Adult Advanced Life Support

Introduction

This section on adult advanced life support (ALS) adheres to the same general principles as in Guidelines 2000, 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 basic life support (BLS) and AED sections.

Guideline changes

CPR before defibrillation

- In the case of out-of-hospital cardiac arrest attended, but unwitnessed, by healthcare professionals equipped with manual defibrillators, give CPR for 2 min (i.e. about 5 cycles at 30:2) before defibrillation.
- Do not delay defibrillation if an out-of-hospital arrest is witnessed by a healthcare professional.
- Do not delay defibrillation for in-hospital cardiac arrest.

Defibrillation strategy

- Treat ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) with a single shock, followed by immediate resumption of CPR (30 compressions to 2 ventilations). Do not reassess the rhythm or feel for a pulse. After 2 min of CPR, check the rhythm and give another shock (if indicated).
- The recommended initial energy for biphasic defibrillators is 150-200 J. Give second and subsequent shocks at 150-360 J.
- The recommended energy when using a monophasic defibrillator is 360 J for both the initial and subsequent shocks.

Fine VF

• If there is doubt about whether the rhythm is asystole or fine VF, do NOT attempt defibrillation; instead, continue chest compression and ventilation.

Adrenaline (epinephrine)

VF/VT

- Give adrenaline 1 mg IV if VF/VT persists after a second shock.
- Repeat the adrenaline every 3-5 min thereafter if VF/VT persists.

Adult Advanced Life Support Algorithm





Pulseless electrical activity / asystole

• Give adrenaline 1 mg IV as soon as intravenous access is achieved and repeat every 3-5 min.

Anti-arrhythmic drugs

- If VF/VT persists after three shocks, give amiodarone 300 mg by bolus injection. 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.
- If amiodarone is not available, lidocaine 1 mg kg⁻¹ may be used as an alternative, but do not give lidocaine if amiodarone has already been given. Do not exceed a total dose of 3 mg kg⁻¹ during the first hour.

Post resuscitation care - therapeutic hypothermia

- Unconscious adult patients with spontaneous circulation after out-ofhospital VF cardiac arrest should be cooled to 32-34°C for 12-24 h.
- Mild hypothermia may also benefit unconscious patients with spontaneous circulation after out-of-hospital cardiac arrest due to a non-shockable rhythm, or after cardiac arrest in hospital.

ALS treatment 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, venous access, administration of adrenaline, and the identification and correction of reversible factors, are common to both groups. The ALS treatment algorithm provides a standardised approach to the management of adult patients in cardiac arrest.

Shockable rhythms (VF/VT)

Sequence of actions

- Attempt defibrillation (one shock 150-200 J biphasic or 360 J monophasic).
- Immediately resume chest compressions (30:2) without reassessing the rhythm or feeling for a pulse.
- Continue CPR for 2 min, then pause briefly to check the monitor:
 - If VF/VT persists:
 - Give a further (2nd) shock (150-360 J biphasic or 360 J monophasic).
 - Resume CPR immediately and continue for 2 min.
 - Pause briefly to check the monitor.



- If VF/VT persists give adrenaline 1 mg IV followed immediately by a (3rd) shock (150-360 J biphasic or 360 J monophasic).
- Resume CPR immediately and continue for 2 min.
- Pause briefly to check the monitor.
- If VF/VT persists give amiodarone 300 mg IV followed immediately by a (4th) shock (150-360 J biphasic or 360 J monophasic).
- Resume CPR immediately and continue for 2 min.
- Give adrenaline 1 mg IV immediately before alternate shocks (i.e. approximately every 3-5 min).
- Give a further shock after each 2 min period of CPR and after confirming that VF/VT persists.
- If organised electrical activity is seen during this brief pause in compressions, check for a pulse.
 - If a pulse is present, start post-resuscitation care.
 - If no pulse is present, continue CPR and switch to the nonshockable algorithm.
- If asystole is seen, continue CPR and switch to the nonshockable algorithm.

Precordial thump

Consider giving a single precordial thump when cardiac arrest is confirmed rapidly after a witnessed and monitored sudden collapse, and a defibrillator is not immediately to hand. A precordial thump should be undertaken immediately after confirmation of cardiac arrest but 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. A precordial thump is most likely to be successful in converting VT to sinus rhythm. Successful treatment of VF by precordial thump is much less likely: in all the reported successful cases the precordial thump was given within the first 10 seconds of VF. There are very rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.

Explanation for the changes in the treatment of VF/VT

CPR before defibrillation

Although Guidelines 2000 recommended immediate defibrillation for all shockable rhythms, recent evidence indicates that a period of CPR before defibrillation may improve survival after prolonged collapse (> 5 min).¹ The duration of collapse is frequently difficult to estimate accurately, so give CPR before attempted defibrillation outside hospital, unless the arrest is witnessed by a healthcare professional or an AED is being used. This advice does NOT apply to lay responders using an AED outside hospital, who should apply the AED as soon as it is available.

In contrast, there is no evidence to support or refute CPR before defibrillation for in-hospital cardiac arrest. For this reason, after in-hospital VF/VT cardiac arrest, give a shock as soon as possible.

Defibrillation strategy

There are no published human or experimental studies comparing a single shock protocol with a three-stacked shock protocol for the treatment of VF/VT cardiac arrest. Experimental studies show that relatively short interruptions in chest compression to deliver rescue breaths or perform rhythm analysis are associated with reduced survival.^{2,3} Interruptions in chest compression also reduce the chances of converting VF to another rhythm.⁴ Significant interruptions in chest compression are common during out-of-hospital and in-hospital cardiac arrest.^{5,6} When using a three-shock protocol, the time taken to deliver shocks and analyse the rhythm causes significant interruptions in CPR. This fact, combined with the improved first shock efficacy (for termination of VF/VT) of biphasic defibrillators, has prompted the recommendation of a single-shock strategy.

With first shock efficacy of biphasic waveforms exceeding 90%, failure to terminate VF/VT successfully implies the need for a period of CPR (to improve myocardial oxygenation) rather than a further shock. Even if defibrillation is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately afterwards, and the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.⁷ If a perfusing rhythm has been restored, giving chest compression does not increase the chance of VF recurring. In the presence of post-shock asystole, however, chest compressions may induce VF.

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. If an initial shock has been unsuccessful it may be worth attempting the second and subsequent shocks with a higher energy level. However, there is no evidence to support either a fixed or escalating energy protocol. Both strategies are acceptable. 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 200 J for the first shock. This 200 J default has been chosen because it falls within the reported range of selected energy levels that are effective for first and subsequent biphasic shocks, and can be provided by every biphasic manual defibrillator available today. It is a consensus default and not a recommended ideal. If biphasic devices are clearly labelled, and providers are familiar with the devices they use in clinical care, there will be no need for the default 200 J. Ongoing research is necessary to establish the most appropriate initial settings for both monophasic and biphasic defibrillators.

Because of the lower efficacy of monophasic defibrillators for terminating VF/VT, and the change to a single-shock strategy, the recommended initial energy level for the first shock using a monophasic defibrillator is 360 J. If needed, second and subsequent shocks should be given at 360 J. A monophasic waveform is less efficient than a biphasic waveform at terminating VF/VT, and most manufacturers now sell only biphasic devices. The urgency with which monophasic defibrillators are replaced must be determined locally, taking into consideration available resources and competing healthcare demands.



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

There is no placebo-controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases 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 experimental data. The alpha-adrenergic actions of adrenaline cause vasoconstriction, which increases myocardial and cerebral perfusion pressure during cardiac arrest.

Immediate resumption of CPR after shock delivery, along with the elimination of a rhythm check at this stage, makes it difficult to select an ideal point in the ALS algorithm at which to give adrenaline. The consensus recommendation is to give adrenaline immediately after confirmation of the rhythm and just before shock delivery (**drug-shock-CPR-rhythm check** sequence). Have the adrenaline ready to give so that the delay between stopping chest compression and delivery of the shock is minimised. The adrenaline that is given immediately before the shock will be circulated by the CPR that follows the shock.

When the rhythm is checked 2 min after giving a shock, if a non-shockable rhythm is present and the rhythm is organised (complexes appear regular or narrow), try to palpate a pulse. Rhythm checks must be brief, and pulse checks undertaken only if an organised rhythm is observed. If an organised rhythm is seen during a 2-min period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life suggesting return of spontaneous circulation (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.

If the patient's rhythm changes to asystole or PEA see non-shockable rhythms below. In patients in asystole or PEA, give adrenaline 1 mg IV immediately intravenous access is achieved.

In both VF/VT and PEA / asystole, give adrenaline 1 mg IV every 3-5 min (approximately every other two-minute loop).

In patients with a spontaneous circulation, doses considerably smaller than 1 mg IV may be required to maintain an adequate blood pressure.

Vasopressin

A recent meta-analysis of five randomised trials showed no statistically significant difference between vasopressin and adrenaline for ROSC, death within 24 h, or death before hospital discharge.⁸ A subgroup analysis, based on initial cardiac rhythm, did not show any statistically significant difference in the rate of death before hospital discharge. Despite the absence of placebo-controlled trials, adrenaline has been the standard vasopressor in cardiac arrest. There is insufficient evidence to support or refute the use of vasopressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm. Thus, adrenaline remains the primary vasopressor for the treatment of cardiac arrest in all rhythms.

Anti-arrhythmic drugs

There is no evidence that giving any anti-arrhythmic drug routinely during human cardiac arrest increases survival to hospital discharge. In comparison with placebo and lidocaine, the use of amiodarone in shock-refractory VF improves survival to hospital admission.^{9,10} There are no data on the use of amiodarone for shock-refractory VF/VT when single shocks are used. On the basis of expert consensus, if VF/VT persists after three shocks, give amiodarone 300 mg by bolus injection during the brief rhythm analysis before delivery of the fourth 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.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as cardiac electrical activity in the absence of any palpable pulse. These patients often have some mechanical myocardial contractions but they are too weak to produce a detectable pulse or blood pressure. PEA may be caused by reversible conditions that can be treated if they are identified and corrected (see below). 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 IV as soon as intravascular access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compressions without pausing during ventilation.
- Recheck the rhythm after 2 min.
 - If there is no change in the ECG appearance:
 - Continue CPR.
 - Recheck the rhythm after 2 min and proceed accordingly.
 - Give further adrenaline 1 mg IV every 3-5 min (alternate loops).
 - If the ECG changes and organised electrical activity is seen, check for a pulse.



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 - If a pulse is present, start post-resuscitation care.
 - If **no** pulse is present:
 - Continue CPR.
 - Recheck the rhythm after 2 min and proceed accordingly.
 - Give further adrenaline 1 mg IV every 3-5 min (alternate loops).

Sequence of actions for asystole and slow PEA (rate < 60 min⁻¹)

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

Asystole

Asystole is a condition that can be precipitated or exacerbated by excessive vagal tone. Theoretically, this can be reversed by a vagolytic drug; therefore, give atropine 3 mg (the dose that will provide maximum vagal blockade) for asystole or slow PEA (rate < 60 min^{-1}).

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. There is no value in attempting to pace true asystole.

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, recognising and treating reversible causes (4 Hs and 4 Ts), and obtaining a secure airway and intravenous access. Healthcare providers must practise efficient coordination between CPR and shock delivery. The shorter the interval between cessation of chest compression 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. 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
- **T**ension 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 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 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 at central venous catheter insertion. The diagnosis is made clinically. Decompress rapidly by 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 needle pericardiocentesis or 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 thought 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.

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 saline or Hartmann's solution. Avoid dextrose; this is redistributed away from the intravascular space rapidly, and causes hyperglycaemia which may worsen neurological outcome after cardiac arrest.

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.

Signs of life

If signs of life (such as regular respiratory effort or movement) reappear during CPR, or readings from the patient's monitors (e.g. exhaled carbon dioxide or arterial blood pressure) are compatible with a return of spontaneous circulation, stop CPR and check the monitors briefly. 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

Safe use of oxygen

In an oxygen-enriched atmosphere, sparks from poorly-applied defibrillator paddles 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.
- 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 rapidly to shave the area intended for electrode placement, but do not delay defibrillation if a razor is not immediately available.



Paddle force

If using paddles, apply them firmly to the chest wall. The optimal force is 8 kg in adults, and 5 kg in children 1-8 years using adult paddles. Place water-based gel pads between the paddles and the patient's skin.

Electrode position

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

Antero-posterior electrode placement may be more effective than the traditional antero-apical position in cardioversion of atrial fibrillation. Either position is acceptable.

An implantable medical device (e.g. permanent pacemaker or automatic implantable cardioverter defibrillator (AICD)) may be damaged during defibrillation if current is discharged through electrodes placed directly over the device. Place the electrode away from the device or use an alternative electrode position. Remove any transdermal drug patches on the chest wall before defibrillation.

Pads versus paddles

Self-adhesive defibrillation pads are safe and effective and are an acceptable alternative to standard defibrillation paddles. They enable the operator to defibrillate from a safe distance, rather than leaning over the patient as occurs with paddles. When used for initial monitoring of a rhythm, both pads and paddles enable quicker delivery of the first shock compared with standard ECG electrodes, but pads are quicker than paddles.

Airway management and ventilation

The principles of airway and ventilation management remain unchanged from Guidelines 2000.

Patients requiring resuscitation often have an obstructed airway. 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 should take precedence over opening the airway.

Give high-flow oxygen. In the spontaneously breathing patient, masks with non-rebreathing reservoir bags are more effective than standard masks.

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 oxygenenriched air. A pocket resuscitation mask enables mouth-to-mask ventilation. 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 facemask, tracheal tube, or alternative airway device. The two-person technique for bag-mask ventilation is preferable. Deliver each breath over approximately 1 sec 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 an airway device 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

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.

The Combitube

A Combitube is relatively easy to insert and ventilation using this device is more efficient and easier than with a bag-mask. Great care must be taken to avoid attempting to ventilate the lungs through the wrong port of the Combitube.

Tracheal intubation

There is insufficient evidence to support or refute the use of any specific technique to maintain an airway and provide ventilation in adults with cardiopulmonary arrest. Despite this, tracheal intubation is perceived as the optimal method of providing and maintaining a clear and secure airway. It should be used only when trained personnel are available to carry out the procedure with a high level of skill and confidence.

The perceived advantages of tracheal intubation over bag-mask ventilation include:

- maintenance of a patent airway, which is protected from aspiration of gastric contents or blood from the oropharynx;
- ability to provide an adequate tidal volume reliably even when chest compressions are uninterrupted;



- the potential to free the rescuer's hands for other tasks;
- the ability to suction airway secretions;
- the provision of a route for giving drugs.

Use of the bag-mask is more likely to cause gastric distension which, theoretically, is more likely to cause regurgitation and aspiration. However, there are no reliable data to indicate that the incidence of aspiration is any higher in cardiac arrest patients ventilated using a bag-mask compared with those ventilated via a tracheal tube.

The perceived disadvantages of tracheal intubation over bag-mask ventilation include:

- the risk of an unrecognised, misplaced tracheal tube;
- a prolonged time without chest compression while intubation is attempted;
- a comparatively high failure rate.

Intubation success rates correlate with the intubation experience of the individual. Healthcare professionals who undertake 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 compression. The intubation attempt will require interruption of chest compression, but once an advanced airway is in place ventilation can continue uninterrupted. Those skilled in advanced airway management should be able to undertake laryngoscopy without stopping chest compression; a brief pause in chest compression will be required as the tube is passed through the vocal cords. Alternatively, to avoid any interruption, the intubation attempt may be deferred until ROSC. No intubation attempt should take longer than 30 sec. If intubation has not been achieved by then, recommence bag-mask ventilation. After intubation, tube placement must be confirmed and the tube secured 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 should include bilateral observation of chest expansion, bilateral auscultation in the axillae (breath sounds should be equal and adequate), and auscultation 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 CO₂ or oesophageal detector device should reduce the risk of unrecognised oesophageal intubation. If there is doubt about correct tube placement, use the laryngoscope and look directly to see if the tube passes through the vocal cords.

None of the secondary confirmation techniques will differentiate between a tube placed in a main bronchus and one placed correctly in the trachea. 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.

During cardiac arrest pulmonary blood flow may be so low that there is insufficient exhaled CO_2 , so the CO_2 detector does not identify a correctly placed tracheal tube. When exhaled CO_2 is detected during cardiac arrest it indicates reliably that the tube is in the trachea or main bronchus, but when it is absent tracheal tube placement is best confirmed with an oesophageal detector device. A variety of electronic as well as simple, inexpensive, colorimetric CO_2 detectors are available for both in-hospital and out-of-hospital use.

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.

Assisting the circulation

Intravenous 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. Central venous line insertion must cause minimal interruption of chest compression.

Intraosseous route

If intravenous access is difficult or impossible, consider the intraosseous route for both children and adults. The intraosseous route also enables withdrawal of marrow for venous blood gas analysis and measurement of electrolytes and haemoglobin concentration.

Tracheal route

If intravenous or intraosseous access cannot be established, some drugs can be given by the tracheal route. The dose of adrenaline is 3 mg diluted to at least 10 ml with sterile water.

Drugs

The use of adrenaline and anti-arrhythmic drugs has been discussed above.

Magnesium

Give magnesium sulphate 8 mmol (4 ml of a 50% solution) for refractory VF if there is any suspicion of hypomagnesaemia (e.g. patients on potassium-losing diuretics). Other indications are:



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

Bicarbonate

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.

Atropine

Blockade of parasympathetic activity at both the sinoatrial (SA) node and the atrioventricular (AV) node may increase sinus automaticity and facilitate AV node conduction. The adult dose of atropine for asystole, or PEA with a rate $< 60 \text{ min}^{-1}$, is 3 mg IV.

Calcium

Calcium is indicated during resuscitation from PEA if this is thought to be caused by:

- hyperkalaemia;
- hypocalcaemia;
- overdose of calcium-channel-blocking drugs;
- overdose of magnesium (e.g. during treatment of pre-eclampsia).

The initial dose of 10 ml 10% calcium chloride (6.8 mmol Ca²⁺) may be repeated if necessary. Remember that 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 it should be given slowly. Do not give calcium solutions and sodium bicarbonate simultaneously by the same venous access.

Post-resuscitation care

Return of spontaneous circulation is just the first step towards the goal of complete recovery from cardiac arrest. Interventions in the post-resuscitation period influence the final outcome significantly. The post-resuscitation phase starts when ROSC is achieved. Once stabilised, the patient should be transferred to the most appropriate high-care area (e.g. intensive care unit or cardiac care unit) for continued monitoring and treatment.

Airway and breathing

Consider tracheal intubation, sedation, and controlled ventilation in any patient with obtunded cerebral function. Adjust ventilation to achieve normocarbia and monitor this using the end-tidal CO_2 and arterial blood gas values. Adjust the

inspired oxygen concentrations to achieve adequate arterial oxygen saturation. 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. Obtain a chest radiograph to check the position of the tracheal tube and central venous lines and exclude a pneumothorax associated with rib fractures from CPR.

Circulation

Haemodynamic instability is common after cardiac arrest. An arterial line for continuous blood pressure monitoring is essential, and the use of a non-invasive cardiac output monitor may be helpful. Infusion of fluids may be required to optimise filling. Conversely, diuretics and vasodilators may be needed to treat left ventricular failure. Infusion of an inotrope may be required to maintain a mean arterial blood pressure that is no lower than the normal pressure for the patient, and achieves an adequate urine output. Maintain the serum potassium concentration between 4.0-4.5 mmol l⁻¹. If there is evidence of coronary occlusion, consider the need for immediate revascularisation by thrombolytic therapy or percutaneous coronary intervention.

Disability (optimising neurological recovery)

Sedation

If sedation is required, short-acting drugs (e.g. propofol, alfentanil, remifentanil) will enable earlier neurological assessment.

Control of seizures

Seizures are relatively common in the post-resuscitation period and may cause cerebral injury. Control seizures with benzodiazepines, phenytoin, propofol, or a barbiturate as appropriate.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia is common in the first 48 h after cardiac arrest. The risk of a poor neurological outcome increases for each degree of body temperature above 37°C. Treat hyperthermia occurring in the first 72 h after cardiac arrest with antipyretics or active cooling.

Therapeutic hypothermia

Mild hypothermia is thought to suppress many of the chemical reactions associated with reperfusion injury. Two randomised clinical trials showed improved outcome in adults, remaining comatose after initial resuscitation from out-of-hospital VF cardiac arrest, who were cooled within minutes to hours after ROSC.^{11,12}

Unconscious adult patients with spontaneous circulation after out-of-hospital VF cardiac arrest should be cooled to 32-34°C.¹³ Cooling should be started as soon as possible and continued for at least 12-24 h. Induced hypothermia may also benefit unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest from a non-shockable rhythm, or cardiac arrest in hospital. Treat shivering by ensuring adequate sedation and giving neuromuscular-



blocking drugs. Bolus doses of neuromuscular blockers are usually adequate but infusions are occasionally necessary. Re-warm the patient slowly $(0.25-0.5^{\circ}C h^{-1})$ and avoid hyperthermia. The optimum target temperature, rate of cooling, duration of hypothermia, and rate of rewarming have yet to be determined; further studies are essential.

External or internal cooling techniques or both can be used to initiate treatment. An infusion of 30 ml kg⁻¹ saline at 4°C decreases core temperature by 1.5°C. Intravascular cooling enables more precise control of core temperature than external methods, but it is unknown whether this improves outcome.

Complications of mild therapeutic hypothermia include increased infection, cardiovascular instability, coagulopathy, hyperglycaemia, and electrolyte abnormalities such as hypophosphataemia and hypomagnesaemia.

Blood glucose control

There is a strong association between high blood glucose levels after resuscitation from cardiac arrest and poor neurological outcome. Tight control of blood glucose (4.4 - 6.1 mmol l⁻¹) using insulin reduces hospital mortality in critically ill adults, but this has not been demonstrated in post-cardiac-arrest patients specifically.

The optimal blood glucose target level in critically ill patients has not been determined. Comatose patients are at particular risk from unrecognised hypoglycaemia, and the risk of this complication occurring increases as the target blood glucose concentration is lowered.

In common with all critically ill patients, patients admitted to a critical-care environment after cardiac arrest should have their blood glucose monitored frequently and hyperglycaemia treated with an insulin infusion. The blood glucose concentration that triggers insulin therapy and the target range of blood glucose concentrations should be determined by local policy. There is a need for studies of glucose control after cardiac arrest.

Prognostication

There are no neurological signs that can predict outcome in the comatose patient in the first hours after ROSC. By three days after the onset of coma related to cardiac arrest, 50% of patients with no chance of ultimate recovery have died. In the remaining patients, the absence of pupil light reflexes on day three, and an absent motor response to pain on day three, are both independently predictive of a poor outcome (death or vegetative state) with very high specificity.

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