic dysfunction and its occurrence has been associated with good neurological outcome.

- Mild induced hypothermia causes a diuresis and electrolyte abnormalities such as hypophosphataemia, hypokalaemia, hypomagnesaemia and hypocalcaemia.

- Hypothermia decreases insulin sensitivity and insulin secretion, and causes hyperglycaemia, which will need treatment with insulin.

- Mild induced hypothermia impairs coagulation and may increase bleeding, although this effect seems to be negligible and has not been confirmed in clinical studies. In one registry study, an increased rate of minor bleeding occurred with the combination of coronary angiography and mild induced hypothermia, but this combination of interventions was the also the best predictor of good outcome.

- Hypothermia can impair the immune system and increase infection rates. Mild induced hypothermia is associated with an increased incidence of pneumonia; however, this seems to have no impact on outcome. Although prophylactic antibiotic treatment has not been studied prospectively, in an observational study, use of prophylactic antibiotics was associated with a reduced incidence of pneumonia. In another observational study of 138 patients admitted to ICU after OHCA, early use of antibiotics was associated with improved survival.

- The serum amylase concentration is commonly increased during hypothermia but the significance of this is unclear.

- The clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34°C. Clearance of sedative and other drugs will be closer to normal at a temperature closer to 37.0°C.

Contraindications to hypothermia

Generally recognised contraindications to TTM at 33°C, but which are not applied universally, include: severe systemic infection and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to mild induced
hypothermia). Two observational studies documented a positive inotropic effect from mild induced hypothermia in patients in cardiogenic shock, but in the TTM study there was no difference in mortality among patients with mild shock on admission who were treated with a target temperature of 33°C compared with 36°C. Animal data also indicate improved contractile function with mild induced hypothermia probably because of increased Ca⁺⁺ sensitivity.

8. Prognostication

This section summarises the Advisory Statement on Neurological Prognostication in comatose survivors of cardiac arrest, written by members of the ERC ALS Working Group and of the Trauma and Emergency Medicine (TEM) Section of the European Society of Intensive Care Medicine (ESICM). For more detail and comprehensive referencing, see the European Advisory Statement.

Hypoxic-ischaemic brain injury is common after resuscitation from cardiac arrest. Two thirds of those dying after admission to ICU following OHCA die from neurological injury. Most of these deaths are due to active withdrawal of life sustaining treatment (WLST) based on prognostication of a poor neurological outcome. For this reason, when dealing with patients who are comatose after resuscitation from cardiac arrest, minimising the risk of a falsely pessimistic prediction is essential. Ideally, when predicting a poor outcome these tests should have 100% specificity or zero false positive rate (FPR), (i.e. no individuals should have a ‘good’ long-term outcome if predicted to have a poor outcome). However, most prognostication studies include so few patients that it is very difficult to be completely confident in the results. Moreover, many studies are confounded by self-fulfilling prophecy, which is a bias occurring when the treating physicians are not blinded to the results of the outcome predictor and use it to make a decision on WLST. Finally, both TTM itself and sedatives or neuromuscular blocking drugs used to maintain it may potentially interfere with prognostication tests, especially those based on clinical examination.

Prognostication of the comatose post-cardiac arrest patient should be multimodal, in other words involve multiple types of tests of brain injury, and should be delayed sufficiently to enable full clearance of sedatives and any neurological recovery to occur – in most cases, prognostication is not reliable until after 72 h from cardiac arrest. The tests are categorised:

- clinical examination – GCS score, pupillary response to light, corneal reflex, presence of seizures
- neurophysiological studies – somatosensory evoked potentials (SSEPs) and electroencephalography (EEG)
- biochemical markers – neuron-specific enolase (NSE) is the most commonly used
- imaging studies – brain CT and magnetic resonance imaging (MRI).

Clinical examination

Bilateral absence of pupillary light reflex at 72 h from ROSC predicts poor outcome with close to 0% FPR but the sensitivity is relatively low (about 19%); in other words, of those who eventually have a bad outcome, only 1 in 5 will have fixed pupils at 72 h. Similar performance has been documented for bilaterally absent corneal reflex.

An absent or extensor motor response to pain at 72 h from ROSC has a high (about 75%) sensitivity for prediction of poor outcome, but the FPR is also high (about 27%). The high sensitivity of this sign enables it to be used to identify the population with poor neurological status needing prognostication. The corneal reflex and the motor response can be suppressed by sedatives or neuromuscular blocking drugs. When interference from residual sedation or paralysis is suspected, prolonging observation of these clinical signs beyond 72 h from ROSC is recommended, in order to minimise the risk of obtaining false positive results.

Myoclonus is a clinical phenomenon consisting of sudden, brief, involuntary jerks caused by muscular contractions or inhibitions. A prolonged period of continuous and generalised myoclonic jerks is commonly described as status myoclonus. Although there is no definitive consensus on the duration or frequency of myoclonic jerks required to qualify as status myoclonus, in prognostication studies in comatose survivors of cardiac arrest the minimum reported duration is 30 minutes.

While the presence of myoclonic jerks in comatose survivors of cardiac arrest is not consistently associated with poor outcome (FPR 9%), a status myoclonus starting within 48 h from ROSC is consistently associated with a poor
outcome (FPR 0% [95% confidence interval (CI) 0–5%]; sensitivity 8–16%). However, several case reports of good neurological recovery despite an early-onset, prolonged and generalised myoclonus have been published. In some of these cases myoclonus persisted after awakening and evolved into a chronic action myoclonus (the Lance-Adams syndrome). The exact time when recovery of consciousness occurred in these cases may have been masked by the myoclonus itself and by ongoing sedation. Patients with post-arrest status myoclonus should be evaluated off sedation whenever possible; in those patients, EEG recording can be useful to identify EEG signs of awareness and reactivity and to reveal a coexistent epileptiform activity.

While predictors of poor outcome based on clinical examination are inexpensive and easy to use, they cannot be concealed from the treating team and therefore their results may potentially influence clinical management and cause a self-fulfilling prophecy.

Electrophysiology

Short-latency somatosensory evoked potentials (SSEPs)

In post-arrest comatose patients, bilateral absence of the N20 SSEP wave predicts death or vegetative state (CPC 4–5) with high reliability (FPR 0–2% with upper 95% CI of about 4%). The few cases of false reports observed in large patient cohorts were due mainly to artefacts. SSEP recording requires appropriate skills and experience, and utmost care should be taken to avoid electrical interference from muscle artefacts or from the ICU environment. In most prognostication studies bilateral absence of N20 SSEP has been used as a criterion for deciding on withdrawal of life-sustaining treatment (WLST), with a consequent risk of self-fulfilling prophecy.

Electroencephalography

Absence of EEG reactivity

Background reactivity means that there is a change in the EEG in response to a loud noise or a noxious stimulus such as tracheal suction. Absence of EEG background reactivity predicts poor outcome with a FPR of 0–2% (upper 95% CI of about 7%). However, most of the prognostication studies on absent EEG reactivity after cardiac arrest are from the same group of investigators. Limitations of EEG reactivity include lack of a standardised stimulus and modest inter-rater agreement.

Status epilepticus

In TTM-treated patients, the presence of status epilepticus (SE) (i.e. a prolonged epileptiform activity), is almost invariably – but not always – followed by poor outcome (FPR 0–6%), especially in presence of an unreactive or discontinuous EEG background.

Burst-suppression

Burst-suppression has recently been defined as more than 50% of the EEG record consisting of periods of EEG voltage <10µV, with alternating bursts. However, most of prognostication studies do not comply with this definition. In comatose survivors of cardiac arrest, burst-suppression is usually a transient finding. During the first 24–48 h after ROSC burst-suppression may be compatible with neurological recovery, while at ≥72 h from ROSC a persisting burst-suppression pattern is consistently associated with poor outcome.

Apart from its prognostic significance, recording of EEG, either continuous or intermittent, in comatose survivors of cardiac arrest both during TH and after rewarming is helpful to assess the level of consciousness – which may be masked by prolonged sedation, neuromuscular dysfunction or myoclonus – and to detect and treat non-convulsive seizures, which occur in about 25% of comatose survivors of cardiac arrest.

Biomarkers

NSE and S-100B are protein biomarkers that are released following injury to neurons and glial cells, respectively. Their blood values after cardiac arrest are likely to correlate with the extent of anoxic-ischaemic neurological injury and, therefore, with the severity of neurological outcome. Advantages of biomarkers over both EEG and clinical examination include quantitative results and likely independence from the effects of sedatives. Their main limitation as prognosticators is that it is difficult to find with a high degree of certainty a consistent threshold for identifying patients destined to a poor outcome. In fact, serum concentrations of biomarkers are per se continuous variables, which limits their applicability for predicting a dichotomous outcome, especially when a threshold for 0% FPR is desirable.
Neuron-specific enolase (NSE)

In TTM-treated patients the thresholds for 0% FPR varied between studies but were as high as 150 mcg L\(^{-1}\) at 24 and 48 h, and up to 80 mcg L\(^{-1}\) at 72 h. The main reasons for the observed variability in NSE thresholds include variation between different analysers, the presence of extra-neuronal sources of biomarkers (haemolysis and neuroendocrine tumours), and the incomplete knowledge of the kinetics of its blood concentrations in the first few days after ROSC. Limited evidence suggests that the discriminative value of NSE values at 48–72 h is higher than at 24 h. Increasing NSE values over time may have an additional value in predicting poor outcome.

Imaging

Brain CT

The main CT finding of global anoxic-ischaemic cerebral insult following cardiac arrest is cerebral oedema, which appears as a reduction in the depth of cerebral sulci (sulcal effacement) and an attenuation of the grey matter/white matter (GM/WM) interface, due to a decreased density of the GM, which has been quantitatively measured as the ratio (GWR) between the GM and the WM densities. The GWR threshold for prediction of poor outcome with 0% FPR in prognostication studies ranged between 1.10 and 1.22. The methods for GWR calculation were inconsistent among studies and, in any case, quantitative measurements are rarely made in clinical practice in the UK.

MRI

Brain MRI is more sensitive than CT for detecting global anoxic-ischaemic brain injury caused by cardiac arrest; however, its use can be problematic in the most clinically unstable patients. MRI can reveal extensive changes when results of other predictors such as SSEP are normal. All studies on prognostication after cardiac arrest using imaging have a small sample size with a consequent low precision, and a very low quality of evidence. Most of those studies are retrospective, and brain CT or MRI had been requested at the discretion of the treating physician, which may have caused a selection bias and overestimated their performance.

Suggested prognostication strategy

A careful clinical neurological examination remains the foundation for prognostication of the comatose patient after cardiac arrest. Perform a thorough clinical examination daily to detect signs of neurological recovery such as purposeful movements or to identify a clinical picture suggesting that brain death has occurred.

The process of brain recovery following global post-anoxic injury is completed within 72 h from arrest in most patients. However, in patients who have received sedatives ≤12 h before the 72 h post ROSC neurological assessment, the reliability of clinical examination may be reduced.\(^{77}\) Before decisive assessment is performed, major confounders must be excluded;\(^{134,135}\) apart from sedation and neuromuscular blockade, these include hypothermia, severe hypotension, hypoglycaemia, and metabolic and respiratory derangements. Suspend sedatives and neuromuscular blocking drugs for long enough to avoid interference with clinical examination. Short-acting drugs are preferred whenever possible. When residual sedation/paralysis is suspected, consider using antidotes to reverse the effects of these drugs.

The prognostication strategy algorithm (Figure 2) is applicable to all patients who remain comatose with an absent or extensor motor response to pain at ≥72 h from ROSC. Results of earlier prognostic tests are also considered at this time point.

Evaluate the most robust predictors first. These predictors have the highest specificity and precision (FPR <5% with 95% CIs <5% in patients treated with controlled temperature) and have been documented in >5 studies from at least three different groups of investigators. They include bilaterally absent pupillary reflexes at ≥72 h from ROSC and bilaterally absent SSEP N20 wave after rewarming (this last sign can be evaluated at ≥24 h from ROSC in patients who have not been treated with controlled temperature). Based on expert opinion, we suggest combining the absence of pupillary reflexes with those of corneal reflexes for predicting poor outcome at this time point. Ocular reflexes and SSEPs maintain their predictive value irrespective of target temperature.\(^{136,137}\)

If none of the signs above is present to predict a poor outcome, a group of less accurate predictors can be evaluated, but the degree of confidence in their prediction will be lower. These have FPR <5% but wider 95% CIs than the previous predictors, and/or their definition/threshold is inconsistent in prognostication studies. These predictors include the presence of early status myoclonus (within 48 h from ROSC), high values of serum NSE at 48–72 h after ROSC, an unreactive malignant EEG pattern (burst-suppression, status epilepticus) after rewarming, the presence of a marked reduction of the GWR or sulcal effacement on brain CT within 24 h after ROSC or the presence of diffuse ischaemic changes on brain MRI at 2–5 days after ROSC. Based on expert opinion, we suggest waiting at least 24 h...
after the first prognostication assessment and confirming unconsciousness with a Glasgow motor score of 1–2 before using this second set of predictors. We also suggest combining at least two of these predictors for prognostication.

No specific NSE threshold for prediction of poor outcome with 0% FPR can be recommended at present. Ideally, every hospital laboratory assessing NSE should create its own normal values and cut-off levels based on the test kit used. Sampling at multiple time-points is recommended to detect trends in NSE levels and to reduce the risk of false positive results. Avoid haemolysis when sampling NSE.

Although the most robust predictors showed no false positives in most studies, none of them singularly predicts poor outcome with absolute certainty when the relevant comprehensive evidence is considered. Moreover, those predictors have often been used for WLST decisions, with the risk of a self-fulfilling prophecy. For this reason, we recommend that prognostication should be multimodal whenever possible, even in presence of one of these predictors. Apart from increasing safety, limited evidence also suggests that multimodal prognostication increases sensitivity.

When prolonged sedation and/or paralysis is necessary, for example, because of the need to treat severe respiratory insufficiency, we recommend postponing prognostication until a reliable clinical examination can be performed. Biomarkers, SSEP and imaging studies may play a role in this context, since they are insensitive to drug interference.

When dealing with an uncertain outcome, clinicians should consider prolonged observation. Absence of clinical improvement over time suggests a worse outcome. Although awakening has been described as late as 25 days after arrest, most survivors will recover consciousness within one week.
9. Rehabilitation

Although neurological outcome is considered to be good for the majority of cardiac arrest survivors, cognitive and emotional problems and fatigue are common. Long-term cognitive impairments are present in half of survivors. Memory is most frequently affected, followed by problems in attention and executive functioning (planning and organisation). The cognitive impairments can be severe, but are mostly mild. Mild cognitive problems are often not recognised by health care professionals and cannot be detected with standard outcome scales such as the Cerebral Performance Categories (CPC) or the Mini-Mental State Examination (MMSE). Emotional problems, including depression, anxiety and posttraumatic stress are also common. Both cognitive and emotional problems have significant impact and can affect a patient’s daily functioning, return to work and quality of life. There is some evidence that follow-up care and rehabilitation after hospital discharge can improve outcome after cardiac arrest.
10. Organ donation

Organ donation should be considered in those who have achieved ROSC and who fulfil criteria for death using neurological criteria. In those comatose patients in whom a decision is made to withdraw life-sustaining therapy, organ donation should be considered after circulatory death occurs.

Non-randomised studies have shown that graft survival at one year is similar from donors who have had CPR compared with donors who have not had CPR.

Organ retrieval from donation after circulatory death (DCD) donors is classified as controlled or uncontrolled. Controlled donation occurs after planned withdrawal of treatment following non-survivable injuries and illnesses. Uncontrolled donation describes donation from patients with unsuccessful CPR in whom a decision has been made that CPR should be stopped. Uncontrolled donation is not yet practised widely in the UK. Once death has been diagnosed, the assessment of which includes a pre-defined period of observation to ensure a spontaneous circulation does not return, organ preservation and retrieval takes place.

11. Screening for inherited disorders

Many sudden death victims have silent structural heart disease, most often coronary artery disease, but also primary arrhythmia syndromes, cardiomyopathies, familial hypercholesterolaemia and premature ischaemic heart disease. Screening for inherited disorders is crucial for primary prevention in relatives as it may enable preventive antiarrhythmic treatment and medical follow-up. This screening should be performed using clinical examination, electrophysiology and cardiac imaging in specialist centres. In selected cases, genetic mutations associated with inherited cardiac diseases should also be searched.

12. Cardiac arrest centres

There is wide variability in survival among hospitals caring for patients after resuscitation from cardiac arrest. Many studies have reported an association between survival to hospital discharge and transport to a cardiac arrest centre but there is inconsistency in the hospital factors that are most related to patient outcome. There is also inconsistency in the services that together define a cardiac arrest centre. Most experts agree that such a centre must have a cardiac catheterisation laboratory that is immediately accessible 24/7 and the facility to provide targeted temperature management. The availability of a neurology service that can provide neuroelectrophysiological monitoring EEG and investigations (e.g. EEG and SSEPs) is also essential.

There is indirect evidence that regional cardiac resuscitation systems of care improve outcome after ST elevation myocardial infarction (STEMI). The implication from all these data is that specialist cardiac arrest centres and systems of care may be effective. Despite the lack of high quality data to support implementation of cardiac arrest centres, it seems likely that post-cardiac arrest care in the UK will become increasingly regionalised.

13. Acknowledgements

These guidelines have been adapted from the European Resuscitation Council 2015 Guidelines. We acknowledge and thank the authors of the ERC Guidelines for Post-resuscitation care: Jerry P. Nolan, Jasmeet Soar, Alain Cariou, Tobias Cronberg, Véronique R.M. Moulaert, Charles D. Deakin, Bernd W. Bottiger, Hans Friberg, Kjetil Sunde, Claudio Sandroni.
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# Prevention of cardiac arrest and decisions about CPR

1. The guideline process
2. Introduction
3. Prevention of out-of-hospital cardiac arrest
4. Prevention of in-hospital cardiorespiratory arrest
5. Resuscitation decisions
6. Recommended strategies for the prevention of avoidable cardiac arrests and inappropriate CPR attempts
7. References

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

## 2. Introduction

Prevention of cardiac arrest is the first link in the Chain of Survival.\(^3\) This section of the Resuscitation Council (UK) guidelines stresses the importance of preventing cardiac arrest in all age groups, and the decision-making process when cardiopulmonary resuscitation (CPR) is inappropriate. This update is based on the European Resuscitation Council Guidelines 2015,\(^4\) and includes updates based on NICE Clinical Guideline 50,\(^5\) and the guidance from the British Medical Association (BMA), Resuscitation Council (UK), and the Royal College of Nursing (RCN) on decisions relating to CPR.\(^6\) The General Medical Council publication, 'Treatment and care towards the end of life: good practice in decision making', also includes advice on decisions relating to CPR.\(^7\)

## 3. Prevention of out-of-hospital cardiac arrest

**Recognising and responding to cardiac chest pain**

Most sudden cardiac death (SCD) victims have a history of heart disease and warning symptoms, most commonly chest pain, in the hour before cardiac arrest.\(^8\) Early recognition of cardiac chest pain and rapid activation of the EMS
is vitally important. When a call to the EMS is made before a cardiac arrest victim collapses, the ambulance arrives significantly sooner after collapse, and the chance of survival is higher. Prompt assessment of people with acute chest pain by the EMS, including recording and interpretation of a 12-lead ECG, enables appropriate treatment of acute coronary syndromes with a minimum of delay (especially reperfusion therapy, usually by primary percutaneous coronary intervention [PPCI] for ST-segment myocardial infarction [STEMI]), reducing the risk of early cardiac arrest and death and of subsequent complications, including death.11-13

Recognising and responding to other causes of cardiac arrest and sudden death

Coronary artery disease is the commonest cause of SCD in people over the age of 35 years.8,14 Other causes of SCD include cardiomyopathies, valve disease, inherited ion channel disorders (e.g. long and short QT syndromes, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia) and congenital heart disease.8,15 Whilst cardiac arrest and SCD are relatively uncommon in people younger than 35, coronary disease is also less common in this age group, so an inherited condition is more likely to be the cause when a younger person suffers unexpected cardiac arrest or SCD.

In patients known to have heart disease, syncope (with or without prodrome – particularly recent or recurrent) is an independent risk factor for increased risk of death.8 Apparently healthy children and young adults who suffer SCD may also have symptoms and signs (e.g. syncope/pre-syncope, chest pain, palpitation, heart murmur) that should alert healthcare professionals to seek expert help to prevent cardiac arrest in those at risk. Features that indicate a high probability of arrhythmic syncope (and potential risk of SCD) include:

- syncope in the supine position
- syncope occurring during or after exercise (although syncope after exercise is often vasovagal)
- syncope with no or only brief prodromal symptoms
- repeated episodes of unexplained syncope
- syncope in individuals with a family history of sudden death or inherited cardiac condition.

Assessment in a clinic specialising in the care of those at risk of SCD is recommended in family members of young victims of SCD or those with a cardiac disorder resulting in an increased risk of SCD.16 Specific and detailed guidance on the care of individuals with transient loss of consciousness is available from the National Institute for Health and Clinical Excellence (NICE).17

4. Prevention of in-hospital cardiorespiratory arrest

Rates of survival and complete physiological recovery following in-hospital cardiac arrest are poor in all age groups. For example, fewer than 20% of adult patients having an in-hospital cardiac arrest (IHCA) will survive to go home.18 Cardiac arrest is rare in both pregnant women and children, but outcomes in these groups after in-hospital arrest are also poor. Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help and an effective response.19

Adults

Most adult survivors of in-hospital cardiac arrest have a witnessed and monitored ventricular fibrillation (VF) arrest and are defibrillated immediately.20 The underlying cause of arrest in this group is usually myocardial ischaemia. In comparison, cardiac arrest in patients in unmonitored ward areas is usually a predictable event not caused by heart disease.21 In this group, cardiac arrest often follows a period of slow and progressive physiological deterioration involving unrecognised or inadequately treated hypoxaemia and hypotension.22 The cardiac arrest rhythm is usually asystole or PEA, and the chance of survival to hospital discharge is extremely poor unless a reversible cause is identified and treated immediately.

Regular monitoring and early, effective treatment of seriously ill patients appear to improve clinical outcomes and prevent some cardiac arrests. However, the quality of monitoring of vital signs may vary and may differ between day and night.23 Closer attention to patients who sustain a ‘false’ cardiac arrest (i.e. a cardiac arrest call to a patient who does not require basic or advanced life support) may also improve outcome, as up to one third of these patients die during their hospital stay.24 Data from the USA suggest that hospitals with the lowest incidence of IHCA have the highest cardiac arrest survival, suggesting better selection of candidates for CPR or better prevention of cardiac arrest or both.25
Deficiencies in acute care

Analysis of the critical events preceding many adult cardiac arrests demonstrates many significant antecedents, usually related to abnormalities of the airway, breathing, and circulation. Additional factors include a failure to use a systematic approach to the assessment of critically ill patients, poor communication, lack of teamwork, and insufficient use of treatment escalation plans.

Hospital processes may also have significant effects on patient outcome. For example, patients who are transferred from intensive care units (ICUs) to general wards at night have an increased risk of in-hospital death compared with those transferred during the day and those transferred to high-dependency units. Higher nurse-patient staffing ratios are also associated with lower cardiac arrest rates and lower rates of pneumonia, shock, and death. These suggest that adequate patient monitoring and assessment are crucial to preventing adverse outcomes.

Recognition of ‘at-risk’, or critically ill, adult patients

When patients deteriorate, they display common signs that represent failing respiratory, cardiovascular, and nervous systems. This is the basis for monitoring patients’ vital signs. Abnormal physiology is common on general wards, yet the important physiological observations of sick patients are measured and recorded less frequently than is desirable. To assist in the early detection of critical illness, every patient should have a documented plan for vital-signs monitoring that identifies which variables need to be measured and the frequency of measurement.

In recent years, early warning scores (EWS), or ‘calling-criteria’ have been adopted by many hospitals to assist in the early detection of critical illness. EWS systems allocate points to routine vital-sign measurements on the basis of their deviation from an arbitrarily agreed ‘normal’ range. The weighted score of one or more total EWS is used to alert ward staff or critical care outreach teams to the deteriorating condition of the patient. Systems that incorporate ‘calling criteria’ activate a response when one or more routinely measured physiological variables reach a pre-defined abnormal value.

The sensitivity, specificity, and accuracy of EWS or calling-criteria systems to identify sick patients have been validated for several outcomes. Several studies have identified abnormalities of heart rate, blood pressure, respiratory rate, and conscious level as possible markers of impending critical events. The ability of these systems to predict cardiac arrest remains less than for other outcomes such as death or unanticipated ICU admission. In the UK, the National Early Warning Score (NEWS) is recommended. Gaps in vital-sign data recording are common; the use of EWS, calling criteria and rapid response systems can increase the completeness of vital sign monitoring. Simpler systems may have advantages over more complex ones. EWS may be better discriminators than calling-criteria systems. Nurse concern may also be an important predictor of patient deterioration.

The clinical response

The medical and nursing response to a patient’s abnormal physiology must be both appropriate and speedy, yet this is not always the case. Traditionally, the response to cardiac arrest has been reactive, with a cardiac arrest team attending the patient after the cardiac arrest. The use of such teams appears to improve survival in circumstances where no coordinated response to cardiac arrest existed previously. However, their impact in other settings is questionable. For example, in one study only patients who had return of spontaneous circulation (ROSC) before the arrival of the cardiac arrest team survived to leave hospital. In some hospitals the role of the cardiac arrest team has been incorporated into that of the medical emergency team (MET). The MET responds not only to cardiac arrests, but also to patients with acute physiological deterioration. The MET usually comprises medical and nursing staff from intensive care and general medicine and responds to specific calling criteria. MET interventions often involve simple tasks such as starting oxygen therapy and intravenous fluids.

The results of research into the benefits of introducing a MET are variable, although evidence for their benefit is increasing. Studies with historical control groups show a reduction in cardiac arrests, deaths and unanticipated intensive care unit admissions, improved detection of medical errors, treatment-limitation decisions, and reduced postoperative ward deaths. A cluster randomised controlled trial of the MET system was unable to demonstrate a reduction in the incidence of cardiac arrest, unexpected death, or unplanned ICU admission.

In the UK, a system of pre-emptive ward care, based predominantly on individual or teams of nurses known as critical care outreach, has developed. Although the data on the effects of outreach care are also inconclusive, it has been suggested that outreach teams may reduce ward deaths, postoperative adverse events, ICU admissions and readmissions, and increase survival.

The role of education in cardiac arrest prevention

The recognition that many cardiac arrests may be preventable has led to the development of postgraduate courses...
specifically designed to prevent physiological deterioration, critical illness, and cardiac arrest (e.g. Acute Life Threatening Events – Recognition and Treatment: ALERT).\textsuperscript{47} Early evidence suggests that they can improve knowledge and change attitudes about acute care. The Immediate Life Support (ILS) and Advanced Life Support (ALS) courses also include sections related to arrest prevention. Implementation of the ILS course is associated with a reduced incidence of cardiac arrest.\textsuperscript{48} Simulation is being used increasingly to train staff in the prevention of patient deterioration. Rapid response teams, such as METs, have a role in educating and improving acute care skills of ward personnel.\textsuperscript{49,50}

**Pregnant patients**

Past reports of the triennial Confidential Enquiry into Maternal and Child Health (CEMACH) have made recommendations for preventing deaths associated with pregnancy, including the need for hospitals to implement, audit, and regularly update multidisciplinary guidelines for the management of women who are at risk of, or who develop, complications in pregnancy. Other recommendations include the development of clinical protocols and local referral pathways, including patient transfer, for pregnant women with pre-existing medical conditions, a history of psychiatric illness, and serious complications of pregnancy (sepsis, pre-eclampsia and eclampsia, obstetric haemorrhage). Maternity teams should be trained to recognise and manage medical emergencies, and to demonstrate their competency in scenario-based training using simulation. CEMACH recommends the routine use of a national modified early obstetric warning score (MEOWS) chart in all pregnant or postpartum women in all hospital settings.\textsuperscript{51} Outreach services for maternity have also been described elsewhere.\textsuperscript{52,53}

**Children**

In children, cardiorespiratory arrest is more often caused by profound hypoxaemia and/or hypotension than by heart disease. Ventricular fibrillation is less common than asystole or pulseless electrical activity. As with adults, there may be opportunities to introduce strategies that will prevent arrest.

There is already evidence of marked, often untreated, abnormalities of common vital signs in the 24 hours prior to the admission of children to an ICU, similar to those reported in adults.\textsuperscript{54} Recognition of the seriously ill child requires determination of the normal and abnormal age-related values for vital signs, and then measuring and reassessing vital signs in the context of the progression of the individual child’s condition. As in adults, serial measurement of heart rate, respiratory rate, temperature, blood pressure, and conscious level, particularly following any clinical intervention, must be performed and acted upon. Intervention at an early stage in an unwell child reduces significantly the risk of developing irreversible shock. Systemic blood pressure decreases at a late stage in shock in the child compared with the adult, and should not be used as the sole determinant of whether or not treatment is required.

Paediatric METs, responding to early warning scores, have been established in some hospitals, but as in adult studies, their impact is difficult to measure. However, there is some evidence that they reduce the incidence of deterioration and cardiac arrest.\textsuperscript{55,56}

**5. Resuscitation decisions**

**Why decisions about CPR are needed**

CPR was originally developed to save the lives of people dying unexpectedly when acute myocardial infarction (AMI) caused sudden cardiac arrest in ventricular fibrillation – ‘hearts too good to die’.\textsuperscript{57} As awareness of CPR increased and resuscitation equipment became more widely available and more portable, attempts at CPR became very common in situations other than a sudden cardiac arrest due to AMI. When a person dies the heartbeat and breathing cease, so the distinction between cardiorespiratory arrest that is sudden and unexpected and cardiorespiratory arrest that occurs in the context of death from an advanced and irreversible cause is not always made. As a result CPR has been attempted commonly in people who are gravely ill, and for whom attempts to re-start their heart either would not work (subjecting them to violent physical treatment at the end of their life and depriving them of a dignified death) or might restore their heart function for a brief period and possibly subject them to a further period of suffering from their underlying terminal illness (prolonging the dying process without prolonging life). A study by the National Confidential Enquiry into Patient Outcome and Death found that CPR was attempted in hospitals in many people for whom there was little or no likelihood of benefit, yet no anticipatory decision had been considered or made about CPR.\textsuperscript{58}

The need for anticipatory decisions arises because those present at the time of death or cardiac arrest must make an immediate decision whether or not to start CPR. Any delay will reduce the chance of success in those for whom CPR
may be beneficial, as emphasised elsewhere in these guidelines. As those present may not know or have instant access to full details of the patient’s circumstances there is a presumption that CPR will be attempted when someone suffers cardiorespiratory arrest or dies, unless there is a clearly recorded decision not to do so. Many healthcare provider organisations have a policy requiring their staff to attempt CPR in such circumstances.

When to consider making decisions about CPR

Recognition of an ‘at-risk’ or critically ill/deteriorating patient should trigger consideration of whether or not attempted CPR would be successful and/or in the patient’s best interests. Critical care outreach and medical emergency teams may contribute to a ‘reduction’ in cardiorespiratory arrests by triggering such consideration and thereby avoiding inappropriate CPR attempts.\(^{59-65}\) However, a crisis situation when someone is acutely unwell and has been admitted to hospital, is not the optimal time to make anticipatory decisions about CPR for most people who have advanced medical conditions and are approaching the end of life. Early consideration of CPR decisions is recommended in the context of broader ‘advance care planning’ (see ‘Discussing decisions about CPR’ below).

Situations where a decision about CPR should be considered

Decisions about CPR should be considered, discussed and recorded:

- At the request of a person with capacity.
- As an important element of end-of-life care for a person who is terminally ill from an advanced and irreversible disease.
- As an important element of care of a patient with an acute severe illness, who continues to deteriorate towards death despite all appropriate treatment or who has suffered a sudden catastrophic event from which no recovery can be reasonably expected.
- As an element of care of people recognised by healthcare professionals as approaching the end of their lives (i.e. within the last year of life).

Making lawful decisions about CPR

Healthcare professionals must follow a good decision-making process that complies with all relevant legislation, including laws relating to capacity, discrimination and human rights.\(^{6,66-69}\) A report of a confidential inquiry into premature deaths of people with learning disabilities found evidence of poor compliance with capacity legislation and poor adherence to guidance on CPR decision-making, resulting in decisions that appeared to discriminate against people with learning disabilities.\(^{70}\) The courts have stated that there should be a presumption that the patient will be involved in decisions about CPR unless discussions about CPR would cause the patient physical or psychological harm. When a do-not-attempt-cardiopulmonary resuscitation (DNACPR) decision is made by clinicians because CPR would not be successful, it is expected that the patient would have the decision and the reasons for it explained to them. Patients and those close to them cannot demand treatment that is clinically inappropriate. However clinicians must not make judgements about a person’s quality of life based on their own perceptions of what would be acceptable. Where there is some chance that CPR may be successful the aim should be to provide the patient and those close to them with information to enable their participation with their healthcare team in a shared decision about whether or not CPR will be attempted, and in what circumstances.

Some patients will have the capacity to participate in the decision-making process, but a substantial number will not. When a person does not have capacity to participate in the decision-making process it is the responsibility of the senior responsible clinician to make decisions about CPR, unless the patient has recorded their wishes in an Advance Decision to Refuse Treatment or has a representative with legal authority (e.g. Power of Attorney) to make such decisions on the patient’s behalf. Where there is a chance that CPR may be successful in restarting the heart and breathing for a sustained period any decision must be made in the patient’s best interests. Whenever possible this should take into account the views of the patient’s family or others close to the patient. Care should be taken to ensure that they understand that they are not being asked to make the decision and (unless they have been given specific legal authority) have no power to make the decision; their role is to help the senior clinician to make the best decision.

Discussing decisions about CPR

Both patients and healthcare professionals find discussions and decisions that focus purely on withholding CPR difficult. They prefer an approach that considers whether or not CPR would be appropriate for the individual person, and contextualises that decision within a broader plan of the person’s wishes regarding other elements of their care and treatment.\(^{71}\)
When possible, this is often best done in the person’s usual home setting, involving one or more healthcare professionals who know the patient well. Discussions about CPR should be undertaken by professionals who have the knowledge and communication skills to support the patient in making an informed decision. Discussions should be supported where possible by information in written or other formats.

Some patients with capacity will choose to discuss and engage in decisions about their end-of-life care whilst others will choose not to do so, and such wishes should be respected.

Recording decisions about CPR

All considerations, discussions and decisions about CPR must be recorded fully and clearly, together with details of the reasons for any decision. Such decisions should also be communicated clearly, where necessary in writing, to all those involved in the patient’s care. It is recommended that decisions about CPR (and about other elements of emergency care and treatment) are recorded on a standard form that crosses geographical and organisational boundaries and is recognised/accepted by all organisations and individuals involved in a person’s care. Such a form should remain close to the patient at all times and be kept in a place where it will be accessible immediately by anyone needing to refer to it in an emergency. Where electronic records are used to generate and store documented decisions about CPR the systems used must be accessible immediately by all those who may need to see the records, must be secure and must be responsive to any reversal of the recorded decision.

Reviewing decisions about CPR

Just as every hospital patient should have a plan detailing their individual needs for type and frequency of vital-sign measurement, so every person with a CPR decision should have a recorded plan detailing their individual need for review of that decision. For a person with an advanced, irreversible condition who is close to the end of their life there may be no need for review of a DNACPR decision. In contrast, when a person is being treated for a severe, acute, life-threatening illness a decision to attempt CPR in the event of cardiac arrest should be reviewed frequently and may warrant changing if they deteriorate or fail to respond to treatment. Similarly a DNACPR decision, made when they are critically ill and highly unlikely to survive cardiac arrest, should be reconsidered frequently so that it can be reversed if appropriate should they respond to treatment and may benefit from and wish to have CPR in the event of cardiac arrest.

Recorded decisions about CPR should be reviewed:

- if the patient requests review
- if those close to the patient request review
- whenever there is a significant change in the patient’s clinical condition
- when the patient moves from one care setting to another (including transfer between wards or teams in a hospital).

Applying decisions about CPR

A recorded decision not to undertake CPR refers only to CPR and not to any other element of a person’s care and treatment. There is evidence of substantial misunderstanding of this by healthcare professionals and by the public. Health professionals must ensure that a DNACPR decision does not compromise any other aspect of a patient’s care and treatment.

Decisions about CPR for children and young people

The ethical principles that underpin decisions about CPR for children and young people are no different from those in adults. Whenever possible, decisions should be made within a supportive partnership that includes patients, parents and healthcare professionals. As with adults, it may be best to make such decisions in the context of a broader decision-making framework. The degree of involvement of the patient varies, as the ability of a child or young person to engage with and contribute to the decision-making process may change quite rapidly. Involvement of those with parental responsibility is usually crucial, but can present difficulties if a child or young person does not want parental involvement, or if the parental view differs from the wishes of the child or young person themselves. Detailed guidance on making decisions to limit treatment in children has been published by the Royal College of Paediatrics and Child Health.

Policies and clinical audit of CPR decisions

Each hospital should have a policy on CPR decisions, based on current national guidance. Each hospital should
undertake on-going clinical audit of adherence to that policy and of the standards achieved with both consideration and recording of decisions about CPR. All hospitals should participate in the National Cardiac Arrest Audit.

6. Recommended strategies for the prevention of avoidable cardiac arrests and inappropriate CPR attempts

1. Ensure that people with symptoms suggestive of acute coronary syndromes are assessed and treated appropriately without delay.

2. In particular ensure that people with STEMI receive reperfusion therapy without delay, wherever possible by timely primary percutaneous coronary intervention (PPCI).

3. Ensure that people with symptoms that may indicate a risk of cardiac arrest and SCD (e.g. unexplained syncope) receive prompt assessment that includes a 12-lead ECG and, where appropriate, are referred for prompt assessment by a heart rhythm specialist.

4. Ensure that people with conditions that may indicate a risk of cardiac arrest and SCD (e.g. complete heart block, severe left ventricular impairment, severe aortic stenosis, hypertrophic cardiomyopathy) receive prompt specialist assessment and appropriate treatment.

5. In hospitals, place critically ill patients and those at risk of rapid deterioration in areas where the level of care is matched to the seriousness of each patient’s condition.

6. Monitor such patients regularly using simple vital-sign observations (e.g. pulse and/or heart rate, blood pressure, respiratory rate, conscious level, temperature and SpO₂).

7. Match the frequency and type of observations to the severity of illness of the patient.

8. Use an EWS system or ‘calling criteria’ to identify patients who are critically ill, at risk of clinical deterioration or cardiorespiratory arrest, or both. In the UK, the National Early Warning Score (NEWS) is recommended.

9. For each patient use a vital-signs chart that encourages and permits the regular measurement and recording of vital signs and of EWS where used, and that also facilitates early recognition of and response to patient deterioration.

10. Ensure that the hospital has a clear policy that requires a timely, appropriate, clinical response to deterioration in a patient’s clinical condition.

11. Introduce into each hospital a clearly identified system for response to critical illness. This will vary between sites, but may include an outreach service or resuscitation team (e.g. MET) capable of responding to acute clinical crises. The service must be available 24 hours per day. An EWS should be used to trigger calls to this team.

12. Ensure that all clinical staff are trained in the recognition, monitoring, and management of critically ill patients, and that they know their role in the rapid response system.

13. Empower staff to call for help when they identify a patient at risk of deterioration or cardiorespiratory arrest.

14. Use a structured communication tool to ensure effective handover of information between staff (e.g. SBAR – Situation-Background-Assessment-Recommendation).

15. Ensure that all policies on CPR decisions are based on current national guidance, and ensure that all clinical personnel understand it.

16. Identify those fully informed patients who do not wish to receive CPR, those patients for whom cardiorespiratory arrest is an anticipated terminal event and for whom CPR would be inappropriate, and those patients who have lost capacity in whom a decision not to attempt CPR is in their best interests.

17. Audit all cardiac arrests, ‘false arrests’, unexpected deaths and unanticipated ICU admissions, using a common dataset. Audit the antecedents and clinical responses to these events.

18. Ensure that all hospitals participate in the National Cardiac Arrest Audit to obtain feedback on each hospital’s individual performance in comparison to others, allowing identification of opportunities for improvement.

19. Audit adherence to each hospital’s policy on CPR decisions and to the adequacy of consideration and recording of decisions about CPR.
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# Peri-arrest arrhythmias

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- The involvement of stakeholders from around the world including members of the public and cardiac arrest survivors.
- These Resuscitation Council (UK) Guidelines have been peer reviewed by the Executive Committee of the 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.  

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\(^+\), Mg\(^{2+}\), Ca\(^{2+}\)).

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).

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 repeat 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](http://resus.org.uk/_resources/assets/attachment/full/0/6477.pdf)

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 arrhythmias with ventricular rate <150 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 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 900 mg over 24 h.

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
- AV re-entry tachycardia (AVRT) – due to WPW syndrome
- atrial flutter with regular AV conduction (usually 2:1).

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\textsubscript{2}DS\textsubscript{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\textsuperscript{-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, hyperkalaemia)
- drugs (e.g., beta-blockade, diltiazem, digoxin, amiodarone) in therapeutic use or overdose.

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.
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.
Pacing

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. References


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

2. Summary of recommendations in education and implementation and teams

Training

- All school children should be taught how to perform CPR and should be made aware of how to use an automated external defibrillator (AED).
- Ambulance Services should have access to a national database of AEDs and their dispatchers should have specific training in how to provide clear and effective instructions to rescuers over the telephone.
- We suggest frequent ‘low-dose’ training may be a beneficial method for providing CPR/AED retraining.
- The outcomes for candidates attending an e-ALS course are the same as those attending a conventional 2-day ALS course.
- High-fidelity manikins are not essential for life support courses.
- Life support courses should incorporate training in non-technical skills (e.g. leadership, team behaviour and
Implementation

- Healthcare systems should evaluate their processes to ensure those with a cardiac arrest have the best outcomes.
- There may be a role for regional cardiac arrest centres, although further work is needed to identify which specific aspects of care are beneficial.
- Teams who manage patients in cardiac arrest should use data-driven performance-focused debriefing.
- Social media and innovative technology have vital roles to play in improving outcomes from cardiac arrest.

3. Introduction

The Mission Statement of the Resuscitation Council (UK) states that it exists to promote high quality, scientific, resuscitation guidelines that are applicable to everybody, and to contribute to saving life through education, training, research and collaboration. The theme of ‘Training people, saving lives’ demonstrates the importance of education in the pursuit of improved outcomes from cardiac arrest.

Similarly, the ‘Formula for Survival’ builds upon the ‘Chain of Survival’ to emphasise the importance of education and also implementation.\(^3,4\) The clinical guidelines tell us what we should be doing according to the latest evidence available to us. The next challenge is to convey this knowledge cost effectively. For this reason, the structure and efficacy of resuscitation courses and other innovative vectors for delivery of education have been subjected to the same rigorous evaluation process as the clinical guidelines.

Finally, healthcare systems at all levels need to be able to implement these new guidelines. It has been stated, “it takes a system to save lives” [www.resuscitationacademy.com](http://www.resuscitationacademy.com). With this in mind, policy makers at local, regional and national levels have a vital role to play in enabling us to train people with these new guidelines and ultimately save lives.

4. Basic life support training

Who to train

Swift bystander CPR and rapid access to defibrillation are vital for successful outcomes from cardiac arrest. Evidence from overseas has shown that training all school children in CPR can dramatically improve bystander CPR rates and survival.\(^5\) This model has the benefit that all members of society, in time, are primed with these essential life saving skills. It has the added benefit that both school children and teachers have been shown to further cascade their learning to family members and friends.\(^6\) The basic concepts of recognition of a person in cardiac arrest and calling for help can be taught to primary school children. Once children reach secondary school, they are physically able to perform CPR and this is therefore an ideal age to teach them these skills. Finally, school children can be educated about the benefit of AEDs as there is a need to improve awareness of their existence and use.\(^7\)

The ambulance services also have a vital role to play in achieving improved bystander CPR rates and rapid use of defibrillators. They should have access to a national database of AEDs and their dispatchers should have specific training in how to provide clear and effective instructions to rescuers over the telephone. This should include emphasis on the identification of agonal breathing and also the importance of seizures as an aspect of cardiac arrest.\(^8\)

How to train

Training must be tailored to the requirements of the learner and kept as simple as possible. Traditional training packages for both lay and healthcare rescuers have focused on face-to-face training with an instructor, although evidence is emerging that the use of self-directed learning and digital media may be as effective either as a replacement or with reduced face-to-face time.\(^9,11\) Those who are expected to perform CPR regularly will also benefit from training in non-technical skills (e.g. communication and team behaviours).\(^12,13\)
The optimal intervals for CPR/AED retraining are not known and will differ according to the characteristics of the learner (e.g. lay or healthcare). It is widely accepted that skills decay within three to six months after initial training. Frequent “low-dose” training may improve CPR skills compared with conventional training strategies.

5. Advanced level training

Life support courses cover the knowledge, skills and attitudes needed for membership and/or leadership of a resuscitation team. There are a variety of courses covering newborn, paediatric, and adult cardiac arrest as well as courses focusing upon trauma, obstetrics, and specified skills such as ultrasound. There are courses designed to train instructors in the various provider courses. These courses are constantly evaluated and updated.

The Immediate Life Support (ILS) course provides training in the prevention and management of adult cardiac arrest. It is primarily targeted at first responders. Its implementation has been associated with a reduction in the number of in-hospital cardiac arrests and unsuccessful CPR attempts.

The Advanced Life Support (ALS) course is designed for healthcare professionals who would be expected to apply the skills as part of their clinical duties as a member or leader of a resuscitation team. Many components of the course have been formally evaluated (e.g. testing scenarios, precourse learning, non-technical skill teaching). There is good evidence that a blended learning course comprising e-learning and reduced face-to-face time (e-ALS) has equally good outcomes as the traditional two-day ALS course.

Whilst high-fidelity manikins provide greater physical realism and are popular with learners, they are expensive and their use is not essential for life support courses. Their use may deliver slight improvements in training outcome on skill performance at the end of courses, but there is otherwise no proven benefit.

All life support courses should include training in non-technical skills. These include situational awareness, communication, team behaviours and leadership skills. These are all vital elements to the successful approach to cardiac resuscitation.

6. Implementation

Systems

All healthcare systems should evaluate their processes to ensure that they are achieving the best possible outcomes from cardiac arrest. The National Cardiac Arrest Audit (NCAA) provides valuable data for participating organisations to benchmark their performance. An out-of-hospital registry is being developed to enable ambulance services to evaluate their performance as well.

Healthcare organisations have an obligation to provide a high quality resuscitation service, and to ensure that staff are trained and updated sufficiently regularly to ensure that they are proficient in resuscitation in relation to their expected role (Resuscitation Council (UK) Quality Standards for CPR practice and training).

Debriefing following resuscitation in the clinical setting

Teams who manage patients in cardiac arrest should use data-driven performance-focused debriefing, as its use has been shown to improve performance.

Social media and innovative technology

Social media has a vital role to play in improving the outcomes from cardiac arrest. It can be used to disseminate awareness and education of the subject to vast audiences. Social media is also a powerful tool for effecting change.
It can be used to engage support for concepts that can then be used to lobby decision makers.

Innovative technology falls into several categories:

1. Simple delivery of information – apps that display resuscitation algorithms (e.g. iResus).
2. Interactive delivery of information – apps that use the geolocation of the user to display the location of the nearest AED.
3. Interactive delivery of education – apps that engage with the user and create an immersive and interactive means of educating the user (e.g. Lifesaver) www.life-saver.org.uk.
4. Feedback devices – real time use of the accelerometer to improve rate, depth of compressions as well as recording data for debriefing.28
5. Notification and activation of bystander schemes – if individuals are willing and able to provide basic life support in a community, the use of these systems may lead to faster response times when compared with emergency service attendance.29,30

The use of technology for the implementation of resuscitation guidelines is constantly evolving. Its development should be encouraged and analysed.

7. Acknowledgements

These guidelines have been adapted from the European Resuscitation Council 2015 guidelines. We acknowledge and thank the authors of the ERC Guidelines for Education and implementation of resuscitation: Robert Greif, Andrew S. Lockey, Patricia Conaghan, Anne Lippert, Wiebe De Vries, Koenraad G. Monsieurs.

8. References


