

Introduction

This chapter on adult advanced life support (ALS) adheres to the same general principles as Guidelines 2005, but incorporates some important changes. The guidelines in this chapter apply to healthcare professionals trained in ALS techniques. Laypeople, first responders, and automated external defibrillator (AED) users are referred to the basic life support (BLS) and AED chapters.

Guideline changes

Defibrillation

- There is increased emphasis on the importance of minimally-interrupted high-quality chest compressions throughout any ALS intervention: chest compressions are paused briefly only to allow specific interventions.
- The recommendation for a specified period of cardiopulmonary resuscitation (CPR) before out-of-hospital defibrillation, following cardiac arrest unwitnessed by the emergency medical services (EMS), has been removed.
- Chest compressions are now continued while a defibrillator is charged – this will minimise the pre-shock pause.
- The role of the precordial thump is de-emphasised.
- The use of up to three quick successive (stacked) shocks is now recommended for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) occurring in the cardiac catheterisation laboratory or in the immediate post-operative period following cardiac surgery.

Drugs

- Delivery of drugs via a tracheal tube is no longer recommended – if intravenous (IV) access cannot be achieved give drugs by the intraosseous (IO) route.
- When treating VF/VT cardiac arrest, adrenaline 1 mg is given once chest compressions have restarted after the third shock and then every 3-5 min (during alternate cycles of CPR). In the 2005 Guidelines, adrenaline was given just before the third shock. This subtle change in the timing of adrenaline administration is to separate the timing of drug delivery from

attempted defibrillation. It is hoped that this will result in more efficient shock delivery and less interruption in chest compressions. Amiodarone 300 mg is also given after the third shock.

- Atropine is no longer recommended for routine use in asystole or pulseless electrical activity (PEA).

Airway

- There is reduced emphasis on early tracheal intubation unless achieved by highly skilled individuals with minimal interruption to chest compressions.
- There is increased emphasis on the use of capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of return of spontaneous circulation (ROSC).

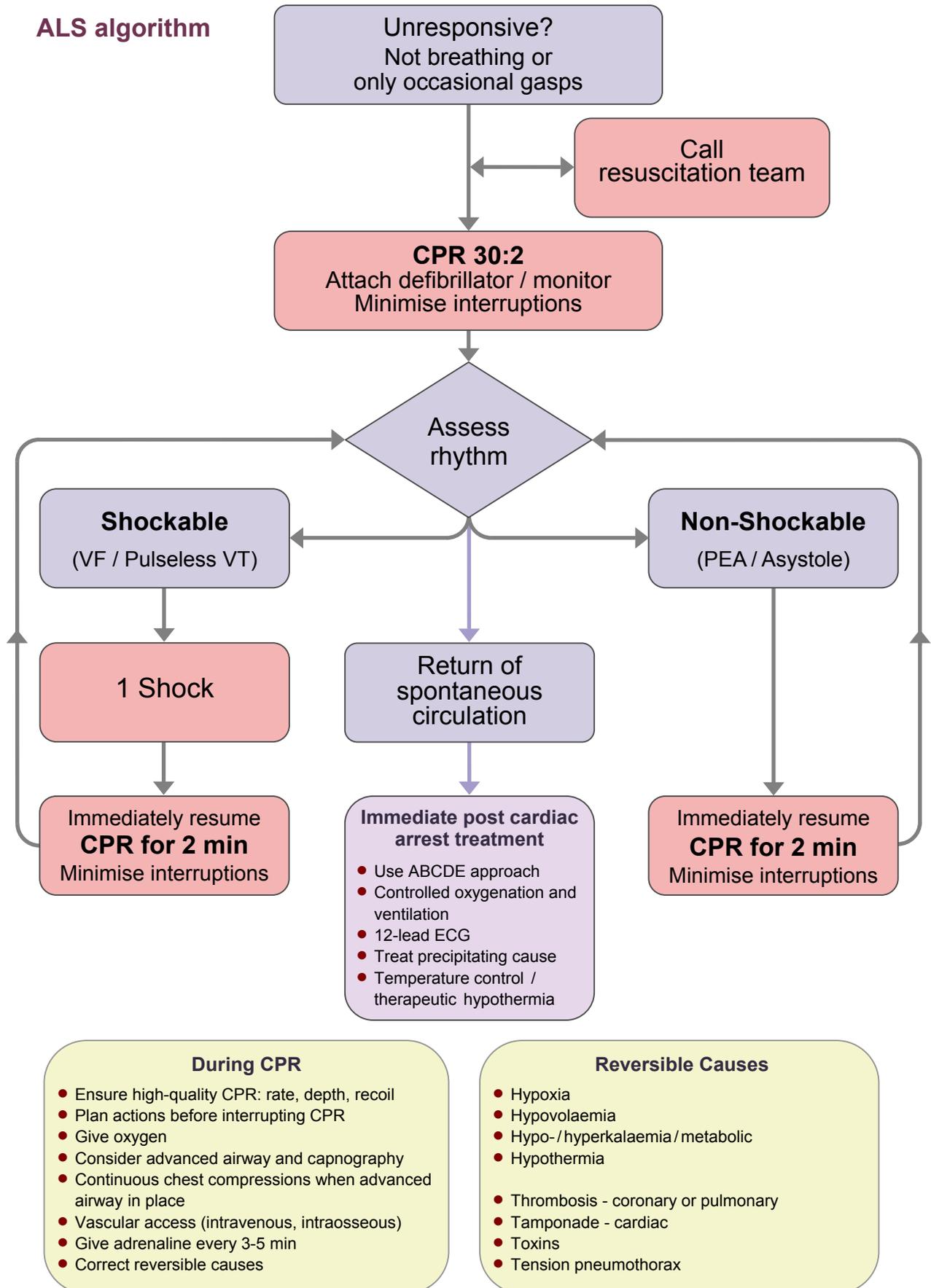
Ultrasound

- The potential role of ultrasound imaging during ALS is recognised.

Post-resuscitation care

- The potential harm caused by hyperoxaemia after ROSC is achieved is now recognised: once ROSC has been established and the oxygen saturation of arterial blood (SaO₂) can be monitored reliably (by pulse oximetry and/or arterial blood gas analysis), inspired oxygen is titrated to achieve a SaO₂ of 94 - 98%.
- There is much greater detail and emphasis on the treatment of the post-cardiac-arrest syndrome.
- There is recognition that implementation of a comprehensive, structured post-resuscitation treatment protocol may improve survival in cardiac arrest victims after ROSC.
- There is increased emphasis on the use of primary percutaneous coronary intervention in appropriate, but comatose, patients with sustained ROSC after cardiac arrest.
- The recommendation for glucose control has been revised: in adults with sustained ROSC after cardiac arrest, blood glucose values >10 mmol l⁻¹ should be treated but hypoglycaemia must be avoided.
- Use of therapeutic hypothermia now includes comatose survivors of cardiac arrest associated initially with non-shockable rhythms as well as shockable rhythms. The lower level of evidence for use after cardiac arrest from non-shockable rhythms is acknowledged.
- It is recognised that many of the accepted predictors of poor outcome in comatose survivors of cardiac arrest are unreliable, especially if the patient has been treated with therapeutic hypothermia.

ALS algorithm



Arrhythmias associated with cardiac arrest are divided into two groups: shockable rhythms (VF/VT) and non-shockable rhythms (asystole and PEA). The principle difference in management is the need for attempted defibrillation in patients with VF/VT. Subsequent 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.

Shockable rhythms (VF/VT)

The first monitored rhythm is VF/VT in approximately 25% of cardiac arrests, both in⁶⁰ or out of hospital.^{42, 129, 130} VF/VT will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.⁶⁰

Treatment of shockable rhythms (VF/VT)

1. Confirm cardiac arrest – check for signs of life or if trained to do so, breathing and 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 from the ECG.
6. Resume chest compressions immediately; simultaneously, the designated person selects the appropriate energy on the defibrillator (150-200 J biphasic for the first shock and 150-360 J biphasic for subsequent shocks) and presses the charge button.
7. While the defibrillator is charging, warn all rescuers other than the individual performing the chest compressions to “stand clear” and remove any oxygen delivery device as appropriate. Ensure that the rescuer giving the compressions is the only person touching the patient.
8. Once the defibrillator is charged, tell the rescuer doing the chest compressions to “stand clear”; when clear, give the shock.
9. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions.
10. Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR.
11. Pause briefly to check the monitor.
12. If VF/VT, repeat steps 6 - 11 above and deliver a second shock.
13. If VF/VT persists repeat steps 6 - 8 above and deliver a third shock. Resume chest compressions immediately and then give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR.

14. Repeat this 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/VT persists.
15. Give further adrenaline 1 mg IV after alternate shocks (i.e., approximately every 3-5 min).

If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of return of spontaneous circulation (ROSC):

- Check a central pulse and end-tidal CO₂ trace if available
- If there is evidence of ROSC, start post-resuscitation care.
- If no signs of ROSC, continue CPR and switch to the non-shockable algorithm.

If asystole is seen, continue CPR and switch to the non-shockable algorithm.

The interval between stopping compressions and delivering a shock must be minimised and certainly should not exceed a few seconds (ideally less than 5 s). Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation.

If an organised rhythm is seen during a 2-minute period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life (this may include a sudden increase in end-tidal carbon dioxide [ETCO₂] if this is being monitored) suggesting ROSC. If there is any doubt about the existence of a pulse in the presence of an organised rhythm, resume CPR. If the patient has ROSC, begin post-resuscitation care.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm⁹³⁻⁹⁵ and is only likely to succeed if given within the first few seconds of the onset of a shockable rhythm.⁹⁶ There is more success with pulseless VT than with VF. Delivery of a precordial thump must not delay calling for help or accessing a defibrillator. It is therefore appropriate therapy only when several clinicians are present at a witnessed, monitored arrest, and when a defibrillator is not immediately to hand. In practice, this is likely to be in a monitored environment such as the emergency department resuscitation room, ICU, CCU, cardiac catheter laboratory or pacemaker room.

A precordial thump should be undertaken immediately after confirmation of cardiac arrest and only by healthcare professionals trained in the technique. 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. There are a very few reports of a precordial thump converting a perfusing rhythm to a non-perfusing rhythm.¹³¹

Explanation for the changes in the treatment of VF/VT

Defibrillation strategy

► Single versus three-shock strategy

Experimental studies show that relatively short interruptions in chest compression to deliver rescue breaths or perform rhythm analysis are associated with reduced survival.¹³² Interruptions in chest compression also reduce the chances of converting VF to another rhythm.¹³³ The interruptions to CPR associated with a three-shock protocol, combined with the improved first shock efficacy (for termination of VF/VT) of biphasic defibrillators, prompted the recommendation of a single-shock strategy in the Guidelines 2005. Subsequent studies have shown a significantly lower hands-off-ratio with the one-shock protocol¹³⁴ and some,^{53, 135, 136} but not all,^{134, 137} have suggested a significant survival benefit from this single-shock strategy. However, all studies except one¹³⁷ were before-after studies and all introduced multiple changes in the protocol, making it difficult to attribute a possible survival benefit to one of the changes.

If VF/VT occurs during cardiac catheterisation or in the early post-operative period following cardiac surgery (when chest compressions could disrupt vascular sutures), consider delivering up to three-stacked shocks before starting chest compressions.¹³⁸ This three-shock strategy may also be considered for an initial, witnessed VF/VT 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 immediately after onset of VF.

► Defibrillation energy

The initial shock from a biphasic defibrillator should be no lower than 120 J for rectilinear biphasic waveforms, and 150 J for biphasic truncated exponential waveforms. For uniformity, it is recommended that the initial biphasic shock should be at least 150 J. Because of the lower efficacy of monophasic defibrillators for terminating VF/VT, and the use of a single-shock strategy, the recommended initial energy level for the first shock using a monophasic defibrillator remains at 360 J.

For second and subsequent shocks, the 2005 guidelines did not distinguish between a fixed and an escalating energy protocol. Since then, several studies have demonstrated that although an escalating strategy reduces the number of shocks required to restore an organised rhythm compared with fixed-dose biphasic defibrillation, rates of ROSC or survival to hospital discharge are not increased.¹³⁹⁻¹⁴² Conversely, a fixed-dose biphasic protocol has demonstrated high success rates with a three-shock fixed-dose protocol.¹⁴³ If an initial shock has been unsuccessful it may be worth attempting the second and subsequent shocks with a higher energy level if the defibrillator is capable of delivering a higher energy, but both fixed and escalating strategies are acceptable, based on current evidence.

Manufacturers should display the effective waveform energy range on the face of the biphasic device. If you are unaware of the effective energy range of the device, use the highest available energy for the first and subsequent shocks.

Fine VF

Fine VF that is difficult to distinguish from asystole is very unlikely to be shocked successfully into a perfusing rhythm. Continuing good quality CPR may improve the amplitude and frequency of the VF and improve the chance of successful defibrillation to a perfusing rhythm. Delivering repeated shocks in an attempt to defibrillate what is thought to be fine VF will increase myocardial injury, both directly from the electric current and indirectly from the interruptions in coronary blood flow.

Adrenaline

Despite the widespread use of adrenaline during resuscitation, and several studies involving vasopressin, there is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases neurologically-intact survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, the use of adrenaline is still recommended, based largely on animal data and increased short-term survival in humans.^{144, 145} The alpha-adrenergic actions of adrenaline cause vasoconstriction, which increases myocardial and cerebral perfusion pressure. The higher coronary blood flow increases the frequency and amplitude of the VF waveform and should improve the chance of restoring a circulation when defibrillation is attempted.¹⁴⁶⁻¹⁴⁸ Although adrenaline improves short-term survival, animal data indicate that it impairs the microcirculation^{149, 150} and can cause post-cardiac-arrest myocardial dysfunction,^{151, 152} both of which might impact on long-term outcome. The optimal dose of adrenaline is not known, and there are no data supporting the use of repeated doses. There are few data on the pharmacokinetics of adrenaline during CPR. The optimal duration of CPR and number of shocks that should be given before giving drugs is unknown. On the basis of expert consensus, for VF/VT give adrenaline after the third shock once chest compressions have resumed, and then repeat every 3-5 min during cardiac arrest (alternate cycles). Do not interrupt CPR to give drugs.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as the absence of any palpable pulse in the presence of cardiac electrical activity that would be expected to produce a cardiac output. These patients often have some mechanical myocardial contractions but they are too weak to produce a detectable pulse or blood pressure – this is sometimes described as ‘pseudo-PEA’. PEA may 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.

Sequence of actions for PEA

- Start CPR 30:2.
- Give adrenaline 1 mg as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compressions without pausing during ventilation.
- Consider possible reversible causes of PEA and correct any that are identified.
- Recheck the patient after 2 min:
 - If there is still **no** pulse and no change in the ECG appearance:
 - Continue CPR.
 - Recheck the patient after 2 min and proceed accordingly.
 - Give further adrenaline 1 mg every 3-5 min (alternate loops).
 - If VF/VT, change to the shockable rhythm algorithm.
 - If a pulse is present, start post-resuscitation care.

Sequence of actions for asystole

- Start CPR 30:2.
- Without stopping CPR, check that the leads are attached correctly.
- Give adrenaline 1 mg as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compression without pausing during ventilation.
- Consider possible reversible causes of PEA and correct any that are identified.
- Recheck the rhythm after 2 min and proceed accordingly.
- If VF/VT, change to the shockable rhythm algorithm.
- Give adrenaline 1 mg IV every 3-5 min (alternate loops).

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.

Atropine

Atropine antagonises the action of the parasympathetic neurotransmitter acetylcholine at muscarinic receptors. Therefore, it blocks the effect of the vagus nerve on both the sinoatrial (SA) node and the atrioventricular (AV) node, increasing the sinus rate and facilitating AV node conduction.

The 2005 guidelines recommended the administration of a single 3 mg dose of atropine for asystole and slow PEA ($< 60 \text{ min}^{-1}$); however, during cardiac arrest asystole is usually caused by primary myocardial pathology rather than excessive vagal tone and there is no evidence that routine use of atropine is beneficial in the treatment of asystole or PEA. Several recent studies have failed to demonstrate any benefit from atropine in

out-of-hospital or in-hospital cardiac arrests¹⁵³⁻¹⁵⁹ and its routine use for asystole or PEA is no longer recommended.

During CPR

During the treatment of persistent VF/VT or PEA/asystole, there should be an emphasis on giving good quality chest compression between defibrillation attempts, whilst recognising and treating reversible causes (4 Hs and 4 Ts), and whilst obtaining a secure airway and intravascular access. Healthcare providers must practise efficient coordination between CPR and shock delivery. The shorter the interval between stopping chest compressions and shock delivery, the more likely it is that the shock will be successful. Reduction in the interval from compression to shock delivery by even a few seconds can increase the probability of shock success. Providing CPR with a CV ratio of 30:2 is tiring; change the individual undertaking compressions every 2 min.

Potentially reversible causes

Potential causes or aggravating factors for which specific treatment exists must be sought during any cardiac arrest.¹³⁸ 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, hypocalcaemia, acidaemia, and other metabolic disorders
- Hypothermia

- Tension pneumothorax
- Tamponade
- Toxic substances
- Thromboembolism (pulmonary embolus/coronary thrombosis)

The four 'Hs'

Minimise the risk of **hypoxia** by ensuring that the patient's lungs are ventilated adequately with 100% oxygen. Make sure that there is adequate chest rise and that there are 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 usually due to severe haemorrhage; this may be precipitated by trauma, gastrointestinal bleeding, or rupture of an aortic aneurysm. Restore intravascular volume rapidly with fluid, coupled with urgent surgery to stop the haemorrhage.

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. A 12-lead ECG may be diagnostic. Intravenous calcium

chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium-channel-blocking drug overdose.

Suspect **hypothermia** in any drowning incident; use a low-reading thermometer.

The four 'Ts'

A **tension pneumothorax** may be the primary cause of PEA and may follow attempts to insert a central venous catheter. The diagnosis is made clinically and/or by use of ultrasound. Decompress rapidly by needle thoracocentesis or urgent thoracostomy, and then insert a chest drain.

Cardiac **tamponade** is difficult to diagnose because the typical signs of distended neck veins and hypotension are obscured by the arrest itself. Rapid transthoracic echocardiography with minimal interruption to chest compression can be used to identify a pericardial effusion. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy.

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.

The commonest cause of **thromboembolic** or mechanical circulatory obstruction is massive pulmonary embolus. If cardiac arrest is likely to be caused by pulmonary embolism, consider giving a thrombolytic drug immediately. Thrombolysis may be considered in adult cardiac arrest, on a case-by-case basis, following initial failure of standard resuscitation in patients in whom an acute thrombotic aetiology for the arrest is suspected. Ongoing CPR is not a contraindication to thrombolysis. Thrombolytic drugs may take up to 90 min to be effective; only administer a thrombolytic drug if it is appropriate to continue CPR for this duration.

Use of ultrasound imaging during advanced life support

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 ultrasound imaging provides information that may help to identify reversible causes of cardiac arrest (e.g. cardiac tamponade, pulmonary embolism, ischaemia (regional wall motion abnormality), aortic dissection, hypovolaemia, pneumothorax).¹⁶⁰ When ultrasound imaging and appropriately trained clinicians are available use them to assist with assessment and treatment of potentially reversible causes of cardiac arrest. The integration of ultrasound into advanced life support requires considerable training to ensure that interruptions to chest compressions are minimised. A sub-xiphoid probe position has been recommended.¹⁶⁰⁻¹⁶² 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 s.

Intravenous fluids

Hypovolaemia is a potentially reversible cause of cardiac arrest: infuse fluids rapidly if hypovolaemia is suspected. In the initial stages of resuscitation there are no clear advantages to using colloid: use 0.9% sodium chloride or Hartmann's solution. Avoid dextrose; this is redistributed rapidly away from the intravascular space and causes hyperglycaemia, which may worsen neurological outcome after cardiac arrest. Try to ensure normovolaemia, but in the absence of hypovolaemia, infusion of an excessive volume of fluid is likely to be harmful during CPR.¹⁶³ Use intravenous fluid to flush peripherally injected drugs into the central circulation.

Open-chest cardiac compression

Open-chest cardiac compression may be indicated for patients with cardiac arrest caused by trauma, in the early postoperative phase after cardiothoracic surgery, or when the chest or abdomen is already open, for example during surgery following trauma.¹³⁸

Signs of life

If signs of life (such as regular respiratory effort, coughing, purposeful movement or eye opening) reappear during CPR, or readings from the patient's monitors (e.g. sudden increase in ET_{CO}₂ or blood pressure monitored from an arterial cannula) are compatible with ROSC, stop CPR and check the monitors briefly. Do not confuse agonal respiration (gaspings), which is common in the immediate few seconds following a cardiac arrest or during good quality CPR, for signs of life. If an organised cardiac rhythm is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care, treatment of peri-arrest arrhythmias, or both. If no pulse is present, continue CPR.

Defibrillation

Strategies before defibrillation

Pads versus paddles

Self-adhesive defibrillation pads have practical benefits over hand-held paddles for routine monitoring and defibrillation.¹⁶⁴ They are safe and effective and, given the change in defibrillation strategy with the 2010 guidelines, are much preferred to standard defibrillation paddles.¹⁶⁵ Use self-adhesive pads in peri-arrest situations and in clinical situations where patient access is difficult. They have a similar transthoracic impedance¹⁶⁶ (and therefore efficacy) to manual paddles and enable the operator to defibrillate the patient from a safe distance rather than leaning over the patient as occurs with paddles. Pads enable a shock to be delivered more rapidly than with paddles.⁹²

Safe use of oxygen

In an oxygen-enriched atmosphere, sparks from defibrillator paddles applied poorly can cause a fire. Taking the following precautions can minimise this risk:

- Remove any oxygen mask or nasal cannulae and place them at least 1 m away from the patient's chest during defibrillation.

- Leave 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.
- The use of self-adhesive defibrillation pads, rather than manual paddles, may minimise the risk of sparks occurring.

Chest hair

It may be necessary to shave the area intended for electrode placement, but do this rapidly and do not delay defibrillation if a razor is not available immediately.

Electrode position

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.

Antero-posterior electrode placement is an acceptable alternative to the traditional right pectoral-apical position for defibrillation and is the preferred choice for cardioversion of atrial fibrillation.

An implanted medical device (e.g. permanent pacemaker or implantable cardioverter defibrillator (ICD)) may be damaged during defibrillation if current is discharged through electrodes placed directly over the device. If possible, place the electrode away from the device if necessary by using an alternative electrode position.

Airway management and ventilation

Most of the principles of airway and ventilation management remain unchanged from Guidelines 2005. There is reduced emphasis on early tracheal intubation unless achieved by highly skilled individuals with minimal interruption to chest compressions. There is increased emphasis on the use of capnography to confirm and continually monitor tracheal tube placement, quality of CPR and to provide an early indication of ROSC.

Patients requiring resuscitation often have an obstructed airway. In these cases, prompt assessment, with control of the airway and ventilation of the lungs, is essential. Without adequate oxygenation it may be impossible to restore a spontaneous cardiac output. In a witnessed cardiac arrest in the vicinity of a defibrillator, attempted defibrillation takes precedence over opening of the airway.

Give high-flow oxygen until ROSC is achieved and reliable monitoring of the oxygen saturation of arterial blood enables the inspired oxygen to be adjusted.

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.

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 supraglottic airway device (SAD). The two-person technique for bag-mask ventilation is preferable. Deliver each breath over approximately 1 s 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 SAD has been inserted, ventilate the lungs at a rate of about 10 breaths min^{-1} and continue chest compression without pausing during ventilation.

Alternative airway devices

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. But there is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (6 - 17% in several studies involving paramedics) is unacceptably high.¹⁶⁷ 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 considered 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, but none of these studies have been powered adequately to enable survival to be studied as a primary endpoint. Instead, most researchers have studied insertion and ventilation success rates. The SADs are easier to insert than a tracheal tube and, unlike tracheal intubation, can generally be inserted without interrupting chest compressions.¹⁶⁸ There are no data supporting the routine use of any specific approach to airway management during cardiac arrest. The best technique is dependent on the precise circumstances of the cardiac arrest and the competence of the rescuer. The Combitube is rarely, if ever, used in the UK and is no longer included in these guidelines.

Laryngeal mask airway (LMA)

A laryngeal mask airway 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.

i-gel

The cuff of the i-gel is made of thermoplastic elastomer gel and does not require inflation; the stem of the i-gel incorporates a bite block and a narrow oesophageal drain tube. It is used commonly for maintenance of the airway during anaesthesia. 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. Use of the i-gel during cardiac arrest has been reported but more data on its use in this setting are awaited.

Laryngeal Tube

The laryngeal tube (LT) was introduced in 2001. A disposable version of the laryngeal tube (LT-D) is available and has been used during resuscitation following pre-hospital cardiac arrest.¹⁶⁹ The LT is not in common use in the UK.

Tracheal intubation

The pros and cons of tracheal intubation have been discussed in the pre-hospital chapter. As with pre-hospital tracheal intubation, intubation in hospital 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 10 s; if intubation is not achievable within these constraints, recommence bag-mask ventilation. After intubation, confirm tube placement and secure the tube adequately.

Confirmation of correct placement of the tracheal tube

Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation. Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube should reduce this risk.

Primary assessment includes observation of chest expansion bilaterally, auscultation over the lung fields bilaterally in the axillae (breath sounds should be equal and heard clearly) and over the epigastrium (breath sounds should not be heard). Clinical signs of correct tube placement (condensation in the tube, chest rise, breath sounds on auscultation of lungs, and inability to hear gas entering the stomach) are not completely reliable. Secondary confirmation of tracheal tube placement by an exhaled carbon dioxide (CO₂) or oesophageal detection device should reduce the risk of unrecognised oesophageal intubation but the performance of the available devices varies considerably. Furthermore, none of the secondary confirmation techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea, so careful primary assessment to ensure equal expansion of both lungs and equally clear breath sounds over each remains important.

There are inadequate data to identify the optimal method of confirming tube placement during cardiac arrest, and all devices should be considered as adjuncts to other confirmatory techniques.¹⁷⁰ There are no data quantifying their ability to monitor tube position after initial placement.

Carbon dioxide detector devices measure the concentration of exhaled carbon dioxide from the lungs. The persistence of exhaled CO₂ after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.⁴⁵ During cardiac arrest pulmonary blood flow may be so low that there is insufficient exhaled CO₂, so the CO₂ detector does not identify a correctly placed tracheal tube. When exhaled CO₂ is detected during cardiac arrest it indicates reliably that the tube is in the trachea or main bronchus. A variety of electronic as well as simple, inexpensive, colorimetric CO₂ detectors are available for both in-hospital and out-of-hospital use. End-tidal CO₂ detectors that include a waveform graphical display (capnographs) are the most reliable for verification of tracheal tube position during cardiac arrest.

Based on the available data, the accuracy of colorimetric 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 should supplement clinical assessment (auscultation and visualisation of the tracheal tube passing between the vocal 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 where intubation is performed, including out of hospital, emergency departments, and in-hospital locations. In the absence of a waveform capnograph it may be preferable to use a supraglottic airway device when advanced airway management is indicated.

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. 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.

Assisting the circulation

Intravascular access

Peripheral versus central venous drug delivery

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. Only those who are skilled and competent in the technique should attempt central venous line insertion and this must be achieved with minimal interruption to chest compressions.

Intraosseous route

If intravenous access cannot be established within the first 2 min of resuscitation, consider gaining IO access. Intraosseous access has traditionally been used for children because of the difficulties in gaining intravenous access, but this route has now become established as a safe and effective route for drug and fluid delivery in adults too.¹⁷¹⁻¹⁷³ Tibial and humeral sites are readily accessible and provide equal flow rates for fluids.¹⁷² Intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations.

Tracheal route

Resuscitation drugs can also be given via a tracheal tube, but the plasma concentrations achieved using this route are very variable and generally considerably lower than those achieved by the IV or IO routes, particularly with adrenaline. Large volumes of intratracheal fluid impair gas exchange. With the ease of gaining IO access and the lack of efficacy of tracheal drug administration, tracheal administration of drugs is no longer recommended.

Drugs

The use of adrenaline has been discussed above.

Anti-arrhythmic drugs

As with vasopressors, the evidence that anti-arrhythmic drugs are of benefit in cardiac arrest is limited. 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 after shock-refractory VF/VT.^{174, 175} There are no data on the use of amiodarone for shock-refractory VF/VT when single shocks are used. Despite the lack of human long-term outcome data, the balance of evidence is in favour of the use of some anti-arrhythmic drugs for the management of arrhythmias in cardiac arrest.

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. Also atrioventricular conduction is slowed, and a similar effect is seen in accessory pathways. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is due more to the solvent (Polysorbate 80 and benzyl alcohol), which causes histamine release, rather than the drug itself.¹⁷⁶ An aqueous amiodarone

preparation that is free from these side effects has recently been approved for use in the United States.

On the basis of expert consensus, if VF/VT persists, give amiodarone 300 mg by bolus injection (flushed with 20 ml of 0.9% sodium chloride or 5% dextrose)¹⁷⁷ after the third shock. A further dose of 150 mg may be given for recurrent or refractory VF/VT, followed by an infusion of 900 mg over 24 h. 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.

Magnesium

Although the benefits of giving magnesium in known hypomagnesaemic states are recognised, the benefit of giving magnesium routinely during cardiac arrest is unproven. Studies in adults in and out of hospital¹⁷⁸⁻¹⁸³ have failed to demonstrate any increase in the rate of ROSC when magnesium is given routinely during CPR. Give an initial intravenous dose of 2 g (= 8 mmol, 4 ml of 50% magnesium sulphate) for refractory VF if there is any suspicion of hypomagnesaemia (e.g. patients on potassium-losing diuretics); it may be repeated after 10-15 min. Other indications are:

- ventricular tachyarrhythmias in the presence of possible hypomagnesaemia;
- torsade de pointes VT;
- digoxin toxicity.

Bicarbonate

Cardiac arrest results in combined respiratory and metabolic acidosis because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. During cardiac arrest, arterial gas values may be misleading and bear little relationship to the tissue acid-base state;¹⁸⁴ analysis of central venous blood may provide a better estimation of tissue pH. Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. It has the following effects:

- it exacerbates intracellular acidosis;
- it produces a negative inotropic effect on 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.

Giving sodium bicarbonate routinely during cardiac arrest and CPR (especially in out-of-hospital cardiac arrest), or after ROSC, is not recommended. Give sodium bicarbonate (50 mmol) if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose. Repeat the dose according to the clinical condition of the patient and the results of repeated blood gas analysis.

Calcium

Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. There are no data supporting any beneficial action for calcium after most cases of cardiac arrest. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Give calcium during resuscitation only when indicated specifically, i.e., in cardiac arrest caused by hyperkalaemia, hypocalcaemia, or overdose of calcium channel-blocking drugs.

The initial dose of 10 ml 10% calcium chloride (6.8 mmol Ca²⁺) may be repeated if necessary. Calcium can slow the heart rate and precipitate arrhythmias. In cardiac arrest, calcium may be given by rapid intravenous injection. In the presence of a spontaneous circulation give it slowly. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.

Mechanical CPR

At best, standard manual CPR produces coronary and cerebral perfusion that is just 30% of normal.¹⁸⁵ Several CPR techniques and devices may improve haemodynamics or short-term survival when used by well-trained providers in selected cases. However, the success of any technique or device depends on the education and training of the rescuers and on resources (including personnel). Although manual chest compressions are often performed very poorly,¹⁸⁶⁻¹⁸⁸ no adjunct has consistently been shown to be superior to conventional manual CPR.

Impedance threshold device (ITD)

The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions; this decreases intrathoracic pressure and increases venous return to the heart. A recent meta-analysis demonstrated improved ROSC and short-term survival but no significant improvement in either survival to discharge or neurologically intact survival to discharge associated with the use of an ITD in the management of adult out-of-hospital cardiac arrest patients.¹⁸⁹ In the absence of data showing that the ITD increases survival to hospital discharge, its routine use in cardiac arrest is not recommended.

Lund University cardiac arrest system (LUCAS) CPR

The Lund University cardiac arrest system (LUCAS) is a gas-driven sternal compression device that incorporates a suction cup for active decompression. Although animal studies showed that LUCAS-CPR improves haemodynamic and short-term survival compared with standard CPR,^{190, 191} there are no published randomised human studies comparing LUCAS-CPR with standard CPR.

Load-distributing band CPR (AutoPulse)

The load-distributing band (LDB) is a circumferential chest compression device comprising a pneumatically actuated constricting band and backboard. Although the use of LDB CPR improves haemodynamics,¹⁹²⁻¹⁹⁴ results of clinical trials have been conflicting.^{195, 196}

The current status of LUCAS and AutoPulse

Two large prospective randomised multicentre studies are currently underway to evaluate AutoPulse and LUCAS. In hospital, mechanical devices have been used to support patients undergoing primary coronary intervention (PCI)^{197, 198} and CT scans¹⁹⁹ and also for prolonged resuscitation attempts (e.g., hypothermia,^{200, 201} poisoning, fibrinolytic therapy for pulmonary embolism, prolonged transport etc) where rescuer fatigue may impair the effectiveness of manual chest compression. The role of mechanical devices in all situations requires further evaluation before firm recommendations on their use can be made.

Post-resuscitation care

The post-cardiac-arrest syndrome

Successful ROSC is the just the first step toward the goal of complete recovery from cardiac arrest. The post-cardiac-arrest syndrome, which comprises post-cardiac-arrest brain injury, post-cardiac-arrest myocardial dysfunction, the systemic ischaemia / reperfusion response, and persistence of the precipitating pathology, often complicates the post-resuscitation phase.²⁰² 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. Post-cardiac-arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypercarbia, hyperoxia, pyrexia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2-3 days.^{203, 204} The whole body ischaemia/reperfusion that occurs with resuscitation from cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection.^{205, 206} Thus, the post-cardiac-arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.^{207, 208}

Airway and breathing

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 causes oxidative stress and harms post-ischaemic neurones.²⁰⁹ A clinical registry study documented that post-resuscitation hyperoxaemia was associated with worse outcome, compared with both normoxaemia and hypoxaemia.²¹⁰ 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%. Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. There are no data to support the targeting of a specific arterial PCO₂ after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocarbia and to monitor this using the end-tidal PCO₂ and arterial blood gas values.

Circulation

It is well recognised that post-cardiac-arrest patients with ST elevation myocardial infarction (STEMI) should undergo early coronary angiography and percutaneous coronary intervention (PCI) but, because chest pain and/or ST elevation are relatively poor predictors of acute coronary occlusion in these patients,²¹¹ this intervention should be considered in all post-cardiac-arrest patients who are suspected of having coronary artery disease as the cause of their arrest.^{55, 211, 212} Several studies indicate that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction.²¹²⁻²¹⁶

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias.²⁰³ If treatment with appropriate fluids and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra-aortic balloon pump.^{212, 213} In the absence of definitive data, target the mean arterial blood pressure to achieve an adequate urine output ($1 \text{ ml kg}^{-1} \text{ h}^{-1}$) and normal or decreasing plasma lactate values, taking into consideration the patient's usual blood pressure (if known), the cause of the arrest and the severity of any myocardial dysfunction.²⁰²

Disability (optimising neurological recovery)

Control of seizures

Seizures or myoclonus or both occur in 5% to 15% of adult patients who achieve ROSC and 10% to 40% of those who remain comatose.²¹⁷⁻²²⁰ Seizures increase cerebral metabolism by up to 3-fold and may cause cerebral injury: treat promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate. No studies address directly the use of prophylactic anticonvulsant drugs after cardiac arrest in adults.

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome.^{221, 222} A large randomised trial of intensive glucose control ($4.5 - 6.0 \text{ mmol l}^{-1}$) 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.²²³ Another recent study and two meta-analyses of studies of tight glucose control versus conventional glucose control in critically ill patients showed no significant difference in mortality but found tight glucose control was associated with a significantly increased risk of hypoglycaemia.²²⁴⁻²²⁶ Severe hypoglycaemia is associated with increased mortality in critically ill patients,²²⁷ and comatose patients are at particular risk from unrecognised hypoglycaemia. Based on the available data, following ROSC blood glucose should be maintained at $\leq 10 \text{ mmol l}^{-1}$.²²⁸ Hypoglycaemia should be avoided. Strict glucose control should not be implemented in adult patients with ROSC after cardiac arrest because of the increased risk of hypoglycaemia.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest.²²⁹ Several studies document an association between post-cardiac-arrest pyrexia and poor outcomes.²³⁰⁻²³² Although the effect of elevated temperature on outcome is not proved, it seems prudent to treat any hyperthermia occurring after cardiac arrest with antipyretics or active cooling.

Therapeutic hypothermia

Animal and human data indicate that mild hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia.^{233, 234} Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen by about 6% for each 1°C reduction in temperature²³⁵ and this may reduce the release of excitatory amino acids and free radicals.²³³ Hypothermia blocks the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response associated with the post-cardiac-arrest syndrome.

All studies of post-cardiac-arrest therapeutic hypothermia have included only patients in coma. There is good evidence supporting the use of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest caused by VF. One randomised trial²³⁶ and a pseudo-randomised trial²³⁷ demonstrated improved neurological outcome at hospital discharge or at 6 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. Extrapolation of these data to other cardiac arrests (e.g., other initial rhythms, in-hospital arrests, paediatric patients) seems reasonable but is supported only by data derived from non-randomised trials.^{212, 238-241}

The practical application of therapeutic hypothermia is divided into three phases: induction, maintenance, and rewarming.²⁴² Animal data indicate that earlier cooling after ROSC produces better outcomes.²⁴³ External and/or internal cooling techniques can be used to initiate cooling. An infusion of 30 ml kg⁻¹ of 4°C 0.9% sodium chloride or Hartmann's solution decreases core temperature by approximately 1.5°C. Other methods of inducing and/or maintaining hypothermia include: simple ice packs and/or wet towels; cooling blankets or pads; water or air circulating blankets; water circulating gel-coated pads; intravascular heat exchanger; and cardiopulmonary bypass.

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. Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling. Thus, rewarming must be achieved slowly: the optimal rate is not known, but the consensus is currently about 0.25-0.5 °C increase in body temperature per hour.²⁴⁰ The well-recognised physiological effects of hypothermia need to be managed carefully.²⁴²

Prognostication

Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury.²⁴⁴ A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Many studies have focused on prediction of poor long term outcome (vegetative state or death), based on clinical or test findings that indicate irreversible brain injury, to enable clinicians to limit care or withdraw organ support. The implications of these prognostic tests are such that they should have 100% specificity or zero false positive rate, i.e., no individuals eventually have a 'good' long-term outcome despite the prediction of a poor outcome.

Clinical examination

There are no clinical neurological signs that predict poor outcome (Cerebral Performance Category [CPC] 3 or 4, or death) reliably less than 24 h after cardiac arrest. In adult patients who are comatose after cardiac arrest, and who have not been treated with hypothermia and who do not have confounding factors (such as hypotension, sedatives or muscle relaxants), the absence of both pupillary light and corneal reflex at ≥ 72 h predicts poor outcome reliably.²²⁰ Absence of vestibulo-ocular reflexes at ≥ 24 h^{245, 246} and a GCS motor score of 2 or less at ≥ 72 h²²⁰ are less reliable. Other clinical signs, including myoclonus, are not recommended for predicting poor outcome. The presence of myoclonic status in adults is strongly associated with poor outcome,^{219, 220, 247-249} but rare cases of good neurological recovery from this situation have been described and accurate diagnosis is problematic.

Biochemical markers

Serum (e.g. neuronal specific enolase, S100 protein) or cerebrospinal fluid (CSF) biomarkers alone are insufficient as predictors of poor outcomes in comatose patients after cardiac arrest with or without treatment with therapeutic hypothermia.

Neurophysiological studies

No neurophysiological study predicts outcome for a comatose patient reliably within the first 24 h after cardiac arrest. If somatosensory evoked potentials (SSEP) are measured after 24 h in comatose cardiac arrest survivors not treated with therapeutic hypothermia, bilateral absence of the N20 cortical response to median nerve stimulation predicts poor outcome (death or CPC 3 or 4).²⁵⁰ Very few hospitals in the UK have the resources to enable SSEPs to be measured.

Imaging studies

Many imaging modalities (magnetic resonance imaging [MRI], computed tomography [CT], single photon emission computed tomography [SPECT], cerebral angiography, transcranial Doppler, nuclear medicine, near infra-red spectroscopy [NIRS]) have been studied to determine their utility for prediction of outcome in adult survivors of cardiac arrest.²⁵¹ There are no high-level studies that support the use of any imaging modality to predict outcome of comatose cardiac arrest survivors.

Impact of therapeutic hypothermia on prognostication

There is inadequate evidence to recommend a specific approach to prognosticating poor outcome in post-cardiac-arrest patients treated with therapeutic hypothermia. There are no clinical neurological signs, neurophysiological studies, biomarkers, or imaging modalities that can predict neurological outcome reliably in the first 24 h after cardiac arrest. Potentially reliable prognosticators of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include bilateral absence of N20 peak on SSEP \geq 24 h after cardiac arrest and the absence of both corneal and pupillary reflexes 3 or more days after cardiac arrest.^{247, 252} Given the limited available evidence, decisions to limit care should not be made based on the results of a single prognostication tool.

Organ donation

Post-cardiac-arrest patients who do not survive represent an opportunity to increase the organ donor pool, either after brain death²⁵³ or as non-heart-beating donors.²⁵⁴

Cardiac arrest centres

There is wide variation in patient survival rates among hospitals caring for patients after resuscitation from cardiac arrest.²⁵⁵⁻²⁵⁸ There is indirect evidence that regional cardiac systems of care improve outcome after STEMI.²⁵¹ The implication from all these data is that specialist cardiac arrest centres and systems of care may be effective but direct evidence is awaited.²⁵⁹⁻²⁶¹