

Learning outcomes

To understand:

- ▶ **The function of the advanced life support (ALS) algorithm**
- ▶ **The importance of minimally interrupted high quality chest compressions**
- ▶ **The treatment of shockable and non-shockable rhythms**
- ▶ **When and how to give drugs during cardiac arrest**
- ▶ **The potentially reversible causes of cardiac arrest**

Introduction

Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation / pulseless ventricular tachycardia (VF/VT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The principle difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in patients with VF/VT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, injection of adrenaline and the identification and correction of reversible factors, are common to both groups.

The ALS algorithm (Figure 6.1) is a standardised approach to cardiac arrest management. This has the advantage of enabling treatment to be delivered expediently, without protracted discussion. It enables each member of the resuscitation team to predict and prepare for the next stage in the patient's treatment, further enhancing efficiency of the team. Although the ALS algorithm is applicable to most cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances (see Chapter 12).

The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander cardiopulmonary resuscitation (CPR), uninterrupted, high quality chest compressions, and early defibrillation for VF/VT. The use of adrenaline has been shown to increase return of spontaneous circulation (ROSC), but no resuscitation drugs or advanced airway interventions have been shown to increase survival to hospital discharge after cardiac arrest. Thus, although drugs and advanced airways are still included among ALS

interventions, they are of secondary importance to high-quality, uninterrupted chest compressions and early defibrillation.

Shockable rhythms (VF/VT)

The first monitored rhythm is VF/VT in approximately 25% of cardiac arrests, both in- or out-of-hospital. 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.

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 (Figure 6.2).
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.
8. Once the defibrillator is charged, tell the rescuer doing the chest compressions to "stand clear"; when clear, give the shock (Figure 6.3).
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.

Adult Advanced Life Support

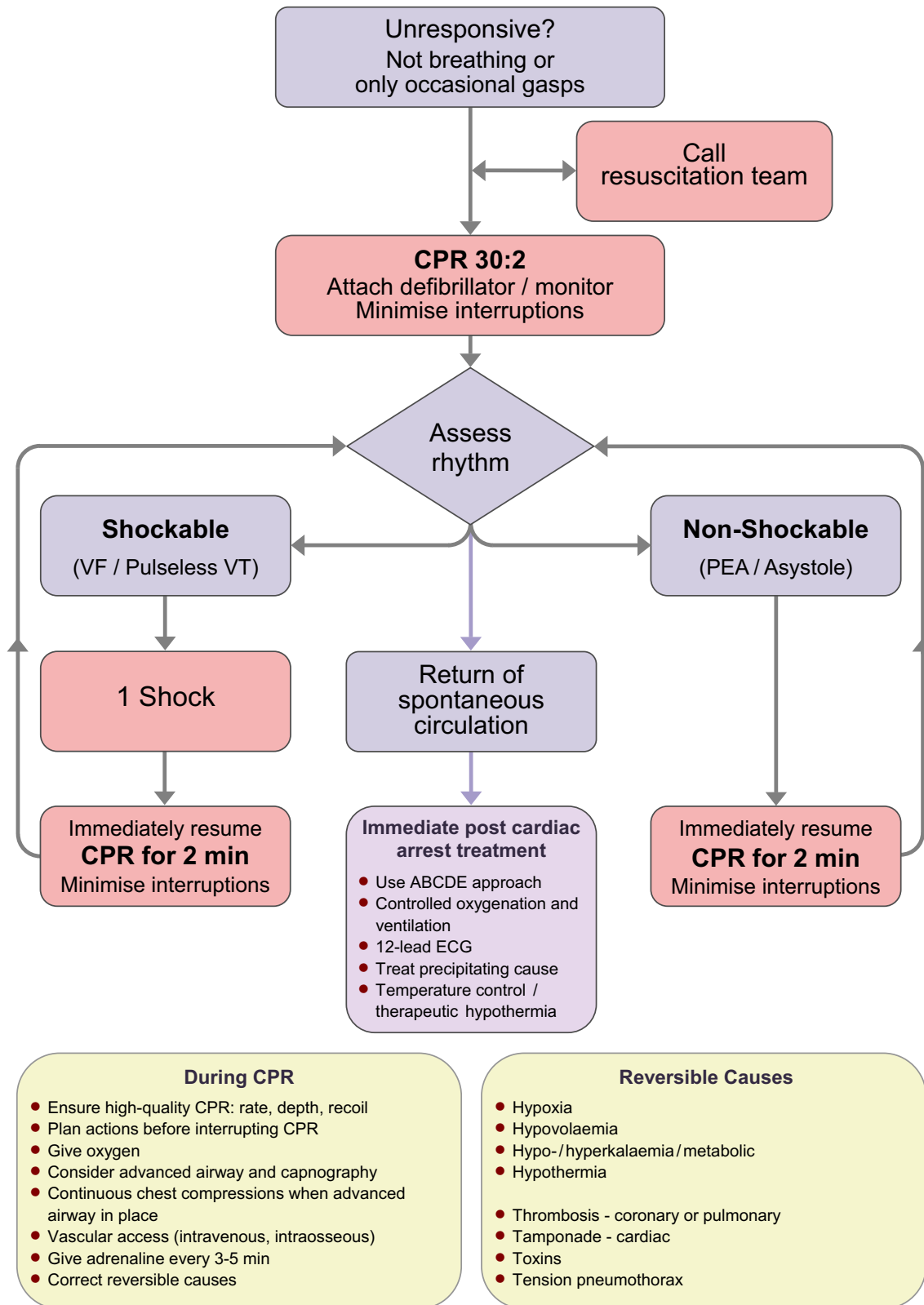


Figure 6.1 Adult advanced life support algorithm

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).



Figure 6.2 Continuing chest compressions during charging with a manual defibrillator

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

- Check a central pulse and end-tidal (ETCO₂) 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, ideally, should not exceed 5 s). Longer interruptions to chest compressions reduce the chance of a shock restoring a spontaneous circulation.

Chest compressions are resumed immediately after a shock without checking the rhythm or a pulse because



Figure 6.3 Shock delivery

even if the defibrillation attempt is successful in restoring a perfusing rhythm, it is very rare for a pulse to be palpable immediately after defibrillation and the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored. If a perfusing rhythm has been restored, giving chest compressions does not increase the chance of VF recurring. In the presence of post-shock asystole chest compressions may usefully induce VF.

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.

The first dose of adrenaline is given immediately after delivery of the third shock; amiodarone 300 mg may also be given after the third shock. Do not stop CPR to check the rhythm before giving drugs unless there are clear signs of ROSC.

Subsequent doses of adrenaline are given after alternate 2-minute loops of CPR (which equates to every 3 - 5 min) for as long as cardiac arrest persists. If VF/VT persists, or recurs, a further dose of 150 mg amiodarone may be given. 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.

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 central pulse and look for other evidence of ROSC (e.g. sudden increase in ETCO₂ or evidence of cardiac output on any invasive monitoring equipment). 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-minute period of CPR, do not interrupt chest compressions to palpate a pulse unless the patient shows signs of life suggesting ROSC. If there is any doubt about the presence 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.

It is important in shock-refractory VF/VT to check the position and contact of the defibrillation pads. The duration of any individual resuscitation attempt is a matter of clinical judgement, and should take into account the perceived prospect of a successful outcome. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing as long as the patient remains in identifiable VF/VT.

If there is doubt about whether the rhythm is asystole or very fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Very fine VF that is difficult to distinguish from asystole is 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 subsequent successful defibrillation to a perfusing rhythm. Delivering repeated shocks in an attempt to defibrillate what is thought to be very fine VF will increase myocardial injury both directly from the electric current and indirectly from the interruptions in coronary blood flow. If the rhythm is clearly VF, attempt defibrillation.

Precordial thump

A single precordial thump has a very low success rate for cardioversion of a shockable rhythm and is likely to succeed only if given within the first few seconds of the onset of a shockable rhythm. 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.

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 very rare reports of a precordial thump converting a perfusing to a non-perfusing rhythm.

Witnessed, monitored VF/VT in the cardiac catheter laboratory or after cardiac surgery

If a patient has a **witnessed** and **monitored** cardiac arrest in the catheter laboratory or early after cardiac surgery:

- Confirm cardiac arrest and shout for help.
- If the initial rhythm is VF/VT, give up to three quick successive (stacked) shocks. Start chest compressions immediately after the third shock and continue CPR for 2 min. With respect to the ALS algorithm, these three quick, successive shocks are regarded as the first shock.

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. There are no data supporting a three-shock strategy in any of these circumstances, but it is unlikely that chest compressions will improve the already very high chance of ROSC when defibrillation occurs early in the electrical phase, immediately after onset of VF.

Non-shockable rhythms (PEA and asystole)

Pulseless electrical activity (PEA) is defined as organised cardiac electrical activity in the absence of any palpable pulses. 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 (see below). Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated quickly and effectively.

Asystole is the absence of electrical activity on the ECG trace. During CPR, ensure the ECG pads are attached to the chest and the correct monitoring mode is selected. Ensure the gain setting is appropriate. Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because in this situation ventricular standstill may be treated effectively by cardiac pacing. Attempts to pace true asystole are unlikely to be successful.

Treatment for PEA and asystole

- Start CPR 30:2.
- Give adrenaline 1 mg IV / IO 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 organised electrical activity is seen, check for a pulse and/or signs of life:*
 - If a pulse and/or signs of life are present, start post resuscitation care.
 - If no pulse and/or no signs of life are present (PEA):
 - Continue CPR.
 - Recheck the rhythm after 2 min and proceed accordingly.
 - Give further adrenaline 1 mg IV every 3 - 5 min (during alternate 2-min loops of CPR).
 - *If VF/VT at rhythm check, change to shockable side of algorithm.*
 - *If asystole or an agonal rhythm is seen at rhythm check:*
 - Continue CPR.
 - Recheck the rhythm after 2 min and proceed accordingly.
 - Give further adrenaline 1 mg IV every 3 - 5 min (during alternate 2-min loops of CPR).

During CPR

During the treatment of persistent VF/VT or PEA / asystole, emphasis is placed on good quality chest compressions between defibrillation attempts, recognising and treating reversible causes (4 Hs and 4 Ts), obtaining a secure airway, and vascular access.

During CPR with a 30:2 ratio, the underlying rhythm may be seen clearly on the monitor as compressions are paused to enable ventilation. If VF is seen during this brief pause (whether on the shockable or non-shockable side of the algorithm), do not attempt defibrillation at this stage; instead, continue with CPR until the 2-minute period is completed. Knowing that the rhythm is VF, the team should be fully prepared to deliver a shock with minimal delay at the end of the 2-minute period of CPR.

Maintain high quality, uninterrupted chest compressions

The quality of chest compressions and ventilations are important determinants of outcome, yet are frequently performed poorly by healthcare professionals. Avoid interruptions in chest compressions because pauses cause coronary perfusion pressure to decrease substantially. Ensure compressions are of adequate depth (5 - 6 cm) and rate (100 - 120 min⁻¹), and release pressure from the chest completely between compressions.

As soon as the airway is secured, continue chest compressions without pausing during ventilation. To reduce fatigue, change the individual undertaking compressions every 2 min or earlier if necessary. Use CPR feedback / prompt devices when available. Be aware that some devices may fail to compensate for compression of the underlying mattress during CPR on a bed when providing feedback.

Airway and ventilation

A bag-mask, or preferably, a supraglottic airway device (e.g. laryngeal mask airway, i-gel) should be used in the absence of personnel skilled in tracheal intubation (Chapter 7). Once a supraglottic airway device has been inserted, attempt to deliver continuous chest compressions, uninterrupted during ventilation. Ventilate the lungs at 10 breaths min⁻¹; do not hyperventilate the lungs. If excessive gas leakage causes inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to enable ventilation (using a compression-ventilation ratio of 30:2).

No studies have shown that tracheal intubation increases survival after cardiac arrest. Incorrect placement of the tracheal tube is common in cardiac arrest if intubation is attempted by unskilled personnel. Tracheal intubation should be attempted **only** if the healthcare provider is properly trained and has regular, ongoing experience with the technique. Avoid stopping chest compressions during laryngoscopy and intubation; if necessary, a brief pause in chest compressions may be required as the tube is passed between the vocal cords, but this pause should not exceed 10 s. Alternatively, to avoid any interruptions in chest compressions, the intubation attempt may be deferred until after ROSC. After intubation, confirm correct tube position, ideally with waveform capnography, and secure it adequately. Once the patient's trachea has been intubated, continue chest compressions, at a rate of 100 - 120 min⁻¹ without pausing during ventilation.

Vascular access

Obtain intravenous access if this has not been done already. Although peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central venous catheter compared with a peripheral cannula, insertion of a central venous catheter requires interruption of CPR and is associated with several

potential complications. Peripheral venous cannulation is quicker, easier, and safer. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid and elevation of the extremity for 10 - 20 s to facilitate drug delivery to the central circulation. If intravenous access cannot be established within the first 2 min of resuscitation, consider gaining intraosseous (IO) access (Figure 6.4). Tibial and humeral sites are readily accessible and provide equal flows for fluids. Intraosseous delivery of resuscitation drugs will achieve adequate plasma concentrations. Several studies indicate that IO access is safe and effective for fluid resuscitation and drug delivery.



Figure 6.4 Intraosseous device

Reversible causes

Potential causes or aggravating factors for which specific treatment exists must be considered 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 (Figure 6.5). More details on many of these conditions are covered in Chapter 12.

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- Hypothermia

- Tension pneumothorax
- Tamponade
- Toxins
- Thrombosis (pulmonary embolism or coronary thrombosis)

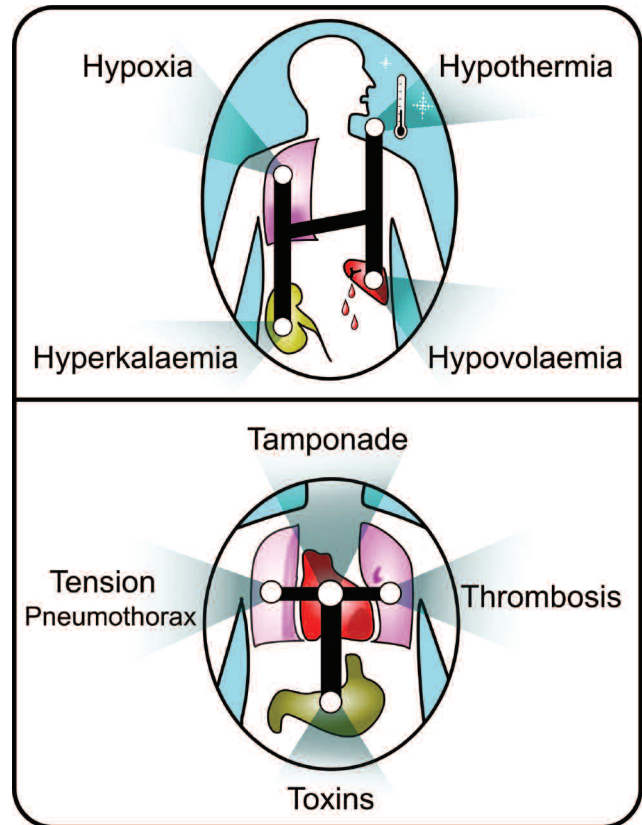


Figure 6.5 The four Hs and four Ts

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 in Chapter 7, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by **hypovolaemia** is due usually to severe haemorrhage. Evidence of haemorrhage may be obvious, e.g. trauma (Chapter 12), or occult e.g. gastrointestinal bleeding, or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with fluid and blood, coupled with urgent surgery to stop the haemorrhage.

Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history e.g. renal failure (Chapter 12).

A 12-lead ECG may show suggestive features. Intravenous calcium chloride is indicated in the presence of hyperkalaemia, hypocalcaemia, and calcium channel-blocker overdose.

Suspect **hypothermia** in any drowning incident (Chapter 12); 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 thoracostomy or needle thoracocentesis and then insert a chest drain.

Cardiac **tamponade** is difficult to diagnose because the typical signs of distended neck veins and hypotension cannot be assessed during cardiac arrest. Cardiac arrest after penetrating chest trauma or after cardiac surgery should raise strong suspicion of tamponade - the need for needle pericardiocentesis or resuscitative thoracotomy should be considered in this setting (Chapter 12).

In the absence of a specific history of accidental or deliberate ingestion, poisoning by therapeutic or **toxic** substances may be difficult to detect but in some cases may be revealed later by laboratory investigations (Chapter 12). Where available, the appropriate antidotes should be used but most often the required treatment is supportive. The commonest cause of **thromboembolic** or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause cardiac arrest consider giving a thrombolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60 - 90 min before termination of resuscitation attempts.

Use of ultrasound during advanced life support

In skilled hands, ultrasound can be useful for the detection of potentially reversible causes of cardiac arrest (e.g. cardiac tamponade, pulmonary embolism, ischaemia (regional wall motion abnormality), aortic dissection, hypovolaemia, pneumothorax). The integration of ultrasound into advanced life support requires considerable training if interruptions to chest compressions are to be minimised. A sub-xiphoid probe position is recommended (Figure 6.6). 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. The Focused Echocardiography Extended Life Support Course (FEEL-UK) provides a valuable introduction to echocardiography in this setting.



Figure 6.6 Use of ultrasound during advanced life support

Signs of life

If signs of life (such as regular respiratory effort, movement) or readings from patient monitors compatible with ROSC (e.g. sudden increase in exhaled carbon dioxide or arterial blood pressure waveform) appear during CPR, stop CPR briefly and check the monitor. If an organised rhythm is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care and/or treatment of peri-arrest arrhythmias if appropriate. If no pulse is present, continue CPR. The use of waveform capnography may enable ROSC to be detected without pausing chest compressions. A significant increase in ETCO₂ during CPR may be seen when ROSC occurs.

Discontinuing resuscitation and diagnosing death

If attempts at obtaining ROSC are unsuccessful the cardiac arrest team leader should discuss stopping CPR with the resuscitation team. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC.

After stopping CPR, observe the patient for a minimum of 5 min before confirming death. The absence of mechanical cardiac function is normally confirmed using a combination of the following:

- absence of a central pulse on palpation;
- absence of heart sounds on auscultation.

One or more of the following can supplement these criteria:

- asystole on a continuous ECG display;
- absence of pulsatile flow using direct intra-arterial pressure monitoring;
- absence of contractile activity using echocardiography.

Any return of cardiac or respiratory activity during this period of observation should prompt a further 5 min observation from the next point of cardiorespiratory arrest. After 5 min of continued cardiorespiratory arrest, the absence of the pupillary responses to light, of the corneal reflexes, and of any motor response to supra-orbital pressure should be confirmed. The time of death is recorded as the time at which these criteria are fulfilled.

Key learning points

- The ALS algorithm provides a framework for the standardised resuscitation of all adult patients in cardiac arrest.
- The delivery of high quality chest compression with minimal interruptions and avoidance of hyperventilation are important determinants of outcome.
- Treatment depends on the underlying rhythm.
- Look for reversible causes and, if present, treat early.
- Whenever possible, secure the airway early to enable continuous chest compressions.

Further reading

Academy of Medical Royal Colleges. A code of practice for the diagnosis and confirmation of death. 2008. <http://www.aomrc.org.uk>

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